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Mitral Chordae Rupture Associated with Thyrotoxic Crisis: An Assessment by 3D Echocardiography

Ruptura de Cuerda Mitral Asociada a Crisis Tirotóxica: Una Evaluación por Ecocardiograma 3D

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SUMMARY

We report a case of a mitral chordaea rupture in a young adult patient presenting with fever, new cardiac murmur and respiratory distress, where the hypothesis of infective endocarditis was primarily selected as the main clinical concern. However, the clinical course in the absence of major criteria for endocarditis, the concomitant presence of a thyrotoxic state and no other echocardiographic detected structural alterations, strengthened the alternative hypothesis of primary rupture due to the hyperkinetic state. These findings were thoroughly documented and confirmed by 3D-echocardiography. The literature review describes that, although mild to moderate mitral regurgitation is highly prevalent in thyrotoxicosis, mitral rupture, in the absence of a previous structural abnormality, is a rare unexpected finding.

Descriptors: Chordae Tendineae; Heart Valve Diseases; Rthyroid Crisis; Echocardiography, Three-Dimensional

RESUMEN

Relatamos el caso de ruptura de cuerda mitral en un paciente adulto joven, con presentación clínica de fiebre, nuevo soplo cardíaco y disfunción respiratoria, condición en que la hipótesis de endocarditis infecciosa es obligatoria. Sin embargo, el curso clínico, con ausencia de criterios mayores para endocarditis, la presencia concomitante de crisis tirotóxica y una válvula mitral sin otras alteraciones estructurales ecocardiográficas, refuerza la hipótesis alternativa de ruptura de cuerda asociada al estado hiperquinético. Los hallazgos fueron apropiadamente documentados y confirmados por ecocardiograma 3D. La revisión de la literatura existente describe que, no obstante la presencia de regurgitación mitral leve a moderada sea altamente prevalente en la tirotoxicosis, la ruptura mitral en la ausencia de otras anormalidades estructurales es un hallazgo raro e inesperado.

Descriptores: Cuerdas Tendinosas, Enfermedad de las Válvulas Cardíacas, Crisis Tiroidea, Ecocardiografía Tridimensional

INTRODUCTION

The thyrotoxic crisis is a hyperdynamic state associated with high mortality rates (20% to 30%)^{1.} The exacerbated effect of triiodothyronine (T3) on the heart and cardiovascular system leads to decreased systemic vascular resistance and increases heart rate at rest, contraction of the left ventricular, systolic

volume, and, consequently, the cardiac output. The most prevalent cardiovascular signs and symptoms in this condition are palpitations, exercise intolerance, and dyspnea. Manifestations such as angina, peripheral edema, and heart failure are less common^{2.} The emergence of an intense heart murmur is unusual and requires the exclusion of other medical conditions with similar

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manifestations, such as infective endocarditis. Echocardiography is a fundamental test in the differential diagnosis.

CASE REPORT

Woman, 24 years, with history of untreated hyperthyroidism, presented with fever, dyspnea, and hemoptysis. The initial presumptive diagnosis was community-acquired pneumonia. The patient evolved with rapid deterioration of her clinical status, respiratory failure, and required mechanical ventilation.

The initial physical examination showed a blood pressure of 140/90 mmHg, heart rate of 130 beats / minute, axillary temperature of 37.3 °C, mild mitral mesosystolic murmur (Grade II / VI), splenomegaly, and patent increased thyroid. Chest radiography showed diffuse pulmonary infiltrates and the initial laboratory evaluation revealed severe anemia (hemoglobin = 7.4 mg / dL), leukopenia (Total WBC = $2.940 / \text{mm}^3$), and hyperthyroidism (TSH < $0.010 \mu \text{UI}$ / mL [reference value = $0.35 \cdot 5.5 \mu \text{UI} / \text{mL}$] and free-T4 = 6.7 ng / dL [reference value = $0.89 \cdot 1.76 \text{ ng} / \text{dL}$]). Samples were collected for blood culture and sputum culture and antimicrobial treatment was implemented with ceftriaxone and azithromycin.

In further diagnostic investigation we performed a chest computerized tomography, which showed an increased thickness of the interlobular septum and interstitial infiltrate in the superior lobes.

During the clinical course, the patient presented with persistent fever (40.3 $^{\circ}$ C), high blood pressure (178/80 mmHg), and tachycardia (120 bpm), which were attributed to thyrotoxic crisis. At that time, heart auscultation revealed a new intense holosystolic mitral murmur (Grade IV / VI). The ECG showed sinus tachycardia and chest radiography remained with pulmonary congestion.

Transthoracic echocardiography revealed a left ventricle of normal size (left ventricular diastolic diameter = 3.0 cm / m²) and hyperdynamic with moderate mitral regurgitation with posteriorly directed jet. Conventional two-dimensional (2D) imaging suggested the presence of flail in the anterior leaflet of mitral valve (Figure I [Longitudinal parasternal 2D.jpg], Figure 2 [2D-4 Chambers.jpg 4]). The left atrium and right cavities had normal dimensions. A functional tricuspid regurgitation jet allowed estimating an increased systolic pressure of the

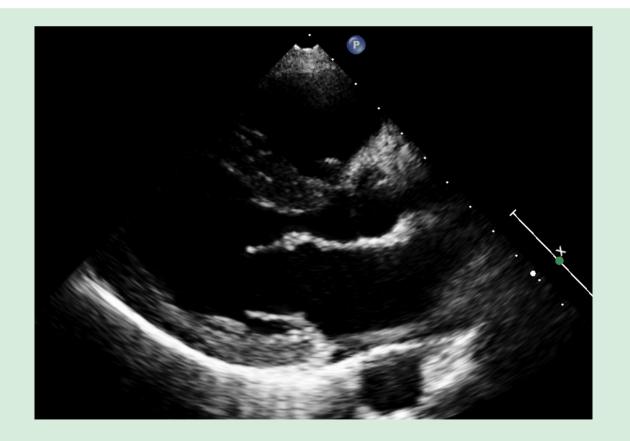


Figure 1: AE: Left Atrium, Ao: aorta, VE: left ventricle, VD: Right Ventricle, VM: Mitral Valve. 2D Echocardiography with parasternal longitudinal image (diastole) showing a structurally normal mitral valve with thin leaflets.



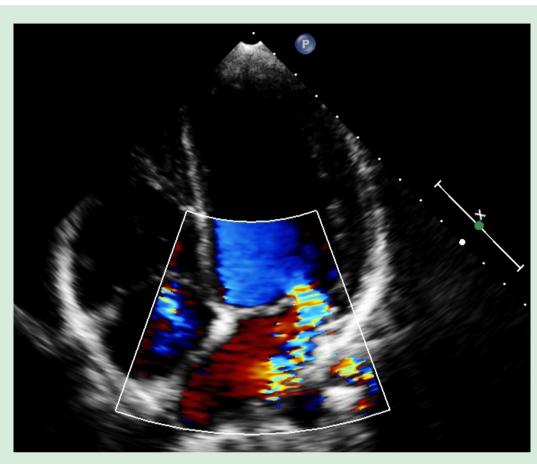


Figure 2: RM: mitral regurgitation, RT: Tricuspid regurgitation, VE: Left Ventricle, VD: Right Ventricle. 2D Echocardiography in apical 4-chamber view showing the eccentric jet of mitral regurgitation.

pulmonary artery of approximately 60mmHg. No images suggesting valve vegetations were identified. These results were confirmed by transesophageal echocardiography performed in the following day.

Treatment for thyrotoxic crises was started with timazol, lugol, hydrocortisone and esmolol. Gram-negative rods have grown on sputum cultures, and serial blood cultures were negative. The initial failure of weaning from mechanical ventilation was attributed to pulmonary congestion, being successfully performed after optimizing diuretics and vasodilators.

Three days after weaning from mechanical ventilation, with clinical improvement and reduction of hyperdynamic state, a transthoracic echocardiography was requested. The examination conducted to reassess the impact of mitral valve injury continued to show moderate mitral regurgitation (vena contracta = 0.38 cm; regurgitation volume = 31 ml), no increase of cardiac chambers, and reduction of systolic pulmonary artery pressure to 38 mmHg. At this time, images obtained by three-dimensional echocardiography (3D) clearly identified the flail in the anterior

leaflet of the mitral valve (segment A2) as a result of a rupture limited to the primary chordae tendineae, and absence of other structural abnormalities in the valve (Figure 3 [static image verified by left atrium].

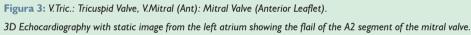
The patient evolved favorably and a strategy of conservative treatment for mitral valve was initially chosen under intensive clinical follow-up. The decision on surgical treatment was postponed to a time after the stabilization of thyroid disease.

DISCUSSION

This case report documents the rupture of mitral valve chordate in the absence of other structural valve abnormalities in a young adult patient during thyrotoxic crises. To our knowledge, this disorder has not been previously reported and must be considered in patients with a marked hyperdynamic status which evolves to respiratory instability. Furthermore, it reinforces the importance of close monitoring of relevant changes in the clinical status (such as the emergence of new murmurs), and the 3D echocardiogram role as tool to support the differential diagnostic.







We attribute the rupture of the mitral valve chordae to thyrotoxic crises, in which some mechanisms may be speculated. Firstly, toxic concentrations of thyroid hormone could lead to dysfunction of papillary muscle, leading to excessive stretching and possible destruction of muscle fibers ³. Secondly, the hyperdynamic status with increased heart output and consequent increased systolic pressure of the left ventricle could predispose to mitral chordae rupture as described by Roberts et al.⁴ A third mechanism is the anatomical predisposition to rupture due to mitral valve prolapse as described by Aronson et al.^{5.} Mercé et al.^{6,} studying patients with hyperthyroidism have found a 13% prevalence of moderate mitral regurgitation, however, none of these cases described by them showed mitral valve prolapse or other structural abnormalities. Patients in this study were assessed only by 2D echocardiography, and subtle abnormalities in mitral leaflets may have not been identified. Our patient underwent a broader and careful assessment by 3D echocardiography and transesophageal echocardiography, which showed no other concomitant structural changes in the mitral valve.

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