Review Article

The evaluation and management of heat injuries in an intensive care unit

Seema Mahant

Abstract

In this summer season (May–June 2014) most of the days temperature was more than 40°C. Our hospital is surrounded by huge slums area. The population which is low in socioeconomic status used to work in such high temperature for more than 8 h daily. Hence, they are very prone to develop heat injuries in the form of heat edema, heat tetany, heat syncope, heat cramps, miliaria rubra, heat exhaustion, and heatstroke. Again it is compulsory to upgrade our knowledge on this life threatening condition.

Keywords: Body cooling unit (BCU) equipments, first aid for heatstroke, golden hour for treatment, heat related injuries, thermoregulatory failure

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Introduction

Heatstroke (HS) (or sunstroke) is the most serious form of heat-related illness and is a medical emergency. It is caused by an excessive rise in deep body temperature due to thermoregulatory failure and is characterized primarily by hyperthermia usually with core temperature above 40.6°C (105°F), due to environmental heat exposure with lack of thermoregulation and usually complicated with central nervous system dysfunction, metabolic derangement, and coma. HS is the most severe form of the heat-related illnesses so it must be distinguished from heat exhaustion, sepsis, diabetic ketoacidosis, closed head trauma, malignant hyperthermia, encephalitis, cerebral malaria, cerebral hemorrhage, amphetamine and cocaine toxicity, strychnine poisoning etc., This is a distinct form of a fever, where there is a physiological increase in the temperature set point of the body. It carries a high mortality nearly 80% if effective treatment is not given immediately.[1]

From:
Department of General Medicine, Intensive Care Unit, RKDF Medical College Hospital and Research Centre, Bhopal, Madhya Pradesh, India

Correspondence:
Prof. Seema Mahant, RKDF Medical College Hospital and Research Centre, Bhopal, Madhya Pradesh, India.
E-mail: pdmahant@gmail.com

Classification

Two forms of HS exist; "classical" non-exertional HS (NEHS) - more commonly affects sedentary elderly individuals, persons who are chronically ill and very young persons and exertional HS (EHS) in young individuals who engage in strenuous physical activity for a prolonged period of time in a hot environment {Table 1]. Both types of HS are associated with a high morbidity and mortality, especially when therapy is delayed.[2]

Epidemiology

With the influence of global warming, it is predicted that the incidence of HS cases and fatalities will become more prevalent. Due to temperature extremes in summer (May–June) in the vast expanses of gangetic and peninsular India (Uttar Pradesh, Bihar, Orissa, Madhya Pradesh, Maharashtra, Rajasthan, and Andhra Pradesh heat-related illness assumes an important public health dimension. India faced the worst heat wave in 1998 when 2600 deaths were reported in a span of 10 weeks. Over 1000 people perished in 2002 due to heat wave when the temperature crossed 122°F, in 2003 heat wave 1600 died of which 1200 were reported from Andhra Pradesh. During the 2005 heat wave, primarily affecting Andhra Pradesh, Orissa, and neighboring Bangladesh, mainly women, children, and people below the poverty line, were taken ill. An epidemiologic study in the United States estimated the

incidence of heatstroke 17.6–26.5 per 100,000 population. In a similar study in Saudi Arabia, the incidence varied from 22 to 250 cases per 100,000 populations with a crude mortality rate of 50%.[3]

Triggers of Heatstroke

Exposure to a hot environment caused by a hot environment that leads to a rise in body temperature, without strenuous physical activity. This type of heatstroke typically occurs in hot, humid weather, especially for prolonged periods. It occurs most often in older adults and in people with chronic illness. [Table 2]

- Strenuous activity - Anyone exercising or working in hot weather can get EHS, but it's most likely to occur if not accustomed to high temperatures
- Wearing excess clothing that prevents your sweat from evaporating easily and cooling your body
- Drinking alcohol which can affect your body's ability to regulate your temperature
- Becoming dehydrated because you're not drinking enough water to replenish fluids you lose through perspiration.

Pathophysiology

Thermoregulation is a highly complex inbuilt mechanism to maintain a constant range of temperature, which essential for normal functioning of the internal milieu. Any abnormal increase in temperature is gauged by the hypothalamic thermoregulatory center resulting in a reflex cutaneous vasodilatation and augmenting the skin blood flow. Thermal sweating is also induced resulting in a loss of as much as 600 Kcal/h by evaporation. Dehydration and salt depletion leads to impaired thermoregulation by decreasing the plasma volume and cardiac output (CO). When heat gain overwhelms the body's mechanisms of heat loss, the body temperature rises, and a major heat illness ensues. Excessive heat (usually temperature >42.2°C [108°F]) denatures proteins, destabilizes phospholipids and lipoproteins, and liquefies membrane lipids, leading to cardiovascular collapse, multi-organ failure due to cellular death, and ultimately, death. Temperatures exceeding 41.1°C (106°F) generally are catastrophic and require immediate aggressive therapy.

Thermoregulatory failure

Response to heat stress leads to a considerable increase in CO (up to 20 l/min), and shift of blood from core to periphery. Various factors such as salt and fluid depletion, cardiac disease, and cardio depressant drugs prevent this increase in CO, and the patients can lapse into HS.

Hyperactive acute phase response

Increase in peripheral blood flow leads to relative splanchnic (mainly hepatic, renal, and intestinal) ischemia, which predisposes to mucosal injury and alteration in immune and barrier. Function of the intestines. The splanchnic ischemia also leads to increased production of reactive. Oxygen and nitrogen species which may induce intestinal mucosal injury and hyperpermeability. This leads to bacterial and other antigens gaining access to circulation contributing to the stimulation of a systemic immune response. Cytokines and endothelial-derived factors such as tumor necrosis factors-á, interleukin-1b, etc., cause alteration of set-point for sweat activation and vascular tone leading to precipitation of hyperthermia and hypotension. Increase in cytokines in the central nervous system leads to increased intracranial pressure and reduced cerebral blood flow causing neuronal injury.

Altered heat shock response and coagulopathy

Heat shock response is protective in heat stress. Attenuation of heat shock response in HS confirms its protective value. Low levels of heat shock proteins are known to occur in old and unacclimatized individuals, and constitutionally in certain genetic polymorphisms favoring early progression from heat stress to HS.

Vascular endothelial damage due to direct cytotoxic effects of heat and inflammatory and coagulation responses to HS leads to diffuse microvascular thrombosis demonstrated by increase in factors like thrombin-antithrombin complexes, fibrin monomers, and fall in protein C, protein and antithrombin. There is marked activation of fibrinolysis as is evident from increased levels of D-dimers, plasmin-a2 antiplasmin complexes, and fall in plasminogen levels.[5]

Clinical Features/Signs and Symptoms of Heatstroke

Heatstroke is characterized by core temperature of more than 40°C and brain dysfunction which may vary from subtle confusion, abnormal behavior to coma. Classical HS occurs during extreme heat waves, the elderly being particularly vulnerable. Patients present with increased body temperature and altered mentation, tachycardia, hyperventilation, and occasionally hypotension. Hence, any patient with altered mentation during heat waves and after exertion should be suspected to have HS irrespective of his core temperature. Cardiovascular alterations include hypotension, tachycardia, ST-T changes, prolonged QT

interval, bundle branch blocks, and myocardial infarction. [Table 3]

Gastrointestinal manifestations are quite common and may comprise of severe diarrhea, jaundice, and deranged liver function tests. Metabolic alterations are characterized by respiratory alkalosis in classical HS, and respiratory alkalosis with lactic acidosis is typically seen in EHS. EHS may be associated with rhabdomyolysis, hyperphosphatemia, hypocalcemia, and hyperkalemia, and these findings are exaggerated after cooling once the normal circulatory state is restored. Jaundice is a frequent occurrence with transaminase levels peaking around day 3 of illness. Renal involvement is seen in almost 30% of EHS cases and is attributed to renal hypoperfusion, rhabdomyolysis, and thermal insult.[7]

Table 1: Differentiating features between heat exhaustion and heat stroke[2]

	Heat exhaustion	Heat stroke
Body temperature	37-40°C	>40.6°C
Skin	Moist	Dry
Sweating	Profuse	Absent
Hydration	Dehydrated	Normal
Pulse	Weak	Full-bound and strong
CNS symptoms	Absent	Delirium, convulsions, coma
Prognosis	Good	May be life threatening

CNS: Central nervous system

Table 2: Predisposing conditions[4]

Increased heat production	Reduced ability to acclimatize
Ambient temperature ≥35°C (e.g., during heat wave)	Children and toddlers
	Elderly persons
Increased metabolism	Obesity
Infections, Sepsis	Sleep deprivation
Encephalitis	Diuretic use
Thyroid storm	Hypokalemia
Decreased heat loss	**Increased muscular activity**
Reduced sweating	Exercise
Humidity >75%	Convulsions
Dermatologic diseases	Tetanus
Burns	Sympathomimetics
Reduced CNS responses	Moderate physical exercise, convulsions, and shivering can double heat production and result in temperature elevations
Advanced age, toddlers, and infants	
Alcohol, Barbiturates	
Other sedatives	Strenuous exercise and status epilepticus can increase heat production 10-fold and when uninterrupted, can overwhelm the body's heat-dissipating mechanisms, leading to dangerous rises in body temperature
Reduced cardiovascular reserve	
Elderly persons	
Drugs-beta-blockers, calcium channel blockers, diuretics, anticholinergics, neuroleptics, antihistamines	
High ambient temperatures effects of dopamine, serotonin, and norepinephrine	Stimulant drugs, including cocaine and amphetamines, can generate excessive amounts of heat by increasing metabolism and motor activity through the stimulatory
High ambient humidity	

CNS: Central nervous system

Differential Diagnosis

Heatstroke needs to be differentiated from certain encephalopathies like falciparum malaria, viral/bacterial meningoencephalitis. Sepsis, thyroid storm, pheochromocytoma, diabetic ketoacidosis, gastroenteritis, and pontine hemorrhage can also be confused with HS. Drug-induced hyperthermia, neuroleptic malignant syndrome, and MH should also be considered in the differential diagnosis and a proper history should be obtained.[8]

Complications

Complications of HS are multiple organ dysfunction syndrome and encephalopathy. The other complications encountered are cerebral edema, seizures,

Table 3: Clinical features/signs and symptoms of heatstroke

Onset-sudden	Mild BP↑-irritability
Core temperature (rectal): >40.6°C or >105°F (occasionally up to 45°C [113°F])	Confusion-dizziness
	Coma seizures
Anhidrosis prodrome (only in 25% cases)	Slurred speech irritability
Rapid and shallow breathing-nausea	Ataxia
Full bound pulse-vomiting	

Table 4: Methods of cooling

External cooling	Internal cooling
Heat loss by	
Evaporation-sprinkling water	Refrigerated intravenous saline infusion
Convection-fanning	Cold water lavage-gastric, peritoneal
Conduction-immersion in ice water (not preferred since limits heat loss due to intense peripheral vasoconstriction)	Extra-corporeal cooling-cardiac bypass, hemodialysis
Putting ice packs in axilla, neck and groin	

Most preferred practice-sprinkling water at 20°C over the body combined with fanning/blowing of warm air. BCU equipment may also be used for this[9]. BCU: Body cooling unit

Figure 1: Body cooling units

Figure 2: Body cooling unit (BCU) equipments

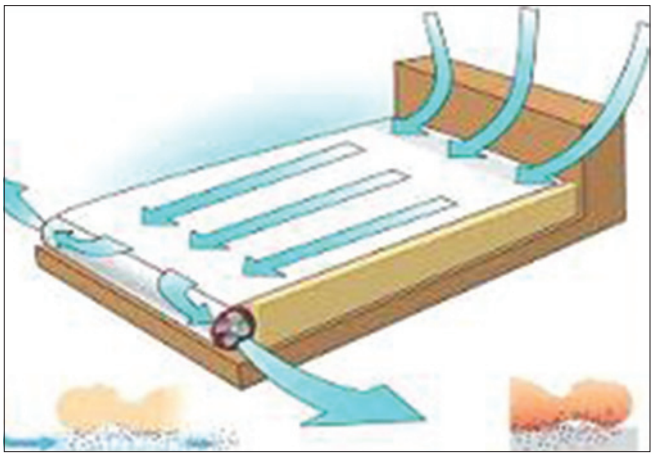


Figure 3: Body cooling unit (BCU) equipments

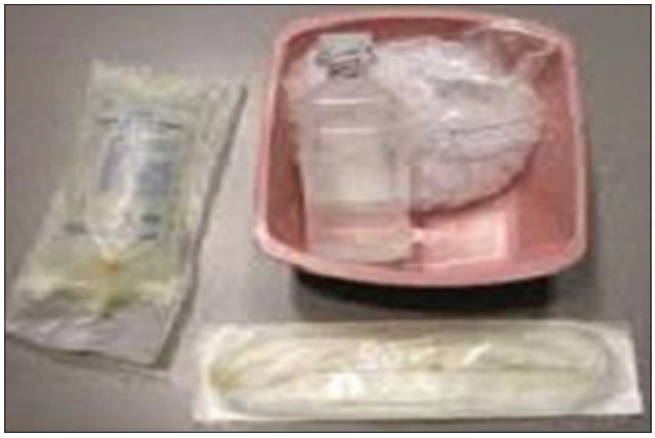


Figure 4: Body cooling unit (BCU) equipments

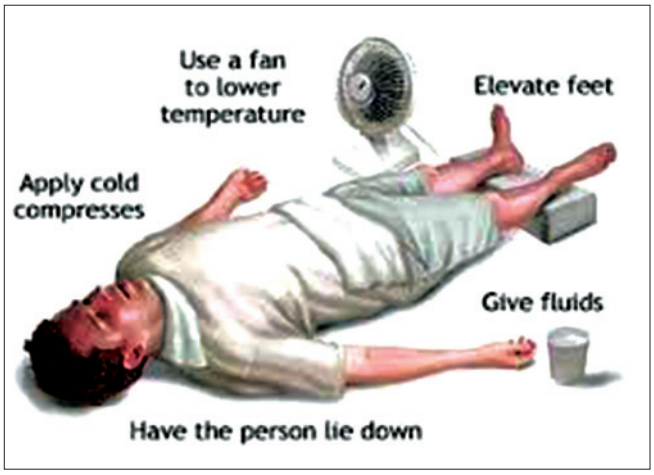


Figure 5: First aid for heatstroke

rhabdomyolysis, acute renal failure, acute respiratory distress syndrome, myocardial, hepatocellular, pancreatic and intestinal injury/ischemia, electrolyte disturbance and hemorrhagic complications like disseminated intravascular coagulation with marked thrombocytopenia.[8]

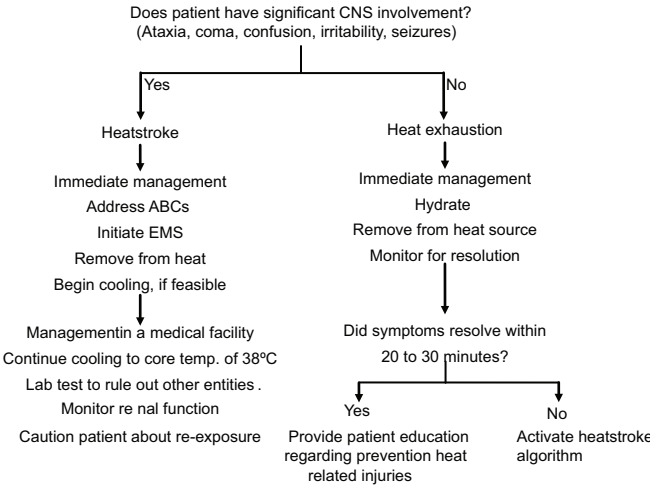
Treatment

First aid for heatstroke

If you suspect that someone has a HS, call 108 immediately or bring the person to a hospital. Any delay in seeking medical help can be fatal. While waiting for the paramedics to arrive, initiate first aid. Move the person to an air-conditioned environment-or at least a cool, shady area-and remove any unnecessary clothing. Fan air over the patient while wetting his or her skin with water from a sponge. Apply ice packs to the patient's armpits, groin, neck, and back. Because these areas are rich with blood vessels close to the skin, cooling them may reduce body temperature [Figure 1-4]. Immerse the patient in a shower or tub of cool water or an ice bath [Table 4] [Figure 5].[9]

Algorithm for a hyperpyrexia patient.[9]

First 3 h are the "golden hour" for heatstroke treatment.



Drugs helpful in malignant hyperthermia

Dantrolene - an agent that impairs calcium release from the sarcoplasmic reticulum and by doing so reduces muscle excitation and contraction. It is used in the treatment of malignant hyperthermia and neuroleptic malignant syndrome, reducing heat production that occurs as a result of muscle rigidity or hyper tonicity typical of these conditions.[9]

Prognosis

Recovery is rapid in EHS; patient recovers fully within half an hour of rectal temperature being brought down to 38°C (100.4°F) that is, approximately 1–2 h after starting treatment.

However, if the initial temperature is >42.2°C (108°F) (temperature at which cellular death occurs), there is 80% mortality. In classic heatstroke (NEHS), there may be a lucid interval of 12–24 h after which the patient may again deteriorate.

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