

## Atrial Fibrillation and Tricuspid Regurgitation as Late Presentations of Contusio Cordis

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### Introduction

Tricuspid regurgitation (TR) is most often seen in association with mitral valve disease and is called “secondary tricuspid regurgitation.” Other etiologies, such as infective endocarditis, Ebstein’s anomaly, endomyocardial fibrosis, carcinoid disease and closed chest trauma are uncommon.<sup>1</sup>

Tricuspid valve lesions resulting from closed chest trauma are rare and often underdiagnosed. The term “contusio cordis” is represented by hemorrhagic or fibrotic lesion of the cardiac myocyte, secondary to closed chest trauma. Commotio cordis, however, involves cardiac electromechanical disorder. Most often, it leads to ventricular fibrillation, coinciding with a critical time in the cardiac cycle — about 20 ms before the peak T wave.<sup>2</sup> Both presentations are usually fatal.<sup>3</sup> We report the case of a patient diagnosed with TR and atrial fibrillation (AF) secondary to contusio cordis, but with an unusual insidious late clinical course diagnosed 11 years after the event.

### Case Report

A 42-year-old male patient, from São Paulo, admitted to the emergency department reporting palpitation and dyspnea on medium exertion. The patient was in New York Heart Association (NYHA) functional class III. No history of myocardial ischemia, infective endocarditis, rheumatic fever, or other rare conditions, such as Ebstein’s anomaly, endomyocardial fibrosis and carcinoid disease. On the other hand, his clinical history included a chest trauma due to an automobile accident 11 years prior. He had been asymptomatic since then, when he started, 3 years before, fatigue and dyspnea, with worsening in the past 8 months.

At the examination, heart rhythm was irregular and holosystolic murmur in the tricuspid valve had 3+/6+ intensity with a positive Rivero-Carvallo maneuver. Hepatomegaly and lower limb edema were present.

Chest X-ray had increased cardiac area at the expense of the right chambers. Electrocardiography showed irregular rhythm and absence of P waves, evidencing AF and right bundle branch block (Figure 1).

### Keywords

Tricuspid Valve Insufficiency/surgery; Atrial Fibrillation; Myocardial Contusions; Commotio Cordis; Arrhythmias Cardiac.

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Two-dimensional echocardiography showing enlarged left atrium (LA) with diastolic diameter of 43 mm and left ventricular (LV) end diastolic diameter of 58 mm. LV ejection fraction 47%. Severe TR secondary to chordal rupture related to the anterior cusp. Traction of the other cusps secondary to right ventricular remodeling, generating severe tricuspid reflux. The ring diameter was 60 mm (Figure 2). LV systolic dysfunction and right ventricle (RV) dysfunction of discrete and moderate degrees, respectively. Systolic pulmonary artery pressure 38 mmHg.

Cardiac magnetic resonance imaging was also performed to evaluate the RV. Imaging showed RV with increased dimensions, with moderate systolic dysfunction (ejection fraction of 34%). LV was increased, with systolic function severely reduced (ejection fraction 30%). Areas of late enhancement were absent, suggesting fibrosis (Figure 3).

With the clinical diagnosis, later confirmed in the surgery, of TR secondary to contusio cordis, the patient was submitted to interventional treatment by median sternotomy. In the intraoperative period, prolapse of the anterior leaflet was confirmed with chordal rupture and tricuspid annulus dilation, causing major regurgitation. Valvuloplasty unsuccessfully tried to attach the leaflet. Tricuspid valve replacement with biological prosthesis was then chosen. The patient progressed postoperatively uneventfully, with improvement of tricuspid regurgitation (Figure 4). On the seventh postoperative day, the patient was discharged with NYHA functional class I.

### Discussion

Cardiac lesions resulting from chest trauma are varied, such as cardiac contusion, free wall rupture, septal rupture and valve lesion.<sup>1</sup> Few cases of traumatic heart lesions were described in the literature,<sup>4</sup> especially when it comes to AF — only two cases were found in our review. Traumatic heart injuries can occur by a simple fall, impact on the chest or after a car accident. They may be benign, with a simple myocardial contusion, or lead to major destruction of intracardiac structures, generating hemodynamic instability, with imminent risk of death.<sup>5</sup> Traumatic TR is a rare occurrence, but because of its anterior location, it remains the most frequently reported valve lesion after blunt chest trauma.<sup>4</sup>

Commotio cordis involves high-impact injuries, such as in American football, martial fights and car accidents. It results from blunt trauma in the precordial region. When it occurs about 20 ms before peak T wave, it may result in ventricular fibrillation.<sup>2</sup> The occurrence of AF is rare. Chest impact during the vulnerable period of atrial repolarization increases RA pressure and triggers premature atrial depolarization with AF. There is induction of the so-called heterogeneity of atrial repolarization, which can perpetuate and sustain AF.<sup>4</sup>

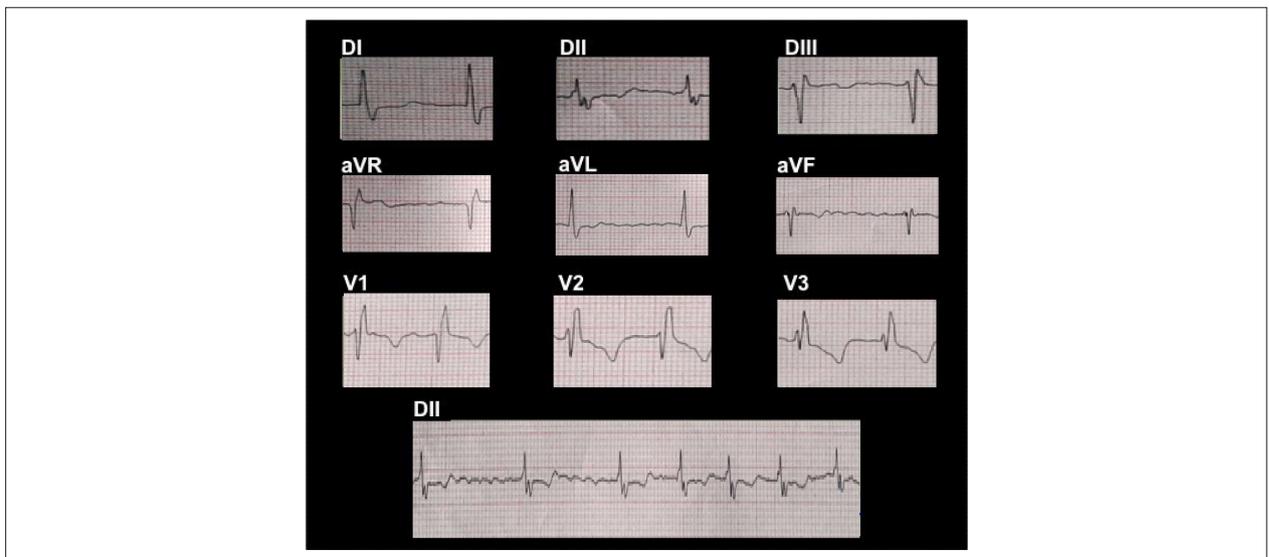


Figure 1 – Electrocardiography with atrial fibrillation associated with right bundle branch block.

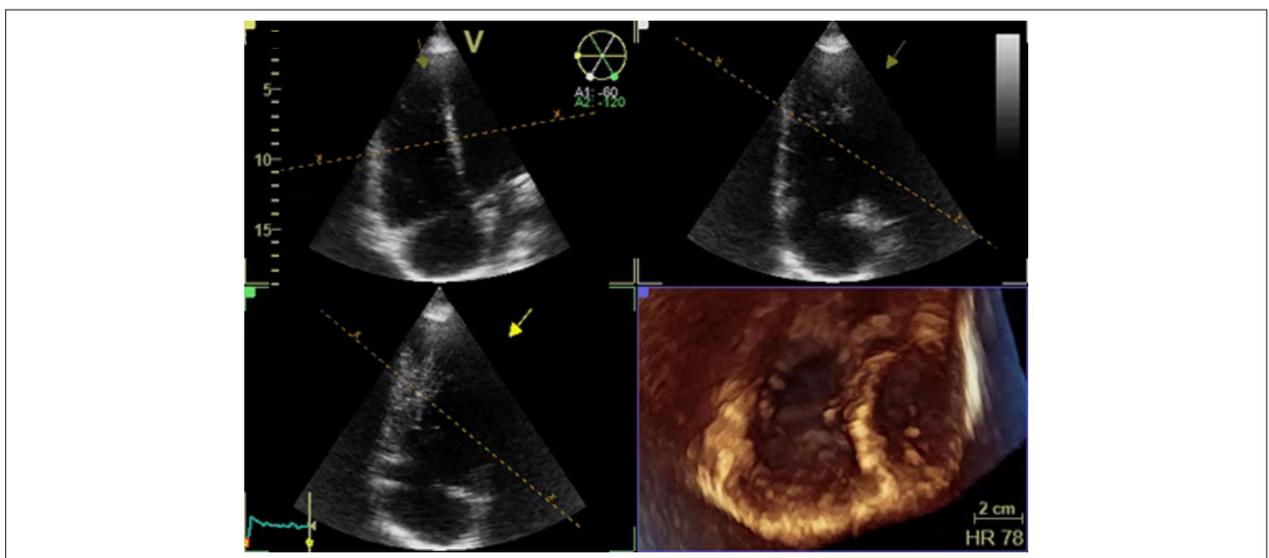


Figure 2 – Two-dimensional and three-dimensional transthoracic echocardiography reveal rupture of the anterior cusp of the tricuspid valve.

Traumatic TR generates clinical manifestations correlated with the severity of the damaged structures, varying from acute heart failure to slow and progressive clinical repercussion. It is usually hemodynamically well tolerated and its incidence is underestimated. The mean time between the trauma and the valve surgery is variable. It may be 17 years approximately.<sup>4</sup> In this case, in fact, the patient presented insidious clinical evolution, and the diagnosis of post-traumatic tricuspid valve injury was late – 11 years after the traumatic event.

The mechanism of tricuspid valve injury, associated with contusio cordis, is secondary to severe and sudden impact during the final diastole. Due to the anatomical position, the RV and the tricuspid valve are vulnerable to injuries after frontal impacts. High hydrostatic pressure in RV increases this vulnerability during diastole. TR is caused by great tension in

both the anterior cusp and in the papillary muscle. In most cases, its progression is insidious and well tolerated.<sup>6</sup>

The most frequently reported lesion is chordal rupture followed by ruptures of the anterior papillary muscle and valvular leaflets.<sup>7</sup> After the chordal rupture, the valve function is usually preserved, with subacute or even late clinical presentation. Rupture of the papillary muscle, in turn, determines immediate and severe hemodynamic instability with acute presentation.<sup>8</sup>

Information on the current clinical course of conservative treatment of traumatic TR is scarce in the literature. There have been successful reports, perhaps explained by remodeling and healing of papillary muscles, with partial resolution of the valvopathy. However, because they are isolated cases, they are insufficient to indicate conservative

## Case Report

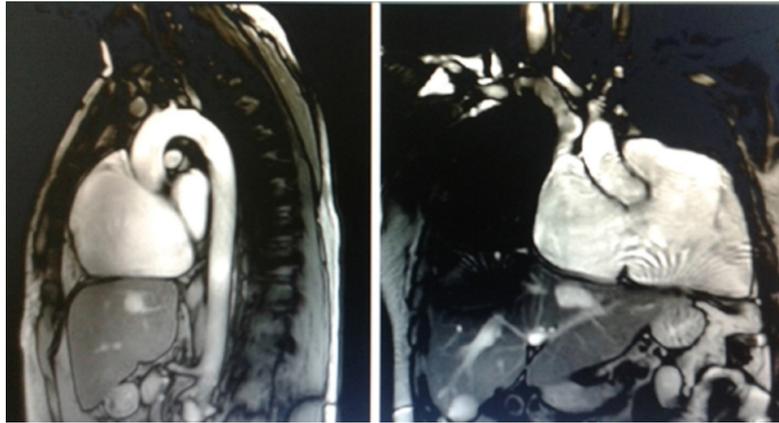


Figure 3 – Cardiac magnetic resonance imaging showing right ventricle with increased dimensions.

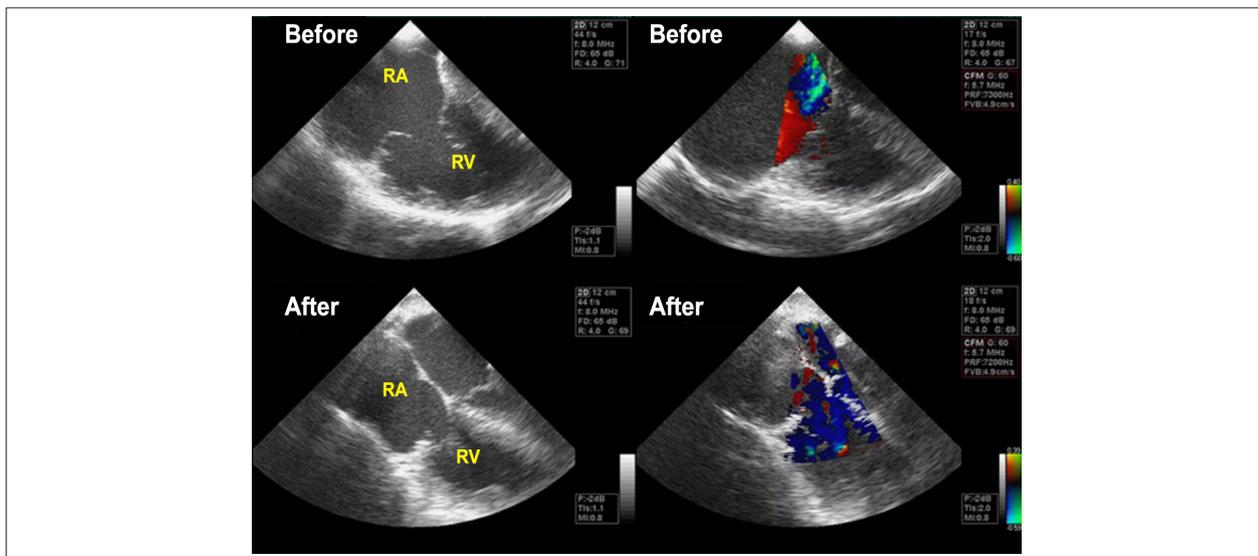


Figure 4 – Intraoperative echocardiography improved tricuspid regurgitation (before and after). RA: right atrium; RV: right ventricle.

management.<sup>6</sup> Unfavorable outcome with conservative treatment, compared to surgical intervention, was confirmed by the high rates of events in the natural history from the time of diagnosis. According to previous reports in the literature, if tricuspid regurgitation is severe, the prognosis is poor even in asymptomatic patients. Increased RV in the presence of tricuspid regurgitation is also predictive of poor prognosis. Therefore, intervention is recommended.<sup>9</sup>

The ideal timing for this in traumatic TR is controversial. Traditional indication for a surgical approach is symptomatic heart failure. Its early realization, after identifying the initial symptoms, could prevent further cardiac remodeling with preservation of cardiac function.<sup>9</sup>

Surgical treatment for tricuspid lesions includes valve repair and replacement. Valve repair has been proven effective and, when possible, is the treatment of choice. Different techniques are available, such as chordae replacement or leaflet re-suture.<sup>9</sup> Early diagnosis and surgical treatment may facilitate the valve repair and prevent RV function deterioration. However, the

possibility of valve repair exists in the minority of cases, either due to the presence of excessive secondary annular fibrosis or dilatation.<sup>10</sup> In the case reported, valvuloplasty was unsuccessful. The patient was then submitted to tricuspid valve replacement with biological prosthesis. After 7 days, the patient presented NYHA functional class I.

### Authors' contributions

Research creation and design: Almeida MM, Alban NRA, Aguay GJV; Data acquisition: Almeida MM, Alban NRA, Aguay GJV; Data analysis and interpretation: Almeida MM; Manuscript writing: Almeida MM, Alban NRA, Aguay GJV; Critical revision of the manuscript as for important intellectual content: Almeida MM.

### Potential Conflicts of Interest

There are no relevant conflicts of interest.

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### Academic Association

This study is not associated with any graduate programs.

## References

1. Nelson M, Wells G. A case of traumatic tricuspid valve regurgitation caused by blunt chest trauma. *J Am Soc Echocardiogr.* 2007;20(2):198,e4-5.
2. Maron BJ, Estes NA 3rd. Commotio cordis. *N Engl J Med.* 2010;362(10):917-27.
3. Alsheikh-Ali AA, Madias C, Supran S, Link MS. Marked variability in susceptibility to ventricular fibrillation in an experimental Commotio Cordis Model. *Circulation.* 2010;122(24):2499-504.
4. Ghose RR. Commotio cordis and atrial fibrillation. *N Z Med J.* 1998;111(1075):394.
5. Krasna MJ, Flancbaum L. Blunt cardiac trauma: clinical manifestations and management. *Semin Thorac Cardiovasc Surg.* 1992;4(3):195-202.
6. Tütün U, Aksöyek A, Parlar AL, Cobanoğlu A. Post-traumatic tricuspid insufficiency: a case report. *Ulus Travma Acil Cerrahi Derg.* 2001;17(6):563-6.
7. Link MS. Commotio cordis: ventricular fibrillation triggered by chest impact-induced abnormalities in repolarization. *Circ Arrhythm Electrophysiol.* 2012;5(2):425-32.
8. Madias C, Maron BJ, Weinstock J, Estes NA 3rd, Link MS. Commotio cordis--sudden cardiac death with chest wall impact. *J Cardiovasc Electrophysiol.* 2007;18(1):115-22.
9. Bolton JW. Traumatic tricuspid valve injury: leaflet resuspension repair. *Ann Thorac Surg.* 1996;61(2):721-2.
10. Schuster L, Graf S, Klaar U, Seitelberger R, Mundigler G, Binder T. Heterogeneity of traumatic injury of the tricuspid valve: a report of four cases. *Wien Klin Wochenschr.* 2008;120(15-16):499-503.