pay special attention to the need for stricter control of not only carbohydrate metabolism, but also lipid metabolism, as well as blood pressure indicators through the prism of vascular risk, or the risk of developing fatal vascular complications of type 2 diabetes mellitus. The most preferred are combinations of oral hypoglycemic drugs that act on both pathophysiological defects of type 2 diabetes mellitus (for example, metformin in combination with sulfonylurea, sulfonylurea in combination with exenatide). The most effective combination is insulin plus metformin. It is important to note that the combination therapy of insulin and thiazolidinediones is currently not approved in the EU countries.

Conclusions. If it is impossible to achieve or maintain "near-normal" glycemic values using one group of drugs, the appointment of combination therapy is indicated. Taking into account the results of international studies, it is recommended to prescribe insulin therapy earlier in patients who have not reached the target glycemic values with the help of oral glucose-lowering drugs.

MEDICINAL METHODS FOR PROATHEROSCLEROTIC STATES TREATMENT

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Introduction. The results of modern studies demonstrate that low density lipoproteins cholesterol(LDL-Ch) and very low density lipoproteins cholesterol (VLDL-Ch) levels are the risk factor for atherosclerosis and correlate with the coronary heart disease. One of the main tasks of treatment is to lower cholesterol level, which can be adjusted by medicinal therapy.

Aim. The purpose of this research was to study the medicinal methods for proatherosclerotic states treatment.

Materials and methods. To fulfill the task, we studied the literature resources concerning the treatment of hypercholesterolemia as reason that caused proatherosclarotic states.

Results and discussion. Several medications that lowering cholesterol are known, particularly, statins, bile acid sequestrants and cholesterol absorption inhibitors. Statins are highly effective because of reducing the cholesterol synthesis in the liver by competitively inhibiting the 3-hydroxy-3-methyl-glutaryl-coenzyme A (HMG-CoA) reductase activity. A decrease in intracellular cholesterol concentration

causes the expression of LDL receptors on the surface of hepatocytes, which contributes to an increase in the LDL-Ch extraction from the blood and a decrease in the LDL-Ch concentration circulating and other lipoproteins containing apoprotein B. Numerous clinical studies have shown that statins significantly reduce cardiovascular morbidity and mortality when used as primary and secondary prevention agents. In clinical trials, statins slowed the progression and even caused regression of coronary atherosclerosis. Statins differ in their absorption, bioavailability, plasma protein binding, excretion, and solubility properties. These enzymes are mainly expressed in the liver and intestinal wall. Although statin use is generally effective in preventing cardiovascular diseases the response to treatment, as well as the incidence of adverse events, varies from patient to patient.

Although many patients achieve their LDL-Ch targets with monotherapy, some high-risk or very high LDL-Ch patients may need additional drugs. There are also patients who do not tolerate statins well or cannot take them in high doses. In such cases, it is necessary to consider the possibility of a combination drug therapy.

Recently, a number of promising new drugs, which are under clinical trials, effectively reduced LDL-Ch in patients with severe hypercholesterolemia. These drugs primarily include microsomal carrier protein inhibitors, thyroid hormone mimetics, and oligonucleotides, which specifically inhibits the synthesis of apo B.

Conclusions. Thus, statins reduce total cholesterol, LDL-Ch and VLDL-Ch in blood plasma. At the same time, medications from this group are can reduce the triglycerides level and slightly increase the level of high density lipoproteins.

ВПЛИВ ДЕФІЦИТУ ВІТАМІНУ Д НА РОЗВИТОК ТА ПРОТІКАННЯ ЦУКРОВОГО ДІАБЕТУ 1 ТИПУ

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Вступ. Цукровий діабет 1-го типу (ЦД1) – це хронічне ендокринно-метаболічне захворювання, викликане інсуліновою недостатністю внаслідок імуноопосередкованої Т-клітинами деструкції β-клітин підшлункової залози, що призводить до необхідності інсулінотерапії. ЦД1 розглядається як багатофакторне захворювання, при якому генетична схильність та фактори навколишнього середовища взаємодіють, сприяючи спрацьовуванню аутоімунних реакцій на β-клітини. Дослідження останніх десятиліть свідчать,