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☐ HYPERKALEMIA IN THE EMERGENCY DEPARTMENT: YES, A NEED FOR FURTHER EVIDENCE, BUT DO NOT DISCOUNT WHAT WE HAVE

☐ To the Editor:

We thank Dr. Lemoine and colleagues for their insightful Letter to the Editor regarding therapies in hyperkalemia. The authors of this letter discuss first-line therapies for transcellular shift and excretion of potassium and raise questions regarding the specific serum potassium level defining a hyperkalemic emergency, what therapies should be initiated in the emergency department (ED), and the specific serum potassium level at which treatment should be started. The authors of this letter also conducted a survey evaluating specific potassium treatment thresholds and therapies, finding significant variation in treatment thresholds and strategies. Such a study is an important contribution to the medical literature, and authors call for "a rigorous evaluation of the first-line treatments of hyperkalemia" in the ED.

Dr. Lemoine is correct in calling for further evidence regarding treatment of hyperkalemia, specifically at what level of serum potassium medical management is indicated. Potassium is an essential ion in cellular physiology, and elevated serum potassium levels can destabilize cardiac membranes and result in dysrhythmia and death (1–4). Classically, electrocardiogram (ECG) changes include peaked T waves, prolonged PR interval, widened QRS complex, loss of the P wave, a "sine wave" appearance, and asystole (5–7). However, providers should not rely on these "classic" findings, as discussed in our review, and patients can progress from sinus rhythm to ventricular fibrillation (5–8). No clear order of ECG changes has been found in animal models or, more importantly, the clinical setting. The sensitivity of ECG for hyperkalemia

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ranges from 34% to 43%, with a sensitivity of 55% to 62% in detecting levels >6.5 mmol/L (5–7). A recent study suggested several ECG findings to be associated with adverse events, including QRS prolongation (relative risk [RR] 4.74), bradycardia <50 beats/min (RR 12.29), and junctional rhythm (RR 7.46), which all occurred prior to treatment with calcium (9). The most important therapy in hyperkalemia management is cardiac membrane stabilization with calcium if these findings are present (1–3).

Patients without clear ECG findings of hyperkalemia present challenges to the emergency provider regarding therapy. As we discuss in our review, providers must evaluate the clinical context and avoid relying solely on a single potassium level or normal ECG (1). Patient comorbidities and the rate of serum potassium increase are directly related to the clinical manifestations of hyperkalemia (1–4,6). The faster the rate of rise, the more drastic the effect on the cardiac membrane. The majority of patients will demonstrate some ECG abnormalities at levels >6.5 mEq/L, especially if patients do not have chronic renal disease/end-stage renal disease (ESRD) (2,3,8). Patients with chronically elevated serum potassium (such as those with ESRD) often do not demonstrate the classic progression of ECG findings with hyperkalemia due to a rebalanced transmembrane gradient (1,8). While we agree that determining evidence-based levels for treatment is vital, this is difficult due to the heterogeneous manifestations of hyperkalemia, in part stemming from different patient populations (ie, those with chronically elevated serum potassium) and the impact of potassium's rate of serum increase.

Dr. Lemoine also discusses the potential treatment strategies and the wide variation found in their survey. We applaud the authors in the undertaking of this study demonstrating the variation in hyperkalemia treatment, with various regimens including sodium polystyrene sulfonate (SPS), insulin/glucose, β-agonists, and sodium bicarbonate, in combination or alone. After transmembrane stabilization with calcium, transcellular shift of potassium with insulin/glucose and β-agonists is important, and a combination of therapies appears to be more efficacious than a single medication (1–3,10). Following transcellular shift, excretion is necessary. Many providers in the survey used a combination of therapies, but interestingly, use of SPS appears to be common, with 44% of the emergency physicians starting treatment with SPS alone for potassium levels 5.5–6.5 mEq/L. The evidence behind SPS is extremely poor, based on studies with no comparators, numerous confounders, and little statistical analysis (1–3). As we discuss in our review, no controlled human studies demonstrate increased fecal potassium excretion with SPS, and the risks of constipation, obstruction, and even colonic necrosis warrant caution (1–3,11,12). If patients are not anuric but are hypovolemic, i.v. fluids should be used, while avoiding normal saline due to the potential for worsening metabolic acidemia. If the patient has normal volume status or is hypervolemic, a diuretic such as furosemide is recommended. Combination diuretic therapy may demonstrate greater ability to excrete potassium. Patients who are anuric require hemodialysis. Other medications include patiromer and sodium zirconium cyclosilicate (ZS-9) (1).

Ultimately, further evidence is required for what serum potassium thresholds require therapy. However, hyperkalemia can result in severe dysrhythmias at various serum levels, dependent on the rate of serum rise and patient comorbidities. Further evaluation of diuretics, patiromer, and ZS-9 is necessary, and we recommend avoiding SPS.

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☐ PLEURAL EFFUSIONS ASSOCIATED WITH SPINAL TUBERCULOSIS

☐ **To the Editor:**

Unusual complications of spinal tuberculosis include not only bilateral pleural effusions, where both effusions occur simultaneously, as in the recent case report (1). There is also the example of a 25-year-old man with spinal tuberculosis, and in whom the right-sided pleural effusion appeared at the time when the left-sided pleural effusion was resolving in response to antituberculous chemotherapy (2).

Chylothorax is another category of pleural effusion associated with spinal tuberculosis. This was radiographically documented as bilateral pleural effusion in a 60-year-old woman with coexisting spinal tuberculosis. Stigmata of chylothorax, in both pleural effusions, included milky appearance and a triglyceride content of 1092 mg/dL. Spinal tuberculosis was characterized by computed tomography (CT) showing bilateral soft-tissue shadowing at T4–T5 and also at T10–T11, in the absence of evidence of lymphadenopathy. Further CT evaluation revealed extensive anterior erosion of T10–T11, and mottled appearance of adjacent vertebral architecture with a sclerotic border demarcating a central cavity. This was strongly suggestive of tuberculous spondylitis, but the patient refused needle aspiration of T10–T11. On the basis of the CT findings and on the basis of a strongly positive tuberculin skin test, the patient was prescribed antituberculous chemotherapy. After 12 months of therapy the pleural effusions resolved, and there was a striking improvement in the bony abnormalities (3).

Finally, tuberculous spondylitis may be complicated both by tuberculous empyema and by the occurrence of chylothorax, the latter subsequent to repeated aspiration of the tuberculous empyema. The above was the course of events in a 67-year-old man in whom these complications were attributable to rupture of a spinal abscess in the T12/L1 region. Initially, the patient had a purulent pleural effusion that communicated with the paravertebral abscess through the diaphragm. The purulent pleural effusion contained acid-fast bacilli fully sensitive to antituberculous chemotherapy. The subsequent chylous effusion was characterized by a triglyceride content > 110 mg/dL (4).

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☐ PLEURAL EFFUSIONS ASSOCIATED WITH SPINAL TUBERCULOSIS

The Reply:

I read with interest the letter by Dr. Jolobe. His argument was that in a patient with thoracic spinal tuberculosis, chylothorax could be the cause of bilateral pleural effusion. I agree with Dr. Jolobe. Chylothorax is, in fact, a collection of chylous lymphatic fluid within the pleural cavity. Obstruction of thoracic duct and disruption of lymphatic flow within the thoracic cavity can result in chylothorax. Chylothorax has many causes, like lymphomas and other thoracic malignancies: iatrogenic after thoracic surgeries, traumatic chest injuries, and congenital duct abnormalities (1,2). In India, filariasis is a relatively more common cause of chylothorax than tuberculosis.

Diagnosis of chylothorax requires demonstration of cholesterol and triglycerides in the pleural fluid. Occasionally, pseudochylothorax develops when an exudative pleural effusion remains in the pleural space for years, and the pleural fluid becomes enriched with cholesterol (1,2).

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