CASE REPORT
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Asymptomatic Isolate Tricuspid Regurgitation with Chordae Tendineae Rupture Caused by Blunt Chest Injury

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The incidence and importance of tricuspid valve regurgitation after a blunt chest injury has risen with the increase in the number of automobile accidents and steering wheel traumas. This kind of injury has been reported more frequently in the last decade because of the better diagnostic procedures and understanding of the pathology. However, tricuspid valve regurgitation following a blunt chest injury can still be easily missed because most patients do not show symptoms at the time of the trauma. A 55-year-old male patient presented himself at our facility after suffering a chest injury from an automobile accident. His transthoracic echocardiography (TTE) revealed severe tricuspid valve regurgitation due to the prolapse of his anterior valve leaflet. We report a case of asymptomatic tricuspid regurgitation that developed after a blunt chest injury.

Key Words: Tricuspid regurgitation, Blunt injury, Transthoracic echocardiography, Chordae tendineae

INTRODUCTION

Tricuspid regurgitation is thought to be a very rare complication of blunt, non-penetrating chest injury.[1] The diagnosis may be delayed or missed, because of its rarity, lack of acute physical findings, and the presence of co-existing more urgent issues.[2] 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.[3] Nevertheless, the real prevalence of traumatic tricuspid regurgitation is probably underestimated because the chronic isolated tricuspid insufficiency is usually well tolerated and most patients experience few or no symptoms early on after the injury.[2] Here, we report a case of severe tricuspid regurgitation which was developed after blunt chest injury by a traffic accident.

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CASE

A 55-year-old male was transferred to our hospital following a traffic accident in July 2011. The patient had 15 pack years of smoking history, without diabetes, hypertension, endocarditis, thrombosis, cardiac disease and medication history. At the time of the traffic accident, he was driving the vehicle and was not wearing a seatbelt. His anterior chest was struck by the steering wheel forcefully. He was diagnosed and treated for bilateral multiple rib fracture, cerebral contusion, cervical sprain, mesenteric laceration, splenic rupture and left acetabular posterior wall fracture in another medical center. Cardiac echocardiography was not performed at the time of initial presentation. The left acetabular posterior wall fracture was required surgical treatment. The other problems have improved with conservative treatment.

Vital signs were stable. Blood pressure was 140/90 mm Hg, pulse rate was 95 beats per minute, body temperature was 36.7℃. Physical examination revealed grade III pansystolic murmur over the left lower sternal border. There were nothing visible injury on the chest. He did not complain of chest discomfort or dyspnea, but complained of tenderness on the right upper chest. There were no signs of right heart

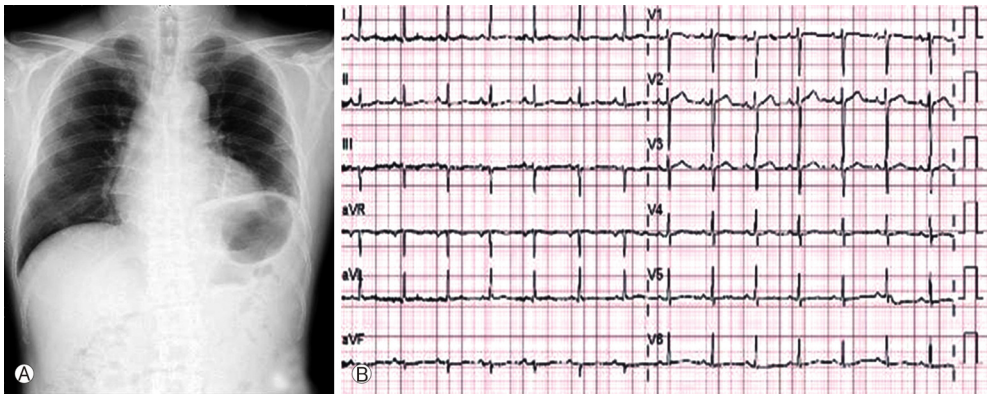

Fig. 1. (A) Chest X-ray of the patient with isolated traumatic rupture of the tricuspid valve. There are fractures on right 6th and 7th rib. (B) Twelve-lead electrocardiography shows normal sinus rhythm with left axis deviation.

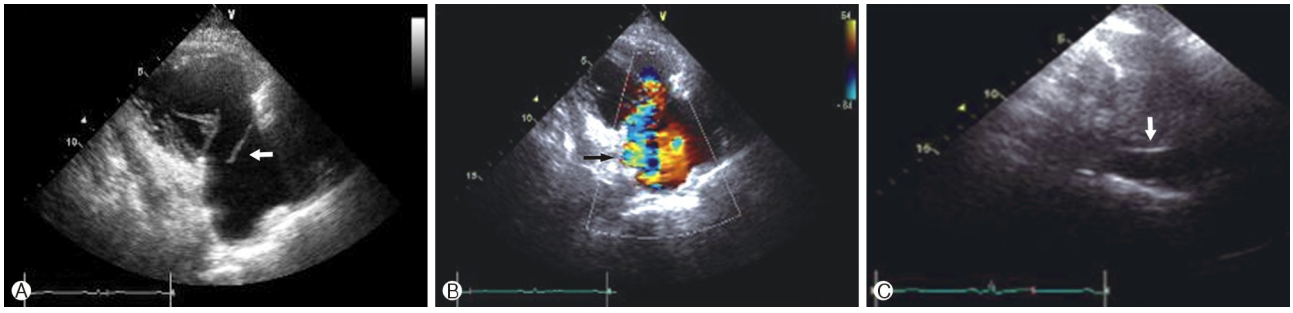

Fig. 2. (A) Echocardiographic right ventricular inflow view shows tricuspid valve chordae tendineae rupture with the prolapsed of the anterior leaflet tip into the right atrium (white arrow). (B) The color-flow Doppler transthoracic echocardiography showed severe tricuspid regurgitation (black arrow). (C) In the echocardiography, distended inferior vena cava is checked (white arrow).

failure. Laboratory tests showed that his hemoglobin, CPK-MB and Troponin I were 15 g/dL, 1.1 ng/mLand 0.02 ng/mL respectively. Chest X-ray showed right 6th and 7th rib fractures, twelve-lead electrocardiography (ECG) revealed sinus rhythm with left axis deviation (Fig. 1).

Because of persistent pansystolic murmur and for his preoperative evaluation, a TTE was performed to evaluate any cardiac lesion. The echocardiogram revealed incomplete coaptation of tricuspid leaflets with rapid systolic movement of the leaflet tip into the right atrium, which was consistent with tricuspid valve chordae tendineae rupture. A distended inferior vena cava (IVC) was also evident (Fig. 2). Color Doppler echocardiography showed severe tricuspid valve regurgitation and the peak pressure gradient of the tricuspid valve was 29.8 mm Hg (Fig. 2). There was no evidence of atrioventricular septal defect and the left ventricular systolic function was normal. A cardiac computed tomography (CT) was performed for confirmation and it showed ruptured tricuspid valve chordae tendineae with systolic valve regurgitation (Fig. 3). No coronary artery abnormality was seen.

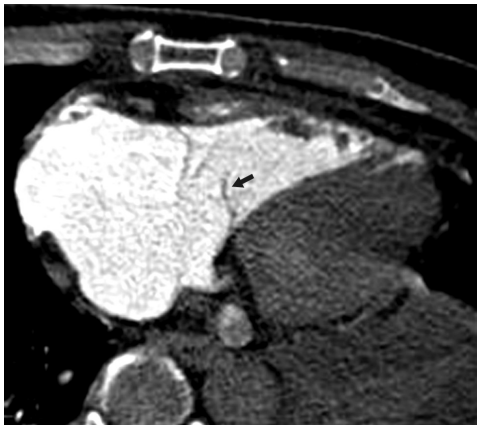

Fig. 3. The cardiac computed tomography shows chordae tendineae rupture with the prolapsed of the anterior leaflet (black arrow).

We opted to observe the tricuspid regurgitation without any surgical treatment since he was not showing any signs of dyspnea, chest discomfort and lower leg pitting edema

during the hospitalization. He had surgery for the correction of left acetabular posterior wall fracture under spinal anesthesia and there was no specific complication. We advised the patient to be repaired the tricuspid valve and consulted to the department of thoracic and cardiovascular surgery, but the patient refused surgery. He was discharged after 36 days of hospitalization, without dyspnea or signs of right-side heart failure.

He had dyspnea of NYHA functional class I and received close follow-up in our outpatient clinic. One year after the accident, the follow-up chest X-ray and twelve-lead ECG revealed no notable changes compared to the previous examinations. When evaluated according to the guidelines for the echocardiographic assessment of the right heart in adults,[4] a follow up TTE showed a more dilated right atrium and right ventricle, but peak pressure gradient of the tricuspid valve and the right ventricle were well preserved (Table 1). The patient is in good condition continues to be asymptomatic and is being followed up with serial echocardiographic examination.

Table 1. Right heart structure and function of the patient[4]

Variable	July 2011	October 2012
RV basal diameter	33.6 mm	38.8 mm
RV mid cavity diameter	25.5 mm	29.7 mm
RV longitudinal diameter	50.6 mm	59.3 mm
RA major dimension	49.5 mm	51.2 mm
RA minor dimension	46.3 mm	48.0 mm
RVOT proximal diameter	36.7 mm	39.0 mm
RV fractional area change	44%	45%
Peak pressure gradient of tricuspid valve	29.8 mm Hg	28.5 mm Hg

DISCUSSION

Cardiac injury resulting from blunt, non-penetrating chest injury has been seen more frequently during the last decade.[3] But symptomatic cardiac injury due to blunt chest injury is very rare, and tricuspid regurgitation accounts for less than 5% of all cases.[5] The diagnosis of this pathology is difficult because of its slow progression and atypical or even asymptomatic clinical presentation. Its incidence rate may be underestimated because of above reasons.[1,2]

In our case, right ventricular dimension was not increased and the chordae tendineae rupture was demonstrated on TTE. The patient's chest X-ray showed no cardiomegaly and signs of right heart failure were not detected on physical examination. Therefore, we concluded that the patient's tricuspid regurgitation developed because of recent blunt chest trauma.

The right ventricle is located immediately posterior to the sternum, predisposing it to an anteroposterior compression type of injury, especially during the end diastolic phase.[6] Acute elevation of the right intraventricular pressure results in injury of the tricuspid valve apparatus.[7,8] The most frequently reported mechanism of injury is chordae tendineae rupture, followed by rupture of the anterior papillary muscle and leaflet tear, primarily of the anterior leaflet.[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]

The clinical course of tricuspid regurgitation following blunt chest injury is variable and the mean interval to diagnosis is 11 years.[8,9] Because tricuspid regurgitation is often clinically unsuspected, patients are often diagnosed at an advanced stage, with frequent symptoms or congestive heart failure, atrial fibrillation and right-sided chamber dilatation.[10]

Optimal treatment for traumatic tricuspid regurgitation is still controversial, from long-term medical therapy to early surgical correction.[11] Chordae tendineae rupture tends to follow a more benign clinical course extending from 10 to 25 years. Papillary muscle rupture is often symptomatic and requires operation usually within weeks to months. The traditional indication for operation is symptomatic heart failure and the operation should be undertaken before right ventricular myocardial dysfunction has occurred.[12] Since post-traumatic tricuspid regurgitation is effectively corrected with reparative techniques, some investigators have proposed earlier intervention, before the development of irreversible right ventricular myocardial dysfunction, to increase the possibility of maintaining normal sinus rhythm.[6,7]

Asymptomatic patients with tricuspid regurgitation exhibited a high incidence of tricuspid-related events after diagnosis, with a 75% rate of severe symptoms or heart failure, atrial fibrillation, death, or need for cardiac surgery at 10 years.[10]

In our case, the patient continues to be asymptomatic, but echocardiography showed more dilated right atrium and right ventricle at follow up examination. Severe enlargement of right-sided chambers predicts poor outcome and progression of tricuspid regurgitation and right ventricular dysfunction. The patient should be closely monitored and early surgical

intervention should be considered.

Furthermore when the operation is delayed, the papillary muscles, chordae tendineae and the valve leaflets are frequently found in a contracted and atrophic state, precluding valve repair. Thus, early operation can facilitate repair of the valve.[7]

This case highlights the fact that physicians should be aware of this potential complication following blunt chest injury. Echocardiography should be considered as initial screening tool. Even if performing an echocardiography is not possible in a situation of acute injury, it should be routinely done as a follow-up screening test. Although many patients can tolerate having traumatic tricuspid regurgitation even several years after its onset, the earlier diagnosis and surgical intervention can provide not only prevention of right ventricular deterioration but also feasibility of tricuspid valve repair.

In this case, echocardiography was performed as an initial examination tool for a healthy male who suffered blunt, nonpenetrating chest injury from an automobile accident, allowing for early diagnosis of traumatic tricuspid regurgitation. Valve repair operation was not done before enlargement of the right sided chambers because the patient refused surgery. Long-term follow-up is needed to monitor his symptoms and right ventricular function. Repair of the valve will be re-recommended as soon as a deterioration of the right ventricular function is detected by an echocardiogram.

REFERENCES

1. Vayre F, Richard P, Ollivier JP. Traumatic tricuspid insufficiency. Arch Mal Coeur Vaiss 1996;89:459-63.
2. Gayet C, Pierre B, Delahaye JP, Champsaur G, Andre-Fouet X, Rueff P. Traumatic tricuspid insufficiency. an underdiagnosed disease. Chest 1987;92:429-32.
3. Jensen SS, Nielsen PL, Grossmann P. Traumatic tricuspid valve insufficiency. Ugeskr Laeger 1997;159:4857-8.
4. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 2010; 23:685-713
5. Parmley LF, Mmnion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. Circulation 1958;18:371-96.
6. Krasna MJ, Flancbaum L. Blunt cardiac trauma: clinical manifestations and management. Semin Thorac Cardiovasc Surg 1992;4:195-202.
7. van Son JA, Danielson GK, Schaff HV, Miller FA Jr. Traumatic tricuspid valve insufficiency. Experience in thirteen patients. J Thorac Cardiovasc Surg 1994;108:893-8.
8. Perlroth MG, Hazan E, Lecompte Y, Gougne G. Chronic tricuspid regurgitation and bifascicular block due to blunt chest trauma. Am J Med Sci 1986;291:119-25.
9. dos Santos J Jr, de Marchi CH, Bestetti RB, Corbucci HA, Pavarino PR. Ruptured chordae tendineae of the posterior leaflet of the tricuspid valve as a cause of tricuspid regurgitation following blunt chest trauma. Cardiovasc Pathol 2001; 10:97-8.
10. Messika-Zeitoun D, Thomson H, Bellamy M, Scott C, Tribouilloy C, Dearani J, et al. Medical and surgical outcome of tricuspid regurgitation caused by flail leaflets. J Thorac Cardiovasc Surg 2004;128:296-302.
11. Maisano F, Lorusso R, Sandrelli L, Torracca L, Coletti G, La Canna G, et al. Valve repair for traumatic tricuspid regurgitation. Eur J Cardiothorac Surg 1996;10:867-73.
12. Bardy GH, Talano JV, Meyers S, Lesch M. Acquired cyanotic heart disease secondary to traumatic tricuspid regurgitation. case report with a review of the literature. Am J Cardiol 1979; 44:1401-6.