Drunk potassium channels

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We report the case of a 42-year-old woman who was admitted due to alcohol induced dehydration leading to cardiac symptoms. On physical examination severe hypokalaemia and long QT syndrome were apparent. During potassium (K⁺) supplementation we simultaneously observed QT interval recovery. This is the first report of continuous documentation of the QTc duration during serum K⁺ rise, highlighting the necessity of electrocardiogram documentation and serum potassium correction in appropriate patients.

Presentation

A 42-year-old woman with impaired nutritional status was referred to the emergency department after a presyncope, shortly before her admission to an alcohol rehabilitation clinic. Prior to her decision of alcohol withdrawal, she noticed some disturbing episodes of tachycardia and dizziness. On admission her blood pressure was 102/68 mmHg with a heart rate of 105 b.p.m., and she was dehydrated with dry skin turgor. In her medical history, depression and alcohol addiction for more than 10 years were known. No cardiovascular or hereditary diseases were present and she was on no regular medication. The woman mentioned that she began to drink much more alcohol and severely neglected her nutrition after she had lost her job a month ago. We made an electrocardiogram (ECG) and took blood samples for laboratory testing.

Assessment

The first ECG showed sinus tachycardia and a prolonged corrected QT interval (QTc) of at least 527 ms (manually calculated using Bazett’s formula). Arterial blood sample revealed normal blood gases but uncovered severe hypokalaemia (K⁺ 2.2 mmol/L; normal range 3.6–5.0 mmol/L). Further laboratory measurements confirmed malnutrition and alcohol-induced dehydration (albumin 23.1 g/L, urea 18.6 mmol/L, sodium 129 mmol/L, calcium 2.7 mmol/L, magnesium 0.62 mmol/L, creatinine 143 μmol/L, glucose 10.1 mmol/L, gamma-glutamyltransferase 122 IU/L, mean corpuscular volume 113 fL) with negative results for cardiac enzymes, D-dimer, blood alcohol concentration, and detailed toxic screening.

Diagnosis, discussion, and management

Potentially proarrhythmic effects of many drugs are well known and have been thoroughly studied, uncovering that inappropriate modulation of ion channels is the main source of cardiac irritation leading to fatal arrhythmias. Two important players that might...
interfere paradoxically with extracellular K\(^+\) are the inwardly rectifying K\(^+\) current (IK\(_1\)) and the human ether-a-go-go-related gene (HERG) potassium channel.\(^1\)–\(^3\) In particular, the inhibition or dysfunction of the HERG channel induces a prolongation of the cardiac action potential leading to delayed repolarization and long QT duration (LQT).\(^2\) Moreover, it is known that extracellular K\(^+\) is essential for HERG function and the cardiomyocytes membrane stability, but also regulates voltage-gated K\(^+\) currents.\(^1\)–\(^3\)

We present a completely documented case of a patient with coincidence of LQT syndrome and hypokalaemia. To test a potential causal link between low K\(^+\) level and the QT prolongation we recorded the QTc duration during electrolyte correction. The time course of QTc duration recovery precluded a direct contributing effect of ethanol itself on QT interval. On the contrary, an impressive inverse correlation of the QTc duration with serum K\(^+\) levels. After 5 days we could discharge our patient cardiopulmonary stable with an unremarkable ECG and normal laboratory values (albumin 38.9 g/L, urea 3.7 mmol/L, sodium 140 mmol/L, calcium 2.42 mmol/L, magnesium 0.78 mmol/L, creatinine 69 \(\mu\)mol/L, and glucose 5 mmol/L).

**Conclusion**

Isolated hypokalaemia-induced LQT syndrome is not only an epiphenomenon.\(^7\) At least in some patients low K\(^+\) levels may induce clinically relevant cardiac conduction disorders.\(^1\) This is the first report of continuous documentation of the QTc duration during serum K\(^+\) rise. Patients at elevated risk for hypokalaemia, e.g. with alcohol abuse or anorexia nervosa, may develop symptomatic LQT syndrome.

**Conflict of interest:** none declared.

**References**