Severe Aortic and Tricuspid Valve Regurgitation after Blunt Chest Trauma: An Unusual Presentation

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INTRODUCTION

Blunt chest trauma (BCT) is a frequent condition in road traffic injuries, and it is related to mortality rates. The most frequent lesion is aortic rupture in the aortic isthmus, followed by heart-specific (blunt cardiac) injuries, of which myocardial contusion is the most frequent.

Motor vehicle accidents represent the most common cause of significant thoracic injury among emergency department patients.[1] The incidence of cardiac injuries ranges from 20% to 76% in individuals involved in this type of trauma.[2] Heart valve injury is an unusual finding after blunt cardiac injury. When it happens, the resulting valvular regurgitation can progress to symptomatic heart failure or death if not diagnosed early; often, these lesions require urgent surgical management.

We describe a young male patient who was involved in a high-speed traffic accident and presented with a new murmur in addition to heart failure symptoms caused by severe aortic and tricuspid valve injuries. We discuss the principles of diagnosis and management of this unusual case.

CASE REPORT

A 43-year-old man, without previous medical history, presented to the emergency room due to a motorcycle collision with traumatic brain injury, multiple facial fractures, and blunt chest trauma (BCT). Initially, he was treated in another hospital at the intensive critical care unit with a successful recovery of head injuries without any neurologic sequelae; 20 days later, he was discharged. No murmur was detected at that time.

One month after the accident, he was admitted to our emergency room due to shortness of breath, abdominal distension, and bilateral lower extremity edema. His physical exam was noticeable for a widened pulse pressure (blood pressure 130/50 mm Hg); a 3/4 diastolic murmur was heard at the right upper sternal border in addition to a 3/6 systolic murmur at the left lower sternal border. Pulmonary findings included bibasilar rales; he had no fever since the onset of symptoms. The hemogram results and blood chemistry were normal.

The electrocardiogram showed normal sinus rhythm with right bundle branch block and left anterior fascicular block. The chest x-ray revealed an increased cardiothoracic index with normal mediastinum (Figure 1). A transthoracic echocardiogram (TTE) showed mild left ventricular dilatation, diffuse hypokinesis with moderate systolic dysfunction (ejection fraction = 40%), and severe eccentric aortic regurgitation due to a flail right coronary cusp (RCC; Video 1, Figure 2). In addition, severe tricuspid insufficiency was noticed due to a partial rupture of the right ventricular anterior papillary muscle causing severe prolapse of the anterior and posterior tricuspid leaflets (Videos 2 and 3, Figure 3). Transesophageal echocardiography (TEE) was performed for further valve characterization and to determine the likelihood of surgical repair (Videos 4 and 5). Three-dimensional echo imaging improved the visualization of the RCC avulsion resulting in severe aortic regurgitation and right ventricle anterior papillary muscle rupture (Videos 6 and 7).

Because of the severity of his symptoms, this patient with severe aortic and tricuspid valve regurgitation was taken emergently to the operating room. The intraoperative view of the aortic valve revealed the traumatic tear in the RCC (Figure 4A). From the right atrium view, surgeons found tricuspid anterior leaflet prolapsed due to an edematous and necrotic anterior papillary muscle that was ruptured and almost totally detached from its base (Figure 5). A successful aortic valvuloplasty and tricuspid valve replacement with a biologic prosthesis were performed without complications (Figure 4B). A TTE performed 4 days after surgery showed normal morphology of the aortic valve with only trace aortic regurgitation and a biologic tricuspid valve prosthesis with normal function (Video 8).

He had an uneventful postoperative course and was discharged 5 days after surgery. Three months later, the patient was asymptomatic without heart failure symptoms.

DISCUSSION

Road traffic injuries are the eighth leading cause of death among all ages and the number one cause of death for people ages 15-29 years.[3] Blunt chest trauma is a common condition following motor vehicle collisions and is the second leading cause of death after brain injury.[1] Principal mechanisms related to BCT include rapid deceleration, torsion forces, high-energy injuries to the thorax, direct precordial impact, and abrupt pressure fluctuations in the chest and abdomen.[2,4]

The leading cardiovascular damages after BCT are thoracic aortic injury and heart-specific (blunt cardiac) injuries. Thoracic aortic injury has the highest mortality rate, up to 80%-85%,[5] and is as frequent as 16% as described in 218 autopsies of people killed after high-speed

traffic accidents.[6] Two-thirds of blunt thoracic aortic injuries occur at the aortic isthmus, just distal to the left subclavian artery where the ligamentum arteriosum attaches to the aorta. At this location, the ligamentum arteriosum creates a tether point, and rapid deceleration can cause shearing, leading to aortic transection or dissection.[7]

Up to 20% of deaths from high-speed accidents are attributable to blunt cardiac injuries.[8] Heart-specific injuries include myocardial contusion, cardiac rupture, valve injuries, papillary muscle dysfunction, pericardial complications, and coronary artery involvement.[1] The most common injury is myocardial contusion, which occurs in approximately 24% of cases. The anterior location of the right atrium and ventricle within the mediastinum is the leading risk factor for severe compression and contusion during chest trauma.[1,4,9]

Heart valve injuries resulting in acute valvar insufficiency are uncommon complications of BCT. Postmortem reports showed an incidence of 5% for valve lesions after blunt cardiac injury.[2] A diagnosis of traumatic heart valve injury often is suggested by history, acute or progressive heart failure, or a new heart murmur. Aortic valve rupture is the most frequently reported valvular injury, followed by the mitral, tricuspid, and pulmonary valves.[10]

Aortic valve injury after BCT can lead to severe aortic regurgitation. The available information corresponding to aortic valve injuries is scarce and mainly derived from case reports. Symptoms are often acute in patients with aortic valve injury.[11] These symptoms can also present subacutely as in our case. Usually, only one cusp is damaged, and the noncoronary cusp is the most commonly involved, followed by the RCC, and, lastly, the left coronary cusp.[12] The mechanism causing aortic valve rupture is a sudden increase in intra-aortic pressure during a vulnerable phase of the cardiac cycle, especially during early diastole when the aortic valve is closed, the pressure in the empty left ventricle is low, and the transaortic gradient is maximal. This high pressure transmitted toward the closed aortic valve can cause detachment of the commissure, a tear, or a rupture of an aortic valve cusp. Since the normal distribution of blood flow to the right and left coronary arteries occurs during diastole, the hemodynamic stress over these cusps decreases, but not over the noncoronary cusp, the most often injured cusp. Also, the closer position of the RCC to the anterior chest wall may expose it to higher pressure, which would explain its second place in the incidence of injury and in our case aortic valve avulsion.[12] The earlier diagnosis of acute aortic valve injury depends on clinical suspicion and the time until the onset of hemodynamic deterioration or heart failure symptoms.

Traumatic tricuspid regurgitation after BCT is less frequent than left heart valves. Anatomically, the right ventricle is immediately behind the sternum, predisposing it to blunt trauma, especially when distended right ventricles are compressed between the sternum and the spine during diastole.[13] Anteroposterior compressive forces cause an increase in the hydrostatic pressure of the ventricle, which is transmitted to the valve and the subvalvular apparatus, resulting in tearing injuries and rupture of the valvular components. The tricuspid valve tends to receive a more severe injury when it is closed and pressures in the ventricles are high. Therefore, the mechanism of tricuspid injury is a sudden increase in intracardiac pressure throughout the vulnerable phase during early systole. Tricuspid injuries include three main mechanisms: chordal rupture, anterior papillary muscle rupture, and less commonly a leaflet tear or rupture, primarily of the anterior leaflet.[14] In our patient, TTE imaging and the intraoperative findings demonstrated the effect of the sudden pressure increase over the subvalvular apparatus, resulting in anterior papillary muscle rupture. Clinical manifestations are related to the extent of tricuspid valve injury. These vary from the rapid onset of heart failure to a slow but progressive clinical deterioration over months to years. A high degree of suspicion and the early use of TTE and TEE are essential in the diagnosis of traumatic tricuspid regurgitation.

With this particular case, it is difficult to determine the chronology of valve dysfunctions. We speculate that the aortic valve regurgitation was severe immediately after the vehicle accident based on the TTE and TEE findings. However, it is probable that tricuspid valve
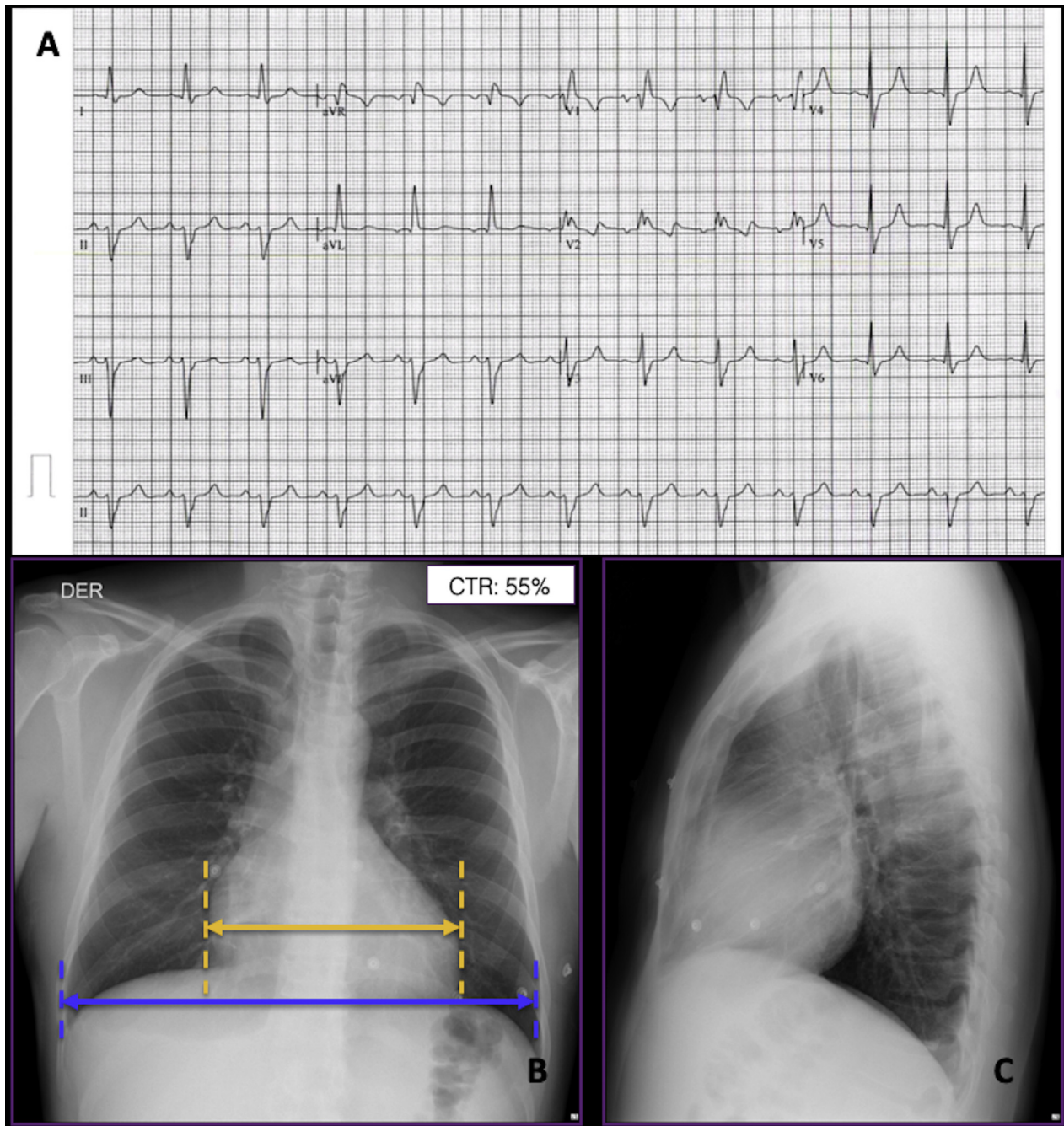
Figure 1 **(A)** Electrocardiogram showed normal sinus rhythm with right bundle branch block and left anterior fascicular block. **(B** and **C)** Chest x-ray with cardiothoracic ratio = cardiac width/thoracic width of 55%, suggestive of mild cardiomegaly. No widened mediastinum was observed.

dysfunction occurred over time, and initially there was only anterior papillary muscle edema that progressed to rupture, leading to a severe anterior and posterior leaflet prolapse resulting in severe tricuspid insufficiency. It may explain why congestive heart failure symptoms happened only 30 days after the accident. Nevertheless, there is no certainty because no TTE was reported from the outside hospital in the first admission just after the accident.

Our case is extremely rare because the patient presented with two unique features: first, the simultaneous combination of severe double

valve injury; and second, each valve injury has different traumatic mechanisms. The delay in the diagnosis of the heart valve injuries (done one month after BCT) may be secondary to the multiple organs involved during the accident (head trauma, pulmonary contusion). Also, no murmur was heard initially in the outside hospital, and early use of TTE may have had a limited role due to the difficulty in obtaining accurate images because of mechanical ventilation, pneumothorax, hemothorax, mediastinal emphysema, and other possible related injuries that may cause trouble for the positioning of the
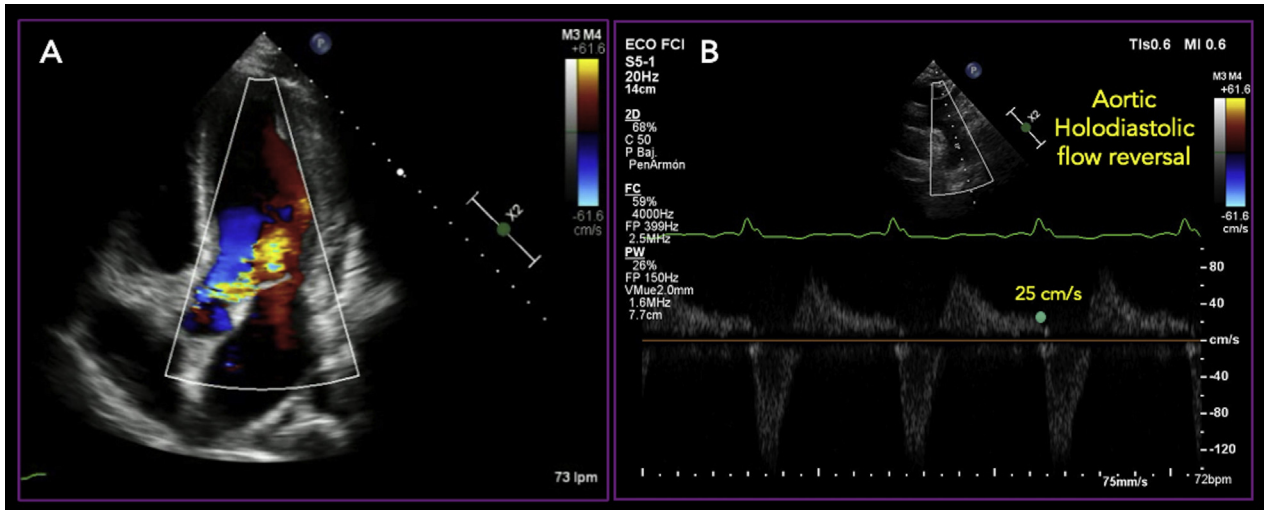
Figure 2 **(A)** Apical five-chamber view showing a significant eccentric aortic regurgitation jet. **(B)** Suprasternal view with pulsed wave Doppler of the aorta showing holodiastolic flow reversal with end-diastolic velocity = 25 cm/sec, a specific sign of severe aortic regurgitation.
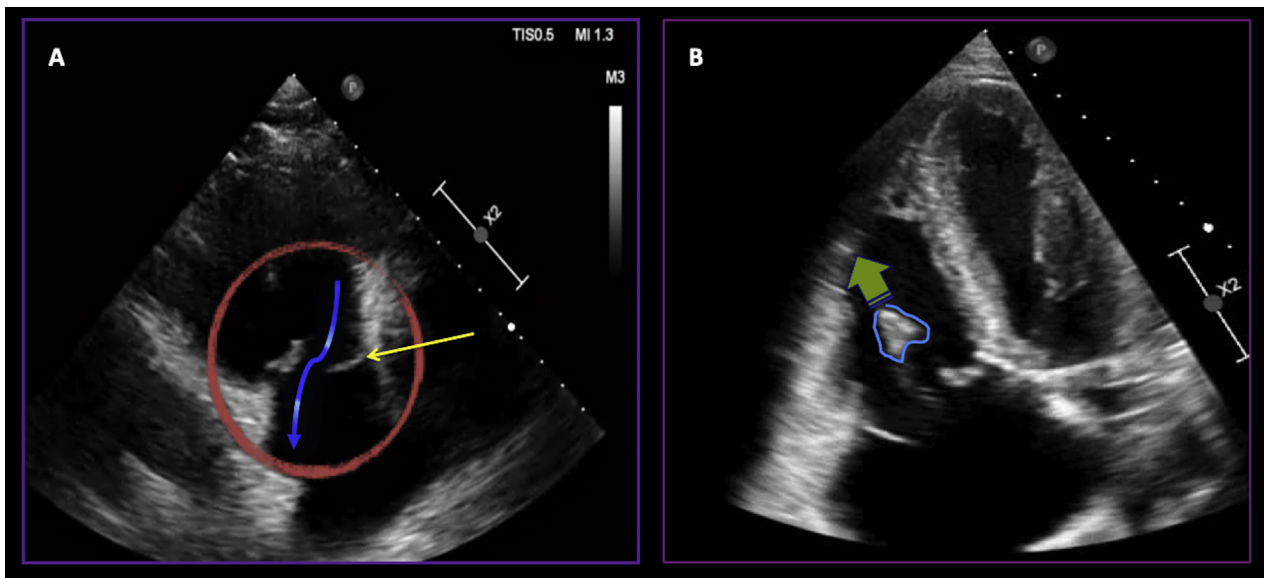

Figure 3 Still-frame images that explain the tricuspid regurgitation mechanism. **(A)** Right ventricular inflow view showing the tricuspid anterior leaflet prolapse (*yellow arrow*) and the coaptation gap where the severe tricuspid regurgitation occurs (*blue arrow*). **(B)** Right ventricular focused view: the demarcated structure corresponds to the ruptured anterior papillary muscle that was detached from right ventricular free wall (*green arrow*) and causes the severe anterior and posterior leaflet prolapse as the mechanism of severe tricuspid regurgitation after BCT.

patient and to the limited optimal visualization of the heart anatomy and function. For these reasons, TEE is the diagnostic procedure of choice for assessing traumatic valve injuries and obtaining accurate information about the injured structures, particularly when TTE images are suboptimal.

In the setting of severe BCT, careful surveillance, repeated physical examination, and echocardiography as the noninvasive test of choice are key for a heart valve injury diagnosis. Symptomatic heart failure is the most reliable indication for surgery and early repair that allows myocardial reserve preservation by preventing subsequent remodeling changes. In our patient, acute severe valvular regurgitation of two valves following BCT led to the rapid onset of heart failure; the decision to undertake surgical intervention was based on the recommendations of the current guidelines for the management of valvular disease.[15] In hemodynamically unstable patients and those with refractory congestive heart failure, surgical intervention should be emergent. Early valve repair is the preferred treatment, although the extensive tricuspid apparatus damage in our case required replacement with a prosthesis.
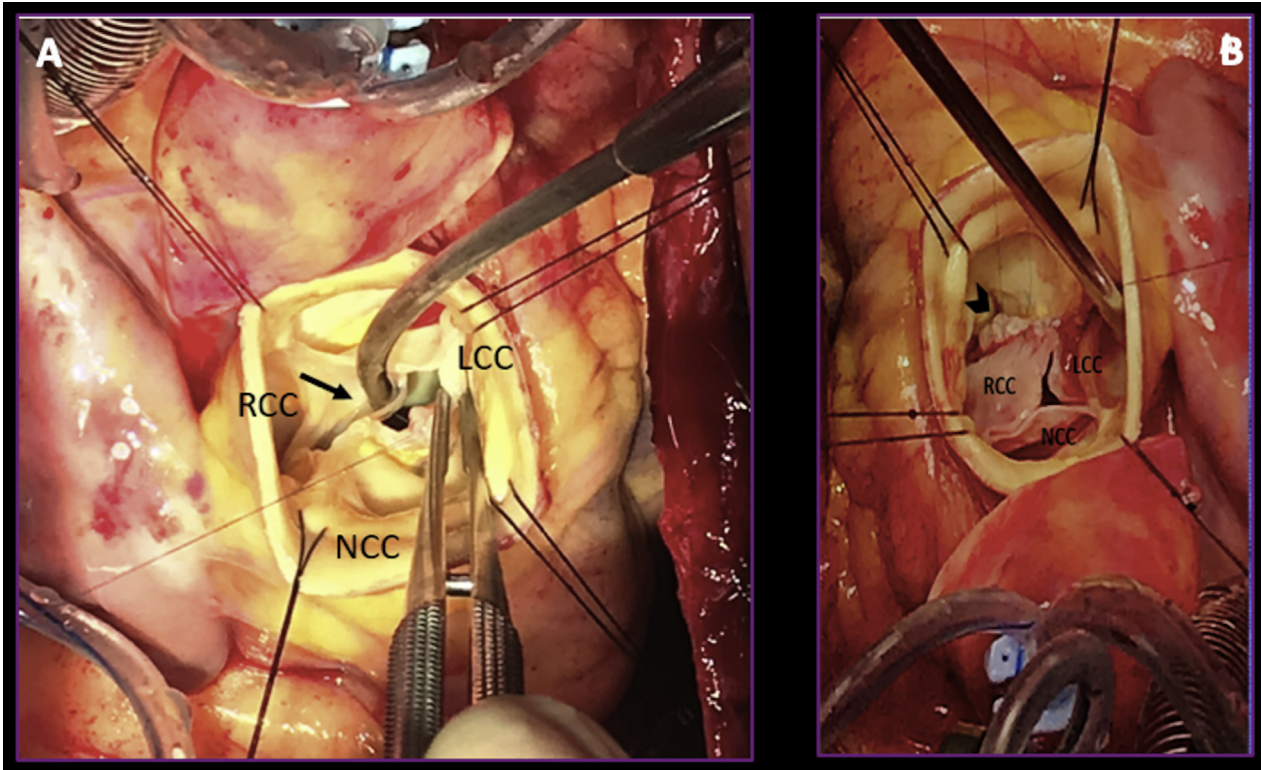
Figure 4 (A) Intraoperative view of aortic valve with a tear in the RCC in which the cannula goes through (*black arrow*). **(B)** RCC valvuloplasty with surgical repair of its tear (*black arrowhead*). No abnormalities were found in the aortic wall and aortic valve annulus. *LCC*, Left coronary cusp; *NCC*: noncoronary cusp.
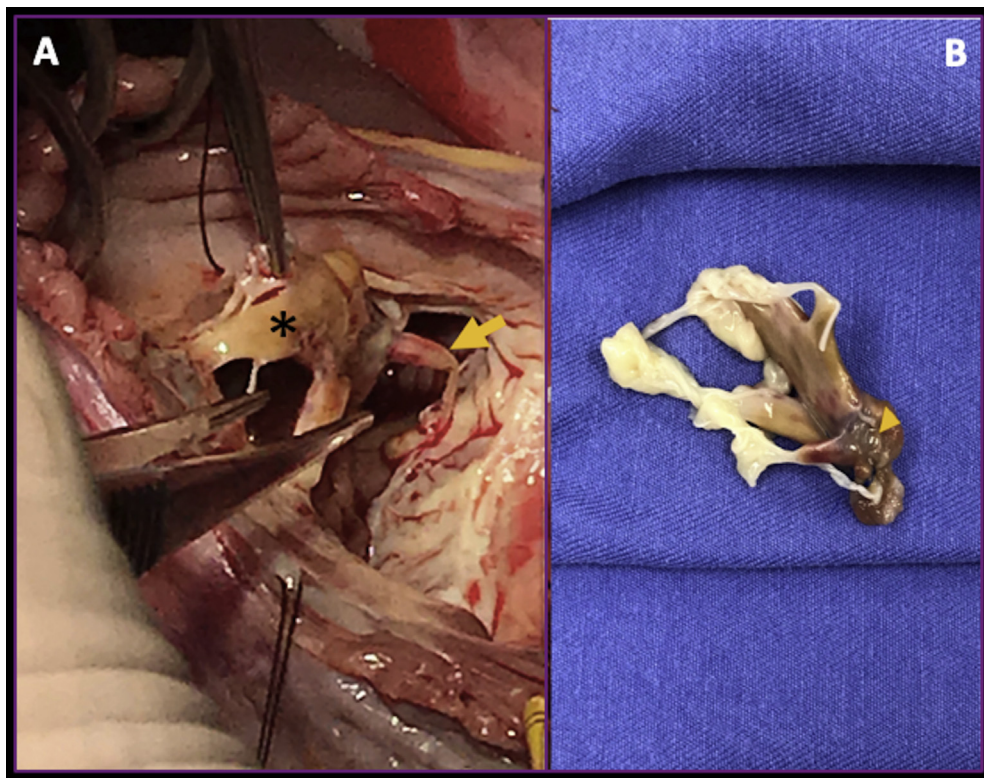

Figure 5 (A) Intraoperative photograph from right atrium view shows the tricuspid anterior leaflet prolapsed (*) because of the anterior papillary muscle rupture (*arrow*), which is almost completely detached from its base. **(B)** Specimen of the resected tricuspid valve with its subvalvular apparatus showing the anterior papillary muscle (*yellow arrowhead*) is ruptured and necrotic. Tricuspid anatomy was unsuitable for repair, so replacement with a biologic prosthesis was performed.

CONCLUSION

Recent high-energy BCT, including traffic accidents and falling from heights, must be promptly and repeatedly explored with physical exam and by echocardiography to rule out either isolated or combined intracardiac lesions. Significant heart valve injuries are rare; however, when valvular dysfunction occurs, it can result in significant morbidity and mortality. The timing of the surgical intervention in heart valve injuries depends on the patient's general clinical condition.

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2020.03.010.

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