Exertional Heatstroke: A Case Report and Review of Articles

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Exertional heatstroke (EHS) is a life-threatening condition to all who engage in vigorous physical exertion. It will result in fatal multi-organ failure. The prognosis of heatstroke is directly related to the degree of hyperthermia and the duration. Thus, early diagnosis and rapid cooling are crucial for the patient’s survival. We present a 19-year-old man who had sudden onset of hyperthermia, seizure and renal function impairment after excessive exercise, and exertional heatstroke was diagnosed in the emergency department. After prompt diagnosis and treatment, the patient was discharged with stable condition. Relevant literature was also reviewed and discussed.

Key words: exercise, exertional heatstroke, multi-organ failure, rapid cooling

Introduction

Exertional heatstroke (EHS) is a state of extreme hyperthermia that occurs when excess metabolic heat, generated by muscular exercise, exceeds the body’s heat dissipation rate\(^1\). It typically affects young athletes or military personnel, who are pushed to their physical limits and suffer a clinical and pathological syndrome\(^2\). Morbidity and mortality are directly related to the duration and intensity of elevated core temperature. Thus, early diagnosis and proper treatment are crucial for the patient’s survival\(^3\). We present a 19-year-old man who had been running a long distance and came to us with a high fever and seizure. Exertional heatstroke with rhabdomyolysis, renal and liver function impairment was diagnosed immediately in the emergency department. After applying aggressive cooling methods and other supportive care, he recovered and was discharged without significant sequela.

Case Report

A 19-year old generally healthy man of average body weight presented to our emergency department with altered mental status, hyperthermia, tachycardia, and diaphoresis after a school sporting event in humid, summer weather. He had just finished running 8.3-km and was on the way back to his classroom when he felt shortness of breath and palpitation, and then lost consciousness and began generalized convulsion. Therefore, he was sent to our emergency department (ED) immediately.

After arriving to our ED, the convulsion had ceased but the conscious status was drowsy, GCS (E3V5M6). His body heat, measured using the tympanic temperature, was 41.1°C. His initial vital signs were: 170 heart beats per minute, 19 respiratory rates per minute, and the blood pressure was measured at a level of 147/39mmHg. We
performed the following emergent management: oxygen supplying via nasal cannula 3L per minute, intravenous isotonic sodium chloride fluid challenge, and body heat cooling measures: including ice pillow, fans, and ice water-soaked towels. Physical monitoring was applied right away. His initial arterial blood pH was 7.3 with a base deficit of 15.2mmol/L. Serum GOT was 58IU/L, BUN was 19mg/dL, creatinine was 2.3mg/dL, glucose was 122mg/dL, K was 4.5meq/L, Na was 145meq/L, Cl was 106meq/L, CK was 178IU/L, and CKMB was 5.1IU/L. Multiple abrasions over his four limbs were noted, so a 0.5mL Tetanus toxoid was given and wound treatment was done. After several minutes of treatment, the patient’s consciousness became clear and no neurological focal sign was found. The chest X-ray showed no significant findings, the EKG showed sinus tachycardia and the brain CT without enhancement showed no significant abnormal findings. After 2 liters of isotonic sodium chloride hydration, there was still no urine output. Neither did inserting a Foley catheter produce any urine. One hour later after beginning cooling measures, the tympanic temperature dropped to 38.5°C; Blood gas pH returned to normal (pH was 7.378; base deficit was 8.1mmol/L). After all the emergent resuscitating management in our ED, this patient was arranged admitted to our hospital under the diagnosis of heat stroke, exertional rhabdomyolysis and impairment of renal function.

In the beginning, to treat rhabdomyolysis and the impaired renal function (Creatinine 2.3mg/dL), sodium bicarbonate and mannitol were used. Creatinine returned to 0.8mg/dL on the fourth day of hospitalization. Urine output returned to normal range on the second day of admission. On the second day of hospitalization, tympanic temperature declined to 37.4°C. On the third day, the CK elevated to 6228IU/L, but declined to 2418IU/L on the sixth day. Renal echo showed mild swelling of the renal parenchyma, which was compatible with renal parenchymal disease.

In addition, the patient’s liver function was noticed to be impaired after admission. On the third day of hospitalization, his serum GOT elevated to 818IU/L and GPT to 738IU/L. After reviewing his medical history, he claimed being physically healthy and denying having any associated liver diseases. He denied suffering any serious abdominal illness as well. Therefore, a series of serum laboratory tests were arranged. The results revealed the following: total bilirubin 1.0mg/dL, direct bilirubin 0.3mg/dL, alkaline phosphatase 54IU/L, negative of HBsAg and HCV-Ab, and lipase 17U/L; No significant abnormalities were found from the abdominal sonography, which was excluded other primary liver and biliary diseases. Heatstroke induced liver damage was suggested and this patient was prescribed Silymarin to protect the injured liver. The level of GOT declined to 115IU/L and GPT to 331IU/L on the sixth day of hospitalization.

After all the treatments, the patient’s general condition, including vital signs, appetite, and urination, were good. Neither physical nor mental discomforts were complained. He was discharged after 7 days of hospitalization. He returned to normal daily life without any significant complications, and continued regular outpatient visits. On the latest visit, he was well.

**Discussion**

Heat exhaustion and heatstroke are part of a continuum of heat-related illnesses. They occur when the body’s thermoregulatory responses are inadequate to preserve homeostasis. Heat exhaustion is usually associated with nonspecific signs and symptoms and mild pyrexia\(^4,5\). Patients may experience headaches, nausea and malaise, and show signs of circulatory collapse. Untreated heat
exhaustion can progress to heatstroke. Heatstroke is the presence of a core temperature more than 40°C accompanied by altered mental status manifesting as delirium, coma, or seizure. Finally, it may result in multi-organ failure and death.

Heat stroke can be divided further into classic and exertional forms. Classic heatstroke occurs when an individual develops heatstroke while exposed to a hot ambient temperature. Anhydrosis, pyrexia, and altered level of consciousness are typical of classic heat stroke. Exertional heatstroke (EHS) occurs when excess heat generated by muscular exercise exceeds the body’s ability to dissipate at the same rate.

In our case, this 19-year-old man ran 8.3-km in humid summer heat and then returned to his classroom. He then lost consciousness and suffered convulsions. We assumed that he produced excessive amounts of heat and sweat during exercise, and did not have time to drink water or supply more fluids, all of which increased the risk of dehydration. Consequently, dehydration led to abnormal heat dissipation and increased heat production continued during exertion, resulted in dysfunction on the central regulation of temperature and EHS developed.

According to his history and physical examination, heat stroke was our preliminary diagnosis. Therefore, intravenous fluid resuscitation, emergent cooling measures and close monitoring were administered based on clinical judgment and resuscitation guidelines. The patient’s condition became stable gradually. The treatment of exertional heatstroke should begin with an assessment of the airway, breathing, circulation, and basic life support, followed by reduction of core temperature as quickly as possible.

After examining the patient’s laboratory data, rhabdomyolysis (CK 6228IU/L), metabolic acidosis (arterial blood gas pH 7.3 with base deficit of 15.2mmol/L), renal function impairment (creatinine 2.3mg/dL) and liver function impairment (GOT 818IU/L and GPT 738IU/L) were defined. If we had not identified heatstroke and started cooling management immediately, the condition could have been life-threatening. As seen in previous reports, when core temperature is elevated long enough, multi-organ dysfunction syndrome (MODS) with disseminated intravascular coagulopathy (DIC), renal failure, liver failure, myocardial injury and adult respiratory distress syndrome (ARDS) may occur. The prognosis of heatstroke is directly related to the degree of hyperthermia and the duration. Thus, the most important feature in the treatment of heatstroke is rapidly cooling the body.

Cooling methods can be categorized as external and internal. External methods include evaporative and immersion cooling. The majority of data suggest evaporative cooling as the method of choice. Splashing copious amounts of water (1-16°C) over the patient with air fanning is strongly suggested. Treatment should be matched to the patient’s age and clinical background. However, recently Smith and Casa et al proposed that ice water immersion was the most effective method of whole body cooling and it should be the gold standard treatment of EHS. Immersion cooling can be achieved using an ice bath, or using cooling blankets in conjunction with ice packs placed on the axilla, groin, neck, and head. Therefore, in the future more and wider randomized controlled studies should be designed to compare the various cooling techniques to find the cooling method of choice. Cooling usually is discontinued once the core temperature has reached 38°C (100.4°F) to avoid hypothermic overshot.

Internal cooling methods are also effective in rapidly decreasing the body temperature. Gastric, bladder, and rectal cold-water lavage can be accomplished with minimal invasion. Peritoneal lavage is difficult to perform, in addition, compared
with evaporative cooling, peritoneal lavage was not found to be advantageous\(^{(14,15)}\). Peritoneal lavage should be performed only in extreme cases. The role of invasive cooling methods has not been fully established\(^{(2)}\).

The acid-base response to heatstroke is predominantly metabolic acidosis\(^{(16)}\). In our patient, metabolic acidosis was corrected after initial treatment. Hart et al observed a significant correlation between lactic acid level and neurological morbidity and mortality in a series of 28 heatstroke patients\(^{(17)}\). Metabolic acidosis occurs late and one might expect heatstroke patients with metabolic acidosis to present with a higher index of severity of illness and increased neurologic morbidity and mortality\(^{(18,19)}\).

Rhabdomyolysis caused by tissue destruction is common and results in myoglobinuria and risk of renal injury. In our patient, we gave him Mannitol, sodium bicarbonate and enough fluids to maintain urine pH above 6.5 and adequate urinary output. Mannitol produces alkaline urine, which will decrease the toxic effects of myoglobin on the renal tubules. It should be used to maintain urinary output of 50 to 100 cc per hour in order to protect the kidneys\(^{(5)}\). Niu et al proposed that mannitol has antioxidant properties and can also reduce intracranial pressure after ischemia-induced cerebral edema\(^{(20)}\).

Liver injury in most cases of EHS is usually asymptomatic and exhibits only mild reversible elevation in plasma aminotransferase levels, however, it may progress to acute hepatic failure in 5% of EHS patients\(^{(8)}\). In our case, we gave him silymarin to protect the injured liver using its antioxidant properties and the plasma aminotransferase level went down gradually. Due to dismal outcome of emergency liver transplantation in cases of heatstroke patients with hepatic failure, conservative management appears to be justified in heatstroke associated liver failure even in the presence of accepted criteria for emergency liver transplantation\(^{(21)}\).

Medications show little efficacy in treating heatstroke. Dantrolene has proved ineffective in decreasing core temperature and increasing survival rate of heatstroke\(^{(4)}\). Antipyretic agents are not recommended now\(^{(3)}\). Ikeda et al proposed that additional blood purification (BP) therapy may provide a better prognosis than conventional therapy only in heatstroke patients with DIC and multi-organ failure. It is useful in removing inflammatory cytokines\(^{(22)}\). In the rat study by Tsai et al, hyperbaric oxygen therapy (HBO) effectively reduced heatstroke-induced arterial hypotension, hypoxia, plasma tumor necrosis factor-\(\alpha\) overproduction, and cerebral ischemia and damage and improves survival. HBO is currently not the treatment of choice but may support conventional treatment options in very severe cases, although further studies are needed\(^{(23)}\).

Prevention of exertional heat injury and EHS is possible through the education of military personnel, athletes and their families, trainers, and coaches\(^{(7)}\). The education includes properly timed rest periods during activity, proper hydration, training schedules that avoid the hottest hours of the day, and physical training matched to each individual’s physical fitness. In addition, it is very important to identify the heat-susceptible subjects (physically unfit, ill, dehydrated, and unacclimatized individuals) and prevent them from performing exercise. Eventually, various education programs should increase awareness to EHS as well as the treatment to medical staff and military commanders\(^{(24)}\).

In conclusion, exertional heatstroke (EHS) is a life-threatening condition caused by an extreme elevated core body temperature. It may result in multi-organ failure and possibly death. The main predictor of outcome in EHS is the duration and degree of hyperthermia. Therefore, prompt recognition, as well as assessment of airway, breathing, circulation and basic life support, followed immediately with cooling management
through evaporation or full-body ice-water immersion are crucial for survival. Most EHS can be prevented. Greater awareness in communities of the early warning signs of exertional heat illness and concentration on decreasing EHS predisposing factors are very helpful for saving lives.

References


運動型中暑之病例報告與文獻回顧

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運動型中暑對所有參與激烈體能運動的人員是一種危及生命的急症，嚴重時會產生體內多重器官衰竭而致命。預後跟高體溫的程度與維持時間有直接相關，及早診斷與快速降溫對於病人的存活與否很重要。本文報告一19歲男性病例，在激烈運動後產生高體溫，癲癇與腎功能受損症狀，運動型中暑於急診被迅速診斷並且經過合宜治療，病患最後穩定出院。本篇文章整理相關文獻並提出討論。

關鍵詞：運動，運動型中暑，多重器官衰竭，快速降溫