

**Clinical Guidelines on
Management of Heat Related
Illness at Health Clinic and
Emergency and Trauma
Department, Ministry of Health,
Malaysia**

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1. INTRODUCTION

- 1.1. Heat related illness is a medical emergency and may lead to mortality as high as 70% in cases of heat stroke. However, if appropriate treatment is started without delay, survival rates can approach 100%. It is common in the tropics and the incidence is rising even in temperate climate due to the effect of global warming.
- 1.2. This condition is also seen in athletes or military personnel while training, especially during warm weather when any necessary precautions are not taken to reduce the risk of heat related illness. Even more tragic, the extreme of heat related illness, is heat stroke which also could affect young children who have been left alone by parents or caregiver in the car even for a short period of time.
- 1.3. Many healthcare providers are not well trained to recognize the situation early enough or are unsure on how to provide treatment for these patients. Information on early recognition and treatment to the public and health personal can save many lives.
- 1.4. Heat stroke occurs when the thermoregulatory responses are inadequate to preserve homeostasis. This can result from extrinsic factors that make heat dissipation less efficient, such as extremes of temperature, physical effort, and environmental conditions. It also can result from physiologic limitations of the body to respond to heat, putting children, elderly persons, and those who are chronically ill at increased risk. Chronic volume depletion, medication use, inability to increase cardiovascular output, normal deficiencies in heat shock protein responses associated with aging, and lack of acclimatization, all can inhibit the body's ability to respond to heat challenges. Meaning, patients with chronic diseases such as diabetes mellitus, heart disease, renal failure and many others are more susceptible to heat related illness. The body tends to maintain the core body temperature between 36°C to 38°C. The thermoregulatory mechanism begins to fail when the core temperature is < 35°C or > 40°C.
- 1.5. When the thermoregulatory respond are overwhelm, **heat exhaustion** and **heat stroke** occur. Excessive heat results in denaturation of protein, interruption of cellular processes and later, cell death. Damaged cells, release inflammatory cytokines and damages the vascular endothelium. This leads to increased permeability of the vascular endothelium, resulting in activation of the coagulation cascade and disseminated intravascular coagulation (DIVC). The serious complications of heat stroke include encephalopathy, rhabdomyolysis, acute renal failure, acute respiratory distress syndrome, myocardial injury and intravascular injury. CNS

dysfunction is attributed to cerebral oedema, metabolic derangement, ischaemia and these effects are universal at a core temperature of more than 42°C. Poor prognostic signs include coagulopathy with liver hepatocyte damage, rectal temperature more than 42.2°C, prolonged hyperthermia, residual brain damage and acute renal failure.

2. OBJECTIVE

Objectives of this guideline are:

- 2.1. To recognize the spectrum of heat related illness.
- 2.2. To recommend the minimum standards of care in management of heat stroke.
- 2.3. To highlight the preventive aspect of heat related illness

3. DEFINITIONS

3.1. Heat related illness

Heat related illness is a group of disorder ranging from minor (heat edema, prickly heat, heat syncope, heat cramps and heat exhaustion) to major (heat stroke). Table 1 below summarizes the various clinical conditions within the spectrum heat related illness.

Table 1: Summary of Spectrum Heat Related Illness

Heat Related Illness	Clinical Presentation	Treatment
Heat edema	<ul style="list-style-type: none"> • Mild swelling of feet, ankle and hands • Appears in few days of exposure in hot environment • Does not progress to pretibial region 	<ul style="list-style-type: none"> • Usually resolves spontaneously within days and up to 6 weeks • Elevate leg • Compressive stocking • Diuretics are not effective
Prickly Heat	<ul style="list-style-type: none"> • Pruritic, maculapapular, erythematous rash normally over covered areas of body • Itchiness • Prolonged or repeated heat exposure may lead to chronic dermatitis 	<ul style="list-style-type: none"> • antihistamine • wearing clean, light, loose fitting clothing • avoiding sweat generating situations • chlorhexidine in a light cream or lotion base
Heat cramps	<ul style="list-style-type: none"> • Painful, involuntary, spasmodic contractions of skeletal muscle (calves, thighs and shoulder) • Occur in individuals sweating profusely and only drinking water or hypotonic solutions • Limited duration • Limited to certain muscle group 	<ul style="list-style-type: none"> • Fluid and salt replacement (IV or oral) • Rest in cool environment
Heat tetany	<ul style="list-style-type: none"> • Hyperventilation • Paresthesia of the extremities and circumoral • Carpopedal spasm 	<ul style="list-style-type: none"> • Calm the patient to reduce respiratory rate • Remove from hot environment
Heat syncope	<ul style="list-style-type: none"> • Postural hypotension • Commonly in non-acclimatized elderly 	<ul style="list-style-type: none"> • Rule out other causes of syncope • Removal from hot environment • Rest and IV drip
Heat exhaustion	<ul style="list-style-type: none"> • Headache, Nausea, Vomiting • Malaise, Dizziness • Muscle cramps • Temperature less than 40°C or normal • May progress to heat stroke if fails to improve with treatment • No CNS involvement 	<ul style="list-style-type: none"> • Removal from heat stress area • Volume replacement • After 30 minutes if no respond need to aggressively cool the patient to core temperature of 39°C

3.2. Heat Stroke

Heat stroke is defined classically as a **core temperature more than 40.5°C accompanied by central nervous system (CNS) dysfunction**. Two clinical presentations of heat stroke (HS) are classical HS (CHS) and exertional HS (EHS). They are defined by etiology but clinical presentation is similar.

Table 2: Differences in various type of heat stroke

Classical Heat Stroke (CHS)	Exertional Heat Stroke (EHS)	Confinement hyperpyrexia
<p>Etiology:</p> <ul style="list-style-type: none"> Occurs slowly within few hours to days giving time for volume and electrolyte loss to occur Population at risk are elderly, the young, those with physiologic, psychiatric and pharmacological impairments of heat loss mechanism. 	<p>Etiology:</p> <ul style="list-style-type: none"> Overly motivated healthy young individuals Exertion beyond physiological capability 	<p>Etiology:</p> <ul style="list-style-type: none"> Subtype of non-exertional hyperpyrexia Found in 3 circumstances :- <ul style="list-style-type: none"> Child left inside car Human trafficking – left in enclosed vehicle Workers exposed to heat in enclosed space
<ul style="list-style-type: none"> Commonly during severe heat wave (environmental temperature > than 39.2°C) Heat gain occurs at environmental temperatures and humidity levels that overwhelm the native heat loss mechanism 	<ul style="list-style-type: none"> Occur over hours in normal or humid or hot environment This condition is commoner in Malaysia 	<ul style="list-style-type: none"> Preventable

4. CLINICAL MANIFESTATIONS

Clinical Manifestations in Heat Stroke

Heat stroke is a clinical diagnosis with a history of exposure to heat and based on the following criteria:

- 4.1. Core body temperature greater than 40°C.
- 4.2. Signs of CNS dysfunction
 - 4.2.1. Confusion
 - 4.2.2. Delirium
 - 4.2.3. Ataxia
 - 4.2.4. Seizures
 - 4.2.5. Coma
- 4.3. Other late clinical findings that can occur:
 - 4.3.1. Anhidrosis
 - 4.3.2. Coagulopathy
 - 4.3.3. Multiple organ failure

5. DIFFERENTIAL DIAGNOSIS

Other possible diagnosis with features of hyperthermia and CNS dysfunction must be ruled out based on focus history and clinical assessment of the patient:

5.1. Intrinsic Factors

- 5.1.1. Central nervous system (CNS) injury
- 5.1.2. Hyperthyroid storm
- 5.1.3. Infection /Septicemia
- 5.1.4. Neuroleptic malignant syndrome (NMS)
- 5.1.5. Pheochromocytoma

5.2. Extrinsic Factors

- 5.2.1. Anticholinergic poisoning
- 5.2.2. Drug ingestion
- 5.2.3. Heat exhaustion

6. HEAT STROKE WORKUP

There is no diagnostic test for heat stroke. Diagnostic studies are for detection of end organ damage secondary to the metabolic derangement and ruling out other differential diagnosis of hyperthermia and CNS dysfunction.

6.1. Laboratory Investigation

6.1.1. **Arterial Blood Gases**

To detect hypoxaemia that can occur in patient with continuous seizure or inadequate respiration secondary to brain injury. Metabolic acidosis can occur secondary to acute renal impairment.

6.1.2. **Glucose / random blood sugar**

Exclude diagnosis of hypoglycaemia in unconscious patient and also hyperglycaemia in patient with underlying diabetes or undiagnosed diabetes.

6.1.3. **Electrolytes**

6.1.3.1. Sodium

Detection of hypernatremia or hyponatremia due to reduced intake fluid and dehydration and guide the choice of fluid for resuscitation

6.1.3.2. Potassium

To detect hypokalemia or hyperkalemia that can occurs in early phases of heat stroke and muscle damages and during treatment

6.1.3.3. Calcium

Hypocalcaemia occur due to binding of calcium to damage muscles

6.1.4. **Liver Function Test (LFT)**

Hepatic transaminases (ALT) usually elevated in heat stroke patient due to centrilobular necrosis of the liver.

6.1.5. **Coagulation studies**

Direct thermal injury also leads to denaturation of proteins exhibited by dysfunctional enzymes. Any derangement of coagulation is a sign of poor prognosis.

6.1.6. **Full Blood Count**

Thermal injury to vascular endothelium causes platelet aggregation and deactivation of protein plasma leading to platelet aggregation and decrease clotting factor. Total white cell may be elevated due to infection and thrombocytopenia. Also haemo concentration shown by elevated PCV and Hb.

6.1.7. **Renal Function Test**

Acute kidney injury due to inadequacy of volume, dehydration and may also due to rhabdomyolysis, or direct thermal injury to renal parenchyma.

6.1.8. **Muscle enzymes** (Creatinine kinase)

6.1.9. **Urine analysis** for protein, cast and myoglobin

6.2. Electrocardiography (ECG)

Arrhythmias is one of the complications of heat stroke and ruling out underlying cardiac disease / myocardial injury.

6.3. Imaging studies

6.3.1. Chest X ray

CXR carried out to detect presence atelectasis, pneumonia, pulmonary infarction and pulmonary oedema complementing clinical examination.

6.3.2. CT Scan

CT scan can be performed once patient is hemodynamically stable and helpful to rule out intracranial bleeding for patient who did not show improvement in neurological signs.

7. MANAGEMENT OF HEAT STROKE

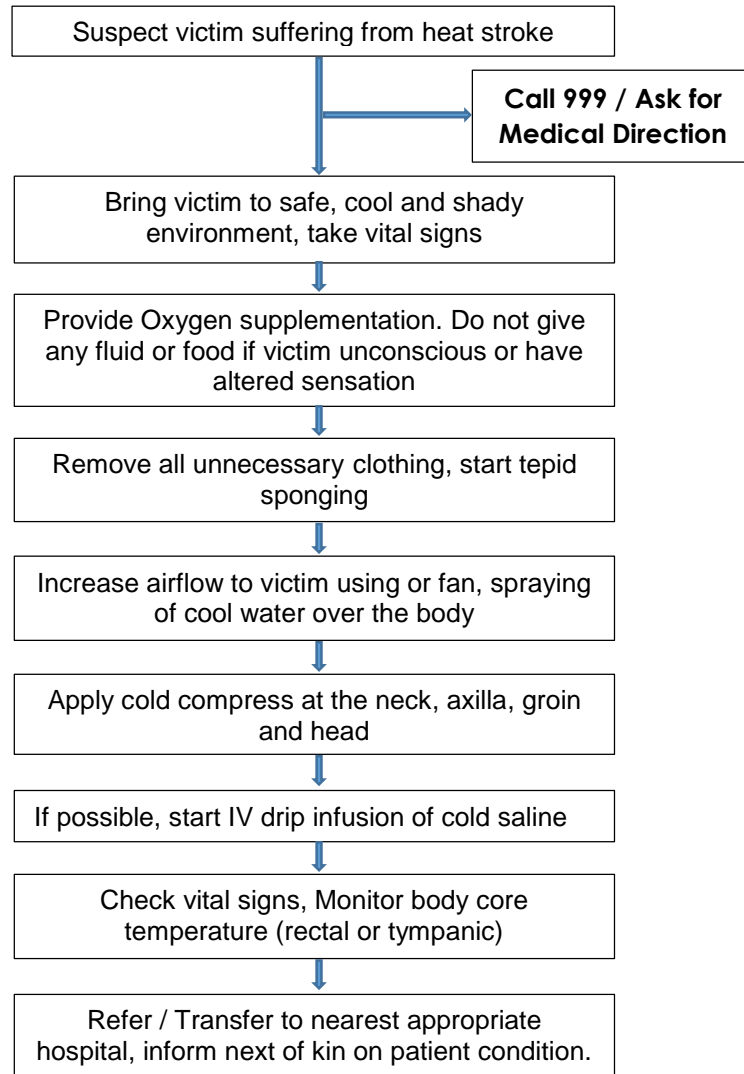
Prompt diagnosis based on focus history (heat exposure or heat exertion) and clinical assessment is vital in ensuring improved clinical outcome. Then followed by effective cooling measures, avoiding a series of metabolic event that may progress into irreversible injury and death.

7.1. Health Clinic or At Scene of Incident

The goal of therapy at health clinic is to **detect the clinical syndrome of heat exhaustion/heat stroke and initiate effective cooling measures immediately, subsequently transfer to nearest appropriate hospital** for definitive treatment.

The following is the recommended management workflow of suspected heat stroke victims that present at the health clinic or found at scene.

Management Workflow of Suspected Heat Stroke Victims



7.2. The Emergency and Trauma Department

The goal of therapy for a heat stroke patient is to prevent further metabolic derangement (rhabdomyolysis, coagulopathy, leading to liver and acute kidney injury) due to thermal injury and institute effective cooling measures, to lower the core body temperature to below 39°C. Initial management of the heat stroke patients is as following:

7.2.1. Focused clinical assessment regarding cardiovascular, respiratory and neurological function.

7.2.2. Exclude other differential diagnoses.

7.2.3. Ensure patent airway, keep patient nil by mouth.

7.2.4. Provide oxygen supplementation.

7.2.5. Ensure adequate respiratory effort.

7.2.6. Insert intravenous cannula and initiate:

7.2.6.1. Intravenous fluids infusion.

7.2.6.1.1. Fluid resuscitation guided based on hemodynamic status, comorbid and ensure urine output (UO) more than 0.5 ml/kg/hr in adult.

7.2.6.1.2. When HR, BP, and UO do not provide adequate hemodynamic information, fluid administration should be guided by other non-invasive and invasive hemodynamic parameters.

7.2.6.2. Take emergent blood samples as per listed in the above (section 5).

7.2.7. Check body core temperature - rectal or esophageal probe or tympanic, ensure the correct placement of probe to the patient.

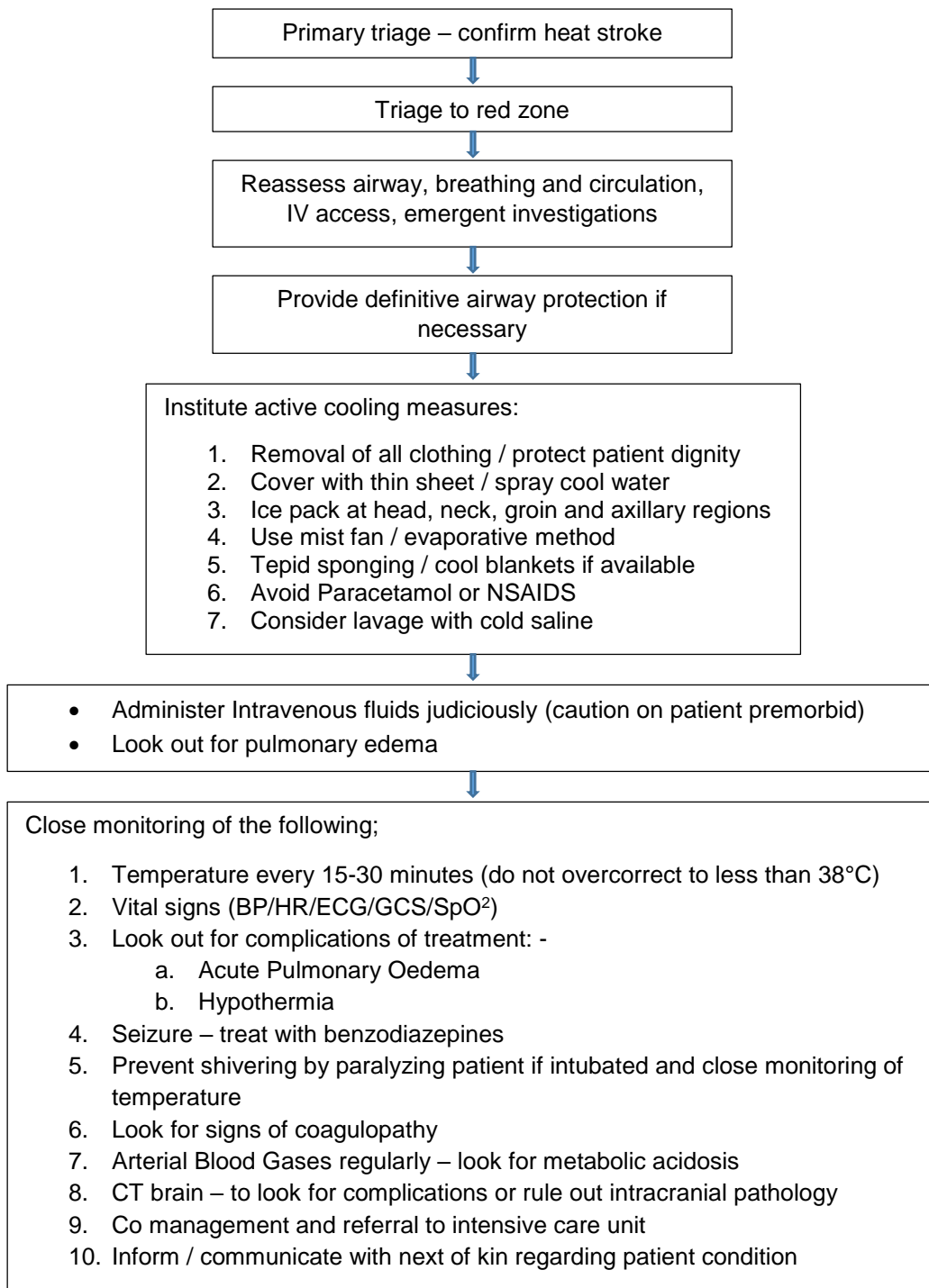
7.2.8. Institute **active cooling** measures such as the following:

7.2.8.1. Removal of body clothing

- 7.2.8.2. Ice packs at groins, neck and axilla, spray cool water
- 7.2.8.3. Use mist fan / air conditioned room / Stand fans.
- 7.2.8.4. Ongoing tepid sponging / cooling blankets.
- 7.2.8.5. Consider lavage with cold saline via nasogastric tube or 3 way urinary catheter.
 - **Please note:** Cooling alone may improve hypotension and cardiac function by allowing blood to redistribute centrally; despite invariably patient with heat stroke has volume depletion.
- 7.2.9. Target to reduce temperature by 0.2°C per minute up to approximately 38°C.
- 7.2.10. **DO NOT** administer Paracetamol or Aspirin or other NSAIDS.
- 7.2.11. Administer benzodiazepine in titrated doses for agitated patient and prepare for securing the airway definitively.
- 7.2.12. Prevent heat production by stopping the seizure and prevent shivering from occurring.
- 7.2.13. Barbiturates may be used for patients having seizures and resistance to benzodiazepines.
- 7.2.14. Close monitoring of the following parameters.
 - 7.2.14.1. Core body temperature.
 - 7.2.14.2. Blood pressure / pulse rate / pulse oximetry.
 - 7.2.14.3. 12 lead ECG and continuous ECG monitoring.
 - 7.2.14.4. Hourly urine output (for patient with continuous bladder drainage).
 - 7.2.14.5. ½ hourly Glasgow Coma Scale (GCS).
 - 7.2.14.6. 4 hourly capillary blood sugar.
 - 7.2.14.7. Nasogastric tube drainage (for intubated patient)

- 7.2.15. Seek and trace the urgent investigation results and treat electrolytes imbalance that can occur.
- 7.2.16. Prevent complications from treatment such as hypothermia and acute pulmonary oedema.
- 7.2.17. Look for signs of coagulopathies, acute kidney injury and liver dysfunction.
- 7.2.18. Co-manage or consult with emergency physician on duty.
- 7.2.19. Co-management and referral to Intensive Care Unit for further care.

Recommended Clinical Workflow in Emergency Department for Management of Heat Stroke Patient



8. PREVENTION

8.1. General preventive measure

- 8.1.1. Decreasing or rescheduling strenuous activity for cooler parts of the day.
- 8.1.2. Wearing clothing that is light colored and loose fitting.
- 8.1.3. Increasing carbohydrate intake and decreasing protein intake to decrease endogenous heat production.
- 8.1.4. Drinking plenty / adequate amount of fluids, even when not thirsty.
- 8.1.5. Avoiding alcoholic beverages, because they promote dehydration.
- 8.1.6. Not using salt tablets.
- 8.1.7. Avoiding direct exposure to the sun.
- 8.1.8. Taking advantage of the shade.

8.2. Public health or public education measures

- 8.2.1. Pay attention to environmental conditions, especially the heat index / weather forecast.
- 8.2.2. Provide access to air-conditioning for individuals at risk (e.g. promote visits to malls, distribute air conditioners) – visit for 2 hours per day reduce risk.
- 8.2.3. Emphasize adequate hydration.
- 8.2.4. Educate public to seek immediate assistance or consult a medical practitioner if developed any signs of heat related illness.
- 8.2.5. Extend social service care for the chronically ill and the elderly.
- 8.2.6. Allow for acclimatization of laborers, workers, athletes, and military personnel and other high risk staffs. Acclimatization is an adaptation of the body's heat stress mechanisms to increase the efficiency of heat loss in a hot climate.

- 8.2.7. Implement paced work schedules for those who must work under adverse conditions.
- 8.2.8. Educate coaches, teachers, youth group other target group leaders about heat-related illnesses.
- 8.2.9. Educate the elderly and patient with multiple co morbids at health clinics / public markets.
- 8.2.10. Limit unnecessary outside activities that may exposure to extreme temperature.
- 8.2.11. Remind parents that they should never leave their children unattended in an automobile.

Appendix 1: Step-By-Step Guide on Initial Management of Suspected Heat Stroke Patient



Step 1:

Remove all unnecessary clothing.



Step 2:

Set IV lines and administer 0.9% normal saline, rate and volume depend on patient's premorbid condition and clinical presentation. Caution of hypoglycaemic event.



Step 3:

Use mist fan for evaporative cooling / use fan to increase airflow (evaporation) / spray with cool water



Step 4:

Apply ice packs at axillary, neck and groin region, continue misty fan / evaporation method.



Step 5:

Tepid sponging and continue misty fan / evaporation method.



Step 6:

Bladder irrigation if necessary or other invasive method of cooling (after consultation) and not to be done routinely.



Step 7:

Monitor temperature every 15 to 30 minutes and vital signs

Step 8:

Repeat step 3 to step 7 again until targeted temperature, and treatment goals achieved

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