A case of hypothermia with significant Osborn Wave: a forgotten electrocardiographic finding

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Abstract

Hypothermia is generally defined as the body’s internal temperature below 35 °C. Hypothermia affects all organs, but the most important effects are on the cardiovascular and central nervous system [1]. Mild hypothermia (32-35 °C) usually manifests with normal electrocardiogram (ECG), but sometimes with Osborn (J) waves, albeit rare [2]. In moderate hypothermia, the j-wave becomes more pronounced in the inferior and lateral leads. In addition, this phase of hypothermia is associated with a prolongation in the PR and QT intervals, increase in QRS duration, decrease in the amplitude of P and T waves, and frequent supraventricular arrhythmias [3,4]. Severe hypothermia is associated, in addition to the above findings, with the disappearance of the P waves in all derivations, whereas J waves and frequent ventricular arrhythmias can be observed on electrocardiogram [5]. Osborn wave is a characteristic ECG finding of hypothermia. The Osborn wave is described as a small positive deflection wave (>1mm) at the junction corresponding to the end of the QRS complex and the beginning of the ST segment in two consecutive beats [6-8].

Keywords: Hypothermia, electrocardiography, Osborn Wave

Introduction

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In this case report, we present a patient, who was unconscious due to ischemic cerebrovascular event and hypothermic due to cold weather, and whose electrocardiogram showed an Osborn wave.

Case Report

A 68-year-old male patient was brought to emergency service because of having lost consciousness.

Initial assessment of the patient showed a blood pressure of 90/60 mmHg, heart rate of 62 beats/min, body temperature of 31°C and respiration rate of 10 breaths/min, and the Glasgow Coma Score was 5/15. Neurological examination revealed neglectful Babinski’s reflex (extensor plantar response) fixed dilated pupils, and bilateral light reflex was positive. Cardiovascular auscultation revealed a 2/6 systolic murmur in the apical region. Capillary blood glucose level was 136 mg/dl. The 12-lead ECG (Figure 1) at admission showed prominent Osborn waves, prolonged QRS duration (140 ms) and increased QTc intervals (486 ms) in all leads. Acute diffuse ischemic foci were detected in the brain by cranial computed tomography scan that was conducted once the patient was optimized for transport after the administration of active and passive heating methods. The laboratory examination of the patient revealed a white blood cell count of 10.800/mm³, hemoglobin level of 12.6 g/dl, hematocrit level of 37.5% and platelet count of 256,000/mm³. The levels of urea, creatinine, liver transaminases, troponin I, potassium, magnesium, chlorine, TSH were within normal limits. The blood gas analysis at admission demonstrated mild
respiratory acidosis (pH: 7.35), carbon dioxide retention (PCO2: 46.4 mmHg, PO2: 52.5 mmHg) and reduced oxygen saturation (78%). At the fourth hour, the rectal body temperature was 34 °C, and there was an increase in the heart rate and a decrease in the amplitude of the Osborn waves and the QTc duration (451 ms) on ECG (Figure 2).

At the 12th hour, the body temperature was measured as 36 °C and the Osborn waves disappeared on ECG. Neurological findings of the patient were not improved during follow-up and the patient died at the 72nd hour because of the deterioration of his neurological findings.

Figure 1. The 12-lead electrocardiogram of the patient with a body temperature of 31 °C shows prolonged QT intervals (489 ms) and prominent J waves with high amplitude in all leads.

Figure 2. The ECG of the patient with body temperature of 34 °C shows an increase in heart rate and marked reduction in the amplitude of Osborn waves.
Discussion

The J wave was first reported by Kraus in a hypercalcemic patient in 1920 and later reported by Tomaszewski in a hypothermic patient in 1938 [7,8]. In 1953, John Osborn described the Osborn waves that resulted in ventricular fibrillation on electrocardiography in an experimental hypothermia model on dogs [9]. As a result of hypothermia, the epicardial potassium current increases relative to that of endocardium during ventricular repolarization. This event is called the "injury current". The action potential notch generated in the epicardium creates a transmural voltage gradient and thus this transmural voltage gradient manifests itself as Osborn waves at the QRS-ST junction on the ECG. In addition, M cells in the deep subepicardial region may contribute to the formation of the Osborn wave in accordance with the order of transmural activation from the endocardium to the epicardium [10].

The development of Osborn waves in hypothermic patients is due to acid-base and electrolyte imbalance. Hypothermia slows the ventricular repolarization and cardiac conduction system, which may prolong all intervals and cause atrioventricular block development on the ECG. Osborn waves are seen in 80% of hypothermic patients and the amplitude of the Osborn waves is inversely proportional to the degree of hypothermia (6-11). There was a decrease in Osborn wave amplitude when the body temperature increased to 34 °C, and a subsequent increase in body temperature to 34 °C was observed to decrease the amplitude of the Osborn waves. Osborn waves disappeared on electrocardiogram at the 12th hour when the body temperature reached 36 °C, and concurrently the heart rate increased and QTc and QRS durations returned to normal limits. Osborn waves can also be seen in hypothermia, acute ischemic events, cocaine use, haloperidol overdose, hypertension-induced left ventricular hypertrophy, brain trauma, subarachnoid hemorrhage, excessive sedation after cardiopulmonary arrest, and rarely normothermic individuals [11]. In our case, we have detected no electrolyte imbalance or a left ventricular hypertrophy on echocardiography. There was no history of cocaine or haloperidol use in the patient. The disappearance of Osborn waves in the ECG with the increase in body temperature despite the deterioration of neurological symptoms suggested that ECG findings were a consequence of hypothermia rather than an acute ischemic event. In addition, simultaneous restoration of QTc and QRS durations supported the idea of Osborn wave formation due to hypothermia.

The determination of the Osborn wave in the ECG, which was assumed to emerge secondary to hypothermia provided convenience for differential diagnosis and treatment. Hypothermia can often develop due to cold traumas in people without metabolic problems, cold exposure due to occupational conditions, and after changes in consciousness due to alcohol and drug use [12]. In our case, the patient had long exposure to the cold in an unconscious state.

In conclusion, the unconscious patients with Osborn waves in ECG should be investigated for hypothermia. Therefore, early identification and differential diagnosis of hypothermia and Osborn waves by physicians may be life-saving.

References