Targeted Electrolyte Replacement in Patients With Ebola Virus Disease

To the Editor—We would like to highlight the importance of performing tests to facilitate targeted electrolyte replacement in patients with Ebola virus disease (EVD).

Patients with EVD often develop gastrointestinal symptoms including abdominal pain, nausea, and anorexia followed by vomiting and profuse diarrhea. The gastrointestinal losses may be significant, leading to profound hypovolemia and electrolyte abnormalities. The mechanism of death remains unknown in many cases, but sudden death has been reported and could be due to electrolyte disturbances. Potassium losses in EVD may be significant, and symptoms of severe hypokalemia include generalized weakness and lassitude, muscle necrosis, impaired respiratory function due to ascending paralysis, and cardiac arrhythmias, some of which have been reported among EVD-positive patients [4–6].

During the initial response to the current outbreak in West Africa, many organizations opened isolation centers aiming to prevent virus transmission and provide symptomatic management of patients [7]. Laboratory or point-of-care electrolyte testing was not always available and many patients did not have electrolytes measured. This was problematic when patients were no longer able to maintain oral hydration and intravenous fluid resuscitation was required [7]. A recent report on the clinical features of EVD-positive patients discussed the limitations of such empiric therapy and emphasized the need for routine blood tests to guide symptomatic treatment [5]. It will be important in future outbreaks for clinicians treating patients empirically to be aware of the quantity of electrolyte replacement required, particularly potassium.

The United Kingdom’s Defence Medical Services opened and staffed an Ebola treatment unit in Kerry Town, Sierra Leone, in November 2014. It included both laboratory and bedside point-of-care testing for blood biochemistry. We reviewed the charts of 36 consecutive patients with confirmed EVD treated there and examined the quantity of potassium replacement given by oral and intravenous routes. Oral potassium supplements were not initially available, so 20 mmol of the intravenous preparation of potassium chloride was mixed with fruit juice to create a well-tolerated oral preparation. Intravenous potassium chloride was delivered as 20–40 mmol/L of sodium chloride, in lactated Ringer’s solution or, if central venous catheter access was available, as 40 mmol potassium chloride in 100 mL sodium chloride solution. Patients were also offered oral rehydration solution.

Potassium replacement varied widely among our patients during admission, ranging from 0 to 630 mmol (mean, 193 mmol; standard error of the mean, 30 mmol) (Figure 1). There was no significant association between potassium replacement and mortality, however, the total quantity of potassium given and length of admission strongly correlated (Spearman $r = 0.61$ [95% confidence interval,.34–.78]; $P < .0001$).

These data support the requirement for rapid deployable point-of-care testing in future outbreaks to identify, monitor, and appropriately treat electrolyte losses.

Figure 1. Potassium replacement among patients with Ebola virus disease in a treatment unit in Sierra Leone, their outcome, and length of admission.
which may be significant and ongoing during patient admission. Given the overall costs associated with the response to an EVD outbreak, this measure is simple and inexpensive [8]. Furthermore, the inventive use of novel oral preparations of potassium replacement, when conventional oral supplements are not available, is also highlighted, demonstrating a role for such an approach in resource-limited settings.

Note

Potential conflicts of interest. All authors: No potential conflicts of interest. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Katherine A. Clay,1 Andrew M. Johnston,1 Alastair Moore,1 and Matthew K. O’Shea1,2
1 Academic Department, Royal Centre for Defence Medicine (Academia and Research), Birmingham, and 2 The Jenner Institute, Nuffield Department of Medicine, University of Oxford, United Kingdom

References