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PERSISTENT SEVERE HYPERKALEMIA IN A PATIENT WITH NORMAL RENAL FUNCTION

Michael L. Tobin, MD

Case Report

A 75-year-old female with a past medical history significant for hypertension, hyperlipidemia, and colon cancer, status post colectomy with ileostomy, presented to the Methodist Hospital emergency room with muscular low back pain, which began a few hours prior, after abruptly bending over. Upon arrival to the hospital, she had no other complaints and noted that the back pain was starting to resolve without intervention.

In triage, her heart rate was measured at 38 beats per minute (BPM) with her other vital signs within normal limits. Her electrocardiogram (EKG) was read as a junctional rhythm with a rate of about 40 BPM, and a wide-complex QRS with what appeared to be a new Right Bundle Branch Block morphology. Previous EKGs, including one from less than a year earlier, were unremarkable. Basic laboratory studies, which returned while the patient was in the ER, revealed a potassium of 7.8 mmol/L with a creatinine of 1.0 mg/dL. Her other labs were unremarkable.

Her hyperkalemia was initially treated in the ER with calcium gluconate, intravenous (IV) insulin, and oral sodium polystyrene sulfonate, which resulted in narrowing of her QRS complex with return of P-waves. Repeat potassium improved to 5.9 mmol/L. Despite this initial treatment, repeat EKG within an hour redemonstrated QRS complex widening with junctional rhythm. Repeat potassium level at this time was 7.2 mmol/L, and at this point, the patient was admitted to the medical intensive care unit (ICU) for further evaluation and treatment.

Over the next day, this pattern of fluctuating potassium levels along with electrocardiographic changes continued. After nearly 24 hours of continued treatment with IV insulin and multiple doses of sodium polystyrene sulfonate, her potassium levels eventually stabilized to the normal range. Of note, the patient never had any subjective complaints, and her creatinine never elevated above her baseline level of 1.0 mg/dL. Urinalysis was unremarkable and urine electrolytes were significant only for mildly elevated urinary potassium. It was confirmed that the patient was taking lisinopril 40 mg daily, which had been at a stable dose for several years for hypertension. Eventually, further questioning done by the patient's nurse revealed that the patient had recently been attempting to improve her health by cutting salt out of her diet. In its place, she began using a "salt substitute" product, which she thought was healthier. In fact, she actually preferred this product to salt, and began using a lot of it over the few weeks leading up to her presentation. Ultimately, it was felt that her excess dietary potassium intake in the form of the salt substitute product led to high total body potassium levels, explaining why it was so difficult to correct her hyperkalemia.

Discussion

Hyperkalemia is a common electrolyte abnormality, which can be lethal in severe cases. As such, a high index of suspicion for hyperkalemia is needed for patients at high risk or who are found to have electrocardiographic changes which are consistent with hyperkalemia. This patient's presentation is unusual in several ways, which partly contributed to the delay in her diagnosis.

First, her physical complaints and her medical background were not typical of a patient with severe hyperkalemia. The patient population most at risk for severe hyperkalemia are those with significant renal disease and who may already be dialysis dependent.¹ In addition to presenting with arrhythmias, hyperkalemia can also present with neuromuscular complaints, ranging from weakness to fasciculations, and frequently with nonspecific gastrointestinal (GI) complaints such as abdominal cramping and vomiting. Although the patient's initial presenting symptom was muscular back pain, the mechanism of her injury does not suggest that hyperkalemia played any role. In addition, she had no other cardiac or GI complaints.

Second, despite the fact that the patient was taking an angiotensin-converting enzyme (ACE)-inhibitor, she appeared to have overall normal renal function. Since potassium is primarily cleared from the body via the kidney, most cases of severe hyperkalemia are seen in patients with a significant degree of renal impairment or those who are on medications such as ACE-inhibitors, angiotensin II receptor blockers (ARBs) or potassium-sparing diuretics, which may affect how efficiently potassium is excreted.^{1,2} In the absence of these predisposing factors, it is very unusual for excess intake of dietary potassium alone to overwhelm the renal excretion mechanisms and cause severe hyperkalemia.³

Third, the patient's EKG was not initially recognized as representing hyperkalemia. Less severe hyperkalemia likely would have been recognized based on its characteristic finding of sinus rhythm with peaked T-waves. Severe hyperkalemia would have been identified based on a sine-wave pattern.² It is important to note the standard progression of electrocardiographic changes that occur with progressive hyperkalemia. The classic finding of narrow-based, peaked T-waves, which are usually best seen in the inferior and mid-precordial leads, occur at potassium levels over 5.5 mmol/L, but are only seen in a relatively small percentage of those with this mild degree of hyperkalemia. At potassium levels beyond 6.5 mmol/L, QRS complex widening and PR segment prolongation can occur. At levels of 8 to 9 mmol/L, a sinoventricular rhythm may be seen, in which the sinoatrial node directly stimulates the ventricles

without evidence of atrial activity. This is because the SA node is less susceptible to the effects of hyperkalemia than is atrial tissue. As such, the P-wave can disappear and the rhythm can appear to be junctional or ventricular. At levels over 10 mmol/L, the SA node no longer functions, and a junctional or ventricular escape takes over the pacemaker function of the heart, eventually with the QRS complex blending with the T-wave to form the classic sine-wave pattern, which is seen just prior to ventricular fibrillation and asystole. This patient's presenting EKG, which showed a wide complex QRS without P-waves at a rate of 40, was consistent with more severe hyperkalemia than would be predicted by her potassium level of 7.8 mmol/L. One additional finding which was useful in making the electrocardiographic diagnosis of hyperkalemia was that the QRS widening was present throughout the entire complex, rather than just at the terminal portion which would be expected with a true Right Bundle Branch Block.²

With the large focus that has recently been put on modifiable risk factors for cardiovascular disease development, dietary sodium intake has become an emerging topic in public health and policy, and has received much attention in the media. Along with this, salt substitutes, which often are composed of potassium chloride, have become more popular and may be viewed by the public as a healthy alternative to standard table salt. Potassium-based salt substitutes have been noted on many occasions to have led to significant hyperkalemia.³ Of particular concern is that patients who are on medications such as ACE-inhibitors are those who may already have cardiovascular disease, renal disease, or diabetes, and therefore would benefit greatly from reducing dietary sodium intake. However, this is

also the population that would be most at risk for significant hyperkalemia from excess dietary potassium intake.⁴ One recent study⁵ suggested that decreasing dietary sodium while increasing dietary potassium may result in overall mortality benefit. This study was reported on in a recent New York Times article,⁶ touting the benefits of patients improving their sodium to potassium intake ratio, but never mentioning that in certain populations, this can lead to potentially severe hyperkalemia and adverse outcomes. This case emphasizes the importance that must be placed on having high clinical suspicion for hyperkalemia, especially in the patient population most at risk, and of course, the diagnostic value of taking a careful history.

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"Ben Franklin Fireworks"

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