NEWSLETTER DC-ISAR

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ABOUT DELHI CHAPTER ISAR

Indian Society for Atherosclerosis Research (ISAR) was founded in 1987 with the major aim to advance knowledge of the causes, prevention and treatment of atherosclerosis, cardiovascular and cerebrovascular diseases in the Indian population, as well as to promote research activities both in the basic sciences and clinical fields of atherosclerosis.

Delhi Chapter ISAR (DC-ISAR) was formed following meeting of the National Executive and General body in November 2014. ISAR- Delhi chapter is devoted to promote dissemination of knowledge of various aspects of atherosclerosis and related fields in India.

The DC-ISAR, like its parent body ISAR, is a multi-disciplinary society whose members are biochemists, molecular biologists, pathologists, cardiologists, epidemiologists, pharmacologists, physicians and vascular surgeons.

Delhi Chapter of ISAR (www.delhichapterisar.co.in) is a state chapter of ISAR (www.isar.co.in) which is a member society of IAS ,the International Atherosclerosis Society based in Italy (www.athero.org). Membership of DC_ISAR as well as ISAR, automatically entitles one for membership of International Atherosclerosis Society (IAS) with all its privileges.

ISAR welcomes all personals working in the field of Atherosclerosis as researchers or as clinicians, to join in its vision and contribute to understanding and managing the enigma of Atherosclerosis.

MESSAGE FROM DIRECTOR, PGIMER, RML HOSPITAL

Dr Prof A.K. GADPAYLE
Additional DG, Director & Medical Superintendent,
PGIMER—RML Hospital, New Delhi

It gives me immense pleasure to know that the second symposium of Delhi Chapter ISAR-2016, on "Preventing atherosclerosis—Nutrition and Lifestyle" is being hosted by PGIMER—RML Hospital.

ISAR has a unique vision in providing a multidisciplinary platform for the research and clinical development in the field of atherosclerosis. Together we have to fight this deadly disease which is affecting our population in epidemic proportions. Nutrition and lifestyle modifications remain the mainstay in the prevention of atherosclerosis.

I congratulate all the members, participants, and organizing committee of DC-ISAR for their initiative, and with the CME all the success.

With best wishes,
Dr Prof A.K. Gadpayle

PRESIDENT DELHI CHAPTER-ISAR: MESSAGE

Dr Jayashree Bhattacharjee
Director Professor, LHMC
Ex-Principal, VMMC & Safdarjung Hospital, New Delhi

Lady Hardinge Medical College

It is proud moment for all of us who have been associated with ISAR (Indian Section of IAS) that Delhi Chapter ISAR is having its 2nd Annual Meeting and academic Symposium (CME) at PGIMER- RML Hospital, New Delhi.

Delhi Chapter is advancing forward with rapid steps exploring different facts of atherosclerosis: a global burden of non-communicable life style disease.

In ISAR different basic or clinical research streams merge into a big river of atherosclerosis research changing its pathway or course quite often in last decades.

I personally have been working on endothelial dysfunction and inflammatory aspects of atherosclerosis for the last many years but lots more need to be done for reliable markers and effective management of atherosclerosis.

I wish a 2nd Symposium (CME) of ISAR- Delhi Chapter a grand academic success.

(Dr. Jayashree Bhattacharjee)

SECRETARY DELHI CHAPTER-ISAR: MESSAGE

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Dear Delhi Chapter ISAR members
It is my immense pleasure that we are organizing the second CME of the Delhi Chapter of the Indian Society for Atherosclerosis research at RML Hospital on 6th Aug 2016 under the young dynamism of Dr Parul Goyal.

Delhi is a centre of excellence in cardiac tertiary care as well as in world class research. Along with my very active involvement in the National body (currently as President Elect), it is a pleasure to see the Delhi Chapter ISAR growing encouragingly well, after we all got together to start this state chapter in 2014. DC-ISAR is incidentally the pioneer in being the first state chapter of Indian Society for Atherosclerosis Research (ISAR) with 80 enthusiastic members till now in the Delhi chapter. It holds regular CME's, has informative newsletters and updates on its website.

As the epidemic of CAD continues to surge in spite of all lipid lowering strategies, my contention is that Atherosclerosis is an inflammatory disease on the foundation of a genetic pre-disposition which also influences lipids. My current Project funded by MoHfw and with GB Pant Hospital is exploring this possibility.

Delhi Chapter of ISAR (www.delhichapterisar.co.in) is a state chapter of ISAR (www.isar.co.in) which is a member society of IAS ,the International Atherosclerosis Society (www.athero.org) based in Milan, Italy. This linked membership is a great asset for our members.

The focus of all medical research is, or should be, the translation of research ultimately to patient care. And this is where this multi-diciplinary ISAR society fulfils this need. I welcome all interested in atherosclerosis research and all clinicians with an interest in CAD to be a part of our society.
I would like to appreciate all those who have been very involved in the working of the Delhi Chapter particularly Dr Jayashree Bhattacharjee, Dr Rajni Dawar and Dr Jagriti Bhatia. I would also like to congratulate the members of Delhi chapter who have been elected as office bearers of National body.

Dr RITU SINGH

ORGANISING SECRETARY: MESSAGE

Dr Parul Goyal
Associate Professor
Biochemistry
PGIMER-Dr RML Hospital New Delhi

Dear Members of DC-ISAR,

It is a matter of great privilege and honor to host the 2nd symposium of Delhi Chapter –ISAR on "Preventing Atherosclerosis-Nutrition and Lifestyle" at PGIMER-Dr RML Hospital, New Delhi. I am grateful to the Delhi Chapter –ISAR for giving me this opportunity.

The vision of this society is to provide a common platform where all the researchers working on different aspects of atherosclerosis can come together and share their knowledge, expertise, and exchange notes on the latest developments in this field.

Through the symposium, we have tried to provide a holistic approach towards prevention of atherosclerosis. To begin with there is a workshop on yoga to de-stress ourselves and an interactive session with the dietician. The symposium includes eminent speakers from Endocrinology, Cardiology and Biochemistry. Dr Abbas Mahdi will be exploring the use of NMR spectroscopy based metabolomics for diagnosis of CAD. Dr Anoop Misra, Chairman Fortis –C-DOC, will be talking about the role of correct nutrition and physical activity to prevent atherosclerosis. The speakers will discuss and enrich us on the preventive aspects of atherosclerosis in order to decrease the incidence of the disease. Dr Neeraj Pandit, will be sharing with us his valuable experiences on prevention of CAD. There is also a poster session wherein the varied research work on atherosclerosis will be displayed for all to read and interact with the researchers. The symposium will have delegates and participants from all the premium institutions and hospitals of Delhi -NCR including LHMC, MAMC, AIIMS, Medanta, UCMS, SGRH, VMMC.

I would like to extend my sincere thanks to all those who have been working with me for this symposium, and to all the faculty and staff of PGIMER-Dr RMLH for their support.

I hope all the members, delegates and students will find this symposium interesting and informative.

Regards
Dr Parul Goyal
Organising Secretary, CME 2016 , DC-ISAR

JOINT SECRETARY DELHI CHAPTER-ISAR: MESSAGE

Dr. Rajni Dawar
Jt. Secretary DC- ISAR
Assistant Professor
Deptt of Clinical Biochemistry
LHMC & SSK Hospital

It is a proud privilege to be associated with Delhi Chapter of ISAR which is pioneer in being the first state chapter to be established. A thought that has been enduring in mind when it becomes real; is truly an interesting and exciting experience. The progress of the society mainly depends on many people who are working behind the scenes and planning things to the smallest. Atherosclerosis research is an ever expanding area with multiple factors involved in its pathophysiology starting from conventional lipids to inflammation to micronutrients and genetic and epigenetic modulation. DC-ISAR being a multidisciplinary society forms a very good platform for exchange of ideas on preventive, promotive, diagnostic and management areas of atherosclerosis research. In a short span of two years we have almost 80 members from varied disciplines. A website is being maintained for DC_ISAR (www.delhichapterisar.co.in). With this website we try to update about the activities of the society especially annual CME's being conducted. Any suggestions/communication is welcome on the email secretary.delhi.chapter.isar@gmail.com.
This CME focusing on nutrition and lifestyle for prevention of atherosclerosis being organized will be an academic feast for all the participants.

TREASURER DELHI CHAPTER-ISAR: MESSAGE

Dr Jagriti Bhatia
Treasurer, Delhi Chapter-ISAR,
Additional Professor,
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All India Institute of Medical Sciences, New Delhi

The Delhi Chapter of Indian Society for Atherosclerosis Research has been steadily marching ahead. It is a proud feeling that we are organizing the second symposium of Delhi Chapter on 6th August, 2016 at PGIMER-Dr. RML Hospital, New Delhi. The progressive growth of our Delhi Chapter ISAR society has been made possible due to the inspiration and guidance of our enterprising and devoted visionary members who have helped to build and nurture this society like Prof S Dwivedi, Prof DK Srivastava, Prof S B Sharma, Prof J Bhattacharjee and Prof Ritu Singh. In a small span of two years, the society has seventy-seven members already and this number is increasing. It was possible due to very far-sighted planning and initiative taken by Prof J Bhattacharjee and Prof Ritu Singh. At the time of the launch of the website, we had little resources but the commitment to make things happen has made a difference and this is the reason we stand confident today.
I take this opportunity to congratulate Dr Parul Goyal, Organizing Secretary, and other members in the organizing committee of Delhi Chapter-ISAR Symposium 2016, for their hard work and dedication. Further, it is my request that we stay committed towards our society and also disseminate information regarding our society, its goals and activities among our peers so that our society grows not only in numbers but is also enriched with learned colleagues.
With best wishes and extending warm welcome to all.

Prevention of Coronary Artery Disease

Dr Neeraj Pandit
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PGIMER, RML Hospital

Coronary Heart Disease (CHD) is the single largest cause of mortality globally. Whereas the prevalence of CHD & associated mortality is showing a downward trend in the developed countries, it is the opposite in developing countries like India. The estimated prevalence of CHD in adults is reported to be 3-4% in rural areas & 8-10% in urban setting in India. This is showing a rapid upward trend. According to WHO, 60% of cases of CHD would be from India by the year 2020.

CHD manifest 5-10 years earlier in Indian patients compared to western counterparts. It affects the individual & society in the most productive years of their life leading to great economic loss. Rapid urbanization along with economic & social transformation of the population has been ascribed as the reason for the substantial increase in CHD. Identification of susceptible population exposed to modifiable /preventable risk factors like diabetes, hypertension, smoking & mental stress can help in planning primary prevention strategies.

Tobacco use, obesity especially with increased waist hip ratio, Diabetes, abnormal lipid profile (High LDL, Low HDL, abnormal apo B:apo A-1ratio), hypertension, low consumption of vegetables & fruits, sedentary lifestyle & psychosocial stress are found to be major risk factors accounting for> 90% of population attributable risk of CHD globally. Amongst all the above mentioned risk factors, diet & ApoB:A-1ratio explain >two third CHD cases.

Formulating strategies to create awareness among general population about the modifiable risk factors & creating enabling environment for people to lead healthy lifestyle would be the cost effective public health policy measures.

Possible Use of Proton Nuclear Magnetic Resonance (^{1}H NMR) spectroscopy based metabolomics for the diagnosis of Coronary Artery Disease

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Keywords: Nuclear magnetic resonance, metabolomics, unstable angina

Introduction and Background: ^{1}H NMR spectroscopy based metabolomics is a system biology approach that detects and helps evaluate perturbations in metabolism associated with a disease condition. Combined with multi-variate statistics, it also exhibits potential to discriminate diseased individuals from their healthy counterparts. Previous studies have supported the role of NMR based metabolomics in defining bio-markers related with Coronary Artery Disease (CAD). Despite this, little attention has been paid to the main culprit behind CAD, that is myocardial ischemia. Moreover, the spectrum of CAD is wide and includes Chronic Stable Angina, Unstable Angina and Myocardial Infarction. Presently, the diagnosis and management of CAD is mainly governed by invasive procedures and Cardiac Specific Troponin-T and I. However, ischemic condition during episode of Unstable Angina (UA) is often mis-judged owing to subjective nature of history and lack of potent bio-chemical markers. Given the scenario, a parallel gaze into metabolism with robust ^{1}H NMR would be a welcome asset. In a quest for insight in subtle metabolic perturbations during ischemia in UA patients, we employed ^{1}H NMR spectroscopy.

Materials and methods: UA patients were classified according to Braunwald's classification. Proton NMR spectra were obtained from filtered serum samples of UA (n=65) and healthy controls (HC) (n= 62) using 800 MHz spectrometer. Orthogonal Partial Least Square-Discriminant Analysis (OPLS-DA) was performed to analyse the differences between NMR profiles of cases and controls.

Results: Amino acid, carbohydrate metabolism and energy metabolism were found perturbed in UA patients as compared to HC. A set of five metabolites played major role in differentiating UA patients from HC with 96% sensitivity and 95% specificity. The level of valine, inosine and adenine were found to be decreased in cases as compared to controls while alanine and glutamine levels were found elevated in cases.

Conclusion: Our results reflected the possible role of ^{1}H NMR metabolomics in serving as a less invasive surrogate and complementary method for appraisal of myocardial ischemia in UA.

RECENT ADVANCES IN CARDIOVASCULAR DISEAS
A compilation

Dr Smita Tripathi
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Atherosclerotic plaque is the local manifestation of a systemic disease. With optimal medical therapy and attention to risk factors firmly established as fundamental aspects of management, in the past year, we have nevertheless perceived a shift in the pendulum toward renewed focus on the local plaque. Arrival of diverse new tools for invasive and noninvasive plaque imaging, provide opportunities to describe, not only the anatomy, but also the biology of the plaque in vivo. Advances in understanding of the cellular and molecular basis for plaque progression have helped to define markers of plaque vulnerability. A first theme has been refinement of our understanding of the role of macrophages, monocytes and inflammation in plaque progression. Macrophages and monocytes have been understood classically to contribute to plaque progression through phagocytosis of cholesterol droplets and debris, yielding a sequence of foam cell generation, foam cell death, and formation of the necrotic core, as well as contributing to inflammation and plaque rupture [1]. Whether plaque accumulation of macrophages and monocytes results from infiltration or not, has now become the subject of controversy following recent surprising results suggesting that local proliferation (rather than infiltration) accounts for the majority of these cells within plaques [2].

Furthermore, there is growing evidence that molecular inflammatory mediators associated with leukocyte activation may relate to risk of atherosclerosis disease progression. A key example is interleukin (IL)-6, an inflammatory cytokine associated with increased C-reactive protein production that is released by activated leukocytes and vascular smooth muscle cells at sites of vascular injury. The potential role of IL-6 signaling in atherosclerosis progression was bolstered by recent observations of significant variation in coronary artery disease (CAD) risk with the Asp358Ala allelic variant of the IL-6 receptor [3].

Another key theme of publications last year has been the role of free hemoglobin and oxidative stress in plaque progression. When free hemoglobin is released into plaque as a consequence of intraplaque hemorrhage, heme iron is a potent generator of reactive oxygen species (ROS). Binding of free hemoglobin by circulating haptoglobin attenuates oxidative activity and promotes clearance of hemoglobin by macrophages via the CD163 scavenger receptor. Unchecked, oxidative activity related to free hemoglobin may contribute to plaque progression. In a study of human aortic plaques, a genetic polymorphism at the haptoglobin locus (*Hp2-2*) linked to defective attenuation of heme iron–mediated oxidation was associated with a significant increase in apoptotic macrophages, oxidized phospholipid, and malondialdehyde-like oxidation-specific epitopes [4].

Emerging evidence suggests that, in addition to and independent of ROS, damage to mitochondrial DNA (mtDNA) may promote atherosclerotic plaque progression. In humans, mtDNA damage in leukocytes was associated with plaque high-risk features on radiofrequency (“virtual histology”) intravascular ultrasound (RF-IVUS) [5].

Another key theme was the role of lipid mediators, in particular, high-density lipoprotein (HDL), in plaque biology. Despite a consistent observation of an inverse relationship between high-density lipoprotein cholesterol (HDL-C) and CAD risk in epidemiological studies; however, the mechanistic role of HDL-C in plaque progression has become increasingly controversial [6], fueled by successive negative

trials of HDL-C–raising therapies, most recently dalcetrapib [7]. Controversy was further stoked this year by results of a Mendelian randomization study showing no association between an allelic variant of the endothelial lipase gene (*LIPG* Asn396Ser) and risk of MI despite a significant associated increase in HDL-C [8]. As a possible explanation for these findings, it is plausible that the level of HDL-C is merely the wrong metric of HDL functionality. Recent evidence has highlighted the importance rather of HDL particle number and the HDL proteome in predicting risk and plaque progression [9], as well as additional candidate mediators such as HDL-associated lipoprotein-associated phospholipase A2 [10]. New data on apo-lipoprotein A1 further underscore the importance of taking caution in interpretation of investigations on the basis of lipoprotein particles isolated from plasma or serum, because the distribution and function of these circulating particles may differ substantially from those found in atherosclerotic plaque [11].

The results of current study suggest that uric acid can be considered as marker and potential modifier of metabolic syndrome. As it is very easy to estimate with the availability of very cheap and reliable methods, its utility as a screening biochemical marker will be highly useful in the early diagnosis and management of metabolic syndrome [12].

A new development among treatment modalities is proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor. It is a new class of cholesterol-lowering medications that provide significant reductions in lipids but at a large cost relative to statins. HMG-CoA reductase inhibitors, or statins, have been the primary therapeutic intervention for hypercholesterolemia for decades. They have been successful in reducing the risk of major cardiovascular events and mortality in a wide range of at-risk individuals. However, there are patients for whom statins alone or in combination with other lipid-lowering therapies are not always adequate to reduce cardiovascular risk, even at maximally tolerated doses. Monoclonal antibodies that inhibit proprotein convertase subtilisin/kexin type 9 (PCSK9) have been under development as novel therapies, potentially filling these gaps in current standard therapies for hypercholesterolemia. The first 2 drugs in this new class of anti-hyperlipidemic treatments received U.S. Food and Drug Administration (FDA) approval in mid-2015 [13]. With 2 PCSK9 inhibitors approved and others in the pipeline, there will not only be questions about the role of this new class of drugs in the treatment of hypercholesterolemia, but also comparative questions about whether there are important differences among the drugs. Because these drugs will be taken for many years and are expensive, there are still questions regarding long-term benefit and harm.

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POSTER ABSTARCTS

1. ROLE OF INFLAMMATORY MARKERS IN PREDICTION OF ATHERSCLEROSIS IN OBESE AND NON-OBESE PRE-PUBERTAL AGE GROUP?

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OBJECTIVES

To estimate and compare levels of IL-6, IL-8, CRP and TNF-α in obese and non-obese individuals in pre-pubertal age group.

MATERIALS AND METHODS

Forty healthy obese and non-obese subjects in the age group of 05 to 11 years were selected as cases and controls. BMI exceeding 95th percentile is defined as obese; between 85th percentile and 95th percentile is considered as overweight; below 85th percentile is considered as normal. Subjects with constitutionally large growth, <5 years and >12 years were not included in the study. Plasma CRP (mg/dl), IL-6 (pg/ml), IL-8 (pg/ml), TNF-α (pg/ml) were determined by double antibody method Enzyme Linked Immunosorbent Assay [ELISA].

Statistical method: Mean, standard deviation and student–t test was calculated. Pearson's correlation coefficient was applied. Statistical calculation was done on SPSS software.

RESULTS

In this study it is found that IL-6 is positively correlated with weight (P=0.211), cholesterol (P=0.331) and triglyceride (P=0.271). IL-8 is positively correlated with weight (0.227), waist/hip circumference ratio (P=0.288), cholesterol (P=0.256), LDL (P=0.266), triglyceride (P=0.287) and TNF-α (P=0.207). TNF-α is having negative correlation with HDL (P= -0.143) and positive correlation with VLDL (P=0.239). While, CRP is positively correlated with weight (P=0.211) and negatively correlated with HDL (P= -0.121).

CONCLUSION

It has been found that obesity causes inflammation which leads to endothelial dysfunction and development of atherosclerosis. It is concluded from this study that obesity is precluding to inflammation and might increase the risk of atherosclerosis later.

2. STUDY OF SEX HORMONE BINDING GLOBULIN AND HIGH SENSITIVE C-REACTIVE PROTEIN IN PREECLAMPSIA

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ABSTRACT

Background:

Gestational hypertension and preeclampsia are classified as hypertensive disorders of pregnancy. They are one of the major causes for increased mortality and morbidity in mother and fetus. In these disorders, Placental ischemia and inflammation occur due to impaired trophoblastic invasion in uterine spiral artery. Inflammatory mediators released from ischemic placenta into the circulation lead to insulin resistance which further lead to endothelial dysfunction and hypertension.

Objectives:

a) To study the levels of Sex Hormone Binding Globulin in pre-eclampsia. (SHBG)
b) To study the levels of high sensitive C- reactive protein in preeclampsia. (hs-CRP)

Methods:

a. SHBG is measured by chemiluminescence method.
b. hs-CRP by immunoturbidimetric method.

Results:

The mean concentrations of Sex Hormone Binding Globulin were significantly decreased in women with preeclampsia when compared with healthy pregnant with p value < 0.001. And mean concentration of high

sensitive C- reactive protein increased significantly in women with preeclampsia when compared with healthy pregnant with p value < 0.0001.

Interpretation and conclusion:

This study suggests that insulin resistance may be involved in pathogenesis of hypertensive disorders of pregnancy. Here insulin resistance is measured by decrease in SHBG levels. If SHBG levels are measured in early pregnancy, then we can prevent the development of preeclampsia and its complications.

Increase in hs-CRP is a indicative of inflammation and endothelial dysfunction. This increase in hs-CRP and decrease in SHBG is also an indicator of future risk of developing cardiovascular diseases in these patient groups.

3. MODULATION OF PPARΓ BY *SYZYGIUM CUMINI* ATTENUATES INSULIN RESISTANCE AND B-CELL DYSFUNCTION IN TYPE 2 DIABETIC RATS

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Key words: β-cell dysfunction, inflammation, oxidative stress, streptozotocin, *Syzygium cumini*

Introduction: Since ancient times, Syzygium cumini (SC), also known as jamun, is used as an anti-diabetic agent but mechanism of its action is not known.

Aims & objectives: Therefore, the present study was undertaken to investigate the role of SC aqueous seed extract (200, 400 and 800 mg/kg; p.o.) on insulin resistance (IR), oxidative stress, inflammation and pancreatic β-cell damage in high fed diet/streptozotocin (HFD-STZ)-induced model of type 2 diabetes mellitus (DM) in rats.

Materials & methods: Rats were fed HFD for 35 days to induce IR. On 10^{th} day, a single injection of streptozotocin (40 mg/kg; i.p.) was administered to induce type 2 DM. Subsequently, after confirmation of diabetes on the 14^{th} day, diabetic rats were treated with SC for the next 21 days.

Results & discussions: HFD-STZ rats showed increased serum glucose, insulin, IR, TNF-α, dyslipidemia, pancreatic oxidative stress along with decreased level of hepatic PPARγ level. In addition, morphological analysis of pancreatic tissue demonstrated, pathological changes in islet and β-cells. Treatment with SC for 21 days revert these changes to near normal level in dose dependent manner.

Conclusions: These finding demonstrates that SC protects against HFD-STZ induced type 2 DM due to preservation of β- cell function and reduction in IR *via* increased PPARγ expression.

4. "STUDY OF SERUM SCLEROSTIN LEVELS AND CORONARY ARTERY CALCIUM SCORE IN CORONARY ARTERY DISEASE."

Presenting Author: Verma Pratima

Co-authors: Gupta SK, Mishra TK, Girish MP, Sharma AK

ABSTRACT:

INTRODUCTION:
Vascular calcification is an active and regulated process. Wnt signalling, required for osteoblast function, is also involved in SMC trans-differentiation in both vascular and bone calcifications. Sclerostin is an endogenous antagonist of wnt/B catenin signaling pathway.

AIMS AND OBJECTIVES:

1. To estimate the serum levels of sclerostin, hs-CRP and lipid profile parameters in CAD patients.
2. To correlate sclerostin level with the coronary artery calcium score (CACS) and other markers of atherosclerosis, hs-CRP, ApoA1, ApoB100, lipid profile and carotid intima media thickness (CIMT) in CAD.

MATERIALS AND METHODS:
A hospital based cross sectional study recruiting 80 intermediate risk CAD patients. Subjects were divided in 2 groups CACS=0(n=50) and CACS>0 (n=30) Serum sclerostin Hs-CRP, ApoA1, ApoB100 levels were estimated by ELISA/ immunoturbidimetry kits. CACS and CIMT measured by CT angiography and Color Doppler.

RESULTS:
Sclerostin levels were higher in males than females (p value<0.01) as well as higher in elderly. Sclerostin was significantly higher in subjects CACS>0 than CACS=0 (p <0.01) significant positive correlation was seen between hs-CRP(p<0.01), CIMT (p<0.014)and sclerostin levels.

CONCLUSION:
In our study serum sclerostin levels significantly correlated with hs-CRP, ApoB100 and CIMT and CACS. Hence we conclude that serum sclerostin can act as a marker of coronary artery calcification in CAD.

5. HPTLC FINGERPRINT PROFILE AND PHARMACOLOGICAL EVALUATION OF *CICHORIUMINTYBUS* AQUEOUS SEED EXTRACT

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The use and search for nutritional supplements and medicine derived from plants have increased in recent years. The present study was designed to analyze the preliminary phytochemical constituents, antioxidant activity and characterization of active constituents of *Cichoriumintybus* aqueous extract by High-performance thin layer chromatography (HPTLC).

Objective: To establish the fingerprint profile of *Cichoriumintybus* using HPTLC technique and its phytochemical, pharmacological evaluation.

Methods: Preliminary phytochemical screening was done and HPTLC studies were carried out. CAMAG HPTLC system equipped with Linomat V applicator, TLC scanner 3, Reprostar 3 and WIN CATS-4 software were used. A sample of Chicory seed aqueous extract received from M/S Hamdard Laboratory (A), India, for quality check. Chicory (*Cichoriumintybus* L.) plant seeds were extracted by different extraction methods viz. Reflux (B), Distillation (C), Freeze dry method (D). The pharmacological effects of Chicory seed were evaluated in Wister rats.

Results: The total yields of aqueous extracts of samples B, C and D were 4.6%, 4.9%, and 3% respectively. The phytochemical evaluation of chicory aqueous seed extract showed sample A and sample B had comparatively maximal concentration of total phenolic content (34.8 %) and total flavonoid content (9.1%) respectively. However, Sample C had comparatively highest antioxidant capacity (85.79 %), while sample D had the lowest DPPH antioxidant capacity (68.9 %). HPTLC finger printing of aqueous extract of Chicory seeds at 250 nm of sample A revealed 12 peaks with Rf values in the range of 0.1 to 0.92; Sample B showed 14 peaks with Rf values in the range of 0.04 to 0.99, sample C showed 12 peaks with Rf value in the range of 0.1-0.92 and Sample D revealed 10 peaks with Rf values in the range of 0.1 to 0.92. HPTLC fingerprint profile at 366 nm in sample A showed 12 peaks with Rf value in the range of 0.11-0.90; Sample B, C, and D revealed 10 peaks with Rf value in the range of 0.11-0.90.

The phytochemical evaluation of Chicory seed extract showed good free radicals scavenging activities. The preliminarypharmacological study of *Cichoriumintybus*aqueous seed extract showed hypotriglyceridemic effect on Wister rats.

Conclusion: In conclusion, the results obtained from the qualitative evaluation of HPTLC fingerprint images will be helpful in the identification and quality control of the herbal drug and ensure therapeutic

efficacy. The preliminary pharmacological evaluation of Chicory seeds showed a good candidate drug for further research.

6. ASSESSMENT OF HDL ANTIOXIDATIVE ACTIVITY IN ACUTE CORONARY SYNDROME SUBJECTS

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Key words: HDL, Antioxidative activity, ACS

Aim: High-density lipoprotein (HDL) is known to possess multiple atheroprotective effects. We investigated HDL antioxidative activity in acute coronary syndrome (ACS) patients and healthy controls.

Methods: Statin naïve ACS patients and healthy volunteers were recruited in the analysis. Dihydrorhodamine (DHR) based fluorescent cell free assay was used to evaluate total HDL antioxidative activity. Paraoxonase activity of PON1 was also assessed.

Results: The mean age of ACS patients (n =40) and controls (n= 30) was 50.97±12.8 years and 37.6±11.7 years respectively. Subjects with ACS had similar HDL (41.8± 3.7 mg/dl vs 46±5.3 mg/dl) but significantly lower total antioxidative activity (52.75±16.65 vs 78.02±13.5) and paraoxonase activity (66.45± 28.54 vs 132.4± 48.36 U/ml) as compared with controls. Paraoxonase activity showed positive correlation with HDL Antioxidative activity (r=0.64).

Conclusion: Impaired antioxidative activity of HDL may responsible for its diminished anti-atherogenic effect and therefore may contribute to increased cardiovascular risk.

7. DESIGN AND SYNTHESIS OF SUCCINAMIC ACID DERIVATIVES FOR MANAGEMENT OF DIABETES AND ITS COMPLICATIONS IN STZ INDUCED RATS.

Nikhil Khurana*, Pankaj Sharma**, SB Sharma* and Sunita Bhagat**
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ABSTRACT

Diabetes mellitus is one of the most challenging health problems of the 21st century. India is the diabetic capital of the world with largest diabetic population i.e. 19.4 million in 1995, 22.9 million in 2000 and predicted to have 57.2 million by the year 2025 (Mohan et al, 2007). The anti-diabetic property of E. *Jambolana* is well documented in the literature. Active antihyperglycemic compound (FIIc, a succinamic acid derivative) has been isolated from fruit pulp of E. *Jambolana* (Sharma et al, patent granted). However, the isolation of this natural compound has limitations as E. *Jambolana* is seasonal fruit and its extraction procedure is cumbersome, expensive and produces very less yield. To overcome these limitations there is growing need for efficient designing and developing synthetic strategy for new chemical entities for possible therapeutic value. Our research group has designed and synthesized few succinamic acid derivatives (*1-hydroxypyrrolidine-2,5-dione, methyl4-amino-4-oxobut-2-enolate, methyl 2-hydroxy-4-(hydroxyamino)-4-oxobutanoate* , 4-(benzyloxl) amino-2-hydroxy-4-oxobutanoic acid)which are assessed for having anti diabetic potential. The fall in fasting blood glucose of STZ induced rats after giving the compounds (90 min later) were 30%, 27%, 26%, 22% respectively. Further experiments need to be done to synthesize the parent herbal compound to establish its role as potent anti diabetic agent.

8. REGULATION OF GENE EXPRESSION BY HERBAL COMPOUND IN DIABETIC RATS

Aiman AJ[1], Sharma SB[1], Mehndiratta M[1], Rani U[2], Luthra K[3]

ABSTRACT

Objective: To study the effect of HPLC purified herbal anti-hyperglycemic active compound (FIIc) on the expression of Glucose transporter level in Streptozotocin- Nicotinamide induced diabetic rats.

Methods: 24 Male Wistar rats were taken and diabetes was induced in group B, C and D rats (n= 6 each) by injecting Streptozotocin at a dose of 45 mg/kg of b.w. 15 minutes after administration of Nicotinamide at a dose 230 mg/kg b.w. intraperitoneally to overnight fasted rats. Active compound (FIIc) was given to group C and Pioglitazone to group D at dose of 20 mg/kg of body weight orally for 4 weeks respectively. Total RNA was isolated by using Trizol method from liver and skeletal muscles. Real time expression of GLUT 4, GLUT 8 and Kv 1.3 potassium channel was measured and compared between healthy and diabetic controls.

Results: After treatment with FIIc for 4 weeks there was a 22-fold increase in GLUT 8 expression and 6 fold increase in GLUT 4 expression in skeletal and liver tissues respectively. However, no significant difference was obtained in kv 1.3 expression in liver and skeletal muscles.

Conclusion: FIIc treatment for 4 weeks significantly improves the expression of GLUT 8 and GLUT 4 expression in Liver and Skeletal muscles respectively. Hence it increases the peripheral insulin sensitivity.

9. EVALUATION OF PAPP-A AND MMP-9 FOR EARLY PREDICTION OF PREECLAMPSIA

Karuna Sharma[1], Ritu Singh[1], Vishwajeet Rohil[3], Manisha Kumar[1], Usha Gupta[1], Jayashree Bhattacharjee[1]
Lady Hardinge Medical College[1], Vallabhbhai Patel Chest Institute[2]

Introduction: Preeclampsia (PE) is one of the foremost causes of maternal and fetal morbidity and mortality. It affects 7-10% of pregnancies world-wide. In India the incidence of pregnancy hypertension is reported to 7.8-10%, out of which 6.2% pregnancies are affected by PE. Delayed diagnosis (after 20th weeks) may be a possible cause of the adverse obstetrics outcome associated with the preeclamptic pregnancies, which can be minimized by early detection. In this study the role of MMP-9 and PAPP-A was assessed for first trimester (11^{+0} -13 $+^{6}$) prediction of PE.

Methodology: This study was a part of an ongoing project of preeclampsia screening in which 1653 antenatal women from ANC outpatient department of Lady Hardinge Medical College and SSK hospital were screened. We have retrospectively selected first trimester serum samples of 45 preeclamptic women and 90 normotensive women who were matched for gestational age and sample storage time.

Results: The serum levels of PAPP-A ($p<0.001$) were significantly low in preeclamptic women as compared to normotensive women while no significant variation was observed in serum levels of MMP-9 ($p=0.06$). A significant positive correlation ($p=0.02$) was observed between MMP-9 and PAPP-A.

Conclusion: Low PAPP-A level is attributed to the impaired implantation. Low PAPP-A levels leads to incomplete trophoblast invasion and spiral arteries modulation. Incomplete spiral arteries modulation causes insufficient Circulation and hypoxic placentas, which is the hall mark of PE. Assessment of PAPP-A levels during first trimester may predict women who are at high risk of PE development. PAPP-A and MMP-9 both are secreted from trophoblast. We have found a positive correlation between MMP-9 and PAPP-A which suggests the involvement of these biomarkers in a common pathway in PE pathophysiology. Low PAPP-A serum levels in first trimester may be used as predictive biomarker for PE.

Keywords: Preeclampsia (PE), Pregnancy associated plasma protein –A (PAPP-A), Matrix metalloproteinase-9 (MMP-9)

10. ASSOCIATION OF 1562 C/T MMP-9 GENE POLYMORPHISM AND MMP-9 LEVELS WITH ACUTE MYOCARDIAL INFARCTION

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Introduction: Cardiovascular disease is an epidemic in India in spite of successful lipid lowering strategies.It has been predicted to be the largest cause of death and disability by 2020 with 4.77 million people dying from coronary artery disease. Research today has justifiably shifted to markers of tissue collagenase and inflammatory pathways.

Aims & Objectives: 1) To study levels of MMP-9 in AMI patients as compared to controls. 2) To study association of 1562C/T polymorphism with levels of MMP-9 in same patients and controls.

Material and Methods: 30 diagnosed patients of AMI were enrolled from cardiology department G. B. Pant Hospital, New Delhi as cases, with age and sex matched controls after informed consent.2 ml blood was collected in plain vial for routine biochemical investigations by automated methodologies and plasma MMP-9 levels was estimated using RayBio Human MMP-9 ELISA Kit.

2ml blood was also collected in EDTA vial for studying MMP-9 gene polymorphism. The study of MMP-9 (1562C/T) gene polymorphism was done by extracting DNA from whole blood and DNA was amplified using Polymerase Chain Reactions, using appropriate primers for MMP-9 gene. The fragments of DNA so obtained were resolved by agarose gel electrophoresis after digestion by Sph1 restriction enzymes.

Results: MMP-9 levels in patients having AMI (22.2 ± 1.2 ng/ml) was significantly more (p-value = 0.031) than in healthy volunteers (21.5 ± 1.2 ng/ml). However, MMP-9 levels with CC genotype (22.05±1.56 ng/ml) were not significantly different (p=0.680) from CT genotype (22.28±1.14 ng/ml). CT genotype was found more in study group compared to control which had more of CC genotype. The frequency of C allele was 60 % in the study group which was lower than in the control group where it was 62%. The frequency of T allele was 40 % in study group was higher than in the control group where it was 38 %. The difference in genotype between cases and controls was not significant.

Conclusion: Matrix metalloproteinases (MMPs) are a group of endopeptidases with capacity to cleave components of extracellular matrix, such as collagen and elastin and these may contribute to the pathogenesis of atherosclerosis by facilitating migration of vascular smooth muscle cells through the

internal elastic lamina into the intima of the vessel wall where they proliferate and contribute to plaque formation and its rupture. **The finding in our study of increased levels of MMP-9 in patients of AMI appear congruent with the etiopathogenesis of vulnerable plaque.**

The variant allele of 1562 C/T MMP-9 gene polymorphism might increase the expression of MMP-9 by binding to the transcriptional repressor factor in the promoter and the over expression of MMP-9 has been reported in vulnerable atherosclerotic plaques. Our results also suggest that MMP-9 1562 C/T polymorphism may play role in the development of MI as the levels of MMP-9 may be related to genotype. However, large scale studies are needed to ascertain its significance in AMI.

Key Words: Acute myocardial infarction, MMP-9, 1562 C/T MMP-9 gene polymorphism

CURRENT ACTIVITIES BY DELHI CHAPTER- ISAR

The second symposia by ISAR-Delhi Chapter on "Preventing atherosclerosis—Nutrition and Lifestyle" is being organized by the Department of Biochemistry, PGIMER, RML Hospital on 6th August 2016. The first symposia "Recent Advances in Cardiovascular Diseases "was at LHMC in 2015, after the formation of DC-ISAR in 2014

It is our proud privilege to have as Guest Speakers eminent speakers from Biochemistry, Endocrinology and Cardiology in this symposia which also has nutrition and yoga workshops

The symposia will include poster presentations and quiz contest in which merit certificates shall be awarded to the poster and quiz winners.

It is indeed a matter of great pride and pleasure to welcome more than 80 members and expected 250 delegates who are interested in DC_ISAR and in the future also, we hope to get such overwhelming response from researchers and clinicians from various disciplines to join us and contribute in a big way towards atherosclerosis related research and patient care.

A newsletter is taken out every year and newsletter 2016 is available at the symposia for members and in e-version at website later. The activities of the society are updated in its website www.delhichapterisar.co.in and it can be traced to its international affiliation of IAS at Milan, Italy (www.athero.org) thru the national society www.isar.co.in .

UPCOMING EVENTS

1. ISARCON 2016 – 29th Annual Conference 21-23rd October At Aster Medicity , Kochi Organizing Secretary: Dr. Anil Kumar R, Lead Consultant, Aster Cardiac Sciences, Kochi, Kerala. Website : http://astermedcity.com/isarcon2016

2. ISA 2018-18th International Symposium on Atherosclerosis: Jun 9-15th 2018 Toronto, Canada. Website : http://www.athero.org/

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This newsletter was compiled by Dr Smita Tripathi and Dr Anita Devi