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A case report: Brugada syndrome in the setting of hypothermia

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1. Introduction

Hypothermia is defined as a core body temperature at about 35 °C (95 °F) [1]. It has many causes that can be due to environmental factors, impaired thermoregulation, or even iatrogenic causes [1]. The severity of hypothermia occurs on a spectrum of mild (32.2–35 °C), moderate (28–32.1 °C), or severe (< 28 °C) [2]. The symptoms of hypothermia vary depending on the severity with mild to moderate symptoms such as elevated blood pressure, shivering, tachycardia, tachypnea, apathy, ataxia, cold diuresis, and hypovolemia [2]. Hypothermia can further progress to more severe symptoms such as altered mental status, coma, shock, and cardiac arrythmias leading to cardiac arrest [2]. These cardiac abnormalities are reflexed on electrocardiograms (EKGs) [3]. EKG changes include, j-waves (Osborn waves), sinoatrial exit block, PR and QT prolongation, QRS widening, ST depression, atrial fibrillation, and ventricular fibrillation [3]. In some rare cases, hypothermia has been known to cause Brugada pattern [4].

Brugada pattern is classified as EKG changes showing ST elevations in the right precordial leads (V_1-V_3) , and accentuated J waves imitating an incomplete right bundle branch block [5]. Brugada is a right ventricular disease and has been linked to several mutations which are inherited in an autosomal dominant fashion [5]. Type One Brugada pattern, also called coved type, is characterized by ST elevation $\ge 2 \text{ mm in } \ge 1$ right precordial lead followed by an R' wave and a concave or straight ST segment which is diagnostic for Brugada Syndrome [6]. Type Two Brugada pattern, also called saddle-back type, is characterized by ST elevation $\ge 0.5 \text{ mm in } \ge 1$ right precordial lead followed by a convex ST [6].

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https://doi.org/10.1016/j.ajem.2022.02.047 0735-6757/© 2022 Elsevier Inc. All rights reserved. ABSTRACT

Hypothermia is a common diagnosis in the Emergency Department. It can cause a multitude of symptoms and complications if not treated promptly. The following case report discusses Brugada pattern on an electrocardiogram in a patient with hypothermia and diabetic ketoacidosis. There was resolution of the Brugada pattern on the electrocardiogram after the patient was warmed to 35.3 °C.

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Brugada, in the inheritable form, is linked to an increased risk for ventricular fibrillation and sudden cardiac death [6]. The risk of cardiac arrythmias and death from Brugada are further increased when the body is placed under stress [5]. For instance, some patients will develop symptoms when they see blood, during micturition, have infections, fever, electrolyte abnormalities (like hyperkalemia) and many other types of stressors [5]. The following case shows a patient with hypothermia and diabetic ketoacidosis (DKA) who developed Brugada pattern on an EKG.

2. Case report

A 65-year-old male with a past medical history of lung adenocarcinoma in remission, Type II diabetes mellitus, hypertension, and a prior cerebral vascular accident presented to the emergency department (ED) for hyperglycemia. The patient also reported a frontal headache and fatigue. The patient was slightly confused and unable to provide much history. He did deny any recent illness, fevers, chest pain, shortness of breath, nausea, or vomiting. While he was at home, his glucose monitor read 'high', and this prompted the patient's wife to call emergency medical services.

The patient's initial vitals were as follows: blood pressure 94/39 mmHg, heart rate 101 bpm, temperature 33.9 °C (93 °F), respiratory rate 31 bpm, and SpO2 100% on room air. A temperature sensing foley was placed as well as a bear hugger to improve and monitor his temperature due to hypothermia. The patient was also given one liter of intravenous normal saline to improve his blood pressure. The physical exam revealed a generally ill and toxic appearing patient with dry mucous membranes, poor skin turgor, and lethargy. He had no focal neurologic deficits and was orientated to person, place, and time. His POC glucose read >600 mg/dL (normal less than 65-99

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C.M. Juybari, S.L. Gaetani, A.H. Miller et al.

American Journal of Emergency Medicine xxx (xxxx) xxx



Fig. 1. Initial electrocardiogram of hypothermic patient at admission. The patient's temperature was 33.9 °C. The blue arrows denote the ST elevations, all greater than 2 mm, followed by R' waves denoted by red arrows in V3, which meet the criteria for a Type One Brugada pattern. The green arrows in V1 and V2 denote RSR' wave complexes, with QRS > 0.12 s suggesting a right bundle branch block. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

mg/dL). An initial EKG revealed ST elevations in V₃ followed by an R' wave which met the requirement for Type One Brugada pattern (Fig. 1). The patient was found to have leukocytosis of 27thou/cmm (normal 4–10.5thou/cmm). He also had a pH of 7.01 (normal 7.35 to 7.45), a Potassium of 6.1 mmol/L (normal 3.6 to 5.2 mmol/L), a bicarbonate level of 5 mmol/L (normal 23-31 mmol/L), an anion gap of 26 (normal 3-11), a blood glucose on chemistry of 805 mg/dL (normal 65-99 mg/dL) and troponin I was <0.02 ng/mL (normal between 0 and 0.04 ng/mL). The patient was noted to be in DKA, and once the Potassium level was resulted, the patient was started on an insulin drip. After about three hours of active resuscitation and warming, his temperature improved. A repeat EKG was ordered which showed the resolution of the Brugada pattern (Fig. 2). At this time, the patient's Potassium level was still elevated and read 5.3 mmol/L. A little over an hour after the EKG was completed, his Potassium level lowered to 4.1 mmol/L which is within normal limits. The patient was ultimately admitted to the intensive care unit for DKA. Throughout the hospital course, his DKA was treated and an outpatient referral to electrophysiology cardiology was placed.

3. Discussion

Hypothermia can cause atrial fibrillation, ventricular ectopic activity, ventricular tachycardia, ventricular fibrillation, and asystole [3]. Also, there are some case reports and animal models that have shown hypothermia causing Brugada like changes on the EKG; however, this is rarely seen in clinical practice as other EKG changes are more commonly associated with hypothermia [4]. In most cases, hypothermic induced EKG changes, notably J waves, will resolve with rewarming/treatment of hypothermia [3]. In the case above, the patient's Type One Brugada pattern experienced full resolution of EKG changes when he was warmed to 35.3 °C. To add to this discussion,



Fig. 2. Electrocardiogram taken after hypothermia improved. This was taken three hours after the initial EKG in Fig. 1. The patient's temperature was 35.3 °C. There is resolution of Type One Brugada pattern, the ST elevations and R' waves in V3, and the RSR' waves and QRS elongation in V1 and V2.

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C.M. Juybari, S.L. Gaetani, A.H. Miller et al.

there is a possibility that the patient's Hyperkalemia could have contributed to the Brugada pattern because it is a known cause of Brugada like pattern on EKGs [5]. As the patient's initial Potassium was 6.1 mmol/L, the Brugada is more likely caused by his hypothermia because the Brugada pattern resolved prior to the patient's Potassium normalizing. The Brugada pattern had resolved shortly after the patient had become normothermic.

Due to hypothermia being very arrhythmogenic, it is imperative for emergency medicine physicians to look for EKG changes, including Brugada pattern when evaluating a patient presenting with hypothermia. It is also important to note that ST elevations in Brugada pattern can resemble those seen in a myocardial infarction [7]. This can lead to misdiagnosis and unnecessary or expensive interventions that may cause more harm than good. It is important for emergency room physicians to be able to identify Brugada pattern on EKGs [7]. Recognizing EKG changes in hypothermia is significant because it can lead to potentially dangerous arrythmias like Brugada pattern, atrial fibrillation, and ventricular fibrillation [3,4,7].

Author contributions

All authors provided substantial contributions to manuscript content. All authors gave final approval of the version of the article to be published.

CRediT authorship contribution statement

Cameron M. Juybari: Writing – original draft. **Samantha L. Gaetani:** Writing – review & editing. **Andrew H. Miller:** Writing – review & editing. **Gavin C. Barr:** Writing – review & editing.

Declaration of Competing Interest

None.

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