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Exercise-Induced Sustained Ventricular Tachycardia without Structural Heart Disease: A Case Report

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Female, 51-year-old
Final Diagnosis: Ventricular tachycardia
Symptoms: Chest pain • shortness of breath
Medication: —
Clinical Procedure: Treadmill stress echocardiogram
Specialty: Cardiology

Objective: Unusual clinical course

Background: Exercise-induced ventricular tachycardia (VT) has been widely reported in patients with preexisting structural heart disease or underlying ischemia and is attributed to reentry tachycardia and abnormal automaticity. However, studies regarding exercise-induced VT in individuals without evident structural heart disease are still limited.

Case Report: A 51-year-old woman came to our practice for a treadmill stress echocardiogram. The patient experienced only mild chest discomfort and was otherwise asymptomatic. Cardiovascular risk factors were significant only for obesity and positive family history of coronary artery disease in the mother. During the exercise stress test, the patient developed wide complex VT with multiple capture beats accompanied by nausea and dizziness, which lasted approximately 2 minutes before resolving spontaneously. Subsequent evaluation with magnetic resonance imaging, transthoracic echocardiography, and coronary angiography revealed an absence of apparent structural heart disease.

Conclusions: Exercise-induced VT in the absence of structural heart disease, although rare, can pose a life-threatening event and requires different considerations for management. The benefits of currently available therapeutic options have yet to be elucidated for this subset of patients. Thus, we assert that there is a need for further investigation on the approach of exercise-induced VT in patients without structural heart disease.

MeSH Keywords: Echocardiography, Stress • Heart Diseases • Tachycardia, Ventricular

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Background

Ventricular tachycardia (VT) is a potentially lethal arrhythmia with a regular, wide QRS complex and a rate greater than 100 beats per minute (bpm). It is considered sustained if it lasts 30 sec or more or causes hemodynamic collapse in less than 30 sec [1].VT often occurs in the setting of underlying heart disease, such as coronary heart disease, dilated cardiomyopathy, and hypertrophic cardiomyopathy. Therefore, patients presenting with VT require a thorough cardiac workup. Diagnostic evaluation often begins with a resting electrocardiogram (EKG) and also includes echocardiography to evaluate left and right ventricular performance, exercise stress testing, coronary angiography, and cardiac magnetic resonance imaging (MRI) [2]. VT in patients with structural heart disease can be induced by exercise and emotional stress, or can occur at rest. In particular, exercise-induced VT in these patients has been described in multiple reports. However, reports of exercise-induced VT in patients without apparent structural heart disease remain limited. Here we report a case of sustained VT in a 51-year-old woman with no underlying heart disease that was induced incidentally by an exercise stress test.

Case Report

An asymptomatic 51-year-old female patient was evaluated at our office at her request due to concerns related to shortness of breath and episodic chest pain both at rest and during physical activity for the previous 6 months. However, history of palpitations, dizziness, syncope, and claudication was denied. Her past medical history revealed iron deficiency anemia (IDA) treated with ferrous sulfate, chronic gastritis treated with proton pump inhibitors, medication-induced constipation, and hypertension treated with amlodipine 5 mg once daily. The patient denied a history of coronary artery disease, diabetes mellitus, and thyroid disease. She also denied use of tobacco, alcohol, and recreational drugs.

The physical examination revealed a resting blood pressure (BP) of 140/80 mmHg, heart rate (HR) of 78 bpm, and respiratory rate of 16 breaths per min. The patient was obese with a body mass index of 32.8 kg/m^2. The cardiovascular evaluation reported a normal heart rhythm with unique S1 and S2, no murmurs, gallop, or rubs. The patient's peripheral pulses were equal and symmetrical. The lung fields were clear and the abdominal evaluation was unremarkable. Laboratory studies ordered included a complete blood count, coagulation profile, complete metabolic panel, uric acid, NT-proBNP, C-reactive protein (CRP), lipid panel, HbA1c, troponin I, thyroid panel, iron studies, and urinalysis. The patient was found to have mild microcytic hypochromic anemia with an Hb level of 11 g/dL, MCV of 71.9 fL, MCH of 22 pg, and MCHC of 30 g/dL. Her red cell width (RCW) distribution was elevated, iron level was low at 26 ug/dL (range, 50–170 ug/dL), iron saturation was 8% (range, 15–50%), and ferritin was 9 ng/mL (range, 5–204 ng/mL), consistent with a diagnosis of IDA. The IDA could potentially explain her initial exertional dyspnea, because after 6 months of rigorous treatment with ferrous sulfate, the patient reported a significant improvement of her chronic IDA-related symptoms (exertional dyspnea). CRP and ESR levels were also elevated, with values of 21 mg/L and 51 mm/h, respectively. Although the patient had no history of diabetes, she was found to be prediabetic, with an HbA1c level of 5.9%. She was also found to have mild dyslipidemia with a total cholesterol level of 214 mg/dL and LDL of 142.4 mg/dL. Other laboratory test results were unremarkable. A treadmill stress echocardiogram was ordered to evaluate her complaints of progressive exertional dyspnea (New York Heart Association class II) and atypical chest pain in relation with her long-standing history of hypertension, obesity, and prediabetic state.

The patient was instructed to exercise on a treadmill according to the Bruce protocol until exhaustion or development of symptoms. Her baseline EKG showed a normal sinus rhythm with no ST segment changes and a HR of 74 bpm (Figure 1). The patient exercised for 6 min and 30 sec, reaching a peak HR of 149 bpm, BP of 162/80 mmHg, and 7 metabolic equivalents, when she suddenly developed nausea, dyspnea, and dizziness. Thus, the test was halted. However, the patient denied chest pain and did not lose consciousness. The EKG at that time revealed multiple episodes of wide complex VT with multiple capture beats at a rate of 230 bpm (Figure 2). The EKG findings lasted for approximately 2 min before resolving spontaneously, accompanied by the disappearance of symptoms. The recovery EKG revealed sinus tachycardia (Figure 3) with a rate of 110 bpm, which gradually returned to baseline.

Further studies were performed to investigate the presence of any structural heart disease in the patient that might have led to the occurrence of exercise-induced VT. Laboratory studies revealed no significant results other than those previously stated. The initial chest X-ray revealed a normal cardiomediastinal silhouette and failed to demonstrate any pathology. A complete 2D transthoracic echocardiogram with color and special Doppler also revealed no significant abnormalities. The patient had no left atrial dilatation (volume index, 18 mL/m^2), no left ventricular hypertrophy, normal end-diastolic diameter (volume index, 49 mL/m^2), no valve abnormality, absent pulmonary hypertension, and normal right atrial pressure (mean, 3 mmHg), normal size and normal wall motion of the left and right ventricles with a good ejection fraction (65%). A subsequent cardiac MRI also showed a normal size of all 4 chambers with normal systolic function. Left ventricle regional function demonstrated normal myocardial motion in all segments. Delayed gadolinium contrast-enhanced images demonstrated no areas of late gadolinium enhancement to suggest

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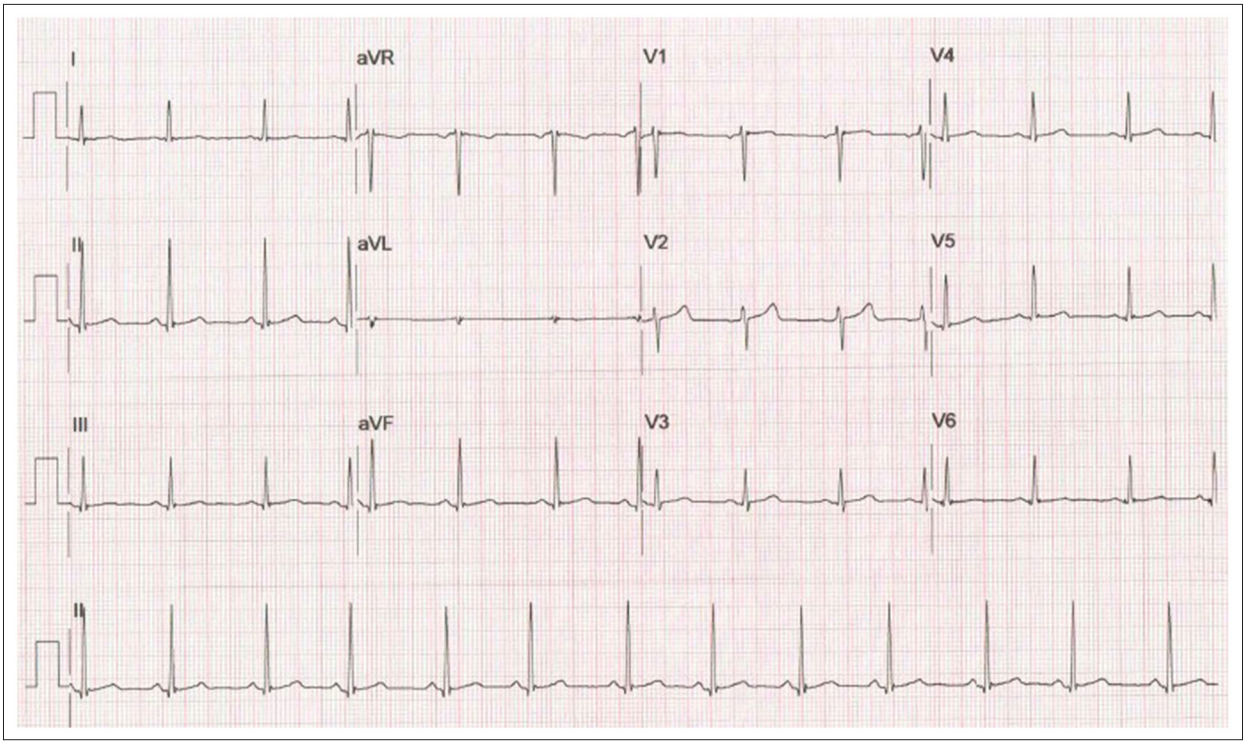

Figure 1. Baseline EKG.

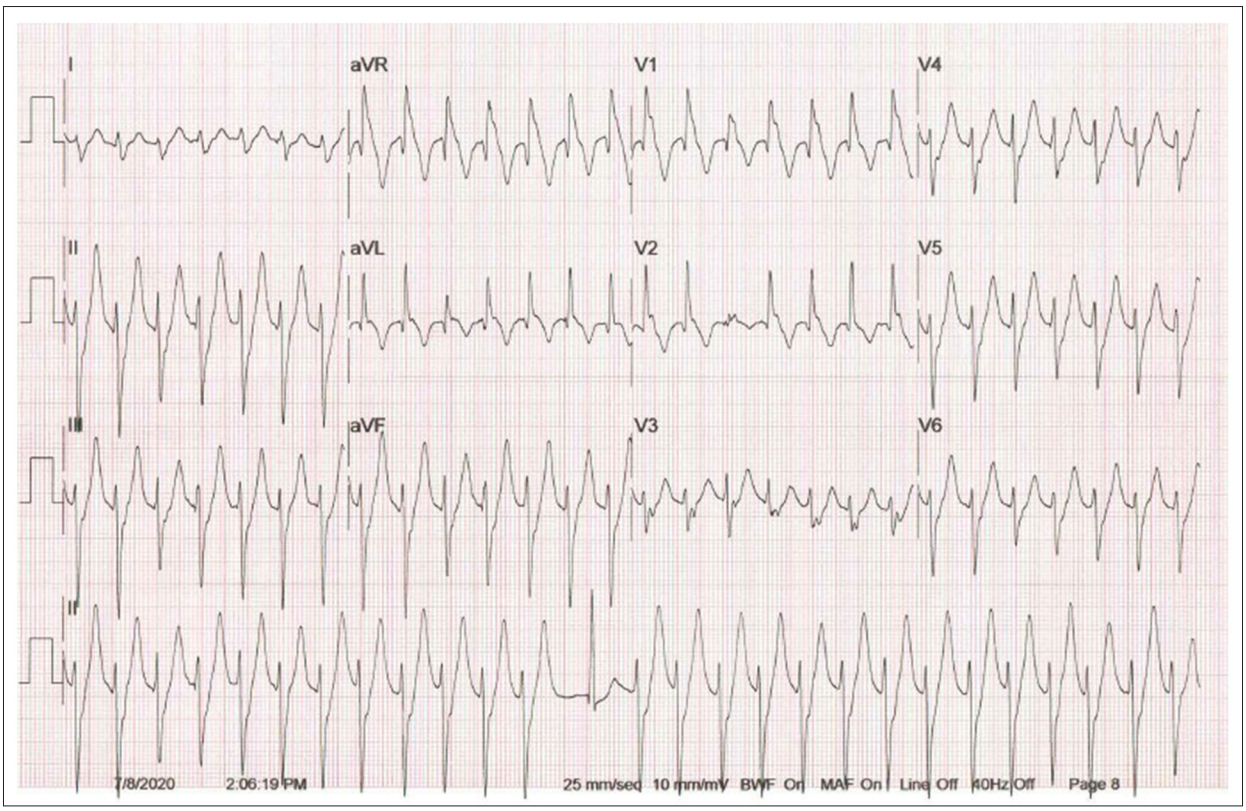

Figure 2. Monomorphic ventricular tachycardia during exercise.

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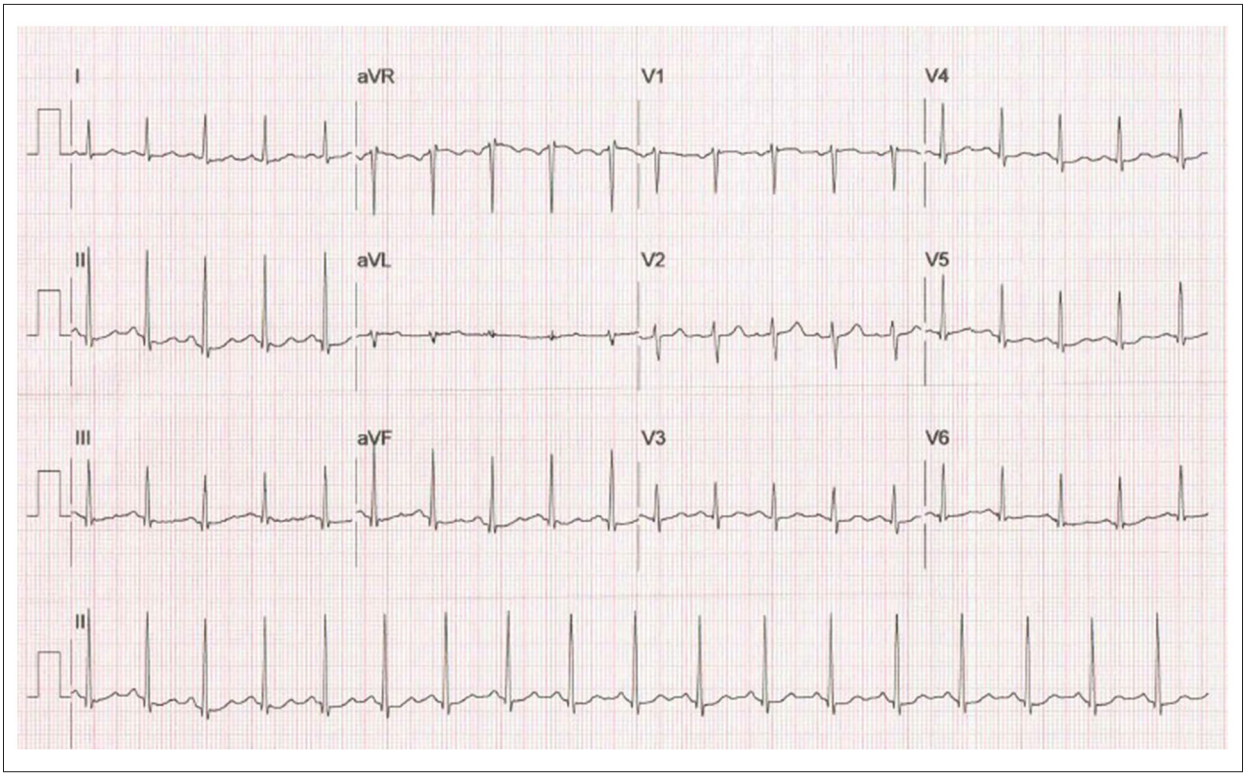

Figure 3. Recovery EKG.

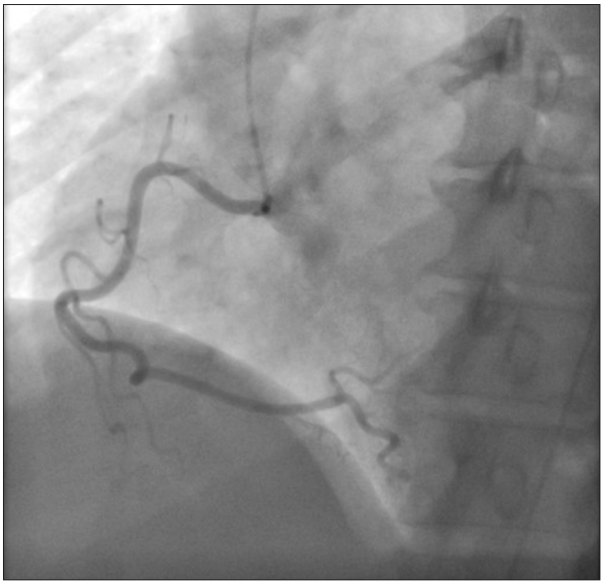

Figure 4. Coronary angiography of the right coronary artery (RCA) showing patent artery with no significant obstruction.

infarction, inflammation, or focal fibrosis, thereby indicating no apparent structural heart disease. Coronary angiography was performed, revealing no significant obstruction as all arteries were patent, as seen in Figures 4, 5, excluding ischemic cardiomyopathy in this patient.

Discussion

Exercise-induced VT is a prevalent event in patients with structural heart disease or underlying ischemia. Although its exact prevalence is yet unknown, coronary artery disease is the most common cause of sudden cardiac death resulting from fatal ventricular arrhythmias [3]. In apparently healthy individuals, exercise-induced VT appears in approximately 1.1% of those who undergo a cardiac stress test [4]. Several studies have shown that a frequent cause of exercise-induced VT in patients without structural heart disease is catecholaminergic polymorphic VT, which is characterized by a 180° beat-to-beat rotation of ectopic QRS complexes, termed "bidirectional" [5]. The absence of this hallmark feature together with the age of the patient in the present case makes this etiology less probable. Structural heart disease, such as ischemia, hypertension, or dilated cardiomyopathy, increases the risk of VT, which can be in the form of reentry tachycardia, abnormal automaticity, or triggered activity [6]. Several cases of polymorphic and monomorphic VT in patients with preexisting, overt cardiac disease have been reported [7,8]. However, the lack of medical literature regarding exercise-induced VT in healthy individuals without underlying cardiac disease created our special interest in reporting this case. Therefore, the absence of structural heart disease in our patient as shown by the coronary angiography (Figures 4, 5) and cardiac MRI raises suspicion of a nonstructural etiology and, therefore, the need to reevaluate the standard classification of exercise-induced VTs.

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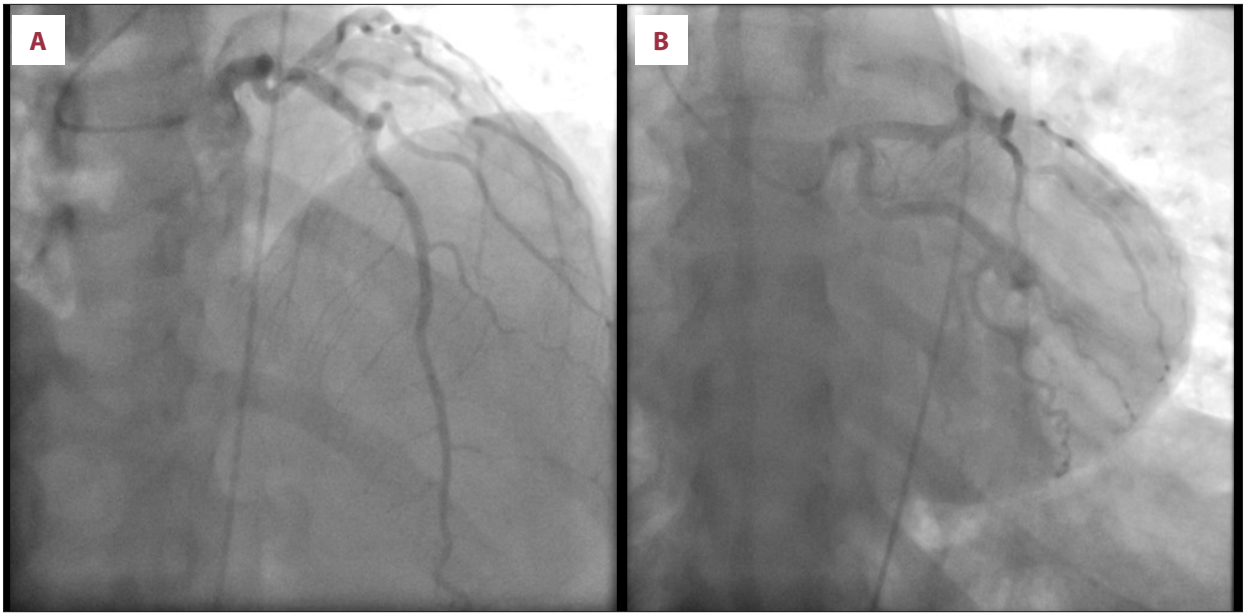

Figures 5. (**A, B**) Coronary angiography of the left coronary circulation showing no significant obstruction on the left coronary artery (LCA), left circumflex artery (LCx) and left anterior descending artery (LAD).

The presence of IDA in this patient might raise concern since anemia has been more frequently found as a comorbidity in heart failure and is associated with poorer outcomes. The conditions share a reciprocal relationship. The prevalence of anemia is increased among heart failure patients, ranging from around 30% in stable patients to 50% in hospitalized patients, compared to 10% in the general population [9]. The pathogenesis is complex and has not been completely elucidated; however, it is suggested to involve the neurohormonal system, inflammatory cytokines, and renal changes [9]. At the same time, severe untreated anemia (4–6 g/dL) causes various physiological changes mimicking heart failure, such as sodium and water retention and an unfavorable cardiorenal response, including reduced renal blood flow and glomerular filtration rate [10]. In addition, there is evidence of neurohormonal activation even without any organic heart disease present [10]. However, studies in our patient demonstrated mild anemia at 11 g/dL that would be unlikely to cause significant heart failure without any underlying cardiovascular disease. Furthermore, studies revealed the patient had a normal ejection fraction, absent left ventricular hypertrophy, normal size of all cardiac chambers, and normal wall motion, excluding suspicion of structural heart disease or heart failure associated with anemia.

The pathophysiologic mechanism of VT has been classified according to its duration, morphology, symptoms, and presence of cardiac disease. A review of the literature on classifications of exercise-induced VT revealed a lack of concordance between the current classifications and the presentation of this case. Therefore, we propose an alternative classification to highlight the importance of this specific type of exercise-induced VT. In this suggested alternative classification, there are 2 categories, depending on whether the patient has structural heart disease or not. Further, in patients without structural heart disease, there are 2 types of VT that can be seen, monomorphic or polymorphic (Figure 6). The most common types of monomorphic VT in patients without structural heart disease are those originating from the septal aspect of the right ventricular outflow tract [11]. It is sustained and presents as syncope, palpitations, and/or dizziness during exercise and is seen almost exclusively in young to middle-aged patients. Other common exercise-induced VTs include those originating in the left ventricular outflow tract and idiopathic left VT, and other forms of VT, including paroxysmal VT.

Right ventricular outflow tract VTs have a characteristic EKG appearance, which includes the presence of the left bundle branch block and/or inferior axis [12]. Our patient had a normal baseline EKG without any evidence of a left bundle branch block or inferior axis. One of the risk factors that has been shown to be associated with an increased incidence of ectopic ventricular arrhythmias during exercise in asymptomatic healthy individuals is obesity [13], which was present in this patient.

Because prognosis and management depend on the type of arrhythmia, several initial distinctions should be made. Most cases of VT are typically diagnosed during cardiac monitoring or an exercise test performed for other reasons. Once the arrhythmia has been identified, the evaluation process should include a complete medical history with pertinent family history and a physical examination. Classification of the type of

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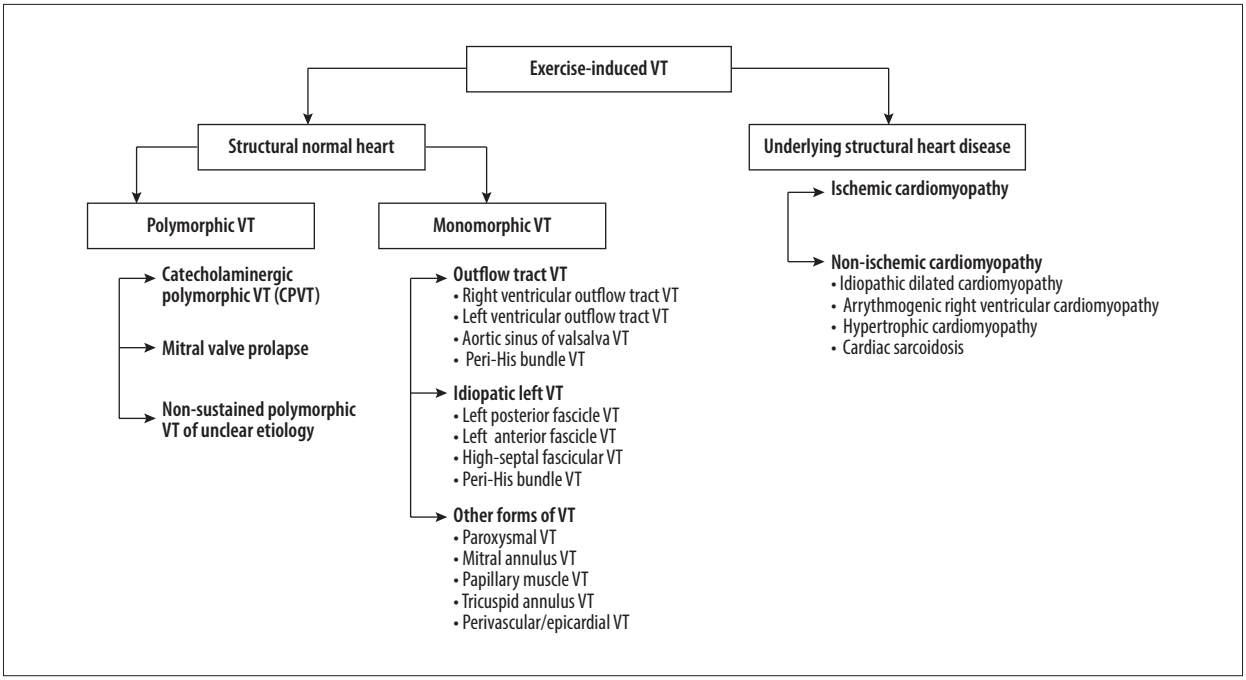

Figure 6. Classification of exercise-induced ventricular tachycardia. Adapted from Tang et al. [10] and Michowitz et al. [14].

arrhythmia should be based on the duration (sustained or nonsustained) and morphology (monomorphic VT and polymorphic VT).

The presence of symptoms can guide the next steps. In asymptomatic patients, a 12-lead electrocardiogram, transthoracic echocardiography, and exercise stress test can help determine the presence or absence of any associated structural heart disease. The latter can worsen the prognosis of any type of VT. The following steps in the risk stratification process should focus on identifying the patients who would benefit from an exhaustive evaluation to exclude the presence of unsuspected structural heart disease, such as past myocardial infarction, arrhythmogenic ventricular cardiomyopathy (ARVC), or dilated and hypertrophic cardiomyopathy [14,15]. The presence of any of these conditions dramatically changes the prognosis because sudden cardiac death is common in these settings if VT is present.

Ambulatory event recorders are valuable regardless of the presence or absence of structural heart disease. Electrophysiologic studies can also be performed to localize a provoked sustained arrhythmia. A cardiac CT/MRI is warranted in patients who are symptomatic or when the VT is sustained or polymorphic. The probability of structural heart disease is higher in these cases and cardiac imaging should aid in excluding structural heart disease and/or ARVC [16].

In 2011, Dadkhah et al. reported a similar case of exercise-induced VT in which an asymptomatic 70-year-old woman developed multiple but shorter bouts of VT, which were categorized as nonsustained [3]. Further evaluation revealed 90% blockage of the patient's proximal left anterior descending coronary artery, and the authors determined the development of exercise-induced nonsustained VT warranted further workup of underlying coronary artery disease. In contrast, the coronary angiogram in our patient demonstrated only mild nonobstructive coronary artery disease. Therefore, although we agree on the importance of a coronary angiogram to exclude coronary artery disease in the evaluation of exercise-induced VT, we also believe it is important to note that exercise-induced VT may develop in patients without underlying coronary artery disease or ischemia.

Medical therapy is based on the presence of symptoms and structural heart disease. If none of these conditions are present and ARVC has been excluded, no specific medical therapy is needed. Beta-blockers are first-line therapy for symptomatic patients. Calcium channel blockers like diltiazem or verapamil can also be used if beta-blocker therapy is contraindicated. Other therapeutic options such as catheter ablation can be performed in cases of persistent arrhythmia, despite pharmacological therapy or in otherwise young and healthy patients where ablative therapy may be preferable over the chronic administration of antiarrhythmic drugs [17].

Overall, prognosis in patients with exercise-induced VT and no structural heart disease is usually favorable. Several studies have demonstrated no significant increase in mortality, latent coronary artery disease, incidence of angina pectoris, myocardial infarction, or sudden cardiac death [14].

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Conclusions

Exercise-induced VTs, particularly sustained, are uncommon in patients without structural heart disease. However, sustained VT is an independent risk factor for increased morbidity and mortality, regardless of concomitant cardiovascular risk factors. This unusual case of exercise-induced VT in a patient without structural heart disease presented a challenge requiring different management considerations. These considerations included a thorough review of the patient's history, a well-equipped testing facility, and awareness of potential adverse events during exercise stress testing.

Therapeutic measures for these events include pharmacological and interventional management; although, long-term benefits of these approaches have only been evaluated in patients with structural heart disease. Their role in exercise-induced VT without underlying heart disease are yet to be elucidated. Our particular case of exercise-induced VT, which was resolved without intervention, highlights the need for further investigation of how to approach the screening, treatment, follow-up, and prognosis in this subset of patients with VT.

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