

CASE REPORT

Cardiac Arrest and Exercise-Induced Polymorphic Ventricular Tachycardia: An Elusive Diagnosis

Nestor Rodrigues de Oliveira,¹ William Santos de Oliveira,² Adalberto Atsushi Porto,³ Fabio Mastrocola,⁴ Ana Eloisa Novaes,⁵ Roberto Moreno Mendonça,⁶ Júlio César Vieira de Sousa⁷

Hospital Universitário Onofre Lopes - Huol-UFRN - EBSERH, Natal, RN - Brazil.

Introduction

Polymorphic ventricular tachycardia (VT) is defined as a rapid rhythm with continuously changing ventricular activation.¹ In the absence of QT interval prolongation, its evaluation is a challenging activity with a series of differential diagnoses. The present study reports on a case of a young man who had an out-of-hospital cardiac arrest and who later presented an episode of exercise-induced polymorphic VT. A rational investigation is discussed based on the patient's clinical findings and baseline ECG.

Case Report

A 30-year-old Brazilian man received medical care at our ambulatory services to evaluate an aborted sudden death two months earlier while running at an airport. Return of spontaneous circulation was achieved using an automated external defibrillator (AED), and he has remained asymptomatic ever since. He had no past medical record, aside from using testosterone esters for competitive sports during one year at the age of 18. He denied smoking tobacco or using other drugs. He always lived in Natal, RN, Brazil, and reported no contact with triatomine bugs. Physical examination was unremarkable, with a BMI of 24.2 kg/m² and BP of 110/80 mm Hg.

A routine laboratory panel showed a hemoglobin of 11.4 g/dL, a leukocyte count of 6490/mm³, a platelet count of 188.000/mm³, a creatinine of 0.6 mg/dL, a blood urea nitrogen of 8.8 mmol/L, a sodium level of 144 mEq/L, and a potassium level of 4.5 mEq/L. His Chagas disease

serologies were negative. His fasting blood glucose, thyroid function, and lipid profile were within the reference range. His baseline ECG (Figure 1) showed a sinus rhythm (HR of 60 bpm) and a discreet ST-segment elevation in leads V3-V5, with no other alterations. A transthoracic echocardiogram revealed ventricular apical hypokinesis and was otherwise normal, depicting a left ventricular ejection fraction (LVEF) of 69%. Afterwards, an exercise stress test elicited an episode of asymptomatic polymorphic VT upon the first minute of the recovery phase (Figure 2A). The patient reached 89% of his predicted maximum HR, and the rest of the stress test was normal. A 24-hr ambulatory ECG monitoring recorded eight monomorphic ventricular premature beats (VPBs).

A coronary CT scan (Figure 2B) revealed a hypodense plaque in the left anterior descending (LAD) coronary artery resulting in a stenosis of 70%. In fact, cardiac catheterization (Figure 2C) showed that the LAD harbored an unstable plaque with 90% stenosis, and catheter angioplasty with the placement of a drug-eluting stent was undertaken uneventfully. At follow-up, the patient was screened for causes of premature coronary artery disease (CAD). Tests for antinuclear antibodies, rheumatoid factor, syphilis, hyperhomocysteinemia, and antiphospholipid syndrome were unrevealing. Therefore, therapy with aspirin, clopidogrel, atorvastatin, enalapril, and propranolol was instituted. Six months later the patient presented no symptoms, and a new exercise test showed normal results.

Discussion

Polymorphic VT evaluation in individuals with normal QT interval depends on underlying structural heart disease, inherited arrhythmia syndromes, early

Keywords

Death Sudden Cardiac; Tachycardia, Ventricular; Exercise Induced; Diagnosis.

Mailing Address: William de Oliveira

Av. Nilo Peçanha, 620. Postal Code: 59012-300, Petrópolis, Natal, RN - Brazil.
E-mail: william.santos0197@gmail.com

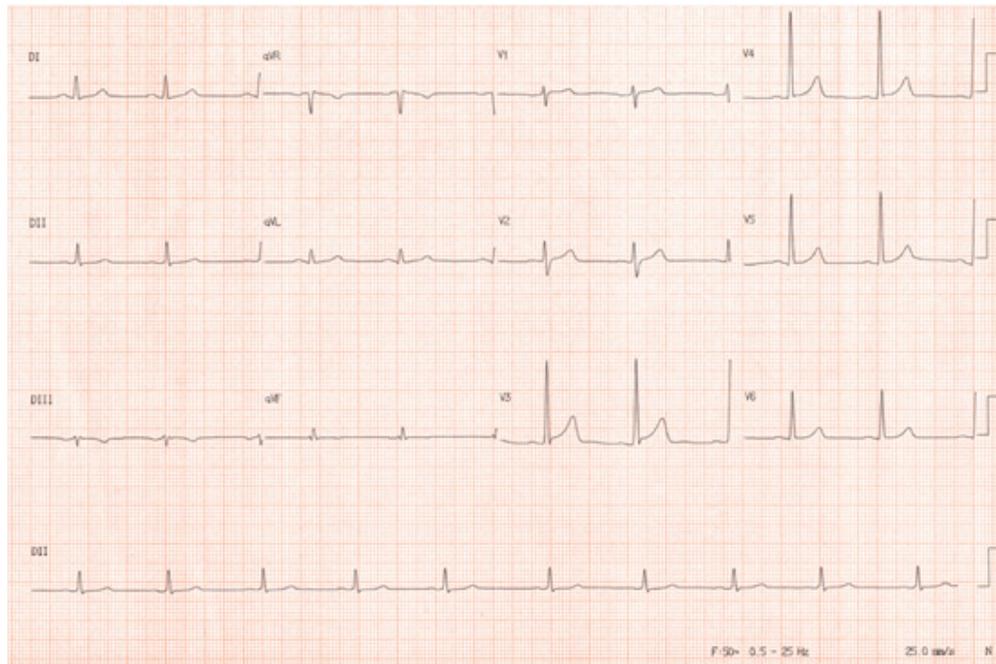


Figure 1 – The baseline tracing showed sinus rhythm (HR of 60 bpm) and ST-segment elevation in leads V3 to V5 with maximum amplitude of 0.15 mV. Lead III presents isolated T wave inversion.

repolarization (ER), and ischemia.^{1,2} Our patient is an otherwise asymptomatic 30-year-old man with a previous cardiac arrest who developed an episode of polymorphic VT during exercise. His baseline ECG showed a discreet ST-segment elevation in leads V3-V5, but it was otherwise unrevealing, while his echocardiogram depicted a ventricular apical hypokinesia. Due to his episode of exercise-induced polymorphic VT, catecholaminergic polymorphic ventricular tachycardia (CPVT) was among the initial hypotheses. CPVT is an inheritable adrenergic-dependent disorder in which VPBs and polymorphic VT may be induced during exertion, emotional stress, and isoproterenol or epinephrine infusion.¹⁻³ Arrhythmias become more complex as the HR rises and resolve upon discontinuation of the inciting factor.³ Our patient's VT, nevertheless, occurred during exercise recovery and was not preceded by progressive rhythm deterioration. On the other hand, as the use of beta-blockers has been found to suppress exercise-induced polymorphic ventricular arrhythmias in up to 65% of the patients,³ his normal testing six months later could be explained by a lack of arrhythmia reproducibility. The diagnosis of CPVT would be supported by a family history of syncope or sudden death and by appropriate genetic testing.¹⁻³

The presence of ST-segment elevation in leads V3-V5 may also resemble ER,^{1,2,4} a typically benign condition characterized by J-point elevation of at least 1 mm in 2 contiguous leads. Some individuals, however, may harbor an increased risk of idiopathic ventricular fibrillation (VF). The strongest association is seen in patients with horizontal or downsloping ST-segment elevation and an end-QRS notch or slur, as well as involvement of inferior leads.⁴ This has led to a 2015 consensus document⁴ that suggests that patients without end-QRS notch or slur should not be characterized as presenting ER. Hence, it is uncertain whether our patient's ascending ST-segment elevation in precordial leads portends an increased risk of arrhythmias.

Importantly, both CPVT and ER syndrome occur in structurally normal hearts.¹⁻⁴ Our patient's echocardiogram, however, revealed a ventricular apical hypokinesia. This finding is typical of Chagas disease,^{2,5} which is a protozoal infection endemic to most of South America, including the region where the patient lives. Transmission may be unnoticed and can also be taken by nonvectorial means,⁵ such as the ingestion of contaminated food or drinks. Up to 45% of patients present late cardiac findings⁵ characterized by myocardial inflammation and fibrosis with a preference for LV apex involvement, often leading

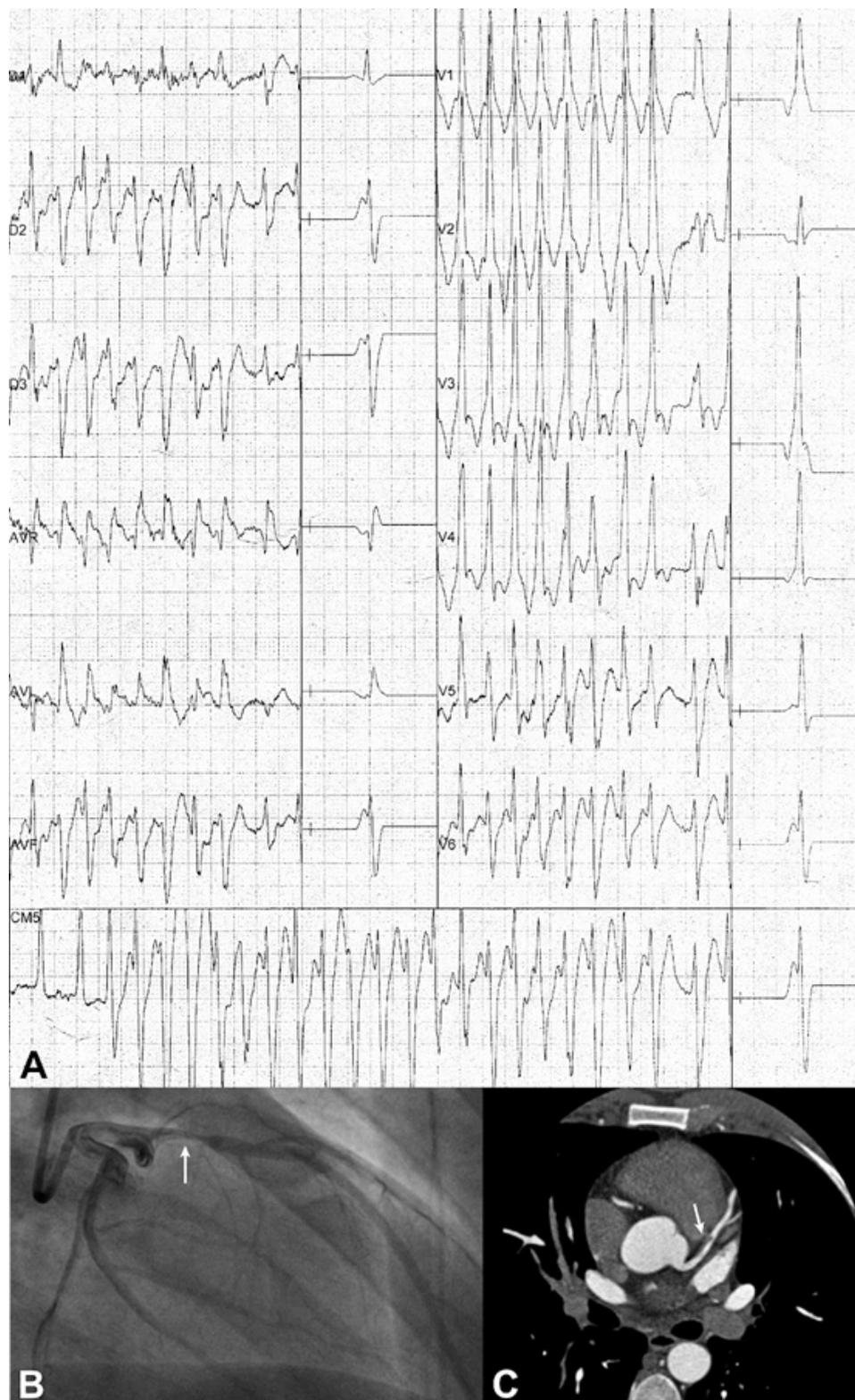


Figure 2 – (A) A polymorphic ventricular tachycardia with HR of 220 bpm is elicited during the recovery phase of an exercise stress test. (B) Coronary CT scan shows a hypodense plaque in the left anterior descending coronary artery with a stenosis of 70% (arrow). (C) Coronary angiography shows the lesion to cause a stenosis of 90% (arrow).

to aneurismal changes.⁵ Nevertheless, the diagnosis during the chronic phase is done with serologic assays, for which our patient had negative results. His hypokinesia could also be explained by left ventricular arrhythmogenic cardiomyopathy (LVAC).⁶ This exceedingly rare disease crosses with fatty infiltration of the LV and may overlap with the more common right ventricular arrhythmogenic cardiomyopathy. The LV shows reduced LVEF and significant dilation, with a median end-diastolic diameter of 65.2±5.6 mm in a Chinese series.⁶ The diagnosis, despite the lack of standardization, is supported by cardiovascular magnetic resonance imaging, genetic testing, and, in select cases, heart biopsy. However, in face of our patient's otherwise normal echocardiogram, his apical hypokinesia was considered as most likely a manifestation of regional ischemia. Accordingly, as about 50% of unexplained sudden cardiac arrests are caused by ischemia,¹ coronary CT or angiography is recommended for assessing the presence of CAD or coronary anomalies in such cases.^{1,2} Arrhythmias may occur in the setting of acute coronary syndromes, due to transient ischemia during exertion, or in patients with stable CAD.² Our patient's CT suggested the presence of a 70% LAD stenosis. Though we deemed angiography as a preferable next step in view of current recommendations,^{1,2} a functional imaging test could have also been performed due to the borderline value of stenosis at CT. Ultimately, his angiography revealed an even more serious lesion with a stenosis of 90%.

Due to the setting of premature CAD in the absence of major cardiovascular risk factors, prothrombotic conditions were investigated with normal results. However, androgenic-anabolic steroid abuse was associated with coronary plaque formation and LV dysfunction in a large prospective study,⁷ which might have contributed to our patient's disease.

References

1. Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis AB, et al. 2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol* 2018 Oct 2;72(14):1677-749.
2. Pedersen CT, Kay GN, Kalman J, et al. EHRA/HRS/APHS Expert Consensus on Ventricular Arrhythmias. *Europace*. 2014 Sep;16(9):1257-83.
3. Bauce B, Rampazzo A, Basso C, Bagattin A, Daliento L, Tiso N, et al. Screening for ryanodine receptor type 2 mutations in families with effort-induced polymorphic ventricular arrhythmias and sudden death: early diagnosis of asymptomatic carriers. *J Am Coll Cardiol* 2002;40:341-9.
4. Macfarlane PW, Antzelevitch C, Haissaguerre M, Huikuri HV, Potse M, Rosso R, et al. The Early Repolarization Pattern: A Consensus Paper. *J Am Coll Cardiol*. 2015 Jul 28;66(4):470-7.
5. Pérez-Molina JA, Molina I. Chagas Disease. *Lancet*. 2018 Jan 6;391(10115):82-94.
6. He, J., Xu, J., Li, G., Zhou, D., Li, S., Zhuang, B., Chen, X, et al. Arrhythmogenic Left Ventricular Cardiomyopathy: A Clinical and CMR Study. *Sci Rep*. 2020;10(1):533.
7. Baggish AL, Weiner RB, Kanayama G, Hudson JI, Lu MT, Hoffmann U, et al. Cardiovascular Toxicity of Illicit Anabolic-Androgenic Steroid Use. *Circulation*. 2017 May 23;135(21):1991-2002.3

Conclusions

Polymorphic VT may be the only manifestation of CAD, sometimes leading to cardiac arrest. In young subjects, this arrhythmia presents a challenging etiologic investigation, in which a comprehensible approach to patient history and baseline ECG is invaluable in order to guide further workup.

Author contributions

Conception and design of the research: Oliveira Neto NR, de Oliveira, WS. Acquisition of data: Porto AA, Oliveira Neto NR, Mendonça RM. Analysis and interpretation of the data: Oliveira Neto NR, Mastrocola F. Writing of the manuscript: Oliveira WS, Novaes AEM. Critical revision of the manuscript for intellectual content: Oliveira Neto NR, de Sousa JCV, Mastrocola F.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any thesis or dissertation work.

Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

