A Case of Repetitive Monomorphic Ventricular Tachycardia

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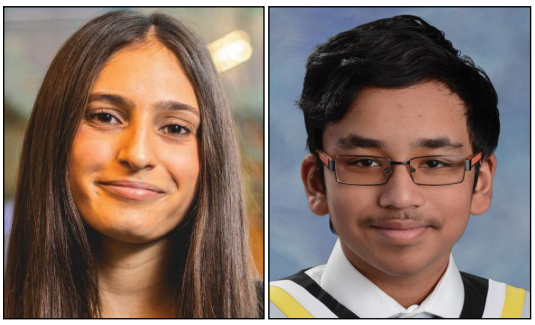

Marwaha Majji

A 56-year-old female with a past medical history of hypothyroidism presents to her family physician with episodes of palpitations and dizziness. She describes these episodes as racing heartbeats and lightheadedness with exercise. She was referred by her family doctor for an outpatient treadmill stress test and a 24-hour ambulatory cardiac monitor. Her resting electrocardiogram (EKG) shows sinus bradycardia at 54 bpm without any significant ST or T wave abnormalities and a normal QT corrected interval (QTc). Her cardiac monitor shows predominantly normal sinus rhythm with 4% premature ventricular contraction (PVC) burden and occasional ventricular couplets and triplets.

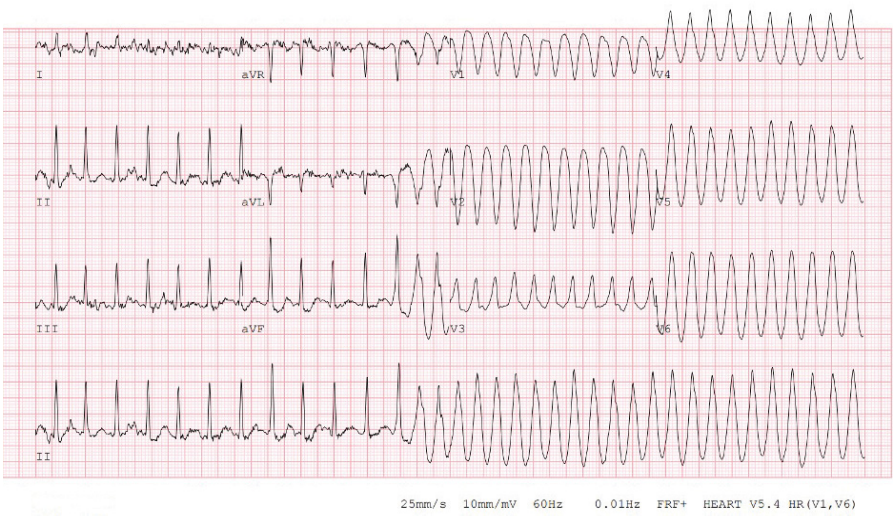

Fig. 1. EKG showing onset of monomorphic ventricular tachycardia.

Her past medical history includes hypothyroidism and exercise-induced asthma. She does not smoke, and only drinks socially and on rare occasions. Her mother had diabetes, hypertension, and dyslipidemia, and had undergone coronary artery bypass grafting in her 50s.

The patient's daily medications include levothyroxine, 88 mcg; vitamin D3 capsule, 2,000 units; fish oil (omega 3) capsule, 1,000 mg; and a multivitamin tablet.

On physical examination, she is afebrile with normal vital signs. She is resting comfortably without any focal findings. Labs reveal a normal thyroid stimulating hormone (TSH) level of 0.90 and normal high-sensitivity troponin levels. The comprehensive metabolic panel and complete blood count are unremarkable. Her lipid profile is normal with a total cholesterol of 147, LDL of 83, HDL of 49, and triglyceride of 74. Her electrocardiogram is normal.

The patient is asymptomatic prior to her treadmill stress test. The patient walks on the treadmill for the first 12 minutes without symptoms. Her EKG does not demonstrate any ST/T wave abnormalities. She has evidence of PVCs and ventricular couplets on EKG during exercise. During the early recovery period, she becomes symptomatic with dizziness

and develops monomorphic ventricular tachycardia (VT) for 30 beats on the cardiac monitor[1] (see Fig. 1 on page 9). She recovers immediately in the supine position, and VT terminates after coughing. After her treadmill stress test, she is admitted to the cardiac telemetry unit for observation and further evaluation of her VT.

During this hospitalization, she undergoes extensive cardiac evaluation including an echocardiogram, coronary computed tomography (CT) angiogram, and cardiac MRI (magnetic resonance imaging). Her echocardiogram reveals a mildly dilated left ventricle with mildly reduced LV function (LVEF 45% to 50%) and mild mitral regurgitation. Her CT angiogram shows a calcium score of 0, consistent with normal coronaries free of stenosis (see Figs. 2-4).

Her cardiac MRI reveals an LV dilation with mildly reduced LVEF of 48% and normal wall motion (see Fig. 5). There is no abnormal myocardial enhancement to suggest prior ischemic damage. However, there is mild basal myocardial septal enhancement (see Fig. 6 on page 12), which could be consistent with a dilated cardiomyopathy or myocarditis. However, there is no evidence of myocarditis on edema-weighted sequences.

DISCUSSION

Ventricular Tachycardia with Structurally Normal Heart

Ventricular arrhythmias are broadly classified based on their duration and morphology. Sustained ventricular arrhythmias are defined as lasting more than 30 seconds in duration, which typically causes hemodynamic collapse and sudden cardiac arrest. Non-sustained ventricular arrhythmias can last less than 30 seconds. Ventricular arrhythmias are also classified as monomorphic (similar QRS morphology) or polymorphic (variable QRS morphology) based on their appearance.

Typically, a malignant ventricular arrhythmia occurs in the presence of structural heart disease such as coronary artery disease with prior myocardial infarction, dilated cardiomyopathy, hypertrophic cardiomyopathy, or arrhythmogenic right ventricular cardiomyopathy. In this situation, ventricular arrhythmia carries a high risk of sudden cardiac death.

Ventricular arrhythmias can also occur with structurally normal hearts. In some situations, these could be associated with underlying electrical channelopathies such as the Brugada syndrome or congenital long QT syndrome. In these patients, ventricular arrhythmias carry a high risk of sudden cardiac death.

Polymorphic VT in the setting of a structurally normal heart can occur with prolonged QT interval and familial conditions such as catecholaminergic polymorphic VT. Ventricular fibrillation in the absence of structural heart disease can be related to metabolic derangements or ischemia. These syndromes are associated with an increased risk of sudden cardiac death.

A monomorphic VT that occurs in the setting of a structurally normal heart carries a benign prognosis. This kind of ventricular tachycardia is commonly grouped together with other idiopathic VT syndromes. Ventricular tachycardia that is not associated with structural organic heart disease can arise in several locations, including the right ventricular outflow tract, tricuspid annulus, right ventricle, left ventricle, left ventricular outflow tract, inferoapical septum, and aortic cusps.

Based on the origin and mechanism, idiopathic VT is broadly classified into three groups:

1. Repetitive monomorphic VT (RMVT), due to triggered activity from the right ventricular outflow tract (RVOT) or left ventricular outflow tract (LVOT).
2. Paroxysmal sustained VT, arising from the right ventricle.
3. Idiopathic left ventricular tachycardia, which is a reentry VT that arises from inferoapical region or mid-septum.

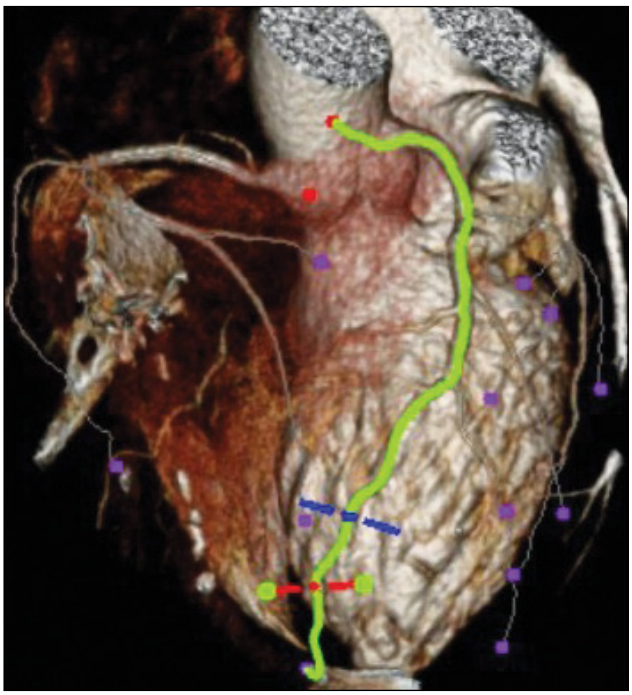

Fig. 2. Cardiac CTA showing three-dimensional volume reconstruction, with LAD coronary artery outlined.

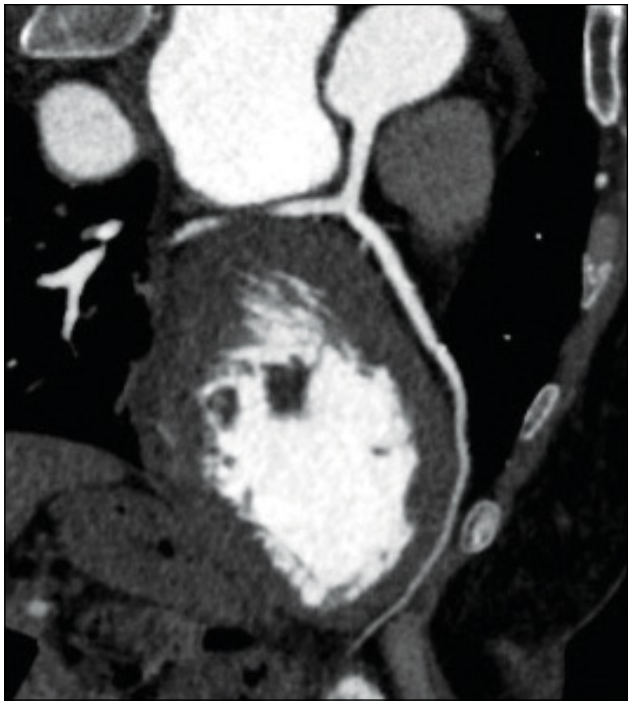

Fig. 3. Cardiac CT of the normal left main and LAD coronary arteries.

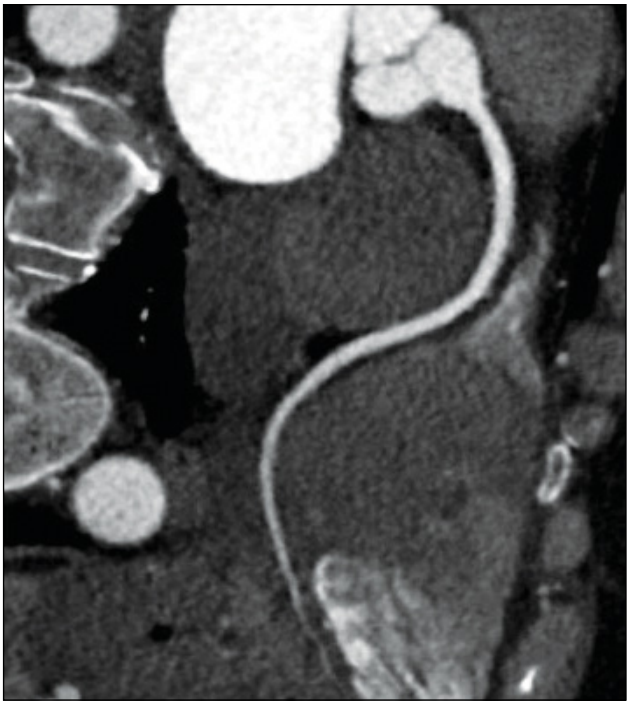

Fig. 4. Cardiac CT showing a normal right coronary artery.

The history may be suggestive. Most arrhythmias are non-sustained (3-15 beats long), although some patients demonstrate sustained episodes. Bursts of non-sustained VT are typically provoked by emotional stress or exercise and may be seen during the warm-down period after exercise, as was noted in our patient.

Arrhythmias may also follow the circadian pattern, with prominent episodes between 7:00-11:00 a.m. and 4:00-8:00 p.m., correlating with periods of high sympathetic activity. Previous studies have demonstrated an association between arrhythmias and hormonal triggers; thus, episodes may be noted during the premenstrual phase, gestational or perimenopausal period, and with use of birth control pills.

To reach this diagnosis means excluding other entities, and thus patients often undergo testing using a variety of diagnostic studies including resting EKG, echocardiogram, exercise stress testing, coronary angiography, and cardiovascular MRI.

Regarding testing, the EKG is diagnostic during VT and shows a left bundle branch block morphology with an inferiorly directed axis. Typically, in LVOT VT, the EKG shows right bundle branch block morphology with an inferiorly directed axis.

Treatment of RMVT includes medications such as beta-blockers, calcium channel blockers, and antiarrhythmic medications. There has also been increasing use of successful radiofrequency ablation[2] in severely symptomatic patients, as well as patients that are refractory to or do not desire long-term drug therapy. Even if the site of origin is not endocardial, epicardial ablation can help successfully treat the arrhythmia.

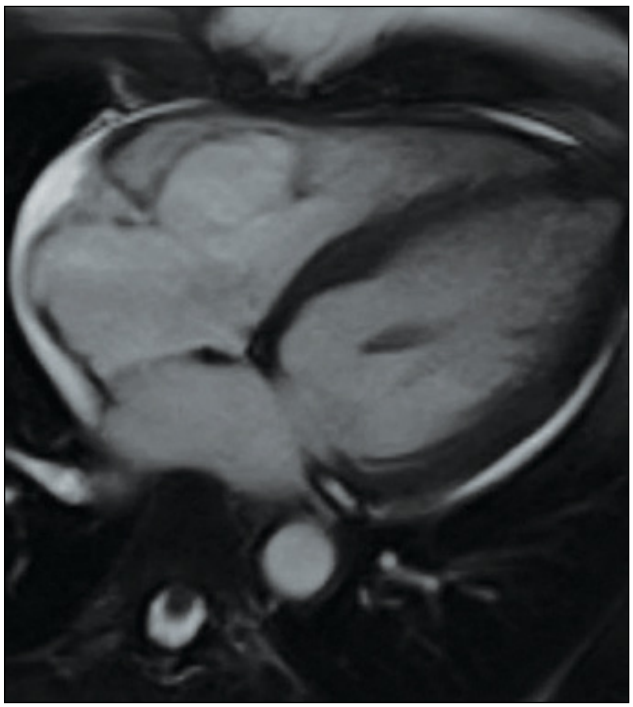

Fig. 5. Cardiac MRI — four-chamber view.

CONCLUSION

Our patient had repetitive monomorphic ventricular tachycardia originating from the right ventricular outflow tract, better known as RVOT VT. This typically occurs in young to middle-aged patients without structural heart disease and is known to have 2:1 female preponderance. It may also be seen in competitive athletes. These patients typically present with symptoms of palpitations and lightheadedness or syncope.

The working diagnosis was thus ventricular tachycardia originating from the RVOT, causing mild cardiomyopathy. The patient was seen by a cardiac electrophysiology consultant and started on oral diltiazem 180 mg daily. She was discharged home for an outpatient electrophysiology study, during which she underwent VT ablation. Her symptoms improved significantly after the ablation. Her follow-up echocardiogram showed normal left ventricular ejection fraction.[3]

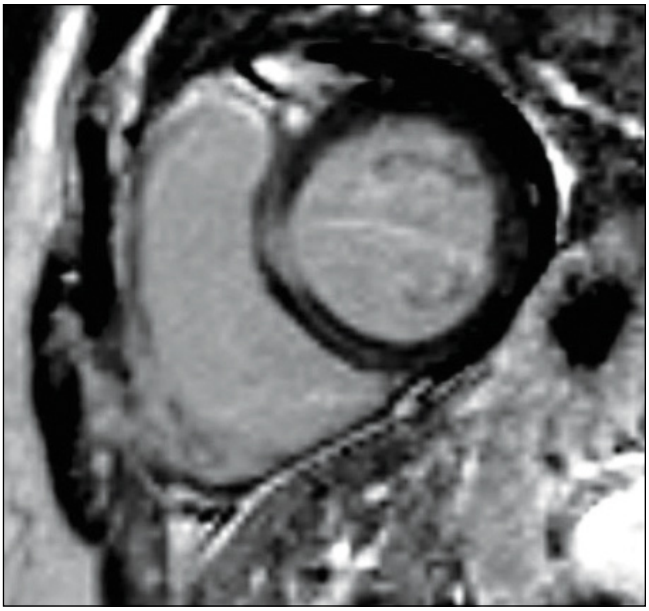

Fig. 6. Cardiac MRI showing delayed enhancement with a mid-myocardial stripe in the basal septum.

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