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EC EMERGENCY MEDICINE AND CRITICAL CARE
Review Article

Heat Stroke Management in the E.D.: A Review Article

J Ashkar*

The Emergency Medicine Department, Hillel Yaffe Medical Center Hadera, Affiliated with Rappaport Faculty of Medicine, The Technion Haifa, The State of Israel

***Corresponding Author**: J Ashkar, Head of Emergency Medicine Department and Lecturer at the Technion's Faculty of Medicine, Hillel Yaffe Medical Center Hadera, Affiliated with Rappaport Faculty of Medicine, The Technion Haifa, The State of Israel.

Received: December 29, 2019; **Published:** January 06, 2020

Abstract

Heat stroke is a life-threatening illness characterized by an elevated core body temperature that rises above 40°C and central nervous system dysfunction that results in delirium, convulsions, or coma.

Despite adequate lowering of the body temperature and aggressive treatment, heat stroke is often fatal, and those who do survive may sustain permanent neurologic damage.

Keywords: *Heat Stroke; Convulsions*

Introduction

Thermoregulatory failure coupled with an exaggerated acute-phase response and possibly with altered expression of heat-shock proteins. multiorgan injury results from a complex interplay among the cytotoxic effect of the heat and the inflammatory and coagulation responses of the host.

Aim of the Study

The aim of this paper is to review Heat stroke, from causes, classifications and symptoms to treatment and prevention.

Review

Physiology

- Under normal physiologic conditions the hypothalamus is the body's thermostat.
- Thermosensors located in the skin, muscles, and spinal cord send information regarding the core body temperature → anterior hypothalamus → increase in the blood flow to the skin (as much as 8 L/min).
- Dilatation of the peripheral venous system.
- Stimulation of the sweat glands.

Path physiology

- In heat stroke the body produces or absorbs more heat than it can dissipate.
- The patient's temperature control system, which produces sweating to cool the body, stops working → temp can rise so high that it denatures proteins, destabilizes phospholipids and lipoproteins, and liquefies membrane lipids, leading to cardiovascular collapse, multiorgan failure, and, ultimately, death.

- Heat stroke may come on suddenly, but usually follows a less-threatening condition commonly referred to as heat exhaustion.
- With heat exhaustion, sweat does not evaporate as it should, possibly because of high humidity or too many layers of clothing. As a result, the body is not cooled properly. Signs include cool, moist, pale/flushed skin; heavy sweating; headache; nausea or vomiting; dizziness; exhaustion. Body temperature will be near normal.

Heat stroke types

1. **Classic:** Occurs without exertion. The elderly, infants and persons with chronic illnesses are at risk.
2. **Exertional heat stroke (EHS):** Among young, healthy adults who are engaging in rigorous exercise in the absence of heat acclimatization (e.g. athletes, construction workers, soldiers).

Glossary of terms

Condition	Definition
Heat wave	Three or more consecutive days during which the air temperature is >32.2°C
Heat stress	Perceived discomfort and physiological strain associated with exposure to a hot environment, especially during physical work
Heat stroke	Severe illness characterized by a core temperature >40°C and central nervous system abnormalities such as delirium, convulsions, or coma resulting from exposure to environmental heat (classic heat stroke) or strenuous physical exercise (exertional heat stroke)
Heat exhaustion	Mild-to-moderate illness due to water or salt depletion that results from exposure to high environmental heat or strenuous physical exercise; signs and symptoms include intense thirst, weakness, discomfort, anxiety, dizziness, fainting, and headache; core temperature may be normal, below normal, or slightly elevated (>37°C but <40°C)
Hyperthermia	A rise in body temperature above the hypothalamic set point when heat-dissipating mechanisms are impaired (by drugs or disease) or overwhelmed by external (environmental or induced) or internal (metabolic) heat
Multiorgan -dysfunction syndrome	Continuum of changes that occur in more than one organ system after an insult such as trauma, sepsis, or heat stroke

Table 1: *Glossary of terms.*

Causes

- Increased heat production
  - Increased metabolism - Infections, Sepsis, Encephalitis, Stimulant drugs, Thyroid storm, Drug withdrawal.
- Increased muscular activity
  - Exercise, Convulsions, Tetanus, Sympathomimetics.
- Decreased heat loss
  - Reduced sweating - Dermatologic diseases, Drugs, Burns.
  - Reduced CNS responses - Advanced age, Toddlers and infants, Alcohol, Barbiturates, Other sedatives.
- Reduced cardiovascular reserve - Elderly persons, Beta blockers, Calcium channel blockers, Diuretics.
- Factors Exogenous - High ambient temperatures, High ambient humidity.

Incidence

- Imprecise - under diagnosed, definition of heat-related death varies.
- From 1979 to 1997 7000 deaths in the US may increase with global warming worldwide increase in frequency and intensity of heat wave.
- During heat waves in urban areas in the US- 17.6 to 26.5 cases per 100,000 population.

Heat-shock response

- Cells response to sudden heating producing heat-shock proteins.
- Gene transcription control.
- Induce a transient state of tolerance to a second, otherwise lethal, stage of heat stress, allowing the cell to survive.
- Blocking the synthesis of heat-shock proteins renders the cells extremely sensitive to a minor degree of heat Heat-shock proteins protect cells.
- Bind to partially folded or misfolded proteins, thus preventing their irreversible denaturation.
- Act as central regulators of the baroreceptor-reflex response during severe heat stress, abating hypotension and bradycardia and conferring cardiovascular protection.

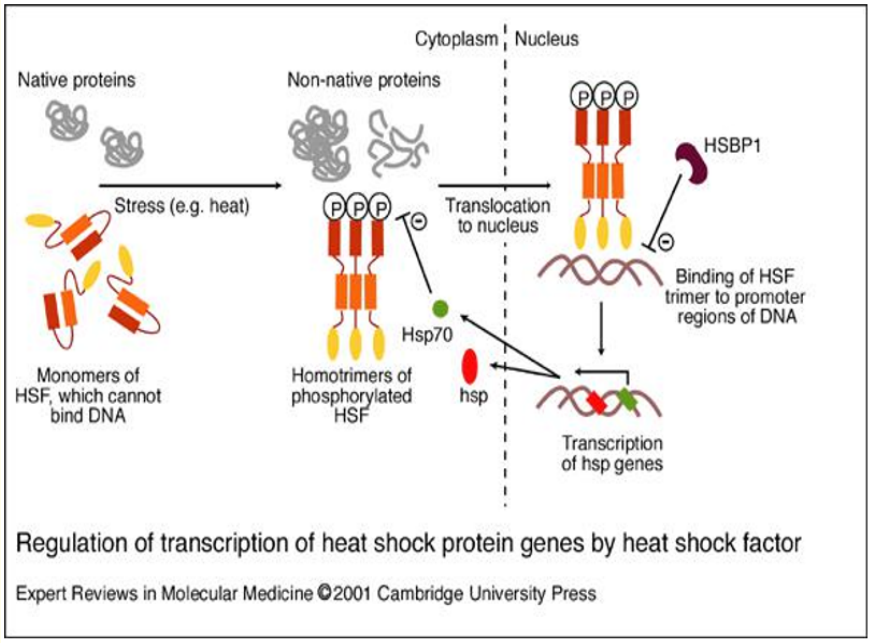

Figure 1

From heat stress to heat stroke

- Thermoregulatory failure.
- Exaggeration of the acute-phase response.
- Alteration of heat-shock response.

Clinical manifestations

- Vital signs:
  - Hyperthermia > 40°C (PR).
  - Tachycardia > 130 bpm.
  - Hypotension is common (vasodilation, dehydration, myocardial damage).
  - Tachypnea and hyperventilation → pco_2 < 20 mmHg.
- CNS:
  - Warning symptoms: Headache, dizziness, and weakness.
  - Confusion to LOC.
  - Seizure, status epilepticus.
- Transient neurologic symptoms - difficulty speaking, impaired concentration, irritability, may persist for several weeks.
- In some cases, permanent damage occurs, the most commonly cerebellar syndrome: gait, articulation, hyperreflexia, and nystagmus.

Laboratory at the time of admission

1. ABG: Respiratory alkalosis and metabolic acidosis due to lactic acidosis.
2. Glucometer: Hypoglycemia
3. LIVER: AST, ALT (peak at 48 hours)
   - If rhabdomyolysis: hypocalcemia, hypomagnesemia, hyperphosphatemia, hyperkalemia.
4. PT, PTT - DIC.
5. CBC - (inc. WBC, dec. PLT)

Imaging

1. CT
2. Chest x- ray (atelectasis, pneumonia, pulmonary infarction, or pulmonary edema).

Treatment

- Oxygen
- External cooling
- Powerful fans
- J.V. saline if BP is low
- Foley catheter to monitor urine output
- Nasogastric tube to monitor for GI bleeding
- Benzodiazepines- for Agitation, shivering, convulsions- (if necessary, barbiturates)
- Treatment of rhabdomyolysis: Infusion of large amounts of i.v fluids, alkalinization of the urine, mannitol.

Discussion

Heat stroke is a preventable illness!!!

- Acclimatize to heat
- Schedule outdoor activities during cooler times of the day

ADAM.

TABLE 3. MANAGEMENT OF HEAT STROKE.*

CONDITION	INTERVENTION	GOAL
Out of hospital		
Heat stress (due to heat wave, summer heat, or strenuous exercise), with changes in mental status (anxiety, delirium, seizures, or coma)	Measure the patient's core temperature (with a rectal probe)	Diagnose heat stroke†
	If the core temperature is >40°C, move the patient to a cooler place, remove his or her clothing, and initiate external cooling‡: cold packs on the neck, axillae, and groin; continuous fanning (or opening of the ambulance windows); and spraying of the skin with water at 25°C to 30°C	Lower the core temperature to <39.4°C, promote cooling by conduction, and promote cooling by evaporation
	Position an unconscious patient on his or her side and clear the airway	Minimize the risk of aspiration
	Administer oxygen at 4 liters/min	Increase arterial oxygen saturation to >90%
	Give isotonic crystalloid (normal saline)	Provide volume expansion
	Rapidly transfer the patient to an emergency department	
In hospital		
Cooling period	Confirm diagnosis with thermometer calibrated to measure high temperatures (40°C to 47°C)	
Hyperthermia	Monitor the rectal and skin temperatures; continue cooling	Keep rectal temperature <39.4°C§ and skin temperature 30°C–33°C
Seizures	Give benzodiazepines	Control seizures
Respiratory failure	Consider elective intubation (for impaired gag and cough reflexes or deterioration of respiratory function)	Protect airway and augment oxygenation (arterial oxygen saturation >90%)
Hypotension¶	Administer fluids for volume expansion, consider vasopressors, and consider monitoring central venous pressure	Increase mean arterial pressure to >60 mm Hg and restore organ perfusion and tissue oxygenation
Rhabdomyolysis	Expand volume with normal saline and administer intravenous furosemide, mannitol, and sodium bicarbonate	Prevent myoglobin-induced renal injury: promote renal blood flow, diuresis, and alkalization of urine
	Monitor serum potassium and calcium levels and treat hyperkalemia	Prevent life-threatening cardiac arrhythmia
After cooling	Supportive therapy	Recovery of organ function
Multiorgan dysfunction		

Figure 2

- Reduce level of physical activity
- Drink additional water
- Consume salty foods and increase the amount of time spent in air-conditioned environments.
- Automobiles should be locked, and children should never be left unattended in an automobile during hot weather [1-5].

Conclusion

On the basis of our understanding of the pathophysiology of heat stroke, we propose an alternative definition of this condition: a form of hyperthermia associated with a systemic inflammatory response leading to a syndrome of multiorgan dysfunction in which encephalopathy predominates.

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Volume 4 Issue 2 February 2020