Folia Cardiologica 2019
tom 14, nr 1, strony 75–78

ISSN 2353–7752

Osborn J wave in a patient with accidental hypothermia — case report

Fala Osborna u chorego z przypadkową hipotermią — opis przypadku

Olga Wajtryt[1], Tadeusz M. Zielonka[1, 2], Katarzyna Życińska[1, 2]

[1]Clinic of Internal Medicine and Metabolic Diseases, Czerniakowski Hospital in Warsaw, Warsaw, Poland

[2]Department of Family Medicine, Medical University of Warsaw, Warsaw Poland

Artykuł jest tłumaczeniem pracy: Olga Wajtryt i wsp. Fala Osborna u chorego z przypadkową hipotermią — opis przypadku. Folia Cardiol. 2019; 14 (1): 71–74. DOI: 10.5603/FC.2019.0011. Należy cytować wersję pierwotną

Abstract

Hypothermia could cause dysfunction of many organs. Any reduction in body temperature affects the circulatory system. Even slightly temperature reduction may result compensatory reactions in the circulatory system, and with significant hypothermia life-threatening arrhythmias are observed. A case of a homeless man, who was found in snow was described. In the course of moderate hypothermia paroxysmal atrial fibrillation and Osborna wave (the so-called J wave) in the electrocardiogram (ECG) was observed. After treatment and warming-up to normal body temperature the sinus rhythm was restored and morphology of the QRS complex was normal. On the basis of the case and literature in this paper cardiological disorders in hypothermia manifested by the Osborn wave in the ECG was presented.

Key words: hypothermia, Osborn wave, J-wave, electrocardiogram

Folia Cardiologica 2019; 14, 1: 75–78

Introduction

Accidental hypothermia according to the guidelines of the European Resuscitation Council from 2015, is an unintentional reduction of bodily temperature below 35°C, or, if its measurement is not possible, exposure to low temperature and a feeling of hypothermic patient [1]. We divide hypothermia into five phases: I mild 35–32°C, II moderate 32–28°C, III severe 28–24°C, IV cardiac arrest or hypoperfusion state < 24°C and V irreversible hypothermia < 13.7°C [1]. It is a significant clinical problem in countries with periodically low temperatures [2]. Due to the influence of temperature on the course of metabolic processes, hypothermia may cause damage to the functions of many organs, especially the cardiovascular, lung, kidney, central and peripheral nervous system [3]. Lowering the body temperature causes changes in the volume and composition of extracellular fluid and influences blood coagulation [3]. Even with a slight decrease in body temperature, compensatory reactions in the circulatory system, such as tachycardia and an increase in minute capacity, are observed [2]. As hypothermia and hypoxemia increase, severe life-threatening cardiac arrhythmias occur [2]. This results in a variety of changes in ECG.

We present a case of a patient with Osborn wave (so called J-wave) in ECG in the course of random hypothermia.

It is characteristic in several specific clinical situations, such as hypothermia, craniocerebral trauma, and less frequently hypercalcemia and ventricular arrhythmias [4].

Address for correspondence: Tadeusz M. Zielonka MD, PhD, Katedra i Zakład Medycyny Rodzinnej, Warszawski Uniwersytet Medyczny, ul. Stępińska 19/25, 00–739 Warszawa, Poland, phone/fax +48 22 318 63 25, e-mail: tadeusz.zielonka@wum.edu.pl

Case report

A 63 year-old homeless man with alcohol dependency syndrome was brought to hospital by the Emergency Rescue Team after a call from the Municipal Police, who found him lying on the snow in the area of allotment gardens where he lived in the winter. The patient negated chronic diseases, allergies, surgeries and the use of drugs. When being admitted he was conscious (Glasgow Coma Scale 10 points), cachectic, with poor hygiene, with trophic skin changes, numerous excoriations and lice. Life parameters: heart rate 75/min, blood pressure 90/60 mm Hg, oxygen saturation (SaO_2) 98% (FiO_2 0.21). Hypothermia with body temperature of 30°C was also found.

Laboratory studies revealed anemia (hemoglobin 6.7 g/dL), increased inflammatory parameters (C-reactive protein 10.1 mg/dL, at 0.5 mg/dL norm), rhabdomyolysis traits (creatine kinase 3119 U/L, at 26–190 U/L norm, heart isoenzyme 207 IU/L, at 2–24 IU/L norm), increased activity of aminotransferases (aspartate [AspAT] 181 U/L, alanine [AlAT] 100 U/L) and normal troponin concentration (0.02 µg/L). In venous blood gasometry acidosis (pH 7.187) was observed, lactate concentration was 16 mmol/L and base excess (BE) 15.6 mmol/L. The chest X-ray shows status post rib fracture. No significant abnormalities were described in the computed tomography (CT) of the head. There were no changes in the organs of the abdominal cavity in ultrasound examination. ECG showed atrial fibrillation with QRS 50–90/min, Osborn wave (so called J-wave) and negative T-waves in the leads: II, III and aVF and V3–V6 (Figure 1).

External physical heating, fluid resuscitation, including heated liquids and oxygen therapy were applied. Packed red blood cells were transfused. Vital parameters were monitored and a decrease in blood pressure to 70/50 mm Hg requiring an increase in fluid supply was observed. Hourly diuresis initially was < 50 mL/h, and after intravenous fluid bolus it was normal. A gradual increase in body temperature was obtained and after 6 hours of treatment at 35°C, the rhythm was converted to sinus rhythm. Sinus bradycardia was observed in ECG at body temperature of 36.3°C and the morphology of QRS, ST interval and T-wave syndromes were normal (Figure 2). In venous blood gasometry the pH was normal and lactates were 1.4 mmol/L. Echocardiography on day 2 in hospital revealed hypokinesis of the lower left ventricular wall. The size of cardiac cavities, left ventricular thickness, ejection fraction and valve function were normal. In the following days the patient was in good general condition, frostbite of the left III and IV toes were observed and after demarcation of the lesions he was qualified for surgical treatment. The patient did not consent for further hospitalization, diagnostics and treatment, or to social assistance and placement in an institution for the homeless. He left the hospital with the recommendation of further treatment and control in the outpatient clinic.

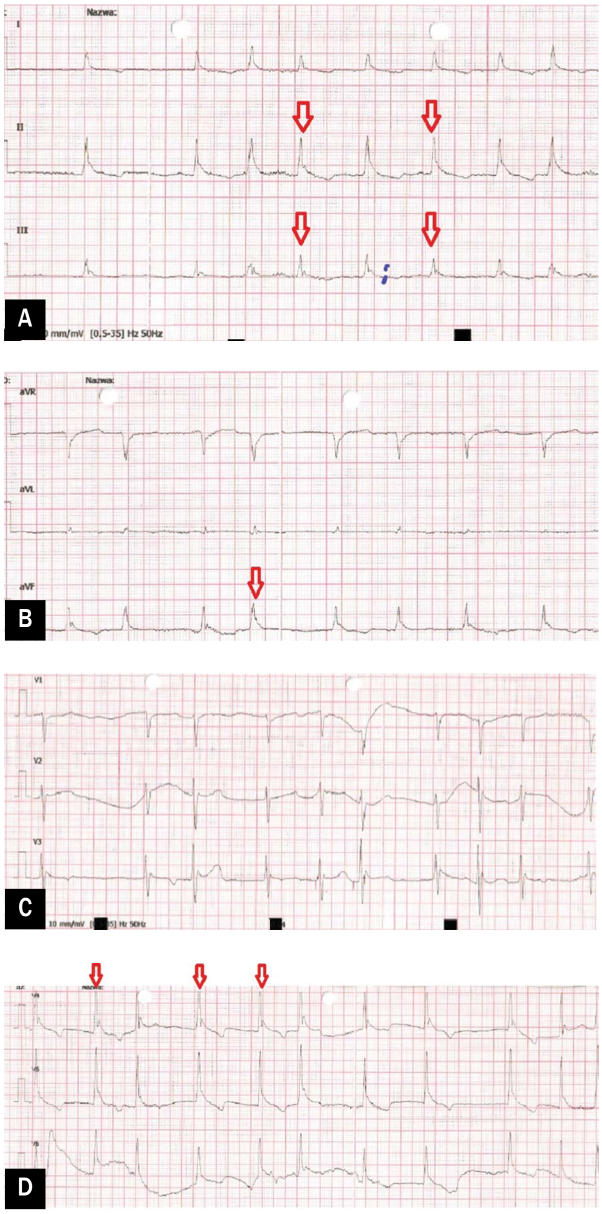

Figure 1A-D. ECG record made on admission to the hospital (limb and precordial limbs) — atrial fibrillation with frequency of QRS 50–100/min, J wave, negative T wave in the inferior wall leads II, III, aVF and antero-lateral wall leads V4–V6

Discussion

Electrocardiographic manifestations of hypothermia include cardiac arrhythmias (sinus bradycardia, atrioventricular block, atrioventricular fibrillation, ventricular arrhythmias), prolongation of the PR interval, QRS

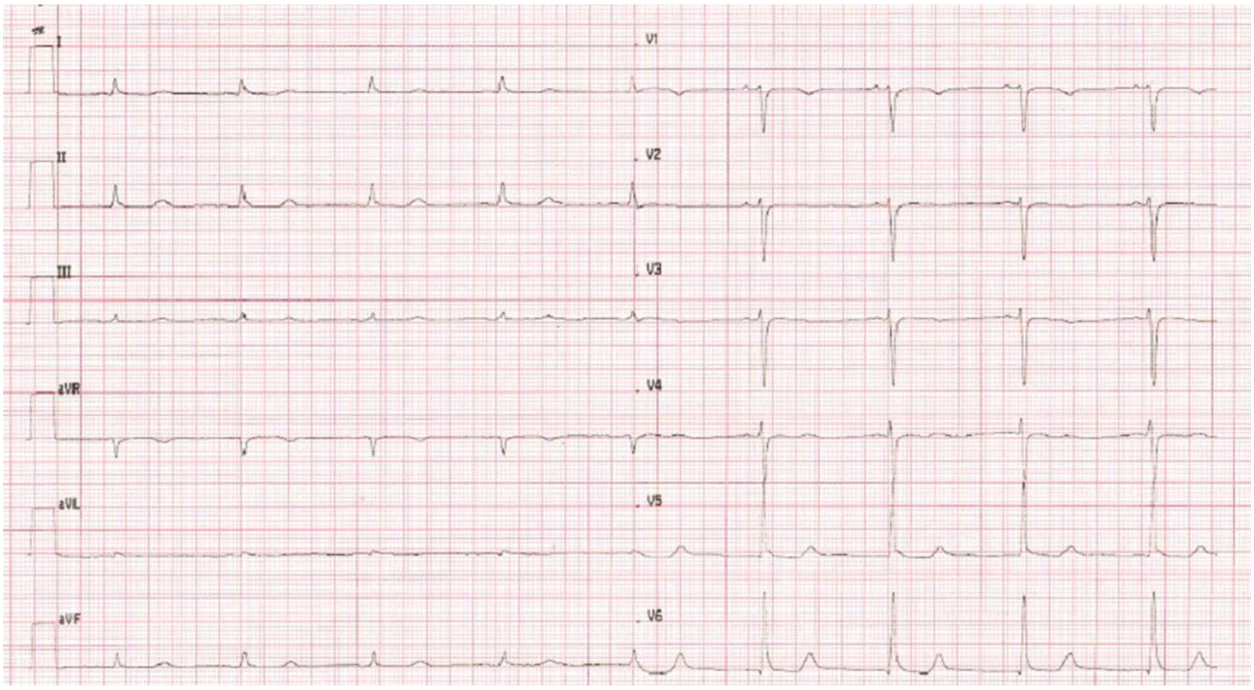

Figure 2. ECG record after 6 hours of treatment — sinus rhythm 53/min, the axis of the heart is normal, PQ 120 ms, QRS 100 ms, prolonged QT/Qtc 528/512 ms, no abnormalities of the ST interval from the T wave

complex and QT interval [2]. These abnormalities also include the Osborn wave characterized by narrowing of the QRS complex at the junction of the R-wave and the ST interval, which imitates the R' wave [2]. It is also called camel hump, J-wave or, less frequently, delta wave [2]. In 1957 Joseph Osborn published the results of research on the influence of hypothermia on the cardiovascular and respiratory system [4]. On the basis of experimental studies, he concluded that this wave appears during hypothermia if it is accompanied by acidosis and abnormal ventilation and disappears after body temperature normalization [4]. The mechanism of its creation is unknown. Hypotheses focus mainly on differences in functional potential of cardiomyocytes and differences in gradients between epicardium and endocardium cells in hypothermia, which results in changes of early repolarization period, *i.e.* phase 1 of functional potential, which can be observed in ECG [5]. The Osborn wave is not a pathognomonic change for accidental hypothermia and also occurs in therapeutic hypothermia [5], hypercalcemia, subarachnoid hemorrhage, craniocerebral trauma, Prinzmetal angina, ventricular fibrillation and sudden cardiac arrest [6]. It is most frequently observed in lower and lateral wall leads (V3–V6) [2], and if it occurs in V1–V2, it requires differentiation with Brugada syndrome [6].

In the described cases the correlation between J-wave amplitude and body temperature [7], the number of leads in which the wave was observed [2], the relationship with acid-base imbalance and gas exchange [4] and ventricular arrhythmias [5] were analyzed. However, individual reports did not allow for a significant correlation to be established. The prognostic significance of the wave is unknown and requires further observation, especially in the situation of increasingly widespread use of hypothermia in the treatment of various diseases [5].

As in the case described above, the Osborn wave has a transient character and subsides with the normalization of body temperature [4]. Awareness of these electrocardiographic changes is helpful in the differential diagnosis of life-threatening conditions, especially in the Emergency Medical Departments.

Conflict of interest

Authors do not report any conflict of interest.

Streszczenie

Hipotermia może powodować zaburzenie funkcji wielu narządów. Każde obniżenie ciepłoty ciała wpływa na układ sercowo-naczyniowy. Już przy niewielkim spadku temperatury obserwuje się reakcje kompensacyjne w układzie krążenia, a przy znacznej hipotermii dochodzi do zagrażających życiu zaburzeń rytmu serca. Opisano przypadek znalezionego w śniegu bezdomnego, u którego w przebiegu umiarkowanej hipotermii w zapisie elektrokardiograficznym (EKG) obserwowano napadowe migotanie przedsionków i falę Osborna (tzw. fala J). Po zastosowanym leczeniu i przywróceniu normalnej ciepłoty ciała powrócił rytm zatokowy, a morfologia zespołów QRS była prawidłowa. Na podstawie przypadku i literatury w niniejszym artykule przedstawiono zaburzenia kardiologiczne w hipotermii objawiające się falą Osborna w badaniu EKG.

Słowa kluczowe: przypadkowa hipotermia, fala Osborna, fala J, elektrokardiogram

Folia Cardiologica 2019; 14, 1: 75–78

References

1. Truhlář A, Deakin CD, Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2015: section 4. Cardiac arrest in special circumstances. Resuscitation. 2015; 95: 148–201, doi: 10.1016/j.resuscitation.2015.07.017, indexed in Pubmed: 26477412.
2. de Souza D, Riera AR, Bombig MT, et al. Electrocardiographic changes by accidental hypothermia in an urban and a tropical region. J Electrocardiol. 2007; 40(1): 47–52, doi: 10.1016/j.jelectrocard.2006.08.094, indexed in Pubmed: 17027018.
3. Wanscher M, Agersnap L, Ravn J, et al. Outcome of accidental hypothermia with or without circulatory arrest: experience from the Danish Præstø Fjord boating accident. Resuscitation. 2012; 83(9): 1078–1084, doi: 10.1016/j.resuscitation.2012.05.009, indexed in Pubmed: 22634431.
4. Osborn JJ. Experimental hypothermia; respiratory and blood pH changes in relation to cardiac function. Am J Physiol. 1953; 175(3): 389–398, doi: 10.1152/ajplegacy.1953.175.3.389, indexed in Pubmed: 13114420.
5. Yan GX, Antzelevitch C. Cellular basis for the electrocardiographic J wave. Circulation. 1996; 93(2): 372–379, indexed in Pubmed: 8548912.
6. Maruyama M, Kobayashi Y, Kodani E, et al. Osborn waves: history and significance. Indian Pacing Electrophysiol J. 2004; 4(1): 33–39, indexed in Pubmed: 16943886.
7. Omar HR, Camporesi EM. The correlation between the amplitude of Osborn wave and core body temperature. Eur Heart J Acute Cardiovasc Care. 2015; 4(4): 373–377, doi: 10.1177/2048872614552057, indexed in Pubmed: 25267877.