



Congenital Tricuspid Valve Dysplasia with Infective Endocarditis Presenting as Cardiac Tamponade

KEYWORDS

Tricuspid valve endocarditis, congenital cardiac anomaly, congenital tricuspid dysplasia

Anil Wanjari

Professor, Department of Medicine, JNMC, DMIMS Univ. Sawangi (M), Wardha; 442004 Maharashtra

Swapnil Chillawar

Resident, Department of Medicine, JNMC, DMIMS Univ. Sawangi (M), Wardha; 442004 Maharashtra

Sourya Acharya

Professor, Department of Medicine, JNMC, DMIMS Univ. Sawangi (M), Wardha; 442004 Maharashtra

S N Mahajan

Prof & HOD, Department of Medicine, JNMC, DMIMS Univ. Sawangi (M), Wardha; 442004 Maharashtra

ABSTRACT

Tricuspid valve endocarditis is mainly a disease of intravenous drug abusers. Tricuspid valve dysplasia is also an uncommon congenital cardiac anomaly which can also predispose to endocarditis making the situation still rarer. We present a case of a 20 year old male who presented with massive pericardial effusion with severe right sided heart failure with congenital tricuspid valve dysplasia and endocarditis.

Introduction:

Tricuspid valve endocarditis is disease of intravenous drug abusers. It is very rare to have endocarditis associated with tricuspid valve dysplasia.

Incidence of Tricuspid valve dysplasia is 1% of all congenital heart disease^[1]. The lower incidence of right-sided endocarditis compared with left-sided endocarditis has been attributed to the lower rate of congenital and rheumatic heart disease affecting the right-sided valves. Isolated native tricuspid valve endocarditis (TVE) accounts for only 5% to 10% of all cases of infective endocarditis^[2]. Pericardial effusion can be a devastating complication of severe right sided heart failure. This is a unique case describe dysplastic tricuspid valve with endocarditis, right heart failure with failure related massive pericardial effusion causing tamponade.

A 20 year old Hindu male presented to us with chief complains of dyspnea on exertion, palpitation, fever on and off since 2 weeks, swelling in both lower limbs since 2 years, There was no history of paroxysmal nocturnal dyspnea, cough, hemoptysis and chest pain. On examination blood pressure was 90/60mmHg in left arm supine position, pulse rate- 120/min regular. Pulsus paradox of 16mm was present. Jugular venous pressure was raised with prominent 'x' descent. Bilateral Pitting edema feet present. There was no evidence of endocarditis.

CVS examination revealed muffled heart sounds, respiratory examination normal, per abdomen revealed soft tender hepatomegaly. Provisional diagnosis of pericardial effusion was made.

Investigations revealed, Normal hemogram, ESR-40 in first hour, three blood cultures were positive for viridians streptococci, chest xray suggested massive pleural effusion(fig 1), 2D- echocardiography suggested tricuspid valve dysplasia with severe tricuspid regurgitation, right ventricular failure, small hyperechoic calcific mass on the tip of the anterior tricuspid leaflet, spontaneous echo contrast in right atrium and large pericardial effusion.(fig 2-4)

Patient was treated with Inj ceftriaxone 2gm iv12 hourly, Inj penicillin, (2-3 million U IV 4 hourly), inj vancomycin(15mg/kg 12hourly) was given along with supportive therapy. In view

of breathlessness pericardiocentesis was done and pigtail catheter was introduced for continuous drainage of effusion. Cytology suggestive of transudative fluid which was negative for Acid fast bacilli, gram staining, culture for AFB. In view of mass lesion and history of fever intravenous antibiotics was given for 2weeks. Pericardial sac became empty by day seven. Fever responded after 2 weeks of treatment. Repeat echocardiography shown resolution of pericardial effusion. Patient was advised for surgery of tricuspid valve repair & was referred to higher centre.



Fig 1- Chest x ray showing massive pericardial effusion .View with orientation on pt. heart, shows grossly dilated RV, RA with large pericardial effusion.



Fig2- the Tricuspid leaflets seen in this view are septal and anterior. The leaflets appear short, malformed, and do not show proper coaptation. A small hyperechoic mass is noted on tip of anterior Tricuspid leaflet which moves with the leaflet.

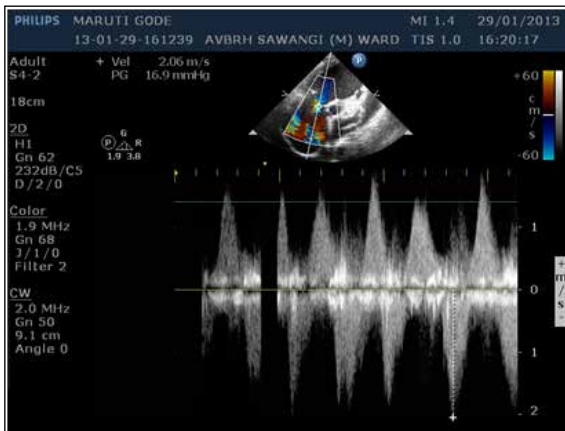


Fig3- A large TR JET is noted.



Fig4-Plax view angulated medially to show RV, RA in flow, shows anterior and posterior tricuspid leaflets. Anterior leaflet shows a small mass as in fig.2.

The posterior tricuspid leaflet appears short, malformed, thickened and thus contributes to non coaptation of leaflet, leading to large TR.

Discussion:

This patient was diagnosed initially as case of pericardial effusion with congestive heart failure. Echocardiography showed tricuspid valve dysplasia with severe tricuspid regurgitation, right ventricular failure, small hyperechoic calcific mass on the tip of the anterior tricuspid leaflet, spontaneous echo contrast in right atrium. The calcified vegetation was probably formed during an unrecognized subacute infective endocarditis. Patient had history of persistent fever. Blood cultures on both admission and day 1 grew viridans streptococci in two of two vials, later confirmed to be *Streptococcus mitis*. Altogether, three sets of blood cultures taken over one week were confirmed to be positive for viridans streptococci. Isolated native Tricuspid valve endocarditis is reported to represent only 5% to 10% of all cases of infective endocarditis. The lower incidence of right-sided endocarditis compared with left-sided endocarditis has been attributed to the lower rate of congenital and rheumatic heart disease affecting the right-sided valves, the lower right heart pressures and the reduced right heart blood oxygen content. The most common predisposing factor for right-sided endocarditis is intravenous drug use^[3]. Our patient gave no history of Intravenous drug use on repeated enquiries. So in our case the dysplastic tricuspid valve was the cause of endocarditis. The association of pericardial effusion and tricuspid valve dysplasia with endocarditis is uncommon. The pericardial effusion in this case could be explained by severe right sided heart failure. Congenital tricuspid incompetence due to valvular dysplasia is a defect involving the leaflets (normally inserted on the ring) the chordae tendinae and papillary muscles of the tricuspid valve. It is a rare condition. Surgery is usually the definitive treatment.

Clinicopathologically: the presentation of such cases has been predominantly divided into 2 groups. The first group comprises the newborn and infants who usually present with cyanosis, cardiomegaly and right ventricular failure in the neonatal period raises the differentials of Ebstein's anomaly and Uhl's disease.^[4] The outcome is usually fatal in several days to weeks. The second group comprises patients who decompensate during adult life, like in our case and the presentation is with right ventricular failure and degrees of tricuspid incompetence. Association of pericardial effusion sometimes arises a confusion of constrictive pericarditis. Only definitive treatment is surgery.

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