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CASE REPORT

# Tricuspid valve avulsion caused by horse kick: A case report and review of literature

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## Abstract

Blunt injury to the chest can affect all components of the chest wall and the thoracic cavity. Tricuspid valve insufficiency, however, is a rare cardiac complication following blunt chest trauma. Moreover, the prevalence of the condition may be underestimated due to subtle clinical symptoms. This may lead to a delay in treatment, and thus result in irreversible dilatation of the right cavities. Here we report a case of severe tricuspid valve insufficiency, caused by papillary muscle and chordal rupture secondary to blunt chest trauma from a horse kick, in a 61-year-old man. Surgery was required, consisting of papillary muscle reattachment and ring annuloplasty. In addition, we review the relevant medical literature regarding the diagnosis and treatment of traumatic tricuspid valve regurgitation.

## Introduction

Traumatic tricuspid valve regurgitation is thought to be a rare complication of blunt, non-penetrating chest trauma. High-speed motor vehicle accidents are a leading cause of traumatic tricuspid valve avulsion. However, this condition can also result from a simple fall to a kick in the chest. The diagnosis may be delayed or missed entirely given its rarity, its lack of acute physical findings and the presence of coexisting urgent issues in the trauma patient.[1]

We report on a case of traumatic tricuspid insufficiency and right ventricular dysfunction in a 61-year old man after a horse kick to the chest and we review the relevant medical literature of traumatic tricuspid valve regurgitation.

## Case report

A 61-year-old man, with an active lifestyle and without relevant previous cardiac or medical history, was admitted to the hospital following blunt chest trauma caused by a horse kick. He presented to the emergency department with chest pain and moderate shortness of breath. He was conscious, alert and haemodynamically stable. Auscultation revealed no heart murmurs. Twelve-lead electrocardiography (ECG) showed normal sinus rhythm, and both creatine kinase (CK) 279 U/l (ref 38-174 U/l) and troponin 0.78 $\mu$g/l (ref <0.08) levels were slightly elevated. Computed tomography (CT) of the chest demonstrated bilateral displaced anterior rib fractures and a limited bilateral pneumothorax. Transthoracic echocardiography (TTE) revealed no pericardial effusion, wall abnormalities or other cardiac lesions. Patient-controlled epidural analgesia (level T5-T6; mixture: ropivacaine (Naropin®) 600 mg and sufentanil (Sufenta®Forte) 0.40 mg in NaCl 0.9% 400 ml; rate: 5 ml/h; bolus: 2 ml; lockout interval: 20 minutes) was initiated before admission

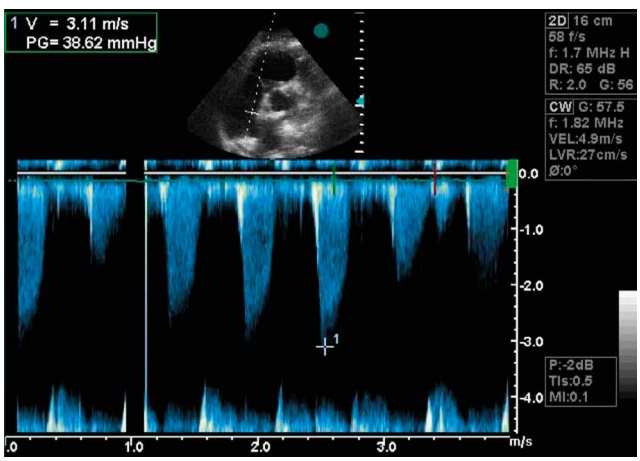


**Figure 1.** The transthoracic echocardiogram shows the ruptured tricuspid valve causing severe tricuspid regurgitation, with jet flow velocities of around 3.0 m/s, and with a pressure gradient of 38 mmHg, indicating pulmonary hypertension

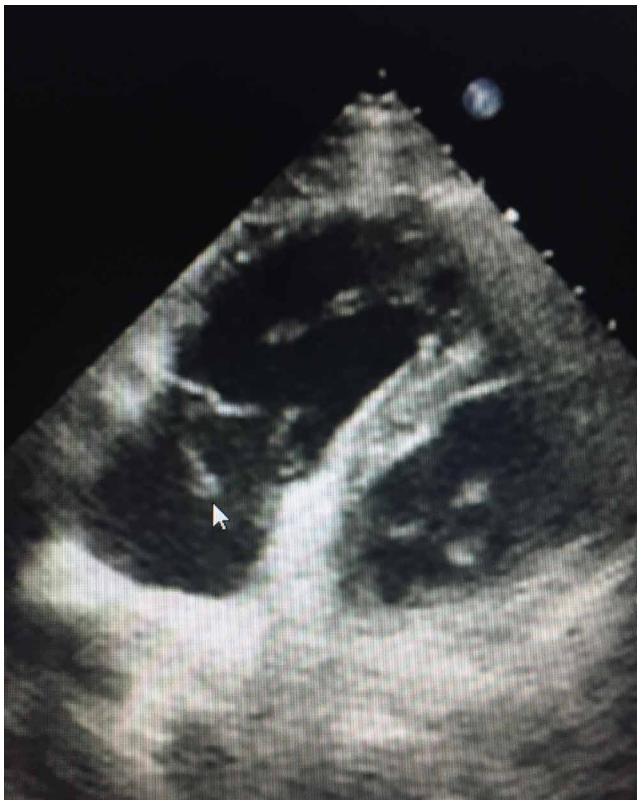

**Figure 2.** 2D transthoracic echocardiographic apical view of the right ventricle. The arrow is located in the right atrium and points to ruptured chordae and papillary muscle. These are right ventricular structures and their presence in the right atrium is proof of chordal rupture

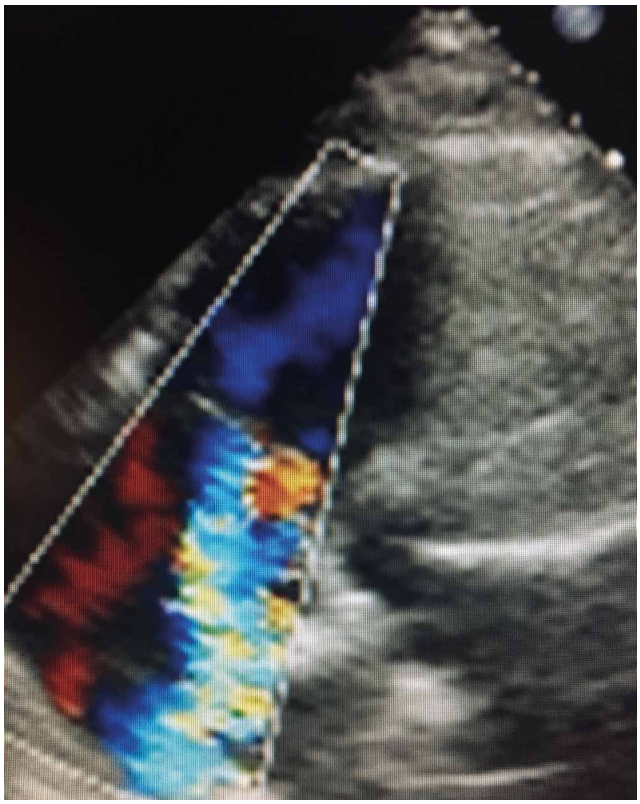

**Figure 3.** 2D transthoracic echocardiographic apical view of the right ventricle with continuous flow mode. Colour flow image showing a large jet of tricuspid regurgitation into the right atrium

to the intensive care unit (ICU) for further management and monitoring. At this moment, no clear clinical signs of right ventricular failure were present.

On the second and third day of hospitalisation, his respiratory parameters improved and on the fourth day he was discharged from the ICU to the medical ward with telemetry monitoring. Later that day, his respiratory condition deteriorated. Negative T waves were seen on his ECG in the anterior and inferior leads, and a new right axis deviation was noted. CT ruled out pulmonary embolism but showed a haemothorax in the right side of the chest. A new TTE revealed severe tricuspid regurgitation secondary to a tricuspid chordae tendineae and papillary muscle rupture and right ventricular dysfunction *(figures 1-3)*.

The patient was referred to a tertiary centre for cardiac surgery. A chest tube was inserted to evacuate the haemothorax. After receiving further conservative treatment, there was only a slight recovery of the right ventricular function. Our patient was scheduled for elective tricuspid valve repair seven weeks after the trauma.

Anaesthesia was induced uneventfully. Cardiac exposure was obtained through a right anterior mini-thoracotomy with a small incision by means of port-access minimally invasive cardiac surgery, as has been previously reported.[2,3] Briefly, the femoral artery and vein were cannulated in the groin to establish cardiopulmonary bypass and the internal jugular vein was used for additional venous drainage of the upper body. Aortic cross-clamping was applied and cardiac arrest was achieved by antegrade cardioplegia using a root cannula and topical cooling. A right atriotomy was performed to visualise the tricuspid valve and subvalvular apparatus. Ruptures of the head of the papillary muscle and of some chordae tendineae associated with the posterior leaflet were noted. The head of the papillary muscle was reattached and cleft closure between posterior and septal cusp was performed *(figure 4)*. Ring annuloplasty was also carried out using a 28-mm Edwards MC3 annuloplasty system (Edwards Life-sciences, Irvine, CA, USA). The right atrium was closed and the patient was weaned from the cardiopulmonary bypass. The postoperative course was uneventful. On the seventh postoperative day, TTE showed satisfactory ventricular function with no paravalvular leaks whereupon the patient was discharged from hospital. Six months after the operation, the patient is in good condition.

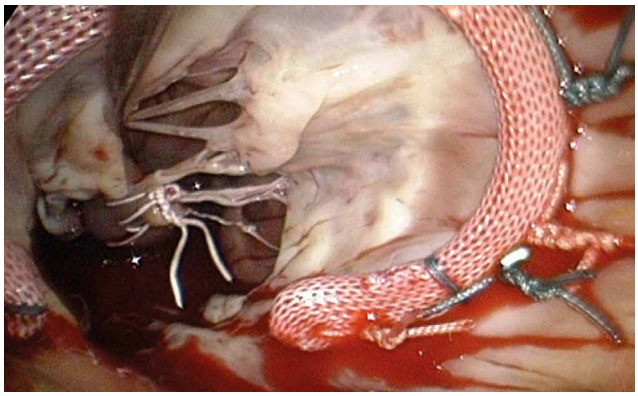

**Figure 4.** Repaired tricuspid valve. The upper side shows the native chordae attaching the tricuspid valve to the papillary muscle. The lower part of the picture shows how the ruptured part of the tricuspid valve is attached with neochords (Goretex) to the papillary muscle. The valve is surrounded by an annuloplasty ring.

## Discussion

Blunt cardiac trauma encompasses a large spectrum of lesions. Injuries can be benign, such as myocardial contusion, or fatal, such as chamber rupture.[4] Modes of injury can vary from a simple fall to a kick in the chest or even automotive airbag deployment.[5] In 2006, the Centres for Disease Control and Prevention estimated the incidence of cardiac injury resulting from blunt chest trauma at 30,000 cases per year in the United States.[6]

Autopsy studies indicate that the aortic valve is most commonly involved, followed by the mitral valve and the tricuspid valve.[7] Although traumatic tricuspid insufficiency is a relatively uncommon lesion, its frequency is probably underestimated given the emphasis on other organ injuries.

The mechanism of tricuspid valve injury is debated. The best postulated theory is extensive tricuspid 'blow out' injury, secondary to severe and sudden impact during end-diastole.[8] As the right ventricle is immediately behind the sternum, it is predisposed to an anteroposterior compression type of injury, especially when the valve is closed. The decelerating force in the right ventricle produces regurgitation, thereby initiating rupture of the papillary muscle and/or chordae tendineae.

The clinical course of tricuspid regurgitation following blunt chest trauma varies greatly and is related to the extent of the tricuspid valve injury. Rupture of papillary muscle, for example, is usually associated with severe and acute symptoms, which are more frequently diagnosed earlier, while mild symptoms appear more often in patients with a rupture of the leaflet of chordae tendineae.[9-11] Consequently, several years may elapse before a diagnosis is made. A delayed presentation of tricuspid regurgitation after trauma can lead to delays in treatment. In a series of 13 cases of tricuspid insufficiency following blunt chest trauma, Van Son et al. reported a median duration between trauma and operation of 17 years (range: 1 month to 37 years).[12]

TTE is considered the golden standard for diagnosing associated lesions and for assessing the mechanism of tricuspid regurgitation because the tricuspid valve is located anteriorly.[13] However, because many of these patients have poor acoustic windows resulting from the trauma-associated complications (e.g., haematoma, pneumothorax) and their treatments (e.g., tubes, bandages), the incidence of missed diagnoses with TTE may be higher than expected. Transoesophageal echocardiography (TEE) can further confirm the TTE findings and optimise the anatomical evaluation of the valvular apparatus.[14]

Optimal treatment for traumatic tricuspid avulsion remains controversial and ranges from long-term medical therapy to early surgical intervention.[11,15] The traditional indication for operation is symptomatic heart failure. However, since post-traumatic tricuspid regurgitation is effectively correctable with reparative techniques, early operation is also considered to relieve symptoms and to prevent right ventricular dysfunction. A trend towards more minimally invasive surgical repair has been seen during the last decade. The case presented here highlights the fact that physicians working at emergency departments or intensive care units should be on the alert for this potential complication of blunt chest trauma. In any case, thorough medical examination including electrocardiography, chest radiography and, with suspicion of cardiac injury, TTE, should be performed early after non-penetrating chest trauma to rule out structural cardiac lesions.

## Disclosures

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