

Osborn waves during therapeutic hypothermia in a young ST-ACS patient after out-of-hospital cardiac arrest

Fala Osborna w trakcie hipotermii terapeutycznej u młodego pacjenta z ostrym zespołem wieńcowym z uniesieniem odcinka ST, powikłanym nagłym zatrzymaniem krążenia

Filip M. Szymański, Grzegorz Karpiński, Anna E. Płatek, Grzegorz Opolski

Department of Cardiology, Medical University of Warsaw, Poland

Abstract

A 37 year-old male patient was admitted to the intensive care unit after an out-of-hospital cardiac arrest due to ventricular fibrillation in a course of ST-segment elevation acute coronary syndrome. On admission, the patient was unconscious with a Glasgow Coma Scale (GCS) score of 5. A percutaneous coronary intervention and mild therapeutic hypothermia (HT), defined as maintaining body temperature between 32°C and 34°C, were performed. During HT on ECG, we observed Osborn waves, which resolved spontaneously after re-warming. After five days of recovery, the patient scored 15 on GCS and did not show any neurological deficits.

Key words: acute coronary syndrome, cardiac arrest, hypothermia therapy

Kardiol Pol 2013; 71, 1: 88–90

INTRODUCTION

Mild hypothermia therapy (HT), defined as maintaining body temperature within the range of 32°C to 34°C, is a procedure of great beneficial potential in preserving cardiac and neurological functions after out-of-hospital cardiac arrest. The worldwide growing acceptance and expanding use of HT emphasises the need for it to be better understood. There are certain non-pathological ECG findings, such as Osborn waves, which may occur during the procedure that physicians must be aware of so that they do not feel the need to terminate the HT prematurely [1].

CASE REPORT

A 37 year-old male patient was admitted to the intensive care unit after an out-of-hospital cardiac arrest due to ventricular fibrillation (VF) in the course of ST-segment elevation acute coronary syndrome. Cardiopulmonary resuscitation (CPR) was initiated immediately by bystanders using an automatic exter-

nal defibrillator. On admission, the patient was unconscious, scored 5 points on the Glasgow Coma Scale (GCS), and his blood pressure (BP) was 150/100 mm Hg. A finger blood stick on site revealed a glucose level of 86 mg/dL. A 12-lead initial ECG revealed: sinus rhythm 98 bpm, pathological Q waves in II, III and aVF, ST segment elevation in II, III and aVF by 1.0 mm, and ST segment depression in V_2 – V_6 to 2.0 mm. The patient was transferred to a catheterisation laboratory, where coronary angiography showed chronic occlusion of the right coronary artery and acute occlusion of the intermediate branch. Primary percutaneous coronary intervention (PCI) with implantation of a bare metal stent (BMS) to the intermediate branch was performed, resulting in complete reperfusion (TIMI 3 flow). Because of haemodynamic and neurological status, a decision was made to apply HT procedure. The procedure was initiated in the cath-lab. The patient was sedated, intubated and connected to a respirator (propofol 1–2 mg/kg/h, fentanyl 1–2 µg/kg/h and vecuronium bromide

Address for correspondence:

Filip M. Szymański, MD, PhD, Department of Cardiology, ul. Banacha 1A, 02–097 Warszawa, Poland, tel: +48 22 599 19 58, fax: +48 22 599-19-57, e-mail: filip.szymanski@wum.edu.pl

Copyright © Polskie Towarzystwo Kardiologiczne

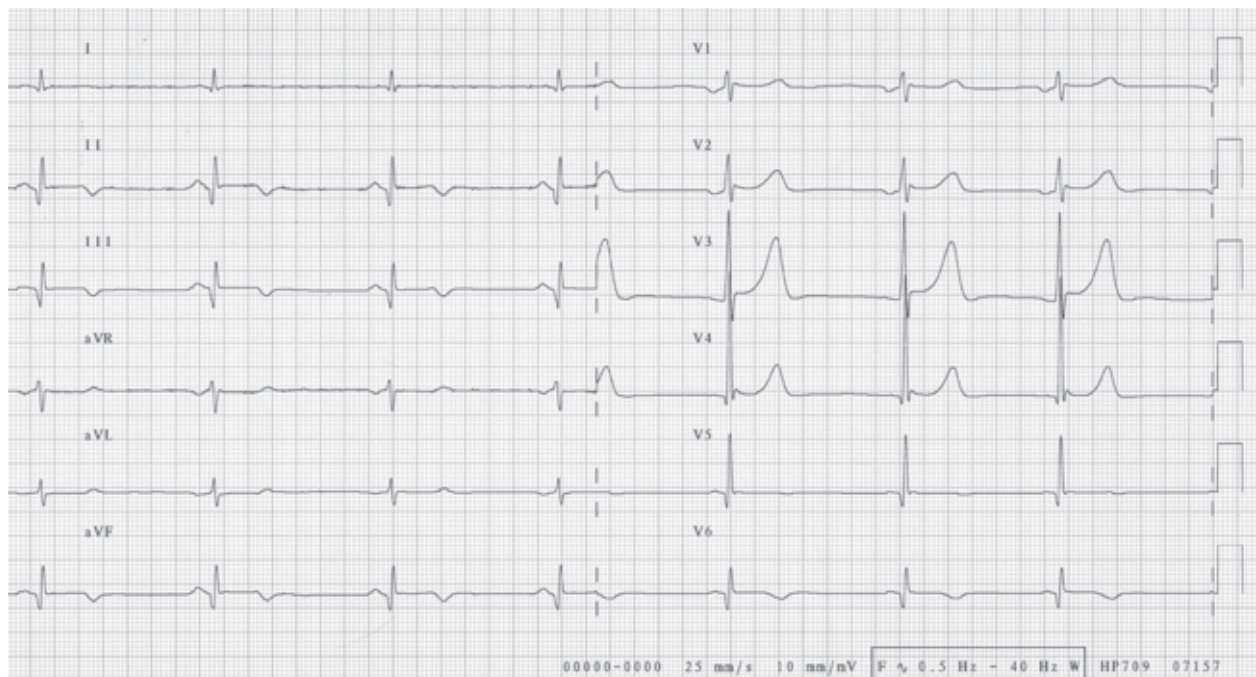


Figure 1. Osborn waves during hypothermia therapy

0.01–0.05 $\mu\text{g}/\text{kg}/\text{min}$). External cooling mattresses were set up, and the cooling phase of the procedure was started. After less than six hours, we achieved desired temperature of $< 34^{\circ}\text{C}$ and proceeded to the maintenance phase of HT. The patient's body temperature was maintained at $33 \pm 0.8^{\circ}\text{C}$, HR 45–59 bpm, BP 95/65–120/60 mm Hg, central venous pressure < 12 mm Hg. On ECG, we observed resolution of the ST segment depression in precordial leads and manifestation of Osborn waves in V_2 – V_4 leads (Fig. 1). After 36 hours, we started to re-warm the patient, obtaining a temperature of $> 35^{\circ}\text{C}$ in the 48th hour. We witnessed a gradual reduction in the amplitude and then a total disappearance of Osborn waves, sinus rhythm 98 bpm, and BP 125/70 mm Hg. An echocardiogram showed slight enlargement of the left ventricle and akinesis of basal and medial segment of the inferior wall. On the fourth day of hospitalisation, sedation was terminated: the patient was breathing on his own and was disconnected from the respirator. He was conscious and oriented, with a GCS score of 15. The patient complained of chest pains, presumably caused by CPR, and headache due to trauma while collapsing during the cardiac arrest. The consulting neurologist found no deficits in neurological functions in an examination. Head computed tomography showed no structural abnormalities. The patient was released home in good overall condition after 21 days of hospitalisation, with zero on the Rankine scale, with no further neurological deficits and neither cardiac nor other symptoms.

DISCUSSION

The International Liaison Committee on Resuscitation recommends using HT (32°C to 34°C for 12–24 hours) in unconscious patients with spontaneous circulation after out-of-hospital cardiac arrest due to VF [2]. It has been shown in both clinical and experimental models that such treatment shows beneficial neuroprotective and cardioprotective properties.

In heart muscle, HT seems to reduce the infarct size and metabolic demand of the myocardium, to increase cellular and mitochondrial membrane integrity and stability, and to improve myocardial microvascular blood flow [3]. The influence of HT on electrical activity of the heart muscle is apparent due to certain ECG findings, which may occur during HT. It appears to have a potential of giving significant prolongation of PR and QTc intervals, heart rate decrease, mild arrhythmia and, rarely, VF (especially when associated with low potassium levels) [4]. But the most obscure finding, which emerged also in our patient, is the Osborn wave (J waves) defined as a late delta wave seen at the end of the QRS complex (or as a small secondary R wave (R')). Named after John J. Osborn, who described it first in 1953 [5], the Osborn wave seems to be caused by action potential of M and epicardial cells, mediated by a transient outward current (I_{to}) that does not occur in the endocardium, originating in a transmural voltage gradient responsible for the spike-and-dome morphology of the end of the QRS complex [6].

An Osborn wave can occur in hypothermia as well as in hypercalcaemia. In our patient, we observed the waves in the V_2 - V_4 leads, when the body temperature dropped below 34°C. After re-warming, the patient's Osborn wave resolved spontaneously.

The second, but equally important, action of HT is neuroprotection. It has been proven that HT modifies cerebral metabolism of oxygen and glucose, reduces brain oedema, and lowers the risk of thrombosis and epileptic activity through electrical stabilising properties [3]. Most importantly, it is also associated with an improvement in neurological outcome and 6-month survival after cardiac arrest due to VF [7]. Our patient was brought to the clinic unconscious with a GCS score of 5, which meets the criteria of severe brain injury. After HT and five days of recovery, his re-evaluated GCS score was 15 and he showed no neurological deficits at all. The applied treatment brought about a tremendous improvement in the patient's health condition and long-term prognosis. HT presumably helped prevent him from suffering physical and neurological disability.

The growing use of HT is fuelling expectations and giving rise to new treatment options in difficult cases such as patients with out-of-hospital cardiac arrests. But it requires up-

to-date knowledge and understanding of its mechanisms from the applying physician.

Conflict of interest: none declared

References

1. Mottillo S, Sharma K, Eisenberg MJ. Therapeutic hypothermia in acute myocardial infarction: a systematic review. *Can J Cardiol*, 2011; 27: 555–561.
2. Nolan JP, Morley PT, Vanden Hoek TL et al. Therapeutic hypothermia after cardiac arrest: an advisory statement by the advanced life support task force of the International Liaison Committee on Resuscitation. *Circulation*, 2003; 108: 118–121.
3. Delhaye C, Mahmoudi M, Waksman R. Hypothermia therapy: neurological and cardiac benefits. *J Am Coll Cardiol*, 2012; 59: 197–210.
4. Lebedez P, Meiners J, Samol A et al. Electrocardiographic changes during therapeutic hypothermia. *Resuscitation*, 2012; 83: 602–606.
5. Osborn JJ. Experimental hypothermia: respiratory and blood pH changes in relation to cardiac function. *Am J Physiol*, 1953; 175: 389–398.
6. Yan GX, Antzelevitch C. Cellular basis for the electrocardiographic J wave. *Circulation*, 1996; 93: 372–379.
7. Bernard SA, Gray TW, Buist MD et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med*, 2002; 346: 557–563.