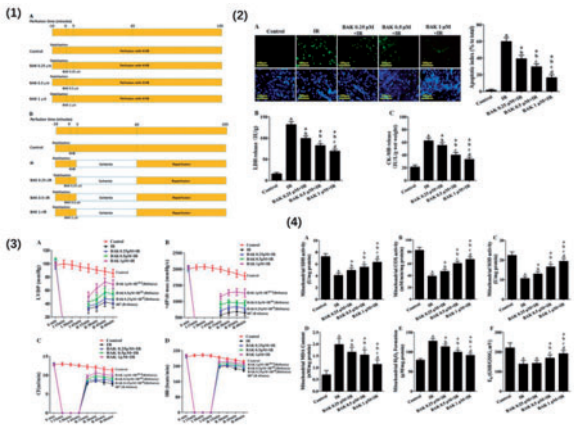
Conclusion: In summary, our results demonstrate that BAK treatment attenuates IRI by attenuating IR-induced mitochondrial oxidative damage via the activation of SIRT3/PGC-1a signaling.



Abstract P504 Figure.

P505

An untargeted metabolomics approach reveals unusual pathways involved in short term low-dose acetylsalicylic acid treatment

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Introduction: Acetylsalicylic acid (ASA) is one of the most commonly antiplatelet drug used for the prevention of cardiovascular events. Although its clinical efficacy has been well documented, a substantial variability in drug response exists and the mechanisms at the basis of this variability are still poorly understood. Purpose. An untargeted metabolomics approach could reveal novel information about biochemical pathways modified by ASA treatment and elucidate the determinants of drug responsiveness and pharmacological properties.
Methods: In this study we defined the urinary metabolomic profile of healthy subjects (n=7) before and 7 days after 100 mg/die ASA treatment, through a liquid chromatography – time of flight mass spectrometry platform, in positive and negative ionization mode.
Results: Through this untargeted approach, we detected 2007 metabolites: among them 64 significantly differed ($p<0.05$) after ASA assumption. Pathway analysis, performed on identified metabolites, reveals low levels of those involved in histidine, alanine, aspartate and glutamate and purine metabolisms, after ASA treatment. In addition, we observed the decrease of several short- and medium-chain acylcarnitines, which suggests an increase in fatty acid β-oxidation process.
Conclusion: The data here reported, revealing relevant pathways altered by ASA, may suggest non-canonical use of ASA in clinical situations characterized by energy depletion.

P506

Adverse left ventricular remodeling and the serum levels of matrix metalloproteinases, biomarkers of myocardium dysfunction and inflammation in patients with acute primary anterior STEMI

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The aim of this study was to assess the changes in serum levels of matrix metalloproteases (MMP)-2, 3, 9, ST2, NTproBNP, IL1β, and hCRP and their impact on the adverse left ventricular remodeling (LVR) in patients with acute primary anterior myocardial infarction with ST segment elevation (STEMI).
The study included 21 patients aged 60.5±7.4 years. All of them received urgent reperfusion therapy; one third of patients received the treatment during the first 3 h. Echocardiography with 2D speckle tracking imaging was performed at day 3 (T2), 7 (T3), and 14 (T4) after STEMI and after 6 months (T5) after AMI (Vivid E9, GE Healthcare).
The concentrations of MMP-2, MMP-3, MMP-9, ST2, IL1β, hCRP, and NTproBNP were determined at the same time point and at a day of admission (T1) by the method of quantitative enzyme linked immunosorbent assay. After that, patients were divided into 2 groups: group 1 comprised patients with the level of ST2 > 35 ng/mL; group 2 comprised patients with ST2 < 35 ng/mL at T1. The study showed that changes in the markers were multidirectional. The level of MMP-2 did not significantly change. The level of MMP-3 increased to T3 and continued to increase to T5; the changes in levels of MMP-9 were reverse over the same period. The level of IL1β decreased to T4 though this parameter as well as the levels of ST2, NTproBNP, and hCRP exceeded the normal range during the entire observation period. The levels of ST2, NTproBNP, and hCRP were changing to T3 and significantly decreased to T5.
Marker ST2 demonstrated the best predictive value for the development of adverse LVR. ST2 level of more than 35 ng/mL at a time of admission was associated with the presence of systolic dysfunction, increased wall motion score index, increased end-systolic volume, increased 2D global longitudinal strain, and reduced ejection fraction in the early postinfarction period

P507

Noncompact left ventricular myocards and pregnancy

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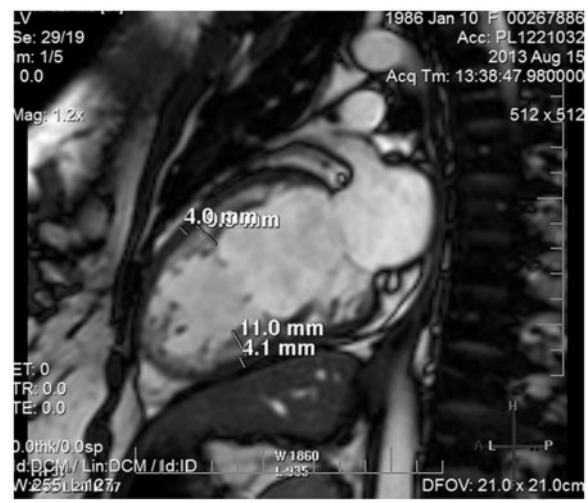
Taking into account the low prevalence of the disease, the lack of research involving a large number of pregnant women with noncompact left ventricular myocardium, single observations are of interest, one of which is presented by us in this abstract.
Patient, 27 years old. Obstetric anamnesis is not burdensome. The first pregnancy. Thrombotic history - in the family of the mother thromboembolism of the pulmonary artery. Based on the results of echocardiography
revealed increased trabecularity in the middle and apiacal regions of the left ventricle. It is impossible to exclude the noncompact myocardium.
According to magnetic resonance imaging, signs of noncompact myocardium of the left ventricle (picture 1). The noncompact myocardium consists of two layers, located in the region of the apex, posterior wall, according to the MRI of the heart it is 2.68 (11.0 / 4.1mm) times thicker than the compact part of the myocardium. Diagnosis of the consultation of cardiologists
bearing pregnancy is contraindicated. The patient refused to abort her pregnancy.
Due to the high risk of thromboembolic complications during pregnancy, sulodexide heparinoide was administered 250 LE 2 per day. Since 28 weeks of pregnancy, the patient has seen an increase in the level of d-dimers.
Given the extragenital pathology, the increase of d-dimer, the violation of the placenta blood flow, a course of anticoagulant therapy with enoxaparin 40 mg once a day, subcutaneously, with the cancellation 12 hours before the proposed delivery and aspirin 75 mg every other day from 16 to 28 weeks. At term of pregnancy of 37 weeks
The patient is delivered by cesarean section. A live full-term boy was born with an Apgar score of 8 points.
Therapy of the early postpartum period: metoprolol 25 mg / day, enoxaparin 40 mg / day. On the seventh day the patient was discharged in satisfactory condition, the child was discharged home.
Conclusion: The question of the possibility of bearing pregnancy in the presence of noncompact myocardium of the left ventricle is solved individually. Prolongation of pregnancy is possible with the joint supervision of obstetricians, cardiologists, hematologists



Abstract P507 Figure.

P508

Massively parallel sequencing of patients affected with arrhythmogenic cardiomyopathy by a targeted gene panel identified a novel nonsense mutation in TP63 gene

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Background: Arrhythmogenic Cardiomyopathy (ACM) is a clinically and genetically heterogeneous heart muscle disease and a leading cause of sudden cardiac death in the young and athletes. The majority of mutations identified in ACM patients involved genes encoding proteins of the intercalated disc but still 40% of patients remained genetically unidentified.
Purpose: The aims of this study was: 1) the identification of the genetic cause in 40 ACM patients; 2) the identification of putative novel genes associated with ACM.
Methods: DNA samples from 40 ACM probands negative for mutations in the 3 major ACM genes (DSP, PKP2 and DSG2) were screened by using a targeted gene panel consisting of 67 genes, 14 known ACM genes and 53 candidate genes. For each sample the exonic and intronic flanking regions of the investigated genes were enriched and sequenced using 150bp paired-end reads on a massively parallel sequencing platform. Only the variants covered at least 15X have been considered as reliable variants. Taking into account that the allele frequency of ACM in the general population span from 0.01% and 0.025%, a genetic variant has been considered a 'mutation' if its minor allele frequency (MAF) is ≤0.01%. In silico predictions of pathogenicity of rare

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