Tricuspid valve lesions caused by penetrating chest trauma are rare and often underdiagnosed. The objective of this report is to describe a case of severe tricuspid insufficiency secondary to a knifing incident with an insidious evolution, diagnosed 19 years after the incident. The case emphasizes the importance of adequate follow-up of patients that are victims of penetrating chest trauma for a long period after the injury, to detect possible late complications of the trauma.

Introduction

Tricuspid valve lesions caused by penetrating chest trauma are rare. The traumatic rupture of the tricuspid valve is well-tolerated hemodynamically and thus, the number of reported cases is often underestimated. The diagnosis is made many months, or even years, after the trauma.

The following report describes a case of traumatic tricuspid insufficiency secondary to a knife incident.

Case Report

A 57-year-old male patient with chest trauma caused by a knife incident that had occurred 19 years before was referred to cardiological assessment with a diagnosis of arterial hypertension. At the time of the chest trauma, he was submitted to hemothorax and hemopericardium drainage and surgical correction of the myocardial laceration.

Currently, the patient has presented dyspnea on great exertion, abdominal discomfort and lower-limb edema. At physical examination, he presented good general status, was eupneic and blood pressure (BP) was 140/90 mmHg. At the assessment of the jugular venous pulse, jugular ingurgitation was observed, with evident v wave. Cardiac auscultation showed a more audible systolic murmur at the lower left sternal border, which was accentuated by inspiration.

The electrocardiogram showed complete blocking of the right branch. The chest X-ray showed an increase in the cardiothoracic index, but no evidence of pulmonary congestion. The echocardiogram study showed rupture of the tricuspid valve leaflets, with bad coaptation at systole (Figure 1), causing massive tricuspid regurgitation (Figure 2). The right chambers showed large dilatation, but normal systolic pressure in the pulmonary artery (25 mm Hg). The contractility of the right ventricle was slightly reduced at the two-dimensional echocardiographic assessment, in spite of the volume overload caused by the valvular regurgitation. The analysis by the strain technique, a parameter used to assess the right ventricular systolic function measured at the basal segment of the free wall of the right ventricle showed a value of 15% (NV = 27 ± 6%), indicating systolic dysfunction of this chamber.

Discussion

Heart lesions caused by chest trauma include several presentations, such as cardiac contusion, free-wall rupture, septal rupture and valvular lesion.

Traumatic tricuspid regurgitation is rare and is often underestimated as it is hemodynamically well-tolerated and due to the attention given to lesions in the other organs. The time between the trauma and the surgery varies greatly, according to the etiology of each case, although the mean is 17 years. In fact, regarding the present case, the diagnosis of post-traumatic tricuspid valvular lesion was made 19 years after the traumatic event.

Due to its anterior location, the right ventricle is the most commonly affected chamber in penetrating chest trauma cases, presenting a predisposition to antero-posterior compression injuries. A sudden elevation in the right intraventricular pressure results in tricuspid valvular apparatus injury. When a sub-valvular lesion, papillary muscle rupture or avulsion occurs, the patient seems to become symptomatic more rapidly. However, as seen in our case, the leaflet lesions due to laceration or rupture close to the annulus evolve with more insidious symptoms.

The mild tricuspid regurgitation constitutes a frequent finding at the echocardiogram, present in more than 75% of normal individuals. However, the regurgitation of any etiology, from moderate to severe, is associated with a worse long-term prognosis. A study with 5,223 patients showed that the 1-year survival rate was 90% in patients with or without mild tricuspid regurgitation; 79% in those with moderate regurgitation and 64% in cases of severe regurgitation, regardless of the age, biventricular systolic function, degree of regurgitation, etc.
function and right ventricular dimensions\textsuperscript{10}. Thus, although the patient had recovered from a severe chest trauma, he presented a residual lesion in the tricuspid valve with impact on survival.

The evaluation of the right ventricular function at the echocardiogram presents several limitations. Recently, a new technique derived from the tissue Doppler, the strain technique (Figure 3), a parameter that directly analyzes the movement of the myocardial fibers, is being employed. A previous study demonstrated a correlation between the
strain and the invasive measurements of the right ventricular systolic function. In the present case, the reduced strain confirmed the right ventricular dysfunction secondary to the tricuspid lesion.

**Conclusion**

In conclusion, the case reported here emphasizes the importance of adequate follow-up of patients that have suffered penetrating chest trauma for several months after the injury, to detect possible late trauma complications.

**References**


