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Licorice Consumption causing Hypokalemia and Lethal Dysrhythmias

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Introduction

This report describes a case in which approximately a two-month exposure of licorice root tea consumption containing glycyrrhizic acid (GA) produced generalized weakness, secondary hypertension, hyporeflexia and hypokalemia, leading to a reentry dysrhythmia of ventricular fibrillation.

Although licorice root has been used for medicinal purposes dating back centuries the potential toxic side effects can be life threatening without early recognition. Most licorice candies in the western world contain artificial flavoring but the use of actual licorice root containing GA is common in many Middle-Eastern, Far Eastern and European countries.

The purpose of this report is two-fold, first to describe the pathophysiological effects of GA that result in severe hypokalemia, muscle weakness and potential dysrhythmias and secondly to reiterate the importance of a complete history, taking into account cultural background when treating a patient emergently.

Presentation of Case

A 54-year-old female of Middle-Eastern descent presented to the emergency department via ambulance with complaints of, severe generalized weakness for about one week. The case was further complicated by a lack of communication as the patient did not speak or understand any English. Interpreter services were requested at this time.

The patient's initial presentation revealed an alert and afebrile patient with normal body mass index (BMI) and moderate hypertension of 165/90 mm Hg, Electrocardiogram revealed sinus rhythm with a prolonged QT. Physical exam findings were as follows cranial nerves II – XII intact, severe weakness of the proximal and distal muscles in all four limbs, without muscle wasting or tenderness and bilateral hyporeflexia to the patellar, ankle and triceps. Intravenous (IV) access was established, complete blood count (CBC), comprehensive metabolic panel (CMP), creatine kinase/myocardial band CK/MB, Troponin, urinalysis (UA) and urine toxicology, chest x ray (CXR) and computed tomography (CT) brain were ordered.

When interpreter services arrived the patient denied any recent history of nausea vomiting or diarrhea, chest pain, dyspnea, excessive sweating or use of diuretics

The patient denied any past medical history or taking any prescription, over the counter or herbal drugs.
Patient soon became unresponsive and pulseless showing ventricular fibrillation (VF) on cardiac monitor (CM). The patient was defibrillated with 200 joules and intubated. Patient returned to previous rhythm and demonstrated adequate signs of ventilation and perfusion.

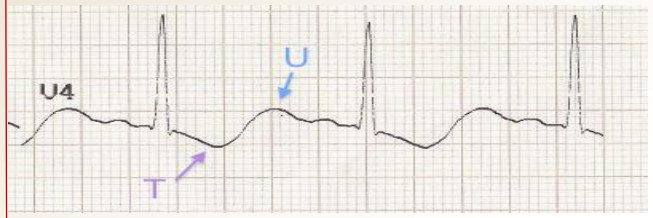
The patient's labs reported severe hypokalemia with a plasma potassium (K) at 1.6 mmol/L and metabolic alkalosis with a bicarbonate (HCO-3) level of 36 mmol/L. The patient's creatine kinase (CK) was moderately elevated at 800 U/L with a negative CKMB and troponin. All other stat labs were unremarkable. The patient was administered IV potassium and admitted to the cardiac intensive care unit (CICU) for further evaluation.

Upon further investigation with the patient's family, it was discovered that the patient had been consuming large quantities of an herbal tea made from licorice root that a family member had obtained from their native country. The most likely diagnosis was exogenously-induced apparent hypermineralocorticoidism (EIAH) secondary to licorice consumption seemed to be the most likely.

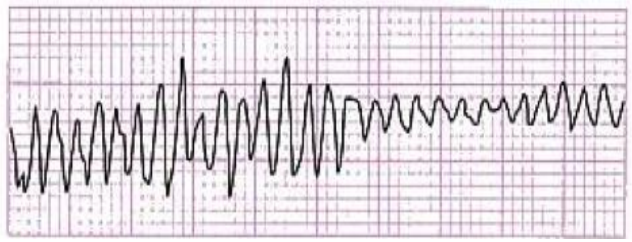
Signs and symptoms

Excessive licorice consumption resulting in symptomatic patients presenting to the emergency department in the United States is rare. However, signs and symptoms of EIAH have a varying range and can mimic other disorders, thus making emergency diagnosis difficult. Excessive licorice ingestion may result in sodium and water retention and suppression of the renin-aldosterone system (RAS) leading to a variety of symptoms that may include fatigue, muscle weakness to paralysis, rhabdomyolysis, new onset hypertension or increased elevation in systolic and diastolic pressures with patients that already have primary hypertension, hypokalemia to varying degrees, and cardiac dysrhythmias.

Initial EKG changes due to hypokalemia



Lethal EKG changes due to hypokalemia



Implications for Nursing Care

Implications for nursing care consist of a detailed patient history, complete physical assessment, interpretation of potential consequences of critical lab values and close observation.

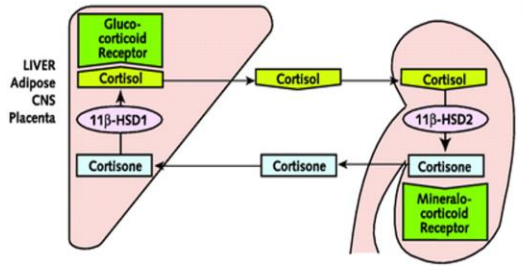
Underlying Pathophysiology and Significance

The underlying pathophysiology lies in the response to the chemical GA contained in licorice. Glycyrrhizic acid causes sodium retention through a cascade of chemical responses in the kidneys and adrenal cortex. The adrenal cortex makes two types of steroids, glucocorticoids such as cortisol and mineralocorticoids such as aldosterone.

The presence of GA inhibits the function of 11-beta-hydroxysteroid dehydrogenase2 (11-BHSD2), the chemical responsible for converting cortisol to cortisone a steroid that does not bind to mineralocorticord receptors (MR) (Hamidon & Jeyabaian, 2006). With the inhibition of cortisol to cortisone it allows for the excess cortisol to activate the renal mineralocorticoid receptors (MR) causing excess production of mineralocorticoid (Kaplan & Young 2014). This in effect suppresses the RAS, leading to increased water and sodium retention and depletion of potassium

. On a cellular level aldosterone creates a mechanism of action by stimulating the gene encoding of Na+ K+ ATPase ultimately leading to an increase in the number of Na+ pumps in the basolateral membranes of the tubular epithelial cells. By the aforementioned mechanism aldosterone stimulates the expression of a sodium
channel that facilitates uptake of sodium from the renal tubules creating an increased reabsorption of sodium and water consequently expanding extracellular fluid volume through the effects of osmosis. In addition there is an increased excretion of potassium (Elinay & Chajek-Shaul, 2003). Severe potassium depletion can lead to severe muscle weakness and cardiac reentry dysrhythmias resulting in ventricular fibrillation.

Cortisol to Cortisone



Conclusion

In conclusion, hypokalemia producing life-threatening results due to licorice root consumption is a rare emergency presentation. However, when hypokalemia exist without other explainable causes one must think of ingestion. The importance of a detailed history is an important element in the findings but not always immediately accessible in an emergent situation therefore emergency medicine often treats the symptoms first then looks for underlying causes.

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