Drugged Driving: A Case of Traumatic Tricuspid Regurgitation in an Inveterate Intravenous Drug User

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**Abstract**

Traumatic tricuspid insufficiency, a rare complication after blunt chest trauma, may be underdiagnosed. Given injuries that demand more immediate attention, variability of presentation, and frequent initial tolerability, acute tricuspid regurgitation can be overlooked. Delays in recognition can lead to subsequent right ventricular deterioration before appropriate intervention can occur. We present a case of an intravenous drug user who sustained multiple injuries in a drug intoxication related motor vehicle accident. Two weeks after the accident, he was found to have severe tricuspid regurgitation secondary to traumatic chordae rupture only because repeat echocardiography was performed to follow up a cardiac contusion. The combination of enhanced awareness of the possibility of tricuspid valve damage, a careful examination, and echocardiography can help assure proper diagnosis and follow up. Drugged driving can lead to traumatic tricuspid injury while inveterate drug use limits therapeutic options.

**INTRODUCTION**

Motor vehicle accidents are the leading cause of traumatic tricuspid valve regurgitation [1]. Due to progressive inclusion of echocardiography following chest trauma, there has been an increased frequency of reporting tricuspid valve damage [2]. However, during the acute phase of injury, the traumatic lesion may go undetected due to the presence of other more apparent thoracic and orthopedic injuries. Additionally, given the initial tolerability of traumatic tricuspid regurgitation in patients with otherwise healthy hearts [3], traumatic tricuspid regurgitation can be easily overlooked. Optimal timing of tricuspid valve intervention depends upon close follow up of a patient’s symptoms, examination, and quantified echo parameters. A lack of an early diagnosis reduces the likelihood of ideal follow up. Inveterate drug abuse complicates treatment decisions and follow up even more.

**CASE REPORT**

A 34 year old male with a history of ongoing intravenous drug use (IVDU) was brought to an outside hospital emergency department following a motor vehicle accident, in which he was not wearing a seatbelt and due to significant opioid intoxication was unable to remember details of the accident. Upon arrival, his temperature was 37.5 degrees Celsius, with a heart rate of 175 bpm, respiration rate of 28 bpm, blood pressure 124/78 mmHg, and pulse oximetry of 96% on room air. On the Glasgow Coma Scale, his score was 14. He had an obvious deformity in the left distal thigh and no obvious deformity in the chest. Cardiac exam revealed a soft systolic murmur, and accentuation with inspiration was not noted. Jugular venous pressure (JVP) or signs of right heart failure were not evaluated. The electrocardiogram at admission showed sinus tachycardia with right axis deviation. Chest radiograph showed mediastinal widening, multiple rib fractures bilaterally, and a small right pneumothorax. Computed tomography of the chest, abdomen and pelvis showed aortic injury with focal outpouching of the medial aspect of the aortic arch, near the origin of the left subclavian artery with a hyperdense periaortic hematoma, bilateral rib fractures, grade II renal laceration, grade II splenic laceration, right distal acetabular posterior wall fracture, and left complex distal femur fracture. The patient was intubated and sedated for pain control and transferred to a tertiary care hospital.

Upon arrival at the tertiary hospital, he was noted to have a heart rate of 121 bpm and blood pressure of 115/63 mmHg, equal in both arms. Examination revealed a III/VI systolic murmur, with respiratory rate of 28 bpm, blood pressure 124/78 mmHg, and pulse oximetry of 96% on room air. The Glasgow Coma Scale, his score was 14. He had an obvious deformity in the left distal thigh and no obvious deformity in the chest. Cardiac exam revealed a soft systolic murmur, and accentuation with inspiration was not noted. Jugular venous pressure (JVP) or signs of right heart failure were not evaluated. The electrocardiogram at admission showed sinus tachycardia with right axis deviation. Chest radiograph showed mediastinal widening, multiple rib fractures bilaterally, and a small right pneumothorax. Computed tomography of the chest, abdomen and pelvis showed aortic injury with focal outpouching of the medial aspect of the aortic arch, near the origin of the left subclavian artery with a hyperdense periaortic hematoma, bilateral rib fractures, grade II renal laceration, grade II splenic laceration, right distal acetabular posterior wall fracture, and left complex distal femur fracture. The patient was intubated and sedated for pain control and transferred to a tertiary care hospital.

left ventricular systolic function with an ejection fraction of 45%, with mild septal hypokinesis. Right ventricular size was normal and systolic function was at lower limits of normal with mild to moderate tricuspid regurgitation.

The patient was taken to the operating room and underwent an open reduction internal fixation of the left femur. Seven days later, he underwent left carotid-subclavian bypass, and then endovascular repair of a thoracic aortic pseudoaneurysm. Two days later, he underwent right acetabular fixation. He was successfully extubated and transferred to the floor.

TTE, repeated 15 days after admission to reevaluate suspected myocardial contusion, showed a borderline dilated left ventricular cavity with an ejection fraction just below normal, 49%. The right ventricular cavity was mildly dilated, with diastolic flattening of the interventricular septum consistent with right ventricular volume overload as well as systolic flattening of the interventricular septum, consistent with right ventricular pressure overload. The right atrium was dilated and the inferior vena cava was noted to have enlarged from 1.2-1.9 to greater than 2.1 cm, without hepatic vein systolic flow reversal. The right ventricular diameter in the parasternal short view had increased from 3.1 to 3.9 cm, with an increase in end systolic area from 7.50 cm² to 13.65 cm², with a percent RV area change of 44% (Table 1). RV systolic pressures were estimated at 54 mmHg. Right ventricular ejection fraction was noted to have decreased slightly, despite the severe tricuspid regurgitation, but there was a preserved tricuspid annular plane systolic excursion of 2.6 cm (Table 1). A highly mobile tip of the anterior tricuspid leaflet was noted consistent with anterior leaflet chordae tendineae rupture (Figure 1). Color-flow Doppler confirmed severe, septally directed tricuspid regurgitation (Figure 2). Detailed exam at that time was notable for flat neck veins, clear lungs, a hyperdynamic precordium, and a nondisplaced PMI. There was a systolic flow murmur that was heard from the apex to the base, which decreased with Valsalva; and there was a tricuspid regurgitation murmur, III/VI, heard at the lower left sternal border and laterally, which incremented with inspiration and Valsalva. The liver was nonpulsatile and nondisplaced. The patient was started on spironolactone and discharged to a rehabilitation facility on hospital day 22 without dyspnea or signs of right-side heart failure.

DISCUSSION

The first case of nonpenetrating traumatic tricuspid insufficiency was reported in 1848 by Todd et al. [4], The

| Table 1: Initial and repeat right ventricular echocardiographic data. |
|-----------------------|----------------------|----------------------|
|                      | Initial TTE          | Repeat TTE           |
| RV End Systolic Area (cm²) | 7.50                | 13.65                |
| RV End Diastolic Area (cm²) | 14.95               | 24.40                |
| Per cent area change (%)   | 50                   | 44                   |
| TAPSE (cm)               | Not performed        | 2.6                  |
| S’ Annular Velocity (cm/s) | Not performed        | 16.0                 |

Abbreviations: RV: Right Ventricle; TAPSE: Tricuspid Annular Plane Excursion.
mechanism of injury is uncertain, though the most accepted theory has been termed “blow out” injury: anteroposterior compression of the right ventricle from the adjacent sternum, particularly in end-diastole when hydrostatic pressure is increased, produces a regurgitant jet which can precipitate rupture of a papillary muscle and/or chordae tendineae [5,6].

The most frequently reported injury is chordal rupture, followed by anterior papillary muscle rupture and leaflet tear [7]. The lack of redundancy of chordal attachments to the tricuspid leaflets adds to the vulnerability of the tricuspid valve to chordal rupture. The presentation of traumatic tricuspid valve injury is heterogeneous based on the spectrum of injury with chordal rupture usually allowing for preserved valve function with a more subacute presentation versus immediate insufficiency and acute right sided heart failure symptoms in papillary muscle rupture [8-10].

As in our patient, traumatic tricuspid injury can be missed in the acute phase due to more apparent and life-threatening orthopedic fractures, abdominal and thoracic lesions necessitating urgent surgery. After initial stabilization, given the clinical subtlety associated with tricuspid regurgitation, tricuspid valve injury again is often overlooked [11,12]. Previous case reports, similar to ours, have also found that this injury can go unnoticed until incidentally discovered during assessment of other more apparent trauma [13]. In our patient, follow up echocardiography was performed to reevaluate the slightly depressed LVEF found on initial TTE, thought secondary to cardiac contusion. Otherwise, his traumatic valvular lesion could have been missed. Routine and early TTE following blunt chest trauma has been advocated [2,14-17]. Furthermore, given that co-existing chest wall injuries can result in poor initial echocardiographic windows, as likely was the case in our patient, repeat TTE can be particularly useful, with careful attention to early changes in RV diameter and percent area change on repeat imaging.

Even with availability of echocardiography, the cardiac physical exam can be important in the acute setting, despite the presence of distracting injuries. Our patient was vaguely noted on admission to have cardiac findings consistent with tricuspid regurgitation. Though inspiratory incrementation of a systolic murmur may only occur in 20-60% of patients with important TR, the finding can be highly specific. The increment in the murmur with Valsalva was interesting, and though usually associated with HOCM or MVP, can be associated with TVP or in this case TV avulsion. More heightened suspicion in our patient with blunt chest trauma and more careful attention to the cardiac exam could have led to a diagnosis, even if a contusion was not being followed up with echocardiography.

Lack of suspicion of this clinical entity in patients with blunt chest trauma and multiple traumatic injuries can lead to delays in diagnosis, less than optimal follow up, and non ideal options for subsequent treatment, particularly with more concealed lesions such as chordal rupture. The time interval to diagnosis has been found to be protracted to over 10 years in many cases, with a median duration between trauma and operative intervention of 17 years [18,19].

Given that repair of the tricuspid valve is preferable to replacement, and that leaflets can become retracted and atrophic with time, thus making valve repair more complicated, prompt recognition is imperative. Furthermore, tricuspid valve intervention should be performed before development of irreversible RV dysfunction and at the earliest signs of a drop in RV percent area change, an alteration in end systolic area, or a decrease in tricuspid annular systolic plane excursion [18-22]. Earlier diagnosis and appropriate follow up and treatment can be achieved by heightened suspicion, close cardiac exam and careful echocardiography.

Therapeutic options can be limited by addiction. In view of the severe TR and the early RV and RA dilation, in addition to a
compromised left side with pulmonary hypertension that would further strain the RV and worsen the TR; there was a consideration for attention to the tricuspid valve, with annuloplasty or bicuspidization [23]. However, given the patient’s professed lack of interest in drug rehabilitation and the appropriate concern for the introduction of any right-sided foreign material, the patient was not offered surgery.

Last, our patient’s case is unusual given his reported drug abuse immediately prior to his motor vehicle accident. Drugged driving remains an important public health problem in the United States, with over 10.1 million people driving under the influence of illicit drugs in 2014 [24]. Opioid use has been shown to cause significant impairment of driving behavior [25], putting drugged drivers at risk for blunt chest trauma and injuries such as seen in our patient. No other case reports of traumatic tricuspid valvular lesions have included patients with IVDU.

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REFERENCES