Severe tricuspid regurgitation secondary to dislodgement of the atrial loop into the right ventricle: an unusual complication of pacemaker implantation in a young adult

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Summary Transvenous pacemaker leads may impair tricuspid valve function. Severe tricuspid regurgitation due to leaflet adhesion to the pacemaker lead has not been reported in a young adult patient in the literature. Our patient underwent a transvenous pacemaker implantation for symptoms of bradycardia. An atrial loop was created in the right atrium for future growth. After 10 years of follow-up, the patient was seen with severe tricuspid regurgitation and enlarged right heart structures due to migration of the atrial loop of the pacemaker lead into the right ventricle and adhesion of the lead to the tricuspid valve. Cardiac surgery and epicardial pacing was the chosen procedure to solve this problem. The venous system and right heart valvular should be carefully observed during the follow-up of children who underwent transvenous pacing.

Keywords Pacemaker – tricuspid regurgitation – atrial loop – complication – adhesion – pacemaker lead.

INTRODUCTION

Transvenous pacing systems are highly preferred for the treatment of bradyarrhythmias. The implantation of a pacemaker (PM) is generally safe and complication rates are low. However, PM leads may impair tricuspid valve function. Mild tricuspid regurgitation (TR) is a commonly reported problem. Severe TR due to entrapment of the PM lead and adhesion of the lead to the tricuspid leaflet has been reported in only a few elderly patients².³ Some authors suggest creating an atrial loop to minimize the lead length/growth mismatch³.⁴ We report a patient with severe TR secondary to the migration of the atrial loop of the PM lead and restriction of movement of the anterior leaflet. To our knowledge, this complication has not been reported before in a young adult patient in literature.

CASE REPORT

A 9-year-old boy with congenital complete atrioventricular block underwent implantation of a transvenous endocardial pacemaker for prevention of symptoms secondary to bradycardia. He had a normal sinus node function and a normal ventricular size and function. The endocardial pacing lead was inserted by percutaneous puncture of the subclavian vein and a PM generator was placed in a subpectoral pocket. A single-lead VDD pacemaker (atrial synchronous ventricular inhibited pacing, model: Medtronic, 8168) with bipolar steroid-eluting lead was implanted into the right ventricular apex with a floating dipole in the right atrium. An atrial loop was created and redundant lead was kept at the right atrium for future growth.

The patient was followed up regularly (every 6 months). He underwent clinical examination, telemetric PM interrogation and a standard electrocardiogram was taken at every follow-up visit. An echocardiogram, Holter monitoring, exercise testing and chest X-ray were performed yearly. During follow-up tricuspid regurgitation (TR) was not reported. The patient had remained asymptomatic for 10 years. When the patient was 19 years old, he was referred to our cardiology department with complaints of palpitations and fatigue.
Physical examination revealed a 2/6 pansystolic murmur on the xiphoid area. A 12-lead electrocardiogram showed a VDD pacemaker rhythm. At the chest x-ray lung fields were clear, the pacing lead appeared intact. The tip of the lead was at the right ventricular apex. The redundant part of the lead formed a loop and moved towards the right ventricle (figure 1). Echo-Doppler examination identified the lead route and position. The lead was passing between posterior and anterior leaflets. Echocardiography revealed that the lead did not move freely and a part of the lead appeared to be adhered to the anterior leaflet (figure 2). There was lead-induced mild obstruction to the tricuspid valve and second- to third-degree TR with a peak velocity of 2.7 m/sec. The colour jet of the regurgitation was approaching the right atrial roof. Right atrium and ventricle were enlarged moderately.

Elective surgical lead change to prevent further valve dysfunction and implantation of an epicardial lead was planned. During surgery the lead was found to penetrate into the right ventricular myocardial tissue at the apex. It was found adhered to the anterior tricuspid valve leaflet and the wall of the right ventricle and covered with granulation tissue. Invasion of the lead tightly to the superior vena cava and the innominate vein was also reported. The parts of the lead in the innominate vein wall and the myocardial tissue were so tightly adhered that these parts could not be extracted. They were left in place and the rest of the lead was extracted. After lead extraction a dual-chamber (DDDR) pacemaker was implanted epicardially. Right atrium and left ventricle apex were the chosen places for pacing.

The patient has been followed for two years after surgery, and there was improvement of the tricuspid valve regurgitation. TR decreased to a mild level due to a dilated tricuspid valve annulus. Clinical and echo-Doppler follow-up examinations showed no increase of tricuspid valve regurgitation or no sign of tricuspid stenosis. In the latest echo-Doppler examination the right atrium and the right ventricle seemed mildly dilated. There was second-degree TR with a peak velocity of 2.5 m/sec.

DISCUSSION

Pacemaker-related long-term complications may occur in about 10-30% of the patients in various reports, mainly related to leads.\textsuperscript{4,5} Endocardial leads may fail for technical reasons. Lead deterioration occurs or lead dysfunction may be due to ageing, or patient's growth. Lead fracture, insulation break, dislodgement, infections, thromboembolic events, abnormalities in pacing or sensing, exit block or abnormal threshold increase are some of the reported complications.\textsuperscript{4-6} Risk factors for lead failure include younger age at implantation, structural congenital heart defects and epicardial lead placement.\textsuperscript{6}
An atrial loop was created in our patient in order to prevent inappropriate lead stretching with somatic growth. In the literature, addition of an atrial loop or leaving some redundancy as slack to avoid tension on the pacing leads is reported to produce a better outcome for ventricular endocardial leads implanted before adolescence. Redundant atrial loops enable a period of reliable ventricular pacing without the need for lead replacement procedures and allow for patient growth and movement. The loops uncoiled successfully in most reports. Silvetti et al. reported 17 children with the addition of an atrial loop. None of them experienced any lead failure; however, their follow-up period has been reported to be short (2.5 ± 1.4 years). In our patient, some part of the atrial loop has uncoiled as the patient grew up; however, the rest of the residual lead was found to be dislodged into the tricuspid valve and the right ventricle. This complication is not reported before in a young adult patient in the literature. PM-related mild TR is a common echocardiographic finding. Severe TR is rare and has been reported in adult patients, mostly in the elderly age group. The literature about PM-related TR is debatable. It is still not clear whether clinically relevant TR is completely due to an interference with valve closure by the lead.

The most likely mechanism of lead-related severe TR was found to be obstruction to leaflet closure in previous reports. Lead adherence to the valve tissue is also reported in a few elderly patients. Other mechanisms reported previously include perforation and laceration of the tricuspid valve. A delay in activity within the right ventricle (RV) or asynchronous RV activity from apex to base or alterations in RV geometry have also been postulated. These are mostly adult and elderly population studies and the most common reason of TR from all these reports seems to be insufficient valve closure.

Seo et al. reported that the leads were positioned on the annulus side between leaflets in most patients with trivial/mild TR. Lead-induced obstruction to the tricuspid valve ring was seen in most patients with severe TR. Septal or posterior leaflet obstructions are reported in the literature in severe TR cases. In our case, anterior leaflet obstruction was the cause of severe TR.

In another report by Lin et al., 39% of the patients had lead impingement only of the tricuspid valve leaflets at surgery, 34% of the patients had adhesion of the PM lead to a tricuspid valve leaflet, 17% had leaflet perforation and 10% had entanglement of the tricuspid valve apparatus. All had severe TR and the age group was 70 ± 10 years when compared with our patient who was a young adult.

There is clear indication for lead extraction if there is lead interaction with the cardiac structures. Most of the literature related to this intervention is focused on transvenous lead extraction. Although it was possible to perform transvenous lead extraction in our case, we chose surgery to extract the lead in order to obtain a direct view of the lead and tricuspid valve. Another advantage of surgery was the epicardial pacing. Avoiding transvenous pacing would prevent further damage to the tricuspid valve apparatus.

CONCLUSION

In conclusion, severe TR from leaflet adhesion to the pacemaker lead may also occur in young patients. During follow-up of children who underwent transvenous pacing one should carefully control the venous system and the right heart valves. Surgical extraction and epicardial pacing may be preferred in cases with lead-induced tricuspid valve problems.

CONFLICT OF INTEREST: none declared.