

# Severe Tricuspid Stenosis related to Endocardial Pacemaker Lead

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**ABSTRACT:** Tricuspid stenosis (TS) is an uncommon complication of transvenous ventricular pacing. We present a case of TS developed 14 years after pacemaker implantation. The etiology of stenosis appeared to be inflammation and fibrosis of tricuspid valve (TV) secondary to leaflet perforation by the pacemaker lead and it was managed successfully with surgical valvuloplasty. Iatrogenic TS may occur more frequently than is clinically suspected and it should be considered in any patient with endocardial leads presenting with right heart failure.

**DESCRIPTORS:** tricuspid valve stenosis, complications, pacemaker, heart failure.

## CASE REPORT

We describe the case of a 62 year old man who had as first manifestation of cardiac disease a hospitalization for unstable angina and third-degree AV block in 1993. He underwent percutaneous coronary intervention of the left anterior descending coronary artery and had a VVI pacemaker implanted.

The pulse generator was replaced in 1997 because of generator pouch inflammation. In July 2002, he was readmitted to the hospital with generator pouch inflammation/necrosis, without signs of systemic infection or endocarditis. The pulse generator was replaced and upgraded to a DDDR unit implanted in the opposite site keeping the original ventricular lead.

In October 2007, he presented to the emergency department with a 3-month history of asthenia, exer-

tional dyspnoea (NYHA III functional class), lower limb edema, and chest pain upon medium effort. Physical examination revealed jugular venous distension, a systolic murmur grade 2/6 and a low-frequency diastolic murmur at cardiac auscultation, hepatomegaly and lower limb edema extending to the upper thigh. ECG showed atrial fibrillation with intermittent ventricular pacing. Laboratory investigation revealed elevated levels of  $\gamma$ -GT, alkaline phosphatase, and B-natriuretic peptide (950 pg/mL). Chest X-ray showed cardiomegaly and a redundant loop of one of the ventricular leads (figure 1).

Transthoracic echocardiography (TTE) revealed a severely dilated right atrium, TV with ill-defined morphology, severe tricuspid stenosis (mean gradient = 10 mmHg; area = 0.8 cm<sup>2</sup>), and mild insufficiency (figure 2). Left ventricular size and function were

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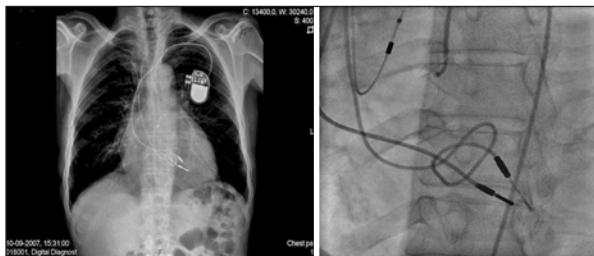


Figure 1 - A) Posterior-anterior Chest X-ray demonstrates two pacemaker leads crossing the tricuspid valve with a redundant loop of the original ventricular lead. B) Ventricular pacemaker leads best visualized in the left anterior oblique view at right coronariography.

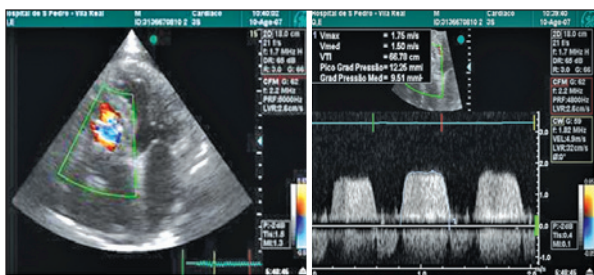


Figure 2 - Transthoracic echocardiography with color flow Doppler revealing a severely dilated right atria with aliasing/turbulence of right ventricular inflow (left) and continuous wave Doppler demonstrating a mean TV gradient of 9.5 mmHg (right).

normal. Transesophageal echocardiography was performed for better morphologic characterization and revealed a TV with pronounced leaflet and subvalvular thickening and reduced mobility, a mean gradient of 12 mmHg, and mild regurgitation. Heart catheterization showed a mean TV gradient of 8 mmHg with normal right ventricular and pulmonary artery pressures; coronary angiography indicated right coronary stenosis of 50%.

The patient was proposed for surgery. The operation showed a severely dilated right atrium, fibrosis of TV leaflets and subvalvular apparatus, predominantly at pacemaker leads interface, and severe TV stenosis. The original pacemaker ventricular lead was perforating the septal leaflet of the TV.

A tricuspid valvulotomy (commissurotomy between anteroseptal and posteroseptal commissures) and a saphenous venous graft to the right coronary artery

were performed. The endocardial pacing system was explanted and replaced with an epicardial system. The procedure was uncomplicated and the patient had an uneventful recovery.

At 15 months of follow-up the patient was well and TTE showed mild TV stenosis (mean gradient of 3 mmHg) and moderate insufficiency.

## DISCUSSION

TV stenosis is a rare complication of transvenous pacing, with few cases reported in the literature. The mechanisms described are obstruction to right ventricle inflow by tricuspid vegetations (endocarditis) or multiple pacemaker leads and TV fibrosis secondary to mechanical trauma by the pacemaker lead<sup>1-4</sup>.

TV trauma by the endocardial pacing system may be induced by laceration, perforation or adherence of redundant loops of the lead to valvular tissue. This endothelial injury promotes a sequence of local events consisting of chronic inflammation, fibrosis, calcification, and eventually valvular stenosis. A similar process has been suggested in an anatomopathological study of ICD patients<sup>1,5</sup>.

There are three previous case reports in which TV stenosis was believed to be secondary to fibrosis induced by leaflet perforation, and four other cases as a result of adherence of redundant loops of the pacemaker lead. In the majority of these cases, there was more than one pacemaker lead crossing the TV and the time that elapsed between first pacemaker implantation and development of symptoms ranged from 7 to 33 years<sup>2</sup>. The reported management included medical management in three, TV replacement in two, surgical valvuloplasty in one and balloon angioplasty in the other<sup>4</sup>.

In our case, TV stenosis was induced by leaflet perforation and was diagnosed 14 years after pacemaker implantation. It was managed successfully with surgical valvuloplasty despite moderate TV regurgitation in the last TTE.

We believe that this chronic complication of pacemaker implantation may occur more frequently than is clinically suspected and it should be considered in any patient with endocardial leads presenting with signs or symptoms of right heart failure.

This entity might even be more common in the future due to the rising number and long-term utilization of intracardiac devices.

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Margato RFPC, Fontes JP, Santos PR, Moreira JI. Estenosis tricuspídea severa inducida por electrocatéter de pacemaker. *Relampa* 2010;23(3):119-121.

**RESUMEN:** La estenosis tricuspídea se constituye en una complicación poco frecuente de la implantación de pacemakers. Los autores presentan un caso clínico de estenosis tricuspídea diagnosticada 14 años tras la implantación de pacemaker. La estenosis valvular fue secundaria a la reacción inflamatoria y fibrosis de la válvula tricuspídea inducida por perforación de uno de los folletos valvulares por el electrocatéter de pacemaker, que fue tratada con éxito por cirugía (valvuloplastia). La estenosis tricuspídea iatrogénica podrá ser más frecuente que se la detecta habitualmente en la práctica clínica y se deberá considerarla en cualquier paciente portador de electrodos endocavitarios que presente insuficiencia cardiaca derecha.

**DESCRIPTORES:** estenosis tricuspídea, complicaciones, marcapasos, insuficiencia cardiaca.

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**RESUMO:** A estenose tricúspide se constitui numa complicação pouco frequente de implante de marcapassos. Os autores apresentam um caso clínico de estenose tricúspide diagnosticada 14 anos após o implante de marcapasso. A estenose valvular foi secundária a reação inflamatória e fibrose da válvula tricúspide induzida por perfuração de um dos folhetos valvulares pelo eletrocatéter de marcapasso, que foi tratada com sucesso por cirurgia (valvuloplastia). A estenose tricúspide iatrogênica poderá ser mais frequente do que é habitualmente detectada na prática clínica e deverá ser considerada em qualquer paciente portador de eletrodos endocavitários que se apresente com insuficiência cardíaca direita.

**DESCRIPTORES:** estenose tricúspide, complicações, marcapasso, insuficiência cardíaca.

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