

## Total body potassium depletion and severe myopathy due to chronic liquorice ingestion

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### Summary

A 33-year-old patient with hypokalaemia associated with severe myopathy following liquorice ingestion is described. Potassium depletion was confirmed by total body potassium measurements and myopathy was established by electromyography and enzyme studies. The potential hazard of chronic liquorice consumption even as a sweet is illustrated by this case.

### Introduction

Although liquorice consumption can be one of the causes of hypokalaemia (Conn, Cohen and Rovner, 1968; Cumming, 1977; Gross, Dexter and Roth, 1966; Salassa, Mattox and Rosevear, 1962) there are no reports of total body potassium measurements in patients with hypokalaemia caused by this. This report concerns a patient with severe myopathy and potassium depletion due to chronic liquorice ingestion, in whom total body potassium was measured.

### Case report

A 33-year-old female Civil Servant, was admitted to the General Infirmary at Leeds with a 4-week history of increasingly severe pain and weakness in proximal muscles. Two weeks before admission she

had noted slight ankle oedema and had been given bendrofluazide 5 mg once/day by her general practitioner. The weakness increased and by the time of admission she was unable to stand. There was no past or family history of similar symptoms. On admission her BP was 180/100 mmHg. She had minimal bilateral pitting ankle oedema. The limbs were hypotonic; the patient could not raise her head, arms or legs from the bed; the deep reflexes were absent. However, her respiration was normal.

Investigations showed severe hypokalaemic alkalosis and elevated 'muscle' enzymes (creatin phosphokinase, aldolase and aspartate amino transferase) (Table 1). The electrocardiogram was normal and electromyography confirmed that she had a myopathy.

On being questioned, the patient admitted that she ate about 500 g (i.e. 4 boxes) of liquorice a week. This and the diuretic were stopped and she was treated with potassium chloride 64 mmol/day. Muscle power dramatically improved within 48 hr and the patient was able to get up from bed and stand unaided. Her total body potassium (TBK), measured in a total body counter on the 4th day after admission was 1940 mmol/l (normal 2355-3071 mmol). As shown in

TABLE 1. Changes in plasma potassium, bicarbonate, enzymes and total body potassium in a patient with liquorice toxicity

Day	Plasma potassium (3.5-5.0) (mmol/l)	Bicarbonate (mmol/l)	AST (GOT) (8-22)	Aldolase (1-9)	CPK (<60)	TBK (mmol/l)
1	1.5	40				
4	2.0	32	315	42	2261	1940
6	2.4	30	146	-		
8	2.8	30	-	4		
11	3.4	28	26	-	160	
12	2.8	24	-	4	97	2231
14	4.2	26	18	-	-	
25				6		2255

Normal ranges are given in brackets, enzyme values in i.u./l. AST = aspartate transaminase; GOT = glutamic oxalo-acetic transaminase; CPK = creatine phosphokinase; TBK = total body potassium.

Table 1, her TBK increased and the plasma potassium, and the muscle enzymes, subsequently returned to normal. Plasma aldosterone was measured 2 months later and was normal. The patient was advised to avoid liquorice in future.

### Discussion

This patient who had been eating 500 g of liquorice per week for many years developed a myopathy and hypokalaemic alkalosis. The administration of a diuretic 2 weeks before admission might have been the aggravating factor in this patient; liquorice has been known to cause oedema (Revers, 1948). The hypokalaemia is thought to be due to the aldosterone-like action of its active principle – glycyrrhizinic acid (Card *et al.*, 1953; Groen *et al.*, 1951, 1952; Louis and Conn, 1956) – and this is known to be associated with muscle weakness, necrosis of muscle fibres and ECG changes (Cumming, 1977; Gross *et al.*, 1966; Salassa *et al.*, 1962). The present patient showed severe muscle weakness, muscle necrosis (increased CPK, aldolase and AST) and oedema, but did not display any ECG changes. A 20% deficit in total body potassium in this patient was also shown. Although weakness due to chronic ingestion has been reported previously (Cumming, 1977; Gross *et al.*, 1966; Salassa *et al.*, 1962), there are no reports on total body potassium measurements in these patients. In spite of a 20% deficit of total body potassium, there were no ECG changes in this case. It is possible that skeletal muscle damage and cardiac changes due to hypokalaemia might have different mechanisms. It is also interesting to note that the patient's muscle power improved dramatically within 48 hr of stopping liquorice, with only a small increase in plasma potassium (0.5 mmol/l) during that

period. Thus, it appears that the severity of muscle weakness does not correlate with the degree of hypokalaemia. The object of this report is also to stress the potential hazard of chronic liquorice consumption, even as a sweet.

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