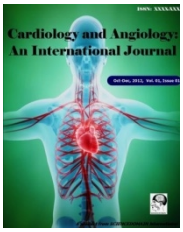

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Tricuspid Valve Replacement in an Adult with Destroyed Tricuspid Valve and Refractory Right Heart Failure

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Authors' contributions

This work was carried out in collaboration between all authors. Author UPP and BSR were the treating physicians of this case. Author UPP wrote the manuscript. Author HKK operated the case. Authors KNR and PKR did Echcocardiograms and Cardiac MRI and helped in key decision making related to management of this rare case.

Case Study

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ABSTRACT

22 year old woman with no previous heart disease history, presented with progressive worsening of right heart failure symptoms due to severe tricuspid valve regurgitation which had become refractory to medical management. Echocardiogram revealed probable rare case of dysplastic tricuspid valve with large calcified mobile masses attached to leaflets. Calcified masses were thought to be due to healed vegetations from silent infective endocarditis of abnormal tricuspid valve which she had suffered in the past. There were no known acquired causes of tricuspid valve endocarditis. She had successfully undergone tricuspid valve replacement with bio-prosthetic valve along with a right atrial reduction surgery after which her heart failure symptoms improved markedly.

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Keywords: Tricuspid valve endocarditis; Ebstein`s anomaly; bioprosthetic valve; calcified vegetations.

1. INTRODUCTION

Isolated severe tricuspid regurgitation due to congenital malformation of the tricuspid valve is a rare anomaly [1,2]. Abnormalities of the tricuspid valve are due to those in which the primary lesion is a downward displacement of the basal attachment of the mural and septal (or both) leaflets, known as Ebstein's malformation and those without downward displacement but with deformation of the leaflets and tension apparatus, an arrangement clubbed together as tricuspid valve dysplasia [3]. Dysplastic valves have variety of changes, including thickening, shortening of the chordae tendineae, rolling off the edges of the leaflets and anomalies of their distal attachment of tricuspid valve [4]. These valves may present with severe regurgitation and may also be predisposed to calcium deposition with or without mural vegetations.

2. CASE REPORT

22 years old woman from poor socioeconomic background presented with symptoms of dyspnea on exertion, palpitation and swelling of feet for 2 years duration. She was referred to our outpatient department with the provisional diagnosis of Ebsteins anomaly. Physical examination revealed elevated jugular venous pressure (JVP) with irregular pulse and generalized anasarca. There were no significant murmurs or additional sounds on auscultation. Electrocardiogram showed right bundle branch block (RBBB) with atrial fibrillation and chest X-ray showed massive cardiomegaly with oligemic lung fields. Her basic blood parameters (hemoglobin, blood counts, erythrocyte sedimentation rate, C-reactive protein) were normal and blood cultures were sterile. Echocardiogram showed completely destroyed tricuspid valve, with large calcified mobile masses attached to redundant leaflets, ruptured chordae and rudimentary papillary muscles. These findings were different from a congenital unguarded tricuspid orifice or Uhl`s anomaly, where there is complete absence of valvular structure. There was also no significant anterior displacement of septal leaflets as expected in Ebsteins anomaly. Right ventricle (RV) was thick and well trabeculated with significant RV dysfunction. The densely calcified mobile masses attached to the anterior and septal tricuspid leaflets represent probably old healed vegetations due to unrecognized silent infective endocarditis in the past. There was another large calcified mass arising from the RV free wall at the attachment of the moderator band Fig. 1. Right atrium (RA) was hugely dilated due to severe tricuspid regurgitation (TR) Fig. 1. Pulmonary artery pressure calculated from TR jet velocity was 25mm Hg. Computed tomography (CT) pulmonary angiogram showed normal pulmonary vasculature with no filling defects. Magnetic resonance imaging (MRI) showed enlarged RA and RV with vegetations on the destroyed tricuspid valve Fig. 2. There is 1 square centimeter (sqcm) mass on septal leaflet moving asynchronously and 7 sqcm mass on RV free wall. RA volume calculated from MRI was 415ml. RV end diastolic volume was 347 ml, end systolic volume was 223 ml and RV ejection fraction was 36%. We diagnosed as a case of the destroyed tricuspid valve due to infective endocarditis with severe tricuspid regurgitation and in right heart failure. She was initially managed medically with diuretics, digoxin and low dose angiotensinogen converting enzyme (ACE) inhibitors. After two years on oral medications and repeated admissions for decompensated heart failure her symptoms became refractory to medical management. She was refused for surgery by three surgeons previously and she herself

was initially not willing for a high risk procedure. But over time her quality of life deteriorated so badly with medical management that she finally gave consent for surgery.

Intra operative findings are large RA, completely deformed tricuspid valve with large clalcifications attached to the chordae of tricuspid valve leaflets. Trucuspid valve replacement with 31mm tissue valve and RA volume reduction was done during surgery Fig. 3. Postoperatively she developed persistent right heart failure due to severe RV dysfunction which was reduced with IV diuretics, levosimendan infusions along with oral digoxin. There were no other perioperative complications and after one year of follow up she was doing well. The histopathlogy of excised tissue showed only large masses of central calcification surrounded by lymphocytes, few neutrophils and fibroblasts with fibrous connective tissue. There was no evidence of endothelial cells in the excised mass.

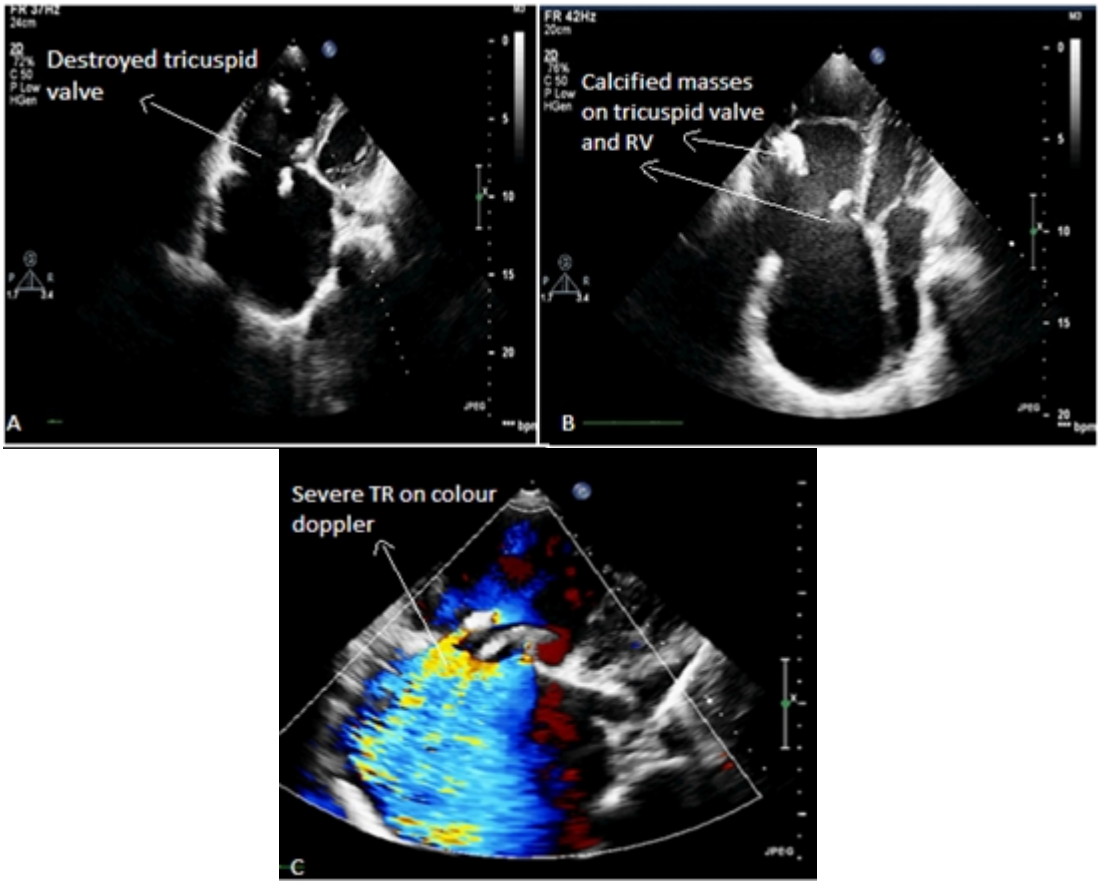

Fig. 1. Echocardiogram showing dilated (A) RA, RV and destroyed tricuspid valve with (B) calcified masses attached to leaflets and (C) severe TR

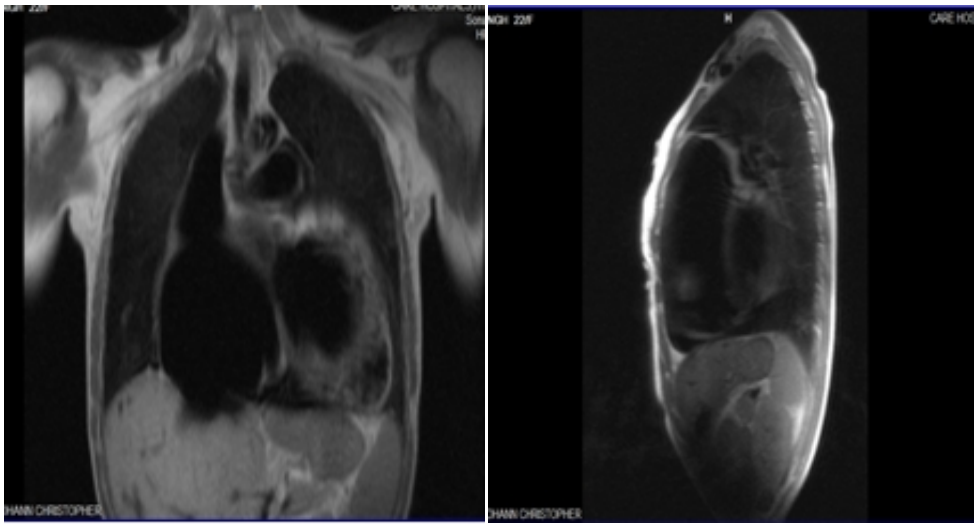

Fig. 2. Cardiac MRI showing dilated RA RV, dysplastic undisplaced tricuspid leaflets with masses attached to leaflets and RV free wall

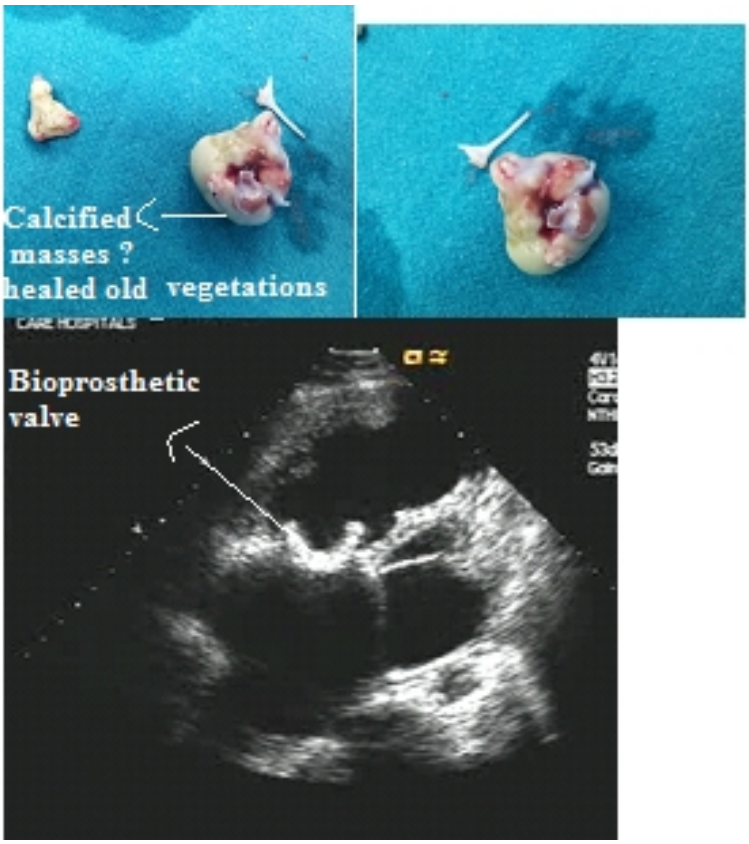

Fig. 3. Excision of calcified masses after surgery and post operative ECHO showing bioprosthetic valve in situ

3. DISCUSSION

Tricuspid valve endocarditis (TVE) is reported to represent only 5% to 10% of all cases of infective endocarditis [5]. The common causes for tricuspid valve endocarditis are intravenous drug abuse and congenital anomalies [6]. Identification of isolated TVE in the absence of predisposing factors and history of intravenous drug is very difficult. It is a rare condition, and as shown in a Canadian study on 135 cases, isolated native TVE was reported in 5% of non-drug users [7]. Although the possibility exists that the patient had an undisclosed history of drug use, there was no evidence of drug abuse on physical examination, and she hails from a simple rural background in India with no previous significant past medical history. On repeated enquires there was no history suggestive of rheumatic fever or congenital heart disease, although she gave a history of prolonged febrile illness during her early childhood which could possibly suggest a period of acute infective endocarditis. Isolated TVE in non addict patients can present with myriad of clinical features but it is rare to detect in such a burnt out stage with large healed vegetations and being relatively asymptomatic in acute stage [8,9]. Purely regurgitant valves have been found to have calcific deposits, some of them which are large due to healed infective endocarditis [10]. There were no other predisposing conditions commonly associated with right-sided endocarditis like dental infections, pelvic or intra abdominal infections, previous catheterization, alcoholism, and immunodeficiency states. Based on Echocardiographic, MRI and intra operative findings we came to conclusion that patient had some form of underlying congenital anomaly of tricuspid valve, possibly tricuspid dysplasia on which she developed infective endocarditis.

Tricuspid valve replacement for dysplastic tricuspid valves has been described since early 1980`s and successful tricuspid valve replacement with bioprosthetic valve has been done in very elderly persons also [11]. In our case there was a complete lack of normal systolic coaptation of anterior and septal tricuspid leaflets producing severe tricuspid regurgitation, hence unsuitable for repair. Bioprosthetic valve was chosen because a mechanical valve in tricuspid location has a very high incidence of valve thrombosis and we were not sure that our patient would be complaint with drug treatment on a long run as she hails from a remote area where medical services are poor. Said et al studied valve replacements in congenital non Ebsteins tricuspid valve and found that porcine bioprosthetic valves are best suited for severely dysplastic tricuspid valves as they have better durability than pericardial valves and less thrombosis than mechanical valves [12]. Our patient had significant RV dysfunction because of late presentation which was the reason for her prolonged postoperative stay. Identifying silent tricuspid endocarditis especially in non drug addicts is challenging and even in gross right heart failure unlike left sided abnormalities, tricuspid valve replacement has favorable outcomes as proven in our case.

4. CONCLUSION

Isolated TVE in non addict patients is a rare presentation and mostly due to congenital abnormalities of the tricuspid valve. It is uncommon to detect in later stages with large healed calcified vegetations and produces refractory right heart failure symptoms. Even in end stage right heart failure, tricuspid valve replacement has better survival chances than continuing medical therapy.

CONSENT

All authors declare that ‘written informed consent was obtained from the patient for publication of this case report and accompanying images**.**

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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