

Liquorice-Induced Hypokalaemia and Rhabdomyolysis

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Abstract

This is a case report of a 60-year-old man who was admitted at the emergency department with tetra paresis, severe hypokalaemia, rhabdomyolysis and hypertension. The cause could have been hyperaldosteronism, but was found to be liquorice intoxication. Liquorice is both available and widely used in both the confectionary and medical sectors. It has glucocorticoid and mineralocorticoid properties, but can, if consuming too much, lead to an acquired form of apparent mineralocorticoid excess syndrome which causes hypokalaemia, hypernatremia, oedema, hypertension, metabolic alkalosis and low plasma renin and aldosterone levels. In this case, the patient developed rhabdomyolysis because of his hypokalaemia. It is believed that potassium depletion causes ischemia in the muscle cells and it therefore can lead to muscle cramps and necrosis.

Keywords: Liquorice intoxication; Hypokalaemia; Rhabdomyolysis

Introduction

Liquorice has been used for thousands of years for confectionary and medical purposes. It is easily available and attractive because of its sweet taste and anti-inflammatory, glucocorticoid effects. It has also less desirable effects due to mineralocorticoid - like properties, mimicking hyperaldosteronism. This case illustrates a life-threatening event due to liquorice over consumption.

Case Report

A 60-year-old man was admitted to the emergency department because of rapidly increasing muscle weakness. He had a history of muscle weakness in his upper arms and pains in the neck and shoulder region after an accident many years ago. For the last few weeks the symptoms had progressed and for the last 4 days also included his legs. Previously he had been able to cycle 10 km without problems, but for the last 4 days he felt his legs couldn't support him. There was no history of crush injury. He had a mild untreated hypertension. For the last 4 days he had small amounts of watery diarrhoea.

On a daily basis he consumed 4 - 5 units of alcohol and sweets, both containing liquorice.

The examination showed that he was tetra paretic; he could barely lift his legs and arms from the bed. There was no sensory disturbance, only a little tenderness in the shoulder and upper arms. Cardiovascular, pulmonary and abdominal examinations were unremarkable. The blood pressure was 180/108, the pulse 90.

Laboratory tests

Potassium 1.5 mmol/L, CK 31,077 U/L, creatinine 66 µmol/L and lactate 4.3 mmol/L, blood gases showed an uncompensated metabolic alkalosis (pH, 7.59, BE 20 mmol/L, HCO₃ 43 mmol/L). ANA, ANCA and immunoglobulins were all normal. ECG was normal.

A rheumatologist was consulted. A CT scan of his upper back showed no signs of cervical herniated disk or spinal stenosis. Polymyositis was disproved. Renin and aldosterone were low and primary hyperaldosteronism was excluded. Hypokalaemia, metabolic alkalosis, muscle weakness, diarrhoea, hypertension and suppression of the renin-angiotensin-aldosterone axis led us to the conclusion of liquorice intoxication.

He was treated with potassium, forced diuresis, spironolactone and amlodipine. After two weeks he was back to normal and was discharged with normalized blood tests and blood pressure.

Manuscript accepted for publication August 14, 2013

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doi: <http://dx.doi.org/10.4021/jem181w>

Discussion

Apart from being used in sweets, liquorice is found in products like smoking and chewing tobacco, chewing gum, herbal teas, alcoholic drinks and herbal remedies for cough, stomach ailments and constipation. The active compound is glycyrrhizic acid (GZA) and consuming large doses of this can among others lead to hypokalaemia due to potassium excretion and hypertension and oedema due to sodium and water retention in the kidneys. GZA and its metabolites inhibit the enzyme 11 β -hydroxysteroid dehydrogenase and subsequent increases activity of cortisol on the mineralocorticoid receptors [1, 2]. Similar symptoms are also seen in primary mineralocorticoid excess, Cushing's syndrome and apparent mineralocorticoid excess syndrome.

The dose-response and the time-response relationship between liquorice ingestion and its side effects show inter-individual variance, but there is a linear dose-response relationship [3]. The lowest observed adverse effect level for GZA is found to be 100 mg/day [4].

In spite of the metabolic alkalosis, the patient had a high lactate. This can be explained by the rhabdomyolysis. The mechanism behind hypokalaemic rhabdomyolysis is not fully understood, but it is believed that decreased potassium levels causes vasoconstrictions and ischemia in the muscle cells, which can lead to muscle necrosis and rhabdomyolysis [5].

Conclusion

With this case we would like to emphasize the importance

of remembering liquorice intoxication especially in patients with unexplained hypertension, hypokalaemia or muscle weakness. A thorough history including eating and drinking habits, especially liquorice consumption, is important. Liquorice is a popular product and it is important to keep in mind the less desirable and possible deadly effects.

Declaration of Interest

The authors report no conflicts of interest.

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