

Case Report

Traumatic severe tricuspid regurgitation diagnosis after the progression of right ventricle function deterioration

Hanna Jung^a, Joon Yong Cho^a, Gun-Jik Kim^a, Young ok Lee^a, Kyoung Hoon Lim^b,
Seong Wook Hong^c, Yehun Jin^c, Shin-Ah Son^{a,*}

^a Department of Thoracic and Cardiovascular Surgery, School of Medicine, Kyungpook National University, Kyungpook National University Hospital, Daegu, Republic of Korea

^b Trauma Center, Department of Surgery, School of Medicine, Kyungpook National University, Kyungpook National University Hospital, Daegu, Republic of Korea

^c Department of Anesthesiology and Pain Medicine, Kyungpook National University Hospital, Daegu, Republic of Korea

ARTICLE INFO

Keywords:

Cardiogenic shock
Heart injury
Multiple trauma
Tricuspid regurgitation

ABSTRACT

Traumatic tricuspid regurgitation is a rare complication of blunt cardiac injury and frequently misdiagnosed during the initial assessment. Unfortunately, it may be diagnosed after deterioration of right ventricle function, which may be fatal to the patient. Here, we report a case of a patient with blunt chest injury complicated by a diagnosis of traumatic severe tricuspid regurgitation after deterioration of the right ventricle function even after the patient was subjected to serum cardiac enzyme normalization. The patient was a driver and admitted to the hospital owing to multiple traumatic injuries. Echocardiography was performed suspicious of blunt cardiac injury, which revealed no abnormal findings. Initial cardiac enzyme levels were high, but after serial follow-up, the levels improved. However, on day 4 of hospitalization, hemodynamic deterioration occurred owing to severe tricuspid regurgitation and delayed right ventricle dysfunction. Immediate tricuspid valve replacement was performed, however, the patient had a prolonged recovery period. We believe that it is important to take into account the nature of the accident and the presentation of clinical signs and symptoms and not be blinded by laboratory test results alone; it is also important to consider performing repeated serial echocardiographic examinations for blunt cardiac injury patients.

Introduction

Owing to the increase in high-speed automobile accidents, the incidence of blunt cardiac injury (BCI) has also increased [1], injury severity may vary from asymptomatic cardiac contusion to fatal heart injury [2]. In multiple traumatic injury, it is challenging to determine the primary injury or the main symptom owing to the coexisting emergent conditions.

Especially, traumatic tricuspid regurgitation (TR) is a rare complication of BCI and frequently misdiagnosed during the initial assessment [3]. Unless the patient initially presents hemodynamic instability with prominent TR on an echocardiography, traumatic TR are asymptomatic. Unfortunately, when traumatic TR is diagnosed after deterioration of the right ventricle (RV) function, it worsens the prognosis of patients [5].

* Corresponding author at: Department of Thoracic and Cardiovascular Surgery, Kyungpook National University Hospital, 130 Dongdeok-ro, Jung-gu, Daegu, Republic of Korea.

E-mail address: sina2-2@daum.net (S.-A. Son).

<https://doi.org/10.1016/j.tcr.2019.100239>

Accepted 25 July 2019

Available online 29 July 2019

2352-6440/© 2019 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

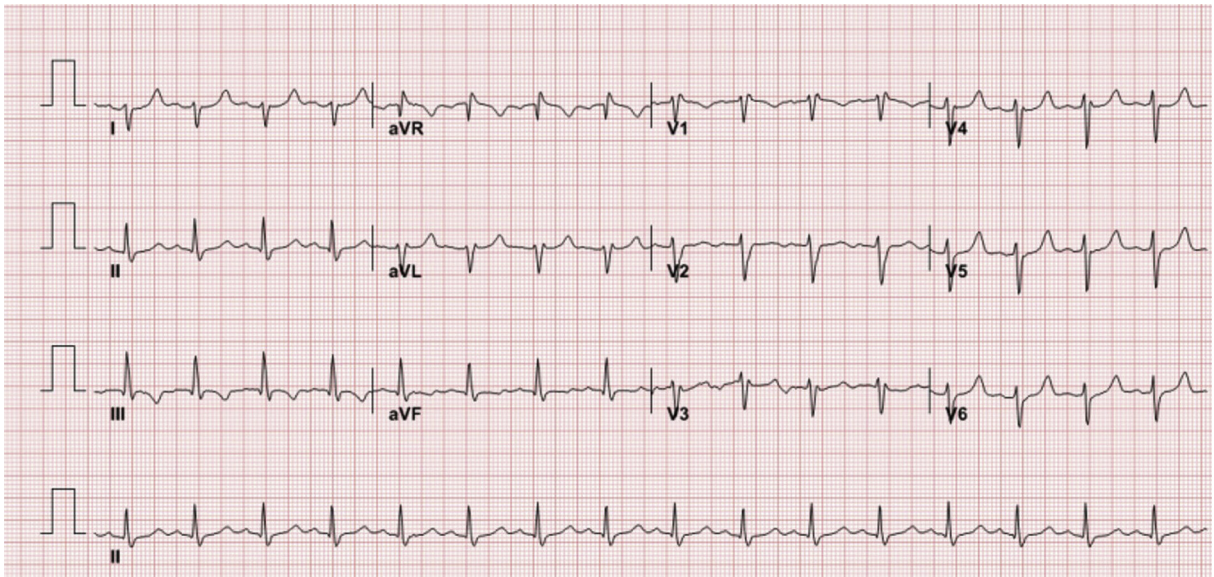


Fig. 1. Initial electrocardiogram showing normal sinus rhythm and a heart rate of 98 beats/min.

Case report

A 58-year-old man with hypertension was transferred to our trauma center after experiencing a traffic accident. The patient showed symptoms of chest pain and abdomen pain. The patient had confused mental status, and his initial blood pressure and heart rate were 88/60 mmHg and 94 beats/min, respectively; his electrocardiogram showed normal sinus rhythm (Fig. 1). The initial computed tomography (CT) scan revealed traumatic subarachnoid and subdural hemorrhage, sternum fracture, multiple rib fractures, and hemoperitoneum owing to omental bleeding (Fig. 2A–D). In the initial laboratory test, the levels of cardiac enzymes including troponin I (cTnI) and creatine kinase MB (CK-MB) were 15.0 ng/mL and 35.2 ng/mL, respectively. Regarding the nature of the accident, the patient was driving the car and he smashed into the median strip of the roadway; along with sternum fracture, cardiac contusion was suspected owing to BCI. Portable transthoracic echocardiography (TTE) was immediately performed in the emergency room, but no abnormal findings were observed.

He was admitted in the intensive care unit to monitor bleeding and manage pain. His condition improved, with no signs of omental bleeding progression. Because his initial cardiac enzyme levels were high, they were followed up daily and the enzyme levels subsequently improved (Fig. 3A). On day 4 of hospitalization, the patient complained of severe dyspnea, and he experienced sudden ventricular tachycardia with hemodynamic deterioration (Fig. 3B). However, the patient did not show neck vein distention or edema of the face and periphery. For a full examination, secondary CT was performed from the head to knee. No active bleeding in the brain and abdomen and no signs of acute pulmonary embolism were observed. Since arrhythmia was sustained, TTE was also performed. Severe TR owing to multiple chorda rupture and right ventricle dysfunction were diagnosed (Fig. 3C), which required emergent surgical intervention.

Through median sternotomy approach, routine cardiopulmonary bypass was performed. In the operative field, hemorrhagic change appeared on RV owing to contusion (Fig. 4A), and RV was dilated. After cardiac arrest, the right atrium was opened and the tricuspid valve (TV) was carefully inspected. Anterior papillary muscle of TV ruptured, and avulsion of the septal papillary muscle was observed on the septum; no part of the valve remained fixed (Fig. 4B, C). The patient underwent TV replacement using a 29-mm bioprosthetic valve. Cardiopulmonary bypass weaning was uneventful; however, arrhythmia and RV failure was sustained, and in the intensive care unit, the patient was in a cardiogenic shock state. The patient was managed in the pulmonary hypertension state and his vital signs were maintained by administering high-dose inotropic agents for more than 10 days. Though his mental state was alert, acute kidney injury required continuous renal replacement therapy and trauma-induced thrombotic thrombocytopenic purpura warranted plasmapheresis. After a long stay in the intensive care unit, he was eventually moved to the general ward but still required hemodialysis.

Discussion

BCI caused by chest trauma may often progress as a fatal condition, and under rare circumstances, it may manifest as traumatic valve injury. Diagnosis of traumatic TV injury may be delayed or overlooked because traumatic valve injury is a rare complication, and traffic accidents are associated with coexisting urgent issues [2,3]. Diagnosis of traumatic TR from TV injury is challenging because of slow pathological progression and atypical or asymptomatic presentation of the former; hence, its incidence rates may be underestimated [1,3].

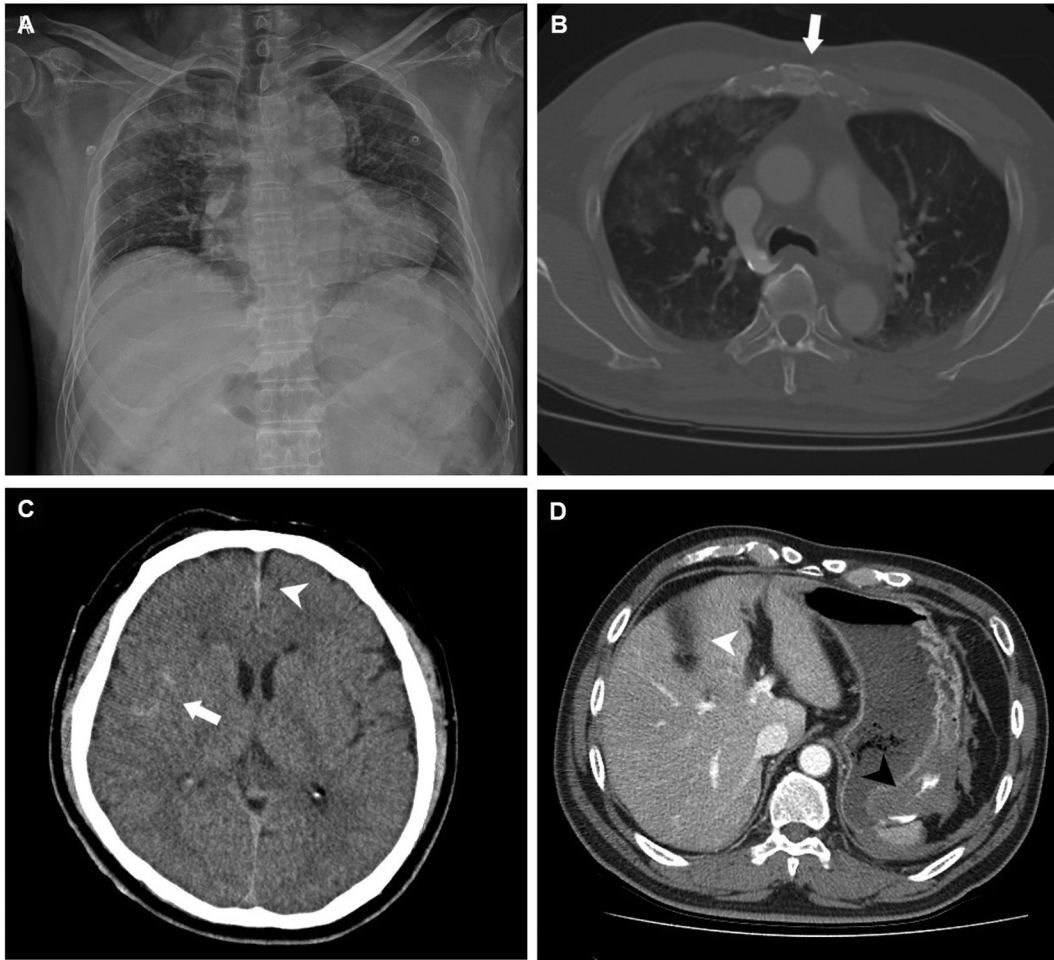


Fig. 2. Initial image work-up in the emergency room. (A) Chest X-ray showing cardiomegaly and pulmonary congestion. (B) Computed tomography (CT) scan of the chest, showing sternum fracture (white arrow) and right lung contusion. (C) Brain CT showing traumatic minimal subarachnoid hemorrhage in the right parietal sulci and sylvian fissure (white arrow) and subdural hemorrhage in the mid-interhemispheric fissure (white arrowhead). (D) Abdomen CT showing omental bleeding (black arrowhead) and liver contusion (white arrowhead).

At the early traumatic TR phase, undetected patients remain asymptomatic until they develop right heart failure. New-onset dysrhythmias are also frequently associated with this injury, ranging from sinus tachycardia to atrial fibrillation, most likely caused by atrial distension [4].

CT scan is a useful tool for initial BCI patient evaluation; however, it is insufficient to diagnose intracardiac structural injury and reflect real-time visualization. Although there is no consensus at present regarding the use of echocardiography as an initial screening tool, echocardiography is the initial method of choice when cardiac injury is suspected [1]. In another report, it has been suggested that patients diagnosed with BCI should undergo more advanced imaging techniques such as TTE or transesophageal echocardiography to rule out the possibility of significant cardiac structural injury [2].

Even asymptomatic patients could have an acute hemodynamic deterioration during the progression of traumatic TR; therefore, surgical treatment should be performed before the development of right heart dysfunction and related symptoms.

In our report, the patient was diagnosed late with severe TR after experiencing hemodynamic deterioration, which was not explained by coexisting injuries. Owing to the nature of the accident and elevated serum cardiac enzyme levels, TTE was initially performed, which did not reveal any significant findings. Serial serum cardiac enzyme measurements were taken daily during follow-up, which showed a trend toward normalization even on the day of surgery. The patient underwent TV replacement immediately after diagnosis. A valve repair might be superior to valve replacement because it is associated with better long-term outcomes and is not associated with artificial valve-related complications [5]. However, the valve deformity and RV dysfunction in our patient made valve repair impossible. Traumatic cerebral hemorrhage and hemoperitoneum were associated with the risk of rebleeding owing to the implementation of cardiopulmonary bypass and heparin administration. Because reducing the time taken for cardiopulmonary bypass would facilitate better patient prognosis, after confirming an absence of TV function, the authors did not hesitate to perform TV replacement.

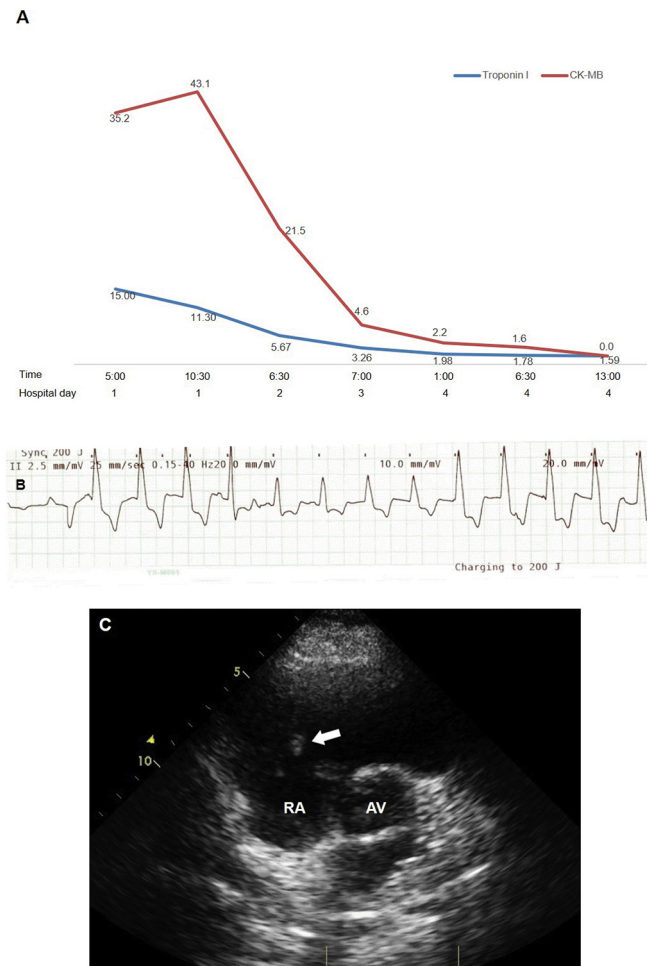


Fig. 3. Examinations after admission. (A) Follow-up of troponin I and creatine kinase MB (CK-MB) levels from the time of admission to the day of surgery. (B) Electrocardiography showing sustained ventricular tachycardia on day 4 of hospitalization. (C) Transthoracic echocardiography showing severe tricuspid regurgitation with chorda rupture (white arrow). RA, right atrium, AV, aortic valve.

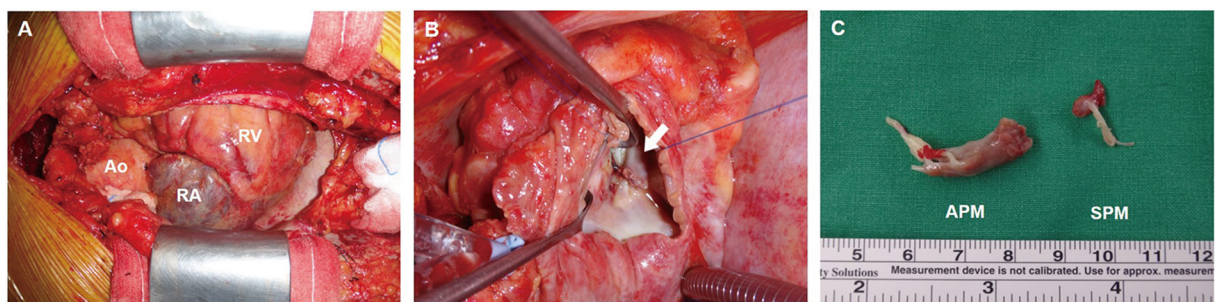


Fig. 4. Operative findings. (A) Hemorrhagic change in the right ventricle. (B) Ruptured anterior papillary muscle (white arrow) and hemorrhagic change of multiple chordae. (C) Closer view of the removed anterior and septal papillary muscle. RV, right ventricle, RA, right atrium, Ao, Ascending aorta, APM, anterior papillary muscle, SPM, septal papillary muscle.

Early detection and elective surgery may facilitate TV repair and prevent progressive deterioration of RV function with rapid recovery. Therefore, to avoid this potentially lethal injury, it is important to be aware of the nature of the accident and presentation of clinical signs and symptoms as well as not be blinded by laboratory test results alone and consider performing repeated serial echocardiographic examinations for BCI patients.

In conclusion, with high-speed injury-related BCI patients should be cared for by taking into account the possibility of traumatic TR. The possibility of traumatic TR in patients should be followed up with serial echocardiographic examinations for earlier diagnosis

and early repair of the valve before deterioration of RV function. This case report may help clinicians with decision-making while treating BCI patients who should be suspicious having traumatic TR.

Acknowledgements

We certify that this case report is our own work and all sources of information used in this report have been fully acknowledged.

References

- [1] H.Y. Jin, J.S. Jang, J.S. Seo, T.H. Yang, D.K. Kim, D.K. Kim, et al., A case of traumatic tricuspid regurgitation caused by multiple papillary muscle rupture, *J. Cardiovasc. Ultrason.* 19 (1) (2011) 41–44.
- [2] G.D. Cresce, A. Favaro, A. D'Onofrio, C. Piccin, P. Magagna, M. Spanghero, et al., Post-traumatic rupture of the anterolateral papillary muscle, *Ann. Thorac. Surg.* 88 (5) (2009) 1664–1666.
- [3] C. Gayet, B. Pierre, J.P. Delahaye, G. Champsaur, X. Andre-Fouet, P. Rueff, Traumatic tricuspid insufficiency. An underdiagnosed disease, *Chest* 92 (3) (1987) 429–432.
- [4] G. Dounis, E. Matsakas, J. Poularas, K. Papakonstantinou, A. Kalogeromitros, A. Karabinis, Traumatic tricuspid insufficiency: a case report with a review of the literature, *Eur J Emerg Med* 9 (3) (2002) 258–261.
- [5] W.G. Ma, G.H. Luo, H.S. Sun, J.P. Xu, S.S. Hu, X.D. Zhu, Surgical treatment of traumatic tricuspid insufficiency: experience in 13 cases, *Ann. Thorac. Surg.* 90 (6) (2010) 1934–1938.