REFERENCES

1. Huerta C., Rivero E., Rodríguez L. G. Incidence and Risk Factors for Psoriasis in the General Population// Arch Dermatol. -2007.-№ 143(12).-P.1559-1565.
2. Azfar RS, Gelfand JM. Psoriasis and metabolic disease: epidemiology and pathophysiology//Curr Opin Rheumatol. -2008.- №20 (4).-P.416-22.
3. Ahdout J., Kim J., Chiu M.. Modifiable metabolic syndrome associated lifestyle factors in psoriasis patients // Acad Dermatol.- 2009.- March.-P.3330
4. Davidovici B. B., Sattar N., Jörg P. C., Puig L., Emery P., Barker J. N.et al. Psoriasis and Systemic Inflammatory Diseases: Potential Mechanistic Links between Skin Disease and Co-Morbid Conditions// Journal of Investigative Dermatology.-2010.-№ 130,.-P.1785–1796.
5. Herron M.D., Hinckley M., Hoffman M.S., et al. Impact of obesity and smoking on psoriasis presentation and management // Arch. Dermatol. – 2005. – Vol. 141, № 12. – P.1527-1534.
6. Mallbris L., Granath F., Hamsten A., et al. Psoriasis is associated with lipid abnormalities at the onset of skin disease // J.Am. Acad. Dermatol. – 2006. – Vol. 54, № 4. – P.614-621
7. Hardingham N, Dachtler J, Fox K. The role of nitric oxide in pre-synaptic plasticity and homeostasis// Frontiers of Cellular Neuroscience. -2013.-V.7.-P. 1-19.
8. Wenner P. Mechanisms of GABAergic homeostatic plasticity //Neural Plast.- 2011.-V.2011.-P.1-6.
9. Cadet J.L., Brannock C. Invited Review Free radicals and the pathobiology of brain dopamine systems// Neurochemistry International .-1997.-Vol. 32. Issue 2.-P. 117–131.

THE SERUM CYTOKINES LEVEL DYNAMICS IN PATENTS WITH HEART FAILURE

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Introduction. The heart failure (HF) syndrome is characterized by impaired systolic and/or diastolic function and various clinical signs such as fatigue, dyspnea, fluid retention, and cachexia. An inflammatory activation in CHF patients has long been recognized. Indeed, immune mechanisms modulate interstitial fibrosis, cardiomyocyte apoptosis, and hypertrophy, all of which are central processes leading to maladaptive remodeling in response to a variety of stimuli [7].

Several reports have demonstrated enhanced expression and release of inflammatory cytokines, as well as several chemokines in HF patients [8]. Ivabradine is a new therapeutic agent designed to reduce heart rate at rest and during exercise by selective inhibition of a novel receptor (If channel) located on the pacemaker-cell membrane within the sinoatrial node. As such, ivabradine joins a list of rate-limiting medications already available to prescribers for the control of heart rate in coronary artery disease (CAD) and HF with systolic dysfunction [5]. The ω-3 polyunsaturated fatty acids (PUFA), such as docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), are known as anti-inflammatory factors, and are using for HF treatment [5]. The data for ivabradine influence for cytokine' cascade are poor; but for PUFA – controversial.

The aim of study was to evaluate of possible influence of ivabradine and ω-3 polyunsaturated fatty acids for pro- and anti-inflammatory cytokines level in patients with ischemic heart failure.

Material and Methods. 357 patients with ischemic HF and sinus rhythm were observed. In accordance to treatment all patients were divided into four groups: I group – basic treatment (89 patients); II group - basic treatment and Ivabradine (Coraxan, Les Laboratoires Servier Industrie, France) – 5 or 7,5 mg twice a day (depends of heart rate); III group - basic treatment and PUFA (Omacor, Abbott Laboratories GmbH, USA) – 1000 mg per day; IV group – basic treatment with Ivabradibe and PUFA in similar doses. All patients were examined before and after 6 months of treatment. Control group – 30 practically healthy persons. The study was performed in accordance with the Helsinki Declaration and Good Clinical Practice Guideline [6]. The study was approved by the local ethics committee and written informed consent was obtained from all patients.

The interleukin 1β (IL-1β), interleukin 6 (IL-6), and interleukin 10 (IL-10) levels in serum were determined using commercial ELISA kits (ProCon, Russia; Amersham Pharmacia Biotech, UK) according to the manufacturer's instructions.

Statistical analyses were performed using the Statistica 12.0 (StatSoft, Tulsa, OK, USA). Statistical significance was assumed at $p<0.05$.

Results and Discussion. HF is characterized by pro-inflammatory cytokine levels increase. The average levels of IL-1β was (49,29±3,78) pg/ml versus (25,31±3,71) pg/ml in control group ($p<0,001$); IL-6 – (15,47±0,52) pg/ml versus (7,19±0,67) pg/ml respectively ($p<0,001$). Their concentrations were increased due raise of HF class. The serum level of anti-inflammatory IL-10 was insignificant lower: (3,12±0,37) pg/ml versus (3,46±0,57) pg/ml in control group ($p>0,05$).

During treatment we observed decrease of serum IL-1β level in all groups of patients. In particular, in first group this parameter was decreased for 25,5% ($p<0,01$). More strong changes were observed in group with additional use of ivabradine, where the IL-1β level decreased for 32,6% ($p<0,001$). Additional prescription of PUFA caused of IL-1 reduction for 35,9% ($p<0,001$). In forth group this parameter decreased for 44,4% ($p<0,001$).

The medications of basic treatment decreased of serum IL-6 level for 30% ($p<0,05$). The its dynamics in second group was higher – 37,6% ($p<0,01$). The more strong changes were caused by additional use of PUFA or their combination with ivabradin. In particular, in third group - for 45,9% ($p<0,01$); in forth – for 48,6% ($p<0,01$).

All therapeutic schemes caused to raising of IL-10 levels in HF patients blood. But more strong changes were observed in groups with PUFA. In third group its growth was 26% ($p<0,01$); in patients of Iv group –26,1% ($p<0,01$). In first group this value increased for 21,4% ($p<0,05$); in second group – for 20,6% ($p<0,05$). For our opinion, this is result of basic treatment influences, but not ivabradine.

IL-10 is a regulatory cytokine with anti-inflammatory properties, potently inhibiting the capacity of innate immune cells to produce inflammatory mediators.

Conclusions. 1. The PUFA medication has an immunomodulatory effects: they decrease of serum pro-inflammatory cytokines (IL-1β, IL-6) levels and increase of level of the anti-inflammatory IL-10.

2. Ivabradine is caused of reduction IL-1β and IL-6 in serum but not influence for IL-10 concentration.

References

1. Прасолов, А.В. Изменение содержания ИЛ-1β, ИЛ-6, ФНО-α в сыворотке крови больных ИБС: стабильной стенокардией напряжения II-III ФК на фоне лечения / А.В. Прасолов // Аллергология и иммунология. – 2009. – Т.10, №1. – С.99-109.
2. Correlation of protein and gene expression profiles of inflammatory proteins after endotoxin challenge in human subjects. / U. Prabhakar, T.M. Conway, P. Murdock, [et al.] // DNA Cell. Biol. -2005. –Vol.24. –P.410–431.
3. Dinarello C. Interleukin 1 in the pathogenesis and treatment of inflammatory diseases / C. Dinarello //Blood. -2011. –Vol.117. –P.3710-3732.
4. Effects of Angiotensin-converting enzyme inhibition and statin treatment on inflammatory markers and endothelial functions in patients with longterm rheumatoid arthritis. /C. Tikiz, O. Ufuk, T. Pirildar [et al.] //The J. Rheum. – 2011. –Vol.32. –P.2095-2101.
5. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012 /J. McMurrey, S. Adamopoulus, S. Anker [et al.] // European Heart Journal. – 2012. –Vol. 33. –P.1787–1847.
6. European Medicines Agency. ICH Harmonised Tripartite Guideline E6: Note for Guidance on Good Clinical Practice (PMP/ICH/135/95). -London: European Medicines Agency, 2002. -46 p.
7. Hofmann U. How we can cure a heart “in flame”? A translation view on inflammation in heart failure / U. Hofmann, S. Frantz //Basic. Res. Cardiol. -2013. –Vol.108. -P. 2-19.
8. Inflammatory cytokine in heart failure: mediators and markers. /L. Gullestad, T. Ueland, L. Vinge [et al.] // Cardiology. -2012. -Vol.122. -P.23–35.

PREVALENCE OF DIFFUSE GOITER AMONG ADULTS IN LVIV REGION IN YEARS 2000-2010

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Iodine deficiency is a common cause of endemic goitre. Recently, WHO calculated that about 740 million people, equivalent to 13% of the world’s population, have goiter due to an excessively low intake of iodine. Iodine deficiency is also a major health problem in regions of western Ukraine. Now, there is an ongoing global iodination program in a collaboration between the International Council for Control of Iodine Deficiency Disorders (ICCIDD), UNICEF and WHO, with the goal of eradicating iodine deficiency throughout the world. The iodination program also includes monitoring goitres and measuring the concentration of iodine in the urine in the population as quality assurance of the program. Incidence of diffuse goitre (DG) in Ukraine is currently higher than in past decades. Therefore, the aim