ATHERO UPDATE

November 2025

An Official Publication of
Indian Society for Atherosclerosis Research
Delhi Chapter

endless process of science...

With 50+ years of expertise, we provide High-Quality Chemicals, Reagents, Solvents, and Culture Media for Research, Diagnostics, and several Industries. Our wide portfolio ensures Purity, Reliability, and Innovation — empowering Scientists and Researchers worldwide.

Our Vision:
To be the gold standard & preferred choice in every scientific lab.

Our Infrastructure & Global Footprint:

- **4** State-of-the Art Production Facilities
- **5** Distribution Warehouses
- **9000+** Products
- **350** Distributors across India
- **5** Quality Certifications
- **15** Trademarks
- **20,000** Stock-Keeping Units (SKU's)
- **130** Distributors Globally

Our Clientele:

Agriculture & Tissue Culture | Diagnostics | FMCG | Hospitals | Nano science & Renewable Energy | Oil & Gas | Pharmaceuticals | Semiconductor | Universities & Institutes | Research & Testing Labs

Connect with us:

Sisco Research Laboratories
marketing@srlchem.com
+91 22-42685800

Certifications:

CONTENTS

EDITORIAL BOARD

Introduction to the Indian Society for Atherosclerosis Research – Delhi Chapter

The Indian Society for Atherosclerosis Research (ISAR) was established in 1987 with the primary objective of advancing knowledge related to the causes, prevention, and treatment of atherosclerosis, as well as cardiovascular and cerebrovascular diseases in the Indian population. ISAR strives to promote both basic and clinical research in these domains and to foster collaboration among scientists and clinicians dedicated to understanding this multifactorial disease.

Like its parent body, the ISAR – Delhi Chapter (ISAR-DC) is a multidisciplinary scientific society, comprising biochemists, molecular biologists, pathologists, cardiologists, epidemiologists, pharmacologists, physicians, and vascular surgeons.

The Delhi Chapter of ISAR (www.delhichapterisar.co.in) is a state chapter of ISAR

(www.isar.co.in), which itself is a member society of the International Atherosclerosis Society (IAS) (www.athero.org) based in Italy. Life Membership of DC-ISAR and ISAR automatically entitles one to membership of the IAS, with all associated professional privileges.

ISAR warmly welcomes researchers and clinicians engaged in cardiovascular and metabolic research to join this shared vision—to collectively understand and manage the enigma of atherosclerosis and to contribute to reducing its burden in India.

Delhi Chapter of ISAR

The Delhi Chapter of ISAR was officially formed with the approval of the National Executive Committee during its meeting held on 25 November 2014 at ISARCON 2014. This milestone marked the establishment of the first-ever state chapter of ISAR in the Delhi–NCR region.

The formation of the Delhi Chapter represented not only organizational growth but also a decisive step toward decentralizing scientific outreach—enabling focused and localized initiatives in research, education, and public awareness on atherosclerosis.

Delhi–NCR, being one of India's leading centers of medical research, healthcare, and academia, provided the ideal environment for setting up this pioneering chapter. It offers a rich ecosystem of researchers, clinicians, and scholars who collectively contribute to the study, prevention, and management of atherosclerosis—a disease that continues to pose a major global and national health challenge.

Since its inception, ISAR-DC has functioned as an active and vibrant arm of the national society, creating opportunities for knowledge exchange, interdisciplinary collaboration, and translation of new scientific insights into clinical practice. Through its conferences, workshops, and publications, the chapter remains dedicated to advancing scientific understanding and improving cardiovascular health in the community.

PROGRAM SCHEDULE

S. No	Time	Speaker	Topic
1	9 A.M – 10 A.M	Registration	
2	10 A.M – 12 P.M	Oral Presentation	
3	12 P.M – 1 P.M	Poster for Display (10 A.M. – 3 P.M) & Poster Evaluation (12 P.M – 1 P.M)	
4	1 P.M. – 2 P.M.	Lunch	
5	2 P.M. – 2.40 P.M.	Inauguration	
6	2.40 P.M – 3 P.M	**Prof. Rajendra K Tangirala**	UNRAVELLING AGE-DIET INTERACTIONS REGULATING ATHEROSCLEROSIS SUSCEPTIBILITY: Novel Precision Nutrition Strategies for Cardiovascular Disease Prevention.
7	3 P.M – 3.20 P.M	**Dr. Safal**	CAD in the young – How early, how often and why?
8	3.20 P.M – 3.40 P.M	**Dr. J.P.S. Sawhney**	Lipoprotein (a) – Neglected risk marker for ASCVD
9	3.40 P.M – 4.10 P.M	**Panelists** **Dr. Ritu Singh** **Dr. Vijay Grover** **Dr. R.K. Nath** **Dr. Jagriti Bhatia** **Dr. Ajay Chauhan** **Dr. Anshita Aggarwal**	Panel Discussion Topic Stress, Speed and Sudden Death: The modern Epidemic of Early CAD **Moderator: Dr. Piyush Jain**
10	4.10 P.M – 4.40 P.M	Valedictory & Vote of Thanks	
11	4.40 P.M	High Tea	

Message from Chief Guest

It gives me immense pleasure to be a part of the Annual Conference of the Delhi Chapter of the Indian Society for Atherosclerosis Research (ISAR-DCCON 2025). Over the years, ISAR has remained steadfast in its commitment to advancing understanding and management of atherosclerosis and related cardiovascular diseases.

The theme this year—"*Changing Face of Coronary Artery Disease: The Young at Risk*"—is both timely and thought-provoking. The rising incidence of premature coronary artery disease in our younger population is an alarming trend that demands renewed attention from clinicians, researchers, and policymakers alike. Beyond the conventional risk factors, our evolving lifestyle, stress burden, and environmental influences have created new challenges in prevention and early detection.

I congratulate the organizers for bringing together experts from diverse disciplines to deliberate on this crucial issue. Such academic exchanges not only enrich scientific knowledge but also inspire collaborative strategies aimed at protecting the health of future generations.

I extend my best wishes for the success of ISAR-DCCON 2025 and hope that the deliberations will lead to impactful ideas and actionable outcomes.

Dir Prof (Dr) Ashok Kumar M.D, PhD

Director
ABVIMS-Dr RML Hospital
New Delhi

DHARMAVIRA HEART CENTRE

Sir Ganga Ram Hospital

Sir Ganga Ram Hospital Marg, New Delhi - 110060
TEL.: 011-42254000, 25750000, Fax : 011-25861002

Prof. S.C. Manchanda
M.D. (Med) D.M. (Cardiology)
Senior Consultant Cardiologist
(Formerly Head, Department of Cardiology, AIIMS)
Reg No. DMC 31430
PADAM SHREE Awardee
M : 9873695460

Dr. Manchanda's
The Caring Touch
Cardiac Dental And Eye Care
R 721 (Basement) New Rajinder Nagar, ND 60

Message from Guest of Honor

It is a matter of great pleasure and privilege to be associated with the Annual Conference of the Delhi Chapter of the Indian Society for Atherosclerosis Research (ISAR-DCCON 2025). The Society's sustained efforts in fostering scientific exchange and promoting research in cardiovascular medicine are truly commendable.

This year's theme, *"Changing Face of Coronary Artery Disease – The Young at Risk,"* highlights an emerging and deeply concerning trend. The increasing burden of coronary artery disease among younger individuals reflects not only genetic and metabolic factors but also the profound impact of changing lifestyles, stress, and environmental influences. It is imperative that as a medical fraternity, we focus on early risk assessment, preventive strategies, and community awareness to curb this growing epidemic.

I congratulate the organizing committee for curating a program that brings together experts, clinicians, and researchers to deliberate on this important issue. I am confident that the discussions and insights emerging from this conference will pave the way for innovative approaches in prevention and management of atherosclerotic diseases in the young.

My best wishes for the success of this CME and for many more years of meaningful academic engagement by the Delhi Chapter of ISAR.

Dr S.C Manchanda M.D, D.M

Former Professor and Head Cardiology AIIMS
Retired Consultant Cardiology, SGRH

Consultant by APPOINTMENT ONLY
Sir Ganga Ram Hospital : Tel : 42254000
Res. Clinic : R-721, New Rajinder Nagar, New Delhi - 110060 Tel. : 011-35679127 , Mobile : 9873695460
email : doctormanchanda@yahoo.com
email - tcteyecare@gmail.com • Mob No. 9560461359

Message from President
Indian Society for Atherosclerosis Research, Delhi Chapter

Dear ISAR Delhi Chapter Members,

Greetings to all members and well-wishers of the ISAR Delhi Chapter. Our society continues to provide a vibrant platform for clinicians, scientists, and students dedicated to advancing knowledge and improving patient care in atherosclerosis and related cardiovascular disorders.

I am proud to share that the Delhi Chapter has completed **ten years of dedicated service**. This milestone is a tribute to our founding members **Dr Shridhar Dwivedi, Dr D K Srivastava, Dr Jayashree Bhattacharjee, Dr Ritu Singh, and Dr Suman Bala Sharma** and to all members who have supported our mission.

This year, we celebrate our **10th Annual Conference**, organized by **Dr Parul Goyal**, Vice President, ISAR Delhi Chapter. I congratulate her and the entire team for their hard work. The conference promises to be an enriching platform for academicians, clinicians, and researchers, while also fostering opportunities for young investigators to learn from experts.

I warmly welcome our new Life and Annual members and encourage everyone to actively participate in the Society's activities. Special thanks to **Dr Harsh Vardhan Singh** and **Dr Aditi Singh** for successfully updating our website and to the Executive team for their continued dedication.

Our Souvenir cum newsletter, **Athero Update**, continues to showcase original mini-reviews and updates on Society activities. My gratitude to **Dr Shridhar Dwivedi, Dr Rachna Agrawal, Dr Harsh Vardhan Singh**, and the editorial team for their efforts.

I invite all members to collaborate and contribute towards the growth as well as strengthening the impact of ISAR Delhi Chapter in the field of atherosclerosis research.

With best wishes,

(Dr Jagriti Bhatia)
President, ISAR Delhi Chapter
Professor
Department of Pharmacology,
AIIMS, New Delhi

Message from the President-Elect

Dear Delegates,

It gives me great pleasure to invite you to the Annual Conference of the Delhi Chapter of the Indian Society for Atherosclerosis Research – ISAR DCCON 2025, organized by the Department of Biochemistry, ABVIMS–Dr. RML Hospital, New Delhi, on Saturday, 1st November 2025 at CSOI, KG Marg, New Delhi.

The theme, 'Changing Face of Coronary Artery Disease – The Young at Risk', highlights an emerging concern that calls for renewed focus on early detection, prevention, and research. Upholding our legacy of academic excellence, this year's conference will feature eminent speakers, insightful panel discussions, and opportunities for young professionals to present their research.

We are heartened by the growing participation and enthusiasm, reflected in the increasing number of scientific submissions and contributions to the DC-ISAR Newsletter & Souvenir. Your continued engagement strengthens our mission to advance knowledge and collaboration in cardiovascular research.

I warmly welcome you all to ISAR DCCON 2025 and look forward to your active participation in making this event a resounding success.

Warm regards,

Sbhargava

(Dr. Seema Bhargava)
President-Elect, Delhi Chapter – ISAR
Chairperson and Senior Consultant,
Department of Biochemistry & Professor, GRIPMER
Sir Ganga Ram Hospital, New Delhi, India

Message from the Secretary

Dear Dignitaries, Senior Colleagues, Delegates, and Students,

It gives me immense pleasure to welcome you all to the Annual Conference – ISAR DCCON 2025, organized by the Department of Biochemistry, ABVIMS–Dr. RML Hospital, under the aegis of the Indian Society of Atherosclerosis Research (ISAR) – Delhi Chapter.

This year's theme, "Changing Face of Coronary Artery Disease – The Young at Risk," reflects a growing public health concern and the urgent need to understand the evolving epidemiology, molecular mechanisms, and preventive strategies for cardiovascular disease among the younger population.

The conference unites leading experts, academicians, and clinicians to share insights and experiences, while providing a valuable platform for young investigators and students to present their work and engage in meaningful academic exchange.

I extend my heartfelt gratitude to all dignitaries, speakers, and participants for their valuable contributions and enthusiastic support in making this event possible. Special appreciation goes to our organizing committee and sponsors for their dedicated efforts. To our students and young colleagues – your curiosity and commitment continue to inspire the spirit of ISAR DCCON and drive the pursuit of excellence in research and innovation.

Wishing you all a scientifically stimulating, productive, and memorable conference.

With warm regards,

(Dr. Harsh Vardhan Singh)
Secretary, DC-ISAR
Editor, Atheros Update
Senior Biochemist,
North DMC Medical College & Hindu Rao Hospital,
Delhi

Message From Treasurer:

Dear Delegates

It gives me great pleasure to welcome all delegates, speakers, and participants to the Annual Conference of the Delhi Chapter of the Indian Society for Atherosclerosis Research (ISAR), being organized by Department of Biochemistry, Atal Bihari Vajpayee Institute of Medical Sciences -Dr RML Hospital, on 1st November 2025.

The theme, "Changing Face of Coronary Artery Disease- The Young at Risk", focuses on a very important transformation seen recently in epidemiology of Coronary Artery Disease. Once regarded as a disease of middle and older age, it is now increasingly affecting younger populations — requiring a relook at its risks, prevention, and management.

This conference aims to encourage collaborative dialogue among scientists, clinicians, and public health professionals working together to address the complex challenges of atherosclerosis and cardiovascular health.

I extend heartfelt appreciation to the organizing committee, dedicated members, speakers and participants, whose efforts have made this event possible. May this conference inspire new ideas, foster meaningful collaborations that improve patient outcomes and promote heart-healthy living.

Wishing all participants, a productive and enriching experience at ISAR–DCCON 2025.As a treasurer of ISAR-DC, I feel privileged to welcome you all to this academic feast.

Looking forward to a fruitful interaction with all of you.

Regards

Rajeev Goyal

(Dr. Rajeev Goyal)
Treasurer, DC-ISAR
Professor, Dept. of Biochemistry,
Lady Hardinge Medical College and associated Hospital,
Delhi

Message From Organising Secretary:

Dear Members,

It gives me immense pleasure to welcome you all to the Annual Conference of the Delhi Chapter of ISAR – ISAR DCCON 2025. This year's theme, "Changing Face of Coronary Artery Disease – The Young at Risk," highlights our growing concern over the rising incidence of cardiac disease among the younger population. It also reflects our shared commitment to address this critical health challenge through enhanced knowledge, greater awareness, and continued advancement in medical science and practice.

Once considered a disease of the middle-aged and elderly, coronary artery disease is increasingly affecting younger populations. This shift calls for renewed attention to early diagnosis, lifestyle modification, preventive cardiology, and innovative treatment strategies. Through insightful scientific sessions, expert deliberations, and multidisciplinary panel discussion, this meet aims to illuminate the evolving landscape of cardiovascular health and inspire actionable change in clinical practice. We are privileged to have with us renowned faculty and enthusiastic participants who will make this platform vibrant and inspiring.

I extend my heartfelt thanks to our distinguished speakers ,esteemed faculty, delegates, and students for their enthusiastic participation and valuable contributions. My sincere appreciation goes to the Delhi Chapter of ISAR for their constant guidance and support, and to the entire organizing team whose dedication and hard work made this conference possible.

I wish all delegates a truly enriching experience of learning and meaningful exchanges. May this conference further strengthen our shared commitment to advancing cardiovascular well-being.

Warm regards,

(Dr. Parul Goyal)
Organizing Secretary ISAR DCCON 2025 & Vice President DC-ISAR
Director Professor , Biochemistry, ABVIMS –Dr RML Hospital

Message from the Patron & Editor-in-Chief

It gives me immense honour to extend my warmest greetings to all participants, delegates, and faculty members on the occasion of ISAR-DCCON 2025 and the release of our newsletter-cum-souvenir, "Athero Update."

This publication reflects the theme of this conference that is , coronary artery disease and its academic spirit of our team , its dedication, and collective effort of our society to promote awareness, research, and collaboration in the field of atherosclerosis and cardiovascular health. I am also delighted to acknowledge the sincere and commendable efforts of the entire editorial team — Dr. Rachana, Dr. Harsh Vardhan, Dr. Aditi, and Dr. Sukanya — along with all members of the Editorial Board, who have worked tirelessly to bring out this excellent issue. Their commitment, creativity, and teamwork have made "Athero Update" both informative and inspiring.

As we gather for ISAR-DCCON 2025, I am confident that the scientific deliberations, discussions, and shared experiences will pave the way for new insights and advancements in preventive and translational cardiology.

My heartfelt best wishes to the President of DC- ISAR Dr Jagriti , Organizing Secretary Dr Parul Goyal and all participants for a highly successful and enriching conference.

With warm regards and best wishes

Dr. Shridhar Dwivedi
Patron & Editor-in-Chief, ISAR-DC

Life Time Achievement Award: Dr. Anita Khalil
DC-ISAR Life time Achievement Awards 2025

Dr. Anita Khalil is a distinguished Consultant Pediatrician and Pediatric Cardiologist at The Heart Centre, with a remarkable career dedicated to advancing pediatric healthcare and cardiology in India.

A graduate of the All India Institute of Medical Sciences (AIIMS) with an MBBS (1965), Dr. Khalil pursued her MD in Pediatrics from Maulana Azad Medical College (MAMC) in 1974. Her academic foundation from these premier institutions set the stage for a lifelong commitment to child health, research, and medical education.

Dr. Khalil's contributions to pediatric cardiology are both pioneering and transformative. She is a founder member of the Cardiology Chapter of the Indian Academy of Pediatrics (IAP) and later served as its Chairperson (1997–2003). Under her leadership, she established the Section on Pediatric Cardiology at MAMC, creating a platform for specialized training and research in the field.

As a Fellow of the Indian Academy of Pediatrics (FIAP) and Life Member of several prestigious organizations—including the Indian Society of Atherosclerosis Research, Pediatric Cardiac Society of India (PCSI), Indian Academy of Echocardiography, and SNEHA (Study of Natal Effects of Heart Ailments)—Dr. Khalil has played a key role in promoting collaborative learning and research.

Her academic curiosity and dedication to evidence-based practice were exemplified in her involvement with the New Delhi Birth Cohort (NDBC), where she conducted extensive studies on low birth weight infants enrolled between 1969 and 1987. Her research extended to advanced assessments such as endothelial function, carotid intima-media thickness, and echocardiographic evaluations in over 600 cohort subjects.

Dr. Khalil is also known for her strong commitment to medical education. She regularly organized Continuing Medical Education (CME) sessions in Pediatric Cardiology every quarter, fostering a culture of continuous learning among medical professionals. These efforts culminated in the publication of "Essentials of Pediatric Cardiology" (Jaypee Brothers, 2010), a compilation of the chapter's newsletters and a valuable resource for practitioners.

Her excellence has been recognized with several honors, including the P. N. Taneja Award (1973) for best paper and the State Award by the Government of Delhi (2003) for outstanding service in the medical profession.

Over the years, Dr. Khalil has authored over 100 research papers published in reputed national and international journals, significantly enriching pediatric literature and clinical practice.

Today, Dr. Anita Khalil stands as an inspiration—a physician, researcher, educator, and leader—whose lifelong dedication has advanced pediatric cardiology in India and improved countless young lives.

Life Time Achievement Award: Dr. Nibhriti Das
DC-ISAR Life time Achievement Awards 2025

Dr. Nibhriti Das (born 1949, Lucknow) is a distinguished biochemist, immunologist, medical educationist, and research scientist of national and international repute. She earned her Ph.D. in Biochemistry (1969–1976) from Lucknow University under Professors P.S. Krishnan and G.G. Sanwal.

Her scientific journey began at the Industrial Toxicology Research Centre (ITRC), Lucknow, where she worked on the toxicological and nutritional impacts of pesticides (1976–1981). In 1982, she joined AIIMS, New Delhi, under Professor L.M. Srivastava, who introduced her to research on coronary artery disease (CAD) and atherosclerosis—fields where she later made landmark contributions. She became a Lecturer in 1984 and served AIIMS for three decades, retiring in 2014.During 1991–1994, she was part of the Arabian Gulf University, Bahrain, where she gained advanced training in problem-based learning (PBL) and later pioneered its implementation at AIIMS and other Indian medical colleges.

Her work on immune-inflammatory pathways in atherosclerosis highlighted the roles of immune complexes, complement activation, and cytokine–complement interplay, paving the way for complement-based therapeutic strategies. A dedicated teacher and mentor, Dr. Das has been core faculty at the K.L. Wig Centre for Medical Education and Technology, AIIMS, and contributed to MCI/NMC competency-based Biochemistry curriculum reforms. With over 110 publications, numerous national and international awards including the G.P. Talwar Oration Award, and extensive mentorship of postgraduate and doctoral students, her academic impact is profound.

The first Indian woman elected to the Council of the International Union of Immunological Societies, she has championed immunology education across India. Honored as Best Teacher in Biochemistry by AIIMS students, she remains active globally, recently presenting at the International Congress of Immunology, Vienna (2025).

After retirement, Dr. Nibhriti Das headed the Department of Biochemistry and Lab Sciences at Nayati Multi-Specialty Hospital, Mathura, achieving NABH accreditation and fostering a strong research culture.

Her pioneering studies on the high susceptibility of Asian Indians to CAD and hypertension explored free radical homeostasis, lipid profiles, adhesion molecules, and genetic markers. She identified low HDL and reduced nitric oxide as independent CAD risk factors and demonstrated that gene variants protective in the West (Apo-lipoproteins AI, AIV, C, E; eNOS, iNOS; paraoxonase) may confer risk in Indians—offering key insights into population-specific disease mechanisms.

Dr. Das exemplifies academic excellence and scientific leadership, shaping India's understanding of cardiovascular and autoimmune diseases while inspiring generations of medical professionals.

PREVENTING CORONARY ARTERY DISEASE IN YOUNG: CATCH THEM YOUNG

'An ounce of prevention is worth a pound of cure'- Benjamin Franklin. (1736)

Cardiovascular diseases (CVDs) continue to be the leading cause of the mortality and morbidity both worldwide including India. CVDs are estimated to cost around 200 billion Euros. Further, non-communicable diseases (NCDs) combined together account for 63% disease burden in India (CVDs - 27%, chronic respiratory – 11%, cancer – 9%, diabetes – 3%, others – 13%[1]. These are seven year old figures. They might have changed for worse. For example the prevalence of obesity the driving force for metabolic dysfunction-associated steatotic liver disease (MASLD) has increased from 3% to 9.4% in India in a span of two decades; thanks to pizza and burger[2-3].

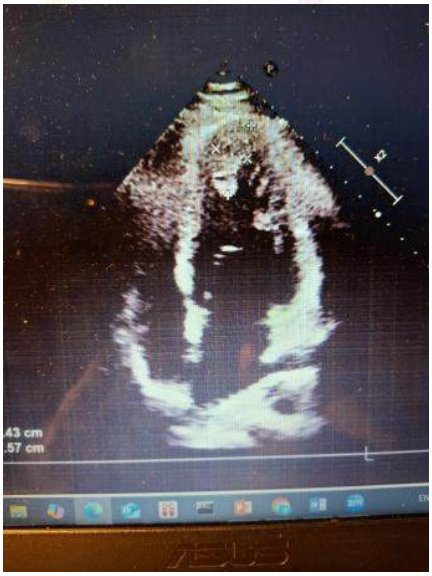
. Among entire gamut of cardiovascular diseases, it is coronary artery disease (CAD) which has emerged as an epidemic and has become a major public health problem in our country. Every other day we get to see the headings in the newspaper and in the mass media about sudden cardiac death in young age (<45) people who are at their peak of life. The economic implications of cardiovascular diseases particularly the acute coronary syndrome is colossal for a common Indian. An Indian who suffers acute coronary event has to spend some 1.57,840 INR[4]. The way is spreading fast, far and wide in India in younger population (age less than 35 years) is a matter of grave concern for health planners as it has damaging implications on the growing economy of our country. Global projections are that CVDs may be responsible for 23.6 million deaths by 2030.

It is not that it has just emerged suddenly like COVID epidemic but a growing noncommunicable epidemic which has been there in early 1970.s and slowly creeping into society on account of socioeconomic improvement , changing dietary pattern , increasing consumption of junk foods and trans-fats , living habit dominated by sedentary activity , adoption of varied forms of tobacco habits , sleep deprivation , increasing tension , stress and strain ,over use of smartphone to addictive levels ,etc. These traits are popularly known as cardiovascular risk factors. Over the years the prevalence of these risk factors among the society has increased to great levels in the younger people on account of paradigm shift in our living and cultural shift in cities and metropolis as enumerated above. This change in lifestyle and possibly some genetics /epigenetic causes has given rise to present epidemic of CAD in our country.

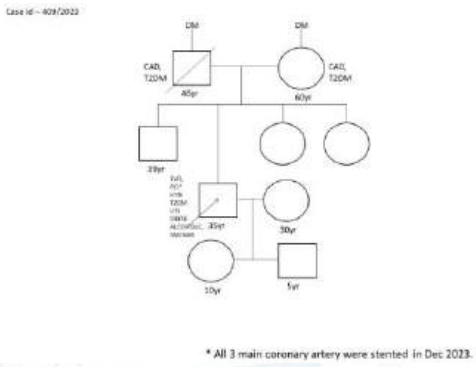
In order to explain our above hypothesis, let us go through some living case examples:

Case 1. A 32 year old young university student presented with history of recent recurrent chest pain. He already suffered two episodes of acute chest pain at age 24 and 30 for which he underwent percutaneous intervention(PCI) . Stents were put in for the same. He was chronic heavy smoker like chimney and had not left the smoking despite previous PCI in left ventricle. He also used to take alcohol very often and was under severe mental stress. There was no obvious history of CAD in family. Echo revealed a large

apical thrombus (Figure -1). This case amply explains the dictum – smoking ,alcohol and stress , cause for cardiac distress .



Case 2 : A - 35-yr old , gentleman ,presented to us with acute chest pain and profuse sweating . He was a chain smoker, gutkha chewer (Look at his teeth, Fig.3) and used to drink very often. He was a known case of diabetes (T2DM) and had a very strong family history of T2DM and CAD (Fig. 2 Pedigree) . His angiogram revealed triple vessel disease for which three stents were put in



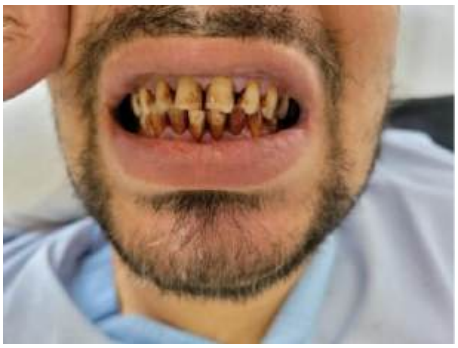
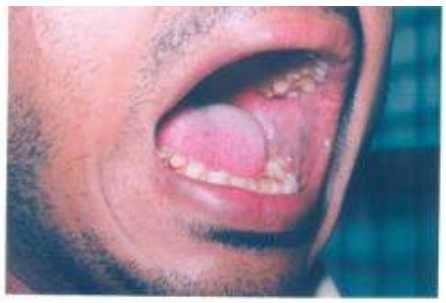


Fig. 3: Case 2 -His most teeth are stained with gutkha . Premature greying is also so evident

Case 3.

A 22 -year- old boy collapsed and died suddenly while doing treadmill in gym.

Case -4. A -35 -year old three wheeler driver developed acute chest pain and sense of impending death. ECG showed evidence of acute extensive myocardial infarction. He was achronic gutkha chewerand beedi smoker. Oral examination revealed submucosal fibrosis in left buccal ucosa (Fig - 5). He thus suffered double whammy -acute coronary syndrome as well as premalignant oral lesion at such a young age on account of his tobacco habit. Further, though he was just 35 year old but his vascular was assessed to be 55.



Case -5. A- 40- year old young man admitted in ICCU on second day of his acute myocardial infarction recovery, he was found to be busy with three mobiles (Fig.6). The intense anxiety and protuberant tummy were very evident. He was a chronic heavy smoker and under severe business stress. This was his second episode of ACS at interval of 3 months.

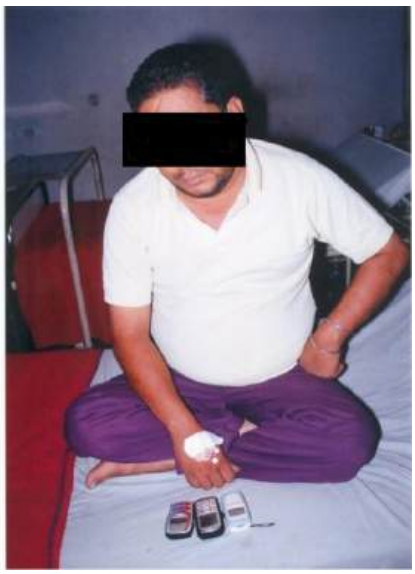


Fig.6 : A -40-year old young man admitted in ICCU for ACS found to have three mobiles. He was a chronic heavy smoker too.

Positive Side of the Healthy Story:

On the other hand there are umpteen number of examples like Marathon runner Fauza Singh living up to 112 years, Rambai, 105-Yr old from Haryana created history by breaking a 100m race record in just 45.40 seconds. Prof. (Dr) Radhakrishna Rao living up to 104 years ,who got Nobel in Statistics at age 96, famous Hindi writer and poet Prof Ram Darash Mishra living and contributing to society at age 100, 92-yr old E Sreedharan, the Metro man of India, Prof JS Guleria (Medicine), Prof SMTuli (Orthopedics) , Prof Neera Agarwal (OBG) , Prof M Khalilullah (Cardiology) all above ninety and still active giving their best to society . One thing common to them all is their healthy lifestyle devoid of tobacco, regular exercise /yoga, dominantly plant based diet, no negativity. Another legendary example Swami Sivananda who lived for 110+. He credited his longevity to a disciplined lifestyle. "I eat simple food—only boiled vegetables without fat, spices, or rice," he told AFP after completing a yoga session in Kolkata. He shared three principles for living a long life: waking up early, eating in moderation, and practicing self-control. (Economic Times ,25th Jan2025). There is much to learn from their lifestyle.

Our Experience: In a recent study carried out at our centre in thirty consecutive angiographically proven acute coronary syndrome (ACS) patients divided in two groups, those belonging to age≤45 (Group A) and those aged 46 years and above (Group B); We noted increased screen time in younger group of patients compared to group B (Table-1). Our youngest patient, a 29-year-old malehad a screen time of 12 hours per day. He was a smoker, obese, with a stressful lifestyle, and reported sleep deprivation. He had a family history of coronary artery disease (CAD). His ECG showed ST elevation, and his echocardiogram revealed an ejection fraction of 45%, along with hypokinetic segments. He was diagnosed with single vessel disease (SVD) and underwent percutaneous coronary intervention (PCI).[5]

Table 1: Screen time, smoking, alcohol and obesity between the <45 years and >=45 years age ACS patients

	Group A	**Group B**	
Variable	< 45 years [n=20] All male	>=45 years [n=10]	P-value
Age yrs (Mean ± SD)	40.05(5.0)	58.0(5.37]	
Screen time (hours) Median[25th to 75th percentile]	5.0[4.0 to 7.8]	3.5[2.0 to 5.8]	0.075*
Smoking /oral tobacco	13(65.0)	3(30.0)	0.122$
Alcohol	10(50.0)	3(30.0)	0.440$
Obesity	5(25.0)	0(0.0)	0.140$

Excessive screen time more than 6 hours a day has recently been incriminated as a strong risk factor for premature CAD .[6-7]

Preventive strategies:

Ever since Dean Ornish convincingly demonstrated that CAD is reversible by simple lifestyle measures there has been umpteen number of papers proving his contention in a convincing matter. [8] Everyone

now agrees that one needs to tackle four ominous 'Ts' - Tobacco, Tummy, Tension and Trans-fats to check the current pandemic of hypertension, obesity, diabetes, heart attack, stroke and cancer. [9-11]. Young students be educated and motivated about these risk factors through school Health Lectures, an initiative which we have undertaken since 2012 and found to be effective in creating awareness about healthy lifestyle.[12-13]. Though not related directly with CAD mouth cancer is primarily due to oral tobacco/gutkha ; obesity in adolescents is due to junk food /trans fats , tummy a manifestation of physical inactivity and junk food consumption and tension /stress plays an important role in causation of hypertension , heart attack and or diabetes . Government of India's estimate is trans-fatty acids are killing 5.4 lacs people a year. WHO says 4.6% of CAD deaths in India may be related to trans-fatty acid intake. Our dictum in this regard is – Eat not much oil, so that heart is not in turmoil. Comprehensively best is to avoid these four dangerous 'Ts'

Our approach in this regard is to start preventive measures right from the day of conception of child and look after mother's health quite well, putting her to a comprehensive check up for gestational diabetes, pregnancy induced hypertension, familial hypercholesterolemia, advising against any kind of tobacco consumption, betel nut or alcohol, and necessity for a calm and stress free environment for the betterment of foetus[14]. Once the child is born the parents and elders in family should present to him what a ideal healthy lifestyle is by leading an ideal healthy lifestyle as enunciated in Srimadbhawad Gita -(*युक्ताहारविहारस्य युक्तचेष्टस्य कर्मसु। युक्तस्वप्नावबोधस्य योगो भवति दुःखहा*॥ (श्रीमद्भगवद्गीता ६.१७) meaning ' eating appropriate diet , doing proper exercise , performing one's own allocated duties properly and going to bed and getting up at appropriate time is yoga way of life which leads you to perfect health[15]. If one integrates this definition of healthy lifestyle as enunciated in Gita with current concepts of American Heart Association (AHA) recommendations it gels so well. The AHA recommends: Be active, Keep healthy weight, Learn about cholesterol, Don't smoke or take Smokeless tobacco, Eat a Heart healthy diet - Plant based diet, best healthy diet, Keep BP healthy, Keep track of your lipids, Learn about DM/ sugar (Fig.7)[16]

Seven countries study lead by Fadness (2024) has shown that sustained change from country-specific typical dietary pattern patterns to longevity-optimized dietary changes, more feasible dietary changes, or optimized vegan dietary changes are all projected to result in substantial life expectancy gains across ages and countries. These changes included more whole grains, legumes, and nuts and less red/processed meats and sugars and sugar-sweetened beverages. Current guidelines recommend diets high in fruits, vegetables, whole grains, nuts, and legumes; moderate in low-fat dairy and seafood; and low in processed food [17] . The Indian concept is that only

those food items be taken which has some medicinal items lest you get trapped with loads of medicine which have been prescribed to manage your plethora of ailments[18].

Putting the entire conceptual model of preventing CAD in young one can integrate the ancient concept of Srimadbhagwad Gita with AHA's seven simple tips very easily and overcome the problem of CAD. There is one caveat in this proposition that the Gita way of life has to be inculcated right from the conception stage of foetus. The expectant mother has to be judicious about her diet, no tobacco , no alcohol , exercise , daily routine and observing the biological clock appropriately . There may be an odd case where despite all lifestyle precautions one develops subclinical signs of CAD prematurely; starting T arjuna bark is one of the option in such cases [19-20]. In case someone develops frank signs of CAD at young age prompt corrective measures be undertaken as per standard guidelines along with healthy lifestyle measures.

Shridhar Dwivedi,

Senior Consultant Cardiologist, National Heart Institute, New Delhi

References:

1. World Health Organization. *NCD India profile 2018*. Geneva: WHO; 2018.
2. UNICEF. *Feeding Profit: How Food Environments are Failing Children*. New York: UNICEF; September 2025.
3. Zhou X, Zhong M, Hua M. Cardiovascular-kidney metabolic syndrome and MASLD: integrated medical perspectives. *Nat Rev Cardiol*. 2025;22:843.
4. Kumar A, Siddharth V, Singh SI, Narang R. Cost analysis of treating cardiovascular diseases in a super-specialty hospital. *PLoS One*. 2022;17(1):e0262190. doi:10.1371/journal.pone.0262190.
5. Dwivedi S, Sharma V, Belwal A. Smartphone addiction, stress, and sleep deprivation: emerging as new risk factors for acute coronary syndrome in young people. *MGM J Med Sci*. 2025;Jan–Mar:65–68.
6. Zhu J, Wang S, Wu Y, Gu L, Ma Y, Wang Y, et al. Smartphone addiction habit is positively associated with coronary artery disease and its severity in Chinese adults: a case-control study. *Front Cardiovasc Med*. 2024;11:1374797. doi:10.3389/fcvm.2024.1374797.
7. Zhang Y. Regular mobile phone use and incident cardiovascular diseases: mediating effects of sleep patterns, psychological distress, and neuroticism. *Can J Cardiol*. 2024;40:2156–65.
8. Ornish D. Can lifestyle changes reverse coronary artery disease? The Lifestyle Heart Trial. *Lancet*. 1990;33:624–6.
9. Dwivedi S, Iyer A. Health aphorisms. *Heart News*. 2024;Oct–Dec:17–18. Available from: www.nationalheartinstitute.com
10. Dwivedi S. *Health Aphorisms (स्वास्थ्य सुभाषित)*. 1st ed. New Delhi: Prabhat Prakashan; 2025.
11. Dwivedi S, Arshad SH. Preventing non-communicable diseases. *Heart News*. 2015;17–2. Dwivedi S. Empowering adolescents in battle against bulge, tobacco and NCDs. *Heart News*. 2021;Oct–Dec:6–7. Available from: www.nationalheartinstitute.com
12. Dwivedi S, Yadava OP, Chugh D. Novel School Health Initiative. *MGM J Med Sci*. 2018;5:195–6.

13. World Health Organization, SEARO. *GenNext – Exit NCDs: Intervention tool for prevention of NCD risk factors in school children*. New Delhi: WHO-SEARO; 2020 Jul 28.

14. Dwivedi S. Keeping heart healthy: conception to centenary. *Heart News*. 2024;Oct–Dec:6–8. Available from: www.nationalheartinstitute.com

15. Jaydayal Goyandka (Commentator). *Śrīmad Bhagavad Gītā (संस्कृत)*. Chapter 6, Shloka 17. 74th reprint. Gorakhpur: Gita Press; 2016 (Samvat 2073). p.151.

16. Hasbani NR, Lighart S, Brown MR, Heath AS, Bebo A, Asley KE, et al. American Heart Association's Life's Simple 7: Lifestyle recommendations, polygenic risk, and lifetime risk of coronary heart disease. *Circulation*. 2022;145(8):e153–e639.

17. Fadnes LT, Arjmand EJ, Økland JM, Celis-Morales C, Livingstone KM, Balakrishna R, et al. Life expectancy gains from dietary modifications: a comparative modelling study in seven countries. *Am J Clin Nutr*. 2024;120:170–7.

18. Dwivedi S. Food decides your innings. *Cardiovascular Network*. 2019;18(4):3. Available from: www.heartacademy.org

19. Dwivedi S. Terminalia arjuna – a useful drug for cardiovascular disorders. *J Ethnopharmacol*. 2007;114:114–29.

20. Kumar V, Sharma N, Saini R, Mall S, Zengin G, Sourirajan A, et al. Therapeutic potential and industrial applications of *Terminalia arjuna* bark. *J Ethnopharmacol*. 2023;310.

Extended Lipid Profile with Atherogenic Indices: A Comprehensive Approach to Cardiovascular Risk Assessment

Rachna Agarwal[1], Aastha Bansal[1], Narendra Singal[2]

[1]Department of Neurochemistry, Institute of Human Behavior and Allied Sciences (IHBAS), Delhi, India

[2]Department of Medicine, Action Balaji Hospital, New Delhi

Cardiovascular diseases remain the leading cause of global mortality, accounting for over 19 million deaths in 2021 followed by conditions viz Cancers, Chronic respiratory illnesses and Diabetes [1]. Early identification of at-risk individuals is essential for effective prevention. Traditionally, lipid assessment using total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) forms the backbone of cardiovascular risk evaluation.

However, growing evidence indicates that a significant residual risk persists despite achieving optimal LDL-C levels, particularly in patients with diabetes, obesity, metabolic syndrome, and chronic inflammatory disorders [2]. Conventional lipid testing, while valuable, does not account for particle number, size, and functionality—all of which are critical in the atherogenic process. This gap has prompted the use of extended lipid profiles and atherogenic indices, which offer a more comprehensive evaluation of lipid metabolism and cardiovascular risk.

Traditional Lipid Profile: Foundation and Limitations

The standard lipid profile includes four basic parameters:

Total Cholesterol (TC): which represents the sum of all cholesterol fractions; elevated levels are associated with coronary risk but lack specificity.

LDL-Cholesterol (LDL-C): The primary target of lipid-lowering therapy; however, LDL-C concentration may underestimate particle number when LDL is small and dense.

HDL-Cholesterol (HDL-C): Known for its anti-atherogenic role through reverse cholesterol transport; low HDL-C is an independent risk factor for CVD.

Triglycerides (TG): Elevated TG levels are common in insulin resistance and metabolic syndrome, contributing to formation of small dense LDL (sdLDL).

Despite their widespread use, these parameters often fail to detect qualitative lipoprotein abnormalities, and individuals with normal LDL-C may still exhibit elevated cardiovascular risk- a phenomenon termed “residual risk” [2-4].

Extended Lipid Profile: Beyond the Conventional Panel

The extended lipid profile introduces additional biomarkers that reflect the particle composition, number, and function of lipoproteins.

Apolipoprotein B (ApoB)

ApoB is the structural protein of all atherogenic lipoproteins including VLDL, IDL, and LDL. Each atherogenic particle contains a single ApoB molecule, making it a direct measure of atherogenic particle number. Elevated ApoB levels correlate more closely with coronary events than LDL-C, particularly in individuals with metabolic syndrome or hypertriglyceridemia [5-6]. Current guidelines recognize ApoB as a secondary therapeutic target when triglycerides are high.

Apolipoprotein A1 (ApoA1)

ApoA1 is the major protein constituent of HDL and plays a vital role in reverse cholesterol transport, antioxidant defense, and endothelial protection. Higher ApoA1 concentrations are inversely related to CVD risk.

ApoB/ApoA1 Ratio

The ApoB/ApoA1 ratio integrates the balance between atherogenic and anti-atherogenic particles, serving as a powerful composite marker of lipid risk. Studies such as the INTERHEART trial demonstrated that this ratio is a stronger predictor of myocardial infarction than any single lipid fraction [7].

Lipoprotein (a) [Lp(a)]

Lipoprotein(a) [Lp(a)] is an apoB100-containing lipoprotein with an additional apolipoprotein(a) component, conferring both atherogenic and thrombogenic properties. LP(a) is an independent causal risk factor for atherosclerotic cardiovascular diseases through mechanisms associated with these properties along with increased inflammation. Its plasma concentration is largely genetically determined and remains relatively stable throughout life. Elevated Lp(a) (>30 mg/dL) is an independent risk factor for premature coronary artery disease and stroke and it remains a risk factor for cardiovascular disease development even after effective reduction of plasma LDL-C and apoB100. [8].

Small Dense LDL (sdLDL)

sdLDL particles are subfraction of low-density lipoprotein (LDL) particles with size 15-20 but are more denser and prone to oxidative modification than larger LDL particles. They penetrate the arterial endothelium more readily, exhibit prolonged plasma residence time, and are strongly linked to plaque instability and acute coronary syndromes particularly in individuals with normocholesterolemia with underlying metabolic disorders [9].

Atherogenic Indices: Integrated Measures of Risk

Atherogenic indices are mathematical ratios derived from lipid parameters that offer an integrated overview of lipid equilibrium and predict cardiovascular risk beyond conventional measures. (Table 1)

Table 1. Atherogenic Indices [10-11]

Index	Formula	Interpretation
Atherogenic Index of Plasma (AIP)	log (TG/HDL-C)	Reflects balance between atherogenic and protective lipoproteins; >0.24 indicates high risk
Castelli's Risk Index I (CRI-I)	TC / HDL-C	Values >4.5 signify increased coronary risk.
Castelli's Risk Index II (CRI-II)	LDL-C / HDL-C	Values >3.0 suggest elevated risk of coronary heart disease.
Atherogenic Coefficient (AC)	(TC–HDL-C)/ HDL-C	Higher values indicate increased atherogenicity.
Non-HDL Cholesterol	TC – HDL-C	Encompasses all atherogenic lipoproteins (LDL, VLDL, IDL, Lp(a)); recommended as secondary target after LDL-C.

Among these, AIP has gained special attention as a surrogate marker for atherogenic dyslipidemia, endothelial dysfunction, and insulin resistance [11].

Clinical Relevance and Utility

Atherogenic indices can provide more holistic assessment of cardiovascular disease (CVD) risk beyond standard lipid panel measurements.

Superiority over standard lipids: Atherogenic indices are often more sensitive than individual lipid levels for assessing atherosclerotic risk. In many cases, these indices provide additional prognostic value even when traditional lipid parameters are within normal ranges. Correlation with lipoprotein particles: AIP is particularly valuable because it strongly correlates with the size and density of lipoprotein particles, especially small, dense low-density lipoprotein (sdLDL). An elevated AIP suggests a higher proportion of these smaller, more atherogenic particles, which are more easily oxidized and deposited in arterial walls. Improved risk prediction: Multiple meta-analyses and large cohort studies have confirmed that a higher AIP is strongly and independently associated with an increased risk of coronary artery disease (CAD), coronary artery calcification, stroke, and major adverse cardiovascular events (MACE). Extended lipid markers can uncover hidden atherogenicity in individuals with apparently normal lipid levels, particularly in diabetes, chronic kidney disease, and obesity. Thus it identifies the residual risk. Enhanced Risk Stratification: ApoB, ApoA1, and their ratio provide superior discrimination of cardiovascular risk compared to LDL-C alone. Assessment of disease severity: In patients with established CAD, higher AIP levels are correlated with more severe coronary lesions, a higher incidence of multivessel disease, and a poorer prognosis. For example, studies have shown that high AIP is linked to higher rates of in-stent restenosis and thrombosis after percutaneous coronary intervention (PCI). Identification of high-risk populations: Atherogenic indices can help identify specific populations with elevated cardiovascular risk. Metabolic syndrome: Significantly elevated AIP levels are found in patients with metabolic syndrome, supporting its use as a screening biomarker. Diabetes: The association between a high AIP and major cardiovascular events is especially pronounced in patients with diabetes, making it a valuable prognostic tool for this high-risk group.

Obesity: These indices correlate with obesity and visceral fat, highlighting the connection between adiposity and adverse lipid profiles.

The indices are calculated from a standard lipid panel, requiring no additional or complex laboratory tests. This makes them an inexpensive and accessible mass screening method for identifying individuals at high risk for CVD, particularly in low-resource settings. Hence, these can serve as cost-effective screening tool. Presenting complex lipid data as a single, intuitive index can help clinicians communicate cardiovascular risk to patients more effectively. Some studies suggest this approach may increase patient motivation to adopt healthy lifestyle changes. Atherogenic indices can be used to monitor the effectiveness of interventions. For example, studies have shown that AIP decreases significantly following successful metabolic surgery, indicating a reduction in CVD risk. For patients who have achieved target LDL-C levels, monitoring AIP can help address residual cardiovascular risk associated with atherogenic dyslipidemia.

AIP can provide prognostic information beyond that of traditional CVD risk factors and risk calculators like the Framingham Risk Score. Incorporating AIP into existing risk models could improve predictive accuracy and lead to more efficient treatment strategies.

ApoB reduction more accurately reflects the efficacy of statins and PCSK9 inhibitors than LDL-C decrease. Genetic predispositions such as elevated Lp(a) can guide early intervention, lifestyle modification, and therapeutic intensity. Thus, offering better therapeutic monitoring and personalized medicine.

Conclusion

The shift from traditional lipid measurement to extended lipid profiling and atherogenic indices marks a significant evolution in cardiovascular diagnostics. These parameters transcend the limitations of conventional testing by assessing particle number, size, and functionality—key determinants of atherogenic potential.

Integration of ApoB, ApoA1, Lp(a), and derived indices such as AIP into in Laboratory and clinical practice can provide a multi-dimensional view of comprehensive and precision-based approach to cardiovascular risk prediction. Routine adoption of extended lipid panels may substantially improve early detection, enabling clinicians to tailor interventions and monitor therapeutic outcomes more effectively particularly among high-risk and borderline populations.

Limitations and future directions

Despite their strengths, atherogenic indices can have certain limitations as well. Most research validating these indices has been conducted in specific ethnic groups, particularly Asian populations. Further studies are needed to confirm their predictive value across different ethnicities. Extensive

research is needed to determine the optimal cutoff points for AIP and other indices in various populations and clinical scenarios.

References

1. Global Burden of Disease Collaborative Network, Global Burden of Disease Study 2021 (GBD 2021) Results (2024, Institute for Health Metrics and Evaluation – IHME) https://vizhub.healthdata.org/gbd-results/
2. Catapano AL, Tokgözoğlu L, Mello e Silva A, Bruckert E. Atherogenic markers in predicting cardiovascular risk and targeting residual cardiovascular risk. Atherosclerosis: X. 2019;1. https://doi.org/10.1016/j.athx.2019.100001.
3. Bergmann K, Sypniewska G, Kretowicz M, Manitius J. Non-HDL cholesterol versus traditional lipid profile as predictor of insulin resistance and cardiovascular risk in young clinically healthy non-obese individuals. Biochim Clin. 2013;37.
4. Dong J, Yang S, Zhuang Q, Sun J, Wei P, Zhao X, et al. The Associations of Lipid Profiles With Cardiovascular Diseases and Death in a 10-Year Prospective Cohort Study. Front Cardiovasc Med. 2021;8. https://doi.org/10.3389/fcvm.2021.745539.
5. Mora S, Szklo M, Otvos JD, Greenland P, Psaty BM, Goff DC Jr, O'Leary DH, Saad MF, Tsai MY, Sharrett AR. LDL particle subclasses, LDL particle size, and carotid atherosclerosis in the Multi-Ethnic Study of Atherosclerosis (MESA). Atherosclerosis. 2007 May;192(1):211-7. doi: 10.1016/j. atherosclerosis. 2006.05.007.
6. Sehayek D, Cole J, Björnson E, Wilkins JT, Mortensen MB, Dufresne L, Pencina KM, Pencina MJ, Thanassoulis G, Sniderman AD. ApoB, LDL-C, and non-HDL-C as markers of cardiovascular risk. J Clin Lipidol. 2025 Jul-Aug;19(4):844-859. doi: 10.1016/j.jacl.2025.05.024.
7. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L; INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004 Sep 11-17;364(9438):937-52. doi: 10.1016/S0140-6736(04)17018-9.
8. Reyes-Soffer G, Ginsberg HN, Berglund L, et al. Lipoprotein(a): A Genetically Determined, Causal, and Prevalent Risk Factor for Atherosclerotic Cardiovascular Disease: A Scientific Statement From the American Heart Association. *Arterioscler Thromb Vasc Biol*. 2022;42(1): e48-e60. doi:10.1161/ ATV.0000000000000147.
9. Bekbossynova M, Saliev T, Ivanova-Razumova T, Andossova S, Kali A, Myrzakhmetova G. Small dense LDL: An underestimated driver of atherosclerosis (Review). *Mol Med Rep*. 2025;32(6):328. doi:10.3892/mmr.2025.13693.
10. Raaj I, Thalamati M, Gowda M N V, Rao A. The Role of the Atherogenic Index of Plasma and the Castelli Risk Index I and II in Cardiovascular Disease. *Cureus*. 2024;16(11):e74644. Published 2024 Nov 28. doi:10.7759/cureus.74644
11. Niroumand S, Khajedaluee M, Khadem-Rezaiyan M, Abrishami M, Juya M, Khodaee G, Dadgarmoghaddam M. Atherogenic Index of Plasma (AIP): A marker of cardiovascular disease. Med J Islam Repub Iran. 2015 Jul 25;29:240.

Trace Element–Cytokine Interactions - Serum Zinc and Interleukin-6 Imbalance reflects Oxidative Stress in Hypertension

Thuraya Abdulsalam A.A.AL-Azazi[1], SB Sharma[2*] ,Manoj Kumar Nandkeoliar [3] and Renu Chane [4]

[1]Ph.D. Scholar, [2,3]Professor, [4] Associate Professor, Department of Biochemistry, School of Medical Sciences and Research, Sharda University, Greater Noida, India.

Corresponding author: S B Sharma[*] (e-mail:drsbs08@hotmail.com)

Abstract:

Background: Hypertension is increasingly recognised as a disorder driven by vascular inflammation and oxidative stress. Zinc (Zn), an essential trace element with antioxidant and anti-inflammatory properties, and interleukin-6 (IL-6), a cytokine central to vascular inflammation, have both been implicated in endothelial dysfunction. However, their combined contribution to blood pressure regulation remains unclear.

Methods: A case–control study was conducted involving 100 hypertensive patients and 100 normotensive participants. Serum Zn was measured using atomic absorption spectrophotometry, and IL-6 using enzyme-linked immunosorbent assay (ELISA). Comparative analysis were performed using *t*-tests, and multiple linear regression showed the associations between Zn, IL-6, and blood pressure both systolic and diastolic.

Results: Hypertensive subjects exhibited significantly lower serum Zn ($p < 0.01$) and higher IL-6 levels ($p < 0.001$) compared with controls. However, regression models showed no significant independent association between Zn or IL-6 and blood pressure (SBP model $p = 0.747$; DBP model $p = 0.432$).

Conclusions:

Hypertension is associated with a disturbance in zinc–inflammatory balance, but neither Zn nor IL-6 independently predicts blood pressure. These findings suggest that trace element and cytokine alterations reflect the oxidative–inflammatory milieu of hypertension rather than act as direct determinants of arterial pressure.

Keywords: Hypertension, Zinc, Interleukin-6, Oxidative stress, Inflammation.

Introduction:

Hypertension (HTN) represents a crucial, modifiable risk factor for cardiovascular diseases (CVDs), yet it often goes undiagnosed or is inadequately managed. Hypertension represents a complex interplay between hemodynamic, metabolic, and inflammatory factors. In India, the burden of hypertension has reached epidemic proportions. Recent data from the National Family Health Survey (NFHS-5) indicate an age-standardized prevalence of 28.1 % among adults aged 18–69 years, with higher rates in urban (32.6 %) than rural populations (25.9 %) (1). The rising prevalence underscores the urgent need to identify biochemical and inflammatory markers that may contribute to vascular dysfunction. Chronic vascular inflammation and oxidative stress are established mediators of endothelial damage and arterial remodelling (2,3).

Interleukin-6 (IL-6), a multifunctional cytokine, contributes to vascular injury through activation of redox-sensitive transcription pathways and smooth-muscle proliferation (4). Zinc (Zn), conversely, functions as a cofactor for antioxidant enzymes such as superoxide dismutase and plays an important regulatory role in inflammation modulation (5,6). Dysregulated Zn metabolism has been associated with enhanced oxidative stress and endothelial dysfunction, but findings on its direct effect on blood pressure are inconsistent (7–9). A recent Mendelian-randomization study found no causal association between circulating Zn levels and hypertension (10). Similarly, although IL-6–targeting therapies have shown modest improvements in vascular health, their effects on blood pressure control remain unclear (11–13).

The present study investigates the relationship between serum Zn and IL-6 levels with blood pressure indices among hypertensive and normotensive adults in India, aiming to determine whether these biomarkers serve as independent predictors or secondary indicators of oxidative–inflammatory vascular stress.

Material & Methods:

A case–control study was conducted involving 200 participants aged 30–65 years: 100 diagnosed with hypertension and 100 normotensive controls matched for age and sex. Participants were recruited from the School of Medical Sciences and Research, Sharda University, Greater Noida, India. Exclusion criteria included secondary hypertension, diabetes, renal or hepatic disease, and recent mineral supplementation. Venous blood samples were collected. Serum Zn concentrations were measured by atomic absorption spectrophotometry (AAS) and IL-6 by enzyme-linked immunosorbent assay (ELISA) according to the manufacturer's protocol. Data were analyzed using SPSS v26. Results were expressed as mean ± SD. Group differences were tested with independent *t*-tests. Multiple linear regression were used to assess the relationships of Zn and IL-6 with SBP and DBP. $P < 0.05$ was considered statistically significant.

Results:

Hypertensive participants had significantly lower serum Zn (30.97±30.97µg/dL) compared with controls (70.44±15.16µg/dL; $p < 0.01$). Serum IL-6 levels were significantly higher in hypertensives (14.93±0.38pg/mL) than in controls (8.77±2.32 pg/mL; $p < 0.001$) Table 1.

Multiple regression analysis revealed no significant relationship between Zn or IL-6 and blood pressure. These findings indicate that while biochemical disparities exist between hypertensive and normotensive groups, Zn and IL-6 do not independently predict blood pressure within individuals.

Table1: showing serum Zn and IL-6 levels in hypertensive and control healthy subjects

Control		N	Hypertension
Zn µg/dL	70.44±15.16	100	30.97±30.97
IL-6 pg/Ml	8.77±2.32	100	14.93±0.38

Independent Samples Test									
	Levene's Test for Equality of Variances		t-test for Equality of Means						
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
								Lower	Upper
Zn	66.811	.000	-22.933	198	.000	-39.4651400	1.7209233	-42.8588309	-36.0714491
IL-6 pg/Ml	125.698	.000	26.164	198	.000	6.15920	.23541	5.69497	6.62343

Multiple regression analysis revealed no significant relationship between Zn or IL-6 and blood pressure.

Regression

Variables Entered/Removeda

Model	Variables Entered	Variables Removed	Method
1	IL-6 pg/Ml, Zn[b]		Enter

a. Dependent Variable: DBP

b. All requested variables entered.

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.151[a]	.023	.003	12.5813

a. Predictors: (Constant), IL-6 pg/Ml, Zn

ANOVAa

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	360.259	2	180.130	1.138	.325[b]
	Residual	15354.101	97	158.290		
	Total	15714.360	99			
a. Dependent Variable: DBP						
b. Predictors: (Constant), IL-6 pg/Ml, Zn						

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	34.906	49.164		.710	.479
	Zn	-.138	.157	-.089	-.885	.378
	IL-6 pg/Ml	4.351	3.313	.133	1.314	.192

a. Dependent Variable: DBP

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.077[a]	.006	-.015	11.2033

Predictors: (Constant), IL-6 pg/Ml, Zn

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	146.054	2.059		70.930	.000
	Zn	.370	.513	.073	.721	.473
	IL-6 pg/Ml	.022	.101	.022	.221	.826

a. Dependent Variable: SBP

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.131[a]	.017	-.003	12.6183

a. Predictors: (Constant), IL-6 pg/Ml, Zn

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	97.246	2.319		41.931	.000
	Zn	-.751	.578	-.131	-1.299	.197
	IL-6 pg/Ml	.015	.114	.014	.135	.893

a. Dependent Variable: DBP

Discussion:

Hypertension is a significant modifiable risk factor for CVDs; however, it often goes undiagnosed and is inadequately managed. The underlying mechanisms of hypertension are complex. This study confirms that hypertensive subjects exhibit decreased Zn and elevated IL-6 levels, consistent with a state of oxidative stress and systemic inflammation (4–6,14). However, neither Zn nor IL-6 showed an

independent predictive relationship with SBP or DBP, suggesting that their alterations reflect secondary consequences of hypertension rather than primary causes.

Recent work supports this view. Liu et al. (10) found no genetic link between serum Zn and hypertension risk, while Nazari et al. (15) observed only minor improvements in cardiovascular parameters after Zn supplementation. Conversely, animal models show that Zn deficiency can increase renal sodium retention via Na^+–Cl^- cotransporter up-regulation, implying an indirect mechanistic role (16).

IL-6 remains a well-established marker of vascular inflammation. Elevated IL-6 correlates with arterial stiffness and endothelial dysfunction (11,12,17). Recent findings indicate that antihypertensive drugs with IL-6-modulating effects may improve vascular outcomes independent of blood pressure reduction (13).

Overall, this evidence supports a model in which Zn and IL-6 participate in the inflammatory–oxidative network underlying hypertension but do not function as independent determinants of arterial pressure.

Conclusion:

Hypertension is associated with reduced zinc and elevated IL-6 concentrations in the Indian population, indicating an oxidative–inflammatory imbalance. However, these biomarkers do not independently predict blood pressure levels. They may serve as secondary indicators of vascular stress rather than direct contributors to hemodynamic regulation.

References:

1. Saju, M. D., Allagh, K. P., Scaria, L., Joseph, S., & Thiyagarajan, J. A. Prevalence, Awareness, Treatment, and Control of Hypertension and Its Associated Risk Factors: Results from Baseline Survey of SWADES Family Cohort Study. *International journal of hypertension*, *2020*, 4964835. https://doi.org/10.1155/2020/4964835.
2. Montezano AC, Touyz RM. Reactive oxygen species and endothelial function—role in hypertension. *Front Physiol.* 2012;3:441.
3. Virdis A, Dell'Agnello U, Taddei S. Impact of inflammation on vascular disease in hypertension. *Maturitas.* 2014;78(3):179-183.
4. Schieffer B, Schieffer E, Hilfiker-Kleiner D, Hilfiker A, Kovanen PT, Kaartinen M, Nussberger J, Harringer W, Drexler H. Expression of angiotensin II and interleukin 6 in human coronary atherosclerotic plaques: potential implications for inflammation and plaque instability. Circulation. 2000 Mar 28;101(12):1372-8. doi: 10.1161/01.cir.101.12.1372. PMID: 10736279.
5. Prasad AS. Zinc in human health: effect of zinc on immune cells. *Mol Med.* 2008;14(5-6):353-357.
6. Jarosz M, Olbert M, Wyszogrodzka G, Młyniec K, Librowski T. Antioxidant and anti-inflammatory effects of zinc. *Nutrients.* 2017;9(10):1211.

7. Al-Daghri, N. M., Al-Attas, O. S., Yakout, S. M., Alnaami, A. M., Wani, K., & Alokail, M. S. The association of serum 25-OH vitamin D with asthma in Saudi adults. *Medicine*, *97*(36), e12286(2018). https://doi.org/10.1097/MD.0000000000012286
8. Huang L, Yuan Q, He Y, et al. The effect of zinc supplementation on blood pressure: a systematic review and meta-analysis. *Eur J Nutr.* 2020;59(2):565-577.
9. Choi SJ, Kim YS, Bae A, et al. Dietary zinc intake is inversely associated with systolic blood pressure in obese Korean women. *Br J Nutr.* 2013;109(9):1741-1750.
10. Liu Y, Bao C, Wang H, Wei D, Zhang Z. Appraising the Role of Circulating Concentrations of Micronutrients in Hypertension: A Two-sample, Multivariable Mendelian Randomization Study. Glob Heart. 2024 Oct 29;19(1):81. doi: 10.5334/gh.1367. PMID: 39479260; PMCID: PMC11523844.
11. Batuo H, van der Linden E, Galenkamp H, Moll van Charante E, Born BV, Chilunga FP. Effects of antihypertensives with and without IL-6 lowering properties on long-term blood pressure control: The prospective HELIUS cohort. Int J Cardiol Cardiovasc Risk Prev. 2024 Dec 11;24:200358. doi: 10.1016/j.ijcrp.2024.200358. PMID: 39760129; PMCID: PMC11699610.
12. Mehta NN, deGoma E, Shapiro MD. IL-6 and Cardiovascular Risk: A Narrative Review. Curr Atheroscler Rep. 2024 Nov 26;27(1):12. doi: 10.1007/s11883-024-01259-7. PMID: 39589436; PMCID: PMC11599326.
13. R Muralitharan R, Marques FZ, O'Donnell JA. Recent advancements in targeting the immune system to treat hypertension. Eur J Pharmacol. 2024 Nov 15;983:177008. doi: 10.1016/j.ejphar.2024.177008. Epub 2024 Sep 18. PMID: 39304109.Tanase DM, Gosav EM, Burlacu A, et al. Arterial hypertension and interleukins: potential therapeutic target in cardiovascular disease. *Int J Mol Sci.* 2019;20(10):2380.
14. Nazari M, Ashtary-Larky D, Nikbaf-Shandiz M, Goudarzi K, Bagheri R, Dolatshahi S, Omran HS, Amirani N, Ghanavati M, Asbaghi O. Zinc supplementation and cardiovascular disease risk factors: A GRADE-assessed systematic review and dose-response meta-analysis. J Trace Elem Med Biol. 2023 Sep;79:127244. doi: 10.1016/j.jtemb.2023.127244. Epub 2023 Jun 15. PMID: 37399684.
15. Williams CR, Mistry M, Cheriyan AM, Williams JM, Naraine MK, Ellis CL, Mallick R, Mistry AC, Gooch JL, Ko B, Cai H, Hoover RS. Zinc deficiency induces hypertension by promoting renal Na^+ reabsorption. Am J Physiol Renal Physiol. 2019 Apr 1;316(4):F646-F653. doi: 10.1152/ajprenal.00487.2018.
16. Epub 2019 Jan 16. Erratum in: Am J Physiol Renal Physiol. 2019 Jul 1;317(1):F218-F219. doi: 10.1152/ajprenal.zh2-8726-corr.2019. PMID: 30649891; PMCID: PMC6483028.
17. Hao XM, Liu Y, Hailaiti D, Gong Y, Zhang XD, Yue BN, Liu JP, Wu XL, Yang KZ, Wang J, Liu QG. Mechanisms of inflammation modulation by different immune cells in hypertensive nephropathy. Front Immunol. 2024 Mar 13;15:1333170. doi: 10.3389/fimmu.2024.1333170. PMID: 38545112; PMCID: PMC10965702
18. .Zhang Y, Zhao Y, Zhong Y, Zeng R, Ye D, Guo D, Li W. Dietary zinc intake is inversely associated with the risk of hypertension in the periodontitis population. Front Nutr. 2025 Jul 15;12:1616989. doi: 10.3389/fnut.2025.1616989. PMID: 40735233; PMCID: PMC12303804.

Stroke: A Overview

Sangeeta Sharma

Professor & Head, Neuropsycholopharmacology Department, Institute of Human Behavior & Allied Sciences, New Delhi.

Stroke in India: where we stand and what to do next

Stroke is now one of India's most urgent cardiovascular problems. Globally, there are almost 12 million new strokes each year and one in four adults over 25 will have a stroke in their lifetime. (1,2) In India, stroke caused an estimated 699,000 deaths in 2019 (7.4% of all deaths) with wide state-to-state variation, reflecting uneven prevention and care. (3) Recent analyses of Indian data from 1990-2021 confirm a heavy and shifting burden driven by modifiable risks: hypertension, diabetes, tobacco, dyslipidaemia, atrial fibrillation, air pollution, and sedentary lifestyles. (4)

Why atherosclerosis matters in stroke

Most strokes are ischaemic, caused by a blocked brain artery. Atherosclerosis is a key mechanismeither large-artery disease (carotid or intracranial plaques) or artery-to-artery embolism from unstable plaque. Atherosclerosis also amplifies risk in cardioembolic stroke via shared factors like age, hypertension, and diabetes. Controlling BP and low-density lipoprotein cholesterol (LDL-C), eliminating tobacco, and treating diabetes and atrial fibrillation are the levers that bend the curve.

Prevention: the highest-yield actions

Blood pressure. After a stroke or transient ischaemic attack (TIA), most patients with hypertension should target <130/80 mm Hg with lifestyle and medication. Tight BP control also prevents first strokes.(5)

Lipids. LDL-C lowering is central. For secondary prevention and very-high-risk patients, guidelines recommend ≥50% LDL-C reduction and a target <55 mg/dL (<1.4 mmol/L). Add ezetimibe and then a PCSK9 inhibitor if goals are not met on maximal statin. (6) The 2025 focused update reinforces early, aggressive LDL-C lowering across risk tiers to reduce cardiovascular events, including stroke. (7)

Atrial fibrillation (AF). Screen systematically after ischaemic stroke; extended rhythm monitoring detects more AF. Most patients with AF and prior stroke should receive oral anticoagulation (preferably a direct oral anticoagulant unless contraindicated). (8)

Lifestyle and cardiometabolic risks. Smoking cessation, salt reduction, weight control, regular physical activity, Mediterranean-style eating patterns, glycaemic control in diabetes, and adherence to antihypertensive and lipid-lowering therapy together deliver the largest absolute risk reduction.

Acute care: time windows are realand expanding

For acute ischaemic stroke, rapid recognition and hospital transfer remain decisive. Intravenous thrombolysis is time-sensitive, and mechanical thrombectomy (MT) is now standard of care for large-vessel occlusions when eligibility criteria are met. Landmark trials show MT benefit up to 6–24 hours from last-known-well in selected patients using perfusion or clinical-core mismatch criteria (DAWN/DEFUSE - 3). Major guidelines reflect this extended window. (9,10) Emerging data explore MT beyond 24 hours in carefully selected cases, but this remains investigational and centre-specific. (12)

For India, the practical message is simple: build prehospital stroke pathways (recognition, dispatch, prenotification), expand 24 by 7 thrombolysis capability, and develop regional MT hubs with tele-stroke support for spoke hospitals.

Secondary prevention package after ischaemic stroke/TIA

1. **High-intensity statin** (e.g., atorvastatin 80 mg) with LDL-C target <55 mg/dL; add ezetimibe/PCSK9 inhibitor as needed. (6)
2. **BP control** to <130/80 mm Hg with ACEi/ARB-based regimens plus thiazide-like diuretic or calcium-channel blocker as required. (5)
3. **Antithrombotic therapy** tailored to mechanism:
   - Non-cardioembolic stroke: single antiplatelet (aspirin or clopidogrel) long-term; consider short dual antiplatelet therapy (DAPT) for minor stroke/TIA per local protocol.
   - AF-related stroke: oral anticoagulation unless contraindicated. (5)
4. **Lifestyle and diabetes management** as above; treat triglycerides and consider icosapent ethyl if triglycerides 135-499 mg/dL on statin and LDL-C 41-100 mg/dL. (10,11)
5. **Carotid disease**: evaluate for revascularization when symptomatic stenosis is present; ensure best medical therapy for all.

What the Indian Society for Atherosclerosis Research can drive

1) Bench-to-community lipid control. Promote routine LDL-C goal-oriented care in cardiology, neurology, and primary care clinics; publish simple treatment algorithms and dose-titration schedules aligned to the <55 mg/dL target in very-high-risk patients. (6)

2) Hypertension action. Support practical home BP monitoring programs with monthly adherence nudges, focusing on states with the highest stroke DALYs. (4)

3) Tobacco and air quality. Partner with state programs on cessation services and clean-air advocacy; both yield stroke gains. (4)

4) Stroke-ready districts. Advocate for district-level stroke pathways: EMS training, hospital prenotification, standard thrombolysis kits, and defined referral routes to thrombectomy hubs using tele-stroke. Align messaging with the extended MT window to reduce nihilism about late presenters. (13)

5) Data and equity. Support audits of door-to-needle and door-to-groin times, LDL-C attainment rates, and medication adherence, with a focus on rural and underserved regions where burden is high and services are thin. (3)

India can cut stroke deaths and disability by doing the basics well - lower BP, lower LDL-C, treat AF, stop tobacco, and organize acute stroke systems. Science is clear; the opportunity is large; the tools are in hand.

References

1. Feigin VL, Brainin M, Norrving B, Martins SCO, Lindsay P, Roth GA, et al. World Stroke Organization (WSO) Global Stroke Fact Sheet 2025. Int J Stroke. 2025;20(2):[epub ahead of print]. doi:10.1177/17474930241308142.
2. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024;23(10):973–1003. doi:10.1016/S1474-4422(24)00369-7.
3. India State-Level Disease Burden Initiative Neurological Disorders Collaborators. The burden of neurological disorders across the states of India: the Global Burden of Disease Study 1990–2019. Lancet Glob Health. 2021;9(8):e1129–e1144.
4. Behera DK, Rahut DB, Mishra S. Analyzing stroke burden and risk factors in India using data from the Global Burden of Disease Study. Sci Rep. 2024 Sep 30;14(1):22640. doi: 10.1038/s41598-024-72551-4. PMID: 39349946; PMCID: PMC11443044.
5. Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D, et al. 2021 guideline for the prevention of stroke in patients with stroke and transient ischemic attack. Stroke. 2021;52(7):e364–e467. doi:10.1161/STR.0000000000000375.
6. European Society of Cardiology; European Atherosclerosis Society. 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. 2025. Available from: https://www.escardio.org (accessed 24 Sep 2025).
7. Koskinas KC, Mach F, Catapano AL, et al. 2025 ESC/EAS Focused Update on dyslipidaemias. Atherosclerosis. 2025;[epub ahead of print]. doi:10.1016/j.atherosclerosis.2025.08.XXX.
8. Nogueira RG, Jadhav AP, Haussen DC, Bonafe A, Budzik RF, Bhuva P, et al. Thrombectomy 6 to 24 hours after stroke with a mismatch between deficit and infarct (DAWN). N Engl J Med. 2018;378(1):11–21. doi:10.1056/NEJMoa1706442.
9. Albers GW, Marks MP, Kemp S, Christensen S, Tsai JP, Ortega-Gutierrez S, et al. Thrombectomy for stroke at 6 to 16 hours with selection by perfusion imaging (DEFUSE-3). N Engl J Med. 2018;378(8):708–718. doi:10.1056/NEJMoa1713973.

10. Bhatt DL, Steg PG, Miller M, Brinton EA, Jacobson TA, Ketchum SB, et al. Cardiovascular risk reduction with icosapent ethyl for hypertriglyceridemia (REDUCE-IT). N Engl J Med. 2019;380(1):11–22. doi:10.1056/NEJMoa1812792.

11. Bhatt DL, Steg PG, Miller M, Brinton EA, Jacobson TA, Ketchum SB, et al. Effects of icosapent ethyl on total ischemic events: the REDUCE-IT trial. J Am Coll Cardiol. 2019;73(22):2791–2802. doi:10.1016/j.jacc.2019.02.032.

12. Endovascular Treatment Beyond the 24-Hour Mark. https://eso-stroke.org/endovascular-treatment-beyond-the-24-hour-mark/

13. Turc, Guillaume. Mechanical Thrombectomy in Late-Time Windows: Time to Treat More Patients. Stroke 2023; 54: 3: pp 731-732. doi:10.1161/STROKEAHA.122.041420

14. India State-Level Disease Burden Initiative Neurological Disorders Collaborators. The burden of neurological disorders across the states of India: the Global Burden of Disease Study 1990–2019. Lancet Glob Health. 2021;9(8):e1129–e1144.

15. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024;23(10):973–1003. doi:10.1016/S1474-4422(24)00369-7.

16. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, et al. 2019 update to the 2018 guidelines for the early management of acute ischemic stroke. Stroke. 2019;50(12):e344–e418. doi:10.1161/STR.0000000000000211.

Dyslipidemia: Current Practices and Guidelines

Anjali Manocha

Senior Consultant, Biochemistry, Ganga Ram Hospital, New Delhi

Dyslipidemia, one of the most commonly detected & treated chronic conditions, is a vital risk factor due to its significant association with cardiovascular disease (CVD). Its importance lies in the fact that it is a modifiable risk factor as about 80% of lipid disorders are related to diet & lifestyle, with the rest being familial. There are no single comprehensive dyslipidemia guidelines, but what is common among them all is that they all identify the routine lipid panel as a practical starting point. For CVD, elevated LDL cholesterol is essentially causal and guidelines worldwise recommend treatment targets for this lipid fraction. Currently, dyslipidemia is an expanding area of research due to recent insight into their role in the molecular basis and genetic origin of the condition. Hence, it ispertinent to discuss, very briefly, the salient recent guidelines for the management of dyslipidemia so as to consider an updated clinical approach forits management.

In the recent Clinical Practice Guidelines for Dyslipidemia Management given by Sawhneyet al in 2024 (1), the focus was on the management of dyslipidemia, specifically in the Indian population. Since India has the distinction of the highest number of dyslipidemia patients globally, the study highlighted specific points as follows:

- A significant epidemiological, genetic and causal association exists between total cholesterol, non-HDL-c,LDL-c & triglycerides with ASCVD events and morality.
- Increasing population level total cholesterol, LDL-c& triglycerides trends have been observed in emigrant South Asians vis-à-vis the developed countries where a declining trend has been observed.
- Raised triglyceridesare a unique feature of dyslipidemia in India.
- Hypercholesterolemia prevalence in southern states is greater than the north Indian states with urban more than rural, while triglycerides& HDL-care similar throughout the country.
- Importantly, the level of awareness in the population and individual levels remains low.

Recommended Levels of Various Lipid Parameters					
Lipid Parameter		**Low Risk**	**Moderate Risk**	**High Risk**	**Very High Risk**
Total Cholesterol		**<200**	**<200**	**200-239**	**≥240**
L.D.L-C		**<100**	**<100**	**<70**	**<55**
H.D.L-C	**M**	**>40**	**>40**	**>40**	**>40**
	F	**>50**	**>50**	**>50**	**>50**
Non H.D.L-C		**<130**	**<130**	**<100**	**<85**
Triglycerides		**<150**	**<150**	**<150**	**<150**

Sawhney JPS, Ramakrishnan S, Madan K et al. CSI clinical practice guidelines for dyslipidemia management: Executive Summary. Indian Heart Journal 2024: 76: S6-S19.

Sawhney et al recommended accurate measurement of the different lipid parameters and measurement of non-fasting levels of total cholesterol, non-HDL-c, LDL-c& triglycerides for risk estimation and guiding treatment. They also advisedthe importanceof risk stratificationin guiding lipid lowering therapy and to identify goals. They suggested that Lp(a) should be measured at least once in a person's lifetime. Significantly, non-HDL-c has been included as a co-primary goal in their guidelines and measurement of non-HDL-c has been recommended as it is a simple calculation and highly predictive of ASCVD events.

The 2025 Focussed Update of the 2019 ESC/EAS Guidelines for the management of dyslipidemia (2) has also stated that epidemiologic and genetic studies strongly support a causal and direct continuous association between high Lp(a) levels and higher risk of ASCVD and aortic valve stenosis. Also, Lp(a) levels above 50 mg/dL (105 nmol/L) should be considered in all adults as a risk enhancing factor. They stated that triglyceride levels are associated with CVD risk, independent of LDL-c levels and recommended statins as the first drug of choice to reduce CVD risk in high-risk patients.

Hence in conclusion, atherosclerosis is a chronic progressive disease that begins early, progresses slowly and along with exposure to high levels of lipids, is associated with a higher risk of CVD. Statins are the drugs of choice for the management of dyslipidemia. A non-fasting lipid profile is acceptable and in acute coronary syndrome, the goal is to achieve LDL-c levels below 55 mg/dL as early as possible. Lifestyle interventions like increased physical activity and dietary modifications are important. Better awareness of dyslipidemia management can go a long way in reducing the morbidity and mortality associated with CVD in our country.

References:

1. Sawhney JPS, Ramakrishnan S, Madan K et al. CSI clinical practice guidelines for dyslipidemia management: Executive Summary. Indian Heart Journal 2024: 76: S6-S19
2. 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of Dyslipidemias. Developed by the Task Force for the management of Dyslipidemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). European Heart Journal 2025; 00;1-20.

Micronutrients with Macro Significance in Cardiovascular Health: Bridging Deficiency and Disease

M. Kankra and S. Bhargava

Department of Biochemistry, Sir Ganga Ram Hospital, New Delhi, India

Abstract

Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide, and nutritional deficiencies in essential micronutrients play a crucial yet underrecognized role in their development. This review examines the links between key micronutrients—magnesium, zinc, copper, iron, selenium, folate, vitamin D, and potassium—and cardiovascular outcomes. Evidence from recent trials indicates that imbalances in these nutrients can affect blood pressure control, endothelial function, oxidative stress, and heart energy production. Low magnesium (<0.75 mmol/L) is linked to a 22% higher risk of coronary events [1], vitamin D deficiency (<20 ng/mL) raises hypertension risk by 1.5 times [2,3], and a high Cu: Zn ratio (>1.5) is associated with a 40% increase in CVD mortality [5]. This paper emphasises the clinical significance, public health impact, and future research directions for optimising micronutrient levels to protect cardiovascular health.

Introduction

Micronutrients are vital for maintaining cardiovascular homeostasis, modulating enzymatic reactions, oxidative balance, and vascular tone. Deficiencies or imbalances of these elements —including magnesium, zinc, copper, iron, selenium, and vitamins —contribute to endothelial dysfunction, oxidative stress, inflammation, and arrhythmogenesis. With the global burden of CVDs rising, understanding the role of micronutrients provides an opportunity for preventive and therapeutic interventions beyond conventional pharmacologic strategies [1–3].

Materials and Methods

A structured literature search was performed using PubMed, Scopus, and Google Scholar from 2000 to 2024. Keywords included ‘micronutrients’, ‘cardiovascular disease’, ‘oxidative stress’, ‘magnesium’, ‘vitamin D’, ‘zinc’, ‘iron’, and ‘Cu: Zn ratio’. Inclusion criteria consisted of human studies, meta-analyses, and large cohort studies assessing micronutrient levels and cardiovascular outcomes [1–9].

Results and Review of Key Studies

Low magnesium levels are linked to a higher risk of arrhythmias, hypertension, and sudden cardiac death (ARIC Study, 2021) [1]. Vitamin D deficiency is associated with increased rates of heart failure and coronary artery disease (VITAL and D-Health Trials, 2022–2023) [2,3]. Iron deficiency negatively impacts exercise tolerance and quality of life in heart failure, as shown by FAIR-HF and AFFIRM-AHF trials [4]. Zinc and copper imbalances, indicated by elevated Cu: Zn ratios, predict systemic inflammation and oxidative stress (NHANES 2015–2020) [5]. Selenium and folate deficiencies contribute to oxidative injury and homocysteine buildup, respectively, both playing a key role in

atherogenesis [6,8]. Potassium supplementation or salt substitution has been shown to reduce stroke and major cardiovascular events (NEJM, 2021) [7].

Clinical Significance

Micronutrient status has direct implications for cardiovascular prevention and treatment. Adequate magnesium supports vascular tone and arrhythmia control [1]; vitamin D improves endothelial function and modulates the renin–angiotensin system [2]; and iron supplementation reduces hospitalizations due to heart failure [4]. Zinc and selenium enhance antioxidant defenses [5,8], while folate and B12 lower homocysteine levels, promoting endothelial repair [6]. Population data indicate that deficiency of one or more micronutrients is common in developing countries like India, highlighting the need for policy-level interventions such as food fortification, routine nutritional screening in cardiac clinics, and personalized supplementation. In India, where vitamin D deficiency affects over 70% of adults and iron deficiency is observed in 30–50% of women, food fortification has become a practical public health strategy [9]. Bhargava et al. (2021) stressed the importance of fortifying staples such as flour and milk to combat urban micronutrient deficiencies and mitigate the risk of related non-communicable diseases [9].

Discussion

Micronutrient imbalances contribute to cardiovascular disease through interconnected mechanisms. Endothelial dysfunction results from magnesium and zinc deficiency, lowering nitric oxide bioavailability. Oxidative stress increases with excess copper or iron, promoting lipid peroxidation [4,5]. Vitamin D, zinc, and selenium help reduce inflammation by modulating NF-κB and cytokines [2,5,8]. Low magnesium and potassium disrupt cardiac repolarization, increasing the risk of arrhythmia [1,7]. Recent evidence indicates that combining micronutrient assessment with conventional risk factors improves cardiovascular risk prediction models.

Clinically, incorporating micronutrient testing into cardiac risk assessments can facilitate early detection of metabolic stress. Magnesium and vitamin D supplementation may slow the progression of hypertension [1–3], while intravenous iron enhances functional capacity in chronic heart failure [4]. However, indiscriminate supplementation without baseline assessment may lead to toxicity or nutrient imbalance, highlighting the importance of personalized therapy [9].

Future research should examine combined nutrient interventions, nutrigenomic responses, and long-term cardiovascular outcomes. Large-scale randomized trials assessing micronutrient repletion across diverse populations are vital to determine optimal thresholds for prevention and management.

Conclusion

Micronutrient homeostasis is essential for cardiovascular health. Evidence indicates that a balanced intake of magnesium, zinc, vitamin D, selenium, and iron helps protect blood vessels, reduce oxidative

stress, and improve heart health [1–9]. Incorporating nutritional monitoring into routine cardiac care is an underused yet promising approach to comprehensive cardiovascular disease prevention.

References

1. Zhang Y, et al. Serum magnesium and cardiovascular mortality: ARIC Study. *J Am Heart Assoc*. 2021;10:e019209.
2. Manson JE, et al. Vitamin D and Omega-3 Trial (VITAL). *N Engl J Med*. 2022;386:105–116.
3. Zhang H, et al. D-Health Trial: Vitamin D supplementation and cardiovascular disease outcomes. *Lancet*. 2023;401:250–261.
4. Ponikowski P, et al. Ferric carboxymaltose in heart failure (AFFIRM-AHF). *N Engl J Med*. 2020;382:121–131.
5. Mocchegiani E, et al. Cu:Zn ratio and oxidative stress in cardiovascular risk. *Nutrients*. 2022;14:3302.
6. Xu J, et al. Folate supplementation and stroke prevention (CSPPT). *JAMA*. 2020;323:1–10.
7. Neal B, et al. Salt Substitute and Stroke Study. *N Engl J Med*. 2021;385:1067–1077.
8. Schomburg L, et al. Selenium status and cardiovascular risk. *Eur Heart J*. 2022;43:2204–2215.
9. Bhargava M, et al. Micronutrient deficiencies and anemia in urban India—Do we need food fortification? *Indian J Clin Biochem*. 2021;37(2):149–158. doi:10.1007/s12291-021-00966-1

Epigenetic modifications due to lifestyle can increase Atherosclerotic risk? – A mini-review

Harsh Vardhan Singh.[1]Parul Goyal,[2] and Ritu Singh [3]

[1]Senior Biochemist, Biochemistry, North DMC Medical College & Hindu Rao Hospital, Delhi

[2] Director Professor, Biochemistry ABVIMS & Dr RML Hospital, Delhi

[3]Director Professor & HOD, Lady Hardinge Medical College & Associated Hospitals, Delhi.

Epigenetics is the study of heritable changes in gene expression which are not coded in the DNA. It involves all processes that alter gene activity without nucleotide sequence modification. The study of epigenetics is relatively new and it moves away from the concept that all heritable characteristics are encoded in the DNA of an individual.

The various regulatory mechanisms studied in epigenetics include DNA methylation, histone post-translational modifications, and modifications in noncoding RNAs. These processes are normally influencing cell differentiation, growth and development as well as environmental adaptation, biological aging and are now known to be modified in many disease states like cardiovascular diseases and cancers. Epigenetic changes can occur in response to environmental changes and lifestyle factors.This alters the gene expression that affect cardiovascular disease risk during the lifetime of the individual as well as be heritable. Biological aging is heavily influenced by traditional risk factors like diet and exercise which cause epigenetic modulation (1) further affecting cardiovascular risk.

The main mechanisms of epigenetics are the influence on histone modifications, DNA methylations and non-coding RNA interferences. The chromatin structure is modified when the DNA methylation happens at CpGs (Cytosine and Guanine). This is an important epigenetic mechanism controlling and modulating gene expression through interactions with histone modifications and chromatin remodeling complexes. This alters the local genomic pattern. DNA methylation plays an important role in maintaining genomic stability. However, DNA methylation patterns can change in response to environmental stimuli such as diet or toxins. The epigenome changes are most likely to happen in prenatal or early childhood. Changes of DNA methylation levels and patterns have been widely studied in cancers, immunological diseases and are now being studied in cardiovascular diseases. Epigenome engineering technologies is a new emerging field which studies functional relevance of DNA methylation (2).

Epigenetic post-transcriptional histone modifications, histone variants, ATP-dependent chromatin remodeling complexes, noncoding RNAs including siRNA and miRNAs, and DNA methylation can all be caused by lifestyle changes (3,4).

Evidence is accumulating that environmental stimuli, such as diet, particularly during pregnancy and early adulthood can induce changes in DNA methylation predisposing to obesity and related comorbidities.

It has been noted, that while genetics of atherosclerosis has been widely researched in the last decade, and has even lead to commercially available genetic risk assays, the performance of these assays and association of genotype analysis to atherosclerotic risk could not be widely accepted (5). On the other hand, lifestyle factors have been shown to be independently associated with susceptibility to Cardiovascular, regardless of an individual's genetic risk. It was also observed that individuals with an high genetic risk of CVD but leading a favourable lifestyle demonstrated a 50% lower relative risk of coronary artery disease compared with those leading an unfavorable lifestyle (6-9).

Researchers have explored the role of methylation in vascular diseases and found that aberrant methylation of endothelial and smooth muscle cell genes affects inflammatory and lipid metabolism pathways (10) These changes contribute to the development of atherosclerosis and hypertension. Environmental exposures, including smoking, pollution, and poor diet, were shown to modulate methylation states, suggesting that DNA methylation could serve as both a biomarker and a therapeutic target for vascular health. Cellular Environmental challenges such as such as vaccines, infections, and hyperglycaemia lead to activation of immune cells via epigenetic mechanisms which further increase inflammation and a risk of vascular disease (1).

Recently, it has been shown that an epigenetic clock based on DNA methylation levels is a potentially precise determinant of biological age and this epigenetic clock has been linked to age-related atherosclerotic disease risk.(1)Smoking has been proven to be a major risk factor for cardiovascular disease and based on DNA methylation analyses it is shown to be a source of accelerated biological aging (11,12,)Oxidative stress and inflammation leading to endothelial dysfunction has been shown to be based on Epigenetic modifications. Many *vitro* studies have provided evidence of the critical role of epigenetic mechanisms in endothelial dysfunction (13,14).

Epigenetic modifications play a pivotal role in cardiometabolic diseases like obesity, metabolic syndrome, type 2 diabetes, hypertension all of which increase atherosclerotic risk. The adiposity, inflammation and endothelial dysfunction, have a direct role in development of atherosclerosis. These heritable changes in gene expression which do not affect DNA sequence can be induced by triggers such as stress, pollution, and cigarette smoking. The modifications so acquired by lifestyle can be transmitted to the offspring and exert their biological effects across multiple generations (14).

The connection between nutrition and epigenetic regulation has also been researched recently. A study has highlighted that nutrients such as folate, vitamin B12, and methionine — essential for one-carbon metabolism — directly influence DNA methylation patterns. Nutritional deficiencies or imbalances can disrupt these patterns, predisposing individuals to cardiovascular, metabolic, and neurodegenerative diseases. The authors propose that nutritional interventions may help maintain or restore proper methylation, offering a preventive and therapeutic approach (15).

Further linking methylation to metabolic disorders, the article *Epigenetics and Mets* discusses how both hypermethylation and hypomethylation of genes involved in insulin signaling and lipid metabolism contribute to the development of metabolic syndrome (15). Altered methylation disrupts energy regulation, leading to obesity and systemic inflammation. Encouragingly, lifestyle modifications — including exercise and dietary regulation — were suggested to reverse some of these methylation changes, improving metabolic outcomes.

There is also compelling evidence that methylation signatures not only mark disease presence but also reflect recovery potential (16). Their study demonstrated that weight loss can partially reverse abnormal methylation profiles associated with obesity and metabolic syndrome. These findings reinforce the concept that DNA methylation is a dynamic and responsive process, acting as both a diagnostic tool and a measure of therapeutic success. Collectively, these studies underscore the central role of DNA methylation in mediating the relationship between genetics, environment, and disease, offering a foundation for personalized medicine and long-term health optimization.

Cardiovascular disease remains one of the leading causes of illness and death (17) As people age, gradual changes in cellular and molecular function, along with chronic low-grade inflammation, increase the risk of heart and vascular disorders. In recent years, researchers have discovered that epigenetic changes—such as DNA methylation, histone modification, and alterations in microRNA expression—play a significant role in how aging affects the cardiovascular system. These changes are strongly influenced by lifestyle and environmental factors including diet, physical activity, smoking, alcohol use, and exposure to pollutants. Encouragingly, evidence suggests that healthy habits, such as eating a balanced diet rich in fruits, vegetables, and essential nutrients, regular physical activity, avoiding tobacco and excessive alcohol, and managing stress, can help prevent harmful epigenetic shifts. Understanding these mechanisms provides valuable insights and scientific proof of how healthy living may delay or reduce the burden of cardiovascular disease.

To summarize, Epigenetics is the study of how genes can be switched on or off without changing the DNA sequence itself. It explains how lifestyle and environmental factors can influence health and disease through changes that affect gene activity. The main epigenetic mechanisms include DNA methylation, histone modification, and the regulation of non-coding RNAs. These processes guide cell growth, development, and aging, and help the body respond to environmental challenges.

In recent years, scientists have discovered that epigenetic changes play a major role in heart and blood vessel diseases. Factors such as smoking, pollution, stress, poor diet, and lack of exercise can alter normal gene expression, increasing the risk of conditions like atherosclerosis, high blood pressure, and heart attacks. These changes can occur early in life and may even be passed from one generation to the next.

On the positive side, healthy lifestyle choices can help protect the heart by promoting beneficial epigenetic patterns. Eating a balanced diet rich in fruits, vegetables, and nutrients such as folate and vitamin B12 supports normal DNA methylation. Regular physical activity, avoiding tobacco, and managing stress can also slow harmful changes linked to aging and cardiovascular disease.

Understanding how epigenetic processes work offers new hope for preventing and treating heart disease. Because these changes are potentially reversible, early interventions focused on lifestyle and nutrition may help restore normal gene function. Epigenetics provides a powerful link between our genes and the environment, showing that everyday choices can shape long-term heart health and overall well-being.

References:

1. Zarzour A, Kim HW, Weintraub NL. Epigenetic regulation of vascular diseases. *Arterioscler Thromb Vasc Biol.* 2019;39(6):879–888. doi:10.1161/ATVBAHA.119.312193
2. Andersen GB, Tost J. A summary of the biological processes, disease-associated changes, and clinical applications of DNA methylation. *Methods Mol Biol.* 2018;1708:3–30. doi:10.1007/978-1-4939-7481-8_1
3. Tost J. *Epigenetics.* Norwich, UK: Horizon Scientific Press; 2008.
4. Dawson MA, Kouzarides T. Cancer epigenetics: from mechanism to therapy. *Cell.* 2012;150(1):12–27. doi:10.1016/j.cell.2012.06.013
5. Schnabel RB, Baccarelli A, Lin H, Ellinor PT, Benjamin EJ. Next steps in cardiovascular disease genomic research: sequencing, epigenetics, and transcriptomics. *Clin Chem.* 2012;58(1):113–126. doi:10.1373/clinchem.2011.170423
6. Khera AV, Emdin CA, Drake I, et al. Genetic risk, adherence to a healthy lifestyle, and coronary disease. *N Engl J Med.* 2016;375(24):2349–2358. doi:10.1056/NEJMoa1605086
7. Abraham G, Havulinna AS, Bhalala OG, et al. Genomic prediction of coronary heart disease. *Eur Heart J.* 2016;37(43):3267–3278. doi:10.1093/eurheartj/ehw450
8. Sotos-Prieto M, Baylin A, Campos H, Mattei J. Lifestyle cardiovascular risk score, genetic risk score, and myocardial infarction in Hispanic/Latino adults living in Costa Rica. *J Am Heart Assoc.* 2016;5(10):e004067. doi:10.1161/JAHA.116.004067
9. Said MA, Verweij N, van der Harst P. Associations of combined genetic and lifestyle risks with incident cardiovascular disease and diabetes in the UK Biobank study. *JAMA Cardiol.* 2018;3(8):693–702. doi:10.1001/jamacardio.2018.1717
10. Zarzour A, Kim HW, Daaka Y, Jo H. Epigenetic regulation of vascular diseases. *Arterioscler Thromb Vasc Biol.* 2019;39(6):879–888. doi:10.1161/ATVBAHA.119.312193
11. Siemelink MA, van der Laan SW, Haitjema S, et al. Smoking is associated with DNA methylation in atherosclerotic carotid lesions. *Circ Genom Precis Med.* 2018;11(8):e002030. doi:10.1161/CIRCGEN.117.002030
12. Zhong J, Agha G, Baccarelli AA. The role of DNA methylation in cardiovascular risk and disease: methodological aspects, study design, and data analysis for epidemiological studies. *Circ Res.* 2016;118(1):119–131. doi:10.1161/CIRCRESAHA.115.305206

13. Khyzha N, Alizada A, Wilson MD, Fish JE. Epigenetics of atherosclerosis: emerging mechanisms and methods. *Trends Mol Med.* 2017;23(4):332–347. doi:10.1016/j.molmed.2017.02.004

14. Ambrosini S, Mohammed SA, Lüscher TF, Costantino S, Paneni F. New mechanisms of vascular dysfunction in cardiometabolic patients: focus on epigenetics. *High Blood Press Cardiovasc Prev.* 2020;27(4):363–371. doi:10.1007/s40292-020-00400-7

15. Kandi V, Vadakedath S. Effect of DNA methylation in various diseases and the probable protective role of nutrition: a mini-review. *Cureus.* 2015;7(8):e309. doi:10.7759/cureus.309

16. Samblas M, Milagro FI, Martínez JA. DNA methylation markers in obesity, metabolic syndrome, and weight loss. *Epigenetics.* 2019;14(5):421–444. doi:10.1080/15592294.2019.1595297

17. Gharipour M, Mani A, Amini Baghbahadorani M, et al. How are epigenetic modifications related to cardiovascular disease in older adults? *Int J Mol Sci.* 2021;22(18):9949. doi:10.3390/ijms22189949

Commentary: Integrating Molecular Biomarkers and Artificial Intelligence to Redefine Risk in Diabetic Coronary Artery Disease

Pradeep Kumar Dabla[1], Vimal Mehta[2], Harsh Vardhan Singh[3]

1Department of Biochemistry, Govind Ballabh Pant Institute of Postgraduate Medical Education and Research, Delhi 110002, India

2 Department of Cardiology, Govind Ballabh Pant Institute of Postgraduate Medical Education and Research, Delhi 110002, India

3 Department of Biochemistry, North DMC Medical College & Hindu Rao Hospital, Delhi

Corresponding Author:

Pradeep Kumar Dabla, Director Professor, Department of Biochemistry, Govind Ballabh Pant Institute of Postgraduate Medical Education and Research, Delhi 110002, India; pradeep_dabla@yahoo.com

Introduction

Coronary artery disease (CAD) remains the leading cause of mortality worldwide, with India contributing disproportionately to the global burden. The coexistence of type 2 diabetes mellitus (T2DM) substantially accelerates CAD onset and severity through hyperglycemia-driven oxidative stress and inflammation. Despite significant progress in risk stratification, the precise molecular and computational integration of diabetic and cardiovascular markers remains underdeveloped. Our recent works have addressed this gap from complementary perspectives—one biochemical, elucidating the role of advanced glycation end products (AGEs) and inflammation in diabetic CAD, and the other computational, using artificial intelligence (AI)-driven association rule mining (ARM) to uncover clinical patterns predictive of CAD in T2DM. Together, these studies offer a composite framework linking molecular pathology with data intelligence to advance predictive cardiometabolic medicine.

The Biochemical Landscape: Nε-Carboxymethyl-Lysine as a Central Mediator

Advanced glycation end products, particularly Nε-carboxymethyl-lysine (CML), have emerged as potent molecular drivers of vascular injury. Generated through non-enzymatic glycation of lysine residues, CML promotes protein crosslinking, endothelial stiffness, and RAGE-mediated inflammatory signaling. The *World Journal of Diabetes* study (Shrivastav et al., 2023) demonstrated that CML levels are significantly elevated in diabetic CAD patients compared with non-diabetic counterparts, accompanied by higher tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) concentrations and diminished nitric oxide (NO) levels. Correlation analyses underscored a strong positive association between CML and IL-6 ($r = 0.596$) and TNF-α ($r = 0.337$), alongside a negative correlation with NO ($r = -0.416$)—revealing a biochemical axis linking oxidative stress to vascular inflammation. Moreover, CML levels in the third quartile (264–364 ng/mL) conferred a threefold increased odds of diabetic CAD, even after adjusting for diet, hypertension, and lipid parameters. These findings position CML as more than a bystander of hyperglycemia; it acts as an active molecular signal of endothelial dysfunction.

Mechanistically, the AGE–RAGE interaction activates NF-κB, upregulates inflammatory cytokines, and suppresses endothelial NO synthase, fostering a pro-atherogenic milieu. This biochemical signature reinforces the concept of glyco-inflammatory cardiomyopathy, a phenotype increasingly recognized in diabetic patients with accelerated coronary pathology.

From Molecules to Machines: AI Discovers Hidden Clinical Patterns

While biomarkers quantify molecular derangement, AI augments the clinician's capacity to interpret multidimensional data. In our subsequent study (*World Journal of Methodology*, 2024), we employed association rule mining (ARM) to extract clinically meaningful rules from a dataset of 300 participants categorized as CAD-with-diabetes, CAD-without-diabetes, and healthy controls. ARM—implemented via the Hotspot algorithm in the WEKA environment—identified high-confidence rules linking glycemic indices with CAD status. The most robust association indicated that random blood sugar >175 mg/dL and HbA1c >6.6% predicted CAD-with-diabetes (Support = 0.33, Confidence = 1.0, Lift = 3). Conversely, low HbA1c (≤5.3%) and absence of angiographic disease predicted the healthy group with equal confidence. Such interpretable AI models transform static clinical data into dynamic decision rules, enabling physicians to anticipate risk trajectories rather than merely react to established disease. Importantly, the approach preserves transparency—critical in clinical AI adoption—allowing direct validation of discovered patterns through biochemical or imaging correlates.

Integrating Biochemistry and Computational Intelligence

The integration of biochemical markers like CML with AI-derived clinical rules marks a paradigm shift from reactive diagnostics to predictive precision cardiology. CML reflects the molecular injury interface, while AI captures its systemic manifestation across biochemical, hemodynamic, and demographic variables. Together, they form a bio-computational model capable of stratifying diabetic CAD risk on multiple axes—metabolic, inflammatory, and clinical.

The translational potential of this integration is significant:

1. **Early prediction** – Routine measurement of CML, TNF-α, IL-6, and NO, coupled with AI-based clinical pattern recognition, can identify high-risk diabetic patients before overt ischemia develops.
2. **Therapeutic targeting** – Interventions aimed at reducing AGE accumulation or RAGE activation may modulate the very pathways highlighted by AI models.
3. **Personalized monitoring** – AI-driven dashboards can continuously learn from patient data, adapting therapy goals for glycemic and lipid control in real time.

This synergy exemplifies the broader movement toward "Augmented Biochemistry", where molecular diagnostics and machine learning converge to interpret patient data as a coherent physiological narrative rather than isolated laboratory values.

Challenges and Future Perspectives

While promising, this integrated framework demands cautious translation. Standardization of CML assays, cross-platform validation of AI algorithms, and prospective multicentric trials remain essential. Moreover, ethical AI deployment requires explainability, data privacy, and clinician oversight to prevent algorithmic bias. From a mechanistic standpoint, further research should investigate whether CML reduction—via pharmacological AGE inhibitors, lifestyle modification, or improved glycemic control—translates into measurable decreases in AI-predicted CAD risk. Longitudinal integration of proteomic and imaging data could refine these predictive models, moving from population-level associations to individualized prognostication.

Conclusion

The confluence of molecular biochemistry and artificial intelligence offers a transformative approach to understanding and managing diabetic coronary artery disease. Elevated CML and inflammatory mediators delineate the biochemical substrate of endothelial dysfunction, while AI-driven rule mining translates this complexity into actionable clinical intelligence. This duality—the molecule and the machine—encapsulates the next evolution in cardiovascular medicine: one that is predictive, personalized, and precise. As these domains converge, they promise not only to decode the hidden patterns of diabetic atherosclerosis but also to reshape clinical decision-making toward preemptive, data-inform.ed care.

References:

1. Shrivastav D, Singh DD, Mir R, Mehra P, Mehta V, Dabla PK. Comparative analysis of Nε-carboxymethyl-lysine and inflammatory markers in diabetic and non-diabetic coronary artery disease patients. World J Diabetes. 2023 Dec 15;14(12):1754-1765. doi: 10.4239/wjd.v14.i12.1754. PMID: 38222780; PMCID: PMC10784802.
2. Dabla PK, Upreti K, Shrivastav D, Mehta V, Singh D. Discovering hidden patterns: Association rules for cardiovascular diseases in type 2 diabetes mellitus. World J Methodol. 2024 Jun 20;14(2):92608. doi: 10.5662/wjm.v14.i2.92608. PMID: 38983667; PMCID: PMC11229869.
3. Dabla PK, Shrivastav D, Mehta V, Singh S, Mir R, Kandar S. Risk assessment analysis of type II Diabetes Mellitus and dyslipidemia with Coronary Artery Stenosis scoring: intervention strategies. eJIFCC2025Vol36No3pp298-304.
4. Kumar Dabla P, Shrivastav D, Ali Beg M M, Meng Q. Exploring Natural Products to Control Type 2 Diabetes Mellitus through Targeting Advanced Glycation End Products. IJDO 2024; 16 (3) :191-201 URL: http://ijdo.ssu.ac.ir/article-1-889-en.html

Lentivirus-Based Gene Therapy: A New Hope for PAH Patients

Pranav Panchbhai, Harlokesh Narayan

Department of Pharmacology, AIIMS, New Delhi-110029

Email: hnyadav@gmail.com

Introduction

Pulmonary Arterial Hypertension (PAH) is a rare, progressive, and deadly disease marked by pulmonary artery (PA) remodeling, which elevates pulmonary vascular resistance (PVR), raising the right ventricular afterload, ultimately leading to heart failure(1). There are currently more therapeutic choices for PAH than there were a few decades ago. However, for many patients, PAH is still a fatal disease with a lesser prospect for recovery(2). In recent years, less frequent or uncommon mutations have been reported, reducing the gap in understanding the pathophysiology of PAH(3). Therefore, gene therapy techniques that would repair aberrant or disrupted gene expression within the lung vasculature are a viable future option in the treatment of PAH. The effective delivery of genes to the target tissue or cells is accomplished through the use of an appropriate gene delivery vehicle, known as a vector, which is a crucial component of any gene therapy-based strategy(4). Vectors made from retroviruses, adenoviruses, or adeno-associated viruses are used in contemporary viral vector-based gene therapy to deliver therapeutic genes to patients(5). However, tissue inflammation and transient expression restrict the applicability of adenoviral vectors (AdVs) in chronic diseases such as PAH(6). Furthermore, Immunogenic reactions to viral gene products are primarily responsible for the short duration of expression with AdVs (7) and the absence of genomic integration makes both naked and viral DNA inherently unstable. Therefore, the Lentiviridae of the Retroviridae family shows potential in the treatment of chronic conditions, including PAH. LVs can stably alter the target cells' genetic architecture by integrating into the chromatin of the host cell (8).

LVs have emerged as a preferred viral vector delivery system in experimental preclinical models of PAH. They are relatively safer, with unique qualities, such as stable transfection, lower immunogenicity, and the capability to carry multiple genes, as well as the ability to be modified into cardiotropic vectors.

Preclinical Insights of LVs in Treating PAH

Lentiviral vectors (LVs) have rapidly gained attention as a preferred gene delivery platform in experimental models of pulmonary arterial hypertension (PAH). Preclinical studies indicate that LV-based strategies are relatively safe in this context(9). The pathogenesis of PAH remains intricate, with ongoing research focused on reversing the vascular remodeling that drives the disease (10). LVs have become an indispensable tool for probing these mechanisms. In various studies, they have enabled the overexpression of protective genes such as PRDX6 (11) and TRB3 (12), which were shown to reduce medial hypertrophy, dampen inflammation, and improve vascular tone. Likewise, knockdown experiments targeting genes like CYLD (13) prevented smooth muscle thickening and promoted

adaptive right ventricular remodeling with preserved systolic function. Together, these findings highlight the versatility of LVs in studying long-term gene modulation in multiple PAH models.

Adding to their potential, LVs have been engineered to carry single, double, and even triple shRNA constructs, allowing simultaneous silencing of multiple targets—an approach first demonstrated in studies on HIV and HCV(14,15). Such multi-shRNA systems could be particularly useful in a multifactorial disease like PAH, where multiple signaling pathways interact. However, despite their strengths, LVs are not without limitations. Their ability to integrate stably into the host genome ensures long-term transgene expression—an advantage over transient systems such as AAV or adenoviral vectors—but it also raises the risk of insertional mutagenesis (16). Moreover, variable transduction efficiency among different pulmonary cell types and challenges in achieving selective targeting remain key barriers to clinical translation (17). Ultimately, animal studies underscore both the promise and the constraints of LV-based gene therapy for PAH, positioning lentiviral systems as a powerful, yet still evolving, tool in the quest to reprogram the diseased pulmonary vasculature.

Barrier to Translation

Over the years, lentiviral vectors (LVs) have been extensively modified to minimize risks and improve their safety profiles (18). Yet, despite these advances, several key challenges continue to limit their clinical translation. One of the foremost concerns is of random genomic integration, which can undesirably activate oncogenes and raise the risk of insertional mutagenesis (19). To address this, researchers have engineered non-integrating lentiviral vectors that maintain broad cell tropism and high transduction efficiency while avoiding genomic integration (20). However, such non-integrating systems are less suited for chronic diseases like pulmonary arterial hypertension (PAH), where long-term gene expression is crucial. Recent studies have offered a promising alternative, where integrase-deficient lentiviral vectors (IDLVs), which can serve as delivery vehicles for genome-editing tools such as CRISPR/Cas systems, enabling durable and controlled gene modification in vivo (21).

Although LVs typically elicit a relatively mild innate immune response, they are still susceptible to phagocytic clearance by immune cells. Incorporating the “don’t eat me” signal molecule CD47 has been shown to significantly reduce this issue (22). To further limit immune activation, researchers have increasingly turned to ex vivo gene therapy, where patient-derived cells are genetically modified outside the body and then reintroduced. Yet even this approach carries risks: adaptive immune reactions against the re-infused autologous cells may occur (23) and cell entrapment in the pulmonary vasculature could lead to potentially fatal embolic events (24).

Together, these findings underscore that while lentiviral systems remain a powerful and versatile tool for gene delivery, their clinical success in PAH will depend on continued innovation—refining vector safety, improving targeting specificity, and mitigating immune and vascular risks for reliable and long-term therapeutic outcomes.

Conclusion

Third-generation self-inactivating lentiviral vectors (LVs) represent a sophisticated and clinically promising platform for gene therapy in pulmonary arterial hypertension (PAH). Compared with adeno-associated viruses (AAVs) and non-integrating systems, LVs enable durable, high-efficiency transgene expression with minimal immunogenicity, making them well-suited for long-term therapeutic interventions. Their large packaging capacity accommodates large or multigenic constructs, while their ability to transduce both dividing and non-dividing cells broadens their applicability across diverse pulmonary cell types. Advances in third-generation vector design have further improved biosafety by eliminating replication-competent elements and reducing insertional risks. Preclinical studies increasingly demonstrate the capacity of LV-based systems to reverse PAH pathophysiology by modulating key molecular pathways involved in vascular remodeling, inflammation, and endothelial dysfunction.

Despite this progress, several translational challenges persist, including achieving selective delivery to the pulmonary vasculature, optimizing tissue-specific promoter systems for targeted expression, minimizing insertional mutagenesis, and overcoming barriers to large-scale vector production. Continued innovation in vector engineering, delivery strategies, and therapeutic gene selection will be essential to fully harness the clinical potential of LV-mediated gene therapy in PAH. Collectively, these advances position lentiviral vectors as a powerful and adaptable platform with the potential to transform the therapeutic landscape of this otherwise intractable disease.

Reference

1. Mocumbi A, Humbert M, Saxena A, Jing ZC, Sliwa K, Thienemann F, et al. Pulmonary hypertension. Nat Rev Dis Primer. 2024 Jan 4;10(1):1–20.
2. Lau EMT, Giannoulatou E, Celermajer DS, Humbert M. Epidemiology and treatment of pulmonary arterial hypertension. Nat Rev Cardiol. 2017 Oct;14(10):603–14.
3. Aldred MA, Morrell NW, Guignabert C. New Mutations and Pathogenesis of Pulmonary Hypertension: Progress and Puzzles in Disease Pathogenesis. Circ Res. 2022 Apr 29;130(9):1365–81.
4. Edelstein ML, Abedi MR, Wixon J, Edelstein RM. Gene therapy clinical trials worldwide 1989–2004—an overview. J Gene Med. 2004;6(6):597–602.
5. Yudaeva A, Kostyusheva A, Kachanov A, Brezgin S, Ponomareva N, Parodi A, et al. Clinical and Translational Landscape of Viral Gene Therapies. Cells. 2024 Jan;13(22):1916.
6. O'Brien T. Adenoviral Vectors and Gene Transfer to the Blood Vessel Wall. Arterioscler Thromb Vasc Biol. 2000 June;20(6):1414–6.
7. Shirley JL, de Jong YP, Terhorst C, Herzog RW. Immune Responses to Viral Gene Therapy Vectors. Mol Ther. 2020 Mar 4;28(3):709–22.
8. Yan KK, Condori J, Ma Z, Metais JY, Ju B, Ding L, et al. Integrome signatures of lentiviral gene therapy for SCID-X1 patients. Sci Adv. 2023 Oct 6;9(40):eadg9959.

9. Dai L, Du L. Genes in pediatric pulmonary arterial hypertension and the most promising BMPR2 gene therapy. Front Genet [Internet]. 2022 Nov 24 [cited 2025 Oct 10];13. Available from: https://www.frontiersin.org/journals/genetics/articles/10.3389/fgene.2022.961848/full
10. Wu X, Ma J, Ding D, Ma Y, Wei Y, Jing Z. Experimental animal models of pulmonary hypertension: Development and challenges. Anim Models Exp Med. 2022 Mar 25;5(3):207–16.
11. Liao J, Xie SS, Deng Y, Wu D dan, Meng H, Lan W fang, et al. PRDX6-mediated pulmonary artery endothelial cell ferroptosis contributes to monocrotaline-induced pulmonary hypertension. Microvasc Res. 2023 Mar 1;146:104471.
12. Cao X, Fang X, Guo M, Li X, He Y, Xie M, et al. TRB3 mediates vascular remodeling by activating the MAPK signaling pathway in hypoxic pulmonary hypertension. Respir Res. 2021 Dec 14;22(1):312.
13. Zhou J jing, Li H, Li L, Li Y, Wang P he, Meng X min, et al. CYLD mediates human pulmonary artery smooth muscle cell dysfunction in congenital heart disease-associated pulmonary arterial hypertension. J Cell Physiol. 2021;236(9):6297–311.
14. Brake O ter, Hooft K 't, Liu YP, Centlivre M, Eije KJ von, Berkhout B. Lentiviral Vector Design for Multiple shRNA Expression and Durable HIV-1 Inhibition. Mol Ther. 2008 Mar 1;16(3):557–64.
15. Henry SD, Wegen P van der, Metselaar HJ, Tilanus HW, Scholte BJ, Laan LJW van der. Simultaneous targeting of HCV replication and viral binding with a single lentiviral vector containing multiple RNA interference expression cassettes. Mol Ther. 2006 Oct 1;14(4):485–93.
16. Dunmore BJ, Jones RJ, Toshner MR, Upton PD, Morrell NW. Approaches to treat pulmonary arterial hypertension by targeting BMPR2: from cell membrane to nucleus. Cardiovasc Res. 2021 Oct 1;117(11):2309–25.
17. Rai N, Shihan M, Seeger W, Schermuly RT, Novoyatleva T. Genetic Delivery and Gene Therapy in Pulmonary Hypertension. Int J Mol Sci. 2021 Jan 25;22(3):1179.
18. Milone MC, O'Doherty U. Clinical use of lentiviral vectors. Leukemia. 2018 July;32(7):1529–41.
19. Bokhoven M, Stephen SL, Knight S, Gevers EF, Robinson IC, Takeuchi Y, et al. Insertional Gene Activation by Lentiviral and Gammaretroviral Vectors. J Virol. 2009 Jan;83(1):283–94.
20. Philpott NJ, Thrasher AJ. Use of Nonintegrating Lentiviral Vectors for Gene Therapy. Hum Gene Ther. 2007 June;18(6):483–9.
21. Dong W, Kantor B. Lentiviral Vectors for Delivery of Gene-Editing Systems Based on CRISPR/Cas: Current State and Perspectives. Viruses. 2021 July;13(7):1288.
22. Milani M, Annoni A, Moalli F, Liu T, Cesana D, Calabria A, et al. Phagocytosis-shielded lentiviral vectors improve liver gene therapy in nonhuman primates. Sci Transl Med. 2019 May 22;11(493):eaav7325.
23. Shirley JL, Jong YP de, Terhorst C, Herzog RW. Immune Responses to Viral Gene Therapy Vectors. Mol Ther. 2020 Mar 4;28(3):709–22.
24. Kwon J, Kim MY, Lee S, Lee J, Yoon HY. Pulmonary passage of canine adipose tissue-derived mesenchymal stem cells through intravenous transplantation in mouse model. J Vet Sci. 2024 May 7;25(3):e36.

Artificial Intelligence in Cardiology: From Concept to Clinical Reality

Snehal Sawant[1], Rajni Dawar [2]

1- Ex-Senior Resident, Department of Biochemistry VMMC and Safdarjung Hospital New Delhi

2- Professor, Department of Biochemistry VMMC and Safdarjung Hospital New Delhi

Introduction

Cardiology is among the most data-intensive medical specialties and is rich in electrocardiograms (ECGs), echocardiograms, angiograms, and continuous monitoring data from wearable devices. This wealth of data has created a natural playground for artificial intelligence (AI), especially machine learning (ML) and deep learning (DL) approaches.

In recent years, AI has transitioned significantly from research laboratories to practical applications in the clinical setting. Breakthroughs anticipated in 2024 and 2025 demonstrate its potential to identify silent cardiac diseases, automate imaging processes, predict patient outcomes, and tailor management strategies. As the global prevalence of cardiovascular disease continues to increase, artificial intelligence (AI) has the potential to enhance the accuracy, speed, and accessibility of cardiac care, especially in resource-limited settings.

AI in Electrocardiography: Revealing Hidden Signals

The 12-lead electrocardiogram (ECG), a longstanding cornerstone of cardiology, is transforming owing to AI. While traditional ECG interpretation primarily concentrates on abnormalities in rhythm and waveforms, deep neural networks can identify subtle electrical signals indicative of structural or functional heart disease, often before the symptoms manifest.

Prediction of Disease and Events

Attia et al. (Mayo Clinic) demonstrated that an AI-enabled ECG can detect asymptomatic left ventricular systolic dysfunction with accuracy comparable to that of echocardiography.[1]

Recent Updates (2025)

A convolutional neural network (CNN) study presented at ACC 2025 analyzed over 140,000 emergency department ECGs. It achieved an AUC ≈ 0.91 for predicting type 1 myocardial infarction and the need for revascularization — outperforming both clinicians and high-sensitivity troponin-T testing. [2]

Columbia University's "EchoNext" project demonstrated the capabilities of AI by utilizing 1.2 million ECG-echo pairs to identify structural heart disease (SHD) with an impressive 77% accuracy, outpacing the performance of 13 expert cardiologists, who achieved an average accuracy of only 64%. [3] These findings indicate a promising future, in which ECG can become an essential tool for screening silent cardiac diseases when paired with AI technology. This approach could help identify patients who require echocardiography or advanced imaging.

AI in Cardiac Imaging: Accuracy, Speed, and Standardization

Cardiac imaging is another significant area of AI integration. Techniques such as echocardiography, computed tomography (CT), and magnetic resonance imaging (MRI) produce extensive image datasets that require expert analysis. AI is capable of automating many of these tasks with impressive precision.

Echocardiography automation

DL models such as EchoNet-Measurements (ASE, 2025) trained on ~155,000 studies can automatically calculate 18 key echo parameters — chamber volumes, ejection fraction, and flow velocities — matching expert accuracy while cutting the reporting time. [4]

Real-World Integration:

In India, SGPGIMS Lucknow was one of the first centers to introduce an AI-enabled intravascular OCT system for precision angioplasty, providing 3D real-time vessel imaging and stent-positioning guidance.

AI for CT and MRI:

AI improves cardiac CT analysis by automating coronary calcium scoring and detecting nonobstructive plaques linked with future coronary events. In MRI, DL accelerates reconstruction and shortens scan times by 30–50 %. AI promises reproducibility, workflow efficiency, and greater access to high-quality imaging, particularly in centers lacking subspecialty expertise.

Wearables, Digital Stethoscopes, and Remote Monitoring

However, cardiac care is no longer confined to hospitals. AI embedded in consumers and medical devices enables continuous real-time monitoring.

AI-powered Stethoscopes

A 2025 multicenter UK trial (Imperial College London) tested an AI stethoscope that detects heart failure, valvular disease, and arrhythmias within 15 s. Among 12,000 patients, it doubled the diagnosis rate of heart failure and tripled the atrial fibrillation detection rate. [5]

Wearables and Patches:

Smartwatches now capture ECGs and photoplethysmographic data analyzed by cloud-based AI, enabling early arrhythmia detection.Hospitals in India (Jaslok and KEM, Mumbai) have recently deployed AI-assisted cardiovascular risk tools that combine wearable data with imaging metrics to flag high-risk patients within seconds. [6] Such devices democratize cardiac diagnostics by providing scalable and cost-effective solutions for early detection and remote disease management, which is an invaluable advancement for preventive cardiology and telemedicine.

AI in Clinical Decision Support and Predictive Analytics

The potential of AI extends well beyond diagnostics to encompass clinical decision-making support. AI systems can forecast adverse outcomes and facilitate personalized therapy by synthesizing patient demographics, laboratory results, imaging, and genomic data.

Outcome Prediction:

ML models have been developed to predict major adverse cardiac events (MACE), readmission risk, and treatment response.

AI for Personalized Therapy

Adaptive algorithms can optimize drug dosages and identify patients most likely to benefit from specific interventions (e.g., CRT, PCI).[7]

Large language Models (LLMs)

In 2025, comparative studies evaluated ChatGPT, Claude, and Gemini in order to answer cardiology queries. Although performance varied, the results suggest that LLMs could eventually support clinical documentation, patient communication, and trial recruitment under human supervision.

Clinical Evidence and Adoption

Until recently, much of the evidence has been retrospective in nature. However, prospective and randomized studies are emerging.

- AI-guided echocardiography helps non-expert operators acquire diagnostic-quality images. [4]
- AI-assisted ECG screening improves the detection of LV dysfunction and atrial fibrillation. [1,2]
- Wearable-based AI trials have demonstrated earlier detection of arrhythmias and reduced emergency visits. [5,6]

Regulatory bodies are responding: as of mid-2025, the U.S. The FDA has cleared over 750 AI/ML-enabled medical devices, many of which are in cardiology. [7,8] Despite regulatory momentum, real-world integration faces barriers, such as interoperability, clinician skepticism, and reimbursement gaps.

Challenges and Ethical Considerations

The success of AI is contingent not only on performance metrics, but also on building trust, ensuring fairness, and maintaining transparency.

1. Data Bias and Generalizability:

A significant number of AI models have been trained on datasets predominantly from Western contexts, which often underrepresent women and low-income populations. This can result in a potential bias in the predictions.

2. Explainability:

Clinicians need easily understandable information. When algorithms function as "black boxes," they can erode trust even if the results are accurate.[9]

3. Integration and Usability:

AI tools should be seamlessly integrated with existing electronic health records (EHR) and imaging systems to prevent disruptions in workflow.

4. Regulation and Liability

Determining who is responsible when AI errs remains legally complex.[6]

5. Data Privacy and Security

Wearables and remote sensors generate vast amounts of sensitive data, and cybersecurity and informed consent are essential.

6. Ethical AI Governance:

Transparent validation, biased audits, and human oversight are necessary to ensure equitable and safe use. [6]

Emerging Research and Future Directions

The application of AI in cardiology has advanced from mere diagnosis to prediction and prevention. The anticipated trends include the following.

Multimodal AI: This approach combines ECG, imaging, genomics, and lifestyle data to deliver comprehensive risk assessments and tailor therapeutic interventions.

Federated Learning: This innovative technique facilitates collaboration across multiple centers without the need to share raw patient data, ensuring the privacy and generalizability of findings.[3]

Interventional AI: This approach is to provide real-time AI support for angioplasty, OCT/OCT-AI analysis, and enhancing the precision of robotic cardiac procedures.

Large Language Models in Cardiology: Preliminary studies, such as the ILLUMINATE trial conducted in 2025, illustrate the potential of large language models (LLMs) to support interventional cardiologists by summarizing imaging reports, clinical guidelines, and notes.[10]

Outcome-Driven Trials: Future research will focus on patient-centered outcomes, including mortality rates, lengths of hospitalization, and cost-effectiveness, to establish clear, tangible benefits.

Global Accessibility: The next challenge is to scale AI solutions for low- and middle-income countries, ensuring an equitable distribution of benefits.[6]

Practical Takeaways for Clinicians

- AI is now a diagnostic tool and not a replacement. [7,8]
- We should look for tools with external validation and regulatory approval (e.g., FDA-cleared).
- We should recognize limitations of models—AI serves to enhance and not replace, clinical judgment.
- We should engage in local AI pilot projects and ensure familiarity with data privacy regulations.
- It promotes collaboration across disciplines by working alongside data scientists and engineers for effective contextual deployment.

Clinicians who develop AI literacy can be optimally positioned to lead the future of digital cardiology.

Conclusion

AI is transforming cardiology at every level from ECG interpretation and imaging automation to risk prediction and personalized therapy. Recent evidence from 2024 to 2025 shows that AI tools can outperform or complement clinicians in specific diagnostic tasks, streamline workflows, and extend cardiac care beyond hospitals. [1–8]

However, challenges of explainability, ethics, and equitable access will persist. The cardiologist in the future will not be replaced by AI, but will be empowered by it.

References

1. Attia ZI, Kapa S, Lopez-Jimenez F, et al. Screening for cardiac contractile dysfunction using an artificial intelligence-enabled electrocardiogram. *Nat Med*. 2019;25(1):70-74. doi:10.1038/s41591-018-0240-2
2. Büscher A. AI model shows high accuracy in heart attack detection. American College of Cardiology. 2025 Mar 29 [cited 2025 Oct 25]. Available from: https://www.acc.org/about-acc/press-releases/2025/03/29/13/45/ai-model-shows-high-accuracy-in-heart-attack-detection
3. Lapid N. Health Rounds: AI expands usefulness of common heart test. Reuters [Internet]. 2025 Jul 18 [cited 2025 Oct 25]. Available from: https://www.reuters.com/business/healthcare-pharmaceuticals/health-rounds-ai-expands-usefulness-common-heart-test-2025-07-18/
4. Sahashi, Y, Ieki, H, Yuan, V. et al. Artificial Intelligence Automation of Echocardiographic Measurements. *JACC.* 2025 Sep, 86 (13) 964–978.**https://doi.org/10.1016/j.jacc.2025.07.053**
5. Gregory A. Doctors develop AI stethoscope that can detect major heart conditions in 15 seconds. The Guardian. 2025 Aug 30. Available from: https://www.theguardian.com/society/2025/aug/30/doctors-develop-ai-stethoscope-that-can-detect-major-heart-conditions-in-15-seconds
6. Economic Times. Revolutionary AI model for heart disease prevention launched by Jaslok Hospital [Internet]. ETHealthworld. 2025 Jul 25 [cited 2025 Oct 25]. Available from: https://health.economictimes.indiatimes.com/news/hospitals/revolutionary-ai-model-for-heart-disease-prevention-launched-by-jaslok-hospital/122899216

7. Visan AI, Negut I. Integrating Artificial Intelligence for Drug Discovery in the Context of Revolutionizing Drug Delivery. *Life*. 2024; 14(2):233. https://doi.org/10.3390/life14020233

8. van der Harst P. Introducing the 'Transforming cardiology with AI' series. Neth Heart J. 2025;33:111. doi:10.1007/s12471-025-01948-1.

9. Marey, A., Arjmand, P., Alerab, A.D.S. *et al.* Explainability, transparency and black box challenges of AI in radiology: impact on patient care in cardiovascular radiology. *Egypt J Radiol Nucl Med* **55**, 183 (2024). https://doi.org/10.1186/s43055-024-01356-2

10. Chen J, Liang Y, Ge J. Artificial Intelligence Large Language Models in Cardiology. *Rev Cardiovasc Med*. 2025;26(7):39452. Published 2025 Jul 8. doi:10.31083/RCM39452

Artificial Intelligence and Coronary Artery Disease: Transforming Diagnosis and Prevention in the Young

Annu Kumari, Kamna Srivastava

Dr. B. R. Ambedkar Centre for Biomedical Research, University of Delhi, Delhi, India- 110007

Corresponding Author: Kamna Srivastava (kamna605@gmail.com; ksrivastava@acbr.du.ac.in)

Introduction

Historically viewed as an age-dependent ailment stemming from cumulative vascular stress, Coronary Artery Disease (CAD) now presents a critical public health challenge due to a global surge in premature atherosclerosis and myocardial infarction in adults under 45 [1]. This emerging phenotype is often metabolically silent, lacking the conventional clinical markers such as hypercholesterolemia and diabetes, yet concealing significant underlying endothelial dysfunction and microvascular inflammation [2]. This profound disconnect highlights the obsolescence of traditional risk prediction tools, such as the Framingham and Atherosclerotic Cardiovascular Disease (ASCVD) scores, which fail to capture the nuanced pathophysiology driven by modern lifestyle, psychosocial stressors, and genetic predisposition [3] [4]. Computational science is uniquely positioned to decipher these complex disease mechanisms, enabling early detection and the development of personalised medicine. The convergence of Machine Learning (ML) and Deep Learning (DL) within Artificial Intelligence (AI) offers an urgent solution to this rising lifestyle epidemic [5]. AI is ushering in a new era of preventive cardiology, moving beyond outdated metrics to provide targeted, precise intervention for the young at-risk population.

Role of computational cardiology in decoding CAD pathophysiology

Computational science has fundamentally redefined the investigation of CAD pathophysiology. Research focusing on premature acute coronary syndrome patients has identified multi-omics signatures, encompassing the transcriptome, metabolome, and gut microbiome, that differentiate this young subpopulation from the general CAD cohort [6]. At the technical core, AI architectures facilitate the multi-omics integration and synthesis of these heterogeneous datasets, crucial for deciphering the disease's complex molecular fingerprint.

DL models are leveraged to generate granular, disease-specific risk quantifications, such as MetScore (derived from hundreds of metabolites) and ProScore (from thousands of proteins) [7], which, in synergy with traditional clinical predictors, enhance risk stratification by offering the mechanistic insights necessary for identifying novel biomarkers and therapeutic targets. Complementary Neural Networks model can predict the nonlinear relationships between gene expression and clinical outcomes, illuminating the cryptic molecular drivers of early atherogenesis [5].

Moreover, ML has successfully delineated inflammatory expression modules in peripheral blood mononuclear cells (PBMCs) [8]. Integrating these modules with serum metabolite profiles indicative of lipid oxidation can construct a holistic view of systemic inflammation. Critically, these multi-omics

signatures often precede overt arterial stenosis, establishing a vital temporal window for primary prevention in young adults [6]. Further in silico analyses using graph theory models elucidate affected molecular networks, predict the systemic impact of molecular perturbations and identify hub molecules that can function as potential therapeutic leverage points, thereby informing the rational design of highly targeted drug interventions. Collectively, this multi-omics AI profiling empowers a shift from traditional risk management to personalised, pre-symptomatic identification and precision primary prevention for this high-risk demographic.

AI innovations in heart imaging and risk assessment

AI is transforming cardiovascular diagnostics by enhancing the interpretation of imaging modalities central to CAD evaluation. DL models, specifically utilising Convolutional Neural Networks (CNNs), are being applied across modalities such as Coronary CT Angiography (CCTA), Intravascular Ultrasound (IVUS), and Optical Coherence Tomography (OCT) for the automated, objective quantification of critical metrics [9]. These models precisely characterise plaque morphology by classifying vulnerable plaque components and estimating physiological parameters like fractional flow reserve (FFR) directly from anatomical images. AI-assisted CCTA excels at subtle detection, identifying high-risk non-calcified plaques and positive remodelling patterns often missed by human readers, thus enabling earlier and more rigorous risk assessment, especially for asymptomatic young cohorts [10].

A significant enhancement comes from Radiomics, which extracts quantitative descriptors of plaque texture, density, and shape from images. Integrating these rich features into ML models significantly boosts the prediction of future cardiac events beyond conventional imaging metrics [11]. Additionally, temporal AI models analyse longitudinal image series to track disease progression and evaluate therapeutic efficacy [12]. For high-risk individuals, such as young diabetics or those with strong family histories, these systems establish personalised, non-invasive surveillance strategies, tailoring monitoring frequency and intervention timing to individual disease dynamics. This integration of advanced imaging AI shifts the focus from simple visual assessment to precise, quantitative, and predictive diagnostic cardiology.

AI and digital phenotyping for heart health

Beyond personalised diagnostics, AI is driving a paradigm shift in healthcare, moving toward proactive public health strategies. At the heart of this transformation is the integration of heterogeneous data streams such as Electronic Health Records (EHRs), biometric data from wearable devices, and lifestyle information, utilised to construct dynamic risk profiles for early disease detection [13].

AI platforms are particularly effective in analysing continuous digital data generated from wearable technologies. Metrics such as heart rate variability, sleep quality, and stress levels are strongly associated with early autonomic imbalance, a recognised precursor to endothelial dysfunction. Detecting these deviations prior to clinical manifestation enables timely intervention, especially in

resource-limited settings where access to advanced healthcare infrastructure is constrained [13]. Natural Language Processing (NLP) further enhances these platforms by extracting clinically relevant insights from unstructured medical data. This ability is essential for identifying subtle indicators of disease progression and uncovering latent risk factors that may otherwise remain obscured in narrative clinical documentation [14].

At the same time, to ensure model robustness and data privacy, Federated Learning (FL) has emerged as a promising solution. FL allows AI systems to be trained across decentralised datasets from multiple hospitals and research institutions without aggregating sensitive patient information [15]. This decentralised approach preserves confidentiality while facilitating the development of accurate, demographically diverse predictive models, particularly effective in identifying coronary artery disease (CAD) risk among younger populations. Collectively, these innovations highlight AI's transformative potential in cardiovascular care, fostering a shift toward anticipatory, data-driven public health interventions that improve early detection, optimise resource allocation, and enhance population health outcomes.

AI-accelerated drug discovery for cad therapeutics

Computational drug modelling, powered by AI, is drastically accelerating the discovery and optimisation of novel therapeutics for CAD. Molecular docking simulations are leveraged to precisely predict the binding affinity and interaction mechanisms of bioactive compounds with critical high-priority targets, such as oxidised LDL receptors, specific mitochondrial chaperones, or key inflammatory kinases [16]. This predictive capacity streamlines target validation. Further extending this capability, deep generative models are now employed to design de novo molecules endowed with specific, desirable pharmacological properties, thus circumventing traditional limitations and dramatically compressing early-stage drug development timelines.

AI-assisted virtual screening libraries enable the simultaneous in silico evaluation of thousands of potential compounds. This allows researchers to rapidly prioritise candidates with the highest likelihood of achieving translational goals, such as reversing endothelial dysfunction or chemically stabilising vulnerable atherosclerotic plaques [16]. The iterative refinement of these virtual models is critically informed by integrating real-world biochemical data, ensuring higher fidelity and maximising their translational value toward clinical application.

Challenges and ethical imperatives in the integration of AI

Despite remarkable potential, the translation of AI and computational tools into clinical cardiology faces hurdles. Data standardisation, interpretability of AI models, and potential biases in training data can limit adoption. Historical clinical datasets often exhibit inadequate representation of specific age cohorts, including healthy young adults, and minority groups. Applying models trained on older populations to young patients risks perpetuating diagnostic bias, potentially leading to the

underestimation of genuine early-stage risk [17]. Ethical implications surrounding patient privacy and algorithmic transparency require continuous oversight. Importantly, clinicians must remain central to AI workflows, acting not merely as end-users but as active contributors guiding model design, validation, and ethical governance. The opacity and black-box problem of understanding complex DL models underscores the need for Explainable AI (XAI), which provides interpretable outputs that clinicians can trust when making high-stakes decisions, especially in asymptomatic young individuals who may benefit from early preventive interventions [18]. Simultaneously, the reliance on sensitive multi-omics data mandates rigorous privacy and security protocols. FL offers a viable solution, enabling models to be trained across decentralised datasets to enhance generalisation while maintaining data confidentiality.

Conclusion

Ultimately, successful clinical integration for this young population requires balanced frameworks where computational precision is meticulously complemented by psychosocial support and targeted interventions, mitigating the risk of over-diagnosis and unnecessary anxiety. This holistic approach is essential to realize the potential of AI to prevent irreversible damage decades in advance.

Bibliography

1. Mir MA, Dar MA, Qadir A (2024) Exploring the Landscape of Coronary Artery Disease: A Comprehensive Review. Am J Biodivers 1:
2. Kayikcioglu M, Ozkan HS, Yagmur B (2022) Premature Myocardial Infarction: A Rising Threat. Balk Med J 39:83–95. https://doi.org/10.4274/balkanmedj.galenos.2022-2-19
3. Bansal A, Hiwale K (2024) Updates in the Management of Coronary Artery Disease: A Review Article | Cureus. Cureus 15:. https://doi.org/10.7759/cureus.50644
4. Abohelwa M, Kopel J, Shurmur S, et al (2023) The Framingham Study on Cardiovascular Disease Risk and Stress-Defenses: A Historical Review. J Vasc Dis 2:122–164. https://doi.org/10.3390/jvd2010010
5. Khatoon F, Ginawi NAA, Alshammeri AAF, et al (2025) The Role of Artificial Intelligence in Early Detection and Risk Stratification of Coronary Artery Disease Using ECG and Imaging Data. J Pioneer Med Sci 14:103–107. https://doi.org/10.47310/jpms2025140913
6. Zhan C, Tang T, Wu E, et al (2023) From multi-omics approaches to personalized medicine in myocardial infarction. Front Cardiovasc Med 10:. https://doi.org/10.3389/fcvm.2023.1250340
7. AI-based multiomics profiling for personalized prediction of cardiovascular disease: A prospective UK Biobank study | Sciety. https://sciety.org/articles/activity/10.21203/rs.3.rs-6139124/v1. Accessed 26 Oct 2025
8. Bondar G, Cadeiras M, Wisniewski N, et al (2014) Comparison of Whole Blood and Peripheral Blood Mononuclear Cell Gene Expression for Evaluation of the Perioperative Inflammatory Response in Patients with Advanced Heart Failure. PLOS ONE 9:e115097. https://doi.org/10.1371/journal.pone.0115097

9. A systematic review on deep learning-enabled coronary CT angiography for plaque and stenosis quantification and cardiac risk prediction - ScienceDirect. https://www.sciencedirect.com/science/article/pii/S235204772500019X. Accessed 26 Oct 2025
10. Advances in Artificial Intelligence-Assisted Coronary Computed Tomographic Angiography for Atherosclerotic Plaque Characterization - PMC. https://pmc.ncbi.nlm.nih.gov/articles/PMC11262402/. Accessed 26 Oct 2025
11. Kahmann J, Nörenberg D, Papavassiliu T, et al (2024) Combined conventional factors and the radiomics signature of coronary plaque texture could improve cardiac risk prediction. Insights Imaging 15:170. https://doi.org/10.1186/s13244-024-01759-9
12. Oladele OK (2025) AI for Longitudinal Analysis: Tracking Disease Progression Over Time. https://www.researchgate.net/publication/389909343_AI_for_Longitudinal_Analysis_Tracking_Disease_Progression_Over_Time?enrichId=rgreq-ddf34364cf738a9db2ef406bdd120c30-XXX&enrichSource=Y292ZXJQYWdlOzM4OTkwOTM0MztBUzoxMTQzMTI4MTMxNjcyNjIzOEAxNzQyMjMzMDUzMTUx&el=1_x_2&_esc=publicationCoverPdf. Accessed 26 Oct 2025
13. Integrating AI-driven wearable devices and biometric data into stroke risk assessment: A review of opportunities and challenges - ScienceDirect. https://www.sciencedirect.com/science/article/pii/S0303846724005766. Accessed 26 Oct 2025
14. Natural Language Processing (NLP) in Healthcare AI: Enhancing Clinical Insight Extraction from Unstructured Patient Data -. https://www.authorea.com/doi/full/10.22541/au.174845034.41890518. Accessed 26 Oct 2025
15. Moshawrab M, Adda M, Bouzouane A, et al (2023) Reviewing Federated Machine Learning and Its Use in Diseases Prediction. Sensors 23:2112. https://doi.org/10.3390/s23042112
16. May LT, Bartolo BA, Harrison DG, et al (2022) Translating atherosclerosis research from bench to bedside: navigating the barriers for effective preclinical drug discovery. Clin Sci 136:1731–1758. https://doi.org/10.1042/CS20210862
17. Predicting coronary heart disease with advanced machine learning classifiers for improved cardiovascular risk assessment | Scientific Reports. https://www.nature.com/articles/s41598-025-96437-1. Accessed 26 Oct 2025
18. Marey A, Arjmand P, Alerab ADS, et al (2024) Explainability, transparency and black box challenges of AI in radiology: impact on patient care in cardiovascular radiology. Egypt J Radiol Nucl Med 55:183. https://doi.org/10.1186/s43055-024-01356-2.

Modern and Investigational Approaches for Treating Atherosclerosis with Novel Mechanisms

Arpan Pathak[1], Swati Kaushik[1]

School of Medical and Allied Sciences, K R Mangalam University, Gurugram

Abstract

Treatment for atherosclerosis is shifting beyond lipid lowering with statins and Proprotein convertase subtilisin/kexin type 9 (PCSK9) monoclonal antibodies toward innovative, durable and mechanism-targeted therapies: specific *In-vivo* Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) of hepatic PCSK9, potent Lipoprotein(a) [Lp(a)]-lowering oligonucleotides/siRNA, and NLR family pyrin domain containing 3 (NLRP3) inflammasome inhibitors. Each of these interventions presents paradigm shifts in a one-time genome editing therapy, treatment of previously untreatable risk factors, and direct modulation of the arterial inflammatory process. While early data in humans and translational studies are reassuring, longer-term safety and outcome data are yet to be observed and understood.

Keywords: Atherosclerosis, Novel therapeutic measures, molecular mechanisms

Introduction

Atherosclerotic cardiovascular disease (ASCVD) remains the leading cause of death globally. Traditional pharmacological options including ezetimibe, monoclonal antibodies to PCSK9 and statins, may effectively lower low-density lipoprotein (LDL)-cholesterol and decrease cardiovascular events, but still, significant residual risk exists. Genetic risk factors like chronic vascular inflammation, lipoprotein(a) [Lp(a)] and long-term treatment adherence demonstrate the need for more durable and mechanistically targeted therapies **(Al-Jabri and Al-Zakwani., 2024; Boffa and Koschinsky., 2023)**. New investigational strategies are currently being studied in clinical trials, with the goal of silencing previously unmodifiable genetic risks, durably modifying these causal pathways, and modifying key mechanisms of inflammation that promote plaque growth and instability **(Nissen SE., 2024; Choudhury et al., 2025)**.

In-vivo Base Editing of PCSK9: A Single Gene Editing Approach

A truly game-changing approach is the In-vivo base editing of the PCSK9 gene, a gene editing technique that is the first use of gene editing for the treatment of cardiovascular disease. This method employs lipid nanoparticle-delivered adenine base editors to add a specific single-base change to the hepatic PCSK9 gene, resulting in a permanent reduction in PCSK9 protein and sustained reduction in LDL-cholesterol **(Musunuru and Chadwick., 2024; Lee et al., 2023)**. Conventional editing systems using CRISPR methods lead to double-strand DNA breaks. Base editing specifically substitutes a single nucleotide without cleaving either strand of DNA, therefore reducing the potential for genomic instability **(Musunuru and Chadwick., 2024)**.

Programs VERVE-101 and next-generation VERVE-102 by Verve Therapeutics are actively testing this idea in humans. VERVE-101 was shown to induce dose-dependent reductions in circulating LDL-C (by 55%) and PCSK9 (by 84%) following a single infusion **(Vafai et al., 2023)**. Then it was placed on a clinical hold based on an adverse event that happened in patients on the lowest dose. After careful evaluation of the emerging safety data, it was determined that the infusion and, therefore, the effects could be optimized, which led to the development of VERVE-102 and an improved delivery system. The early results of the ongoing Heart-2 study of VERVE-102 indicate that high-dose cohorts have achieved sustained reductions in LDL-C of over 50%, and the tolerability profile is favorable in very small groups of patients **(Verve Therapeutics, 2025)**. VERVE-102 has also received U.S. FDA Fast Track Designation **(Verve Therapeutics, 2024)**. Overall, these results, to this point, speak to the potential for permanent LDL-C lowering with a single intervention; however, long-term safety needs to be documented, particularly hepatic toxicity and off-target genomic effects, before clinical implementation.

Lipoprotein(a) Lowering with Oligonucleotides and siRNA

In addition to genome editing, RNA-based therapies aimed at lipoprotein(a) have emerged as extremely promising. Elevated Lp(a) is a genetically determined, independent risk factor for ASCVD and calcific aortic valve disease - and no conventional therapies have lowered Lp(a) levels. Agents targeting RNA, such as antisense oligonucleotides (ASOs) and small interfering RNA (siRNA), can inhibit hepatic production of apolipoprotein(a), the building block of Lp(a), and lower plasma levels of Lp(a) by >90% **(Furtado and Jukema., 2023)**.

Pelacarsen, an **antisense oligonucleotide (ASO)** agent developed by Novartis, and Olpasiran, a siRNA agent from Amgen, both have demonstrated remarkable and sustained biochemical efficacy in phase 2 studies. Pelacarsen is currently being tested in the phase 3 Lp(a)HORIZON trial to determine if lowering Lp(a) will result in fewer cardiovascular events **(Cho et al., 2025)**. Olpasiran data reveals a sustained 40-50% lower Lp(a) levels in participants nearly one year after dosing **(O'Donoghue et al., 2024)**. Collectively, both new agents are a monumental event in precision lipid management - targeting a causal risk factor that until now was not treatable. Successful development of these therapies may alter the future of primary and secondary cardiovascular prevention in genetically predisposed populations, while event reduction has yet to be demonstrated.

Table 1: *Mechanistically Targeted Investigational Therapies for Atherosclerotic Cardiovascular Disease (ASCVD).*

Therapy	Target	Mechanism of Action	Stage of Development	Key Outcomes / Notes
In-vivo PCSK9 Base Editing (VERVE-101 / VERVE-102)	PCSK9 gene	Adenine base editor introduces single-nucleotide change in hepatic PCSK9 gene leads to permanent LDL-C reduction	Early human trials (Phase 1/2)	Up to 84% PCSK9 reduction, 50% LDL-C reduction after a single dose; long-term safety under evaluation.
NLRP3 Inflammasome Inhibitors (MCC950, Dapansutrile)	NLRP3 inflammasome	Small molecules inhibit NLRP3 inflammasome and also reduce IL-1beta/IL-18 mediated vascular inflammation	Preclinical / Early human trials	Reduces vascular inflammation and potentially stabilizes plaques; safety and immune modulation monitored.
Lipoprotein(a) siRNA (Olpasiran)	Apolipoprotein(a)	Small interfering RNA silences hepatic apo(a) production lowers plasma Lp(a)	Phase 2 / long-term follow-up	Sustained 40-50% Lp(a) reduction off-treatment for 1 year; durable effect.
Lipoprotein(a) ASO (Pelacarsen)	Apolipoprotein(a)	Antisense oligonucleotide reduces hepatic apo(a) synthesis lowers plasma Lp(a)	Phase 3 [Lp(a)HORIZON]	>90% Lp(a) reduction in phase 2; cardiovascular outcome trial ongoing.

ASO: Antisense Oligonucleotide; LDL-C: Low-Density Lipoprotein Cholesterol; Lp(a): Lipoprotein(a); NLRP3: Nucleotide-binding Oligomerization Domain, Leucine-rich Repeat and Pyrin Domain-containing 3; PCSK9: Proprotein Convertase Subtilisin/Kexin type 9; siRNA: Small Interfering RNA; IL-1beta/ IL-18: Interleukin-1 beta / Interleukin-18 (Pro-inflammatory cytokines).

NLRP3 Inflammasome Inhibitors - Targeting Vascular Inflammation

In addition to lipid modification, chronic vascular inflammation is increasingly being recognized as a primary driver of plaque vulnerability and atherosclerosis progression. The favorable results of the CANTOS trial with canakinumab, an IL-1beta monoclonal antibody, highlighted the potential value of anti-inflammatory approaches to therapy. Following up on these findings, oral, small-molecule inhibitors of the NLRP3 inflammasome are being developed as an alternative way to down-regulate upstream inflammatory signals **(Latz and Duewell., 2023)**.

The NLRP3 inflammasome regulates the activation of IL-18 and IL-1beta and is an important mediator of sterile inflammation within atherosclerotic plaques. For example, dapansutrile (OLT1177) and

MCC950 have shown preclinical evidence of inhibition of NLRP3 activation and downstream cytokine expression while early-phase trials show biological activity and safety in humans **(Abbasi et al., 2024)**. Targeting NLRP3 is an attractive mechanism for reducing vascular inflammation aimed at stabilizing a vulnerable plaque while providing adjunctive therapy to statins. However, there remains the challenge of systemic immune suppression which can lead to infection risk and will require careful clinical assessment.

Translational Perspective and Implications

The combination of gene editing, RNA silencing, and inflammasome inhibition signifies a transformative development in the treatment of atherosclerosis. The *In-vivo* PCSK9 base-editing method allows for the potential of a single treatment option with lifelong results in reducing LDL-cholesterol; oligonucleotide therapies that target Lp(a) provide a mechanism of precision therapy toward a major genetic determinant of risk and NLRP3 inhibitors tackle the inflammatory basis of plaque development itself. Collectively, these developments expand the scope of cardiovascular therapeutics from controlling disease to possible molecular alterations **(Prajapati et al., 2024)**.

The potential for these novel treatments is embedded not only in the mechanistic frameworks, but also in their ability to reduce the complexity of lifelong disease management. But we now need long-term safety data, large outcome studies, and cost-effectiveness data to help us confirm that any perceived mechanistic advantage is eventually translated into reducing stroke, myocardial infarction, or mortality.

Future Directions and Research Priorities

Future strategies should place strong consideration on longitudinal safety registries for *In-vivo* gene editing, to ensure genomic monitoring and functional assessment of outcomes. Larger event driven studies, such as Lp(a)HORIZON, are likely to demonstrate whether Lp(a) reduction is clinically beneficial. Important translational biomarkers need to be developed to quantitatively link inhibiting the inflammasome with plaque stabilization using advanced imaging, such as Medical Resonance Imaging (MRI) and Positron Emission Tomography (PET). Finally, investigating combination strategies that reduce lipids along with controlling inflammation has potential to completely attenuate cardiovascular risk in atherosclerosis.

Conclusion

The most innovative and least studied therapies of clinical development for atherosclerosis are the *In-vivo* base editing of PCSK9, siRNA agents and potent oligonucleotides that lower Lp(a), and inhibitors of the NLRP3 inflammasome. All these agents target different mechanistic pillars of the disease (i.e. genetic predispositions, inflammation and lipid metabolism) and signify the beginning of durable and personalized cardiovascular therapy. Early data are promising, but the key challenge for these innovative therapies will be moving from biomarker efficacy to proven reductions in cardiovascular events.

References

1. Abbasi A, et al. Targeting the NLRP3 Inflammasome in Atherosclerosis: Mechanisms and Therapeutic Potential of Small-Molecule Inhibitors. *Cardiovasc Res*. 2024;120(5):1018-1033.
2. Al-Jabri AA, Al-Zakwani I. Residual Cardiovascular Risk: Beyond Low-Density Lipoprotein Cholesterol—Inflammation, Triglycerides, and Lipoprotein(a) as Emerging Targets. *Curr Atheroscler Rep*. 2024;26(5):42. doi:10.1007/s11883-024-01201-8.
3. Boffa MB, Koschinsky ML. The Genetics, Biochemistry, and Emerging Treatments for Lipoprotein(a) in Cardiovascular Risk. *J Clin Invest*. 2023;133(22):e172778. doi:10.1172/JCI172778.
4. Cho L, et al. Design and rationale of the Lp(a)HORIZON trial: a phase 3 randomized controlled trial to evaluate the effect of pelacarsen on cardiovascular outcomes in patients with elevated lipoprotein(a) and established cardiovascular disease. *J Clin Lipidology*. 2025.
5. Choudhury RP, et al. Next-generation treatments for atherosclerosis. *Cell Rep Med*. 2025;6(2):100361.
6. Furtado DV, Jukema J W. The Rise of Lipoprotein(a): Current Evidence and Future Therapies with Focus on Olpasiran. *Eur Heart J Cardiovasc Pharmacother*. 2023;9(6):506-515. doi:10.1093/ehjcvp/pvad023.
7. Latz E, Duewell, P. The NLRP3 Inflammasome: A Target for Atherosclerosis Therapy. *Nat Rev Cardiol*. 2023;20(8):517-531. doi:10.1038/s41569-023-00839-8.
8. Musunuru K, Chadwick AC. Base Editing for In Vivo Genome Editing: Implications for Cardiovascular Disease. *Circ Res*. 2024;134(3):253-268. doi:10.1161/CIRCRESAHA.123.323533.
9. Nissen SE. Targeting Residual Risk in Atherosclerotic Cardiovascular Disease: New Approaches to an Old Problem. *N Engl J Med*. 2024;390(13):1238-1249. doi:10.1056/NEJMra2311749.
10. O'Donoghue ML, et al. The Off-Treatment Effects of Olpasiran on Lipoprotein(a): Results from the OCEAN(a)-DOSE Study. *JAMA Cardiol*. 2024;9(4).
11. Prajapati V, et al. The Future of Atherosclerosis Therapy: From Lipid Lowering to Gene Editing and Beyond. *Cardiology*. 2024;149(3):145-155.
12. Lee RG, Mazzola AM, Braun MC, Platt C, Vafai SB, Kathiresan S, Rohde E, Bellinger AM, Khera AV. Efficacy and safety of an investigational single-course CRISPR base-editing therapy targeting PCSK9 in nonhuman primate and mouse models. Circulation. 2023 Jan 17;147(3):242-53.
13. Vafai SB, Gladding PA, Scott R, Kerr J, Taubel J, Cegla J, et al. Safety and Pharmacodynamic Effects of VERVE-101: An Investigational DNA Base Editing Medicine Designed to Durably Inactivate the PCSK9 Gene and Lower LDL-C. *Circulation*. 2023;10(2):89.
14. Verve Therapeutics. VERVE-102 Program: A Single-Course Gene Editing Medicine Targeting PCSK9. 2025 Apr 14. [company press release].
15. "Verve Therapeutics Receives U.S. FDA Fast Track Designation for VERVE-102." [Press release]. 2024.

Targeted Innovation: GalNAc-Steered Delivery of Inclisiran to Hepatocytes for the Treatment of Hypercholesterolemia.

Aditi Singh[1], Pinky Garg[2], Harsh Vardhan Singh[3], Rajeev Ranjan[4].

[1]Associate Professor, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi.

[2] Professor, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi.

[3]Senior Biochemist, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi.

[4]Head, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi.

Abstract:

Inclisiran is a small interfering RNA(SiRNA), designed to mute the PCSK9 gene. SiRNA is a double stranded RNA molecule, which is non coding. These are short 21-25 base pairs with hydroxylated 3' end and phosphorylated 5' end. SiRNA enables gene targeted silencing. PCSK9(Proprotein Convertase Subtilin/Kexin type 9) protein produced from liver and secreted in blood stream, interacts with LDL receptors on the hepatocytes. It forms a complex with LDL particles, which undergo degradation in the lysosome. This produces decreased number of LDL receptor on hepatocytes and hence decreased ability to remove bad cholesterol ,ie LDL from blood.

Mechanism of action of SiRNA is that it interferes with the gene expression of PCSK9 in hepatocytes, causing decreased PCSK9 levels and increased LDL receptors with better removal of LDL Cholestrol from circulation. Moreover, conjugation of Inclisiran with GalNAc (N-acetyl galactosamine) ensures specificity of Inclisiran on targeted hepatocyte action. The Asialoglycoprotein receptor(ASGPR) are present on liver cells only. These receptors have high affinity for glycoprotein, terminating with GalNAc.

The Inclisiran SiRNA is chemically linked with three GalNAc residues. These groups are recognised by ASGPR on hepatocytes. Once bound the complex is internalised into the liver cells through endocytosis. SiRNA released into cytoplasm meets RNA induced Silencing Complex(RISC), to degrade PCSK9 mRNA. Later the ASGPR receptors are recycled by returning back on the cell membrane of the hepatocytes.

Summary:

Inclisiran (GalNac conjugate)→Binds to ASGPR on liver cell→Receptor mediated endocytosis→Inclisiran internalised into liver cell→Enters RNA induced Silencing Complex(RISC) of PCSK9→Cleaves PCSK9 mRNA→Decreased synthesis of PCSK9→Increased LDL Receptor recycling→Increased LDL uptake by hepatocytes→Decreased Plasma LDL-C levels.

Introduction:

Cardiovascular diseases remains to be a leading cause of morbidity and mortality across the world. Elevated LDL-C is a major modifiable risk factor. Traditional lipid-lowering therapies like statins significantly improves the outcome. Still a major population with hypercholesterolemia is not able to achieve LDL-C goals despite taking all the therapies. Thus improved technologies on novel mechanism of action has always been focussed upon in innovation.

Inclisiran is one of such innovation, a small interfering SiRNA therapeutics that attacks mRNA of PCSK9. PCSK9 is a liver derived protein that binds to hepatocyte LDL receptor (LDLRs) and assist in their degradation. Moreover, reduced number of LDLR causes decreased hepatic clearance of LDL-C from plasma. LDLR levels increased by inhibiting production of PCSK9 protein and enhanced clearence of LDL-C. Inclisiran produces sustained LDL-C reduction by 50%. However, delivery is the key challenge in nucleic acid based therapies like SiRNAs.

To avoid rapid plasma degradation and off target uptake, therapeutic molecule must ensure to reach the correct cell type. So targeted delivery innovation plays a critical role in the treatment.

GalNAc -Mediated Hepatocyte Targeting:

The hepatocytes express high level of Asialoglycoprotein receptor(ASGPR) on its surface. It recognises GalNAc molecules. The Inclisiran conjugated with triantennary N-Acetylgalactosamine ligand bind with high affinity ASGPR and is rapidly taken up into liver cells via receptor-mediated endocytosis. Once internalised, the SiRNA enters RNA-induced silencing Complex(RISC) and guides cleavage of PCSK9 mRNA and thereby reducing production of PCSK9 protein.

The Advantages of GalNAc-steered mechanisms:

1. Hepatocyte Specificity-Directing the therapeutic primarily to liver cell. Off target tissue exposure is minimal. Improving safety and reducing the required dose.
2. Low Systemic Exposure- The plasma level of Inclisiran declines rapidly and is undetectable in 24-48 hrs and thereby reducing systemic side effects.
3. Sustained duration of Action-One SiRNA loaded RISC Complex can cleave many mRNA in liver cells. The LDL-C lowering effect persists for months. Dose every six months after initial loading.
4. Lower dose requirement-Due to high specificity and efficient uptake, smaller cumulative dose required. Reduces risk of immune reaction, systemic side effects and cost as well.

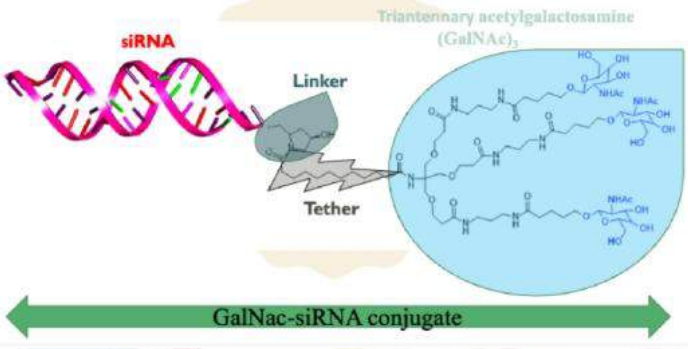


Figure: Chemical structure of triantennary acetylgalactosamine (GalNAc) with siRNA.

Mechanism of Action of Inclisiran:

Once Inclisiran GalNAc-SiRNA is administered subcutaneously, the GalNAc ligand binds to ASGPR strand on hepatocyte, triggering internalisation of complex via clathrin-coated vesicle and endosomal transport. The ASGPR is recycled back to liver cell surface while GalNAc is degraded. In hepatocytes SiRNA emerges into the cytoplasm and combines with RISC. The messenger strand is degraded and guide strand remains complexed. The guide strand pairs with PCSK9 mRNAand directs the cleavage, preventing translation of PCSK9 protein . Reduced PCSK9 level allow more LDLRs on the liver cell surface, increases hepatic LDL-C uptake and reduces the circulating LDL-C.

The upstream intervention due to targeting hepatocyte have advantage in durability and mechanism of action. The drug act at the site of PCSK9 mRNA and production of PCSK9 protein is reduced .

Clinical Innovation and Implications:

Inclisiran represent an important innovation in hypercholesterolemia management from therapeutic point of view. In the pivotal phase 2 and phase 3 trials, patients receiving Inclisiran achieved 50% reduction in LDL-C with acceptable safety profile. Twice yearly dose ie a loading dose enhances patient adherence, a major real world barrier in lipid loweing therapy. Regulatory approval reflect the innovative broader context of targeted innovation. Inclisiran GalNAc delivery strategy sets a model for future level targeted RNA therapies for metabolic disorder. This suggest that other genes expressed in hepatocytes may be manipulated with similar specificity and durability.

Challenges and Considerations:

Safety data in long term is still awaited, inspite of hepatocyte specificity. Specially in diverse populations and persons with hepatic impairment. Some mild elevation of liver enzymes observed may be attributed to concomitant therapy rather than Inclisiran itself.

The cost effectiveness relative to conventional therapies and monoclonal antibodies need to be evaluated in practice.

Patients selection and long term cardiovascular outcome beyond LDL-C reduction like major cardiovascular accident is still under study. The delivery mechanism relies on ASGPR intact function. Patients with mild/moderate /severe liver disease on altered receptor expression respond differently.

Conclusion:

The GalNAc mediated targeting of Inclisiran to liver cell exemplifies an elegant and highly effective example of targeted innovation by leveraging advanced Biochemistry (SiRNA), Precision delivery (GalNac-ASGPR) and metabolic Biology(PCSK9-LDLR axis) to manage Hypercholestremia. The success of this platform underscore the significance of not only identifying the right molecular target(PCSK9) but also delivering therapy to the right cell(hepatocyte) at right time, with optimal

duration and safety. Future therapies may adopt similar liver targeting approaches for other metabolic and cardiovascular diseases.

REFERENCES:

1. Lamb Y N. Inclisiran;4th approval.Drugs2021;81(3):389-395.
2. Ray K K et al. Dawn of new era of Targeted Lipid Lowering Therapies. Eur Heart J.2022;43(34):3108.
3. Nishikid T, Ray KK. Harnessing RNA Interference for cholesterol lowering: The bench to bedside story of Inclisiran. Clin Pharmacol Ther 2022.
4. E Oluwakemi, C Pietro, Wong GK ,et al. Development of Novel SiRNA Therapeutics: A Review with a focus on Inclisiran for the treatment of Hypercholestremia. Int J Mol Sci.2023.
5. Dec A, Niemiec A, Wojciechowska E, et al. Review Reports-Inclisiran: A Revolutionary Addition to a Cholestrol lowering therapy. Int J Mol Sci.2023:24(7):6858.

Silent but Deadly: Oxidative Stress and Lipid Indices in Early Atherogenesis

AK Singhal[1], AK Harith[2], (Maj Gen) TK Saha[3]

[1]Associate Professor, Department of Biochemistry, Amrita School of Medicine Faridabad

[2]Professor, Department of Biochemistry, Amrita School of Medicine Faridabad

[3]Professor & Head, Department of Biochemistry, Amrita School of Medicine Faridabad

Introduction

Lipids play an indispensable role throughout all stages of the atherosclerotic process, beginning with endothelial dysfunction and culminating in the formation and progression of atheromatous plaques. Atherosclerosis is now globally recognized as a chronic, progressive disease and characterized by both lipid metabolism dysregulation and persistent vascular inflammation.

The accumulation and subsequent modification of lipids within the arterial intima trigger a complex cascade of cellular and molecular events involving endothelial cells, macrophages, vascular smooth muscle cells, and various inflammatory mediators. These interactions collectively contribute to plaque development, progression, and vascular remodeling.

Lipid profile

Traditionally, the conventional lipid profile—comprising total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C)—has served as the cornerstone for evaluating serum lipid status and assessing the risk of atherosclerotic cardiovascular disease (ASCVD). These parameters have been extensively validated as major biomarkers for cardiovascular risk prediction and have guided both preventive and therapeutic strategies for decades.

Despite the well-recognized utility of the traditional lipid profile, it often does not fully capture the complexity of lipid metabolism or its interplay with inflammatory and metabolic pathways. Notably, many individuals with seemingly normal lipid levels still develop premature atherosclerosis or cardiovascular events, indicating that conventional lipid measures may underestimate residual risk. To address this limitation, several non-traditional lipid parameters and derived indices have been proposed to offer a more comprehensive assessment of lipid-associated cardiovascular risk.

Role of Oxidative Stress in Atherosclerosis

Oxidative stress refers to an imbalance between the generation of ROS (reactive oxygen species) and the antioxidant system's ability to neutralize them. The oxidative stress plays a pivotal role in the initiation and progression of atherogenesis.

The following are potential mechanisms by which oxidative stress mediates atherosclerosis:

Endothelial dysfunction.
LDL Oxidation and Foam cell formation.
Inflammatory cascade activation.
Smooth muscle cell (SMC) migration and proliferation.
Plaque destabilization.

Endothelial dysfunction

The endothelium is the first site affected by oxidative stress. Endothelial dysfunction thus represents the earliest functional marker of atherosclerosis. ROS degrade nitric oxide (NO), which results in reduced bioavailability of NO and formation of peroxynitrite ($ONOO^-$). This results in increased vascular permeability, enhanced leukocyte adhesion, migration and impaired vasodilation.

LDL Oxidation and foam cell formation

LDL molecules infiltrate the subendothelial space, where they undergo oxidation mediated by ROS and forms Oxidized LDL (ox-LDL); which is pro-atherogenic in nature and leading to foam cell formation followed by fatty streak in the intima, the earliest visible lesion of atherosclerosis.

Inflammatory Cascade Activation

Oxidative stress activates redox-sensitive transcription factors (NF-κB and AP-1), leading to upregulation of numerous inflammatory mediators which sustain vascular inflammation, smooth muscle proliferation, and plaque progression. Over a period of time plaques became unstable and prone to rupture and thrombosis — the hallmark of acute coronary events.

Oxidative stress markers

Markers of oxidative stress—such as malondialdehyde (MDA), ox- LDL, and F_2-isoprostanes -correlate with the severity of atherosclerosis and cardiovascular risk.

In nut shell, oxidative stress acts as the driving force through all the stages of atherosclerosis — from endothelial injury, lipid oxidation to plaque formation and rupture. Understanding its basic mechanisms not only clarifies disease pathogenesis but also offers potential targets for early detection and intervention of atherosclerosis.

Lipid Indices

lipid indices integrate interactions between pro-atherogenic and anti-atherogenic lipoproteins, providing a broader perspective on an individual's atherogenic potential. These indices correlate more strongly with surrogate markers of subclinical atherosclerosis—such as carotid intima-media thickness (CIMT) and coronary artery calcium score—than isolated lipid measurements. They are especially

valuable in populations with mixed dyslipidemia, metabolic syndrome, or diabetes mellitus, where traditional lipid parameters may underestimate cardiovascular risk.

Key indices include:

- Atherogenic Index of Plasma (AIP): log (TG/HDL-C), reflecting the balance between triglyceride-rich lipoproteins and protective HDL.
- Atherogenic Coefficient (AC): (TC – HDL-C)/HDL-C, indicating the proportion of non-HDL cholesterol relative to HDL.
- Castelli Risk Indices (CRI-I and CRI-II): TC/HDL-C and LDL-C/HDL-C, respectively, assessing global lipid-related risk.
- Lipoprotein Combine Index (LCI), Lipid Tetrad Index (LTI), Lipid Pentad Index (LPI), and Advanced Atherogenic Index (AAI): composite indices incorporating multiple lipid fractions to better reflect overall atherogenicity.

Incorporating lipid indices into routine assessment may enhance early identification of high-risk individuals, guide therapeutic decisions, and allow for more personalized strategies for atherosclerosis prevention and management.

Conclusion

Early atherosclerosis is marked by a pronounced elevation of oxidative stress biomarkers alongside unfavorable changes in lipid indices. Incorporating these parameters into routine clinical assessment may facilitate the early detection of individuals at heightened cardiovascular risk, enabling timely preventive and therapeutic interventions. Further studies involving larger populations and longitudinal follow-up are warranted to confirm and extend these observations.

"Could ApoB Predict COVID-19 Risk? Exploring a New Biomarker"

Aastha Bansal

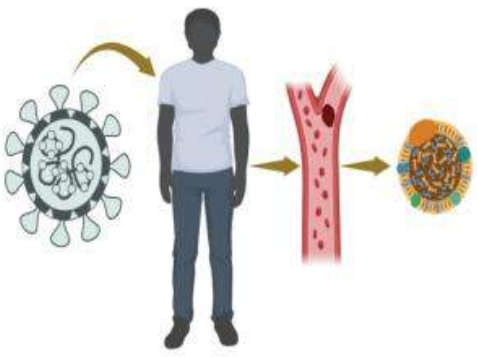
Assistant Professor, Neurochemistry Department, Institute of Human Behavior & Allied Sciences, New Delhi

The COVID-19 pandemic has presented unprecedented challenges to global health, particularly concerning cardiovascular complications. Understanding and mitigating these risks has become paramount as we navigate the ongoing crisis. Scientists are still looking for biomarkers that can assist anticipate the severity of diseases and direct early therapies, even though the introduction of vaccinations and antivirals has saved countless lives. ApoB protein that has historically been linked to cardiovascular health, can be one such biomarker. Could the severity of COVID-19 also be predicted by this lipid transport protein? In the battle against pandemic viral infections, this newsletter examines the most recent research findings, therapeutic applications, and potential future paths for ApoB as a novel biomarker.

Lipid Metabolism and COVID-19: An Odd Association

Cholesterol regulates the entry of the virus causing COVID-19, SARS-CoV-2, into host cells as cholesterol-enriched rafts are specific platforms that enable viral endocytosis through the Angiotensin converting enzyme (ACE2) receptor. This causes immunological dysregulation and an inflammatory cascade and the resulting inflammation and cytokine storms inturn negatively impacts natural lipid biosynthesis pathways. As SARS-CoV-2 comprises a bilayer lipid envelope, the viral replication inside the host cell also disrupts lipid metabolism and lipid resources. This has been observed that patients with COVID-19 infection, who are hospitalised frequently have dyslipidaemia with increased triglycerides and decreased HDL-C, and that is quite common in critical cases. These changes imply that the pathogenesis of COVID-19 may include lipid-related mechanisms, such as lipoproteins containing ApoB.

Apolipoprotein B (APO B): The Key Player



APO B is a primary protein component of atherogenic lipoproteins, such as low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL), and intermediate-density lipoprotein (IDL) essential for cholesterol transport in the bloodstream. ApoB comes in two main forms: ApoB-100 produced by Liver, a structural protein for LDL and VLDL particles and ApoB-48 produced in intestine aids Chylomicron production. Unlike LDL cholesterol (LDL-C), which measures the cholesterol within these particles, APO B provides a more accurate assessment of cardiovascular risk by quantifying the actual number of lipoprotein particles since there is just one ApoB molecule per atherogenic particle. Elevated ApoB levels are thus linked to

a higher risk of cardiovascular disease due to the increased number of atherogenic particles capable of penetrating arterial walls and triggering inflammation.

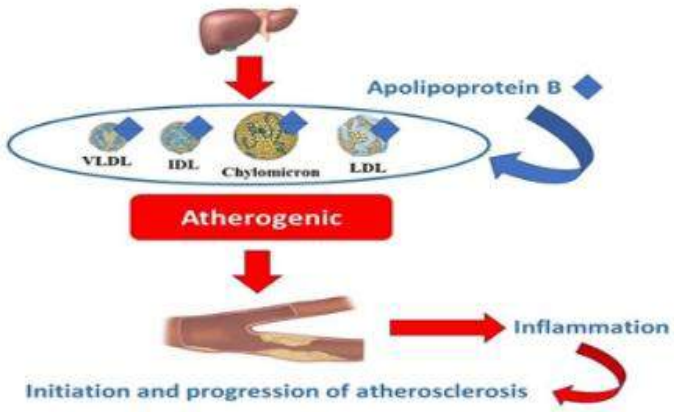
Mechanistic Perspectives: The Potential Impact of ApoB on COVID-19 Results

How ApoB may contribute to COVID-19 risk is explained by a number of tenable mechanisms:

1. Endothelial dysfunctioning and ApoB are involved in endothelial inflammation and atherosclerosis. These are the two conditions that are related to the vascular damage brought on by COVID-19. Endothelial damage caused by the virus results in: Coagulopathies, thrombosis of the microvascular system and an embolism in the lungs.
2. Elevated ApoB levels thus may intensify these consequences, perhaps making infected patients' prognosis worse.
3. The virus's inflammatory response can destabilize atherosclerotic plaques, further leading to acute cardiovascular events.

The link between Apo B and atherosclerosis.

Immune modulation and ApoB



ApoB-rich lipoproteins have the ability to interact with immune cells, including dendritic cells and macrophages. According to certain research, ApoB-lipoproteins may change viral entrance or clearance by binding to viral particles. The cytokine storm observed in severe COVID-19 may be exacerbated by elevated ApoB, which may trigger the production of pro-inflammatory cytokines.

Shared Risk Factors: Obesity and Metabolic Syndrome

Both elevated ApoB and severe COVID-19 are associated with obesity, diabetes, and metabolic syndrome. ApoB may act as a proxy marker reflecting underlying metabolic derangements that predispose individuals to worse COVID-19 outcomes.

ApoB as a Hospitalisation Risk Predictor

The lipid profiles in COVID-19 patients were examined in many cohort studies. The findings demonstrated that, regardless of LDL-C or triglyceride levels, increased ApoB levels were associated with a greater probability of hospitalisation and intensive care unit admission. Researchers assessed the extended lipid profiles of hospitalised COVID-19 patients and found that ApoB-to-ApoA1 ratios were

much greater in patients with increased levels of inflammatory markers- C-reactive protein (CRP), Interleukin-6 (IL-6- a cytokine that promotes inflammation) and D-dimer, a coagulopathy marker. One reliable indicator of respiratory failure and the requirement for mechanical ventilation can be ApoB/ApoA1 ratio.

ApoB and Long COVID: A Path Forward

Long COVID is one of the most confusing problems of the pandemic, since patients continue to suffer symptoms like as exhaustion, fogginess, and dyspnoea months after they have recovered. These symptoms might be caused by chronic metabolic and endothelial dysfunction, according to some studies. ApoB may be an indicator of persistent inflammation linked to lipids, offering information on the long-term pathophysiology of COVID-19. Long-term COVID therapy regimens may include an investigation of ApoB-lowering techniques.

A Hidden Biomarker in the Public Eye

Integrative biomarkers spanning immunological, cardiometabolic health, and infectious illness have become increasingly important as a result of the COVID-19 pandemic. As a long-standing indicator of cardiovascular risk, ApoB may provide important information about: Immune and endothelial dysfunction, post-acute sequelae of viral infections, and susceptibility to severe COVID-19.

Although clinical guidelines has not yet included ApoB-based COVID-19 screening, the mounting evidence is however strong. Future studies may confirm ApoB's status as a vital pandemic preparation tool, with potential uses in other inflammatory and viral illnesses.

Call to Action: What Are the Next Steps for Researchers and Clinicians?

1. Risk Stratification: Measuring APO B levels and ApoB/ApoA1 ratio in addition to inflammatory markers in COVID-19 patients could become a standard practice to identify those at higher risk for severe disease, enabling more tailored and proactive treatment approaches.
2. Preventive Measures: For individuals with known high APO B levels, stringent preventive measures against COVID-19, such as vaccination and booster shots and lifestyle modifications, are highly recommended.
3. Therapeutic Interventions: Incorporating lipid-lowering treatments in the management plan for COVID-19 patients with elevated APO B levels might be beneficial.
4. Researchers may Conduct prospective research correlating COVID-19 results to pre-infection ApoB levels. Investigate the mechanisms that link ApoB to viral clearance and immunological respons alonside examining the possibility of ApoB-targeting therapies for both acute and chronic COVID or viral infections

References:

1. Myrou A, Barmpagiannos K, Papathanasiou E, Kachtsidis V, Kiouli C, Tziomalos K. Association Between Lipid Profile and COVID-19 Severity: Insights from a Single-Center Cross-Sectional Study in Northern Greece. J Clin Med. 2025 Jun 9;14(12):4082.
2. Kowalska K, Sabatowska Z, Forycka J, Młynarska E, Franczyk B, Rysz J. The Influence of SARS-CoV-2 Infection on Lipid Metabolism-The Potential Use of Lipid-Lowering Agents in COVID-19 Management. Biomedicines. 2022 Sep 18;10(9):2320.
3. Abu-Farha M, Thanaraj TA, Qaddoumi MG, Hashem A, Abubaker J, Al-Mulla F. The Role of Lipid Metabolism in COVID-19 Virus Infection and as a Drug Target. Int J Mol Sci. 2020 May 17;21(10):3544.
4. Garcia-Gil M, Barrios V, Benito B. Dyslipidemia and COVID-19: Possible Role in Severe Disease. *Curr Cardiol Rep.* 2021;23(10):140.
5. Dorobanțu M, Halațiu VB, Gheorghe-Fronea O, Bala CG, Moldovan H, Irinel-Parepa R, Rodean IP, Benedek I, Benedek T. The Association between Apolipoprotein B, Cardiovascular Risk Factors and Subclinical Atherosclerosis-Findings from the SEPHAR National Registry on Hypertension in Romania. Int J Mol Sci. 2023 Feb 1;24(3):2813.

Cardiovascular Benefits of Dapagliflozin: From Glycaemic Control to Multisystem Protection

Arti Srivastava, Jagriti Bhatia, Dharmaveer Singh Arya
Department of Pharmacology, All India Institute of Medical Sciences, New Delhi-29

Introduction

Dapagliflozin, a selective sodium–glucose co-transporter 2 (SGLT2) inhibitor originally approved for the management of type 2 diabetes mellitus (T2DM), has rapidly evolved from a glucose-lowering agent to a cornerstone therapy in cardiovascular (CV) medicine. Its unexpected benefits on heart failure outcomes, renal protection, and mortality across diverse populations have redefined therapeutic strategies for patients with metabolic and cardiovascular diseases. Unlike traditional antidiabetic medications that act via insulin-dependent pathways, dapagliflozin exerts systemic hemodynamic, metabolic, and inflammatory effects that collectively offer substantial cardio protection. The discovery of its cardiovascular benefits emerged serendipitously from large outcome trials mandated by regulatory agencies to evaluate CV safety of antidiabetic drugs. However, rather than demonstrating mere safety, dapagliflozin reduced heart failure hospitalisation rates, cardiovascular deaths, and renal decline in both diabetic and non-diabetic populations, heralding a paradigm shift in therapeutic positioning. This review synthesises current evidence on the cardiovascular role of dapagliflozin, spanning mechanistic insights, major clinical trials, guideline recommendations, and emerging therapeutic frontiers.

Mechanistic Insights into Cardiovascular Protection

The cardiovascular benefits of dapagliflozin are attributed to a combination of hemodynamic, metabolic, renal, and molecular effects that extend far beyond glycemic reduction. At the hemodynamic level, dapagliflozin promotes osmotic diuresis and natriuresis, leading to volume offloading, reduced preload and afterload, and subsequent improvement in cardiac filling pressures [1]. Unlike conventional diuretics, which can activate neurohormonal pathways and lead to electrolyte disturbances, dapagliflozin induces a more balanced fluid redistribution without significant impact on serum potassium or sympathetic activation [2]. Additionally, dapagliflozin lowers systolic blood pressure by approximately 3 to 5 mmHg without triggering reflex tachycardia, contributing further to vascular unloading.

Metabolically, dapagliflozin facilitates a shift in myocardial substrate utilisation from glucose and free fatty acids to ketone bodies, particularly β-hydroxybutyrate, which yield more ATP per unit of oxygen consumed [3]. This metabolic shift enhances cardiac efficiency, particularly in failing hearts that struggle with impaired energy utilisation. Weight reduction and decreased visceral adiposity induced by dapagliflozin further alleviate cardiometabolic stressors linked to left ventricular hypertrophy and systemic inflammation.

Renoprotective effects contribute significantly to cardiovascular outcomes, as chronic kidney disease (CKD) is a major driver of heart failure progression. Dapagliflozin reduces intraglomerular pressure through tubuloglomerular feedback modulation, thereby delaying nephron loss and subsequent cardiorenal deterioration [4]. At the molecular level, dapagliflozin suppresses oxidative stress, reduces pro-fibrotic signaling pathways such as TGF-β, and attenuates NLRP3 inflammasome activation, collectively preventing structural cardiac remodeling [5]. Improvement in endothelial function, reduction in arterial stiffness, and inhibition of sympathetic overactivation add to its multifaceted cardioprotective mechanisms.

Evidence from Randomised Cardiovascular Outcome Trials

The cardiovascular legacy of dapagliflozin was cemented through a series of rigorously conducted outcome trials. The DECLARE–TIMI 58 trial, enrolling over 17,000 patients with T2DM, demonstrated a 27% relative risk reduction in hospitalisation for heart failure, establishing dapagliflozin as the first SGLT2 inhibitor with a primary prevention benefit in patients without established atherosclerotic cardiovascular disease [6]. While the reduction in major adverse cardiovascular events (MACE) did not reach statistical significance across the overall cohort, the consistency of heart failure benefit was striking.

The DAPA-HF trial marked a major breakthrough by recruiting patients with heart failure with reduced ejection fraction (HFrEF), irrespective of diabetic status. Dapagliflozin reduced the composite endpoint of cardiovascular death or worsening heart failure by 26%, with benefits evident within 28 days of treatment initiation [7]. Importantly, the magnitude of benefit was similar in non-diabetic patients, confirming that glucose reduction was not the primary driver of cardiac protection.

Subsequently, the DELIVER trial extended dapagliflozin's indications to heart failure with preserved ejection fraction (HFpEF), a phenotype notoriously resistant to conventional treatments. In this cohort, dapagliflozin reduced the risk of cardiovascular death or worsening heart failure by 18%, making it the first therapy with consistent efficacy across the entire left ventricular ejection fraction spectrum [8].

In parallel, the DAPA-CKD trial demonstrated significant reduction in CKD progression, cardiovascular events, and mortality among patients with chronic kidney disease, regardless of diabetic status [9]. Given the strong interrelationship between renal dysfunction and cardiovascular mortality, these renal benefits indirectly reinforce the cardioprotective profile of dapagliflozin. Across all trials, dapagliflozin consistently reduced sudden cardiac death, hospital readmissions, and need for loop diuretics, supporting its integration into core heart failure management.

Impact Across Patient Subgroups

A remarkable feature of dapagliflozin is its uniform efficacy across diverse patient phenotypes. Elderly patients and those with multiple comorbidities derived similar benefit to younger individuals, highlighting its therapeutic robustness [10]. Patients with atrial fibrillation, obesity, and hypertension also exhibited significant risk reduction, suggesting potential applications in broader cardiometabolic syndromes. In post-myocardial infarction patients, preliminary evidence suggests reduction in adverse remodeling and prevention of heart failure onset, though dedicated endpoint trials are ongoing. The neutral effect on arrhythmogenesis, coupled with reduction in cardiac fibrosis, raises interest in its role as an upstream therapy for atrial and ventricular arrhythmias.

Patients with advanced renal impairment previously excluded from most heart failure drugs were safely treated with dapagliflozin down to estimated glomerular filtration rates (eGFR) of 25 mL/min/1.73 m^2, without increased risk of ketoacidosis or electrolyte imbalance. Importantly, dapagliflozin demonstrated no increased incidence of major hypoglycemia, regardless of background insulin therapy, distinguishing it from other antihyperglycemics with cardiovascular concerns. Collectively, these findings underscore dapagliflozin's translational applicability beyond diabetic heart failure populations.

Comparison With Other Cardioprotective Drug Classes

Traditional anti-heart failure therapies such as ACE inhibitors, beta-blockers, and mineralocorticoid receptor antagonists confer benefits primarily through neurohormonal suppression. In contrast, dapagliflozin operates via volume modulation, metabolic reprogramming, and inflammation reduction.

Rather than competing with existing agents, it complements them, leading to additive reductions in cardiovascular events when used in combination. In network meta-analyses, dapagliflozin ranked among the most effective agents in preventing heart failure hospitalisation, outperforming GLP-1 receptor agonists, DPP-4 inhibitors, and sulfonylureas [11]. Unlike loop diuretics, dapagliflozin does not induce rebound sodium retention or activate renin-angiotensin pathways, allowing for stable volume management without neurohormonal toxicity.

Comparisons with empagliflozin and other SGLT2 inhibitors have generally shown class homogeneity in cardiovascular outcomes, though dapagliflozin has stronger renal outcome data in non-diabetic populations. In contrast to sacubitril/valsartan, which shows maximal benefit in HFrEF, dapagliflozin maintains efficacy in HFpEF and CKD populations. These unique features position dapagliflozin as a foundational therapy that bridges metabolic, renal, and cardiac advantages.

Safety Considerations and Clinical Practicality

Dapagliflozin is well tolerated, with a favorable safety profile in both acute and chronic care settings. The most commonly reported adverse events include genital mycotic infections, which are typically mild and manageable with hygiene counselling. The risk of diabetic ketoacidosis is low but higher in insulin-dependent diabetics during periods of acute illness or reduced carbohydrate intake, necessitating patient education rather than drug discontinuation. Hypotension and volume depletion are uncommon but warrant dose adjustment of concomitant diuretics in frail patients. Unlike thiazolidinediones, dapagliflozin does not induce fluid retention or exacerbate heart failure. Its once-daily oral administration offers a practical advantage over injectable cardiometabolic therapies, promoting adherence.

Guideline Integration and Real-World Adoption

Following the results of DAPA-HF and DELIVER, major international societies including the European Society of Cardiology (ESC) and American Heart Association (AHA) have incorporated dapagliflozin as a Class I recommendation for the treatment of symptomatic HFrEF, irrespective of diabetes status in 2023[12]. It is now considered a foundational pillar alongside ACE inhibitors/ARNI, beta-blockers, and mineralocorticoid antagonists. In HFpEF, dapagliflozin holds the strongest recommendation among available therapies. Real-world registry analyses confirm that early initiation after hospitalisation for acute decompensated heart failure reduces 30-day readmissions and mortality, supporting inpatient adoption.

Future Directions and Emerging Applications

Beyond established indications, several trials are exploring novel cardiovascular applications of dapagliflozin. In post-myocardial infarction patients without heart failure, dapagliflozin is being investigated for its potential to prevent adverse remodeling and new-onset heart failure. Preclinical models suggest attenuation of ischemia–reperfusion injury via mitochondrial protection and reduced oxidative stress [13]. Emerging data propose benefits in pulmonary hypertension through reduction in right ventricular afterload and vascular inflammation. Additionally, dapagliflozin is undergoing evaluation in patients with cardiomyopathy secondary to chemotherapy, obesity, or sleep apnea. Its potential role in atrial fibrillation suppression via reduction in atrial stretch and fibrosis represents another evolving frontier. Combination therapies with GLP-1 agonists or mineralocorticoid antagonists may further enhance cardiometabolic synergy.

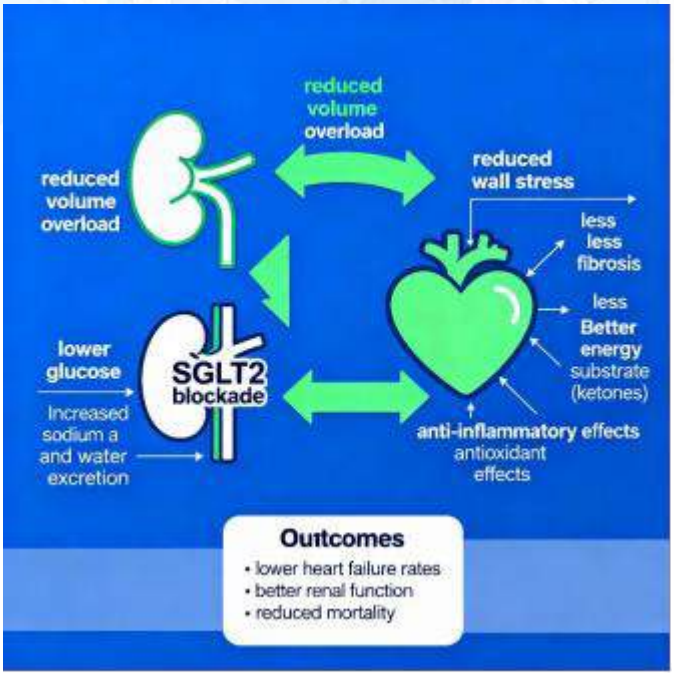


Figure 1: *Dapagliflozin inhibits renal SGLT2, reducing glucose and sodium reabsorption. This decreases blood volume, lowers cardiac workload, improves myocardial energy, and reduces inflammation, resulting in fewer heart failure events, better renal outcomes, and reduced mortality.*

Conclusion

Dapagliflozin has transitioned from a glucose-lowering drug to a pivotal cardiovascular and renal protective therapy with applications extending far beyond diabetes. Its cardioprotective mechanisms encompass hemodynamic unloading, metabolic reprogramming, renal preservation, and anti-inflammatory remodeling, collectively yielding robust reductions in heart failure hospitalisations, cardiovascular death, and progression to end-stage renal disease. Evidence across landmark trials confirms consistent benefit in both diabetic and non-diabetic populations, and across varying ejection fraction phenotypes. With strong safety and practicality, dapagliflozin now stands as a paradigm-shifting agent that bridges endocrinology, cardiology, and nephrology. As ongoing investigations explore its role in arrhythmia prevention, post-infarct remodeling, and pulmonary vascular disease, its therapeutic legacy is poised to expand further. In an era of precision cardiometabolic medicine, dapagliflozin exemplifies the evolution of drugs beyond their initial indication into versatile multisystem protectors.

References

1. Heerspink HJL, Perkins BA, Fitchett DH, Husain M, Cherney DZI. Sodium Glucose Cotransporter 2 Inhibitors in the Treatment of Diabetes Mellitus: Cardiovascular and Kidney Effects, Potential Mechanisms, and Clinical Applications. *Circulation*. 2016;134(10):752–772.
2. Verma S, McMurray JJV. SGLT2 inhibitors and mechanisms of cardiovascular benefit: a state-of-the-art review. *Diabetologia*. 2018;61(10):2108–2117.
3. Ferrannini E, Mark M, Mayoux E. CV Protection in the EMPA-REG OUTCOME Trial: A "Thrifty Substrate" Hypothesis. *Diabetes Care*. 2016;39(7):1108–1114.
4. Cherney DZI, Dekkers CCJ, Barbour SJ, Cattran D, et al. Effects of the SGLT2 inhibitor dapagliflozin on proteinuria in non-diabetic patients with chronic kidney disease. *Lancet Diabetes Endocrinol*. 2020;8(7):582–593.
5. Li C, Zhang J, Xue M, Li X, Han F, Liu X. SGLT2 Inhibition with Dapagliflozin Reduces Myocardial Infarct Size via Promoting M2 Macrophage Polarization in Mice. *Cardiovasc Diabetol*. 2021;20(1):81.
6. Wiviott SD, Raz I, Bonaca MP, et al. Dapagliflozin and Cardiovascular Outcomes in Type 2 Diabetes. *N Engl J Med*. 2019;380(4):347–357.

7. McMurray JJV, Solomon SD, Inzucchi SE, et al. Dapagliflozin in Patients with Heart Failure and Reduced Ejection Fraction. *N Engl J Med*. 2019;381(21):1995–2008.
8. Solomon SD, McMurray JJV, Claggett B, et al. Dapagliflozin in Heart Failure with Mildly Reduced or Preserved Ejection Fraction. *N Engl J Med*. 2022;387(12):1089–1098.
9. Heerspink HJL, Stefánsson BV, Correa-Rotter R, et al. Dapagliflozin in Patients with Chronic Kidney Disease. *N Engl J Med*. 2020; 383:1436–1446.
10. Kato ET, Silverman MG, Mosenzon O, et al. Effect of Dapagliflozin on Heart Failure and Mortality in Type 2 Diabetes Mellitus. *Circulation*. 2019;139(22):2528–2536.
11. Zelniker TA, Wiviott SD, Raz I, et al. SGLT2 inhibitors for primary and secondary prevention of cardiovascular and renal outcomes: a systematic review and meta-analysis of cardiovascular outcome trials. *Lancet*. 2019;393(10166):31–39.
12. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J*. 2021;42(36):3599–3726.
13. Lim VG, Bell RM, Arjun S, et al. SGLT2 Inhibition with Dapagliflozin Reduces Myocardial Infarct Size and Improves Cardiac Function in a Preclinical Model of Ischemia–Reperfusion Injury. *Cardiovasc Drugs Ther*. 2022; 36:587–596.

From Kitchen to Heart: Revisiting Cow Ghee and Mustard Oil as Anti-Atherosclerotic Agents

Vaishali Prajapati, Vipin Kumar Verma, Priya Bhardwaj, Jagriti Bhatia, Dharamvir Singh Arya
Cardiovascular Research Laboratory, Department of Pharmacology, All India Institute of Medical Sciences, New Delhi-110029

Abstract:

Cow ghee and mustard oil, traditionally used cooking fats, show promising anti-atherosclerotic properties. Cow ghee improves lipid profile by lowering LDL, VLDL, and TGs, while increasing HDL cholesterol. Its conjugated fatty acids and antioxidants reduce inflammation, enhance bile acid secretion, and support reverse cholesterol transport. Mustard oil, rich in omega-3 fatty acids and allyl isothiocyanate, exhibits antioxidant and vasodilatory effects and reduces lipid peroxidation and improves endothelial function. Hence, these fats mitigate atherosclerosis through anti-inflammatory, hypolipidemic, and antioxidant mechanisms, and reduce the risk of cardiovascular diseases.

Introduction:

Atherosclerosis is a chronic inflammatory disease characterised by the buildup of fatty plaques in the arteries, leading to cardiovascular diseases (CVD) such as heart attacks and strokes. Diet plays a crucial role in the development and prevention of atherosclerotic disease. In South Asia, cow ghee and mustard oil are traditional dietary fats, and recent research has begun to explore their potential anti-atherosclerotic properties. This review summarizes the evidence and mechanisms by which cow ghee and mustard oil may protect against atherosclerosis.

Cow Ghee and Mustard Oil: Composition and Cardiovascular Effects

Cow ghee, made from clarified cow's milk butter, contains saturated fats, short and medium-chain fatty acids, conjugated linoleic acid (CLA), butyric acid, and fat-soluble vitamins A, D, E, and K (Anamika FN et al., 2025It has been shown that moderate ghee consumption can lower total, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) cholesterol while increasing high-density lipoprotein (HDL) cholesterol (Munisekhar K et al., 2022). Cow ghee neutralises free radicals that reduce vascular stress and inflammation, which are the key drivers of atherosclerosis (Marcone S et al., 2015; Anamika FN et al., 2025). However, ghee's high saturated fatty acid (SFA) content (around 65%), cholesterol level, and an unfavourable omega-6 to omega-3 ratio (3:1) may raise LDL and worsen lipid balance if consumed excessively. Thus, moderate, controlled intake may offer benefits without elevating cardiovascular risk (Mishra S et al., 2012).

Mustard oil (known as *sarson ka Tel*), extracted from mustard seeds, is rich in healthy fats like Monounsaturated Fatty acids (MUFA; erucic and oleic acid), Polyunsaturated Fatty Acids (PUFA; omega-3 and omega-6 fatty acids), along with antioxidants such as vitamin E and allyl isothiocyanate (Anamika FN et al., 2025). These compounds help lower LDL cholesterol, raise HDL, and protect blood vessels from inflammation and oxidative damage. Though it contains erucic acid, once linked to heart issues in animals, recent studies show overall heart-protective effects due to its fatty acid balance. With low saturated fat (8%) and high MUFA (70%), mustard oil supports healthy lipid metabolism and reduces atherosclerosis risk when used moderately and in pure, unadulterated form (Mishra S et al., 2012; Poddar KH et al., 2022).

Safety and Traditional Use

Ayurvedic medicine has long advocated the use of cow ghee for heart health. Modern studies confirm that moderate intake is safe and may even be beneficial for cardiovascular health, provided it is consumed as part of a balanced diet (Munisekhar K et al., 2022). Mustard oil is widely used in South Asian cooking and has a long history of traditional use for heart health. Modern research supports its safety and efficacy when consumed in moderation. Both fats, when used in moderation, may offer complementary benefits for cardiovascular health.

Traditional and Modern Perspectives

Ayurveda and traditional medicine have long promoted cow ghee and mustard oil for heart health. Modern research is beginning to validate these claims, showing that both fats can be part of a heart-healthy diet when consumed in moderation and as part of a balanced nutritional pattern.

Mechanism of action

Ghee helps maintain a balance between LDL and HDL cholesterol (lowers LDL, VLDL, and TGs and increases HDL), thereby reducing the risk of plaque formation. Bioactive lipids and butyric acid suppress inflammatory cytokines and inhibit NF-κB signalling, a major pathway in atherosclerosis. Vitamins and fatty acids in ghee scavenge free radicals, protecting endothelial cells from damage (Munisekhar K et al., 2022). Ghee may enhance the removal of cholesterol from arterial walls, a process known as reverse cholesterol transport (Park SH et al., 2024).

MUFAs and omega-3s in mustard oil reduce LDL cholesterol and increase HDL cholesterol, preventing plaque buildup by decreasing lipid peroxidation. Vitamin E and other antioxidants in mustard oil neutralise free radicals, protecting endothelial cells. Mustard oil improves endothelial function and inhibits platelet aggregation, which is crucial for preventing atherosclerosis (Munisekhar K et al., 2022; Park SH et al., 2024; Poddar KH et al., 2022).

Evidence from Human and Animal Studies

Cow Ghee

Animal studies show that cow ghee lowers cholesterol and TGs in a dose-dependent way and helps protect against atherosclerosis. Mice fed ghee developed smaller artery plaques and showed less oxidative damage. Long-term ghee intake improved liver antioxidant activity and proved safer than hydrogenated oils for heart health. Mouse studies found that ghee-derived peptides reduced vascular inflammation via the NF-Kb pathway, supporting antiatherogenic mechanisms (Marcone S et al., 2015). In another animal study, replacing other fats with cow ghee reduced plaque buildup, improved cholesterol levels, lowered inflammation, and enhanced blood vessel function, showing ghee's protective effect on heart and vascular health.

Experimental studies in Wistar rats demonstrated that adding 2.5-10% of total dietary calories from cow ghee resulted in a dose-dependent reduction in serum total cholesterol. LDL, VLDL, and triglycerides, along with decreased liver peroxidation and anti-inflammatory eicosanoids such as leukotrienes and prostaglandins. Translating this to human dietary energy intake, a typical 2000-kcal diet would correspond to 10-25 ml/day of cow ghee (roughly 2-3 teaspoons daily), which aligns with Ayurvedic recommendations for safe therapeutic consumption (Munisekhar K et al., 2022).

Recent human studies show that cow ghee and its milk fat globule membrane (MFGM) components have lipid-lowering and vascular-protective benefits. A meta-analysis found that MFGM supplementation significantly reduced total and LDL cholesterol, suggesting a positive effect against atherosclerosis (Kanon AP et al., 2024). Cohort and clinical studies report that replacing meat-based animal fats with dairy fats like ghee does not increase cardiovascular disease (CVD) risk and may even offer protection (Kanon AP et al., 2024; Munisekhar K et al., 2022). Human intervention trials and plasma studies show that ghee supports healthy HDL levels, improves endothelial function, and does not raise atherogenic risk in either healthy or hyperlipidaemic adults (Marcone S et al., 2015; Givens DI, 2022). Population-based studies in South Asians associate traditional ghee use with lower coronary event rates compared to refined oil diets (Givens DI, 2022).

Mustard Oil

Mustard oil (MO) demonstrates potent lipid-lowering and cardioprotective effects in hyperlipidaemic animal models. Supplementation with MO significantly decreases total and LDL cholesterol levels while elevating HDL, resulting in up to a 52.5% reduction in total cholesterol and an 89.3% improvement in the LDL/HDL ratio (Al-Fartosi KG et al., 2017). Its nearly optimal omega-6 to omega-

3 ratio of 1.2:1 supports cardiovascular health by enhancing platelet stability and lowering the risk of thrombosis (Mishra et al., 2012). Mechanistically, MO reduces hepatic cholesterol accumulation, mitigates oxidative stress, upregulates antioxidant enzymes (glutathione peroxidase, catalase), and suppresses vascular inflammation and foam cell formation. Chronic administration improves endothelial function and limits atherosclerotic plaque formation, offering superior protection compared to diets rich in saturated or refined oils (Vesnina et al., 2022). Preclinical data on rats showed that diets containing diacylglycerol-rich mustard oil improved LDL, HDL, triglyceride ratios and reduced plasma lipid peroxidation, suggesting cardioprotective potential (Dhara R et al., 2013).

The cold-pressed mustard oil, rich in erucic acid, demonstrates significant cardioprotective benefits. It's supplementation reduces TG and TC levels, and increases HDL cholesterol by approximately 13% (Prakash et al., 2019). Clinical studies associate mustard oil use with lower IHD risk compared to sunflower oil (Mishra et al., 2012). Regular consumption among South Asian populations correlates with improved lipid profile, reduced inflammation, and lower CAD rates vs. refined oil users. Randomized trials have confirmed declines in TGs, CRP, and antioxidant activity. MO further enhances endothelial function, promotes vascular health, and reduces oxidative stress. Epidemiological and clinical evidence support moderate intake of pure, cold-pressed MO as part of a heart-healthy diet (Vesnina et al., 2022; Khatun et al., 2021). Clinical practice literature suggests 5-10 ml once or twice daily (10-20 ml/day total) as a safe and effective internal dose for improving circulation, lipid metabolism, and vascular health when used as the primary cooking oil or dressing (Gupta S. 2025).

Practical Recommendations

Excessive intake of any fat can be harmful. Use cow ghee and mustard oil in moderate amounts. Choose pure, unadulterated cow ghee and cold-pressed mustard oil for maximum health benefits. Combine these fats with a diet rich in fruits, vegetables, whole grains, and lean proteins. For individuals seeking anti-atherosclerosis effects through dietary fats, including cow ghee, up to 2-3 teaspoons/day (10-25 ml/day) and mustard oil, up to 2 teaspoons twice a day (10-20 ml total). According to the ICMR-National Institute of Nutrition (NIN) 2024 Dietary Guidelines for Indians, adults should maintain total visible fat intake (including all cooking oils, butter, and other added fats) below 25-30 ml/day (Manoj Shah R et al., 2024).

Conclusion

Cow ghee and mustard oil, traditional fats of South Asia, both show anti-atherosclerotic action when used in moderation. Cow ghee, rich in antioxidants and good fats, helps to lower bad cholesterol and supports healthy blood vessels. Mustard oil is full of omega-3 and omega-6 fats, which reduce inflammation and improve circulation. Together, they protect against artery blockage and support overall health. A smart balance is to let cow ghee make up one-third and mustard oil two-thirds of your daily visible fat (around 25-30 ml total), giving you a perfect mix of taste, tradition, and cardiovascular protection.

References

1. Al-Fartosi KG, Roomi AB, Al-Badry SH. Study of Mustard Oil (Brassica nigra L.) As A Hypolipidemic. Educational studies. 2017 Jan 1;37:285-302.
2. Anamika FN, Mahajan S, Munjal P, Aggarwal K, Jain R. From Ancient Wisdom to Modern Health:'Let Food Be Thy Medicine'–Examining the Cardiovascular Impact of Ghee and Seed Oils. Eastern Journal of Medical Sciences. 2025 Jul 4;10(3).1
3. Dhara R, Dhar P, Ghosh M. Dietary effects of diacylglycerol rich mustard oil on lipid profile of normocholesterolemic and hypercholesterolemic rats. Journal of food science and technology. 2013 Aug;50(4):678-86.
4. Givens DI. Saturated fats, dairy foods and cardiovascular health: No longer a curious paradox?. Nutrition Bulletin. 2022 Dec;47(4):407-22.
5. Gupta S, BAMS M. Mustard Oil: Health Benefits, Nutrition, Uses & Side Effects (2025).
6. Kanon AP, Spies SJ, MacGibbon AK, Fuad M. Milk fat globule membrane is associated with lower blood lipid levels in adults: A meta-analysis of randomized controlled trials. Foods. 2024 Aug 28;13(17):2725.
7. Manoj Shah R, Sachdeva G, Chaudhari UK. Dietary guidelines across different countries & comparisons to dietary guidelines for Indians, 2024. Indian Journal of Medical Research. 2025 Jul 1;162(1).

8. Marcone S, Haughton K, Simpson PJ, Belton O, Fitzgerald DJ. Milk-derived bioactive peptides inhibit human endothelial-monocyte interactions via PPAR-γ dependent regulation of NF-κB. Journal of Inflammation. 2015 Jan 20;12(1):1.
9. Mishra S, Manchanda SC. Cooking oils for heart health. J Prev Cardiol. 2012 Feb;1(3):123-31.
10. Mohammadifard N, Nazem M, Naderi GA, Saghafian F, Sajjadi F, Maghroon M, Bahonar A, Alikhasi H, Nouri F. Effect of hydrogenated, liquid and ghee oils on serum lipids profile. ARYA atherosclerosis. 2010;6(1):16.
11. Munisekhar K, Rao PS, Sitaram B, Sharvani N, Kiranmayi VS, Hemalatha D. Lipid profile in healthy human volunteers before and after consuming ghee. Bioinformation. 2022 Sep 30;18(9):742.
12. Park SH, Kang MK, Kim DY, Lim SS, Kang IJ, Kang YH. Ellagic acid, a functional food component, ameliorates functionality of reverse cholesterol transport in murine model of atherosclerosis. Nutrition Research and Practice. 2024 Apr 1;18(2):194-209.
13. Poddar KH, Sikand G, Kalra D, Wong N, Duell PB. Mustard oil and cardiovascular health: Why the controversy?. Journal of clinical lipidology. 2022 Jan 1;16(1):13-22.
14. Prakash S, Singh N, Priyatma VS, Singh C. Assessment of mustard oil components against free radicals and oxidized LDL cholesterol. IJCS. 2019;7(3):423-8.
15. Vesnina A, Prosekov A, Atuchin V, Minina V, Ponasenko A. Tackling atherosclerosis via selected nutrition. International journal of molecular sciences. 2022 Jul 26;23(15):8233.

Repurposing CD80/86-CD28 Blockade to Combat Cardiac Hypertrophy in Experimental Rat Model via MAPK/Nrf-2-HO1/Smad-TGF-beta pathways

Vaishali Prajapati, Jagriti Bhatia, Dharamvir Singh Arya
Department of Pharmacology, All India Institute of Medical Sciences, New Delhi-110029
Email (presenting author): vaishalivaishali220@gmail.com

Introduction: Cardiac hypertrophy, a major precursor to heart failure, is strongly driven by inflammation and immune cell activation. Isoproterenol-induced hypertrophy in rats closely mimics these pathological processes, with T-cell infiltration and macrophage recruitment contributing significantly to cardiac remodelling. Abatacept, a CTLA4 analogue widely used in autoimmune disorders, blocks T-cell co-stimulation and suppresses inflammatory signalling, making it a promising candidate for repurposing in cardiovascular disease. This study aimed to evaluate the therapeutic potential of Abatacept in counteracting isoproterenol (ISO)-induced cardiac hypertrophy through immune-mediated mechanisms.

Materials and Methods: Male Wistar albino rats were divided into six groups (n=6): control, disease control (ISO 3 mg/kg, s.c.), three experimental groups (Abatacept at 2.5, 5, and 10 mg/kg with ISO), and Abatacept control (10 mg/kg). Treatments were administered daily for 42 days. On day 43, hemodynamic assessments were performed, followed by collection of blood and cardiac tissue for biochemical, molecular, and histopathological analyses.

Results: Abatacept attenuated ISO-induced cardiac hypertrophy in a dose-dependent manner, preserving cardiac function and improving myocardial architecture. It significantly reduced cardiac injury markers (CK-MB, LDH), enhanced antioxidant defenses, and suppressed inflammatory cytokine levels. Histopathology (H&E and MT staining) revealed improved structural integrity, while immunofluorescence confirmed a reduction in activated T-cell subpopulations. Molecular analysis indicated Abatacept's regulatory effects on apoptotic, necrotic, autophagic, and inflammatory pathways, particularly involving MAPK, Nrf-2/HO-1, and Smad/TGF-β signaling cascades.

Conclusion: Abatacept exerts cardioprotective effects by mitigating oxidative stress, inflammation, and immune cell infiltration, thereby preserving myocardial function and structure. These findings highlight Abatacept as a potential preventive and therapeutic strategy for pathological cardiac hypertrophy and for individuals at risk of developing heart failure.

Keywords: Abatacept, Cardiac hypertrophy, Immune cell infiltration, Isoproterenol

Role of Receptor for advanced glycation end products (RAGE) in coronary artery disease (CAD) patients with diabetes mellitus

Mohit Singh[1], Pradeep Kumar Dabla[2], Vimal Mehta[3]
[1]Post Graduate Resident, Department of Biochemistry, GB Pant Institute of Postgraduate Medical Education and Research, associated Maulana Azad Medical College, New Delhi
[2]Director Professor and Head, Department of Biochemistry, GB Pant Institute of Postgraduate Medical Education and Research, associated Maulana Azad Medical College, New Delhi
[3]Director Professor, Department of Cardiology, GB Pant Institute of Postgraduate Medical Education and Research, associated Maulana Azad Medical College, New Delhi

Email id of presenter: Dr. Mohit Singh; mohit99101@gmail.com

Introduction: Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide. Diabetes mellitus exacerbates CAD through chronic hyperglycaemia, which promotes advanced glycation end product (AGE) formation. Interaction of AGEs with their receptor (RAGE) triggers activation of transcription factors, promoting the expression of inflammatory cytokines such as IL-1β, IL-6, and TNF-α. This chronic pro-inflammatory and pro-oxidative environment accelerates

atherosclerosis and endothelial dysfunction, thereby aggravating CAD progression in diabetic patients. Therefore, this study was conducted to correlate the concentration of RAGE amongst patients of CAD with and without diabetes mellitus.

Methods: A total of consented 160 angiographically proven CAD patients were enrolled at GB Pant Hospital (GIPMER) during catheterization. Patients were divided into two groups A&B, Group A including 80 patients with diabetes mellitus and Group B including 80 patients without diabetes. Samples collected under aseptic precautions and serum analysed for Lipid profile, HsCRP, HbA1c via auto analysers and RAGE concentrations using ELISA.

Results: Serum RAGE levels were significantly elevated in CAD patients with diabetes mellitus compared to non-diabetic CAD patients ($p < 0.05$). A significant positive correlation was observed between RAGE and HbA1c ($p = 0.004$). Elevated RAGE levels correlated with the pro-inflammatory state and may contribute to enhanced atherosclerotic burden in diabetic individuals.

Conclusion: RAGE is significantly upregulated in CAD patients with diabetes mellitus, underscoring its role in promoting vascular inflammation and atherosclerosis. These findings suggest that RAGE may serve as a potential biomarker of disease severity and a therapeutic target to reduce cardiovascular risk in diabetic populations.

Keywords: RAGE, CAD, diabetes mellitus, inflammation, atherosclerosis

Erdosteine-Mediated Modulation of Myocardial Tolerance to Ischemia-Reperfusion Injury in Diabetic Rats

Priya Bhardwaj[1], Vipin Kumar Verma [1], Ekta Mutneja [1], Jagriti Bhatia [1], Dharamvir Singh Arya[1]*
[1]Department of Pharmacology, All India Institute of Medical Sciences, New Delhi-110029
Presenting author: Priya Bhardwaj; Email: pripray23@gmail.com

Introduction: Ischemic heart disease (IHD) is one of the most prevalent and life-threatening cardiovascular complications of diabetes mellitus (DM), remains a major cause of mortality among diabetic patients. Current therapeutic strategies are directed toward achieving both glycemic control and reducing cardiovascular risk, particularly IHD. This study aimed to evaluate the cardioprotective effects of erdosteine in modulating the myocardial tolerance to ischemia-reperfusion (I/R) injury in diabetic rats.

Materials and Method: Forty adult male Wistar rats were divided into five groups: Sham, diabetic control (Dia-C), disease control (Dia+IR), Erdosteine (80 mg/kg, s.c.) + Dia+IR, and Erdosteine (80 mg/kg) per se. Diabetes was induced with streptozotocin (STZ, 50 mg/kg, i.p.), and rats with fasting blood glucose >400 mg/dL were included. Erdosteine was administered for 28 days as a pretreatment. On day 29, myocardial ischemia was induced by LAD coronary artery occlusion for 60 mins followed by 45 mins reperfusion. Hemodynamic, biochemical, and molecular parameters were assessed. Data were analyzed using one-way ANOVA followed by Tukey–Kramer post hoc test.

Results: Erdosteine pretreatment significantly decreased advanced glycation end products (AGEs) and improved hemodynamic parameters compared to Dia+IR rats. It enhanced antioxidant defenses (GSH, SOD, CAT), reduced lipid peroxidation (MDA), and minimized cardiac injury markers (CK-MB, LDH). Inflammatory cytokines (TNF-α, IL-6) were lowered, and myocardial HMGB1 and RAGE expression were downregulated, while PPAR-γ expression remained unchanged.

Conclusions: Erdosteine enhances myocardial tolerance to IR injury in diabetic rats by attenuating AGE-RAGE signalling and downstream activation of HMGB1 and NF-κB pathways, along with reducing free radical generation.

Keywords: Cardiovascular disease; Diabetes; Erdosteine; Myocardial Ischemia-Reperfusion Injury (MIRI); Immune response

DISLIPIDAEMIA IN PATIENTS WITH SUBCLINICAL HYPOTHYROIDISM

Ajanabh Goswami, Lokesh Kumar Sharma, Bindu Kulshreshtha, Sanjeet Panesar
Department of Biochemistry and Endocrinology, ABVIMS & DR RMLH, New Delhi

ABSTRACT:

Hypothyroidism is a common endocrine disorder resulting from deficient thyroid hormone levels, affecting about 4–5% of the population in developed countries. Its causes are multifactorial, involving both environmental and genetic factors, and it impacts multiple organ systems, particularly the cardiovascular system. Subclinical hypothyroidism (SCH), as defined by the American Thyroid Association (2021), is a mild form characterized by elevated thyroid-stimulating hormone (TSH) levels, usually below 10 mIU/L, with normal thyroid hormone levels. SCH is often asymptomatic and more prevalent in women (7.5–8.5%) than in men (2.8–4.4%). Its prevalence increases with age, necessitating better screening and monitoring strategies.

Subclinical hypothyroidism (SCH) has been associated with dyslipidaemia, which increases cardiovascular risk. Several studies support this association. Hussain et al. reported higher total cholesterol and triglycerides with lower high-density lipoprotein (HDL) in SCH patients. Kumar et al. found that SCH patients positive for anti-thyroid peroxidase (anti-TPO) antibodies had significantly elevated triglycerides, total cholesterol, and low-density lipoprotein (LDL). Thyroid hormones regulate lipid synthesis, mobilization, and clearance in the liver. In SCH, decreased hepatic LDL receptor expression reduces LDL clearance, increasing circulating LDL cholesterol. Impaired lipoprotein lipase activity elevates triglycerides and very-low-density lipoprotein (VLDL) levels, while reduced cholesterol ester transfer protein (CETP) activity lowers high-density lipoprotein (HDL). These alterations lead to an atherogenic lipid profile. Overall, evidence suggests that higher TSH levels and autoimmune involvement in SCH predispose individuals to dyslipidaemia.

Apolipoprotein A1 polymorphism (rs670) and serum Apolipoprotein A1 levels in patients with Metabolic Syndrome

Parul Bansal*,Ritu Singh**, Rajeev Goyal***, Ritika Sood****
*Resident, Department of Biochemistry, Lady Hardinge Medical College & Associated Hospitals, New Delhi.
**Director Professor & Head of Department, Department of Biochemistry, Lady Hardinge Medical College & Associated Hospitals, New Delhi.
***Professor, Department of Biochemistry, Lady Hardinge Medical College & Associated Hospitals, New Delhi.
****Professor, Department of Medicine, Lady Hardinge Medical College & Associated Hospitals, New Delhi.
Email ID: drparulbansal97@gmail.com

Introduction: Metabolic Syndrome (MetS) is a cluster of abnormalities including abdominal obesity, insulin resistance, high blood pressure and dyslipidaemia which collectively increase the risk of chronic diseases, such as cardiovascular disease and Type 2 Diabetes Mellitus. Dyslipidaemia in MetS is characterised by elevated triglyceride levels and low high-density lipoprotein cholesterol (HDL-C). Apolipoprotein A1 (ApoA1), the major structural and functional important protein in HDL-C, plays a critical role in lipid metabolism and cardiovascular protection. This study was done to evaluate the role if any of ApoA1 in MetS.

Materials and methods: The serum ApoA1 levels and ApoA1 gene rs670 polymorphism were studied in 80 subjects (40 patients with MetS and 40 healthy volunteers). The serum ApoA1 level was estimated using immunoturbidometry. Extracted DNA from blood was amplified by polymerase chain reaction, digested with MspI restriction enzyme and analysed using restriction fragment length polymorphism.

Results: Mean serum Apo A1 levels were higher in control subjects(152.7±24.5mg/dl) than the study group(164.7±26.9mg/dl) and difference was found to be statically significant (p= 0.04). Homozygous

genotype (GG & AA) was found to be higher in controls, whereas heterozygosity (AG) was higher in cases. However this was not statistically significant.

Conclusion: The study reinforces the importance of ApoA1 levels as metabolic indicator of lipid dysregulation. The genetic findings though not significant in this study may indicate a role of homozygosity versus heterozygosity of the ApoA1 rs670 polymorphism in contributing to dyslipidaemia. Further studies with larger sample size is required to affirm or refute the findings.

Key words: metabolic syndrome, dyslipidaemia, Apolipoprotein A1, polymorphism, zygosity.

ASSOCIATION OF SERUM LEPTIN-ADIPONECTIN RATION WITH CAROTID ATHEROSCLEROSIS IN PRE DIABETES AND TYPE 2 DIABETES"

Dasapalli Veena Priya, Dr (Prof) Parul Goyal, Dr (Prof) Ajay Chauhan, Dr (Prof)Sarita Jilowa
Department of Biochemistry, Department of Medicine, Department of Radiodiagnosis.
ABVIMS & Dr RAM MANOHAR LOHIA HOSPITAL, NEW DELHI.
d.veenapriya@gmail.com

INTRODUCTION

Leptin, a 167-amino-acid hormone secreted by adipocytes in proportion to fat mass, exerts an inhibitory effect on food intake and regulates appetite and energy balance. Elevated leptin contributes to atherosclerosis through cholesterol accumulation, oxidative stress, foam cell formation, thrombogenesis, and activation of pro-inflammatory cytokines. Adiponectin, a 30 kDa hormone secreted by adipocytes, enhances insulin sensitivity and exerts anti-inflammatory, anti-atherogenic, and cardioprotective effects. Hypoadiponectinemia is linked to endothelial dysfunction and increased carotid intima-media thickness (CIMT), promoting coronary artery calcification independent of traditional cardiovascular risk factors.

The Leptin–Adiponectin Ratio (LAR), reflecting the opposing physiological actions of leptin and adiponectin on insulin sensitivity and vascular regulation, demonstrates superior predictive accuracy in identifying metabolic dysregulation and vascular pathology. CIMT, a surrogate marker of subclinical atherosclerosis and vascular inflammation, is a reliable predictor of cardiovascular risk and is routinely assessed using high-resolution B-mode ultrasonography.

MATERIAS AND METHODS

A cross-sectional observational study was conducted in the Departments of Biochemistry, General Medicine, and Radiology at ABVIMS & Dr. RML Hospital, New Delhi, from 1st February 2024 to 30th June 2025. The study included 90 participants—30 healthy controls, 30 prediabetic, and 30 newly diagnosed diabetic patients count.

RESULTS:

LAR levels were significantly higher in diabetics (88.37 ± 46.18) than in prediabetics (49.02 ± 38.60) and healthy controls (22.26 ± 20.55). LAR showed a strong positive correlation with CIMT ($r = 0.374$, $p = 0.036$). ROC analysis for predicting increased CIMT demonstrated high diagnostic accuracy (AUC = 0.85) with 85% sensitivity and specificity at a cutoff of 0.85.

CONCLUSIONS:

Our study concludes that LAR is a superior biomarker for early vascular alterations, outperforming individual adipokines and insulin resistance markers. Elevated LAR identifies individuals at risk for endothelial dysfunction and subclinical atherosclerosis, as indicated by increased CIMT.

Polystyrene Nanoplastics Promote Oxidative Stress and Dysregulation of Lipid-Handling Proteins in Cardiomyoblasts

Sana Naaz[1], Ankita Yadav[1], and Kamna Srivastava[1]*
[1]Dr. B.R. Ambedkar Centre for Biomedical Research, University of Delhi, Delhi, India- 110007

Abstract

Introduction: Environmental nanoplastics are emerging contaminants linked to cardiovascular toxicity. Among them, polystyrene nanoplastics (PS-NPs) have been implicated in oxidative stress and lipid dysregulation. However, their molecular effects on cardiac lipid-regulating genes remain unclear. This study integrates bioinformatics and in-vitro approaches to investigate the impact of carboxylate-modified PS-NPs on H9C2 cardiomyoblasts, focusing on SORT1 and PCSK9 proteins associated with atherosclerosis.

Materials and Methods: Protein interaction and pathway enrichment of SORT1 and PCSK9 were analyzed using STRING and Enrichr-KG databases. H9C2 cells were exposed to PS-NPs (0–500 µM) for 24 h. Cell viability (MTT), nitric oxide (Griess), and ROS (DCFH-DA) were assessed, while SEM and TEM examined morphology and intracellular NP uptake. Protein expression was quantified via western blotting.

Results: STRING and KEGG analyses revealed strong association of SORT1 and PCSK9 with lipid metabolism and atherosclerosis pathways. PS-NP exposure induced dose-dependent cytotoxicity (IC_{50} = 4.83 µM), elevated NO and ROS, and showed mitochondrial swelling, vacuolization, and nanoplastic accumulation by TEM. Western blotting revealed upregulation of SORT1 (~1.4-fold) and PCSK9 (~1.6-fold), indicating lipid-handling disruption.

Conclusion: Polystyrene nanoplastics trigger oxidative and nitrosative stress and activate lipid-regulatory genes promoting atherosclerotic signaling. This combined computational-experimental study identifies SORT1 and PCSK9 as potential biomarkers for nanoplastic-induced cardiovascular risk.

Keywords: Nanoplastics, Cardiomyoblasts, Oxidative stress, SORT1, PCSK9.

"Study of epigenetic modification (DNA Methylation), serum Paraoxonase 1 levels and Paraoxonase 1 gene (Q192R) polymorphism in smokers"

Srishti*, Ritu Singh,** Rajeev Goyal***, Ashok Kumar Singh****
*Post Graduate Student, Department of Biochemistry, LHMC, New Delhi.
**Director Professor and HOD, Department of Biochemistry, LHMC, New Delhi.
***Professor, Department of Biochemistry, LHMC, New Delhi.
****Director Professor and HOD, Department of TB & Chest, LHMC, New Delhi.
Address- RMO-A, LHMC, New Delhi
Email- srishtic49@gmail.com

Abstract

Introduction: Smoking is a major global health problem causing over 8 million deaths annually and leading to cancer, respiratory and cardiovascular diseases. Beyond genetic factors, smoking influences disease risk through epigenetic changes, particularly DNA methylation. Smoking also produces oxidative stress, affecting enzymes like Paraoxonase 1 whose reduced activity in smokers reflects impaired antioxidant defense.

Materials and Methods: A cross sectional study was conducted on 40 smokers and 40 age and gender matched healthy controls. DNA was extracted for measuring % DNA methylation and Paraoxonase 1(Q192R) polymorphism. % DNA methylation, Paraoxonase 1, glutathione peroxidase were estimated

by ELISA. hs-CRP levels were measured by spectrophotometry. PCR-RFLP of Paraoxonase 1 (Q192R) polymorphism was also performed. Statistical analysis was performed using SPSS version 29.

Results: % DNA methylation was significantly lower in cases ($p<0.001$). The mean levels of Paraoxonase 1 ($p=0.035$) and Glutathione Peroxidase ($p=0.004$) showed significantly low value in cases. QQ genotype of Paraoxonase 1 (Q912R) polymorphism was significantly more frequent in controls ($p=0.044$). The mean levels of hs-CRP were found to be significantly increased in cases ($p<0.001$).

Conclusion: DNA was significantly hypomethylated in smokers. Paraoxonase 1 and glutathione peroxidase levels were reduced in smokers, because they help to reduce the oxidative stress generated by smoke. Large scale studies with individuals from diverse ethnic groups (including family history) are needed to confirm whether global DNA methylation changes, reduced Paraoxonase1 levels and Q192R polymorphism affect gene expression.

Keywords: Smoker, Epigenetics, DNA methylation, Paraoxonase 1, Glutathione peroxidase.

ASSESSMENT OF ANDROGEN LEVELS AND ITS RELATION WITH GONADAL FUNCTION IN TYPE 2 MALE DIABETICS

Sangeeta Haldar[1], Neera Sharma[2], Bindu Kulshreshtha[3], Lokesh Kumar Sharma[4]
ABVIMS & DR. RML HOSPITAL

KEY WORDS: Free androgen index, Erectile dysfunction, Loss of libido, Hypogonadism, Diabetes mellitus

INTRODUCTION:

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Patients with diabetes mellitus experience sexual dysfunction such as loss of libido & erectile dysfunction. There is bidirectional interaction between androgen & diabetes in men in which low testosterone predisposes to diabetes.

MATERIAL & METHODS:

Estimation of hormonal parameters like LH, FSH, Total Testosterone (TT), DHEAS, SHBG were done on fully automated Beckman Coulter Unicel DXi 600 immunoassay analyzer using Chemiluminescence technology. Estimation of Insulin was done by CLIA on VITROS Eci. Assessment of DHT was done by ELISA method. Free Androgen Index was calculated as (TT/SHBG×100).

RESULTS:

Testosterone showed no significant relationship with LH ($p = 0.136$) and significant difference was found for FSH ($p < 0.05$). SHBG concentrations are not impacted by levels of LH or FSH. DHEAS showed no significant association with LH ($p = 0.705$), but a significant association was found with FSH ($p < 0.05$). There were no significant associations between DHT levels and either LH ($p = 0.846$) or FSH ($p = 0.986$). FAI exhibits significant positive correlations with Total Testosterone and SHBG and a significant negative correlation with DHT.

CONCLUSION:

The study reaffirms reciprocal link between T2DM & male hypogonadism, emphasizing the need for integrated management strategies. Addressing underlying factors like obesity, inflammation, & glycemic control while monitoring hormonal profiles could offer significant benefits in improving metabolic and reproductive health outcomes in affected indiviuals.

"A study of Surfactant Protein D Gene Polymorphism (rs721917) and SP-D Protein Levels in Pediatric Sepsis-Associated Acute Kidney Injury"

Vandana Lath*, Smita Tripathi**, Shalu Gupta***
*Post Graduate Student, Department of Biochemistry, LHMC, New Delhi
** Director Professor, Department of Biochemistry, LHMC, New Delhi
*** Director Professor, Department of Pediatric, LHMC, New Delhi.
Email id- drvandanabiochemistry@gmail.com

Abstract

Introduction: Surfactant Protein D (SP-D) is an innate immune defence molecule that regulates inflammatory and immune responses during sepsis. Its role in the pathogenesis of sepsis-associated acute kidney injury (AKI) has gained increasing importance, given the significant morbidity and mortality linked to this condition. Previous studies have shown a possible association between SP-D and sepsis-associated AKI. However, our present study is the first to investigate this relationship in the pediatric population.

Materials and Methods: A case–control analytical study was conducted on 30 children with sepsis-associated AKI and 30 septic controls without AKI admitted to the Pediatric Intensive Care Unit. SP-D gene polymorphism (rs721917) was analysed using DNA isolation, polymerase chain reaction (PCR), and restriction fragment length polymorphism (RFLP). Serum SP-D levels were estimated by ELISA. Statistical analysis was performed using SPSS version 29.

Results: The CC genotype of the rs721917 polymorphism was significantly more frequent in cases than in controls ($p = 0.038$), while the TT genotype predominated among controls. The CC genotype was associated with severe AKI (Stage 3), whereas the CT genotype was linked with milder stages ($p = 0.026$). Mean serum SP-D levels were markedly higher in cases compared to controls ($p < 0.001$) and showed a progressive increase with AKI severity ($p < 0.001$).

Conclusion: Elevated serum SP-D levels and the rs721917 CC genotype are significantly associated with sepsis-associated AKI in children. Larger multicentric studies are warranted to validate SP-D as a potential biomarker and genetic indicator for predicting susceptibility and severity of pediatric sepsis-associated AKI.

Keywords: Surfactant Protein D (SP-D); Sepsis-associated Acute Kidney Injury (AKI); rs721917 Polymorphism; Pediatric Sepsis; Biomarker.

Evaluation of Markers of Inflammation and Total Antioxidant Capacity in Diabetic Retinopathy

Gloria Taiveinai Ro K[1], Smita Roy[2], Taru Dewan[3], Parul Goyal[4], Ajay Chauhan[5]
Post graduate, Department of Biochemistry[1],
Associate Professor, Department of Biochemistry[2],
Professor, Department of Ophthalmology[3]
Professor, Department of Biochemistry[4],
Professor, Department of Medicine[5],
ABVIMS & Dr RML Hospital, New Delhi
Email ID: glotaivei123@gmail.com

Abstract

Introduction:

Diabetic Retinopathy (DR) is one of the micro-vascular complications of diabetes which leads to blindness among the working age individuals. Chronic hyperglycemia activates the inflammatory

pathways and oxidative stress mechanisms with consequent damage to nerve tissue and retina. The present study focuses on the early identification of the disease process, thereby minimizing retinal function loss and heralding an improvement in the management of diabetic retinopathy.

Methods and Material:

A cross-sectional observational study was conducted on 96 patients comprising of three groups, 32 T2DM patients complicated with diabetic retinopathy, 32 T2DM patients without diabetic retinopathy and 32 healthy controls. Estimation of serum insulin, ferritin, ceruloplasmin and NLR was done on chemiluminescence based, immunoturbidimetric and haematology analysers respectively. Serum TAC was assayed using ELISA technology. Data was analysed using SPSS version 26.0.

Results:

Mann Whitney U analysis showed that levels of HbA1c%, HOMA-IR, ferritin, ceruloplasmin, NLR and TAC were significantly different among the three study groups. Spearman test showed a significant positive correlation between these parameters and an inverse correlation with TAC. The ROC curve, shows TAC as the best predictor of diabetic retinopathy with AUC of 0.936 followed by ceruloplasmin (0.844), ferritin (0.760) and NLR (0.646).

Conclusion:

Elevated levels of ceruloplasmin and ferritin coupled with decreased TAC levels are good indicators for diabetic retinopathy and may be used as a diagnostic tool to predict the occurrence of retinopathy.

Keywords:

Ferritin, ceruloplasmin, NLR, Total antioxidant capacity, Diabetic retinopathy

UNDERSTANDING THE EFFECT OF DIFFERENT COVID VACCINES ON COAGULATION STATE IN PREMATURE CORONARY ARTERY DISEASE

Cheteny*[1], Sharma Manushri[2], Sympli Enitarast[3], Batra Vishal[4], Mahajan Bhawna[5]
[1]Second year Post Graduate Resident, Department of Biochemistry, GB Pant hospital, 1 Jawaharlal Nehru Marg, 64 Khamba, Raj Ghat, New Delhi, Delhi, 110002, (drchetenya@gmail.com, MOB: 8750102573)
[2]Associate Professor, Department of Biochemistry, GB Pant hospital, New Delhi
[3]First year Post Graduate Resident, Department of Biochemistry, GB Pant hospital, New Delhi
[4]Proffesor, Department of Cardiology, GB Pant hospital, New Delhi
[5]Professor, Department of Biochemistry, GB Pant hospital, New Delhi

Introduction:

Coronary artery disease (CAD) remains a leading cause of global morbidity and mortality. Alarmingly, premature CAD (pCAD) cases— aged 18–50 has surged post–COVID-19 pandemic, with reports indicating a 12–15% rise by 2025. COVID-19 infection and its vaccination linked to hypercoagulable states through endothelial dysfunction, platelet hyperreactivity, and impaired fibrinolysis, Altering PT, INR, aPTT, and D-dimer, potentially accelerating atherothrombotic processes and predisposing susceptible individuals to pCAD. We aim to compare the effect of different covid vaccines on coagulation profile of pCAD cases.

Materials and Methods:

In this cross-sectional study, a total of 57 confirmed pCAD patients were included, out of which 20 were vaccinated with Covaxin, 21 with Covishield, and 16 unvaccinated. PT/INR and aPTT were measured by Turbidimetric method while D-dimer by immunoturbidimetry method. One-way ANOVA was used to compare coagulation profiles across the different groups.

Results:

Among pCAD, statistically significant differences were observed in PT and D-dimer levels across three groups. Patient received Covaxin had significantly higher D-dimer levels (1.08 ± 0.80 µg/mL; p = 0.01) in comparison to Covishield (0.54 ± 0.43 µg/mL) and unvaccinated group (0.53 ± 0.39 µg/mL). On the other hand, patient received Covishield vaccine had lower PT (11.24 ± 0.78 s; p=0.04), INR (0.95 ± 0.08; p=0.07) and elevated aPTT levels (43.45 ± 12.93 s; p=0.69) than patient received Covaxin (11.68 ± 1.05 s; 0.99 ± 0.12, 42.41 ± 10.53 s) or unvaccinated (12.06 ± 1.05 s; 1.04 ± 0.12, 40.53 ± 5.82 s).

Conclusion:

Our study shows that Covaxin vaccine produce more pro-coagulable state in pCAD as compared to Covishield reflecting subtle fibrinolytic or endothelial activation.

Keywords: Premature coronary artery disease, Hypercoagulable state, Coagulation, Vaccine, COVID.

Correlating serum MMP-9 level and Gensini score in patients of coronary artery disease

Kaviyarasan[1], Charanjeet Kaur[2], Isser[3]
1.PG 3 Resident VMMC & Safdarjung Hospital New Delhi
2.Director & Professor Dept of Biochemistry, VMMC & Safdarjung Hospital New Delhi
3.HOD , Director & Professor , Dept of Cardiology VMMC & Safdarjung Hospital New Delhi
Key words : CAD ,MMP-9,GENSINI SCORE,SEVERITY OF CAD , CAD MARKER

INTRODUCTION

Coronary artery disease is a major cause for mortality and morbidity in Indian population.

Gensini score widely used to quantify the severity and extent of coronary artery disease based on findings from a coronary angiogram

MATERIALS AND METHODS:

case & control study,70 cases & 70 controls

Results:

Serum MMP 9 levels were significantly higher in the CAD patient group (350+-125ng/ml) compared to the healthy control group (210 +-75.4ng/ml) with statistical significance of p<0.001

OR = 4.25 suggesting high MMP 9 is an independent risk factor for CAD

CORRELATION between serum MMP 9 & Gensini score: A significant positive correlation was observed between the serum MMP 9 levels and the Gensini scores among the CAD patients (r=0.58)p<0.001.

This indicates that patients with higher MMP 9 levels tends to have more extensive and severe coronary atherosclerosis and triple vessel disease

Conclusions:

This case -control study demonstrated that serum MMP 9 levels were significantly elevated in patients with coronary artery disease compared to healthy controls .Furthurmore, MMP 9 levels were positively and significantly correlated with severity of CAD quantified by Gensini score .

STUDY OF SERUM LEVELS OF HEART TYPE FATTY ACID BINDING PROTEIN AND CAROTID INTIMA-MEDIA THICKNESS IN PREDIABETICS

Sweta Saroha, Priyamvadha.R, Parul Goyal, * Ajay Chauhan,
**Dr Sarita Jilowa
Department of Biochemistry, *Department of Medicine, **Department of Radiodiagnosis, ABVIMS & Dr RML Hospital, New Delhi

Email: swtsaroha@gmail.com

INTRODUCTION:

Prediabetes is increasingly being studied in the context of its association with cardiovascular disease (CVD). Besides raised HbA1c and sugar levels, the major underlying defect seems to be insulin resistance (IR). Subclinical atherosclerosis, measured by high sensitivity C reactive protein (hsCRP) and carotid artery intima media thickness (CIMT) underlies the pathogenesis of CVD in prediabetes. Heart-type fatty acid binding protein (H-FABP), a novel cardiac biomarker, also might have a role in predicting prediabetic heart disease.

METHODOLOGY

50 prediabetic patients and 50 age, sex and BMI matched controls were employed in the case control study. Serum F & PPBS, (HbA1c), fasting insulin levels were measured in cases and controls. Serum H-FABP was measured in both cases and controls. All cases and controls were subjected to bilateral CIMT measurements and Serum hsCRP levels. The values were compared between both the groups and subjected to appropriate statistical analysis.

RESULTS:

The mean serum levels of H-FABP among cases and controls were 6.38 ± 2.76 ng/ml and 3.24 ± 2.47 ng/ml respectively ($p < 0.0001$). Mean CIMT was found to be higher in prediabetics (0.59 ± 0.11 mm) compared to controls (0.45 ± 0.07mm) ($p<0.0001$). Serum hsCRP levels were also statistically higher in prediabetics (5.75 ± 4.16 mg/l) than that of controls (1.86± 1.67 mg/l) ($p<0.0001$). The correlations of the two variables, hsCRP and CIMT with H-FABP were both strongly positive ($r = 0.687$) & ($r = 0.779$) respectively [both cases ($p< 0.0001$)].

CONCLUSION:

The novel cardiac biomarker H-FAPB might be a good predictor of cardiovascular risks in prediabetics.

KEYWORDS: Heart type fatty acid binding protein (H-FABP), Cardiovascular disease, Insulin Resistance, Carotid artery intima media thickness (CIMT).

ASSOCIATION OF SERUM FETUIN-A AND ADIPONECTIN WITH CAROTID ATHEROSCLEROSIS IN PRE-DIABETES AND DIABETES

S Naveen Kumar, Parul Goyal, * Ajay Chauhan, ** Sarita Jilowa

Department of Biochemistry, *Department of Medicine, **Department of Radiodiagnosis, ABVIMS & Dr RML Hospital, New Delhi

Naveengvmc10@gmail.com

INTRODUCTION:

Serum Fetuin-A is a multifunctional glycoprotein which is exclusively secreted from hepatocytes. Adiponectin, a 30 kDa hormone derived from adipocytes, plays an important role in regulating glucose metabolism and increases insulin sensitivity. Adiponectin and Fetuin-A are tightly associated with T2DM and cardiovascular diseases, working in opposite direction. Early detection of comorbidities, including the early stages of CVD, is crucial for effective management and prevention of adverse outcomes. Using easily measurable biomarkers, such as proteins or glycoproteins, that are associated

with an increased cardiovascular risk can be useful in early detection and active management of cardiovascular disease.

METHODODLOGY:

A cross-sectional observational study was conducted in department of Biochemistry and in the out-patient departments of Medicine and Radiodiagnosis, ABVIMS & Dr RML hospital, New Delhi with study duration from April 2023 to June 2024. The study population included 90 patients in three groups: 30 patients with T2DM, 30 patients of prediabetes and 30 healthy controls. Serum Fetuin-A, Adiponectin, HbA1c%, HOMA-IR, AIP and CIMT were measured. Statistical analysis was performed using excel sheet and IBM SPSS v.26.

RESULTS:

Mann-Whitney U analysis showed that serum Fetuin-A, Adiponectin, HbA1c%, HOMA-IR, AIP and CIMT values were significantly different among the three study groups. Spearman test showed a significant positive correlation between Fetuin-A and other markers and negative correlation between Adiponectin and other markers. ROC Curve analysis demonstrated none of the parameters show statistically significant discriminatory power in prediabetes. Among them, HOMA-IR (AUC - 0.511) have slightly higher AUC values, suggesting moderate potential for discrimination, without reaching significance. Among Diabetes group the conventional biomarker HbA1C% (0.970) had the highest AUC, but Fetuin-A (0.934) and Adiponectin (0.948) also had nearly the same AUC followed by CIMT (0.922).

CONCLUSION:

In conclusion, although our study is limited by its low sample size and cross-sectional design, we have demonstrated association of Fetuin-A and Adiponectin with carotid atherosclerosis, which provides evidence for their role in CVD development beyond conventional inflammatory markers and cardiovascular risk factors. This suggests that they may serve as potential diagnostic marker for early detection of atherosclerosis. However, a greater number of studies with larger sample size and of prospective design, are necessary to establish the role of these markers in CVD development and CVD-related mortality.

Role of Receptor for advanced glycation end products (RAGE) in coronary artery disease (CAD) patients with diabetes mellitus

Mohit Singh[1], Pradeep Kumar Dabla[2], Vimal Mehta[3]

[1]Post Graduate Resident, Department of Biochemistry, GB Pant Institute of Postgraduate Medical Education and Research, associated Maulana Azad Medical College, New Delhi

[2]Director Professor and Head, Department of Biochemistry, GB Pant Institute of Postgraduate Medical Education and Research, associated Maulana Azad Medical College, New Delhi

[3]Director Professor, Department of Cardiology, GB Pant Institute of Postgraduate Medical Education and Research, associated Maulana Azad Medical College, New Delhi

Email id of presenter: Dr. Mohit Singh; mohit99101@gmail.com

Introduction: Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide. Diabetes mellitus exacerbates CAD through chronic hyperglycaemia, which promotes advanced glycation end product (AGE) formation. Interaction of AGEs with their receptor (RAGE) triggers activation of transcription factors, promoting the expression of inflammatory cytokines such as IL-1β, IL-6, and TNF-α. This chronic pro-inflammatory and pro-oxidative environment accelerates atherosclerosis and endothelial dysfunction, thereby aggravating CAD progression in diabetic patients. Therefore, this study was conducted to correlate the concentration of RAGE amongst patients of CAD with and without diabetes mellitus.

Methods: A total of consented 160 angiographically proven CAD patients were enrolled at GB Pant Hospital (GIPMER) during catheterization. Patients were divided into two groups A&B, Group A including 80 patients with diabetes mellitus and Group B including 80 patients without diabetes.

Samples collected under aseptic precautions and serum analysed for Lipid profile, HsCRP, HbA1c via auto analysers and RAGE concentrations using ELISA.

Results: Serum RAGE levels were significantly elevated in CAD patients with diabetes mellitus compared to non-diabetic CAD patients ($p < 0.05$). A significant positive correlation was observed between RAGE and HbA1c ($p = 0.004$). Elevated RAGE levels correlated with the pro-inflammatory state and may contribute to enhanced atherosclerotic burden in diabetic individuals.

Conclusion: RAGE is significantly upregulated in CAD patients with diabetes mellitus, underscoring its role in promoting vascular inflammation and atherosclerosis. These findings suggest that RAGE may serve as a potential biomarker of disease severity and a therapeutic target to reduce cardiovascular risk in diabetic populations.

Keywords: RAGE, CAD, diabetes mellitus, inflammation, atherosclerosis.

ASSOCIATION OF ANGIOPOIETIN LIKE PROTEIN-3 WITH CAROTID ATHEROSCLEROSIS IN PRE-DIABETES AND TYPE 2 DIABETES

Santosh Kumar, Parul Goyal, * Ajay Chauhan, ** Sarita Jilowa

Department of Biochemistry, *Department of Medicine, **Department of Radiodiagnosis,

ABVIMS & Dr RML Hospital, New Delhi

INTRODUCTION:

Type 2 diabetes (T2DM) and prediabetes are major risk factors for atherosclerotic cardiovascular disease (ASCVD). Carotid intima-media thickness (CIMT) is a reliable marker for early atherosclerosis. Angiopoietin-like protein 3 (ANGPTL-3), a liver protein, regulates fat metabolism and may promote atherosclerosis, but its link with CIMT and heart disease risk is mixed. This study aimed to examine the association of serum ANGPTL-3 with CIMT and ASCVD risk factors across normal glucose, prediabetes, and newly diagnosed T2DM.

METHODOLOGY:

A cross-sectional observational study was conducted on 120 patients (40 T2DM, 40 prediabetes, 40 healthy controls) at ABVIMS & Dr RML Hospital. We measured serum ANGPTL-3, HbA1c, HOMA-IR, AIP, and CIMT. Quantitative assessment of hsCRP was done in our lab using hsCRP reagent on vitros 5600 integrated system.

Estimation of ANGPTL3 was done by using ELK biotech ELISA kit.

Estimation of CIMT was done in the dept of Radiodiagnosis using ECUBE ALPINION USG system.

RESULTS:

The mean age was 42.3 ± 7.4 years, and mean BMI was 27.05±3.2 kg/m^2. Fasting glucose, HbA1c, HOMA-IR, hsCRP, and CIMT all rose progressively from controls to prediabetes and diabetes ($p<0.001$), reflecting increasing insulin resistance, inflammation, and vascular thickening. Serum ANGPTL-3 levels were significantly lower in prediabetes and diabetes compared to controls ($p=0.004$). ANGPTL-3 showed a negative correlation with fasting glucose, HbA1c, and insulin resistance indices.

CONCLUSION:

Early metabolic and vascular parameters progressively worsen from normoglycemia to T2DM. ANGPTL-3 levels were paradoxically lower in prediabetic and diabetic individuals, suggesting regulation by metabolic disturbances16. The lack of a direct association between ANGPTL-3 and CIMT indicates its impact on vascular health may be indirect. These findings emphasize the early start of subclinical atherosclerotic changes, necessitating early detection and prevention.

Exploring the Role of MMP-2 in cardiovascular disease: A Potential Biomarker for Disease Progression

Ankita Yadav, Kamna Srivastava
Dr. B.R. Ambedkar Centre for Biomedical Research, University of Delhi, Delhi, India- 110007
Email: ankitaofficial1116@gmail.com

Abstract

Introduction: Cadmium (Cd) is a toxic heavy metal and environmental pollutant that contaminates agricultural soil, water, and air through various sources. Cd exposure induces inflammation, oxidative stress, and endothelial dysfunction, contributing to cardiovascular diseases. It may also regulate matrix metalloproteinases (MMPs), key enzymes involved in cardiac remodeling. This study aimed to investigate the effect of acute low-dose cadmium exposure on MMP-2 regulation in H9c2 cardiomyoblasts.

Materials and Methods: H9c2 cells were exposed to cadmium chloride ($CdCl_2$) for 24 hours. Cell viability was evaluated using the MTT assay, and intracellular nitric oxide (NO) levels were measured using the Griess assay. The expression of MMP-2 was analyzed at mRNA and protein levels using qPCR and Western blotting, respectively.

Results: Low-dose cadmium exposure significantly decreased cell viability and increased intracellular NO levels, indicating oxidative stress. Both MMP-2 mRNA and protein levels were upregulated in Cd-treated cells compared to untreated controls. A positive association was observed between elevated NO levels and increased MMP-2 expression, suggesting oxidative stress-mediated regulation of MMP-2.

Conclusion: Acute cadmium exposure induces oxidative stress and enhances MMP-2 expression in H9c2 cardiomyocytes. The upregulation of MMP-2 may contribute to cardiac fibrosis and remodeling, indicating its potential role as a biomarker of cadmium-induced cardiotoxicity and cardiovascular disease progression.

Key Words: MMP-2, Cardiovascular Diseases, Cadmium, Cardiomyoblast, oxidative stress

Pregnancy Specific Beta-1- Glycoprotein 1 (PSG1) levels in early-onset preeclampsia

Shahana Aziz*, Parijat Gogoi**, Rajeev Goyal***, Binita Goswami****, Manisha Kumar*****
*Resident, Department of Biochemistry, Lady Hardinge Medical College and Associated Hospitals, New Delhi
**Professor, Department of Biochemistry, Lady Hardinge Medical College and Associated Hospitals, New Delhi
***Professor, Department of Biochemistry, Lady Hardinge Medical College and Associated Hospitals, New Delhi
****Professor, Department of Biochemistry, Maulana Azad Medical College, New Delhi
*****Professor, Department of Obstetrics and Gynaecology, Lady Hardinge Medical College and Associated Hospitals, New Delhi
Email : shahanaaziz2014@gmail.com

Introduction: Pregnancy Specific Beta-1-Glycoprotein 1 (PSG1) is a major trophoblastic protein secreted by syncytiotrophoblasts and extravillous trophoblasts. Alterations in PSG1 expression have been implicated in abnormal placentation and hypertensive disorders of pregnancy such as preeclampsia. The **objective of our study was** to evaluate serum PSG1 levels in women with early-onset preeclampsia and assess its association with disease severity.

Methods: A cross-sectional analytical study was conducted on 80 women diagnosed with early-onset preeclampsia (20–34 weeks' gestation). Maternal serum PSG1 concentrations were measured using enzyme-linked immunosorbent assay (ELISA) and compared between severe and non-severe

preeclampsia groups. Statistical analysis was performed using unpaired *t*-test and receiver operating characteristic (ROC) curve analysis to determine predictive value.

Results: The mean serum PSG1 levels were lower in the preeclampsia group (62.33 ± 19.49 µg/mL) compared to the normal physiological range. Among those with preeclampsia, mean PSG1 levels were slightly lower in the severe preeclampsia subgroup (61.44 ± 17.31 µg/mL) compared to the non-severe subgroup (63.17 ± 21.54 µg/mL); however, this difference was not statistically significant ($p = 0.693$. Reduced PSG1 concentrations were associated with impaired placental function, consistent with previous findings that link low PSG1 to an increased risk of preeclampsia.

Conclusion: Although PSG1 levels did not differ significantly between severity groups, their downward trend compared with normal pregnancy suggests potential involvement in the pathophysiology of preeclampsia. Larger, longitudinal studies are required to validate PSG1 as an adjunctive biomarker for early prediction and risk stratification of preeclampsia.

Key words: Early onset Preeclampsia, Pregnancy Specific Beta-1-Glycoprotein 1, ELISA, severe and non severe preeclampsia cases.

Multi-dataset analysis highlighting potential role of HSPE1, CRYAB and Pentraxin3 in Coronary Artery Disease

Annu Kumari, Mayank Deep Singh, Kamna Srivastava
Dr. B. R. Ambedkar Centre for Biomedical Research, University of Delhi, Delhi, India- 110007
Corresponding Author: Dr. Kamna Srivastava (kamna605@gmail.com; ksrivastava@acbr.du.ac.in)

Introduction

Cardiovascular diseases (CVDs) continue to pose a major global health challenge, highlighting the need to identify novel biomarkers that can aid in diagnosis, prognosis, and therapeutic development. This study aimed to explore the expression profiles of key inflammation-associated genes in patients with acute myocardial infarction (AMI).

Materials and methods

Multiple microarray gene expression datasets were retrieved from the GEO database, comprising peripheral blood samples from healthy individuals, as well as patients with Single Vessel Disease (SVD) and Triple Vessel Disease (TVD). Data processing and analysis were performed using R-based bioinformatics tools, with a focus on genes implicated in coronary artery disease (CAD) and inflammation, specifically Pentraxin3 (PTX3), Alpha Crystallin B chain (CRYAB), and Heat Shock Protein 10 (HSPE1). Reference genes were used for normalization and validation of expression patterns. Differential gene expression, log2 fold change, heatmap visualisation, and co-expression mapping were employed to assess expression dynamics across cohorts.

Results

Results revealed a significant upregulation of PTX3 post-MI, elevated HSPE1 expression, particularly in TVD patients, and increased CRYAB expression across all disease groups compared to healthy controls. These findings indicate stage-specific expression of inflammation-related genes, suggesting their potential utility as biomarkers for CAD.

Conclusions

The study highlights the potential of PTX3, HSPE1, and CRYAB as promising candidates for enhancing clinical stratification and management of coronary artery disease.

Keywords

Coronary artery disease, Biomarker, Differential expression, Heat shock protein, Inflammation

GESTATIONAL DIABETES AND ITS INTERGENERATIONAL RISK OF CARDIOVASCULAR DISEASES: A SYSTEMATIC REVIEW AND META-ANALYSIS

Ashley Chen, Breanna Tan, Ruochen Du, Yap Seng Chong, Cuilin Zhang, Angela S.Koh, Ling-Jun Li
Joshilla Oinam, Department of Biochemistry, ABVIMS & RML Hospital, Delhi
joshillaoinam@gmail.com

INTRODUCTION : Gestational diabetes mellitus (GDM) - a glucose intolerance first detected during pregnancy, affects about 14% of pregnancies worldwide. It increases

long-term maternal risk of CVS including hypertension, coronary artery disease, stroke and heart failure, and predisposes offspring to hypertension, endothelial dysfunction and subclinical atherosclerosis.

MATERIALS AND METHODS : A systematic search was conducted in PubMed, Embase, Web of Science and Scopus for studies published between January 1980 and June 2024. Data extraction and meta-analysis were performed up to July 31, 2024 following a predefined PROSPERO protocol. Eligible full-text English studies reporting GDM and its association with subsequent cardiovascular diseases in mothers or offsprings were included. Study quality was assessed using the Newcastle-Ottawa Scale. Maximally adjusted risk estimates were pooled using a random-effects model to calculate risk ratios for overall and specific CVD outcomes.

RESULTS : The meta-analysis included 38 studies comprising 77,678,684 participants. Mothers with prior GDM has a 46% higher risk of developing overall CVD (RR 1.46, 95% CI 1.34-1.59) while offspring showed a 23% increased risk (RR 1.23, 95% C1 1.05-1.45). GDM was also associated with a 20% to twofold higher risk of specific CVD subtypes, including coronary artery disease, heart failure and venous thromboembolism.

CONCLUSIONS : These findings emphasize the increased risk of cardiovascular diseases in mothers and offspring affected by GDM, emphasizing on the need for early preventive measures to reduce the long-term CVD burden in these populations.

KEYWORDS : Gestational diabetes mellitus, inter-generational, cardiovascular diseases, systematic review, meta-analysis.

Network Analysis Evidence for a Novel ATHEROSCLEROSIS- ACE2-DYSBIOSIS Triad: A System-level Approach to Understand Pathological Flow and Drug Repurposing

Prabhakaran V[1], Dharamvir Singh Arya[1], Jagriti Bhatia[1].
Department of Pharmacology, AIIMS New Delhi[1], India.
Corresponding Author: Dr. Jagriti Bhatia; Email: jagritiaiims2012@gmail.com

Introduction: Chronic inflammation is the central driver of atherosclerosis, with gut dysbiosis and angiotensin-converting enzyme 2 (ACE2) acting as key modulators. Although individual links between these components have been reported[1][2][3], a unified mechanistic framework connecting them remains unexplored.

Aims: This study aims to unravel the molecular network linking atherosclerosis, gut dysbiosis, and ACE2 pathways through network analysis.

Methods: A multi-omics network was constructed using literature curation and the OmicsNet platform used to construct network within the protein, metabolite, and microbial interactions. The network was

then analyzed using Guilt by Association (GBA) and topological clustering to identify influential nodes, modules, and pathways. Finally, drug target mapping was applied to this network to identify potential drugs for repurposing.

Results: Network analysis validated the triad by identifying key molecular connectors like interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), solute carrier family 6 member 19 (SLC6A19), and flavin-containing monooxygenase 3 (FMO3). Guilt by Association (GBA) analysis suggested dysbiosis, driven by metabolites like tryptophan and arginine, as the origin of the cascade. This inflammatory response leads to ACE2 downregulation, completing the triad. The analysis revealed 9 statistically significant molecular pathways ($P<0.05$), leading to the identification of 21 repurposable drug candidates including minocycline, canakinumab, progesterone and cefdinir.

Conclusion: This study establishes a novel framework where gut dysbiosis initiates a pathological triad with reduced ACE2 and atherosclerosis, driven by chronic inflammation. Crucially, our findings provide a data-driven basis for repurposing 21 existing drugs as targeted cardioprotective therapies by modulating this triad.

Keywords: Atherosclerosis, Ace2, Dysbiosis and Network pharmacology

Reference(s)

[1] Zhang YH, Zhang YH, Dong XF, et al. ACE2 and Ang-(1-7) protect endothelial cell function and prevent early atherosclerosis by inhibiting inflammatory response. *Inflamm Res*. 2015;64(3-4):253-260. doi:10.1007/s00011-015-0805-1

[2] Hashimoto, T., Perlot, T., Rehman, A. *et al.* ACE2 links amino acid malnutrition to microbial ecology and intestinal inflammation. *Nature* 487, 477–481 (2012). https://doi.org/10.1038/nature11228

[3] Alexandrescu L, Suceveanu AP, Stanigut AM, et al. Intestinal Insights: The Gut Microbiome's Role in SAtherosclerotic Disease: A Narrative Review. *Microorganisms*. 2024;12(11):2341. Published 2024 Nov 16. doi:10.3390/microorganisms12112341

Repurposing FDA-Approved Drugs to Inhibit VAV2 and Prevent Early Foam Cell Formation in Atherosclerosis: Addressing the Young at Risk

Prerna Bansal, Kamna Srivastava
Dr. B.R. Ambedkar Center for Biomedical Research, University of Delhi, Delhi, India–110007

Keywords: VAV2, atherosclerosis, FDA, drug repurposing, foam cells, premature coronary artery disease

Introduction

The growing incidence of coronary artery disease (CAD) among younger individuals underscores an urgent need to target its earliest molecular triggers. Atherosclerosis, the precursor to CAD, begins when macrophages transform into foam cells within arterial walls. VAV2, a guanine nucleotide exchange factor in the CD36–oxidized LDL pathway, mediates this transformation, making it a promising yet unexplored therapeutic target. Intervening at this stage could prevent early vascular changes responsible for premature CAD in the young population.

Materials and Methods

To identify potential inhibitors, FDA-approved drugs from five databases were screened using structure-based computational approaches. Molecular docking, pharmacokinetic filtering, molecular dynamics simulations, MM-PBSA binding free-energy analysis, and principal component analysis were performed to identify stable binders. Lead compounds were validated in THP-1–derived macrophages through MTT cytotoxicity assays and Oil Red O staining to measure lipid accumulation and foam cell formation.

Results

Three drugs—Talazoparib (anticancer), Tivozanib (anti-angiogenic), and Isradipine (antihypertensive)—demonstrated stable binding with VAV2 in silico. Sub-cytotoxic concentrations of Talazoparib significantly reduced foam cell formation, decreasing lipid accumulation by 2.24-fold compared with untreated controls. These results suggest effective inhibition of VAV2-driven macrophage activation in early atherogenesis.

Conclusions

Our study positions VAV2 as a critical and targetable driver of early atherogenic signaling. Repurposing FDA-approved drugs, particularly Talazoparib, offers a rapid and translational opportunity to prevent the onset of foam cell formation that precedes CAD. This approach aligns with the changing face of coronary artery disease—prioritizing early, preventive strategies to protect younger populations now increasingly at risk.

ASSOCIATION OF INSULIN-LIKE GROWTH FACTOR-1 AND INSULIN RESISTANCE IN OBSTRUCTIVE CORONARY ARTERY DISEASE

Akansh Kirodiwal, Prof. Neera Sharma, Prof. Ranjit K. Nath
1. PG Resident, Department of Biochemistry, ABVIMS &RMLH, New Delhi
2. Professor, Department of Biochemistry, ABVIMS &RMLH, New Delhi
3. Professor, Department of Cardiology, ABVIMS &RMLH, New Delhi akanshkirodiwal@gmail.com

Introduction:

Coronary artery disease (CAD) remains a major global cause of morbidity and mortality. Insulin-like Growth Factor-1 (IGF-1) and insulin resistance (IR) are key metabolic regulators influencing vascular function, plaque stability, and disease severity. Their interrelationship in obstructive CAD, however, remains inadequately defined.

Materials and Methods:

A cross-sectional observational study was conducted in the Department of Biochemistry, ABVIMS & Dr. RML Hospital, New Delhi, from February 2024 to June 2025. Sixty angiographically proven obstructive CAD patients aged 18–50 years were enrolled. Fasting glucose, insulin, lipid profile, growth hormone, and IGF-1 were estimated. IR was calculated using the HOMA-IR formula, and IGF-1 was measured by competitive ELISA. Statistical analysis was performed using SPSS v24, with $p < 0.05$ considered significant.

Results:

IGF-1 levels were significantly higher in the high-severity CAD group (221.7 ± 35.2 ng/mL) than in the low-severity group (139.4 ± 34.4 ng/mL; $p < 0.001$). The IGF-1/GH axis and AST also showed strong correlations with disease severity, while HOMA-IR showed only moderate association (AUC = 0.613). ROC analysis identified IGF-1 (AUC = 0.916) and IGF-1/GH axis (AUC = 0.948) as highly sensitive markers of CAD severity.

Conclusion:

Elevated IGF-1 and altered IGF-1/GH axis are significantly associated with obstructive CAD severity, suggesting their potential role as biomarkers for disease progression and risk stratification.

Keywords:

Insulin-like Growth Factor-1, Insulin Resistance, HOMA-IR, Obstructive Coronary Artery Disease.

Docking-Based Investigation of Hydroxycinnnamic acid Interaction with PPARγ and C/EBPα: Implications for Metabolic Regulation

Adra Parvathy T J[1], Dr. A. K. Singhal[2], Dr. (Maj Gen) T.K. Saha[3]
[1]Ph. D. Scholar, Department of Biochemistry, Amrita School of Medicine, Faridabad,
email id: adratjp@gmail.com
[2]Associate Professor, Department of Biochemistry, Amrita School of Medicine, Faridabad, [3]Professor and Head, Department of Biochemistry, Amrita School of Medicine, Faridabad

Keywords: metabolic regulation, Peroxisome proliferator-activated receptor gamma (PPARγ), CCAAT/enhancer-binding protein alpha (C/EBPα), molecular docking, hydroxycinnamic acid

Introduction:
Peroxisome proliferator-activated receptor gamma (PPARγ) and CCAAT/enhancer-binding protein alpha (C/EBPα) are crucial transcription factors that regulate adipocyte differentiation, lipid metabolism, and glucose homeostasis. PPARγ activates the expression of C/EBPα, while C/EBPα further induces and maintains PPARγ expression. This synergistic effect drives the differentiation of fat cells. Targeting these proteins with phenolic compounds represents a promising strategy for managing metabolic disorders such as obesity and type 2 diabetes mellitus. This study aimed to evaluate the binding affinity of hydoycinammic acid toward PPARγ and C/EBPα using computational molecular docking.

Materials and Methods:

Three-dimensional structures of PPARγ and C/EBPα were obtained from the Protein Data Bank, and the ligand structure was obtained from PubChem. Molecular docking was performed with AutoDock 4.0 employing the Lamarckian genetic algorithm. Binding energies, hydrogen bonds, and hydrophobic interactions were analyzed to identify potential active site residues involved in ligand binding.

Results:
Docking results revealed that the hydroxycinnamic acid exhibited a strong binding affinity to both PPARγ and C/EBPα. The ligand formed stable hydrogen bonds with key residues in the ligand-binding domain of PPARγ and showed complementary interactions within the DNA-binding domain of C/EBPα. These findings indicate that the compound may modulate transcriptional activity associated with lipid and glucose metabolism.

Conclusions:
The study highlights the potential dual modulator effect of hydoycinammic acid toward PPARγ and C/EBPα. This, in turn, inhibits the positive feedback loop of PPARγ and C/EBPα promoting fat cell differentiation.

PCSK9 rs505151 Polymorphism and Serum PCSK9 Levels in Acute Coronary Syndrome

Kumari Khushboo[1], Smita Tripathi[2], Saroj Choudhary[3], Ritika Sud[4]
[1]Resident, Dept. of Biochemistry, LHMC
[2]Director Professor, Dept. of Biochemistry, LHMC
[3]Associate Professor, Dept. of Biochemistry, LHMC
[4]Professor, Dept. of Medicine, LHMC

Introduction:

Acute Coronary Syndrome (ACS) is a major global health concern, increasingly affecting younger populations. Proprotein Convertase Subtilisin/Kexin Type 9 (PCSK9) regulates lipid metabolism by promoting degradation of LDL receptors, thereby influencing circulating LDL cholesterol levels. A gain-of-function mutation at the rs505151 single nucleotide polymorphism (SNP) of the PCSK9 gene has been associated with elevated LDL levels and increased cardiovascular risk. This study aimed to investigate the prevalence of this polymorphism in ACS patients and explore its potential association in the Indian population.

Methods:

A cross-sectional observational study was conducted on 50 ACS patients in the Department of Medicine. Blood samples were collected after informed consent and ethical approval. Genomic DNA was extracted, and the PCSK9 rs505151 polymorphism was analyzed using PCR–RFLP. Serum PCSK9 levels were quantified by ELISA, and statistical analysis was performed using the unpaired t-test to compare levels among ACS subgroups.

Results:

The AA genotype predominated (96%), AG genotype was 4%, and GG genotype was absent ($p = 0.88$). Serum PCSK9 levels were highest in STEMI patients (39.83 ± 45.79 ng/mL; p = 0.15), followed by unstable angina (29.70 ± 2.31 ng/mL; p = 0.27) and NSTEMI (27.45 ± 3.46 ng/mL; p = 0.73); differences were not statistically significant.

Conclusion:

The AA genotype of rs505151 predominates among ACS patients. Due to the small sample size, no statistically significant associations were observed. Serum PCSK9 levels were highest in STEMI cases, though not significant. Larger studies are required to confirm these findings. PCSK9 genotyping may help identify individuals at higher risk for ACS in susceptible populations.

Key Words : ACS , STEMI , NSTEMI , ELISA , PCSK9

Association Between Serum Prolactin and Coronary Artery Disease in Chronic Kidney Disease

Rahul Kumar[1], Mugesh Kannan[2], UP Yadav[3], R. K Ranjan[4], Aditi Singh[5], Harsh Vardhan Singh[6]

1- Senior Resident, Department of Biochemistry, NDMC Medical College & HRH
2- DNB Resident, Department of General Medicine, NDMC Medical College & HRH
3- Unit Head, Department of General Medicine, NDMC Medical College & HRH
4- Head, Biochemistry, NDMC Medical College & HRH
5- Associate prof. Department of Biochemistry, NDMC Medical College & HRH
6- Senior Biochemist, Department of Biochemistry, NDMC Medical College & HRH

Keywords: coronary artery disease (CAD), chronic kidney disease (CKDe), Prolactin, electrocardiogram (ECG), echocardiogram (ECHO)

Introduction

chronic kidney disease (CKD) frequently leads to hyperprolactinemia due to reduced renal hormone clearance. Given the elevated cardiovascular risk in CKD patients, this study investigated the association between serum prolactin levels and coronary artery disease (CAD).

Methods

We conducted a cross-sectional study of 100 advanced CKD patients. Comprehensive data collection included demographic information, comorbidities, and laboratory parameters. Serum prolactin levels were measured, and CAD was assessed through electrocardiogram (ECG) and echocardiogram (ECHO). Statistical analyses employed appropriate comparative tests with significance at $p<0.05$.

Results

The study revealed a striking association between hyperprolactinemia and cardiovascular disease. Patients with elevated prolactin levels showed significantly higher prevalence of CAD, confirmed by both ECG and ECHO (60.3% vs 4.8%, $p<0.001$). Additionally, elevated prolactin correlated strongly with increased carotid intima-media thickness ($p<0.001$), indicating accelerated atherosclerosis. These associations remained significant after adjusting for traditional risk factors including age, gender, diabetes, and dyslipidemia.

The study also confirmed high prevalence of hyperprolactinemia (58%) in our CKD cohort. Furthermore, elevated prolactin levels demonstrated strong correlation with disease severity, showing significant associations with higher blood urea, elevated serum creatinine, and reduced creatinine clearance (all $p<0.001$).

Conclusion

Hyperprolactinemia is highly prevalent in advanced CKD and demonstrates a strong, independent association with coronary artery disease. These findings position serum prolactin as a valuable biomarker for cardiovascular risk stratification in CKD patients, potentially enabling earlier intervention and improved management of cardiovascular complications in this high-risk population.

Recurrent Acute Coronary Events Associated with Left Ventricular Thrombus in a Young Adult

Aabiru Aijaz[1], S. Dwivedi[2]
[1]Final-year B.Sc. Cardiovascular Technology Intern, Sharda University, Greater Noida (Internship at National Heart Institute, New Delhi)
[2]Head of Academics, National Heart Institute, New Delhi
Email: aabirukhanak@gmail.com

Introduction

The Incidence of coronary artery disease (CAD) in young adults is increasing progressively due to faulty lifestyle such as smoking, stress, consumption of junk food, overuse of smartphones and disturbed sleep pattern. We recently saw a young adult suffering recurrent myocardial infarction (MI) associated with LV thrombus. This prompted us to report this case.

Materials and Methods:

A single clinical case of a 32-year-old male with recurrent acute coronary events was studied. Patient history, echocardiography, and lifestyle assessment were performed to determine potential risk factors and complications.

Results:

The patient, a chronic smoker under severe psychological stress, experienced two MIs at ages 24 and 30. He reported smoking 15–20 cigarettes per day. Echocardiography revealed a large LV apical thrombus measuring 3.4 × 1.6 cm. No family history of premature CAD was reported. Calculations based on cigarette consumption indicated an estimated 11.7 years of life expectancy lost. Smoking, stress, and sleep deprivation were identified as key contributors.

Conclusion:

This case emphasises the critical importance of early detection of LV thrombus in young adults with MI. Strong counselling for smoking cessation, lifestyle modification, and anticoagulation therapy are essential to prevent recurrent events and improve long-term outcomes.

Keywords:

Recurrent myocardial infarction, Young adults, Chronic heavy smoking, Stress, Left ventricular thrombus, Lifestyle modification.

PREVALENCE OF HYPOMAGNESEMIA AND ITS ASSOCIATION WITH GLYCAEMIC CONTROL IN PATIENTS WITH T2DM

Priyanka Sharma[1], A. K.Singhal[2], Maj Gen T.K. Saha[3]
[1]PhD Scholar, Department of Biochemistry, Amrita School of Medicine, Faridabad
Email- drprisha1991@gmail.com
[2]Associate Professor, Department of Biochemistry, Amrita School of Medicine, Faridabad
[3]Professor and Head, Department of Biochemistry, Amrita School of Medicine, Faridabad

Introduction- Diabetes is a major public health concern having high morbidity due to complications. Mg deficiency has been shown to cause endothelial cell dysfunction, inflammation, and oxidative stress, which are major contributors to atherosclerosis. Mg has received considerable attention for its potential role in improving insulin sensitivity and preventing diabetes and its complications. However, studies on the association between serum magnesium levels and glycaemic control in diabetes have shown inconsistent results.

Aim- To determine the prevalence of Hypomagnesemia in patients with type 2 diabetes mellitus and to evaluate its association with glycaemic control.

Materials and Methods-Cross sectional observational study was conducted on 150 patients with T2DM attending the OPD, casualty or admitted in various wards of tertiary care Centre, North India. Serum Mg levels and HbA1c were estimated. For comparison hypomagnesemic and normo-magnesemic groups of patients were categorized.

Results- Out of 150 Diabetic cases, **39 (26%)** have hypomagnesemia. In present study we found that mean HbA1c was significantly higher in hypomagnesemia group as compared to normo-magnesemia group, indicating poor glycaemic control. A positive association was observed between hypomagnesemia and poor glycaemic control.

Conclusion- A considerable prevalence of hypomagnesemia was found among patients with T2DM.Low magnesium levels were significantly associated with poor glycaemic control. Routine monitoring of serum magnesium level and appropriate supplementation may help improve glycaemic management and reduce the risk of long-term diabetic complications.

Keywords- Type 2 Diabetes Mellitus, Glycaemic control, Hypomagnesemia, Serum Magnesium, HbA1c.

Postmortem study of Atherosclerotic Lesions: Insights into Vascular Pathology and Disease Burden"

Niharika[1], Harsh Vardhan Singh[2], Dhawal Patil [3], Garima Singh [4], Sompal Singh[5]
1- Senior Resident, Department of Pathology, North DMC Medical College and Hindu Rao Hospital, Delhi
2- Senior Biochemist, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi
3- DNB Resident, Department of Pathology, North DMC Medical College and Hindu Rao Hospital, Delhi
4- Specialist, Department of Pathology, North DMC Medical College and Hindu Rao Hospital, Delhi
5- Senior Specialist & HOD, Department of Pathology, North DMC Medical College and Hindu Rao Hospital, Delhi

Introduction: Atherosclerosis is a leading cause of morbidity and mortality worldwide, primarily due to its role in ischemic heart disease. The present study aimed to evaluate the extent and pattern of atherosclerotic changes in the coronary arteries in post-mortem hearts specimens.

Materials and Methods: A retrospective autopsy study was conducted on 100 hearts. Gross examination of heart was done by conventional Inflow-outflow method. Detailed gross and microscopic assessment were done including 4 major coronary arteries - Right Coronary artery (RCA), Left Coronary artery (LCA), Left Anterior Descending artery (LAD) and Left Circumflex artery (LCX).

Results: Out of 100 subjects, 63% had atherosclerotic changes in one or more coronary artery. Mean age of subjects with CAD was 48.38±12.11 years whereas mean age of subjects without CAD was 30.99±12.75years which was statistically significantly higher (p -value <0.001). There was male preponderance among CAD subjects (92.6%). The RCA was the most frequently involved vessel (68% cases) followed closely by LCA (65% cases), LAD (57% cases) and LCX (55.5% cases).

Calcified plaques were seen in 24 cases (38.1%). Among CAD patients with complicated plaque, the mean age was 50.33±10.39 years whereas CAD patients without complication had mean age of 47.18±13.04 years, however this difference was not statistically significant (p -value 0.304). Among the CAD with complications, males comprised of 41.4% cases. This male propensity was statistically not significant (p –value = 0.502).

Conclusion: In contrast to other Indian articles the present study found RCA as the most common vessel involved. Though atherosclerosis of coronary vessels is associated with advanced age and male gender, the frequency of complicated plage has no such association.

Keywords:

Atherosclerosis; Coronary arteries; Autopsy study; Ischemic heart disease; Histopathology.

A STUDY OF GENETIC POLYMORPHISM OF KCNJ11 GENE (rs5219 and rs5215) IN TYPE 1 DIABETES MELLITUS - A CASE CONTROL STUDY"

Aliya Arshad*, Preeti Chauhan**, Anju Seth***
*Post Graduate Student, Department of Biochemistry, LHMC, New Delhi
** Professor, Department of Biochemistry, LHMC, New Delhi
*** Director Professor, Department of Paediatrics, LHMC, New Delhi.
Email id- aliya325@gmail.com

Introduction: The KCNJ 11 gene encodes for ATP-sensitive potassium channel that regulates release of insulin in response to a metabolic state. Single nucleotide polymorphism (SNP) of the KCNJ11 gene especially rs5219 has been linked to Type 2 Diabetes Mellitus. Fewer studies involving Type 1 Diabetes Mellitus have also shown a similar association. Along with rs5219, we also investigated the role of another SNP rs5215 in children suffering from Type 1 Diabetes Mellitus.

Materials and Methods: A case–control analytical study was conducted on 72 children diagnosed with Type 1 Diabetes Mellitus and 72 controls without Diabetes Mellitus. Gene polymorphisms (rs5219 and rs5215) were analysed using DNA isolation, polymerase chain reaction (PCR), and restriction fragment length polymorphism (RFLP). Serum Kir6.2 levels were estimated by ELISA. Statistical analysis was done using SPSS version 29.

Results: The KK genotype of the rs5219 polymorphism and TT genotype of the rs5215 polymorphism was found to be more frequent in cases than in controls. There was no inter-association of two SNPs. Mean serum Kir 6.2 levels were slightly higher in cases compared to controls ($p < 0.001$) and showed a positive correlation with HbA1C levels.

Conclusion: The rs5219 KK genotype and rs5215 TT genotype are associated with Type 1 Diabetes Mellitus along increased levels of Kir6.2. Further large-scale and longer duration studies are required to use kir 6.2 as a potential marker for early recognition of Type 1 Diabetes Mellitus in Children and its association with SNPs involving KCNJ11 gene.

Keywords: Kir 6.2 , Type 1 Diabetes mellitus, rs5219, rs5215 Polymorphism, KCNJ11,

To evaluate Martin's formula and ApoB-100 against direct LDL-C measurement in Diabetics and Non-Diabetic groups

Lalita Barjatiya[1], Kailash Chandra[2], Rajeev Ranjan[3] , Harsh Vardhan Singh [4] , Pinky Garg[5] , Aditi Singh[6], Sompal Singh[7]
[1]Senior Resident, HRH and NDMC College, Malka Ganj
[2]Associate Professor, Faculty of Biochemistry, HIMSR, Jamia Hamdard
[3]HOD, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi
[4]Senior Biochemist, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi
[5]Professor, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi
[6]Assocaite Professor, Department of Biochemistry, North DMC Medical College and Hindu Rao Hospital, Delhi
Email ID- lalitabarjatiya22@gmail.com

Introduction

Dyslipidemia is a significant determinant of ischemic heart disease and stroke. Martin et al. at Johns Hopkins University developed an improved formula for accurate estimation of LDL-C using adjustable parameters. The present study aims to evaluate the efficacy of the Martin formula and apolipoprotein B-100 levels relative to enzymatically measured LDL-C in diabetic and control cohorts.

Material and method

The study included 121 diabetic and 95 non-diabetic participants. Fasting blood samples were analyzed for glucose, HbA1c, total cholesterol, triglycerides, HDL-C, LDL-C (direct), and ApoB100. LDL cholesterol was also calculated using the Martin formula. Data were expressed as mean ± SD, and statistical significance was assessed using standard tests.

Result

Both groups showed strong agreement between the Martin formula and direct LDL-C measurement. Control group: High correlation ($r \approx 0.97$) with minimal bias on Bland–Altman analysis, and Diabetic group similarly strong correlation ($r \approx 0.96$) with acceptable limits of agreement. Overall, Martin's formula reliably estimates LDL-C in both control and diabetic group comparison to ApoB100.

Conclusion

From both groups, Martin's formula showed slightly better agreement with directly measured LDL-C than ApoB-100. However, ApoB-100 remains a strong complementary marker because it reflects LDL particle number rather than cholesterol content. Martin's formula is more accurate for LDL-C estimation, while ApoB-100 provides additional risk assessment value.

Keywords: Diabetes Mellitus, Dyslipidemia, LDL-C, Martin's formula, ApoB100.

ANNUAL CONFERENCE ISAR-DCCON 2024

DELHI CHAPTER OF INDIAN SOCIETY FOR ATHEROSCLEROSIS RESEARCH

in collaboration with Department of Biochemistry,
Hindu Rao Hospital and North DMC Medical College, Delhi

Theme:- Integrating Basic Science and Clinical Insights in Atherosclerosis Management

Venue: Dr. Passey Sabhagaar,
Hindu Rao Hospital & North Delhi Medical College, Delhi

REGISTRATION CHARGES

DC-ISAR Members: **INR 500/-**
*DC-ISAR Non-Member: **INR 800/-**
UG/PG Student (Non-Presenter): **INR 250/-**

*Rs 500/- Annual membership charges are mandatory for non members presenting oral/poster category.

LAST DATE OF ABSTRACT SUBMISSION IS 30TH OCTOBER 2024

Conference Secretariat
Dr. Harsh Vardhan Singh
Organizing Secretary & Secretary DC-ISAR
Department of Biochemistry, Hindu Rao Hospital and North DMC Medical College, Delhi
(M.) +91-98109-10824, Email ID: dcisarconference@gmail.com, www.delhichapterisar.co.in

About Delhi Chapter ISAR

The **Delhi Chapter of ISAR** (Indian Society for Atherosclerosis Research) was established with approval from the national ISAR body during the ISARCON conference on November 25, 2014. It holds the distinction of being the first state chapter in the Delhi-NCR region, created to support the national body's mission of promoting research in the field of atherosclerosis.

Objectives and Activities:

- **Knowledge Promotion:** The primary focus of the ISAR-Delhi chapter is to disseminate knowledge and advancements related to atherosclerosis; a disease characterized by the buildup of plaque in arteries**.**
- **Annual Events:** The chapter organizes annual meetings, symposia, and workshops to raise awareness about the latest developments in research and clinical practice concerning atherosclerosis and its related fields.
- The society brings together professionals from diverse fields, including **Biochemists, Molecular Biologists, Cardiologists, Cardiovascular Scientists, Epidemiologists, Pharmacologists, and Internists.**
- Membership is open to individuals involved in basic and clinical research on atherosclerosis, with the aim of fostering collaboration and innovation across disciplines.

By facilitating collaboration and promoting knowledge exchange, ISAR-Delhi contributes to the advancement of cardiovascular research in India

Welcome Message from Organizing Secretary

We are excited to announce the **Annual Conference of the Delhi Chapter of the Indian Society for Advanced Research (DC-ISAR)**, scheduled for **9th November 2024 at Hindu Rao Hospital & North DMC Medical College.**

Hindu Rao Hospital, one of Delhi's oldest and most respected healthcare institutions, holds the unique distinction of being among the few government hospitals converted into a medical college. It serves as a hub for healthcare education and innovation, hosting a range of academic and professional medical programs.

On behalf of my department and the organizing committee, we warmly invite seniors, colleagues, juniors, and students to join us for this prestigious event. Upholding our tradition of excellence in knowledge sharing, this year's conference promises an enriching experience, featuring **leading experts as faculty, engaging panel discussions**, and an invaluable **platform for young professionals to present their work.**

We are now accepting submissions for **Oral and Poster Presentations**, as well as **Research Articles**, to be featured in the **DC-ISAR Newsletter & Souvenir**. This is a fantastic opportunity for **students, research scientists, clinicians, and faculty members** to showcase their research. Your contributions will bring diverse perspectives and groundbreaking insights to the conference.

We encourage you to spread the word about the conference and this **call for submissions** among your peers, colleagues, and fellow researchers. Together, we can ensure a broad range of contributions that reflect the diverse expertise within our community.

We eagerly look forward to your participation and valuable submissions, and we warmly welcome you to **Hindu Rao Hospital**!

Dr: Harsh Vardhan Singh

DC ISAR OFFICE BEARES

PATRONS


Dr. S. Dwivedi


Dr. D. K. Srivastava


Dr. Jayshree Bhattacharya


Dr. S. B. Sharma

ADVISORS


Dr. Sanjay Tyagi


Dr. Anupam Prakash
President, ISAR


Dr. Amitesh Aggarwal
Secretary, ISAR

ISAR DELHI CHAPTER OFFICE BEARERS


Dr. Ritu Singh
President


Dr. Jagriti Bhatia
President Elect


Dr. Gajender Singh Ranga
Vice President


Dr. Harsh Vardhan Singh
Secretary DC-ISAR


Dr. Anita Bhatia
Joint Secretary


Dr. Rajeev Goyal
Treasurer

DCISAR Executive Members


Dr. Parul Goyal


Dr. Abhishek Jaiswal


Dr. Kamna Srivastava


Dr. Anjali Manocha


Dr Harlokesh Narayan Yadav


Dr. Pradeep Dabla


Dr. Sudheer Arava


Dr. Rachna Agarwal


Dr. Archna Singh


Dr. Bhumika Upadhyay

Chief Guest

Dr. J P Sahwney
Chairperson, Cardiology (SGRH)

Guest of Honour

Dr. Akshay Dharmhara
Director Hospital Administration, MCD

Dr V K Tiwari
Dean

Dr. Anand Narnoliya
Medical Superintendent

Dr. Renu Pathak
Additional MS

Dr Ritu Singh
Organizing Chairperson

Dr Jagrati Bhatia
Organizing Chairperson

Dr. Seema Bhargava
Chairperson, Scientific Committee

Dr Rajeev Ranjan
Organizing Chairman

Dr Harsh Vardhan Singh
Organizing Secretary

Dr Parul Goyal
Joint Secretary

Dr Rajeev Goyal
Joint Secretary

Dr Pinky Garg
Joint Secretary

Dr Aditi Singh
Joint Secretary

Dr Rakesh Dogra
Event Co-ordinator

Dr. Raman Kumar
Event Co-ordinator

Local Organizing Committee

Dr. Aditi Gupta (RGCIRC, Delhi)
Dr. Anjali Arora (SGRH, Delhi)
Dr. Deepak Das (HRH, Delhi)
Dr. Devender Singh Yadav (NIMS, Noida)
Dr. Hemant Sharma (HRH, Delhi)
Dr. Juhi Aggarwal (VPCI, Delhi)
Dr. L.M Srivastava (Advisor, DC ISRCON 24)
Dr. M.N Khan (SIMS, Hapur)
Dr. Mansi Modi (HRH, Delhi)
Dr. Mamta Kankra (SGRH, Delhi)
Dr. Mukesh Meena (LHMC, New Delhi)
Dr. Neera Sharma (RML, Delhi)
Dr. NR Saklani(HRH, Delhi)
Dr. Poonam Aggarwal (SMC, Ghaziabad)
Dr. Rahul (HRH, Delhi)
Dr. Raj Narayan Gupta (SMC, Ghaziabad)
Dr. Rakesh Gupta (JROP, Delhi)
Dr. Ram Sharma (HRH, Delhi)
Dr. Rishabh Rajpoot (MDRC, Gurugram)
Dr. Sangeeta Sharma (IHBAS, Delhi)
Dr. Sanjay Jain(HRH, Delhi)
Dr. Shyam Prakash (AIIMS, Delhi)
Dr. Sompal Singh(HRH, Delhi)
Dr. Sudip Dutta (AIIMS, Delhi)
Dr. Sunil Attri (HRH, Delhi)
Dr. Zahid Ashraf (JMI, Delhi)

Report on ISAR DC Conference 2024

The **ISAR DC Conference 2024** gathered an impressive community of healthcare professionals, medical researchers, and academics dedicated to advancing knowledge in atherosclerosis, cardiovascular diseases, and related biomedical sciences. Held on 9th November 2024 at Passey Sabhagaar, Hindu Rao Hospital & North DMC Medical College, Delhi, the conference featured dynamic sessions, enlightening presentations, and interactive discussions aimed at fostering collaboration and sharing the latest breakthroughs in cardiovascular research and patient care.

1. Conference Kick-off and Opening Ceremony

The event commenced with registration, setting a seamless and organized start to the day. **Dr. Harsh Vardhan Singh** from HRH delivered the welcome address, followed by a traditional **Vandana** led by **Dr. Rajeev Ranjan and Ritika Sinha** to invoke auspicious beginnings. Concurrently, poster presentation sessions showcased cutting-edge research in biochemistry and cardiology, offering attendees a space for active engagement, knowledge sharing, and mutual learning.

2. Young Scientist Oral Presentations

The conference spotlighted young researchers through two engaging sessions of oral presentations:

- **Session 1:** Chaired by **Dr. Pinky Garg (HRH)**, this session featured young scientists presenting their oral presentations & was chaired by **Dr. S.B. Sharma (ESIC)**, **Dr. Pradeep Kumar Dabla (GBP)**, and **Dr. Sudip Dutta (AIIMS)**. The participants presented pioneering research with potential for significant impacts on cardiovascular science. Their presentations highlighted advancements in diagnostic methods, innovative treatment approaches, and novel research findings.
- **Session 2:** Chaired by **Dr. Harnam Kaur (ESIC)**, **Dr. M.N. Khan (SIMS)**, and **Dr. Rajni Dawar (VMCC)**, the session brought forth studies by emerging scientistsin oral presentation category exploring cardiovascular health improvement strategies, disease prevention, and therapeutic advancements, with a focus on real-world applications to enhance patient outcomes.

3. Young Scientist Poster Presentations

4. Interactive and Educational Segments

Throughout the day, structured interactive segments encouraged networking and learning:

- **Tea Break:** This interval allowed attendees to connect, exchange insights, and discuss ongoing research, fostering collaborations within the cardiovascular research community.
- **Mystery of the Mind: Riddles to Relieve Stress:** Conducted by the Biochemistry Department of Hindu Rao Hospital, this session featured riddles to stimulate and relieve stress, adding a playful yet educational break to the conference. Prizes were distributed by a "Santa" figure, creating an enjoyable, engaging experience for participants.

5. Guest Speakers Presentations

The conference's guest speakers delivered in-depth presentations on critical cardiovascular topics:

- **Dr. Shyamal Goswami (JNU)** discussed the role of free radicals and reactive oxygen species in atherosclerosis and cardiovascular diseases. Chaired by **Dr. Nibhriti Das (AIIMS)**, **Dr. Kalpana Luthra (AIIMS)**, and **Dr. Sunil Attri (HRH)**, the session explored oxidative stress and its clinical implications for cardiovascular health.
- **Dr. Anjali Manocha (SGRH)** presented on **Biochemical Markers of Myocardial Damage**, emphasizing these markers' role in early diagnosis, clinical decisions, and patient monitoring.
- **Dr. Deep Narayan Srivastava (HRH)** highlighted imaging and interventional radiology's evolving roles in detecting and managing atherosclerosis, focusing on technology-driven advancements for accurate diagnoses.
- **Dr. Rachana Agarwal (IHBAS)** presented on **Apolipoprotein E Genotype and Stroke Risk**, delving into genetic factors that influence stroke susceptibility and genetic testing's potential in personalized treatment.

6. Inaugural Function

The **Inaugural Ceremony** was a distinguished gathering with **Chief Guest Dr. JPS Sawhney** , (Chairman, Cardiology,SGRH) and **Guest of Honor Dr. Akshay Dharmhara (DHA,MCD)**. Dignitaries also included **Dr. Anand Narnoliya (Medical Superintendent)**, **Dr. VK Tiwari (Dean)**, **Dr. Ritu Singh (President)**, **Dr. Rajiv Ranjan (HOD, Biochemistry)** , **Dr. Harsh Vardhan**

Singh (Organizing Secretary, ISAR DCCON 2024), and **Dr. Rajeev Goyal (Treasurer)**, were felicitated. The ceremony included Lamp lightining, the release of the **conference's souvenir cum newsletter** and the presentation of **DC ISAR Lifetime Achievement Awards** to **Dr. S.B. Sharma** and **Dr. Ritu Singh** in recognition of their outstanding contributions to healthcare and DC-ISAR.

7. Chief Guest Expert Discussion

Focused expert session provided actionable insights on cardiovascular care and disease management

Guidelines on Dyslipidemia Management: Presented by **Dr. JPS Sawhney, Chairman of Cardiology (SGRH)**, this session outlined the latest clinical guidelines on dyslipidemia management, chaired by **Dr. Shridhar Dwivedi (UCMS)**, **Dr. D.K. Srivastava (UCMS)**, and **Dr. Jagrati Bhatia (AIIMS)**.

He emphasized that dyslipidaemia is a significant risk factor for coronary artery disease (CAD), and the Cardiological Society of India (CSI) has developed clinical practice guidelines to address this concern in the Indian population. The guidelines focus on managing elevated low-density lipoprotein (LDL) cholesterol, non-high-density lipoprotein cholesterol (non-HDL-C), triglycerides, and lipoprotein(a). They provide recommendations for various groups, including the elderly, young adults, children, and patients with comorbidities like stroke, kidney failure, and familial hypercholesterolemia. Key recommendations include using non-fasting lipid profiles for risk assessment, starting routine lipid screening at age 18, and setting LDL-C goals based on risk levels, such as <100 mg/dL for low-to-moderate risk, <70 mg/dL for high risk, and <55 mg/dL for very high risk. The guidelines also stress screening for genetic causes and suggest combination therapy with statins and non-statin agents for CAD management. Additionally, for patients with high triglycerides, targeting non-HDL cholesterol is recommended to reduce CAD-related morbidity and mortality.

8. Panel Discussion on Atherosclerosis Prevention and Management:

Moderated by **Dr. Ghanshyam Pangtey (LHMC)**, this insightful panel featured **Dr. J.P.S. Sawhney**, **Dr. Hemant Sharma (HRH)**, **Dr. Sangeeta Sharma (IHBAS)**, **Dr. Ram Sharma (HRH)**, **Dr. Sompal (HRH)**, and **Dr. Ritu Singh (LHMC)**. The panelists engaged in a thorough discussion covering a broad range of strategies for atherosclerosis prevention and management, including lifestyle modifications, pharmacological approaches, advances in laboratory science, and the latest research findings. Atherosclerosis prevention and

management require a comprehensive, multifaceted approach involving cardiologists, pathologists, pharmacologists, and biochemists. Cardiologists focus on clinical management, emphasizing early detection, risk assessment, and lipid-lowering therapies. Pathologists provide insights into the disease's underlying mechanisms and risk factors, including the role of plaque formation and inflammation. Pharmacologists are exploring new treatments, especially for statin-intolerant patients or those at high risk, including novel drugs like PCSK9 inhibitors and therapies targeting inflammation. Biochemists delve into the molecular and biochemical processes of atherosclerosis, contributing to the identification of new biomarkers and therapeutic targets. The combined expertise of these professionals is crucial in reducing the burden of atherosclerosis and improving patient outcomes. During panel discussion, these experts exchanged their experiences and knowledge, fostering a deeper understanding of atherosclerosis and its management among the audience.

Dr. Pangtey's skilled moderation facilitated an interactive and accessible session, making it a standout event that resonated with attendees across all fields. His organization and delivery were instrumental in ensuring the session was both informative and smoothly executed, drawing high participation and engagement from the audience.

9. Special Presentation and Closing presentation

Dr. Zahid Ashraf (Jamia Millia Islamia) concluded the presentations with insights on **Long Non-Coding RNA in Cardiovascular Diseases**, introducing new perspectives in cardiovascular genomics. Chaired by **Dr. Parul Goyal**, **Dr. Rajeev Ranjan**, and **Dr. M.N. Khan**, the session captivated the audience with discussions on the future of genetic research in cardiovascular health. He concluded that, lncRNAs hold great promise to serve as important future therapeutic targets of cardiovascular disease. Future research should examine the potential role of interventions that increase or normalize lncRNA expression.

Following the presentations, the **DC-ISAR General Body Meeting** convened to discuss ongoing projects and future objectives. In a ceremonious handover, the outgoing president and executive committee welcomed **Dr. Jagrati Bhatia** as the new **President of DC-ISAR**. The new Executive Committee was also officially recognized and felicitated.

10. Valedictory Function and Prize Distribution

The **Valedictory Function** celebrated the achievements of young scientists, honoring them with awards for outstanding oral and poster presentations. In the handover ceremony, **Dr. Jagrati Bhatia** was warmly welcomed as the new DC-ISAR President.

11. Vote of Thanks:

Dr. Harsh Vardhan delivered a final vote of thanks, extending appreciation to the Chief Guest, Guest of Honor, and all dignitaries. Special acknowledgments were made for **Dr. Ritu Singh**, **Dr. Anand Narnoliya**, **Dr. VK Tiwari**, **Dr. Renu Pathak**, **Dr. Rekha Jain**, **Dr. Seema Bhargava (Chair of Scientific Committee)**, **Dr. Anjali**, and **Dr. Mamta** for curating the scientific program.

Special thanks were extended to **Dr. Pinky Garg** and **Dr. Aditi Singh** for managing the hall and stage including oral presentations, **Dr. Parul Goyal**, **Dr. Raman Kumar**, and **Dr. Raj Narayan** for preparing the souvenir cum newsletter, and **Dr. Mansi** and **Dr. Rahul** for overseeing registration and poster evaluations. **Dr. Rajeev Goel** acknowledged for efficient account management, and **Dr. Rakesh Dogra** for his pivotal remarkable support as Conference Coordinator.

Industry support was also highlighted with gratitude, as Dr. Harsh Vardhan acknowledged the sponsors and industry partners for their engagement, stalls, advertisements, and product information displays that enriched the experience for attendees.

The conference concluded with a **High Tea** session, fostering further networking among participants. The ISAR DC Conference 2024 proved to be a successful and inspiring platform for advancing research, sparking meaningful discussions, and affirming ISAR's commitment to progress in cardiovascular research and healthcare innovation.

(Dr Harsh Vardhan Singh)

Organising Secretary, ISAR DCCON 2024 &

Secretary, DC-ISAR


ANNUAL CONFERENCE ISAR - DCCON 2024
DELHI CHAPTER OF INDIAN SOCIETY FOR
ATHEROSCLEROSIS RESEARCH
in collaboration with Department of Biochemistry,
Hindu Rao Hospital and North DMC Medical College, Delhi


ANNUAL CONFERENCE ISAR - DCCON 2024

DC-ISAR Lifetime Achievement Award 2024

Dr. Ritu Singh

Dr. S.B.Sharma

ISAR DCCON 2024 Oral Presentation Award

Dr. Komal Sagar
First Prize

Dr. Pratip Jana
Second Prize

Dr. Pratibha Sharma
Third Prize

ISAR DCCON 2024 Poster Presentation Award

Dr. Pulkit Soni
First Prize

Dr. Lamthrila Monzgar
Second Prize

Dr. Anshul Jangra
Third Prize

Dr. Parul Bansal
Third Prize

ISARCON 2025

"Translating Vascular Science into Cardiovascular Health"

Organized By

Indian Society for Atherosclerosis Research

6TH-7TH DECEMBER 2025

36TH ANNUAL CONFERENCE OF INDIAN SOCIETY FOR ATHEROSCLEROSIS RESEARCH

Venue : The Fort Unchagaon by Aspen, Garhmukteshwar, UP

ISAR DCCON 2026

Hosted by Jamia Millia Islamia, New Delhi

The Delhi Chapter of the Indian Society for Atherosclerosis Research (ISAR-DC) is pleased to announce its upcoming annual conference — ISAR DCCON 2026 — to be held at Jamia Millia Islamia (JMI), New Delhi.

Jamia Millia Islamia, a distinguished Central University, is renowned for its excellence in life sciences and biomedical research. With its vibrant academic atmosphere and modern facilities, JMI provides the perfect venue to foster meaningful scientific exchange and collaboration in the field of cardiovascular and metabolic research.

We warmly invite all delegates and researchers to join us for another inspiring and academically enriching event.

- Venue: Jamia Millia Islamia (Central University), New Delhi
- Date: To be announced Soon

Prof. Mohammad Zahid Ashraf
Dean, Faculty of Life Sciences
Professor, Department of Biotechnology
Jamia Millia Islamia, New Delhi

Organized by:
Delhi Chapter, Indian Society for Atherosclerosis Research (ISAR-DC)

ANNUAL MEMBERSHIP OF INDIAN SOCIETY FOR ATHEROSCLEROSISRESEARACH–DELHICHAPTER

Annual Membership of ISAR, Delhi Chapter –Rs 500/-

Special Annual Membership package for Students only–Rs1,000 (for 3 years)

First Name

Last Name

Academic Qualification

Designation

Office Address

Please paste your recent colour passportsize photograph

Office Tel No

E-mail

Mobile Number

Residence Address

Residence Tel No

Area of Research

Details of Publication/Presentation/Poster/ Thesis in Atherosclerosis

Nominated by (Name of Life Member of ISAR National Body/Life Member of ISAR Delhi Chapter and their Life Membership Number)

Please tick one of the following Membership options:

1. **Annual Membership fee: Rs 500**
2. **Special Annual Membership Package for Students* only: Rs1,000 (validfor3years from the date of application)**

   *** Please note: The student has to submit valid proof (Student ID) along with the letter from guide/Head of the Department/Institution verifying that you are currently student in their department/Institution and your date of joining the department/Institution.**

Please tick the payment option A or B

A) For online payment: "Delhi Chapter ISAR"
Account No.3432232021, Central Bank of India, Lady Hardinge Medical College, New Delhi110 001; IFSC code: CBIN0283462

B) Demand draft/Multicity or local cheque in favour of "Delhi Chapter ISAR" and should reach Secretary or Treasurer of ISAR, Delhi Chapter.

DD no /Cheque no **Dated**
Bank details (including Name and Branch)

ESIDENT

Jagriti Bhatia

fessor,
partment of Pharmacology,
India Institute of Medical Sciences,
sari Nagar, New Delhi 110029
bile No. +91-9868394054
mail: jagritiaiims2012@gmail.com

SECRETARY

Dr. Harsh Vardhan Singh

Senior Biochemist
Department of Biochemistry,
North Delhi Municipal Corporation Medical College & Hindu Rao Hospital, Delhi-110007
Mobile No. 91 + 9810910824
E-mail: secretary.delhi.chapter.isar@gmail.com

TREASURER

Dr Rajeev Goyal

Associate Professor,
Department of Biochemistry,
Lady Hardinge Medical Colleg
SSK and KSC Hospitals, New D
110001
Mobile No:+91-8191861234
E-mail:docrajeev@gmail.com

INDIAN SOCIETY FOR ATHEROSCLEROSIS RESEARCH

APPLICATION FORM FOR MEMBERSHIP

PHOTO

Name: Dr. /Mr./Mrs.: - First Name | Middle Name | Surname

Designation & Affiliation: -

Home Address: -

Mobile No.:- E-mail :-

Academic Qualifications: -

Applicant's Signature

Date:

Date of birth:

LIFE MEMBERSHIP FEES ISAR & IAS & STATE CHAPTER: **Rs. 7000** only

PLEASE CHOOSE ANY ONE STATE CHAPTER: DELHI ☐ UP ☐ BIHAR ☐ ASSAM ☐

CHEQUE/ ONLINE TRANSFER DETAILS:

Cheque should be made in name of " Indian Society for Atherosclerosis Research" payable at Delhi.
For online transfer: Indian Society for Atherosclerosis Research; Account no.3009101059892; IFSC Code: CNRB0003009; UCMS AND GTB BRANCH, DELHI.
Send the complete application with DD/ Cheque/ Online transfer details by post to:

Dir. Prof. Amitesh Aggarwal Secretary, ISAR D 72, Sector 20, Noida, UP 201301 Mobile :- 9811060025 E-Mail :- isarsecretary@gmail.com	Scanned copy of form & UPI details can be mailed to-isarsecretary@gmail.com

Date of Approval **Membership Number**

Signature Secretary

LOCAL ORGANIZING COMMITTEE

DR SMITA ROY
ORGANIZING CHAIRPERSON

DR NEERA SHARMA
ORGANIZING CHAIRPERSON

DR PARUL GOYAL
ORGANIZING SECRETARY

DR LOKESH KUMAR SHARMA
JOINT ORGANIZING SECRETARY

DR SANDHYA LAL
JOINT ORGANIZING SECRETARY

DR RASHMI VERMA
JOINT ORGANIZING SECRETARY

DR RAJEEV GOYAL
JOINT ORGANIZING SECRETARY

ADVISORS

DR RITU SINGH
DR JAGRITI BHATIA
DR HARSH VARDHAN SINGH

SCIENTIFIC COMMITTEE

DR SUKANYA
DR RAJNI DAWAR

ORGANIZING COMMITTEE MEMBERS

DR SHAKSHI SINGH
DR PRASHANT SINGH
DR NIRMAL KUMAR
DR APOORVA AGGARWAL
DR MD MUNTAKHAB
DR VANLALTHIAMSANGI

DR ROHAN RAJPUT
DR B RAMESH
DR NAVEEN KUMAR
DR ABHISHEK KUMAR
DR GLORIA TAIVEINAI RO K
DR SANGEETA HALDAR
DR MAHEK NAREDA

DR DASAPALLI VEENA PRIYA
DR SWETA SAROHA
DR SANTOSH KUMAR
DR YOGESH KUMAR
DR AJANABH GOSWAMI
DR AKANSH KIRODIWAL
DR DEVASISH M

DR MOHAMMED SUHAIL SYED ALI
DR JOSHILLA OINAM
DR SONIYA SIWACH
DR SUBHADRA SAHA
DR AASTHA YADAV
DR ANURAG VATS

CHIEF GUEST

DR. (PROF.) ASHOK KUMAR, MD, PHD
DIRECTOR
ATAL BIHARI VAJPAYEE INSTITUTE OF MEDICAL SCIENCES
& DR. RAM MANOHAR LOHIA HOSPITAL
NEW DELHI

GUEST OF HONOR

DR. S.C MANCHANDA, MD,DM
FORMER PROFESSOR AND HEAD, CARDIOLOGY
AIIMS, NEW DELHI
RETIRED CONSULTANT, CARDIOLOGY
SIR GANGA RAM HOSPITAL

MEET THE SPEAKERS

DR. J.P.S. SAWHNEY
CHAIRPERSON
DEPT OF CARDIOLOGY
SIR GANGA RAM HOSPITAL
NEW DELHI

PROF. RAJENDRA K TANGIRALA
DIVISION OF CLINICAL CHEMISTRY
DEPARTMENT OF LABORATORY MEDICINE
KAROLINSKA INSTITUTE, STOCKHOLM
SWEDEN
PRESIDENT & CEO, GUT LEBEN INC
SAN DIEGO, CALIFORNIA, USA

DR. SAFAL
PROFESSOR
DEPT OF CARDIOLOGY
G. B. PANT HOSPITAL
NEW DELHI

PANELISTS

DR. RITU SINGH
HEAD OF THE DEPARTMENT
DEPT OF BIOCHEMISTRY
LHMC & SSK HOSPITAL

DR. VIJAY GROVER
HEAD OF THE DEPARTMENT
DEPT OF CTVS
ABVIMS & DR RML HOSPITAL

DR. R.K. NATH
HEAD OF THE DEPARTMENT
DEPT OF CARDIOLOGY
ABVIMS & DR RML HOSPITAL

DR. JAGRITI BHATIA
PROFESSOR
DEPT OF PHARMACOLOGY
AIIMS, NEW DELHI

DR. AJAY CHAUHAN
DIRECTOR & PROFESSOR
DEPT OF GENERAL MEDICINE
ABVIMS & DR RML HOSPITAL

DR. ANSHITA AGGARWAL
PROFESSOR
DEPT OF ENDOCRINOLOGY
ABVIMS & DR RML HOSPITAL

MODERATOR
DR. PIYUSH JAIN
PROFESSOR
DEPT OF MEDICINE
ABVIMS & DR RML HOSPITAL

AUTHOR INDEX

Fast, Accurate True High-Sensitivity Troponin I Results

They're counting on you

Powered by Atellica® Solution

When every minute counts for cardiac patients, you can be confident in the Siemens Healthineers true High-Sensitivity Troponin I assay to provide results with improved sensitivity and precision at the low end of the assay range.

Combined with the revolutionary STAT capabilities of the Atellica Solution, this 10-minute assay enables clinicians to quickly triage chest pain patients.

See our website or ask your sales representative for more information.

siemens-healthineers.com/TrueTroponin

SIEMENS Healthineers