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LETTER TO THE EDITOR
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# Atrial fibrillation and QTc prolongation associated with hypokalemia and hypomagnesemia: a case report

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Atrial fibrillation (AF) is the most common arrhythmia in clinical setting and has been increasingly prevalent due to the aging population. AF is associated with a three to fivefold increased risk of stroke. Treatment options include pharmacological and electrical cardioversion, each carrying specific risks. AF may resolve spontaneously, but often recurs, making a "watch-and-wait" strategy a reasonable approach to avoid unnecessary antiarrhythmic therapy. Additionally, research has shown that hypomagnesemia and hypokalemia contribute to AF development. Thus, potassium and magnesium supplementation may be a rational strategy to enhance spontaneous sinus rhythm conversion rates.[1,2]

A 70-year-old female patient was brought to the emergency department by her relatives due to fatigue and altered consciousness. History revealed that she had diarrhea for several days and had been unable to eat properly. Her medical history included high blood pressure and diabetes mellitus. On admission, the patient appeared ill, with a lethargic mental state. Vital signs were as follows: pulse rate of 120 beats/min, body temperature of 36.2 °C, blood pressure of 110/70 mmHg, respiratory rate of 22 breaths/min, and oxygen saturation of 96%. Blood glucose was measured at 312 mg/dL. Cardiovascular examination revealed an irregular tachycardia. Neurological examination showed a Glasgow Coma Scale score of 12, without any focal neurological deficits or neck stiffness. Electrocardiogram (ECG), venous blood gas analysis, and laboratory parameters were assessed. ECG revealed an irregular rhythm with a high ventricular response, indicating AF. The ventricular rate was 131 beats/min, QRS duration 92 ms, and QTc interval of 491 ms (Figure 1). Arterial blood gas analysis showed pH of 7.47, $HCO_3$ of 19%, lactate of 2.1 mmol/L, base excess of 3.4, and calcium of 1.09 mmol/L. Intravenous access was established, and isotonic fluid therapy was initiated, with fluid replacement adjusted according to urine output. The patient was evaluated by the cardiology department, and amiodarone therapy was recommended. However, laboratory findings revealed the following: sodium of 143 mmol/L, chloride of 96 mmol/L, potassium of 2.62 mmol/L, magnesium of 1.77 mg/dL, urea of 21 mg/dL, creatinine of 0.39 mg/dL, high-sensitivity troponin I of 28.83 ng/mL, and thyroid-stimulating hormone (TSH) of 0.17 μU/mL (reference range: 0.55–4.78 μU/mL). Due to hypokalemia and hypomagnesemia, amiodarone was not administered. Potassium deficit was calculated, and intravenous potassium infusion was initiated. A 500 mL isotonic saline solution containing 40 mEq potassium chloride (KCl) was administered intravenously at a rate of 250 mL/h. Concurrently, 2 g of magnesium sulfate was provided via intravenous infusion. Follow-up laboratory results showed potassium of 2.7 mmol/L and magnesium of 2.76 mg/dL. Subsequently, an additional 500 mL isotonic saline solution containing 40 mEq KCl was administered intravenously over two hours. After four hours of treatment, the patient converted to sinus rhythm, with a PR interval of 122 ms, ventricular rate of 96 beats/min, QRS duration of 86 ms, and QTc interval of 469 ms (Figure 2). The patient was admitted to the ward for further monitoring, and subsequent potassium and magnesium levels were 3.29 mmol/L and 2.2 mg/dL, respectively. ECG confirmed sinus rhythm with a QTc interval of 451 ms. KCL replacement was given as 10 mEq/h in the ward. The control potassium value was measured as 4.1 mmol/L.

In the emergency department, AF management aims to control heart rate or rhythm and prevent thromboembolic complications through anticoagulation. The choice of treatment depends on symptom duration and severity. Conditions such as hyperthyroidism, acute coronary syndro-

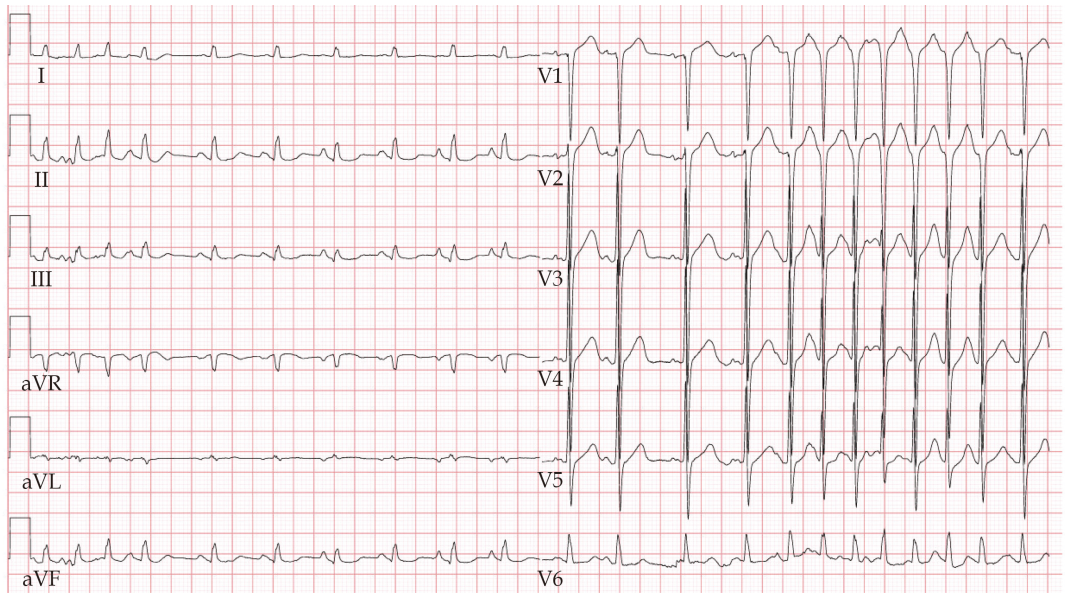


**Figure 1 Electrocardiogram: atrial fibrillation with rapid ventricular response, ventricular rate of 131 beats/min, QRS duration of 92 ms, and QTc interval of 491 ms.**

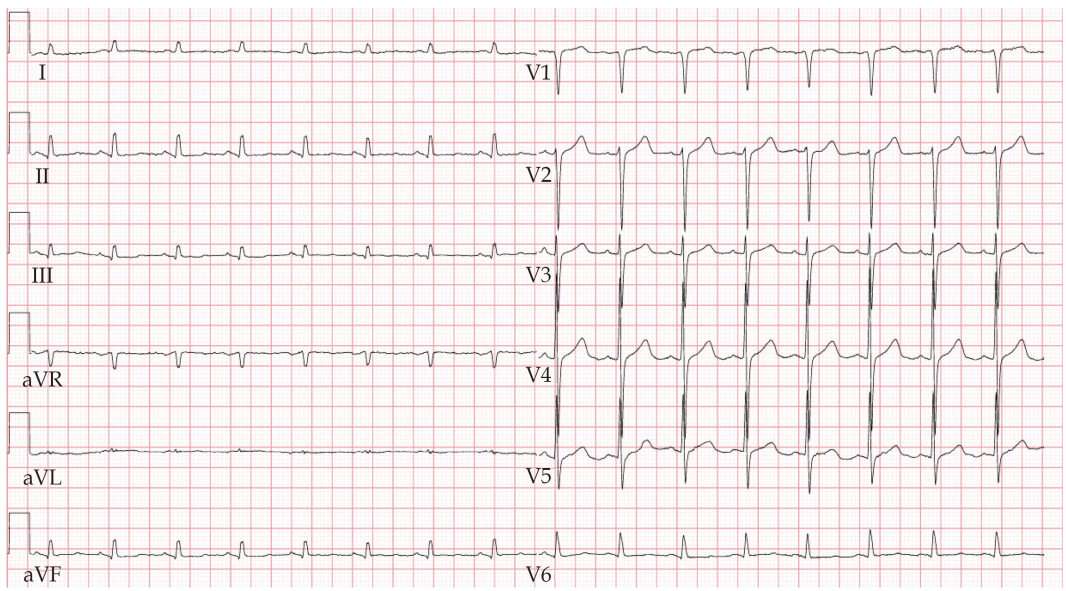


**Figure 2 Control electrocardiogram: sinus rhythm, PR interval of 122 ms, ventricular rate of 96 beats/min, QRS duration of 86 ms, and QTc interval of 469 ms.**

me, and exacerbation of chronic obstructive pulmonary disease may trigger AF, requiring targeted treatment.[2]

Electrolyte imbalances are common in clinical practice. Hypomagnesemia and hypokalemia may result from intracellular electrolyte shifts, gastrointestinal losses, or renal excretion. Hypomagnesemia potentiates the proarrhythmic effects of hypokalemia. Studies have reported an increased AF risk when potassium levels fall below 3.5 mmol/L. Low serum magnesium levels have been moderately associated with AF, particularly in individuals without structural heart disease.[3] Furthermore, postoperative AF incidence following coronary artery bypass grafting has been linked to hypomagnesemia.[4] Thus, potassium and magnesium supplementation may enhance spontaneous sinus rhythm conversion rates.

Our patient presented with acute AF and QTc prolongation. Normal QTc values range from 350–450 ms in adult males and 360–460 ms in the adult female. Prolonged QTc

may result from genetic disorders (e.g., long QT syndrome), pharmacological agents (e.g., antiarrhythmics, antipsychotics, antibiotics), electrolyte abnormalities (e.g., hypokalemia, hypomagnesemia), and their interactions.[5] In our case, hypokalemia and hypomagnesemia were attributed to diarrhea, leading to QTc prolongation. A meta-analysis found a significant correlation between prolonged QTc and AF risk, with a 10-ms increase in QTc associated with a higher likelihood of AF development.[6] Our patient responded well to electrolyte replacement therapy, achieving spontaneous sinus rhythm conversion and normalization of QTc without additional medications or complications.

AF is the most common cardiac complication of hyperthyroidism and may be its initial manifestation. The clinical presentation of AF in hyperthyroid patients varies based on age, gender, and comorbidities. Patients over 60 years old are at a higher risk for developing AF.[7] Routine thyroid function tests are recommended in new-onset AF cases. Our patient had a suppressed TSH level but no history of thyroid disease or medication use. Studies have established an association between subclinical hyperthyroidism (defined as low TSH with normal free T4 and free T3 levels) and an increased risk of AF in patients over 60 years old.[8] Conversely, the relationship between hyperthyroidism and QTc prolongation remains poorly understood. One hypothesis suggests that thyroid hormone affects cardiac myocyte Na (+)/K (+)-ATPase activity. Higher free T4 levels have been linked to greater QTc prolongation.[9,10] In our case, both hyperthyroidism and electrolyte imbalances may have contributed to AF and QTc prolongation, which, if left untreated, could lead to life-threatening arrhythmias such as ventricular tachycardia and fibrillation.

AF is a frequently encountered arrhythmia in the emergency department. Careful differentiation between acute and chronic AF is essential, particularly in elderly patients. Electrolyte and thyroid function assessments should be performed before initiating pharmacological treatment in stable AF patients. In cases of AF with concurrent QTc prolongation, clinicians should consider hypokalemia, hypomagnesemia, and hyperthyroidism as potential underlying causes.

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