Case Report

Severe Tricuspid Stenosis related to Endocardial Pacemaker Lead

Renato Filipe de Paiva e Cunha MARGATO¹ José Paulo FONTES² Policarpo Rosa SANTOS³ José Ilídio MOREIRA⁴

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, exertional 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 γ-GT, alkaline phosphatase, and B-na-triuretic 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 cm²), and mild insufficiency (figure 2). Left ventricular size and function were...

Article done in the Centro Hospitalar de Trás-os-Montes-e-Alto-Douro, Unidade Vila Real.
(1) Resident in Cardiology.
(2) Cardiologist, board member of Portuguese Association of Arrhythmology Pacing and Electrophysiology (APAPE).
(3) Cardiologist.
(4) Cardiologist. Director of Cardiology Department.
Mailing address: Serviço de Cardiologia do Centro Hospitalar de Trás-os-Montes-e-Alto-Douro. Av. da Noruega - Lordelo 5000-508. Vila Real - Portugal. E-mail: renatomargato@yahoo.com
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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 (comissurectomy between anteroseptal and posteroseptal comissures) 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.

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.

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

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 à reação inflamatória e fibrose da válvula tricúspide induzida por perforação 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 que é habitualmente detectada na prática clínica e se deverá considerar em qualquer paciente portador de eletrodos endocavitários que presente insuficiência cardíaca direta.

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

BIBLIOGRAPHICAL REFERENCES