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Gina Grosscup
Otterbein University, gina.grosscup@otterbein.edu

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Licorice Consumption causing Hypokalemia and Lethal Dysrhythmias
Gina Grosscup BA, RN, CEN
Otterbein University, Westerville, Ohio

Introduction

This report describes a case in which approximately a two-month exposure of licorice root tea consumption containing glycyrrhizinic acid (GA) produced generalized weakness, secondary hypertension, hypokalemia, and hypokalemia, leading to a near death 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 teas are consumed in the western world for its diuretic 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 sodium and water consequently expanding the membranes of the tubular epithelial cells, ultimately leading to an increase in the concentration of sodium which 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 secretion of potassium (Ouwendijk & Dees, 2005, April 2005). Severe hypokalemia can deadline to severe muscle weakness and cardiac reentry dysrhythmias resulting in ventricular fibrillation.

Presentation of Case

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 jolts and intubated. Patient returned to previous rhythm and demonstrated adequate signs of ventilation and perfusion.

The patient’s initial presentation revealed an alert and able patient with normal body mass index (BMI) and moderate hypertension of 105/90 mm Hg. Electrocardiogram revealed sinus rhythm with a prolonged QT. Physiologic and biochemical findings were as follows: normal serum levels of lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), creatinine, blood urea nitrogen (BUN), and uric acid. Urinalysis was positive for lipiduria, ketonuria, and proteinuria.

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 diuretic-induced apparent hyperreninemic hyperaldosteronism (AHH) secondary to licorice root consumption seemed to be the most likely.

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

Underlying Pathophysiology and Significance

The underlying pathophysiology lies in the chemical glycyrrhizinic acid (GA) contained in licorice. Glycyrrhizinic acid causes sodium retention through a cascade of chemical responses in the kidney 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 11b-hydroxysteroid dehydrogenase (11-BHDS) which results in an increased secretion of potassium (Ouwendijk & Dees, 2005, April 2005). Severe potassium depletion can deadline to severe muscle weakness and cardiac reentry dysrhythmias resulting in ventricular fibrillation.

Signs and symptoms

Excessive licorice consumption resulting in symptomatic patients presenting 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 the time.

The patient’s labs reported severe hypokalemia with a plasma potassium (K) at 1.6 mmol/L, and metabolic alkalosis with a bicarbonate (HCO3-) level of 46 mmol/L. The patient’s creatine kinase (CK) was moderately elevated at 802 U/L, with a negative CKMB and troponin. All complete blood count (CBC), and triceps. Intravenous (IV) access was established, complete blood count (CBC), and triceps. Intravenous (IV) access was established, complete blood count (CBC), and triceps. Intravenous (IV) access was established, complete blood count (CBC), and triceps. Intravenous (IV) access was established, complete blood count (CBC), and triceps. Intravenous (IV) access was established.

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.

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 look for underlying causes.

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