Case Report

Thyroid storm and myxoedema: two reversible causes of acute heart failure

J Neiva, G Fernandes, D Carvalho, B Ferreira, R Duarte & R Araújo

Abstract

Cardiovascular signs and symptoms can prevail in both hypothyroidism and hyperthyroidism. Case 1: 30-year-old female with acute right heart failure as first manifestation of Graves' disease. It illustrates some of its cardiovascular complications, particularly pulmonary hypertension, tricuspid regurgitation, atrial fibrillation, mitral valve prolapse and mitral regurgitation. Case 2: 83-year-old-man with a severe primary hypothyroidism/ myxoedema crisis presented to emergency department with extreme bradyarrhythmia with hemodynamic impact and heart failure signs, which led to implantation of definitive pacemaker. Thyroid disorders are treatable entities and most cardiovascular changes are reversible. Early diagnosis is mandatory and can avoid severe complications and, eventually, invasive therapeutics interventions. Pulmonary hypertension and acute heart failure, especially in young adults, should be screened for underlying hyperthyroidism.

Keywords

Thyroid storm; myxoedema; right acute heart failure; pulmonary hypertension; extreme braycardia

Key points

- Cardiovascular symptoms and signs are particularly common in decompensated thyroid disease, and can prevail in both hypothyroidism and hyperthyroidism.
- Pulmonary hypertension and acute heart failure, especially in young adults, should be screened for underlying hyperthyroidism.
- An early diagnosis of thyroid failure can avoid severe complications and, eventually, invasive therapeutics interventions.
- Most cardiovascular changes are reversible when the underlying disorder is treated.

Introduction

Thyroid disease is common. Current estimates suggest that it affects 9-15% of adult female population and a smaller percentage of adult males.[1,2] Thyroid hormone is an important regulator of cardiac function and cardiovascular hemodynamics. Cardiovascular symptoms and signs are common in the context of thyroid disease, and can prevail in both hypothyroidism and hyperthyroidism.

Case 1

A 30 year-old woman, without any relevant medical history, presented to the emergency room with few days of evolution of peripheral oedema, palpitations and minimal exertional dyspnoea. The patient described a few months of weight loss, excessive sweating, irritability, hair thinning, amenorrhea, tremor and fatigue.

At admission there was extreme lethargy, tachycardia of 150 beats per minute (bpm), arterial blood pressure (BP) 175/91mmHg, jugular venous distention, systolic murmur III/IV more audible in tricuspid and mitral area, bibasal crepitations, marked bilateral lower limb pitting oedema, and goitre.

Laboratory results showed elevated brain type natriuretic peptide (proBNP) 485.5pg/mL; suppressed thyroid stimulating hormone (TSH); free thyroxine (FT4) higher than 7.70ng/dL, 99pmol/L (0.70-1.48ng/dL, 9-25pmol/L); triiodothyronine 16.6pg/mL, 25.5pmol/L (1.71-3,71pg/mL, 3.5-7,8pmol/L) and positive TSH-receptor antibodies (TRAbs).

She had hypoxaemic respiratory failure (PaO2 6.7 kPa). Electrocardiogram (ECG) demonstrated atrial fibrillation (AF) at 125bpm (figure 1). Transthoracic echocardiography (TTE) showed moderate dilation of the right chambers, a hyperdynamic left ventricle, mitral valve prolapse of segment A2 with apparently moderate, eccentric mitral regurgitation (MR), moderate to severe tricuspid regurgitation (TR), dilated inferior vena cava (32mm) with reduced respiratory variation, and moderate pulmonary hypertension (PHT), with estimated pulmonary artery systolic pressure (PASP) of 55mmHg.

Joana Neiva[1]
MD

Gonçalo Fernandes[2]
MD

Daniela Carvalho[2]
MD

Bruno Ferreira[1]
MD

Rita Duarte[1]
MD

Rui Araújo[2]
MD

[1] Department of Cardiology, Vila Franca de Xira Hospital, Vila Franca de Xira, Portugal
[2] Department of Intensive Medicine, Unidade de Saúde Local de Matosinhos, Matosinhos, Portugal

Correspondence
Joana Boaventura Penteado Neiva
Estrada Carlos Lima Costa Nº2, Povos,
2600-009
Vila Franca de Xira,
Portugal
E-mail: joana_neiva@hotmail.com

© 2018 Rila Publications Ltd.

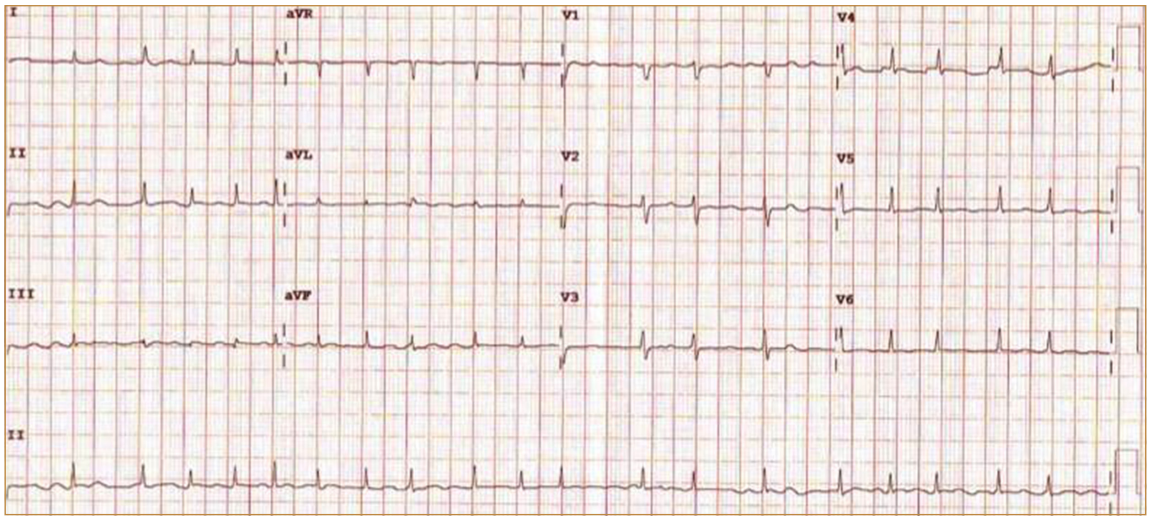


Figure 1. Twelve-lead electrocardiography showed atrial fibrillation of 125 beats per minute.

Thoracic CT angiography confirms the presence of a diffuse goitre and excluded pulmonary thromboembolism. Workup for other possible causes of PHT and right heart failure was negative. She was diagnosed with Graves' disease (GD), meeting the criteria for a thyroid storm. Electrical cardioversion was attempted unsuccessfully. During hospitalisation in intensive care unit, symptomatic therapy with beta-blockers and diuretics, antithyroid therapy with methimazole and adjuvant therapy with glucocorticoids and cholestyramine was initiated. Favourable clinical progress was observed. At 6-months follow-up visit there were no signs of heart failure, there was maintenance of sinus rhythm and significant regression of initial TTE findings, only showing mild PHT, mild to moderate TR and mild MR.



Figure 2. Macroglossia and marked non-pitting oedema in hands.

Case 2

A 83 year-old man, with history of hypertension, dyslipidemia, permanent AF, sleep apnoea and obesity, hospitalised due to fatigue on mild activity. Over the last two months he developed lethargy, asthenia, widespread oedema, weight gain, xerostomia, constipation and generalised osteoarticular pain. On examination he had bradyarrhythmia (25-38bpm), BP 105/57mmHg, dry and pale skin, macroglossia, brittle and thinning hair and generalised non-pitting oedema, more marked in the face, periorbital region, hands and pretibial region (figure 2); bibasilar crepitations; and a palpable thyroid.

Laboratory results revealed a creatinine 203mg/L, creatine kinase 1974U/L, proBNP 2159pg/mL, TSH 140.16mU/L (0.35-5.50mU/L), FT4 0.22ng/dL,2.8pmol/L (0.70-1,48ng/dL, 9-25pmol/L), total cholesterol 247mg/dL, low density lipoprotein cholesterol (c-LDL) 169mg/dL, triglycerides 190mg/dL; positive thyroid peroxidase and thyroglobulin antibodies. ECG demonstrated AF at 36bpm, low voltage QRS and flattened T waves. Chest X-ray revealed right pleural effusion. TTE showed mild circumferential pericardial effusion, mild dilation of left atrium and preserved biventricular systolic function. Thyroid ultrasound detected diffuse enlargement, with multiple nodules. The clinical picture was compatible with severe primary hypothyroidism/ myxoedema crisis. Levothyroxine therapy and supportive care were initiated, with progressive improvement of symptoms. Due to persistence of bradyarrhythmia, with hypotension, a permanent pacemaker was implanted. At a 6-month follow-up visit there was almost complete regression of all hypothyroidism signs, ventricular pacing was less than 20%, and both TSH (3.5mU/L) and lipid profile had normalised.

© 2018 Rila Publications Ltd.

Discussion

Cardiovascular manifestations are the leading cause of death in hyperthyroidism. It is associated with an increased risk of arrhythmias; mostly AF (10-20%).[1,3] Approximately 55-75% return to sinus rhythm within 3-6 months after treatment of thyrotoxic state.[2] Electrical cardioversion is the preferable antiarrhythmic strategy, but should only be attempted when euthyroid. Anticoagulation is controversial; should be considered in elderly and persistent AF. PHT can be presented in up to 47% of hyperthyroid patients.[1,4] Three possible mechanisms have been proposed: increased cardiac output (CO), elevated vascular pulmonary resistance and an autoimmune-mediated endothelial remodelling, since there is proved a correlation between TRAbs and PHT.[2,5] Most cases of PHT recover by the time euthyroidism is reestablished. Methimazole (although not used in the UK) is the preferable therapeutic, since it might cause direct vasodilation of the pulmonary vasculature. Long-standing untreated hyperthyroidism may lead to refractory PHT. TR has occasionally been reported as a predominant manifestation in hyperthyroidism; it may be related to overload of the right heart side, either by increased CO or by PHT. It is more frequent in advanced age and in presence of AF. It is usually reversible if there is correction of the predisposing factors. Predominant or isolated right heart failure is uncommon. It can develop due to direct effects of hormone excess on the right ventricle myocardium leading to 'stunned myocardium', or due to afterload increase; mostly occurs in consequence of PHT. Mitral valve prolapse was found in 6-43% of all hyperthyroidism, more commonly reported in GD.[5,6] Reversible MR has also been reported (13-17%).[4] and a transient dysfunction in the mitral valve apparatus is the most probable cause.[4]

The cardiovascular manifestations of hypothyroidism are not as dramatic as those of thyrotoxicosis and are usually only evident with profound longstanding thyroid failure. It includes bradycardia, conduction abnormalities, pericardial effusion and heart failure, mainly due to diastolic dysfunction. CO can be reduced up to 30-40%.[3] ECG changes include bradycardia, low voltage QRS, prolonged PR and QT intervals and flattened or inverted T waves. Hypothyroidism has also been linked to accelerated atherosclerosis and coronary artery disease.[2,3,6] About 90% have hyperlipidemia.[1,3] LDL fraction is the most affected, since there is both a decrease in LDL-receptor expression and in biliary excretion.[1,2,3] The increase occurs in proportion to TSH levels.[2] Most of these anomalies are generally reversible after return to euthyroidism.

The authors present these two cases due to their rare presentation and severity. PHT and acute heart failure, especially in young adults, should be screened for underlying hyperthyroidism. An early diagnosis of thyroid failure can avoid severe complications and, eventually, invasive therapeutics interventions, such was the implantation of pacemaker in our case. Thyroid disorders are treatable entities and most cardiovascular changes are reversible when the underlying disorder is treated.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Disclosure summary

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics, consent and permissions

Informed consent was obtained from all patients for whom identifying information is included in this article.

References

1. Klein I, Danzi S; Thyroid Disease and the Heart. *Circulation.* 2007;116:1725-1735.
2. Available: https://www.ncbi.nlm.nih.gov/pubmed/17923583
3. Klein I, Danzi S; Thyroid Disease and the Heart. *Current Problems in Cardiology.* 2016; 41(2):65-92.
4. Rhee SS, Pearce E; Sistema endocrino y corazón: una revisión. *Rev Esp Cardiol.* 2011;64(3):220–231.
5. Available: http://www.revespcardiol.org/es/sistema-endocrino-corazon-una-revision/articulo/90000665/
6. Siu CW, Zhang XH, Yung C, *et al;* Hemodynamic Changes in Hyperthyroidism-Related Pulmonary Hypertension: A Prospective Echocardiographic Study. J Clin Endocrinol Metab. 2007; 92(5):1736–1742.
7. Available: https://www.ncbi.nlm.nih.gov/pubmed/17327384
8. Mercé J, Ferrás S, *et al;* Cardiovascular abnormalities in hyperthyroidism: A prospective Doppler echocardiographic study. *The American Journal of Medicine.* 2005;118:126-131.
9. Available: https://www.ncbi.nlm.nih.gov/pubmed/15694895
10. Dillmann WH; Cardiac function in thyroid disease: clinical features and management considerations. *Ann Thorac Surg.* 1993;56:S9-15.
11. Available: http://www.annalsthoracicsurgery.org/article/0003-4975(93)90549-W/fulltext

© 2018 Rila Publications Ltd.