Medical Emergency

Heat Stroke

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Introduction

Heat Stroke (HS) - a life threatening medical emergency - is defined clinically as a core temperature (temperature of blood perfusing the hypothalamus, rectal temperature being the closest approximation) >40.6°C accompanied by central nervous system (CNS) dysfunction [1]. It is an important treatable form of Multiple Organ Dysfunction Syndrome (MODS) resulting from thermo-regulatory failure coupled with an exaggerated acute phase response and possibly altered expression of heat-shock proteins [2]. It is a common problem in the tropics, and with ever-increasing global warming its incidence is rising even in temperate climate. The highest incidence of heat illness of 45-1300/lac is reported from Saudi Arabia [3]. In India, HS occurs frequently in areas of Northern and Western India, and sporadic cases of Exertional Heat Stroke (EHS) are reported in military recruits. Despite the advances in last 50 years, mortality due to HS continues to be as high as 10 to 50% [4].

Two clinical presentations of HS are : classic HS and exertional HS (EHS). Classic HS is usually seen in extremes of age related to either limited mobility and / or chronic diseases or in predisposed individuals on exposure to excessive environmental temperature and humidity. EHS victims are active individuals who over-exert themselves in the heat. EHS can occur in rather more temperate climates, and cases have been reported in individuals exerting at temperature as low as 21°C [5]. A combination of the two types is frequently seen. Women are at lower risk of EHS probably due to lower muscle bulk, effects of estrogens and a lower threshold for, activation of thermo-regulatory reflexes [6].

Pathogenesis / Pathophysiology

Genetic factors may determine the susceptibility to HS via genes encoding cytokines, coagulation proteins and heat shock proteins [6]. Risk factors for HS include male sex, sleep deprivation, obesity, poor physical conditioning, lack of acclimatization, diuretic therapy, dehydration, febrile illness, alcohol abuse, skin diseases that affect sweating, heavy protective gear, drugs impairing normal thermoregulatory response, and a past history of HS.

Failure of normal cardiovascular adaptation to severe heat stress, exaggerated acute phase response and attenuated heat-shock proteins response are the main reasons which lead to HS [7]. HS and its progression to MODS result from complex interaction of acute physiological alterations associated with hyperthermia (increased metabolic demand, circulatory failure and hypoxia), direct cytotoxicity of heat, and inflammatory and coagulation responses of the host. This results in alteration in microcirculation and consequent damage to vascular endothelium and tissues [8]. Increased intestinal permeability mainly due to gut ischaemia (most of the cardiac output is diverted to exerting muscles and other peripheral organs) may lead to endotoxemia resulting in excessive production of inflammatory cytokines which induce endothelial cell activation and release of nitric oxide and endothelins [9].

Clinical Features

Raised body temperature and CNS dysfunction are common to both classic and exertional HS [2]. Classic HS victims usually present with hot dry skin, tachypnoea, tachycardia and hypotension. In EHS cases, sweating may be profuse or absent, and circulation is often hyperdynamic with marked tachypnoea [10,11,12]. Core temperature may be lower in patients given pre-hospital treatment. The single clinical finding that distinguishes HS from other forms of heat illness is altered mental status. Hence, any person who becomes irrational or confused or collapses following heat stress with or without physical activity, should be presumed to have HS regardless of core temperature and immediately given appropriate treatment. The characteristics of classical and EHS are give in Table 1. The clinical features noted by us in EHS are shown in Table 2 [13]. The degree and duration of hyperthermia may have equal importance. CNS damage is attributed to cerebral oedema, metabolic

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alterations and ischaemia. CNS dysfuction is usually severe (manifesting as encephalopathy, delirium, convulsions or coma) but may be subtle showing only as mild confusion, inappropriate behaviour or impaired judgement [4,5].

Table 1

Characteristics of classical and exertional heat stroke

Characteristics	Classical HS	Exertional HS
Age group	Older	Young
Occurrence	Epidemic	Sporadic
Predisposing illness	Frequent	Rare
Weather	Heat wave	Variable
Acid-base status	Respiratory alkalosis	Respiratory alkalosis + lactic acidosis
Rhabdomyolysis, renal failure, DIC	Rare	Common
Hyperuricemia	Mild	Marked

Table 2

Clinical and laboratory features of heat stoke

Feature	Frequency %	Feature	Frequency %
Violent behaviour	15	Convulsions	29
Confusion	15	Pre-existing skin boils	29
Coma	43	Gastroenteritis	43
Shock	29	Haematuria	29
Absent sweating	43	Leucocytosis	100
Jaundice	29	Raised aminotransferases	86
Cyanosis	29	Transient right bundle branch block	15
Dehydration	29		

Source : Adapted from Mehta SR, et al 1987 [13]

Although complete neurological recovery is the rule in survivors of HS, deficit may persist in 20% cases. Cerebellum is the most susceptible to thermal damage; the delayed manifestations are seen weeks after the insult and progress for a variable period thereafter. Rarely, intracerebral haemorrhage, central pontine myelinolysis and a clinical picture like Guillian Barre syndrome may occur. All patients of HS have tachycardia and hyperventilation. Hypotension is noted in 25% cases and is probably related to shift of blood to peripheral circulation and increased nitric oxide production [11]. Diffuse myocardial injury, raised CK-MB, tachyarrhythmias, non specific ST-T changes, prolongation of QT interval (probably due to hypokalemia, hypercalcemia or hypomagnesemia), bundle branch blocks and myocardial infarction may occur. Acute pulmonary oedema may occur due to excessive fluid administration during resuscitation. Gastrointestinal manifestations noted within hours of injury include diarrhoea, vomiting, gastrointestinal haemorrhage and elevated liver enzymes. Fulminant hepatic failure is rare. In survivors, jaundice and coagulation disturbances are seen 2-3 days following the thermal insult.

Acute renal failure seen in 30% of EHS cases is strongly related to rhabdomyolysis, hypotension, and in severe cases, to direct heat injury to renal parenchyma [6]. Coagulation disturbances are common and multifactorial. Thrombocytopenia, DIC and deranged prothrombin time may also occur. Other serious complications of HS are acute respiratory distress syndrome (ARDS) seen in around 23% of cases and rhabdomyolysis. Semenza et al in a study during Chicago heat wave of 1995 have brought out in detail important clinical features and complications of HS [11].

Biochemical abnormalities also include respiratory alkalosis (alongwith lactic acidosis in EHS), hypophosphatemia, hypokalemia, hypercalcemia and hypoglycemia. In EHS, rhabdomyolysis, hyperphosphatemia, hypocalcemia and hyperkalemia may be noted after complete cooling. Common febrile encephalopathies which may cause a diagnostic dilemma include falciparum malaria, meningo-encephalitis, sepsis and pontine haemorrhage.

Baseline tests should include chest radiography, ECG, cardiac enzymes, arterial blood gas study, blood for malarial parasite, blood culture, prothrombin time, fibrinogen level, blood chemistry profile, CK and urinalysis including urine for myoglobin.

Treatment

Better preparedness, prevention of heat related disorders and keeping and /or taking cooling items like ice and cold water etc., during exercises or exertions likely to lead to these disorders are the best strategies. A “heat stroke van” like a coronary care van, is the need of the hour. The most critical steps in the management of HS are immediate on-site initiation of rapid cooling and concurrent major resuscitation procedures. In the military setting, education of soldiers to recognize subtle behavioural signs possibly attributable to heat injury, helps in early detection of cases. Difficulties arise when collapse due to HS occurs unexpectedly in a person labouring in cool environment, temperature measurement is delayed, or inaccurate axillary or oral temperatures are not confirmed by true core temperature.

On the Spot Management

Move the patient to a cooler place, remove his or her clothing and initiate external cooling. Place cold packs on the neck, axillae and groin and carry out continuous fanning along with spraying of skin with water at 25-30°C. Position an unconscious patient to side and clear the airway. Administer oxygen at 4L/

min and give IV normal saline. The goals of these measures are immediate lowering of core temperature to <39.0°C and promote cooling by conduction and evaporation. The cooling process must be continued enroute when the patient is being transported to hospital.

Treatment in Hospital

In hospital, cooling measures of various kinds are in vogue [14,15]. The techniques should be readily available and rapidly instituted, and the core temperature should be lowered at least 0.1°C/min. All cooling techniques have similar efficacy. Give benzodiazepine to control seizures. Elective intubation is done for impaired gag and cough reflex to protect airway and augment oxygenation to keep SpO_2 >90%. Hypotension refractory to IV fluids and cooling measures may be due to vasodilatory shock and primary myocardial dysfunction. Vasopressors and CVP monitoring may be considered for such patients. In rhabdomyolysis, measures include, volume expansion with normal saline, IV frusemide, mannitol and sodium bicarbonate. Serum potassium and calcium levels must be monitored, and hyperkalemia treated to prevent life-threatening cardiac arrhythmias. The course of HS may be complicated by acute renal failure, ARDS, myocardial injury, hepatic failure, intestinal and pancreatic injury, and coagulopathies like DIC and MODS. The crux of management lies in supporting the patient through the afore mentioned life threatening complications.

The important methods of cooling are :-

I Techniques based on conductive cooling

(a) External :

(i) Cold water immersion or ice water bath i.e. placing the patient in a tank of iced water. Shivering and agitation are quite common in iced baths and can be treated with slow IV diazepam.

(ii) Application of cold packs or ice slush over part or whole of body.

(iii) Use of cooling blankets.

Concomitant vigorous massaging is recommended with all measures of external cooling to counter cutaneous vasoconstriction.

(b) Internal (not frequently used) - iced gastric lavage or iced peritoneal lavage.

II Techniques based on evaporative or convective cooling

(a) Fanning the undressed patient at room temperature (20-22°C).

(b) Spraying the uncovered patient with lukewarm water with continuous fanning

(c) Use of a body-cooling unit - a special bed that sprays atomized water at 15°C admixed with warm air at 45°C over the whole body surface to keep the temperature of wet skin between 32°C and 33°C [15].

No drugs that accelerate cooling have proved helpful in HS. Antipyretics have not been evaluated in standard trials and are presently contraindicated. Despite its relation to malignant hyperthermia, dantrolene has not been found effective in HS. The normalization of body temperature does not reverse the chain of inflammatory cascade precipitated by heat stress though cooling the patient to <38.9°C within 30 minutes of presentation is known to improve survival [16-18].

HS is a relatively immunosuppressed state and chances of secondary infection exist. Aspiration pneumonia is a well-known complication and antibiotics should be used in all such patients. Residual brain damage, especially the cerebellar syndrome and spinal cord lesions with motor neuron loss, may occur despite prompt treatment in about 20% of patients, and these are associated with higher mortality and morbidity [6,13]. Adverse prognostic factors in HS are delayed presentation to medical attention, hypotension, haemodynamic instability, raised enzymes specially LDH and aminotransferases, and residual neurological disability.

Conclusion

Heat related illnesses are increasing with increased global warming. Greater awareness regarding them will help in recognizing and treating these disorders at an early stage. HS is a preventable fatality warranting a high index of clinical suspicion in appropriate setting. Public education on heat illnesses, behavioural changes, restricted use of alcohol, enforced rests and fluid protocols, acclimatisation and ready availability of cooling facilities in hot areas will help decrease morbidity and mortality.

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The 1982 Israeli invasion of Lebanon resulted in many dogfights between Syrian and Israeli jet fighters. In the end, the Syrians lost over 80 planes and had a number of SAM batteries knocked out, while the Israelis lost no planes. Sometime later, the Syrian Defense Minister was shopping for weapons in Moscow. His host, the Soviet Defense Minister, was embarrassed about the scorecard from Lebanon. He told his Syrian guest, "Take anything you want - our best tanks, rifles, or surface-to-air missiles." "No, no - you don't understand!" the Syrian replied. "Last time you gave us surface-to-air missiles. This time we need surface-to-*jet* missiles!"

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A high school teacher was giving a true/false test. He was strolling up and down the aisles surveying the students at work. He came upon one student who was flipping a coin, then writing.

Teacher: What are you doing?

Student: Getting the answers to the test.

The teacher shook his head and walked on. A little while later, when everyone was finished with the test, the teacher noticed the student was again flipping the coin.

Teacher: Now what are you doing?

Student: I'm checking the answers

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