Tricuspid valve regurgitation management with valve replacement Late Presentation of Traumatic Tricuspid Valve Regurgitation after Motor Vehicle Accident – Role of 3D Echocardiography

Hong Seok Lee[1] and Tasneem Naqvi[2]

[1]Affiliation not available
[2]Mayo Clinic

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Abstract

A 51-year-old unrestrained female driver with history of a high-speed motor vehicle accident had been followed due to progressively worsening tricuspid valve regurgitation (TR). Three dimensional (3D0 transesophageal echocardiogram (TEE) showed a TR jet through a perforation in the an avulsed anterior leaflet from the tricuspid valve and another central tricuspid TR jet regurgitation due to tricuspid leafletresulted from malcoaptation of the leaflets from tricuspid annulus annular dilatation.

Introduction

Car accidents induced blunt chest trauma can cause many cardiac complications such as right ventricular free-wall rupture, tricuspid valve rupture, tricuspid valve regurgitation, ventricular septal rupture, coronary artery dissection or thrombosis, left ventriclular chordae and papillary muscle rupture, heart failure and arrhythmias[1-3]. One of the most common valvular complication is tricuspid regurgitation (TR)[4], which can be diagnosed by transthoracic echocardiography (TTE) acutely and followed by TTE for sequelue during follow-up[5].

Unlike rupture of cardiac wall or left sided valves, tricuspid valve rupture usually does not result in immediate hemodynamic instability and may go undiagnosed[5,6]. TR after chest-wall trauma may involve any part of valvular apparatus[5,7] and depending upon the severity of the structural damage to the tricuspid apparatus, the manifestations can vary. Herein we present the case of a car driver who sustained blunt chest-wall trauma and then developed TR due to tricuspid valve leaflet perforation and subsequently another TR jet from tricuspid annular dilation due to right ventricle remodeling during long term follow up.

History of presentation

51-year-old unrestrained female driver was involved in a high-speed motor vehicle accident 27 years ago. At that time, she had a thoracic aortic injury requiring urgent repair. Since then, the patient had developed moderate-severe tricuspid regurgitation with peripheral edema and had been taking a small dose of Furosemide. TTE at presentation showed severe TR, right ventricular systolic pressure of 30 mmHg with normal inferior vena cava size and inspiratory collapse. TAPSE (Annulus Systolic Excursion by M-Mode) was 22 mm. Global averaged RV global longitudinal peak systolic strain (GLS) was -35%. Intraoperative transesophageal echocardiogram (TEE) showed a cresentric perforation along the basal anterior leaflet of the tricuspid valve near the annulus from 8 to 10 O'clock and central malcoaptation of leaflets causing 2 jets of severe TR **(Figure 1,2)** , one jet through the basal anterior leaflet perforation and the other one from the tricuspid valve malcoaptation from a markedly dilated tricuspid annulus **(Figure 3)** . There was hepatic systolic flow reversal **(Figure 4).** There was moderately enlarged right ventricular chamber size

with normal RV systolic function with RV fractional area change of 54%. Based on TEE, TR appeared to have a primary and a secondary etiology. Primary etiology was likely the perforation at the tricuspid valve anterior leaflet, and as a secondary etiology was likely the progressive right ventricular (RV) remodeling from volume overload of severe TR, resuling in malcoaptation of the leaflets from annulus dilatation (5.5x 5.9 cm), causing further increase in TR. Finally, she underwent minimally invasive tricuspid valve replacement with a 31 mm Edwards Life sciences bioprosthesis in 2019. After surgery, follow- up TTE showed normal LV size with an LV ejection fraction of 61% and no regional wall motion abnormalities. There was mild RV enlargement with normal RV function and an estimated RVSP of 21 mmHg. There was normal function of the tricuspid valve prosthesis with a mean diastolic gradient of 1 mm of Hg at a heart rate of 65 bpm and trivial prosthetic valve regurgitation.

Discussion

We presented the case of a car driver who sustained chest-wall trauma and then developed severe TR due to tricuspid valve leaflet perforation and annulus dilatation after blunt trauma. The mechanism of tricuspid valve injury is still controversial. Anatomically, the right ventricle is likely to have vulnerability to blunt trauma due to increased hydrostatic pressure by anteroposterior compression from the adjacent sternum[7]. When a deceleration force especially during the end-diastolic phase is transmitted through the ventricle, a forceful regurgitant blood flow can cause the rupture of a papillary muscle or of the chordae tendineae[8]. Delayed valvular rupture or avulsion may occur due to papillary muscle contusion with hemorrhage, inflammation, and late necrosis, leading to disruption over time[6]. If the damage is severe, symptomatic clinical deterioration and hemodynamic compromise can result. Blunt chest-wall trauma during high-speed motor vehicle accidents is common; however, valvular rupture or perforation is rare (less than 1%) and may present late[9,10] which is similar to our case worsening progressively and causing severe TR later.

In our case, TR had been monitored and managed appropriately ,however, etiology of TR was not clear. The etiology of TR may be missed by 2-Dimentional(2-D) imaging and 3-Dimentional (3-D) TEE may provide definitive anatomic evaluation of the tricuspid apparatus for treatment planning[11]. As was seen in our patient, damage to tricuspid valve leaflet was missed on 2-D imaging which only showed annual dilatation and severe TR. Intraoperative 3-D echocardiographic imaging was able to show detailed anatomical and functional evaluation of the tricuspid apparatus.

Generally, tricuspid valve repair is preferable to valve replacement[9,11]. Whether to perform early surgery in patients who have sustained severe traumatic TR is still controversial. Traumatic TR is amenable to reparative techniques; however, delayed presentation impairs the success of surgical repair[12]. Particularly in patients who present late, surgical findings include contracted and atrophic papillary muscles, chordae, and valve leaflets[5]. Performing surgery before right ventricular dysfunction occurs will enhance the possibility of an adequate result and the subsequent maintenance of sinus rhythm[12]. In this case, complex anatomical valve dysfunction confirmed by intraoperative TEE allowed a decision for valve replacement.

Conclusion

Echocardiography plays a key role in evaluation of patients, who may have traumatic tricuspid regurgitation. It is also used to evaluate valvular apparatus , which is important when surgery is being considered. Recent advances in 3-D TEE can provide better assessment of valvular heart disease. Physicians should be aware of immediate and long term sequele of blunt chest trauma that requires close follow up years after the accident.

Figure 1:

A. TEE biplane view showed two TR jets, one arising from the basal anterior leaflet and the other from central leaflet coaptation (red arrows)

B. TEE deep transgastric view showing color doppler regurgitant jet through anterior leaflet (red arrow)

C. TEE midesophageal right ventricle inflow view showing central and anterior regurgitant jet (red arrows)

D. TEE mid-esophageeal four-chamber view showing tricuspid annual dilatation (red arrow)

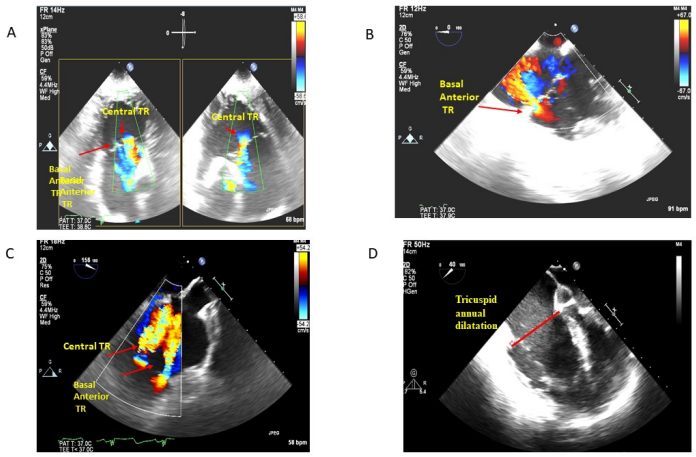


Figure 2

A, Three- dimensional TEE image showing avulsion of anterior leaflet of the tricuspid valve near the annulus from 8 to 10 O'clock (red arrow)

B. Three- dimensional TEE showing malcoaptation of tricuspid leaflets (red arrow)

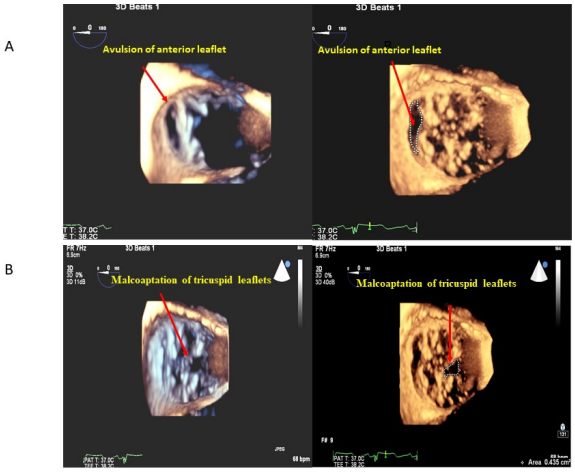
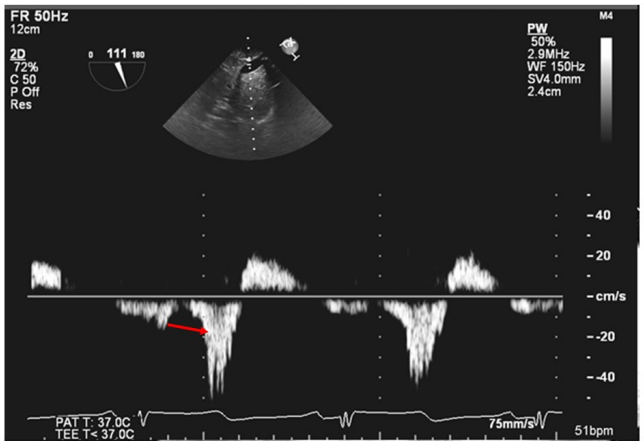


Figure 3.

Offline 3D TEE full volume loop reconstruction showing an anterior jet through the basal anterior leaflet (arrow) and a central TR jet from the coaptation site (arrow). There are no arrows pointing at the jets

Figure 4. TEE image of modified-transgastric hepatic vein view showed severe systolic flow reversal (arrows)



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