Pacemaker Lead–Induced Tricuspid Valve Stenosis?

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INTRODUCTION

Tricuspid valve (TV) stenosis (TS) is a rare condition, occurring in 2.4% of cases of primary TV disease, and is most often due to rheumatic disease. Other causes are congenital heart disease, endocarditis, and large right atrial tumor. Rarely, TS secondary to pacemaker (PM) leads is reported. We report a case of PM lead–induced TS and describe a multidisciplinary approach to evaluate such patients.

CASE PRESENTATION

A 54-year-old woman with no previous medical history had been diagnosed with dilated cardiomyopathy 12 years earlier. Her primary complaint was functional dyspnea classified as New York Heart Association (NYHA) class II–III. Despite optimal heart failure medical treatment, the patient remained symptomatic with an ejection fraction (EF) of 20%. The electrocardiogram showed sinus rhythm with left bundle branch block and QRS width of 160 msec; therefore, the patient had a cardiac resynchronization therapy device implanted. The patient demonstrated an excellent response to the resynchronization therapy with relief of symptoms (NYHA I), and the EF increased afterward to 50%. Twelve years after the cardiac resynchronization therapy, a high-risk finding given the presence of patent foramen ovale and large right atrial tumor. Rarely, TS secondary to pacemaker (PM) leads is reported. We report a case of PM lead–induced TS and describe a multidisciplinary approach to evaluate such patients.

DISCUSSION

The most common dysfunction of the TV related to PM implantation is tricuspid regurgitation, while TS has been reported very rarely. Based on the few available cases of TS caused by PM lead implantation, the pathology of TS appears to be due to either mechanical damage to the endothelium or an obstruction based on a loop formation of the lead. The mechanical damage causes an inflammatory response resulting in fibrosis, calcification, and eventually stenosis. The mechanical damage can be caused by lead perforation, lead adherence and tethering, lead loop restricting the opening, and adherence to subvalvular apparatus.
In the present case no findings indicated a mechanical obstruction due to loop formation of the PM lead, and the cause of TS was considered likely to be due to mechanical damage of the endothelium of the valve with induction of inflammation and fibrosis leading to the development of TS. In addition, no thrombus material was demonstrated at the TV. The fact that the therapeutic anticoagulation therapy treatment did not alter the morphological or functional performance of the TV supports these considerations.

The most commonly used treatment for TS caused by PM implantation has been surgical removal of the lead and subsequent repair or replacement of the TV. Other treatment options are medical treatment or percutaneous balloon valvotomy. Although long-term data are limited, percutaneous treatment of PM lead--induced TS seems to be safe and effective.

Due to the complex structure and function of the TV, TS can be challenging to evaluate. Therefore, it is of great importance to use a multidisciplinary and multimodality approach, which can provide anatomical and functional data, to thoroughly evaluate the degree of stenosis and plan the optimal treatment strategy.

With the use of a transthoracic echocardiography we were able to establish TS as a working diagnosis for the patient. To further characterize the TS and confirm the diagnosis, 3D TEE offered a more accurate TV area and a more detailed anatomical characterization of the valve pathology. Using right-sided catheterization, the TV area was estimated to be 0.5 cm², which was almost equivalent to the TEE finding (0.7-1.0 cm²). Cardiac CT was also used, but due to artifacts from the PM lead it could not be used to describe the TV and the underlying pathology. Thus, by use of advanced echocardiography and right-sided catheterization, we were able to ensure the rare diagnosis and provide an optimal strategy for treatment.

Autopsy studies and heart surgical data have shown that PM leads develop fibrotic attachment to the TV during long-term follow-up and will result in a greater degree of fibrotic adhesion. With an increasing number of PM implantations, the risk of TS may be expected to increase, but further studies must be conducted to determine a possible increased incidence of TS.

CONCLUSION

Tricuspid valve stenosis caused by PM lead implantation is very rare, and to ensure the diagnosis and optimal treatment strategy, a multidisciplinary approach is recommended. Given the increasing number of PM implantations, a lead-induced TS must always be included in the

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Figure 3 (A, B) Intraoperative picture of the TV seen from the RA. The valve leaflets are marked “anterior,” “posterior,” and “septal.” Retractor placed on the left side of the patient. Venous cannulas are shown. The PM lead passing through the valve is shown with widespread adhesions to the valve tissue as seen in panel C. The valve leaflets are fibrotic and in combination with the adhesions make the valve stenotic. (B) Forceps holding a fibrotic valve leaflet released from the annulus. (C) Removed PM lead.

Figure 2 Left panel: Resting simultaneous pressure tracings from the right atrium (light green) and right ventricle (red). Right panel: TV area calculated as 0.49 cm² using the Gorlin equation. Heart rate, 89 beats per minute; cardiac output, 2.9 L/minute; RA mean gradient, 9 mm Hg; pulmonary artery mean gradient, 9 mm Hg; tricuspid mean gradient, 7.73 mm Hg; and TV flow, 60 mL/sec. In both panels the corresponding electrocardiogram tracings are at the top. Note that the waveforms are shown with different sweep speed, not different heart rate.
differential diagnosis for patients with a PM who present with unexplained TS.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2021.07.003.

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