

A Case Of Traumatic Tricuspid Insufficiency With Patent Foramen Ovale Causing Cyanosis

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Abstract: Traumatic tricuspid regurgitation is a rare complication after blunt chest trauma and frequently misdiagnosed during the initial assessment. Traumatic tricuspid valve injury associated with right-to-left shunting via a patent foramen ovale causing hypoxemia and desaturation is noted even less frequently. Here we report a case of a patient with right heart failure symptoms and desaturation due to right-to-left shunting via a patent foramen ovale secondary to traumatic tricuspid insufficiency detected 20 years after a car accident. Surgery was required, consisting of tricuspid valve replacement. This case reminds that physicians in the emergency department should be aware of this potential complication following nonpenetrating chest trauma and its different clinical presentation. Patients with suspected valve lesion should undergo close echocardiographic follow-up to permit early detection of valve injury and timely surgical treatment.

Keywords: Tricuspid regurgitation; Tricuspid valve; Heart injury; Blunt trauma; Case report

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I.

Introduction

Traumatic tricuspid regurgitation is a rare complication after blunt chest trauma and frequently misdiagnosed during the initial assessment [1]. Despite the expanding use of echocardiography in trauma units, it is not uncommon to be diagnosed with tricuspid valve injury after several months to years [2]. This can be explained by the coexisting urgent issues of traffic accidents and the variable clinical course of tricuspid insufficiency. Besides that, traumatic tricuspid valve injury associated with right-to-left shunting via a patent foramen ovale causing hypoxemia and desaturation is noted even less frequently [6].

In this report we describe a patient with right heart failure symptoms and desaturation due to right-to-left shunting via a patent foramen ovale secondary to traumatic tricuspid insufficiency detected 20 years after a car accident.

II.

Case Report

A 44-years-old man was brought to our emergency department presenting symptoms of acute decompensated congestive heart failure, class III New York Heart Association (NYHA), with dyspnea and pulmonary congestion. Vital signs in the emergency room were pulse rate 95 / minute, blood pressure 108/72 mmHg, body temperature 36.1°C, respiration rate 26 per minute and pulse oximetry 85% with oxygen catheter 5 liters / min. Physical examination revealed rhythmic normophonetic heart sounds with grade 3/6 systolic murmur in tricuspid focus, absence of jugular turgence or hepatojugular return and extremities without edema. It also exhibited bilateral digital clubbing and cyanosis.

The patient was under follow-up at valvular heart disease ambulatory for etiological investigation of severe tricuspid regurgitation and hypoxemia. The outpatient transthoracic echocardiography revealed right cardiac chamber growth, significant tricuspid regurgitation and signs of rupture of tricuspid valve chordae, with preserved biventricular function and no signs of pulmonary hypertension. A congenital heart disease or Eisenmenger syndrome was discarded by the congenital heart disease department. During the investigation, the case was evaluated by the pneumology team and a lung disease that justified hypoxemia were also ruled out.

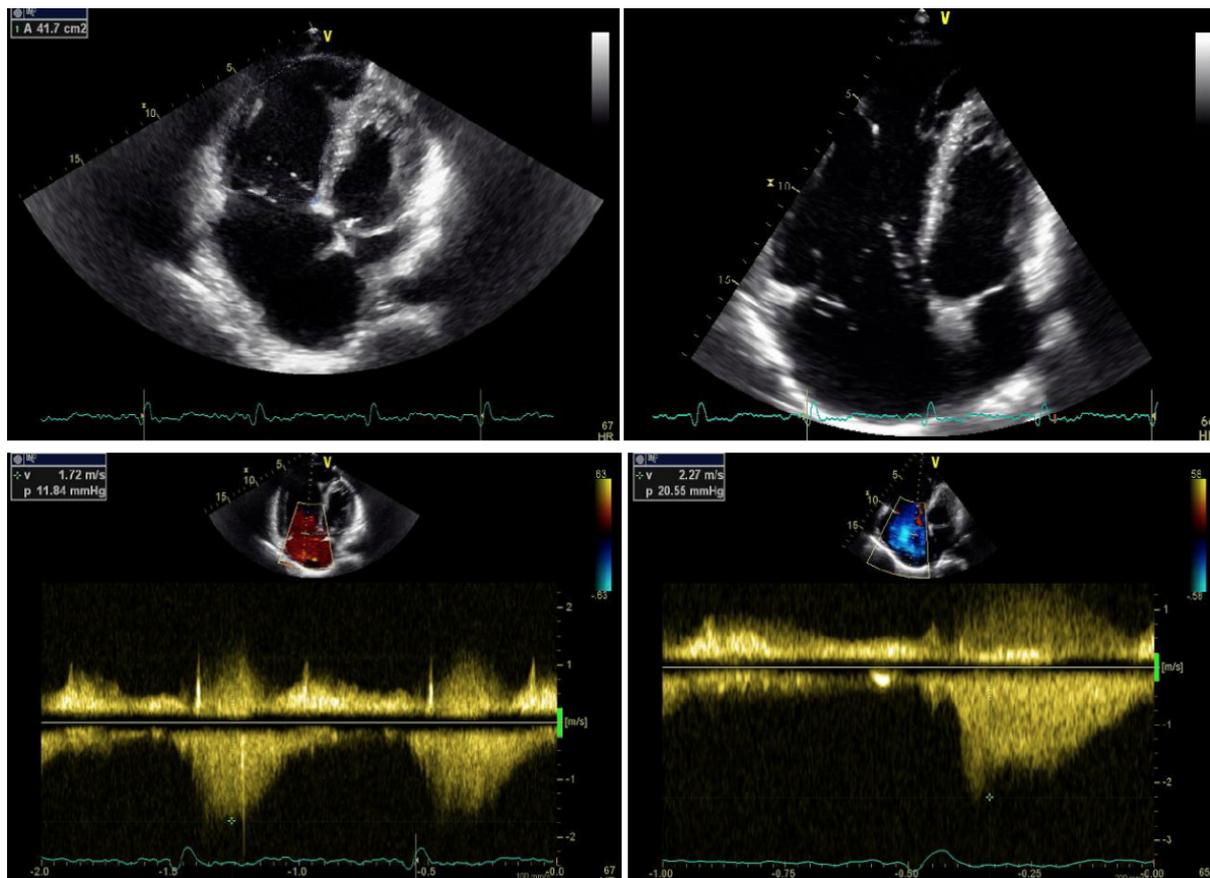


Figure 1 - Transoesophageal echocardiography showing significant tricuspid regurgitation and signs of rupture of tricuspid valve chordae

In addition, he had no history of infectious endocarditis and 20 years ago the patient was involved in a high-energy car accident, with severe traumatic brain injury requiring neurosurgery and blunt chest trauma. At that time, no cardiac disorder had been suspected and no echocardiogram had been performed.

For initial investigation at the emergency department, complementary exams were requested, including chest X-ray, twelve-lead electrocardiography, transthoracic echocardiography and chest computed angiography and there were no changes beyond those previously diagnosed. After another discussion in the Heart Team of our Institution, transoesophageal echocardiography and cardiovascular magnetic resonance imaging were requested.

Transoesophageal echocardiography showed a patent foramen ovale tunnelled, with 11mm extension and larger opening diameter of 2mm, with right-left shunt; tricuspid valve with rupture of anterior cusp chordae and severe regurgitation, estimated right ventricular systolic pressure of 25mmHg and severe dilation of right ventricle with preserved biventricular systolic function.

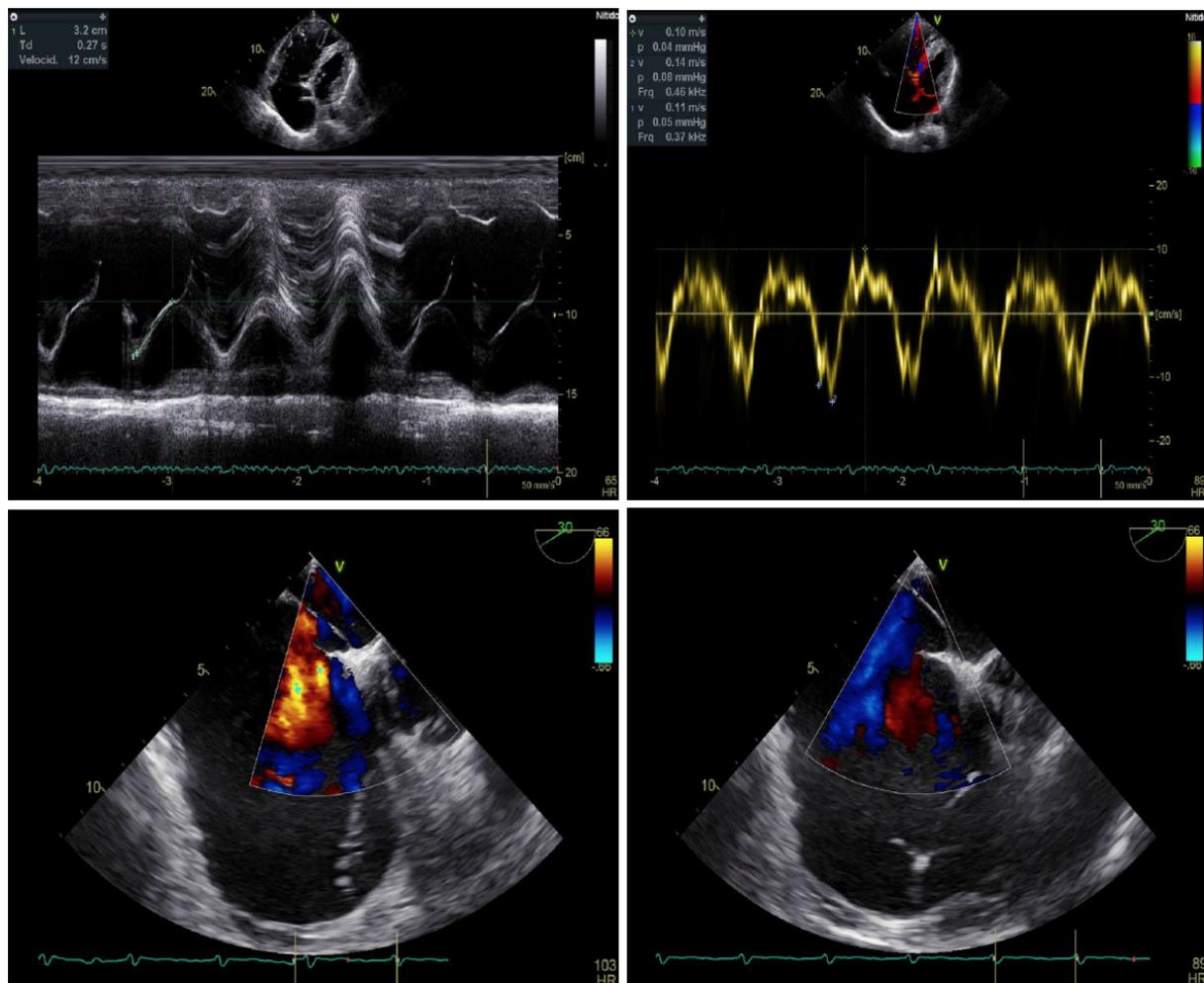


Figure 2 - Echocardiography transoesophageal showing patent foramen ovale tunnelled, with 11mm extension and larger opening diameter of 2mm and right-shunt. Tricuspid valve with severe regurgitation.

Cardiac magnetic resonance imaging showed major tricuspid regurgitation, right heart chambers with significant enlargement; preserved biventricular systolic function and absence of myocardial fibrosis. Right cardiac catheterization revealed no signs of pulmonary hypertension.

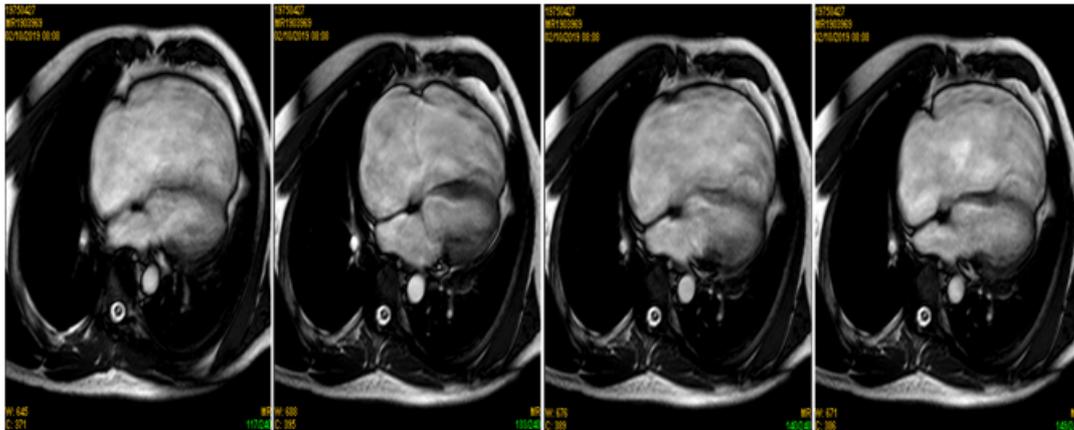


Figure 3 - Cardiac magnetic resonance showing right heart chambers with significant enlargement and severe tricuspid regurgitation

After evaluation of all exams, the Heart Team decided for surgical treatment of severe tricuspid regurgitation. Intraoperative findings confirmed retraction of the tricuspid anterior leaflet, with multiple ruptured chords and a patent foramen ovale. Tricuspid valve replacement was performed with a size 33 biological prosthesis. It was opted to maintain patent foramen ovale due to considerable risk of right ventricular dysfunction. The postoperative transoesophageal echocardiography showed normal tricuspid prosthetic functioning with no perivalvar leak and normal biventricular systolic function.

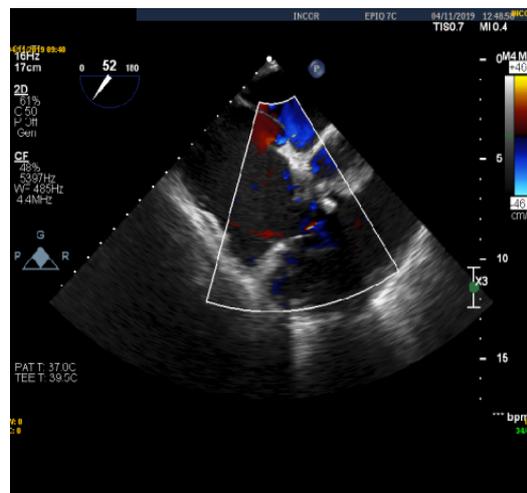


Figure 5- Intraoperative echocardiography transoesophageal showing patent foramen ovale

The patient had an uncomplicated postoperative course and left the hospital 11 days after surgery in excellent clinical condition, asymptomatic and with scheduled outpatient follow-up.

III.

Discussion

Traumatic tricuspid insufficiency as a result of blunt chest trauma is a relatively uncommon lesion. Its prevalence is probably misdiagnosed during the initial assessment due to subtle clinical symptoms and more clinically acute lesions [1,3]. When it does not occur rapid onset of heart failure, diagnosis of traumatic tricuspid regurgitation could be difficult because its clinical presentation can be atypical or asymptomatic for many years, with a slow but progressive course [1]. This may lead to a delay in treatment and thus result in dilatation of the right-sided cavities and irreversible right ventricular insufficiency.

Benhassen and cols [4] reported a case of traumatic tricuspid valvular insufficiency in a 44-year old man detected 28 years after a moped-accident. Bortolotti and cols [5] described a tricuspid regurgitation in a 48-

year old man diagnosed 20 years after an apparently minor thoracic trauma. This patient had a marked tricuspid insufficiency with a patent foramen ovale and a mild right-to-left shunt, with no desaturation.

Desaturation and cyanosis due to right-to-left shunting via a patent foramen ovale can be observed when elevated right atrial pressure occurs. In the context of tricuspid valve insufficiency, similar cases have been reported by others. Schuster and cols. [6] reported a patient presenting with signs of right heart failure and cyanosis with a peripheral oxygen saturation of 84% nine years after a blunt thoracic trauma. Contrast and Doppler echocardiography demonstrated severe tricuspid regurgitation and a significant right-to-left shunt at the atrial level via a patent foramen ovale. This patient became symptomatic 10 months after accident but a valve injury was undiagnosed. Bardy and cols. [7] described a patient with cyanosis and right-to-left shunting via a patent foramen ovale secondary to traumatic tricuspid insufficiency. These authors suggest that the right-to-left shunt across a patent foramen ovale without a high pulmonary arterial pressure, like our case reported, can be explained by an increased right atrial pressure due to tricuspid insufficiency that surpasses left atrial pressure; by the streaming of blood from inferior vena cava to the left atrium that can be independent of pressure relations and by an enlarged right atrium that stretches the foramen ovale, which becomes patent with a "ventricularized" right atrial pressure [7].

The clinical course of tricuspid regurgitation following a non-penetrating chest trauma is variable. The severity of symptoms and the duration between trauma and onset of cardiac symptoms were related to the type of damage to the tricuspid valve incurred [7].

Transthoracic echocardiography is the investigation of choice in patients with blunt chest trauma, in particular if cardiac injury is suspected. In addition, earlier diagnosis may increase the feasibility of tricuspid valve repair and surgical treatment may prevent progressive deterioration of right ventricular function [8,9].

The traditional indication for surgery is heart failure symptoms. However, surgery should be performed before the development of right ventricular dilatation and insufficiency [10,11]. Schuster and cols. suggest [6] that early operation should generally be preferred, since significant annular dilatation and leaflet retraction make repair of the valve impossible.

The usual lesions observed at surgery are subvalvular rupture of the anterior papillary muscle, leaflet prolapse, rupture of the chordae tendinae and laceration of valve leaflets [12].

In the case reported here, the important valve deformity and a right ventricular dilation made valve repair impossible and a valve replacement was performed. It has been reported that there is an association between failure of tricuspid valve repair and prolonged interval between trauma and treatment [13]. In general it is suggested that valve repair is more difficult if complex lesions are present and the time interval between trauma and surgery is long. However, there is a consensus that valve repair, when feasible, is superior to replacement [14].

In addition, in all of our literature references, the foramen ovale was closed at surgery of the traumatic tricuspid regurgitation.

This case reminds us that physicians in the emergency department should be aware of this potential complication following nonpenetrating chest trauma and its different clinical presentation. Patients with suspected valve lesion should undergo close echocardiographic follow-up to permit early detection of valve injury and timely surgical treatment. Earlier diagnosis and surgical treatment may increase the feasibility of tricuspid valve repair and prevent progressive deterioration of right ventricular function [8].

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