

ORIGINAL STUDY

Study of Patients with Heat Stroke Admitted to the Intensive Care Unit of Hamad General Hospital, Doha, Qatar During Summer 2004

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Abstract:

Summer shade temperatures in the State of Qatar are commonly above 40 °C making heat stroke a big problem in unacclimatized outside immigrant workers. Seven males were admitted with heatstroke to the ICU, Hamad General Hospital between 4th July and 24th August 2004. Presenting signs varied but included coma, abnormal behavior, aggression, mental confusion, fits, hypovolemic shock and respiratory failure, metabolic acidosis, hypokalemia, hyponatremia, elevated serum enzymes and sinus tachycardia. All developed renal insufficiency but none died and there appeared to be no residual brain or organ damage. This has been taken as a measure of the effectiveness of the treatment and management in the ICU. It is emphasized that heat stroke is a medical emergency that can result in major organ failure and death and that early recognition and correct treatment are crucial.

Key words: Heat stroke, hyperthermia, complications, outdoor activities, Intensive Care Unit

Introduction:

Heat stroke (HS) is a serious and potentially life-threatening condition worldwide, especially in regions characterized by high summer temperatures and humidity as in Qatar. Traditionally heatstroke is divided into exertional and classic varieties, which are defined by the underlying etiology but are clinically indistinguishable. Exertional heatstroke typically occurs in younger athletic patients who exercise vigorously in the heat until the body's normal thermoregulatory mechanisms are overwhelmed. Classic heatstroke occurs more commonly in older patients or in patients with underlying illnesses who are exposed to extreme environmental conditions.

In the summer of 2004 between 4th July and 24th of August more than 50 patients with acute heat-related illness arrived at the A & E Department of Hamad General Hospital. Seven had severe heat stroke and were admitted to the ICU in critical condition. Their clinical courses during hospitalization in ICU are described including responses to treatment and implications for future management of this disorder.

Patients and Methods:

A descriptive observational study was conducted prospectively on seven patients admitted to the intensive care unit of Hamad General Hospital, Qatar, for heat stroke defined by an elevated core body temperature above 40 °C with central nervous system dysfunction resulting in bizarre behavior, hallucinations, altered mental status, confusion, disorientation, and coma. Exclusion criteria included abnormal head CT findings suggesting infarction, hemorrhage, and tumor, CSF study suggesting CNS infection, EEG findings suggesting tumor or epilepsy, and any abnormal test result that might explain hyperthermia, e.g. positive blood culture.

All patients were treated with a standard regime of IV fluids and sponging in the ICU. Data collected on a standard form included demographic characteristics, clinical presentation, history of previous disease, co-existing medical conditions, drug history, physical examination, electrocardiography and echocardiography results, fluid resuscitation, radiography results and laboratory findings. This was fed into a software program for epidemiological analysis (Epi info 2000) being entered twice to minimize errors before analysis and tabulation using X2, student t-test and odd ratio to identify possible significant association risk factors relating to heat stroke.

Results:

Seven males, median age 24 years, were admitted to the ICU for heat stroke after arriving in Doha only recently. The major risk factor for heat stroke was working outdoors for prolonged periods (mean time of 8.6 hours/day) in a hot and humid atmosphere and none knew how to protect themselves. (Table 1)

Five patients (71.4%) presented in a coma; abnormal behavior, e.g. aggression and mental confusion was exhibited by

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the other two. The mean Glasgow Coma Scale rating being 4.5 +/- 1.5. Three patients were admitted initially as suspected cases of meningoenephalitis. Fits were seen in three patients (42.8%); four patients (57%) were in hypovolemic shock and respiratory failure, necessitating mechanical ventilation. (Table 2)

Metabolic acidosis was seen in six patients (85.7%); hypokalemia in two patients (28.5%); and hyponatremia in two (28.5%). (Table 3)

All seven developed renal insufficiency. Five tested positive for urine myoglobin. Creatine phosphokinase (CK) was elevated in all patients but CK-MB levels were normal. Elevated liver aminotransferase levels were present in all patients. Treponine T (cardiac marker) was normal in all patients. ECG traces showed sinus tachycardia in all seven patients (Table 4). No residual brain damage was detected. None of the seven died.

Table 1: Means of different parameters

Patient No.	Age	Hours of work under the sun
1	21	8
2	22	8
3	24	8
4	24	8
5	26	8
6	27	8
7	42	12
Mean	27	8.6

Table 2: Clinical presentation and Glasgow coma scale of the patients

Patient No.	Clinical presentation	Glasgow Coma Scale
1	Coma, fits, hypovolemic shock and respiratory failure	3
2	Coma, fits, hypovolemic shock and respiratory failure	3
3	Coma, fits, hypovolemic shock and respiratory failure	3
4	Coma, hypovolemic shock and respiratory failure	3
5	Coma	3
6	Agitated, semi-conscious	3
7	Confused	3
Mean		4.5

Table 3: PH value and serum electrolyte of the patients

Patient No.	PH value	S. Potassium meq/L	S. Sodium meq/L
1	6.9	2.8	121
2	7.0	2.8	130
3	7.1	3.5	135
4	7.1	3.5	136
5	7.1	3.5	137
6	7.2	3.6	137
7	7.36	4.1	140

Table 4: ECG trace and Cardiac makers including, Creatinine phosphokinase, CK-MB, and Treponine T of the patients

Patient No.	S. Creatinine Phosphokinase	CK-MB	Treponin T	ECG trace
1	438 U/L	Normal	Normal	Sinus tachycardia
2	623 U/L	Normal	Normal	Sinus tachycardia
3	815 U/L	Normal	Normal	Sinus tachycardia
4	1316 U/L	Normal	Normal	Sinus tachycardia
5	2565 U/L	Normal	Normal	Sinus tachycardia
6	11400 U/L	Normal	Normal	Sinus tachycardia
7	12825 U/L	Normal	Normal	Sinus tachycardia

Discussion:

During the summer months of July and August the weather in the State of Qatar is hot and humid; daily shade temperatures vary from 45 to 50°C and the unshaded solar radiation is intense. Each year this results in 50 or more visits to the Emergency Department of the General Hospital and admissions to the intensive care unit with heat stroke defined by an elevated core body temperature rising above 40°C and central nervous system dysfunction resulting in bizarre behavior, hallucinations, altered mental status, confusion, disorientation, and coma⁽¹⁾. Reported risk factors for heat stroke include non-acclimatization to high temperatures, high humidity, pre-existing illness with fever, obesity, diabetes, alcoholism, excessive muscular activity, administration of sweat inhibiting drugs⁽⁷⁾.

Of the seven critically ill persons discussed in this report the factors pre-disposing to heat stroke were prolonged exposure to hot and humid weather in persons not-acclimatized to such an environment. Co-existing medical conditions were absent. None died and there was remarkably no functional impairment on discharge despite previous multiple organ system dysfunctions and only four of the seven having been cooled within an acceptable

time frame (body temperature $< 38.9^{\circ}\text{C}$ within 30 minutes of presentation to the Emergency Department).

Data on the incidence of heat stroke are imprecise because this illness is underdiagnosed and because the definition of heat-related death varies^(2, 3). According to the National Oceanic and Atmospheric Administration (NOAA), during an average year in the United States approximately 175-200 persons die from heat-related disorders. In Saudi Arabia the incidence varies seasonally from 22 to 250 cases per 100,000 population and the crude mortality rate is estimated at 50 percent⁽⁴⁾.

Tachyarrhythmia and hypotension have been reported in patients with heat stroke^(5, 6). All our patients developed sinus tachycardia. Renal dysfunction, well documented in exertional heat stroke, has been attributed to numerous factors including direct thermal injury, pre-renal insult, rhabdomyolysis, and disseminated intravascular coagulation^(7, 8-11). Most of our patients also developed substantial renal insufficiency caused in part by rhabdomyolysis^(9, 12-16). Creatine phosphokinase levels were elevated in all patients. A high prevalence of elevated liver aminotransferase levels, as in our patients, has been reported in experimental and exertional heat stroke and has been attributed to ischemia and direct thermal injury^(5, 17, 18).

Despite the risk of heat-related morbidity and mortality, many cities lack written heat response plans. In a review of plans from 18 cities at risk of heat-related deaths, it was found that many cities had inadequate or no heat response plans. This is an important area for further investigation and government attention⁽¹⁹⁾.

The key to preventing excessive heat stress is educating the employer and worker (especially recent arrivals in the country), on the hazards of working in heat and the benefits of implementing proper controls and work practices. The employer should establish a program designed to acclimatize workers who must be exposed to hot environments and provide necessary work-rest cycles and water to minimize heat stress. At the same time national public health authorities need to update the current heat emergency response plans with emphasis on their ability to predict mortality and morbidity associated with specific climatologic factors and their public health effect.

Precautions that can help protect workers against the adverse effects of heat stroke include:

- The worker should drink plenty of fluids during outdoor activities, especially on hot days. Water and sports drinks are

the drinks of choice; avoid tea, coffee, soda and alcohol as these can lead to dehydration. Salt replacement is required; the best way to compensate for the loss is to add a little extra salt to the food. Salt tablets *should not* be used.

- To wear lightweight, tightly woven, loose-fitting clothing in light colors.

- To schedule vigorous activity and sports for cooler times of the day. When feasible, the most stressful tasks should be performed during the cooler parts of the day (early morning or at night). Double shifts and overtime should be avoided whenever possible. Rest periods should be extended to alleviate the increase in the body heat load.

- He should protect himself from the sun by wearing a hat, sunglasses and using shade, even when possible, an umbrella.

- To increase time spent outdoors gradually to get your body used to the heat. Acclimatization typically requires 90 minutes per day of exercise in hot conditions for at least one week. Gradually increase exercise intensity and duration

- During outdoor activities, the worker should take frequent drink breaks and mist himself with a spray bottle to avoid becoming overheated.

- Try to spend as much time indoors as possible on very hot and humid days.

Three patients were admitted initially as suspected cases of meningoencephalitis. Such incorrect diagnoses delay the proper management of heat stroke and increase the risk of the complications. Failure to diagnose (or delay in diagnosis), with consequent failure to treat is due to:

- Reliance on classic heatstroke symptoms for diagnosis (eg, extreme hyperpyrexia, anhydrosis) may be misleading.

- Sweating often is maintained in heatstroke; loss of sweating is typically a late sign.

- Pre-hospital cooling can lower a patient's temperature at presentation to the Emergency Department.

Heat exhaustion and heatstroke occupy points along a continuum and patients may progress from heat exhaustion to heatstroke rapidly, even when in hospital. The mortality rate may reach 70% if treatment is delayed more than two hours. Accordingly, heat stroke should form part of the differential diagnosis when any patient who has been working under the sun comes to the Emergency Department with a fever $\geq 40^{\circ}\text{C}$.

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