Partial Rupture of the Tricuspid Valve after Extraction of Permanent Pacemaker Leads: Detection by Transesophageal Echocardiography

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ASSAYAG, P., ET AL.: Partial Rupture of the Tricuspid Valve after Extraction of Permanent Pacemaker Leads: Detection by Transesophageal Echocardiography. Traumatic lesions of the tricuspid valve complicating pacemaker lead extractions appear to be rare. We report two cases of partial rupture of the tricuspid valve, following apparently uneventful extraction of permanent ventricular leads, resulting in severe regurgitation and, in one case, chronic heart failure. TEE was useful to identify the traumatic mechanism of tricuspid regurgitation (TR) and the extent of valvular lesions in these patients. Such etiology should be suspected, and TEE performed, in patients developing TR or heart failure late after lead extraction. (PACE 1999; 22[Pt. I]:971-974)

tricuspid regurgitation, pacemaker lead, lead extraction, transesophageal echocardiography

Introduction

Tricuspid regurgitation (TR) complicating extraction of endovenous pacemaker leads is rare. Most cases occurred during implantation procedures with entrapment of tined leads in the tricuspid valve apparatus, needing their immediate withdrawal.1,2 TR complicating extraction of chronically implanted leads has been exceptionally reported,3 with no clinical consequences. We describe two cases of partial rupture of the tricuspid subvalvular apparatus, following apparently uneventful extraction of permanent leads, identified by transesophageal echocardiography (TEE), and resulting in chronic heart failure in one case.

Case Report

Case 1

A 71-year-old man was referred to our institution for congestive right heart failure. He had a history of atrioventricular (AV) block, with a permanent ventricular pacemaker implanted 20 years ago. Five months before admission, he underwent the extraction of a dysfunctioning ventricular tined lead (Medtronic type 5064, Medtronic Inc., Minneapolis, MN, USA) by manual traction, and the procedure was uneventful. A transthoracic echocardiography (TTE) performed before extraction documented the absence of TR or tricuspid lesion, and normal left ventricular function. At admission the patient had painful hepatomegaly, jugular vein congestion, and bilateral leg edema. Auscultation revealed a significant tricuspid insufficiency. The patient was afebrile, and blood cultures were sterile. TTE demonstrated a severe TR (Fig. 1), and volume overload of the right ventricle. Biplane TEE identified multiple ruptured chordae of the tricuspid valve with a full prolapse of the anterior leaflet (Fig. 1). The patient was improved with diuretic therapy, but had recurrent episodes of cardiac failure during the 5-year follow-up.

Case 2

A 65-year-old man with a history of sick sinus syndrome and with a sequential AV pacemaker since 3 years (ventricular tined lead: A.P.C.I. 3272M), was referred after prostatic surgery for nosocomial staphylococcus aureus septicemia
and secondary septic osteo-arthritis of the knee, and was treated 30 days with vancomycin and rifampycin. He was readmitted 5 months later with chronic septic osteo-arthritis and bacteriemias; the pulse generator site was normal, there was no peripheral stigmata of endocarditis, and cardiac auscultation revealed no murmur. Staphylococcus aureus was recovered from blood cultures and joint puncture. Antibiotic therapy was initiated, with immediate aprexy. TTE and TEE examination, at this time, showed no evidence of vegetation on cardiac valves or on pacing leads, no tricuspid insufficiency, and no lesion of the tricuspid apparatus. Lead extraction was decided because of repeated episodes of systemic infection. The procedure necessitated a prolonged external traction during 10 days in the department of electrophysiology: no complication was reported, and cultures of the leads were sterile. However cardiac
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auscultation revealed a new holosystolic murmur increasing with the Carvallo maneuver, with no peripheral sign of congestion, and a new echocardiographic control was performed. TTE demonstrated the onset of an important TR, and biplane TEE showed the rupture of a papillary muscle head (Fig. 2), with complete prolapse of the posterior leaflet. New leads were implanted a few days later, and the patient remained asymptomatic during 1-year follow-up, with stable TR.

Discussion

Traumatic lesions of the tricuspid apparatus complicating extraction of pacemaker ventricular leads have been rarely reported. Most cases occurred during complicated implantation procedures, with entrapment of tined leads or ventricular arrhythmias necessitating immediate withdrawal with forceful traction. In these reports, the trauma was immediately diagnosed as partial avulsion of tricuspid tissue which was visible on the tip of the removed lead.

Extraction of permanent ventricular lead implanted for a long time may be difficult, due to fibrosis along the lead or at the endocardial site of the tip. Fatal or life-threatening complications occur in 2.5% cases, including myocardial avulsion and hemopericardium, ventricular arrhythmias, migrating lead fragment, and pulmonary embolism, and most complications are related to difficulties from freeing leads from scar tissue. However, tricuspid injury appears to be exceptional: one case was immediately evidenced after prolonged external traction, as tricuspid valve tissue and chordae were attached to the removed electrode tip, and the patient remained asymptomatic during follow-up. We report two cases of partial rupture of the tricuspid subvalvular apparatus secondary to permanent lead extraction, resulting in important regurgitation, one case presenting as right-sided cardiac failure. This complication was initially unrecognized during the procedure, which was reported to be uneventful, and the traumatic lesions of the tricuspid valve were documented later by echocardiography. In our patients, as in the previous report, extraction was performed using external traction. No tricuspid lesion or insufficiency was reported in large series using intravascular techniques with countertraction. The use of intravascular sheaths over the lead could be less deleterious for the tricuspid apparatus. However, in these studies, there were no systematic ultrasound assessment of the tricuspid valve function, and the incidence of such complication may be underestimated.

TEE was particularly useful to identify the traumatic mechanism of TR in our patients, demonstrating rupture of a papillary muscle head or of multiple chordae tendineae. It allowed us to eliminate other possible causes of TR in patients with permanent leads, such as valve perforation, adherence between the lead and the leaflets, or tricuspid endocarditis complicating lead infection, particularly in patient case 2 who had had septicemia. TEE also disclosed the type and extent of traumatic lesions. Identification of a partial rupture of papillary muscles is of particular interest, as complete rupture may occur later. Traumatic TR is often well tolerated for a long time. However, severe regurgitation may result in progressive heart failure, necessitating valve surgery. In both patients, partial rupture of the tricuspid apparatus resulted in important regurgitation, leading in case 1 to congestive heart failure.

In conclusion, partial rupture of the tricuspid apparatus and significant regurgitation may occur during apparently uneventful extraction of permanent leads, and the frequency of this complication might be underestimated. Traumatic lesions should be suspected in patients developing TR and unexplained heart failure late after an extraction procedure. Echocardiographic assessment of the tricuspid valve after lead extraction is useful, and in case of significant tricuspid insufficiency, TEE should be performed to identify the extent of the traumatic lesions, which can be a determinant of its long-term tolerance.

References

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