CASE OF THE MONTH

A Fluttering Heart: A Storm is Brewing

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CASE HISTORY

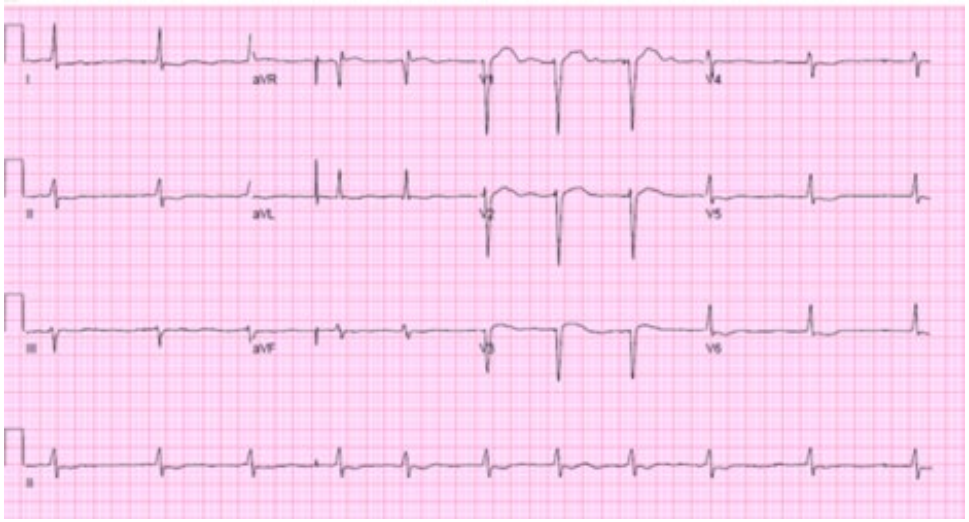
A 79-year-old man with an implantable cardiac defibrillator (ICD) experienced three ICD discharges on the same day. The ICD had been implanted because of a history of ischemic cardiomyopathy with a previous, silent myocardial infarction (MI) and depressed ejection fraction (EF). He also had history of paroxysmal atrial fibrillation and hypertension. Prior to the ICD discharges he felt "uneasy," but did not faint. He decided to go to a local emergency department. The patient denied chest pain, dyspnea, or near syncope. He gave a history of a remote appropriate ICD discharge for sustained ventricular tachycardia (VT) and was currently taking amiodarone.

Other pertinent past medical history included repair of an inguinal hernia. His family history was negative for premature CAD. His home medications included:

- Amiodarone 200 mg twice daily
- Carvedilol 12.5 mg twice daily
- Digoxin 125.0 mcg daily
- Rivaroxaban 20.0 mg po daily
- Simvastatin 10.0 mg daily
- Tamsulosin 0.4 mg daily
- Finasteride 5.0 mg daily

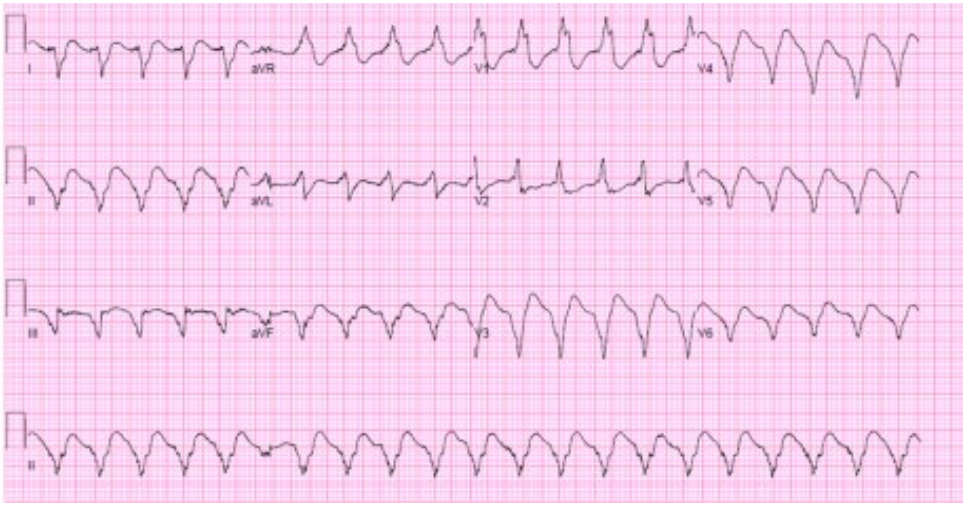
Physical examination at the local hospital showed temperature 97.9° F, respiratory rate 20 breaths per minute, blood pressure 154/86, heart rate 63 beats per minute, and O_2 saturation 95% on room air. He was oriented to person, place, and time and in no distress. Overall, the examination was unremarkable except for an ICD noted in the left sub-clavicular region. An electrocardiogram was also performed (Figure 1).

Figure 1. Electrocardiogram from the local hospital.



An echocardiogram done was interpreted as showing an EF in the mid 30s and apical akinesis. Soon after, the patient felt palpitations and "impending doom." A repeat electrocardiogram (ECG) was done (Figure 2). The patient's blood pressure at time of ECG was 92/63 mm Hg.

Figure 2. Repeat electrocardiogram



QUESTION 1: Where is the likely source of VT?

a) Left ventricular outflow tract (LVOT)
b) Right ventricular outflow tract (RVOT)
c) LV apex
d) Basal inferior LV wall

ANSWER

C: LV apex

Explanation: The vector of the QRS is going away from Lead 1 and V6, and towards AVR and V1, making the LV apex a likely source.

CASE CONTINUED

The patient was admitted to the critical care unit. Because his condition was stable, his ICD was deactivated to prevent continuous discharges. He was initially started on an amiodarone and lidocaine drip, and digoxin was discontinued. The patient was taken to the catheterization laboratory for coronary angiography (Video 1) to evaluate for benefit of percutaneous coronary intervention (PCI) revascularization.

Video 1a. Coronary angiography
https://youtu.be/XzO9WIuWGso

Video 1b. Coronary angiography
https://youtu.be/Nj_n5JmOSuQ

QUESTION 2: What would you do next? The patient is still in VT.

a) Proceed with PCI of left anterior descending artery
b) Cardiovert patient
c) Abort PCI and continue medical treatment
d) Abort PCI and transfer patient to a higher level of care facility

ANSWER

D: Abort PCI and transfer patient to a higher level of care facility

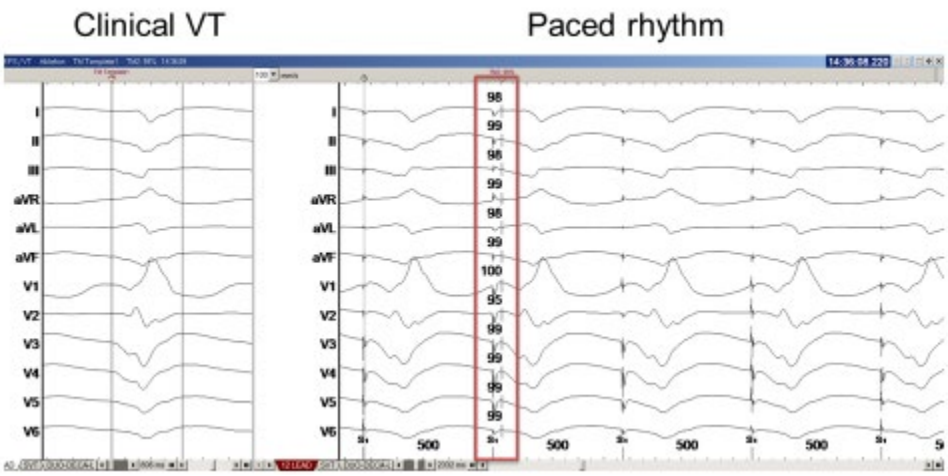
Explanation: Video 1b depicts an aneurysm of the mid left anterior descending artery with a speckle of calcium, followed by total occlusion of the vessel, consistent with a chronic process. This indicated that acute ischemia was unlikely to be the source of the VT storm. The patient had already failed a medical trial with lidocaine and amiodarone; thus, transfer to a tertiary care facility was the most appropriate decision.

CASE CONTINUED

The patient was transferred to our institution while in VT and on amiodarone and lidocaine drip. His blood pressure remained low but stable. A review of outside transthoracic electrocardiogram (TTE) showed an apical aneurysm with preserved wall motion on non-infarcted segments. This finding together with the VT vector on the ECG suggested an apical origin. Consequently, the patient was taken directly to the electrophysiology lab for an ablation.

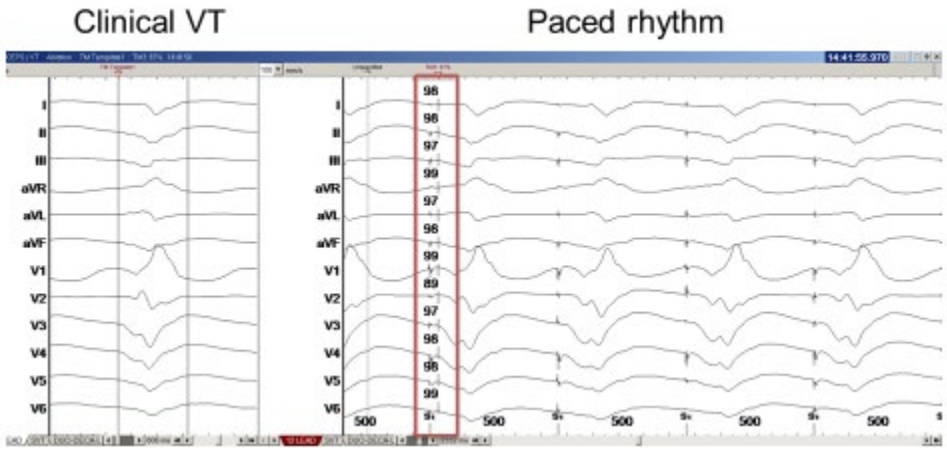
Figure 3 shows a 12-lead ECG of the clinical VT next to the 12 leads observed during endocardial pacing within the LV aneurysm. The numbers highlighted by the red box indicate the percent match of the paced QRS with the clinical QRS.

Figure 3. Endocardial pacemap from inferolateral apex



Similarly, Figure 4 shows the clinical and paced match obtained with epicardial pacing. The numbers highlighted by the red box indicate the percent match of the paced QRS with the clinical QRS.

Figure 4. Epicardial pacemap opposite to endocardial side: 97% match



An ablation was performed in the critical isthmus of the reentrant circuit. This led to elimination of the clinical VT, which had until then been incessant.

Video 2 shows a VT propagation map. Local activation times were collected during VT using a multipolar catheter (PentRay, Biosense Webster). Propagation initiates at the lateral apex, spreading rapidly through the lateral and anterior walls and reaching the septum, where propagation slowly proceeds towards the apex, completing a reentrant circuit.

Video 2. Ventricular tachycardia propagation map.
https://youtu.be/Oz0MWL03-Xw

A repeat TTE with contrast was performed after the ablation to better define the aneurysm and the function of the remaining myocardium (Video 3). Note the discrete apical dyskinetic region and the preserved wall motion in the noninfarcted segments.

Video 3a. Transthoracic electrocardiogram without contrast. This parasternal long-axis view does not capture the left ventricular aneurysm.
https://youtu.be/tuRH5COuHEc

Video 3b. Transthoracic electrocardiogram with contrast. Note the aneurysm visible in this apical four-chamber view.
https://youtu.be/f4sljUueePY

The patient remained in sinus rhythm with stable hemodynamics. He was discharged 3 days later and has remained stable over 6 months of follow-up.

DISCUSSION

Our case demonstrates the devastating sequelae of nonrevascularized MI. In the era of coronary reperfusion therapy, the incidence of LV aneurysm after MI is decreasing.[1] However, despite advances in the treatment of acute MI, some patients still develop LV aneurysm. In our case, the patient had a remote "silent" anterior MI that was not revascularized and subsequently developed a LV aneurysm. Complications of LV aneurysm include systemic thromboembolism, heart failure, angina, and ventricular arrhythmias.

Our patient presented with electrical storm in the form of incessant VT and repeated ICD shocks. Medical management of incessant VT is limited and frequently ineffective. In our case, intravenous amiodarone and lidocaine was tried and was unsuccessful. It is not uncommon that escalation of antiarrhythmic drugs fails to control VT. In the setting of recurrent VT despite already being on antiarrhythmic therapy, VT ablation is superior to escalation of antiarrhythmic drugs.[2] The 2017 AHA/ACC /HRS guideline for the management of ventricular arrhythmias and prevention of SCD recommends catheter ablation in patients with prior MI and recurrent episodes of symptomatic sustained VT who have failed or are intolerant of antiarrhythmic drugs.[3]

Patients who present to the hospital with recurrent VT frequently already have an ICD. This is because ICD has been shown to be more effective than antiarrhythmic drugs alone in preventing SCD.[4] Unfortunately, these patients can experience repeated appropriate ICD shocks. Besides causing pain and anxiety, ICD shocks are associated with an increased mortality risk even after adjustment for Seattle Heart Failure Model predicted mortality.[5] Fortunately VT ablation can reduce ICD shock burden. There is also evidence that patients who have an ICD shock and are treated with VT ablation have a lower risk of death and heart failure hospitalization.[5]

TAKE-HOME POINTS

Recurrent ventricular arrhythmias can be challenging to treat. Fortunately, we have techniques available to use beyond medical therapy. Catheter ablation of ventricular tachycardia is effective and patients presenting with electrical storm should be transferred to high-volume centers capable of VT ablation.

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