Angiotensin I-Converting Enzyme Levels and Renin-Aldosterone Axis Recovery after Cessation of Chronic Licorice Ingestion

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Dear Sir,

Excessive licorice ingestion may result in sodium and water retention, hypertension, hypokalemia and suppression of the renin-aldosterone system. Stewart et al. [1] proposed that licorice acts by inhibiting renal 11- β -hydroxysteroid dehydrogenase activity, thereby diminishing the conversion of cortisol to cortisone and resulting in high renal levels of cortisol, which is available to mineralocorticoid receptors.

Limited data exist concerning the recovery of the renin-aldosterone axis after the suppression that occurs during chronic ingestion of licorice [2,3]. We describe the recovery of the renin-aldosterone system and the normalization of angiotensin I-converting enzyme (ACE) levels in a patient with chronic licorice ingestion after licorice withdrawal.

A 34-year-old man was referred to us for evaluation of edema, hypertension and hypokalemia. One week before seeking medical advice, he noted the appearance of edema at the lower extremities. He also complained of weakness during this time. He was not taking any medications, and he did not report any more symptoms. The pharmacological dietetic history revealed he had been eating approximately 40 g of licorice candies daily for the past 2-3 years, and he had increased licorice ingestion to more than 100 g/day the week before the symptoms appeared. On examination the patient was a thin man with a blood pressure of 140/100 mm Hg. Pitting edema was present in the lower extremities. The rest of the physical examination was normal. Plasma sodium was 150 mmol/l, and plasma potassium was 3 mmol/l. Plasma renin activity, plasma aldosterone, and urinary aldosterone excretion were undetectable, plasma cortisol and ACTH levels were normal, and ACE was elevated during licorice ingestion (data shown in table 1). The patient stopped eating licorice after the initial evaluation, and he was seen at intervals of 2-6 weeks for continued clinical follow-up and hormonal measurements. Blood pressure returned to normal levels and edema disappeared 10 days after licorice withdrawal.

Data in mks/cgs are shown in parentheses.

ACE, plasma renin activity and aldosterone returned to normal levels by day 48 after licorice withdrawal (table 1).

The clinical features of our patient are similar to those described in previous cases of severe licorice intoxication [4]. Suppression of the renin-aldosterone system after chronic licorice ingestion varies widely, with a range of 2-12 months after the cessation of licorice intake. Recovery of the renin-aldosterone axis in our patient was slightly shorter, though urinary aldosterone was still low, a fact that has also been described [2]. These suppressive effects, although variable, are shorter than those that occur after the removal of an aldosterone-producing adenoma, which creates a similar state of chronic mineralocorticoid excess.

ACE levels are increased in patients with essential and renovascular hypertension, but they remain normal in patients with hyperaldosteronism [5]. There are no reported values of ACE levels in patients with other types of hypermineralocorticism, such as chronic licorice ingestion. Our patient showed high levels of ACE during the licorice ingestion period, which returned to normal values after licorice withdrawal. This fact could be helpful in the diagnosis of hypermineralocorticism syndromes, however, more studies to appropriately establish the utility of this test are mandatory before any definite conclusion is reached.

References

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Martinez Licorice Withdrawal