An uncommon case of thermal burn from repetitive implantable cardioverter-defibrillator shocks seen on positron emission tomography/computed tomography scan

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Introduction

The use of implantable cardioverter-defibrillators (ICDs) has increased significantly after clinical trials have shown that they reduce mortality in selected patients with heart disease.[1] The advantage of these devices is related to their ability to reduce sudden cardiac death (SCD) by providing antitachycardia pacing and high-energy shocks in malignant ventricular arrhythmias.[2] First- and second-degree burns can be a common side effect of external electrical therapy such as cardioversion or defibrillation. Deep dermal burns may also occur but are less common.[3,4] However, skin and subcutaneous tissue burns or injury due to ICD shocks have never been reported. We present the case of a 22-year-old male patient who received multiple ICD defibrillations resulting in thermal injury from the device.

KEY TEACHING POINTS

- The widespread use of implantable cardioverter-defibrillators (ICDs) is leading to more physicians encountering patients who have had a defibrillation.
- Multiple ICD discharges can lead to subcutaneous tissue injury.
- Subcutaneous tissue injury can be seen on positron emission tomography/computed tomography scans.

Case report

A 22-year-old male patient with a history of myocarditis, ventricular fibrillation, and prior out-of-hospital cardiac arrest status post single-chamber ICD placement was admitted to the hospital for multiple appropriate ICD shocks. His medical history was notable for gene mutations of unknown significance, including *KCNH2*, *PKP2*, and *TTN*. Prior to this admission, he reported being in good health, exercising regularly, and without ICD discharge since placement. He reported nonadherence to sotalol. The patient reported intense exercise preceding the delivery of 15 shocks. The physical examination was notable for tenderness and noticeable swelling at the site of the implantation, but otherwise no fever. Device interrogation revealed monomorphic ventricular tachycardia at 300 beats per minute. Initial troponin was 0.39 ng/mL (0.0–0.9 ng/mL), which peaked at 20.98 ng/mL. An ultrasound of the chest showed no evidence of fluid collection or soft tissue abnormalities. An echocardiogram showed normal left ventricular function with a 55%–60% ejection fraction, normal diastolic function, and mildly dilated right atrium and ventricle. A high-resolution computed tomography (CT) scan of the chest, which was obtained owing to concern for sarcoidosis, did not reveal features of sarcoidosis or other interstitial lung disease. Cardiac magnetic resonance imaging showed nonspecific left ventricular wall thinning and delayed gadolinium enhancement, suggestive of infarct vs previous infectious or inflammatory process such as myocarditis or sarcoidosis. To complete the evaluation of possible cardiac sarcoidosis, a positron emission tomography (PET) CT scan was obtained that showed no increased FDG (F-fluorodeoxyglucose) activity in the myocardium suggestive of active cardiac sarcoidosis. Incidentally, there was increased FDG activity posterior to the ICD along the anterior left pectoralis major muscle related to inflammation from multiple ICD shocks, as shown in Figure 1. Ultimately, his episode of ventricular

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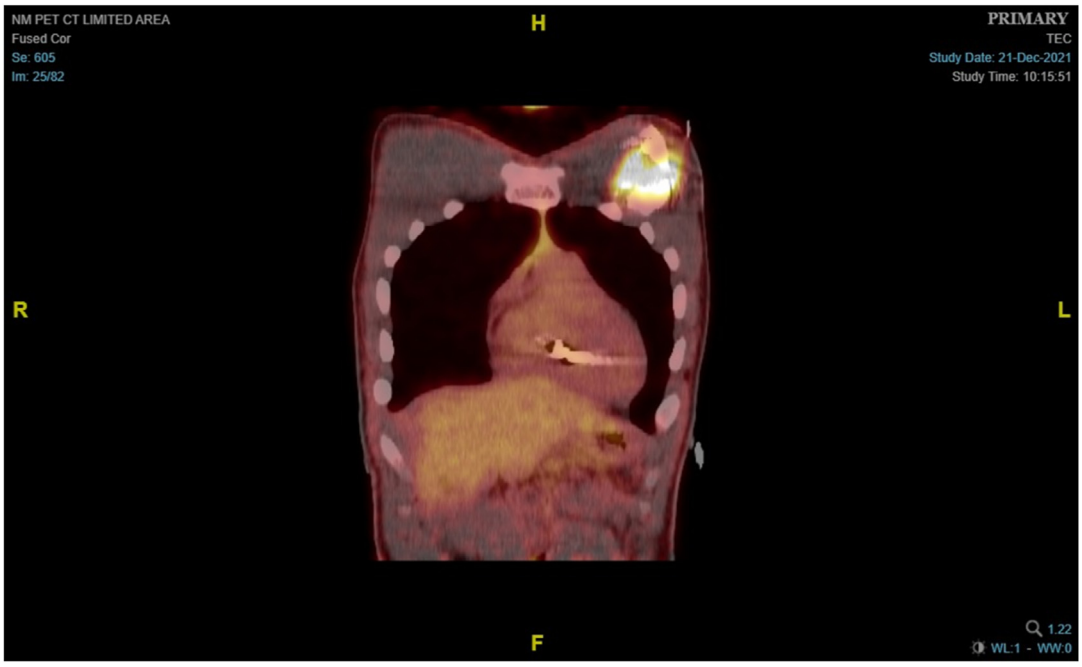

Figure 1 Positron emission tomography/computed tomography scan demonstrating evidence of increased FDG (F-fluorodeoxyglucose) activity posterior to the cardiac conduction device along the anterior left pectoralis major muscle related to inflammation.

tachycardia was thought to be scar mediated. Sotalol was resumed on discharge and the patient was referred for catheter-based ablation.

Discussion

SCD is often the result of malignant arrhythmias such as ventricular fibrillation or monomorphic or polymorphic ventricular tachycardia. The only effective approach to preventing SCD in these situations is high-energy (and, sometimes, repetitive) electrical defibrillation. The benefit of ICDs derives from the ability to provide antitachycardia pacing and high-energy shocks.[1,2] Of patients who have an ICD implanted, approximately one-third will have an appropriate shock while another one-third will experience an inappropriate shock. Whereas the physical side effects of external defibrillation include first-, second-, and, rarely, third-degree burns, the effects of ICD shock on the subcutaneous tissues are typically minimal.[3,4] Soft tissue evaluation after ICD shocks is rarely needed because of the lack of significant impact on treatment. However, it is important to recognize that repetitive ICD shocks can cause inflammation of the chest wall owing to thermal energy released from the device. This consequently may cause pain, swelling, and/or tissue damage, which may mistakenly be confused for infection or bleeding.

There have been some reports of skeletal muscle injury following an ICD shock, which are not necessarily demonstrated by imaging modalities. Specifically, studies have shown that ICD shocks can result in elevations of creatine kinase and cardiac enzymes, indicating the occurrence of muscle damage.[5,6] However, despite these enzyme elevations, imaging studies such as magnetic resonance imaging and CT may not always detect visible muscle injury.

PET CT is a useful tool that can aid in the diagnosis of infectious and inflammatory conditions. Despite the potential benefits of using PET CT to assess for soft tissue damage after an ICD shock, there is a paucity of literature on this topic. This may be owing in part to the fact that soft tissue injuries are often less visible than other types of injuries, making them more difficult to detect and quantify.[7] Furthermore, because soft tissue injuries are often self-limited and may resolve over time without intervention, they may be overlooked or dismissed as minor inconveniences.[8] In addition, PET scans are not routinely performed after an ICD shock owing to their high cost and limited availability, which further limits our understanding of the long-term impact of these shocks on subcutaneous tissue and surrounding structures.

Conclusion

ICDs are a well-established therapy for the prevention of SCD. Given their widespread use, more physicians will encounter patients who have received an ICD shock. It is important to know that multiple discharges from an ICD can lead to thermal injury of the subcutaneous tissues. Though such testing is not typically needed for management, this case highlights rare evidence of subcutaneous tissue injury on PET CT.

Given the potential long-term consequences of soft tissue injury, such as chronic pain or functional impairment, it is important that further research be conducted in this area to better understand the impact of ICD shocks on soft tissue structures and to develop effective management strategies.

A more comprehensive understanding of the soft tissue effects of ICD shocks can help to inform clinical practice and improve patient outcomes.

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