EJCRIM
European Journal
of Case Reports
in Internal Medicine

SHORT-COUPLED PREMATURE VENTRICULAR CONTRACTIONS IN A YOUNG MALE WITH SUDDEN CARDIAC ARREST

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Received: 12/04/2025
Accepted: 28/04/2025
Published: 20/05/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: The authors declare no conflict of interest.
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How to cite this article: Mansoor T, Costa Filho FF, Furlan A, Madeiro M, Kelly M, Koshy SKG, Franzon T. Short-coupled premature ventricular contractions in a young male with sudden cardiac arrest. *EJCRIM* 2025;**12**:doi:10.12890/2025_005443

ABSTRACT

Introduction: Idiopathic ventricular fibrillation secondary to short-coupled premature ventricular contractions (scPVCs) is a rare cause of sudden cardiac arrest and predominantly originates from the His-Purkinje system or right ventricular outflow tract.

Case description: A 36-year-old man with prior episodes of palpitations and a normal Holter monitor evaluation as well as a family history of sudden cardiac death presented following an out-of-hospital ventricular fibrillation cardiac arrest. After exclusion of other etiologies including structural, ischemic, and primary electrical disease, as well as the demonstration of scPVCs with moderator band origin, a diagnosis of scPVCs leading to sudden cardiac arrest was made. Following implantable cardioverter defibrillator and treatment of other medical concerns, he was discharged home.

Conclusion: Idiopathic ventricular fibrillation is an uncommon cause of sudden cardiac arrest. The diagnosis is made after the exclusion of other etiologies and the demonstration of characteristic electrocardiogram findings.

KEYWORDS

Short-coupled premature ventricular contractions, cardiac arrest

LEARNING POINTS

- Short-coupled premature ventricular contractions are a rare cause of sudden cardiac arrest.
- The diagnosis is based on the demonstration of characteristic electrocardiogram findings and the exclusion of other etiologies including structural, ischemic, and primary electrical disease.
- Treatment involves pharmacotherapy, implantable cardioverter defibrillator placement, and cardiac ablation.

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

INTRODUCTION

Idiopathic ventricular fibrillation is a rare cause of sudden cardiac arrest and is characterized by premature ventricular contractions (PVCs) with a short coupling interval of <300 ms, a short QRS duration, and a normal QT interval[1]. There is evidence to support that these short-coupled PVCs (scPVCs) may originate from the His-Purkinje system or the right ventricular outflow tract but uncertainties regarding the pathophysiology remain[2]. Furthermore, although there is a potential hereditary component for the occurrence of scPVCs, evidence regarding causal genetic variants remains limited. Diagnosis of scPVCs can be challenging due to the intermittent nature of this arrythmia.

CASE DESCRIPTION

A 36-year-old obese male with hyperlipidemia, prior episodes of palpitations and a normal Holter monitor evaluation as well as a family history of sudden cardiac death in 2 maternal non-first-degree male relatives presented to the emergency department after suffering an out-of-hospital ventricular fibrillation (VF) cardiac arrest with return of spontaneous circulation after cardiopulmonary resuscitation and defibrillation in the field. The patient arrived at the emergency department intubated but hemodynamically stable and was admitted to the intensive care unit for anoxic encephalopathy, respiratory failure, and post cardiac arrest care.

Initial electrocardiogram on presentation demonstrated normal sinus rhythm, scPVCs with a left bundle and left superior axis. The QT interval was normal and there was no evidence of early repolarization or Brugada syndrome. Toxicology screen was also negative. Transthoracic echocardiogram at admission demonstrated severe global left ventricular dysfunction with an ejection fraction (EF) of 20% to 25%.

Following extubation, the patient underwent coronary angiography with left ventriculography on hospital day 4 which demonstrated normal coronary arteries with a recovered EF of 60%. Further evaluation with a cardiac magnetic resonance imaging with contrast demonstrated normal cardiac morphology and no evidence of myocardial scar. Genetic testing revealed variants of unknown significance in the *ANK2* and *SCN5A* genes. Ultimately, after exclusion of other etiologies, and demonstration of scPVCs with morphology consistent for moderator band or right ventricle papillary muscle origin on electrocardiogram (ECG), a diagnosis of idiopathic PVC-induced VF was made. The patient underwent subcutaneous implantable cardioverter defibrillator implantation on hospital day 9 and returned to work approximately 7 weeks after his cardiac arrest. An analysis of 42 genes in known association with inherited arrythmias revealed a variant of unknown significance with the absence of clinically actionable alterations. Further genetic testing and genetic counseling of the patient as well as the family members was not pursued.

DISCUSSION

We describe a case of out-of-hospital cardiac arrest in a relatively healthy 36-year-old male secondary to idiopathic VF after exclusion of underlying structural heart disease, primary electrical disease, and other reversible causes. The presence of short-coupled PVC's consistent with moderator band origin supported the diagnosis in this case *(Fig. 1)*.

Idiopathic VF is a rare cause of sudden cardiac arrest in individuals in the absence of structural heart disease triggered by a scPVC with a coupling interval of <300 ms originating predominantly from the His-Purkinje system or right ventricular outflow tract[3]. Idiopathic VF has been shown to account for approximately 5 to 10% of all out of hospital cardiac arrests, with 25% of those demonstrating scPVCs[4]. A family history of sudden cardiac death is present in approximately 25 to 30% of presentations with multiple implicated genetic variants. Underlying mechanisms are thought to include increases in transient outward potassium current in the Purkinje network, intracellular calcium dysregulation, and transmural heterogeneity of autonomic innervation[1,3].

Implantable cardioverter defibrillator implantation is necessary for patients after scPVC cardiac arrest for secondary prevention. Pharmacological treatment with



Figure 1. Electrocardiogram demonstrating short coupled premature ventricular contractions in the patient.

verapamil for scPVCs leading to cardiac arrest reduces the ectopic burden and lengthens the PVC coupling interval[1]. Class 1A antiarrhythmic agents, especially quinidine, can reduce the incidence of scPVCs[1]. Catheter ablation has also been shown to reduce the risk of recurrent VF. In an analysis of patients with recurrent episodes of idiopathic VF who underwent ablation, 89% were shown to have absence of VF recurrence during a follow-up of 24±28 months[4].

CONCLUSION

Short-coupled PVCs represent an uncommon cause of sudden cardiac arrest in patients in the absence of structural heart, disease, primary electrical disease, and other reversible causes. The diagnosis is made after the exclusion of other etiologies and the demonstration of characteristic ECG findings. Treatment includes pharmacological therapy, implantable cardioverter defibrillator implantation, and catheter ablation.

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