Reel syndrome in a patient with an implantable cardioverter-defibrillator

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ABSTRACT
The Twiddler syndrome and its variant, the Reel syndrome, are uncommon complications in patients with an implantable cardioverter-defibrillator, but they can cause severe device dysfunction. The identification of the predisposing factors and the intervention on them, as well as the periodic monitoring of the operation by means of telemetry and chest radiography, are useful in the prevention of these syndromes. Here is described the case of a patient with Reel syndrome at the third month after the implantation of a cardioverter-defibrillator, as a result of a sepsis in the generator pocket.

Key words: Reel syndrome, Implantable cardioverter-defibrillator, Electrodes, Complications

INTRODUCTION
The Twiddler syndrome was described for the first time in a patient with a single-chamber pacemaker in 1968, by Bayliss et al. It consists in the rota-
tion of the generator on its axial axis with torsion of the electrodes, which can reach its fracture or displacement and, therefore, to cause the device’s dysfunction. Veltri et al.\textsuperscript{2} reported for the first time the presence of this syndrome in a patient with an implantable cardioverter-defibrillator (ICD) in 1984.

The Reel syndrome is a variant of the Twiddler syndrome, in which the generator's rotation occurs in its transversal axis, with reel of the electrodes around the device. Carnero-Varo et al.\textsuperscript{3} made the first description in a patient with a single-chamber pacemaker, in 1999. Next, we describe the case of a patient with Reel syndrome in the third month of an ICD implantation.

**CASE REPORT**

A 38-year-age male patient, normal weight, without personal pathological history of interest, and with the diagnosis of primary ventricular fibrillation. An ICD VDD (Iforia 5 VR-T-Dx) was implanted via left cephalic access. The active fixation electrode (Protego DF-1-Pro MRI Dx 65/15) was located at the apex of the right ventricular and fixed to the pectoralis muscle fascia with the corresponding fixing rings. The measurements for the implant were the following: threshold 0.7 V; amplitude of the R wave 6.7 mV; amplitude of the P wave 2.2 mV; and impedance 552 Ω. The generator was located in a subcutaneous pocket and no attention was paid to the muscle fascia. A basic pacing rate of 40 beats per minute (bpm) and a window of ventricular atrial fibrillation of 200 bpm were programmed.

At the third month of the implant, the patient came to the first follow-up and referred slight discomfort in the pocket, but without signs of acute inflammation. The electrocardiogram (Figure 1) showed sinus rhythm with a heart rate of 80 bpm, capture failure of the ventricular channel and of detection of both channels. The device was applied telemetry, which demonstrated an impedance of 560 Ω of the ventricular electrode and the defibrillation electrode of 54 Ω, absence of R and P waves' detection, and ventricular capture failure. The anteroposterior chest radiography (Figure 2) showed a retraction of the electrode, with location of the distal end at the level of the innominate vein, reel around the generator, compatible with a Reel syndrome. There was neither antecedent in the patient of excessive manipulation of the generator, nor of the performance of repetitive postural vices.

When performing the surgical procedure for repositioning the electrode, a pocket sepsis was observed, with dehiscence of the electrode fixation.
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points. The infected system was totally removed and no fracture of the electrode was found. The patient refused the reimplantation of a new system on the contralateral side, i.e., a treatment with quinidine sulfate, 200 mg every 8 hours, was started.

**COMMENT**

The Twiddler and Reel syndromes are rare in patients who carry pacing cardiac systems. They take place by different mechanisms, but are a common cause of device dysfunction. In most Reel syndrome reports, the electrodes are displaced, without damage, unlike the Twiddler syndrome, where the rotation mechanism produces fractures of the cables. In this patient was observed a displacement of the electrode to the innominate vein, with reel around the generator, but no fracture. The normal values of the impedances' stimulation and defibrillation demonstrated the integrity of the probe.

The forms of presentation of these syndromes are variable and their prevalence is approximately 0.07%5. The displacement or fracture of the electrodes can cause detection failure, increase of the impedance or of the stimulation threshold, with capture failure and phrenic, brachial or pectoral stimulation. In ICD carriers can be presented failures in the detection by defect or excess, as well as the increase of the defibrillation threshold with ineffectiveness of the therapy6,7. The detection and capture failure had an asymptomatic course in this patient, non-dependent on pacing stimulation. No dysfunctions of the ICD therapy were registered either.

The chest radiography confirms the diagnosis when proving the generator rotation within the pocket, the displacement of the endocardial electrodes and the reel of them around the generator8.

In some cases, it has been possible to demonstrate excessive voluntary or involuntary manipulation of the generator by the patient, but in the vast majority, the cause is unknown. Nevertheless, there have been described the predisposing factors for the development of these syndromes, such as: female sex, older age, obesity, neuro-psychiatric disorders, repetitive postural vices, very large subcutaneous pockets, weight and size of the generator, the abdominal location and device replacement by another one smaller9,10.

Some measures are proposed at the time of the implantation, in order to prevent these syndromes, such as: fixing the generator to the muscle fascia and the electrodes with their corresponding fixing rings, locating the subpectoral generator, using a dacron bag and limiting the size of the pocket, although the latter measure may favor aseptic necrosis.
An infection in the first ICD implantation is rare and ranges from 0.4-5.0%, according to different series\textsuperscript{11}. We consider that the loss of electrode fixing points as a result of the sepsis was the predisposing factor for the development of the Reel syndrome in this specific case\textsuperscript{9,12}.

The early identification of patients with predisposing factors and regularly monitoring the functioning and location of the electrodes, telemetry and chest radiography, will guarantee an early diagnosis of these syndromes, and major complications due to device dysfunction are avoided.

REFERENCES