IJBPAS, April, 2023, 12(4): 1417-1421

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

EXERTIONAL HEAT INJURY RESULTING IN MULTIORGAN FAILURE INVOLVING THE PANCREAS, LIVER AND GASTROINTESTINAL TRACT

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Received 8th May 2022; Revised 16th June 2022; Accepted 27th Aug. 2022; Available online 1st April 2023

https://doi.org/10.31032/IJBPAS/2023/12.4.7001

ABSTRACT

Due to internal heat produced in response to strenuous physical activity, external heat illness ranges from heat exhaustion to heat injury and heat stroke. Here we describe a case of multiorgan dysfunction after a marathon with average body temperature, i.e. exertional heat injury, the intermediate spectrum of exertional heat illness. A 20-year male had a transient loss of consciousness after the marathon recovered but had episodes of vomiting and altered sensorium the next day. Urine was cola coloured, and he gradually became anuric. Blood urea nitrogen, creatinine, liver enzymes and creatinine kinase were elevated and hepatic dysfunction worsened. Noncontrast computerized tomography showed a bulky pancreas. The patient developed respiratory distress and had grade II gastrointestinal injury. He succumbed to his illness after 19 days. Hence, exertional heat injury is a concept separate from heatstroke. Genetic susceptibility and lack of expression of heat shock protein may contribute to multi-organ dysfunction.

Keywords: Exertional heat illness, Intensive Care Unit, Acute Kidney Injury, Heat Shock Protein

INTRODUCTION

Heat stress is defined as discomfort and physiological strain on exposure to a warm and humid environment, especially during physical work. This stress response, when not compensated, progresses to heatstroke and, if associated with vigorous physical exercise, is called exertional heatstroke. The spectrum of physiological derangements associated with heat stress is quite large, but most reported cases of multi-organ dysfunction are only associated with heatstroke **[1]**. We present a case of exertional heat stress resulting in rhabdomyolysis, acute kidney injury (AKI), hepatic dysfunction, pancreatitis, and acute gastrointestinal injury.

Case Report

A 20 yr old male had gone for recruitment in security forces where he participated in the marathon. After the marathon, he had a loss of consciousness transiently for 5 minutes. There was no report of any hyperthermia episode. He was observed by the physician at the site for 2 hours, improved, became fully conscious and was sent home. The following day he had episodes of vomiting with altered mentation and was admitted to the hospital. On catheterisation of the bladder, cola coloured urine came. He was conscious but drowsy, hemodynamically stable and had no respiratory distress. Laboratory investigations showed raised blood urea nitrogen, creatinine and bilirubin. The creatinine kinase was elevated to more than five times the upper normal level. There was three times increase in amylase and lipase. The next day patient became anuric and was started on dialysis. On day 9 of his illness patient developed fever and respiratory distress, was intubated and kept on mechanical ventilation.

The patient was referred to our ICU on day 11 of his illness. He was deeply sedated, intubated on mechanical ventilation, hemodynamically stable and oliguric. His laboratory parameters on admission were hemoglobin 12 g/dl, total leukocyte count (TLC) 8,000 (×109/L) and platelet count of 165 (×109/L). His creatinine was 7.5 mg/dL, total bilirubin was 10.1mg/dl, and elevated liver enzymes. He had acute gastrointestinal injury grade II, high gastric residual volumes, and started post pyloric feeding. Viral markers for hepatitis were negative, and there was no evidence of any obstructive pathology. He had edematous pancreatitis evident from the CT scan but no necrosis or any intra-abdominal collections. There was a progressive worsening in liver function according to the laboratory parameter. On

day 17 of his illness, he developed ventilator-associated pneumonia (VAP) and severe septic shock. He succumbed to his illness on day 19 of his illness **(Figure 1)**.

Discussion

A life-threatening condition with hyperthermia and profound encephalopathy (delirium, agitation, stupor, seizures, coma) defines heatstroke. There is an increasing incidence worldwide due to global warming. Classic form occurs with passive exposure (very young or elderly), and Exertional form is due to intense exercise (young, healthy adults). It is the most severe form of heat illness and is most commonly described in the literature **[2]**.

The heat illness disease consists of heat exhaustion, heat injury & heat stroke. Heat fatigue is defined as mild to moderate illness characterized by the failure to maintain cardiac output; moderate (>38.5ºC; 101ºF) to high (>40.0ºC; 104ºF) core body temperature. Heat injury is defined as moderate to severe illness with organ and/or tissue damage; high core body temperature, but usually not more significant than 40ºC. It is the intermediate form of heat illness. Our patient belonged to this category. He never had very high body temperatures, yet he had a severe multi-organ failure. This group of patients are reported less in the literature **[3]**.

The exertional heat illness and exertional rhabdomyolysis are part of the same disease profile. In exertional rhabdomyolysis, muscles are involved primarily due to overuse strain and secondarily due to severe inflammatory response syndrome (SIRS), as seen in sepsis. There is the extensive release of cytokines and inflammatory mediators which affect various organ systems **[4]**.

The question that has been most engaging in the development of the heat illness continuum is why some people develop this severe reaction to heat or exertion while others do not. The severe stress response of exertional heat injury results in heat shock proteins (HSP). It is a profoundly conserved family of proteins in the evolutionary method with molecular sizes ranging from 10 to 110 kDa. In molecular biology, it's a chaperone that helps in the synthesis, folding, transport and degradation of proteins and is induced by stressful conditions **[5]**.

Transgenic knock-in mice overexpressing HSP72 has not been shown to have expedited tissue injury recovery by lessening NF-κB signalling. In transgenic mice that had knock-out for HSP70, there was an increased incidence of pancreatitis. What appears to predispose specific individuals to heat injury and multiorgan dysfunction is the lack of expression of heat shock protein. This has

not yet been studied in humans and may add to our knowledge in the management and prevention of this deadly disease entity.

Our patient had a rapid onset of acute kidney injury with altered sensorium within one day of disease onset. He also developed edematous pancreatitis and hepatic dysfunction. This signified an extensive inflammatory response as edematous pancreatitis developing in a patient of exertional rhabdomyolysis and exertional heat injury is not reported in the literature. The development of pancreatitis could be explained by the heat shock protein theory that has shown to have a protective response in patients of acute stress.

This patient had acute gastrointestinal (GI) injury grade II and was given nasojejunal feeds due to intolerance to nasogastric feeding. The severe inflammatory response possibly resulted in lowering the immunity in this patient. This resulted in the development of VAP, which resulted in severe septic shock, which did not respond to management and ultimately resulted in his death.

Our case describes an exertional heat injury that resulted in multiorgan failure, which has not been reported before in literature. The reason being genetic susceptibility which predisposed this individual to develop such a severe response to heat and exertion. Our case highlights the need to investigate the entity of heat injury separate from heatstroke and look at possible genetic predisposition.

Patient Consent: A written consent was approved from the patient family that the following case can be published and can be used as a reference study.

Abbreviations:

ICU: Intensive Care Unit

AKI: Acute Kidney Injury

CT: Computerised Tomography

TLC: Total Leucocyte Count

k DA: Kilo Dalton

VAP: Ventilator-Associated Pneumonia

SIRS: Systemic Inflammatory Response Syndrome

HSP: Heat Shock Protein

Conflict of Interest: Nil

Funding Source: Nil

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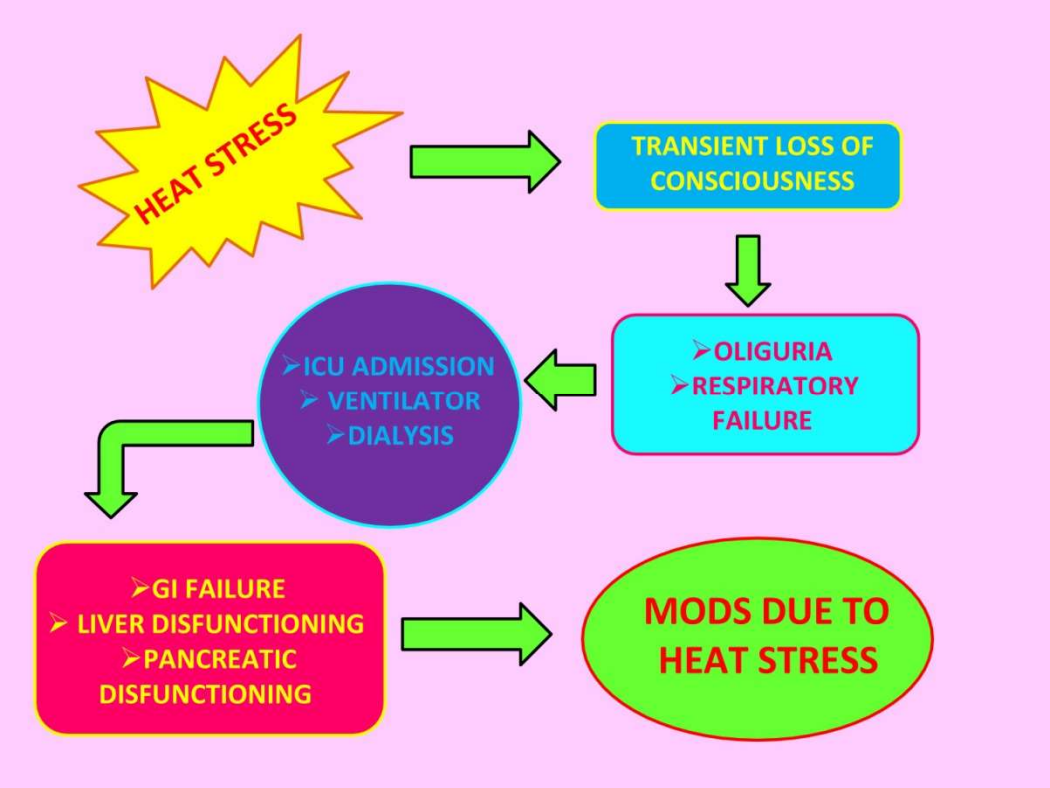

Figure 1: Diagrammatic representation of the entire case report