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Case Report

High traffic congestion in right atrium

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ABSTRACT

A 62-year lady presented with limb swelling and heart failure due to leads induced venous fibrosis and severe tricuspid stenosis, 33 years after pacemaker implantation. After undergoing surgical removal of all leads and tricuspid valve replacement under cardiopulmonary bypass, she regained a normal functional status and tricuspid and right ventricular functions.

A 62-year-old female had permanent pacemaker implantation (VVI Mode, St. Jude Medical, St. Paul, MN) via right cephalic venous cut down for symptomatic complete heart block (CHB) in 1982. A year later, a new pacing unit (VVI mode, St. Jude Medical) was implanted from the right side, following pacemaker pocket infection. The old right-sided pacing lead could not be retrieved, and hence left in situ. Three years later, she again got admitted with right side pocket infection, for which a new pacing unit (VVI mode, St. Jude Medical) was implanted from left subclavian vein. The two ventricular leads from right side could not be retrieved, and hence remained in situ. Five years later, she again got admitted with pocket infection, this time on the left side. Again a new pacing unit (VVI mode, St. Jude Medical) was implanted from the left side after the removal of infected generator and a course of antibiotics. The left-sided old pacing lead could not be retrieved. By this time, the patient had four pacing leads – two superfluous ventricular leads from right side, one superfluous ventricular lead from left side, and one functional VVI pacing lead from left side (Fig. 1). Twenty years later in 2001, the depleted battery was replaced by a new VVI generator on the left side. Twenty-eight years later, she developed insidious onset, gradually progressive swelling of neck, both upper limbs, and facial puffiness. Ultrasound confirmed the thrombosis of bilateral subclavian veins. She was started on oral anticoagulants and target INR was maintained between 2 and 3. Thirty-one years later, she presented with dyspnea NYHA class II, lower limb edema, and abdominal distension. Physical examination revealed hepatomegaly, lower limb edema, and a diastolic murmur in tricuspid area. Two-dimensional echocardiography revealed dilated right atrium, with thickened tricuspid valve with stenosis (TS). Tricuspid valve area by continuity equation was 0.5 cm^2, and a mean diastolic gradient across it was 7 mmHg

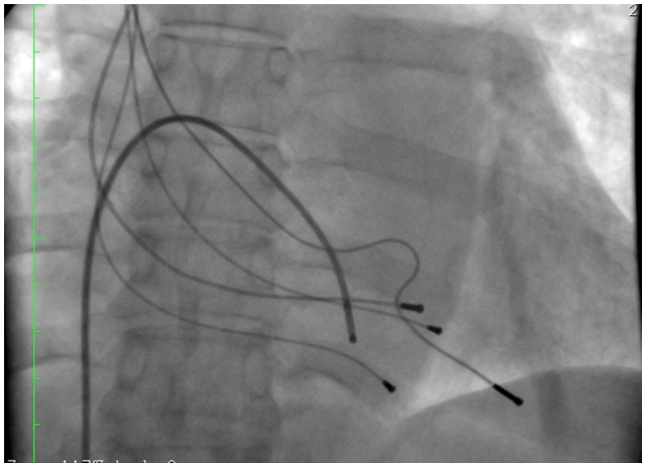

Fig. 1. Fluoroscopy showing four leads across tricuspid valve.

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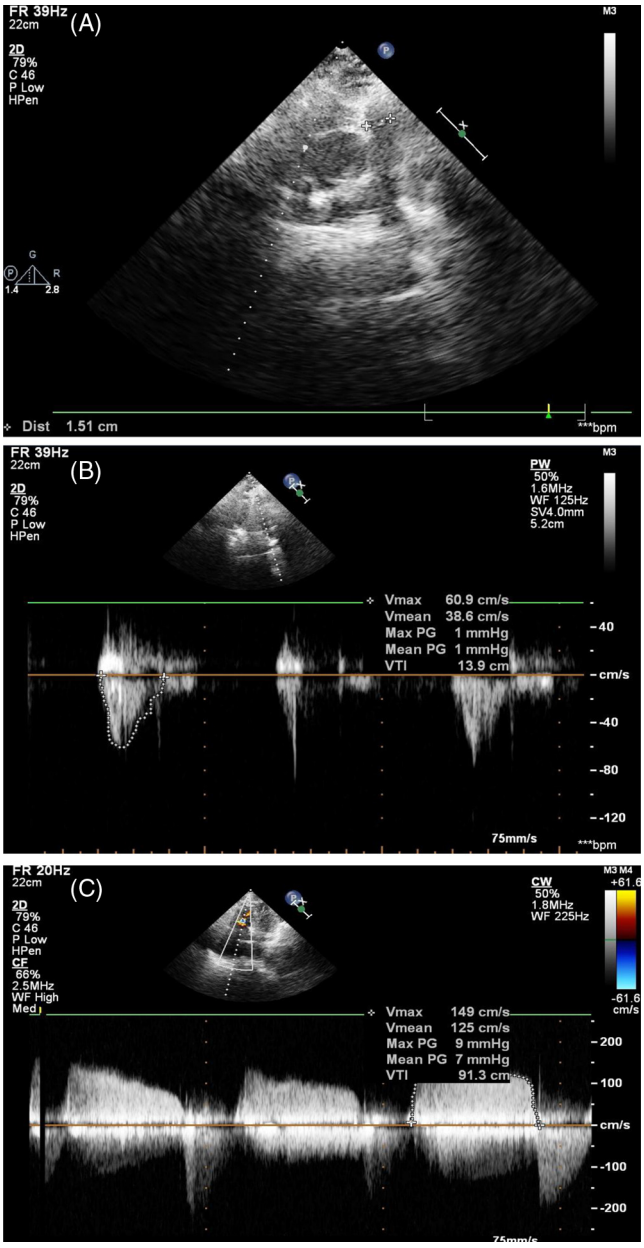

Fig. 2. (A) CW Doppler showing mean gradient across the tricuspid valve of 7 mmHg S/O severe T.S., tricuspid valve VTI was 91.0. (B and C) Calculation of stroke volume across RVOT.

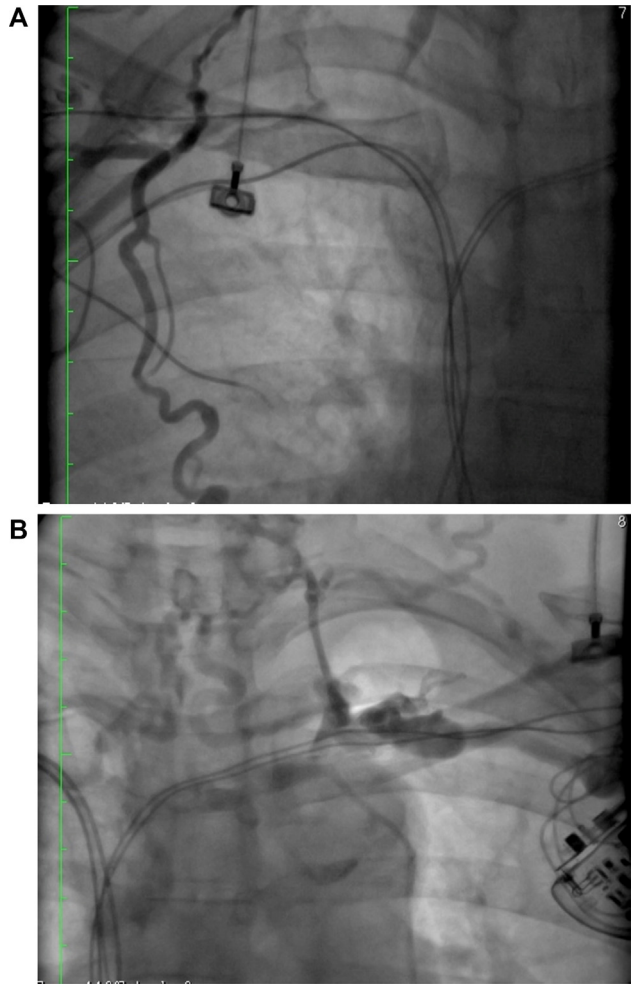

Fig. 3. (A) Venogram showing occlusion of right subclavian and internal jugular veins. (B) Venogram showing occlusion of left internal jugular and brachiocephalic veins with collaterals.

(Fig. 2A–C). She was managed with diuretics and anticoagulation as the family refused surgery. In April 2015, 33 years later, she presented with lead fracture of ventricular lead. Venography (Fig. 3A and B) revealed bilateral subclavian and superior vena cava occlusion, which was confirmed on cardiac CT scan. Cardiac catheterization revealed transtricuspid mean gradient of 7 mmHg, mean pulmonary artery pressure of 15 mmHg, and normal coronaries. She underwent surgery for removal of pacing leads, tricuspid valve and superior vena cava reconstruction, and a new epicardial pacemaker implantation.

Following median sternotomy, superior vena cava (SVC) and right atrium (RA) were cannulated. Tricuspid valve was thickened, fibrosed, and significantly stenosed. The commissures of all three leaflets were fused. All four leads were trapped and fused with tricuspid leaflets (Fig. 4). Tip of all four leads were firmly embedded in RV with severe fibrosis, preventing their removal despite applying an adequate traction. Tricuspid valve along with leads from the RV apex, interventricular septum, and inferior wall was excised. A 31-mm bioprosthetic valve (EPIC valve, St. Jude Medical) was implanted at tricuspid position. The fibrosed and stenosed SVC and bilateral brachiocephalic veins were reconstructed by autologous pericardium. A 10 mm PTFE graft was sutured to the pericardial augmented mouth of the left brachiocephalic vein using 6–0 prolene. An epicardial lead was sutured at LV apex and generator was placed in left rectus sheath (VVI Myodex lead, St. Jude Medical). The postsurgery course was uneventful. A repeat echocardiography showed 2 mmHg mean gradient across bioprosthetic tricuspid valve. Patient was asymptomatic at 3 months of follow-up.

1. Discussion

Permanent pacing leads in heart causes fibrosis of adjacent structures, such as RV apex, tricuspid valve, SVC, and brachiocephalic veins.[1] Tricuspid valve stenosis[2] as a consequence of ventricular pacing leads is infrequent, and only few cases have been published in the literature.[3–7] The mechanism for tricuspid valve stenosis includes continuous friction between leads and valve leaflets leading to endothelial damage and later fibrosis.[8,6,9] Multiple leads across tricuspid valve as in index case would have accelerated the pathogenetic process. Moreover, these pacemaker

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