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DIABETES MELLITUS IN COMBINATION WITH CORONARY HEART DISEASE

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Abstract:

The article is devoted to the study of the prognosis and course of Diabetes mellitus in combination with coronary heart disease. Also in this article, the features of biochemical blood parameters in patients in the study group are highlighted. An important problem at the moment is carbohydrate metabolism disorders, which in turn worsen the prognosis of patients with CVD. The prevalence of diabetes mellitus (DM) in patients with coronary heart disease (CHD) reaches 20-30%, 70% have newly diagnosed DM or impaired glucose tolerance (HTG).

Keywords: Diabetes mellitus, coronary heart disease, homocysteine, glucose, carbohydrate metabolism, insulin resistance, dyslipidemia, glycated hemoglobin.

INTRODUCTION

According to the International Diabetes Federation (IDF), the incidence of diabetes mellitus (DM) in the world is growing (from 8.8% in 2017 to 9.9% by 2045). The incidence of coronary heart disease in men with diabetes is 2 times, and in women it is 3 times higher than the incidence of coronary heart disease in the general population. In between the ages of 30 and 55, 35% of diabetic patients die from coronary heart disease, while in the general population, coronary heart disease is the cause of death only in 8% of men and 4% of women of the same age category. Causes, risks and features of ischemia in diabetes mellitus. The chance of heart ischemia formation in patients with diabetes mellitus is significantly higher than in other categories of patients – by 3-5 times. To a greater extent, the development and course of heart disease in this case depends on its duration, rather than on the severity of the diabetic condition. In patients with DM complications of ischemia they develop much earlier than in all other risk groups. At the initial stage, they are most often asymptomatic, which makes it difficult to make a timely diagnosis. The disease may not manifest itself until a painless myocardial infarction.

It is necessary to take into account the relevance of screening examinations for early detection of diabetes, since the main goal of treatment of coronary heart disease is to reduce the risk of development and progression of complications and mortality in patients with diabetes.

The high frequency of death from CVD among patients with DM is primarily due to objective difficulties in early diagnosis of coronary heart disease. According to the submissions of various researchers: the asymptomatic nature of myocardial ischemia in the presence of proven Coronary heart disease is registered in 30-48% of patients. The frequent development of the cardiac form of autonomic neuropathy leads to the inability to accurately use the standard criterion for achieving the diagnostic level of the stress test for the level of heart rate recommended by the American Heart Association and By the American College of Cardiology. As a result of diabetic autonomic neuropathy, progressive denervation of the heart and blood vessels occurs. Absence of a characteristic pain syndrome it may be the cause of delayed recognition of ischemia and myocardial infarction, which delays the appointment of the necessary therapy. Loss of pain sensitivity means the absence of a limiting factor during exercise, which, accordingly, increases the risk of heart attack myocardium. It is the pain-free form of a heart attack myocardium is one of the causes of sudden death in DM.

One of the important goals of reducing cardiovascular events is the control of lipid profile, glycemia, homocysteinemia, and risk factors for CVD.

High mortality in patients with DM is associated with systemic atherosclerotic vascular lesion. Dyslipidemia mixed or isolated (an increase in the level of triglycerides and / or serum cholesterol) is determined, as a rule, in every second elderly patient Type 2 SD. In DM, well-studied risk factors are identified in the pathogenesis of atherosclerosis, including including non-correctable (age, gender, heredity) and correctable (hypertension, smoking, unbalanced diet, obesity and physical inactivity), as well as partially correctable (dyslipidemia, insulin resistance, psychoemotional stress.

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Hyperglycemia in diabetes contributes to the development of atherogenesis in the vascular wall with a high prevalence of atherosclerotic lesions with endothelial damage, smooth muscle cell growth, fibrinolysis, thrombosis, proliferation and increased oxidative stress with the trigger role of cytokines. Chronic hyperglycemia is one of the main factors leading to vascular wall damage in diabetes, and leads to an increase in glycosylation and oxidation of proteins involved in lipid metabolism, blood clotting system and vascular hemostasis. In addition to glycemia, there are a number of important factors of vascular wall damage that lead to cascading vascular wall damage.

Insulin deficiency and insulin resistance. If autoimmune destruction of beta-cells of the pancreas occurs in type 1 diabetes mellitus (DM1) and there is an absolute deficiency of insulin secretion, then type 2 diabetes (DM2), which accounts for 90-95% of all cases of diabetes, is characterized by resistance to the action of insulin and a compensatory reaction in the form of its hypersecretion. As a result, an insulin-resistant state is formed, which affects the change in platelet function using various mechanisms.

One of them is associated with the substrate of the insulin receptor (SIR), a large cytoplasmic protein. Previously, it was assumed that it is specific only for insulin, but in recent years it has been proven that SIR is the basis for many receptor systems, including insulin-like growth factor-1 (IGF-1), similar in structure to pro-insulin. This hormone is called insulin-like in connection with the ability to activate the absorption of glucose by muscle and adipose tissue, similar to insulin.

In vitro platelets not only express SIR and IGF-1, but also stimulate phosphorylation of IGF-1 receptors, tyrosine residues of SIR and protein kinase C. This process is dose-dependent: the higher the insulin resistance, the more The effect of SIR and IGF-1 on the increase of platelet aggregation properties is expressed. Another insulin-mediated mechanism affecting the creation of an abnormal platelet structure in DM is an increase in the concentration of intracellular calcium and its ionization. Normally, ionized calcium, through the activation of enzymes such as phospholipases C and A, triggers a cascade of arachidonic acid, followed by the formation of thromboxane and prostacyclin (prostaglandin I2). In conditions of insulin resistance and relative insulin deficiency, its interaction with platelets through SIR and IGF-1 reduce the expression of the prostacyclin receptor. At the same time, the balance between prostacyclin, a powerful inhibitor of platelet aggregation, and the stimulator of their aggregation, thromboxane, is disturbed in favor of the latter. The importance of insulin resistance in the development of platelet dysfunction is demonstrated by the use of thiazolidinediones: rosiglitazone increases the sensitivity of platelets to endothelial secreted nitrogen monoxide and reduces the expression of P-selectin, thereby confirming the hypothesis that the benefits of reducing the glucose level depends on the way this was achieved. Metabolic and cellular disorders associated with DM. DM2 is associated with a number of metabolic conditions, such as dyslipidemia, obesity, systemic inflammation. A typical violation of the lipid profile of the "diabetic triad" type for diabetes is manifested by hypertriglyceridemia, as well as an increase in the levels of very low-density lipoproteins. The structure of the latter includes apolipoprotein E, similar in its physico-chemical properties to plasminogen, which makes it possible to prevent the formation of plasmin from plasminogen in a competitive way and reduce the fibrinolytic response to the formation of an intravascular thrombus. In turn, the severity of diabetic dyslipidemia is closely related to hyperinsulinemia, in which conditions lipolysis in adipose tissue increases and the concentration of free fatty acids (FFA) and triglycerides increases. Obesity, especially visceral, is a frequent companion of diabetes. With obesity, the number and average volume of platelets is significantly higher compared to people with a normal body mass index. In addition, the peptide hormone in adipose tissue, leptin, the amount of which depends on the severity of obesity, enhances platelet adhesion through leptin receptors.

The results of numerous studies have allowed us to formulate a hypothesis of a close relationship between thrombotic occlusion of the arteries due to the formation or damage of atherosclerotic plaque and inflammatory factors such astumor necrosis factor (TNF-α), C-reactive protein, interleukin-6. These factors affect the blood clotting system through the expression of the platelet receptor Fcgamaria. In patients with DM, even in the absence of coronary heart disease, the levels of inflammatory markers are elevated. Under such conditions, excessive formation of oxidants – superoxide anions – leads to suppression of the synthesis of antioxidants, including platelet, and to the expression of platelet adhesion integrins – P-selectin, as well as surface proteins and glycoproteins (GP): Ib and IIb/IIIa.Increased platelet reactivity is not the only cause of atherothrombotic complications, other links of hemostasis are also disrupted.

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Abnormalities of pro-coagulation, such as increased synthesis of coagulation factors in blood plasma (factor VII and thrombin) and coagulants concentrated in the injury zone (tissue factor), excessive decrease in the level of endogenous anticoagulants (protein C and thrombomodulin), as well as increased synthesis of fibrinolysis inhibitor increase the risk of thrombosis in patients with DM. Thus, in DM, the hemostasis system is characterized by dysregulation of a number of signaling pathways both from the interaction of the receptor and the surface membrane of the cells of the blood coagulation system, and from subsequent intracellular changes. At the same time, he the platelet is extremely variable and depends not only on hereditary, but also acquired factors, where chronic hyperglycemia and its consequences play a significant role. It is obvious that these processes affect a higher probability of developing ACS and its worse prognosis, as well as the insufficient effectiveness of standard antiplatelet therapy in patients with DM.

There is also evidence of a new metabolic risk factor for cardiovascular complications of DM - hyperhomocysteinemia. Homocysteine is a sulfur–containing amino acid that is synthesized from methionine in the process of multistage metabolism. Homocysteine can be converted back to methionine with the help of group B vitamins. Homocysteine also acts as an allosteric antagonist of dopamine D2 receptors. It has even been suggested that homocysteine may have played an important role in the origin of life on earth. Homocysteine levels are usually higher in men than in women, and are constantly increasing with age. Average levels of homocysteine in adults are in the range of 10-12 mmol/l, and values from 20mmol / l and higher is observed in the elderly or with vitamin B12 deficiency.

Homocysteine values above 15 mmol/l indicate hyperhomocysteinemia, which is a significant risk for the development of thrombosis, neuropsychiatric diseases, bone fractures, and is also considered a marker of increased risk of cardiovascular diseases and kidney disease.

Approximately 70% of homocysteine in the kidneys is converted to methionine, so kidney diseases and a decrease in their effective work contribute to an increase in homocysteine levels and an increase in risks development of cardiovascular pathologies. Homocysteine affects the tissues of blood vessels so that they become loose, local inflammation occurs due to the action of immune cells, and cholesterol and calcium are deposited on this surface, which contributes to the formation of plaques. As a consequence of diabetic nephropathy, an auxiliary mechanism is an intense lesion of the vascular system of the entire macroorganism. This explains that the main function of converting homocysteine is assigned to the kidneys. Under the influence of homocysteine, low-density lipoproteins are oxidized to form small dense particles prone to aggregation. These microparticles are absorbed by macrophages to form "foam cells", which with the blood flow enter various tissues of the body, including the tubulointerstitial kidney tissue. Foam cells are a new additional source of reactive oxygen radicals, which leads to even greater damage to the endothelium of the vascular wall. The endothelium plays a leading role in the control of vascular tone with the help of biologically active substances. One of these the substance is nitric oxide, the production of which is carried out by the endothelium continuously. Nitric oxide is characterized by several protective properties, which include vasodilation, inhibition of proliferation of smooth muscle cells, reduction of platelet aggregation and other blood cells. Under normal conditions, nitric oxide has the ability to react with homocysteine and thus "neutralizes" it. Homocysteine reduces the production of endothelin-1(ET1). ET-1 is a protein consisting of 21 amino acids synthesized by the vascular endothelium. ET-1, binding to specific transmembrane receptors of smooth muscle cells, stimulates their proliferation, and also has a powerful vasoconstrictor effect. These basic properties of ET-1 determine its role in the development of vascular pathology. However, ET-1 is also capable of causing a depressive reaction by interacting with transmembrane receptors, but already endothelial cells. Numerous studies have shown that GHZ leads to a significant increase in vascular wall density due to increased synthesis and accumulation of collagen in it. These changes are explained by the ability of homocysteine to stimulate collagen synthesis by fibroblasts of smooth muscle cells of the vascular wall, and the accumulation of collagen in the cell layer occurs in parallel with the increase in the concentration of amino acids. As a result of the accumulation of collagen and proliferation of smooth muscle cells of the vascular wall, its deformation, thickening and increased rigidity occur. In the literature there are information that homocysteine disrupts the function of the tissue plasminogen activator, promotes the binding of lipoprotein (a) to fibrin, which leads to inhibition of fibrinolysis. GHZ inhibits the function of natural anticoagulants such as antithrombin III and protein C.

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THE PURPOSE OF THE WORK

1. To study the statistical data on the incidence of diabetes mellitus in KGP at the from 2018 to 2020.
2. To conduct a comparative analysis of clinical (including correctable and uncorrectable risk factors) and laboratory data in patients.
3 Duration of the study (duration of the study) – the duration of observation is 3 years.
4. Characteristics of the subjects The study is planned to include patients _247____ (number) with diagnoses of type 2 diabetes mellitus, as well as Coronary heart disease in combination with type 2 diabetes mellitus. The age of patients is from 39 years to 74 years. The study included a group of patients diagnosed with type 2 diabetes mellitus (total number - group 1) and patients diagnosed with coronary heart disease in combination with type 2 diabetes mellitus (group 2).
5. Research methodology
5.1 Describe the study design: A closed clinical prospective comparative controlled study is conducted on this group of patients. The study scheme will include patients according to their group, taking into account the indicators: fasting glycemia, glycated hemoglobin, lipid profile, hemostasis; instrumental: diagnostic coronary angiography, stenting, CABG, risk factors.
5.2 Distribution of patients into groups
5.3 Criteria for inclusion of subjects: participation in the PUZ program (Disease Management Program), written consent of the patient to participate, dynamic observation.
5.4 Criteria for non-inclusion of subjects: refusal of laboratory studies.
5.5 Criteria for excluding subjects: death or relocation of the patient.

MATERIALS AND METHODS

247 patients with type 2 diabetes (98 men and 149 women) were examined, the average age of patients was 51 years. The average life expectancy was 8-11 years. In order to study fasting glycemia, glycated hemoglobin, lipid profile, hemostasis; instrumental: diagnostic coronary angiography, stenting, CABG, risk factors – patients were divided into 2 groups. The first group included 74 patients with type 2 diabetes with coronary heart disease (the average age was 60 years). The second group included 173 patients with type 2 diabetes without coronary heart disease (the average age was 53 years).
The data of a study of 247 patients with DM in combination with coronary heart disease were analyzed.

RESULTS

Studies in a cohort of 247 people with diabetes mellitus, 74 of them in combination with coronary heart disease were identified: general clinical and biochemical (fasting glycemia, glycated hemoglobin, lipid profile, hemostasis); instrumental: diagnostic coronary angiography, stenting, CABG, risk factors). Conclusions. The number of patients with diabetes in the Pavlodar region has increased by 20.6% over the past 3 years. There were 95% of patients with type 2 diabetes, including the middle-aged and elderly accounted for 87%. It follows from Table 3 that the majority of patients underwent percutaneous intervention and the main part underwent coronary artery stenting (41%), compared to those who have a number of corrected risk factors, they are more susceptible to myocardial infarction and, consequently, fatal outcomes. The age over 50 was 76.1%, of which 60.4% were women. Out of the general group of patients with DM with coronary heart disease, 8.5% underwent AMI. A heavily controlled lipidemic profile is observed in 74.4%.

CONCLUSION

So, it can be assumed that the decompensation of laboratory parameters indicating a violation of carbohydrate metabolism contributes to the development of endothelial dysfunction, which leads to the activation of thrombosis and hypercoagulation. Violation of lipid metabolism can affect the function of platelets. Dyslipidemia plays the most important role in the violation of the fluid and oxygen -carrying properties of blood, as well as the development of tissue hypoxia in DM. When studying the lipid spectrum of patients, an increase in the level of OHS, LDL in the blood serum was revealed. In patients with type 2 diabetes, hypercholesterolemia was detected in 85%, high LDL levels were detected in 89%. When comparing the indicators of the hemostasis system of patients with DM with coronary heart disease (group 1) and DM without Coronary heart disease (group 2), there were no differences in BMI in patients in the observed groups. The level of glycemia on an empty stomach and after meals, HbA1c was significantly higher in patients with coronary heart disease.
Thus, the developing imbalance between damage and restoration of the endothelium in type 2 diabetes in combination with coronary heart disease may affect the formation and prognosis of the disease. The study of pathogenetic mechanisms of formation of diabetic complications, as well as their prevention. Recent

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studies show that a number of pathological changes occur in the development of vascular complications, including activation of the inflammatory cascade, oxidative stress and impaired hemocoagulation. Circulating biological markers of these processes can potentially be used for early diagnosis of vascular complications, as well as become targets for new treatment methods.

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