CARDIOTHYREOSIS AND HEART FAILURE IN A YOUNG MAN WITH GRAVES’ DISEASE

SUMMARY:
It is presented a case of a young male subject diagnosed in emergency room with severe cardiothyreosis and thyroid goiter. During the hospitalization Graves’ disease is confirmed with concomitant severe heart failure. In this paper are presented clinical and paraclinical findings, diagnostic and treatment management, etiological aspects and case particularities.

Keywords: cardiothyreosis, heart failure, Graves’ disease

INTRODUCTION
Effects of hyperthyroidism on the cardiovascular system and especially on heart manifest even at small changes in serum thyroid hormones. In the context of cardiothyreosis while supraventricular arrhythmias (especially atrial fibrillation) and unmasked ischemic heart disease are found more frequently, heart failure rarely occurs and denotes a severe pathological heart condition or intense and prolonged effect of thyroid hormones.

CASE PRESENTATION
Male patient, 26 years old, currently unemployed, former undertaker, without prior cardiovascular disorders, presented himself in the emergency room of County Emergency Hospital Timisoara for malaise, rapid palpitations for about a month, small and medium effort dyspnea during last two weeks, accentuated in the last 3 days, 12h fever, sweats, significant weight loss. On physical examination we have found warm and wet skin, mild exophthalmia, peripheral tremor of extremities, enlarged thyroid gland, tachyarrhythmic heart sounds with heart rate 210/min, grade 3 systolic murmur in mitral focus, blood pressure of 140/80 mmHg; turgid jugular veins, decreased vesicular murmur basal in right pulmonary area, also liver was sensitive and palpable 4 cm below the costal margin and lower splenic pole was palpable.

Electrocardiogram at presentation in emergency room showed atrial fibrillation with rapid heart rate (Fig.1).

The first biological investigations revealed mild normocytic normochromic anemia (Hb=10.5 g/dl), a slightly increased CKMB = 26 U/l, but normal troponin (Tnl = 0.008 ng/ml) and mild cholestasis (total bilirubin =2.38 mg/dl). At chest radiography we noted global cardiomegaly and mild central pulmonary stasis.

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Based on history, objective clinical examination and primary emergency investigations we established the following diagnosis:

- Recent atrial fibrillation with rapid heart rate. Heart failure NYHA class III. Suspicion of Graves’ disease with cardiothyreosis.

The determination of thyroid hormone serum concentrations showed the following: FT3 > 35 pmol/l (normal values: 3.39-6.80 pmol/l), FT4 > 90 pmol/l (normal values: 10.55-22.77 pmol/l), TSH < 0.015 mU/L (normal values ranged between 0.27-4.20 mU/L), antiTPO antibodies 230 IU/mL (normal <12 IU/mL), anti TSH receptor antibodies: 25.2 IU/mL (normal <1.5 IU/mL).

Echocardiography has brought additional elements of subsequent cardiac disturbance: dilation of the four chambers with right ventricle of 3.6 cm, left ventricle end-diastolic diameter of 6.8 cm, left ventricle end-systolic diameter of 5.5 cm, left atrium of 6.3 cm and right atrium of 5.2 cm, a significant systolic dysfunction with LVEF=34%, severe mitral regurgitation, tricuspid regurgitation grade II, moderate pulmonary hypertension, without pericardial fluid (Fig.3,4,5,6); also at ultrasound we found pleural fluid in small amount in right side, hepatomegaly and splenomegaly (14 cm in length).

Thyroid ultrasound confirmed a diffuse goiter (thyroid volume of 47.2 ml) with marked hypoechochogenic homogeneous appearance of parenchyma and intense, diffuse vascularization at Doppler ultrasound (Fig.7)

The final diagnostic was:

Fig. 3. Echocardiography (four chambers view, Color Doppler): severe mitral regurgitation

Fig. 4. Echocardiography (four chambers view, 2D mode): dilation of the four cardiac chambers suggesting dilated cardiomyopathy

Fig. 5. Echocardiography (four chambers view, continuous doppler): velocity and gradient of mitral regurgitation
We initiated treatment with antithyroid drug (Thyrozol 40 mg/day), beta-blocker (metoprolol in progressively increasing doses), diuretic, injectable then oral anticoagulant. The patient evolution during hospitalization was favourable with improvement of symptoms, hemodynamic control, INR control.

Given the large size of the heart dimensions we considered also other etiologies such as tachycardiomyopathy, because of undefined onset of arrhythmia, a preexisting (non)familial dilated cardiomyopathy, a dilated cardiomyopathy. We have taken into account a postmiocarditis viral etiology, ethanol or drug abuse, infectious etiology. Markers of B, C hepatitis and HIV serology were negative, full blood count analysis, erythrocytes sedimentation rate and inflammation markers were normal. Additional immunological dosing and cardiac MRI could not be performed. Hepato-splenomegaly and association of cholestatic syndrome could be an expression of the liver ischemia in the context of heart failure or a chronic liver preexisting pathology.

**DISCUSSION**

The most important hemodynamic effects of the thyroid hormones include peripheral vasodilation, increased heart rate, increased cardiac and stroke output, increased myocardial contractility and improved ventricular relaxation.

Tachycardiomyopathy occurs in the context of persistent supraventricular tachyarrhythmia (atrial fibrillation or atrial tachycardia) and is characterized by persistent ventricular dysfunction. Severity of the heart failure depends on the duration of arrhythmia and the heart rate and improves after controlling the frequency and/or heart rate, the duration ranging from several days to several months (1).

Graves’ disease, the most common cause of hyperthyroidism, is an autoimmune thyroid disorder which often affects middle-aged women.

Atrial fibrillation occurs in about 10 % of cases of Graves’ disease, especially in young people. Embolic risk
is reduced to this category (< 2 % / year) and it is recommended only antiplatelet therapy. In this case, the significant systolic dysfunction and associated cardiomegaly imposed the anticoagulation treatment. Excepting the cases with hemodynamic instability, the conversion to sinus rhythm it is not recommended as the risk of recurrence lasts until the thyroid function returns to normal. The euthyroidism is restored usually in few (3-4) months of antithyroid treatment. Restoring euthyroid status leads to spontaneous conversion to sinus rhythm in about 6-12 weeks to more than 50 % of patients newly diagnosed with atrial fibrillation (3), the main contributing factor being the duration of arrhythmia, patient age and the absence of cardiac disease. In patients who do not recover the sinus rhythm in more than four months after restoring the euthyroid status cardioversion may attempted if the necessary CHA2 DS-VAS 2C criteria are met.

Heart failure in a patient with hyperthyroidism per primam is the expression of overt increased cardiac output but often unmask a latent cardiopathy, most frequently coronary heart disease. Other cardiac failure mechanisms are: the shortening of diastole by loss of the atrial pump when atrial fibrillation occurs, myocardial structural changes with the appearance of diastolic dysfunction, deficit between demand and myocardial oxygen consumption, ventricular systolic dysfunction by direct toxic action of excess thyroid hormones on cardiac myocytes.(2). The recommended treatment is classic, primarily beta -blockers, but also ACE inhibitors, diuretics, digitalis are used in severe systolic dysfunction and lack of adequate control of heart rate. The most severe complication of thyrotoxicosis is represented by thyroid storm, characterized by impairment of general health, associating fever, nausea, vomiting, diarrhea, dehydration and coma appearance.

CASE CHARACTERISTICS
- Relatively good clinical tolerance to a very rapid heart rate and a previously damaged heart
- Cardiac dilation associated to Graves’ disease with atrial fibrillation in a young patient, which customizes the therapeutic management and evolution
- Graves’ disease occurrence in a young male subject.

References: