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Cardiac Complications of Endocrine Diseases: A Literature Review

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Abstract

Endocrine disorders in almost all cases lead to pathological changes in metabolism, including dyslipidemia and deterioration of carbohydrate metabolism. Thus, insufficient glycemic control in diabetes mellitus contributes to an increase in triglyceride levels and the development of atherogenic dyslipidemia. In turn, this promotes changes in the cardiovascular system at the structural and functional level. Frequent phenomena may include: increased stiffness of the arterial wall, hypertension, cardiomyopathy, vascular atherosclerosis, coronary heart disease and heart failure. Hypercholesterolemia contributes to the occurrence of microvascular damage, increased blood viscosity, inflammation, thrombosis, vascular occlusion, and atherogenesis. In addition, endocrine diseases activate other pathogenic mechanisms, including increased sensitivity of the myocardium to the adrenergic system, and a direct toxic effect on the heart muscle. In the absence of timely and adequate treatment, the risks of death from late cardiovascular complications increase. Properly selected therapy allows not only to normalize metabolism, but also to significantly neutralize pathological processes in the cardiovascular area or push back the time of their occurrence to a later stage of life.
Keywords: cardiovascular system, dyslipidemia, atherosclerosis, arterial hypertension, coronary heart disease

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Резюме

Эндокринные нарушения почти во всех случаях приводят к патологическому изменению метаболизма, включая дислипидемии и ухудшение обмена углеводов. Так, недостаточный контроль гликемии при сахарном диабете способствует повышению уровня триглицеридов и развитию атерогенной дислипидемии. В свою очередь это способствует изменениям в сердечно-сосудистой системе на структурном и функциональном уровне. Частыми явлениями могут быть: повышение жесткости артериальной стенки, гипертензии, кардиомиопатии, атеросклероз сосудов, ишемическая болезнь сердца и сердечная недостаточность. Гиперхолестеринемия способствует возникновению микрососудистых повреждений, повышению вязкости крови, воспалению, тромбозу, окклюзии сосудов, атерогенезу. Кроме того, эндокринные заболевания активируют и другие патогенные механизмы, включая усиление чувствительности миокарда к адренергической системе и прямое токсическое действие на сердечную мышцу. При отсутствии своевременного и адекватного лечения возрастают риски смерти от поздних сердечно-сосудистых осложнений. Правильно подобранная терапия позволяет не только нормализовать обмен веществ, но и значительно нивелировать патологические процессы в кардиоваскулярной области или отодвинуть время их возникновения на поздний этап жизни.
Ключевые слова: сердечно-сосудистая система, дислипидемия, атеросклероз, артериальная гипертензия, ишемическая болезнь сердца

■ INTRODUCTION

Cardiovascular diseases (CVD), in the pathogenesis of which hyperlipidemia and atherosclerosis play the most important role, are one of the main mortality reasons in the developed world. The highest risk of CVD progression is connected with the increased

cholesterol (CS) level in LDLP. The examination for dyslipidemia is aimed, among other issues, at eliminating the secondary reasons of lipid disorders, including different endocrine diseases. A great many endocrine disorders are complicated by hypertension, ischemic heart disease (IHD), cardiomyopathies, etc. [1]. Dyslipidemias and other metabolic disorders in endocrine diseases increase the risk of cardiovascular damages, including coronary atherosclerosis, myocardial infarction, cardiac insufficiency and mortality from CVD [2]. When dealing with endocrine disorders, it is necessary to evaluate all delayed risks and choose the therapy so that to decrease the frequency of realization of these risks or shift them to a later period of life.

The purpose of the work – to analyze possible cardiovascular risks increased on the background of endocrine disorders.

■ DIABETES MELLITUS

If the relevant treatment is not provided, diabetes mellitus (DM) leads to hypertriglyceridemia and atherogenic dyslipidemia. Apart from the increased level of triglycerides, patients with DM2 have an elevated content of CS-LDLP, decreased concentration of high-density lipoproteins (HDLP) and apolipoprotein A1 (Apo A1), which is connected with insulin resistance. An intensified flow of fatty acids from the fatty tissue to the liver worsens the liver insulin resistance and enhances dyslipidemia [3].

Even against the background of statin therapy, a high content of triglycerides in the blood serum is registered in a half of patients with DM2, which is also the result of dietary violations: high glycemic index of the food taken and prevalence of saturated fat in the diet. A considerable level of postprandial triglycerides is provided by the food fat, which comes to bloodstream from intestines in the composition of chylomicrons. The enhanced synthesis of triglycerides and their excretion into VLDLP take place in the liver due to insulin resistance and fat excess. Decreased lipoprotein lipase activity as a result of the increased Apo-C3 synthesis is another reason of the elevated level of triglycerides. Patients have fine dense LDLP particles, related to high-atherogenicity particles, circulating in their blood. With high hyperglycemia, the ongoing processes of glycation and oxidation of lipoproteins result in the accelerated atherosclerosis and its complications [4]. Hypertriglyceridemia in combination with atherosclerosis is the most significant CVD risk factor. Hypercholesterolemia leads to pathological changes in the vessel walls and thrombosis through the initiation of microvascular injuries. A high cholesterol level in the blood activates endothelial proteins (p-selectin), increases the blood viscosity and activity of neutrophils in the microvascular stream that results in inflammatory vascular thrombosis [5]. Besides, the combination of hyperglycemia and insulin resistance triggers the oxidative stress, interferes the transmission of protein kinase C signals, increases the amount of glycation products that provokes inflammation, occlusion of vessels, thrombosis and atherogenesis. The intensive DM2 treatment results in decreasing the risk of macro- and microvascular events more than by 10% [6]. Despite the recent tendency to decreased CVD mortality, the double prevalence of mortality among people with DM in contrast to patients without diabetes is preserved [7].

Not only DM2 but pre-diabetes is also characterized by increased cardiovascular risks. In particular, people with pre-diabetes (impaired fasting glycemia and inadequate glucose tolerance) have the increased arterial stiffness, more aggressive coronary sclerosis course, intensified frequency of cardiac insufficiency. The timely treatment can

decrease the frequency of DM2 progression in this group and decrease cardiovascular risks [8, 9].

The therapy in the considered cohort of patients is mainly aimed at normalizing CS-LDLP level, and triglycerides are considered to be the target of the second line. The efforts of medical specialists should be aimed at changing the lifestyle envisaging the diet modification and increased physical activity: only this can result in decreased CS-LDLP content by 10–15%. For patients with DM2 it is recommended to decrease CS-LDLP concentration below 70 mg/dl. The decreased content of triglycerides below 500 mg/dl can be achieved by the therapeutic lifestyle and prescription of statins. In case of severe triglyceridemia, fibrates and/or omega-3 fatty acids are prescribed [4].

The glucose content is monitored individually. For monotherapy, the prescription of metformin is optimal. If hemoglobin A1c is elevated, it is necessary to consider the possibility of applying inhibitor of sodium/glucose-2 cotransporter (SGLT2) or inhibitor of dipeptyl peptidase-4 (DPP-4). In some cases, antilipedimics are prescribed (ezetimibe, fibrate, proprotein convertase, subtilisin/kexin of type 9), inhibitors of sodium/glucose-2 cotransporter (SGLT2), as well as nutraceutical medications (berberine, omega-3 fatty acids, red yeast rice) complementing the main therapy. Patients with DM and acute myocardial infarction (MI) are recommended to undergo angioplasty/stenting and with multivessel disease – coronary artery bypass graft [3, 6].

■ DISEASES OF THYROID GLAND

Endothelium of myocardium and vessels contains receptors to thyroid hormones and is sensitive to changes in the concentration of circulating hormones. Even insignificant changes in the level of thyroid gland hormones in blood (in subclinical hypothyroidism, hyperthyroidism, low-level tri-iodothyronine syndrome) unfavorably affect the cardiovascular system. These effects are mediated via such states as dyslipidemia, endothelium dysfunction, changes in the arterial pressure and via the direct thyroid gland action onto the myocardium. The treatment of thyroid gland disorders decreases the risk of cardiovascular diseases. The connection between the thyroid gland function disorder during the acute MI and further unfavorable cardiovascular outcomes was found. The thyroid gland hormones play an important therapeutic role in decreasing the infarction size and improving the myocardium function [10].

Deviation from the normal concentration of thyroid gland hormones leads to deep changes in the regulation of cardiovascular hemodynamics. Thyroid gland hormones enhance the expression of calcium-activated ATP-ase of sarcoplasmic reticulum and suppress the expression of phospholamban (key diastolic function regulator). The main effects onto myocardium are mediated by T3 hormone (mainly via TRα1 receptors), which modifies the activity of sodium, potassium and calcium channels, changing the intracellular signal ways in smooth-muscle cardiac cells and vessels. T3 increases myocardium sensitivity due to the increased number of adrenergic membrane receptors. Besides, T3 decreases the systemic vascular resistance due to the relaxation of smooth muscles of the vessels (intensified NO production), that, in turn, decreases the kidneys' perfusion and activates the renin-angiotensin-aldosterone axis. The relaxation of vessels increases the heart oxygenation. T3 activates metabolism and oxygen consumption, inducing the release of vasodilating mediators. In the experiment, the thyroid hormones produced the dose-related direct effect in rats' coronary arteries that indicates the work

of nongenomic regulatory mechanisms. Besides, they produce the proangiogenic effect, stimulating the growth of arterioles in a normal heart and after MI [11].

The increased frequency of cardiac beats at rest, increase in the systolic discharge, myocardial contractility and output fraction are characteristic for hyperthyroidism. The cardiac insufficiency progression is connected with high output in hyperthyroidism and can be conditioned by the cardiomyopathy developed as a result of tachycardia. The long-term functional load onto the heart in toxic goiter results in myocardium dystrophy followed by the decreased exercise tolerance, occurrence of dyspnea, abnormal heart rhythms [12]. Cardiac insufficiency is the main reason of increased CVD mortality rate, both in distinct and subclinical hyperthyroidism.

The deficiency of thyroid gland hormones in hypothyroidism results in the drop of frequency of cardiac beats, decreased myocardium contractions and lusitropy with the extension of systolic and early diastolic time. The heart preload decreases due to the diastolic function disorder. The heart preload increases, and the chronotropic and inotropic functions decrease (Table 1). The subclinical dysfunction of thyroid gland is often observed in patients over 65. Subclinical hypothyroidism increases the mortality risk from IHD and its complications. The mortality risk from IHD and atrial fibrillation increases at very low level of thyreotropin. Hypothyroidism in elderly people can have a subclinical form. TSH levels can increase with age and inconsiderable increase does not always indicate the subclinical hypothyroidism. If the diagnosis is verified, it is necessary to carefully titrate the levothyroxine dose to avoid such unfavorable complications as atrial fibrillation, angina, osteoporosis. Iodine-containing medications, such as amiodarone, can induce hypothyroidism (Wolff-Chaikoff effect) and hyperthyroidism (Jaud-Basedow effect) [11, 13, 14]. In the late post-infarction period, the thyroid gland hormones modulate the structure, function and geometry of the heart left ventricle [11]. The unattended hypothyroidism (myxedema) can result in severe cardiac insufficiency with ascites, anasarca, hydrocardia and death [15].

It was demonstrated in Mendelian randomized study (atrial fibrillation n=5514; reference group n=482 295) that an elevated ratio of FT3:FT4 and hypothyroidism were directly connected with the increased risk of atrial fibrillation (AF), and normal values of thyreotropin and hypothyroidism were inversely correlated with AF risk. The direct involvement of FT4 reference values and increased titer of antibodies to thyroid peroxidase in atrial fibrillation progression was not proved [16].

The connection between thyroid gland dysfunction, on the one hand, and the class of cardiac insufficiency by New-York Cardiologic Association, atrial fibrillation and combined

Table 1
Influence of thyroid gland dysfunction on cardiovascular system [11]

Hypothyroidism: distinct or subclinical	Hyperthyroidism: distinct or subclinical
↓ pulse	↑ pulse
↓ inotropism	↑ myocardium contractility
↑ peripheral vascular resistance	↑ cardiac output
Diastolic hypertension	Increased pulse pressure
Sinus bradycardia	Vascular distention
↑ increased risk of atherosclerosis and dyslipidemia	↑ blood volume
Pericarditics, pericardial effusion, cardiac tamponade	↓ medium arterial pressure
	Atrial fibrillation, pulmonary hypertension, atrioventricular valve insufficiency

end point of supplementary ventricular device installation, heart transplantation or death of 1365 participants with cardiac insufficiency, on the other hand, was studied in another work. The subclinical hyperthyroidism with TSH level ≥7 mMU/l and isolated T3 low level in patients with cardiac insufficiency previously detected was connected with the unfavorable forecast regarding all tested outcomes [17].

■ PATHOLOGY OF ADRENAL GLANDS

About 52% of patients with hypercorticoidism are subject to secondary lipid disorders. Sometimes this can be the result of treatment with glucocorticoids. Even in cases with nonfunctional tumors of adrenal glands, the risk of cardiovascular complication is increased. Glucocorticoids can result in dyslipidemia connected with abdominal obesity and metabolic syndrome [4].

When "Cushing disease" is diagnosed, the majority of patients have cardiovascular, metabolic and endocrine disorders. Obesity, diabetes or glucose tolerance decrease, dyslipidemia, arterial hypertension, cardiac and vascular pathology are detected in the patients. Hypercortisolemia remission decreases but does not completely eliminate cardiovascular complications. The increased risks of IM and insult are noted in patients with Cushing disease (CD) during the long-term monitoring. High levels of triglycerides and total CS with nonpermanent fluctuations of HDLP level are detected in the blood. An excess of glucocorticoids stimulates the lipolysis and production of free fatty acids resulting in the liver steatosis [18].

With active CD, hypertonia is detected in 49–78% of cases and is connected with hypercorticoidism duration and severity. The activity of mineralocorticoids, increased production of vasoconstrictors (endothelin-1), enhanced CVS reactivity to vasoconstrictors, modulation of renin-angiotensin-aldosterone system, inhibition of vasodilators' release (NO, prostaglandins E2 and I2), activation of sympathetic nervous system count in pathogenesis. When CD remission is reached, hypertonia is preserved in the considerable part of the patients, probably, as a result of remodeling of the vessels and irreversible changes in the heart. The hypertrophy of left ventricle (LV) and concentric remodeling are observed in the patients during the long-term CD treatment. The systolic and diastolic dysfunctions are the result of the considerably decreased output fraction of LV, right ventricle (RV) and left atrium (LA). Sometimes patients with CD have dilated cardiomyopathy or marked cardiac insufficiency. The intensified response to angiotensin II, myocardium fibrosis and activation of receptors to mineralocorticoids matter in the pathogenesis of such changes. The latter circumstance can result in hypokalemia and fatal arrhythmias [18]. The cases of spontaneous dissection of coronary arteries of patients with CD and lack of other popular reasons of such complication occurrence were described. Nevertheless, the indicated connection needs a wider clinical study [19]. The increased intima thickness of large arteries, growth of atherosclerotic plaques, cases of arterial thrombosis are detected on the level of vessels. In people with CD, the risk of IHD increases in 3.6 times in comparison with those without it. Even when CD remission is achieved, it is still impossible to completely eliminate pathological cardiovascular changes [18]. Cardiovascular diseases are the main mortality reason of patients with CD [20].

Apart from CD, other diseases of adrenal glands also produce cardiovascular complications. Thus, when examining 163 patients with pheochromocytoma, 63 of them had dilatational cardiomyopathy, 38 – Takotsubo cardiomyopathy, 30 – inverted

Takotsubo cardiomyopathy, 10 – hypertrophic cardiomyopathy, and 14 – unspecified cardiomyopathy. Such disorders as heart pounding and arterial hypertension are characteristic for pheochromocytoma [21]. In the review by Monticone et al. the connection between initial aldosteronism and cardiovascular complications was studied. After 8.8 years of observations from the moment of diagnosing arterial hypertension, the patients with the initial aldosteronism demonstrated the increased risk of insult (odds ration [OR] 2.58, 95% CI 1.93–3.45), ischemic heart disease (1.77, CI 1.10–2.83), atrial fibrillation (3.52, 2.06–5.99), cardiac insufficiency (2.05, CI 1.11–3.78), left ventricle hypertrophy (2.1, CI 1.65–3.17) in comparison with patients only with AH [22].

■ HYPOPHISIAL DISORDERS

Hypophisial disorders are rather varied and changes in the patient's lipid profile often underlie cardiovascular complications (Table 2).

Cardiovascular diseases (together with oncologic) are the main mortality reason in active acromegalia. Patients with acromegalia are often diagnosed with arterial hypertension, cardiomyopathy, arrhythmia, diabetes mellitus, dyslipidemia. With acromegalia the growth hormone and IGF-1 contribute to endothelium dysfunction and atherosclerosis by many different mechanisms. The coronary blood flow in persons with acromegalia is often decreased. Nevertheless, such patients do not always have distinct atherosclerotic damages and IHD. The growth hormone hypersecretion duration, age and body weight index are the key factors of cardiovascular pathology progression. The majority of patients, especially women, have arterial hypertension with elevated diastolic AP and frequent lack of AP decrease at night. Effective neurosurgery of hypothesis adenoma and pharmacological therapy decrease the LV weight and improve the cardiac function [23].

The growth hormone deficiency (GHD) is accompanied by unfavorable cardiometabolic risk profile. Apart from cardiovascular risk factors, patients with GHD have the endothelial dysfunction, subinflammation, disordered profile of adipokines, oxidation stress and D hypovitaminosis. Substitution therapy has a positive effect on some risk factors but does not eliminate all cardiometabolic disorders connected with the disease. GHD is connected with immature atherosclerosis and the increased thickness of intima-media complex of carotid arteries (IMCCA) is considered to be one of the earliest morphological changes. The hormonal substitution therapy can neutralize these changes. Other features of endothelial dysfunction are the decreased aorta elasticity and decreased endothelium-dependent vasodilatation mediated by the blood flow. The untreated patients with GHD have the systolic function disorder, and after 6 months of hormonal substitution therapy a considerable improvement of cardiac index, increased cardiac output, systolic discharge, decreased diastolic arterial pressure are observed [24].

Table 2
Changes in the lipid profile in disorders [4]

Pathology	Total CS	CS-LDLP	Triglycerides	CS-HDLP
Hyperprolactinemia	↑	↑	↑	
Acromegalia			↑	
Growth hormone deficiency	↑/N	↑/N	↑	↓
Combined deficiency of hypophisial hormones	↑	↑		

Hyperprolactinemia is able to cause considerable metabolic changes which, in turn, increase cardiovascular risks. Patients with prolactinoma have the increased total CS and CS-LDLP: their content decreases against the background of the therapy with dopamine agonists and normalization of prolactin level in the blood serum. The increased levels of triglycerides and decrease in CS-HDLP can be also observed. Prolactin can modulate inflammatory reactions, stimulate proliferation of vascular smooth muscle cells and contribute to adhesion of circulating mononuclear cells to endothelium that result in endothelial dysfunction. Besides, a high hormone level decreases NO production, resulting in the increased arterial pressure. Hyperprolactinemia can contribute to the formation of peripartum cardiomyopathy characterized by systolic dysfunction leading to the decreased output fraction and cardiac insufficiency symptoms. The product of prolactin decomposition 16 kDa induces apoptosis, vasoconstriction, decreased metabolism and functional activity of cardiomyocytes resulting in cardiomyopathy progression. Bromocryptine therapy of women with peripartum cardiomyopathy resulted in the improved output fraction in half a year in comparison with the group with standard therapy [25].

Hypopituitarism affects several endocrine organs and metabolic effects vary greatly. Patients with hypopituitarism frequently have the excessive body weight, abdominal obesity, hypertonia, diabetes, low level of HDLP, hypertriglyceridemia. The revealed hormonal disorders usually comprise hypogonadism, central hypothyroidism, growth hormone deficiency, adrenal insufficiency. The patients usually demonstrate the increased frequency of cardiovascular pathology. Hormonal substitution therapy provides positive results in this group of patients and leads to the decreased frequency of cardiovascular complications [26].

■ CONCLUSION

The analysis of publications demonstrates that each endocrine disorder is accompanied by metabolic changes underlying unfavorable cardiovascular events. Lipid spectrum disorders triggering the mechanism of atherosclerosis of coronary vessels represent the most obvious long-term danger. Besides, many endocrinopathies result in thickened vessels' intima and increased arterial stiffness, and such patients are most often diagnosed with arterial hypertension contributing to further unfavorable changes in cardiovascular system (myocardium hypertrophy, decreased cardiac function efficiency, cardiac insufficiency progression, etc.). Some hormones (thyroid) can directly toxically affect the cardiac muscle. Cardiovascular pathology eventually leads to the increased mortality risk among patients with endocrine disorders. This is the reason to start the treatment of the considered group of diseases as early as possible to reverse or slow down fatal metabolic disorders, eliminate or postpone the emergence of life-threatening complications.

Recommendations

1. Treatment of endocrine disorders should be started as early as possible to prevent late complications.
2. For patients with endocrine disorders it is recommended to carefully monitor the level of corresponding hormones (glucose, glycosylated hemoglobin) to timely correct the treatment.

3. Laboratory and instrumental examination of cardiovascular system should be a part of the general examination of patients with endocrine pathology.

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