CASE REPORT

Prolonged Brackish Water Exposure: A Case Report

Scott B. Hughey, MD¹; Kari L. McCoy, DO²; Aaron E. Mills, DO²¹Naval Medical Center Portsmouth, Portsmouth, Virginia; ²Eastern Virginia Medical School, Norfolk, Virginia

Exposure to and consumption of brackish water are associated with an elevated risk of infection, hypernatremia, and hypothermia. Minimal data exist to support the diagnosis and treatment of patients with long-term brackish water exposure. We present a case of a patient who spent 5 to 10 d semisubmerged in the Elizabeth River in coastal Virginia. A 55-y-old male presented via ambulance after 5 to 10 d of being “stuck in the mud.” He was hypernatremic, with a sodium of $176 \text{ mEq} \cdot \text{L}^{-1}$, hypothermic to 34.5°C (94.1°F), and hypotensive at $88/50 \text{ mm Hg}$, with a sodium concentration of $176 \text{ mEq} \cdot \text{L}^{-1}$ and an osmolality of $412 \text{ mosm} \cdot \text{kg}^{-1}$. He developed pneumonia, with respiratory cultures growing *Vibrio parahaemolyticus*, *Klebsiella oxytoca*, and *Shewanella* algae. He had pustules, which grew *Aeromonas hydrophilia* and *Aeromonas caviae*. A nasogastric tube was placed. Using suction, 500 mL of coarse sand and gravel was removed from his stomach. Antibiotics and intravenous fluids were given. The patient fully recovered after 3 wk and was discharged to rehabilitation. Exposure to brackish water can present a unique set of infectious and metabolic complications. Initial care should include treatment of metabolic derangements, such as hypovolemia, hypernatremia, and hypothermia, and treatment of infections with antibiotics based on knowledge of the most likely causative organisms.

Keywords: hypernatremia, *Vibrio parahaemolyticus*, *Aeromonas caviae*, Elizabeth River, Virginia

Introduction

Tidewater Virginia is an area of the United States with brackish and freshwater waterways. Brackish water estuaries are coastal habitats where the salt concentration is less than that of ocean water but generally greater than that of true freshwater, usually between 0.5 to 35 parts per thousand.³ The Elizabeth River is located in southeastern Virginia. Its waters empty into the Chesapeake Bay, which leads to the Atlantic Ocean. Short-term exposures to brackish water are common, with patients falling from boats, capsizing, or getting lost in the water. However, long-term exposure to this environment is uncommon and provides a unique set of diagnostic and treatment challenges. Elevated salinity makes water unsuitable for human consumption. Patients exposed to brackish water are at risk for hypernatremia, hypothermia, and waterborne infections.^{1,2} There is minimal published research to guide therapy, and the ability to conduct controlled research is

limited. In this article, we add to the limited body of literature by presenting a case of a 55-y-old male who was trapped in brackish water for 5 to 10 d.

Case

A 55-y-old man fell into the water of the Elizabeth River, a brackish water tributary of the Chesapeake Bay, and was stuck in a mud-silt layer adjacent to a jetty. The patient reported walking along the riverbank at night and slipping into the water. He was unable to swim but floated for a short period until he ended up on the edge of a jetty, which allowed him to lie semisupine with his head above the water. He described being unable to free himself from the mud, although he had sustained no injuries. He was there for an unknown period of time, but reported seeing 5 to 10 sunrises. He drank the water because he was thirsty. He also reported crabs, described as being 10 to 12 cm (4 to 5 in) wide, pinching him. The National Oceanic and Atmospheric Administration (NOAA) reported the temperature at that time to have been 28°C (83°F).

Upon arrival at the emergency department, the patient's rectal temperature was 34.5°C (94.1°F), blood pressure was

Corresponding author: Scott B. Hughey, MD, Naval Medical Center Portsmouth, Anesthesia, 620 John Paul Jones Circle, Portsmouth, VA 23708; e-mail: scott.b.hughey@gmail.com.

Submitted for publication October 2019.

Accepted for publication April 2020.

88/50 mm Hg, heart rate was 82 beats·min⁻¹, respiratory rate was 41 breaths·min⁻¹, and oxygen saturation was 94%. The physical examination was notable for coarse breath sounds bilaterally, diffuse bullae, significant lower extremity edema, maceration of the feet and hands, and pallor of the extremities. Neurologically, he was able to move his extremities and could answer questions, although his speech was limited because of his worsening respiratory status. Dried mud and sand covered his entire body.

The initial computed tomography (CT) scan showed an infiltrate consistent with pneumonia. A follow-up chest CT performed approximately 36 h later demonstrated bilateral necrotizing pneumonia, along with an area concerning for septic emboli. CT of the abdomen was notable for the appearance of intestinal contrast despite no agent having been administered. This finding was later identified as sand the patient had ingested. The initial laboratory evaluation was significant for a sodium level of 176 mEq·L⁻¹ (normal 136–144), chloride of 136 mmol·L⁻¹ (normal 96–106), bicarbonate of 13 mmol·L⁻¹ (normal 23–29), blood urea nitrogen of 152 mg·dL⁻¹ (normal 6–20), creatinine of 6.3 mg·dL⁻¹ (normal 0.8–1.2), and a white blood cell count of 35,900 (normal 4000–11,000) with 90% segmented neutrophils (normal 40–60).

He was cautiously resuscitated because of the hypernatremia, to avoid too rapid a correction of his sodium. He was hypothermic and actively rewarmed with infrared lamps.⁴ Two hours after arrival to the emergency department, the core temperature had improved to 36.1°C (97.1°F). His lower extremities became erythematous starting at the knees and then progressing distally. Over a period of approximately 1 h, he began developing pustular lesions on the entire body. These were more concentrated on the lower extremities and posterior thorax. During the third hour, he developed worsening hypotension requiring vasopressor support and respiratory distress with increased work of breathing requiring intubation. Blood cultures were obtained, as well as respiratory cultures and cultures from the pustular lesions.

Vancomycin and ceftazidime were started for empiric and broad-spectrum coverage, owing to clinical evidence of septic shock. Additional coverage with doxycycline was added out of concern over aquatic organisms, specifically *Vibrio parahaemolyticus*. A nasogastric tube was placed and returned approximately 500 mL of coarse sand. The hypernatremia was slowly corrected with a goal of 6 to 8 mEq·L⁻¹ daily. This was initially managed with Lactated Ringers but was transitioned to D5W with sodium bicarbonate because of slow response in sodium correction. Calculated free water deficit was determined to be 22 L. Free water, administered through a nasogastric tube, was also started on the first hospital day when sodium was 176

mmol·L⁻¹. Blood cultures had no growth, whereas the respiratory cultures grew *Vibrio parahaemolyticus*, *Klebsiella oxytoca*, and *Shewanella algae* gram-negative marine bacteria. The pustules grew *Aeromonas hydrophilia* and *Aeromonas caviae*. The patient's respiratory status slowly improved, and he was extubated after 8 d. The skin lesions improved and resolved with systemic antibiotics and local wound care. Vancomycin was discontinued after no methicillin-resistant *Staphylococcus aureus* was identified in cultures. Because of poor response to ceftazidime, he was transitioned to meropenem after 10 d, for a 30-d course to treat pulmonary septic emboli and necrotizing pneumonia. Doxycycline was continued for 14 d after repeat bronchoscopy showed no further *Vibrio parahaemolyticus*. Eventually, he made a full recovery.

Discussion

Prolonged marine exposure is associated with a variety of medical issues. This case highlights many of these issues, in particular marine pathogens, aspiration, drowning, dehydration, saltwater ingestion, and hypothermia. Marine pathogen exposure via breaks in the skin are commonly reported in fishermen, recreational boaters, and others who spend time in and around the water. The patient reported crabs pinched his legs. This may have caused breaks in the skin that could be the route for introduction of pathogens. Common pathogens include *Vibrio parahaemolyticus*, *Aeromonas*, and *Pseudomonas*. *Aeromonas* spp infections are common in marine wounds. *Vibrio* pneumonia has been reported in individuals who had drowning episodes.^{5,6} The presence of *Shewanella algae* in the lungs is consistent with a drowning incident; this gram-negative rod is only found in marine environments. If the patient was truly stuck in the mud, the water level change with tides could certainly have put him at risk for aspiration. Seawater, when aspirated, leads to parenchymal lung damage that impairs oxygen exchange across the alveoli and can lead to pneumonitis. In addition, the amount of sand aspirated from the stomach suggests that a significant amount of seawater was ingested. Doxycycline was used to treat waterborne organisms. It is our recommendation to use doxycycline for empiric coverage against marine organisms until culture data are available to guide therapy. Previous studies investigating seawater aspiration have shown that the majority of organisms collected were *Enterobacteriaceae* with low rates of antibiotic resistance.⁷

The water temperature measured by a nearby NOAA buoy was 28°C (83°F). This temperature had been nearly consistent for the previous 10 d based on NOAA records. In a 1946 study of shipwreck victims,⁸ water temperature

greater than 27°C (80°F) was associated with survival after prolonged water immersion. Our patient arrived with mild hypothermia at a temperature of 34.5°C (94.1°F). We initially suspected the patient's estimate of 5 to 10 d was incompatible with survival. Based on the 1946 study, however, it is possible that he was in the water for an extended period of time.

The patient drank brackish owing to extreme thirst. The salinity of brackish water varies by location and tidal changes. From where the patient was extricated, the salinity may have been close to that of seawater. Significant morbidity is associated with a serum osmolality above 370 mmol·L⁻¹ and serum sodium greater than 170 mEq·L⁻¹.^{9,10} These data are based on hypernatremia caused by dehydration, whereas our patient had dehydration and had ingested hypertonic saltwater. On arrival his calculated serum osmolality of 412 mmol·L⁻¹ and a serum sodium of 176 mEq·L⁻¹. Because of the patient's rapid deterioration and exhaustion on arrival to the hospital, it was difficult to assess neurologic function during the limited time between arrival and intubation. He was oriented to person and place, although he was unsure of the date.

Volume replacement in hemodynamically unstable patients is critical, but rapid correction of hypernatremia can cause harm. The primary focus of resuscitation should be on fluid replacement. Data are limited on the appropriate speed for correction of hypernatremia. Rapid correction is associated with cerebral edema and seizures.¹¹⁻¹³ However, recent evidence showed that critically ill patients who were corrected faster than 0.5 mmol·L⁻¹·h⁻¹ did not develop neurologic side effects from rapid correction of hypernatremia. In one study, there was a higher mortality rate associated with slow correction (<0.5 mmol·L⁻¹·h⁻¹).¹⁴ We corrected the hypernatremia at a rate of 0.5 mmol·L⁻¹·h⁻¹, with 0.45% saline.

Conclusions

This case demonstrates an unusual situation of long-term exposure to brackish water and illustrates some of the possible complications. It demonstrates some of the uncommon brackish water microbiota of this region and suggests antibiotic management, specifically with the empiric use of doxycycline. It can be a challenge to balance the management of hypernatremia with the need for volume resuscitation in patients with brackish water exposure, because of the need for slow correction of hypernatremia and rapid correction of dehydration.

Author contributions: AEM, KLM, and SBH all contributed equally to the patient identification and manuscript preparation.

Financial/Material Support: None.

Disclosures: The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the US government. Lt Hughey is a military service member. This work was prepared as part of his official duties. Title 17 USC 105 provides that "Copyright protection under this title is not available for any work of the United States Government." Title 17 USC 101 defines a US government work as a work prepared by a military service member or employee of the US government as part of that person's official duties.

References

1. Diaz JH, Lopez FA. Skin soft tissue and systemic bacterial infections following aquatic injuries and exposures. *Am J Med Sci*. 2015;349(3):269–75.
2. Addleman M, Pollard A, Grossman RF. Survival after severe hypernatremia due to salt ingestion by an adult. *Am J Med*. 1985;78(1):176–8.
3. US Department of Commerce, National Oceanic, Atmospheric Administration. National Ocean Service Education: Estuaries. Available at: https://oceanservice.noaa.gov/education/kits/estuaries/estuaries01_what.html. Accessed December 19, 2019.
4. McCullough L, Arora S. Diagnosis and treatment of hypothermia. *Am Fam Physician*. 2004;70(12):2325–32.
5. Ender PT, Dolan MJ. Pneumonia associated with near-drowning. *Clin Infect Dis*. 1997;25(4):896–907.
6. Kelly MT, Avery DM. Lactose-positive *Vibrio* in seawater: a cause of pneumonia and septicemia in a drowning victim. *J Clin Microbiol*. 1980;11(3):278–80.
7. Robert A, Danin PÉ, Quintard H, Degand N, Martis N, Doyen D, et al. Seawater drowning-associated pneumonia: a 10-year descriptive cohort in intensive care unit. *Ann Intensive Care*. 2017;7(1):45.
8. Molnar GW. Survival of hypothermia by men immersed in the ocean. *J Am Med Assoc*. 1946;131:1046–50.
9. Arieff AI, Guisado R. Effects on the central nervous system of hypernatremic and hyponatremic states. *Kidney Int*. 1976;10(1):104–16.
10. Madea B, Lachenmeier DW. Postmortem diagnosis of hypertonic dehydration. *Forensic Sci Int*. 2005;155(1):1–6.
11. Peces R, Ablanado P, Alvarez J. Central pontine and extrapontine myelinolysis following correction of severe hyponatremia. *Nephron*. 1988;49(2):160–3.
12. Laureno R, Lamotte G, Mark AS. Sequential MRI in pontine and extrapontine myelinolysis following rapid correction of hyponatremia. *BMC Res Notes*. 2018;11(1):707.
13. Laureno R, Karp BI. Pontine and extrapontine myelinolysis following rapid correction of hyponatremia. *Lancet*. 1988;1(8600):1439–41.
14. Alshayeb HM, Showkat A, Babar F, Mangold T, Wall BM. Severe hypernatremia correction rate mortality in hospitalized patients. *Am J Med Sci*. 2011;341(5):356–60.