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Author
Sohrabian, Sahar

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CLINICAL VIGNETTE

A Red Herring or a Lighthouse? A Case of Ventricular Tachycardia in a Middle-Aged Man

Sahar Sohrabian, MD

A 49-year-old man with a prior history of smoking, hypertension and dyslipidemia presented with intermittent chest symptoms for the past three months. The patient had noted chest tightness and diaphoresis during exertion and rest associated with occasional palpitations. Both his parents were diagnosed with coronary artery disease in their late sixties. He denied drug use, syncope, or prior heart disease.

Physical exam was unremarkable including a normal cardiac examination with regular heart rate and no murmurs or gallops. Electrocardiogram (ECG) demonstrated sinus bradycardia at a rate of 56 beats-per-minute (BPM) with normal axis, intervals, QRS, and ST-T wave patterns. A transthoracic echocardiogram, treadmill stress echocardiogram, and Holter monitor were obtained. Holter monitor demonstrated sinus rhythm with infrequent premature ventricular contractions (PVC). Echocardiogram was unremarkable. During treadmill stress testing, he exercised for 10 minutes and achieved 11.7 METs without symptoms. Two-millimeter horizontal ST depressions were observed in the inferior leads of the ECG at 9 minutes of exercise. Wide complex tachycardia consistent with monomorphic ventricular tachycardia (VT) was observed at peak exercise that resolved after 40 seconds in the recovery stage (Figure 1). His prior reported symptoms were not reproducible during stress testing. He denied palpitations, shortness of breath, or chest pain during the entirety of the test including during VT. Aspirin 81 mg daily and metoprolol succinate 25 mg daily were prescribed and he was referred for coronary angiography given the marked abnormality in the ST-segments coupled with non-sustained VT.

Coronary angiography revealed no epicardial coronary artery disease. Cardiac MRI showed left and right ventricles with normal size and systolic function. Delayed ventricular enhancement was absent thereby excluding myocardial scar or infiltrative disease. A diagnosis of idiopathic VT was established, and the patient was maintained on medical therapy after a discussion of risks and benefits of invasive catheter ablation for treatment. Beta-blocker intolerance was observed after 6 weeks of use. Therefore, an extended day Holter monitor was obtained to document arrhythmias with and without beta-blocker therapy and no VT was observed with or without therapy. Subsequently, beta-blocker was withdrawn due to side-effects. The patient did not have recurrence of chest symptoms while continuing to exercise normally off medical therapy.

Discussion

This case highlights the challenging diagnostic decision making throughout the clinical spectrum of VT. Specifically, this patient was observed to have a single incident of asymptomatic sustained (>30 seconds) monomorphic VT during a stress-test for intermediate risk exertional chest pain. All other diagnostic testing, including Holter monitoring, was unrevealing. Ventricular tachyarrhythmias, unlike atrial tachyarrhythmias, may lead to life-threatening conditions, hence the sense of urgency is heightened for the clinician. However, certain "benign" forms of VT portend a good prognosis and do not carry an increased risk of SCD therefore obviating the need for specialized medications or life-saving therapies such as implantable cardioverter-defibrillators. Differentiating between "malignant" and "benign" forms of VT may be difficult and requires a thorough diagnostic evaluation with consideration for advanced cardiac imaging such as cardiac MRI or positron-emission tomography (PET).[1] Differentiating between polymorphic and monomorphic VT varieties is the initial step to determining risk. Sustained polymorphic VT (on the spectrum of ventricular fibrillation) is a life-threatening disorder usually associated with ion-channel dysfunction (acquired or congenital) or ischemia in the absence of fibrotic heart disease. A detailed evaluation is necessary to exclude monomorphic VT related to underlying structural, ischemic, or genetic heart disease or systemic disorder (ie hyperthyroid state). Cardiac evaluations for arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D), inflammatory cardiomyopathies (CMY) (i.e. cardiac sarcoidosis), dilated CMY, and infectious CMY may be necessary. Basic laboratory evaluation may suggest a systemic illness.

Idiopathic monomorphic VT is a benign condition associated with normal cardiac structure and function in the absence of fibrotic heart disease. Symptoms may include chest pain, palpitations, exercise intolerance, lightheadedness, and fatigue. However, some patients may be asymptomatic. Most cases of idiopathic VT are associated with sympathetic tone as a result of triggered activity secondary to delayed after-depolarizations (DADs) mediated by cyclic adenosine monophosphate and intra-cellular calcium loading. A variety of anti-arrhythmic medical therapies targeting the biochemical basis of triggered activity may be instituted for idiopathic VT including beta-blockers and ion-specific calcium, sodium and potassium

channel blockers.[2] The goals of medical therapy are to lessen VT burden and reduce symptoms although success and compliance rates may not be ideal. Prognosis is generally good with low risk of sudden death or malignant ventricular arrhythmias with some exceptions.[3,4] Catheter ablation of idiopathic VT is an alternative to medical therapy with potential for cure and a generally low risk procedural profile.[4]

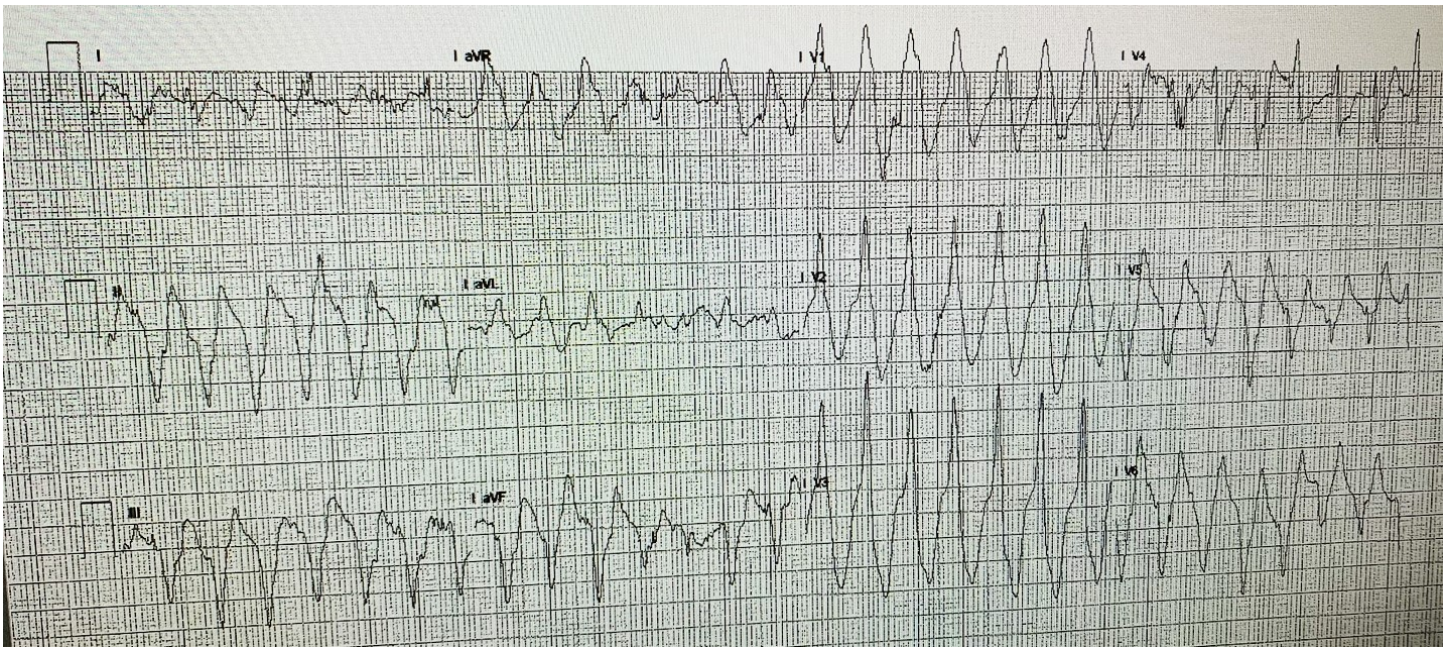

Figure 1: Electrocardiogram of ventricular tachycardia during treadmill stress test.

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