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Editorial

Heat Stroke and Related Health Hazards: Bangladesh Perspective

A.H.M. Feroz[1]
Professor and Head, Department of Medicine, Monno Medical College, Manikganj, Bangladesh

Heat stroke is a life-threatening condition that necessitates neurocritical care. Heat stroke results from exposure to a high environmental temperature or from strenuous exercise. Between 2006 and 2010, there were at least 3,332 deaths attributed to heat stroke in the USA.[1] The 28-day and 2-year mortality rates for heat stroke have been reported at 58% and 71%, respectively.[2] Additionally, climate change is expected to increase the number of heat stroke deaths. By the 2050s, heat stroke-related deaths are projected to be nearly 2.5 times the current annual average of approximately 2,000 deaths.[1] Data on the incidence of heat stroke are imprecise because this illness is underdiagnosed and because the definition of heat-related death varies.[3] In an epidemiologic study during heat waves in urban areas in the United States, the incidence of heat stroke varied from 17.6 to 26.5 cases per 100,000 population.[3] In Saudi Arabia, the incidence varies seasonally, from 22 to 250 cases per 100,000 population. The crude mortality rate associated with heat stroke in Saudi Arabia is estimated at 50 percent. The incidence of heat exhaustion in Saudi Arabia, in contrast, ranges from 450 to more than 1800 cases per 100,000 population.[4] Heat stroke is defined by patient symptoms at the time of clinical admission, including profound central nervous system (CNS) abnormalities like delirium, seizures, coma and severe hyperthermia like core temperature typically but not always above 40°C.[5] 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. Recent epidemiological studies indicate that multi-organ system dysfunction can continue to manifest in patients following clinical treatment, increasing the risk of mortality during the subsequent months and years of recovery.[2] Preexisting conditions, such as mental illness, alcoholism, or drug use like diuretics, anticholinergic may compromise an individual's physiological adjustments to heat stress and increase the incidence of passive heat stroke. Athletes like marathon runners, race car drivers, occupational workers like fire fighters, agricultural workers and military personnel are highly motivated populations at risk for exertional heat stroke while performing strenuous physical work or exercise in temperature or hot climates. A recent epidemiological study identified a variety of factors that are associated with increased incidence of exertional heat illness, including sex (women are more than men), geographic region of origin (Northern is predominant than Southern states), and race/ethnicity (Caucasian are more than African American).[6] The incidence of exertional heat stroke is influenced by a multitude of factors, including pre-existing illness, drug use like alcohol, amphetamines, ecstasy and wearing protective clothing like uniforms in athletes that limits heat dissipation. The inability to properly anticipate, diagnose, and treat the long-term consequences of heat stroke is a significant limitation in modern medicine, reflecting our limited understanding of the pathophysiological mechanisms mediating tissue injury.

Hyperthermia due to passive heat exposure facilitates the leakage of endotoxin from the intestinal mucosa into the systemic circulation and the movement of interleukin IL-1 and IL-6 proteins from muscles into the bloodstream. This results in excessive activation of leukocytes and endothelial cells, leading to the release of various cytokines and high-mobility group box [1] protein (HMGB[1]), a prototypic alarming signaling tissue and cellular damage. These processes collectively trigger the systemic inflammatory response syndrome (SIRS). The inflammatory and coagulation responses to heat stroke, combined with the direct cytotoxic effects of heat, injure the vascular endothelium, causing microthrombosis. Platelet counts decrease due to micro thrombosis, secondary platelet consumption, and hyperthermia -induced platelet aggregation.

Correspondence: Prof. Dr. A.H.M. Feroz, Professor and Head, Department of Medicine, Monno Medical College, Gilondo, Manikganj, Bangladesh. Email: ahmferoz5@gmail.com Cell no: +8801817059292

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Heat stroke also suppresses platelet release from bone marrow because megakaryocytes are susceptible to high temperatures. Consequently, heat stroke-induced coagulation activation and fibrin formation clinically manifest as disseminated intravascular coagulation (DIC).[5] Heat is a leading cause of natural-hazard-related deaths in the United States, as evidenced by the significant morbidity and mortality associated with recent heat waves. Recent research has shown that heat stroke survivors have a substantially elevated 30-year mortality rate compared to individuals who have never experienced heat stroke. Despite this, our knowledge of the pathophysiology of heat stroke and the mechanisms of the systemic inflammatory response syndrome (SIRS) that predispose individuals to morbidity and mortality remains severely limited.

From 1961 to 2020, Bangladesh experienced an average temperature increase of 0.13°C per decade and a relative humidity rise of 0.3% per decade, leading to a rapid increase in the Discomfort Index (DI) by 0.13°C per decade, Heat Days (HD) by 0.22°C per decade, and Wet-Bulb Temperature (WBT) by 0.17°C per decade.[6] These increases were more pronounced in coastal regions, where thermal stress was already high. The rapid temperature rise significantly contributed to the increase in annual and monsoon DI, HD, and WBT, while rising relative humidity drove the increase in these indices during the pre-monsoon season.[7] The study also revealed a sharp rise in severe DI or dangerous HD days, with the number of such days tripling in the densely populated city of Dhaka and increasing twelvefold in Sylhet over the decades.[6]

Currently, there is a lack of data supporting the efficacy of existing clinical treatments, highlighting the urgent need for more effective therapeutics. The use of novel biotechnologies, including radiotelemetry, genomic, and proteomic analyses, will be crucial in advancing our understanding of heat stroke pathophysiology. These technologies, combined with innovative in vivo, in vitro, and in silico models, will be essential for enhancing our understanding of SIRS and developing new strategies to reduce the morbidity and mortality associated with heat stroke.[7]

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