6th BALTIC ATHEROSCLEROSIS CONGRESS

October 11-12, 2013 Riga, Latvia
RADISSON BLU HOTEL LATVIJA, Elizabetes iela 55, Riga

PROGRAM AND ABSTRACTS

www.kardiologija.lv

WELCOME

Dear colleagues and friends,

On behalf of the Organizing Committee I am very pleased to welcome you at the 6th Baltic Atherosclerosis Congress!

The meeting is a continuum of the tradition to hold the Baltic Atherosclerosis Congress every three years in Tallinn, Vilnius or Riga. Despite the recent advances in the cardiovascular prevention and atherosclerosis research, the burden of the cardiovascular morbidity and mortality remains very high in all the three Baltic countries. We believe this meeting will be an excellent platform where views and experiences will be shared among local and international experts for the benefit of our patients and nations.

It is our conviction that the program of the Congress should help practitioners to find answers to the questions they are facing in their daily work. The program will tackle all the major risk factors and mechanisms of atherosclerosis as usually, with special emphasis on lipids, familial dyslipidemias such as familial hypercholesterolemia, and risk estimation in primary prevention.

I wish you an enriching experience and a very pleasant stay in Riga!

Gustavs Latkovskis
Chairman of the Organizing Committee
President-elect of the Baltic Atherosclerosis Society

ABOUT THE CONGRESS

CONGRESS VENUE
Congress will be held in Radisson blu Hotel Latvija Conference center, Elizabetes 55, Riga, Latvia. Hall Omega 1, Hall Omega 2.

OFFICIAL LANGUAGE
The official Congress language is English. Translation to Russian will be provided.

CERTIFICATE
The Certificate of attendance will be available for all delegates. Delegates may pick up the Certificate from 12 October 9:00 at the Registration desk.

ONSITE REGISTRATION FEE
Participant 150 EUR
Student, fellow, resident 75 EUR

REGISTRATION DESK
The registration desk is located on the 1st floor of the Radisson blu Hotel Latvija Conference center. Opening hours:
Friday, 11 October 08:00 – 16:30
Saturday, 12 October 08:00 – 13:30

CONGRESS IDENTIFICATION BADGES
Congress Identification Badge will be provided at Registration desk. There is no admittance to the Scientific Sessions without the Congress badge.

SPEAKERS ROOM
The Speakers are welcome to check their presentations in the Speakers room, located on Conference center, hall Sigma.

POSTER SESSION
The Poster Session will be located on Hall Omega 2, next to Exhibition and Coffee breaks. The Poster viewing is scheduled all day long and will be shown electronically on Plasma screens.

INTERNET
WiFi will be provided in all Congress area. Internet access point will be also provided.

CONGRESS IDENTIFICATION BADGES
Congress Identification Badge will be provided at the Registration desk.
There is no admittance to the Scientific Sessions without the Congress badge.

CONGRESS SECRETARIAT
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ORGANIZING COMMITTEE

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President-elect, Baltic Atherosclerosis Society
Board Member, Latvian Society of Cardiology

Andrejs Erglis MD, PhD, FESC, FACC
Co-Chairman, 6th Baltic Atherosclerosis Congress
President, Latvian Society of Cardiology

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President, Baltic Atherosclerosis Society

Margus Viigimaa MD, PhD, FESC
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Olar Pullisaar MD, Estonia
Arvo Mesikepp MD, PhD, Estonia
Silvia Noodla MD, PhD, Estonia
Remigijus Zaliunas MD, PhD, Habil. Dr., Lithuania

INVITED SPEAKERS

Anu Ambos MD (Estonia)
Center of internal medicine, North Estonia Medical Center

Karl Andersen MD, PhD, FESC (Iceland)
Professor of Cardiology, University of Iceland, Department of Medicine, division of Cardiology, Landspitali University Hospital
Director, Risk Assessment Clinic of the Icelandic Heart Association,
Member of the European Chronic Disease Alliance and the EU Relations Committee on CVD Prevention of the European Society of Cardiology.

Edwin D. Blumberg MD, FACC (USA)
CNJ Cardiology, South Plainfield, NJ

Maija Dambrova PhD (Latvia)
Head, Laboratory of Latvian Institute of Organic Synthesis

Ingrida Domarkiene MD (Lithuania)
Department of Human and Medical Genetics of the Faculty of Medicine, Vilnius University

Vilnis Dzerve MD, PhD, FESC (Latvia)
Chief, Scientific Board, Research Institute of Cardiology, University of Latvia
Vice-President, Latvian Physician's Association

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Chief, Latvian Centre of Cardiology, Pauls Stradins Clinical University Hospital

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Cardiac Centre, East-Tallinn Central Hospital
G. Kees Hovingh MD, PhD (The Netherlands)
Department of Vascular Medicine, Academic Medical Center, Amsterdam

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President-elect, Baltic Atherosclerosis Society
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Department of Physiology, Biochemistry, Microbiology and Laboratory Medicine of the Faculty of Medicine, Vilnius University

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Faculty of Medicine, Vilnius University

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Latvian Centre of Cardiology, Pauls Stradins Clinical University Hospital
Board Member, Latvian Society of Cardiology

Valdis Pirags MD, PhD (Latvia)
Center of Endocrinology, Clinic of Internal Medicine, Pauls Stradins Clinical University Hospital

Zeljko Reiner MD, PhD, FRCP, FESC, FACC (Croatia)
Head, Department of Internal Medicine, University Hospital Center Zagreb
School of Medicine, University of Zagreb
Member of the Croatian National Academy of Sciences and Arts
President, Croatian Atherosclerosis Society
Chairman, EAS Committee for National Atherosclerosis Society Relations

Ligita Ryliskyte MD (Lithuania)
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Rimvydas Slapikas MD, PhD (Lithuania)
Department of Cardiology, Kaunas University of Medicine Hospital, Lithuania

Siim Sober PhD (Estonia)
Researcher, Tartu University, Estonia

Margus Viigimaa MD, PhD, FESC (Estonia)
Past president, Baltic Atherosclerosis Society
Head, Heart Health Centre, North Estonia Medical Centre
Vice president, Estonian Society of Hypertension
Past President, Estonian Society of Cardiology

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INNOVATION&INTELLIGENCE sistēmu inovācijas

VENUE

Radisson blu Hotel Latvija Conference center, Elizabetes 55, Riga, Latvia.
HALL OMEGA 1, HALL OMEGA 2

1. OLAINFARM
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PROGRAM

FRIDAY, OCTOBER 11

09:00 – 09:10 WELCOME
Gustavs Latkovskis, Chairman of the Baltic Atherosclerosis Congress,
President-Elect of the Baltic Atherosclerosis Society
Zita Kucinskiene, President of the Baltic Atherosclerosis Society

SESSION 1
Burden and pathobiology of atherosclerosis
Chairmen: Zita Kucinskiene, Gustavs Latkovskis

09:10 – 09:30 Burden of atherosclerotic diseases in the Baltic States. Vilnis Dzerve (Latvia)
09:30 – 10:00 Understanding atherosclerosis: a historic perspective. Kristaps Zarins (USA)
10:00 – 10:20 Current concepts of plaque instability and neoatherosclerosis. Andrejs Erglis (Latvia)
10:20 – 10:40 New concepts derived from genome wide analyses. Siim Sober (Estonia)

10:40 – 11:00 COFFEE BREAK

11:00 – 11:10 WELCOME ADDRESS
Ingrida Circene, Minister of Health of Latvia
Andrejs Erglis, President of the Latvian Society of Cardiology,
Co-Chairman of the Baltic Atherosclerosis Congress

SESSION 2
Risk estimation for primary prevention
Chairmen: Andrejs Erglis, Margus Viigimaa

11:10 – 11:40 Challenges of risk estimation in primary prevention. Zeljko Reiner (Croatia)
11:40 – 12:00 Advantages and disadvantages of the risk prediction models. Karl Andersen (Iceland)
12:00 – 12:20 Arterial markers of early atherosclerosis: clinical relevance and prognostic value. Ligita Ryliskyte (Lithuania)
12:20 – 12:40 Novel imaging techniques of atherosclerosis hemodynamic assessment. Kristaps Zarins (USA)
12:40 – 13:00 Are we ready to incorporate genes in risk estimation? Lithuanian way towards genomics of coronary heart disease. Ingrida Domarkiene (Lithuania)

13:00 – 14:00 LUNCH

SESSION 3
Management of dyslipidemias
Chairmen: Anu Hedman, Valdis Pirags

14:00 – 14:30 Familial hypercholesterolemia: cascade screening and clinical implications. G. Kees Hovingh (The Netherlands)
14:30 – 14:50 LDL-C goals as the mainstay of prevention. Gustavs Latkovskis (Latvia)
14:50 – 15:10 Statin side effects and safety. Rimvydas Slapikas (Lithuania)
15:10 – 15:30 Do we care about lp(a) and Lp-PLA2? Edwin Blumberg (USA)

15:30 – 15:50 COFFEE BREAK

SESSION 4
Management of dyslipidemias
Chairmen: Rimyldas Slapikas, Anu Ambos

15:50 – 16:20 Emerging therapies of dyslipidemias. G. Kees Hovingh (The Netherlands)
16:20 – 16:40 Update on niacin and fibrates. Anu Hedman (Estonia)
16:40 – 17:00 Omega-3 fatty acids. Edwin Blumberg (USA)
17:00 – 17:20 Understanding HDL. Gustavs Latkovskis (Latvia)

17:20 – 17:40 Elections of the board of the Baltic Atherosclerosis Society

17:40 Welcome reception

SATURDAY, OCTOBER 12

SESSION 5
Lifestyle and traditional risk factors
Chairmen: Vilnis Dzerve, Edwin Blumberg

08:30 – 08:45 High-risk vs. population based strategy for cardiovascular prevention. Karl Andersen (Iceland)
08:45 – 09:00 What is new in the European Hypertension Society 2013 guidelines? Margus Viigimaa (Estonia)
09:00 – 09:15 Are all antihypertensive drugs equal? Gustavs Latkovskis (Latvia)
09:15 – 09:30 Diabetes - targets and means, individualized approach. Valdis Pirags (Latvia)
09:30 – 09:45 CVD risk factors perception and reality in different subpopulations – physicians, medical students, general population, elderly. Zeljko Reiner (Croatia)
09:45 – 10:00 Evidence-based lifestyle changes and nutrition. Iveta Mintale (Latvia)

10:00 – 10:20 Coffee break

SESSION 6
Novel risk factors and targets
Chairmen: Iveta Mintale, Ligita Ryliskyte

10:20 – 10:35 L-carnitine and atherosclerosis: a new potential therapeutic target. Maija Dambrova (Latvia)
10:35 – 10:50 Cardiovascular risk and prevention in patients with chronic kidney disease. Marius Miglinas (Lithuania)
10:50 – 11:05 Vitamin D, calcium metabolism and cardiovascular risk. Virginijus Sapoka, Andrius Bleizgys (Lithuania)
11:05 – 11:20 Thyroid dysfunction and cardiovascular disease. Anu Ambos (Estonia)
11:20 – 11:35 Role of polyphenols in cardiovascular disease. Andrejs Erglis (Latvia)
11:35 – 11:50 Why lycopene is beneficial against cardiovascular disease? Asta Mazeikiene (Lithuania)

11:50 – 12:00 ADJOURN

ABSTRACTS

ASSOCIATION BETWEEN SERUM LYCOPENE CONCENTRATION AND CARDIOVASCULAR MORBIDITY

Mazeikiene A.[1], Kucinskiene Z. A.[1], Kucinskas V.[2]
[1]Department of Physiology, Biochemistry, Microbiology and Laboratory Medicine, Faculty of Medicine, Vilnius University, Lithuania;
[2]Department of Human and Medical Genetics, Faculty of Medicine, Vilnius University, Lithuania

BACKGROUND: Lycopene is a potent antioxidant, and it has been suggested that higher blood lycopene concentration is associated with a decreased risk of cardiovascular diseases (CVD). The goal of this study was to evaluate the association of serum lycopene concentration with CDV morbidity and biochemical blood risk factors of CVD in Lithuanian population.

METHODS: 520 subjects aged 2–85 years were randomly selected from 33 Lithuanian cities. Study group involved 48 professionally diagnosed self-reported cases of CVD (non-fatal myocardial infarction and stroke). Serum concentrations of total (*tarns*+ *cis*) lycopene was determined using high performance liquid chromatography (HPLC) method; total cholesterol, HDL-Ch, LDL-Ch, triglyceride, Apo A-1, Apo B, Lp(a), glucose and hsCRP were measured using standardized procedures. Due to the non-normal distribution of variables, nonparametric tests were applied using IBM/SPSS v20.0.

RESULTS: The median serum lycopene concentration in general study population was 0.566 (SD 0.28) μmol/l. Lycopene concentration varied significantly between different age groups: 2-17 years 0.48(SD 0.23) μmol/l, 18-65 years 0.62(SD 0.28) μmol/l, older than 65 years 0.31(SD 0.25) μmol/l; p=0.00. No significant variation between genders was found (women 0.58(SD 7.78) μmol/l and men 0.55(SD 7.22) μmol/l; p=0.32). Median serum lycopene concentration was significantly lower (p=0.00) in patients with CVD compared to the control group: 0.38(SD 0.28) and 0.60(SD 0.29) **μmol/l, respectively.** Higher serum lycopene concentration significantly correlated with HDL-Ch (r=0.218; p<0.001) and total cholesterol concentrations (r=0.422; p<0.001).

CONCLUSIONS: Median serum lycopene concentration in the study population was one of the lowest compared to concentrations reported in other European countries. Patients with CVD had significantly lower serum lycopene concentration. These findings could partly explain the fact, that cardiovascular morbidity rates in Lithuania are one of the highest in Europe.
The study was supported by LITGEN Project (VP1-3.1-ŠMM-07-K-01-013).

NONINVASIVE FOLLOW-UP – A KEY POINT AFTER LEFT MAIN PERCUTANEOUS CORONARY INTERVENTION

Milana Zabunova[1, 2], Iveta Mintale[1, 2], Inga Narbute[1, 2], Sanda Jegere[1, 2], Ilja Zakke[1], Andrejs Erglis[1, 2]
[1]Pauls Stradins Clinical University Hospital, Latvian Centre of Cardiology
[2]University of Latvia, Faculty of Medicine

BACKGROUND: The purpose of the study: to show the role of exercise test follow-up by reflecting the control of patients' compliance and medication adherence in order to optimize management process and monitoring of the outcomes after percutaneous coronary intervention (PCI).

METHODS: The observational study (2009 – 2011) included the patients (n=405) with left main (LM) stenotic lesion (more than 50% of the artery lumen diameter) and performed LM PCI, which were followed by performing exercise test for every three months after invasive treatment.
The patients groups with regular and irregular exercise test follow-up were analyzed.
Phone follow-up survey performed in 364 patients, patients with exercise test follow-up – 136. Mean follow-up period – 19.38 ± 8.16 months. The information included clinical events and medication adherence.

RESULTS: Myocardial infarction developed in 1.2% vs 3.8% of the patients with regularly and irregularly performed exercise test and stroke was documented in one patient with irregular control visits (12 months after LM PCI).
Significant difference was observed in use of statins therapy – 97.6% vs 86.3% in both groups (p=0.01).

CONCLUSIONS: 1) Difference in use of medications shows the significance of regular follow-up, especially in high-risk patients compliance (LM group); 2) Regular follow-up is evaluated as significant as invasive treatment in management process of the outcomes; 3) Precisely preplanned, regularly performed of high quality follow-up can be like „a tool" to stabilize the positive benefit of invasive treatment and clinical outcomes control in the future.

PREVALENCE OF CONCOMITANT USE OF CLOPIDOGREL AND PROTON PUMP INHIBITORS IN LATVIAN PATIENTS: A WAY TO PERSONALIZED TREATMENT DEVELOPMENT

Katrina Pukite[1], Inga Urtane[2], Silvija Berzina[2], Aina Silvija Stokmane[2]
[1]Magister Study Programme "Clinical Pharmacy"
[2]Department of Pharmaceutical Chemistry

BACKGROUND: Dual antiplatelet therapy (DAT) has become a cornerstone of management of patients with acute coronary syndrome (ACS) and after percutaneous coronary intervention (PCI). Patients receive PPIs (proton pump inhibitors) to reduce the risk of gastrointestinal bleeding that is the most important side-effect of DAT. The aim of this study was to analyze the prevalence of use of DAT with concomitant PPIs therapy in patients after PCI.

METHODS: In this retrospective descriptive study were involved 70 patients from first care physicians' clinical practices in Riga, Latvia, receiving DAT, during 2010-2011 years. The risk factors of increased gastrointestinal bleeding was: age >70 years, male, smoking status, diabetes mellitus, heart failure symptoms, gastrointestinal bleeding events. Data were analyzed using SPSS statistics 17.0.

RESULTS: From 70 patients 40 (57.1%) were male. Median age of patients was 64±8 years, 21 (30.0%) after 70 years. The main clinical diagnosis were: myocardial infarction (23 patients (32.9%)), PCI (61 (97.1%)), stroke (12 (17.1%)) and unstable angina (25 (36.8%)). Patients were divided into two groups, depending on used PPI type. In the study drug-drug interaction in high-risk group were 27 (67.5%) patients, received potential CYP2C19 inhibitors - omeprazole or esomeprazole. Approximately one half of the study participants (30 (42.86%) patients) did not use PPI at all.

CONCLUSIONS: A significant number of patients were taking a combination of DAT and PPIs, mainly omeprazole. It is important to consider pantoprazole as an alternative to omeprazole patients individually.

NONINVASIVE IDENTIFICATION OF LEFT MAIN CORONARY RESTENOSIS

Milana Zabunova[1, 2], Iveta Mintale[1, 2], Inga Narbute[1, 2], Sanda Jegere[1, 2], Ilja Zakke[1], Andrejs Erglis[1, 2]
[1]Pauls Stradins Clinical University Hospital, Latvian Centre of Cardiology
[2]University of Latvia, Faculty of Medicine

BACKGROUND: The purpose of the study: noninvasive identification of patients with left main (LM) coronary artery restenosis using exercise test.

METHODS: The observational study was implemented from 2002 till 2011. The patients (n=513) with LM stenotic lesion (more than 50% of the artery lumen diameter) and performed percutaneous coronary intervention (PCI) of LM were included and consequently observed in defined follow-up visits by performing exercise test every three months after LM PCI. All patients underwent control coronary angiography. Two study patients' groups were extracted – with and without LM restenosis.

RESULTS: There was no significant difference between both groups in evaluation of demographic parameters and risk factors. Restenosis was detected by clinical evaluation according to classical criteria (ST-segment deviation, ST/pulse index) and by control coronary angiography. The additional predictable parameter had been revealed on exercise testing (diagnostics in early follow-up period after PCI) – double [rate-pressure] product (DP). The results show the significance of DP targeted monitoring at exercise testing follow-up – in patients with LM restenosis it was less than 200 and without LM restenosis – more than 220 ($p < 0.001$).

CONCLUSIONS: 1) Powerful predictable capability of exercise test to identify high-risk patients; 2) Exercise testing – early identification of left main coronary restenosis; 3) Exercise testing follow-up – the control and prediction of stable period for high-risk patients.

ASSOCIATIONS OF SERUM 25-HYDROXY-VITAMIN D WITH BIOMARKERS OF ENDOTHELIAL DYSFUNCTION: FIRST RESULTS FROM REPEATED CROSS-SECTIONAL STUDY OF YOUNG MEN

Andrius Bleizgys, Virginijus Šapoka
Vilnius University

BACKGROUND: Soluble cell adhesion molecules (AM) could serve as biomarkers of endothelial dysfunction (ED), and vitamin D was shown to have beneficial effects on endothelium. We hypothesized that associations between levels of 25-hydroxy-vitamin D (25OH-D) and levels of soluble AM could have a reciprocal character.

METHODS: Randomly selected healthy men of age 20-39 were examined at February or March (cold season) and reexamined at August or September (warm season). Data about some nutrition, smoking and physical activity habits were recorded. Anthropometrical (height, weight, waist and hip circumference, arterial blood pressure) and laboratory measurements (general blood count, lipids, high sensitivity CRP, glucose, 25OH-D, and serum soluble adhesion molecules - ICAM-1, VCAM-1, E-selectin and P-selectin) were performed.

RESULTS: 64 men were examined during cold season, 96.9 % had low vitamin D. 61 men were reexamined during warm season, and 25.2 % retained low vitamin D status. In warm season, levels of soluble VCAM-1 were significantly lower, while soluble ICAM-1 and P-selectin levels were higher in comparison to cold season. Cross-sectional analysis of the cold season data showed associations of 25OH-D with soluble E-selectin; however, levels of soluble E-selectin have changed insignificantly.

CONCLUSIONS: Firstly, low vitamin D appears to be an actual problem not only during cold season. Secondly, only one soluble AM demonstrated clear seasonality reciprocal to 25OH-D changes, and two other AM have changed in a manner opposite to our hypothesis. We suggest that there could be different modulation paths, depending on vitamin D, for AM expression and soluble AM generation.

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- **In angina patients**[1]
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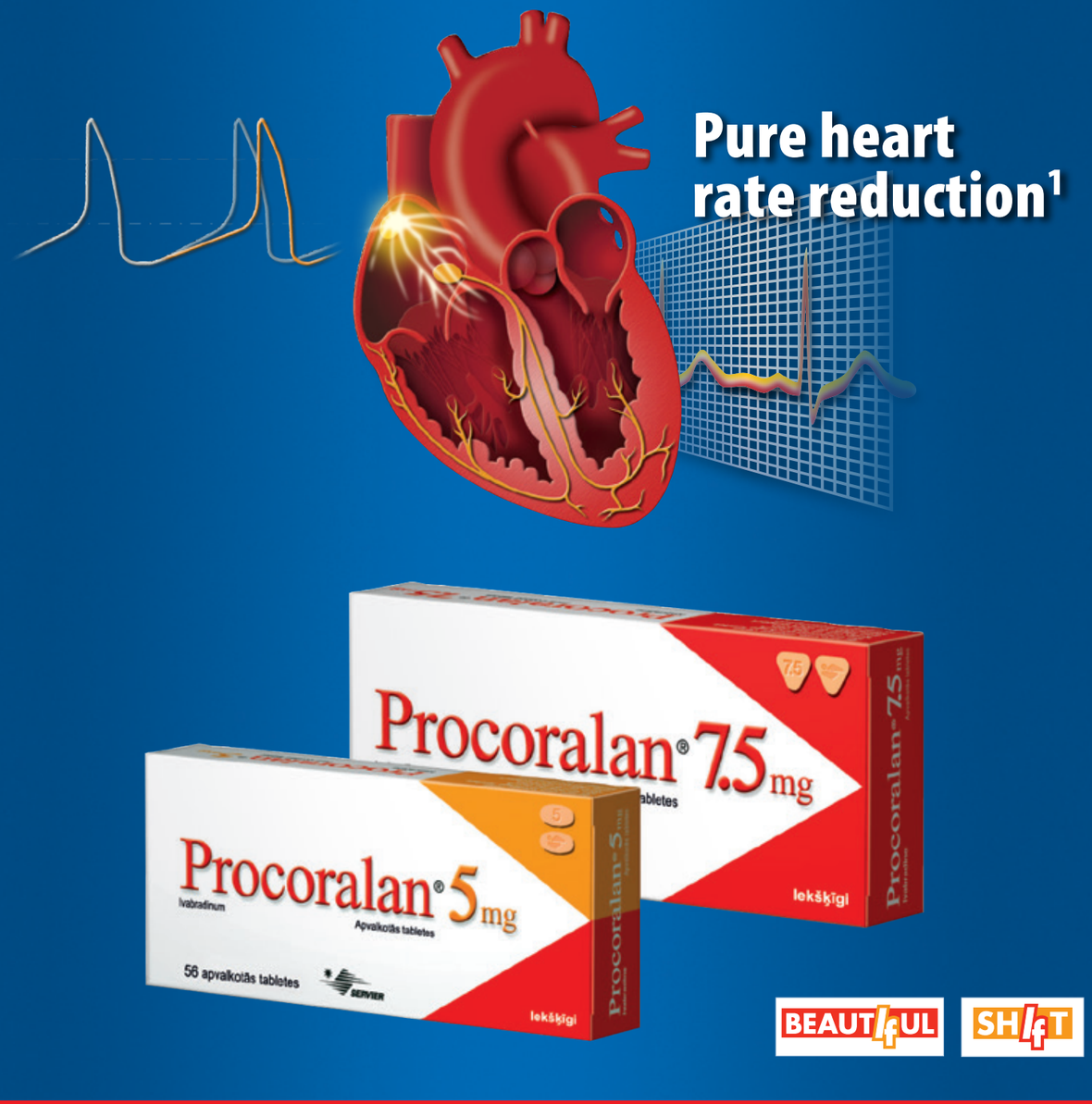
- **In heart failure patients**[3]



1 tablet 2 times daily

1. Procoralan Summary of Product Characteristics (SPC): www.ema.europa.eu
2. Fox K, Ford I, et al; BEAUTIFUL Investigators. Effect of ivabradine on cardiovascular outcomes in patients with stable coronary artery diseaseand left-ventricular systolic dysfunction with limiting angina: a subgroup analysis of the randomized, controlled BEAUTIFUL trial. Eur Heart J. 2009;30:2337-2345.
3. Swedberg K, Komajda M, Böhm M, et al. Ivabradine and outcomes in chronic heart failure (SHIFT): a randomised placebo-controlled study. Lancet. 2010;376:875-885.

COMPOSITION*: Procoralan 5 mg: film-coated, scored tablet containing 5 mg ivabradine; Procoralan 7.5 mg film-coated tablet containing 7.5 mg ivabradine. Contains lactose as an excipient.
INDICATIONS*:
Treatment of coronary artery disease:
Symptomatic treatment of chronic stable angina pectoris in coronary artery disease adults with normal sinus rhythm. Procoralan is indicated:
- in adults unable to tolerate or with a contra-indication to the use of beta-blockers, or
- in combination with beta-blockers in patients inadequately controlled with an optimal beta-blocker dose and whose heart rate is >60 bpm.

Treatment of chronic heart failure: Ivabradine is indicated in chronic heart failure NYHA II to IV class with systolic dysfunction, in patients in sinus rhythm and whose heart rate is ≥75 bpm, in combination with standard therapy including beta-blocker therapy or when beta-blocker therapy is contra-indicated or not tolerated.
DOSAGE AND ADMINISTRATION*: The starting dose in coronary artery disease patients and in patients with stable heart failure is 5 mg orally twice daily during meals: breakfast and dinner. Depending on the therapeutic response, the dose may be increased to 7.5 mg twice daily after 3 to 4 weeks of treatment in coronary artery disease patients, and after 2 weeks in heart failure patients for whom heart rate is persistently above 60 bpm. In heart failure patients, if heart rate is between 50 and 60 bpm, the dose of 5 mg twice daily should be maintained. If heart rate decreases persistently below 50 bpm at rest or in the presence of symptoms related to bradycardia such as dizziness, fatigue or hypotension, treatment should be downtitrated from 7.5 mg to 5 mg or from 5 mg to 2.5 mg twice daily. Treatment must be discontinued if heart rate is below 50 bpm or symptoms of bradycardia persist.
CONTRAINDICATIONS*: Hypersensitivity to the active substance or to any of the excipients; resting heart rate below 60 bpm prior to treatment; cardiogenic shock; acute myocardial infarction; severe hypotension (< 90/50 mmHg); severe hepatic insufficiency; sick sinus syndrome; sino-atrial block; unstable or acute heart failure; pacemaker dependent (heart rate imposed exclusively by the pacemaker); unstable angina; AV block of 3rd degree; combination with strong cytochrome P450 3A4 inhibitors such as azole antifungals (ketoconazole, itraconazole), macrolide antibiotics (clarithromycin, erythromycin per os, josamycin, telithromycin), HIV protease inhibitors (nelfinavir, ritonavir) and nefazodone (see interactions section); pregnancy, lactation (see fertility, pregnancy and breastfeedingsection).
WARNINGS*: Special warnings: Cardiac arrhythmias: ivabradine is not recommended in patients with atrial fibrillation and other cardiac arrhythmias that interfere with sinus node function, monitor regularly ivabradine-treated patients for the occurrence of atrial fibrillation. Monitor also closely patients with chronic heart failure and intraventricular conduction defects; AV-block of 2nd degree: use not recommended; low heart rate: treatment must not be initiated below 60 bpm, during treatment, if resting heart rate decreases persistently below 50 bpm or in case of symptomatic bradycardia, the dose must be down-titrated or treatment discontinued if it persists; combination with calcium channel blockers (e.g. verapamil, diltiazem): not recommended; chronic heart failure NYHA class IV patients: use with caution; stroke: not recommended immediately after a stroke; visual function: use with caution in patients with retinitis pigmentosa. Precautions for use: Hypotension: use with caution; atrial fibrillation - cardiac arrhythmias: non urgent DC-cardioversion should be considered 24 hours after the last dose of ivabradine; patients with congenital QT syndrome or treated with QT prolonging medicinal products: use should be avoided; hypertensive patients requiring blood pressure treatment modification: blood pressure should be monitored; excipients: contains lactose.
INTERACTIONS*: *Contra-indicated:* strong CYP3A4 inhibitors. *Not recommended:* QT prolonging medicinal products, Moderate CYP3A4 inhibitors (verapamil and diltiazem). *With precautions:* Potassium-depleting diuretics (thiazide diuretics and loop diuretics), other moderate CYP3A4 inhibitors, grapefruit juice, CYP3A4 inducers.
FERTILITY*. PREGNANCY and BREASTFEEDING*: contra-indicated.
DRIVE AND USE MACHINES*: Possible occurrence of transient luminous phenomena should be taken into account.
UNDESIRABLE EFFECTS*: *Very common:* Luminous phenomena (phosphenes). *Common:* Headache, blurred vision, dizziness, bradycardia, AV 1st degree block (ECG prolonged PQ interval), ventricular extrasystoles, uncontrolled blood pressure. *Uncommon:* Eosinophilia, hyperuricaemia, syncope, vertigo, palpitations, supraventricular extrasystoles, hypotension, dyspnoea, nausea, constipation, diarrhoea, angioedema, rash, muscle cramps, asthenia, fatigue, elevated creatinine in blood, ECG prolonged QT interval. *Rare:* Erythema, pruritus, urticaria, malaise. *Very rare:* Atrial fibrillation, AV 2nd degree block, AV 3rd degree block, sick sinus syndrome.
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SERVIER

LITHUANIAN WAY TOWARDS GENOMICS OF CORONARY HEART DISEASE

Ingrida Domarkienė[1], Aidas Pranculis[1], Šarūnas Germanas[1], Audronė Jakaitienė[1], Vilma Dženkevičiūtė[3], Vaidutis Kučinskas[1], Zita Aušrelė Kučinskienė[2]
[1]Department of Human and Medical Genetics, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
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BACKGROUND: Previous Linkoping-Vilnius CHD risk assessment study demonstrated differences of atherosclerotic process between Lithuanian and Swedish male individuals. Other study aimed to identify the potential causes of these particular disparities and developed an array of genetic markers' for the search of candidate genes for atherosclerosis and CHD. The results lacked significant values of SNP and disease association. Novel genotyping techniques and platforms provide an improved opportunity for a more precise analysis of whole genome variation associated with human complex diseases. The aim of the study was to find novel (previously not analysed) significantly associated SNPs in potential candidate genes and evaluate their possible effect on the susceptibility of CHD.

METHODS: The DNA samples of 31 families of CHD patients were genotyped using 770K SNPs Illumina® genome-wide genotyping array HumanOmniExpress-12 v1.0. For statistical analysis of autosomal SNPs the transmission disequilibrium test (TDT) using McNemar test was performed. The empirical power and OR (CI 95%) were calculated. The significance level was set at 10^{-4}. Adaptive permutation procedure was used.

RESULTS: The analysis extracted 12 SNPs statistically significantly associated with the CHD phenotype in the group of 31 males and their parents. Regarding the statistical power and the size of the effect represented by the OR values, two candidate genes, *RTN4* and *FBXL17,* emerged. Their function has been analyzed in other studies worldwide and the role in the atherogenesis mechanisms has been shown. Therefore our results are consistent with these findings.

CONCLUSIONS: Study results suggest *RTN4* and *FBXL17* genes as potential candidate genes involved in CHD risk in the Lithuanian male population. In addition, the genotypes of the significantly associated SNPs may be informative and specific for the genetic risk of CHD evaluation in the Lithuanian population and could be taken under consideration in further hypothesis validation.

ASSOCIATIONS BETWEEN RISK FACTORS FOR ATHEROSCLEROSIS AND RATIO OF INTRA-ABDOMINAL TO TOTAL ABDOMINAL FAT VOLUME

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BACKGROUND: Excess body fat distributed in the abdominal area increase the risk of cardiovascular disease (CVD). Intra-abdominal (IA) and subcutaneous adipose tissue (SA) alone provide insufficient information regarding the relative distribution of body fat. We hypothesised that the propensity to store energy in IA relative to total abdominal fat (TA) depots may be associated with risk factors for atherosclerosis, and tested this hypothesis using the IA_{vol} /TA_{vol} ratio as a metric of fat distribution.

METHODS: The study included 80 subjects *(40/40 F/M; age of 38.8 4.3*). We obtained CT scans starting at the upper edge of the left kidney and continuing until the lower edge of the right kidney (Th_{11}–L_3). The IA_{vol} was measured by drawing a line within the muscle wall surrounding the abdominal cavity. TA_{vol} was calculated by adding SA_{vol} volume to IA_{vol}. Subjects were divided into quartiles for IA_{vol}/TA_{vol} ratio: Gr.-1 (Q_1 < 25%), Gr.-2 (25% < Q_2 < 75%) and Gr.-3 (Q_3 > 75%). Total plasminogen activator inhibitor (tPAI)-1, vascular adhesion molecule (sVCAM)-1, intercellular adhesion molecule (sICAM)-1 and E-selectin serum levels were measured by Luminex xMAP technology. *Biochemical parameters (total cholesterol, TC; triglycerides, TG; high and low density lipoproteins, HDL and LDL; apolipoprotein, Apo-A1; LDL/HDL ratio; non-HDL) were measured* using *standard* procedures.

RESULTS: IA_{vol} was higher in men than women ($p < 0.01$), while TA_{vol} was not significantly different ($p = 0.42$). Higher IA_{vol}/TA_{vol} ratio was associated with dyslipidemia – the level of HDL and Apo-A1 were significantly ($p = 0.02$) decreased but levels of TG, TC, LDL as well as non-HDL and LDL/HDL ratio were significantly ($p < 0.01$) increased in Gr.-2 and Gr.-3 in comparison with Gr.-1. Serum level of tPAI-1, sICAM, sVCAM and E-selectin were significantly higher ($p < 0.01$) in Gr.-3 in comparison with Gr.-1 and Gr.-2.

CONCLUSIONS: IA_{vol} /TA_{vol} ratio has strong associations with significant changes in lipid profile and increased levels of tPAI-1 and adhesion molecules resulting in dyslipidemia and an increased risk for atherosclerosis.

ASSOCIATION OF CORONARY AND CAROTID ARTERY PLAQUE COMPOSITION BY IVUS-VH WITH STENT RESTENOSIS AND PLAQUE PROGRESSION

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BACKGROUND: Atherosclerosis is a systemic inflammatory disease involving multiple arterial beds. Despite differences in the carotid and coronary vasculature, both vascular distributions are believed to share common pathway in disease progression. The aim of the study was to compare frequency of carotid and coronary restenosis and its composition by virtual histology IVUS in patients with relevant atherosclerosis involving coronary and carotid arteries.

METHODS: In Latvian Center of Cardiology one hundred consecutive patients with concomitant coronary and carotid artery disease defined as ≥50 % stenosis were included into a single-center, prospective study. All patients were scheduled for carotid and/or coronary artery stenting and underwent IVUS-VH (Eagle Eye™; Volcano Therapeutics Inc; CA, USA) examination of coronary and carotid plaque. Angiography and IVUS-VH follow-up was done after 16 month.

RESULTS: A total of one hundred consecutive patients (60 men and 40 women), mean age 69.6±8.4 years, were enrolled. 78 patients with significant carotid stenosis and underwent carotid stenting, 36 patients underwent PCI for investigated plaque. Follow-up was performed after mean 489 days (95% CI 507.0 - 631.8). 75 patients underwent follow-up angiography with IVUS-VH for the same carotid and coronary artery, 7 patients completed only clinical follow-up, 11 patients died (7 CV deaths), 7 patients refused follow-up. In 1 case of 57 implanted carotid stents (1.8%) stent restenosis ≥ 50% was found. 3 of 17 patients (17.6%) had significant atherosclerotic plaque progression and consequent carotid stenting was done on follow-up. Coronary stent restenosis rate was 25.8% (8 of 31 patients). Comparison between coronary and carotid artery plaque composition according to IVUS-VH was done. We haven't found difference in unstented carotid plaque tissue composition by VH-IVUS at baseline between progressive (n=3) and nonprogressive (n=14) carotid plaques (fibrotic tissue 56.7±8.4% vs 57.3±7.4%, p=0.898, fibrolipids 15.0±7.5% vs 18.7±9.3%, p=0.531, dense calcium 5.7±3.5% vs 5.8±4.0%, p=0.959, necrotic core 22.3±12.9% vs 18.4±9.7%, p=0,548). Similarly, no association with IVUS-VH characteristics of culprit lesion at baseline was found between coronary restenosis (n=8) and no-restenosis (n=23) group (fibrotic tissue 54.4±11.5% vs 51.7±12.4%, p=0.590, fibrolipids 10.0±3.7% vs 13.1±11.4%, p=0.457, dense calcium 12.8±7.7% vs 12.9±9.7%, p=0.975, necrotic core 24.1±9.0% vs 22.7±8.5%, p=0,680).

CONCLUSION: Tissue characteristics by IVUS-VH were not associated with carotid plaque progression and frequency of restenosis in coronary arteries in these series. Restenosis rate in carotid arteries is low in comparison with coronary arteries regardless of the stenosis morphological differences.

THE PREVALENCE OF METABOLIC SYNDROME IN MIDDLE-AGED LITHUANIAN SUBJECTS

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BACKGROUND: Cardiovascular disease (CVD) is still a major cause of premature death across Europe and in Lithuania. The metabolic syndrome (MS) is a cluster of risk factors including obesity, glucose intolerance, dyslipidemia, and hypertension that increase the risk for CVD and type 2 diabetes.

METHODS: In 2006 the Lithuanian High Cardiovascular Risk programme was started. LitHiR programme recruited men – at the age of 40-54 years and women – 50-64 years without overt CVD. This report describes the relationship between MS and other risk factors in the group of 23 204 subjects.

RESULTS: The average age of the participating patients was 52.60 (± 6.11) years; 59.9% were females. The prevalence of MS in the whole sample was 31.5%. MS was more prevalent in the enrolled females (35.9%) than in males (24.9%)(p<0.001). The most important MS parameters in the study subjects were hypertension (58.4%), central obesity (46.6%), elevated triglycerides (38.4%), hyperglycemia (29.1%) and low HDL cholesterol (15.0%). Gender differences were seen as statistically significant regarding waist circumference, blood pressure, triglyceride and glucose levels while they were similar regarding HDL cholesterol. All main risk factors, except for smoking were more expressed (p<0.001) in the subjects with MS. The number of risk factors in both groups also differed significantly (p<0.01) – median 5 in the MS group and 3 – in the group without MS.

CONCLUSIONS: Our study documents not only the high prevalence of MS, but also MS association with other cardiovascular risk factors. MS seems to be a major health problem and is likely to be important contributor to the epidemic of CVD in Lithuania.

PREVALENCE OF DYSLIPIDEMIA IN MIDDLE AGE LITHUANIAN ADULTS AND ITS ASSOCIATION WITH OTHER RISK FACTORS. ANALYSIS OF PATIENTS FROM LITHIR COHORT STUDY

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BACKGROUND: Lipid disorders are common worldwide and contribute to significant proportion of the burden atherosclerotic cardiovascular diseases. Information concerning lipid disturbance in Lithuania middle age adults is scare.
The objective of this study were to describe the lipid profile in a large cohort of Lithuanian middle age adults and to explore the relation between dyslipidemia and other cardiovascular risk factors as a positive family history, obesity, smoking, dietary habits and physical activity.

METHODS: This is an observational, multi-center, cross sectional study. A total of 23204 patients were enrolled into the study. Anthropometric measurements were taken and blood investigations including glucose, lipid panel were done. Information on smoking, hypertension status, family history, dietary habits and physical activity were collected.

RESULTS: The mean age was 52.6 ± 6.11 years. The prevalence of lipid abnormalities was 91.3 % for high total cholesterol (TC), 32.2 % for high triglycerides (TG), 16.7% for low high-density lipoprotein cholesterol (HDL) and 88.6% for high low density lipoprotein cholesterol (LDL). The severe dyslipidemia observed in 12.1 % of all lipid abnormalities. The subjects with dyslipidemia has significant higher rates of MetS (34% vs. 10%, p<0.001), diabetes mellitus (10.5% vs. 7.3, p<0.001), arterial hypertension (58.8% vs. 47.5%, p<0.001), abdominal obesity (48% vs. 34.2%, p<0.001), positive family history (23.9% vs. 21%, p<0.001) and insufficient physical activity (50.5% vs. 44.7%, p<0.001). The clustering number of risk factors (3.09 vs. 2.42, p<0.001) were greater in the patients with dyslipidemia.

CONCLUSIONS: The very high prevalence of lipid disorders and its association with cardiovascular risk factors in an apparently healthy Lithuanian population which is cause for concern as these individuals are at high risk of developing cardiovascular disease in later life. This indicates urgent need for targeted intervention to reduce the cardiovascular risk.

RESTING HEART RATE CONTROL OVER THREE-YEAR PERIOD IN OUTPATIENTS WITH STABLE CORONARY ARTERY DISEASE IN LATVIA

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BACKGROUND: Increased resting heart rate (HR) is independent cardiovascular (CV) risk factor (RF). HR ≥70 beats per minute (bpm) increases CV risk in coronary artery disease (CAD) patients. We aimed to analyze changes of HR control over three-year period in patients with established CAD in Latvia.

METHODS: We surveyed 120 treated out-patients with stable CAD. From 2009 to 2013 during baseline and three annual visits following data were analyzed: resting HR (measured by pulse palpation and ECG), physical examination and clinical data as well as treatment.

RESULTS: Median (Me) of HR by pulse palpation at baseline, year (Y)1, Y2, Y3 was 65.5 (interquartile range (IQR)=9.5), 66.0 (IQR=8.0), 64.0 (IQR=10.0), 65.5 (IQR=12.3) bpm, respectively. Me HR by ECG at baseline, Y1, Y2, Y3 was 65.0 (IQR=14.0), 65.0 (IQR=15.3), 63.0 (IQR=12.0), 67.0 (IQR=14.5) bpm, respectively. Only in Y2 HR by pulse palpation was significantly different (lower) vs baseline (p=0.027). Increased resting HR (≥70 bpm) when measured by palpation was present in 35.8% of cases at baseline, in 35.6%, 29.8% and 35.1% of cases in Y1-Y3, respectively; when measured by ECG: in 33.6% (baseline), 36.8% (Y1), 26.7% (Y2), 33.7% (Y3) of cases. No differences in proportions of patients with increased HR in Y1-Y3 vs baseline were found. Positive correlation between HR and diastolic blood pressure (BP) was observed (by palpation at baseline (r=0.404; p=0.007), in Y1 (r=0.464; p=0.002), in Y3 (r=0.340; p=0.029) and by ECG in Y1 (r=0.260; p=0.004). No significant correlations between HR and other analyzed RFs (systolic BP, diabetes, smoking, body mass index and waist circumflex) were found.

CONCLUSIONS: Over three-year period HR control did not improve in the analyzed sample of treated out-patients with stable CAD. Substantial (around one third) and stable proportion of patients with increased HR ≥70 bpm clearly indicates need for better control of this RF.

HIGH SENSITIVITY C-REACTIVE PROTEIN LEVELS ARE ASSOCIATED WITH TOTAL CORONARY ARTERY OCCLUSIONS

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BACKGROUND: We aimed to test whether high-sensitivity C-reactive protein (hs-CRP) is associated with specific manifestations of coronary artery disease (CAD): chronic total occlusions and left main (LM) disease.

METHODS: We prospectively investigated digital coronary angiography results of 974 patients referred to elective invasive diagnostic procedure. Among these patients we selected 712 cases with clear evidence of CAD defined as coronary artery lesion of at least 50%. Occlusion was defined as total lack of flow distally to the lesion in a coronary artery. Left main disease was defined as stenosis of at least 50%. In all patients hs-CRP was measured on the day of angiography. Results were adjusted for confounders known to be associated with CRP levels or CAD: age, gender, diabetes, smoking, hypertension, family history of premature myocardial infarction and pre-hospital use of statins. Due to highly skewed distribution hs-CRP levels were log-transformed when entered in logistic regression models.

RESULTS: The prevalence of total occlusions and LM disease were 39.3% (n=280) and 6% (n=43), respectively. CRP levels ranged from 0.2 to 98.1 mg/L. In 11.2% of cases with hs-CRP levels were above 10 mg/L. The median hs-CRP levels were 2.4 and 1.8 mg/L in patients with and without coronary occlusions, respectively (Mann-Whitney p=0.030). In logistic backward regression analysis only log-CRP was associated with coronary occlusions (OR 1.40, 95%CI 1.04-1.88, p=0.028). The difference of median hs-CRP levels in patients with or without LM disease was not statistically significant: (2.8 and 2.0 mg/L, respectively, p=0.335).

CONCLUSIONS: Increased level of hs-CRP is associated with total coronary artery occlusions and it appears to be stronger marker of coronary occlusions than traditional risk factors. We did not find correlation between hs-CRP levels and LM disease, which may be due to the small number of LM cases in the sample.

ROLE OF GENETIC FACTORS ON THE EFFECT OF ADDITIONAL LOADING DOSES AND TWO MAINTENANCE DOSES USED TO OVERCOME CLOPIDOGREL HYPORESPONSIVENESS

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BACKGROUND: Hyporesponsiveness to clopidogrel is associated with thrombotic events following coronary stent implantation. We aimed to investigate whether genetic polymorphisms associated with high platelet reactivity modify effect of additional loading doses (LDs) used to overcome hyporesponsiveness to clopidogrel.

METHODS: In a prospective single-center study we included patients after DES implantation receiving clopidogrel LD of 300mg or 600mg, respectively. According to the first VASP platelet reactivity index (PRI) test patients were classified as responders or hyporesponders (PRI<60% or >=60%, respectively). Hyporesponders received up to 3 additional LDs (600mg) and higher maintenance dose (MD, 150mg) for one month and 75mg thereafter. The following polymorphisms were tested: CYP2C19*2, *3, *17, CYP2C9*2, *3.

RESULTS: A total of 94 patients were enrolled. Among patients 68(72.3%) were hyporesponders after the first LD. After second and third LD 24(25.5%) and 11(11.7%) patients had PRI>=60%, respectively. After fourth LD 2(2.1%) patients were non-responders and were switched to ticagrelor. At the follow-up, only 8(12.3%) of hyporesponders had PRI>=60% while on MD of 150mg compared to 33(48.5%) while on 75mg MD (p<0.001).
Patients carrying at least one CYP2C19*2 allele had a higher PRI after the first LD than those carrying the wild-type genotype (77.2±13.3 vs 65.3±19.5, p=0.012). After the first additional LD of clopidogrel PRI decreased similarly in both groups of CYP2C19*2 genotype (-35.3±1.9%p vs -27.3±17.4%p, p=0.348).
In carriers of CYP2C19*2 allele PRI was higher with both MD of 150mg and 75mg: 53.3±12.1 vs 40.4±13.5(p=0.001) and 63.1±11.3 vs 50.3±17.1(p=0.002), respectively.
Patients carrying at least one CYP2C19*17 allele had a lower PRI after the first LD compared with those carrying wild-type genotype (65.1±20.7 vs 72.5±14.9, p=0.071).

CONCLUSIONS: Carriers of the CYP2C19*2 less frequently reach adequate platelet inhibition and therefore require more additional LDs. The MD of 150mg was more effective than 75mg both in hyporesponders and in carriers of CYP2C19*2.

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