JACC: CASE REPORTS VOL. 3, NO. 8, 2021

CASE REPORT

ADVANCED

CLINICAL CASE

Spontaneous Termination of Ventricular Fibrillation

Is Organization Required?

Braden Vogt, BS,[a] Michael Kwok, MS,[a] Michael Wu, MD[b]

ABSTRACT

Ventricular fibrillation is a life-threatening arrhythmia that can result in sudden cardiac death and almost always requires emergency electrical defibrillation. This paper presents a unique case of a 74-year-old woman with spontaneous termination of a 2-min 13-s ventricular fibrillation episode without organization before termination. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:1108–13) © 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 74-year-old woman presented to the emergency department (ED) with a syncopal episode 1 week after atrial fibrillation (AF) ablation. Vitals and pertinent physical exam were unremarkable on presentation. Electrocardiography (ECG) in the ED showed sinus rhythm at 61 beats/min with a PR interval of 188 ms, QRS interval of 92 ms, and QTc of 600 ms (**Figure 1**). Laboratory values were remarkable for potassium at 3.1 mEq/l (normal: 3.6 to 5.1 mEq/l), magnesium at 1.5 mEq/l (normal: 1.3 to 1.9 mEq/l), and troponin I level at 0.191 ng/ml (normal: 0.006 to 0.060 ng/ml). Chest x-ray was unremarkable.

LEARNING OBJECTIVES

- To discuss spontaneous termination of ventricular fibrillation and its possible mechanisms.
- To recognize potential adverse events of amiodarone-induced QT prolongation.

PAST MEDICAL HISTORY

The patient had a past medical history of remote cerebral vascular accident with full recovery, persistent AF, and hyperlipidemia. A week before ED presentation, she underwent an AF ablation. After ablation, the patient redeveloped AF. Therefore, metoprolol was discontinued, amiodarone was initiated, and she was recardioverted. She was discharged from the hospital on 400 mg amiodarone 3 times daily for 1 week, then 400 mg twice daily for 1 week, followed by 200 mg daily. Other medications on discharge included 5 mg apixaban orally twice daily, 10 mg rosuvastatin orally daily, and 40 mg pantoprazole orally twice daily. Of note, the patient had an implantable loop recorder for stroke by means of which AF was subsequently diagnosed.

From the [a]Division of Biology and Medicine, Warren Alpert Medical School of Brown University, Providence, Rhode Island, USA; and the [b]Division of Cardiovascular Medicine, Arrhythmia Services Section, Rhode Island Hospital, Warren Alpert Medical School of Brown University, Providence, Rhode Island, USA.

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 December 6, 2020; revised manuscript received March 31, 2021, accepted April 29, 2021.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2021.04.022

DIFFERENTIAL DIAGNOSIS

The striking finding on presentation was a very prolonged QTc on ECG. The etiology for a prolonged QT interval includes ischemia, heart failure, channelopathies, medication, and electrolyte imbalance.

INVESTIGATIONS

Baseline ECG 1 week before showed sinus rhythm at 78 beats/min with a QTc of 456 ms (**Figure 2**). Interrogation of the implantable loop recorder revealed an episode of ventricular fibrillation (VF) for 2 min 13 s (**Figure 3**) during the syncopal event, followed by spontaneous conversion to sinus rhythm. The VF was triggered by a premature ventricular contraction (PVC) during which the QTc was prolonged. Unfortunately, no 12-lead ECG was available at the time of the event to localize the PVC. Echocardiography 1 month before and transesophageal echocardiography a week before the event revealed normal left ventricular size and systolic function, normal right ventricular function, a dilated left atrium, and mild mitral insufficiency. She underwent coronary angiography after the event, which showed minimal coronary artery disease and minimally elevated left ventricular end-diastolic pressure at 15 mm Hg. These findings along with her history are suggestive of PVC-induced VF in the setting of amiodarone-induced QT prolongation.

MANAGEMENT

Amiodarone was discontinued, and the QTc decreased from 650 ms to 468 ms during the hospitalization. An implantable cardioverter-defibrillator was recommended to avoid possible future episodes of VF.

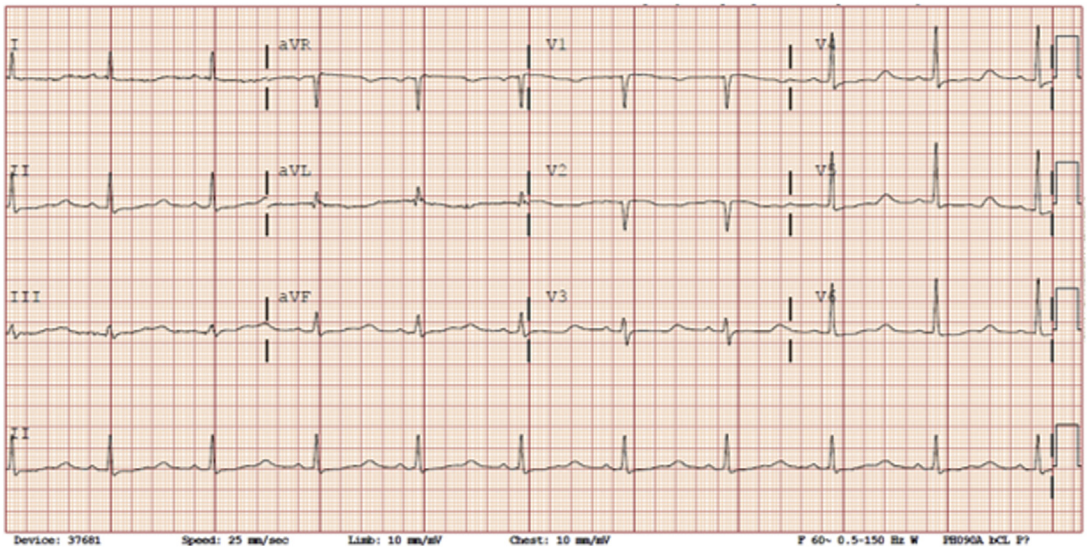
DISCUSSION

In almost all cases, defibrillation is required to terminate VF. Although spontaneous termination of VF has been previously reported in the literature, it is rare. Most of these cases describe organization of VF to a more stable ventricular tachycardia before spontaneous termination (1-3). The unique aspect of the present case is the lack of clear organization before spontaneous termination. The precise mechanism of spontaneous termination of VF is unclear, and there are various theories. In most cases, organization of ventricular rhythm is necessary. This is achieved by confinement of VF to a small region through either an anatomic or a functional boundary,

ABBREVIATIONS AND ACRONYMS

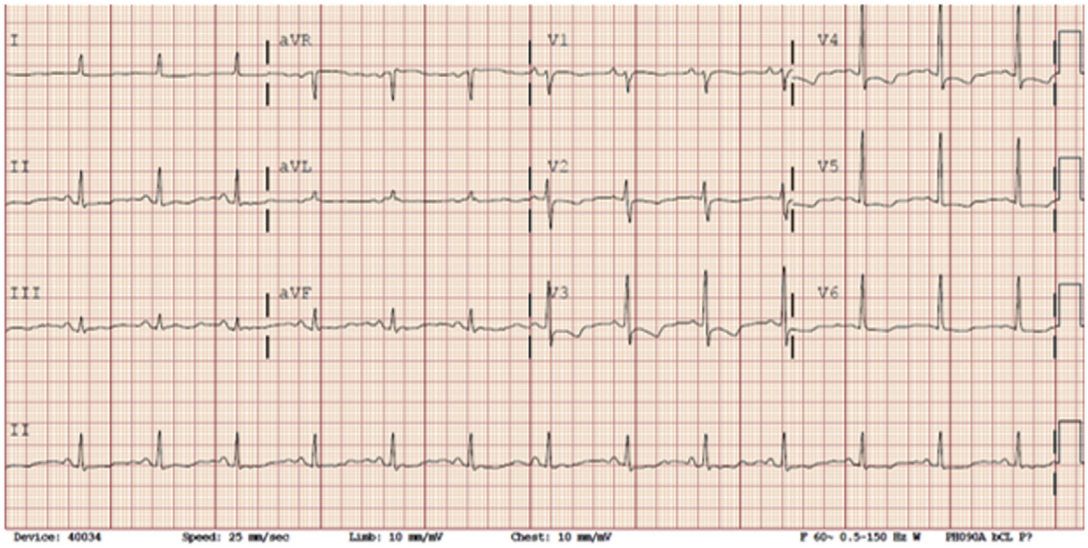
AF = atrial fibrillation
ECG = electrocardiography
ED = emergency department
PVC = premature ventricular contraction
VF = ventricular fibrillation

FIGURE 1 Electrocardiogram on Admission After Syncopal Event



JACC: CASE REPORTS, VOL. 3, NO. 8, 2021
JULY 2021:1108–13

FIGURE 2 Electrocardiogram 1 Week Before Syncopal Event



which then facilitates organization of ventricular rhythm. In certain nonhuman animal models, high levels of cardiac catecholamines can increase cardiac myocyte intercellular coordination. This phenomenon results in organization of rhythm and eventual conversion to sinus rhythm (4). In this case, the mechanism of spontaneous VF termination is likely due to reduced myocardial excitability secondary to global cardiac ischemia during a prolonged VF episode in combination with collision of all meandering spiral waves (5).

Given that the VF event occurred within 1 week following amiodarone loading, amiodarone was likely responsible for QT prolongation, although underlying genetic susceptibility (concealed form of long QT syndrome) and hypokalemia may also have played a role. Pantoprazole is also known to prolong QT, but in this case the QT interval shortened with discontinuation of amiodarone alone.

Amiodarone is used relatively frequently in cardiology for rhythm control; however, in approximately 1% of the cases, amiodarone-induced QT prolongation resulting in torsades de pointes can occur (6). Some centers choose to schedule an ECG after amiodarone loading to evaluate the QT interval and potential presence of U waves. This is uncommon in our center, but in patients with a borderline prolonged QT interval before amiodarone loading, it could be considered by clinicians.

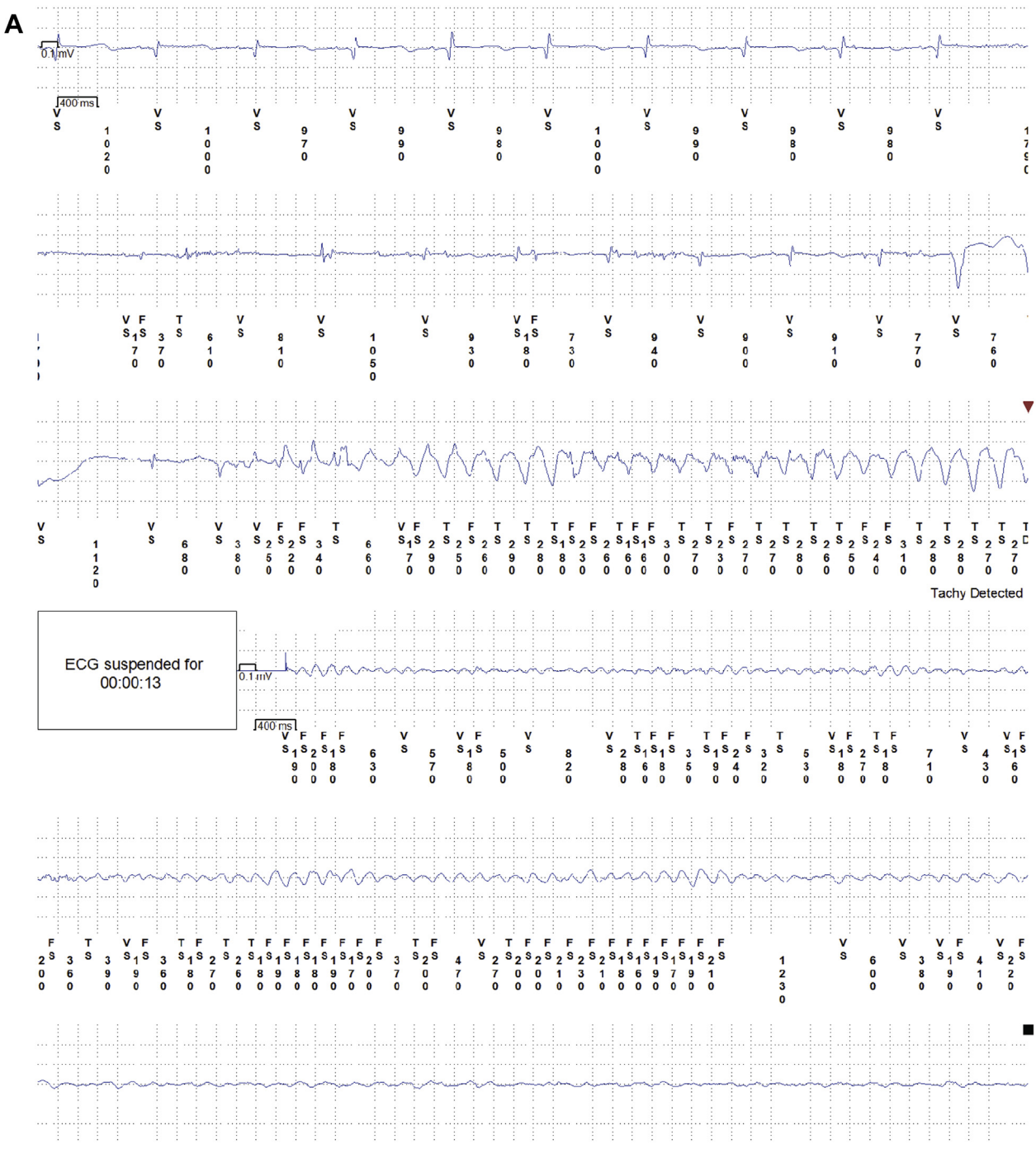
FOLLOW-UP

After discharge from the hospital, the patient had no more episodes of VF.

CONCLUSIONS

Spontaneous termination of VF is rare. This case demonstrates that organization may not be necessary before spontaneous VF termination even after a prolonged episode. In addition, amiodarone use could result in fatal arrhythmia. Careful patient selection and monitoring are imperative.

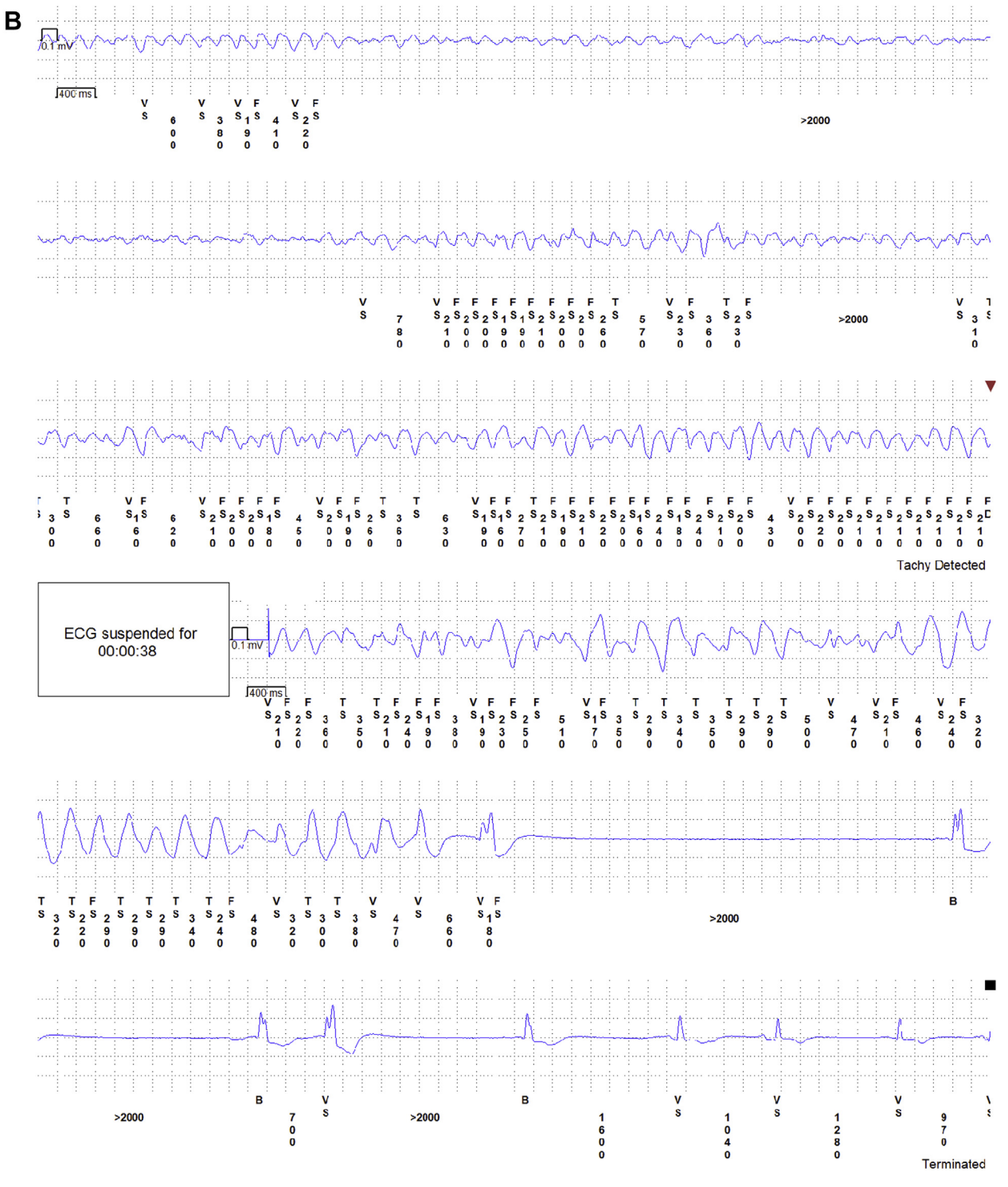
FIGURE 3 **ECG During Syncopal Event**



Notice baseline prolonged QTc at almost 600 ms. A premature ventricular contraction after a long-short sequence triggered ventricular fibrillation. The ventricular fibrillatory episode lasted 2 min 13 s before it spontaneously converted to sinus rhythm. ECG = electrocardiogram.

Continued on the next page

FIGURE 3 Continued



FUNDING SUPPORT AND AUTHOR DISCLOSURES

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

ADDRESS FOR CORRESPONDENCE: Mr. Braden Vogt, Division of Biology and Medicine, Warren Alpert Medical School, 70 Kennedy Plaza, Unit 2, Providence, Rhode Island 02903, USA. E-mail: braden.r.vogt@gmail.com. Twitter: @Braden_vogt.

REFERENCES

1. Konstantino Y, Morello A, Zimetbaum PJ, et al. Self-terminated ventricular fibrillation and recurrent syncope. Am J Cardiol 2011;107:638–40.

2. Calabrò MP, Luzza F, Carerj S, et al. Spontaneous termination of ventricular fibrillation. Heart 2004;90:133.

3. Konishi S, Minamiguchi H, Masuda M, et al. Self-terminated long-lasting ventricular fibrillation: what is the mechanism? J Cardiol Cases 2014;10:136–9.

4. Reisin L, Blayer Y, Manoach M. Spontaneous ventricular defibrillation. Br Heart J 1993;70:590–1.

5. Chen PS, Wu TJ, Ting CT, et al. A tale of two fibrillations. Circulation 2003;108:2298–303.

6. Hohnloser SH, Klingenheben T, Singh BN. Amiodarone-associated proarrhythmic effects. A review with special reference to torsades de pointes tachycardia. Ann Intern Med 1994;121:529–35.

KEY WORDS amiodarone, atrial fibrillation, premature ventricular contraction, ventricular fibrillation