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Case report

Surgical repair for primary tricuspid regurgitation related to trauma



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ABSTRACT

We report a case of a 45-year-old man presenting with tachycardia and palpitation. Echocardiography indicated severe tricuspid regurgitation. We suspected traumatic tricuspid damage due to high energy trauma in a motor vehicle accident 17 years earlier. He underwent a sternotomy, and his tricuspid valve was repaired with chordal reconstruction, indentation closure, and ring annuloplasty. The postoperative period was uneventful, and he was discharged 10 days after the operation. This report highlights the value of echocardiography for diagnosis of primary tricuspid regurgitation related to trauma, and the importance of early diagnosis to allow surgical intervention before irreversible damage occurs.

Learning objective: Traumatic tricuspid regurgitation is a rare cardiovascular complication of blunt chest trauma. The mechanism of the tricuspid valve injury is thought to be secondary to sudden impact causing an anteroposterior compression of the right ventricle by the sternum in end-diastole. This injury is often incidentally identified or can be missed until the patient experiences symptoms of right heart failure resulting from severe tricuspid regurgitation.

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Introduction

Traumatic tricuspid regurgitation (TR) is a rare complication after blunt chest trauma [1,2]. However, the real prevalence of traumatic TR is probably underestimated because symptoms of traumatic TR are nonspecific and often tolerable. It can result in severe and irreversible damage to the right ventricular function, impacting the long-term survival and quality of life. Therefore, it is important to perform surgical intervention while the right ventricular function is still preserved [3]. In this report, we present a case of a patient who developed TR related to trauma causing by a motor vehicle accident, which was successfully treated by valve repair before right ventricular dysfunction.

Case report

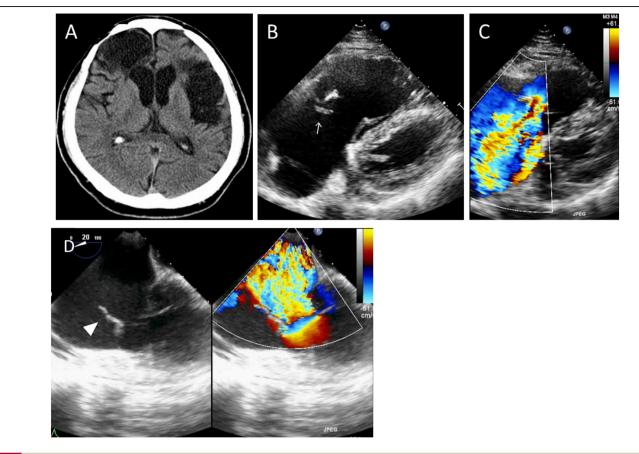
A 45-year-old man with no previous cardiac history presented at our department of cardiovascular surgery, reporting a few months of tachycardia and palpitation. At the age of 28 years, while in a car, he had suffered multiple injuries in a frontal impact against a truck. His

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injuries then included intracerebral trauma with loss of vision in his right eye, and he required surgery for injuries to his head, his right hip joint, and his left knee. He spent a half year in hospital but recovered with no symptoms of heart trauma.

Recent physical findings revealed a Levine grade 4/6 pansystolic murmur at the left lower parasternal border and no peripheral edema. Amlodipine 5 mg per day and atenolol 25 mg per day were prescribed. Chest X-ray revealed a cardiothoracic ratio of 50 % and a sharp costophrenic angle. Computed tomography revealed an old brain contusion in his right frontal lobe and left temporal lobe (Fig. 1A). Electrocardiography showed a sinus rhythm and incomplete left bundle branch block. Blood tests showed no abnormalities in the complete blood count or coagulation system, no liver dysfunction, and no renal dysfunction, but the white blood cell count, hemoglobin, and brain natriuretic peptide levels were slightly increased (11,500 /mm², 17.5 g/dL, and 17.9 pg/dL). Transthoracic echocardiography showed that the right atrium and right ventricle were dilated (right atrium dimensions 71.2 mm × 74.2 mm by 4-chamber view, and right ventricle enddiastolic dimension 36.6 mm by 4-chamber view). Indicators of right ventricular function were preserved [tricuspid annular plane systolic exclusion 26 mm, S' (right ventricle) 12.9 cm/s, and right ventricular fractional area change 39.7 %]. Although the left ventricle was not dilated (left ventricle end-diastolic dimension 36.7 mm, and left ventricle

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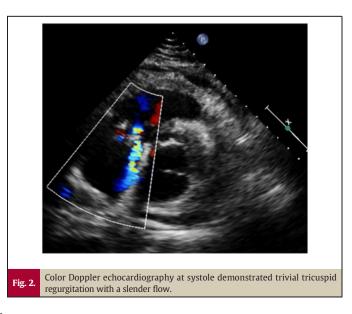
(A) Computed tomography shows brain contusion in the right frontal lobe and left temporal lobe. (B) Transthoracic echocardiographic image at diastole shows an isoechoic cord (a white arrow) on the anterior leaflet. (C) Color Doppler echocardiography at systole demonstrated severe tricuspid regurgitation (TR) with a massive flow due to anterior leaflet prolapse of the tricuspid valve. (D) Transesophageal echocardiographic image shows the anterior leaflet prolapse (a white triangle) and color Doppler echocardiography demonstrated severe TR.

end-systolic dimension 22.7 mm), the left ventricular ejection fraction was 69.3 %. Color Doppler echocardiography showed severe TR with a massive flow due to anterior leaflet prolapse with chordae tendineae avulsion (Fig. 1B, C); TR velocity was 2.89 m/s, and TR pressure gradient was 33.0 mmHg. Cardiac catheterization revealed the right atrium, right ventricle, pulmonary artery, and pulmonary capillary wedge mean pressures were 11, 27/14, 31/11, and 8 mmHg, respectively. The cardiac output and cardiac index were 4.26 L/min and 2.09 L/min/m² by thermodilution method. Calculations by Fick method were not performed. The diagnosis of severe TR was suggested as traumatic in origin because the pattern of damage (ruptured chordae tendineae of the anterior leaflet, and the severe injuries 17 years previously) matches the known pattern of late-onset non-penetrating traumatic TR. His symptoms and progression of congestive heart failure could not be controlled by medications, so surgical repair was deemed necessary.

A median sternotomy was performed. Mild hypothermic cardiopulmonary bypass was established via ascending aortic and bicaval venous cannulation. Intraoperative transesophageal echocardiography showed anterior leaflet prolapse and severe TR with color Doppler (Fig. 1D). Cardiac arrest was induced with aortic cross clamp and infusion of antegrade cold blood cardioplegia. Right atriotomy was performed. Careful inspection of the tricuspid valve (TV) revealed an anterior leaflet prolapse due to ruptured chordae tendineae and anterior leaflet rupture. Two chords of GORE TEX (W. L. Gore & Associates, Inc., Newark, DE, USA) CV-5 polytetrafluoroethylene neochordae were installed from the anterior papillary muscle to the anterior leaflet. A 32-mm Edwards MC3 annuloplasty ring (Edwards Lifesciences, Irvine, CA, USA) was sutured to the tricuspid annulus with horizontal mattress

sutures of 2–0 Teflon-coated braided suture. The ruptured anterior leaflet was repaired using 5–0 polypropylene suture. The aortic cross clamp and cardiopulmonary bypass times were 84 and 91 min, respectively.

Postoperative TTE showed trivial TR (Fig. 2), and the symptoms resolved promptly. The patient recovered uneventfully and was discharged to home on postoperative day 10.



Discussion

In most cases of TR, the etiology is secondary and functional; rarely is it primary, due to infective endocarditis, trauma, iatrogenesis, etc. Other causes were ruled out in this case, and there was a history of high-energy trauma. Therefore, we assessed that traumatic TR was most likely. Relevant key features of traumatic heart-valve damage include the following:

- 1. Violent non-penetrating or "blunt" thoracic trauma, typically anterior, as in a deceleration accident or a violent physical attack can cause it [1,2].
- 2. Asymptomatic internal damage may go unnoticed, especially if attention is diverted by obvious injuries elsewhere.

According to the annual report by The Japanese Association for Thoracic Surgery in 2016, among the 19,820 patients who underwent surgery for acquired valvular disease, 5512 patients (23.7%) underwent TV surgery [4]. Of those 5512 patients, 5209 had concurrent left heart valve surgery, and the remaining 303 patients underwent TV surgery only. Primary TV surgery made up 5.5 % of all TV surgery and 1.5 % of all valve surgery. Partly for lack of reports on outcomes, the indications for intervention in isolated TV disease remain controversial. According to the 2014 ACC/AHA recommendations, indications for isolated TV procedures include symptomatic, severe TR or asymptomatic progressive right ventricle annular dilation (>40 mm) and/or systolic dysfunction [5]. Since right heart failure due to TR responds well to medical management, medication is recommended first for primary TR which is not planned for left heart valve surgery. However, the long-term prognosis of medical treatment for isolated severe TR, especially concomitant with pulmonary hypertension and right ventricle dysfunction, tends to be poor [3]. In addition, patients who already have right ventricular dysfunction have a high surgical risk and may not benefit from TV surgery.

Some reports recommended early surgical management for traumatic TR because of the low perioperative morbidity and mortality, and improvement in the right cardiac function [1,2]. In this case, the right atrium and ventricle were enlarged at the time of our diagnosis, which suggested that a long period had passed since the onset of the TR. In addition, this was a young patient whose mid- to long-term prognosis needed to be considered. Therefore, we selected early surgical intervention by TV repair before right ventricular dysfunction and arrhythmia in the present case. After the operation, there were no operative complications, and his symptoms resolved.

Traumatic TR can present many years after a trauma due to subtle clinical symptoms, which may lead to a delay in treatment. Since

patients with traumatic TR are generally young and active, valve repair is preferable to valve replacement, because of the limited durability and life-long anticoagulation associated with biological and mechanical valve prosthesis, respectively. A previous series with a mean time to operation of 16 years reported that 20 % of patients required valve replacement [1]. In comparison, the other case series with time to operation of 3 years achieved valve repair in all 13 patients [2]. Therefor it is important to diagnose as soon as possible and perform early surgery while the right ventricular function is preserved, to obtain the best results and to avoid the late complications associated with TV insufficiency.

Patient permission/consent statement

We informed the patient that his images will be used in this scientific publication and informed consent was obtained.

Declaration of competing interest

The authors declare that there is no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jccase.2024.02.005.

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