

A CASE OF HYPOKALAEMIA AND DEPENDENT OEDEMA INDUCED BY LIQUORICE

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ABSTRACT

Excessive intake of liquorice can cause hypokalaemia and hypertension and generally, the onset and severity of symptoms depend on the dose and duration of liquorice intake, as well as individual susceptibility. We describe a patient with hypokalaemia caused by long term consumption of liquorice. The case emphasizes the importance of considering a detailed patients' history, which often lead the treating physician to the correct clinical diagnosis.

Key Words: Hypokalaemia, Dependent Oedema, Liquorice

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INTRODUCTION

Liquorice (scientific name *Glycyrrhiza glabra*) is historically used for GI complaints. Now it is primarily used as a flavoring agent in the tobacco, confectionery and to some extent in the pharmaceutical and beverage industries. The active ingredient of Liquorice is glycyrrhetic acid. Glycyrrhetic acid inhibits the conversion of active cortisol to inactive cortisone in the kidneys by inhibiting 11 β -Hydroxysteroid dehydrogenase¹ as well as competitively inhibit it as well². As a result, cortisol levels are high within the collecting duct of the kidney. Cortisol has intrinsic mineralocorticoid properties that work on NaCl channels in the collecting duct. Hypertension develops due to this mechanism of sodium retention. People often have high blood pressure with hypokalaemia and a low renin and low aldosterone blood level.

The mineralocorticoid stimulation by licorice is reversible, usually recovering within days, but may be sustained for several weeks according to amount taken and individual susceptibility³

CASE REPORT

A 69 years old Caucasian lady who was previously fit and well, presented through her General Practitioner with low serum potassium of 2.0 mmol/L (normal 3.5 -5mmol/L) and dependent oedema. No history of diar-

rhoea or vomiting and any diuretics or laxative use. She was an ex smoker stopped when she was a teenager and consumed a bottle of wine per week.

Physical examination showed mild hypertension 173/88mmHg, Pulse 85/min regular. Dependent oedema up to the ankles. Rest of the physical examination was normal.

Her initial blood investigations showed serum potassium of 2.0 mmol/L (normal 3.5 -5.5) serum sodium 146 mmol/L (normal 135-145), Urea and creatinine normal. Normal bone profile. Serum Magnesium 0.57mmol/L (normal 0.7- 1) Her Arterial Ph was 7.50 with Bicarbonate 42mmol/L (metabolic alkalosis) Her Serum TSH 2.25 mIU/L (normal) Haemoglobin, White cell count and Platelets were normal. She was given intravenous replacement of potassium and when serum potassium improved, she was started on oral potassium supplements. She had Serum Renin (<0.2pmol/ml/h) and Aldosterone (<70pmol/L) checked which were both low. She had urine cortisol creatinine ratio checked which was raised 75.6 nmol/mmol (Normal 5-55 nmol/mmol). However, Subsequent over night Dexamethasone suppression test was normal at 24 nmol/L. Her ultrasound scan of the kidneys was normal.

Her 24 hours urinary calcium was normal 2.82mmol/day (normal 2.5-7.5mmol/day)

Figure 1: Mechanism of action of Liquorice

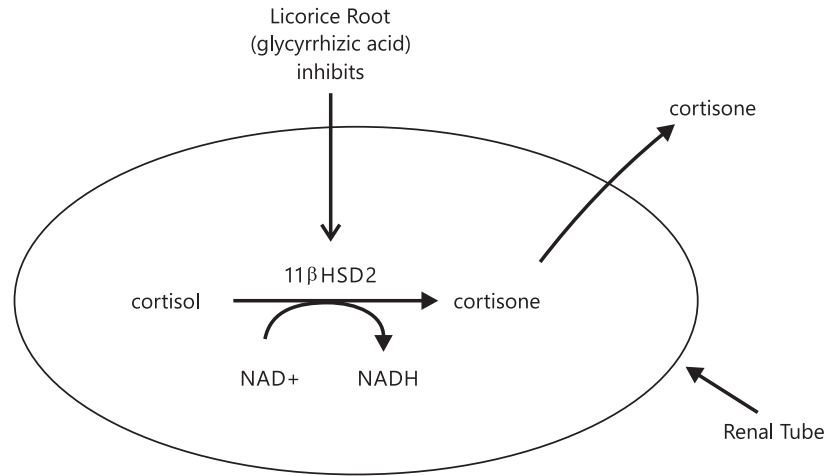
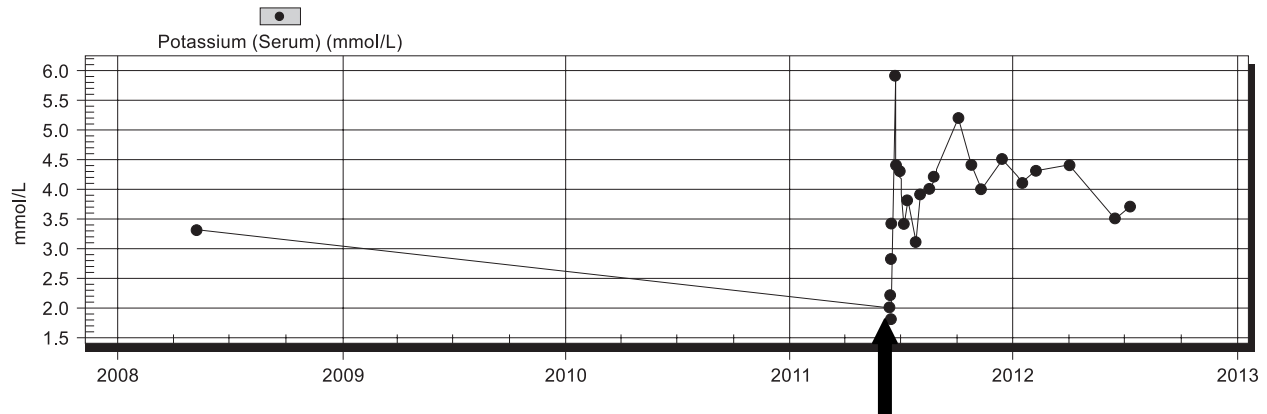


Figure 2: Serum Potassium levels. Arrow indicates admission to the hospital



She was seen by the Endocrinology team and her history at that time revealed that she had been taking liquorice in sweets for as long as she could remember. Her previous potassium readings couple of years back were low. She stopped Liquorice after that admission and was discharged home on oral potassium supplements Sando K 2 tablets three times a day which she gradually reduced and was not on any potassium supplements for about two months when seen July 2012 and was normotensive at 120/60 mmHg and no dependent oedema.

DISCUSSION

Liquorice ingestion can present with fatigue, muscle cramps, hypokalaemia, myoglobinuria⁴, weakness/paralysis⁵, oedema, dyspnoea or Paresthesias/dysesthesias (e.g., burning sensations of extremities). It is known to cause hypertension and hypertensive encephalopathy⁶

The differential diagnoses in our case initially were primary hyperaldosteronism, Cushing's syndrome, Re-

no-vascular disease, Liddle's syndrome, Gitelman's syndrome and Bartter syndrome.

Primary hyperaldosteronism was ruled out by low plasma renin and aldosterone levels. She had no features of Cushing's syndrome and overnight dexamethasone suppression test was normal as well so excluding Cushing's syndrome from the list of differentials. The ultrasound scan of the kidneys was normal which ruled out renal artery stenosis⁷

Liddle's syndrome is an autosomal dominant disorder characterized by early, and frequently severe, hypertension associated with low plasma renin activity, metabolic alkalosis due to hypokalemia, and hypoaldosteronism. Although Liddle's syndrome can present in elderly⁸, our patient potassium stabilized after discontinuing Liquorice ingestion

Gitelman's and Bartter's syndrome patients are usually normotensive, hypokalaemic, hypocalciuric and have metabolic alkalosis and raised plasma renin and

aldosterone levels while our patient was initially hypertensive and had low plasma renin and low plasma aldosterone levels⁹

In our patient, no history of liquorice ingestion was recorded initially. She did not have vomiting, diarrhea, and denied using drugs, including diuretics, herbal medication, and laxatives. When seen by the Endocrine team as in patient and specifically asked for she admitted to be using liquorice.

CONCLUSION

A detailed history is vital to reach the clinical diagnosis. In patients who present with hypokalaemia and hypertension liquorice ingestion should be kept in mind particularly those with low renin and aldosterone levels.

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