Liquorice Induced Hypertension and Hypokalaemia

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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. Excessive intake of liquorice can cause hypokalaemia and hypertension 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 may lead the treating physician to the correct clinical diagnosis.

Case Presentation

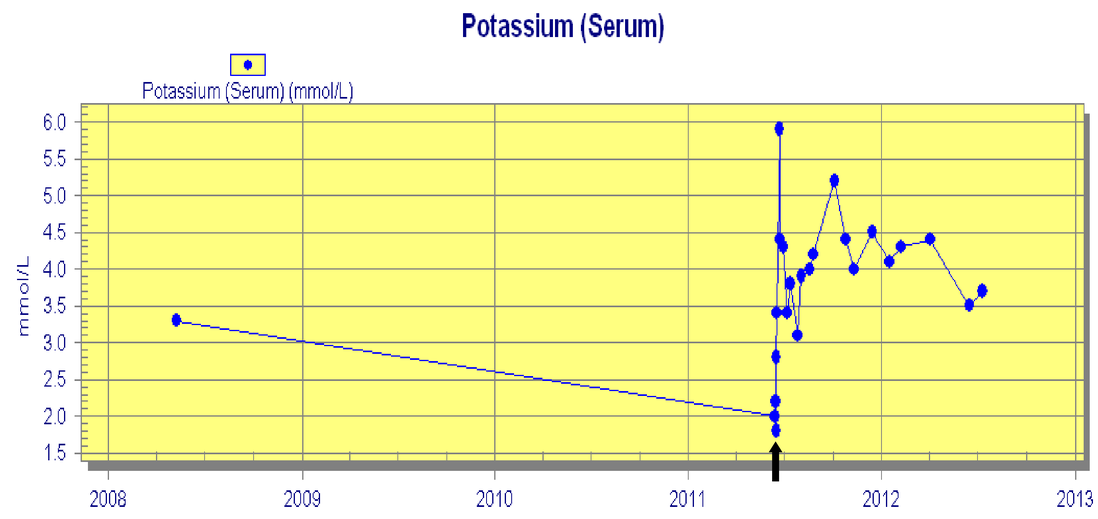
A 69 years old caucasian lady was referred by General Practioner with low serum potassium of 2.0 mmol/L and dependent oedema. No history of diarrhoea or vomiting and no diuretics or laxative use. She was an ex smoker stopped when she was a teenager and consumed a bottle of wine per week.
Physical examination showed mild hypertension 173/88mmHg, Pulse 85/min regular. Dependent oedema up to the ankles. Rest of the physical examination was normal

Progress and Management

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 remembered.Her previous potassium readings couple of years back showed that she was hypokalaemic. She stopped Liquorice after that admission and was discharged home on oral potassium supplements which she gradually reduced and was not on any potassium supplements for about two months when seen in July 2012. She was normotesive at 120/60 mmHg with no dependent oedema.

Investigations

- Serum potassium of 2.0 mmol/L, Serum sodium 146 mmol/L,Urea and creatinine normal
- Normal bone profile.
- Serum Magnesium 0.57mmol/L(Normal 0.7- 1)
- Arterial Ph 7.50,Bicarbonate 42mmol/L(metabolic alkalosis)
- TSH 2.25 (Normal)
- Heamoglobin, White cell count and Platelets were Normal
- .Serum Renin <0.2pmol/ml/h and Aldosterone <70pmol/L
- over night Dexamethasone suppression test was normal at 24 nmol/L.
- Her ultrasound scan of the kidneys was normal.
- 24 hours urinary calcium was normal 2.82mmol/day



Dig.Serum Potassium levels. Arrow indicates admission to the hospital

Discussion

- Glycyrrhetinic acid inhibits the conversion of *active* cortisol to inactive cortisone in the kidneys by inhibiting 11β-Hydroxysteroid dehydrogenase as well as competitively inhibitining it[1,2] Cortisol has intrinsic mineralocorticoid properties thus leading to hypertension, hypokakalamia,low renin and aldosteone levels
- It can present with fatigue, muscle cramps, hypokalaemia, myoglobinuria[3], weakness/paralysis[4], oedema, dyspnoea or Paresthesias/dysesthesias (e.g., burning sensations of extremities). It is known to cause hypertension and hypertensive encephalopathy[5]
- In our patient ultrasound of the kidneys was normal ruling out renal artery stenosis[6] and normal over night dexamethasone ruled out cushing's syndrome
- Although Liddle's syndrome can present in elderly[7], our patient's potassium stabilized after discontinuing liqourice ingestion
- Gitelman's and Barter's syndrome patients are usually normotensive, hypokalaemic,hypocalciuric and have metabolic alkalosis and raised plasma renin and aldosterone levels[8]

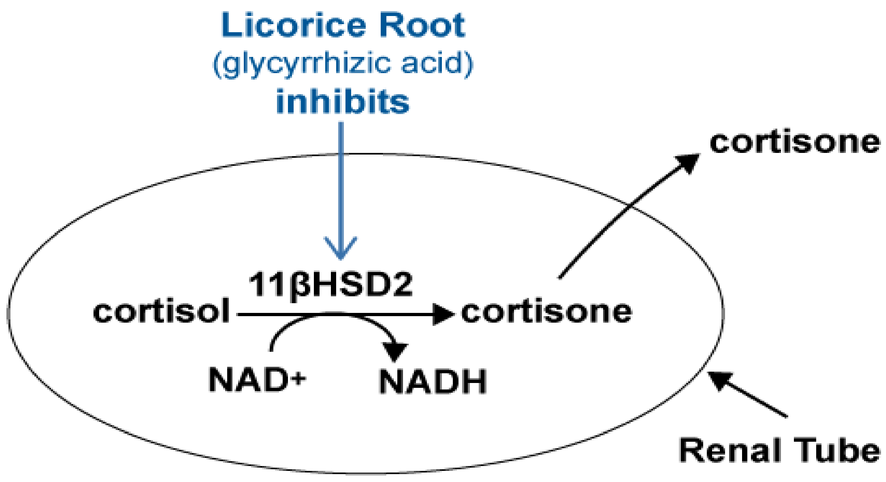


Fig Mechanism of action of Liquorice

Conclusion

A detailed clinical history is vital to reach 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

References
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