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Myocardial infarction as a manifestation of thyreocardiac syndrome

Thyreocardiac syndrome is a well-known manifestation of hyperthyroidism. Most definitions connect the symptoms of thyreocardiac syndrome with the synergy of excessive thyroid hormones with other heart injuring factors. Some authors believe that cardiovascular symptoms may develop without preexisting myocardial damage, in completely healthy people. Clinical consequences of cardiovascular alterations are: tachycardia, high systolic-diastolic amplitude of arterial blood pressure, systolic murmurs, dilation of aorta, pulmonary trunk and left ventricle, increased myocardial contractility, increased ejection volume, raised cardiac output, decreased vascular resistance and increased blood flow, elevation of QSR tone and ST segment depression. Thyreocardiac syndrome may proceed as persistently recurrent supraventricular arrhythmia, coronary failure and circulatory failure resistant to conventional treatment.

CASE DESCRIPTION

51-year-old B.M. was admitted to the Intensive Cardiologic Care Unit at the Clinic of Internal Diseases and hospitalized from April 21 to May 29, 1992. On admission she reported a choking sensation in the throat, recurrent retrosternal pains with a burning sensation lasting for a few months, first connected with effort, in the last days before admission occurring at rest. Besides, the patient complained about a heat sensation, profuse sweating, palpitation, swollen feet and shanks, excessive thirst, hand tremor, general weakness, increased nervous excitability, body weight loss of about 10 kg. A week before admission a rash appeared. For 2–3 months earlier she was treated for arterial hypertension, irregularly. Other ailments included arthralgia, hot flushes. Gynaecological history revealed regular menstruation. During physical examination psychomotor agitation, moist skin and micromaculate rash all over the skin were found. Mebius, Graefe and Kocher symptoms were negative. Thyroid gland was slightly enlarged as a whole, on the border of the right lobe and isthmus there was a 1.5 cm nodule. Rhythmic heart action – about 120/ min., RR 120/60, low systolic apex murmur. Percussion sound over the lungs was evident, vesicular murmur was regular (normal). Soft abdomen. Liver and spleen not enlarged. On lower limbs feet and shanks swollen. Supplementary examinations: haemoglobin – 12 g%, leucocytosis – 11,500, erythrocyte sedimentation reaction – 25/35, thrombocytes 189,000, cholesterol 149 mg%, chest X-ray picture: no changes, eye fundus normal, negative reaction to antinuclear and antimitochondrial antibodies. ECG on admission to the Clinic: traits of subendocardial infarct, successive records showed the curve evolution. ECG on discharge: sinus rhythm, regular, about 50/min., levogram, negative symmetric T waves in II, III, aVf and V2–V6.

The level of thyroid hormones confirmed the diagnosis of hyperthyroidism: total thyroxine level (DELFA) – 20.8 ug/100 ml (own norm – 4.5 10 ug/100 ml), free triiodothyronine level (DELFA) – 39.1 p mol/l (own norm 3.4 – 8.9 p mol/l), free thyroxine level >84.0 p mol/l (DELFA) – (own norm 8.5 – 19 p mol/l), TSH level – 0.4 – 5.5 mIU/l. Dermatological consultation: allergic changes on the skin.

The analysis of history taking and physical examination allowed to diagnose myocardiac infarction and hyperthyroidism, which were confirmed by enzymatic and hormonal examinations and ECG changes. The course of infarction uncomplicated, the patient was discharged from the Clinic in good general state.

In the clinical picture cardiovascular symptoms in the form of arterial blood pressure rises, tachycardia and symptoms of unstable cardiac angina were predominant; while in the last days before the admission to the Clinic – the symptoms of circulatory failure with swelling of lower limbs. The ailments increased during 2–3 months; the patient reported to the Outpatients' clinic a few times in that period. The delay in diagnosing thyrotoxicosis may have resulted from the similarities of the hyperthyroidic ailments with those accompanying the period of menopause. The common ailments are: palpitation, heat sensation, increased nervous irritability, arterial blood pressure rises, profuse sweating. The menopausal period and predominant symptoms of coronary failure had masked the symptoms of hyperthyroidism.

Intensification of coronary ailments in the course of thyreocardiac syndrome is connected with metabolic disturbances leading to the increased cardiac output and increased oxygen demand. Under the influence of thyroid hormones the use of oxygen grows, especially in skeletal muscles, heart muscle, liver, kidneys. It is currently assumed that various metabolic mechanisms are responsible for thyreocardiac syndrome.

The excess of thyroid hormones causes metabolic disturbances on the cellular level. The existence of specific receptors for triiodothyronine (T3) was proved within the cellular membrane, cell nucleus chromatin and in mitochondrial internal membrane. The interaction of triiodothyronine with nuclear receptor regulates the transcription phenomenon and leads to the generation of mRNA. However, the interaction of T3 with the receptor in the mitochondrium leads to quick metabolic reactions and is clinically manifested by the increased oxygen consumption. Under the influence of thyroid hormones the energy produced during metabolic changes is not bound in high-energy compounds but turns into heat. The excess heat is transferred by the increased blood circulation rate with simultaneous dilatation of vascular bed and increase in cardiac output. In that situation the oxygen consumpion becomes disproportionately high in relation to ATP produced.

The metabolic mechanism which may be responsible for the changes in the cardiovascular system is the interaction of thyroid hormones and sympathetic nervous system. Symptoms such as tremor, tachycardia, anxiety are suggestive of the stimulation of adrenergic system, however, this is not reflected in the level and excretion of catecholamines, but depends on the density of receptors within the cells. Alpha 1, beta 1 and beta 2 receptors have close affinity with triiodothyronine, alpha 2 receptors, however, are thought to be T3 antagonists. Receptor proteins undergo a modification under the influence of catecholamines.

Thyroid hormones also exert an influence on myocardial contractility, which is independent of catecholamines. Triiodothyronine may stimulate the heart Ca-ATP-ase on which relaxation is dependent. The relaxation index is secondary to the activation of Ca ions of sarcoplasmic reticulum ATP-ase pump. Diastolic compliance of the left ventricle may be decreased in hyperthyroidism.

Apart from the impact on the cardiac muscle through the increase in cardiac work load, thyroid hormones may affect preload and afterload. Studies on humans show that circulating blood volume is increased under the influence of excessive thyroid hormones. The increase of cardiac output is secondary to vasodilatation. The increase in systolic blood pressure depends on the increased stroke volume and peripheral dilatation reduces diastolic pressure. Peripheral resistance can be 30% lower.

RAA system plays a minor role in the development of hyperthyroidism. Plasmic renin level is lowered. However, RAA system may stimulate myocardial hypertrophy synergically with T4 (quadriiodothyronine) and adrenergic system.

In literature, the influence of low TSH level was described to increase cardiovascular mortality with diagnosed left ventricular hypertrophy. What seems significant is the fact that even in subclinical manifestation of hyperthyroidism tachycardia, impaired left ventricular relaxation, arrhythmias, left ventricular mass growth and growth of cardiovascular mortality were observed.

Thyroid fuction may influence the condition of the cardiovascular system. In the case of cardiovascular manifestations the elimination of thyreotoxicosis is necessary in the search of causes of myocardial damage.

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SUMMARY

Thyreocardiac syndrome is manifested by disturbances in the cardiovacular system, resistant to conventional treatment, which is connected with metabolic changes. The mechanisms of metabolic changes are: increased oxygen consumption in mitochondria, myocardial hypertrophy induced by quadriiodothyronine, decrease in diastolic susceptibility (compliance) through the stimulation of Ca-ATP-ase influenced by triiiodothyronine, the synergy of excess thyroid hormones with sympathetic nervous system. A case of myocardial infarction in the course of thyreocardiac syndrome and diagnostic difficulties were described in a 51-year-old female patient, connected with overlapping of the symptoms of hyperthyroidism, menopause and ischaemic heart disease. In hyperthyroidism many authors observe tachycardias, left ventricular relaxation damage, arrhythmias, left ventricular mass growth and increase in cardiovascular mortality. Thyroid function may have an influence on the condition of the circulatory system, hence in the case of clinical symptoms of heart damage it is necessary to eliminate thyreotoxicosis.

Zawał serca jako manifestacja zespołu tarczycowo-sercowego

Zespół tarczycowo-sercowy manifestuje się zaburzeniami w układzie sercowo-naczyniowym opornymi na konwencjonalne leczenie, co jest związane ze zmianami metabolicznymi. Mechanizm zmian metabolicznych to: zwiększone zużycie tlenu w mitochondriach, przerost mięśnia serca z udziałem czterojodotyroniny, zmniejszenie podatności rozkurczowej poprzez stymulację Ca- ATP-azy pod wpływem tyrójjodotyroniny, współdziałanie nadmiaru hormonów tarczycy i sympatycznego układu nerwowego. Opisano przypadek zawału serca w przebiegu zespołu tarczycowo-sercowego i trudności diagnostyczne u 51-letniej chorej, związane z nałożeniem się objawów nadczynności tarczycy, okresu przekwitania i choroby niedokrwiennej serca. W nadczynności tarczycy wielu autorów obserwuje tachykardie, uszkodzenie relaksacji lewej komory, arytmie, wzrost masy lewej komory i wzrost śmiertelności sercowo-naczyniowej. Czynność tarczycy może mieć wpływ na stan układu krążenia, stąd w przypadku objawów klinicznych uszkodzenia serca konieczność wykluczenia tyreotoksykozy.