

# Brugada Pattern in an Afebrile Patient with COVID-19

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## Learning Objectives

1. This is a classic arrhythmia that has been known to be exacerbated by fever. We have seen brugada pattern with a number of COVID-19 cases and do not know the long-term cardiac risk factors in these patients.
2. These patients should be closely monitored on telemetry or in the intensive care unit if there is any respiratory compromise.
3. There is no current indication to for any invasive interventions if there is no family history of channelopathies, syncope or sudden cardiac death.

## Case Presentation

A 44-year-old Hispanic man with no significant medical history presented with generalized fatigue, headaches, productive cough of whitish to yellow sputum, myalgias, abdominal pain and non-bloody diarrhea with fevers (T.max of 102.6°F) for six days prior to admission and worsening shortness of breath for two days. He reported getting a COVID-19 test one week prior to admission at an urgent care center which was positive. A chest x-ray at that time showed clear lungs without any acute cardiopulmonary abnormalities. He denied any significant family history including sudden cardiac death, arrhythmias, myocardial infarction or syncope. He did not recall any exposure to coronavirus and had no sick contacts. He lives at home with his wife and children. He works in maintenance, denies any alcohol, tobacco and illicit drug use. He denied any lightheadedness, dizziness, chest pain/palpitations, nausea, vomiting, loss of taste and smell.

### Physical Exam:

Vital signs- afebrile (98.2°F); BP, 136/86 mm Hg; heart rate, 110 beats/min; respiratory rate, 24 breaths per minute and oxygen saturation 95% on room air. Pupils were equal, round, and reactive to light with normal conjunctiva and no scleral icterus. Lungs with decreased breath sounds; no wheezing, rales, or rhonchi. Cardiovascular examination revealed increased rate and normal rhythm, no murmurs. The abdomen was diffusely tender to palpation, but not distended and with normal bowel sounds and no hepatosplenomegaly. The bladder was not distended, and there was no costovertebral tenderness. There was full range of motion in all joints without swelling, tenderness, or edema. There were no focal neurologic deficits or meningeal signs.

### Labs

Laboratory data demonstrated mild hyponatremia 134 mmol/L (135-145 mmol/L), and normal potassium 3.9 mmol/L (3.5-5.0 mmol/L), magnesium 2.0 mmol/L (1.5-2.0 mmol/L), and brain natriuretic peptide level 20 pg/mL (0-120 pg/mL). Liver enzymes tests showed AST 92 units/L (10-45 units/L), ALT 120 units/L (10-45 units/L), normal alkaline phosphatase 84 units/L (40-125 units/L). His ferritin was normal at 234 ng/mL (20-450 ng/mL) however his other inflammatory markers were elevated; sedimentation rate 49mm/hr (0-15 mm), lactate dehydrogenase 782 units/L (300-600 units/L), D-dimer (0.48 mcg/mL FEq unit (0.2-0.38 mcg/mL FEq unit) and procalcitonin level 0.113 ng/mL (0.00-0.099 ng/mL). The remaining laboratory values were unremarkable, and the troponin level was normal.

He tested positive for COVID via polymerase chain reaction (PCR).

## Imaging:

The electrocardiogram (ECG) showed a brugada-type 1 pattern “coved” ST-segment elevation that concaves down with inverted T waves in V1-V2 leads with no reciprocal changes (Figure 1). Portable chest radiograph demonstrated moderate pulmonary interstitial and alveolar edema with enlarged heart (Figure 2).

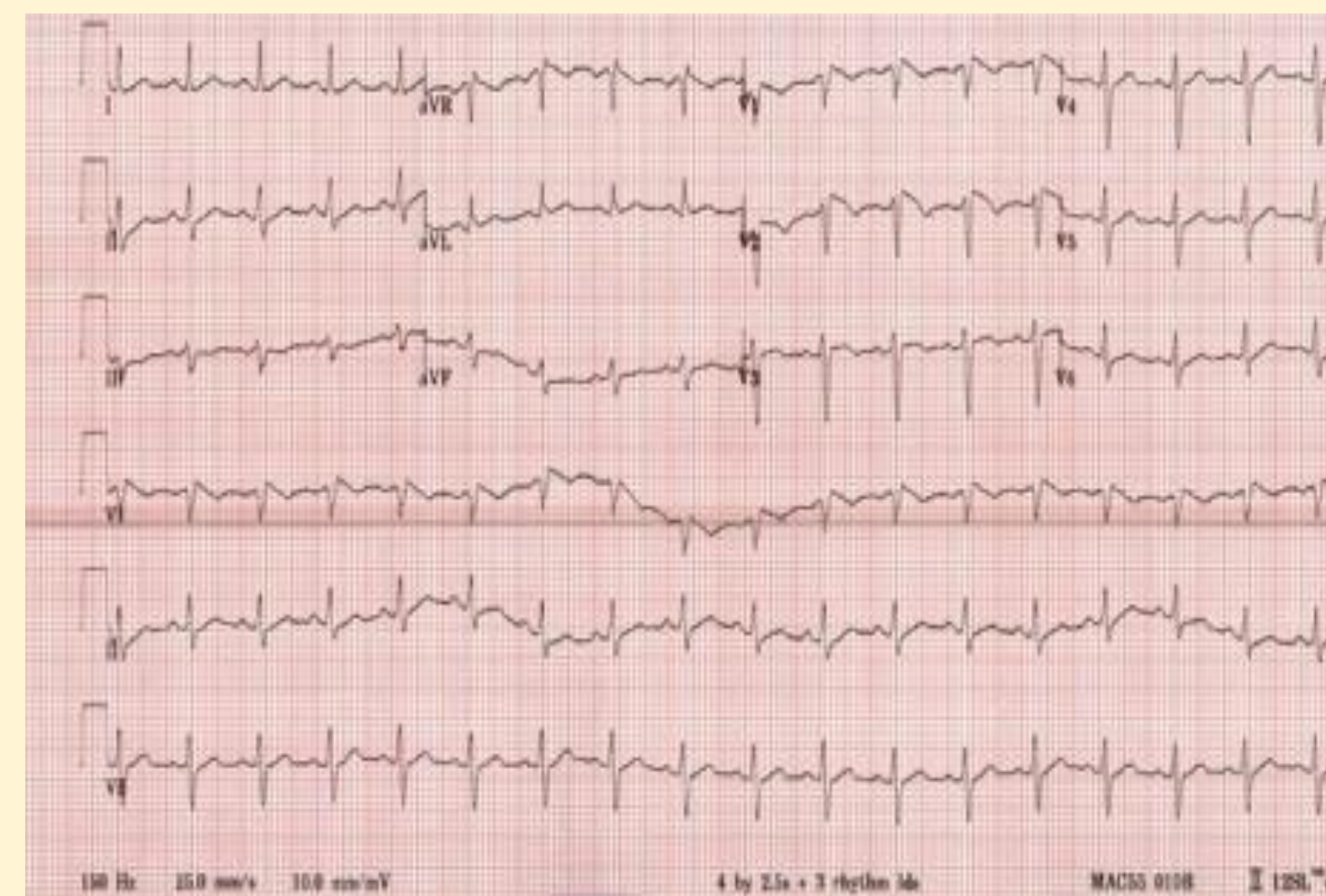


Figure 1: Initial WKG showing brugada type 1 pattern “coved ST elevation” with inverted T waves in V1-V2



Figure 2 – Chest x-ray significant for pulmonary edema.

## Treatment:

The patient was managed conservatively with supportive care: antipyretic (Tylenol) and benzonatate for symptomatic relief of his cough. He was monitored overnight, remained afebrile and did not require supplemental oxygen therapy, so there was no need to initiate remdesivir or dexamethasone for treatment of his COVID. He was discharged home after 48 hours of monitoring with follow-up with cardiology in three weeks given his low adverse cardiac risk. A repeat EKG was done in cardiology clinic which showed normal sinus rhythm, without ST-T elevations or signs of ischemic and QTc of 413ms (400-440 ms)

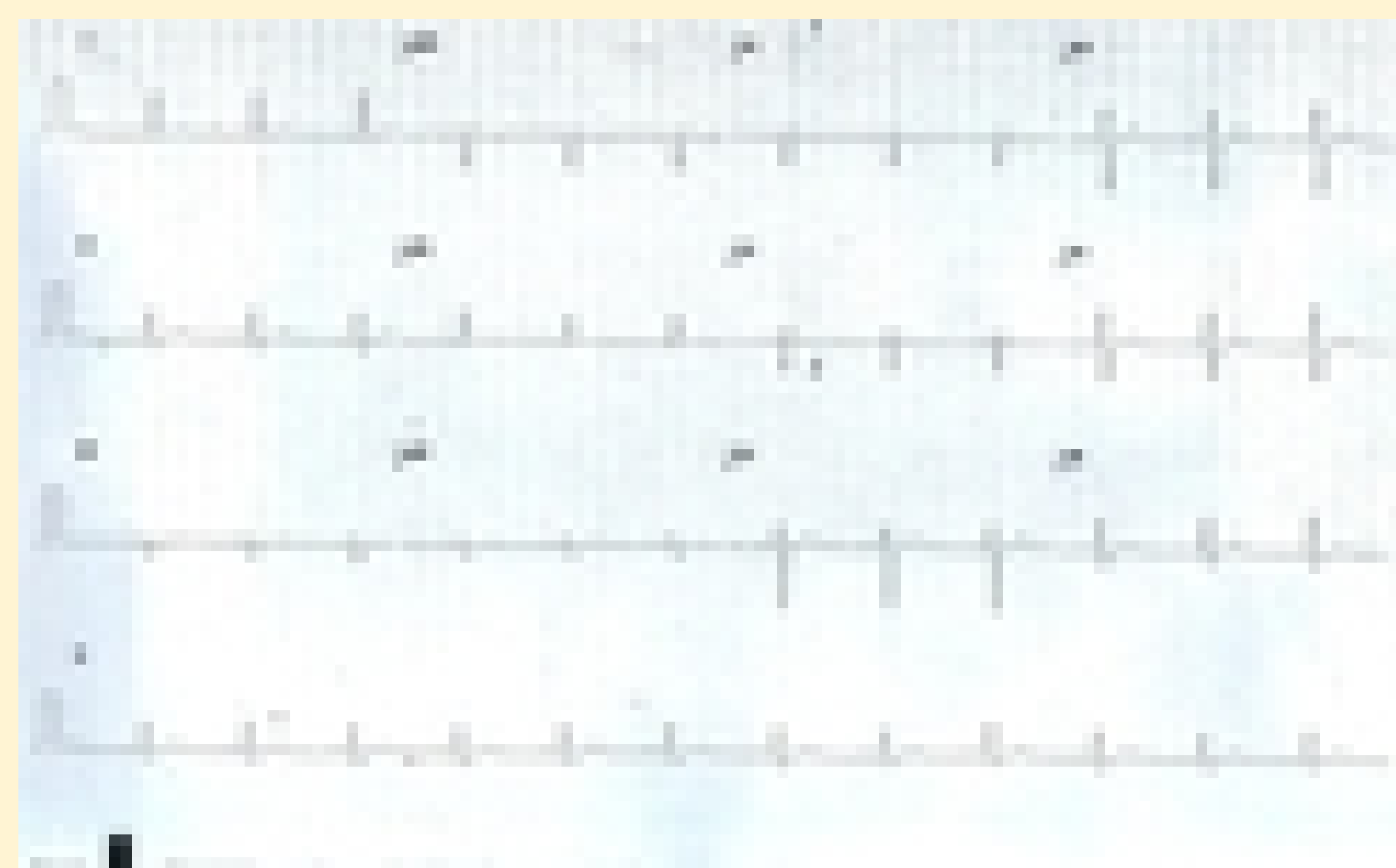


Figure 3: Repeat EKG in follow up demonstrates normal sinus rhythm without any abnormalities.

## Discussion

Brugada syndrome (BrS) is an inherited cardiac channelopathy that compromises ion channel integrity and leads to transmembrane current alterations in cardiomyocytes.

The distinction between BrS and Brugada pattern (asymptomatic but with characteristic ECG findings) has important implications for patient management. Placement of an implantable cardioverter-defibrillator is indicated in BrS patients who have survived sudden cardiac arrest or have sustained ventricular tachycardia (VT), but individuals with BrS pattern and no signs, symptoms, or family history of BrS do not benefit from a defibrillator. Despite being asymptomatic, those who have Brugada pattern on ECG carry a risk of VF up to 12% at 10 years, so the clinical threshold to perform an ECG should remain low if there is any suspicion of BrS. BrS is often elicited from various inciting stressors, such as fever, alcohol, and certain medications, including those commonly used in critical care scenarios. Fever is a particularly well described variable in the etiology and prognosis of BrS, with higher temperatures increasing the risk for cardiac arrest in BrS. It is known that the biochemical capacity of cardiac sodium channels declines at higher temperatures, and, importantly, fever-exacerbated BrS appears to not be mutation-specific. COVID-19 disease is typified by the presence of fever, among a constellation of other constitutional and/or pulmonary symptoms, and thus can be an important risk factor in unmasking BrS. The severe inflammatory response frequently seen with COVID-19 can lead to fever-induced arrhythmias and, to some extent, dictate the prognostic outcomes. In our case it is notable that the patient was afebrile during the hospital stay. A prior case report had demonstrated unmasking of Brugada pattern in a febrile patient with COVID-19. To our knowledge, our case is unique in that the patient remained afebrile suggesting another mechanism for unmasking the Brugada pattern perhaps related to the inflammatory response. Health care providers should be aware of this phenomenon and prioritize treatment of fever and avoidance of drugs that could provoke BrS, such as sodium channel blockers or tricyclic antidepressants, if characteristic ECG changes are recognized in the setting of COVID-19 infection.

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