A Case of Hyperkalemia Secondary to Table Salt Alternative in a Patient with Normal Renal Function

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Introduction

Hyperkalemia is a common electrolyte derangement. In one retrospective study by Singer et al of 100,260 Emergency Department (ED) patient visits it was found that 1 in 11 ED patients presented with a potassium derangement (1). Presentations can vary widely, from asymptomatic to life threatening arrhythmias. While hyperkalemia is usually secondary to chronic kidney disease or medication use, a less commonly recognized cause of is dietary substitutions for everyday table salt. Ayach et al reported a patient with potassium of 9.8 mmol/L secondar to dietary substitutions but with acute kidney injury and ACE inhibitor use (2). We present a case of a patient with normal kidney function, and no other known risk factors for hyperkalemia, presenting with an initial potassium of 9.0 mmol/L after the initiation of common table salt replacement.

Case History and Studies

A 60-year-old female with no PMH presented to the ED for generalized weakness, nausea and 1 episode of emesis. See Table 1 for initial vital signs. EKG (Figure 1), chest x-ray, labs (CBC, CMP, Troponin, pro BNP, Lipase, TSH w/ reflex T4, Urinalysis) were ordered. Results were significant only for **Potassium 9.0 mmol/L** on the CMP. Creatinine was within normal limits at 0.70 mg.dL.

The patient was treated with 3 grams of calcium gluconate, 10mg of nebulized albuterol and 5 units Insulin with 25 mL of D50W. The intensive care team was consulted, and the patient was admitted to the ICU overnight for close monitoring. She was discharged in stable condition, and her final potassium level was **3.5 mmol/L**.

The patient revealed that she had recently started a table salt replacement called Nu-Salt because her primary care physician advised her to decrease her sodium intake. Nu-Salt is a popular salt substitute that is formulated with potassium chloride rather than sodium chloride. We strongly suspect this was the cause of her hyperkalemia.

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Figure 1

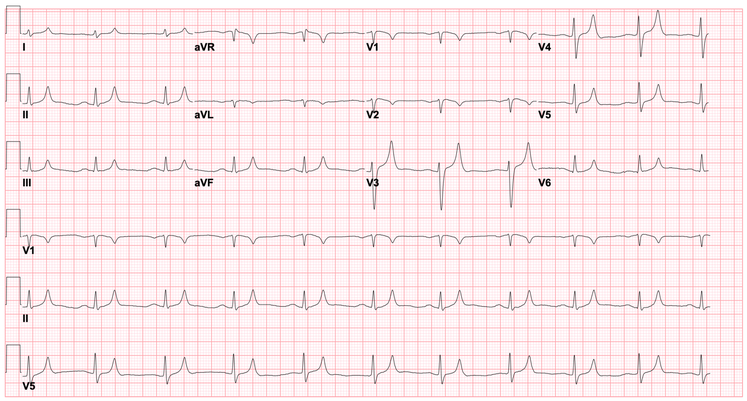

Table 1

Temp	BP	HR	RR	O2 Sat
97.3 F	84/62 mmHg	62/min	18/min	99%

Discussion

Hyperkalemia is a common cause of high morbidity/mortality in the Emergency Department (ED) due to the risk of dysrhythmias. The most common cause of hyperkalemia is impaired kidney function. Reports have described incidence as high as 40-50% in people with chronic kidney disease, in comparison to the general population, where prevalence is as low as 2-3% (3). A less commonly thought of cause of hyperkalemia includes exogenous supplementation. It is imperative that etiologies other than renal injury be considered as patients can have cardiac effects merely from potassium supplementation/substitution (John et al. 2010).

Primary care physicians often recommend to their patients that they decrease sodium intake. However, further education on nutrition is normally very limited. Patients aren't commonly educated on proper food substitutes or the risks involved with dietary modifications. Our case describes a patient who was advised to diminish her sodium intake and conversely increased potassium intake, thereby causing potentially life-threatening hyperkalemia despite normal kidney function.

Hyperkalemia is easily diagnosed through a metabolic profile and electrocardiography. Classic ECG findings of hyperkalemia include peaked T waves, shortened QT intervals, lengthened PR intervals, lost P waves, widened QRS complexes that can lead to sine waves (6). This can then lead to ventricular fibrillation, asystole, and death.

Clinical symptoms will vary broadly, from asymptomatic to severe hemodynamic instability. Management aims to stabilize the cardiac membrane to prevent dysrhythmia (7) followed by an attempt to both shift potassium out of the blood and then ultimately increase its excretion. Prognosis often depends on serum or plasma levels; patients with chronic hyperkalemia could generally be asymptomatic (8) while acute rises may lead to significant morbidity/mortality.

In this case, we describe a potentially fatal sequela of an apparently minor dietary change. The patient's acute presentation with hypotension, EKG changes and generalized weakness may have led to dysrhythmias if it had not been appropriately and aggressively treated.
Proper education about all the etiologies of the disease, including exogenous ingestion and dietary supplementation/substitution, is paramount.

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