

# Managing Exertional Heat Stroke Among the Combatants

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## ABSTRACT

Exertional Heat Stroke (EHS) is a medical emergency which when not addressed timely can lead to high mortality and morbidity. Armed forces are vulnerable to heat stroke because of their involvement in strenuous physical exercises in extremes of climatic conditions.

The present case series aimed to analyse the clinical features, complications and effect of prompt treatment on the final outcome of the patients. Soldiers reporting with heat related cases were screened to identify those suffering from EHS and various relevant information was collected. It was found that 33.3% of patients were not acclimatised to the environment before taking part in strenuous physical activities and had deranged liver and renal function. Most (77.8%) of the patients had run 5 km just before having EHS. All patients had delirium on presentation and 16.7% had seizures. Majority (77.8%) of the patients had normal blood pressure on presentations. All patients developed diarrhea whereas liver enzymes were raised in 11.1%, 33.3% had renal failure and 11.1% developed multiorgan failure. On aggressive management with rapid cooling, intravenous fluids and other supportive medications, all patients of EHS recovered completely without any mortality and were discharged from the hospital within one week of admission, without any residual neurological deficit except two patients who developed multiorgan failure requiring haemodialysis for six weeks. Despite taking innumerable precautionary measures, EHS cannot be entirely prevented but continuous monitoring and aggressive management can lead to a great reduction in morbidity and mortality of the patients.

**Keywords:** Delirium, Exercise, Hot temperature, Military personnel, Seizures

First described in 24 BC, in the Roman soldiers, heat stroke is proposed to occur because of derangement of normal physiologic processes of thermoregulation, which happens under overwhelming circumstances, contributed both by extreme environmental onslaught as well as poor acclimatisation of the affected individual [1-5]. Armed forces, world over indulge in intense training activities like Battle Physical Efficiency Test (BPET) and cross-country run in preparedness of war [6]. These intense physical exercises present massive heat load to the soldier's body, putting him at risk for EHS. Exertional Heat stroke is a lethal medical emergency which when not addressed within a proper duration results in Multiorgan Dysfunction syndromes and death [1,3,7] and mortality rate approaching nearly 10%-50% in last 50 years [1,2,8]. The objective of this case series is to impress upon the medical fraternity the different presentations, complications and the effect of aggressive treatment on the final outcome of the patients suffering from exertional heat stroke.

## CASE SERIES

### Materials and Methods

We describe the clinical features and treatment outcomes of 18 patients who were diagnosed of exertional heat stroke, in a semi-urban region of northern India in March 2013 to September 2014 in a medical facility centre that provides the primary and the secondary level of healthcare for all military personnel residing in the study area. Institutional Ethics Committee approval was taken along with informed written consent from the patients for the conduct of the study. All cases of heat related illness were screened to find out the cases of EHS which was diagnosed using the criteria of having high core body temperature >40°C (i.e., 104°F) (core body temperature was measured using a rectal electronic thermometer probe) with

neurological involvement (coma, stupor, delirium, and seizures) [1].

Relevant background information was also recorded, including patient age, unit, and relevant data for identification of the individual. Along with this pre-exertion acclimatisation, level of physical fitness, prior alcohol consumption, distance of run, any pre-existing or comorbid illness, which could influence the outcome of the illness, were also recorded. The entire data was gathered using personal interview, previous health records pertaining to the individual and interviews of the team members and respective commanders.

Laboratory evidence for hepatic involvement (S. Bilirubin, AST, ALT), renal involvement (blood urea, creatinine), hematologic involvement (Hb, platelets, leukocyte count), coagulation profile (PT/INR), electrolyte disturbances, blood gas analysis and serum lactate levels were analysed. Patients were continuously monitored for changes in vitals and cardiac conditions till the time of recovery. All patients were followed up for subsequent eight weeks.

### Results

In the cantonment area, a total of 50 heat related illness were reported in the said population in the given time period among which 18 cases were identified as a case of EHS. All the cases were reported in the month of March 2013 to September 2014 when average maximum temperature was around 42°C-47°C and humidity was around 60-80%. Mean age of the patients suffering from EHS was 30 (SD 7.0) years.

[Table/Fig-1] shows that all patients who were diagnosed as a case of EHS, had history of strenuous physical activity, among which 14 patients (77.8%) had run for 5 km while 4 patients (22.2%) had run for 2.4 km. Only 12 (66.7%) patients were acclimatised to the environment.

Predisposing Factors	Number (%)
<b>1. Acclimatisation</b>	
Yes	12 (66.7)
No	6 (33.3)
<b>2. Strenuous physical exercise preceding heat stroke</b>	
5 km run	14 (77.8)
2.4 km run	4 (22.2)

**[Table/Fig-1]:** Distribution of the predisposing factors among the individuals suffered from exertional heat stroke (n=18).

Clinical features	Number (%)
<b>1. Pulse rate</b>	
Tachycardia	16 (88.9)
Normal	2 (11.1)
<b>2. Blood pressure</b>	
Hypotension	2 (11.1)
Normotension	14 (77.8)
Hypertension	2 (11.1)
<b>3. Cyanosis</b>	
Present	6 (33.3)
Absent	12 (66.7)
<b>4. Wheeze</b>	
Present	2 (11.1)
Absent	16 (88.9)
<b>5. Neurological symptoms</b>	
Delirium	18 (100.0)
Seizures	3 (16.7)

**[Table/Fig-2]:** Clinical features of the individuals suffered from exertional heat stroke (n=18).

All the 18 cases of EHS had Core Body Temperature (CBT) of 40°C (i.e., 104°F) or more. All cases presented with neurological symptoms in the form of delirium (100%). [Table/Fig-2] shows that tachycardia was evident in 16 (88.9%) patients at the time of presentation. Blood pressure was normal in 14 (77.8%) patients, while 4 (22.2%) patients were having deranged blood pressure. Cyanosis was noted in 6 (33.3%) patients at presentation and 2 (11.1%) patients were having wheeze on chest auscultation. Seizures were seen in 3 (16.7%) cases.

Patients were shifted to 'Cool Room', which is provided with air conditioning, ice packs and other necessary equipments. The initial management included removal of clothes and drenching the patients with tepid water (20°C -25°C) along with fanning in cool room to facilitate evaporative cooling. Ice packs were placed near points of major heat transfer like axillae, groin, perineum, chest and neck. In one patient nasogastric lavage with ice-cold normal saline was also required. Prompt IV fluid infusion was started with normal saline and DNS alternatively and titrated on the basis of Central Venous Pressure (CVP) and urinary output. The treatment given to the patients resulted in dropping of the CBT to 37.2°C (99°F) within 45 minutes of initiating the treatment. All patients of EHS required 8 to 10 liters of fluid during resuscitation. Patients, with renal output less than 30 mL/hour, received high ceiling diuretic (Inj. furosemide 20 mg). Inj. diazepam was used in the maximum dose of 5-10 mg hourly not only for controlling seizures but also for providing sedation. In addition to diazepam, 12 (66.7%) patients needed continuous sedation using Inj. Propofol (0.5 mg/kg body weight) for 12 to 16 hours.

Complications developed among the patients suffering from EHS are shown in [Table/Fig-3]. All the patients suffered from diarrhoea. Urine output less than 30 mL/hour. with raised urea and creatinine values was observed among 6 (33.3%) patients. Liver enzymes were raised in 2 (11.1%) patients, whereas 12 (66.7%) patients had

Complications	Number (%)
1. Diarrhea	18 (100.0)
2. Acute lung injury	1 (0.06)
3. Raised liver enzymes	2 (11.1)
4. Raised urea, creatinine	6 (33.3)
5. Hypocalcaemia and lactic acidosis	12 (66.7)
6. Cardiac Ischemic changes	2 (11.1)
7. Multi Organ Failure Syndrome (MODS)	2 (11.1)
8. Rebound Hyperthermia	2 (11.1)

**[Table/Fig-3]:** Complications developed among the individuals who suffered from exertional heat stroke (n=18).

hypocalcaemia and lactic acidosis.

Patients with EHS recovered completely without any mortality and were discharged from the hospital within one week of admission, without any residual neurological deficit except two patients who developed multi organ failure requiring haemodialysis for six weeks.

## DISCUSSION

EHS is caused due to overwhelming response of the homeostatic mechanism involved in tackling the heat load presented to the body, generated intrinsically via intense muscular activity [1,6].

Heat stroke is identified when CBT i.e., rectal temperature rises above 40°C [1,3,5,9,10]. We also realised, that inclusion of this aspect in recognition of EHS is not only unnecessary, but in some instances, also hinders initiation of treatment within stipulated time. Recent research states that heat stroke can be defined with CBT ranging between 38.9°C-41°C (102°F-105.8°F); when they are specifically associated with neurological symptoms [1,4,5,6,11]. Though, all the 18 cases of EHS we had seen had CBT of 40°C or more, we still intend to introduce this slight change in the criteria for defining EHS on the basis of CBT<38.9°C, as it would reduce the threshold for initiating preventive and timely action by healthcare observers, as well as, prompts medical practitioners to initiate treatment at the appropriate time.

Though, it is expected that heat stroke would occur only in extremes of climate [1,2,12] and all cases identified as EHS in our study, had evidence of intense physical activity under extreme temperature (Temperature >30°C and humidity >70%), which puts them at Wet Bulb Globe Temperature (WBGT) of over 28 (ACSM recommends canceling sports activity above WBGT 28) [13], still we wish to point out here that extreme climatic conditions are not a prerequisite for occurrence of EHS [14-16]. Giercksky T et al., describe the case of a 31-year-old male who developed heat stroke after running 5 km at 21°C in Norway, who went on to develop severe liver failure as complication [17]. Similarly Boersma LVA et al., list the case of a 20-year-old male who developed EHS after biking for 3 hour in 26°C in forest of Netherlands [18].

Norms and regulations [3,19] regarding pre-requisites for environmental conditions have already been laid down, which need to be satisfied before initiating events involving intense physical activity and our experience shows that whenever these norms have been violated the risk for EHS increases manifold. From our study we could conclude that certain factors, like improper rest/work cycles (77.78% patients had long distance runs continuing over minimum five days), poor rehydration post activity and disregard for environmental conditions (high temperature and humidity) proved to be a major factor for development of EHS.

Lack of acclimatiation is a risk factor for EHS [3,20]. Acclimatiation is the term given for the physiological response to a new environment or climate [21]. In our study, all patients were trained soldiers who are regularly involved in long distance runs and had high standards of physical fitness. However, 33.3% of our patients were not

acclimatised to the surrounding environment (they had recently reported to this station from previous unit where temperature and humidity was low). Incidentally, these patients went on to develop severe liver failure and renal failure as complications of EHS. This observation brings out the basic requirement of acclimatising the soldiers to the surrounding environment before they are involved in intense physical activity. Evidence exists, that major physiological adaptation to heat stress, in the form of increased sweating causing fall in intravascular volume followed by expanding intravascular volume and ultimately adjusting cardiac output to maintain cardiac efficiency; occurs in the first week and takes up to two months [22]. For all practical purposes we recommend that at least 15 days of prior acclimatisation should be allowed to all soldiers, before indulging in rigorous physical activity.

Ice cold water immersion is the method of choice for rapid cooling of patients [1,3,7,12,22-24]. Without doubt the immersion of a patient in ice cold water has resounding effect in lowering the body temperature at the maximal rate, but it is a method that is not readily available everywhere, especially in the scenario of the military, which often trains in the hardest and most desolate of places. Under such circumstances, 'Evaporative cooling' which involves throwing tepid water over patient followed by blowing air over his body, is used, and is generally recommended for military world over [1,15,25]. This approach requires to strip the patient naked followed by covering the patient with wet gauge sheet and fanning along with water splashes [12]. In combination with ice water gastric lavage, placing ice packs over major arteries [12], and further placing in a cool room equipped with air conditioning proves to be effective in bringing down the temperature, as was observed in our study where all the cases of heat related illness, regardless of being EHS or not were given the same treatment and their core body temperature fell to 37.2°C (99°F) within first hour of treatment. As is evident from above, the most pertinent issue in EHS is to initiate cooling of CBT, using the best means possible, regardless of the comparative effectiveness of the method used. Various researches have laid down different end points of CBT where active cooling should cease. Though all of them emphasise on rapid cooling of the body, they also emphasise avoiding hypothermia occurring due to excessive cooling down of the body temperature [1,3,22,25]. American College of Sports Medicine has questioned the validity of the range of end point of reduction of CBT to be 38.3°C-38.9°C [13]. We continued our efforts of decreasing the CBT to normal body temperature (37.2°C) to assess the response in our patients. In our study, only 6 (33.3%) patients developed subsequent mild hypothermia, which could be attributed to lowering of body temperature below 37.2°C (99°F). This mode of treatment combined with the results in the form of nil mortality with lesser complications, has strengthened our belief that lowering the CBT further to 37°C using aggressive cooling methods is definitely desirable.

Fluid management is indeed, an integral part of managing cases of EHS, as EHS is a state of hyperthermia along with dehydration [1,3,5,22] faced with thermoregulatory stress, this shunting of blood away from the viscera leads to the clinical manifestations of tachycardia, acidosis [5,11]; neurological symptoms [5,18]; and renal failure [5,14,26]. Under such circumstances maintaining the hydration status of the individual is very pertinent. Though hypotension is not seen in all patients, the ones with hypotension are at a greater risk of a worse outcome [27]. Hence prompt fluid resuscitation, guided by urinary output of at least 50 mL/hour should be a standard of care [26]. In our study, 33.3% patients had urinary output less than 30 mL/hour upon catheterization at the time of presentation and the condition did not improve even after liberal fluid administration. This prompted the addition of diuretics of Inj. furosemide, following which the urinary output could be achieved at a higher and satisfactory rate. Only 11.1% patient developed frank renal failure, doubling of serum creatinine was observed in 33.3%

of cases. Rhabdomyolysis is inherent to exertional heat stroke [5] compounded by cellular injury due to lactic acidosis [5,11] resulting in myoglobin being released in blood. This myoglobin has potential to cause mechanical obstruction and incite an inflammatory reaction into renal tubules causing renal failure, and hence needs to be flushed out of the kidneys [1,3,5,22]. Hence, concluding from the existing evidence and our research, we believe that fluid resuscitation needs to be augmented with high ceiling diuretics like furosemide so as to allow an uneventful recovery and prevent renal injury caused by accumulation of myoglobin [1,3,5,22,26].

Benzodiazepines should be used for controlling seizure activity [1,14,26]. Benzodiazepines are recommended to be given to those patients with evidence of seizure activity. In our study three patients had seizure, and one had suspected syncope; however, all of them had delirium and violent behaviour on presentation. Inj. diazepam and inj. propofol were used to provide sedation and control of seizures in these patients.

Exertional heat stroke patient have a higher chances of leaving the hospital with residual deformity [10,12,14]. Several studies suggest that EHS patients develop significant complications during the course of treatment and in most instances, are destined to be discharged from the hospital with some residual deformity [5,10,26]. However, in our study, all our patients of EHS who did develop complications, recovered completely without any residual disabilities. One patient did go on to develop severe derangement in renal function and eventually required haemodialysis for six weeks. However, he had a complete recovery. We attribute our results to continuous monitoring of CBT of patient using electronic rectal probe, vigorous efforts were made to keep the body temperature within normal limits, and prompt treatment was initiated for emerging complications.

## CONCLUSION

It takes a colossal effort to recruit, train and prepare a soldier, and to lose him to an ailment like heat stroke, can only be viewed as a despicable loss of a precious resource and a demoralising failure for medical services. Despite the innumerable precautionary measures, EHS cannot be entirely prevented or eliminated but with measures like improved and accurate recording of temperatures, initiating treatment within 10 minutes of occurrence of EHS, prompt reduction in body temperature using combination of methods, adequate fluid administration, monitoring and maintaining urine output and continuous monitoring of CBT, a vigilant watch over the complications, it's blow may be softened. The above measures, especially when initiated within the first "Golden" hour of occurrence of any heat related illness, can reduce morbidity and prevent deaths.

## REFERENCES

- [1] Bouchama A, Knochel James P. Heatstroke, *N Engl J Med*. 2002;346:1978-88.
- [2] Schär C, Jendritzky G. Climate change: hot news from summer 2003. *Nature*. 2004;432:559-60.
- [3] Khosla R, Guntupalli KK. Heat-related illnesses. *Critical Care Clin*. 1999; 15(2):251-63.
- [4] Bricknell MCM. Heat illness- A review of Military Experience (Part I). *J R Army Med Corps*. 1995;141(3):157-66.
- [5] Grogan H, Hopkins PM. Heat stroke: implications for critical care and anaesthesia. *Br J Anaesth*. 2002;88(5):700-07.
- [6] Duthie DJR. Heat related illness. *Lancet*. 1998;352:1329-30.
- [7] Bouchama A, Muhammad D, Chaves-Carballo E. Cooling and haemodynamic management in heatstroke: Practical recommendations. *Critical Care*. 2007;11(3):R54.
- [8] Ghaznawi HI, Ibrahim MA. Heat stroke and heat exhaustion in pilgrims performing the Hajj (annual pilgrimage) in Saudi Arabia. *Ann Saudi Med*. 1987;7:323-6.
- [9] Winkenwerder W, Sawka M. Disorders due to heat and cold. In: Goldman L, editor. *Cecil Medicine*. 23rd edition, Philadelphia: Saunders Elsevier;2008.
- [10] American College of Sports Medicine, Armstrong LE, Casa DJ, Millard-Stafford M, Pyne SW, et al. American College of Sports Medicine position stand. Exertional heat illness during training and competition. *Med Sci Sports Exerc*. 2007;39(3):556-72.
- [11] Knochel JP. Exertional heatstroke- Pathophysiology of heatstroke. In: Hopkins PM, Ellis FR, eds. *Hyperthermic and Hypermetabolic Disorders*. Cambridge: Cambridge University Press, 1996;42-6.

- [12] Douglas J, Armstrong LE. Exertional heat stroke in competitive athletes. *Current Sports Medicine Reports*. 2005;4(6):309-17.
- [13] O'Connor FG, Casa DJ, Bergeron MF, Carter R, Deuster P, Heled Y, et al. American college of sports medicine roundtable on Exertional heat stroke - return to duty/return to play: conference proceedings. *Curr Sports Med Rep*. 2008;9(5):314-21.
- [14] Dematte JE, O'Mara K, Buescher J, Whitney CG, Forsythe S, McNamee T, et al. Near-fatal heat stroke during the 1995 heat wave in Chicago. *Ann Intern Med*. 1998;129(3):173-81.
- [15] Shapiro Y, Seidman DS. Field and clinical observations of Exertional heat stroke patients. *Med Sci Sports Exerc*. 1990;22(1):6-14.
- [16] Roberts WO. Exertional heat stroke during a cool weather marathon: a case study. *Med Sci Sports Exerc*. 2006;38(7):1197-203.
- [17] Giercksky T, Boberg KM, Farstad IN, Halvorsen S, Schmpf E. Sever liver failure in exertional heatstroke. *Scand J Gastroenterol*. 1999;34(8):824-27.
- [18] Boersma LV, Leyten QH, Meijer JWR, Strubbe EJ, Bosch FH. Cerebral haemorrhage complicating Heatstroke. *Clin Neurol Neurosurg*. 1998;110(2):112-15.
- [19] Minard C. Prevention of heat casualties in marine corps recruits. *Mil Med*. 1961;126:261-72.
- [20] Hajat S, Madeline O'Connor, Tom Kosatsky. Health effects of hot weather: from awareness of risk factors to effective health protection. *Lancet* 2010; 375(9717):856-63.
- [21] Bligh J, Johnson KG. Glossary of terms for thermal physiology. *J Appl Physiol*. 1973;35:941-61.
- [22] Stine RJ. Heat illness. *JACEP*. 1979;8(4):154-60.
- [23] Gaffin SI, Gardner KW, Flinn SD. Cooling methods for heatstroke victims. *Ann Intern Med*. 2000;132(8):678.
- [24] Smith J, Wallis L. Cooling methods used in the treatment of Exertional heat illness. *Br J Sports Med*. 2005;39(8):503-07.
- [25] Weiner JS, Khogali M. A physiological body-cooling unit for treatment of heat stroke. *Lancet*. 1980;1(8167):507-09.
- [26] Lugo-Amador NM, Todd R, Moyer P. Heat related illness. *Emer Med Clin N Am*. 2004;22(2):315-27.
- [27] Howe AS, Boden BP. Heat-Related Illness in Athletes. *Am J Sports Med*. 2007;35(8):1384-95.

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