

Case Report

J-wave change during rewarming therapy for accidental hypothermia

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Background: J waves are abnormal electrocardiogram findings that indicate an elevation at the junction between the QRS complex and the ST segment. Hypothermia is associated with fetal arrhythmia, along with the increase of J-wave manifestation.

Case Presentation: A 68-year-old woman with a medical history of old cerebral infarction and dementia was admitted to the emergency department with accidental hypothermia. An admission, electrocardiogram (ECG) showed prominent J waves with the highest amplitude recorded in limb and precordial leads. Continuous ECG monitoring was recorded during the rewarming therapy. As the body temperature increased, the amplitude of J waves became lower and disappeared. This case clearly showed that the degree of core body temperature is related to the height of the J-wave amplitude. There was no fatal arrhythmia during rewarming therapy.

Conclusion: This case describes serial changes in the J-wave amplitude and morphology during rewarming therapy. Continuous ECG monitoring is important in a patient with severe hypothermia.

Key words: J wave, J-wave amplitude, hypothermia, rewarming therapy

INTRODUCTION

J WAVES, ALSO known as Osborn waves or camel-hump waves, are characterized by notching or slurring at the terminal part of the QRS complex on a standard 12-lead electrocardiogram (ECG). They are observed in various pathological conditions.^{1,2} Previous studies have reported a high prevalence of J wave as a risk marker for fatal arrhythmia under specific conditions, including idiopathic ventricular fibrillation,²⁻⁴ Brugada syndrome,²⁻⁴ vasospastic angina, acute myocardial infarction, and hypothermia.⁵

Accidental hypothermia is defined as an involuntary decrease in the core body temperature to <35°C.⁶ Severe hypothermia (body core temperature < 28°C) is specifically associated with a high risk of sudden cardiac arrest.⁷

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Numerous studies have reported on the relationship between J-wave amplitude and hypothermia.^{8,9} However, the change in the J-wave amplitude according to the rewarming therapy remains unclear. The present study aimed to describe a case of accidental hypothermia, with a change in the J-wave amplitude along with the rewarming of core temperature.

CASE REPORT

IN JANUARY 2016, a 68-year-old woman with a medical history of hypertension, dyslipidemia, old cerebral infarction, and mild dementia was admitted to the emergency department with unconsciousness, disquiet, and severe anasarca. She had been bedridden and living alone. On the particular day with an outside temperature of approximately 0°C, she was lying on the bed at home without heating. Her relatives noticed that she was unconscious and called for an emergency. Her initial heart rate was 15 b.p.m., respiration rate was 8/min, blood pressure and O₂ saturation could not be measured, and urinary bladder temperature was 25.4°C. Respiratory acidosis (pH 7.092, PO₂ 65 mm Hg, and PCO₂ 80 mm Hg) was observed in arterial blood gas. The following laboratory results were obtained: white blood cell count $15.4 \times 10^3/\mu\text{L}$, hemoglobin 10.8 g/L, platelet count

446 $\times 10^3/\mu\text{L}$, C-reactive protein 4.57 mg/mL, total bilirubin 0.5 mg/mL, aspartate transaminase 69 U/L, alanine transaminase 52 U/L, lactate dehydrogenase 266 U/L, blood urea nitrogen 30.0 mg/mL, creatinine 1.06 mg/dL, sodium 140 mEq/L, potassium 4.7 mEq/L, and chloride 104 mEq/L.

According to her initial vital signs, she immediately underwent tracheal intubation without anesthesia and given i.v. atropine 0.5 mg to improve breathing and cardiac output, respectively. After an i.v. injection of atropine, an ECG revealed 85 b.p.m. with indeterminate rhythm, wide QRS complexes, and prominent J waves at the end of the QRS, with the highest amplitude recorded in limb leads I and II and precordial leads V_{3-6} (Fig. 1). External and internal heating (2°C per hour) was immediately applied using an electric heat blanket, a forced-air warming device, and a respiratory warming/humidifying device, because there were no extracorporeal circulation circuit devices in the hospital at this time. Consecutive ECG monitoring during external heating showed that the J-wave amplitude decreased with the increase in the urinary bladder temperature, and the J wave disappeared at 32°C (Fig. 2). Rewarming by external heating was continued until the core body temperature reached 36°C. After the patient regained consciousness, she was extubated the next morning. During rewarming therapy, ventricular premature contractions were observed, but no lethal arrhythmia was observed. The patient had severe systemic edema. A diuretic was used to reduce the body fluid for a while, and she was discharged after complete recovery after 1 month.

DISCUSSION

A CHANGE IN the J-wave amplitude over time due to the rewarming therapy in a patient with accidental hypothermia was demonstrated in this study. Notched J

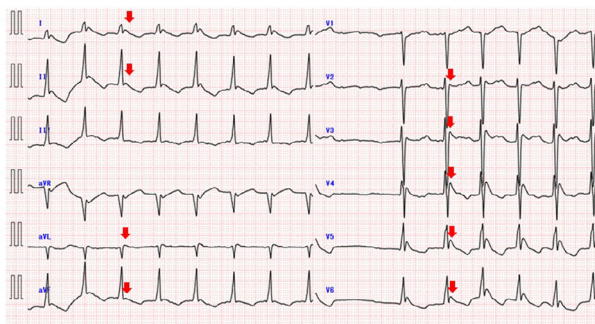


Fig. 1. Initial electrocardiogram after i.v. injection of atropine on admission of a 68-year-old woman with accidental hypothermia. Prominent J waves in limb leads I and II and precordial leads V_{3-6} (red arrows) were observed.

waves were constantly observed with a heart rate of 85 b.p.m. (after atropine injection) and a core temperature of 25.4°C on admission, although the 12-lead ECG was not obtained before an i.v. injection of atropine at the emergency department. During the rewarming therapy for accidental hypothermia, continuous ECG monitoring disclosed that the J-wave amplitude in the inferolateral leads became attenuated with a change in the J-wave morphology from notching to slurring, and finally disappeared with an increased core temperature of 32.0°C. The J waves were not observed in ECG at discharge, and no fatal arrhythmias occurred during her follow-up for 2 years. As shown in Fig. 3, a significant negative correlation was observed between the core body temperature and the J-wave amplitude, indicating a strong relationship between the two. Although previous studies suggested that the degree of hypothermia correlated with the height of J waves,^{8,9} none of them reported the detailed and serial changes in the J-wave amplitude and morphology during rewarming therapy for accidental hypothermia, which was secondary to a medical history of old cerebral infarction and dementia. J waves were observed at the R-ST junction (J point) of the ECG, usually in leads II, III, aVR, and aVF, and the midlateral precordial leads.^{2,5,8} Aizawa et al.¹⁰ reported that J waves were associated with fatal arrhythmia; the clinical significance of this association is under investigation. On the basis of cellular electrophysiology, J waves were caused by a more prominent Ito-mediated spike-and-dome action potential morphology in ventricular epicardium than in endocardium.^{2,5,8} Severe hypothermia could precipitate fatal arrhythmia and lead to a high mortality rate.⁷ Previous studies experimentally described that the J-wave amplitude augmented along with the decreased core temperature in dogs.⁷ A decrease in the core temperature resulted in an increase in the action potential in the epicardium but not in the endocardium, leading to a prominent J wave.^{2,8} The J-point elevation of at least 0.2 mV in inferior leads increased the risk of cardiac death from cardiac causes and fatal arrhythmia.^{3,4} Therefore, careful and frequent recording and evaluation of the 12-lead ECGs are indispensable so as not to miss the change in the J wave to prevent cardiac arrhythmia in patients with severe accidental hypothermia regarding “after drop” phenomenon. Bedside continuous single-lead ECG monitoring might be insufficient to evaluate the change in the J waves in the inferolateral leads.

CONCLUSIONS

THIS STUDY DESCRIBED the detailed and serial changes in the J-wave amplitude and morphology

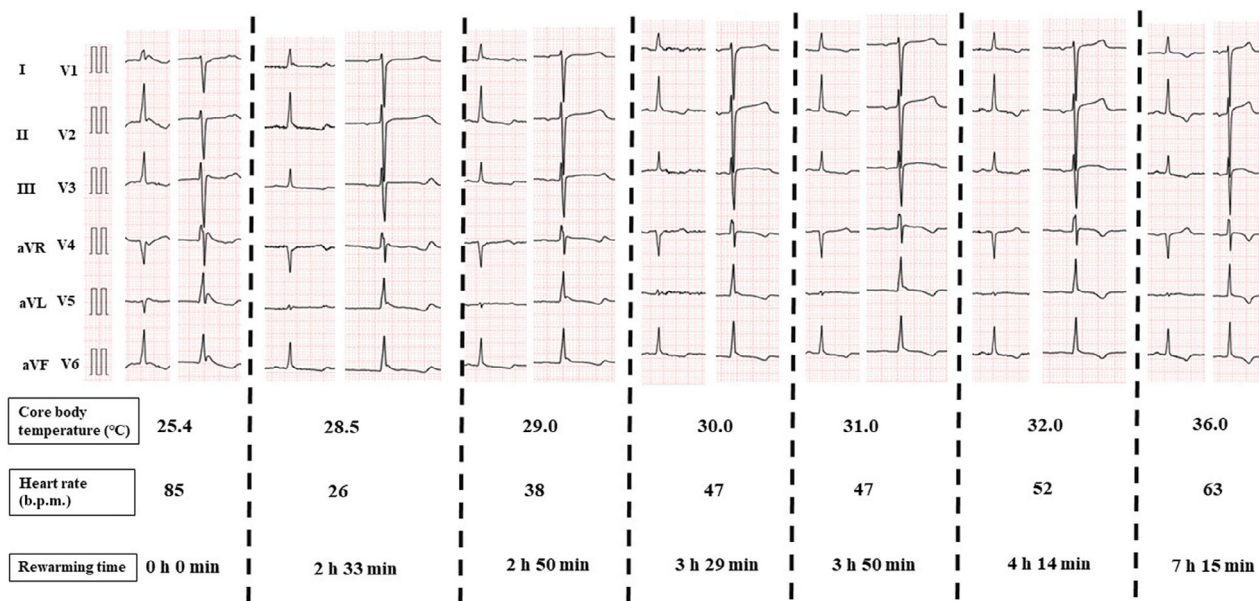


Fig. 2. Serial changes in the J-wave amplitude and morphology in precordial leads at each core body temperature (from 25.4°C to 36.0°C), heart rate, and elapsed time since the initiation of rewarming therapy in a 68-year-old woman with accidental hypothermia. The J-wave amplitude became attenuated with changes in morphology from notching to slurring and disappeared at 32°C.

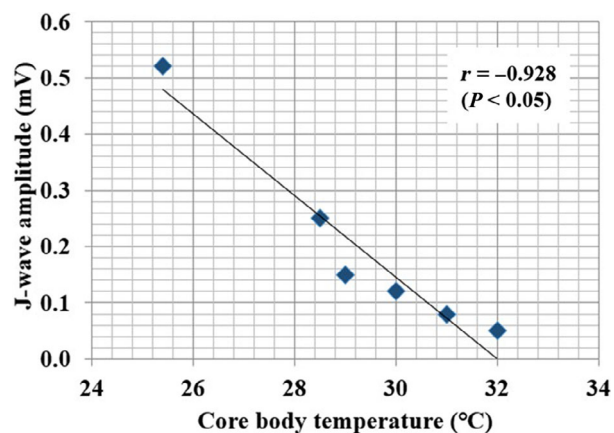


Fig. 3. Relationship between the J-wave amplitude in V_4 lead and the core body temperature in of a 68-year-old woman with accidental hypothermia. The J-wave amplitude was measured from baseline (TP-segment) to peak of notch in V_4 .

during rewarming therapy for accidental hypothermia. It highlighted the importance of continuous ECG monitoring to prevent cardiac fatal arrhythmia in a patient with severe accidental hypothermia.

DISCLOSURES

Approval of the research protocol: N/A.

Informed consent: Yes.

Registry and the registration no. of the study/trial: N/A.

Animal studies: N/A.

Conflict of interest: None.

REFERENCES

- Haïssaguerre M, Derval N, Sacher F, *et al.* Sudden cardiac arrest associated with early repolarization. *N. Engl. J. Med.* 2008; 358: 2016–23.
- Antzelevitch C, Yan GX. J wave syndromes. *Heart Rhythm* 2010; 7: 549–58.
- Tikkanen JT, Anttonen O, Junttila MJ, *et al.* Long-term outcome associated with early repolarization on electrocardiography. *N. Engl. J. Med.* 2009; 361: 2529–37.
- Shinohara T, Takahashi N, Saikawa T, Yoshimatsu H. Characterization of J wave in a patient with idiopathic ventricular fibrillation. *Heart Rhythm* 2006; 3: 1082–4.
- Hada Y, Nishizaki M, Yamawake N, Sakurada H, Hiraoka M. Appearance of J wave in the inferolateral leads and ventricular fibrillation provoked by mild hypothermia in a patient with Brugada syndrome. *Heart Rhythm* 2016; 2: 407–11.
- Zafren K. Out-of-hospital evaluation and treatment of accidental hypothermia. *Emerg. Med. Clin. North Am.* 2017; 35: 261–79.
- Paal P, Gordon L, Strapazzon G, *et al.* Accidental hypothermia—an update. *Scand. J. Trauma Resusc. Emerg. Med.* 2016; 24: 111.

- 8 Yan GX, Antzelevitch C. Cellular basis for the electrocardiographic J wave. *Circulation* 1996; 93: 372–9.
- 9 Aizawa Y, Hosaka Y, Oda H, *et al.* Dynamicity of hypothermia-induced J waves and the mechanism involved. *Heart Rhythm* 2019; 16: 74–80.
- 10 Aizawa Y, Tamura M, Chinushi M, *et al.* Idiopathic ventricular fibrillation and bradycardia-dependent intraventricular block. *Am. Heart J.* 1993; 126: 1473–4.