



Original Contribution

Osborn wave in hypothermia and relation to mortality

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ARTICLE INFO

Article history:

Received 2 March 2018

Received in revised form 17 August 2018

Accepted 18 August 2018

ABSTRACT

Background & aim: The aim of this study was to compare hypothermia patients with and without an Osborn wave (OW) in terms of physical examination findings, laboratory results, and clinical survival.

Methods: The study was carried out retrospectively on hypothermic patients. The hypothermic patients were divided into two groups. Group 1 comprised patients with OW on electrocardiogram (ECG), and Group 2 comprised patients without OW on ECG. The Mann–Whitney *U* test was used to compare the two groups, and the relationships between the variables and the presence of OW and mortality were analyzed with ANOVA. A value of $p < 0.05$ was considered statistically significant.

Results: OW was detected on ECG of 41.9% of the patients (Group 1). The mean body temperature was 30.8 ± 4.1 °C in Group 1 and 33.3 ± 1.6 °C in Group 2 ($p = 0.106$). The mean creatinine level was 1.01 ± 0.6 mg/dl in Group 1 and 0.73 ± 0.5 mg/dl in Group 2 ($p = 0.046$). The mean bicarbonate level was 15.9 ± 3.8 mmol/l in Group 1 and 18.6 ± 3.5 mmol/l in Group 2 ($p = 0.038$). A relationship was determined between the presence of OW and pH, bicarbonate, and creatinine levels ($p = 0.026$; 0.013; 0.042, respectively). The mortality rate was 69.2% in Group 1 and 77.8% in Group 2 ($p = 0.689$).

Conclusion: Although there is a relationship between the decrease in bicarbonate levels, changes in kidney functions that cause acidosis, and the presence of OW, it has no effect on mortality. The presence of OW in hypothermic patients is insufficient to make a decision regarding mortality.

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1. Introduction

An Osborn wave (OW) is a deflection wave in the shape of a dome formed after the QRS complex in electrocardiography (ECG), which is also known as the “Giant J wave” [1,2]. Although OW is frequently observed in hypothermic patients, it has also been reported in normothermic patients as a result of metabolic imbalances such as hypokalemia, hypocalcemia, diabetic ketoacidosis, and in other conditions such as neuroleptic medication, Brugada syndrome, Chagas disease, sepsis, ischemic heart disease, cardiac arrest, and brain damage [3–5]. Therefore, in addition to a decrease in body temperature, the development of OW is thought to be particularly caused by acid–base imbalance (acidosis) and electrolyte disturbances [6]. Hypothermia is defined as a body temperature < 35 °C and is divided into three categories of mild (35 – 32 °C), moderate (32 – 28 °C), and severe (< 28 °C) [7]. The rate of

OW in hypothermic patients differs depending on the temperature. Prevalence rates have been reported as 10.7% in mild hypothermia, 75% in moderate hypothermia, and 100% in severe hypothermia, in which fatal heart rhythms are more frequently observed and the patient’s clinical findings worsen [8–10]. It has therefore been suggested that the presence of OW in hypothermic patients increases mortality, but the opposite view has also been reported [11–15].

The purpose of this study was to compare patients diagnosed with hypothermia following emergency department assessment, and with or without OW on ECG, in terms of physical examination findings, laboratory results, and clinical survival.

2. Methods

2.1. Study design

Following receipt of local ethics committee approval (No: 2017-16/06), the study was conducted retrospectively, with patients admitted to the emergency departments of Kirikkale University Faculty of

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Medicine Hospital and Cekirge State Hospital and diagnosed with hypothermia between January 1, 2012, and December 31, 2017.

2.2. Selection of patients and data collection

All patient data were retrieved from the hospital computer system and archived records. The records were searched for ICD-10 codes starting with “T.33, T.34, T.35, T.68, T.69, or X.31” associated with hypothermia and frostbite, in order to access data related to hypothermia. Hypothermia was treated and managed following the algorithms developed for this purpose [16]. The patients were fully monitored during the follow-up period. Both infrared thermometers (Freely FT3010 infrared thermometer, Zhongshan City, Guangdong, China) and rectal thermometers used for body core temperature measurement (Arctic Sun@5000 Temperature Management System, Medivance, Louisville, USA) were employed in the measurement of body temperature. Temperature was recorded in “degree Celsius (°C)”. J-point elevation or the OW is defined as a 1-mm height elevation at the end of the QRS complex occurring in two consecutive beats. ECGs were reviewed by two emergency medicine specialists blinded to the temperature of the patient at the time of each ECG. Cardiac rhythm was recorded for each ECG (initially and finally). The presence or absence of the classic J-point elevation characteristic of hypothermia was examined, and the height of the elevation was noted. J-point elevation (sometimes referred to as OW) was defined as a 1-mm height elevation at the end of the QRS complex occurring in two consecutive beats.

The hypothermic patients were divided into two groups based on the observation of OW on ECG:

- Group 1: patients with OW on ECG
- Group 2: patients with no OW on ECG.

Patients with a history of Chagas disease, or Brugada syndrome, or patients with intracranial bleeding, or ischemic heart disease accompanying hypothermia, patients who were dead on arrival at the ED, patients undergoing therapeutic hypothermia, and subjects under the age of 18 years were excluded from the study.

2.3. Statistical analysis

Statistical analysis was performed on SPSS 21.0 (IBM SPSS Statistics 21.0, IBM Corporation, Armonk, NY, USA) software. The Kolmogorov-Smirnov test was used in the analysis of normal distribution. Parametric data were expressed as the mean and \pm standard deviation (SD), and categorical variables as number (n) and percentage (%). The Mann-Whitney *U* test was used for intergroup comparisons. The effects of variables on OW formation and mortality were analyzed using ANOVA. A value of $p < 0.05$ was regarded as statistically significant.

3. Results

A total of 42 patients diagnosed with hypothermia were admitted to our hospital emergency departments during the study period. After exclusion of 11 patients who met the exclusion criteria or for whom data were unavailable, the study was completed with 33 subjects. OW was detected in 13 (41.9%) patients (Group 1). The mean age of the entire study group was 45.3 ± 15.8 years (range 19–82 years), 41.4 ± 17.7 years in Group 1 and 48.5 ± 14.1 years in Group 2. There was no difference between the groups in terms of age ($p = 0.183$). Males represented 74.2% ($n = 23$) of all the patients in the study, as 76.9% ($n = 10$) of Group 1, and 72.2% ($n = 13$) of Group 2. There was no difference between the two groups in terms of gender ($p = 0.825$). The demographic data of the hypothermic patients are presented in Table 1 and the data for patients with OW in Table 2.

Table 1
Demographic data of hypothermic patients.

Parameter	Group 1 (n = 13)	Group 2 (n = 18)	Total (n = 31)
	n(%)	n(%)	n(%)
Hypothermia classification			
• Mild (32–35 °C)	7 (53.8)	13(72.2)	20(64.5)
• Moderate (28–32 °C)	2(15.4)	5(27.8)	7(22.6)
• Severe (<28 °C)	4(30.8)	–	4(12.9)
Where hypothermia developed			
• Outside (homeless, on the street, on exposed land)	8(61.6)	13(72.2)	21(67.7)
• Living in a tent	4(30.8)	2(11.1)	6(19.3)
• Nursing home	1(7.7)	3(16.7)	4(12.9)
Hypothermia etiology			
• Accidental	6(46.1)	11(61.1)	15(48.4)
• Carbon monoxide poisoning	4(30.8)	–	4(12.9)
• Decreased cold tolerance	1(7.7)	7(38.9)	8(25.8)
• Alcohol or drug abuse	4(30.8)	4(22.2)	8(25.8)
• Presence of chronic disease	3(23.1)	9(50)	12(38.7)
• Trauma	2(15.4)	3(16.7)	5(16.1)

Mean body temperature (MBT-rectal) was 30.8 ± 4.1 °C in Group 1 and 33.3 ± 1.6 °C in Group 2. There was no difference in MBT between the groups ($p = 0.106$). There was also no difference between the groups in terms of other physical examination findings (Table 3).

In terms of laboratory results, mean creatinine levels were 1.01 ± 0.6 mg/dl in Group 1, and 0.73 ± 0.5 mg/dl in Group 2. Creatinine levels differed significantly between the groups ($p = 0.046$). Mean bicarbonate levels were 15.9 ± 3.8 mmol/l in Group 1, and 18.6 ± 3.5 mmol/l in Group 2. The difference was also significant ($p = 0.038$). There was no difference between the groups in terms of other laboratory results (Table 3).

In the mortality analysis, 69.2% ($n = 9$) of the patients in Group 1 and 77.8% ($n = 14$) of those in Group 2 did not survive. There was no difference between the groups in terms of mortality rates ($p = 0.592$) (Table 3). No relationship was determined between mortality rates and laboratory values in the two groups (Table 4). The presence of OW was correlated with some laboratory parameters; correlations were determined between the presence of OW and pH ($p = 0.026$), bicarbonate levels ($p = 0.013$), and creatinine ($p = 0.042$) levels. No relationship was determined between presence of OW and partial oxygen pressure (PO_2) ($p = 0.186$), partial carbon dioxide pressure (PCO_2) ($p = 0.069$), or other laboratory results. In the evaluation of relationships between mortality and the presence of OW and laboratory results, no relationship was determined between mortality and the presence of OW ($p = 0.062$), pH ($p = 0.09$), bicarbonate levels ($p = 0.167$), creatinine levels ($p = 0.127$), PO_2 ($p = 0.285$), PCO_2 levels ($p = 0.196$), or other laboratory results.

4. Discussion

The results obtained from this study show a relationship between blood pH and bicarbonate levels that maintain the acid-base balance (acidosis), and kidney functions and the development of OW in hypothermic patients. However, no relationship was determined between mortality and any physical examination findings or laboratory results.

Although OW is characteristic of hypothermia, it is not pathognomonic [4]. Moreover, detection of OW on the initial ECG in hypothermic patients may not provide accurate information regarding the degree of hypothermia [17]. Previous studies have emphasized that the development of OW is not solely related to body temperature, as the wave disappears when pH values are normalized and that it may be related to acidosis, rather than to hypothermia [1,18]. Similarly, Vasallo et al. stated that the OW observed in hypothermic patients is caused by electrolyte disturbances, particularly acid-base imbalance (acidosis), and not by the decrease in body temperature [6]. Omar et al. reached a

Table 2
Physical examination findings and laboratory results of patients with OW.

Case	Age/sex	Place of hypothermia development	Hypothermia etiology	CBT (°C)	Pulse (min)	ECG findings		Clinical outcome
						Initial	Final	
1	82/f	Nursing home	DM, Alzheimer, osteoporosis (confined to bed due to femur fracture)	31.3	30	SB	Complete AV block, Asystole	Death
2	40/m	Street	Epilepsy (postictal period)	34.8	42	SB	SB	ICU
3	52/m	Nature land	Direct cold exposure (be caught in the blizzard)	27.1	28	NSR, Prolonged QT	VF, asystole	Death
4	56/m	Street	Alcohol abuse	33.9	44	SB	First degree AV block, SB	ICU
5	44/m	Living in a tent	CP	33.6	68	NSR	NSR	Death
6	42/f	Living in a tent	CP, and DM	33.0	52	SB	VF, asystole	Death
7	21/m	Living in a tent	CP	33.9	72	NSR	NSR	ICU
8	19/f	Living in a tent	CP	32.6	62	SB	SB, Asystole	Death
9	28/m	Nature land (Riverside)	Alcohol abuse, and fall in cold water	27.9	28	SB	VF, asystole	Death
10	32/m	Homeless	Direct cold exposure	34.7	54	SB	SB	ICU
11	52/m	Homeless	DM, alcohol abuse	27.8	24	SB	VF, asystole	Death
12	49/m	Homeless	Direct cold exposure	31.3	32	NSR	Complete AV block, asystole	Death
13	21/m	Homeless	Alcohol abuse	27.2	28	SB	VF, asystole	Death

CBT, Core Body Temperature; CP, Carbon monoxide Poisoning; DM, Diabetes Mellitus; ECG, Electrocardiography; ICU, Intensive Care Unit; NSR, Normal Sinus Rhythm; SB: Sinus Bradycardia, VF: Ventricular Fibrillation.

different conclusion and suggested that the development of OW is not related to the electrolytes involved in pH or acid-base balance nor to PO₂. However, they also added that the small number of cases in their study was insufficient to provide definitive data, as pH was tested in only one-third of their patients [17]. In the current study, although the pH level was lower in the group with OW compared to the group without OW, the difference between them was not significant. There was also no difference between the groups in terms of PO₂ or PCO₂, which are among the factors determining pH levels. These two factors were also determined to have no effect on the development of OW. Bicarbonate levels were significantly lower in the patients with OW compared to those without. Moreover, the decrease in both bicarbonate and pH levels (acidosis) was related to the development of the OW. Creatinine levels indicating kidney function, as one of the principal organs regulating acid-base levels, were higher in patients with OW. Although this increase did not cause acute kidney failure, the rise in creatinine levels was statistically significant. These results are similar to those of previous studies reporting a relationship between OW and acidosis. A decrease in levels of bicarbonate, one of the blood buffer molecules, will reduce pH levels and lead to acidosis. The impairment of kidney functions, another buffer mechanism, can also be considered to facilitate OW formation.

Table 3
Comparison of the hypothermia groups.

	Group 1 (n = 13) mean ± SD	Group 2 (n = 18) mean ± SD	p
Physical examination findings			
• Core body temperature (rectal, °C)	31.5 ± 2.9	33.3 ± 1.6	0.106
• Pulse (min)	44.9 ± 16.5	53.6 ± 8.3	0.157
• Respiration rate (min)	9.7 ± 1.8	9.8 ± 1.1	0.737
• P _{Systolic} (mm Hg)	94.2 ± 10.7	96.6 ± 15.9	0.622
• P _{diastolic} (mm Hg)	54.9 ± 8.8	51.8 ± 12.3	0.540
Laboratory results			
• Glucose (mg/dl)	91.5 ± 32.3	108.6 ± 37.9	0.056
• Creatinine (mg/dl)	1.01 ± 0.6	0.73 ± 0.5	0.046
• Sodium (mmol/L)	135.9 ± 2.2	135.4 ± 2.8	0.798
• Potassium (mmol/L)	4.4 ± 0.9	4.7 ± 0.9	0.312
• Calcium (mg/dl)	9.7 ± 0.9	9.5 ± 0.4	0.828
• White blood cell (10 ³ /mm ³)	12.4 ± 8.9	11.6 ± 6.3	0.890
• Hemoglobin (g/dl)	13.8 ± 1.8	13.8 ± 1.8	0.984
• Platelets (10 ³ /mm ³)	236.5 ± 121.7	262.8 ± 80.1	0.441
• pH	7.21 ± 0.2	7.29 ± 0.1	0.062
• HCO ₃ (mmol/L)	15.9 ± 3.8	18.6 ± 3.5	0.038
• PO ₂ (mm Hg)	69.9 ± 26.1	55.3 ± 11.1	0.170
• CO ₂ (mm Hg)	54.2 ± 10.3	47.3 ± 7.5	0.062
Mortality	9 (69.2)	14 (77.8)	0.592

HCO₃, Bicarbonate; PO₂, Partial oxygen; PCO₂, Partial carbon dioxide.

Studies have suggested that observation of OW in hypothermic patients may be linked to mortality [11–13]. Such a relationship may be expected, since OW occurs in all hypothermic patients with a body temperature < 28 °C and with further deteriorations in clinical condition [10]. Many arrhythmias, and particularly ventricular fibrillation (VF), occur in severe hypothermia [19,20]. Aissou et al. reported a higher prevalence of VF and in-hospital mortality among cases with OW on ECG and a previous history of coronary artery disease [21]. While some studies have reported a relationship between mortality and OW, others have suggested no relationship between the OW and clinical outcomes or mortality [14,15]. Omar et al. suggested that OW amplitude does not provide any information about clinical course and mortality, whereas other studies have proposed that mortality is particularly closely associated with pre-existing diseases in hypothermic patients [17]. The pH levels in our study were lower in patients with OW than in those with no OW, but the difference was not statistically significant. Although pH, PO₂, PCO₂, and bicarbonate determine acidosis or alkalosis in blood gasses, the only statistically significant difference between the two groups of patients in the current study was in bicarbonate levels. The lower pH in our Group 1 patients, those with an OW, may be attributed to the low bicarbonate levels. Although OW was seen in patients with acidosis and low bicarbonate levels, no relationship was determined with mortality. Furthermore, no relationship was determined between mortality and other laboratory values. This suggests that observation of an OW in hypothermic patients by itself provides no indication regarding mortality. To draw any conclusion regarding mortality in hypothermic patients, it can be considered essential to know the patient's age, gender, history, existing diseases, medications, type of exposure to hypothermia, duration of exposure, and many other factors.

Table 4
Relations between mortality and laboratory results.

Laboratory results	Death (n = 23) mean ± SD	No death (n = 8) mean ± SD	p
• Glucose (mg/dl)	101.2 ± 40.5	91.4 ± 7.8	0.580
• Creatinine (mg/dl)	0.97 ± 0.6	0.66 ± 0.2	0.219
• Sodium (mmol/L)	135.5 ± 2.6	135.8 ± 2.4	0.947
• Potassium (mmol/L)	4.5 ± 0.9	4.9 ± 0.7	0.317
• Calcium (mg/dl)	9.6 ± 0.7	9.6 ± 0.5	0.362
• White blood cell (10 ³ /mm ³)	12.4 ± 8.5	14.7 ± 10.5	0.808
• Hemoglobin (gr/dl)	13.8 ± 1.9	13.7 ± 1.2	0.674
• Platelets (10 ³ /mm ³)	254.5 ± 109.1	244 ± 65.8	0.947
• pH	7.24 ± 0.1	7.3 ± 0.1	0.09
• HCO ₃ (mmol/L)	16.6 ± 4.3	18.5 ± 2.9	0.167
• PO ₂ (mm Hg)	59.1 ± 18.9	68.3 ± 22.4	0.285
• PCO ₂ (mm Hg)	51.5 ± 9.9	46.4 ± 6.2	0.196

HCO₃, Bicarbonate; PO₂, Partial oxygen; PCO₂, Partial carbon dioxide.

5. Limitations

There are a number of limitations to this study. First, since hypothermia usually occurs in winter, when exposure to cold is greatest, the number of patients included in the study was limited, and this may have affected the statistical results. Second, OW can also occur due to hypokalemia or hypocalcaemia. Moreover, hypothermia causes both OW and biochemical and metabolic changes (hypokalemia, hypocalcaemia, hypo/hyperglycemia, etc.) [4,5]. The medical backgrounds of the patients admitted to the Emergency Department were therefore investigated in detail during the study, and it was assumed that electrolyte disturbances causing OW had not existed previously and that the changes in the laboratory results resulted from hypothermia. Another limitation is that only the presence or absence of OW was investigated on ECG. There is a need for further studies investigating other arrhythmias in hypothermia to be able to shed light on this.

6. Conclusion

The development of OW in hypothermic patients is not solely related to a decrease in body temperature. Decreases in the levels of buffer molecules that result in acidosis, and particularly bicarbonate levels, and impairment of the kidney functions that regulate the acid-base balance are also associated with the development of OW, but not with mortality. The detection of OW in hypothermic patients is not sufficient to make a decision regarding mortality, and several factors should be evaluated together.

Conflict of interest

None.

Financial support

None.

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