HEATSTROKE IN THE "MATRA" HEALTH PERSPECTIVE

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ABSTRACT

Heatstroke is a life-threatening condition if not recognized and treated effectively. It is classified into two types: exertion heatstroke (EHS) and non-exercise heatstroke (NEHS) or classic heatstroke. In 2017, for example, there were 464 heatstroke cases and 1,699 heat exhaustion cases. The overall gross incidence rates of heatstroke and heat exhaustion were 0.38 cases and 1.41 cases per 1,000 people per year, respectively. Both diseases are more common among younger members, Asian/Pacific Islanders, Marine Corps and Army personnel, and those in combat-related occupations.

Classical heatstroke can be caused by a variety of factors, including the weather, physiology, social factors, and underlying disease, whereas exertion can be triggered by a variety of other factors, including social (excessive exercise motivation), functional issues, underlying disease, and medication use. Heatstroke can harm multiple organs because of its pathophysiology, which includes compensable and non-compensable effects. The triad of hyperthermia, neurological abnormalities, and exposure to hot or cold weather or physical activity is used to make the diagnosis. The first task of field management is to maintain a constant internal temperature. Large quantities of water and fanning can be used in military or desert environments to achieve cooling rates of 0.10 oC per minute, even if ice is not available. Field planning and adherence to experience-based preventive measures can significantly reduce the incidence of exhaustional heatstroke.

INTRODUCTION

Hyperthermia is a symptom of heatstroke, which is the most dangerous condition in a continuum of disease that begins with heat exhaustion and ends with heatstroke (i.e., an increase in body temperature when exposed to heat and no heat dissipation occurs during exercise and exposure to continuous environmental heat stress). Extreme hyperthermia (often > 40.5 oC) and dysfunction of the central nervous system (CNS) are the hallmarks of clinical heatstroke. Exertion Heat Stroke (EHS) and NEHS (Non-Exertion Heat Stroke) are two distinct types of heatstroke. EHS occurs when someone engages in strenuous physical activity for a long period of time in a hot environment; NEHS occurs when someone is elderly, chronically ill, or very young[2].

In the most severe form of heat injury, heatstroke, mortality, and morbidity rates can be extremely high if not treated quickly[2]. Heatstroke is reported to have a mortality rate of between 10 and 50%. In addition, 7–20% of the survivors are left with persistent neurological damage.[3]. Between 2006 and 2010, Americans suffered 3,332 deaths as a result of heatstroke. A 28-day mortality rate of 58% and a two-year mortality rate of 72% have been reported for heatstroke[4].

Heatstroke and heat exhaustion were diagnosed in 464 and 1,699 cases, respectively in 2017. An estimated 0.38 and 1.41 deaths per 1,000 person-years were caused by heatstroke or exhaustion, respectively. Over the past year, service members under the age of 19 and residents of Asia/Pacific Islands, Marine Corps/Army personnel and combat-specific occupations were the most likely groups to suffer from heatstroke. Heatstroke rates were significantly higher among men than women. In contrast, the rate of heat exhaustion among women was similar to that amone men. During 2013-2017, a total of 359 heat illnesses were documented among service members in Iraq and Afghanistan; 8.6% (n=31) diagnosed as heatstroke[5].

Exercise in a hot environment can result in a heat injury that can lead to heatstroke. The body's temperature regulation system and cardiovascular system may be severely taxed if exercise is performed in such a hostile environment. For this reason, it is critical to know how the body responds to extreme environmental conditions in order to prevent injury to those who exercise or compete in extreme conditions[2].

Hemorrhagic shock occurs when the body's thermoregulatory system fails to keep hemostasis stable. Extrinsic factors, such as extreme temperatures, strenuous physical activity, and environmental conditions, are to blame for the disruption of hemostasis. An athlete's or

Kostrad's performance in exercises or competitions can be harmed if their body temperature rises, both physiologically and psychologically[7].

In the Military Health System, the most serious heat-related illnesses are considered notifiable medical events, including heatstroke and heat exhaustion. All cases of heat illness requiring medical intervention or resulting in a change in duty status should be reported. Commanders, small unit leaders, training cadres, and medical support personnel must ensure that the military personnel they supervise and support are informed about the risks, precautions, early signs and symptoms, and first responder actions associated with heatstroke.[5] Preventing heatstroke is a good idea because it can lead to lower morbidity and mortality rates and not stop a Kostrad personnel or an athlete from training. This can be done by planning the right field, assessing the risks, and managing the field properly, as well as getting the right treatment.

DISCUSSION

The Concept of Heatstroke

To date, there is no universally accepted definition of heatstroke. The Bouchama definition of heatstroke is the most widely accepted definition. For Bouchama, a heatstroke occurs when the core body temperature rises to or exceeds 40^0C, which is accompanied by hot, dry skin and abnormalities of the central nervous system such as delirium, seizures, or coma. Strenuous exercise or exposure to high temperatures can cause heatstroke. The pathophysiology of heatstroke, as proposed by Bouchama, suggests that it is a form of hyperthermia associated with a systemic inflammatory response that leads to a multiorgan dysfunction syndrome, particularly encephalopathy[8].

The Cause of Risk Assessment Heatstroke

The following are some of the risk factors for heatstroke quoted from Yorem (2019).

Table 1. Risk Factors Underlying Heatstroke* [1]

Heatstroke Types & Risk Factors	
Classic heatstroke	**Description**
Weather	A heat wave with consecutive hot days and nights.
Physiological Factor	Cardiovascular insufficiency that impedes normal cardiovascular adaptation to heat stress: inability to maintain acceptable stroke volume in heat, inadequate peripheral vasodilation due to structural changes and nitric oxide-mediated vasodilation mechanisms, decreased capillary density and quality of skin microcirculation, decreased sweating and sweating glandural secretation in response to heat stress.
Social Factor	Social isolation, unventilated and air-conditioned living space, inability to care for oneself, and confinement in bed.

Underlying Disease	Eksaserbasi penyakit mental, kardiovaskular, serebrovaskular, dan paru-paru dan multiple sclerosis oleh paparan stres panas.
Drugs	Beta-blockers, diuretics, calcium-channel blockers, laxatives, anticholinergic drugs, salicylates, thyroid agonists, benztropine, trifluoperazine, butyrophenones, α-agonists, monoamine oxidase inhibitors, sympathomimetic medications, tricyclic antidepressants, SSRIs.
Exertional Heatstroke	
Social Factor	Exessive motivation, and peer and coach pressure
Functional Factor	Low physical fitness (physical effort incompatible with physical fitness "killing workout"), lack of acclimatization (habituation to heat, low work efficiency, overweight (reduced skin area to mass ration and greater heat storage capacity in the body fat layer), protective clothing (reduces sweating efficiency).
Obtained Factor	Viral of bacterial infection (even if subclinical), dehydration, lack of sleep, sweat gland dysfunction, e.g., deep burns, skin scars > 40% of total body surface area).
Innate Factor	Chronic familial or idiopathic anhidrosis, ectodermal dysplasia.
Drug Abuse	Amphetamines and amphetamine-like agets (e.g., ephedra, MDMA, cocaine, PCP and LSD, synthetic stimulants of the cathinone class cathinone (e.g, α-PHP), alcohol.
*LSD denotes lysergic acid diethylamide, MDMA 3,4 methylenedioxymethamphetamine (ecstasy), PCP phencyclidine, α-PHP α-pyrrolidinohexanophenone, and SSRI selective serotonin-reuptake inhibitor.	

Heatstroke Classification

Heatstroke can be classified as either classic (passive) or activity/exertional heatstroke, depending on the underlying cause. In both cases, excessive body heat is the root cause, but the underlying mechanisms are different. Classic heatstroke is a result of prolonged exposure to high temperatures and inadequate heat dissipation mechanisms in the environment, whereas exertional heatstroke is a result of excessive metabolic heat production and physiological heat loss mechanisms during physical exercise[1].

Classic Heatstroke

Classic heatstroke is common in the elderly, the chronically ill, and people who are unable to take care of themselves because of their impaired physiological ability to adapt to heat stress[9]. Heat waves are being caused by an increase in global temperatures, and urbanization is another major extrinsic factor[10]. The elderly is more vulnerable to heat because of a number of intrinsic physiological, social, and medical risk factors[10]. As a result, many elderly patients with classic heatstroke are hospitalized or found dead within one or three days of reporting the onset of their symptoms. More than half of all elderly heatstroke deaths are preventable[10].

Prepubescent children are also at risk for the classic heatstroke because of a high surface area to mass ratio (leading to an increased rate of heat absorption), underveloped thermoregulatory system (impairing effective heat dissipation), a small blood volume relative

to body size (limitng potential heat conductance and increasing heat accumulation), and lower sweating rates (reduced heat dissipation potential through evaporation of sweat)[11]. Infants who are left in a hot car for long periods of time are the greatest risk of death during the hottest months of the year[12].

Exertional Heatstroke

As a medical emergency, heatstroke on exerction can occur at any time during strenuous physical activity. Athletes, workers (e.g., firefighters and agricultural workers), soldier, and others who engage in similar levels of exertion and exposure to the environment, such as these, may be affected. Even in the first 60 minutes of exertion, heatstroke can occur and can be triggred even if there is no direct exposure to high ambient temperatures[13].

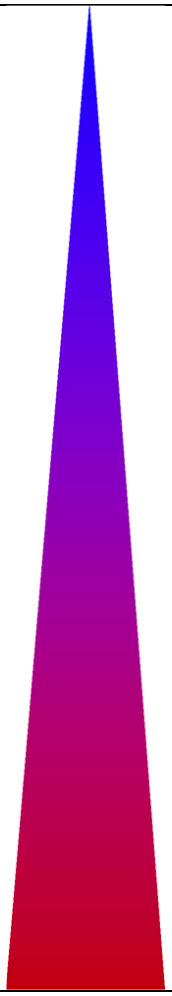
Excessive peer and coach pressure and motivation are the main causes of exertional heatstroke. These factors push athletes to the limit of their physical capabilities. As a result, some people are more susceptible to heatstroke because of functional and acquired factors, as well as some inherited conditions that make them more vulnerable to heat. Psychedelic trance music festivals and parties frequently feature alcohol and drug abuse, which increases the metabolic response to energetic music and increases the risk of heatstroke among participants. The use of amphetamines and other stimultants by athletes increases their risk of heatstroke.[14]

Heatstroke on exertion is frequently misdiagnosed, obscuring the true incidence of the condition (e.g., dehydration or heat exhaustion). Over the past decade, heatstroke deaths and morbidity among high school football players and military personnel have steadily increased, according to epidemiological surveys. However, the mortality rate is low (<5%) because heatstroke on exertion most commonly affects healthy young people and is usually recognized and treated quickly.[5]

The Japanese Assocation for Acute Medicine (JAAM) has been compiling data on patients with heat-related illness (including heatstroke) since 2006, regardless of their core body temperature, through the national heat-related disease register in Japan. The JAAM has established and published criteria for disease related to heat, including heatstroke.[4]

Recommended Classification by Japaness Association of Acute Medicine Committee Related to Heatstroke"

Tabel 2. Japanese Association of Acute Medicine Heat Related Illness Classification 2015

	symtoms	Severity	Treatment	Classsification from clinical presentation
Stage 1 (first aid and observasi)	Dizziness, faintness, sligt yawning heavy sweating, muscle pain, stiff muscles (muscles cramps) Impaired consciousness is not observed (JCS = 0)		May be handled on site under normal conditions→ resting in a caal place, cooling the body surface, and orally supplying water and Na^+	Hear Cramp Heat Syncope
Stage II (Should be taken to a medical institution)	Headache, vomiting, fatigue, sinking feeling, and declined concentration and judgement (JCS <1)		Examination at a medical institution is necessary→ body temperature management, resting, and sufficiently supplying water and Na (by drip infusion if oral intake is difficult)	Heat exhaustion
Stage III (Inpatient hospital care)	Includes at least on of the following: (c) Central nervous system manifestation (impaired consciousness JCS >2, cerebellar symptoms, convulsive seizures) (H/K) hepatic/renal dyfunction (follow – up following admission to hospital, hepatic or renal impairment requiring inpatient hospital care (D) Coagulation disorder (diagnosed as DIC according to acute phase DIC diagnostic criteria (Japanese Association of Acute Medicine) → most severe of the three types		Inpatient hospital care (depending on the case, intensive care) is necessary → body temperature management (internal body cooling, intravascular cooling, etc. are carried out along with body surface cooling) Respiratory and circulatory care DIC treatment	Heat stroke

First aid can be conducted and patien is monitored only when Stage I symptoms gradually improve

The patient should immediately be taken to the hospital in the event when stage II symptoms occur or improvement in Stage I is not observed (assessed by others)

Whether or not is stage III is determined by ambulance staff or at examination/checkup after arrival at hospital

Japanese Association of Acute Medicine Heat-Related Illness criteria, DIC, (Disseminated Intravascular Coagulation), JCS (Japan Coma Scale) Classification Related Illness quoted from[4]

Pathophysiologi

The main pathogenic mechanism of heatstroke involves the transition from a compensated themoregulatory phase (where heat loss exceeds heat gain) to an uncompensated

phase (where heat gain is greater than heat loss). This occurs when the heart's ability to pump blood is insufficient to maintain a stable temperature.

There is a cytotoxic effect and an inflammatory response that happen because the body's core temperature rises. This causes a cycle that leads to multiorgan failure. The can be seen in the **Image 1** below.

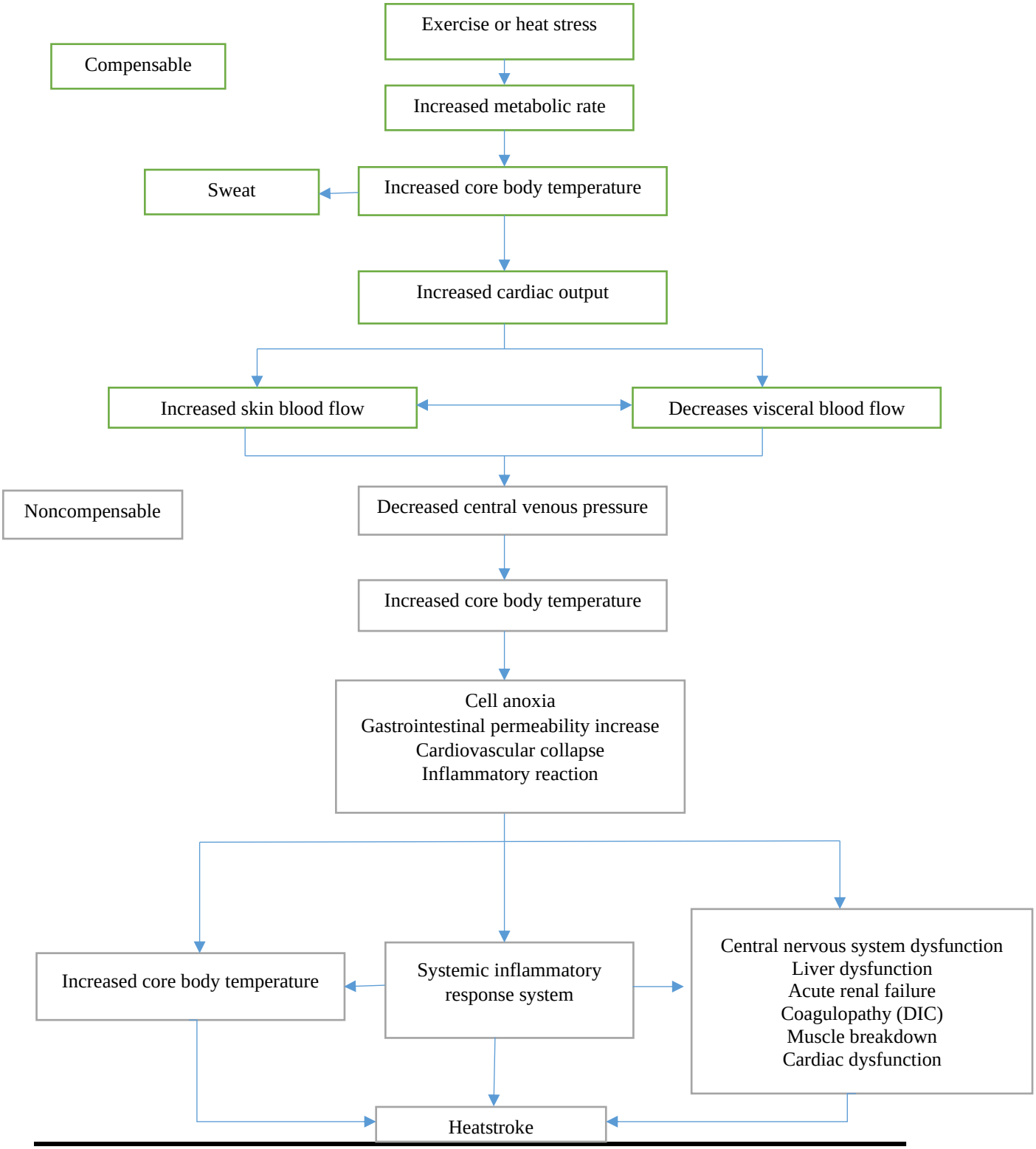


Image 1. Pathophysiology Path towards Heatstroke Quoted from[1]

The transition from a compensable to an uncompensated state of themoregulation is what causes heatstroke. In response to increased cardiac output and redistribution of blood flow, the body's themoregulatory system is activated. The core temperature rises rapidly and uncompensated when the central venous pressure drops significantly. When the body is unable to regulate its temperature, pathological processes, such as inflammation, get worse. This leads to multiorgan failure, which is what causes heatstroke. DIC indicates disseminated intravascular coagulation.

Inflammatory Response

Coordination of endothelial cell, leukocyte, and epithelium cell stress responses provides protection against tissue injury and promotes cell repair in the presence of hyperthermia. Inflammatory and anti-inflammatory cytokine levels in the blood and tissues are affected by heat shock protein molecules of the molecular chaperone family. Prolonged hyperthermia causes acute physiologic changes (such as hypoxemia, increased metabolic demands, and circulatory failure) and heat-related cytotoxic effects that directly increase, leading to dysregulation of the inflammatory reaction.[15]

The heatstroke-associated inflammatory response is similar to that of systemic inflammatory response syndrome (SIRS).[8] According to some research, SIRS is caused by the release of cytokines and high mobility group 1 box protein (HMGB1) by circulating messenger RNA, which then causes leukocytes and endothelial cells to become excessively activated. [16] Like septic shock, SIRS can lead to DIC, multiorgan failure, and death. Since heatstroke is characterized by an inflammatory response and multiple organ dysfunction, it is defined as "an extreme form of hyperthermia with an accompanying encephalopathy." 84% of patients admitted to the hospital for heatstroke on exertion were also diagnosed with SIRS, and their hospitalization was extended.[1]

As evidenced by clinical and experimental evidence, neutrophil activation in classic heatstroke serves as a link between inflammation and coagulation.[15] However, SIRS is often mistaken for heatstroke, which is why it is not commonly seen in the intensive care unit and does not appear to be a common cause of SIRS. It is critical to recognize the link between the

two disorders and get the right treatment as soon as possible to lessen the negative effects of SIRS.

Heatstroke causes gastrointestinal ischemia, which has a negative impact on cell viability and cell wall permeability in the intestines. Oxidative and nitrogenous stress can damage cell membranes, allowing endotoxins and other possible pathogens to leak into the bloodstream. This overloads the liver's ability to detoxify and causes endotoxemia, which can be dangerous.

Clinical Manifestation

Table 3. Epidemiologic and Clinical Features of Classic and Exertional Heatstroke[1]

Feature*	Classic Heatstroke	Exertional Heatstroke
Age group	Puberty and old age	Postprubertal and active, young age
Occurance	Epidemic (when heat waves occur)	Sporadic (occur yearly)
Concurrent Activity	Concurent ccurance	Settled and heavy
Health Status	Chronic disease	Common healthy people
Drugs	Often used (drug prescription)	Usually nothing is used (sometimes ergogenic aids, illegal drugs)
Mechanism	Poor environmental heat absorption and heat dissipation	Excessive heat production, which interferes with the heat loss mechanism
Sweating	Probably do not exist (dry skin))	Sometimes exist (wet skin)
CNS Disfunction	Common	Common
Acid-base Disturbance	Respiratory Alkalosis	Metabolic acidosis
Rhabdomyolisis	No	Sometimes exist
Liver Dysfunction	Light	Heavy
Renal failure	Uncommon (<5%)	Common (25-30%)
DIC	Light	Heavy
ARDS	Common	common
Creatinin kinase	Slightly increase	Subtantially increase
Calcium	Normal	Low (hypocalcemia)
Potassium	Normal	Rarely increase (hyperkalemia)
*ARDS denotes acute respiratory distress syndrome, CNS central nervous system, and DIC disseminated intravascular coagulation.		

The triad of hyperthermia, neurologic abnormalities, and recent exposure to hot weather (in the classic form) or physical activity are the primary diagnostic criteria for heatstroke (in the form of activity). Tachycardia, tachypnea, and hypotension are common. Exertion-induced heatstroke is marked by excessive sweating and moist skin, whereas classic heatstroke is marked by dry skin because of the characteristic decreased sweat gland response and output in elderly people. It is possible for the skin to appear reddened, reflecting excessive peripheral vasolidation, or pale, indicating vascular collapse.[1]

Laboratory

Laboratory tests reflect organ function which should be monitored systematically for at least 72 hours to avoid possible deterioration in clinical quality.[9] However, experimental

evidence suggests that this metric may not accurately reflect the disease's severity or long-term prognosis. Yorem (2019) argued that circulating biochemical markers such as HMGB1, neutrophil gelatinase-associated lipocalin (also known as 24p3, uterocalin, and lipocalin), 17 troponin Cardiac I, 72-hour urine protein ratio to urinary creatinine, histones, and peptide cryptdin 2 are being investigated (an intestinal alpha-defensin).[1,18] However, these biomarkers are preliminary and have not been tested or approved for clinical use.[1] According to Melinda (2019), routine blood, blood urea nitrogen, serum creatine, liver function, serum albumin, bilirubin, potassium, pH, and coagulation factors, as well as D-dimer fibrinogen which increased on day 4, were performed at the start of the ED patient.[6]

Heatstroke Care Plan

Preventing heatstroke is both more effective and less time consuming than treating it once it has occurred. Protective measures must be taken in hot weather and especially during heat waves in order to avoid classic heatstroke. In order to combat loneliness and isolation, some people choose to live in climate-controlled environments, use fans, take cold showers on a regular basis, reduce their physical activity, or all of the above.[9,19] Elderly people should also be inspected frequently by family members and social workers to ensure their well-being.

Heat exhaustion can be prevented to a great extent if people and organization follow tried-and-true preventive measures. It is important to adjust to changing environmental conditions, ensure that physical activity levels are matched to physical fitness levels, avoid training in the heat of the day, remove sweat-blocking equipment and clothing, stay hydrated during physical activity, and refrain from participating in physical activity if you have early signs of diseases.[1]

Field Management of Heatstroke

Temperature-related illness is treated symptomcatically and minimally. Preventing hyperthermia is the primary objective.

Body Temperature Control

Prognosis worsens if body temperature is above the threshold (>40.5%^{o}C). Indeed, rapid and effective cooling is the cornerstone of treatment and should only be delayed for essential cardiopulmonary resuscitation.[20] In order to minimize the risk of clinical deterioration after cooling, the safe target temperature is below 29^{o}C, (preferably 38.5^{o}C to 38.0^{o}C). Cooling at a rate faster than 0.10^{o}C per minute is safe and helps the prognosis for exertional heatstroke.[21] In

order to achieve a cooling rate of 0.20ºC to 0.35ºC per minute, but this method is rarely used. A cooling rate of about 0.10ºC per minute can be achieved by pouring large amounts of water over the body and fanning the patient with heatstroke in military or desert conditions where ice is not available. The following is a guideline for the management of heatstroke (Yorem, 2019).

Table 3. Heatstroke Management Guide

Management Guide	**Description**
Field Guide	
CPR	Comply with ACLS protocol; give oxygen at a rate of 4 liters/minute to increase oxygen saturation up to >90%.
Body Temperature	Monitor rectal temperature and perform cooling in cases of hyperthermia; for exertional heatstroke in cold water; for classic heatstrike, cooling by conductive or evaporative means.
Fluid	Give isotonic saline IV (1−2 liter/hour); dehydration is not really a problem.
Seizure Medicine	Give benzodiazepin IV till the seizure stops (5 mg and not over 20 mg).
Evacuation	For classic heatstroke, transfer to emergency unit care; for excessive heatstroke, transfer to emergency unit care after cooling to body temperature <39.0 °C.
Care in Emergency Center	
Pantau suhu tubuh	Monitor rectal intravesical temperature and cool to core temperature <38.0 °C; use cooling clothes or cold liquid (4°C, 1000 ml/30 minute) infused through a central catheter; toxic antipyretics should be avoided; has not been proven effective
Seizure Medicine	Give benzodiazepin IV (5 mg, repeating) or phenytoin IV (loading dose, 15-20 mg/kg in15 minute) till the seizures stop.
Laboratory Check	Perform CBS, urinalysis, blood culture, kidney function and liver function test (ALT, AST, amonia, INR); glucose test, electrolytes, arterial blood gases and acid-base balance, freeze function, CK, LDH, mioglobin, CRP.
Circulation Control	For circulation failure, give liquid (30 ml/kg), control CVP or control the invasive hemodynamics, maintain mean arterial pressure at > 65 mm Hg (or >75 mm Hg for elderly patient or those with hypertension issue), all with the aim of normal lactate levels and urine output >50 ml/kg/hour; vasopressors should be considered if fluid therapy fails.
Management Guide in Emergency Center	
General	Perform CPR as per ACLS protocol; ECMO can be used as needed.
	Monitor rectal, intravesical, or blood temperature; continue cooling to maintain core temperature at <38,0°C by infusion of cold liquid (4°C, 1000 ml/30 minute) via a central cathether or use extracorporeal blood cooling for resistant hyperthermia; toxic antipyretics should be avoided; dantrolene has not been proven effective
	Perform laboratory check: CBC, glucose, arterial blood gases and acid-base balance, clotting function, CK, LDH, liver function (ALT, AST, ammonia, INR) myoglobin, renal function, urinalysis, CRP, blood culture; repeat every 12 hours for the first 48 hours, then every 24 hours.
Hear failure	Perform CPR according to the ACLS protocol; perform invasive hemodynamic monitoring and echocardiography; for mild multiorgan failure, give IV dobutamine (1g/kg/minute, then 2-20g/kg/minute as needed) or IV milrinone (loading dose, 50 g/kg over 10 minutes, then 0.2-0, 75 g/kg/minute) IV adrenaline (1g/minute); for severe multiorgan failure, ECMO can be used as needed.
Acute Kidney Injury	Give crystalloid solution to maintain urine output >50ml/kg/hour; give IV furosemide (10-20 mg in patients without previous diuretic exposure; follow-up dose dependent on urine output); provide hemodialysis or CVVH in cases

	of volume overload, severe acidosis, hyperkalemia, or uremia; adjust the rate of fluid infusion according to blood pressure and urine output; monitor electrolytes and correct it as needed.
Ensefalopati & brain edema	For scores <8 on GCS, intubation and ventilation; for mild hyperventilation (Pco2, 34-36 mm Hg) give hypertonic saline 3% IV (initial dose, 100 ml/30 minute, then adjust patient's total body water to achieve an increase in sodium level of 12 mmol.day) or mannitol 20% IV (0.25-2 g/kg in 30 minutes); keep the head at an angle of 45 degrees, administer a sedative; patients with huperammonemia require hemofiltration or MARS therapy; conditions improve with cooling; consider monitoring ICP.
Rhabdomyolysis	Give IV fluid infusion, 1-2 liters/hour (aggressive fluid treatment within first hour), then 300 ml/hour' IV furosemide 910-20 mg in patients without previous diuretic treatment; follow-up dose dependent on urine output) in case of fluid overload; sodium bicarbonate, 30 mmol/hour (to achieve urine pH >6.5); myoglobinuria is expected; hypercalcemia and metabolic alkalosis (pH >7.5) should be avoided.
DIC and oheer coagulase disorders	For bleeding and thrombosis, give fresh frozen plasma (bolus dose, 10-15 ml/kg, then 200-400 ml according to the coagulation index); give cryoprecipitate (5-10 U each time) for fibrinogen levels <180 mg/dl; give platelet concentrate (infusion of one therapeutic dose) if the platelet count is <20 per mm3 or if there is bleeding and the platelet count is <50 per mm3; in patients with liver failure, consider PCC to achieve a target INR of 1.5; inject PCC dose according to INR and patient's body weight; avoid heparin; beware of hypothermia and metabolic acidosis.
ARDS	Perform intubation and mechanical ventilation; avoid excess fluid.
Liver failure	Monitor liver function and mental status for at least 4 days; provide supportive treatment: hemodynamic stability, N-acetycysteine IV (bolus dose, 150 mg/kg in 200 ml 5% glucose solution for 20 minutes, then 50 mg/kg in 500 ml 5% glucose solution for 4 hours, then 100 mg/kg in 1000 ml of 5% glucose solution for 16 hours); give 3% hypertonic saline IV or IV mannitor 90.25-2 g/kg over 30 minutes in 20% solution), hemofiltration, laxative (e.g., oral lactulose, 30 ml every 2 hours until diarrhea occurs), oral rifaximin (400 mg 3 times a day) in case of fulminant hepatic failure; liver transplantation is rarely needed, and there is no evidence that it is effective .
EKG Changes	Monitor continuously for possible arrhythmias; ECG changes are not specific
SIRS	Treat the same as sepsis; consider antibiotics

There are no hard and fast rules in the table above. Treatment should be tailored to the specific needs of each patient. Only 24 to 48 hours after the event can the full picture of organ failure be seen. As a result, at least 96 hours of follow-up should be required.

Conclusion

If heatstroke is not diagnosed and treated effectively, it can be fatal. Preventing classifc and on-the-go heatstroke can be as simple as avoiding strenuous activity in hot environments, reducing exposure to heat stress, and addressing socioeconomic issues that increase risk. Our knowledge of risk assessment, field management, planning, and pathophysiology of heatstroke and mechanism-based treatment approaches is still in its infancy. Future research is likely to focus on three areas: identifying genetic traits that might reduce a person's ability to cope with heat stress, seeking new biomarkers that can better predict the short-and long-term outcomes

of heatstroke, and developing new adjubant treatments that can effectively control the inflammatory reaction and prevent multiorgan complications.

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