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Case Report

Primary tricuspid regurgitation: a rare case report

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ABSTRACT

Tricuspid regurgitation is no longer a forgotten valve as TR can be clinical challenge for the treating physician especially patients with moderate to severe tricuspid regurgitation. The aetiology can be primary and secondary TR of which secondary TR is more common with female predominance. Another entity called isolated TR is now seen. Primary TR due to infective endocarditis is a rare condition. Clinical examination must look for signs of right heart failure. Investigations like Transthoracic and transesophageal echocardiogram has to meticulously done to grade the severity of TR and look for morphological changes. Medical management must be tailored made for patients based upon the severity and the clinicians must know when to refer to the valve team for appropriate intervention.

Keywords: Tricuspid regurgitation, Infective endocarditis, Right heart failure

INTRODUCTION

Tricuspid regurgitation (TR) is often found to be an incidental finding on screening echocardiogram. Functional TR is the commonest form and is mainly caused by cardiomyopathies, left heart failure, mitral or aortic valve disease, or pulmonary disease.[1] The prevalence of TR is higher among women than men and it is increasing in increasing ages. The prevalence of moderate and severe TR reaches up to 1.5 % and 5.6 %, respectively.[2] TR is classified as primary or secondary depending upon underlying cause. But nowadays an independent entity called isolated TR (iTR) is increasingly recognized by clinicians. Patients with moderate and severe TR in clinical scenario is a challenge for physicians. Previously tricuspid valve had been a forgotten valve. The disorder of the valve was either being dealt as benign or else thought to be well tolerated by affected patients. The current mind set about the tricuspid valve has significantly changed. Mild or moderate functional TR, if uncorrected during left-sided heart valve surgery, can progress in ≥25 % of patients and result in right heart failure, increased hospitalizations for heart failure, and decreased survival regardless of the ejection fraction, right ventricle (RV) size, or pulmonary artery pressure.[3]

CASE REPORT

A 50 years old male presented with progressive breathlessness on exertion and abdominal distension for six months duration. He also had bilateral leg swelling along with scrotal swelling. He underwent open laparotomy for perforated gangrenous appendix 8 years back. He also had a history of tricuspid valve infective endocarditis (*Staphylococcus aureus*) with bilateral basal pneumonia 16 years back and was treated with antibiotics. He is currently on oral Digoxin 0.25 mg OD, oral warfarin 2 mg and oral Torsemide+spironolactone 10 mg/25 mg once daily. General examination revealed bilateral pitting pedal edema and scrotal edema. Jugular venous pressure was elevated with prominent ‘v’ wave. Pulse-92 bpm, irregularly irregular rhythm. Blood pressure-110/70

mmHg in left upper limb in supine position.[4] Respiratory rate-24 cycles per minute, afebrile and his BMI was 30.[4] Systemic examination revealed apical impulse felt in left 5th intercostal space half an inch lateral to mid clavicular line. Pulsations were seen in epigastric region. He also had systolic thrill in left lower sternal border. Tender hepatomegaly and ascites (Figure 1) were present. Auscultation revealed variable first heart sound suggesting atrial fibrillation and a high-pitched soft blowing holosystolic murmur of grade 4 is heard with diaphragm of the stethoscope with the patient in supine position with breath held in inspiration in the tricuspid area. Bilateral infrascapular fine crepitations was present.

Laboratory investigations showed hemoglobin 10.4 g/dl Serum total bilirubin-2.1 mg/dl. Renal parameters and serum electrolytes were within normal limits. Viral markers were non-reactive. His APTT-49.2s, PT-21.3s and INR-1.6. Chest x-ray showed cardiomegaly (Figure 2). Electrocardiogram revealed atrial fibrillation with controlled ventricular rate and RBBB. USG abdomen showed congestive hepatopathy, mild splenomegaly, bilateral grade 2 renal parenchymal changes and left renal cortical cyst.

Two-dimensional echocardiogram showed AF during study. Dilated right atrium and right ventricle along with D shaped septum. Dilated pulmonary arteries and its branches. Right ventricle systolic dysfunction present. Non coapting Tricuspid valve was seen. Severe lowpressure TR. No pulmonary hypertension. Adequate LVSF (EF-50 %). IVC dilated and non-collapsing. (Image 3 and 4). Other investigations to rule out secondary TR was done and were ruled out. A diagnosis of primary tricuspid regurgitation due to infective endocarditis with right heart failure with atrial fibrillation was made. Patient was started with intravenous furosemide, oral digoxin 0.25 mg, oral spironolactone 25 mg, oral anticoagulants and other supportive medications. Cardiology opinion was sought and advised to continue diuretics and anticoagulants. Patient improved symptomatically with medications and is currently under regular follow up.

Figure 1: Denoted ascites with dilated veins with facial plethora.

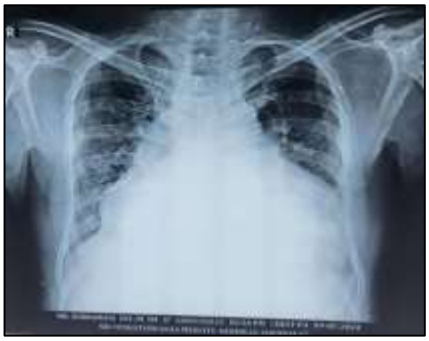

Figure 2: Chest X ray reveals cardiomegaly with pulmonary congestion.

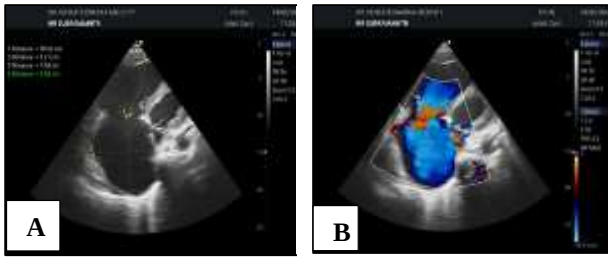

Figure 3 (A and B): Echocardiogram reveals dilated RS and RV with TR.

DISCUSSION

The tricuspid valve (TV) is the largest cardiac valve and sits most apical and anterior. The valve apparatus has 4 anatomic components, fibrous annulus, 3 leaflets, papillary muscles, and the fibrous chordae tendinae. The tricuspid annulus is vulnerable for morphologic changes depending on degree of pressure variations.[4] The anatomy of the complex fibrous crescent shaped annulus and the small septal leaflet without a dedicated papillary muscle make for vulnerable structures that when diseased can lead to TR. These anatomic considerations become relevant during transcatheter interventions.

The aetiology can be divided into primary and secondary of which secondary TR is more common. Primary TR is caused by an abnormality of the tricuspid valve and/or its subvalvular apparatus due to congenital heart disease like Ebstein's anomaly, atrioventricular defects and myxomatous prolapse. Acquired causes of primary TR include carcinoid disease, myxoma, drug-induced damage (ergot alkaloids, dopamine agonists, anorectic drugs), iatrogenic injury during transvenous pacing or defibrillator leads, endomyocardial biopsy, infective endocarditis, systemic diseases (lupus erythematosus, sarcoidosis), radiation, rheumatic disease, and trauma.[5] The incidence of isolated TR appears to be rising along with the prevalence of atrial fibrillation (AF) and intracardiac devices.[6] In secondary TR which is characterised by RV dilation and dysfunction, leading to tethered leaflet, annular dilation and leaflet malcoaptation.[7] This is most

often caused by significant left-sided valvular and myocardial disease, which ultimately leads to increased left-sided pressures, pulmonary hypertension, increased RV afterload and remodeling of the RV. In the absence of pulmonary hypertension, intrinsic RV disease like arrhythmogenic RV cardiomyopathy can lead to TR through papillary muscle displacement, along with leaflets tethering and maladaptation.

A large observational study by Wang TKM et al showed 9 % patients with moderate to severe TR were secondary. Secondary TR due to left heart disease, atrial function abnormalities, pulmonary disease and pericardial disease were 54%, 24%, 17%, 4% and 1% respectively.[8] Primary TR was due to endocarditis in 47%, degeneration or prolapse in 18%, prosthetic valve failure in 16% and implantable device in 10%. It also showed that secondary TR has significantly higher rates of morbidity and mortality. Secondary TR due to pulmonary disease and left heart disease had the poorest prognosis with the highest mortality rate.

Patients with long-standing significant TR, develop clinical signs like ascites, bilateral pedal edema, congestive hepatomegaly and jaised jugular venous pulse suggestive of right heart failure. Auscultation reveals pansystolic murmur heard at the left lower sternal border and the intensity increases on inspiration (Carvallo sign). Two-dimensional (2D) transthoracic echocardiography remains the mainstay investigation in establishing the diagnosis of TR and grading the severity. Qualitative parameters include assessment of tricuspid valve morphology, leaflet mobility, colour flow TR jet and continuous wave Doppler spectral signal.[9] Central jet area >10 cm^2, proximal isovelocity surface area >0.9 cm, vena contracta diameter >0.7 cm, effective regurgitant orifice area ≥40 mm^2 and regurgitant volume ≥45 ml denotes the echocardiographic criteria for severe TR. Medical management for TR is very limited. The severity progression has to be monitored by TTE surveillance. There is no specific management guidelines for the frequency of surveillance. TEE once in 3 to 6 months may be reasonable to use in patients with severe TR to monitor the degree of annular dilation and RV function. Loop diuretics are the mainstay in the medical management of TR to relieve symptoms of right heart failure.[10] However there is no particular loop diuretic that has demonstrated significant advantages and has no morbidity or mortality benefit. According to 2020 AHA/ACC recommendation which suggests the consideration of intervention in TR patients with refractory right HF. Progressive annular dilation and presence of congestive hepatopathy are associated with poor prognosis and should be referred to valve team for intervention. In severe TR there is progressive right heart remodeling, leads to increased morbidity and mortality. Ref RV ejection fraction and end-diastolic volumes can be correlated for outcome after TV intervention and repair; however, this is not well defined and no widely accepted quantifiable cutoffs. Early referral to a valve team in patients with symptomatic severe TR has to be considered to prevent chronic irreversible right heart remodeling.[11-13]

CONCLUSION

Primary TR due to infective endocarditis is a rare clinical scenario. Tricuspid regurgitation is no longer a forgotten valve as TR can be clinical challenge for the treating physician especially patients with moderate to severe tricuspid regurgitation. The prevalence of isolated TR is also increasing. Secondary causes of TR have to ruled out. Medical management must be tailored made for patients based upon the severity and the clinicians must know when to refer to the valve team for appropriate intervention.

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