

CASE REPORT

Severe Mitral Regurgitation by Hyperthyroidism in the Absence of Left Ventricular Dilatation

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Introduction

Graves' disease (GD) is an autoimmune disease and the most common cause of thyrotoxicosis,^{1,2} with multisystem involvement, that mainly affects women between 40 and 60 years of age. It is the most prevalent cause of autoimmune hyperthyroidism in pregnancy,^{1,2} and it can be distinguished from gestational thyrotoxicosis due to the presence of diffuse goiter and previous history of hyperthyroidism.³ Clinical hyperthyroidism occurs in 0.2% of pregnant women.⁴

Heart failure (HF) is a rare manifestation of decompensated GD and it represents a diagnostic challenge due to low clinical suspicion of this etiology.¹

We report a case of a pregnant woman with GD who presented thyrotoxicosis and HF with severe mitral dysfunction towards the end of pregnancy, but without ventricular dilatation.

Case Report

A 23 year-old female patient, 37 weeks pregnant, with a history of previous GD, under irregular treatment of Propylthiouracil for four years, was admitted into the emergency department due to dry cough and high fever. She was admitted to the maternity section at the hospital for investigation, where a FT4 level of 6.0 mg/dL and a TSH of 0.011 ng/dL were found. She denied previous surgeries, high blood pressure, diabetes mellitus, rheumatic fever, tuberculosis, illicit drug use, alcoholism or smoking. She progressed to labor and after four days a caesarean was indicated due to acute fetal distress. She was discharged five days after delivery on Tapazole 20 mg per day and

Propranolol 80 mg per day. She was readmitted two days after discharge complaining of tiredness, prostration, orthopnea and paroxysmal nocturnal dyspnea. She was lucid, oriented, emaciated, tachydyspneic with accessory muscle use, pale (2+ / 4+), febrile and with mild tremor of the extremities. Blood pressure of 150 / 80 mmHg; HR 110 bpm; RR 32rpm; axillary temp 101.12°F (38.4°C); exophthalmos and goiter with fibroelastic consistency and no nodulation; regular heart rate, hyperphonic sound, systolic murmur +++/6 in the mitral focus; fine crackles heard at the lung bases, without edema.

Hemoglobin of 7.4 g/dL; 18.800 leukocytes; TSH: 0.034 ng/dL and FT4: 1.92 ng/dL. Chest radiograph showing consolidation of the right hemithorax and pulmonary congestion. A transthoracic echocardiography was performed, which showed preservation of the ventricular cavity size and function, mildly increased biatrial size and severe mitral valve regurgitation, without structural damage, with eccentric jet. Severe mitral regurgitation (MR) was characterized by an eccentric jet, which occupied greater than 40% of the left atrial area. Color Doppler showed prominent holosystolic flow. (Figure 1).

An antibiotic was started, furosemide 80 mg/day, methyl dopa 750 mg/day, propranolol 120 mg/day and the tapazole dose was increased to 30 mg/day. On the eighth day of hospitalization, the patient was asymptomatic and a new echocardiography showed expressive regression of mitral regurgitation (Figure 2). The patient was discharged from the hospital asymptomatic, in NYHA functional class I, and clinically stable.

Discussion

It is reported the case of a pregnant woman, with previous hyperthyroidism without adequate treatment, who presented pulmonary infection and HF associated with severe mitral regurgitation, confirmed

Keywords

Mitral Valve Insufficiency; Hyperthyroidism; Graves' Disease; Pregnant Women.

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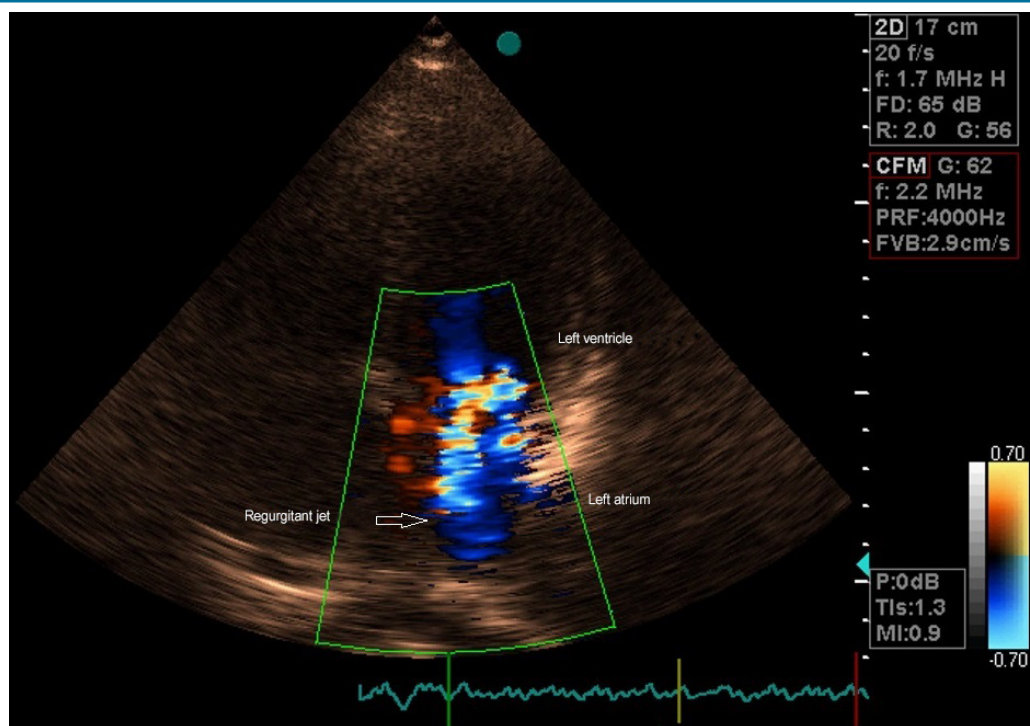


Figure 1 – Initial color echocardiogram showing severe mitral regurgitation (maximum area greater than 8 cm²).

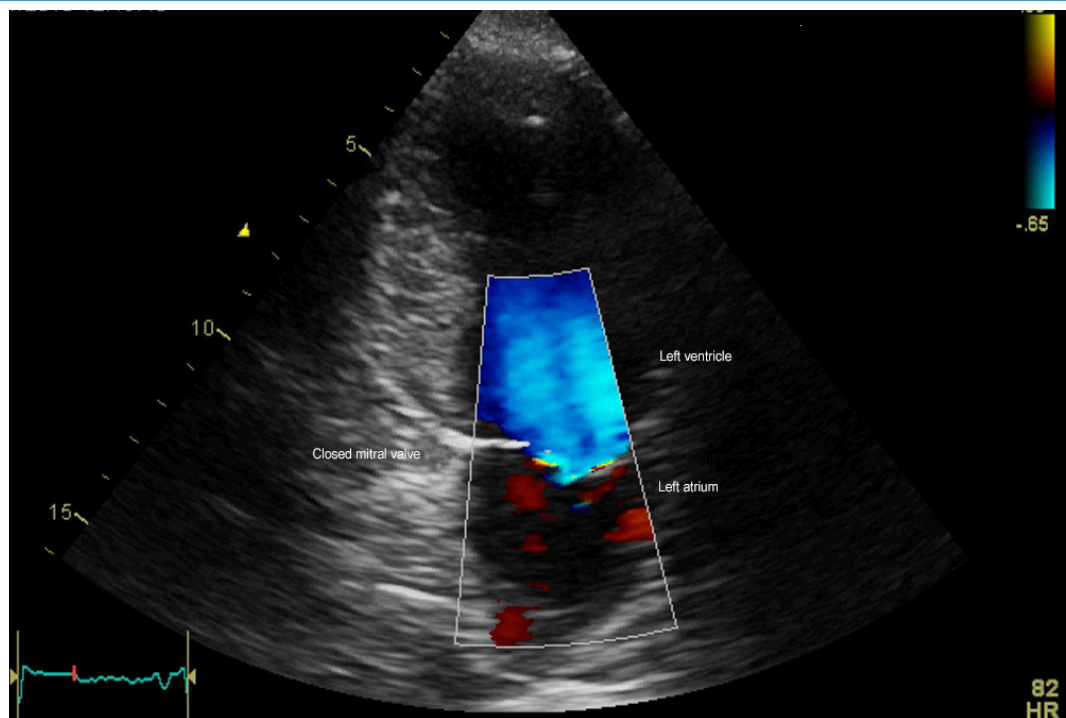


Figure 2 – Color echocardiogram performed one week after the first examination showing the disappearance of the regurgitant jet.

by echocardiography, and who, after treatment optimization, showed complete regression. It is possible that anemia and lung infection have contributed to exacerbate the hemodynamic changes that caused the patient's high output.

The development of heart failure in these patients occurs due to changes in contractility, caused by poor oxygen supply optimization and changes in the myosin isoform expression in the cardiomyocyte.⁵⁻⁷ In addition, there is an increase in blood volume and in the final diastolic pressure and, therefore, increased cardiac work.⁵⁻⁷ The presence of functional RM in GD may occur secondary to an accumulation of glycosaminoglycans or intrinsic papillary muscle dysfunction.⁸

The patient presented elevated FT4 levels. However, the cardiovascular manifestations of hyperthyroidism may occur due to minimal changes in hormone levels and include an increased heart rate at rest, in myocardial contractility, in ventricular muscle mass and a predisposition to atrial arrhythmias.⁹

HR in GD is unusual and, when it occurs, it is often due to high biventricular rate with regular or reduced pulmonary and systemic vascular resistance. It affects mostly patients at extremes of age or those with previous heart disease - conditions not presented by the patient in this case.

MR in GD may be caused by ventricular dilatation. However, it is important to highlight that, unlike the case report written by NG Cravos et al.¹⁰, there was no evidence of such increase in this patient. The cause of MR, in this case, must be associated with an increased synthesis of glycosaminoglycans

of the endocardium or with an intrinsic dysfunction of papillary muscle activity.⁸

In this case, there was a relation between the levels of circulating thyroid hormone and the severity of RM and HR. The use of diuretics and antithyroid agents led to regression of the clinical picture within one week, with the disappearance of mitral regurgitation.

Author contributions

Conception and design of the research: Jorge AJL, Martins WA, Almeida BM. Acquisition of data: Gripp EA, Almeida BM, Figueroa CCRP, Sabino CL. Analysis and interpretation of the data: Jorge AJL, Martins WA. Writing of the manuscript: Jorge AJL, Martins WA, Gripp EA, Almeida BM, Figueroa CCRP, Sabino CL. Critical revision of the manuscript for intellectual content: Jorge AJL, Martins WA, Gripp EA. Supervision / as the major investigator: Jorge AJL.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

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