

## Severe Hypothermia With Osborn Waves in Diabetic Ketoacidosis

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### Introduction

Diabetic ketoacidosis causes substantial morbidity and mortality.<sup>1</sup> Though the management of acute diabetic ketoacidosis centers on correcting hyperglycemia and electrolyte abnormalities, other important metabolic derangements may be seen. Specifically, hypothermia may be both a cause and consequence of diabetic ketoacidosis, and the severity of the hypothermia correlates with the severity of the illness.

The case presented below highlights a presentation of severe diabetic ketoacidosis associated with hypothermia, which caused distinctive changes (Osborn waves) on the electrocardiogram (ECG). In the setting of diabetic ketoacidosis the recognition of ECG changes characteristic of hypothermia will help guide management and avoid the misdiagnosis of an ST-segment elevation myocardial infarction.

### Case Report

A 30-year-old man was found unresponsive in his apartment by a family member. He was intubated by the paramedics. At evaluation in the emergency department, he had a core temperature of 26.8°C and a serum glucose of 1,070 mg/dL.

The patient had been given the diagnosis of non-insulin-dependent diabetes mellitus 4 months earlier and was taking metformin 500 mg orally, twice daily. According to his family, he ran out of the medication one month prior to this presentation and did not seek medical follow-up. He had no other known medical conditions and was not taking

any medications. He drank alcohol on occasion. He did not smoke tobacco or use illicit drugs.

On examination he was intubated and nonresponsive. His temperature had risen slightly, to 27.8°C, shortly after admission. His blood pressure was 107/58 mm Hg, and his heart rate was 52 beats/min. His blood oxygen saturation was 100% while breathing 100% oxygen on assist-control ventilation. His lung and cardiac examinations were normal. Abdominal examination revealed hypoactive bowel sounds, without masses or organomegaly. Neurologic examination revealed absent gag and corneal reflexes, sluggishly reactive pupils, a normal plantar reflex, and 1+ symmetric patellar and Achilles deep tendon reflexes. The patient did not demonstrate purposeful movements, and painful stimuli did not elicit any response.

Initial arterial blood gas values were pH 6.65,  $P_{CO_2}$  28 mm Hg, and  $P_{O_2}$  320 mm Hg. Serum chemistries showed sodium 132 mEq/L, potassium 6.8 mEq/L, chloride 100 mEq/L, bicarbonate 3 mEq/L, phosphate 7.6 mg/dL, creatinine 3.6 mg/dL, blood glucose 1,111 mg/dL, and serum osmolality 371 mOsm/kg. Triglycerides were 853 mg/dL. His lipase level was 3,178 U/L (normal range 12–64 U/L). His pancreatic amylase was 491 U/L (normal range 14–77 U/L). Urinalysis revealed 4+ ketonuria and 3+ glucosuria. Urine and serum toxicology panels were negative. Cardiac troponins were negative. A noncontrast head computerized tomogram was unremarkable.

Twelve-lead ECG showed sinus bradycardia with peaked T waves, prominent J (Osborn) waves, and QT prolongation (Fig. 1). An abdominal ultrasound revealed a normal-appearing liver and pancreas.

He received a 15-unit bolus of regular insulin, followed by intravenous fluids and an insulin infusion, and was admitted to the medical intensive care unit.

Successful rewarming was achieved with an external rewarming blanket and warm intravenous fluids over 24 hours, and his profound acidemia progressively resolved; more aggressive rewarming maneuvers were not performed, given the patient's hemodynamic stability. His anion gap slowly corrected, with resolution of the ketoacidosis. His phosphate and potassium levels fell to < 0.5 mg/dL and 3.3 mEq/L, respectively, but corrected and remained stable throughout admission. Follow-up ECG showed resolution

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The author reports no conflicts of interest related to the content of this paper.

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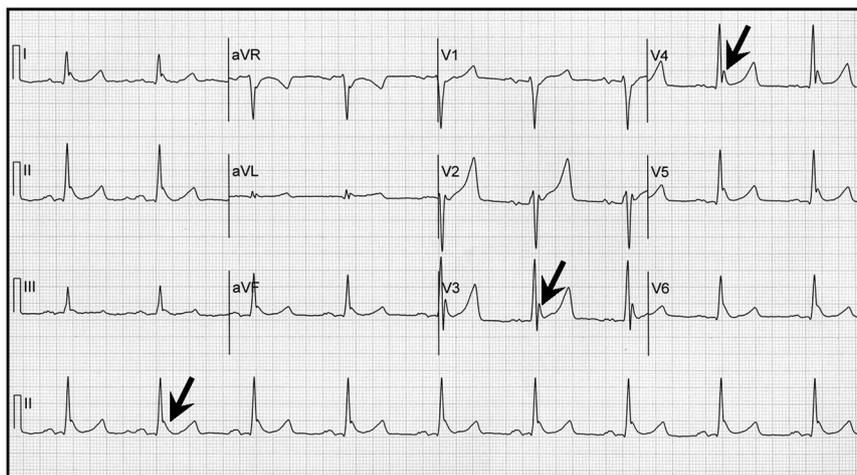


Fig. 1. This 12-lead electrocardiogram shows classic signs of systemic hypothermia. Sinus bradycardia (52 beats/min) and QT prolongation (540 ms) are present, with J point elevations (Osborn waves) at the R-ST junctions (arrows). Hyperkalemia (serum potassium 6.8 mEq/L) may account for the prominent T waves superimposed on the hypothermia pattern. These findings normalized on follow-up electrocardiogram after successful therapy.

of the Osborn waves and the T wave amplitude. After 4 days his serum lipase decreased to 115 U/L, and his triglycerides decreased to 34 mg/dL. However, he became anuric shortly after admission, and his creatinine rose to a peak of 6.1 mg/dL. His neurologic status improved and he was extubated one week after admission.

The patient was discharged from the hospital after an 8-day admission during which he required 5 units of neutral protamine Hagedorn insulin twice a day, with prandial short-acting insulin. He denied any symptoms suggestive of acute pancreatitis. His urine output improved, reflecting the resolution of acute tubular necrosis, and his creatinine was 2.3 mg/dL at the time of discharge. Follow-up chemistries 2 weeks after discharge were normal, and a fasting lipid profile 6 weeks after discharge was normal.

### Discussion

The patient had severe accidental hypothermia on presentation. The most common cause of hypothermia is cold weather exposure. Nonenvironmental causes include cerebrovascular accident, medications, infection, and endocrine abnormalities (eg, hypothyroidism, hypopituitarism, hypoglycemia, diabetic ketoacidosis).<sup>2</sup> Hypothermia may be both a contributor to and a result of severe diabetic ketoacidosis.<sup>3</sup>

The hypothermia seen with diabetic ketoacidosis is probably related to the inability of glucose to endocytose, due to the insulin deficit, which leads to a lack of substrate for cellular heat production.<sup>2,4</sup> One report cited diabetic ketoacidosis as the cause of 12% of cases of hypothermia over a 7-year period.<sup>5</sup>

Prompt recognition of severe hypothermia is critical in the early management of diabetic ketoacidosis because hypothermia portends a poor prognosis: mortality among such patients is 30–60%.<sup>5,6</sup> Furthermore, hypothermia potentiates insulin resistance and renders exogenously administered insulin much less effective, which further complicates management.<sup>2</sup>

Notably, the patient presented here had negative cardiac troponins throughout his admission. In addition, despite the presence of hyperlipasemia and hyperamylasemia, acute pancreatitis was probably not the cause of diabetic ketoacidosis in this young, previously healthy individual. Elevation of these enzymes can occur in critical illness in the absence of pancreatitis.<sup>7,8</sup>

In cases of marked hypothermia, an elevated J point on the ECG (Osborn wave) may be present, usually best seen on the precordial leads.<sup>9,10</sup> The pathophysiology of Osborn waves is not entirely understood but appears to be related to altered transmural action potential properties.<sup>11</sup> Hypothermic patients with this pattern are at increased risk of ventricular fibrillation during rewarming.<sup>12</sup> Recognizing this ECG pseudo-infarct pattern will avoid unwarranted treatment of suspected acute coronary syndrome.

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