Introduction

Left ventricle outflow tract (LVOT) obstruction is not uncommon in patients with transposition of great artery (TGA). The reported incidence varies from 30% to 38%.\(^1\)\(^2\)\(^3\) It may occur at various levels and may be caused by several anatomical structures including tricuspid valve tissue.\(^3\) The septal leaflet of tricuspid valve may prolapse through perimembranous VSD or rarely tricuspid valve tissue may override to produce LVOT obstruction. Occasionally, this may be mistaken for vegetation due to associated pulmonary valve endocarditis.\(^3\)

We report a case of d-TGA with presumptive pulmonary valve endocarditis and LVOT obstruction that was found to be due to tricuspid valve straddling on transesophageal echocardiography, resulting in change in the surgical plan and thus avoiding catastrophe.

Case Report

An 8-year-old male patient weighing 15 kg with congenital heart disease was referred to our institute with provisional diagnosis of infective endocarditis following two episodes of seizure and episodes of fever for the past 20 days. His general physical examination revealed cyanosis, clubbing, and features of developmental delay. Auscultation of chest revealed Grade IV pansystolic murmur over precordium. Neurological examination was unremarkable. Laboratory investigation showed Hb 17.3 g%, normal total leukocyte count, platelet count, and sterile blood culture. A transthoracic echocardiogram demonstrated situs solitus, levocardia, atrioventricular concordance, ventriculoarterial discordance, a large VSD (size 27 mm) with severe pulmonary stenosis (peak gradient of 80 mmHg), and a large (18 mm × 14 mm) mobile mass attached to the pulmonary valve. Computerized tomography pulmonary angiogram showed normal-sized pulmonary vessels with no evidence of pulmonary thromboembolism. The diagnosis of d-TGA with pulmonary valve stenosis and pulmonary valve endocarditis was made. He was planned for excision of vegetation.

His preoperative systemic oxygen saturation on room air was 85%. A transesophageal echocardiography examination after induction of anesthesia in midesophageal five-chamber view confirmed the presence of a large VSD and a mass attached to the undersurface of the pulmonary valve.

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In addition, a thick cord-like structure originating from the endocardial surface of the left ventricle was found attached to the mass [Figure 1 and Video 1]. Color Doppler examination showed turbulent flow across the pulmonary valve [Figure 2]. Continuous-wave Doppler confirmed the gradient across the obstruction. In midesophageal four-chamber view, chordae from the tricuspid valve were seen attached to the tip of interventricular septum (IVS) [Figure 3 and Video 2]. Midesophageal aortic long-axis view showed a mobile mass attached to the undersurface of the pulmonary valve, a large VSD, and normal-appearing pulmonary and aortic valves. A careful look showed the mass moving with the movement of the pulmonary valve and a chord-like structure attached to the mass after originating from both right and left ventricles [Figure 4 and Video 3]. The surgeon was informed about tricuspid valve straddling and LVOT obstruction due to tricuspid valve tissue. However, in view of the clinical presentation of fever, we decided to proceed with surgery.

The intraoperative surgical finding confirmed straddling of the tricuspid valve, and the mass attached to the pulmonary valve appeared as normal tricuspid subvalvular tissue. The pulmonary valve leaflets appeared normal. Excision of mass was abandoned, and a small piece of tissue from it was excised for histopathological confirmation. Postoperatively, the patient was discharged from hospital after a week of uneventful course with long-term plan for single-ventricle pathway. The histopathology confirmed the excised tissue as normal myocardium.

Discussion

The LVOT obstruction in d-TGA with VSD may be variously caused by fibromuscular tunnels or diaphragm, abnormal insertion and/or abnormal movement of the mitral valve, aneurysmal membranous IVS, and tricuspid valve tissue. Most VSD in d-TGA are conoventricular with some degree of malalignment; however, these are not necessarily juxta-tricuspid. Inlet location or inlet extension of a VSD causes tricuspid valve tensor apparatus to straddle the defect. The other potentially contributing factor is the malalignment of the muscular outlet ventricular septum. The overriding of the tricuspid valve causes LVOT obstruction when the inlet VSD extends anteriorly and includes a perimembranous component, and when the chords insert into the papillary muscle of the left ventricle, crossing part of the LVOT. In the index case, midesophageal five-chamber view showed turbulent flow across the LVOT due to a mobile mass attached to the pulmonary valve [Figure 2]. A chord-like structure arising from the mass was attached to the papillary muscle of left [Figure 1 and Video 1] and right ventricles [Figure 4 and Video 3]. The midesophageal four-chamber view showed an inlet VSD, relatively small-sized right ventricle, and the tricuspid valve chordae attached to the IVS [Figure 3], suggesting tricuspid valve straddling that was confirmed in an orthogonal plane [Figure 4].

Echocardiography may help in differentiating tricuspid valve straddling from other causes of LVOT obstruction in d-TGA such as aneurysm of the membranous IVS, anomalous insertion of the mitral valve, or a large vegetation due to associated infective endocarditis. Aneurysm of the membranous IVS appears as an outpouching around the VSD, while anomalous insertion of mitral valve at the lower edge of VSD and altered papillary muscle position is seen in cases of prolapsing mitral valve tissue. In the index case, mass was mistaken for vegetation due to pulmonary valve endocarditis.

Isolated pulmonary valve endocarditis is rare, affecting <1.5%–2% of patients suffering from infective
endocarditis.[6] In order of decreasing frequency, the valves affected by infective endocarditis are the mitral, aortic, tricuspid, and pulmonary. Right heart endocarditis is more common in patients who are intravenous drug users and can also occur in association with right-sided devices such as pacemaker leads.

Transesophageal echocardiography has higher sensitivity (90%–100%) than transthoracic echocardiogram (25% for 5 mm or less, and 70% of those between 6 and 10 mm).[7,8] Specificity of echocardiography is related to the clinical indication and the type of population studied.

The characteristic echo appearance of vegetation is of an echogenic mass, often irregular in shape, attached to the “upstream” side of a valve leaflet (i.e., the atrial side in the case of the mitral and tricuspid valves, the ventricular side for the aortic and pulmonary valves). It can be attached to any part of the valve, but most commonly at the coaptation line. Vegetation varies in size, often being just a few millimeters in diameter but sometimes reaching 2–3 cm. One resulting from fungal infections (e.g., Candida, Aspergillus) is usually much bigger than bacterial vegetation and can be so big that they are mistaken for a cardiac tumor. Vegetation moves with the leaflet but in a more chaotic (“oscillating”) manner. It is common for a vegetation to prolapse through the valve as it opens and regurgitation of the infected valve is always constant. However, echocardiography does not differentiate septic and aseptic vegetation.

In the index case, clinical feature of fever and seizure along with the presence of mobile mass on the undersurface of pulmonary valve leads to misdiagnosis of pulmonary valve endocarditis. A closer look on transesophageal echocardiography showed the presence of tricuspid valve straddling as a cause for LVOT obstruction that corroborated with intraoperative surgical and histopathological findings. Transesophageal echocardiography played a crucial role in altering the surgeon about change in diagnosis and thus avoiding the catastrophe of resection of whole mass culminating into pulmonary plethora, free pulmonary regurgitation, LV dysfunction, and its sequel.

**Conclusion**

The LVOT obstruction in TGA with ventricular septal defect may be rarely caused by tricuspid valve straddling. Detailed transesophageal echocardiography examination may helpful in differentiating it from other common causes of LVOT obstruction, thus avoiding catastrophe.

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**Conflicts of interest**

There are no conflicts of interest.

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Figure 3: Midesophageal four-chamber view showing chordae from the tricuspid valve attached to the tip of interventricular septum. LA: left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle, VSD: ventricular septal defect.

Figure 4: Midesophageal aortic long-axis view showing a mobile mass attached to the undersurface of the pulmonary valve, a large ventricular septal defect, and normal-appearing pulmonary and aortic valves.
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