Abstract: P595

Early paradoxical steroid induced ventricular tachycardia in cardiac sarcoidosis

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Topic(s):
Implantable Cardioverter Defibrillator (ICD)

Citation:
A 39 year old former athlete was admitted with recurrent shocks from his ICD (implantable cardiac defibrillator). Medication history included sotalol, eplerenone and an angiotensin receptor neprilysin inhibitor. Electrolytes were within normal range and his ECG (electrocardiogram) was unremarkable.

A year prior he had been diagnosed with a dilated cardiomyopathy (left ventricular ejection fraction 40%) after an out of hospital VF (ventricular fibrillation) arrest. A secondary prevention ICD was implanted. PET-CT (Positron emission tomography–computed tomography) was suggestive of cardiac sarcoidosis (CS) and this diagnosis was later confirmed on a second cardiac biopsy.

The patient completed a three day course of intravenous steroids (methylprednisolone 970mg followed by oral prednisolone) two days prior to the ICD therapies. Device interrogation revealed two episodes of VF (ventricular fibrillation) each successfully terminated by defibrillation. The device EMG (electromyography) revealed a slow rhythm with an occasional PVC (premature ventricular contraction) leading to polymorphic VT (ventricular tachycardia) and subsequent VF. Sotalol was changed to bisoprolol and amiodarone. The lower rate of his pacemaker was increased to supress PVCs and prevent a long cycle post PVC. He experienced frequent runs of NSVT (non sustained ventricular tachycardia) and on day seven of steroid use he experienced VT with successful ATP (anti tachycardia pacing). The patient was unable to tolerate a higher base rate of 70bpm due to marked anxiety. His mood improved with clinical psychology input and his symptoms and arrhythmias abated with steroid reduction and commencement of methotrexate. He maintained sinus rhythm and has had no further ICD shocks since discharge.

Problem: Cardiac sarcoidosis is a multisystem infiltrative condition characterised by non-caseating granuloma. It is termed the “great masquerader” due to the diverse range in presenting symptoms and the difficulty in diagnosis. CS carries a higher risk of life-threatening tachyarrhythmia and can manifest as VT without any prior clinical features. There is a high recurrence rate of VT and it is notoriously difficult to control with antiarrhythmic therapies. Inflammation and re-entry due to scar are postulated mechanisms of VT, however there are currently no models to predict risk. Gallium scintigraphy, reduced ejection fraction and a prior VT have been suggested as predictors of VT recurrence. Firstline steroid therapy reduces the progression of myocardial inflammation and fibrosis, however steroid commencement is also associated with VT in the first year. This case illustrates an early paradoxical proarrhythmic effect of steroid use in CS. In this case arrhythmias abated after antiarrhythmic adjustment, pacing at a higher rate and with persistence of steroid use. We advise close monitoring on a cardiac unit for patients with cardiac sarcoidosis commencing steroid therapy.