Traumatic Tricuspid Insufficiency with Chordae Tendinae Rupture: A Case Report and Literature Review

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With the increase in the number of automobile accidents, traumatic tricuspid insufficiency, a rare complication of non-penetrating blunt chest injury, has become an important problem. This kind of injury has been found more frequently during the last decade, partly because of better diagnostic procedures and a better understanding of the pathology. Here, we report a 22-year-old male patient who suffered chest trauma from an automobile accident. Echocardiography demonstrated tricuspid chordae tendinae rupture with remarkable tricuspid regurgitation. We discuss this case in comparison with the previous literature. This case reminds us that physicians in the emergency department should be aware of this potential complication following non-penetrating chest trauma.

Key Words: chordae tendinae rupture, trauma, tricuspid regurgitation
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Valvular lesions following blunt thoracic injury are uncommon. Tricuspid insufficiency is thought to be a rare complication of blunt, non-penetrating chest trauma [1]. However, it has been reported with increasing frequency during the last decade, which may be due to the increase in the number of high-speed automobile accidents, better diagnostic procedures, and a better understanding of the pathology [2,3]. Here, we report a 22-year-old male patient who developed tricuspid chordae tendinae rupture with remarkable valvular insufficiency, identified by transthoracic echocardiography (TTE), after blunt chest trauma sustained in a traffic accident.

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Case Presentation

A 22-year-old male without previous cardiac or medical history was sent to our emergency department following a traffic accident in February 2006. The car's front end had crashed against a tree. His chest was forcefully propelled into the steering wheel. He had cerebral contusion and immediately lost consciousness. Endotracheal intubation was immediately done in the emergency department for airway protection.

Vital signs in the emergency room were blood pressure 124/98 mmHg, pulse rate 130/minute, and body temperature 36.7°C. Physical examination revealed grade III pansystolic murmur over the left lower sternal border, with breathing sounds decreased over the left chest. He had no lower leg edema. Twelve-lead electrocardiography revealed sinus tachycardia. Chest X-ray showed hemopneumothorax in the left chest and a chest tube was inserted immediately. TTE revealed no pericardial effusion. He had a Glasgow

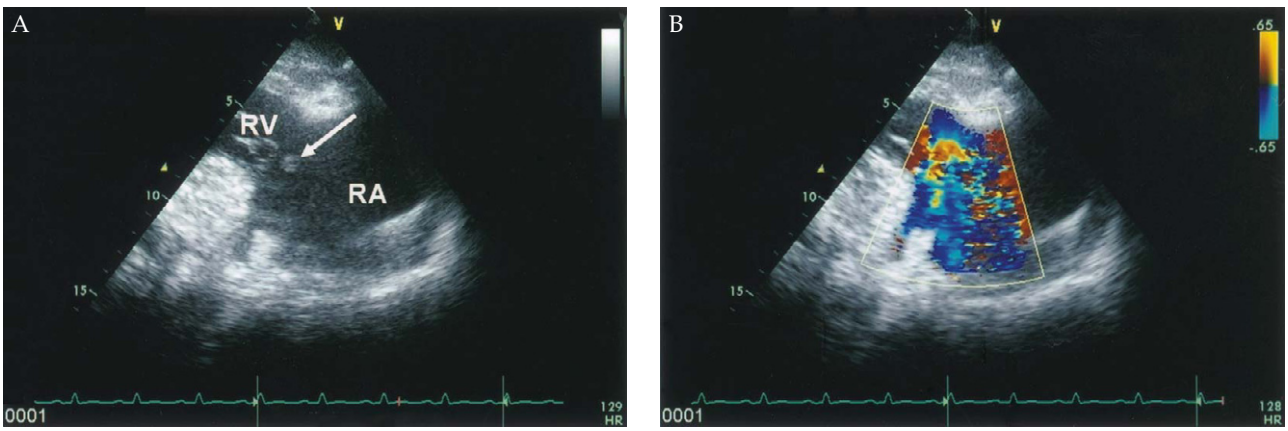

Figure. *Echocardiographic right ventricular inflow view shows: (A) tricuspid chordae tendinae rupture with leaflet tip into the right atrium (RA) (arrow); and (B) tricuspid regurgitation. RV = right ventricle.*

coma scale of $E_1V_1M_4$, multiple rib fractures, and multiple lacerations over the face, trunk and limbs. Blood examinations showed that creatine kinase (CK), CK-MB, and troponin I were 497 U/L (normal range, 26–174 U/L), 4.2 U/L (normal range, 2.3–9.5 U/L), and 0.435 ng/mL (normal range, < 0.04 ng/mL), respectively. He was then admitted to the intensive care unit for further evaluation and management.

On the 2nd day of hospitalization, the endotracheal tube was removed successfully. Because of persistent pansystolic murmur and tachycardia, TTE was arranged to evaluate the cardiac lesion and it revealed failure of the tricuspid leaflet coaptation with rapid systolic movement of the leaflet tip in the right atrium, which is consistent with tricuspid chordae tendinae rupture (Figure A). Color Doppler echocardiographic imaging showed remarkable, eccentric tricuspid regurgitation (Figure B). During admission, physical examination had disclosed jaundice and bilirubin 3.45/1.39 mg/dL (total/direct; normal range, 0.1–1.0/0–0.2 mg/dL). He received conservative treatment with a smooth course. After 19 days of hospitalization, he was discharged uneventfully without dyspnea or signs of right-side heart failure. He had class I New York Heart Association (NYHA) cardiac function and received close follow-up in our outpatient department.

Discussion

Cardiac injury resulting from blunt, non-penetrating chest trauma has been seen more frequently during the last decade [3]. Although traumatic tricuspid insufficiency is a relatively uncommon lesion, its frequency is probably underestimated [1,4]. The diagnosis is difficult because of the slow progression of this pathology and the presence of more clinically acute lesions. Non-penetrating chest trauma is responsible for 90% of cases [1].

The right ventricle is immediately behind the sternum, predisposing it to an anteroposterior compression type of injury, especially during the end diastolic phase [5]. The decelerating force in the right ventricular chamber produces a regurgitation, thereby initiating rupture of the papillary muscle and/or chordae tendinae. The usual lesion observed at surgery is subvalvular rupture of the anterior papillary muscle [6]. Tricuspid leaflet prolapse, rupture of the chordae tendinae, and leaflet rupture have also been reported [7,8]. TTE is the investigation of choice for diagnosing associated lesions and for assessing the mechanism of tricuspid regurgitation because the tricuspid leaflet is located anteriorly [1]. Transesophageal echocardiography (TEE), which can further confirm the TTE findings and optimize the anatomic evaluation of the valvular apparatus in patients with poor TTE window, is an invasive procedure and usually used as a second line examination [9].

The clinical course of tricuspid regurgitation following blunt chest trauma varies greatly [10]. The mean interval to diagnosis is long (11 years) [6]. The delay before diagnosis may be explained by the diversity of lesions of the tricuspid valve, associated cardiac disease, and the request of echocardiography. Rupture of papillary muscle is associated with severe and acute symptoms, which are more frequently diagnosed

earlier [11]. Mild symptoms usually appear in patients with a rupture of the leaflet or chordae tendinae. Optimal treatment for this condition is still controversial and ranges from long-term medical therapy to early surgical correction [12]. However, the operation should be undertaken before right ventricular myocardial dysfunction. Since post-traumatic tricuspid regurgitation is effectively correctable with reparative techniques, early operation is also considered to relieve symptoms and to prevent right ventricular dysfunction [12]. In a recent Chinese population study, patients with NYHA classes II–IV received benefit from earlier surgery; however, the intervals between trauma and operation ranged from 1 month to 20 years (mean, 52.5 ± 80.3 months) [13]. Surgical indications are also difficult to determine and depend on the patients' symptoms and the type of anatomic lesions [1,6,14].

In the case reported here, a healthy young male suffered from blunt, non-penetrating chest trauma from a traffic accident. Tricuspid chordae tendinae rupture was identified by TTE. Because conservative treatment resulted in an uneventful clinical course (NYHA class I), he did not receive surgical intervention. Long-term follow-up was mandatory to monitor his symptoms and right ventricular function. This case highlights the fact that physicians in emergency departments should be aware of this potential complication of non-penetrating chest trauma. Patients should be triaged early on the basis of physical examination, symptoms, and echocardiographic examination. Although good functional results can be achieved many years after the onset of traumatic tricuspid valve insufficiency, earlier diagnosis and surgical treatment may increase the feasibility of tricuspid valve repair, prevent progressive deterioration of right ventricular function, and increase the possibility of maintaining late sinus rhythm [15].

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外傷性三尖瓣閉鎖不全合併腱索斷裂－病例報告及文獻回顧

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隨著高速撞擊車禍的數目增加，外傷性三尖瓣閉鎖不全，這種非穿刺性胸部挫傷造成的罕見合併症已經成為重要的課題。這種形式的傷害近幾十年來較為普及，部份是因為診斷工具的改進以及對其病理學上有更進一步的瞭解。我們報告一位二十二歲年輕男性在一場車輛當中遭受胸部挫傷。心臟超音波證實三尖瓣腱索斷裂及明顯的三尖瓣逆流。我們討論此病例並且和之前的文獻做一番比較。這個病例亦提醒我們急診醫生應警覺到非穿刺性胸部挫傷造成三尖瓣閉鎖不全的潛在危險性。

關鍵詞：腱索斷裂，外傷，三尖瓣逆流
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