Clinical Case Report

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A female patient with hypokalaemia-induced J wave syndrome

An unusual case report

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Abstract

Rationale: Prominent J waves can be seen in life-threatening cardiac arrhythmias such as Brugada syndrome, early repolarization syndrome, and ventricular fibrillation. We herein present an unusual case report of hypokalemia-induced J wave syndrome and ST (a part of ECG) segment elevation.

Patients concerns: A 52-year-old woman with chief complaints of chest pain for 2 hours and diarrhea showed a marked hypokalemia (2.8 mmol/L) and slightly elevated creatine kinase-MB (CK-MB) (57.5 U/L). The electrocardiographic (ECG) recording was normal upon admission and computed tomography (CT) aorta angiography excluded an aorta dissection. ECG done 17 hours after admission showed ST segment elevation and elevated J wave in leads II, III and aVF, and fusion of T and U wave in all leads.

Diagnosis: We first thought that the diagnosis of this patient was acute myocardial syndrome.

Intervention: Potassium chloride and oflocaxin treatment was given to the patient.

Outcomes: Laboratory test showed the level of serum potassium ion increased to 3.4 mmol/L and CK-MB did not have any significant change. The infusion of potassium chloride-induced disappearance of the elevated J wave, although QT (a part of ECG) intervals were still longer than that upon admission.

Lessons: This case tells us that hypokalaemia might induce J wave and elevated ST segments which should be distinguished from acute myocardial syndrome.

Abbreviation: CT = computed tomography scan.

Keywords: hypokalemia, J wave, ofloxacin

1. Introduction

J wave syndromes was first reported by Yan et al in 2004 and defined by J wave (elevated J point more than 0.5–1 mm with 20 ms duration) with potentially fatal arrhythmia including Brugada syndrome and early repolarization syndrome which share common cellular mechanism affecting the transient outward potassium current (ITO).[1] Prominent J wave has been used as a marker to identify generation of potentially fatal ventricular arrhythmias.[2] We herein report an unusual case of hypokalemia-induced J wave syndrome.

2. Case presentation

A 52-year-old woman was admitted to our hospital because of acute chest pain with sweating for approximately 2 hours at midnight on February 15, 2017. She denied any history of previous heart disease, hypertension, diabetes, and hyperlipidemia. She was a nonsmoker. There was no family history of sudden death and she has never been on oral contraceptive. The patient had slight diarrhea 2 days before admission, which exacerbated after admission. Physical examination showed a normal heart. No audible murmurs were heard and lungs were clear. However, there was tenderness but no rebound tenderness below the xiphoid. Otherwise, other examinations were normal.

The first ECG done upon admission showed T waves depression and presence of U wave (Fig. 1). After admission, chest pain was gradually relieved within 1 hour. Laboratory test showed hypopotassium (2.8 mmol/L) and slightly elevated CK-MB (57.5 U/L). We initially thought that the diagnose of this patient was acute myocardial syndrome because of the symptom of chest pain, slightly elevated CK-MB (57.5 U/L) and ECG findings (Figs. 1 and 2). Aorta dissection, pulmonary embolism with hypokalemia should also be considered. So we did a computed tomography (CT) aorta and pulmonary artery angiography to rule out aorta dissection and pulmonary

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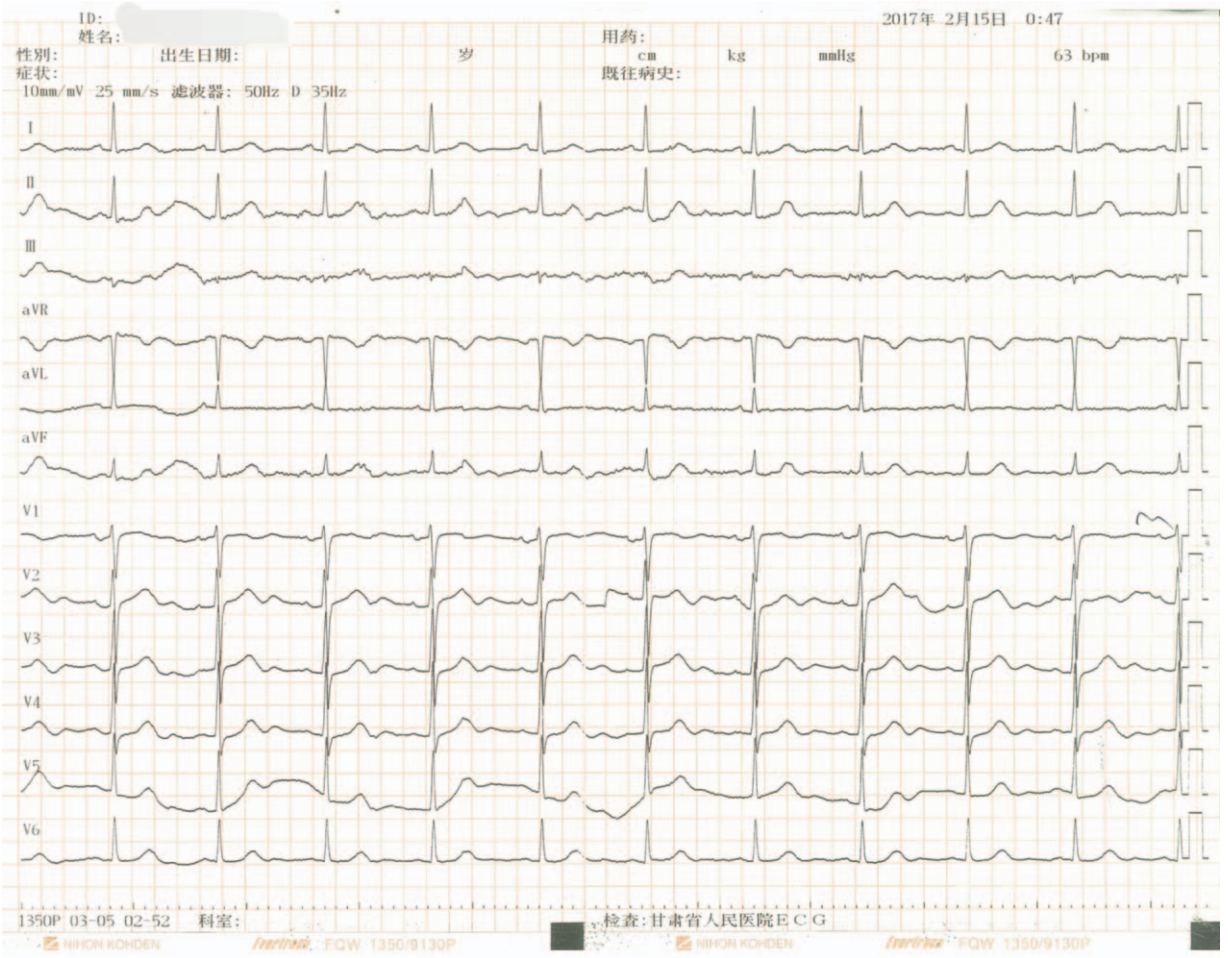

Figure 1. The first ECG done upon admission. ECG = electrocardiographic.

embolism. Accordingly, the patient was given supplementation of potassium chloride (concentration of 10%), rehydration, and antibiotics (ofloxacin).

A second ECG done 17 hours after admission showed ST-segment elevation and J wave in leads II, III, and aVF with obvious fusion of T and U wave in all leads (Fig. 2). At that time, the laboratory test still revealed hypokalemia (2.9 mmol/L) and there was no significant change in CK-MB (53.2 U/L) as compared with that upon admission. Supplement of potassium chloride (10%, the total dose was 240 mL), rehydration solution, and levofloxacin were continued. Abdominal pain and diarrhea were relieved in 1 day. The serum potassium level increased to 3.4 mmol/L. Repeated ECG recording showed disappearance of J wave and ST segment with slight T wave depression, although the QT intervals were still longer than that upon admission (Fig. 3). This shows that our treatment was effective. According to the classification proposed by Antzelevitch and Yan,[2] the patient was assigned as type 2, which is associated with a higher risk of developing ventricular fibrillation and hence cardiac death. Due to personal reasons, the patient wanted to be discharged against medical advice. The patient's condition was associated with high risk of unanticipated events that may occur outside the hospital. Unfortunately, we learned that the patient eventually died from cardiac arrest at discharge.

3. Discussion

J waves with elevated ST segments are often presented with patients with chest pain and suspected acute ST segment-elevated myocardial infarction which is vital important to be diagnosed on time in the adult population.[3] So it is necessary to be acquainted with other conditions, which can induce similar ECG changes. In this case, even the patient complained of chest pain with slightly elevated CK-MB level, there were no dynamic changes in CK-MB. Thus acute coronary syndrome, especially STEMI, was ruled out. Also, CT aorta angiography ruled out aortic dissection. Moreover, the lab test showed obvious hypokalemia. It is well known that the typical ECG appearances in patients with hypokalemia are ST-interval depression, T wave inversion, prolonged PR-interval, and U waves.[4] However, after supplementary with potassium chloride, J waves and elevated ST segments disappeared, supporting that hypokalemia contributed to the formation of J waves and elevated ST segments. In addition, it is possible that the residual changes of prolonged QT intervals were relative to ofloxacin treatment.[5]

Since J wave syndromes were firstly reported by Yan et al in 2004,[6] many studies have shown that J wave was effected by various factors, such as hypoxia, acute ischemic syndrome, delayed ventricular depolarization, and early ventricular repolarization.[7] The cellular basis of the electrocardiographic J wave

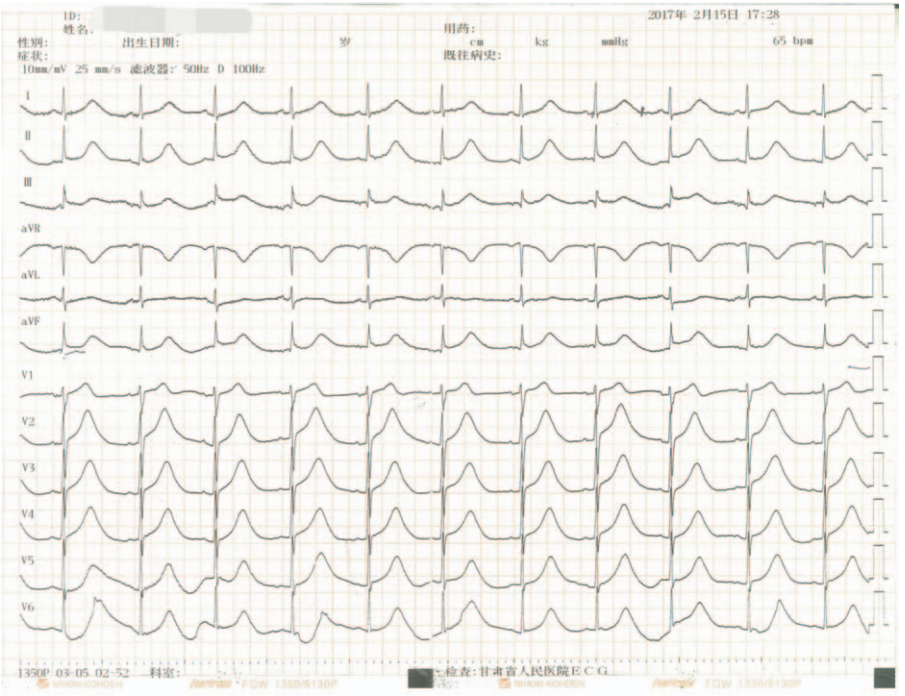

Figure 2. A second ECG done 17 hours after admission. ECG = electrocardiographic.

is the transient outward current that mediates the action potential of epicardium and epicardial cell.[8] Ventricular activation from endocardium to epicardium sequentially corresponds to the J wave of ECG. The transient outward in epicardium is attributed to a transmural voltage gradient at the end of the QRS complex.[8] Low serum potassium might affect multiple ion channels, which disturb Ito current and induce J wave formation and ST segment elevation. The aim in the management of this case was hypokalemia-induced J wave, which is rarely presented in the clinic.

4. Conclusion

According to this case, hypokalemia might induce J wave and elevated ST segments which should be distinguished from acute myocardial syndrome.

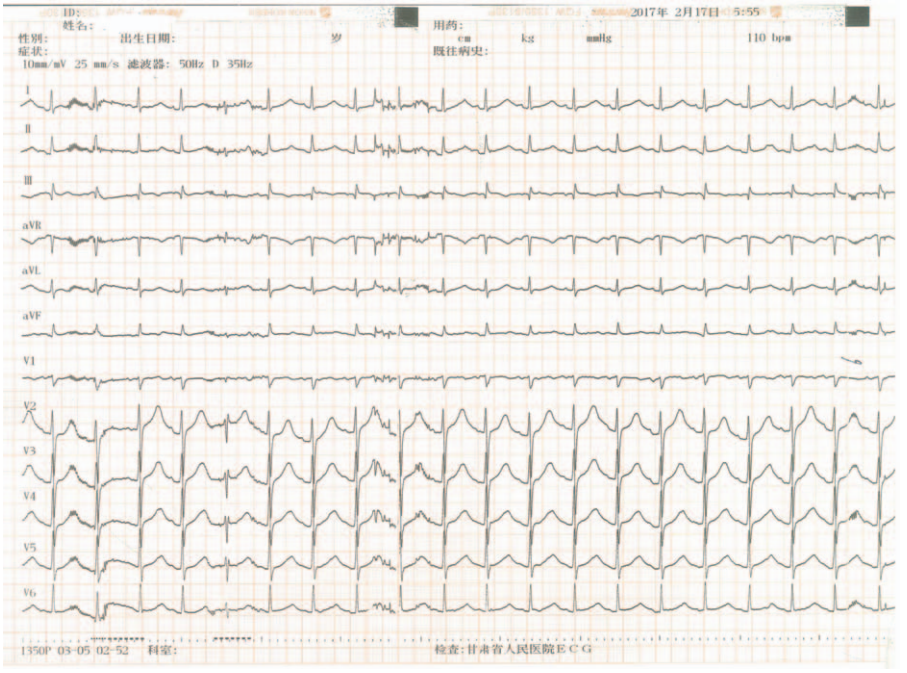

Figure 3. Repeated ECG recording in 1 day after supplementary with potassium chloride. ECG = electrocardiographic.

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