JACC: CASE REPORTS VOL. 3, NO. 3, 2021


MINI-FOCUS ISSUE: ELECTROPHYSIOLOGY

INTERMEDIATE

CASE REPORT: CLINICAL CASE

# Asymptomatic Athlete With Short-Coupled Premature Ventricular Contractions

Anat Milman, MD, PHD,[a,b,c] Bernard Belhassen, MD,[c,d] Avi Sabbag, MD,[a,c] Gal Dubnov-Raz, MD,[b,c] Roy Beinart, MD[a,c]

## ABSTRACT

We present an asymptomatic 26-year-old athlete, with no family history of sudden cardiac death and no structural heart disease, who displayed short-coupled premature ventricular contractions on exercise test and Holter monitoring. The rarity of the case as well as management dilemmas are discussed. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:496-501) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

## HISTORY OF PRESENTATION

A 26-year-old male athlete was referred to our center by his workplace for a physical screening test. He was very physically active, training 6 times per week (CrossFit). He was asymptomatic and had no personal history of syncope or any family history of sudden cardiac death (SCD). Results of his physical examination were normal, and he was first scheduled for a maximal exercise electrocardiogram (ECG) test (EET).

**Figure 1** represents his resting ECG at the beginning of the EET. There was a sinus arrhythmia (rate ranging from 41 to 62 beats/min) with normal QRS pattern and duration (80 ms). The QTc interval was normal at approximately 400 ms; there was a bifid T-wave in lead $V_2$. An early repolarization pattern (ERP) was observed in the infero-lateral leads including a notched J-wave with upsloping ST-segment interval maximal in leads $V_3$ and $V_4$. Finally, a single premature ventricular contraction (PVC) with a short coupling interval of 290 ms was noted.

The EET was performed using Bruce protocol, with maximal capacity for age reached (93% of maximal heart rate; total exercise time 14 min and 35 s). The patient was asymptomatic during the test. The monofocal PVCs persisted during the test with the same short coupling interval but the ERP disappeared (**Figure 2**). During the recovery phase, the short-coupled PVCs recurred as intermittent bigeminy (**Figure 3**). They had a left bundle branch block morphology with a QRS duration of 110 ms and axis of −30°. A saddle ST-T elevation was present in $V_1$ but disappeared on resumption of the periods of ventricular bigeminy.

### LEARNING OBJECTIVES

- To make a differential diagnosis of short-coupled PVCs.
- To understand the malignant risk of this finding and what workup should be performed.

From the [a]Leviev Heart Institute, Sheba Medical Center, Tel Hashomer, Israel; [b]Sports and Exercise Medicine Clinic, Sheba Medical Center, Tel Hashomer, Israel; [c]Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel; and the [d]Heart Institute, Hadassah University Hospital, Jerusalem.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received November 6, 2020; revised manuscript received November 30, 2020, accepted December 14, 2020.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2020.12.039

A 24-h ECG Holter revealed 943 single, short-coupled PVCs (1.3% burden) equally distributed during the recording. The morphology of the single PVCs was similar on the three ECG leads to that observed during exercise recovery; however, the coupling interval was slightly longer ranging from 360 to 364 ms at 10-min intervals between 12:25 AM and 12:45 AM when the patient was awake (Figure 4). There were also 1 couplet (Figure 5) and 1 triplet. No significant changes in QTc intervals were found at both slower and faster heart rates.

## MEDICAL HISTORY

The patient was healthy, never suffered from syncope episodes, did not take any medication, and did not smoke or use illicit drugs.

## DIFFERENTIAL DIAGNOSIS

Very short-coupled PVCs have been recognized as a trigger for malignant ventricular arrhythmias in patients with no structural heart disease. These arrhythmias, which usually originate in the Purkinje system and less commonly in the ventricular myocardium, have been documented at the onset of malignant arrhythmic events in patients with idiopathic ventricular fibrillation (1). Although their prevalence in normal asymptomatic subjects is unknown, our experience suggests that such arrhythmias are exceptional in the normal population. Their observation in our young athletic subject who exhibited various repolarization disorders warranted comprehensive investigation.

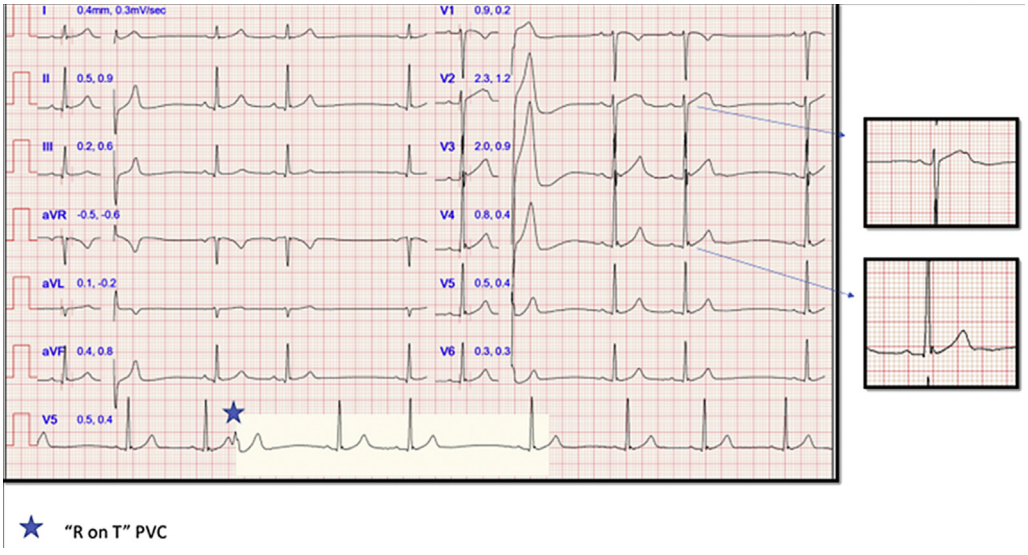
## INVESTIGATIONS

Structural heart disease was ruled out using normal transthoracic echocardiogram and cardiac magnetic resonance imaging. The diagnosis of Brugada syndrome was ruled out using the ajmaline test (1 mg/kg over 10 min), suggesting that the pseudo-Brugada type 2 ECG pattern observed in $V_1$ during ventricular bigeminy was actually the result of a "memory phenomenon" secondary to the prior PVC (2). The bifid pattern of T-wave in $V_2$ was considered as a normal variant with no suspicion of any type of long QT syndrome. Finally, the ERP observed in the inferolateral leads was considered to be of unclear prognostic significance according to an expert consensus (3); actually, the notched J-wave is thought to suggest a worse prognosis while an upsloping ST-segment is considered to suggest a good one (3). Our presumptive final diagnosis was short-coupled Purkinje PVCs originating at a medial insertion site of the moderator band near the right bundle branch (4,5).

### ABBREVIATIONS AND ACRONYMS

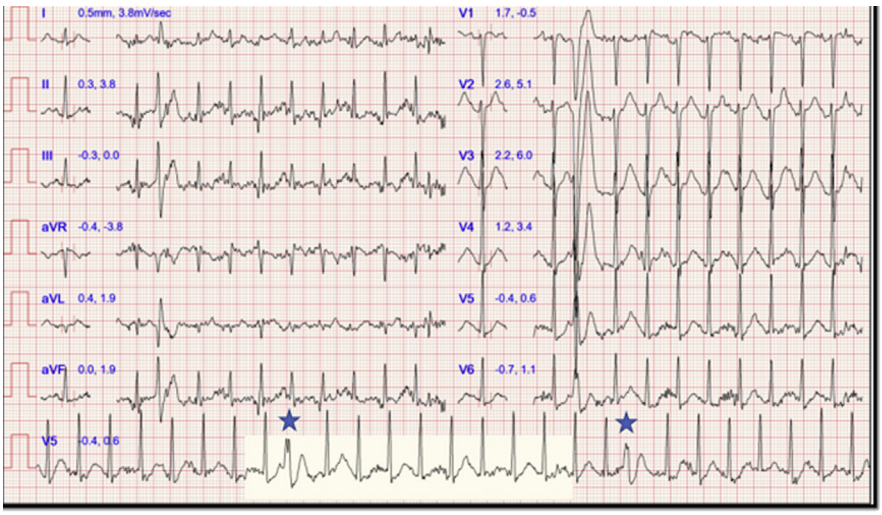
**ECG** = electrocardiogram
**EET** = exercise ECG test
**ERP** = early repolarization pattern
**PVC** = premature ventricular contraction
**SCD** = sudden cardiac death

**FIGURE 1** Resting Electrocardiogram



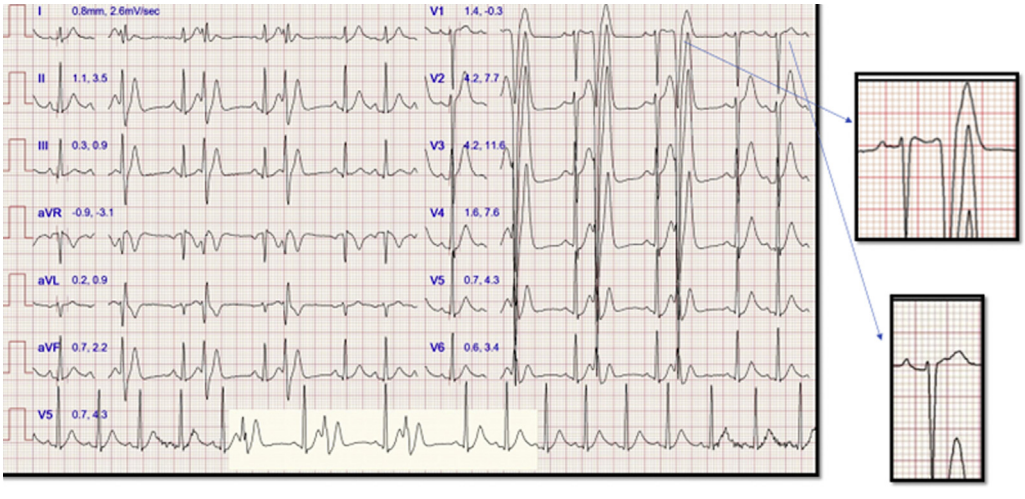

Sinus arrhythmia with normal QRS and QTc (400 ms). A bifid T-wave and an early repolarization pattern are present in lead $V_2$ and the inferolateral leads, respectively. A single premature ventricular contraction (PVC) with a short coupling interval of 290 ms is observed.

**FIGURE 2 Maximal Exercise Electrocardiogram Test**



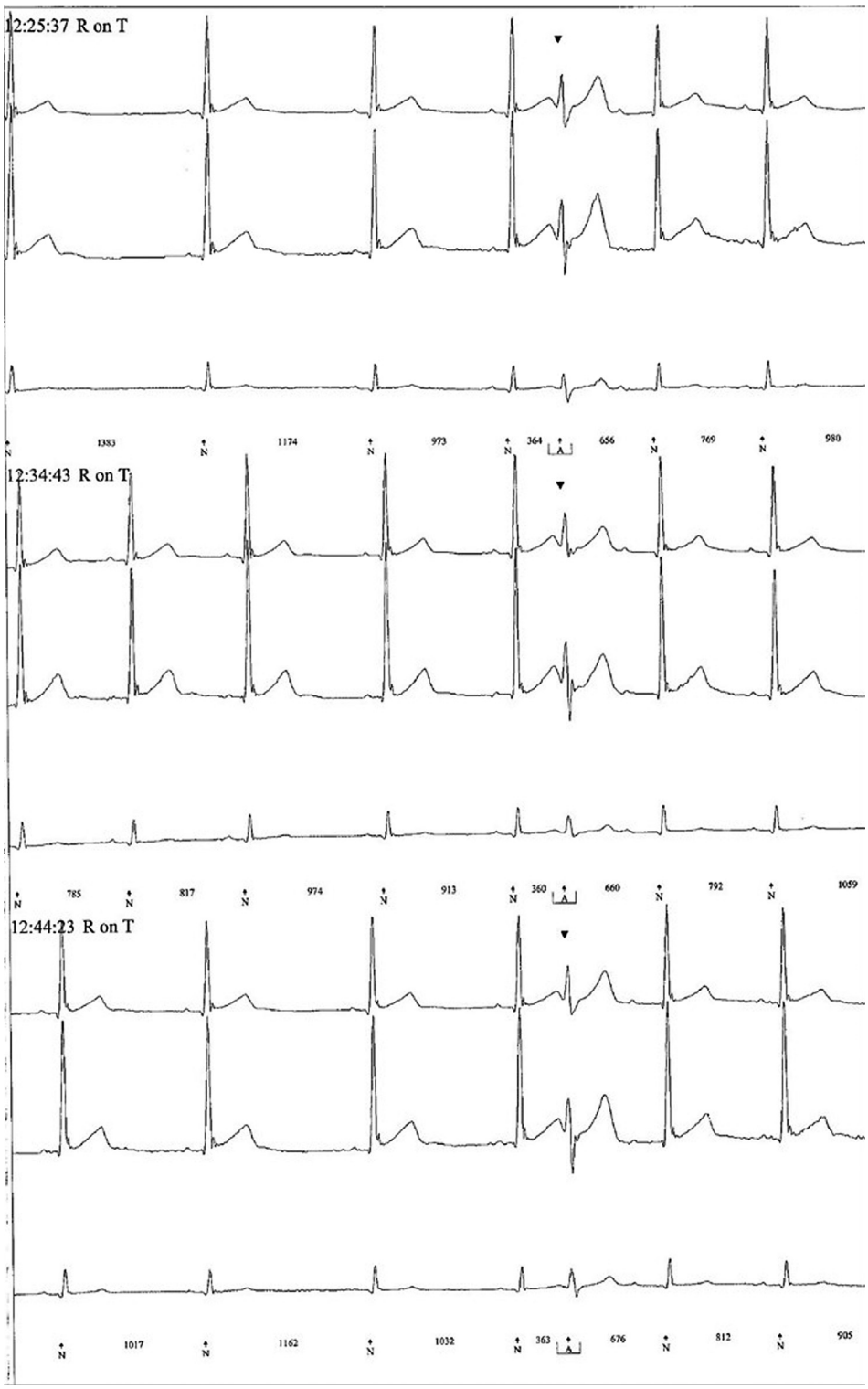

Monofocal premature ventricular contractions (PVCs) persist during the test with the same short coupling interval but the early repolarization pattern disappears.

**FIGURE 3 Recovery**



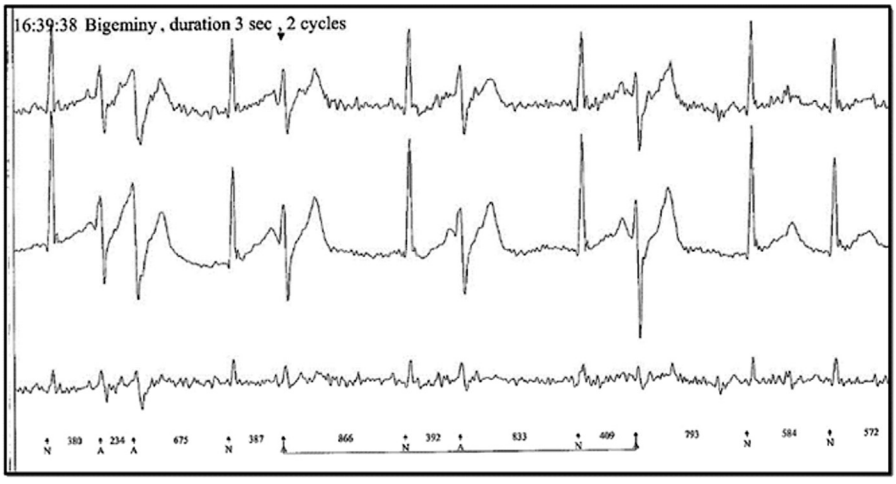

Short-coupled premature ventricular contractions (PVCs) as intermittent bigeminy. PVC have a left bundle branch block morphology with a QRS duration of 110 ms and axis of −30°. A saddle ST-T elevation is present in $V_1$ only during the periods of ventricular bigeminy. Note the normalization of the repolarization on the last beat in $V_1$.

**FIGURE 4** **Holter Recording**




Monofocal single short-coupled premature ventricular contractions (PVCs) recorded at 10-min intervals. Note that the morphology of the PVCs on the 3 electrocardiogram leads is similar to that observed during exercise recovery; however, the coupling interval is slightly longer (360 to 364 ms).

**FIGURE 5 Holter Recording**




One single couplet is also recorded initiated with a short-coupled premature ventricular contraction.

## MANAGEMENT

Therapeutic options include the following: 1) a "wait and see" option while strenuous physical activity is forbidden and a loop recorder ECG implanted; 2) an empirically guided treatment with oral quinidine (beginning with low doses that are usually well tolerated), taking into account the great efficacy of this medication in patients with arrhythmic storms of idiopathic ventricular fibrillation (6,7); 3) electrophysiologically guided quinidine therapy, which may be recommended for those patients who also have ventricular fibrillation inducible with programmed ventricular stimulation (7,8); 4) radiofrequency ablation of the PVCs, which could be an excellent option in patients with mappable monofocal arrhythmias (9); and 5) implantation of an implantable defibrillator also may be discussed, but only as an ultimate resort. As far as genetic testing, a single study showed multiple ectopic Purkinje-related premature contractions and attributed them to an *SCN5A* cardiac channelopathy (10). In our subject, the lack of any family history of arrhythmias and sudden death would not support a familial channelopathy.

## DISCUSSION

The asymptomatic 26-year-old athlete, with no family history of SCD and no structural heart disease hereunder reported, presented with a PVC burden of 1.3% short-coupled PVCs on Holter monitoring, presumably originating from the right ventricle moderator band. The arrhythmias did not disappear during exercise. We have not encountered a similar case during up to 45 years activity in cardiology (by our most senior author, BB), nor did other physicians with vast experience in the field of ventricular arrhythmias in athletes (Alessandro Zorzi, October, 2020, personal communication).

In the recent guidelines on sports cardiology published by the European Society of Cardiology (11), athletes with PVCs on baseline ECG have received a Class I recommendation to be evaluated for excluding a structural heart disease or arrhythmogenic conditions. After ruling out a family history of SCD and structural heart disease, all sports activities are permitted. However, the guidelines state that very rarely, otherwise "benign" PVCs arising from the Purkinje network may give rise to polymorphic ventricular tachycardia or fibrillation due to their short coupling interval; in such patients, the malignant electrical presentation mandates aggressive treatment. However, specific recommendations for the clinical management of such subjects are lacking.

## FOLLOW-UP

Our patient decided to continue his athletic activity. He definitely declined receiving an implantable loop recorder or undergoing any invasive procedure despite our repeated explanations that such a refusal

may have lethal consequences. He has remained asymptomatic during 4 months of follow-up.

## CONCLUSIONS

This unique case report should prompt the performance of multicenter studies exploring the prevalence of short-coupled PVCs in subjects with apparently normal hearts, especially those engaged in athletic activities. The question whether such PVCs could lead to potentially lethal ventricular arrhythmias during strenuous exercise or as a result of commotio cordis should be raised.

## FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

**ADDRESS FOR CORRESPONDENCE:** Dr. Anat Milman, Leviev Heart Institute, Sheba Medical Center, Tel Hashomer, Israel. E-mail: anatmilman@gmail.com.

## REFERENCES

**1.** Haïssaguerre M, Shoda M, Jaïs P, et al. Mapping and ablation of idiopathic ventricular fibrillation. Circulation 2002;106:962–7.

**2.** Chiale PA, Etcheverry D, Pastori JD, et al. The multiple electrocardiographic manifestations of ventricular repolarization memory. Curr Cardiol Rev 2014;10:190–201.

**3.** Antzelevitch C, Yan GX, Ackerman MJ, et al. J-Wave syndromes expert consensus conference report: emerging concepts and gaps in knowledge. Europace 2017;19:665–94.

**4.** Sadek MM, Benhayon D, Sureddi R, et al. Idiopathic ventricular arrhythmias originating from the moderator band: electrocardiographic characteristics and treatment by catheter ablation. Heart Rhythm 2015;12:67–75.

**5.** Barber M, Chinitz J, John R. Arrhythmias from the right ventricular moderator band: diagnosis and management. Arrhythm Electrophysiol Rev 2020;8:294–9.

**6.** Kontny F, Dale J. Self-terminating idiopathic ventricular fibrillation presenting as syncope: a 40-year follow-up report. J Intern Med 1990;227:211–3.

**7.** Belhassen B. A 25-year control of idiopathic ventricular fibrillation with electrophysiologic-guided antiarrhythmic drug therapy. Heart Rhythm 2004;1:352–4.

**8.** Belhassen B, Viskin S, Fish R, Glick A, Setbon I, Eldar M. Effects of electrophysiologic-guided therapy with Class IA antiarrhythmic drugs on the long-term outcome of patients with idiopathic ventricular fibrillation with or without the Brugada syndrome. J Cardiovasc Electrophysiol 1999;10:1301–12.

**9.** Haïssaguerre M, Duchateau J, Dubois R, et al. Idiopathic ventricular fibrillation: role of Purkinje system and microstructural myocardial abnormalities. J Am Coll Cardiol EP 2020;6:591–608.

**10.** Laurent G, Saal S, Amarouch MY, et al. Multifocal ectopic Purkinje-related premature contractions: a new SCN5A-related cardiac channelopathy. J Am Coll Cardiol 2012;60:144–56.

**11.** Pelliccia A, Sharma S, Gati S, et al. ESC Scientific Document Group. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. Eur Heart J 2021;42:17–96.

**KEY WORDS** electrophysiology, exercise, ventricular tachycardia