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Nocturnal phantom shock cessation with zolpidem

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ABSTRACT

A 77 year old man with a biventricular ICD-pacemaker complained of painful, electric jolts disturbing him nightly from sleep. Extensive work-up including device interrogation revealed no defibrillations or arrhythmia, and he was subsequently diagnosed with phantom shocks (PS). His nightly PS symptoms terminated after starting zolpidem 10 mg each night. To date, literature review reveals fifteen articles reporting 163 phantom shock (PS) cases**.** PS affects 5–9% of ICD recipients. Risk factors include psychiatric disease, atrial fibrillation, NYHA functional status III or greater, prior shock storm, and intraoperative awareness during ICD placement, with defibrillation threshold testing. This report describes a successful PS intervention, and reviews the current knowledge available in the pathophysiology and treatment of PS.

Introduction

An implantable cardioverter defibrillator (ICD) delivers a life-saving discharge, or antitachycardia pacing (ATP) to prevent sudden cardiac arrest, the leading cause of death in the United States. Proven mortality reduction by ICDs has resulted in over a million implantations in the United States.[1] However, complications from ICDs do arise, necessitating hospital visits, evaluation, reoperation, and replacement.[2] One unusual, often frustrating, complication is a phantom shock (PS).

PS is the patient perception of an ICD discharge in the absence of actual device activity, verified on device interrogation. Since the initial description of PS in 1992,[3] PS has been mentioned in 49 publications to date. Symptoms of PS include electric, shock-like sensations of the whole body or chest, chest tightness, and dizziness. PS is often clinically indistinguishable from ICD discharge and prompts emergency department visits, manufacturer interrogations, and even coronary angiography.[4,5] PS often occurs during sleep, although reports of daytime events exist.[6,7] Suffers of PS complain of frustration, confusion, helplessness, vulnerability, and device uncertainty.[8] Patients may seek device replacement, in-home monitoring, or even deactivation.[9] In one extreme case, recurrent ambulatory PS incited a suicide attempt.[10] Unfortunately, to date, there are no reports of successful treatment for PS published.

Case report

A 77 year old man with ischemic cardiomyopathy, well-controlled type-2 diabetes mellitus, obstructive sleep apnea (OSA), anxiety, and chronic bilateral hip pain from osteoarthritis presented to the emergency department (ED) complaining of brief jolts of electricity-like pain across his chest, which awakened him from sleep. These “mini-shock” sensations occurred as often as

Abbreviations: ATP, antitachycardia pacing; DFT, defibrillation threshold; EKG, electrocardiography; PS, phantom shock; ICD, implantable cardioverter defibrillator; NYHA, New York Heart Association; OSA, obstructive sleep apnea; RCT, randomized control trial; SSRI, selective serotonin reuptake inhibitors; VT, ventricular tachycardia.

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three times per night for three months and were similar to a confirmed ICD discharge that he experienced for sustained ventricular tachycardia (VT). His biventricular ICD-pacemaker was implanted for secondary prevention of VT over 3 years prior.

At presentation, vital signs were unremarkable except for hypertension to 155/85 mmHg and a body mass index of 28.7 kg/m^2. Physical examination of other organ systems were noncontributory. His medications included amiodarone, low-dose aspirin, carvedilol, levothyroxine, lactulose, metformin, warfarin, and hydrocodone-acetaminophen. Despite exertional dyspnea when walking up a flight of stairs, he cycled 5–6 miles three times weekly.

ECG showed a ventricular-paced rhythm at 59 beats per minute. Interrogation of his Medtronic (Dublin, Ireland) model DDBC3D ICD-pacemaker revealed no shock discharges. The manufacturer was contacted by telephone, who confirmed that the ICD showed no activity correlating with his diary entries of shock events. Chest roentgenogram demonstrated well-positioned ICD leads and no acute cardiopulmonary processes. At the time of his third and current ICD placement, his defibrillation threshold (DFT) was less than 15 J. Cardiac catheterization from six months prior revealed a left ventricular ejection fraction of 10–15% and diffuse coronary artery disease.

Upon discharge from the ED, he was seen in the cardiology clinic, where his device-related anxiety was reviewed and improved with ongoing counseling. Previously, he had resorted to sleep aids to manage nighttime disturbances, but neither doxylamine succinate (25 mg) nor melatonin (5 mg) improved his nocturnal PS symptoms. He was started on zolpidem 10 mg each night. Upon initiation, his nightly PS symptoms abated. His latest follow up at twelve months revealed that his PS events had ceased altogether.

Discussion

We describe the first case of successful treatment of nocturnal PS. The patient's PS events resolved after initiating a non-benzodiazepine sleep aid. While a placebo effect cannot be excluded from this intervention, this finding warrants further investigation given the limited evidence available for the treatment of this painful, disturbing, and frustrating complication of ICDs. A comprehensive review of publications indexed through PubMed, Google Scholar, and Web of Science revealed no reports of successful treatment of PS.

The existing literature does provide some insight into the epidemiology and risk factors related to PS. Fifteen articles report 163 PS cases (Table 1). PS occurred in approximately 5–9% of ICD-implanted patients. Timing of onset after implantation varied between four months to two years.[11–13] Reported risk factors include major depressive disorder, anxiety disorder, and post-traumatic stress disorder (PTSD). Atrial fibrillation and New York Heart Association (NYHA) functional status were associated with PS in patients whose ICD indication was primary prevention.[12] No studies demonstrated an association of PS with age, gender, left ventricular ejection fraction, prior shock, history of ventricular tachycardia/fibrillation, or indication for ICD therapy. No reports of PS with non-ICD implantable pacemakers or wearable defibrillation devices exist to date.

Investigations of risk factors for PS have focused on psychiatric disturbances. Depression and anxiety affect approximately 25% of patients with ICDs,[14] and psychiatric illness is common among heart failure patients even without ICDs.[15] PS was first described as a manifestation of psychiatric disease or difficulty with device coping.[3,13,16] Populations at risk for ICD-related anxiety include woman and young adults.[17] While no gender difference has been described in PS studies,[7,11,13] one survey of 55 young adults with ICDs found that over 20% experienced PS.[18] High anxiety levels from substance abuse (e.g., cocaine) may predispose ICD patients to PS.[13] A prospective study of 300 consecutive patients before and after ICD implantation found no association between preexisting anxiety or depression and PS.[11] An Israeli study similarly questioned this association, as patients without prior psychiatric illness also developed PS after ICD placement.[19] Psychopharmacologic treatments have not been rigorously studied in PS, but patients with signs of comorbid anxiety, depression, and PTSD merit psychiatric attention.

The development of PTSD following device discharge has been considered, yet prior shock therapy (i.e., number of prior shocks, appropriateness of shock therapy) was not associated with PS in two studies.[12,16] However, shock storm, defined as three or more device discharges within 24 h, is a putative risk factor.[13] Defibrillation or semi-conscious DFT testing may serve as the traumatic substrate for PTSD, and PS could represent memory reactivation.[8,13,18,20] As such, electrophysiologists and anesthesiologists may play an integral role in preventing PS when an ICD is inserted and tested. Adequate anesthesia and amnesia during DFT testing may prevent PTSD.[13] Risk-stratification for DFT testing may be warranted, as one systematic review found that DFT testing provides only modest shock efficacy, and mortality benefit at *de novo* ICD implantation.[21] While ATP and long-detection intervals decrease unnecessary shocks and improve quality of life, program technologies have not been studied in preventing PS.[22,23]

PS commonly occurs during sleep, but the stages of sleep during which PS occurs are unknown.[3,13] Interventions aimed at the improving sleep hygiene or treatment with somniferous agents have not been addressed in the literature. In the present case report, zolpidem demonstrated effectiveness. This medication selectively binds the alpha-1 subunit of the gamma-aminobutyric acid-benzodiazepine receptor. Zolpidem shortens sleep onset latency and prolongs stages 3 & 4 (deep) sleep, thereby decreasing nighttime awakenings.[24] Additionally, dose-dependent clonic inhibition and reduction in startle response may theoretically decease shock perception in PS.[25] The risks of zolpidem must be considered and discussed prior to initiation, including suicidal ideation, exacerbation of depression, somnambulation, amnesia, hallucinations, morning drowsiness, abuse potential, head injury from falls, and oxygen desaturation in patients with OSA.[26–28] Consideration of concomitant sleep disorders (e.g., circadian rhythm disruption, central sleep apnea) is also important prior to the initiation of zolpidem. Co-administration of SSRIs and zolpidem is accepted, and generally considered safe.[29]

Post-shock pain control in addition to adequate anesthesia and amnesia during DFT testing may serve as preventative measures for PS.[13] Screening for PS among ICD patients is warranted, as sufferers are not always forthcoming with PS events.[6] Among ICD recipients with PTSD symptoms, cognitive behavioral therapy may be beneficial if PS is an associated symptom.[30] Nightmares accompanying or triggering PS may improve with sympatholytic agents such as central alpha-1 adrenergic antagonists and alpha-2 adrenergic agonists.[13,31]

Studies of PS have shortcomings which limit the understanding of this process (Table 1). Diagnostic standards for PS are needed, as patients in the reported literature have misinterpreted seizures, syncope, and atrial fibrillation as PS.[7,18,32] Only half of PS studies disclose the prevention indication (i.e., primary or secondary) for ICD therapy.[5–7,10–12,18] Omission of device characteristics (e.g., lead/electrode count, ATP capability), prior DFT testing, tunneling of subcutaneous arrays, and lead placement approach (i.e., transvenous or subcutaneous) further limits understanding of hardware-associated risk factors.[33] Whether ICD components and