Reel syndrome: dislodgement of an active fixation lead

Reel sendromu: Aktif fiksasyonlu leadin yer değiştirimesi

Introduction

Reel syndrome occurs due to spontaneous retraction of pacemaker leads, which causes lead dislodgement and severe complications (1-3). We reported a case of Reel syndrome with dual-chamber pacemaker in which an actively fixated ventricular lead was coiled around the generator and retracted back into the pocket. The tined atrial lead remained in post implantation position.

Case Report

A 79-years-old woman was admitted to hospital with presyncope, lightheadedness, and right pectoral pulsation. Patient had been undergone a dual-chamber pacemaker implantation 1 week ago because of total AV-block. Chest radiography showed the tined atrial lead near the superior tricuspid annulus. The active right ventricular lead was retracted under the right clavicle (Fig. 1). A fluoroscopic image performed just after the first implantation demonstrated that the tined atrial lead was placed near the superior tricuspid annulus and active ventricle lead implanted to right ventricular apex (Fig. 2). Comparison of fluoroscopic image with new chest radiography showed the rotation of generator (Fig. 1b, 2a). The atrial lead was in same location and ventricular lead was retracted under the right clavicle. A new surgical procedure was performed after 24 hours. During revision procedure we observed a large and deep sub pectoral pocket containing hematoma. The ventricular lead was out of the vein and coiled around the generator. The generator was not sutured to the fascia of the pectoral muscle, and only single loose sleeve sutures were observed on both leads. The ventricular lead was implanted successfully after performing a new vein puncture. The atrial lead was successfully repositioned to right atrial appendage. The sleeves and the generator were tightly sutured to the fascia. During 1 month follow-up period no new complications occurred.

Discussion

Reel syndrome describes the spontaneous retraction of pacemaker leads into the pocket without patient manipulation (1, 2). However, this syndrome was thought to be a variant of Twiddler’s syndrome, consciously or unconsciously manipulation by the patient is not required (1-5). We thought that Reel syndrome is better defines the clinical scenario occurred in our case. During reimplantation procedure we observed that the ventricular lead was circled around the generator and fluoroscopic image demonstrated the rotation of generator (Fig. 1, 2). There are a few case presentations reporting this syndrome either in patients with single lead or with multiple leads (1-7). Interestingly the actively fixated ventricular lead dislodged in our case instead of tined atrial lead, which was not optimally implanted. Twisting the generator consciously or unconsciously by the patient is the mechanism of Twiddler’s syndrome (8, 9). Previous reports demonstrated that not suturing the generator to fascia and a large pacemaker pocket can cause moving the generator by itself (3). Moreover inadequately suturing the lead sleeves facilitates the retraction of leads. Patel et al. (3) described 4 cases with Reel syndrome and in all cases a single securing suture had been placed on leads. In our case the pacemaker generator was implanted under the pectoral muscle. Additionally the leads were loosely fixated with single sutures, and the active fixation ventricular lead was dislodged. In clinical practice some operators focus on placing the leads to appropriate location. They do not pay enough attention to pocket preparation nor to adequate lead and generator fixation. But preparing an adequate pocket, suturing lead sleeves at least with 2 separate tight sutures, and suturing the generator to the fascia are as important as lead placement for successful pacemaker implantation.

Conclusion

The Reel syndrome is a rare but potentially life threatening complication in patients with bradycardia pacing. There is no consensus on underlying mechanisms and diagnostic criteria of Reel syndrome which may cause underestimation of this complication.

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References

Successful percutaneous balloon mitral valvuloplasty in patients with left atrial appendage thrombus

Sol atriyal apendikte trombüs olan iki hastada başarlı mitral balon valvüloplasti

Introduction

Percutaneous balloon mitral valvuloplasty (PBMV) has become the treatment of choice for patients with symptomatic mitral stenosis since its successful use by Inoue et al. (1) in 1984. One of contraindications to this technique is the presence of thrombus in the left atrium (LA) or left atrial appendage (LAA). Nevertheless, there are some publications indicating that Inoue technique can be safely performed in patients with LAA thrombus (2).

In this report, we describe two patients with rheumatic mitral stenosis referred for PMBV and were found to have LAA thrombus. Despite the presence of LAA thrombus, successful PMBV, with the help of transthoracic echocardiography (TTE), was undertaken without complications.

Case Reports

Case 1

A 51-year-old woman was diagnosed with rheumatic mitral stenosis and referred to our hospital for PBMV. TTE revealed mild mitral regurgitation, moderate-severe mitral stenosis with a mitral valve area (MVA) of 1.1 cm² and systolic pulmonary artery pressure (PAP) of 70 mmHg.

Maximum and mean gradients across the valve were 23 and 11 mmHg respectively. Transesophageal echocardiography (TEE) revealed thrombus in the LAA (Fig. 1). Wilkins mitral valve score was calculated as 7. Mitral valve replacement (MVR) was offered to the patient, but she refused. PBMV was explained to the patient with risks of complications. After her informed consent for the procedure, along with TTE guidance, interatrial septum was punctured from more basal than usual and dilation was performed by Inoue balloon with as less manipulation as possible (Fig. 2). The catheter equipment was kept at the mid LA level and away from the appendage. When the balloon was deflated, great caution was exercised to avoid the catheter tip springing up to the appendage. The procedure was completed successfully without complications. TTE showed reduction of valve gradients, maximum gradient was 8.5 mmHg and mean gradient was 4 mmHg with MVA of 1.8 cm². Systolic PAP was 30 mmHg.

Case 2

A 56-year-old woman was diagnosed with mitral stenosis and atrial fibrillation in 2007. She was being followed on β-blocker and anticoagulant therapy. She was admitted to our clinic with progressive dyspnea, which limited her daily activity. On TTE, bialtral dilatation, moderate mitral...