Texas Heart Institute Journal

Focus on ECGs: Case #26

Cardiac Arrest in the Presence of Alcohol Abuse, Hypokalemia, and Possible Brugada Syndrome

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A 44-year-old man was brought to our emergency department after having a ventricular fibrillation cardiac arrest and initial emergency defibrillation. He had elevated blood alcohol levels (318 mg/dL), hypokalemia (potassium level, 2.7 mEq/L), and a mildly elevated cardiac troponin I level (0.2 ng/mL). A coronary angiogram showed no coronary artery disease or other abnormality. An electrocardiogram (ECG) showed a QT interval of 416 ms, a prolonged corrected QT interval (QTc) of 602 ms, and coved ST-segment elevations in leads V_1 and V_2 (Fig. 1).

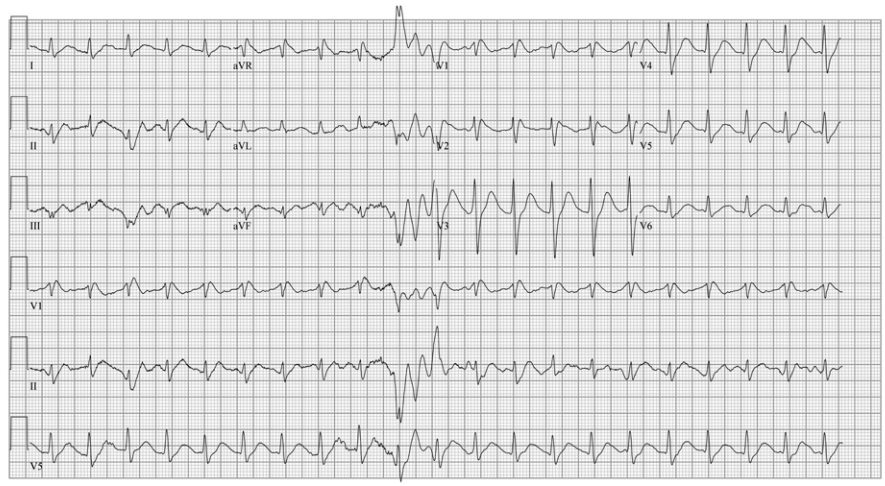

Fig. 1

Which initial action is most appropriate?

A) Place an implantable cardioverter-defibrillator (ICD) for secondary prevention with no further investigation

B) Prescribe oral nadolol and perform genetic testing for long QT syndrome

C) Prescribe oral nadolol and place an ICD, with no further investigation

D) Obtain cardiac magnetic resonance images

E) Monitor patient on telemetry while investigating nonreversible causes of sudden cardiac death

See next page for the answer.

Citation: Mogalapalli A, Kumar S, Ghani A, Mar PL. Cardiac arrest in the presence of alcohol abuse, hypokalemia, and possible Brugada syndrome. Tex Heart Inst J 2021;48(4):e207281. doi: 10.14503/THIJ-20-7281

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E) Monitor patient on telemetry while investigating nonreversible causes of sudden cardiac death

The prolonged QTc seen on the presenting ECG resolved when the patient's potassium levels normalized, indicating that the patient had acquired long QT syndrome, which would seemingly contraindicate ICD implantation.[1] However, the pattern in the precordial leads suggested Brugada syndrome (BrS), which also resolved (QT interval, 420 ms; QTc, 446 ms) after potassium levels normalized (Fig. 2).

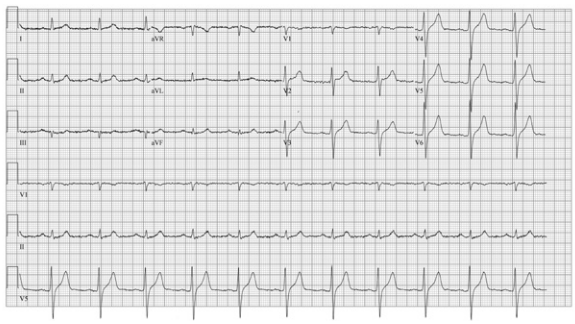

Fig. 2

Procainamide infusion (10 mg/kg for 30 min) induced a type 1 BrS pattern[2]: a widened QRS interval, pseudo-right bundle branch block, and coved ST-segment elevation in leads V_1 and V_2, followed by a negative T wave (Fig. 3). The QT interval was 386 ms, and the QTc, 495 ms.

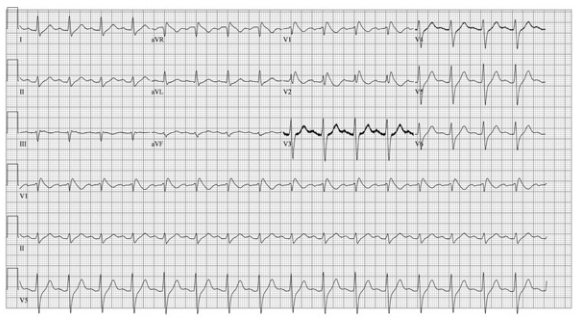

Fig. 3

The patient's recent cardiac arrest and these findings put him at high risk of recurrent ventricular fibrillation, so we placed an ICD. Three months after ICD placement and while the device was charging for defibrillation, the patient had another event that was aborted by antitachycardia pacing.

Placing an ICD without further intervention would have been inappropriate because the patient had a familial condition that warranted screening of first-degree relatives. Nadolol was not indicated because he had acquired (not congenital) long QT syndrome. Cardiac magnetic resonance imaging was reasonable but not the most appropriate response. The patient had just experienced a cardiac arrest with ECG findings suggesting BrS and therefore needed monitoring in the intensive care unit with further workup.

This case highlights the importance of seeking nonreversible causes of cardiac arrest even when reversible causes are obvious.

Published: 15 October 2021

Section Editors: Yochai Birnbaum, MD, FACC; Mohammad Saeed, MD, FACC

References

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