CASE REPORT

Licorice-Induced Severe Hypokalemia with Recurrent Torsade de Pointes

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A 38-year-old obese woman presented with recurrent polymorphic ventricular tachycardia secondary to persistent hypokalemia necessitating more than 40 DC shocks. All endocrine investigations for hypokalemia were negative with impression of "mysterious hypokalemia." On repeated inquiry, a hidden history of licorice use was elicited causing persistent hypokalemia. The case highlights a life threatening complication of licorice use. In addition, it reiterates the importance of repeated history taking in a patient with undiagnosed hypokalemia and torsade de pointes which avoided a device therapy.

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Licorice, the root of the plant *Glycyrrhiza glabra*, has been used all over the world for its sweet taste and for medical purposes.^{1,2} We report a rare case of recurrent polymorphic ventricular tachycardia secondary to persistent hypokalemia because of licorice ingestion.

CASE REPORT

A 38-year-old obese woman, mother of nine children, nondiabetic, hypertensive, presented to a regional hospital with recurrent syncope. She was postpartum 6 months, on methyldopa for hypertension. She was not on any other medication and denied any vomiting or diarrhea. On presentation, she was hypotensive with monitor showing polymorphic ventricular tachycardia (PVT). She was defibrillated promptly. However, she developed recurrent PVT with hypotension and syncope necessitating 40 DC shocks over 2 hour's period (Fig. 1). She was electively intubated and transferred to our institute on inotropic support. Her echocardiogram showed global hypokinesia with an ejection fraction of 25%. Her blood investigations including glucose,

sodium, magnesium, calcium, thyroxine, creatinine, creatine phosphokinase, cortisol, plasma renin, and aldosterone levels were within normal limits except for serum potassium which was persistently low at 2.4 mmol/L (N = 3.5–5). Arterial blood gas analysis showed metabolic alkalosis with pH 7.50 [n = 7.35–7.45], PaCO₂ 35 mmHg [n = 35–45], bicarbonate 28 mmol/L [n = 22–26], base excess of 4 mmol/L. Serum osmolality was calculated to be 300 mOsm/kg. Her spot urinary potassium was 29 mmol/L [n < 20 mmol/L] suggesting renal loss. Twelve-lead electrocardiogram demonstrated U waves in V1; prolonged QTc interval at 485 milliseconds with frequent ventricular premature complexes producing R on T phenomenon initiating a PVT (Fig. 2).

She was treated with magnesium sulphate and potassium infusions followed by oral replacement bringing the potassium levels to normal within 12–24 hours resulting in disappearance of PVT. During this period of replacement she needed another 8 DC shocks for her PVT. After 48 hours she was extubated and weaned of inotropes. A repeat echocardiogram after 2 weeks showed an ejection fraction of 45%. Endocrinology investigations including magnetic resonance imaging

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Figure 1. ECG monitor rhythm strips showing recurrent sustained polymorphic ventricular tachycardia (torsades de pointes) necessitating multiple DC shocks in a patient with licorice-induced severe hypokalemia.

Figure 2. Chest leads of 12-lead ECG in sinus rhythm demonstrating prominent U wave (A, arrow), prolonged QTc interval of 485 milliseconds and R on T phenomenon initiating a polymorphic ventricular tachycardia (B, arrow) in a patient with licorice-induced severe hypokalemia.

Figure 3. Twelve-lead ECG at discharge, demonstrating sinus rhythm with normal QTc interval of 435 milliseconds in a patient with licorice-induced hypokalemia and Torsades de Pointes.

of abdomen and renals showed no indication of Cushing's syndrome, primary hyperaldosteronism or renal artery stenosis. Endocrinologists opined as "mysterious hypokalemia." However, on taking repeated history, for the past 2 months, she was ingesting licorice root tea thrice daily to reduce weight. She was hiding this history so that her husband and family do not know about her licorice use. This history clinched the diagnosis to licorice-induced hypokalemia with secondary PVT, thus avoiding an internal cardioverter defibrillator. At 1 month follow-up and off licorice use, she was well with normal potassium level (4.2 mmol/L), a normal QTc interval (435 msec; Fig. 3) and 24-hour Holter study. Her blood pressure was well controlled with Atenolol.

DISCUSSION

Licorice contains glycyrrhetic acid which inhibits renal 11 beta-hydroxysteroid dehydrogenase type 2, which normally inactivates cortisol.^{1,2} Inhibition of this enzyme by licorice leads to excess cortisol which stimulates mineralocorticoid receptors resulting in a state of "pseudohyperaldos-

teronism" causing hypertension, renal potassium loss, high bicarbonate and metabolic alkalosis. Thus, cortisol effects mimic aldosterone excess (apparent mineralocorticoid excess syndrome), although aldosterone remains low or normal during licorice overdose.^{1,2} In addition, licorice is known to reduce body fat by inhibiting 11 beta-hydroxysteroid dehydrogenase type 1 at the level of fat cells, thus increasing fat oxidation.³ Studies have indicated that licorice consumption should be no more than 10-30 g licorice per day or no more than half a cup of licorice tea a day.⁴

Pseudohyperaldosteronism presents with low aldosterone and low renin and may result from genetic or acquired etiologies. Genetic causes are very rare and present early during childhood or in adulthood. In this patient all features of acquired "pseudohyperaldosteronism" were apparent like renal potassium loss, high serum bicarbonate and metabolic alkalosis, even though aldosterone level was apparently normal, which is known during licorice use.^{1,2} This patient developed severe hypokalemia secondary to excessive licorice use leading to acquired long QT syndrome and recurrent PVT suggestive of torsades de pointes.

However, she responded to potassium replacement and magnesium, which is the drug of choice for suppressing early after depolarizations of PVT, even in patients with normal magnesium levels as noted in this patient. There was resolution of PVT and hypokalemia following stoppage of licorice use. This case highlights a life threatening complication of licorice use, as well as importance of taking a comprehensive history which avoided a major device intervention.

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