



Post-Traumatic Chordae Rupture of Tricuspid Valve

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Abstract

Blunt injury to the chest can affect any one or all components of the chest wall and thoracic cavity. The clinical presentation of patients with blunt chest trauma varies widely and ranges from minor reports of pain to florid shock. Traumatic tricuspid valve regurgitation is a rare cardiovascular complication of blunt chest trauma. Tricuspid valve regurgitation is usually begotten by disorders that cause the right ventricle to enlarge. Diagnosis is made by physical examination findings and is confirmed by echocardiography. We report two cases of severe tricuspid regurgitation secondary to the rupture of the chordae tendineae of the anterior leaflet following non-penetrating chest trauma. Both patients had uneventful postoperative courses.

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Introduction

Blunt thoracic trauma has become a regular occurrence courtesy of high-speed motor vehicle accidents,¹ especially in the last decade.² Such accidents are the principal cause of isolated tricuspid valve regurgitation (TR).³ Traumatic TR is a rare cardiovascular complication of blunt chest trauma. The most frequently involved valve in blunt chest trauma is the aortic valve, followed by the mitral and tricuspid valves.⁴ Traumatic TR is usually well tolerated;⁵ however, as far as treatment is concerned, the tricuspid valve replacement has been the conventional procedure, especially in cases with delayed presentation.⁶ We herein report two cases of severe TR secondary to the rupture of the chordae tendineae of the anterior leaflet following non-penetrating chest trauma.

Case Reports

Case # 1

A 28-year-old man was admitted to our hospital suffering from exertional dyspnea and palpitation. One year previously, he had sustained blunt chest trauma in a car accident, but no cardiac abnormality had been noticed at the time. His symptoms started about two months before his referral to us.

At presentation, the patient had a blood pressure of 120/90 mmHg and pulse of 70 beats/min. The electrocardiogram revealed sinus rhythm and right bundle branch block with left-axis deviation. Transthoracic echocardiography (TTE) demonstrated a normal left ventricle (LV) size, mild LV systolic dysfunction, septal motion abnormality due to the right ventricle (RV) volume overload, severe RV and right

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atrium (RA) enlargement, and finally, severe low pressure TR due to the flail anterior leaflet of the tricuspid valve. Transesophageal echocardiography (TEE) was conducted as a complementary study and confirmed the flail leaflet secondary to the ruptured chordae. The patient was, therefore, scheduled for tricuspid valve repair.

The operation was performed through median sternotomy. Following the institution of cardiopulmonary bypass (CPB), the heart was arrested via cardioplegia and the RA was explored: there was chordae rupture of the anterior leaflet. The rupture was repaired by first constructing two near chordae with 5/0 Gortex suture and then placing edge-to-edge stitches on the edge of the three leaflets. Additionally, ring annuloplasty using a Carpenter (31mm) was performed.

Postoperative TEE demonstrated mild to moderate TR and no significant tricuspid valve stenosis (TS) (tricuspid valve mean gradient = 2.5 mm Hg). The patient's postoperative course was uneventful.

Case # 2

A 53-year-old man was referred to our hospital because of exertional dyspnea. One year previously, he had sustained multiple traumas in a bus accident. The accident left him in a deep coma for 17 days, after which he suffered a stroke, right hemi-paresis, and aphasia due to infarction in the left temporal lobe. Also, he had fractures in the left femoral bone, left tibia bone, right hand, and two ribs on the superior right side. At the time, no cardiac symptoms were detected but the patient was subjected to splenectomy.

On admission, the electrocardiogram showed sinus rhythm, right bundle branch block with left-axis deviation. TTE revealed a normal LV size with mildly reduced LV systolic function, abnormal septal motion due to the RV volume overload, severe RV and RA enlargement with mild RV systolic dysfunction, and flail anterior tricuspid valve leaflet with severe low pressure TR. In addition, a large, mobile filamentous mass was detected on the tip of the flail leaflet, in favor of ruptured chordae. These findings were all confirmed by preoperative TEE, which also demonstrated an aneurismal inter-atrial septum with a small secondary type of atrial septal defect (ASD). Cardiac catheterization showed a small left-to-right shunt with no evidence of coronary artery disease.

Intraoperatively, the chest was explored via median sternotomy. CPB was instituted, the heart was arrested by cardioplegia, and the RA was explored. The anterior papillary muscle was ruptured, resulting in the prolapse of the anterior tricuspid leaflet. The De Vega technique was applied to the tricuspid annulus. Pledged 4/0 Prolene suture was used for the reconstruction of the anterior tricuspid valve papillary muscle to scar tissue in the base of the papillary muscle. Linear repair was thereafter performed for the closure of the ASD. Saline test was carried out next and the result was

so optimal that it obviated the need for TEE. Postoperative TTE revealed mild RV enlargement with mild systolic dysfunction. Doppler and contrast study showed mild TR and no residual shunt flow.

The patient made an uneventful recovery. One year after surgery now, the patient is well and asymptomatic with trivial TR on echocardiography.

Discussion

Traumatic TR is a rare sequela in the wake of blunt thoracic trauma. The most likely cause is compression, decompression, or deceleration of the thorax. These mechanisms give rise to intraventricular pressure peaks, which in combination with a closed valve and the systolic pressure in the RV most often affect the chordae tendineae and the papillary muscles.¹

TR frequently goes undiagnosed at blunt chest trauma because of its asymptomatic nature; sometimes several or even more than ten years elapse before a diagnosis is made.⁷⁻¹⁰ Nevertheless, severe TR eventually leads to right ventricular dilatation and progressive right heart failure, even if it is associated with no pulmonary hypertension.⁶ The clinical course of TR following blunt chest trauma varies largely. Little is known about the clinical course of isolated ruptured chordae tendineae. Indeed, the only detailed case to have been reported thus far is that by Kleikamp et al., whose patient developed TR due to ruptured chordae tendineae of the anterior leaflet seven years after a blunt chest trauma, which was surgically corrected.¹ It is deserving of note that the rupture of the chordae tendineae is believed to be the consequence of an acute increase in the RV pressure associated with a closed tricuspid valve.¹¹

Conclusions

The two cases presented herein demonstrate that the rupture of the chordae tendineae of the tricuspid valve could be another cause of TR following blunt chest trauma. This is, however, a condition that can be repaired surgically without the need for valve replacement. Advances in echocardiography have enabled earlier diagnosis and ergo more effective treatment.

We recommend that physicians working at emergency departments be on the alert for this potential complication of non-penetrating chest trauma and subject all patients admitted to the emergency department due to blunt chest trauma to TTE or TEE for accurate diagnosis.

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