

When the Valve Doesn't Explain Everything: Secondary Heart Failure in a Young Woman

Cuando la válvula no explica todo: insuficiencia cardíaca secundaria en una mujer joven

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Hyperthyroidism profoundly alters cardiovascular physiology by increasing heart rate, contractility, and stroke volume, expanding circulating volume, and decreasing systemic vascular resistance. Consequently, some patients develop a hyperdynamic state that can progress to heart failure (HF), atrial fibrillation (AF), pulmonary hypertension, and functional valvular regurgitation. (1–5)

High-output heart failure is a rare phenotype in which congestion, elevated filling pressures, and elevated natriuretic peptides may coexist despite a preserved left ventricular ejection fraction (LVEF). (6)

We present the case of a young woman with thyrotoxicosis due to Graves' disease, AF, right-sided HF, and functional valvular heart disease, in whom the integration of cardiac magnetic resonance imaging (CMRI), echocardiography, and computed tomography (CT) angiography allowed us to redefine the etiological interpretation of a heart condition initially considered predominantly structural.

This is a 33-year-old female patient with no conventional cardiovascular risk factors, a history of hyperthyroidism, iron-deficiency anemia, and previous hospitalizations for HF. She was being evaluated for mitral and tricuspid regurgitation, pulmonary hypertension estimated by echocardiography, and an atrial septal defect (ASD).

Gadolinium-enhanced CMR performed on January 29, 2026, showed moderate dilatation of both atria and a severe increase in right ventricular volumes (indexed end-diastolic volume 116 mL/m²), with preserved systolic function, as indicated by a right ventricular ejection fraction (RVEF) of 56% and tricuspid annular plane systolic excursion (TAPSE) of 17 mm. There was moderate to severe tricuspid and mitral re-

gurgitation, moderate left ventricular dilation, LVEF 61%, with no late enhancement. An ASD of approximately 4 mm was identified in the fossa ovalis, with bidirectional flow predominantly left-to-right and a Qp/Qs ratio of 1.18 (Figure 1). The cardiac index was 4.73 L/min/m², consistent with hyperdynamic physiology.

An echocardiogram on June 24 showed LVEF of 59%, a markedly dilated left atrium (70 mL/m²), a right atrium of 39 cm², a pulmonary artery with left-to-right shunt, a dilated right ventricle, an inferior vena cava measuring 22 mm with no inspiratory collapse, an estimated pulmonary systolic pressure of 48 mmHg, moderate mitral regurgitation, and moderate-to-severe tricuspid regurgitation. (Figures 2 and 3)

On July 26, she was admitted due to a new episode of HF decompensation with systemic congestion. During her hospitalization, AF and markedly elevated natriuretic peptides were documented. Endocrinological testing revealed thyroid-stimulating hormone (TSH) <0.01 mIU/L, free T4 5.47 ng/dL, and thyroid-stimulating hormone receptor antibodies (TRAb) >40 IU/L. Thyroid ultrasound showed diffuse enlargement of the gland and a predominantly hypoechoic structure, findings consistent with Graves' disease. A chest CT angiogram performed on July 29 ruled out pulmonary thromboembolism and revealed a moderate right pleural effusion.

Treatment was initiated with methimazole, iodine, glucocorticoids, and monitoring of ventricular response, combined with diuretic therapy for congestion.

The follow-up echocardiogram on July 31, performed in AF at 90 beats per minute, showed preserved LVEF, significant biauricular dilation, a dilated

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right ventricle with mild impairment of function, and a 42-mm tricuspid annulus with moderate-to-severe functional regurgitation. Mitral regurgitation, which had previously been moderate, was now mild. AAS could not be visualized. On that date, the patient was hemodynamically stable, without dyspnea or need for oxygen, and with mild residual signs of congestion. Free thyroxine decreased from 5.47 to 2.9 mIU/L and subsequently to 1.26 mIU/L.

The main diagnostic challenge was to determine whether the marked right ventricular remodeling and valvular regurgitations corresponded to primary structural heart disease or were, at least partially, consequences of a systemic hemodynamic disturbance.

Thyrotoxicosis increases cardiac output due to its positive chronotropic and inotropic effects, a decrease in systemic vascular resistance, and plasma volume expansion. AF and associated anemia can amplify this mechanism. (4,5) The resulting HF does not require impaired LVEF: patients with high-output HF may present with congestion and elevated filling pressures while maintaining preserved left ventricular systolic function. (6)

In this patient, the cardiac index of approximately 4.73 L/min/m², documented six months prior to decompensation, constitutes objective evidence supporting preexisting hyperdynamic physiology. The persistently preserved LVEF, therefore, should not be interpreted as evidence against the diagnosis of HF.

ASD was a relevant etiological differential diagnosis. However, its approximate diameter of 4 mm and Qp/Qs ratio of 1.18 make it unlikely that it alone accounts for the extent of right ventricular remodeling. Likewise, the absence of late enhancement reduced the likelihood of overt cicatricial or infiltrative cardiomyopathy, while CT angiography ruled out pulmonary embolism as the cause of decompensation.

The course of mitral regurgitation is of pathophysiological significance. Its reduction from moderate to mild during hospitalization, in parallel with hormonal control and decongestion, supports a predominantly functional mechanism. The persistence of right ventricular dilation, a 42-mm tricuspid annulus, and moderate-to-severe tricuspid regurgitation, however, suggests a more established right-sided remodeling.

Current recommendations for HF emphasize identifying and correcting etiological and precipitating factors. (1,2) In AF associated with hyperthyroidism, treatment of the thyroid disorder is essential; rate control should be individualized based on hemodynamic status, and the indication for anticoagulation should be determined according to conventional thromboembolic risk. (3)

The combination of markedly positive TRAb, suppressed TSH, very elevated free thyroxine, and diffuse thyroid enlargement points to Graves' disease as the most likely endocrinological mechanism. In this context, it is more appropriate to interpret the presentation as complex HF with hyperdynamic physi-

ology induced by thyrotoxicosis, associated with AF and right ventricular remodeling, rather than as HF with primarily preserved ejection fraction or isolated valvular disease.

The main limitation of this report is the absence of invasive hemodynamic evaluation and follow-up after the sustained restoration of euthyroidism. A delayed echocardiographic reevaluation will allow us to determine what proportion of the right ventricular remodeling and tricuspid regurgitation is reversible and whether the ASD retains hemodynamic significance.

Fig. 1. Cardiac magnetic resonance imaging. Four-chamber view showing biauricular dilation with an atrial septal defect.

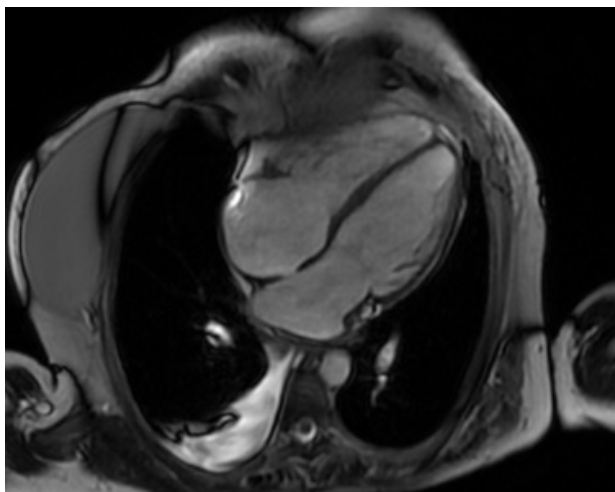
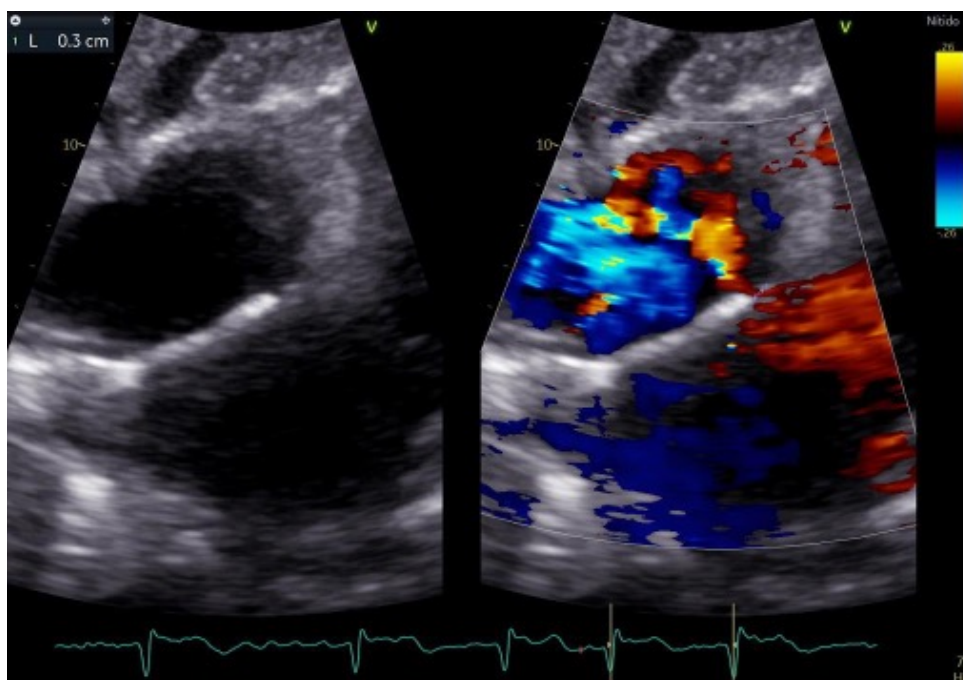


Fig. 2. Two-dimensional transthoracic echocardiogram, left parasternal short-axis view. Septal flattening due to right ventricular overload, with right ventricular dilation.



Fig. 2. Two-dimensional echocardiogram (left) with color Doppler (right). Subxiphoid view showing flow from the left to the right atrium through the atrial septal defect.



In conclusion, thyrotoxicosis can cause severe HF even with preserved LVEF. The demonstration of an elevated cardiac index, along with AF, right ventricular remodeling, functional valvular heart disease, and Graves' thyrotoxicosis, allowed us to reconsider a heart condition initially attributed primarily to valvular disease.

The main clinical message is that, in the face of unexplained heart failure in a young patient, the systematic search for reversible causes and the integration of serial cardiovascular imaging can substantially alter the diagnosis and prevent premature structural interventions.

Conflicts of interest

None declared.

(See conflicts of interest forms on the website).

Ethical considerations

Not applicable

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