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Licorice-induced Pseudohyperaldosteronism: Insights from Clinical Cases

Asaf Bijariakia*

Department of Medicine, Soroka University Medical Center, Beer Sheva 8453227, Israel

Introduction

Licorice, derived from the root of Glycyrrhiza species, is a widely consumed herb known for its sweet flavor and medicinal properties. However, its excessive consumption or prolonged use can lead to significant health consequences, particularly licorice-induced pseudohyperaldosteronism. This condition arises due to the presence of glycyrrhizin, a compound in licorice root that inhibits the enzyme 11β-Hydroxysteroid Dehydrogenase type 2 (11β-HSD2). This enzyme normally converts cortisol to cortisone in the kidney, thereby preventing cortisol from binding to mineralocorticoid receptors and causing mineralocorticoid-like effects. Pseudohyperaldosteronism manifests clinically with symptoms akin to primary hyperaldosteronism, including hypertension, hypokalemia, metabolic alkalosis and occasionally, muscle weakness. The condition presents diagnostic challenges due to its resemblance to other forms of aldosteronism, necessitating awareness among healthcare providers regarding licorice consumption and its potential health impacts [1].

Description

Mechanism of action of glycyrrhizin: Glycyrrhizin, the active component in licorice root, inhibits 11β-HSD2 enzyme activity in the kidney. This inhibition allows cortisol to persistently bind to mineralocorticoid receptors, mimicking the effects of aldosterone. As a result, sodium retention increases, leading to fluid retention and hypertension. Concurrently, potassium excretion is enhanced, resulting in hypokalemia, which further contributes to the pathophysiology of licorice-induced pseudohyperaldosteronism [2].

Clinical presentation and diagnostic challenges: The clinical presentation of licorice-induced pseudohyperaldosteronism varies but commonly includes hypertension, which may be severe and resistant to conventional antihypertensive treatments. Hypokalemia is a hallmark feature, often asymptomatic initially but potentially progressing to muscle weakness, cardiac arrhythmias and even rhabdomyolysis in severe cases. Metabolic alkalosis, due to excessive renal hydrogen ion excretion secondary to potassium wasting, may also be observed. Diagnosing licorice-induced pseudohyperaldosteronism requires a high index of suspicion, especially in patients with unexplained hypertension or electrolyte abnormalities [3]. Laboratory investigations typically reveal low serum potassium levels, elevated urinary potassium excretion and metabolic alkalosis. Measurement of plasma renin activity and aldosterone levels may initially suggest primary hyperaldosteronism; however, the absence of suppressed renin activity distinguishes licorice-induced pseudohyperaldosteronism.

Epidemiology and risk factors: The prevalence of licorice-induced pseudohyperaldosteronism is influenced by cultural dietary practices and the widespread availability of licorice-containing products. Countries where licorice consumption is common, such as parts of Europe and Asia, report higher incidences of this condition. Prolonged and excessive intake of licorice in various forms, including candies, teas, dietary supplements and herbal remedies, increases the risk of developing pseudohyperaldosteronism.

Management and treatment strategies: Management of licorice-induced pseudohyperaldosteronism primarily involves discontinuation of licorice consumption. This may be challenging due to its presence in a wide range of products and variations in glycyrrhizin content. Healthcare providers play a crucial role in educating patients about the potential health risks associated with licorice consumption and advising on alternative therapies for managing conditions for which licorice is traditionally used. For symptomatic patients, correction of electrolyte imbalances, particularly potassium supplementation, is essential to prevent complications such as cardiac arrhythmias and muscle weakness. Monitoring of blood pressure and electrolyte levels is recommended until normalization, which typically occurs within weeks after cessation of licorice exposure. In severe cases or when immediate correction is necessary, hospitalization and intravenous potassium administration may be warranted [4].

Public health implications and regulatory measures: Public health initiatives are essential to raise awareness about the potential health risks associated with licorice consumption. Regulatory measures aimed at limiting the glycyrrhizin content in licorice-containing products have been implemented in some countries to mitigate the incidence of licorice-induced pseudohyperaldosteronism. Labelling requirements and consumer education efforts can further aid in informing individuals about the safe consumption limits of licorice products and potential interactions with medications [5].

Conclusion

Licorice-induced pseudohyperaldosteronism exemplifies a unique form of secondary hypertension and electrolyte imbalance resulting from excessive licorice consumption. The condition underscores the importance of dietary habits in influencing health outcomes and the need for healthcare providers to consider licorice intake when evaluating patients with unexplained hypertension or electrolyte abnormalities. While the diagnosis can be challenging, early recognition and cessation of licorice consumption are crucial for preventing complications and achieving clinical resolution. Moving forward, continued research into the mechanisms of glycyrrhizin's action and its effects on mineralocorticoid receptors will enhance our understanding of licorice-induced pseudohyperaldosteronism. Public health strategies aimed at promoting awareness and regulating licorice-containing products can contribute to reducing the incidence of this potentially serious condition. By integrating these efforts, healthcare providers can effectively manage and prevent licorice-induced pseudohyperaldosteronism, thereby improving patient outcomes and promoting safer dietary practices.

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***Address for Correspondence:** Asaf Bijariakia, Department of Medicine, Soroka University Medical Center, Beer Sheva 8453227, Israel, E-mail: asafbijarkia@hotmail.com*

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Conflict of Interest

No conflict of interest.

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