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Percutaneous tricuspid valvotomy for pacemaker lead-induced tricuspid stenosis



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ABSTRACT

Permanent pacemaker lead-induced tricuspid regurgitation is extremely uncommon. We report a patient with severe tricuspid stenosis detected 10 years after permanent single chamber pacemaker implantation in surgically corrected congenital heart disease. The loop at the level of the tricuspid valve may have caused endothelial injury and eventually led to stenosis. Percutaneous balloon valvotomy for such stenosis has not been reported from India.

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An 8-year-old girl underwent surgical repair of subaortic ventricular septal defect without any temporary detachment of the septal tricuspid leaflet. Twelve days later, she developed complete atrio-ventricular block. A permanent VVIR pacemaker implantation was performed with a customary alpha loop retained in the right atrium. A pre-procedural echocardiography done revealed no residual shunt or regurgitation. Ten years later, she presented with fatigability and exertional presyncope. Constitutional symptoms such as fever or weight loss were absent. Clinical examination revealed prominent jugular 'a' waves and left parasternal mid-diastolic murmur and mild hepatomegaly. The hemogram, serum liver and renal function assays, and blood cultures were unremarkable. Pacemaker interrogation suggested that the lead impedance and lead threshold histograms were stable, but the battery life had reached ERI (elective replacement indicator).

Echocardiography revealed congested inferior vena cava and right atrial dilation. The anterior and septal tricuspid leaflets were thickened and tethered to the ventricular lead with characteristic diastolic doming (Fig. 1a and b). Subvalvular crowding was present with thickened, fused chordae. Doppler interrogation revealed peak and mean gradients of 15 and 9 mm Hg, respectively, across the tricuspid valve (Fig. 1c and d); color flow imaging showed grade II/IV central regurgitation. There was no vegetation. Tricuspid annulus measured 21 mm. Findings were confirmed on transesophageal echocardiography (Fig. 1e and f). A diagnosis of acquired (lead-induced) tricuspid stenosis without infective endocarditis was made. Percutaneous balloon tricuspid valvotomy using a 22 mm (100% of the tricuspid annulus diameter) Inoue mitral valvuloplasty balloon (Toray Industries Inc.) was planned. After reaching the right atrium with the slenderizing Inoue

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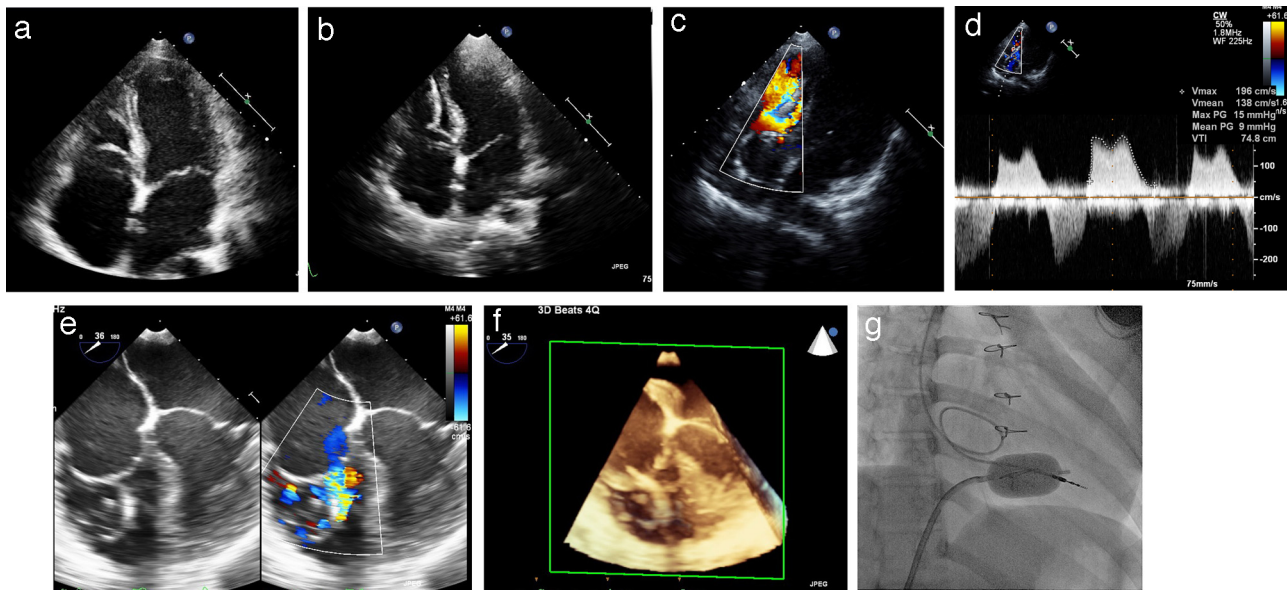


Fig. 1 – (a) Transthoracic echocardiography in apical four-chamber view demonstrating a thickened tricuspid valve with diastolic doming with the in situ ventricular lead better visualized at slight angulated view. (b and c) Transthoracic echocardiographic with color flow mapping showing turbulence across the tricuspid valve as two jets on either side of the ventricular lead. (d) Continuous Doppler interrogation across the tricuspid valve demonstrating high gradients. (e) Transesophageal echocardiography image in four-chamber view showing thickened and doming tricuspid valve with in situ ventricular lead with 3D reconstruction. (f and g) Fluoroscopic image in right 30° angulation demonstrating inflated balloon across the tricuspid valve. Also seen is the ventricular-lead loop in atria at tricuspid.

Balloon, the golden stylet was withdrawn up to the two-inch mark. With the help of the J-shaped torquer and anticlockwise rotation, the balloon was advanced across the stenotic valve. The distal balloon was then inflated in the right ventricle and the catheter shaft was slightly withdrawn until it got hitched and straddled at the tricuspid valve. Three full dilations were attempted (Fig. 1g). Fall in the mean transvalvular gradient from 8 mm Hg to 5 mm Hg was noted. This was followed by the replacement of the pulse generator. The patient tolerated the procedure well.

Tricuspid stenosis is a rarely encountered late complication of endocardial pacemaker implantation, generally in middle-aged patients. The usual mechanisms described include (i) obstruction to right ventricular inflow by tricuspid valve vegetations (i.e. infective endocarditis), (ii) leaflet perforation or laceration with resultant fibrosis, or (iii) adherence between the redundant portion of the lead and subvalvular tissue. Our patient was young and had lead encasement at both valvular and subvalvular levels. The loop at the level of tricuspid valve may have caused endothelial injury and resultant tricuspid stenosis in our patient. Most patients have a protracted subclinical course and present late with features of right heart failure or cardiac cirrhosis. Management revolves around decongestive therapy and surgical lead removal with tricuspid valve replacement.¹ Percutaneous management has also been lately reported.² The design of an Inoue balloon gives it an advantage over other balloons at even the tricuspid valve.

Though there was a symptomatic benefit post-procedure, the suboptimal procedural outcome in our case may be explained by a distorted valve and subvalvular apparatus, which was adherent to the pacing lead and did not yield sufficiently to balloon dilation. At 6 months post-procedural follow-up, the patient was apparently asymptomatic and the mean gradient across the tricuspid valve was 5 mm Hg. We believe that if lead parameters do not mandate lead replacement, then balloon valvotomy may be attempted with intent to cause controlled commissural rupture for symptomatic relief.

Conflicts of interest

The authors have none to declare.

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