Echocardiography was done in patients presentation, VEGF-A level was determined on the 7th day of STEMI by enzyme-linked immunoassay. Anxiety level assessed by Taylor questionnaire during the period of 10-14 days before STEMI. After 6-month observation 47 patients were assessed. Statistics were performed with the help of Statistica 8.0.

Results. In patients with STEMI in comparison with the control group significant rise of serum VEGF-A level was observed which accompanied with positive correlation with creatine kinase level. The level of VEGF-A below median 160 pg/ml in comparison with its level above the median 160 pg/ml associates with higher frequency of myocardial infarction in men (P=0.023), anxiety before infarction (P=0.019), end diastolic diameter (EDD), end diastolic volume (EDV) increase in acute phase, EDD, ESD, EDV, left ventricular myocardial mass, E/A, lower exercise tolerance after 6-month observation. This processes indicates about unfavorable role of low VEGF-A levels and protective - high VEGF-A levels for cardiac remodeling after myocardial infarction. ROC-analysis was performed to find out the VEGF-A level which is prognostic for pathologic remodeling development – ΔEDV>0 assessed as unfavorable result after 6-month observation. With sensitivity 57.9% and specificity 85.7%, AUC=0.711, 95% CI 0.513-0.908, P=0.036, the level of VEGF-A≥201.86 pg/ml is prognostic for investigated parameter. So, VEGF-A level – important indicator to estimate the extent of myocardial injury and pathologic remodeling development.

Prospects for future research: in future we plane to research the influence of different single gene polymorphisms (Lys198Asn – endothelin-1, T344C – aldosterone synthase, A1166C – receptor R1 to angiotensin-II) on the VEGF-A level.

Key words: ST-elevation myocardial infarction, VEGF-А, left ventricular remodeling

References:

Key words: ST-elevation myocardial infarction, VEGF-A, left ventricular remodeling

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EPIDEMIOLOGICAL PROFILE OF TORCH-INFECTION IN PREGNANT WOMEN IN KHARKOV REGION

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Diagnostics of TORCH infections (toxoplasmosis, rubella, cytomegalovirus and herpes simplex virus type 1 and 2) remains actual problem. Among possible causes of these difficulties are peculiarities of these diseases: lifelong persistence of the pathogen in the organism in absence of disease clinical manifestations; wide prevalence among all population groups; absence of representative clinical symptoms; high risk of fetal or newborn pathology in case of primary infection at pregnancy; exacerbation of latent infection in immunocompromised. This group of infections
Background. It is known that children with asthma are susceptible to the upper respiratory tract infections (URTIs). Likewise, respiratory infections play an essential role in the onset and progression of asthma [1]. In recent years a growing body of evidence have been obtained showing that immunological shift towards type 2 inflammatory response with overproduction of interleukins (IL) 4, 5, and 13 plays an essential role in the pathogenesis of asthma [2]. The study objectives were to investigate the serum cytokine response during viral URTIs in asthmatic children.

Materials and Methods. We enrolled a total of 95 children aged 3-7 years suffering viral URTIs on the 1-3rd day of the disease. The main group were 62 asthmatic patients with mild to moderate asthma, and 33 non-allergic patients were the controls. Serum levels of interferon α and γ (IFN-α, IFN-γ) were measured on the 1-2nd, 4-5th and 7-9th day of URTI, and serum IL-4, IL-5 and IL-13 concentrations were studied in the early and late disease.

Results. Compared to the control group, asthmatic children showed significantly lower serum levels of both interferons in the early disease (IFN-α 60.7±4.5 pg/ml and IFN-γ 4.3±0.2 pg/ml vs. 75.9±3.5 and 9.5±1.6 pg/ml in controls, resp., p< .01) and showed no significant increase in interferon levels in the middle (IFN-α 37.5±2.8 pg/ml vs. 131.0±26.8 pg/ml in controls, IFN-γ 6.6±0.4 pg/ml vs. 9.5±1.6 pg/ml in controls, p< .001) and late disease (IFN-α 28.7±1.6 pg/ml, IFN-γ 5.9±0.3 pg/ml vs. 59.9±4.1 pg/ml and 5.9±0.6 pg/ml in controls, p< .05). Levels of pro-allergic cytokines IL-4 and IL-13 in asthmatic children showed substantial increase during URTI (IL-4: 0.8±0.1 pg/ml vs. 0.5±0.1 pg/ml in controls initially, 1.5±0.3 pg/ml vs. 0.5±0.1 pg/ml in controls, p< .001) and late disease (IL-13: 12.6±2.8 pg/ml vs. 2.5±0.4 pg/ml in controls, and 2.3±2.3 pg/ml vs. 2.8±0.2 pg/ml in controls, in the early and late disease, resp.; p< .001).

Conclusion. Results of the study reviewed that not only intensification of allergic inflammation but also depression of interferon response during viral URTIs is typical for asthmatic children.

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SYSTEMIC INFLAMMATION AND THE STATE OF CENTRAL HEMODYNAMICS IN PATIENTS WITH CORONARY HEART DISEASE

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Cardiovascular disease, and coronary heart disease (CHD) first and foremost, is one of the leading causes of mortality in developed countries of the world [1]. The prognosis for patients with coronary heart disease depends, predominantly, on the progression of coronary atherosclerosis (CA). The pathogenetic basis of CA is chronic systemic inflammation (CSI) [2]. CSI in CA is supported by induction of cytokine-associated pathways of intracellular signaling. The central component of CSI is the nuclear factor kappa B (NF-kB), the main activators of which are proinflammatory cytokines (CK), especially interleukin 1 (IL-1) and tumor necrosis factor α (TNFα) [3, 4]. The effector unit of the CSI is CK

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