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Reminder of important clinical lesson

A salty cause of severe hypertension

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Summary

A 51-year-old lady was referred to our clinic because of severe hypertension; blood pressure 214/119 mm Hg despite treatment with an angiotensin receptor antagonist and a calcium channel blocker. Her initial laboratory results showed hypokalaemic alkalosis with normal urea and creatinine levels. Her 24-h urinary sodium excretion was markedly elevated at 244 mmol (equivalent to a daily intake of approximately 16 g of salt). Hyperaldosteronism was suspected but her plasma aldosterone level was subsequently found to be normal. On further questioning, the patient admitted to eating considerable amounts of salted liquorice and a diagnosis of acquired apparent mineralocorticoid excess was made. Liquorice has a well-known mineralocorticoid activity as it inhibits the action of 11-hydroxysteroid dehydrogenase 2 and can induce mineralocorticoid hypertension. After stopping eating the salted liquorice, the patient's blood pressure quickly normalised and all her antihypertensive medications were stopped.

BACKGROUND

This case describes unusual cause of severe hypertension and electrolyte abnormalities. The case highlights the importance of taking adequate dietary history, underscores the importance of suspecting and diagnosing potentially curable secondary hypertension and highlights the perilous interaction between high dietary salt intake and the ingestion of liquorice in inducing severe hypertension.

CASE PRESENTATION

In June 2004, a 51-year-old retired microbiologist was referred to us because of severe essential hypertension. She was treated with an angiotensin receptor antagonist (candesartan 16 mg once daily) and a calcium channel blocker (amlodipine 5 mg once daily) but her blood pressure (BP) remained very high at 214/119 mm Hg. Four years before her presentation her BP was normal and only after she had seriously dieted and lost almost half her body weight by losing eight stones that her BP started to increase. She was a non-smoker and drank three to four units of alcohol per month. When the patient was first seen in our unit she underwent 24 h ambulatory blood pressure monitoring which confirmed that her blood pressure was not adequately controlled as it revealed a mean day time blood pressure of 141/90 mm Hg, and a mean night time blood pressure of 140/85 mm Hg. Her initial laboratory results showed that her plasma sodium was 139 mmol/l, plasma potassium was 3.1 mmol/l and her bicarbonate was 33 mmol/l, with normal urea and creatinine levels. Her 24-h urinary sodium excretion was markedly elevated at 244 mmol (equivalent to a daily intake of approximately 16 g of sodium chloride) and potassium excretion was 141 mmol. We suspected hyperaldosteronism because of the hypokalaemic alkalosis and hypertension. After correcting her hypokalaemia with oral potassium supplements, we checked her plasma renin activity (PRA) and plasma aldosterone levels, both of which were found to be within normal limits; plasma aldosterone was 113 pmol/l (100–600) and PRA was 1.28 ng/ml/h (0.6–4.5). We suspected apparent mineralocorticoid excess (AME) and on further questioning, the patient admitted to her extreme fondness for eating salted liquorice. Since she was a young schoolchild she has eaten liquorice on a regular basis. However, when her husband started travelling to Norway on a weekly basis in 1999 she discovered 'the delights of Salty and Salmiak flavoured Norwegian liqorice' (unavailable at the time in UK but widely available in continental European countries) and her consumption level rocketed. In fact, in the months prior to her presentation she had approximately 750–800 g of salted liquorice per day. The patient was not a very keen cook and she relied heavily on ready-made meals which have a high salt content.

The diagnosis was then established as acquired AME secondary to liquorice ingestion. We advised the patient to stop eating liquorice, to cut down on her salt intake and to eat more fruits and vegetables. The patient initially found it difficult to comply with this advice but on our persistent recommendation she eventually stopped and also reduced her salt intake. The patient's BP quickly improved with the discontinuation of liquorice ingestion and all her antihypertensive medications were stopped. When the patient was reviewed in our clinic 6 months later, her 24 h sodium excretion was reduced to 87 mmol and her BP remained normal at 124/82 mm Hg while on no treatment with normalisation of her plasma potassium and bicarbonate levels.

This case emphasises the importance of taking adequate dietary history, underscores the importance of suspecting and diagnosing potentially curable secondary hypertension, and highlights the perilous interaction between high dietary salt intake and the ingestion of liquorice in inducing severe and difficult to treat hypertension. The finding of hypertension and hypokalaemia, whether

spontaneous or induced by diuretics, should always raise the suspicion of secondary hypertension and in particular hyperaldosteronism. Measuring PRA and aldosterone levels under standardised conditions and determining the aldosterone-renin ratio will help in establishing the correct diagnosis.[1]

Liquorice is the root of *Glycyrrhiza glabra* and has been used as a medicinal herb in ancient Egypt and Greece to relieve symptoms in individuals with adrenal insufficiency, chronic hepatitis, cystitis, gastric ulcers, kidney stones and diabetes.[2] The active ingredient of liquorice is glycyrrhizic acid and together with its hydrolytic product glycyrrhetinic acid, have a well-known mineralocorticoid activity as they inhibit the action of 11-hydroxysteroid dehydrogenase 2.[3] As little as 50 g of liquorice per day may induce mineralocorticoid hypertension because of salt and water retention, and hypokalaemia which could be severe enough to cause myopathy or arrhythmias including ventricular fibrillation.[4] The combination of high salt intake[5] and the ingestion of salted liquorice can cause a large rise of BP and precipitate severe hypertension. Correct apt diagnosis and simple dietary advice to this patient resulted in normalisation of her BP and her electrolytes abnormalities.

OUTCOME AND FOLLOW-UP

The patient's BP quickly improved with the discontinuation of liquorice ingestion and all her antihypertensive medications were stopped. When the patient was reviewed in our clinic 6 months later, her 24 h urinary sodium excretion has reduced to 87 mmol and her BP remained normal at 124/82 mm Hg while on no treatment with normalisation of her plasma potassium and bicarbonate levels.

DISCUSSION

We could not find any previous publications of severe and difficult to treat hypertension caused by salted liquorice.

Learning points

- Taking adequate dietary history is very crucial in the evaluation of patients with hypertension.
- It is important to suspect and diagnose secondary hypertension, as it is potentially curable.
- High salt intake combined with liquorice ingestion can induce severe and difficult to treat hypertension.
- The finding of hypertension and hypokalaemia, whether spontaneous or induced by diuretics, should always raise the suspicion of secondary hypertension and in particular hyperaldosteronism.

Competing interests None.

Patient consent Obtained.

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