RCE: Glu/Glu = 0.71; Glu/Asp = 0.27; Asp/Asp = 0.02 (X2 = 0.01; P = 0.9 (between expected and observed.). The frequency of allele distribution of RCE in patients with FC III CHF: Glu = 0.81; Asp = 0.19. The expected frequency distribution of genotypes in patients RCE: Glu/Glu = 0.66; Glu/Asp = 0.30; Asp/Asp = 0.035. The observed frequency distribution of genotypes in patients RCE: Glu/Glu = 0.65; Glu/Asp = 0.33; Asp/Asp = 0.02. (X2 = 0.5; P = 0.5 (between expected and observed)).

Relative deviation from the expected heterozygosity observed (D) polimorfizma- Glu298Asp NOS-3 gene was calculated using the formula: D = (hobs-hexp)/hexp, where hobs and hexp — expected and observed heterozygosity, respectively. **Conclusion.** Thus, the study of the distribution of alleles and genotypes Glu298Asp NOS-3 gene in patients with heart failure showed that progressirovinie CHF depends on the polymorphism of this gene and gene Glu298Asp NOS-3 is effective prognostic markers.

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Evaluation of the effect of omega-3 PUFA on lipid profile in patients with postinfarction cardiosclerosis complicated by congestive heart failure

Abstract: The effect of omega-3 PUFA on lipid profile in patients with postinfarction cardiosclerosis complicated by heart failure.

Key words: chronic heart failure, omega-3 PUFA, hypercholesterolemia.

The most common cause of heart failure (congestive heart failure) is coronary heart disease (CHD), which is 54–68,6%. About 50% of patients with CHF, despite the use of combination therapy, died within 5 years after the onset of clinical symptoms. The problem of the treatment of coronary heart disease (CHD) complicated with chronic heart failure (CHF) remains relevant, as it has great social and economic importance. Today the main cause of morbidity, disability and mortality worldwide are cardiovascular disease. The prevalence of chronic heart failure (CHF) in the general population is 1.5–2%, and in persons over 65 years amounts to 10%. Forecasts are also disappointing: the researchers suggest that the prevalence of heart failure in

the population will increase in the next 20 years by 40–50%. Despite the advances made in diagnosis and treatment, in recent years, mortality remains high [1]. According to the Framingham study die about 80% of men and 65% of women over 6 years after the first signs of heart failure. Currently, along with the use of antianginal and lipid-lowering drugs, the search for new drugs that can korregirovat atherosclerotic lesions of the vascular bed. In particular, a new group of drugs, ω-3 polyunsaturated fatty acids (ω-3 PUFA), Omacor (Abbott), the use of which is due to their ability to work on various parts of the atherosclerotic process: hypertriglyceridemia, growth of atherosclerotic plaque, endothelial dysfunction, and others. [2]. The study GISSI-Prevenzione, which involved 11,324 patients with myocardial infarction, studied the effectiveness of standardized capsules of omega-3 PUFAs. It was found that after 3.5 years of mortality (both general and cardiovascular) decreased significantly in patients treated with omega-3 fatty acids, the addition of vitamin E did not significantly affect the results. In men, acute myocardial infarction and treated with omega-3 PUFAs in fish oil at a dose of 500–800 mg/day showed a reduction in mortality associated with cardiovascular disease, by 62%, and a reduction of total mortality by 56%. (Burr M. L. et al., 1989). It is shown that omega-3 fatty acids may slow the growth of atherosclerotic plaque, as evidenced when playing cholesterol atherosclerosis in experimental animals (pigs, rabbits, rats and monkeys). And based on the results of clinical work C. vonSchacky et al (1999), AT Erkkilä et al (2004) found that a diet with sufficient intake of omega-3 PUFAs from fish helps to slow down the progression of angiographically documented coronary atherosclerosis. In a randomized controlled clinical trials (level of evidence A) shows that people who have had cardiac events, the reception of oily fish (200–400 g/week, which corresponds to 500–800 mg daily produce omega-3 PUFA) or supplements of omega-3 PUFA (containing 850–1800 mg of EPA and DHA) reduced the risk of nonfatal heart attack, sudden death and death from all causes (TullS. P., YatesC. M., MaskreyB. H. et al. 2009). Of particular interest is the lipid metabolism plays an important role in the pathogenesis of coronary heart disease complicated by heart failure.

Purpose of the study Assess the impact of long-term therapy of omega-3 PUFA on lipid profile in patients with postinfarction cardiosclerosis complicated by heart failure.

Materials and methods:

In 125 patients with CHF FC I–III (with the original content of total cholesterol> 4.5 mmol/L and/or the original content of triglycerides> 1.7 mmol/L.) To evaluate the comparative effectiveness of lipid-lowering statin — atorvastatin and omega 3 PUFAs. The patients were divided into two groups: the first group (I) accounted for 67 patients who on the background of basic therapy (beta blockers, ACE inhibitors, antiplatelet agents, statins, spiranolakton) taken against a background of basic therapy (Omacor) at a dose of 1 g/day, the second group (II) — 58 patients receiving basic therapy.

In Group I — patients with I, II and III CHF FC were 16, 26 and 25 patients, respectively, in group II — 15, 24 and 19 patients, respectively. All patients were determined the levels of total bilirubin, ALT, AST, the number of erythrocytes, leukocytes, erythrocyte sedimentation rate in blood, determination of total cholesterol (MBF), high density lipoprotein (HDL) and low density lipoprotein (LDL), triglycerides (TG) at baseline and after 6 months treatment.

Results and discussion

Evaluation of the 6-month course of treatment ω-3 PUFAs in patients with CHF showed a good tolerability of Omacor: side effects have been reported in any of the patients.

Inclusion in the complex therapy of Omacor has led to increased lipid-lowering efficacy of traditional treatments CHF. The level of TC in the blood by the end of 6 months. treatment decreased by 16,7% (p <0,001), while in the control group changed its concentration of 15.5% (0.01).On Omacor therapy was revealed changes in the level of LDL cholesterol. By the end of 6 months. therapy group showed decrease in value of the level of low density lipoprotein (LDL) to 22,3% (p <0,01). In the control group, the rate changed to 15.9% (0.01). The content of HDL in serum by the end of 6 months. in the intervention group increased by 30.9% compared to baseline and was 1,1 (p <0,0005). In the control group the level of HDL during the study changed to 23,1% (p <0,0005). By the end of 6 months. Omacor therapy serum Tg levels decreased by 30,5% (p <0,0005) compared with baseline. Traditional therapy has reduced this figure by 21.3%.

Thus, according to the results, Omacor therapy influenced the individual parameters of lipid profile. This is consistent with other meta-analysis of clinical traylov [1,7], where it was found the effect of concentrate ω-3 PUFAs in the blood lipid profile. The evaluation was conducted ω-3 PUFAs in the form of dietary supplements in various dosages.

In our study, it was confirmed gipotriglitseridemicheskoe effect of Omacor due to a decrease in the synthesis of triglycerides and apolipoprotein B (apo-B) in the liver [9], as well as inhibition of the synthesis of chylomicrons in the intestine and to facilitate contacts with unsaturated chylomicrons lipoprotein lipase [8; 5]. We identified changes in the level of LDL cholesterol may be associated with a decrease in the initial substrate for their education. [9] Raising HDL complex therapy Omacor is associated with increased formation of HDL-like fragments of VLDL cholesterol particles, with their ω-3 PUFA-mediated lipoproteidlipolize having high ability to absorb cholesterol from the cell membrane [3; 4].

Our results showed a significant effect of Omacor on the level of TC, LDL, HDL, and triglycerides, as opposed to jobs where food additives were used, which showed less significant changes in lipid profile [4]. Also, similar results may be associated with symptoms of synergies between ω-3 fatty acids and a statin that has been demonstrated in a number of large international

studies — JELIS [10], COMBOS [6]. In the GISSI-HF study included approximately 7000 patients with heart failure. Use of Omacor in a dose of 1 g/day for an average of 3.9 years, caused a significant reduction in the risk of death from any cause by 9% compared with placebo. According to the results of many studies have highlighted the effectiveness of Omega 3 PUFA-in terms of their ability to reduce the severity of hypertriglyceridemia. It has been shown that their use will reduce the serum concentration of triglycerides by more than 60%. Application ω3-PUFA during the initial hypertriglyceridemia also contributes to a marked reduction in the likelihood of thrombus formation, decrease cardiovascular risk.

Findings

The preparation of omega-3 PUFA (Omacor) has lipid-lowering effect of reducing the level of TC, LDL–C, TG and increasing the concentration of HDL in patients with coronary artery disease and heart failure. Our experience of Omacor has shown that the drug has a sufficient efficiency, well tolerated and can be recommended for use in patients with coronary artery disease and heart failure with hypercholesterolemia.

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Non-invasive Brain Cooling in Severe Multisystem Brain Injury Complicated Septic Shock

Abstract: The basis of the therapeutic effect of non-invasive brain cooling in severe traumatic brain injuries is to protect brain tissue from the damaging effect of oxygen deficiency. From this perspective, the results of studying the effect of nasopharyngeal cooling on the state of the brain stem structures both in primary and secondary ischemic injury are of interest.

Keywords: severe brain injury, non-invasive brain cooling, nasopharyngeal cooling, brain stem structures.

Traumatic brain injury (TBI) is a critical public health and socio-economic problem throughout the world. It is a major cause of death, especially among young adults [1], and lifelong disability is common in those who survive. Although high-quality prevalence data are scarce, it is estimated that in the USA, around 5.3 million people are