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Case Report

Electrocardiographic Manifestations in Three Psychiatric Patients with Hypothermia – Case Report

Pelechas E[1]*, Tsigaridas N[2], Kyrama S[3], Trogganis E[4] and Kardamis Ch[5]

[1]Accident and Emergency Department, Scarborough General Hospital, United Kingdom

[2]Department of Cardiology, Chatzikosta Hospital, Ioannina, Greece

[3]Department of Cardiology, General Hospital of Arta, Greece

[4]Department of Cardiology, General Hospital of Kastoria, Greece

[5]Department of Cardiology, General Hospital of Corfu, Greece

***Corresponding author:** Eleftherios Pelechas, Accident and Emergency Department, Scarborough General Hospital, Scarborough, North Yorkshire, United Kingdom

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Abstract

Hypothermia occurs when the core body temperature falls below 35°C and, in severe cases, it can lead to electrocardiographic changes. Several conditions which can occur in the psychiatric population increase the risk of hypothermia which can be aggravated by the use of several classes of medications such as antipsychotics, beta-adrenergic antagonists, benzodiazepines and other sedatives. Three psychiatric patients have been admitted for hypothermia and electrocardiographic manifestations (sinus bradycardia, QT prolongation and Osborn waves) which reversed completely after treatment.

Keywords: Hypothermia; Osborn waves; Electrocardiographic changes; Psychiatric patients.

Introduction

Hypothermia is associated with a spectrum of electrocardiographic changes [1]. The degree of hypothermia leads to various electrocardiographic manifestations [2]. In mild hypothermia (35°C - 32°C), the Electrocardiogram (ECG) is usually normal but it can rarely show J waves (Osborn waves) [3]. The presence of Osborn waves in inferior and lateral leads, in combination with the appearance of other electrocardiographic manifestations such as increase in PR and QT intervals, increase in QRS complex duration, decrease in amplitude of P and T waves and frequent supraventricular arrhythmias, are noted in moderate hypothermia (32°C – 28°C) [4-7]. In severe hypothermia (<28°C), additional ECG changes such as J waves in all leads, absence of P waves and frequent ventricular arrhythmias [8-9]. Osborn wave is considered the most specific ECG change in hypothermia [10-12].

Case Presentation

Three psychiatric inmates,within a month (December), have been transferred to the emergency department by ambulance due to low responsiveness (two of them) and coma (the third patient). The medical and drug history of those three patients is presented in (Table 1). Their electrocardiogram showed sinus bradycardia (38bpm – 43bpm), QT prolongation (.52sec - .72sec) and Osborn waves (Figure 1, Figure 2 and Figure 3). There is also a "shivering artifact" on the electrocardiograms of the first and the third patient.

All of those three patients were under treatment with benzodiazepines (diazepam and lorazepam), one of them was also suffering from enuresis and the other two from Parkinson's disease, which was poor-controlled. Their full blood count, urea and electrolytes and chest x-ray film did not show any signs of infection. Because of the fact that we had three patients in so short period of time, we found out that the heating system was not working as it should

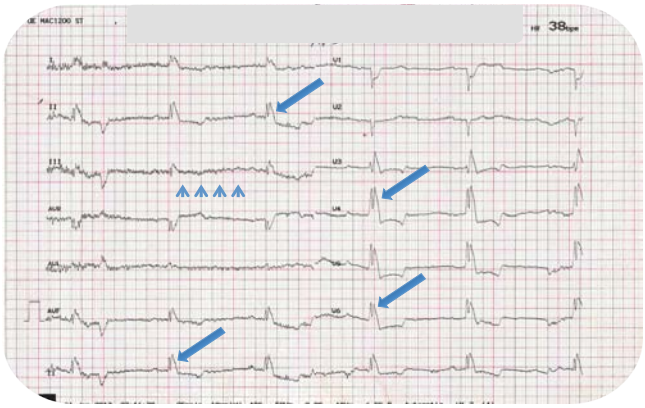

Figure 1: Electrocardiogram in severe hypothermia (27,2°C): sinus bradycardia (38bpm), QT prolongation, J waves in all leads, shivering artefact.

Table 1: Medical and drug history of the three cases of hypothermia.

	Age (in years)	Drug history	Core body temperature
Case 1	50	Diazepam, haloperidol, zolpidem	27,2oC
Case 2	61	Diazepam, haloperidol, zolpidem, clozapine, biperiden	27,6oC
Case 3	64	Levodopa, memantine, amlodipine/ valsartan, lorazepam	28,8oC

and it could be one of the factors as well as the enuresis problems and the shakings of the poor-controlled Parkinson's disease, that led those patients in hypothermia.

Case nr. 1 was taking orally diazepam and haloperidol when both increase sedation and drowsiness while combining zolpidem and diazepam increases risk of central nervous system depression.

Case nr. 2 was using the same medication with case nr. 1 plus clozapine. Combination of diazepam, haloperidol and clozapine

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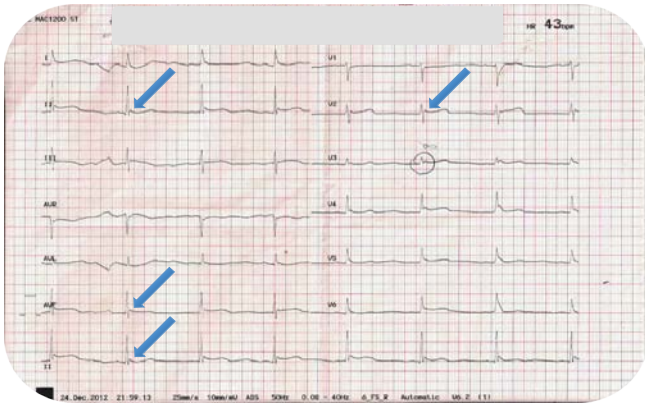

Figure 2: Electrocardiogram in severe hypothermia (27,6°C): sinus bradycardia (43bpm), QT prolongation, J waves and myocardial infarction-like ST elevation in $V_4 - V_6$.

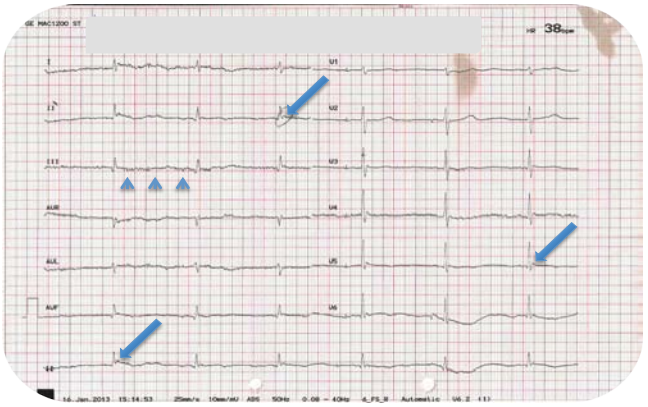

Figure 3: Electrocardiogram in moderate to severe hypothermia (28,8°C): sinus bradycardia (38bpm), QT prolongation, J waves in inferior leads (II, III, avf), shivering artefact.

increase sedation and drowsiness and there is risk of heart or respiratory failure.

The only possible interaction in case nr. 3 is that of amlodipine and valsartan with levodopa, which both can produce added drug effects and should be monitored closely.

After the progressive slow rewarming of the patients with special rewarming blankets, the electrocardiographic manifestations came back to normal. They have been discharged a few days later.

Discussion

J wave was initially reported by Kraus 1920 and then in 1922 in a patient with hypercalcemia and later Tomazewski (1938) in a patient with hypothermia [13-15]. John Osborn reported experimentally induced J wave in hypothermic dogs in 1953. He described J wave as a "current of injury" and postulated its occurrence secondary to hypothermia-induced acidosis [16]. Osborn waves (also known as camel-hump sign, late delta wave, hathook junction, hypothermic wave, prominent J wave), are positive deflections occurring at the junction between the QRS complex and the ST segment, where the S point (also known as the J point), has a myocardial infarction-like elevation [17-18]. The mean vector axis of the J wave is oriented

Table 2: Causes of Osborn waves in normothermic patients.

Acute ischemic events
Cocaine use
Haloperidol overdose
Left ventricular hypertrophy
Hypercalcemia
Brugada's syndrome
Central nervous system injury
After resuscitation of cardiac arrest

anteriorly, inferiorly and leftward across the left ventricle and septum [6,19] J waves are present in 80% of patients with a temperature less than 35°C [2]. There is no consensus on the prognostic significance of J waves. The physiological basis of J wave has been described by Antzelevitch and colleagues [19]. Responsible for the characteristic spike and dome pattern of action potential in the ventricular epicardial and endocardial cells is the presence of 4-aminopyridine-sensitive transient outward potassium current [20]. This current is more prominent in the ventricular epicardium compared to the endocardium. This difference creates a voltage gradient between the epicardial and endocardial cells [21]. This voltage gradient across ventricular myocardium is accentuated by the hypothermia and this results in prominent Osborn waves.

Osborn waves are notpathognomic of hypothermia. Normothermic patients can also present those waves. Some causes of Osborn waves in normothermic individuals are presented in (Table 2) [22-26]. Although the arrhythmogenic implications of the Osborn waves are not fully understood, the existence of this characteristic deflection may represent some underlying critical conditions. Different medical backgrounds of each patient could lead to ventricular arrhythmias but this should be considered individually. The true significance of the Osborn waves needs further studies in order to be considered as a potential distinguished characteristic for the patients who present it.

Several conditionsthat can occur in the psychiatric population, increase the risk of hypothermia. Mental retardation, debilitating physical illness, nocturnal enuresis and seizure disorders are some of those. This risk can be further increased by the use of several classes of medications used to treat psychiatric disorders such as benzodiazepines and other sedatives, antipsychotics, beta adrenergic antagonists [27-28]. Air-conditioning or poor supervision of those patients, regarding the room temperature, can lead to hypothermia.

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