

[CASE REPORT]

Improvement of the Left Ventricular Function after Tricuspid Valve Plasty for Traumatic Tricuspid Regurgitation

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Abstract:

Traumatic tricuspid regurgitation (TR) is a rare cardiovascular complication in chest trauma. Changes in the left ventricle (LV) function after operation are unclear. A 61-year-old woman who had been involved in a traffic accident 1 month earlier presented with exertional dyspnea. Transthoracic echocardiography (TTE) showed severe tricuspid regurgitation (TR) accompanied by LV dysfunction due to anterior leaflet prolapse with papillary muscle rupture. After tricuspid plasty, the LV function improved, as evidenced by TTE and speckle tracking echocardiography. In conclusion, the early diagnosis of traumatic TR is important, and early surgical intervention might be effective for achieving ventricular function improvement.

Key words: trauma, tricuspid valve prolapse, valve repair, left ventricle dysfunction, speckle tracking echocardiography

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Introduction

Traumatic tricuspid regurgitation (TR) is a rare cardiovascular complication and may be undetected in chest trauma due to other organ damage (1). However, advanced echocardiographic techniques have facilitated the diagnosis of traumatic TR (2). Although symptoms of traumatic TR are non-specific and often tolerable, it can result in severe and irreversible damage to the right cardiac chamber, impacting the long-term survival and quality of life (1). Therefore, surgical management, such as valve repair or replacement, should be considered at an early stage. While some reports have shown surgical success in improving the right cardiac function (3), none have described the changes in the left ventricle (LV) function.

We herein report a case of traumatic severe TR that underwent successful surgical repair, with the LV function assessed by echocardiography before and after the operation.

Case Report

A 61-year-old woman presented to our hospital with exertional dyspnea. One month earlier, she had been involved in a traffic accident and was treated for multiple rib fractures as well as fracture of the sternum and right hemothorax. Although she was on antihypertensive medication, she had no history of cardiac disease. On an examination, her blood pressure was 169/116 mmHg, her heart rate (HR) was 86 beats/minute, her percutaneous oxygen saturation was 97% by room air, her respiratory rate was 18 breaths/minute, and her body temperature was 36.5°C. Physical findings revealed remarkably distended jugular veins in a standing position, hepatomegaly, and abdominojugular reflux. She had no heart murmur and no peripheral edema. Chest X-ray revealed a cardiothoracic ratio of 64% and a blunt costphrenic angle (Fig. 1a). Electrocardiography (ECG) showed a sinus rhythm and complete left bundle branch block (CLBBB) (Fig. 1b). Blood tests showed that there were no abnormali-

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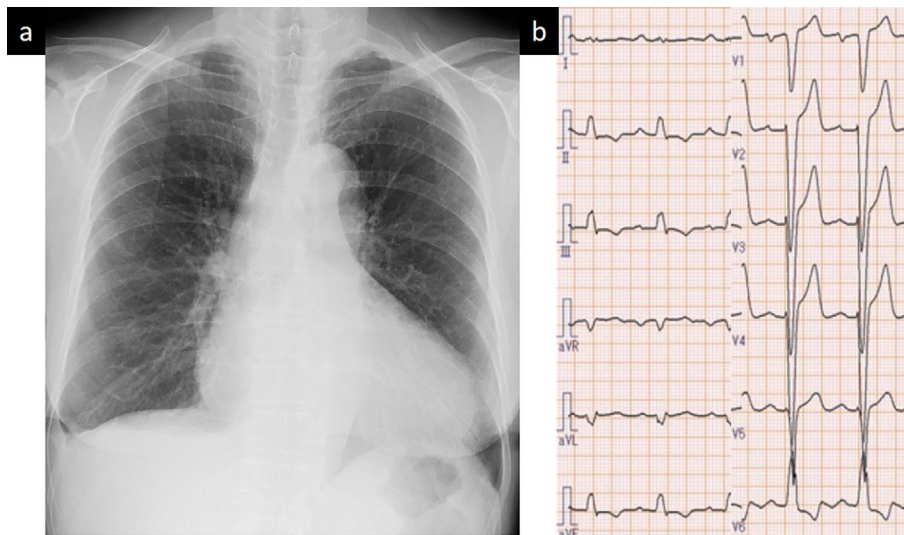


Figure 1. (a) Chest radiograph showing an enlarged cardiac silhouette and a blunt costophrenic angle. (b) Electrocardiography showing a sinus rhythm and complete left bundle branch block.

ties in the complete blood count or coagulation system, no liver dysfunction, and no renal dysfunction, but the brain natriuretic peptide was markedly increased (554.3 pg/mL).

Transthoracic echocardiography (TTE) showed that the right atrium (RA) and right ventricle (RV) were dilated [RA end-systolic area (RA ESA) 27 cm², RV end-diastolic area (RV EDA) 23 cm², and end-systolic area (RV ESA) 14 cm²]. Although the LV was not dilated [LV end-diastolic dimension (LVDd) 44 mm], the interventricular septum (IVS) showed paradoxical motion, and the LV ejection fraction (LVEF) was 39% (modified Simpson's method) (Fig. 2). Tissue Doppler imaging showed an early diastolic tissue velocity of the lateral mitral annulus (lateral E') that was lower than that of the septal mitral annulus (septal E') (3.6, 4.3 cm/s, respectively) and mean E/E' was 15.6 (trans mitral flow E/A (TMF-E/A) 61/120 cm/s). The velocity-time integral at the left ventricular outflow (LVOT-VTI) was 16.5 cm (HR 72 beats/min). Two-dimensional speckle tracking echocardiography (2D-STE) (QLAB v10.8; Phillips Medical Systems, Andover, USA) showed that the global longitudinal strain (GLS) was decreased (-11.0%), and in particular, the peak systolic longitudinal strain of the septal segments of the LV was reduced compared to that of the lateral segments. In addition, the myocardial shortening of the septal segments peaked at an early stage before aortic valve closure (Fig. 3a). Color Doppler echocardiography demonstrated severe TR with a laminar flow (Fig. 4a, b) due to anterior leaflet prolapse of the tricuspid valve with papillary muscle rupture (Fig. 4c, d), and the RV-RA pressure gradient was 4 mmHg. The RV fractional area change (RV FAC) was 40%. The tricuspid annular plane systolic excursion (TAPSE) was 21 mm. The peak systolic velocity (S') of the tricuspid annulus was 12.9 cm/s. Cardiac catheterization revealed pulmonary artery wedge (PCW), and the pulmonary artery (PA), RV, RA, and LV pressure were 6 (mean), 17/9,

17/8 (end-diastole), 9 (mean), and 157/9 (end-diastole) mmHg, respectively (Fig. 5). The cardiac index was 1.96 L/min/m² according to the Fick method.

We diagnosed the case as traumatic severe TR due to papillary muscle rupture with LV dysfunction. Her symptoms that were due to a low cardiac output could not be controlled by medications. Therefore, we consulted with a cardiovascular surgeon, and surgical repair of the tricuspid valve was performed. The operative findings showed that prolapse of the whole anterior leaflet of the tricuspid valve had been caused by rupture of the anterior papillary muscle. The anterior leaflet was repaired using artificial chordae and annuloplasty (Physio 30 mm; Edward Lifesciences, Irvine, USA).

TTE seven days after operation showed that TR was mild, the size of the right heart had decreased (RA ESA 19 cm², RV EDA 22 cm², and RV ESA 13 cm²) (Fig. 4e, f), the RV function was preserved (RV FAC 41%), and the RV-RA pressure gradient was 16 mmHg. Although paradoxical motion of the IVS remained, the LVEF was improved (65%), and the LVOT-VTI was increased (22 cm, HR 60/min). The lateral E' was greater than septal E' (10.5, 4.1 cm/s, respectively), and the mean E/E' was 15.8 (TMF-E/A 109/88 cm/s). 2D-STE after tricuspid valve plasty showed that the LV GLS had increased (-17.1%), and the timing of the peak of myocardial shortening had improved (Fig. 3b).

Her postoperative course was uneventful. Her dyspnea and distended jugular veins disappeared, and she was discharged 13 days after the operation.

Discussion

We herein report a woman who had traumatic TR with LV dysfunction. She underwent successful surgical repair of the tricuspid valve, and the LV function was improved after

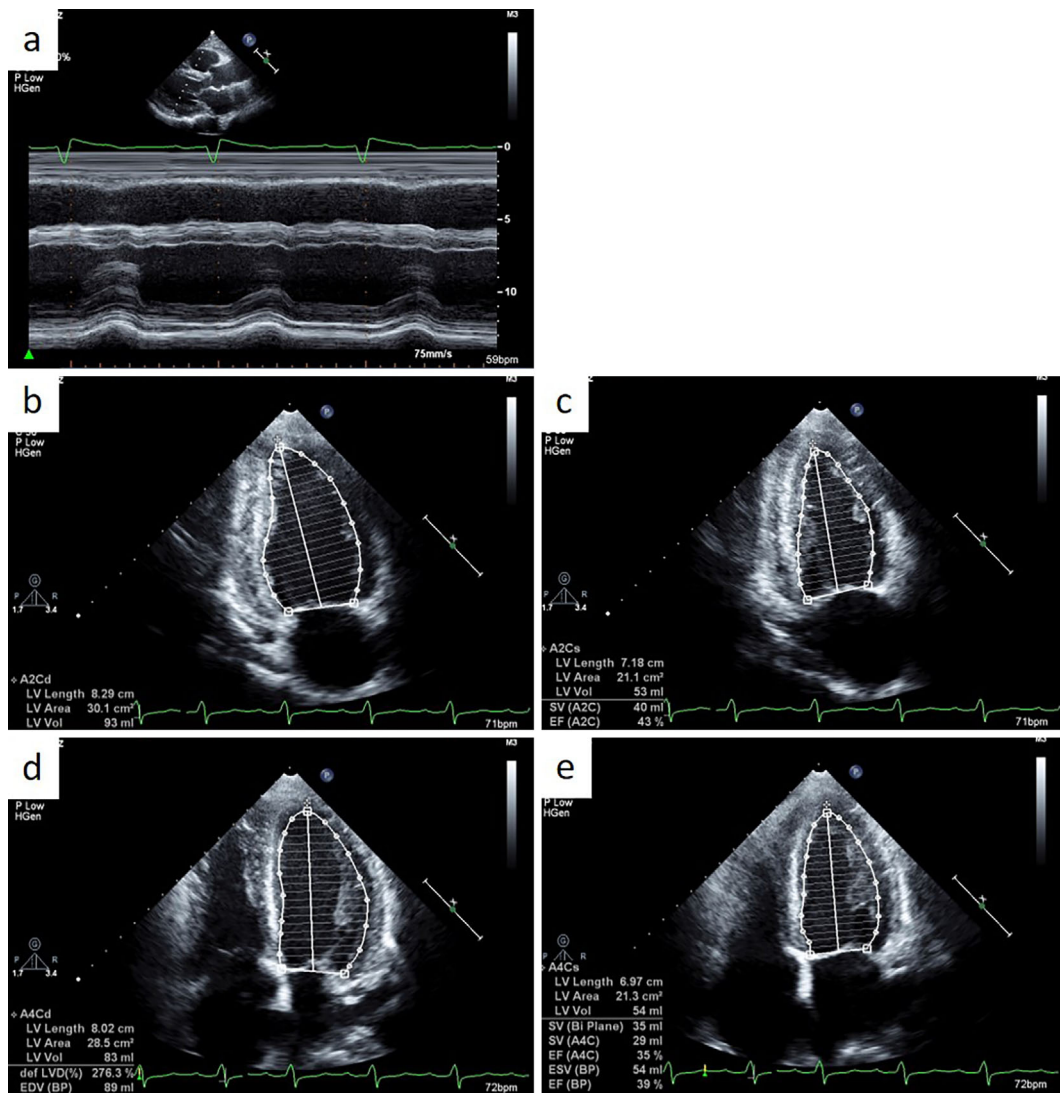


Figure 2. (a) Transthoracic echocardiography in the parasternal long axis view by M-mode showed paradoxical motion of the interventricular septum in the early diastolic period. (b and c) A two-chamber view during the end-diastolic and end-systolic periods, and (d and e) a four-chamber view during the end-diastolic and end-systolic periods showed that the left ventricular ejection fraction was decreased by the modified Simpson's method.

operation based on 2D echocardiography. As she had no evidence of cardiomyopathy, we considered that the LV dysfunction observed in the acute traumatic TR was due, in part, to the interdependence of the LV and RV. Traumatic TR caused a sudden onset of right heart volume overload. As a result, the left heart could not compensate, and LV systolic and diastolic dysfunction was observed.

Traumatic TR is a rarer complication of non-penetrating chest trauma than aortic valve and mitral valve (4). The frequency of tricuspid valve lesions was approximately 0.13% of all injury discharges in the United States (5). The presentation may be delayed for even years after the responsible incident; indeed, von Son et al. reported a median duration between the trauma and operation of 17 years (1). The most frequent cause of traumatic TR was a motor vehicle accident (1, 6). Symptoms of traumatic TR are usually exer-

tional dyspnea, chest pain, and palpitations (6, 7), and they are often tolerable. The long-term prognosis of medical treatment for isolated severe TR, especially concomitant with pulmonary hypertension and RV dysfunction, tends to be poor (8). Some reports recommend early surgical management for traumatic TR because of the low perioperative morbidity and mortality and improvement of the right cardiac function (1, 3, 6, 7).

Traumatic severe TR is acute and may alter the LV and LA geometry because of RV and RA volume overload, which can cause LV diastolic dysfunction and decrease LV filling (8). In this case, TTE showed that RV and RA were dilated while the LV showed no dilatation, and the LVEF was decreased with paradoxical motion of the IVS and decreased lateral E'. Cardiac catheterization showed that the PA, RV, and RA attained equal pressure during systole, indi-

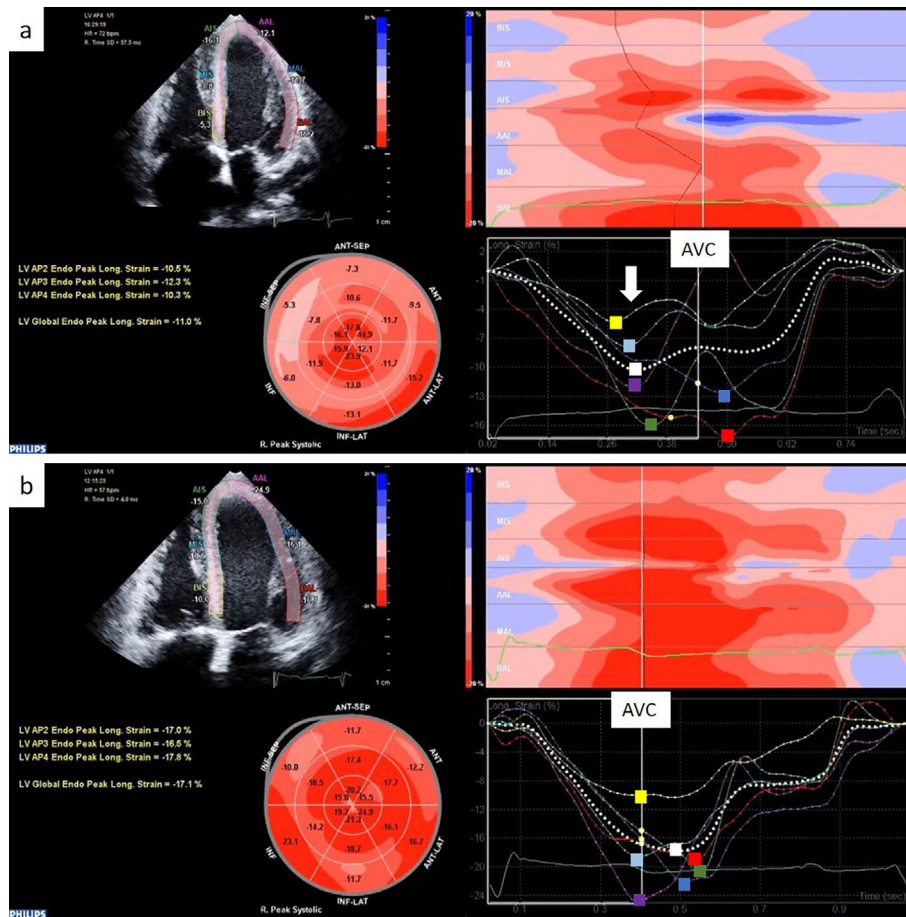


Figure 3. (a) Two-dimensional speckle tracking echocardiography (2D-STE) before the operation showing a reduced peak systolic longitudinal strain of the septal segments of the LV compared to the lateral segments. The myocardial shortening of the septal segments also peaked at an early stage (arrow) before aortic valve closure (AVC). (b) 2D-STE after tricuspid valve plasty showed improvement in the peak systolic longitudinal strain and the timing of the peak of myocardial shortening. The square indicates the peak point of the strain value. Yellow: base of inferoseptal, light blue: middle of inferoseptal, green: apex of inferoseptal, red: base of anterolateral, blue: middle of anterolateral, violet: apex of anterolateral, white: global. LV: left ventricle

cating the presence of severe TR. In addition, there was equalization of the end-diastolic pressure in all cardiac chambers. This is an important finding in the presence of interdependence of the LV and RV, like constrictive pericarditis (9) and cardiac tamponade, as an increase in the right heart volume affects the left heart volume due to the limited intra-pericardium space. An increased right heart volume in the limited intra-pericardium space and low output from the RV to the PA results in a decreased LA volume and consequent early diastolic suction of the LV (10). Decreased LV filling and shuffle motion due to CLBBB might have caused the decreased LVEF.

2D-STE reflects deformation of a structure and therefore directly describes the contraction and relaxation pattern of the myocardium (11). Some reports have shown an association between RV and LV using 2D-STE. Acute right ventricular pressure overload impairs septal strain and apical rotation (12) and influences the intervals from the QRS onset

to peak systolic displacement due to a shift in the IVS (13). RV dyssynchrony caused by pulmonary hypertension is associated with LV dyssynchrony and a reduced LVEF (14). Although the current case suffered from acute volume overload due to TR and not pressure overload, the peak systolic strain was reduced, and displacement also occurred early in the septal segments and improved after the operation (Fig. 2 yellow and light blue). In contrast, the peak systolic strain was delayed after AVC in the lateral segments (Fig. 2 red and blue) and showed no change after operation, which reflected CLBBB. In patients with CLBBB, disability of septal contraction precludes the equilibration of contractive forces during late systole (15). The differential diagnosis between LV dysfunction and cardiomyopathy with CLBBB was difficult in this patient. STE may be useful for the differential diagnosis of LV dysfunction based on the assessment of the contraction and relaxation patterns of the myocardium, such as the attitude and timing of peak systolic

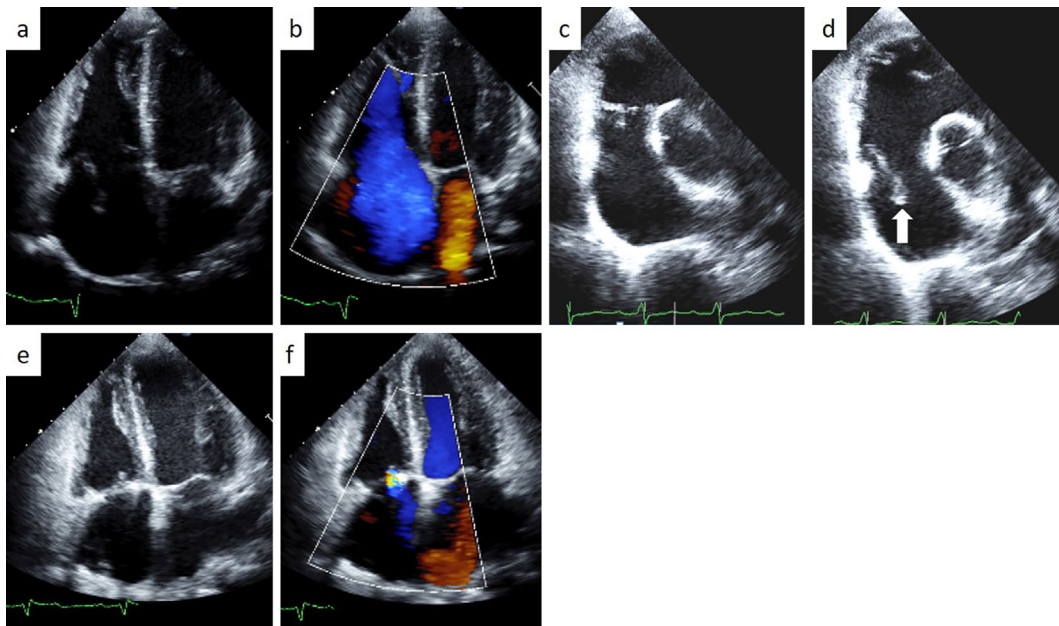


Figure 4. (a and b) Transthoracic echocardiography in the four-chamber view with color Doppler showed severe tricuspid regurgitation with laminar flow. (c) A short-axis view during the diastolic period and (d) a short-axis view during the systolic period showed anterior leaflet prolapse of the tricuspid valve with papillary muscle rupture (arrow) before operation. (e and f) A four-chamber view with color Doppler showed that tricuspid regurgitation improved after tricuspid valve plasty.

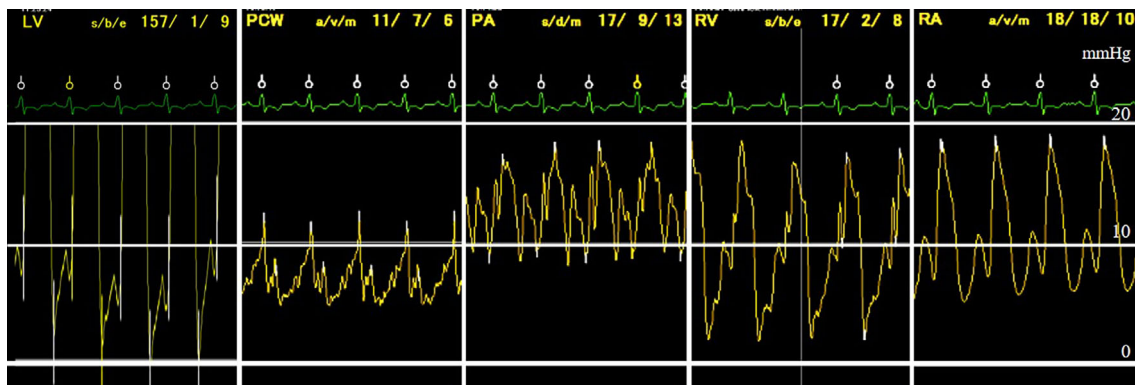


Figure 5. Cardiac catheterization demonstrated that the pulmonary artery (PA), right ventricle (RV), and right atrium (RA) achieved equal pressure during systole and that the end-diastolic pressure was equalized in all cardiac chambers. PCW: pulmonary artery wedge, LV: left ventricle

strain.

The primary treatment option for traumatic TR is surgery. However, the indications, timing, and choice of valve repair or replacement remain controversial. We selected early surgical intervention by tricuspid repair in the present case. After the operation, there were no operative complications, and her symptoms resolved. In addition, the LV function improved, as evidenced by the increased LVEF and lateral E' and improved LV GLS, and the RV function was also preserved.

In conclusion, the findings of this case indicate that traumatic severe TR can cause LV dysfunction. An early diagnosis is important, and early surgical intervention before ir-

reversible damage occurs might be effective for improving the ventricular function. 2D-STE may be useful for the diagnosis of LV dysfunction in patients with CLBBB and RV volume overload.

The authors state that they have no Conflict of Interest (COI).

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