Infective endocarditis-induced complete closure of a ventricular septal defect and complete heart block in a child

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ABSTRACT

We hereby report rare occurrence of irreversible complete heart block in a child with tricuspid valve infective endocarditis. The tricuspid valve vegetation also caused complete closure of perimembranous ventricular septal defect, which was later discovered during surgery.

Keywords: Complete heart block, tricuspid valve endocarditis, ventricular septal defect

INTRODUCTION

Infective endocarditis (IE) in childhood is rare. Unlike adults, majority (75%–80%) of children with IE have underlying heart disease.[1] While 10%–15% of children have other risk factors, approximately 8%–10% of patients have no risk factor for IE.[1] The data on right-sided IE are further limited, but it is uncommon in the absence of structural abnormality of the heart and/or risk factors such as indwelling central venous catheter.[2] IE of the tricuspid valve usually presents with vegetation with or without manifestations of embolization of vegetation to the lungs. Rarely, tricuspid valve IE is complicated by conduction abnormality,[3] acquired ventricular septal defect (VSD),[4] and acquired Gerbode defect.[5] Complete closure of a preexisting VSD by the vegetation on the tricuspid valve is also reported albeit only a few times.[6,7] We hereby present an unusual case of tricuspid valve IE with complete heart block (CHB), wherein the VSD was concealed by the vegetation.

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CASE REPORT

An 8-year-old boy was admitted with a history of high-grade intermittent fever, generalized malaise, and easy fatigability for 3 weeks. He also had a single episode of syncope, 15 days before admission. At presentation, he was febrile (temperature - 103°F) and ill appearing. His pulse was regular but bradycardic at 44/min. This was accompanied by intermittent cannon A wave in the jugular venous pulse. He also had elevated jugular venous pressure, pedal edema, and hepatomegaly. Cardiovascular examination revealed bradycardia and variable intensity S_1 and S_2 consistent with CHB. No murmur was audible. Electrocardiogram [Figure 1] confirmed CHB with an atrial rate of 148 beats/min and ventricular rate of 42 beats/min with complete right bundle branch block (QRS duration - 110 ms). Transthoracic echocardiography revealed two large vegetations, with one measuring 14 mm × 10 mm attached to the septal tricuspid leaflet and the other measuring 10 mm × 6 mm attached to anterior tricuspid

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leaflet [Figure 2]. There was moderate tricuspid regurgitation with no pulmonary arterial hypertension. No other cardiac abnormality was seen [Supplementary Videos 1 and 2].

Laboratory investigations showed moderate anemia (Hb–9.2 g/dL), neutrophilic leukocytosis (15,500/mm^3), thrombocytopenia (82,000/mm^3), and elevated erythrocyte sedimentation rate (44 mm in 1st h), and C-reactive protein level (116 mg/dl). He was treated with intravenous antibiotics (ceftriaxone, gentamycin, and vancomycin) and isoprenaline infusion (at 0.1 mcg/kg/min). Despite CHB with low escape rate, a transvenous pacemaker was not placed in view of the large vegetations in the right atrium. Blood culture grew methicillin-resistant *Staphylococcus aureus*. He continued to have fever and CHB persisted. Hence, surgical vegetectomy with repair of the tricuspid valve was planned after 12 days of antibiotics. During surgery, surprisingly, a 10 mm × 5 mm perimembranous VSD was found underneath septal leaflet of tricuspid valve [Figure 3]. The child underwent vegetectomy, VSD closure, and repair of the tricuspid valve. Postoperative echocardiography confirmed no VSD and tricuspid regurgitation. Pacemaker implantation was deferred in view of the possibility of spontaneous resolution of CHB following surgery. However, CHB persisted, and therefore, an epicardial single-chamber pacemaker for ventricular pacing was implanted 2 weeks after the first surgery. Endocardial pacemaker was not preferred in view of endocarditis of the tricuspid valve. He was continued on intravenous antibiotics for a total of 6 weeks. Unfortunately, 4 weeks after discharge, he had recurrence of fever which proved to be due to fungal endocarditis of the tricuspid valve. He had large vegetations on the tricuspid valve, and the blood culture grew *Candida tropicalis*. This time, he required surgical resection of tricuspid valve with an implantation of 25 mm bioprosthetic valve (St. Jude Medical; St. Paul, Minneapolis, Minn, USA). At the time of writing this report, he is asymptomatic and has completed 6 weeks of intravenous antifungal therapy. Currently, he is on oral fluconazole prophylaxis along with oral anticoagulation with strict monitoring of international normalized ratio. He continues to have CHB with continuous requirement of ventricular pacing.

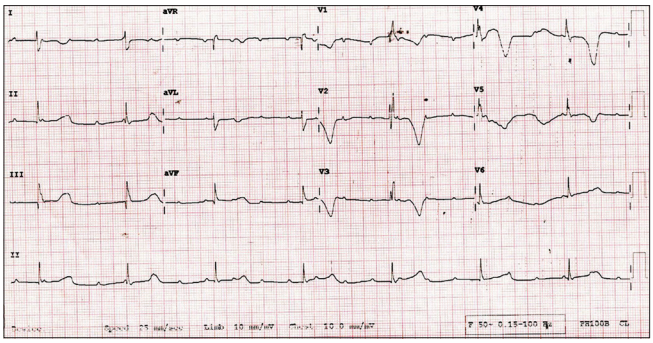

Figure 1: Twelve lead electrocardiography shows complete heart block with atrial rate of 148/min and ventricular rate of 42/min with right bundle branch block

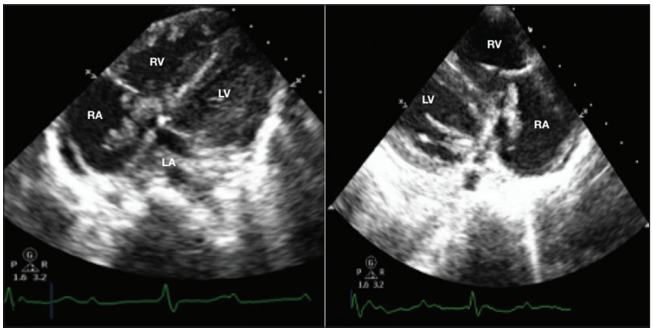

Figure 2: Transthoracic echocardiogram in apical four-chamber (panel A) and parasternal long axis (panel B) view shows large vegetation on septal and anterior leaflet of the tricuspid valve, respectively. RA: Right atrium, RV: Right ventricle, LA: Left atrium, LV: Left ventricle

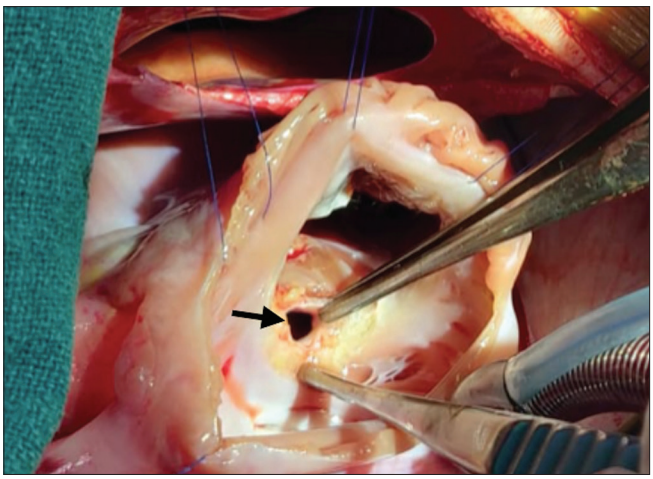

Figure 3: Intraoperative photograph, as seen from the right atrial aspect, shows a perimembranous ventricular septal defect (arrow) unmasked after removal of the vegetation on the septal leaflet of the tricuspid valve

DISCUSSION

Congenital heart disease (CHD) is the most common risk factor for right-sided IE in children. Among congenital heart diseases, small VSD is the most common lesion predisposing to IE with vegetations on tricuspid valve, right ventricular aspect of VSD, and rarely the lateral wall of the right ventricle.[8] Usually, the VSD is easily identifiable on echocardiography. The complete closure of the VSD by the vegetation on tricuspid valve is extremely rare with only two such reports in the literature.[6,7]

High-grade atrioventricular conduction block is rarely seen in IE and usually indicates the development of perivalvar complications. Most such reports are in the

setting of aortic or mitral valve IE. Despite anatomic proximity, conduction abnormalities are rare in tricuspid valve IE with only a few cases reported in the literature.[3,9,10] None of these cases had underlying CHD. The rarity of conduction abnormalities in tricuspid valve IE may be explained by the fact that penetrating portion of the His bundle pierces the central fibrous body through the atrial portion of membranous septum above the attachment of septal tricuspid leaflet and emerges on the left side at the level of the noncoronary aortic cusp, in proximity to the mitral ring.[11] We believe that the extension of the vegetation into the VSD in our case possibly brought it closer to the conduction system and caused permanent damage. Unlike few cases in the literature, CHB in our case did not reverse despite antibiotic therapy and surgical removal of the vegetation. This poor response to antibiotic therapy and surgery in our case is also possibly the result of prolonged illness and virulent causative organism.

CONCLUSIONS

Vegetation on the septal leaflet of the tricuspid valve can mask a perimembranous VSD. Extension of the vegetation into the VSD possibly brings it closer to the conduction system. Prolonged illness by a virulent organism and proximity of the vegetation to the conduction system can cause irreversible CHB, and therefore, one needs to be vigilant for conduction abnormalities, even in the setting of tricuspid valve IE.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the legal guardian has given his consent for images and other clinical information to be reported in the journal. The guardian understands that names and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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