CASE REPORT

Ventricular fibrillation induced by high-output ICD shock: report of cases and review of literature

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Accepted 20 June 2017

SUMMARY

This report highlights the importance of realising that even the modern-day implantable cardioverter defibrillators (ICDs) with R wave synchronised appropriate shocks have a potential proarrhythmic effect. We present two cases of ventricular fibrillation induction resulting from an appropriate ICD shock observed in two different patients at our institution. Such cases have not been reported before. We discuss the possible reasons for our observations and are also submitting a pertinent literature review with our reports.

BACKGROUND

Cardioversion was described clinically for the first time by Lown *et al* in 1962.¹ Cases of ventricular fibrillation (VF) and other arrhythmias following synchronised external cardioversion were reported soon after.^{2,3} The implantable cardioverter defibrillator (ICD) was first used in clinical practice in 1980⁴ and has seen a significant amount of evolution and sophistication since then. Long-term reduction in mortality with use of ICDs in the correct clinical setting has been well validated⁵ and ICDs have been proven effective in preventing sudden cardiac death (SCD).⁶ Therapies delivered by ICDs have also been reported to induce arrhythmias⁷ and these proarrhythmic effects can impact morbidity, patient comfort, and hospital stay related to device therapies. VF emergence following an appropriate ICD shock for ventricular tachycardia (VT) is extremely rare. Ours is the first report of two cases of ventricular flutter or fibrillation (VF) emergence from a clinically appropriate, R wave synchronised, high-energy ICD cardioversion requiring a second high-energy shock to terminate the VF.

CASE PRESENTATION**Case 1**

A 68-year-old woman with a history of ischaemic heart disease and preserved left ventricular ejection fraction (LVEF) underwent ICD implantation 11 years ago due to recurrent symptomatic VT. At generator change a year earlier, it was noticed that the Riata™ lead had externalisation of conductor and that the left subclavian vein was occluded. Thus, the existing left-sided lead was capped and abandoned and a new ICD was implanted in the right pectoral area (St. Jude Medical Fortify Assura VR1257-40Q with a single coil lead 7122Q). Since the reimplant the patient had one occurrence of symptomatic VT treated by an uneventful

ICD shock. Patient presented to the hospital with shortness of breath and dizziness for a brief period while at rest, immediately followed by an ICD discharge. Transthoracic echocardiogram showed an unchanged LVEF of 51%–55% with normal-sized left and right ventricles. Labs on presentation showed a normal troponin I level. B-type natriuretic peptide (BNP) 3740 pg/mL. Haemoglobin 11.6 g/dL from anaemia of chronic disease. Thyroid-stimulating hormone (TSH) was normal. Serum creatinine was baseline at 1.3 mg/dL. Electrolyte panel was unremarkable including a normal serum potassium level. Patient had not been on any antiarrhythmic therapy as an outpatient. Patient had no significant family history of ventricular arrhythmias.

Interrogation of the ICD confirmed that the patient had a sustained monomorphic VT that did not respond to two attempts of burst antitachycardia pacing (ATP), and was successfully terminated with a 36 J shock. However, the interrogation also showed a previous event about 3 months prior to current presentation that the patient had no recollection of. This event of VT at approximately 250 bpm was treated with a shock of 36 J with emergence of VF (figure 1) for which a second shock at 40 J was delivered with successful resumption of a paced rhythm (figure 2). Patient did not seek medical attention for this because she did not feel the shocks. All shocks were delivered with the default waveform parameters (biphasic waveform, 65% tilt, right ventricular (RV) to the defibrillator box (Can), the anode being the RV).

Patient remained asymptomatic during her stay in the hospital with no recurrence of VT. Over the next 2 years of follow-up there were three recurrences of VT that were successfully treated with ICD shock with no resulting postshock arrhythmia. She was also subsequently started on amiodarone in the outpatient setting for suppression of recurrent VT.

Case 2

A 56-year-old man with ischaemic cardiomyopathy and LVEF <20%, status post single vessel coronary artery bypass grafting, mechanical aortic valve replacement for severe aortic regurgitation and a single chamber ICD (Medtronic Protecta VR D334VRM with a dual coil lead 6947 Sprint Quattro Secure) placed approximately 3 years prior to current presentation for primary prevention of SCD, presented with recent worsening shortness of breath, orthopnoea and syncope. Physical

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To cite: Sattar A, Inbar S. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-220098

Figure 1 (Patient 1) VF induced by a 36 J shock. VF, ventricular fibrillation.

examination documented an elevated jugular venous pressure and bilateral lower extremity pitting oedema. Complete blood count and electrolytes were within normal limits. N-terminal proBNP was 11822 pg/mL. Serum TSH was within normal limits. Patient did not require intubation or inotropic support during his stay in the hospital. ICD interrogation on admission documented VT storm (seven episodes of VT in 24 hours) all appropriately treated by ATP or shocks. The patient was admitted for decompensated heart failure and was treated with amiodarone for episodes of atrial fibrillation with rapid ventricular response. Three days later, the patient received two consecutive ICD shocks. His laboratory work-up remained unremarkable. Device interrogation showed appropriate 34.5 J shock (B>AX) for VT at cycle length of 260 ms (figure 3) which resulted in acceleration to ventricular flutter at cycle length of 170–180 ms for which a second 34.5 J shock (AX>B) was delivered with resulting emergence of paced rhythm followed by sinus rhythm (figure 4). Patient was treated with diuretics for 3 more days in the hospital and was eventually discharged with amiodarone as antiarrhythmic therapy. Over the next 19 months of follow-up there were no recurrences of VT.

DISCUSSION

The first report of an ICD shock-induced malignant arrhythmia was in 1990.⁸ This report presented a case where a synchronised shock of unreported energy was triggered by sinus tachycardia which resulted in VT at a rate of 250 bpm. Later, in the era of epicardial lead systems there was a report of low-energy ICD shock inducing VF.⁹ However, this report was done in a generation of ICDs (Medtronic Models 7216A and 7217B) that did not have the ability to deliver R wave synchronised shock in the 'VF' zone. Low-energy shocks have subsequently been reported to result in VF in other reports as well.^{7 10–12} Another case in 1997 reported of a VT that evolved into a self-terminated VT with different QRS morphology and slower rate after an appropriate high-energy ICD defibrillation.¹³ Our cases are the first reported events in a modern ICD system of high-energy R wave synchronised cardioversions which resulted in degeneration of VT into VF.

ICD therapies can be classified as being 'appropriate' or 'inappropriate.' Inappropriate therapies are those that are delivered in response to a rhythm other than rapid ventricular tachyarrhythmia, most commonly due to a malidentification

Figure 2 (Patient 1) Termination of ventricular fibrillation with a 40 J shock.

Figure 3 (Patient 2) 34.5 J shock-induced VF. Upper channel: Can to SVC, middle channel: RV tip to RV ring. SVC, superior vena cava; VF, ventricular flutter.

Figure 4 (Patient 2) Defibrillation of the ventricular flutter with 34.5 J.

of the rhythm and have a well-documented proarrhythmic potential.¹⁴ Appropriate therapies are delivered when ventricular tachyarrhythmias are sensed correctly and are treated according to the programmed parameters. Worsening of arrhythmias can occur even after clinically appropriate ICD therapies. Appropriate ATP therapies have also been shown to induce a number of arrhythmias including accelerated VT, VF, supraventricular tachycardia and bradyarrhythmias.⁷ The mechanism of arrhythmia induction or deterioration after an appropriate synchronised ICD defibrillation remains unclear. Low-energy synchronised shocks (<6 J) have been found to have a 35% likelihood of VF induction or VT acceleration according to a study done on 40 patients with coronary artery disease and left ventricular dysfunction.¹²

As for possible aetiologies of our reported cases, first, the ventricular myocardium has a 'vulnerable period' near the apex of the T wave, and defibrillation during this period of the T wave consistently results in VF. R wave synchronised defibrillation is helpful in avoiding this vulnerable period. Nevertheless, the mechanism of VF induction with R wave synchronised low-energy ICD shock remains unclear. A possible explanation relates to the properties of scarred, diseased and ischaemic myocardium. Such tissue has slowed conduction and increased dispersion of refractoriness. Thus, a shock wave that is synchronised to the local R wave in the sensing electrode in the right ventricle may eventually encounter myocardium which is still in its vulnerable period, hence precipitating VF.¹² Furthermore, cardioversion can modify 'excitable' VT circuit components along with altering intraventricular conduction and creating new areas of conduction block that can destabilise the circuit and result in VF.¹⁵

Second, repolarisation during tachycardia appears more dispersed than during normal heart rates. In an experimental sheep model, VF induction was evaluated under different cycle lengths during synchronised electrical cardioversion for atrial fibrillation.¹⁶ This study showed that the risk of VF was significantly higher when the preceding ventricular cycle length was <300 ms. No VF was observed if preceding cycle length was >300 ms which supports the fact that once all of the myocardium has had time to fully repolarise, VF is not observed.

In addition, induction of VF can also be explained by timing of the shock itself. ICD shock delivery timing relies on locally

Learning points

- ▶ ICDs have a well-documented survival benefit in prevention of sudden cardiac death but therapies delivered by these devices can also be proarrhythmic. Our cases demonstrate a rare observation of degeneration of ventricular tachycardia into ventricular fibrillation (VF) following an appropriate R wave synchronised high-energy shock.
- ▶ Altered myocardial refractoriness due to scar formation and variable myocardial repolarisation are two possible explanations.
- ▶ In patients with severe cardiomyopathy, the dyssynchronous left and right ventricular depolarisations can lead to erroneous shock synchronisation in regard to the global ventricular QRS and can in turn lead to VF which can potentially add to the morbidity related to device therapies.

sensed electrograms for synchronisation. It has been shown that during periods of tachycardia the RV apical sensed depolarisation can vary in timing with respect to the global ventricular depolarisation¹⁷ and depends on the site of origin of the VT. The delay in local R wave sensing can result in delayed shock delivery into the global vulnerable period and thus lead to VF. This dispersion of repolarisation can lower the VF threshold and prolong the vulnerable period, especially in patients with severe cardiomyopathy that can have dyssynchrony of left and right ventricular depolarisations, and hence a higher risk of VF induction.

According to current American College of Cardiology/American Heart Association guidelines, patients with VT should have their underlying condition appropriately treated. It is reasonable to treat patients with recurrent VT with ICD in conjunction with beta blockers and antiarrhythmic drugs. However, percutaneous catheter-guided VT ablation is also reasonable for non-responding patients. Guidelines currently do not mention the appropriate interval for device interrogation for evaluation of subclinical VT; however, it remains prudent that these patients have close follow-up until their clinical condition stabilises.

Contributors Both authors contributed equally to the preparation of this manuscript.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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