Title: A case report of congestive heart failure in patient with hyperthyroidism

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Introduction: Hyperthyroidism is a common metabolic disorder with cardiovascular manifestations. It can cause classical high-output heart failure because of decreased systemic vascular resistance and increased resting heart rate, left ventricular contractility, blood volume and cardiac output.
Clinical case: We presente the case of a 37-year-old woman followed-up at endocrinology center with clinical features of hyperthyroidism and CHF. The patient was initially admitted to the local hospital with toxic hepatitis of unknown etiology. She had progressive weakness, fatigue, reduced exercise tolerance, shortness of breath, peripheral edema and ascites over the last two months. Patient denied of alcohol and drug abuse. Physical examination showed sinus tachycardia, elevated jugular venous pressure and peripheral edema. Computer tomography revealed right-sided hydrothorax, splenomegaly and ascites. Liver function tests (LFT) showed more than 9 fold increase in transaminases and increased bilirubin. Markers of viral hepatitis were negative. TSH level was 0,1mMe/l, Free T4 = 28,2 pmol/l. After getting results TSH level she was admitted at endocrinology centre. Ultrasound study and scintigraphy demonstrated diffuse toxic goiter. ECG revealed sinus tachycardia with HR 119/min. The transthoracic echocardiography showed normal left ventricular systolic function (EF = 62%, global longitudinal strain = - 20,7%), biatrial enlargement (LA index - 62,5ml/m2, RA index - 59ml/m2), moderate mitral and tricuspid regurgitation, PAH of 50mmHg, grade II diastolic dysfunction.
The patient was started on the beta-blocker with further dose up-titration (Metoprolol tartrate 75mg/ daily), loop diuretics (Torasemide 10 mg/daily) and thyreostatics (Methimazole 30mg/daily). After a month from admission the patient was treated with radioactive iodine therapy.
Over the next three months, the patient fully recovered with complete resolution of ascites and fluid overload, free from dyspnea, fatigue or weakness, normal exercise tolerance and LFT.
Control echocardiography performed after 3 months showed improvement of atrial sizes (LA index – 40ml/m2, RA index – 39ml/m2), resolution of mitral and tricuspid regurgitations, normalized diastolic function and systolic pulmonary arterial pressure.
Conclusion: We showed that this disease can be diagnosed by a detailed history and physical examination and that the TSH level should be checked as a part of the initial laboratory work-up of patients with new-onset CHF. Therefore, based on this case report, we suggest that atrial remodeling and functional mitral and tricuspidal regurgitation secondary to atrial dilatation can be resolved rapidly after antithyroid treatment. Manifestation of hyperthyroidism can be varied and in our patient began with liver failure.