Medi Quest BRS Hospital

A monthly News letter from BRS Hospital

HEAT RELATED ILLNESSES - PART 1 HEAT STROKE

Dr Praveen Balachandran

MD Int Medicine Asst Professor SRM Medical College Hospital Kattankalathur Visiting Consultant BRS Hospital Dr S Ramesh MD DCH

Consultant Pediatrician BRS Hospital

Price Rs. 5/- Only

April - 2025

Medi - 19

Ouest - 04

Yearly Subscription

Rs 50/- only

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Editors

Dr.B.Madhusudhan,
MS.MCh., DNB(Plastic)

Dr.S.Ramesh, MD, DCh

28,Cathedral garden Rd, Nungambakkam, Chennai - 600 034. Phone: 044 - 61434250 044 - 61434230

Email:

brsmediquest@gmail.com

Web:

www.brshospital.com

DEFINITION

Heatstroke is the most hazardous condition in a spectrum of illnesses progressing from heat exhaustion to heatstroke, in which a shared finding is hyperthermia (i.e., the rise in core body temperature when heat accumulation overrides heat dissipation during exercise or exposure to environmental heat stress). Clinically, heatstroke is characterized by central nervous system (CNS) dysfunction, multiorgan failure, and extreme hyperthermia (usually >40.5°C or 104.9°F)

CLASSIFICATION

Heatstroke may be categorized as either classic (passive) or exertional.

Classic heatstroke is due to exposure to environmental heat and poor heat-dissipation mechanisms. Classic heatstroke frequently occurs as an epidemic among elderly persons whose ability to adjust physiologically to heat stress has become compromised, chronically ill persons, and those who cannot care for themselves. Prepubertal children are also regarded as a population at risk. Children's susceptibility to classic heatstroke is attributed to a high ratio of surface area to mass, an underdeveloped thermoregulatory system, small blood volume relative to body size, and a low sweating

rate.

In infants, a major risk factor for death during hot weather is confinement in a closed car, where death can occur within a few hours

Exertional heatstroke is associated with physical exercise and results when excessive production of metabolic heat overwhelms physiological heat-loss mechanisms. Exertional heatstroke is a medical emergency, sporadic in nature, and directly related to strenuous physical activity. Exertional heatstroke can occur even within the first 60 minutes of exertion and may be triggered without exposure to high ambient temperatures.

PATHOGENESIS

The primary pathogenic mechanism of heat stroke involves transition from a compensable thermoregulatory phase (in which heat loss exceeds heat gain) to a noncompensable phase (in which heat gain is greater than heat loss), when cardiac output is insufficient to cope with the high thermoregulatory needs.

PATHOPHYSIOLOGY

Heat stress initiates a thermoregulatory response to increased cardiac output and redistribution of blood flow. When central venous pressure begins to decrease substantially, core temperature begins to increase rapidly and becomes noncompensable. The thermoregulatory failure aggravates pathophysio-logical processes at the cellular level, including an inflammatory reaction, and multiorgan failure occurs as a result of the combination of high body temperature and circulatory collapse, and ultimately is expressed as heatstroke.

RISK FACTORS OF HEAT STROKE

CLASSIC HEAT STROKE

Weather-Heat waves, with successive hot days and nights

Physiological factors- Cardiovascular insufficiency impeding normal cardiovascular adjustments to heat stress

Social factors- Social isolation, unventilated and nonair-conditioned living space

Underlying illness- Exacerbation of mental, cardiovascular, cerebrovascular, and pulmonary illnesses by exposure to heat stress

Medication- Beta-blockers, diuretics, calciumchannel blockers, laxatives, salicylates, thyroid agonists, tricyclic antidepressants, SSRIs

EXERTIONAL HEAT STROKE

Acquired factors - Low physical fitness, over weight

Viral or bacterial infection

Functional - familial anhydrosis, ectodermal dysplasia

Drug abuse-Amphetamines, alcohol

DIAGNOSIS

The diagnosis of heatstroke is largely clinical, based primarily on the triad of hyperthermia, neurologic abnormalities, and recent exposure to hot weather (in the classic form) or physical exertion (in the exertional form). Tachycardia, tachypnea, and hypotension are common. Profuse sweating and wet skin are typical of exertional heatstroke, whereas in classic heatstroke, the skin is usually dry, reflecting the characteristic decrease in the sweat-gland response and output in elderly people under heat stress.

CLINICAL FEATURE AND COMPLICATION

The disorder has three phases

Hyperthermic - neurologic acute phase
Hematologic - enzymatic phase (peaking 24 to 48

hours after the event)

Renal - hepatic phase (if clinical symptoms are sustained for 96 hours or longer).

Adequate measurement of core (rectal) temperature is critical in persons who may have heatstroke.

CNS disturbances - Behavioral changes, confusion, delirium, dizziness, weakness, agitation, slurred speech, nausea, and vomiting. Seizures and sphincter incontinence may occur in severe cases.

Multiorgan system dysfunction and failure (more pronounced in exertional heatstroke than in classic heatstroke) may peak within 24 to 48 hours.

COMPLICATION

Alteration in consciousness to DIC, acute respiratory distress syndrome, and acute renal, cardiac, and hepatic dysfunction and failure. Rhabdomyolysis, although not pathognomonic, is typical of exertional heatstroke

To be continued next issue...

Advance copy of Heat Related Illnesses Part II covering management available on website

http://www.brshospital.com/BRS-Mediquest.php









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No.28, Cathedral Garden Road, Nungambakkam, Chennai - 600 034.

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www.brshospital.com

: care@brshospital.com

Owned and Published by Dr. Madhusudhan 28, Cathedral Garden Road, Chennai - 34. Printed by S. Baktha at Dhevi Suganth Printers 52, Jani Batcha Lane, Royapettah, Chennai -14. Publication on: Final week of every month posted on 29.04.2025