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Inappropriate ICD Shocks caused by T-wave Oversensing due to Acute Alcohol Intoxication

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Abstract

T-wave oversensing can be a serious problem that often results in inappropriate device therapy. We report here a patient with binge alcohol use who received multiple, inappropriate ICD shocks due to T-wave oversensing from repolarization changes induced by acute alcohol intoxication and no other relevant metabolic derangements. Following recovery from his alcohol intoxication a few days later, the T-wave amplitude decreased so the device no longer inappropriately sensed or delivered therapies. This case represents an uncommon, but reversible cause of T-wave oversensing, that should be considered before more aggressive measures are taken to correct the abnormality.

Keywords

implantable defibrillator; alcohol intoxication; inappropriate shocks; T-wave oversensing; anion gap

Case Report

A 65 year-old male with a longstanding history of non-ischemic cardiomyopathy, left bundle branch block and a primary prevention internal cardioverter defibrillator (ICD), presented to the Emergency Room (ER) after receiving 3 ICD shocks while at home watching television. Specifically, he had implanted a Medtronic Concerto-II Biventricular ICD with a Medtronic Sprint Fidelis LFJ6949 RV ICD lead using true bipolar sensing from the tip-to-ring, a Capturefix Novus 5076 atrial lead and a Medtronic Attain OTW 4193 LV lead. On admission to the ER the patient was also complaining of abdominal pain, nausea, vomiting and diarrhea during the past few days, but denied chest pain, shortness of breath or palpitations. His medical history is significant for long-standing alcoholism with recent binge drinking for 2–3 days prior to presentation. In the ER he received 5 further witnessed ICD shocks while he was in sinus rhythm. He had no symptoms prior to, or following the shocks. Initial blood work was pertinent for an anion gap (gap = 22) without acidosis (pH=7.36 on a venous blood gas) and normal lactate, potassium, calcium and magnesium levels. His admission EKG revealed normal sinus rhythm at 88 bpm interchanged with biventricular pacing. T-waves were biphasic after conducted complexes and lower in

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amplitude following biventricular paced complexes (Figure 1). Device interrogation showed the T-wave amplitude in the ventricular channel was significantly higher during sinus rhythm with intrinsic conduction than during biventricular pacing (4 mV versus 1.5mV). In addition, the T-wave amplitude of the conducted beats was significantly higher than at the time of implant or previous interrogations. A relatively higher sinus rate at the time of admission resulted in a significant percentage of intrinsic conduction with T-wave oversensing and inhibition of biventricular pacing. Review of stored electrograms showed inappropriate device therapies due to T-wave oversensing during sinus rhythm (Figure 2). Lowering the ventricular sensitivity, which could have led to failure to detect ventricular arrhythmias, or changing sensing polarity did not help avoid T-wave oversensing. Therapies were deactivated, and to maximize biventricular pacing a shorter AV-delay was programmed. The patient was considered for lead revision (i.e. implantation of a new sensing lead away from the RV apex to maximize R-wave sensing and reduce T-wave oversensing), as this was his second admission for inappropriate ICD therapies.

The patient's hospital course was further complicated by alcoholic transaminitis and pancreatitis from his recent binge drinking. He was ruled out for myocardial infarction by cardiac markers. A transthoracic echocardiogram revealed severely decreased left ventricle ejection fraction ~30% with stage I diastolic dysfunction that was not significantly changed from an echocardiogram obtained 2 years before. The patient was treated with IV hydration and supportive measures for alcohol withdrawal with normalization of his bicarbonate and anion gap. Repeat ICD interrogation was performed on hospital day 3 and the T-wave amplitude of conducted beats was now ~2 mV. Device parameters were reprogrammed to those before admission with 100% biventricular pacing and no further T-wave oversensing on discharge, and at a 6 week follow-up appointment (Figure 3A). The T-wave amplitude of biventricular paced complexes on his EKG prior to discharge was similar to those following biventricular pacing on his admission EKG (Figure 3B). Review of past records showed he had been hospitalized for inappropriate ICD shocks due to T-wave oversensing following binge drinking; however during his previous admission he did have overt ketoacidosis.

Discussion

This case illustrates a rare consequence of alcohol intoxication and its association with ventricular repolarization abnormalities. To the best of our knowledge no previous reports exist describing the effects of acute alcohol intoxication upon ICD function in the absence of secondary metabolic derangements. On the other hand, considerable attention has been given to abnormalities of the electrocardiographic T-wave and the non-specificity of T-wave changes. In a now classic article, Harold Levine listed 67 factors affecting T-wave morphology on the surface EKG which included causes such as acidemia, electrolyte derangements and alcohol abuse¹. It is now known alcohol has a number of pharmacological effects on cardiac conduction, including prolongation of ventricular repolarization and sympathetic stimulation^{2,3}. Various repolarization abnormalities have also been associated with alcohol intake including tall spinous T-waves in the precordial leads, cloven T-waves in leads I and II and dimpled T-waves in lead I⁴. Interestingly, the QRS and T-wave amplitudes on the surface ECG appear to be visibly affected by acute alcohol intoxication in non-alcoholic patients, but not as dramatically in patients with chronic alcoholism⁵. As with the patient in this case, the changes in T-wave amplitudes were not obvious on the surface ECG. The more clear changes we observed in repolarization on local electrogram later reverted back to normal with abstinence from alcohol.

With regards to the mechanism by which acute alcohol intoxication induces local repolarization changes, these may be due to the direct effect of ethanol on cardiac ion channels. Several studies suggest ethanol inhibits the conductance of most voltage-

dependent cardiac ion channels but with varying potencies for different channels⁶. Although the evidence in this area is sparse, it appears significantly higher concentrations of ethanol are required to block cardiac potassium repolarization channels than to inhibit cardiac L-type calcium channels^{6,7,8}. In this regard, an overview of the limited literature suggests the IC₅₀ of ethanol for inhibiting cardiac L-type calcium currents ranges from 300–550 mM^{6,7}, while it is more on the order of 1000–2000 mM for inhibiting cardiac potassium outward repolarization currents^{6,8,9}. Therefore, at clinically relevant ethanol blood concentrations that induce intoxication, 0.1% to 0.3% (22 mM to 66 mM), it is likely cardiac L-type calcium currents are partially inhibited with little effect upon the outward potassium repolarization currents. In this situation the outward or positive component of the cardiac repolarization wave front will be augmented and the local repolarization electrogram will look larger to an implantable defibrillator. It should also be mentioned recent recommendations regarding driving restrictions following an ICD shock are now available¹⁰. With regards to the case presented here where the patient received an inappropriate ICD shock, these guideline recommend that driving may be resumed once the underlying cause of the shock is corrected.

Conclusions

This case highlights an uncommon complication of alcohol intoxication in a patient with an ICD. While most T-wave oversensing is due to metabolic disturbances, alcohol is usually not a cause most physicians consider as an offending agent in the absence of overt metabolic derangements. We suggest alcohol intoxication should be considered as a cause of inappropriate ICD shocks in alcoholic patients, and these patients should be advised against alcohol binging to prevent such incidences.

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Figure 1.

Admission EKG on presentation to the Emergency Room showed normal sinus rhythm with intrinsic atrioventricular conduction as well as biventricular pacing. Note that the T-wave amplitude of the conducted beats are biphasic and of normal amplitude

Figure 2.

Stored electrograms retrieved after delivery of five ICD shocks in the Emergency Room showed an atrial rate of 95–100 bpm (Atip to Aring) with identical ventricular rate and large amplitude repolarization electrograms (RVtip to RVring). Note in the right ventricular electrogram the ICD is sensing both the ventricular depolarization (~5 mV) and repolarization (~4 mV) electrograms and counting them in the tachycardia therapy zones (TS & FS) at alternating cycle lengths from 320–330 ms (depolarization to repolarization) and 250–270 ms (repolarization to depolarization). Due to space limitations only the first inappropriately detected episode is shown with delivery of antitachycardia pacing during

charging (TP) followed by VF redetection (VF Rx 1 Defib) and delivery of a 30 J shock (CD, arrow). The same detection pattern resumes 6 depolarizations after this shock.

Figure 3.

(A) ICD electrograms recorded during device interrogation on the day of discharge during intrinsic rhythm. Note on the RV near-field electrogram (EGM2) the R-waves are now ~6 mV and the T-waves now measure ~2 mV. EGM1 = atrial electrogram; EGM3 = RV far-field electrogram. (B) Surface EKG obtained just prior to hospital discharge now shows biventricular pacing. Note that the T-wave morphology and amplitude appear normal following paced complexes. While the T-waves are no longer biphasic, their amplitude is similar to those following biventricular paced complexes on the admission ECG.