CLINICAL VIGNETTE

Hypothermic Osborn Waves Mimicking Acute ST-Segment Elevation Myocardial Infarction

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Abstract

Osborn waves (or J waves) are an electrocardiogram finding seen with significant hypothermia. The morphology is described as a deflection with a dome shape in the same direction as the R wave immediately following the QRS complex. They are typically seen in the inferior and precordial V2-V6 leads. We present a hypothermic patient with profound Osborn waves mimicking an acute anterior ST-segment elevation myocardial infarction (STEMI). It is important for clinicians to recognize the electrocardiographic Osborn wave pattern as a STEMI mimic as it greatly affects patient management.

Keywords: Osborn wave, STEMI mimic, arrythmia, myocardial infarction, electrocardiogram

Introduction

Osborn waves (or J waves) are an electrocardiogram (ECG) finding seen with significant hypothermia. They are present in approximately 80% of ECGs in patients with body temperature under 30 degrees Celsius (°C). The morphology is described as a deflection with a dome shape in the same direction as the R wave immediately following the QRS complex. They are typically seen in the inferior and precordial V2-V6 leads. We present a hypothermic patient demonstrating profound Osborn waves mimicking an acute anterior ST-segment elevation myocardial infarction (STEMI).

Case Summary

A 47-year-old male with type 2 diabetes, presented with altered mental status. He was hypothermic to 31.5°C (Figure 1), hypotensive with a systolic blood pressure of 70 mmHg, and in diabetic ketoacidosis (DKA). His initial ECG showed Osborn waves (Figure 2) which resolved with rewarming to 35.9°C (Figure 3). Initial hypothermia evaluation was unremarkable including a normal TSH of 0.751uIU/mL, random cortisol of 15.6 mcg/dL, blood glucose of 1517mg/dL, and negative blood cultures. The patient was residing in Southern California in early Fall during a heat wave, making environmental exposure unlikely. He was admitted to the intensive care unit for treatment, however shortly after admission he underwent PEA cardiac arrest likely secondary to metabolic derangements from DKA. The patient's temperature at the time of arrest was 35.8°C. Return of Spontaneous Circulation was achieved after three rounds of cardiopulmonary resuscitation. After intubation and therapeutic temperature management (TTM) was initiated. Approximately twenty hours later, while still on TTM, the patient again became hypothermic to 33.3°C. Repeat ECG showed recurrence of the Osborn waves (Figure 4), however this time they were more profound raising concern for anterior STEMI. The patient's troponin level at that time was 2.391 ng/mL, and he was still intubated so he could not report chest pain. TTM was discontinued, the patient's hypothermia improved to 37.6°C, and the Osborn waves resolved (Figure 5), lowering the concern for true acute coronary syndrome. The elevated troponin was attributed to demand ischemia secondary to his cardiac arrest. Over the next two weeks, the patient's metabolic derangements improved, he was extubated, and discharged home.

Discussion

Hypothermia affects the heart by causing dysfunction of the calcium, sodium, and potassium channels in the myocyte membrane. This channel dysfunction leads to numerous morphological ECG changes such as prolonged PR and TP intervals, widened QRS complexes, flattened T waves, and Osborn waves. Osborn waves (also known as J waves) are positive deflections at the J point (the point where the QRS complex meets the ST segment) and are thought to be due to specific dysfunction in the potassium channel. Altered intracellular potassium levels have been speculated to cause either a late depolarization or early repolarization in one area of the ventricle, which gets reflected as an upward deflection immediately after the QRS complex. This deflection is most prominent in the precordial leads V2-V6 and is negative in aVR and V1. Generally, the duration and amplitude of Osborn waves are inversely related to core temperature and re-warming causes them to resolve. This wave morphology has been reported in approximately 80% of hypothermic patients with a core body temperature less than 30°C.[1,2]

While Osborn waves are mostly observed in hypothermic patients, they can be seen in severe hypercalcemia, CNS injury, cocaine use, haloperidol overdose, cardiopulmonary arrest (specifically from oversedation) vasospastic angina, or idiopathic ventricular fibrillation.[3] Our patient's Osborn waves emerged only while he was hypothermic and resolved with re-

warming, making hypothermia the leading etiology. However, this patient did not have a clear etiology for his hypothermia, as common causes (sepsis, adrenal insufficiency, hypothyroidism, and hypoglycemia) were ruled out. Interestingly, it has been postulated that DKA can cause hypothermia due to the inability of sufficient glucose to enter the cell, leading to decreased metabolism and a lack of heat production.[4] Therefore, our patient's DKA may be a possible etiology of his hypothermia, and consequent, development of his Osborn waves.

There is conflicting evidence about the clinical significance of Osborn waves. While early studies initially reported Osborn waves were associated with higher mortality,[5,6] later multi-center studies showed no correlation between Osborn waves and the development of ventricular arrythmias.[3,7] In a clinical context, it is important for providers to recognize that Osborn waves are the physiologic consequence of hypothermia and do not represent a second disease process. We believe this is the first report of a hypothermic patient whose Osborn waves were so profound that they caused concern for STEMI. Osborn waves mimicking STEMI have been reported in patients with central nervous system disorders or severe hypercalcemia,[8] but never in hypothermia. Many other processes can also mimic the ECG tracings in STEMIs, including left ventricular hypertrophy, early repolarization patterns, hyperkalemia, hypercalcemia, Brugada syndrome, Wolf-Parkinson-White syndrome, and Taktsubo cardiomyopathy. It is important for providers to recognize these mimics, as their management greatly differs from that of STEMIs.

Figures

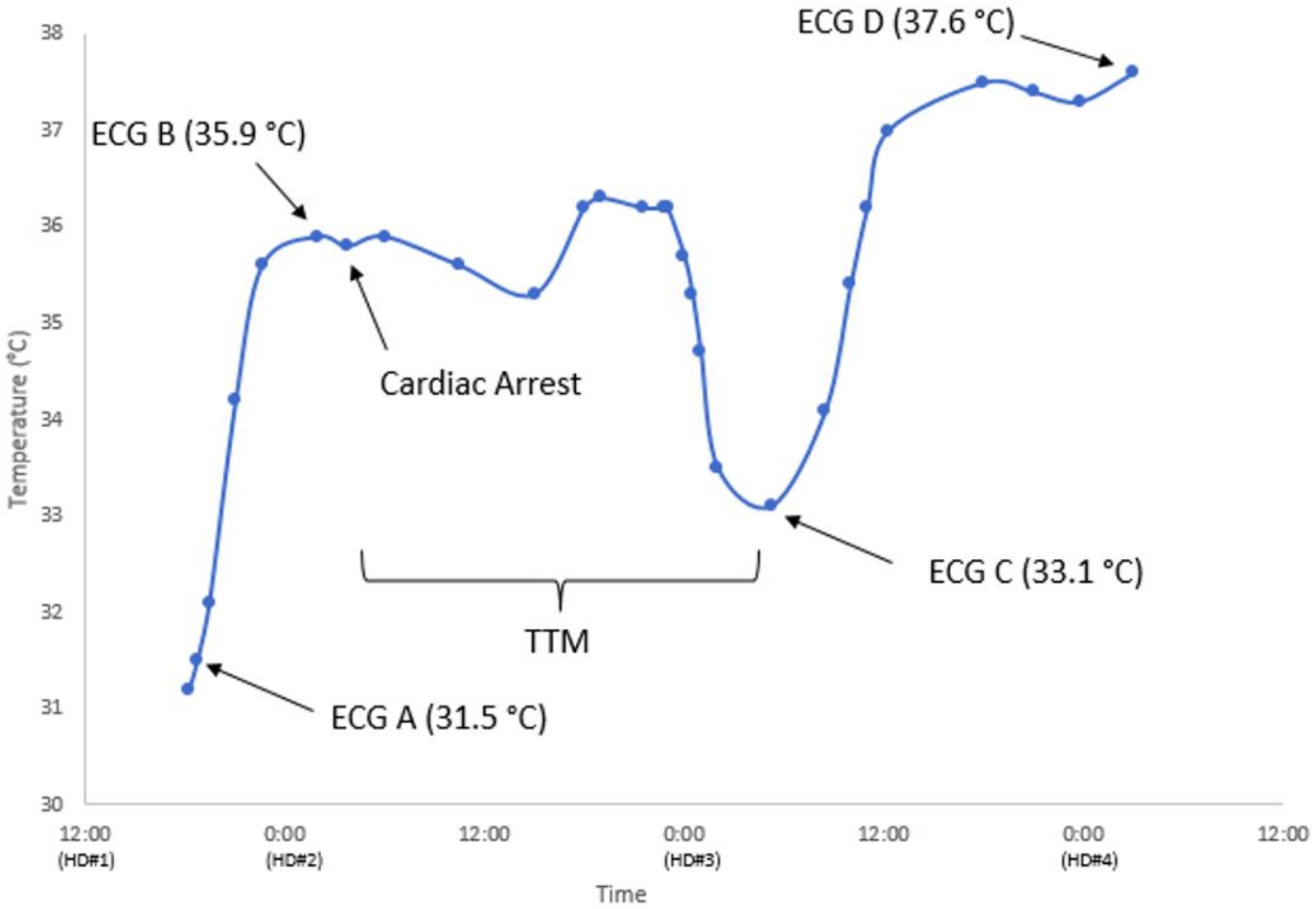

Figure 1. Graphical depiction of patient's temperature over first four days of hospitalizations. The time points and corresponding temperatures of when the ECGs in Figures 2-5 were obtained. The time of the patient's cardiac arrest and the time TTM was administered are also depicted.

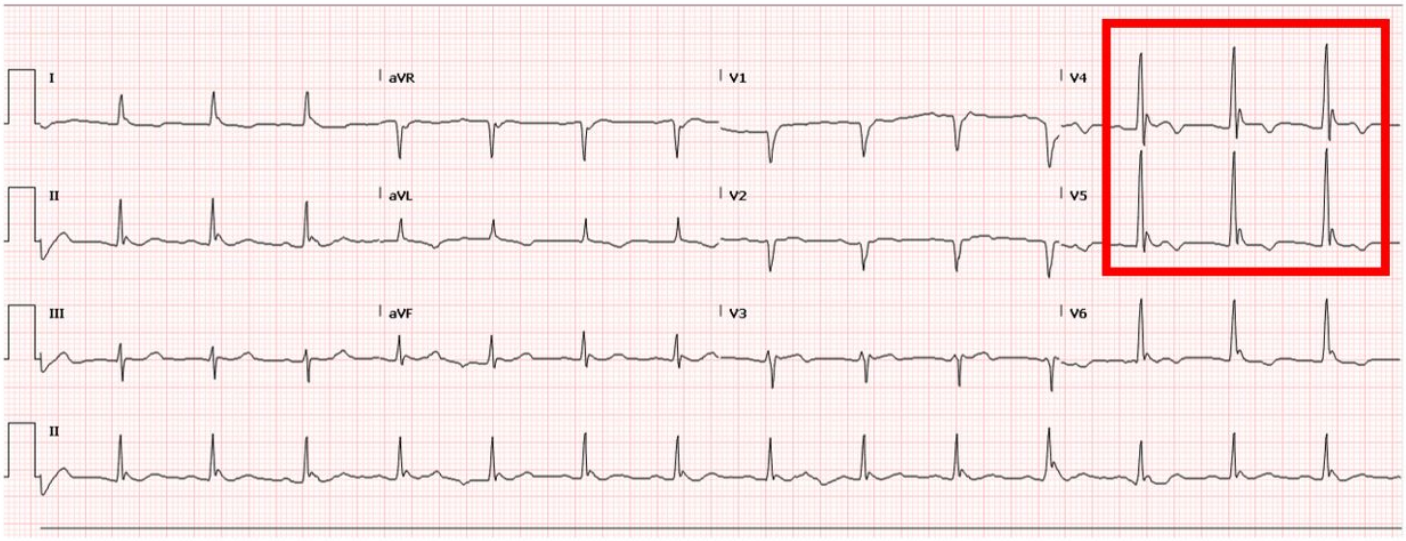

Figure 2. ECG A on hospital day 1 at core body temperature of 31.5°C. Osborn waves are demonstrated in leads V4 – V5, as depicted by the red box.

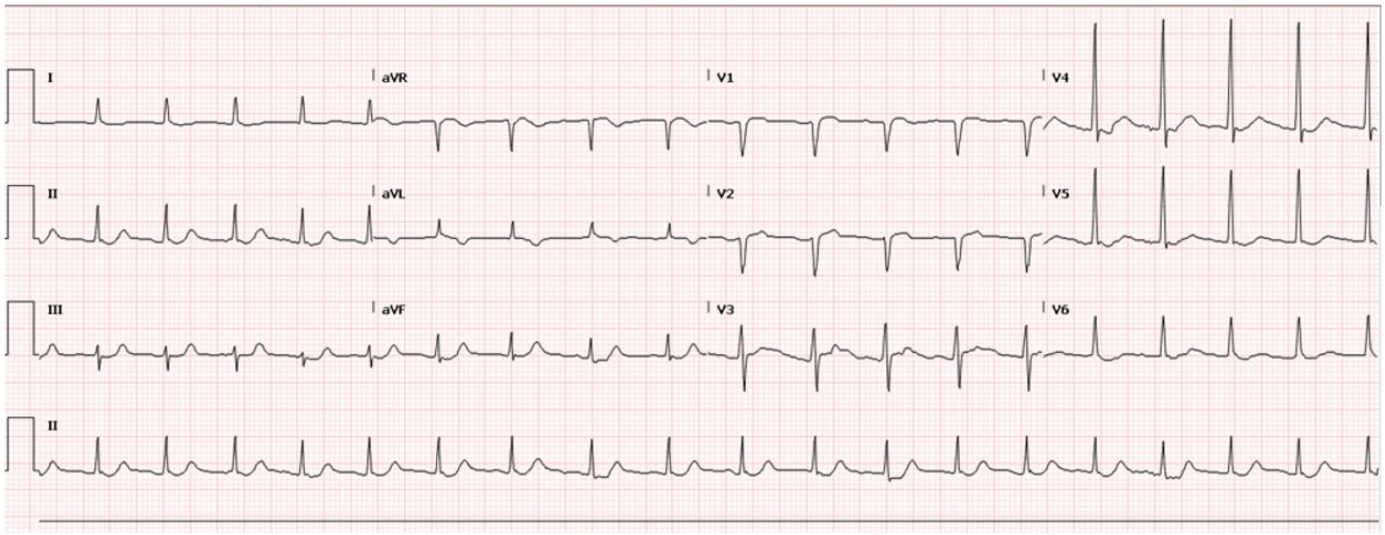

Figure 3. ECG B on hospital day 2 with resolution of Osborn waves with re-warming to temperature of 35.9°C.

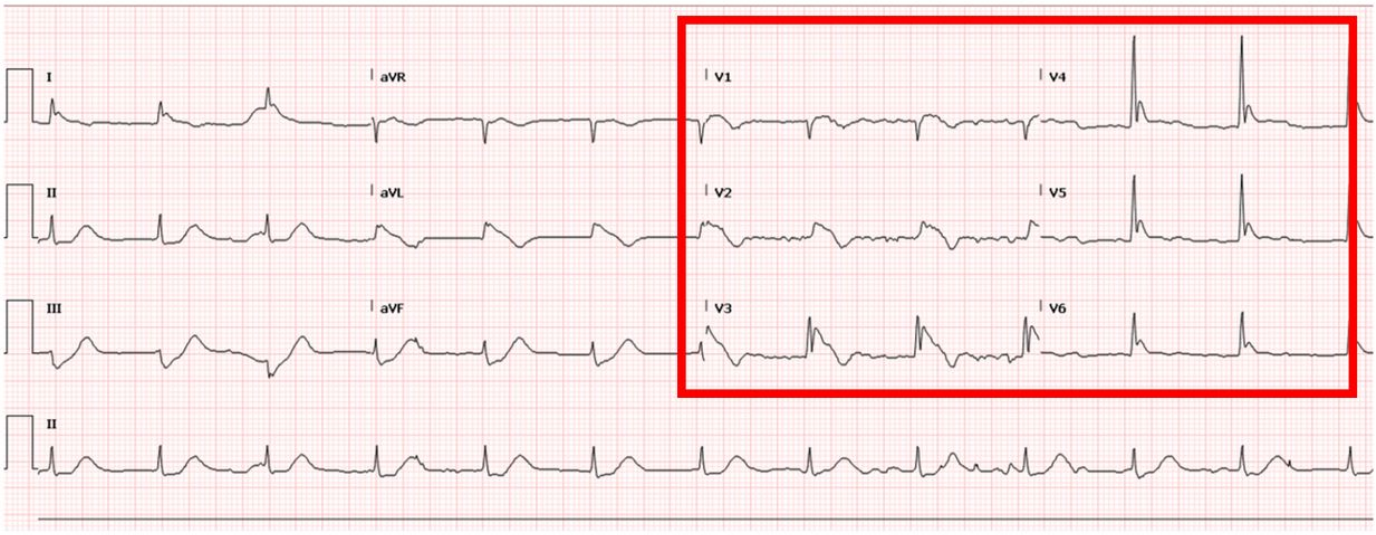

Figure 4. ECG C re-demonstrating Osborn waves on hospital day 3 when temperatures cooled again to 37.6°C.

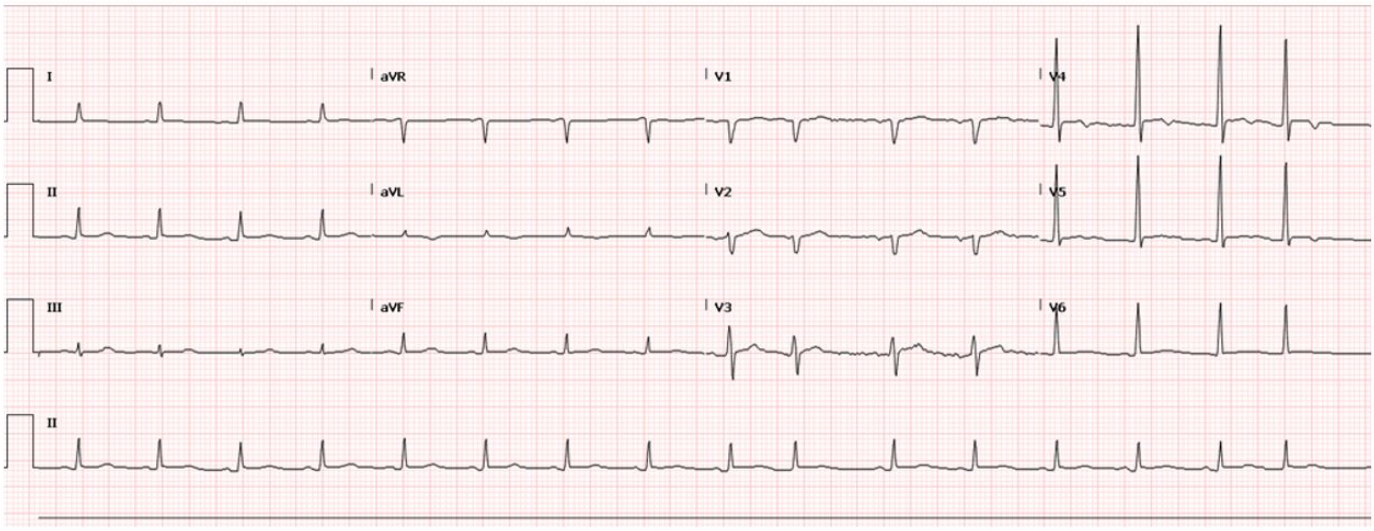

Figure 5. ECG D on hospital day 4, again demonstrating resolution of Osborn waves with rewarming to 33.1°C.

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