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Licorice with a Bitter Taste: A Case Report of Pseudo Hyperaldosteronism

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Background: The consumption of licorice can cause pseudo hyperaldosteronism. Glycyrrhizin, the major active component in licorice, inhibits the enzyme 11β hydroxysteroid dehydrogenase type 2, that converts cortisol to cortisone, increasing cortisol concentrations with excessive mineralocorticoid receptor activation.

Case History: A 55 years old man was admitted to Emergency Room due to high blood pressure values and headache. In the Internal Medicine Unit: normal CT brain, abdomen ultrasound, renal Doppler ultrasound and chest x-rays; laboratory exams revealed k 2.6 mEq/l (n.r. 3.5-4.5 mEq/l), urinary cortisol 435.5 mcg/24h (n.r. 28.5-213.7), serum cortisol 18.7 mcg/dl (n.r. 5-25), renin in orthostatism 8.30 uUI/ml (n.r. 4.40-46.10), renin in clinostatism 3.43 uUI/ml (n.r. 2.80-39.90), ACTH 40 pg/ml (n.r. up to 46), aldosterone in clinostatism 2.65 ng/dl (n.r. 1.75-23.20), aldosterone in orthostatism 1.790 ng/dl (n.r. 2.53-39.20). Abdominal MRI excluded adrenal pathologies. Patient who had quit smoking 5 months ago reported consuming large amounts of licorice every day. A diagnosis of pseudo hyperaldosteronism was made and the patient was discharged with normal potassium levels and antihypertensive therapy at home.

Discussion: Excessive intake of licorice can cause pseudo hyperaldosteronism. Aldosterone dosage is normal. The prognosis is generally favorable suspending licorice intake and correcting the electrolyte disorder. Often despite the suspension of licorice arterial hypertension persists.

References

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