The key role of ECG in diagnosing secondary hypothermia in a patient with shock: a case report

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Abstract

Even if hypothermia is described as one of the main causes of cardiac arrest, in medical literature, it is not often mentioned as a main cause of shock. Nevertheless, it is known to contribute to shock development, maintenance, and worsening of its prognosis by lowering cardiac performance and blood pressure; this is particularly relevant in sepsis. Diagnosing hypothermia can be challenging, especially in patients not suffering an obvious environmental exposure. Digital medical thermometers cannot accurately estimate hypothermia, so when suspected, the placement of an esophageal thermometer is indicated. Below 32°C, the Electrocardiogram (ECG) often shows a characteristic elevation of the J point called the Osborne Wave (OW). We present a case of a patient with mixed shock in which this electrocardiographic disturbance allowed for an early suspicion of hypothermia, enabling prompt initiation of adequate treatment.

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Introduction

Shock is a clinical syndrome due to circulatory failure that results in end-organ hypoperfusion and so inadequate cellular oxygen utilization, leading to high patient morbidity and mortality.[1] Many etiologies have been described as causing or contributing to shock, and more than one can be involved in the clinical scenario of a shocked patient. Hypothermia causes a reduction in metabolic activity[2,3] and, when moderate or severe, can lower heart rate, blood pressure, and cardiac performance, resulting in a worsening of the multiorgan failure.[4] Even if it has classically been considered as one of the 8 most frequent causes of cardiac arrest, hypothermia is not often mentioned in literature as a cause or contributor to shock.[5] Nevertheless, it is well documented that patients affected by sepsis who present with hypothermia show an increased incidence of shock and mortality compared to their normothermic or febrile counterparts.[6] While usually used digital axillary thermometers can fail to detect moderate to severe hypothermia, a characteristic Electrocardiogram (ECG) alteration known as Osborne Wave (OW) often appears. Recognizing OW can lead to suspecting and promptly treating hypothermia while confirming it invasively, especially in non-traumatic shocked patients.

Case Report

A 67-year-old man with a history of obesity, hypertension, ischemic heart disease with normal cardiac function, and chronic obstructive pulmonary disease was found unconscious. He was last seen 12 hours before, apparently asymptomatic. The pre-hospital medical team found him at home unconscious with 5 points on the Glasgow Coma Scale, a blood pressure of 90/60 mmHg, heart rate of 65 bpm, respiratory rate of 10/min, and oxygen saturation of 69% when breathing room air (this data could be distorted because of bad perfusion). Body temperature couldn't be determined, while blood sugar was normal. An intravenous line was taken, fluid resuscitation was started, and a rapid sequence intubation with etomidate, midazolam, and rocuronium was performed before transferring the patient to the Emergency Department (ED). When the

patient arrived at the ED, he was still profoundly hypotensive. The oxygen saturation was 99%, with supplementary 100% oxygen delivered by an invasive ventilator via a correctly placed orotracheal tube. Regular digital thermometers were not able to detect axillary and rectal temperature. His skin was cold and mottled. His heart was arrhythmic at 90 bpm. Air passed bilaterally, while crackles could be heard in the right inferior lung. The abdomen was apparently normal. He showed edema of the left inferior limb with tight, glossy, and swollen skin. Arterial blood gases showed an acute respiratory failure with associated lactic acidosis (lactate 10 mmol/L). A 12-lead ECG was obtained (Figure 1), which showed a new onset of atrial fibrillation with diffuse ST segment depression and a subtle elevation in aVR, as well as a deflection wave in the shape of a dome after the QRS complex, consistent with OW. A chest x-ray showed a right inferior consolidation. The complete blood test showed white blood cells 12.700/mm^3, hemoglobin 14.8 g/dL, platelets 174x10^9/L, Na 145 mmol/L, K 2.94 mmol/L, urea 49 mg/100 mL, creatinine 1.58 mg/100 mL, total bilirubin 1.61 mg/100 mL, conjugated bilirubin 0.89 mg/100 mL, aspartate transaminase 408 U/L, alanine transaminase 337 U/L, C reactive protein 57 mg/L, procalcitonin 6.26 ng/mL, and troponin 386 ng/L. A point-of-care ultrasound ruled out a deep venous thrombosis. It documented a diffuse, slightly impaired left ventricular contractility with lowered cardiac output (Velocity Time Integral 12 cm), a normal right ventricular function, and no significant valvular disease or pericardial effusions. The patient was suspected of septic shock from cutaneous and respiratory focus (left limb cellulitis and aspiration pneumonia) with associated cardiac stunning induced by hypothermia and hypoxia; boluses of warm fluids and empiric antibiotics with piperacillin/tazobactam were started, while an inflation air warming thermal blanket was placed. After 3 liters of crystalloids, hypotension persisted, so noradrenaline and dobutamine were started, a central venous line was placed, and he was admitted to the ICU, where an esophageal thermometer showed a central temperature of 32°C, confirming the diagnosis. A nasogastric tube and a urinary catheter were inserted to start warm fluid infusion in order to guarantee controlled and progressive warming. Potassium was supplemented.

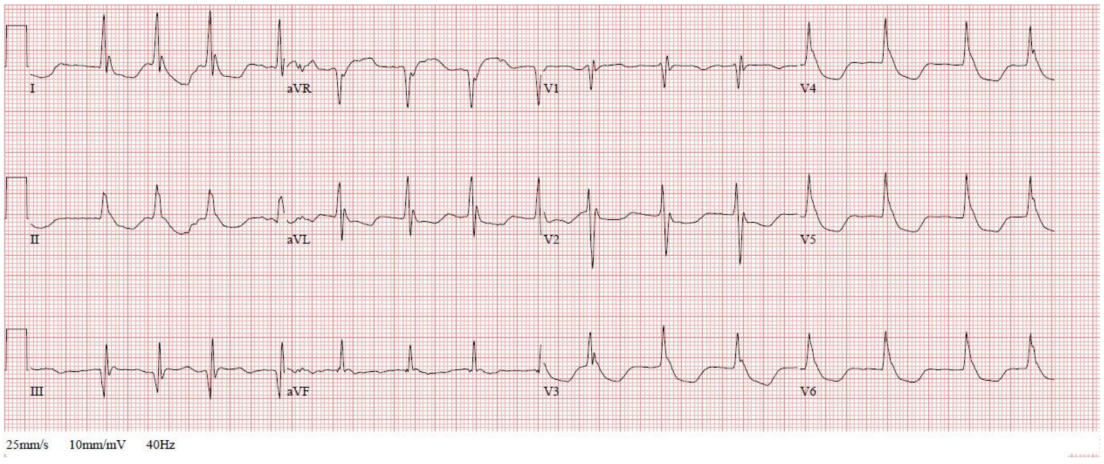

Figure 1. ECG 1, TC 32°C. Prominent convex deflections at the junction between QRS and the beginning of the ST segment consistent with Osborn waves.

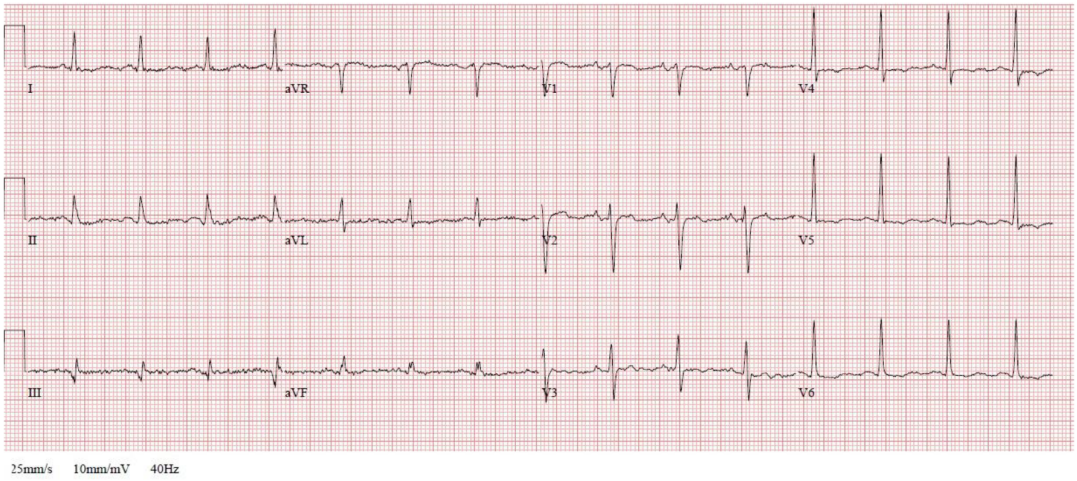

Figure 2. ECG 2. Normalization of the ECG when temperature is normal.

The patient showed progressive improvement; vasoactive and inotropic support were suspended after 48 hours. He was extubated on day 4 and discharged fully recovered by day 14, with no readmission within 30 days. The ECG and cardiac ultrasound alterations resolved as soon as normal temperature levels were obtained and hypoxia and shock subsided.

Discussion

Hypothermia is defined as a body temperature less than 35°C and is classified into mild (35 to 32°C), moderate (32-28°C), and severe (less than 28°C); the last one represents a life-threatening condition. It usually develops as a result of prolonged exposure to cold environments, as it happens in avalanche victims, drowning, homeless people, and severe trauma. This is known as primary hypothermia and can easily be suspected by its context.

Nevertheless, hypothermia can develop even in a warm environment; in that situation, it is called secondary hypothermia. Secondary hypothermia can occur in ill persons with a wide variety of medical conditions such as acidosis, malnutrition, hypoglycemia, endocrine disorders, central and peripheral nervous system lesions, Parkinson's disease, toxins, burns, cancer, infection, and shock.[7] In patients with secondary hypothermia, death is often caused by the underlying condition rather than by hypothermia itself, but its contribution must be taken into account. In all types of hypothermia, consciousness, breathing, and circulation gradually deteriorate as the body cools, progressively leading to coma, hypoxia, and shock. Today, sepsis is the most common cause of non-traumatic shock worldwide, with its prognosis closely linked to the timely administration of antibiotics. A notable proportion of septic patients do not develop fever, and 10 to 20% of them present with hypothermia, complicating the diagnosis.[8] It remains uncertain whether sepsis-related secondary hypothermia represents a manifestation of severe inflammation or an adaptive response aimed at reducing metabolism and preventing hypoxia;[9] on the contrary, it is well known that septic patients presenting with hypothermia suffer a higher incidence of shock and mortality compared to those who are normothermic or febrile.[6]

Given these considerations, measuring body temperature in all patients with shock is mandatory, as well as maintaining a high suspicion for sepsis while other causes of shock are ruled out. The European law[10] requires manufacturers of standard digital thermometers to ensure they can measure temperatures between 34°C and 43°C, so, depending on the brand, lower and upper values are usually not estimated and simply marked as "lo", "hi" or "err". For this reason, hypothermia diagnosis is sometimes tricky, especially when not immediately suspected because of evident exposure reasons (secondary hypothermia). ECG is an essential tool in the initial evaluation of all acutely ill patients, allowing the quick identification of pathologies such as arrhythmias, myocardial ischemia, metabolic disorders, or intoxications that may cause or contribute to the patient's clinical condition. Its immediate interpretation facilitates critical therapeutic decisions and improves the patient's prognosis. This fact is even more relevant when facing an unconscious and critical patient when obtaining a correct past medical history and detailed anamnesis is not usually feasible, and rapid action is needed. A series of ECG alterations can be observed during hypothermia, including lengthening of the PR, QRS complex and QT interval, bradycardia, atrial fibrillation, and even ventricular fibrillation.[11] Below 32°C, a characteristic wave is produced in the ECG due to elevation of the J point, called the Osborn wave. This finding has been described in the early '50s as a deflection with a dome or hump morphology in the same direction as the R wave, immediately following the QRS complex, and its prevalence rates have been reported as 75% in moderate hypothermia and 100% in severe hypothermia.[12] Although best known as a marker of hypothermia, OW has also been occasionally described in other conditions such as acidosis,[13] hypercalcemia,[14] and myocardial ischemia.[15] OW tends to be most evident in the inferior and lateral precordial leads (II, III, aVF, V4-V6) and disappears when normalizing the temperature or treating the underlying cause.[12]

Our patient was found unconscious and shocked in a city with a warm climate. He was intubated by the pre-hospital medical team, so it was not possible to get a detailed medical history in the ED. The thermometer could not determine the body temperature. Still, the initial ECG (Figure 1) showed a diffuse concave ST depression with slight ST elevation in aVR, which are common findings in severe hypoxia, probably emphasized by the associated hypokalemia.

Moreover, especially in DI, it was evident a prominent convex deflection at the junction between QRS and the beginning of the ST segment, consistent with OW. These alterations subsequently resolved (Figure 2) with the normalization of the patient's body temperature and the correction of hypoxia, hypokalemia, and shock. As the conventional thermometer couldn't determine exactly the body temperature, in our case the ECG played a crucial role in suspecting (and so promptly treating) secondary hypothermia, subsequently confirmed with an esophageal thermometer. Furthermore, promptly detecting the association of shock with hypothermia heightened suspicion of sepsis as the primary cause of the patient's condition, which was later confirmed. In our patient, the complementary tests and confirmation of the normalization of ECG and ultrasound abnormalities after temperature recovery allowed for ruling out the other mentioned causes of OW.

Conclusions

Because of the unreliability of regular thermometers, hypothermia can be difficult to suspect and diagnose in patients without a history of environmental exposure to cold; therefore, it can potentially be kept underdiagnosed and consequently undertreated. Nonetheless, it can represent a relevant issue, contributing to shock development and maintenance by lowering cardiac performance and blood pressure, leading to acidosis and coagulopathy, and overall worsening a patient's prognosis, especially when related to sepsis. In a patient with shock and indeterminate body temperature, a simple, easily available, affordable, and quick test as a 12-lead ECG can show a characteristic elevation of the J point known as OW, which can be useful to suspect secondary hypothermia and stratify the mortality risk when the underlying cause is sepsis.

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