Challenges in Clinical Electrocardiography

# An Abnormal ECG Finding in a Patient With COVID-19

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**A patient in their 50s** was admitted to the intensive care unit with severe COVID-19 pneumonia. Their medical history was notable for hypertension and diabetes for 10 years. At presentation, the patient's oxygen saturation was 87% on room air and improved to 94% with oxygen supplementation. They were treated with steroids and parenteral anticoagulation. However, there was progressive deterioration that required mechanical ventilation and inotropic support by the seventh day of hospitalization. A 12-lead electrocardiogram (ECG) was performed because a ST-segment abnormality was observed on telemetry (**Figure**).

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**Questions:** What is the abnormality in the ECG? What is the immediate concern regarding management?

## Interpretation

The ECG in the Figure shows sinus bradycardia with a heart rate of 50 beats per minute. There was a distinctive end-QRS notch followed by J point elevation in all leads except $V_1$ and $V_2$, eponymously known as *Osborn waves*.[1] The amplitude was highest in the lateral precordial leads. The QTc interval was prolonged at 474 milliseconds. The immediate concern was hypothermia.

## Clinical Course

The patient's core body temperature was normal at 98.8 °F; thus, other causes of Osborn waves were considered. Nonhypothermic causes of Osborn waves include hypercalcemia, hypothyroidism, diabetic ketoacidosis, acute myocardial ischemia, myocarditis, and Takotsubo cardiomyopathy.[2] The prolonged QTc interval made hypercalcemia unlikely. Nevertheless, an evaluation of serum electrolytes revealed normal levels of serum calcium, magnesium, and potassium. Thyroid function test results and blood glucose levels were normal. A bedside transthoracic echocardiogram showed normal ventricular systolic function. Troponin levels were slightly elevated at 0.13 ng/mL (normal, <0.01 ng/mL [to convert to µg/L, multiply by 1]) but did not display any rising titers. Thus, severe myocarditis or stress-induced cardiomyopathy, both of which have been known to be associated with nonhypothermic Osborn waves, were excluded. The patient died of COVID-19 pneumonia 5 days later.

## Discussion

Osborn waves were first described as J point elevation in the QRS complex in experimentally induced severe hypothermia in dogs.[1] Although classically associated with hypothermia, Osborn waves are not pathognomonic of hypothermia. They may also be seen in several nonhypothermic conditions, including severe hypercalcemia, central nervous system injury, acute myocardial ischemia, Takotsubo cardiomyopathy, severe myocarditis, early repolarization syndromes, cocaine use, haloperidol overdose, and resuscitation from cardiac arrest.[2] Osborn waves are often seen in patients with critical illness and is regarded as a predictor of ventricular fibrillation and sudden cardiac death.

There is no consensus on definitive physiological cause for the J point elevation. It is believed to be due to a difference in electrophysiologic properties of ventricular epicardium and endocardium. Transmural voltage gradient associated with heterogenous expression of 4-aminopyridine sensitive transient outward current ($I_{to}$) has been proposed to be the driving factor for development of Osborn waves.[2] Ventricular activation from endocardium to epicardium, with the latter being activated last, is a prerequisite for development of J waves.[3]

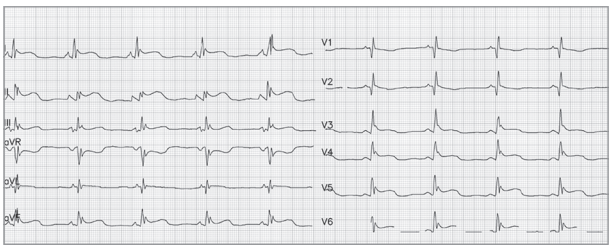
Cardiac injury is not uncommon in COVID-19 infection. Myocardial injury is more pronounced in severe infection.[4] Myocardial damage appears to be associated with increased cardiometabolic demand that is associated with the systemic infection and ongoing hypoxia caused by severe pneumonia. The role of proinflammatory cytokines is considered paramount to the development of cardiac injury.

Osborn waves have been reported in hospitalized patients with COVID-19–associated pneumonia in the absence of hypothermia.[5] The mechanisms proposed in these reports include myocardial and/or conduction system damage. Owing to disturbed electrolyte current in the cardiomyocytes on a background of oxidative stress, it has been proposed as a predictor of future fatal cardiac arrhythmias.[6] Presence of Osborn wave was shown to be predictive of short-term mortality in patients with COVID-19–associated pneumonia in a small retrospective study.[5]

## Take-Home Points

- Repolarization abnormalities may be encountered in patients with severe COVID-19 infection.
- Osborn waves, typical of hypothermia, have rarely been described in COVID-19 infection and are associated with poor prognosis.
- Hypothermia and dyselectrolytemias need to be excluded first in patients with Osborn waves.

Figure. Electrocardiogram (ECG) Findings




A 12-lead ECG showing sinus bradycardia with heart rate of 50 beats per minute with diffuse J point elevation and a prolonged QTc interval.

## ARTICLE INFORMATION

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