Exertional Heat Stroke: Clinical Significance and Practice Indications for Special Operations Medics and Providers

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ABSTRACT

Exertional heat stroke is an acute injury associated with high morbidity and mortality, and is commonly encountered within military and Special Operations environments. With appropriate planning, rapid diagnosis, and aggressive treatment significant mortality reduction can be obtained. Planning for both training and real world operations can decrease the patient's morbidity and mortality and increase the chances of successful handling of a patient with exertional heat stroke. The mainstay of treatment is rapid reduction of the core body temperature. This is paramount both at the field level of care as well as in a clinical setting. Diligent surveillance for commonly encountered complications includes anticipating electrolyte abnormalities, rhabdomyolysis, acute renal failure, and hepatic injuries. Treatment with dantrolene may be indicated in patients with continued hyperthermia despite aggressive traditional treatment.

Case Presentation

A 26 year-old Special Forces (SF) student collapsed at the eleventh mile of a ruck march. The outdoor temperature was 23 °C/74 °F with 53% relative humidity. Upon initial assessment, he was conscious, but had altered mental status (AMS) and could not obey commands. No body temperature was obtained in the field, and due to his AMS, the patient was transported to the Womack Army Medical Center Emergency Department (ED) without further diagnostics or treatment prior to transport. Upon arrival at the ED, approximately 45 minutes after the patient's initial collapse, the patient was still unable to follow commands. He was severely diaphoretic and had an elevated core temperature of 40.5 °C/104.6 °F rectal. Rapid and aggressive temperature reduction was implemented by a combination of conductive and evaporative heat loss through the use of a fan in addition to ice packs to the groin and axilla.

Clinically, based on history and the patient's hypotension and tachycardia, the patient was resuscitated with normal saline (NS) solution. Because of the concern of hypotensive-related renal failure, which could lead to hyperkalemia, lactated ringers (LR) solution was avoided due to its potassium content. Laboratory studies indicated the patient had significantly elevated aspartate aminotransferase/alanine aminotransferase (AST/ALT) indicating acute liver injury, elevated creatinine consistent with acute renal failure, creatine kinase (CK) elevation diagnostic of rhabdomyolysis, and elevated troponin likely indicative of myocardial muscle injury. Given this constellation of findings, the patient was diagnosed with heat stroke and multi-system organ failure, and was admitted to the intensive care unit (ICU). With continued temperature reduction and fluid resuscitation, his symptoms gradually resolved and after an eight-day hospital stay he was discharged in good condition. After several months, his laboratory abnormalities resolved, he gradually returned to exercise, and he was placed on a one-year heat injury profile.

Introduction

Heat Injury is a spectrum of disease that starts with heat cramps and heat exhaustion characterized by an elevated temperature and decreased performance, with many non-specific symptoms such as fatigue, nausea and vomiting, cramps, and dizziness. At the other end of the spectrum is Heat Stroke (HS), a medical emergency, which by definition, consists of an elevated body temperature, usually greater than 40 °C/104 °F, with altered mental status (AMS).[1] One does not have to have go through stages of heat injuries; patients can develop heat stroke without prior signs of heat exhaustion. Two forms of heat stroke are described in this article.

Classical heat stroke (CHS) is due to an increase in ambient heat with impaired thermoregulation and is seen typically in patients at both extremes of age. Infants and young children have immature physiology, increased body surface area, and rely on adults to regulate their environment to include adequately hydrating themselves. The elderly have poor compensatory mechanisms to efficiently mitigate heat stress; their declining thermoregulatory and sensory mechanisms produce poor responses to high temperatures.

Exertional heat stroke (EHS) is caused by increased metabolic production of heat due to physical exertion and is found typically in young and early middle-aged healthy adults. Exertional heat stroke is therefore commonly encountered within military settings. In 2010, the United States (U.S.) military saw 2,576 patients with heat-related injuries; of these, 311 were heat stroke.[2] Initial studies on heat injuries showed a case fatality rate of 50%;[3] however, many of these studies did not differentiate between EHS and CHS. Typically, patients susceptible to CHS are older and have more medical co-morbidities, resulting in a higher mortality rate. Newer studies focusing specifically on EHS have shown case fatality rates of 5–6%.[4,5]

Hypothetically, some predisposing factors for EHS include underlying dehydration, exercise in hot and humid conditions, thermogenic supplements that uncouple oxidative phosphorylation, elevated body mass index (BMI), poor baseline physical fitness levels, lack of acclimatization to climate, sleep deprivation, and prior heat injury.[6] Additionally, Special Operation Forces (SOF) spend long durations of time in body armor and helmets that do not breathe or allow the core to radiate heat properly. Furthermore military uniforms for combat are meant to protect the Soldier against the elements and other potential threats such as fire and do not dissipate heat as effectively as athletic training apparel such as Coolmax®. It is not completely understood why specific individuals are affected by heat injury and others are not, despite the overall similarities in Soldiers' environments. Because of this, a genetic component is likely, but yet to be proven.

Pathophysiology

Thermoregulation

In order to understand heat injury and specifically heat stroke, one must first understand thermoregulation. Body heat is gained both from the environment and from cellular metabolism. Many cellular enzymes function at optimum temperatures and any significant deviation in temperature not associated with a fever can result in decreased cellular performance and ultimately cellular death. Because of this, the body tightly regulates the temperature to keep the core temperature at approximately 37 °C/98.6 °F. When the core temperature rises, the hypothalamus—the primary regulator of body temperature—causes peripheral vasodilatation and shunts blood away from the visceral organs to the skin, thus resulting in increased heat dissipation. This process of thermoregulation specifically involves the kidneys and the intestines. Therefore, increased body temperature can predispose patients to acute renal failure and intestinal ischemia.[7]

Increased body temperature also induces sweating and if the surrounding air is not significantly humid, evaporation of sweat will significantly increase heat dissipation. This thermal gradient is critical to the transfer of heat. Heat reduction through evaporation of sweat is severely impaired in hot humid conditions. This is why increased numbers of heat injuries occur in humid rather than dry climates. Losses of salt and water through sweating also can be significant and need to be replaced in order to avoid dehydration and electrolyte deficiencies, both of which can further impair thermoregulation.

Acute-Phase Response and Heat-Shock Proteins

Increased heat load and strenuous exercise cause an acute-phase response, which results in the release of several cytokines, helps to protect against tissue injury, and promotes cellular repair.[8] A similar acute-phase reaction is seen in sepsis, which might explain why a prolonged systemic inflammatory response syndrome (SIRS) is commonly seen in patients with EHS.[9] Temperature elevation also causes cells to produce heat-shock proteins that help protect cells from damage caused by elevated temperature, inflammatory cytokines, ischemia, and hypoxia.[10] These heat-shock proteins cause a transient state of tolerance to the elevated temperature, which temporarily increases the survival of the cell. Decreased levels of heat-shock protein transcription have been observed in poorly acclimatized individuals.[10] This could potentially explain why EHS is seen more commonly in poorly acclimatized people. For example, Soldiers from bases in colder climates, such as Alaska, are initially at an increased risk of EHS when they report to bases with hot, humid summers such as Fort Bragg, NC. In addition, certain genetic polymorphisms are associated with decreased heat-shock protein production,[11] which could explain why certain individuals develop EHS and others do not, given the same ambient and core temperatures.

Inflammatory Cytokines

Reduced visceral perfusion seen during the body's response to increased heat load can result in the translocation of intestinal bacteria and the subsequent release of endotoxins, which leads to the release of inflammatory cytokines. These inflammatory cytokines can interfere with normal thermoregulation and promote the progression from heat exhaustion to heat stroke.[1] Inflammatory cytokines also help generate a SIRS-like response as seen in heat injuries.

Prevention

Given that EHS in many instances is self-induced, significant effort should be made to mitigate and prevent the injury from happening in the first place. This can be accomplished by risk factor reduction and by proper planning.

Risk factors should be identified and reduced. Soldiers should insure that they are not taking thermogenic

supplements or excessive amounts of energy drinks. A high level of physical fitness should be maintained, and individuals should get adequate amounts of sleep. Soldiers should undertake a proper acclimatization program after arrival in hot/humid locations. A reasonable acclimatization program would include daily exercise in the heat, gradually increasing intensity and duration over the course of two to four weeks.[6] Heat retaining clothing should be reduced if possible, and an attempt should be made to perform physical fitness early or late in the day when the heat index is at the lowest point. Furthermore, increased rest periods should be incorporated during high intensity exercise. Unfortunately, during combat operations the risk mitigation strategies discussed above often cannot be employed give operational necessity resulting in a higher likelihood of heat injuries occurring.

Proper planning at the team level by the medic is also critical to both prevention of EHS and if prevention is not possible, the effective treatment of EHS. Medics need to be able to recognize training that could potentially create a heat casualty. Medics should also be involved in planning in order to mitigate any risk factors that might be present. Furthermore, medics need to educate leadership on the need for training plans to include potential scenarios of patients with heat injury. These scenarios should include initial treatment and a "battle drill" plan. Although logistics in the field vary depending on the environment, medics need to ensure they have adequate logistics available to effectively treat heat-related injuries during high-risk training.

Clinical Presentation

Patients with heat injury can present initially with nonspecific symptoms such as muscle cramping as well as nausea and vomiting; however, sometimes collapse is the first sign. Diaphoresis often varies. As the patient's temperature and the extent of the injury increases, altered mental status, seizures, and cardiovascular collapse can occur. It is not unusual for trained athletes to sustain prolonged periods of hyperthermia without noticeable impairment. Conditioned athletes can tolerate core temperatures up to 41.9 °C/107.4 °F during exercise and show no clinical signs of heat-related injury most likely due to their level of acclimatization and conditioning.[12] The exact mechanism that causes a patient to go from heat exertion to heat stroke is not fully understood. In many instances, central nervous system (CNS) dysfunction can be subtle, presenting as inappropriate behavior or impaired judgment. Although by definition altered mental status will be present in EHS, loss of conciousness (LOC) cannot be used as a reliable indicator for heat stroke. Patients have been documented conversant with core temperatures above 106. These patients often "decompensate" quickly and lose consciousness. When patients present with signs of heat injury, a rectal temperature should be obtained for an accurate determination of core body temperature. Unfortunately, in the field there is no other reliable indictor except for core temperature. The medical provider must know the possibility of heat injury exists and based on the clinical situation be ready to treat empirically if no thermometer is available. Although traditional instruction teaches cessation of sweating is a cardinal sign of heat stroke, this is a late sign and the presence of sweating should not be used to rule out heat stroke. Unless an alternate cause is obvious, a previously healthy individual that develops AMS during or after physical exertion or collapses should be considered to have heat injury until proven otherwise.

Treatment

Prehospital

Upon an initial assessment, the medic should get a good set of vitals including a rectal temperature. Activation of the planned or improved evacuation plan should be done as soon as a patient with EHS is identified. It is likely that evacuation will not be immediate, so early activation will limit delays in the evacuation process. Field care during training should include proper exposure of the patient and monitoring of the patient's vital signs. In addition, their airway, breathing, and circulation status should be evaluated.

Initial treatment focuses on the rapid reduction in core temperature. Studies have shown a trend of decreasing fatality rates in patients with more rapid cooling rates.[13,14,15] Although these studies are limited in the numbers of patients studied, and include CHS patients, common sense dictates that the longer a patient is hyperthermic, the greater the resulting damage. Studies show that the sooner the patient can begin being cooled the better. For field treatment this must be by any means possible. One should think that the first thirty minutes after a recognized onset is critical to your patient's outcome. For this reason immediate treatment must be initiated in the field and provide a means of continued cooling and monitoring while being evacuated. Therefore, rapid reduction in temperature is essential, and some providers' advocate cooling HS patients first and transporting second.[16]

In order to facilitate temperature reduction the patient should be placed in a shaded area, clothing should be loosened, and water should be drizzled or poured over the patient. One should avoid dumping a Gatorade jug of ice water on a patient as most of the water will end up in the dirt. If limited treatment equipment is available, the use of a poncho or other similar piece of kit will help keep water and ice in contact with the skin surface of the patient as long as possible. Having dedicated equipment

to treat heat injuries is preferred and the authors recommend a modified body bag with top flap cut off. Patients should be placed on their back in bag, and ice and water should be poured into body bag over patient. It is important to maintain control of head and airway and not to fill the bag up past the back of the patient's posterior ear lobe; this positioning prevents airway issues and allows vitals signs to be monitored. One needs to be prepared for the patient to vomit and if this does occur the patient should be repositioned by rolling the patient on their side to maintain their airway. If needed, water can be rebathed over patient. The bag and patient can be placed on a litter to facilitate cooling and allows simultaneous transportation. The bag can be prepared to include a rectal thermometer and the entire package can be placed in a medical coverage bag or with pre-staged kit. Further planning and logistics will significantly improve patient care and do not require significant resources. The authors recommend two five-gallon water cans filled with ice and water strategically staged with a modified body bag. When using ice water immersion, the patient's temperature should be reassessed every 10–15 minutes. If ice water is not available and a less effective cooling process is being used for the patient should be reassessed every 25–45 minutes. It is important to prevent iatrogenic hypothermia from prolonged cooling.

Combat field care should include as many as the above-mentioned techniques as possible, tactical situation permitting. While this is not a care-under-fire scenario, it is important to realize that patients with EHS are critically injured. Untreated EHS has a mortality rate as great as many of the injuries seen on the battlefield. As such, EHS patients should be MEDEVAC and be categorized as urgent.

Cooling

Studies on hyperpyrexic (fevers above 41.5 °C/106.7 °F) patients without evidence of EHS have shown that ice water immersion provides the fastest reduction in core temperature with a cooling rate of 0.35 °C/min.[17] This is due to water's unparalleled volume-specific heat capacity, which is nearly 3500 times greater than air. This method is recommended by the American College of Sports Medicine[12] and is used by Navy medical providers working at Parris Island Recruit Depot. Over a twelve-year span, military medical providers at Parris Island treated 252 cases of HS with ice water immersion with no fatalities.[18] However, ice water immersion in deep tubs complicates concurrent patient care by limiting providers' access to the patient for performing clinical interventions, electric monitoring and procedures. Furthermore, because exposure to ice water results in peripheral vasoconstriction and impaired heat loss, there is the concern of impaired heat dissipation with ice water immersion. Based on this argument, some believe that evaporative heat loss is best accomplished by applying a large fan and cool water mist as the most effective way to treat patients with EHS. Unfortunately there are no randomized controlled trials looking at the best method of cooling in patients with EHS. The majority of data available on cooling rates comes from studies of hyperthermic individuals that show no signs of EHS. Given that patients with EHS have impaired thermoregulation, extrapolating the data on hyperthermic individuals to patients with EHS is problematic. Because of the lack of research on the topic, one cannot definitively conclude the best method of cooling. However, given ice water immersion's documented cooling rates, and its reported low mortality rate associated with its clinic use,[18] it does seem prudent to use as the primary means of cooling especially in the prehospital environment where its use does not complicate cardiac monitoring and invasive procedures such as central lines. This recommendation is not based on large amounts of research and because of this the common clinical practice of wrapping a patient in wet sheets, applying a large fan to the patient, and placing ice packs in locations where major blood vessels are close to the skin such as the groin and axilla is not unreasonable.

Figure 1 *Shows how small the preparation of equipment could be.*

Figure 2 *Patient on a litter in the "Johnston/Donham improvised cooling bag".*

Once the patient's core temperature drops below 39 °C/102.2 °F, active cooling should stop. If shivering develops during treatment, benzodiazepines should be given to prevent further heat production from muscle contraction induced during shivering. Aspirin and acetaminophen should be avoided as the elevation of temperature in EHS is not due to an increase in set point of the hypothalamus as seen in infectious fevers and thus will not be effective in temperature reduction. Also, these medications have a deleterious effect on the patient's hepatic, renal, and hematologic system. In extreme circumstances where a significant elevation in core temperature continues when the recommended treatments are not effective, more invasive treatment, including peritoneal lavage or cardiopulmonary bypass, is sometimes indicated.

Volume Resuscitation

Patients with EHS typically need vigorous intravenous fluid resuscitation to restore intravascular volume loss and to treat renal injuries that result from rhabdomyolysis and direct thermal injury. Although EHS can be present with normovolemia, especially in conditioned athletes, most patients are dehydrated in combat settings. In one of the largest case series of EHS, Sithinamsuwan et al. found that 28.6% of Soldiers with EHS presented in hypovolemic shock.[15] Servicemembers fight while wearing heavy body armor in environmentally hot areas, and are often unable to drink sufficient fluids to maintain hydration. These same servicemembers often drink volume-depleting fluids that induce diuresis, such as energy drinks that contain significant amounts of caffeine and sodium. In these patients, because of the concern for renal injury, normal saline solution is the resuscitative fluid of choice. Lactated ringers solution (LR) with potassium concentrations should be avoided due to the concern for hyperkalemia. Traditional variables such as vital signs, physical examination (specifically mental status), and urine output should be the guide for volume resuscitation.

Electrolytes

Patients with EHS should have targeted electrolyte monitoring, and any deficiencies should be addressed. Recently, increased awareness and education about dehydration and its deleterious effects on human performance have addressed over-hydration and subsequent hyponatremia. Potassium levels should be monitored, as EHS patients can have increased plasma levels of potassium due to muscle cellular breakdown and release of intracellular potassium. Given that these patients are also at risk for acute renal failure, hyperkalemia, if present, should be treated aggressively with calcium to stabilize the cardiac membrane, with insulin/albuterol/bicarbonate to cause an intracellular shift of potassium, and with kayexalate or dialysis to ultimately remove excess potassium.

Medications

Dantrolene is a drug that reduces skeletal muscle contractions, and thus heat production, by inhibiting calcium release from the sarcoplasmic reticulum. It is commonly used to treat neuroleptic malignant syndrome and malignant hyperthermia. Given that neuroleptic malignant syndrome and malignant hyperthermia share common symptoms of muscular rigidity/contraction and subsequent hyperthermia, some have speculated that treatment with dantrolene might effectively treat EHS.[19] However, randomized controlled trials show no reduction in morbidity or mortality with dantrolene treatment in EHS.[20] Some literature speculates that certain subsets of EHS patients have genetically de-regulated calcium metabolism similar to malignant hyperthermia, and that these patients could potentially benefit from dantrolene therapy.[19] Because of this, Hadad et al. recommend that patients with EHS who do not respond to conventional therapy should be given dantrolene empirically.[19] Given dantrolene's lack of significant side effects, this seems to be a reasonable treatment decision.

Other medications commonly carried by SOF medical providers such as steroidal and non-steroidal anti-inflammatory medications have not been shown to be beneficial and could potentially be harmful because of their deleterious effect on the patient's hepatic, renal, and hematologic system. If shivering develops during cooling, benzodiazepines are a useful adjunct, but should not be routinely used because they could potentially make any AMS present worse.

Complications

Patients with EHS are prone to developing multiple complications. Depending on the degree of temperature elevation and the duration of exposure, multiple organ systems can be severely damaged. Studies have shown that the critical component is not the peak temperature, but instead the overall duration of temperature elevation sustained by the patient.[5] Exertional heat stroke causes the release of pro-inflammatory mediators and patients can develop a SIRS-like response similar to what is seen in sepsis. Zeller et al. found that 50% of patients with significant EHS had prolonged SIRS even after their initial temperature elevations were corrected.[9] Cardiovascular collapse, myocardial injury, lactic acidosis, elevation in liver enzymes, and rarely hepatic failure can all occur. Rhabdomyolysis is seen in 25–100% of patients with EHS[9,14,21] and renal failure is seen in 13–87.5% of patients.[9,14,21] As a result, EHS patients need to have their creatine phosphokinase and renal function monitored frequently if any elevations from baseline occur. Disseminated intravascular coagulation (DIC) is seen in 32–45%[14,15] of heat injury patients likely due to microvascular thrombosis.[1]

Prognosis

The prognosis for EHS is variable depending on the extent of the injury. With prompt recognition and treatment these patients can have very good outcomes despite the critical nature of the injury. However, even in the best cases, the recovery period can be extensive taking several months to even a year before individuals are able to return to unrestricted duty and able to exercise at maximal intensity in hot humid climates.

Conclusion

Exertional heat stroke (EHS) is commonly encountered in military operations and has a high rate of morbidity and mortality when unnoticed or when left untreated. The cornerstone of management is prompt diagnosis with rapid lowering of core body temperature by any means available. With appropriate treatment, morbidity and mortality can be reduced significantly.

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