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Letters to the Editor

Reply to Yalta and Yetkin—Cardiac Sarcoidosis: Indications for Implantable Cardioverter-Defibrillator Therapy

To the Editor:

We thank Drs Yalta and Yetkin for their article on indications for implantable cardioverter-defibrillator (ICD) therapy in patients with cardiac sarcoidosis.[1] They have provided a helpful summary document that nicely reviews the guidelines for ICD therapy in this population and outlines challenges of management in these cases.

The main indication for ICD therapy in our case was the presenting symptoms, and not merely a case of primary prophylaxis.[2] The extent of the disease was impressive, and presumed sarcoidosis masquerading as cardiac tumours was the main reason for presenting the case. This was not the primary reason for device implant. The patient presented with recurrent episodes of palpitations, chest tightness, and profound weakness. This occurred over the course of a couple of weeks. During 2 of these episodes, she felt very unwell, with near syncope. We did not document any sustained arrhythmia during hospital monitoring. However, her symptoms sounded very concerning for hemodynamically significant ventricular arrhythmia. We did discuss doing an electrophysiological study among our group, but we felt that the presenting symptoms warranted a prophylactic ICD even if the electrophysiological study were to be negative. We felt that she met the guideline for near-syncope with arrhythmic origin.[3] Admittedly, the severity of these episodes is not well captured in the original case report.

Following a tapering dose of prednisone, a repeat positron emission tomography (PET)-computed tomography scan demonstrated increased fluorodeoxyglucose uptake in the cardiac masses and hilar nodes. The masses appeared to be slightly smaller on follow-up magnetic resonance imaging, but they were active again on PET. Given that we did not have definitive pathology on the prior lymph node biopsy, a cardiac biopsy was performed of the inferior mass using intracardiac echo guidance. Unfortunately, this procedure demonstrated nonspecific inflammatory changes. The patient was restarted on high-dose prednisone, this time with the addition of methotrexate and pending follow-up PET. Unfortunately, she had presented recently with an appropriate shock from her device (Fig. 1). Of note, her device is programmed to deliver anti-tachycardia pacing (ATP) during charging, but it did not do so in this case, due to Smart Mode.[4] A preceding shorter episode had failed to terminate with ATP, so further ATP attempts were disabled for this zone. This first episode stopped spontaneously without a shock. The episode shown in Figure 1 is the second episode that day. This second episode required a shock for termination. The only finding on electrocardiogram were isolated premature ventricular contractions. The arrhythmia appears to initiate following a premature ventricular contraction. No QT prolongation and no pause-dependent arrhythmia occurred. The patient was started on amiodarone, and short-term follow-up has been arranged.

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Ethics Statement

This letter adhered to health research ethics guidelines.

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Disclosures

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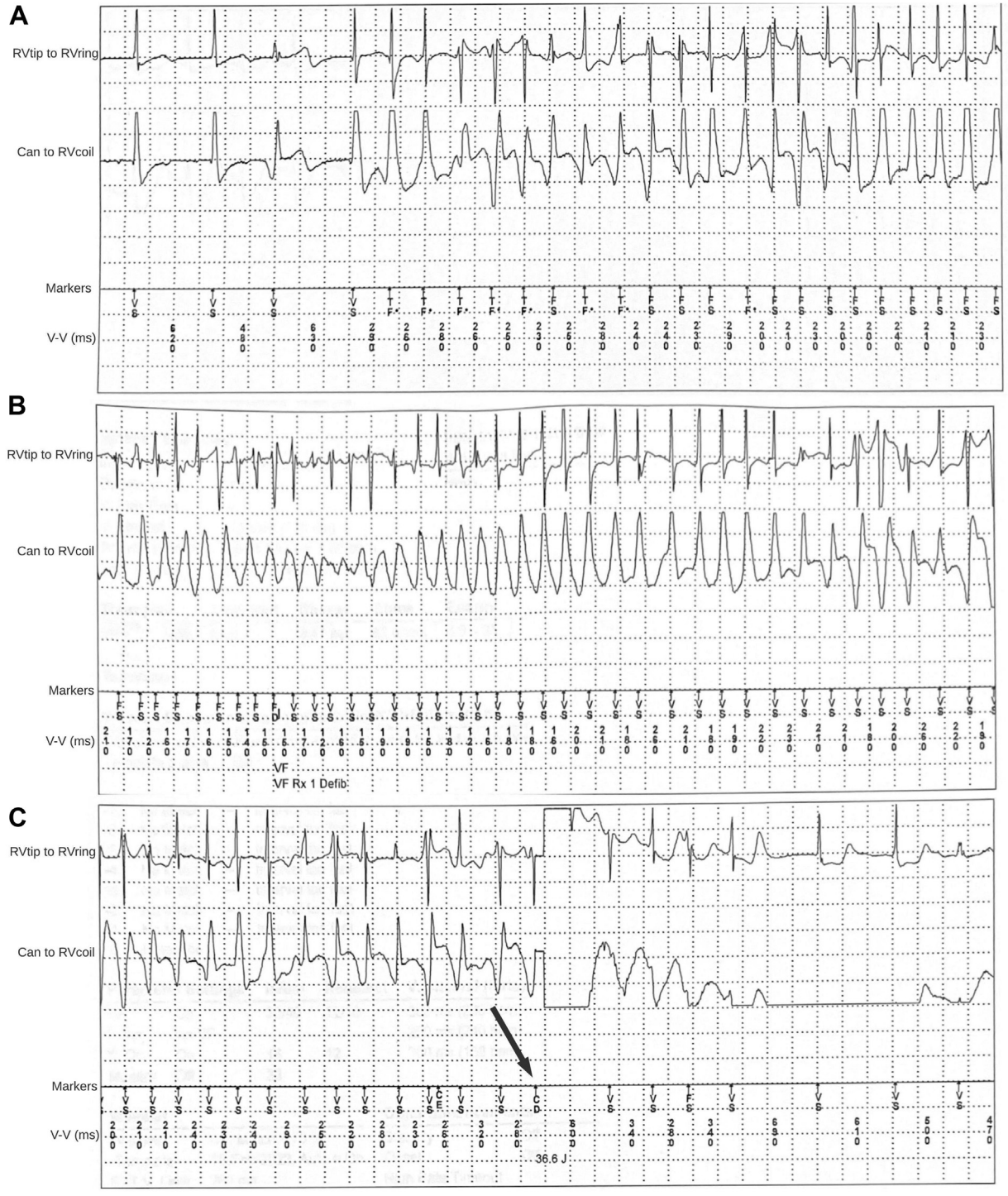

Figure 1. Episode of polymorphic ventricular tachycardia—(**A**) initiation; (**B**) ongoing; (**C**) termination following shock (**arrow**).