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Case Report

Tricuspid stenosis: An emerging disease in cardiac implantable electronic devices era. Case report and literature review

Aysha Husain (MD MRCP)[a,*], Faris Tufail Raja (MD)[a], Ahmed Fatallah (MD)[b], Bahaa Fadel (MD FACC)[a], Aly Alsanei (MD)[a], Fahad Tufail Raja (MD)[a], Bandar AlGhamdi (MD)[a]

[a] Heart Center, King Faisal Hospital and Research Center, Riyadh, Saudi Arabia
[b] Radiology Department, King Faisal Hospital and Research Center, Riyadh, Saudi Arabia

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ABSTRACT

Tricuspid valve dysfunction and in particular tricuspid stenosis has recently been described secondary to cardiac implantable electronic devices. The valve is subjected to different mechanisms of injury related to the endocardial lead passing through its plane. The lead can form a loop or perforate one of the leaflets and initiate inflammatory response and fibrotic changes. Multimodality cardiac imaging is required to diagnose this clinical entity and decide on the best treatment plan. Here we present a case of a young female who developed tricuspid stenosis secondary to permanent pacemaker lead that was implanted 24 years before. We performed a review for all cases reported in the literature with a similar condition and various treatment approaches.

<**Learning objectives:** 1. Tricuspid valve dysfunction can develop secondary to cardiac implantable electronic devices. Tricuspid regurgitation is the most common valve lesion however, tricuspid stenosis is reported as well. 2. Endocardial leads can cause injury to the valve initiating a cascade of inflammatory response and fibrosis. 3. Trans-thoracic echocardiography is the initial diagnostic modality but visualization of lead injury requires further cardiac imaging such as cardiac computed tomography and trans-esophageal echocardiography. 4. Various treatment modalities are reported in the literature; medical therapy, percutaneous valvoplasty; and surgery.>

Introduction

Cardiac implantable electronic devices (CIEDs) lead-related tricuspid valve (TV) dysfunction is considered one of the causes of TV disease in the modern endocardiac devices era [1]. This clinical entity is not yet clearly defined, and it is likely underestimated. The complexity underlies the quantification of TV dysfunction secondary to the lead versus the underlying clinical disease. The most common TV dysfunction related to lead implantation is TV regurgitation; this association was first described by Gibson and colleagues in 1980 [2]. TV stenosis, on the other hand, is reported less frequently with diverse treatment approaches that include percutaneous and surgical techniques. We report a case of pacemaker-induced TV stenosis after 24 years of pacemaker lead implantation with a literature review of this problem.

Case report

A 38-year-old female underwent ablation of her right posteroseptal accessory pathway in September 1992 for recurrent supraventricular tachycardia. One year later, she developed high-grade atrioventricular block for which a dual chamber pacemaker device was inserted. Generator change was done in 2001 and 2012, but the leads were not replaced. She started to complain of exertional shortness of breath that was progressive and limiting her daily activities, 24 years after the implantation of the endocardial leads. She also noticed lower limb swelling with an increase in abdominal girth. On examination, her pulse was regular with heart rate of 90/min and blood pressure of 128/79 mmHg. Her cardiovascular examination revealed engorged jugular vein with diastolic murmur III/VI over left the sternal border. Her abdomen was distended with ascites, and her legs were edematous. Her

* Corresponding author at: Adult Cardiology Fellow, King Faisal Hospital and Research Center, MBC16, Heart Center, P.O. Box 3354, Riyadh 11211, Saudi Arabia.
E-mail address: aishaonco@gmail.com (A. Husain).

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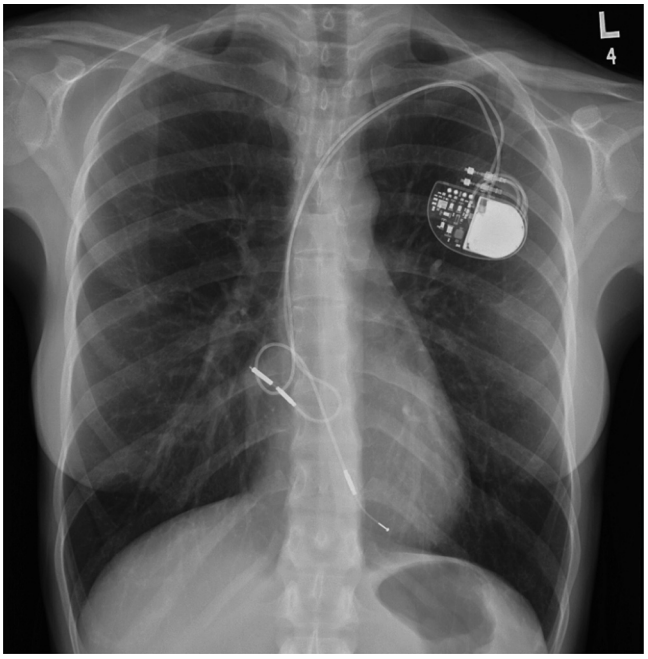

Fig. 1. Chest X-ray showing the loop of the atrial pacemaker lead as it crosses down and returns back to the right atrium.

chest X-rays showed the atrial lead forming a loop as it crossed down to the right ventricle (RV) and returning to right atrium (Fig. 1). Transthoracic echocardiography identified TV stenosis with a mean gradient of 11 mmHg and a maximum gradient of 29 mmHg. The right atrium was dilated and left ventricle was normal in size and function. Transesophageal echocardiography

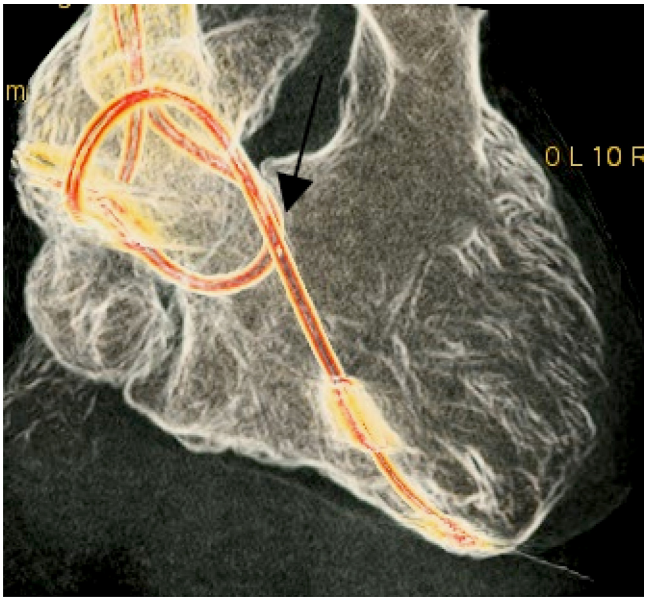

Fig. 2. Contrast-enhanced cardiac computed tomography of the heart (volume rendered image) demonstrates the atrial pacemaker lead forming a loop as it crosses down to the right ventricle and returning to the right atrium against the septal leaflet.

demonstrated the involvement of the lead in TV, as there was evidence of restricted opening of the septal and anterior leaflets.

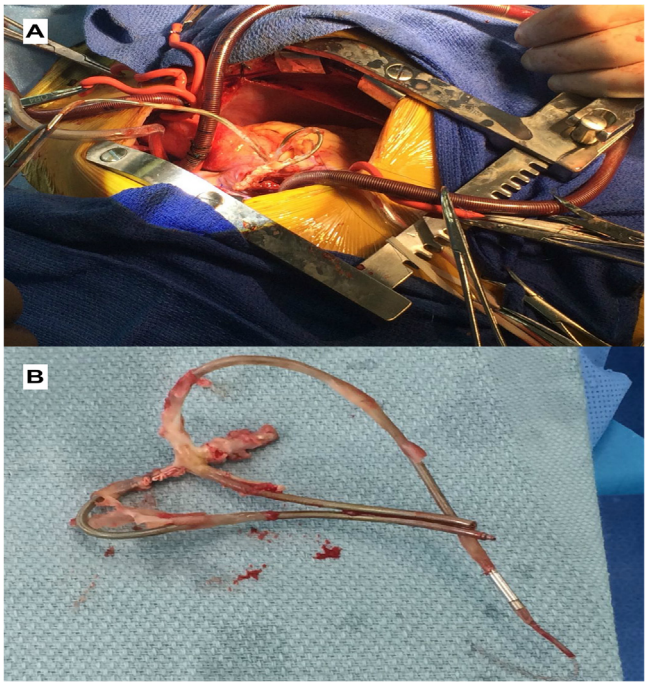

Fig. 3. (A) Pacemaker lead looping found during surgical extraction distorting the tricuspid valve. (B) Extracted pacemaker lead with a loop with formation.

This was due to the anterior leaflet either stuck to the pacemaker wire or perforated by the wire as it crossed into the RV. To confirm it further computed tomography cardiac imaging was requested, and it showed the atrial lead was looped and stretched against the septal leaflet (Fig. 2) The case was discussed in our heart team meeting and we decided to go for lead extraction surgically and TV repair as it was felt that balloon valvoplasty would likely yield a suboptimal result due to excessive fibrosis. During surgery, the atrial lead was noticed to be looping against the septal leaflets restricting its opening and perforating the anterior leads as it returned to right atrium causing leaflet adhesions and fibrosis (Fig. 3). TV annuloplasty was done and old endocardial leads were extracted with new dual chamber leads inserted. She was discharged home five days postoperatively with no complications.

Discussion

TV stenosis secondary to endocardial leads is reported scarcely in the literature. By doing a Pubmed search for (TV stenosis) and (lead), we found 16 case reports in English literature after excluding the endocarditis-related cases (Table 1) [3–10].

All these cases had permanent pacemaker leads that caused TV stenosis rather than other endocardial devices. The time from implantation to the clinical presentation can vary from being as early as few weeks to a late presentation in 30 years. The underlying mechanisms for TV stenosis related to endocardial leads can be either due to a mechanical injury or obstruction by loop formation. The mechanical injury causes a cascade of inflammatory response resulting in fibrosis and adhesions. Types of mechanical injury observed included (1) lead perforation (2) lead adherence and tethering (3) lead loop restricting the opening, and (4) adhesion to subvalvular apparatus. Some cases of an early presentation were primarily related to mechanical obstruction by loop formation. This patient suffered from severe TV stenosis likely secondary to endothelial injury at the level of the TV that may