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Case Report

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Retained Pacemaker Lead Fragment-Induced Fibrosis, Resulting in Impaired Tricuspid Valve Function, Managed with a Valve-Sparing Open Surgical Approach

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Abstract

Retained pacemaker lead fragments can induce fibrosis which can affect valve function. In this case, a female patient in her fifties had undergone pacemaker insertion in her teens for symptomatic bradycardia. Due to pacemaker pocket erosion, she had undergone a lead extraction where lead fragments were left in-situ. Over time, she gradually developed symptomatic tricuspid dysfunction. Due to the severe impact on her quality of life, the patient opted for an open surgical approach. Intra-operatively, electrocautery was used to debride the fibrotic tissue inhibiting the leaflets of the tricuspid valve. This resulted in marked improvement of valve function and additional repair/replacement was not necessary. To our knowledge, such a case has not been previously described.

Keywords: Pacemaker, Fragment, Inflammation, Fibrosis, Tricuspid Dysfunction

Case Report

In 2015, a fifty-year-old female presented with worsening tricuspid dysfunction. She had undergone pacemaker implantation in her teens due to symptomatic bradycardia. The pacemaker and leads were extracted ten years later due to pocket erosion. During otherwise uncomplicated laser extraction, several lead fragments were left in-situ which could not be safely retrieved without high-risk intervention. A new pacemaker generator and leads were relocated.

The patient then slowly developed proximal venous congestion compatible with superior vena cava (SVC) syndrome. A venogram in 2010 demonstrated completely occluded brachiocephalic and innominate veins with significant adjacent venous collateralization. The patient underwent an uneventful generator change in 2011 and a computed tomography (CT) scan showed partial obstruction of the SVC and tricuspid stenosis (TS). A conservative approach was taken.

The patient later developed hepatic congestion and cirrhosis secondary to elevated right sided pressures and right atrial congestion due to TS. She was unable to lie flat and her limbs were edematous. She was unable to walk more than a few meters due to abdominal distention and pain. The patient's condition was the result of occluded proximal veins, SVC syndrome, and functional TS, all of which were the result of fibrotic tissue, secondary to pacemaker lead-induced inflammation. The lead fragments left in-situ likely contributed. The patient was scheduled to have all leads removed from the venous system. She would then be implanted with an epicardial pacing system with the lead tunneled to a position above the right epigastrium; access to the pacemaker would then be simplified. The major surgical concern was the unknown state of the TV; there was concern that the valve would be so scarred that repair might not be possible. Should a valve replacement be necessary, the patient agreed to a bioprosthetic valve, despite the possibility for redo surgery in the future, as a bio prosthesis would avoid anticoagulation.

Intra-operatively, the SVC was small caliber, non-mobile, and rigid. The IVC was of normal caliber but the IVC/RA junction had a white-yellow discoloration consistent with fibrosis; it was rigid and stenosed (Figure 1A). The RA was of small size with reduced

contractility. The patient was placed on cardiopulmonary bypass with bi-caval cannulation and a right atriotomy was performed after SVC and IVC snares were tightened around the cannulae. On atriotomy, it was immediately apparent that the atrial wall was significantly thickened. Once inside the atrium, the SVC snare was carefully released to assess SVC flow while also avoiding an airlock. However, there was no visible inflow into the RA from the SVC. Locating the TV was difficult due to the fibrosed and contracted RA. Once visualized, it was clear that the exaggerated fibrotic reaction that had formed around the pacemaker leads had caused significant reduction in the right atrial size and entrapped the TV, impairing its function (Figure 1B).

Using electrocautery, the fibrotic tissue that fixed the pacemaker leads to the RA walls and TV leaflets was debrided. This was surprisingly beneficial and further valve repair/replacement was not indicated. Fibrotic tissue at the atrial wall and IVC/RA junction was also debrided allowing for better inflow. Unfortunately, inflow could not be restored from the SVC even after removal of the leads, and the patient was left with SVC syndrome. After removal of the leads and remnants (Figure 2A), the atriotomy was closed with a bovine patch (Figure 2B). Since the intra-operative findings were unexpected, and the patient had not given consent, SVC bypass or alternative intervention was not considered. This would have required extensive dissection and post-operative anti-coagulation.

Comment

Severe TV dysfunction due to device implantation is uncommon [1]. The presence of foreign bodies can result in localized inflammation, fibrosis, and encapsulation which can complicate lead removal. This can result in raised central venous pressures due to increased right sided preload which can increase right atrial volume. In turn, this can reduce venous return and result in low cardiac output. Diagnosis is usually by examination and echocardiography. Transesophageal echocardiography +/- CT may provide additional benefit [1]. The first reported case of TV leaflet adhesion caused by pacemaker leads was described in 2006 [2]. Prior cases have been treated with valve replacement, surgical valvuloplasty, or percutaneous balloon angioplasty [2-4]. No prior case has been described where a patient has been successfully managed with an open surgical valve-sparring approach.

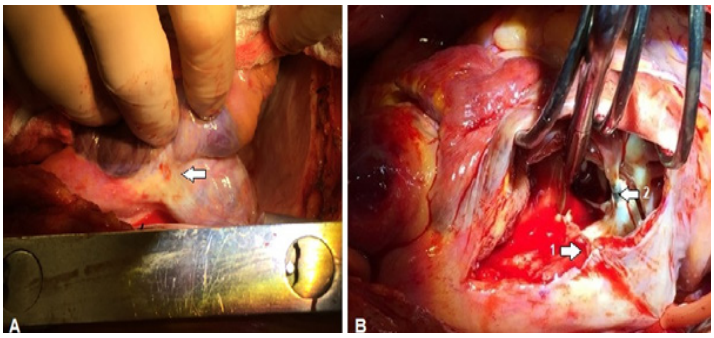

Figure 1: (A) Fibrosis at the IVC/RA junction (arrow). (B) The right atriotomy and thickened atrial wall (arrow 1). The tricuspid valve bound to a pacemaker lead (arrow 2).

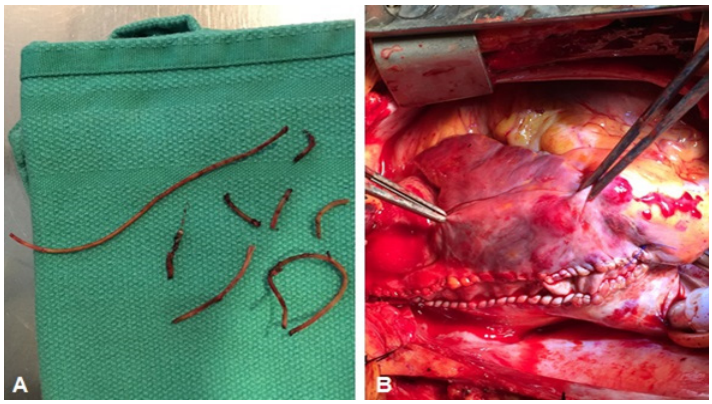

Figure 2: (A) The leads/lead remnants removed from the patient. (B) Closure of the right atrium with a bovine patch

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