Transient Phrenic Stimulation due to Twiddler Syndrome

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Twiddler syndrome is a rare complication of implanted permanent pacemaker and implantable cardioverter defibrillator (ICD), with a reported incidence between 0.07% and 7% [1]. The syndrome is caused by patient conscious or unconscious device manipulation in the pocket, resulting in torsion and painless lead dislodgement with subsequent malfunction of the implanted device.

We report a case of transient phrenic stimulation due to Twiddler syndrome.

PATIENT DESCRIPTION

A 59-year-old man, who underwent a primary prevention ICD implantation for non ischemic dilated cardiomyopathy presented to the emergency department complaining of abdominal contraction.

A week earlier an ICD (St. Jude Medical S.C., Inc, Ellipse™ DR, USA) was implanted in the left subcutaneous pectoral region. The leads were inserted via left axillary vein puncture: the atrial lead (Solia JT 53 Biotronik, Germany) and positioned in the right atrial appendage. The screw-in right ventricular lead (SJM 7122Q-65) was screwed in the high intraventricular septum. Both were secured to the muscle fascia with non absorbable suture. Satisfactory leads parameters for pacing and sensing were obtained.

The patient was not known to have cognitive deficit and denied that he manipulated the device.

At entrance chest X-ray showed the atrial lead retracted into the superior vena cava [Figure 1A]; the day after revision procedure for lead reposition was planned. At this time at fluoroscopy the screw-in right ventricular lead was retracted into the left brachiocephalic vein, while the atrial lead was totally retracted in the pocket encircling the pulse generator [Figure 1B]. The abdominal contractions were absent. After that the pocket was opened a cluster of twisted electrodes connected to the ICD was found [Figure 1C]. Both leads have been deformed and needed to be replaced. The ICD was enveloped in a Tyrx absorbable antibacterial envelope and repositioned in a sub pectoral pocket.

COMMENT

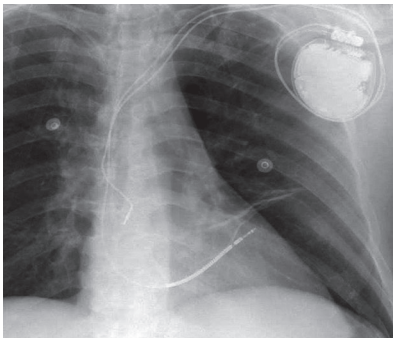
In a pacemaker dependent patient Twiddler syndrome may be a lethal complication. In ICD patients the treatment of life threatening ventricular arrhythmias would be disabled; moreover inappropriate ICD therapy may be pro arrhythmic.

The clinical manifestations that bring the patients to medical attention depend on the site of the dislodged lead. A retracted lead into the superior vena cava may stimulate the phrenic nerve and induce abdominal contraction [2,3]. A further retracted lead may induce pectoral muscular twitching [4] or stimulate the brachial plexus and cause arm twitching [5]. Inappropriate shock was reported during Twiddler syndrome [1].

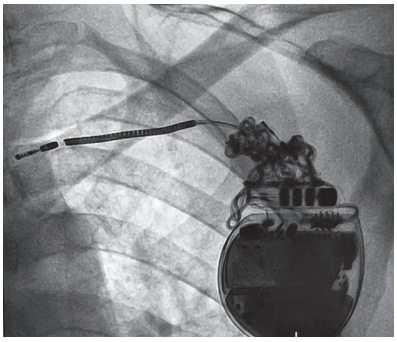
Symptoms are not passing before revision of the device will be performed. In our patient abdominal contraction was the clinical manifestation of Twiddler syndrome, but, differently than in previous reports, it was transient. Most probably the patient continued to ma-

Figure 1. Entrance chest X-ray. Atrial lead dislodged in the superior vena cava

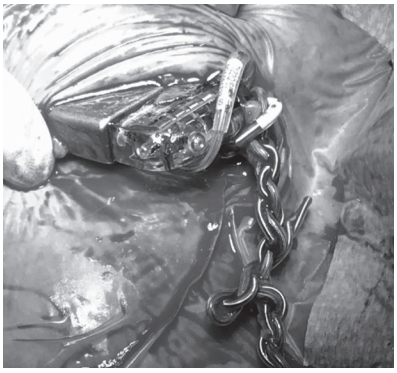
[A] Atrial lead dislodged in the superior vena cava



[B] Ventricular lead retracted in the left brachiocephalic vein and the atrial lead in the pocket encircling the pulse generator



[C] Cluster of twisted electrodes connected to the implantable cardioverter defibrillator



nipulate the device and retracted the lead from the site that stimulated the phrenic nerve; involuntarily he cured the symptom.

Temporariness of the symptom may lead to delay of the recognition and treatment of this potentially dangerous syndrome. In our case no delay happened because Twiddler syndrome was already had been diagnosed before the symptom resolution.

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It's amazing what you can accomplish if you do not care who gets the credit

Harry S. Truman (1884–1972), was the 33rd president of the United States, serving from 1945 to 1953, assumed office following the death of Franklin D. Roosevelt

Capsule

Immunogenicity of Ad26.COV2.S vaccine against SARS-CoV-2 variants in humans

The Ad26.COV2.S vaccine has demonstrated clinical efficacy against symptomatic coronavirus disease-2019 (COVID-19), including against the B.1.351 variant that is partially resistant to neutralizing antibodies. However, the immunogenicity of this vaccine in humans against severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) variants of concern remains unclear. **Alter** and co-authors reported humoral and cellular immune responses from 20 Ad26.COV2.S vaccinated individuals from the COV1001 phase 1/2 clinical trial against the original SARS-CoV-2 strain WA1/2020 as well as against the B.1.1.7, CAL.20C, P.1., and B.1.351 variants of concern. Ad26.COV2.S induced median pseudovirus neutralizing antibody titers that were 5.0- and 3.3-fold lower against the B.1.351 and P.1 variants, respectively, as compared with WA1/2020 on day 71 following vaccination. Median binding antibody titers were 2.9- and 2.7-fold lower against the B.1.351 and P.1 variants, respectively, as compared with WA1/2020. Antibody-dependent cellular phagocytosis, complement deposition, and NK cell activation responses were largely preserved against the B.1.351 variant. CD8 and CD4 T cell responses, including central and effector memory responses, were comparable among the WA1/2020, B.1.1.7, B.1.351, P.1, and CAL.20C variants. These data show that neutralizing antibody responses induced by Ad26.COV2.S were reduced against the B.1.351 and P.1 variants, but functional non-neutralizing antibody responses and T cell responses were largely preserved against SARS-CoV-2 variants. These findings have implications for vaccine protection against SARS-CoV-2 variants of concern.

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Capsule

A public anti-COVID antibody repertoire

Most analyses of the antibody responses induced by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection have focused on antibodies cloned from memory B cells. This approach has led researchers to conclude that neutralizing antibodies (nAbs) primarily target the receptor-binding domain (RBD) of the virus's spike protein. **Voss** et al. took a different approach using proteomic deconvolution of the serum immunoglobulin G antibody repertoire from four COVID-19 convalescent patients. They found that the nAb response was largely directed against epitopes such as the N-terminal domain (NTD), which lie outside the RBD. Several of these nAbs were shared among donors and targeted an NTD epitope that is frequently mutated by variants of concern.

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