Results 10 animals were included in the control group and 9 in the Colchicine group (blinded adjudication). All the animals were successfully operated and survived at 24 hours. The AAR/total area ratios were respectively 54.4±4.3% in the Colchicine group versus 51.9±4.4% in the control group, p=n.s. No difference in infarct size could be found in Colchicine treated mice in comparison to control mice with IA/AAR ratios respectively at 49±3.4% versus 53.7±5.3%, p=n.s.

Conclusion In conclusion, reduction of infarct size could not be obtained by treatment with Colchicine at the present concentration after induced myocardial infarction in mice. Further analysis should be performed to evaluate inflammatory cells accumulation in myocardium and inflammation pathways as well as LV remodeling.

Abstract 0140 – Figure: Impact of Colchicine on infarct size in mouse

The author hereby declares no conflict of interest

0527

Changing in level of microRNA-1, –155, –210, –208a and –499 in blood plasma, platelets and monocytes of patients with ST-segment-elevation myocardial infarction

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Background The study of the changing levels of microRNAs (miRNAs) in different biological body environments as diagnostic and prognostic marker for ST-elevation myocardial infarction (STEMI) calls attention of scientists. We conducted a research to determine the level of several microRNAs in blood plasma, isolated monocytes and platelets of STEMI pts in the dynamics of disease development (the 1st and the 7th days). Into the study we included the evaluation of some cardiac microRNAs (1, 208a, 208b, 499), inflammation-related and hypoxic-response microRNA-155 and –210.

Methods 20 STEMI pts (mean age 54.8±2.2 years) were identified in National Scientific Center Institute of Cardiology M.D. Strazhesko. The control group included 10 subjects of comparable sex and age without any evidence of coronary artery disease. Expression miRNAs was quantified from plasma, isolated monocytes and platelets using quantitative Real-Time polymerase-chain reaction system.

Results In plasma the microRNA-1 level was significantly higher only on day 7; the microRNA-155 level was lower on the first day and 15 times higher on day 7. In platelets the microRNA-1 level was 4.2 times higher on the first day of STEMI in comparison with the control group and on day 7 of STEMI returned to the level of the control group. The changes in monocytes were more revealing: the microRNA-1 level was higher by 28 times on the first day of STEMI, the microRNA-208a level was higher by 2 times on day 1 and by 19 times on day 7 of STEMI, and the microRNA-155 level was higher by 17 times on day 1 and by 260 times on day 7 of STEMI in comparison to the control group.

Conclusions The miRNA distribution is unique amongst blood plasma, isolated monocytes and platelets in pts with STEMI. Our results show higher informational content of evaluation of certain miRNAs in isolated cells comparing with blood plasma, and expresses the necessity of determining miRNA levels in the dynamics of STEMI.

The author hereby declares no conflict of interest

0183

A common polymorphism in CD40 Kozak sequence (+1C/T) is associated with myocardial infarction in the Tunisian male population

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Background Coronary artery disease is influenced by both environmental and genetic factors. Current evidence shows that the CD40-CD40 ligand (CD40-CD40L) system plays a crucial role in the development, progression and outcome of acute coronary syndrome. A single nucleotide polymorphism (SNP) located at position +1 in the Kozak sequence of the CD40 gene (rs1883832; C>T) has been associated with the development of acute coronary syndrome. The purpose of the study was to explore the association between the C/T (+1) single nucleotide polymorphism in the CD40 gene and myocardial infarction (MI) in the Tunisian male population.

Methods A total of 273 unrelated Tunisian patients with MI and 219 healthy controls were genotyped using the polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) method. Clinical parameters such as fasting serum total cholesterol, high-density lipoprotein cholesterol (HDL-C), triglycerides, and plasma glucose were detected by autoanalyzer assay. Weight, height and blood pressure (BP) were measured and body mass index (BMI) was calculated.

Results Patients with MI had significantly higher frequency of the TT genotypes compared to controls (12.5% vs. 5.9%; OR=1.43, 95% CI: 1.08 – 1.89, p=0.011). The MI patient group showed a significant higher frequency of the T allele compared to the controls (0.33 vs. 0.26; OR=1.45, 95% CI: 1.09 – 1.94, p=0.008). The association between the C/T (+1) single nucleotide polymorphism in the CD40 gene and MI remained significant after adjustment for other well-established cardiovascular risk factors.

Conclusion Our study suggests that C/T (+1) single nucleotide polymorphism in the CD40 gene might contribute to the susceptibility to MI in the Tunisian male population. Further replication with larger numbers, and populations of different ethnicities, are needed to confirm our finding.

The author hereby declares no conflict of interest

0261

Inflammatory response phenomena mediate by cytokines in the myocardial remodeling post myocardial infarct

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Introduction Myocardial infarction (MI) is a major cause of morbidity and mortality and is a main etiology of heart failure. The process of MI is modulated by the inflammatory response. Many studies have shown a significant inflammatory response phenomenon in pathogenesis of MI.

Objectives The aim of this study is to discuss the role of pro-inflammatory (IL8,TNFα) and anti-inflammatory (IL10) cytokines that are among others described in involved in the process of remodeling post-myocardial infarction.

Methods Prospective, monocentric case-control study that included 82 patients diagnosed with a first episode of myocardial infarction and low LVEF and 30 healthy subjects. The quantification of IL10 was based on the technique of sandwich ELISA. The CLIA technique was used for quantification of IL8 and TNFα. All patients were evaluated by an echocardiography after revascularisation and one month after to study cardiac remodeling.