Luminal vena cava superior and right atrial obstruction associated with severe tricuspid valve stenosis due to endocardial pacemaker leads: an extremely rare complication of permanent pacemakers

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In this report, we describe a case with severe tricuspid stenosis associated with partial luminal obstruction of the right atrium and vena cava superior secondary to transvenous pacemaker leads in a 49-year-old patient who had a permanent pacemaker implanted 17 years ago. The patient had no specific symptoms related to above findings; however, after clinical suspicion, transthoracic and transoesophageal echocardiographic examination showed large mobile masses attached to the thickened transvenous pacemaker leads. Leads and generator were removed surgically, but some parts of transvenous leads could not be extracted due to massive fibrotic adhesions. It is possible that such cases will be more common due to increasing number of cardiac rhythm device implantations.

Keywords

Permanent pacemaker • Pacemaker leads • Tricuspid valve stenosis • Infective endocarditis • Luminal vena cava superior obstruction

Introduction

Permanent pacemakers and implantable cardioverter-defibrillators (ICD) are becoming increasingly used for the treatment of cardiac rhythm disturbances worldwide and early and long-term complications related to these devices are also becoming more common. Although various tricuspid valve abnormalities secondary to pacemaker leads are reported in the literature,1,2 iatrogenic tricuspid valve stenosis due to pacemaker lead associated masses and lead adhesion is an extremely rare complication of permanent cardiac pacemakers. In this case report, we describe a patient with iatrogenic severe tricuspid stenosis associated with partial luminal obstruction of the right atrium and vena cava superior secondary to transvenous pacemaker leads.

Case report

A 49-year-old male with unstable angina pectoris presented to our cardiology clinic and underwent coronary angiography.

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right heart chambers (see Supplementary data online, Movie 1). Using continuous-wave Doppler echocardiography, significant tricuspid valve stenosis (mean diastolic gradient $= 9 \text{ mmHg}$) with moderate regurgitation was noted (Figure 1). On the subcostal view, it was found that inferior vena cava diameter was 2.2 cm without inspiratory collapse. Subsequent transoesophageal echocardiography (TEE) showed significant thickening of subvalvular apparatus of tricuspid valve (Figure 1 and also see Supplementary data online, Movie 2). Also, partial obstruction of vena cava superior lumen was noted. His serial white blood cell count, erythrocyte sedimentation rate and C-reactive protein values were within normal limits and no fever was detected during clinical follow-up. Liver function and coagulation tests were also normal. Three separate blood cultures revealed no specific microorganism. With the above findings, patient was referred for surgery. Intraoperatively, massive fibrotic tissue causing partial obstruction of right atrium and vena cava superior lumen was observed at gross inspection. Also, one of the transvenous leads was found to perforate tricuspid annulus close to posterior leaflet. Massive fibrosis was present particularly at the subvalvular level (Figure 2). Leads were excised from the surrounding tissue but small parts of the lead material were not able to be removed because of excessive fibrotic tissue. After tricuspid annuloplasty and valvotomy, right atrium and vena cava superior were enlarged with patch. Postoperative TTE revealed mild-to-moderate regurgitation and mean diastolic gradient of 3 mmHg at the tricuspid valve (Figure 2). Dense fibrosis with thrombotic material was found at the histopathologic examination. Also extracted lead material was sent to culture but no microorganism grew. Since multiple preoperative 24-h Holter electrocardiography recordings demonstrated very rare paced rhythms, pacemaker reimplantation was not planned before surgery. Two months after surgical operation, patient was still asymptomatic and no significant pauses or bradycardia episodes were present at Holter recordings.

**Discussion**

Although numerous complications after the implantation of permanent pacemakers were reported in long term, luminal vena cava superior and right atrium narrowing associated with tricuspid valve stenosis secondary to transvenous pacemaker leads are extremely rare. Lead related leaflet perforation or laceration may cause endothelial injury with subsequent formation of thrombus and scar tissue around the tricuspid valve leaflets and subvalvular apparatus. Looping of endocardial leads at tricuspid valve level may cause similar changes as well. Also, atrial wall perforation by the pacemaker lead may cause significant adhesions, calcification, and scar tissue formation along the leads and eventually result in luminal narrowing and tricuspid valve stenosis. Tricuspid valve abnormality is also reported to be more frequent in the presence of more than one right ventricular lead across the tricuspid orifice. In our case, there were more than one right ventricular lead and one of the leads was found to perforate the tricuspid annulus. Infective endocarditis related to transvenous pacemaker leads is another postulated mechanism. In recent series of pacemaker-related infective endocarditis, retaining the device and leads

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**Figure 1** (A) and (C) Transthoracic echocardiogram showing thickened pacemaker leads, and mobile masses (arrows) from the apical four-chamber view (LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle). (B) Transoesophageal echocardiogram showing the tricuspid valve in the four-chamber view. (D) Tricuspid valve gradient with continuous-wave Doppler echocardiography.
intact was associated with higher morbidity and mortality rates due to relapsing or persistent infection.6,7 Therefore, complete hardware removal is recommended as a class I indication, even only for generator pocket infections.8 In our case, however, complete hardware removal had not been performed despite recurrent pocket infections. It can be speculated that healed infection of transvenous leads might have caused excessive fibrosis and adhesions to surrounding tissue.

Echocardiography is a useful clinical tool for the diagnosis of pacemaker-related infective endocarditis. Owing to its higher sensitivity and better negative predictive value, TEE evaluation should always complete the TTE examination. TEE has superior features particularly for the evaluation of number and size of lead-associated masses. Besides, assessment of tricuspid valve involvement and better visualization of right atrium and vena cava superior are other advantages.9,10 Some authors suggested that TEE examination could also be helpful in deciding the most appropriate lead extraction strategy. By identifying the presence of myocardial abscess and very large vegetations, surgical lead removal is recommended rather than a percutaneous lead extraction method.6

Supplementary data
Supplementary data are available at European Journal of Echocardiography online.

References