

Asymptomatic Patient with Complex Exercise-induced Ventricular Tachycardia

Michael Jeserich^{1*}, Simone Kimmel², Heinrich Bechtold², Martin Arnold³, Gabor Pavlik⁴, Bela Merkely⁴, Christoph Bode¹ and Annette Geibel^{1#}

¹Department of Cardiology and Angiology I, Heart Center Freiburg University, Germany

²Cardiology and Angiology, Koenigstr, Nuernberg, Germany

³Department of Cardiology, University of Erlangen-Nürnberg, Ulmenweg, Erlangen, Germany

⁴Heart and Vascular Center, Semmelweis University, Városmajor str, Budapest, Hungary

Author Deceased

*Corresponding author: Michael Jeserich, Koenigstr. 39, 90402 Nuernberg, Germany, Tel: 0049911209209; Fax: 00499112059962; E-mail: info@praxis-jeserich.de

Rec date: Aug 24, 2014, Acc date: Oct 25, 2014, Pub date: Oct 30, 2014

Copyright: © 2014 Jeserich M, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

We present a 58-year-old patient who had planned to start running and bicycling after the winter break. He took no medications on a regular basis, had no known risk factors, and his blood pressure and cholesterol levels were normal. His GP carried out an exercise bicycle test. Ventricular ectopic beats started at 200 watts at increasing frequency. The exercise bicycle test had to be stopped at 225 watts because of a polytope ventricular tachycardia. A sophisticated diagnostic procedure including CMR enabled us to diagnose asymptomatic myocarditis with oedema as cause of the complex ventricular tachycardia in this patient. EPU ruled out other causes such as Brugada syndrome, ion channel diseases or QT syndromes. The patient had no structural heart disease such as previous myocardial infarction, dilated or hypertrophic cardiomyopathies or arrhythmogenic right-heart cardiomyopathy. Coronary angiography ruled out coronary artery disease. This patient was at possible risk of sudden cardiac death while engaging in intensive sport activities. A diagnostic approach including cardiac CMR enabled this potentially life-saving diagnosis.

Keywords: Myocarditis; Ventricular tachycardia; Inflammation; Cardiovascular magnetic resonance; T2-imaging; STIR

Case report

A 58-year-old man presented at his general practitioner (GP) for a cardiac check-up. He had planned to start running and bicycling after the winter break, as he used to be very active in recreational sports. He takes no medications on a regular basis, had no known risk factors, and his blood pressure and cholesterol levels were normal. There are no cardiac disorders in first relatives family history under the age of 60 yrs. His father died aged 79 of (presumably) heart failure. His GP carried out an exercise bicycle test. Ventricular ectopic beats (VPCs) started at 200 watts at increasing frequency. The exercise bicycle test had to be stopped at 225 watts because of a polytope ventricular tachycardia (VT; Figures 1a and 1b). The patient was asymptomatic during exercise and during VPCs or VT.

The patient was referred to our cardiology center. He had been in his usual state of health months before. During the previous winter he had suffered a brief viral respiratory tract infection, about 8 weeks prior to this cardiological examination. He complained of no palpitation, exertional dyspnoea, fatigue or retrosternal pain.

On examination, his temperature was 37.30°C, blood pressure was 133/92 mm Hg. The pulse rate was 64 beats per minute at a regular rate and his respiratory rate was normal. He weighed 90 kg and was 181 cm tall with a body-mass index of 27.5. Heart sounds were normal without murmur. The lungs were free. There was no palpable hepatomegaly and no apparent peripheral oedema. At admission the ECG revealed a sinus rhythm at a rate of 64/min and a left anterior hemibloc. His AV-conduction time was normal and there were no ST-

T-wave-abnormalities. QRS duration was modestly increased to 112 ms, as was QT time with 438 ms (QTc 115%). This was in contrast to the ECG done by his GP two weeks beforehand with flattened T-Waves (Figure 1a).

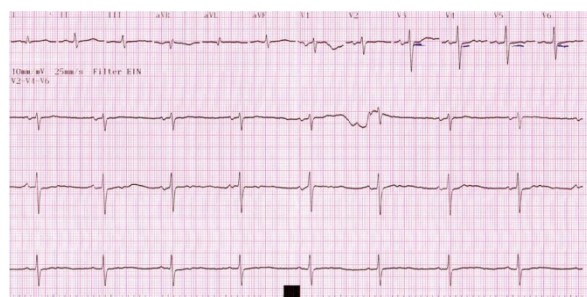


Figure 1a: 12 lead-ECG at rest. T-wave changes are visible and moderately increased QT time (QTc 115%)

Echocardiography showed a left atrium (38 mm) and ventricle of normal dimensions. Left end-diastolic and end-systolic diameters were 52 and 34 mm, respectively. Left-ventricular function was normal (65%) without regional wall abnormalities. There was no significant valve regurgitation. Holter ECG showed a sinus rhythm with a mean frequency of 61. There were 730 ventricular premature beats, and 4 couplets present. The patient underwent coronary angiography to rule out an ischaemic cause of the arrhythmias.

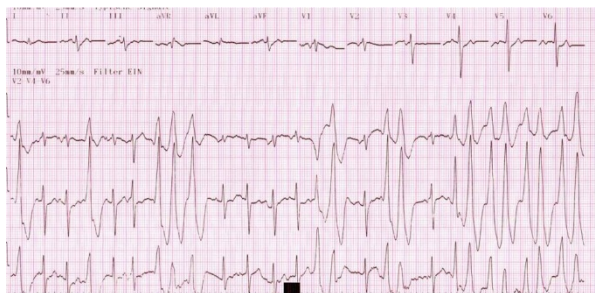


Figure 1b: ECG during maximum exercise on a treadmill (225 watts). There are ectopic ventricular beats and polytopal ventricular tachycardia visible

The coronary arteries were without any atherosclerotic lesions and without coronary artery anomalies. Laboratory tests were normal including high sensitive troponin-T and BNP values. Cardiac magnetic resonance (CMR) imaging and an electrophysiology test were also performed. CMR revealed anterolateral oedema indicated by an elevated STIR (a triple inversion recovery spin echo sequence) myocardium/muscle ratio and midmyocardial/subendocardial late-contrast enhancement ten minutes after IV administration of 0.2 mmol/kg intravenous gadolinium, and a 3D inversion recovery turbo gradient echo sequence (Figures 2 and 3). The patient was advised to stop sport activities, and was given tablets containing potassium and magnesium. We decided against beta-blocker therapy because of intrinsic low heart rate. After six weeks the oedema had disappeared and there was less contrast enhancement on the control CMR. Electrophysiology testing helped us rule out Brugada syndrome as well as conduction, congenital abnormalities or QT syndromes. During electrophysiology testing six weeks after the index data there were no inducible ventricular ectopy. The bicycle test revealed less ventricular ectopy, and no ventricular tachycardia occurred.

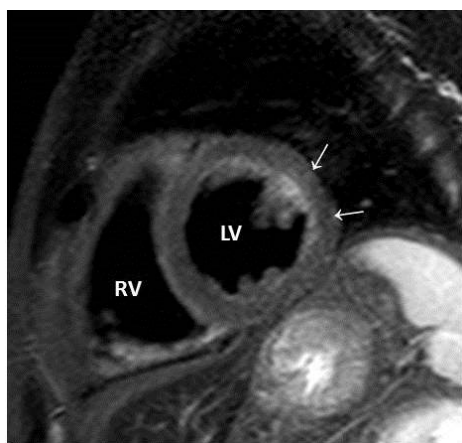


Figure 2: An example of T2-weighted (STIR) image of this patient. Enhanced signal intensity of the myocardium of the left ventricle, especially anterolateral (arrows). LV: Left Ventricle

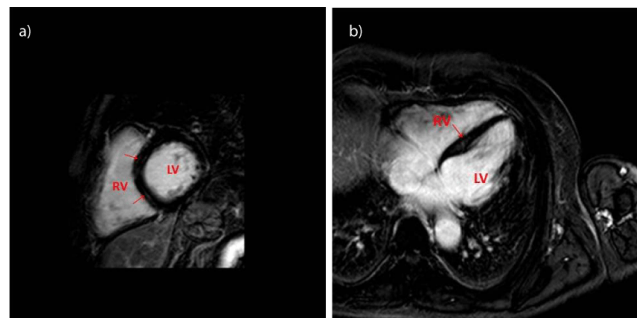


Figure 3: Late-enhancement image of this patient. Note the patchy enhancement of the midwall anterior wall (↑). Two and four chamber view. LV: Left Ventricle; RV: Right Ventricle.

CMR imaging has recently emerged as an important procedure for assessing cardiovascular disease. Functional parameters of LV function, dimensions, and flow can be measured accurately [1,2]. In acute and chronic myocarditis, myocardial regions suffering from irreversible inflammatory injury show bright signal intensity due to increased gadolinium-distribution volume long after intravenous administration [3,4]. A short-term triple inversion recovery spin echo sequence (STIR) serves as an indicator of excess water content in tissue and has been used in primary inflammation (acute myocarditis) and infarct-related ischaemic injury within the perfusion bed [3-7]. This STIR sequence has been tested in patients with accompanying myocarditis after viral or gastrointestinal tract infections [8,9]. We combined this approach with the late gadolinium enhancement (LGE) contrast-enhanced technique widely used as an accurate and robust method in detecting myocarditis [3,4,10,11]. Both parameters were positive in this patient (Figures 4a and 4b). It revealed anterolateral oedema and a typical midmyocardial/subepicardial late-enhancement pattern as a sign of myocarditis [3,6,8,9,12].

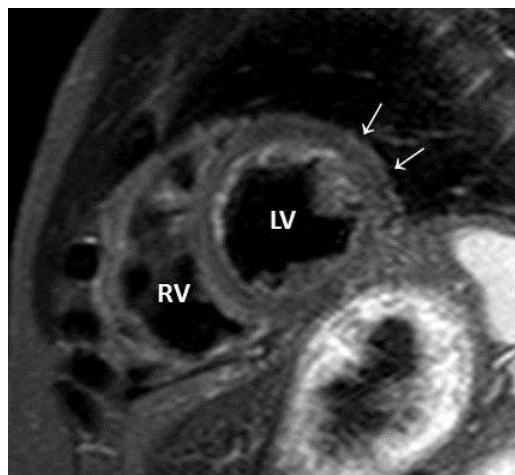


Figure 4a: T2-weighted (STIR) image of this patient after six weeks. The enhanced signal intensity of the myocardium of the left ventricle, especially anterolateral (arrows) had disappeared. LV: Left Ventricle

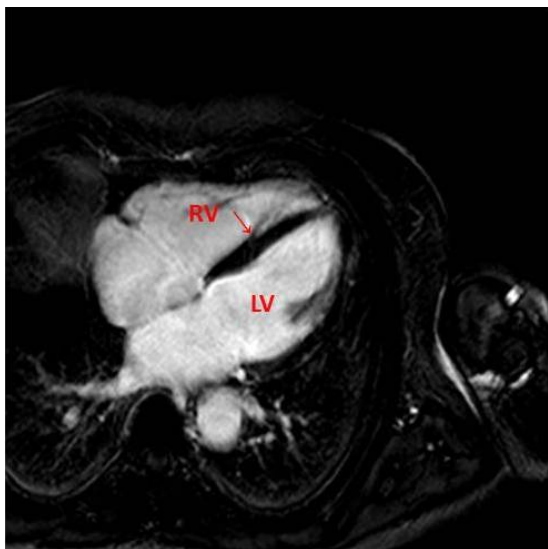


Figure 4b: Late-enhancement image of this patient after six weeks. There was less contrast enhancement visible (↑). Four chamber view. LV: Left Ventricle. RV: Right Ventricle

Patients with ventricular tachycardia without underlying heart disease have a good prognosis [13]. However, ventricular tachycardia is a key cause of sudden cardiac death in developed countries [14]. Coronary artery disease is the most frequent cause of ventricular tachycardia and sudden cardiac death in individuals over the age of 30, while hypertrophic cardiomyopathy (HCM), myocarditis and congenital heart disease are the most frequent causes of death in those below 30 years of age [14,15]. Thus it is very important to detect any potential cardiac disease as an underlying cause of ventricular tachycardia. Moreover, long-term studies have detected an association between exercise-induced ventricular premature beats as our patient experienced and a higher risk of cardiovascular or all-cause mortality rates [16,17]. Our patient appeared to be clinically healthy. Only a sophisticated diagnostic procedure including CMR enabled us to diagnose asymptomatic myocarditis as highly probable cause of the complex ventricular tachycardia in this patient. EPU ruled out other causes such as Brugada syndrome, ion channel diseases or QT syndromes. The patient had no structural heart disease such as previous myocardial infarction, dilated or hypertrophic cardiomyopathies or arrhythmogenic right-heart cardiomyopathy. Interestingly, in the Framingham population, exercise-induced ventricular premature beats were associated with increased all-cause mortality rates during follow-up of a mean of 15 years but they were not associated with increased myocardial infarction, coronary insufficiency, or cardiac death [17]. There might be other underlying causes therefore, such as occult myocarditis.

In conclusion, we detected an asymptomatic myocarditis in an apparently clinically-healthy patient with exercise-induced VPCs and complex VT upon intensive exercise. This patient was at possible risk of sudden cardiac death while engaging in intensive sport activities. A diagnostic approach including cardiac CMR enabled this potentially life-saving diagnosis.

References

1. Marcu CB, Beek AM, van Rossum AC (2006) Clinical applications of cardiovascular magnetic resonance imaging. *CMAJ* 175: 911-917.
2. Gibbons RJ, Araoz PA, Williamson EE (2009) The year in cardiac imaging. *J Am Coll Cardiol* 53: 54-70.
3. Friedrich MG, Sechtem U, Holmvang G, Alakija P, Cooper LT, et al. (2009) Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J Am Coll Cardiol* 53: 1475-1487.
4. Abdel-Aty H, Boye P, Zagrosek A, Wassmuth R, Kumar A, et al. (2005) Diagnostic performance of cardiovascular magnetic resonance in patients with suspected acute myocarditis: comparison of different approaches. *J Am Coll Cardiol* 45: 1815-1822.
5. Assomull RG, Lyne JC, Keenan N, Gulati A, Bunce NH, et al. (2007) The role of cardiovascular magnetic resonance in patients presenting with chest pain, raised troponin, and unobstructed coronary arteries. *Eur Heart J* 28: 1242-1249.
6. Jeserich M, Konstantinides S, Pavlik G, Bode C, Geibel A (2009) Non-invasive imaging in the diagnosis of acute viral myocarditis. *Clin Res Cardiol* 98: 753-763.
7. Viallon M, Mewton N, Thuny F, Guehring J, O'Donnell T, et al. (2012) T2-weighted cardiac MR assessment of the myocardial area-at-risk and salvage area in acute reperfused myocardial infarction: comparison of state-of-the-art dark blood and bright blood T2-weighted sequences. *J Magn Reson Imaging* 35: 328-339.
8. Gutberlet M, Spors B, Thoma T, Bertram H, Denecke T, et al. (2008) Suspected chronic myocarditis at cardiac MR: diagnostic accuracy and association with immunohistologically detected inflammation and viral persistence. *Radiology* 246: 401-409.
9. Jeserich M, Brunner E, Kandolf R, Olschewski M, Kimmel S, et al. (2013) Diagnosis of viral myocarditis by cardiac magnetic resonance and viral genome detection in peripheral blood. *Int J Cardiovasc Imaging* 29: 121-129.
10. Laissy JP, Hyafil F, Feldman LJ, Juliard JM, Schouman-Claeys E, et al. (2005) Differentiating acute myocardial infarction from myocarditis: diagnostic value of early- and delayed-perfusion cardiac MR imaging. *Radiology* 237: 75-82.
11. Babu-Narayan SV, McCarthy Karen P, Siew Yen Ho, Alan G Magee, et al. (2007) Images in cardiovascular medicine. Myocarditis and sudden cardiac death in the young extensive fibrosis suggested by cardiovascular magnetic resonance in vivo and confirmed post mortem. *Circulation* 166: 122-125.
12. Mahrholdt H, Wagner A, Deluigi CC, Kispert E, Hager S, et al. (2006) Presentation, patterns of myocardial damage, and clinical course of viral myocarditis. *Circulation* 114: 1581-1590.
13. Sellal JM, Brembilla-Perrot B, Suty-Selton C, Sadoul N, Huttin O (2011) Ventricular tachycardia without apparent heart disease: long-term follow-up. *J Electrocardiol* 44: 786-791.
14. Mavrogeni S, Petrou E, Kolovou G, Theodorakis G, Iliodromitis E (2013) Prediction of ventricular arrhythmias using cardiovascular magnetic resonance. *Eur Heart J Cardiovasc Imaging* 14: 518-525.
15. Maron BJ (2003) Sudden death in young athletes. *N Engl J Med* 349: 1064-1075.
16. Jouven X, Zureik M, Desnos M, Courbon D, Ducimetière P (2000) Long-term outcome in asymptomatic men with exercise-induced premature ventricular depolarizations. *N Engl J Med* 343: 826-833.
17. Morshedi-Meibodi A, Evans JC, Levy D, Larson MG, Vasan RS (2004) Clinical correlates and prognostic significance of exercise-induced ventricular premature beats in the community: The Framingham Heart Study. *Circulation* 109: 2417-2422.