Melinda Sharon, MPH; Brandon Wilson, MD; Bradley End, MD; Clara Kraft, MD; Joseph Minardi, MD

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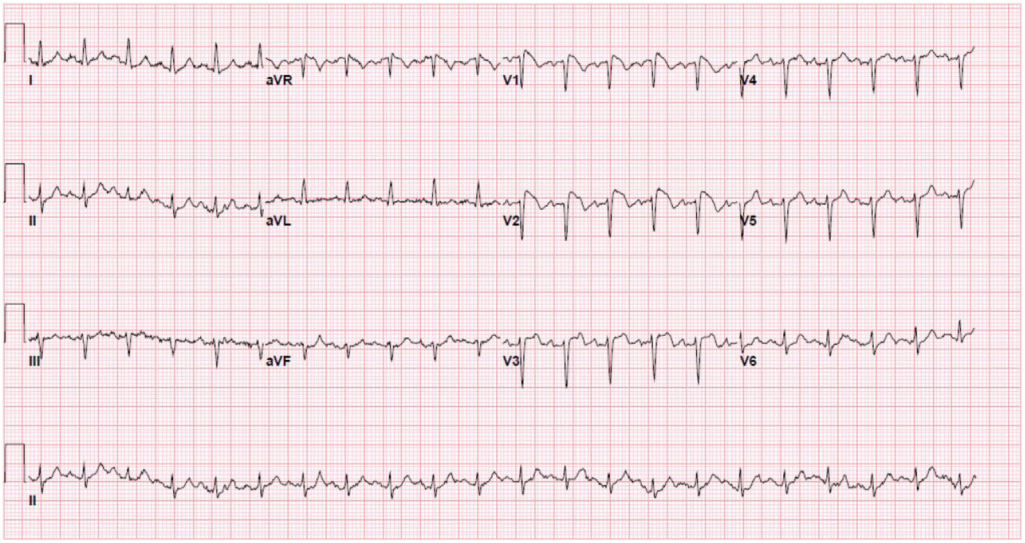

Figure 1. Initial ECG in the ED.

[Ann Emerg Med. 2019;74:782-785.]

A 49-year-old man presented to our emergency department (ED) with right-sided chest and abdominal pain. The emergency physician was called to the bedside during the initial nursing evaluation because his automated ECG interpretation read "STEMI" (ST-segment elevation myocardial infarction) (Figure 1).

The patient was in obvious distress because of pain and dyspnea. He was hypertensive, tachycardic, tachypneic, and hypoxic. He reported having right-sided flank pain for 1 to 2 days that was now also involving the right side of his chest. The pain was pleuritic and had been worsening during the past 6 hours. Medical history included type 2 diabetes and hypertension. He had a coronary angiogram revealing minimal coronary artery disease 6 months before.

What are the critical findings on this ECG? Should this patient be taken for emergency cardiac catheterization? If cardiac catheterization is not immediately available, should systemic thrombolytics be administered?

For the diagnosis and teaching points, see page 783.
To view the entire collection of ECG of the Month, visit www.annemergmed.com

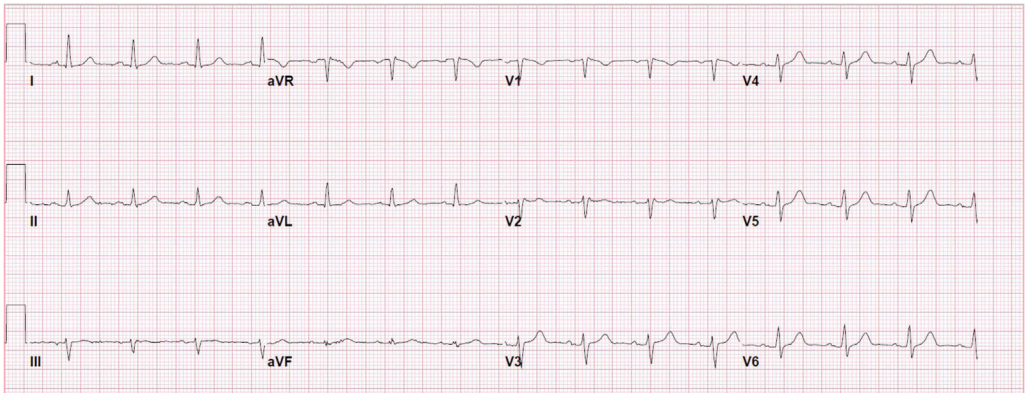

Figure 2. Repeated ECG the next day, after defervescence.

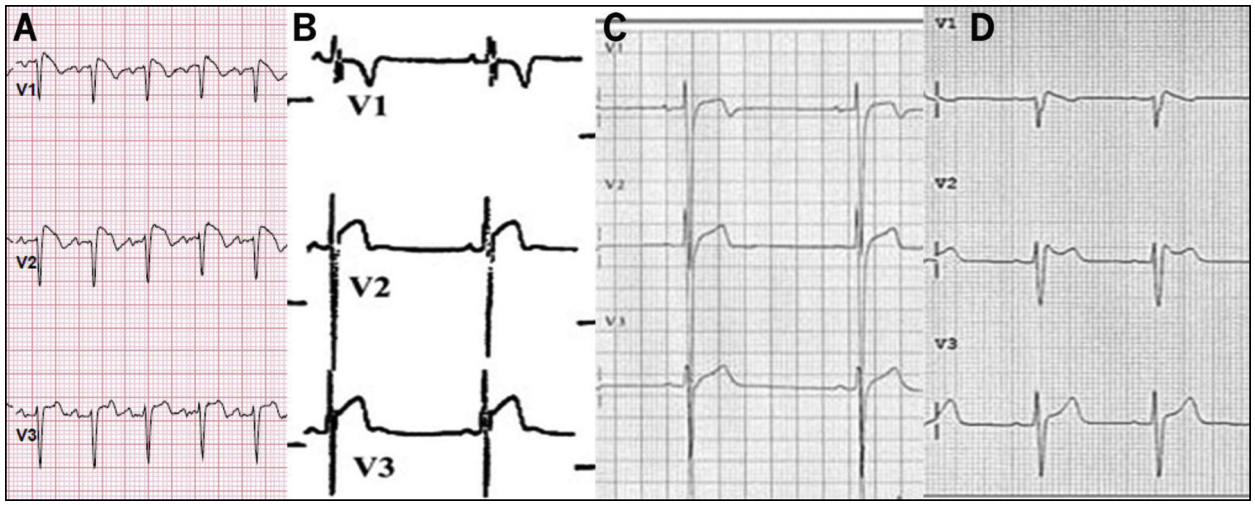

Figure 3. *A*, Brugada's ECG pattern, type I. A portion of our patient's ECG with the diagnostic Brugada's type I pattern that includes ST-segment elevation, starting immediately at the J point and then downsloping into the T wave without an isoelectric interval. *B*, Normal athlete's heart. There is ST-segment elevation in V2 and V3, but the ST segment slopes upward toward the T wave.[3] *C*, Early repolarization. There is mild ST-segment elevation, but the ST segment slopes upward toward the T wave.[4] *D*, Incomplete right bundle branch block. There is slight ST-segment elevation and borderline right ventricular conduction delay. There is slight downsloping of the ST segment in V1 and a saddle-back appearance of the ST segment in V2.[4]

ECG OF THE MONTH

(continued from p. 782)

DIAGNOSIS:

Interpretation

The ECG demonstrated sinus tachycardia with an rSR′ pattern and downsloping ST-segment elevation in V1 through V3. His previous ECG result 5 months before was normal.

CLINICAL COURSE

Initial evaluation was focused on pulmonary embolism, aortic dissection, and acute coronary syndrome. Hollow viscous perforation or other intra-abdominal process was also considered. Bedside echocardiogram revealed no pericardial effusion, a normal-sized right ventricle, a normal-appearing aortic root, and vigorous left ventricle without obvious wall-motion abnormalities. ST-segment elevation myocardial infarction alert was not

activated. Supplemental oxygen, intravenous fluids, and analgesics were administered. He had a temperature of 100.9°F.

He was taken for computed tomography angiography of the chest, abdomen, and pelvis, which revealed a right lower lobe consolidation consistent with pneumonia. Broad-spectrum antibiotics and antipyretics were administered. A Brugada's ECG pattern unmasked by febrile illness was suspected. Cardiology was consulted. Initial troponin-level results were normal. A repeated ECG was obtained that appeared similar. Although acute coronary syndrome was thought less likely, given his acute severe pain with new ST-segment elevation, known risk factors, and previously documented minor coronary artery disease, coronary angiography was believed to be warranted; but the result was negative, confirming the suspected Brugada's pattern as the cause for the acute ST-segment elevation. The patient was admitted to the ICU and treated for his pneumonia, with good results. A repeated ECG performed the next day when the patient was afebrile showed reversion of the Brugada's findings to a type III (nondiagnostic) pattern, as shown in Figure 2. He was discharged on hospital day 7.

DISCUSSION

Brugada's syndrome is an autosomal-dominant genetic disorder that increases an individual's risk for sudden cardiac death and ventricular dysrhythmias, which was first described by the Brugada brothers in 1992.[1] Current estimates of the disease in the literature are between 1 and 5 persons per 10,000.[1,2] The classic Brugada's ECG pattern includes characteristic cove-shaped ST-segment elevation that begins at the J point and then slopes downward into the T wave, with little to no isoelectric interval between the J point and the T wave. These findings occur in leads V1 through V3, often associated with a complete or incomplete right bundle branch block appearance, and are considered type I patterns. Type II and III patterns have lesser degrees or different contours of ST-segment elevation and are not considered diagnostic of the disease.[1] Brugada's syndrome is diagnosed when a patient has the Brugada's ECG pattern and documented ventricular tachydysrhythmia or history consistent with ventricular tachydysrhythmia, such as syncope or sudden cardiac death. Some findings can mimic the appearance of the Brugada's pattern, such as athlete's heart, early repolarization, and right bundle branch blocks.[1,3] Most mimics, on close inspection, do not have the characteristic ST-segment elevation beginning at the J point, which is followed by rapid, steep downsloping toward the T wave (Figure 3).[3,4] The patient in this case displayed a Brugada's type I pattern during his acute febrile illness (Figures 1 and 3*A*) and reverted to a type III pattern on defervescence (Figure 2).

Previous literature has demonstrated that the loss of function of a sodium-channel current, as observed in Brugada's syndrome, is accentuated at higher temperatures and thus is more likely to trigger ventricular arrhythmias.[1,2,5] Additionally, it has been shown that the prevalence of type I Brugada's pattern is 20 times higher among patients presenting with fever than in afebrile patients.[2] Our patient presented with a fever of 100.9°F and continued to have persistent bouts of fever during his inpatient stay. In addition to exposure by fever, Brugada's pattern may be unmasked by antidysrhythmics and other medications, electrolyte abnormalities, or other physiologic disruptions.[1-3] Recommendations include reducing fever in patients with this syndrome and avoiding excessive alcohol intake, which was communicated to our patient on discharge. For symptomatic patients (including symptoms or findings of ventricular tachydysrhythmia), or in cases in which those recommendations are ineffective, further electrophysiologic testing may be required to determine the need for implantable cardiac defibrillator.[1,3]

If a Brugada's pattern is identified on ECG, a detailed family history should be obtained to elicit sudden cardiac death, drowning, or other evidence of tachydysrhythmia. It is estimated that a family history is present for 20% to 30% of patients.[1] Additionally, the patient should be asked about syncopal episodes or other dysrhythmic symptoms. If there are symptoms of tachydysrhythmia, the patient should be referred and possibly admitted for potential implantable cardiac defibrillator placement, depending on the particular scenario.[1,3] If there is no suggestive family or patient history, education and general cardiology referral are adequate.

Emergency physicians should recognize the Brugada's pattern on 12-lead ECG and be aware of its physiologic triggers and implications in patient management decisions.

PEARLS

Brugada's type I ECG pattern is demonstrated with immediate ST-segment elevation beginning at the J point, which slopes downward toward the T wave, with minimal to no isoelectric interval in V1 to V3, usually with some component of right ventricular conduction delay.

Brugada's pattern may cause acute ST-segment elevation, mimicking ST-segment elevation myocardial infarction, making interpretation challenging in some scenarios.

Brugada's pattern may be unmasked by fever or other metabolic or physiologic disruptions.

If the Brugada's pattern is identified, a careful patient and family history is required to inform the decision for implantable cardiac defibrillator referral.

Author affiliations: From the Department of Emergency Medicine, West Virginia University School of Medicine, Morgantown, WV.

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