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Using Esophageal Temperature Management to Treat Severe Heat Stroke: A Case Report

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ABSTRACT

BACKGROUND: Exertional heat stroke (EHS) is defined by a core body temperature that exceeds 40°C with associated central nervous system dysfunction, skeletal muscle injury, and multiple organ damage. The most important initial focus of treatment involves reduction of patient temperature. First approaches to achieve temperature reduction often include ice packs, water blankets, and cold intravenous fluid administration. When these measures fail, more advanced temperature management methods may be deployed but often require surgical expertise. Esophageal temperature management (ETM) has recently emerged as a new temperature management modality in which an esophageal heat transfer device replaces the standard orogastric tube routinely placed after endotracheal intubation and adds a temperature modulation capability. The objective of this case study is to report the first known use of ETM driven by bedside nursing staff in the treatment of EHS. **METHOD:** An ETM device was placed after endotracheal intubation in a 28-year-old man experiencing EHS over a 5-day course of treatment. **RESULTS:** Because the ETM device was left in place, when the patient experienced episodes of increasing temperature as high as 39.1°C, which required active cooling, nursing staff were able to immediately adjust the external heat exchange unit settings to achieve aggressive cooling at bedside. **CONCLUSION:** This nurse-driven technology offers a new means to rapidly deploy cooling to critically ill patients without needing to implement advanced surgical approaches or obstruct access to the patient, freeing the provider to continue optimal care in high-morbidity conditions.

Keywords: cooling devices, esophageal cooling, exertional heat stroke, targeted temperature management, therapeutic hypothermia

CASE STUDY

Exertional heat stroke (EHS) is a life-threatening medical condition defined by a core body temperature that exceeds 40°C with associated central nervous system dysfunction that typically includes skeletal muscle injury and multiple organ damage.[1] Risk factors for EHS include strenuous exercise in high ambient temperature and humidity, a lack of acclimatization, poor physical fitness, obesity, dehydration, acute illness, and external load, including clothing, equipment, and protective gear.[2] Certain medications and dietary supplements may also predispose to EHS.[1,2] Despite efforts to improve awareness among those well positioned to recognize and prevent EHS, such as athletes, coaches, and clinicians, deaths related to EHS have increased in recent years.[3] In 2018, EHS was the leading cause of preventable death in high school student athletes.[4] Reduction of patient temperature and continued maintenance of normothermia are crucial to EHS patient survival.[5]

Typical first approaches to reduce body temperature include providing a patient with ice packs, fans, water blankets,[6] and advanced surface cooling hydrogel pads[7,8] and administration of cold intravenous fluids via intravascular catheters.[9] When these measures fail, more invasive temperature management methods may be deployed, such as implementing body cavity lavage via the stomach or bladder or, in extreme cases, implementing thoracic or peritoneal lavage, which requires advanced surgical training.

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M.N. was an employee of Attune Medical, Chicago, IL at the time of the original drafting of this article. For the remaining authors, no conflicts of interest or sources of funding were declared.

DOI: 10.1097/JNN.0000000000000488

Recent efforts to bring to market pharmaceutical EHS treatments that are effective in the treatment of other exertional heat illnesses such as malignant hyperthermia have been unsuccessful.[10,11] Currently, dantrolene is the only drug that specifically treats patients with malignant hyperthermia but has shown promise to effectively treat EHS. Although malignant hyperthermia and EHS are different exertional heat illnesses, the common adverse effect between them is the breakdown of metabolism and sarcoplasmic function. When this occurs, increased levels of calcium in the cells cause the muscles to contract and hypermetabolize. Dantrolene works to stop this process by inhibiting ryanodine receptors on the surface of the sarcoendoplasmic reticulum to release calcium into the cells.[12] However, there are several setbacks to using dantrolene. Dantrolene is costly to prepare, is requiring at least 36 vials of 20 mg prepared within 10 to 15 minutes of presentation, and has a short half-life, and peak plasma concentration of the drug lasts only 5 hours.[12,13] When given to patients with EHS, fever reducing medications such as acetaminophen and anti-inflammatory ibuprofen can actually cause further complications such as disseminated intravascular coagulation, acute kidney injury, and hepatic failure.[1,2,14] Laxatives, antihistamines, benzodiazepines, diuretics, and β-blockers have been known to predispose patients to EHS.[12] With β-blockers, the vasodilation mechanism of the drug causes the body to remain hyperthermic. Because of the difficulties in finding effective pharmaceutical treatments, mechanical and physical means of temperature reduction remain necessary in the in the treatment of EHS.[5]

Esophageal temperature management (ETM) has emerged as a new temperature management modality. An esophageal heat transfer device replaces the standard orogastric tube, routinely placed after endotracheal intubation, and adds a temperature modulation capability.[11,15,16] A nurse verifies proper placement by using an x-ray to verify the esophageal tube is in the correct position. The device is similar to an esophageal tube that is normally used in standard-of-care settings with the additional cooling component built in via water flowing in a current in the outer part of the tubes (see Figure 1). Proper placement of the heat transfer device in the esophagus is verified by x-ray. The temperature of the esophageal tube is controlled by a heat exchange device, which can be operated by a nurse at bedside. When a patient's temperature rises unexpectedly, adjusting the temperature on the device deploys cooling to the inner core of the body within seconds. The esophagus' heat exchange environment allows heat to diffuse up through the esophagus and out of the body. Esophageal temperature management has successfully modulated temperature in a wide variety of critical care scenarios such as cardiac arrest, hypothermia, burns, and traumatic brain injuries.[11,15–21] This case report presents a novel application of ETM in the treatment of severe EHS over a 5-day course of treatment.

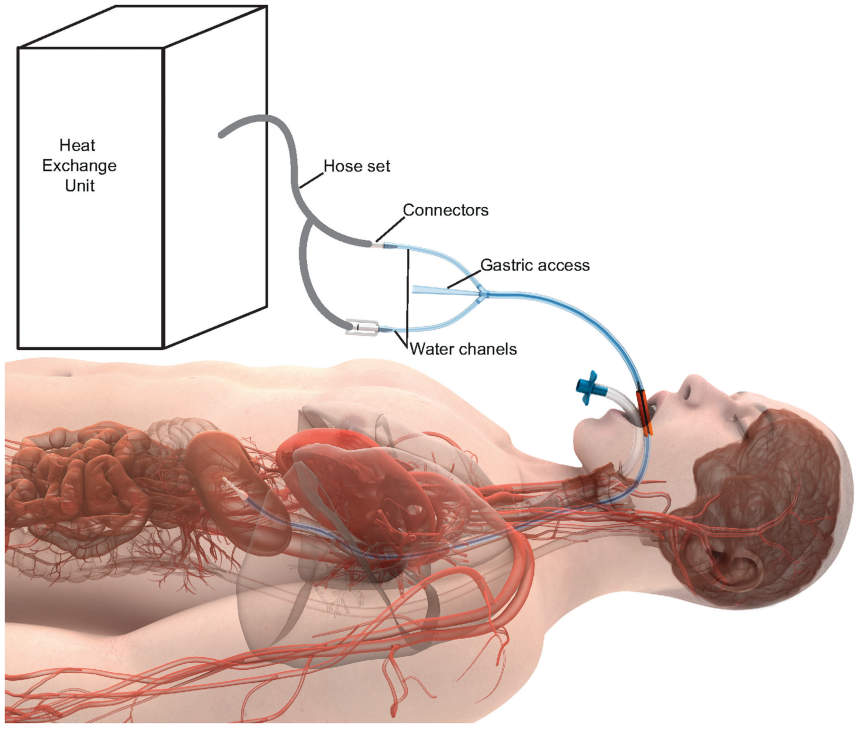
Case

A 28-year-old man with no previous medical history collapsed during an outdoor hot weather training mission as part of a firefighter curriculum. The patient had recently entered an elite program that included strenuous training exercises for 16 hours a day for 5 weeks. In the weeks since starting the program but before his collapse, the patient lost 20 lb. The night before his collapse, he had severe muscle cramps in all extremities.

On the day of his collapse, he was participating in a strenuous uphill hike in 26.7°C weather. The patient reported lightheadedness, sunk to his knees, and then lost consciousness. He was subsequently dragged to a medical evacuation helicopter landing site and transported via helicopter to the nearest hospital. His initial temperature reading upon evacuation was 41.7°C, and cooling measures were initiated in-flight with ice packs. On arrival at the first hospital, patient temperature was recorded at 40°C, and the patient was described as agitated and incoherent, with questionable seizurelike activity. As a result, the patient was sedated, and his airway was secured via endotracheal intubation. The patient was initially treated with water blankets and cold intravenous fluids. Laboratory tests were drawn, showing elevated levels of potassium at 5.4 mmol/L, creatinine of 2.23 mg/dL, glucose of 198 mg/dL, international normalized ratio of 1.94, and troponin I of 0.32 ng/mL.

The patient had a normal creatine kinase of 155 U/L, and evidence of normal liver function with an alkaline phosphatase of 79 U/L, aspartate aminotransferase of 38 U/L, and alanine aminotransferase of 49 U/L. Sodium levels were normal for the patient, ranging from 141 on admission to 135. A computed tomography scan of the head was performed without abnormalities identified, and a computed tomography of the chest showed mild bronchovascular opacities in the left lower lobe and subplural regions (left greater than right), likely aspiration pneumonitis versus atelectasis. A urine drug screen and blood alcohol levels were negative. The patient required high doses of propofol to attain adequate sedation. Uncertainty regarding details of the patient's preceding clinical condition and concern for possible infectious etiology prompted initiation of vancomycin, cefepime, metronidazole, and acyclovir. Concern for seizure activity and possible need for continuous electroencephalogram prompted initiation of levetiracetam and transfer to a tertiary care center.

FIGURE 1 Representation of the System, With Esophageal Temperature Management Device in Place



Upon arrival at the tertiary care center, a physical examination was performed while the patient received minimal propofol (sedation was not completely discontinued because of risk of self-extubation); the patient was noted to open his eyes to name, but not follow commands. He had 2-mm pupils that would react sluggishly to pinpoint, did not track or fixate on the examiner, withdrew to noxious stimuli in all 4 extremities, had weak corneal reflexes, and presented oculocephalic reflexes, and cough and gag reflexes. The patient was noted to have foul-smelling diarrhea.

It was noted that, despite the cooling measures already implemented, the patient's temperature remained at 38.1°C. Although other vital signs showed improvement (heart rate had decreased from 150 to 64 beats per minute, blood pressure was 97/55, and oxygen saturation of 100% on 100% FIO_2 via endotracheal tube), an ETM device (EnsoETM, Attune Medical) was placed by nursing staff to address the persisting hyperthermia. The external heat exchange unit (Blanketrol III, Cincinnati Subzero) was set to a cooling mode (Figure 1).

Within 30 minutes of ETM device placement, the patient's temperature reached 37.6°C. Per the tertiary care center's protocol, the target was normothermia at 37°C. Despite attainment of normothermia and no evidence of seizure activity on continuous electroencephalogram, the patient continued to exhibit signs of severe encephalopathy while developing thrombocytopenia, transaminitis, and rhabdomyolysis over the next 3 days. The patient's platelets decreased to 51 000 per U/μL, whereas aspartate aminotransferase and alanine aminotransferase increased to greater than 600 U/L, and creatine kinase increased to 1326 U/L. Results of a spinal tap performed by interventional radiology showed no evidence of meningitis. During this time, the patient continued to have episodes of temperature spikes as high as 39.1°C, which required active cooling. With the ETM device left in place, more aggressive cooling was reinitiated by nursing staff through adjustments to the external heat exchange unit settings. Chest x-rays showed improving pulmonary edema and consolidation/atelectasis in the patient who began to show signs of improving thrombocytopenia and coagulopathy, with decreasing international normalized ratio and uptrending fibrinogen. His renal function recovered with a creatinine of 0.7 mg/dL. By day 5, the patient's temperature remained stable, allowing removal of the ETM device, and his neurologic examination improved sufficiently to allow extubation. His creatine kinase levels and liver function tests subsequently returned to normal.

Upon discharge from the intensive care unit to the acute rehabilitation unit, the patient was noted to have some residual vocal cord dysfunction secondary to his prolonged endotracheal intubation, with voice improving daily. His neurologic examination revealed an awake and alert patient answering questions appropriately, with no facial asymmetry, 4/5 strength to bilateral upper extremities, and 3/5 strength to bilateral lower extremities.

After 22 days in the acute rehabilitation unit, the patient was discharged. At discharge, the patient's family reported that the patient was at neurologic baseline, and at 30-day follow-up, no residual deficits were found on examination.

Discussion

The constellation of signs and symptoms observed in patients with EHS like the one described in this case result from what is referred to as the "multiple hit" hypothesis, which proposes that a rapid onset of core body temperature elevation compromises molecular tissues' protective mechanisms.[10,22] Even with early medical intervention, EHS may have lasting effects, including damage to the nervous system and other vital organs.[23] Organ damage can include rhabdomyolysis, acute renal failure, acute respiratory distress syndrome, myocardial injury, hepatocellular injury, intestinal ischemia or infarction, pancreatic injury, and hemorrhagic complications, especially disseminated intravascular coagulation, with pronounced thrombocytopenia.[1,2] Central nervous system dysfunction can include disorientation, headache, irrational behavior, irritability, emotional instability, confusion, altered consciousness, coma, or seizure.[1,2] Clinical findings vary, but most patients with EHS are tachycardic and hypotensive and may have hyperventilation, dizziness, nausea, vomiting, diarrhea, weakness, profuse sweating, dehydration, dry mouth, thirst, muscle cramps, loss of muscle function, and ataxia. The patient described in this case exhibited most of these end-organ and neurologic insults.

The key to treating EHS is that the severity of illness may not be apparent upon initial presentation, and mortality is directly related to the duration of core temperature elevation, implying that rapid, aggressive cooling and continued maintenance of normothermia is crucial.[1,5,22] In this patient, cooling was initiated immediately with ice packs, followed by water blankets, but these measures did not sufficiently reduce his temperature. An ETM device was therefore deployed, which provided the necessary heat extraction to facilitate the attainment of normothermia. Within 30 minutes of ETM device placement by nursing staff, the patient's temperature reduced to 37.6°C. Because the ETM device was left in place, when the patient experienced recurrent episodes of temperature spikes, aggressive cooling was reinitiated by nursing staff through adjustments to the external heat exchange unit settings. The esophageal route of the device allowed for rapid heat extraction and helped staff avoid covering the patient with water blankets, freeing up patient access for ongoing care and monitoring. By avoiding surgical intervention, hospital staff limited vascular placement complications such as deep venous thrombosis, central line–associated bloodstream infection, and the implementation of peritoneal lavage. The device was only one part of the recovery of the patient, but it allowed nurses to react quickly to deteriorating vital signs in a minimally invasive manner, thereby limiting the need for immediate physician intervention.

Conclusions

The technique used in this case report is novel in that this was the first time the described temperature management system was used in a patient with EHS as an adjunct therapy to normal standard-of-care routine during the course of 5 days. The implications of such nursing-driven technology offer nursing staff new means to take control over sudden deteriorating vital signs in a minimally invasive manner without obstructing access to the patient, freeing the bedside nursing staff and physicians alike to continue to give rapid optimal care in high-morbidity conditions. Further testing of ETM when used not only as an adjuvant but also as initial treatment and sole treatment in pilot studies would further solidify the effectiveness of ETM in the treatment of EHS.

References

1. Bouchama A, Knochel JP. Heat stroke. *N Engl J Med*. 2002; 346(25):1978–1988.
2. O'Connor FG, Casa DJ. Exertional heat illness in adolescents and adults: epidemiology, thermoregulation, risk factors, and diagnosis. In: Post TW, ed. *UpToDate*. Waltham, MA: UpToDate; 2017.
3. Kerr ZY, Casa DJ, Marshall SW, Comstock RD. Epidemiology of exertional heat illness among U.S. high school athletes. *Am J Prev Med*. 2013;44(1):8–14.
4. Rodgers J, Slota P, Zamboni B. Exertional heat illness among secondary school athletes: statewide policy implications. *The Journal of school nursing*. 2018;34(2):156–164.
5. Heled Y, Rav-Acha M, Shani Y, Epstein Y, Moran DS. The "golden hour" for heatstroke treatment. *Mil Med*. 2004; 169(3):184–186.
6. Lee EJ, Lee SW, Park JS, Kim SJ, Hong YS. Successful treatment of severe heat stroke with selective therapeutic hypothermia using an automated surface cooling device. *Resuscitation*. 2013;84(6):e77–e78.
7. Lee B-C, Kim JY, Choi SH, Yoon YH. Use of an external-cooling device for the treatment of heat stroke. *Clinical and Experimental Emergency Medicine*. 2014;1(1):62–64.
8. Hong JY, Lai YC, Chang CY, Chang SC, Tang GJ. Successful treatment of severe heatstroke with therapeutic hypothermia by a noninvasive external cooling system. *Ann Emerg Med*. 2012;59(6):491–493.
9. Hamaya H, Hifumi T, Kawakita K, et al. Successful management of heat stroke associated with multiple-organ dysfunction by active intravascular cooling. *Am J Emerg Med*. 2015;33(1): 124.e125–124.e127.
10. Krau SD. Heat-related illness: a hot topic in critical care. *Crit Care Nurs Clin North Am*. 2013;25(2):251–262.
11. Naiman M, Shanley P, Garrett F, Kulstad E. Evaluation of advanced cooling therapy's esophageal cooling device for core temperature control. *Expert Rev Med Devices*. 2016; 13(5):423–433.

12. Muehlschlegel S, Sims JR. Dantrolene: mechanisms of neuroprotection and possible clinical applications in the neurointensive care unit. *Neurocrit Care*. 2009;10(1):103–115.
13. Kim HJ, Koh WU, Choi JM, Ro YJ, Yang HS. Malignant hyperthermia and dantrolene sodium. *Korean J Anesthesiol*. 2019;72(1):78–79.
14. Glazer JL. Management of heatstroke and heat exhaustion. *Am Fam Physician*. 2005;71(11):2133–2140.
15. Hegazy AF, Lapierre DM, Butler R, Martin J, Althenayan E. The esophageal cooling device: a new temperature control tool in the intensivist's arsenal. *Heart Lung*. 2017;46(3):143–148.
16. Markota A, Fluher J, Kit B, Balažič P, Sinkovič A. The introduction of an esophageal heat transfer device into a therapeutic hypothermia protocol: a prospective evaluation. *Am J Emerg Med*. 2016;34(4):741–745.
17. Williams D, Leslie G, Kyriazis D, O'Donovan B, Bowes J, Dingley J. Use of an esophageal heat exchanger to maintain core temperature during burn excisions and to attenuate pyrexia on the burns intensive care unit. *Case Reports in Anesthesiology*. 2016;2016:6.
18. Markota A, Košir AS, Balažič P, Živko I, Sinkovič A. A novel esophageal heat transfer device for temperature management in an adult patient with severe meningitis. *Journal of Emergency Medicine*. 2017;52(1):e27–e28.
19. Markota A, Kit B, Fluher J, Sinkovic A. Use of an oesophageal heat transfer device in therapeutic hypothermia. *Resuscitation*. 2015;89:e1–e2.
20. Khan I, Haymore J, Melinosky C, et al. Novel esophageal cooling device for therapeutic normothermia. Paper presented at: 14th Annual Neurocritical Care Society Meeting; 2016; National Harbor, MD.
21. Hegazy AF, Lapierre DM, Butler R, Althenayan E. Temperature control in critically ill patients with a novel esophageal cooling device: a case series. *BMC Anesthesiol*. 2015;15:152.
22. Nichols AW. Heat-related illness in sports and exercise. *Curr Rev Musculoskelet Med*. 2014;7(4):355–365.
23. Grogan H, Hopkins PM. Heat stroke: implications for critical care and anaesthesia. *Br J Anaesth*. 2002;88(5): 700–707.