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Tetraparesis with Major Hypokalaemia and Rhabdomyolysis Induced by Chronic Liquorice Ingestion

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ABSTRACT

Chronic ingestion of liquorice induces a syndrome with findings similar to those for primary hyperaldosteronism. This is characterized by hypokalaemia, hypertension, metabolic alkalosis and suppression of the renin-aldosterone system.

We describe a 30-year-old woman who, with a plasma potassium level of 1.5 mmol/l, presented with tetraparesis and severe rhabdomyolysis (CK up to 35,460 U/l). She admitted to a daily consumption of nearly 300 g of liquorice sweets during the previous 6 months.

This case emphasizes the importance of a detailed anamnesis, which is essential for diagnosis, avoids unnecessary and expensive investigations and reduces the duration of hospitalization.

LEARNING POINTS

- Liquorice, the root of *Glycyrrhiza glabra*, has been used throughout the millennia for its taste and for medicinal purposes.
- Chronic ingestion of liquorice can mimic primary hyperaldosteronism.
- It is important for physicians to keep liquorice consumption in mind as a cause of hypokalaemic paralysis and rhabdomyolysis.

KEYWORDS

Hypokalaemia, rhabdomyolysis, liquorice abuse

CASE DESCRIPTION

A 30-year-old woman presented to the emergency room with muscular weakness that progressed to paralysis involving all extremities.

Her family and past medical histories were unremarkable. She did not use alcohol. She had no history of diarrhoea, vomiting or diuretic use.

On examination, blood pressure was 160/80 mmHg, heart rate was 80/min, respiratory rate was 16/min and body temperature was 37.5°C. There was a symmetric flaccid paralysis with areflexia in the lower and upper extremities. The muscles were tender to palpation but no rash was found. She was unable to stand up from the sitting position. The remainder of the clinical examination was unremarkable.

Laboratory studies found profound hypokalaemia of 1.5 mmol/l, pH of 7.6 and creatine kinase (CK) levels of approximately 35,550 U/l. Urine chloride was 80 mmol/24h (NR <20). Both oral and intravenous potassium were administered and within 4 days, serum levels of potassium normalized and all clinical symptoms improved. Serum CK levels returned to normal within 2 weeks. A diagnosis of primary hyperaldosteronism was suspected but plasma renin activity and aldostero**ne levels were below normal. In addition, thyroid function tests and cortisol serum levels were normal. Computed tomography scanning showed normal adrenal glands.

Finally, the patient admitted to a daily consumption of nearly 300 g of liquorice sweets during the previous 6 months.

One year after she stopped consuming the liquorice sweets, her plasma potassium concentration was 4.1 mmol/l and she is still asymptomatic.

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DISCUSSION

Liquorice, the root of *Glycyrrhiza glabra*, has been used throughout the millennia for its taste and for medicinal purposes (hypotension, stomach ulcers). Liquorice-induced hypokalaemia is a rare disorder first described by Revers in 1946[1]. Since then, numerous case reports[2–6] have been published on this disorder.

Our patient had severe hypokalaemia and rhabdomyolysis in combination with metabolic alkalosis. She admitted to a daily consumption of nearly 300 g of liquorice sweets during the previous 6 months.

The main active component of liquorice is glycyrrhizic acid, which inhibits an enzyme (11β-hydroxysteroid dehydrogenase) required to convert cortisol to a less active metabolite, cortisone. This causes excess cortisol, simulating the syndrome of apparent mineralocorticoid excess[2]. Russo et al. suggested that low doses of liquorice can also induce hypertensive encephalopathy[5].

Since liquorice consumption is very common and not all of its consumers develop a hypertensive syndrome, Miettinen et al.[7] reasoned that genes influencing liquorice action [encoding the β and γ subunits of the epithelial sodium channel] may at least partially determine susceptibility to its side effects.

In conclusion, liquorice has become widely available as a flavouring agent in foods and drugs. Our case emphasizes the importance of a detailed anamnesis, which is essential for diagnosis, avoids unnecessary and expensive investigations and reduces the duration of hospitalization.

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