Chapter

Acute Management of Heat Stroke: Facts and Figures

Gamal Al-Ameri, Waleed Salem, Galal Alessai, Mohammed Hellboob, Mubarak Alhatemi, Umme Nashrah and Nissar Shaikh

Abstract

Heat-related illnesses range from heat exhaustion to heat stroke. Heat stroke is a life-threatening medical emergency causing multiple organ dysfunction that if not treated, can be fatal. It is a severe heat illness with a body temperature of more than 40^0c and organ dysfunction. Epidemiology of heat stroke varies depending on geographic location, and reported incidences range from 1.98 to 2.89/100000 per year. Heat stroke is classified as exertional or non-exertional. Pathophysiology is exposure to higher temperatures with impaired thermoregulation. Patients will present with high core body temperature; tachypnea, tachycardia, and hypotension may be present. The manifestations of organ dysfunction range from coagulopathy to altered levels of consciousness, and pulmonary edema. Accurate core body temperature measurement with clinical manifestations will diagnose the heat stroke. Early diagnosis, earlier temperature management, and organ supportive care are essential.

Keywords: core body temperature, emergency, heat stroke, temperature management, organ supportive care

1. Introduction

Heat-related illness is a spectrum of conditions that severely increase from heat exhaustion and heat injury to life-threatening emergency called heat stroke. The manifestation of heat stroke ranges from delirium to seizures. Heat stroke, or sunstroke, is a life-threatening emergency. It is a severe heat illness that increases body temperature to more than 40°C (104 F), heat stroke causes more than 600 deaths per year in the US [1].

2. Epidemiology

In the United States, from 2006 to 2010, it was reported to have caused at least 3332 deaths. Heat stroke mortality is correlated with the degree of body temperature elevation, time of initiation of cooling, and number of organs affected [2].

Various aspects can affect the incidence of heat stroke, including gender, age, geographic location, and occupation. Heat stroke is higher in females, with a female-to-male ratio of up to 2.89 versus 0.98–1.98 per 1000 person-years [3].

Heat stroke has different rates of occurrence in different geographic areas. During the European heat wave in 2022, thousands of people died due to heat stroke. Heat stroke mortality is expected to rise by 2.5 times by 2050 [3].

3. Classification and definition

Bouchama's definition is the most commonly accepted for heat stroke, and it is a rise in core body temperature of more than 40°C with hot, dry skin and central nervous system abnormalities. Another alternative definition of heat stroke is the hyperthermia associated with SIRS (Systemic Inflammatory Response Syndrome), which causes multiple organ dysfunction and mainly central nervous system dysfunction [4].

The JAAM heat stroke committee (Japanese Association for Acute Medicine) heat stroke committee, after analysis of the data collected by the working group, modified the heat stroke definition. Heat stroke is defined as patients exposed to high environmental temperatures who meet one or more of the following criteria [5]:

1. Glasgow Coma Scale (GCS) score of ≤ 14,

2. Creatinine or total bilirubin levels of ≥ 1.2 mg/dL,

3. JAAM DIC score of ≥ 4.

4. Classification

Heat stroke is typically classified into two groups depending upon the presence or absence of exertion [4, 5].

1. The exertion of heat stroke occurs in healthy individuals performing rigorous physical activity in higher environmental temperatures.

2. The non-exertional heat stroke develops during minimal physical activity in elderly persons with comorbidities including hypertension, diabetes mellitus, obesity, renal and cardiac disease, or alcoholism.

Non-exertional or classic heat stroke occurs in individuals at extremes of age, elderly over 70, or children in the vehicle. The main issue in this type of heat stroke is an anatomical or physiological predisposition or the patient's comorbidities, all of which impair thermoregulation, prevent removal from a hot environment, or interfere with access to hydration or attempts at cooling. Prescribed or recreational medication, anticholinergics, beta-blockers or diuretics, and cocaine and alcohol, respectively, also contribute to non-exertional heat stroke. Exertional heat stroke occurs when a healthy individual heavily exercises at a higher ambient temperature.

5. Pathophysiology

5.1 Thermoregulation

The human body maintains a temperature of 37 C, through convection, conduction, radiation, and vaporization, which are controlled by the hypothalamus. In cases of an increase in body temperature, the sympathetic system is activated, causing cutaneous vasodilation and thermal sweating. These also cause a loss of intravascular volume and salt, leading to dehydration. Increased severity causes further loss of water with salt, leading to impaired thermoregulation and reduction of visceral blood volume with shunting to the skin and muscles, resulting in organ dysfunction (above). In response to stress, body cells generate heat stroke proteins (HSP). An increase in HSP is essential for heat tolerance, and overexpression of HSP protects against organ impairment.

The increase in body temperature causes intestinal mucosal injury, and the release of endotoxins and proinflammatory mediators interleukins 1 and 6 from muscles to the systemic circulation, these chemicals leucocytes and endothelial cells, and the release of various cytokines and HMGB (high mobility group box 1 protein), all in combination stimulate SIRS (systemic inflammatory response syndrome) (**Figure 1**) [5].

The heat stroke thus generates inflammatory and coagulopathy responses; the direct effect of heat also causes endothelial injury and generates microthromboses, leading to a disseminated intravascular coagulation (DIC) response.

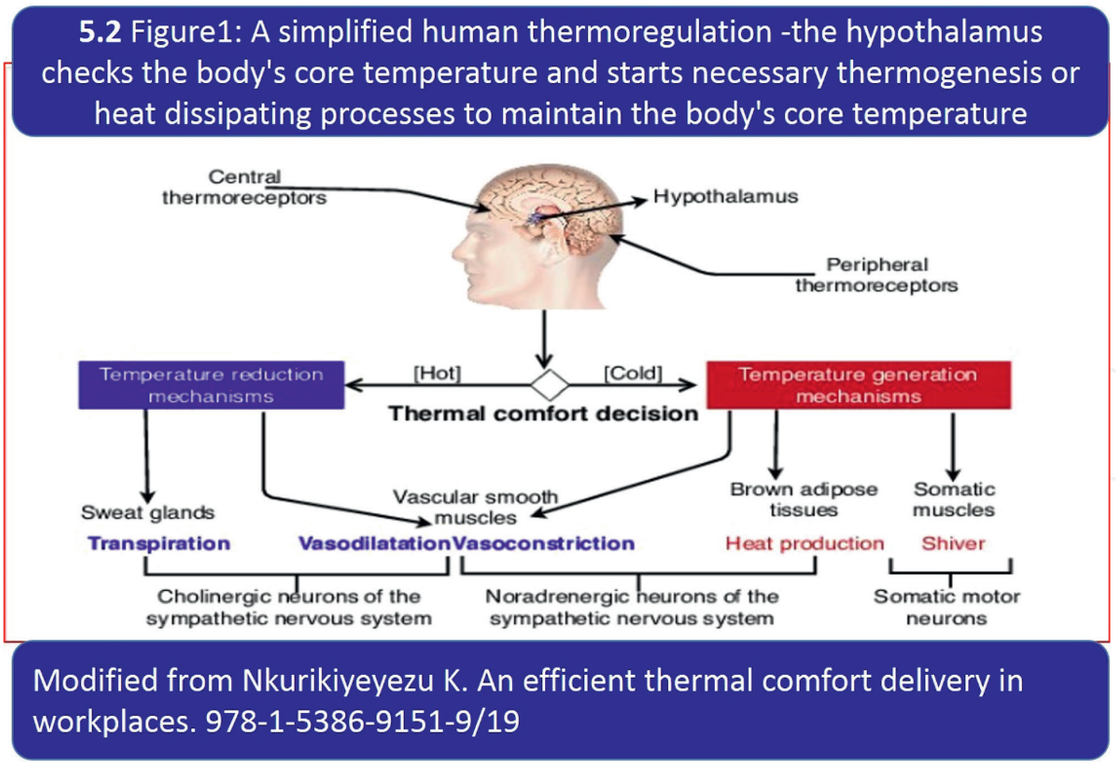

Figure 1.
A simplified human thermoregulation –the hypothalamus checks the body's core temperature and starts necessary thermogenesis or heat dissipating processes to maintain the body's core temperature. Modified from Nkurikiyeyezu K. An efficient thermal comfort delivery in workplaces. 978-1-5386-9151-9/19.

Diuretic therapy
Elderly (> 70 years)
Cardiac disease
Oncological disorders
Core body temperature > 40°C
Hypotension
Hypertension
Renal dysfunctions
Coagulopathy
Requirement of vasopressors
Decrease conscious level
hypoxia
No air cooling,

Table 1.
Prognostic factors.

6. Prognostic factors

Table 1 shows prognostic factors in patients with heat stroke. These factors increase mortality and morbidity.

7. Risk factors for heat stroke

The factors that increase the chances of a classic heat stroke are age, obesity, poor physical condition, lack of acclimatization, no air conditioning, and social isolation. Other risk factors are the use of illicit drugs, recreational drugs, and comorbidities such as cardiovascular disease and diabetes mellitus.

The factors associated with increased mortality and morbidity in heat-exertion patients are the degree of temperature elevation and the number of organ dysfunction [1, 5].

8. Clinical presentation

Elevated core body temperature and central nervous system disorder are diagnostic presentations of heat stroke.

With increased body temperature or hyperthermia, there will be tachycardia, tachypnea, wide pulse pressure, and hypotension mostly due to dehydration. Organ dysfunction can manifest as noncardiogenic pulmonary edema with crepitation and desaturation.

If coagulopathy is severe will be skin rashes, petechiae, and ecchymosis, the neurological dysfunction will be manifested by an altered level of consciousness, speech distortion, agitation and irritability, seizure, or coma [6].

9. Diagnosis

A missed diagnosis of heat stroke increases mortality and morbidity. Accurate measurement of core body temperature is essential in these patients. The X-ray chest may show diffuse infiltrates and signs of pulmonary edema. ECG may show different changes, from dysrhythmias to ST segment changes [1, 4].

CBC (complete blood count), renal function, coagulation parameters, and serum urinary electrolytes may be deranged. Usually, liver function takes a day or two to be deranged.

10. Management

Early diagnosis, quicker cooling, correction of electrolyte abnormalities, and supportive care are essential for a better patient outcome.

Depending on the patient's level of consciousness, if the patient is still able to obey commands, oxygen supplementation via face mask or nasal cannula should be enough. If the patient has lower GCS and cannot protect the airway, intubation, and mechanical ventilation are mandatory.

Hypotension or shock management should be guided in critically ill patients with the use of novel tools for hemodynamic monitoring and the use of point-of-care ultrasound technologies.

These patients, if still hypotensive after resuscitation, may require vasopressors and inotropes. Cooling with temperature monitoring. Rapid cooling with accurate core body temperature monitoring is essential for patients with heat stroke.

Evaporative and connective cooling are found to have a better outcome, as they are effective maneuver and easy to perform.

Evaporative and connective cooling are performed with a spray of a moist tube of warm water and using fans to blow air over the moist skin of a naked patient.

The technique may require sedation with shorter-acting agents, particularly if patients are irritable or have altered mental status.

Immersing these patients in ice water is also a rapid, noninvasive method of cooling patients. Those patients are immersed, and it is difficult to have a venous cannula. Another alternative is to place the patient on a porous stretcher positioned on top of a tub of ice water and have paramedical staff continuously pour ice water from the bath onto to patient and manage the major muscular area [7]. Here, we can better monitor patients and have intravascular accesses.

Otherwise continuously applying ice to the axilla, neck, and groin will also help in cooling.

More invasive is peritoneal and pleural cold water. Continuous lavage is an alternative but is invasive, and one has to be careful in patients with coagulopathy. More recently, intranasal cooling was also used in those patients [7].

Pharmacological therapy and alcohol sponges should be avoided. Alcohol may get absorbed by dilated skin vessels and may cause toxicity, whereas those anti pyritic medications, aspirin, and paracetamol are not useful in heat stroke patients as the mechanism does not involve a change in the hypothalamic set point. These may cause adverse effects on the liver [5, 7, 8].

All these patients' temperatures should be monitored continuously and accurately, either by rectal or oropharyngeal temperature monitoring.

Cooling measures should be stopped once a temperature of 38 or 39C is achieved in order to reduce the iatrogenic hypothermia [8].

11. Organ dysfunction and its management

Heat stroke can cause multiple organ dysfunction. We will describe the heat stroke causing multiple organ dysfunction in system-wise order.

11.1 Respiratory dysfunction

Heat stroke patients can have aspiration pneumonia, bronchospasm, non-cardiogenic pulmonary edema, pneumonitis, pulmonary hemorrhage, and infection. Invasive ventilation with endotracheal intubation enables protection of the airway and management of increased metabolic demand through oxygen supplement and increased minute ventilation [9].

11.2 Cardiac dysfunction

Heat stroke can cause acute cardiac decompensation and myocardial injury, with reversible cardiac biomarkers elevation. Cardiac dysfunction and tachyarrhythmias respond well to cooling, hence anti-arrhythmics are seldom necessary, and electric cardioversion should be avoided until cooling is achieved unless necessary to treat VF (ventricular fibrillation) or PVT (pulseless ventricular tachycardia) [9].

11.3 Hypotension

Hypotension in these patients is due to volume depletion, vasodilation, and cardiac dysfunction, therapy is isotonic or crystalloid fluid resuscitation, and alpha-adrenergic medication impairs cooling and should be avoided [9].

11.4 CNS dysfunction

Cerebral edema and seizure are frequent CNS complications in heat stroke patients.

A variety of cerebrovascular disorders ranging from neuropathies, Gillian-Barre Syndrome, and Parkinsonism are reported in heat stroke patients [10].

Standard anticonvulsant and anti-edema measures can be followed in these patients with continuous EEG monitoring.

11.5 Rhabdomyolysis

Rhabdomyolysis is frequent in heat stroke patients, and can be typically managed by hydration-forced diuresis and electrolyte management.

11.6 Acute kidney injury

Heat stroke patients will have AKI due to severe dehydration, or rhabdomyolysis. Serum electrolyte and renal functions should be monitored regularly and depending on AKI severity, may require renal replacement therapy.

11.7 Hepatic injury

The liver function should be monitored in patients with heat stroke, liver injury in these patients is self-limited but can progress to hepatic failure and require transplant [11].

11.8 Disseminated intravascular coagulation (DIC)

DIC is more frequent in patients with heat stroke during the initial 72 hours. All these should be monitored with traditional coagulation parameters (PT, aPTT, INR, fibrinogen, D-Dimer) and state of art management such as ROTEM. If required, DIC can be treated with the replacement of clotting factors, fresh frozen plasma, or prothrombin complex.

12. Mortality and outcome

Heat stroke patients' outcomes depend on early diagnosis, early cooling, and prevention and treatment of organ dysfunction. These patients are at higher risk compared to the control group for myocardial ischemia, kidney injury, and ischemic stroke in the long term [12].

In these patients regardless of a number of organ dysfunction, the heat stroke survivors will have long-term functional and neurological disorders and impairment, with reported 28-day mortality of 58% and 2-year mortality of 71% [13].

13. Conclusion

Heat stork is a severe temperature-related illness, an acute medical illness, and a medical emergency. It is more common in females than males. Heat stroke is defined as a core body temperature of more than 40^0c with hot and dry skin associated with central nervous system dysfunction. Typically divided into exertional and non-exertional heat stroke. Heat stroke generates a systemic inflammatory and coagulopathy response in combination with heat injury causes organ dysfunctions. Diagnosed with raised body temperature with clinical manifestation and signs and symptoms of organ dysfunction or failure.

In the management of heat stroke ABCDE approach should be followed. Early body temperature management in combination with organ-supportive care will improve the clinical outcome. The mortality from heat stroke ranges from 58 to 71%.

Author details

Gamal Al-Ameri[1*], Waleed Salem[2], Galal Alessai[2], Mohammed Hellboob[2], Mubarak Alhatemi[2], Umme Nashrah[3] and Nissar Shaikh[1]

1 Surgical Intensive Care, Hamad Medical Corporation, Doha, Qatar

2 Accident and Emergency Department, Hamad Medical Corporation, Doha, Qatar

3 Deccan College of Medical Science, Hyderabad, India

*Address all correspondence to: drgamalabdulrahman@gmail.com

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