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CASE REPORT

Pulmonary hypertension, heart failure and hyperthyroidism: A case report☆

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Abstract We present a case of acute heart failure as the first manifestation of Graves' disease. It illustrates some of its cardiovascular complications, particularly atrial fibrillation, pulmonary hypertension and heart failure. This case report highlights the importance of considering hyperthyroidism as a cause of idiopathic pulmonary hypertension, and demonstrates the potential reversibility of its complications.

PALAVRAS-CHAVE
Hipertiroidismo;
Hipertensão pulmonar;
Insuficiência cardíaca

Hipertensão pulmonar, insuficiência cardíaca e hipertiroidismo: caso clínico

Resumo Apresenta-se um caso de insuficiência cardíaca aguda como manifestação inicial de doença de Graves, ilustrando-se as suas complicações cardiovasculares: fibrilhação auricular, hipertensão pulmonar e insuficiência cardíaca. O caso mostra como as complicações do hipertiroidismo são potencialmente reversíveis, analisando-se assim a importância da sua inclusão nas causas de hipertensão pulmonar não explicada.

Case report

We present a case of acute heart failure (HF) in a 41-year-old woman who went to the emergency department due to progressive dyspnea. At admission, she was in New York Heart Association (NYHA) class IV, with lower limb edema of around one week's duration. She reported no fever, cough, hemoptysis, chest pain, weight loss or joint pain. The patient, a farmer, had been previously healthy and took no regular

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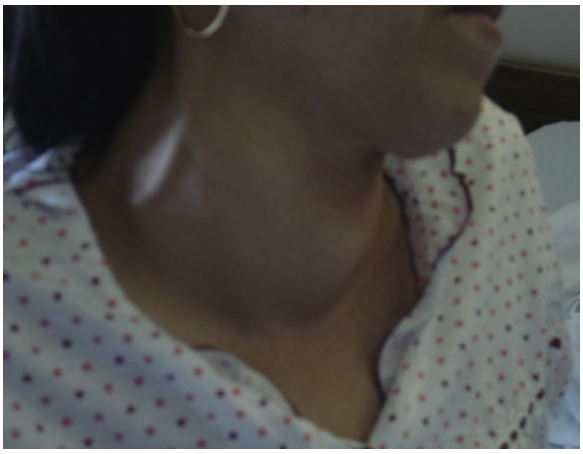

Figure 1 Enlarged thyroid gland.

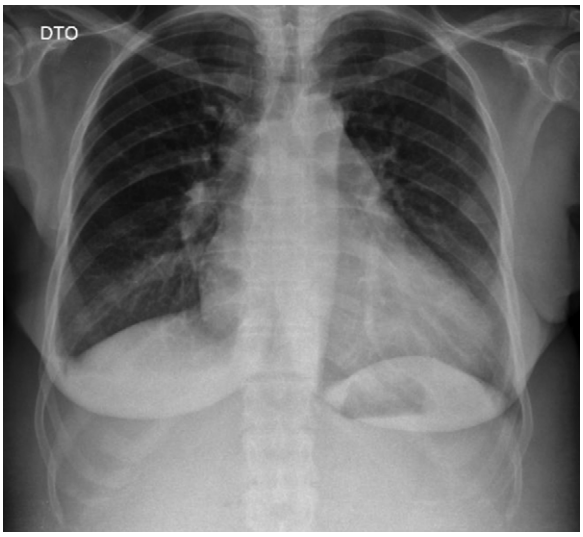

Figure 2 Chest X-ray.

medication, did not smoke or drink alcohol, and had not traveled in the recent past.

Her pulse was irregular, heart rate 115 bpm, blood pressure 147/86 mmHg; she was apyretic and peripheral oxygen saturation was 97% in room air. Physical examination revealed clammy skin and fine hair, exophthalmos and diffuse enlargement of the thyroid gland with thrill but no palpable nodules (Figure 1).

Jugular venous distension of 8 cm at 45° was visible. Cardiac auscultation showed no murmurs, and pulmonary auscultation revealed rales in both lung bases. The patient had painless hepatomegaly 2 cm below the ribcage and bilateral lower limb edema up to the knee.

Initial clinical investigation included laboratory tests, which revealed anemia, negative D-dimers and myocardial necrosis markers, and elevated pro-BNP (Table 1), and the chest X-ray showed increased cardiothoracic index (Figure 2).

The setting was interpreted as acute HF, and etiological investigation included electrocardiography (ECG) and transthoracic echocardiography (TTE). The ECG showed atrial fibrillation (AF) and a mean ventricular rate of 130 bpm, but no ST-T segment abnormalities (Figure 3).

TTE, performed with the patient in AF, showed degenerative changes in valve structures with no significant hemodynamic compromise and mild to moderate tricuspid regurgitation, with pulmonary artery systolic pressure (PASP) estimated at 36 mmHg and right atrial pressure at 20 mmHg. There was mild right chamber dilatation (right atrial area 26 cm^2), with flattening of the interventricular septum secondary to right ventricular (RV) overload (Figure 4), RV systolic function at the lower normal limit (tricuspid annular plane systolic excursion 1.7 cm; S wave 13 cm/s) and mild inferior vena caval dilatation (28 mm) with reduced respiratory variation, all compatible with pulmonary hypertension (PH). There was also moderate left atrial dilatation (area 31 cm^2), with normal left ventricular (LV) dimensions (end-diastolic diameter 52 mm) and ventricular wall thickness. LV systolic function (LVSF) was mildly impaired, with a mean ejection fraction of 46% and increased LV filling pressures (septal E/E′ 21.9).

Table 1 Laboratory test results.

	Result	Reference value
Leukocytes	5200/μl	4000–11 000
Hemoglobin	9.8 g/dl	11.5–16.0
Platelets	161×10^3/μl	$150–450 \times 10^3$
Creatinine	0.4 mg/dl	0.7–1.2
CRP	0.9 mg/dl	<0.5
Pro-BNP	1852.6 pg/ml	<125
Troponin T	0.01 ng/ml	<0.50
D-dimers	0.31 μg/ml	0–0.5

CRP: C-reactive protein.

Findings on physical examination suggested thyroid disease, and thyroid function tests were compatible with hyperthyroidism: free T_4 100 pmol/ml (normal 12–22) and thyroid-stimulating hormone (TSH) <0.005 μU/ml (0.27–4.20). TSH-receptor antibodies (TRAb) were elevated (12.28 U/l; normal <1) and thyroid ultrasound showed diffuse enlargement of the thyroid gland, which was generally heterogeneous in texture with solid hyperechogenic nodules.

The combination of diffuse thyroid enlargement, laboratory evidence of thyrotoxicosis, exophthalmos and positive TRAb led to a diagnosis of Graves' disease (GD), complicated by AF, PH and HF. Treatment was begun with metibasol (30 mg/day), propranolol (60 mg/day), enalapril (5 mg/day), furosemide (60 mg/day) and aspirin (150 mg/day). It was decided to institute antiplatelet therapy since older age is the main risk factor for embolic phenomena in hyperthyroidism. In younger patients, the risks of anticoagulation are greater than its potential benefits.[1]

After six months of medical therapy, and normal thyroid function for three months (TSH 2.13 μU/ml and free T_4 0.881 pmol/l), the patient showed improvement in dyspnea (from NYHA class IV to II) and no peripheral edema. The ECG showed sinus rhythm, with heart rate of 49 bpm. Reassessment by TTE showed PASP of 38 mmHg, RV dimensions at the upper normal limit, and mean ejection fraction of 54%. Despite normal thyroid function, the patient still had an enlarged thyroid gland and positive TRAb, predictive of recurrence.[2] She has poor tolerance of antithyroid drugs and