Pilsicainide intoxication mimicking hyperkalemia effectively managed by prompt diagnosis and emergency haemodialysis

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DESCRIPTION

A man in his 80s with paroxysmal atrial fibrillation and stage 3b chronic kidney disease without renal replacement therapy was presented to the hospital because of anorexia for several days and a general sense of weakness for several hours. He had been taking pilsicainide 150 mg/day for paroxysmal atrial fibrillation for the past 16 years. On examination, bradycardia (40/min) and normal blood pressure (122/45 mm Hg) were observed. Laboratory tests revealed hyperkalemia (6.1 mmol/L) and acute kidney injury with a creatine level of 3.84 mg/dL. The initial ECG demonstrated bradycardic atrial fibrillation (40/min) with wide QRS (figure 1A). Calcium gluconate and glucose-insulin therapy were administered based on ECG changes due to the initially suspected hyperkalemia. However, the treatment was ineffective, and the ECG changed to an accelerated idioventricular rhythm of 105/min (figure 1B) and decreased blood pressure. Based on the clinical course, we suspected the diagnosis of pilsicainide intoxication induced by dehydration due to anorexia and subsequent acute kidney injury. Therefore, it was discontinued, and emergency haemodialysis was performed. Two hours after initiating dialysis, the ECG returned to sinus rhythm (figure 1C) and blood pressure returned to baseline.

The serum pilsicainide concentration was 3.54 µg/mL on admission, which was significantly higher than the therapeutic range (0.20–0.90 µg/mL), confirming the diagnosis of pilsicainide intoxication. After haemodialysis, pilsicainide concentration decreased to 2.6 µg/mL. The patient’s postadmission course was uneventful, and he was discharged on the ninth day of hospitalisation. Since paroxysmal atrial fibrillation did not recur without pilsicainide, it was discontinued as is. The patient has no recurrence of the arrhythmia.

Pilsicainide is a pure sodium channel blocker frequently used in East Asia for atrial fibrillation and other tachyarrhythmias as an antiarrhythmic drug with fewer adverse effects. Because most pilsicainide is excreted from the kidney, its dosage should be adjusted according to renal function.1 However, awareness of the need for dose adjustment has been limited among clinicians, and pilsicainide intoxication may often be overlooked. Although its usual dosage is 150 mg/day, in the present case, the patient should have been started at 25 mg every other day of pilsicainide, and its serum concentration should have been monitored very closely thereafter.2

Typical clinical presentation of pilsicainide intoxication includes arrhythmia, such as ventricular fibrillation,1 ventricular tachycardia,4 and torsades de pointes.3 Because pilsicainide intoxication more likely occurs in patients with renal impairment and the ECG waveform is similar to that of hyperkalemia (eg, wide QRS complex), pilsicainide intoxication may mimic hyperkalemia.

As pilsicainide intoxication may potentially lead to life-threatening conditions, such as severe cardiogenic shock and death,6 early diagnosis and treatment are crucial. However, no treatment strategy has been established. Magnesium sulfate and sodium bicarbonate may be effective,7 8 but there are few reports of actual use of these drugs and their effectiveness is not certain. The effect of haemodialysis on prognosis among patients with pilsicainide intoxication is also controversial.

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Figure 1 (A) First ECG showed bradycardic atrial fibrillation with wide QRS. (B) Second ECG showed accelerated idioventricular rhythm. (C) Two hours after initiating haemodialysis, the ECG showed sinus rhythm.
because of limited data. However, such as this case, several case reports suggest that, although the removal rate of pilsicainide by haemodialysis is not high, a slight decrease in blood levels of the drug can improve symptoms. Thus, haemodialysis is a treatment that should be considered in emergency settings.

Patient’s perspective

I am glad that my illness is gone. I was very careful not to forget to take my medicine, but I was surprised to find out that it could make me sick.

Learning points

► Pilsicainide is a pure sodium channel blocker frequently used in Asia for tachyarrhythmias.
► Because there is scant awareness of the need for dose adjustment of pilsicainide among clinicians, the diagnosis of pilsicainide intoxication may often be delayed.
► Pilsicainide intoxication may mimic hyperkalemia because of its ECG waveform (e.g., wide QRS complex).

Contributors

YH drafted the manuscript. HF reviewed and supervised the manuscript. SS acquired data and reviewed and supervised the manuscript. All authors read and approved the final manuscript.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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