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CASE REPORT

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Traumatic tricuspid regurgitation following equine related blunt chest trauma and review of the literature

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Abstract

Myocardial injury following blunt chest trauma may be difficult to detect. We advocate for cardiac screening in such scenarios. Observation versus intervention should be based on symptoms and the degree of intracardiac disease.

KEYWORDS

blunt thoracic trauma, pediatric echocardiography, tricuspid regurgitation, tricuspid valve

1 | CLINICAL PRESENTATION

A patient was kicked in the chest by a horse with initial evaluation significant for chest pain, elevated troponins, and right bundle branch block. Echocardiogram demonstrated moderate tricuspid regurgitation secondary to an elongated anterior leaflet. Managed conservatively, serial echocardiograms demonstrated eventual resolution of tricuspid insufficiency and resumption of normal activities.

A 14-year-old healthy woman with no significant past medical history was seen at an outside emergency department 2 h after traumatic chest injury by a horse. She was kicked in the chest as she approached the animal from behind causing her to fall backward. She had a brief loss of consciousness following the initial trauma. She was seen in an outside emergency room with complaints of chest pain and chest contusion.

Vital signs at presentation were heart rate 95 beats/min, oxygen saturation 98%, respiratory rate 22 breaths/min, and blood pressure 120/61 mmHg. Chest X-ray showed normal lung fields, no bone or rib abnormalities, and normal cardiac silhouette. CT-angiography of the chest showed no pulmonary embolism and again normal lung fields, no rib fractures, and intact aortic arch. Initial troponin and creatinine-kinase MB (CK-MB) were 0.957 (normal value <= 0.003) and 6.3 (normal value <=0.00–2.37 ng/ml), respectively, 2 h after the injury. Electrocardiogram (ECG) showed a right bundle branch block (RBBB; Figure 1). Given the concern for myocardial injury, she was transferred to our center for further cardiac evaluation. She was hemodynamically stable, but physical examination was

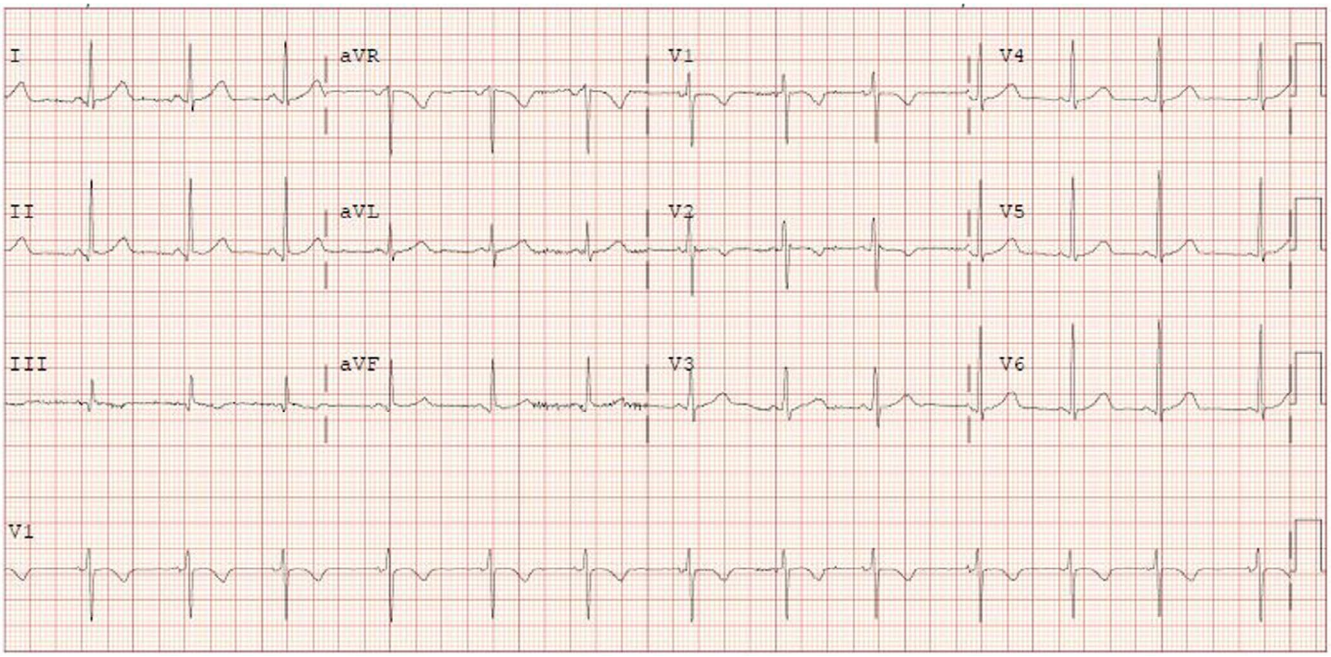

FIGURE 1 Initial ECG form outside hospital showing right bundle branch block

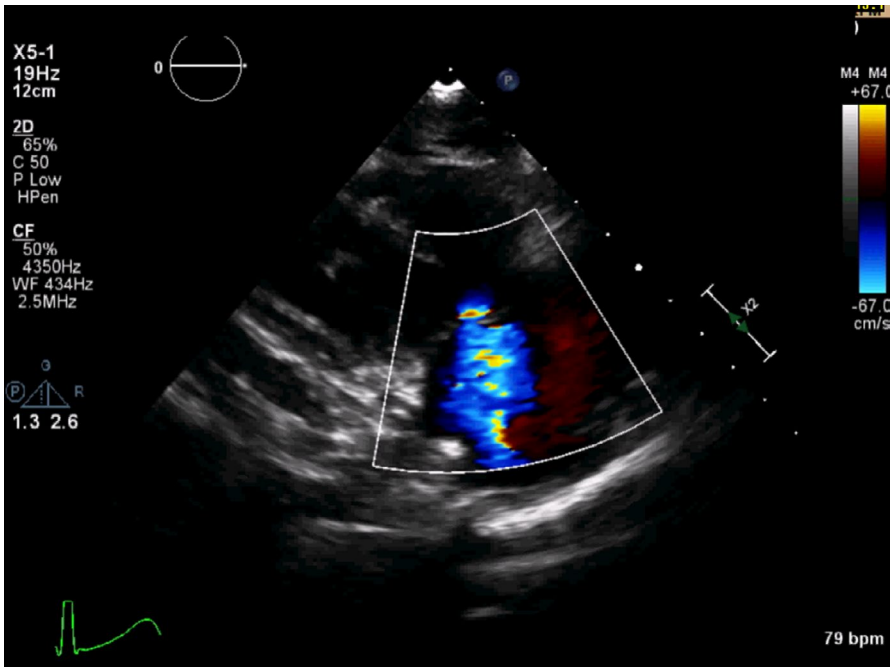

FIGURE 2 Parasternal long-axis image with posterior angulation showing the tricuspid valve with moderate regurgitation at initial presentation

significant for mild sternal bruising and tenderness with a low pitched I-II/VI holosystolic regurgitant murmur loudest at left-mid sternal border. Differential diagnoses entertained included myocardial contusion, myocardial rupture, traumatic valvar injury, and pericardial effusion.

Investigation at our institution included repeat ECG, which had normalized. Echocardiography demonstrated moderate tricuspid valve regurgitation (TR) with a peak tricuspid valve insufficiency gradient of 23 mmHg. Vena contracta measurement of 0.51 cm characterized tricuspid regurgitation as moderate, which agreed with subjective assessment.[1] The mechanism of TR was thought to be from elongation of chordae to the anterior leaflet of the tricuspid valve causing a prolapse of that leaflet (Figures 2 and 3). This created a regurgitant jet along the anterior leaflet with the largest regurgitant jet between the anterior and posterior leaflets. Right and left ventricular systolic wall motion were normal. She was admitted to the cardiology service for observation in setting of chest pain with new tricuspid regurgitation. The highest troponin and CKMB of 1.22 and 10.54 were recorded hours later at our institution. Troponins down trended during her inpatient admission prior to discharge.

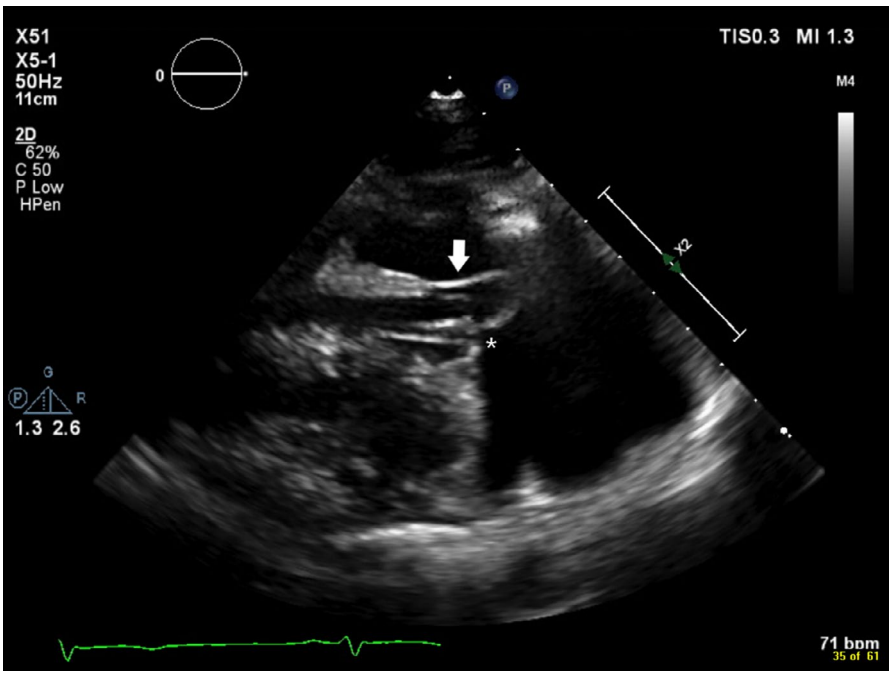

FIGURE 3 Parasternal long-axis image demonstrating hyperechoic chordal tissue (arrow) and coaptation defect (*) at initial presentation

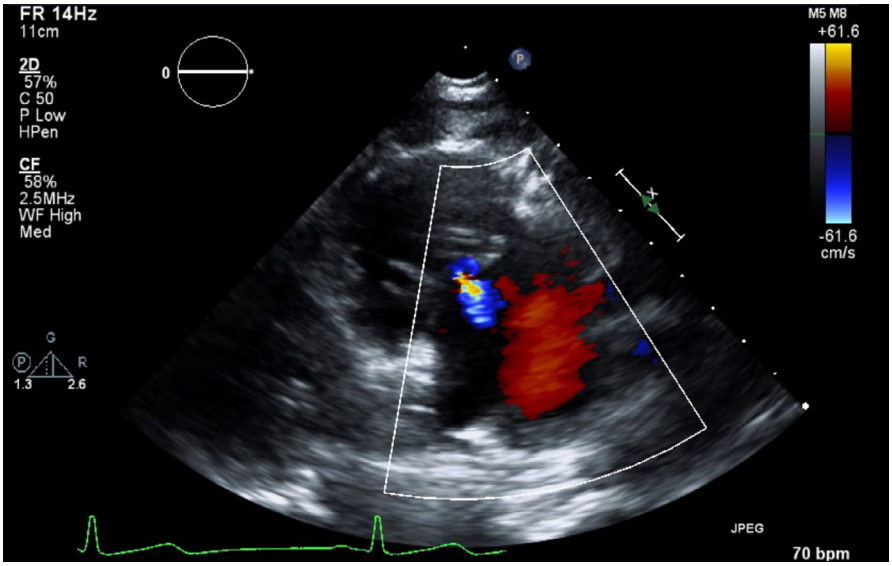

FIGURE 4 Parasternal long-axis image with posterior angulation 3 weeks after trauma showing tricuspid valve with mild regurgitation

2 | MANAGEMENT

She was managed conservatively with NSAIDs for pain control, serial echocardiography to evaluate competency of the tricuspid valve, and repeat ECG. After an uneventful overnight observation, she was discharged home once pain had been adequately controlled and laboratory-work showed down-trending troponins. Her echo at the time of discharge demonstrated moderate tricuspid regurgitation, normal right ventricular systolic wall motion, and no other intracardiac abnormalities.

She was followed in the outpatient setting with serial echocardiograms (Figures 4 and 5), which demonstrated improvement in tricuspid regurgitation. She now has physiologic TR, and she is back to full activity without limitations 3 months following her initial presentation.

3 | DISCUSSION

Blunt chest trauma can be associated with intracardiac injuries and is a significant cause of morbidity and mortality. Infrequently, the blunt chest trauma leads to myocardial contusion. The exact incidence of myocardial contusion in patients with blunt chest trauma is difficult to estimate as diagnostic criteria vary between institutions, but has been reported between 10% and 70%.[2] Myocardial contusion may predispose a patient to hypotension and arrhythmia. Arrhythmia typically occurs between 24 and 48 h after the injury.[2,3]

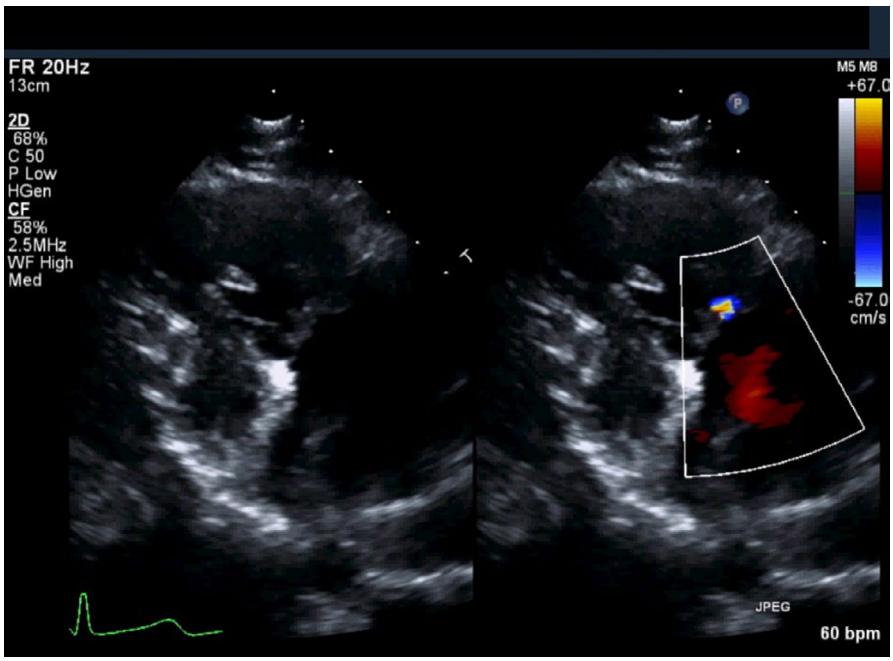

FIGURE 5 Parasternal long-axis image with posterior angulation 3 months after trauma showing tricuspid valve with physiologic regurgitation

The mechanism of injury relates to the sudden deceleration forces and compression within the chest cavity. Increases in intravascular pressure due to the mechanical compression may also contribute to injury. Among the intracardiac structures, the right ventricle (RV), including the tricuspid valve and the papillary muscles, appears to be more prone to injury. The right ventricle sits anteriorly within the thoracic cavity; therefore, it receives the full impact of blunt chest trauma. The low-pressure system also ill prepares the RV for sudden elevations of intraventricular pressure. This puts the tricuspid valve and its apparatus at increased risk of injury.[4] Other intracardiac injuries including mitral and valve injury, intraventricular septum rupture, and aortic transection have been reported.[5-7] Ismailov et al[8] in a systematic review highlighted both acute and chronic sequelae with blunt chest injuries. In their population, tricuspid regurgitation occurred with 12-fold increase followed second by aortic regurgitation with a 3.4-fold increase. Tricuspid regurgitation is quantified in the adult population with measures such as vena contracta, regurgitant jet area, and effective regurgitant orifice areas; however, these have not been well validated in children and thus have limited utility leading the pediatric cardiologist to be dependent on subjective assessment.[1]

The diagnostic approach to patients with mild-to-moderate blunt chest trauma can be challenging. Alborzi et al. recommend obtaining ECG and troponin in all blunt chest trauma patients to serve as an initial screen as patients may be asymptomatic from a cardiac perspective or could have more severe distracting injuries at the time of presentation. CK-MB can be used as a secondary test though it may be affected by traumatic muscular injury as due to its non-specificity in inflammatory conditions.[9,10] Overall, troponin levels may be more useful compared to other conventional markers in myocardial injury and could help with stratifying patients with respect to clinical severity and outcome.[3]

Transthoracic echocardiography (TTE) is helpful in identifying global and regional wall motion abnormalities, valvar injuries, and associated intracardiac damage. In patients with associated pulmonary injuries, such as hemothorax, pneumothorax, and pulmonary edema, poor acoustic windows could obscure intracardiac details. In such cases, transesophageal echocardiography may be useful to delineate the etiology in valvular pathology.[10] In this case, the patient was able to tolerate the transthoracic study and had adequate sonographic windows to identify the etiology of her tricuspid regurgitation. Thus, a more invasive modality was not required.

Intervention versus conservative management, in the setting of isolated tricuspid valve trauma, must be based on the presence of clinical symptoms, degree of damage to the tricuspid valve apparatus, and etiology of tricuspid valve injury. The latter two criteria will likely determine chance of recovery and thus impact the need for intervention. Assessment of the right ventricular function should also be included in serial echocardiograms as declining function may warrant intervention. RV dysfunction at the time of presentation may be secondary to cardiac contusion and thus may improve with temporal distance from the inciting event. Severe RV dysfunction in the setting of severe tricuspid regurgitation that is unlikely to improve should prompt surgical evaluation. Conservative management could be an option for patients who are hemodynamically stable and without severe intracardiac disease, as in this case. Patients without intervention at presentation should have longitudinal cardiology follow-up in the

setting of persistent tricuspid regurgitation to monitor for secondary right-sided chamber enlargement, subsequent change in right ventricular function, or development of clinical symptoms which may warrant intervention particularly in those with persistent lesions. Most studies have demonstrated persistent and progressive valvar injuries following blunt chest trauma require surgical intervention.[5,7,11,12]

In the case above, the mechanism of trauma is such that it might not raise suspicion for intracardiac injury. Modest elevation of troponin and right bundle branch block pattern on ECG necessitated formal cardiac evaluation. Elongation of the chordal tissue caused tricuspid valve prolapse and resultant tricuspid regurgitation. As the inflammation of the chordal tissue was able to remit, the coaptation improved and the TR dissipated.

4 | CONCLUSION

Blunt chest injuries can cause significant cardiovascular morbidity and mortality. Most often the mechanism involved may be insignificant to alert providers to consider significant myocardial injury. Therefore, we propose that medical providers maintain a high index of suspicion for intracardiac injury following blunt chest trauma, even in those with hemodynamic stability at presentation, to identify and expedite assessment of patients at significant risk.

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CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

SS and BA performed the literature review, drafted the manuscript, and critically revised the manuscript in preparation for submission. EF critically reviewed and edited the manuscript in preparation for submission.

ETHICAL AND CONSENT STATEMENTS

The study conforms to recognized standards for ethical research as outlined in US Federal Policy for the Protection of Human Subjects. Consent was obtained for the clinical case and images to be included in this report.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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