A Case of Traumatic Tricuspid Valve Regurgitation Caused by Blunt Chest Trauma

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A case of severe tricuspid valve regurgitation caused by ruptured chordae tendineae secondary to blunt chest trauma from a motor vehicle accident is described. A transthoracic echocardiogram did not demonstrate these findings, which were clearly visualized on a transesophageal echocardiogram. We propose that patients with blunt chest trauma be considered for transesophageal echocardiography (unless surface images are of high quality) to evaluate not only the tricuspid valve apparatus but to examine other cardiac structures as early (ie, presymptomatic) treatment is preferable. (J Am Soc Echocardiogr 2007;20:198.e4-e5.)

Traumatic tricuspid valve regurgitation (TTR) is believed to be a rare complication of nonpenetrating chest trauma. High-speed automobile accidents are a leading cause of isolated tricuspid valve regurgitation (TR). 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] It is not uncommon for patients to present years beyond the trauma with symptomatic TR.[2] Advances in echocardiography have allowed easier diagnosis of this condition resulting in earlier and, hence, more effective treatment. However, transthoracic echocardiography (TTE) is often difficult in the patient with blunt chest trauma because of coexisting chest injuries. Transesophageal echocardiography (TEE) allows better visualization of much of the cardiac anatomy involved in TTR. We describe a case of severe TTR visualized by TEE but not appreciated by TTE in a patient who presented to the emergency department after blunt anterior chest wall trauma from a motor vehicle accident.

CASE REPORT

An 89-year-old man with a history of coronary artery disease status postcoronary artery bypass grafting with no significant TR previously documented presented to the emergency department with anterior chest wall tenderness after a motor vehicle accident in which he was a restrained passenger. Primary and secondary surveys revealed normal vital signs, symmetric rising of the chest with inspiration, and left-sided chest ecchymosis with compression tenderness. A murmur was not auscultated. A chest radiograph demonstrated unchanged cardiomegaly. Computed tomography of the chest demonstrated bilateral anterior rib fractures with small bilateral pleural effusions. The pericardium was intact and there was no pericardial effusion. He was admitted to the trauma intensive care department for monitoring.

His posttrauma course was complicated by atrial fibrillation with rapid ventricular response and *Staphylococcus aureus* bacteremia. Biomarkers were mildly positive for myocardial injury (troponin I = 4.35). This was thought to be a result of either a supply-demand mismatch from the atrial fibrillation with rapid ventricular response or of direct myocardial injury secondary to trauma, although no coronary angiogram was performed. An echocardiogram was ordered by the primary team. This study was suboptimal because of poor acoustic windows; however, all cardiac structures were identified. The left ventricular systolic function was normal. Mild TR was noted and there was a question of an echodensity on the atrial side of the tricuspid valve. This finding raised the suggestion of a vegetation. A TEE demonstrated a flail anterior tricuspid valve leaflet caused by chordal rupture with associated severe TR (Figure 1). The chordal structures prolapsed into the right atrium accounting for the echodensity present on the TTE. Because of the patient's debilitated state, he was deemed not to be a candidate for valve operation. The TR was well tolerated in the patient. He was later discharged to a skilled nursing facility in stable condition.

DISCUSSION

Cardiac injury resulting from blunt chest trauma encompasses a large range of presentations, including cardiac contusion, free wall rupture, septal rupture, and valvular disruption.[3] Although TTR is

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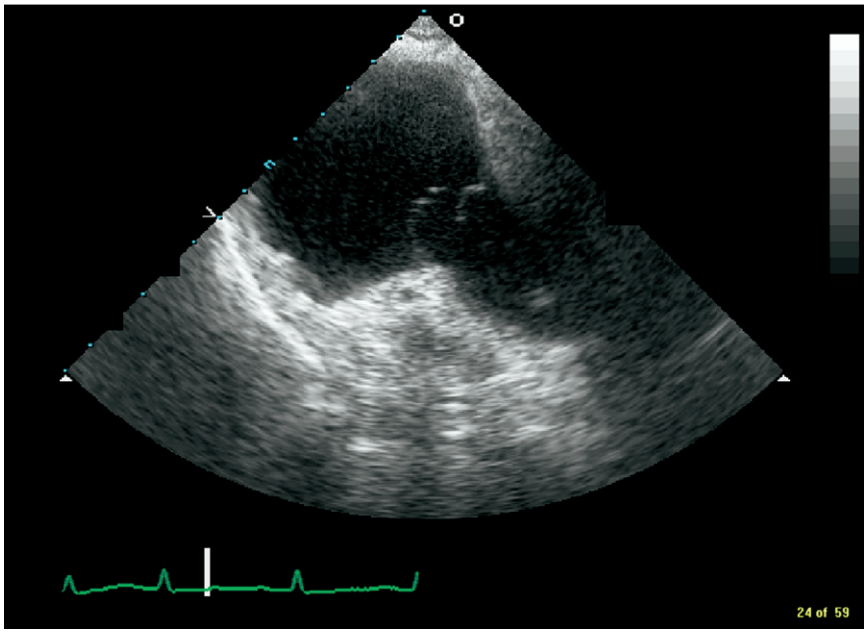

Figure 1 Transesophageal echocardiogram from midesophagus 45-degree angle showing flail anterior tricuspid valve leaflet caused by blunt thoracic trauma.

reported to be rare, its frequency is probably underestimated given the emphasis on other organ injuries. The right ventricle is immediately posterior to the sternum predisposing it to blunt trauma. Acute elevation of the right intraventricular pressure results in injury of the tricuspid valve apparatus.[2,4] The most frequently reported mechanism of injury is chordal rupture, followed by rupture of the anterior papillary muscle and leaflet tear, primarily of the anterior leaflet.[4]

The treatment for TTR is usually surgical, preferably valve repair. Although chordal rupture is associated with a more benign course extending from 10 to 25 years, papillary muscle rupture becomes symptomatic and requires operation, usually within weeks to months. The traditional indication for operation is symptomatic heart failure.[5] However, some investigators have proposed earlier intervention, before the development of irreversible right ventricular myocardial dysfunction, to facilitate valve repair rather than replacement.[2] Therefore, identification of a valve abnormality before development of symptomatic heart failure is optimal.

In our case report, the patient had blunt chest trauma caused by a motor vehicle accident. His elevated biomarkers in the setting of normal left ventricular systolic function likely reflected right ventricular injury as a result of cardiac contusion. Both the TR and chordal rupture were missed on the TTE. The incidence of this missed diagnosis may be higher than expected as many of these patients have poor acoustic windows resulting from trauma (eg, hematoma, pneumothorax) and treatment of these complications (eg, tubes, bandages). TEE could be considered in any patient with blunt chest trauma, particularly those where cardiac injury is suggested as the images are superior to TTE and the procedure is safe in trauma patients.

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SUPPLEMENTARY DATA

Supplementary data associated with this article can be found, in the online version, at 10.1016/j.echo.2006.09.003.