Heat-Related Illnesses

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ABSTRACT

Heat related illnesses include a spectrum of disorders from heat syncope, muscle cramps, heat exhaustion to medical emergency like heat stroke. Dissipation of heat occurs by cutaneous vasodilatation and diaphoresis. Factors that interfere with diaphoresis significantly increase the risk of heat illnesses. Acclimatization is a constellation of physiologic adaptations to lose heat more efficiently. Heat wave exacerbate the mortality rate, particularly among the elderly and among persons lacking adequate nutrition and accesses to cooler environments. Heat stroke is a Triad after exposure to a Heat of CNS Dysfunction, Anhidrosis and a Core Temperature >40.5^{0}C (105^{0}F). It is usually best to follow "cool first, transport second" guideline in these patients. Special therapeutic measures are requiredfor hyperthermia, acute kidney injury, hypotension, pulmonary dysfunction, arrythmias, metabolic disorders, rhabdomyolysis and coagulopathies.

Introduction :

Heat Wave is an extreme weather event. It has recently become a concern for disaster management in India due to widespread and severe impact on health and environment. Since last few years, heat wave during summer season is increasingly affecting morbidity and mortality in the country. Heat waves are known to be "silent killers" amongst the natural disasters. Since beginning of the 21st century, the average global temperature has increased by nearly a degree Centigrade. This weather pattern, coupled with the El-Nino effect, is increasing the temperatures in Asia. There has been an increasing trend of heat-wave in India over the past several years whereby several cities in India have been severely affected. In India, 2014 surpassed 2010 as the warmest year in a global temperature record that stretches back to 1880s. In India heat wave killed about 3000 people in year 1998, more than 2000 in year 2002 and more than 2400 died in the heat wave of 2015. Heat wave should be declared when actual maximum temperature remains 45^{0}C in plains. So we in Vidarbha live in a state of persistent heat wave during the summer months.

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Heat related illnesses include a spectrum of disorders from Heat Syncope, Muscle Cramps, Heat Exhaustion to medical emergency like Heat Stroke. The International Classification of Diseases (ICD) contains 10 categories of heat illnesses. Heat Stroke in patients who present to the hospital have mortality, with rate ranging from 21 - 63%. Humans are capable of significant heat generation. The heat load from metabolic heat production and environmental heat absorption is balanced by a variety of heat dissipation mechanisms. The central integrative dissipation pathways are orchestrated by the central thermostat, which is located inpreoptic nucleus of the anterior hypothalamus. Dissipation of heat occurs by cutaneous vasodilatation and diaphoresis. Factors that interfere with diaphoresis significantly increase the risk of heat illnesses.

Acclimatization is a physiologic adaptation to lose heat more efficiently. It requires one to several weeks after exposure to high temperature environment. During acclimatization, thermoregulatory set point is altered and this alteration affects the onset, volume and content of diaphoresis. The threshold for initiation of sweating is lowered, and the amount of sweat increases, with a lowered salt concentration. Expansion of plasma volume also occurs and improves cutaneous vascular flow and the heart rate lowers with a higher stroke volume.

When there is an excessive heat load, unacclimated

individuals can develop a variety of heat-related illnesses. Heat waves exacerbate the mortality rate, particularly amount the elderly and among persons lacking adequate nutrition and access to air-conditioned environments.

Table 1 : **Predisposing Factors for Heat-related illness and associated mechanism :**

ILLNESS	PREDISPOSING FACTORS
Cardiovascular inefficiency	Elderly age group Beta/calcium channel blockade Congestive heart failure Dehydration Diuresis Obesity Poor physical fitness
Centralnervous system illness	Cerebral hemorrhage Hypothalamic cerebrovascular accident Psychiatric disorders Status epilepticus
Impaired heat loss	Antihistamines Heterocyclic antidepressants Occlusive clothing Skin abnormalities Ectodermal dysplasia Anhidrosis Sickle cell trait Autonomic neuropathy
Endocrine-related illness	Diabetic ketoacidosis Pheochromocytoma Thyroid storm
Excessive heat load	Environmental conditions Exertion Fever Hypermetabolic state Lack of acclimatization
Infectious illness	Cerebral abscess Encephalitis Malaria Meningitis Sepsis syndrome Tetanus Typhoid
Toxicologic illness	Amphetamines Anticholinergic toxidrome Cocaine Dietary supplements Hallucinogens Malignant hyperthermia Neuroleptic malignant syndrome Salicylates Serotonin syndrome Strychnine Sympathomimetics Withdrawal syndromes (ethanol, hypnotics)

Classification and Definition of Heat-related illnesses :

A. Minor Heat-Related illnesses :

1. Heat edema is characterized by mild swelling of the hands, feet, and ankles during the first few days of significant heat exposure. The principle mechanism involves cutaneous vasodilatation and pooling of interstitial fluid in response to heat stress. Heat also increases secretion of antidiuretic hormone and aldosterone. Simple leg elevation or thigh-high support hose is sufficient for treatment. Diuretics are not effective and, in fact, predispose to volume depletion and the development of more serious heat-related illnesses.
2. Prickly heat (miliariarubia, lichen tropicus)- It is a maculopapular, pruritic, and erythematous rash due to blockage of sweat pores by debris from macerated stratum corneum, it causes inflammation in sweat ducts as the ducts dilate and rupture to produce superficial vesicles. The predominant symptom is pruritis. Treated with antihistamines and local applications of chlorhexidine. Clothing with breathable fabric should be clean and loose fitting. Activities or environment that induce diaphoresis should be avoided.
3. Heat syncope (exercise-associated collapse) can follow endurance exercise or occurs in the elderly. Other scenarios include prolonged standing while stationary in the heat and sudden standing after prolonged exposure to the heat. Heat stress causes volume depletion, decreased vasomotor tone and peripheral vasodilatation which leads to postural hypotension in unacclimatized elderly individuals. After removal from the heat source, most of the patients will recover promptly with cooling and rehydration.
4. Heat exposure stimulates hyperventilation producing respiratory alkalosis, paresthesias and carpopedal spasm. Treatment includes removal of patient from exposure, reassurance and addressing the hyperventilation.

B. Major Heat-Related Illnesses

Heat Cramps

Heat cramps are intermittent, painful and involuntary spasmodic contractions of skeletal muscles. Typically occurs in unacclimated individual after vigorous exertion in a humid, hot environment There is relative deficiency of sodium, potassium and intracellular fluid. The Hypotonic fluid intake and a large amount of sodium in diaphoresis cause further hyponatraemia and hypochloraemia which result in muscle cramps. Treatment includes ingestion of electrolyte solutions or dissolved two 650 mg salt tablets in 1 quart of water to produce a 0.1% saline solution.

Heat exhaustion

Core temperature is elevated but is generally < 40.5°C (<105°F) and the thermoregulatory and CNS function are maintained. Two physiologic precipitants are water depletion and sodium depletion, which often occur in combination. Labourers, athletes and elderly individuals exerting themselves in hot environments, without adequate fluid intake, tend to develop water-depletion and heat exhaustion. Persons working in the heat frequently consume only two-thirds of their net water loss and are voluntarily dehydrated. In contrast salt-depleted heat exhaustion occurs more slowly in unacclimated persons who have been consuming large quantities of hypotonic solutions. Mild neurologic and gastrointestinal influenza-like symptoms are common. These symptoms may include headache, vertigo, ataxia, impaired judgment, malaise, dizziness, nausea, and muscle cramps. Orthostatic hypotension and sinus tachycardia develop frequently. Aggressive cooling of non-responders is indicated until their core temperature is 39°C (102.2°F). Except in mild cases, free water deficits should be replaced slowly over 24-48 h to avoid a decrease of serum osmolality by >2 mOsm/h.

HEATSTROKE :

There is total loss of thermoregulatory function. Triad after heat exposure of Anhidrosis, CNS dysfunction, and a Core temperature > 40.5°C

(105°F) helps establish the preliminary diagnosis. If the patient's mental status does not improve with cooling, toxicologic screening may be indicated, and Cranial CT and spinal fluid analysis can be considered. Sudden onset of heatstroke occurs when the maintenance of adequate perfusion requires peripheral vasoconstriction to stabilize the mean arterial blood pressure. As a result, the cutaneous radiation of heat ceases. At this juncture, the core temperature rises dramatically.

There are two forms of heatstroke :

1. Classic Heatstroke (CHS)
2. Exertional Heatstroke (EHS)

Table 2 : **Difference between CHS and EHS**

CLASSIC	EXERTIONAL
Older patient	Younger patient
Predisposing health factors / medications	Healthy condition
Epidemiology (heat waves)	Sporadic cases
Sedentary	Exercising
Anhidrosis	Diaphoresis (common)
Central nervous system Dysfunction	Myocardial / hepatic injury
Oliguria	Acute renal failure
Coagulopathy (mild)	Disseminated Intravascular coagulation
Mild lactic acidosis	Marked lactic acidosis
Mild creatine kinase elevation	Rhabdomyolysis
Normoglycemia/calcemia	Hypoglycemia/calcemia
Normokalemia	Hyperkalemia
Normonatremia	Hyponatremia

Patients with CHS are usually elderly with some predisposing factors which make them prone to heat related illnesses. Skin of these patients is usually hot and dry because of dehydration. If cooling is delayed severe hepatic dysfunction (AST and ALT often increase to >100 times of normal) , renal failure, disseminated intravascular coagulation occurs and fulminant multisystem organ failure may occur. Coagulation studies commonly demonstrate decreased platelets, fibrinogen, and prothrombin. Hypernatremia is secondary to dehydration in CHS. Many patients exhibit significant stress leukocytosis, even in the absence of any infection. Most patients with CHS require cautious crystalloid resuscitation, electrolyte monitoring.

EHS

Athletes, labourers, and military recruits are common victims for exertional heat stroke (EHS) and affected individuals are profusely diaphoretic despite significant dehydration. Hyponatremia, hypoglycemia, and coagulopathies are more frequent than CHS. Lab investigations can show elevated creatine kinase and lactate dehydrogenase levels, which suggest EHS. Oliguria is a common finding. Renal failure can result from direct thermal injury, untreated rhabdomyolysis or volume depletion. Common urinalysis findings include microscopic hematuria, myoglobinuria, and granular or red cell casts. Heatstroke often causes thermal cardiomyopathy resultingin an elevated CVP despite significant dehydration. Additionally the patient often presents with potentially deceptive noncardiogenic pulmonary edema and basilar rales despite being significantly hypovolemic. The electrocardiogram commonly displays a variety of tachyarrhythmias, nonspecific ST-T wave changes, and heat-related ischemia or infarction. Antiarrythmics are seldom necessary and electrical cardioversion should be avoided until cooling is achieved, unless necessary to treat ventricular fibrillation or pulseless ventricular tachycardia.

COOLING STRATEGIES

Heatstroke victims must be immediately and aggressively cooled. This must be initiated concurrently, with control of the airway, breathing and circulation. Morbidity and mortality are directly associated with the duration of elevated core temperature. Continuous core-temperature monitoring should be considered, as peripheral methods to measure temperature are notreliable. Peripheral vasoconstriction delays heat dissipation, repeated administration of discrete boluses of isotonic crystalloid for hypotension is preferable over the administration of á-adrenergic agonists. Evaporative coolingis frequently the most practical and effective technique. Cool water (15°C [60°F]) is sprayed on the exposed skin while fans direct

continuous airflow over the moistened skin. Cold packs applied to the neck, axillae, and groin are useful cooling adjuncts.

Immersion cooling in ice-cold water is an alternative option in EHS but can induce peripheral vasoconstriction and shivering. This technique presents significant monitoring and resuscitation challenges in many clinical settings. The safety of immersion cooling is best established for young, previously healthy patients with EHS. Active cooling should be terminated ~ 38°39°C (100.4°F102.2°F), to avoid hypothermic after drop associated with cooling after immersion.

Other methods **-** Commercial cooling blankets should not be used as the sole cooling technique as the rate of cooling is far too slow. Cold thoracic and peritoneal lavage are efficient manoeuvres but are invasive and rarely necessary. Cardiopulmonary bypass provides effective cooling but is labor intensive and is rarely necessary.

RESUSCITATION

Aspiration commonly occurs in heatstroke, and endotracheal intubation is usually necessary to secure the airway. Metabolic demands are high, and supplemental oxygenation is essential due to hypoxemia induced by thermal stress and pulmonary dysfunction. Pneumonitis, pulmonary infarction, hemorrhage, edema, and acute respiratory distress syndrome occur frequently in heatstroke patients. The circulatory fluid requirements, particularly in CHS may seem falsely modest. Many patients present with a thermally induced hyperdynamic circulation accompanied by a high cardiac index, low peripheral vascular resistance, and an elevated CVP caused by right-sided heart failure. In contrast, most patients with EHS require far more zealous isotonic crystalloid resuscitation. Hypotension as an initial presentation in patients with heat stroke results from dehydration as well as high-output cardiac failure. Inotropes causing -adrenergic stimulation (e.g., norepinephrine), impede cooling by causing significant vasoconstriction. But at the same time vasoactive catecholamines such as dopamine or dobutamine may become necessary if the cardiac output remains depressed despite an elevated CVP, particularly in patients with a hyperdynamic circulation.

The administration of atrial or ventricular antiarrhythmic medications is rarely indicated for tachyarrhythmias that are routinely observed on presentation, as they resolve spontaneously during cooling. With a cardiac rhythm that sustains perfusion, electrical cardioversion of the hyperthermic myocardium should be deferred until the myocardium is cooled.

Significant shivering, discomfort, or extreme agitation is preferably mitigated with short-acting benzodiazepines, which are ideal due to their renal clearance. Whereas chlorpromazine may lower the seizure threshold, has anticholinergic properties, and can exacerbate the hypotension or cause neuroleptic malignant syndrome.

Metabolic acidosis and respiratory alkalosis are the most common abnormalities on blood gas analysis. Myoglobinuria should be suspected in any patients with brown urine supernatant that is heme positive and clear plasma.

Coagulopathies more commonly occur after the first day of illness and more so after cooling, during which the patient should be monitored for disseminated intravascular coagulation, and replacement therapy with fresh-frozen plasma and platelets should be considered.

ROLE OF ANTIPYRETICS

Antipyretics have no role in the control of environmentally induced hyperthermia as these drugs block the actions of pyrogens at hypothalamic receptor sites. Salicylates can further uncouple oxidative phosphorylation in heatstroke and exacerbate coagulopathies. Acetaminophen may further stress the hepatic function.

OUTCOME

Mortality (<10%) has improved over past years with increased awareness, rapid diagnosis and aggressive management. Overall mortality is higher in the classic heatstroke group, and up to 33% have a residual neurological deficit. Poor prognostic indicators in classical heatstroke include

hypotension, requirement for endotracheal intubation, coagulopathy and advanced age. The majority of exertional heatstroke victims should make a full recovery when optimally managed. Severe brain injury accounts for most of the fatalities in those who do not survive.

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