HEAT STROKE: A CLINICAL REVIEW OF 27 CASES

M K Tham, J Cheng, K M Fock

ABSTRACT

Heat stroke is the most serious entity amongst the heat disorders. Whilst potentially fatal, it is preventable and salvageable. A retrospective study involving 27 patients admitted for exertional heat stroke to the Medical Unit, Toa Payoh Hospital, Singapore from January, 1984 to January, 1987 was carried out. These patients presented with a rectal temperature of > 40°C and central nervous system disturbances. All were males and, except for three, were local born. All except two were below thirty years old. The patients were treated with a standard regime of IV fluids and sponging in the ICU.

19 patients (70.4%) presented in coma whilst abnormal behaviour, e.g. aggression and mental confusion was seen in the remainder. Fits were seen in only 5 patients (18.5%). Metabolic acidosis was seen in 93.3%. Hypokalaemia was present in 3 patients (11.1%). Of the enzymes, creatinine phosphokinase was elevated in all except 1 patient (mean: 4868.8, range: 146-28850).

There were no deaths recorded in this series. Complications include reversible DIVC (3 instances), oliguric renal failure (4 instances) and residual neurological deficit (2 instances). 9 patients (39.1%) took more than 4 hours for the rectal temperature to attain 37.5°C. All the above complications except for 1 instance of DIVC occurred in this group with delayed cooling.

Heat stroke is a serious condition with serious complications and require prompt treatment.

Key words: Exertional Heat Stroke, Clinicopathophysiological Aspects, Potentially Fatal, Rapid Cooling.

INTRODUCTION

Living in the tropics exposes oneself to the discomforts of heat. The maintenance of an optimal internal mileiu for physiological functions is dependent upon the function of the thermoregulatory mechanisms of the body. Should the accumulation of environmental and metabolic heat exceed the dissipatory capacity of the thermoregulatory mechanisms, a spectrum of symptom complexes known collectively as heat disorders occur. Heat disorders may be further classified into several categories (1) of which heat stroke occupies the severe end of the spectrum.

Two major groups of heat stroke exist. Classical heat stroke occurs in epidemic proportions following a rapid rise in environmental temperature. The very young and the elderly are particularly susceptible (2, 3, 4). Exertion in hot humid climate may result in exertional heat stroke which affects fit, young and healthy individuals such as athletes, military recruits at training (5) and construction workers. The cases tend to occur sporadically and casualties are often unacclimatised (6) and may be in suboptimal physical condition (7, 8). Other predisposing factors have also been recognised, for example, physical illnesses (9, 11) obesity (7), restrictive clothing (8), fatigue (8) and lack of sleep (10). Hart et al (12) noted that exertional heat stroke tended to have a lower incidence of predisposing factors such as alcoholism and chronic illnesses seen with classical heat stroke. He also found that lactic acidosis was the predominant acid-base disturbance in exertional heat stroke. Rhabdomyolysis, disseminated intravascular coagulation (DIVC) and oliguric renal failure are recognised complications.

Mortality from heat stroke varies from 5 to 9.5% and early recognition and treatment is important in reducing morbidity and mortality (12, 13).

This paper on exertional heat stroke is to study the clinicopathophysiological aspects of the disorder based upon our own experiences. We also wish to highlight the condition to increase the awareness of clinicians and paramedical personnel to this potentially fatal but treatable condition.

METHOD AND MATERIALS

There were twenty-seven cases of exertional heat stroke and they were admitted to the Medical Unit, Toa Payoh Hospital, Singapore between January 1984 and January 1987. All the patients received a standard treatment of intravenous fluids and sponging as outlined in a protocol.

The patients included had:

1) a rectal temperature of 40°C or more; and
2) central nervous system disturbances.

Cases of pyrexia due to a current infection and patients with persistent fever following adequate attempts at cooling were excluded from the study.

RESULTS

1. Age, Sex and Nationality Distribution

All cases were males. Twenty-four cases were local born Singaporeans and the other three were foreigners: a Korean, a Thai construction worker and a New Zealander Serviceman. The mean age was 20.9 ± 4.7 years with twenty-five cases (92.6%) below the age of thirty-one (Figure 1). The oldest being forty years old.

2. Preceding illnesses

Seven patients (25.9%) had evidence of an upper

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respiratory tract infection a week prior to admission while two (7.4%) had diarrhoea.

Figure 1 — Age Distribution of 27 Heat Stroke Cases

Age In Years
25 (92.6%) cases — below age 31
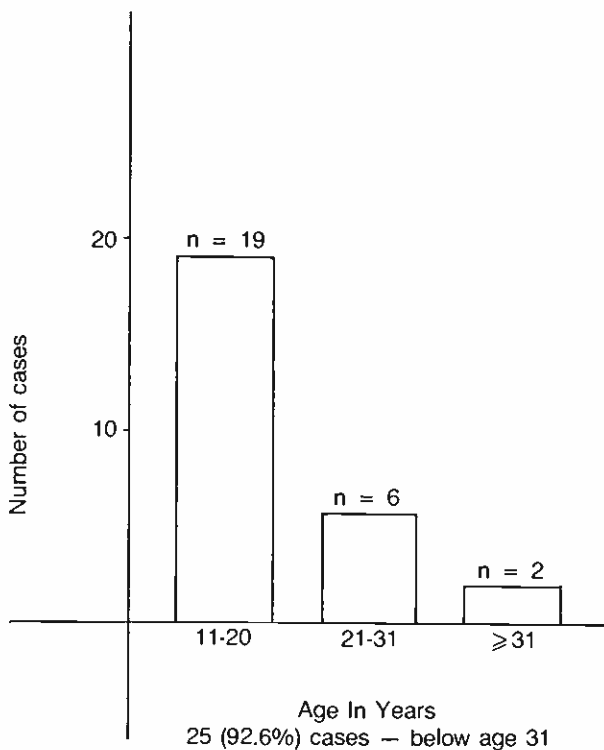
3. Time taken for rectal temperature to attain 37.5°C

Documentation was available in twenty-three cases. Fourteen patients (60.9%) required less than four hours for the rectal temperature to reach 37.5°C whereas nine (39.1%) required more than four hours.

4. Presenting features

(i) Neurological

Nineteen cases (70.4%) presented in coma III or IV. Eight cases (29.6%) had abnormal behaviour manifesting as apathy, aggression and self-mutilation. There were five instances (18.5%) of convulsions, all being generalised in nature and were transient. Only two cases (7.4%) sustained persistent central nervous system defects. One had cerebellar signs while the other had signs of corticospinal tract release. These two had been in coma for more than forty-eight hours.

(ii) Gastrointestinal

There were two instances (7.4%) of diarrhoea and four instances (14.8%) of vomiting. Stool cultures in the cases of diarrhoea were negative for pathogens.

(iii) Urea, electrolytes and glucose

The mean values of urea, electrolytes, and serum glucose are laid out in Table 1. Two patients (7.4%) had hyponatraemia with sodium values less than 132 mmol/L. Hypokalaemia (values less than 3.0 mmol/L) was seen in three cases (11.1%) and two cases (7.4%) had potassium values greater than 5.0 mmol/L. Urea values greater than 50 mg/dl were seen in seven patients (25.9%) of whom four went on to develop oliguric renal failure. Hyperglycaemia occurred in 5 cases whilst there was one case of hypoglycaemia. None of these cases had diabetes mellitus and the hyperglycaemia was transient.

Table 1.
BIOCHEMICAL INDICES IN THE EXERTIONAL HEAT STROKE CASES

Biochemical Index	No. of Subjects	Mean ±1 SD	Range
Sodium (mmol/L)	27	141.0 ± 14.9	121.0 — 149.0
Potassium (mmol/L)	27	4.1 ± 0.5	2.8 — 6.3
Urea (mg/dl)	27	49.9 ± 35.8	17.0 — 157.0
Glucose (mg/dl)	25	133.8 ± 70.5	40.0 — 320.0

(iv) Acid-base status (Table 2)

Of the fifteen cases where results were available, fourteen (93.3%) had metabolic acidosis. In nine cases (64.3%), the metabolic acidosis was compensated. One patient had normal acid-base status.

Table 2.
ACID-BASE STATUS IN EXERTIONAL HEAT STROKE CASES

pH	Uncompensated n (%)	Compensated n (%)
Metabolic Acidosis	5 (33.3%)	9 (60%)
Normal in 1 (6.7%)		

(v) Plasma enzymes (Table 3)

The mean value of each of the plasma enzymes, creatinine phosphokinase (CPK), aspartate transaminase (AST) and lactate dehydrogenase (LDH) was elevated. Five cases (20%) had CPK values greater than 10,000 U/L.

Table 3.
PLASMA ENZYMES IN THE EXERTIONAL HEAT STROKE CASES

Enzyme	No. of Subjects	Mean	Range
CPK (U/L)	25	4868.8	146.0 — 28850.0
AST (U/L)	25	153.5	18.0 — 999.0
LDH (U/L)	25	841.5	264.0 — 2070.0

Note: 5 cases had CPK more than 10,000 U/L

(vi) Haematological

Three patients (11.1%) developed disseminated intravascular coagulation as evidenced by two or more of the following:

(a) an increase in fibrinogen degradation products
(b) prolonged thrombin clotting time
(c) platelet count of less than 130,000/mm3
(d) prolonged prothrombin time

Ten patients (37%) had mild prolongation (ratio > 1:1.2) of the prothrombin time.

5. Complications

The major complications encountered in our series were persisting neurological deficits occurring in two instances, oliguric renal failure in four patients and disseminated intravascular coagulation in three.

DISCUSSION

Exertional heat stroke is a cause of regular hospital admissions. Morbidity is appreciable and mortality has been reported. The predisposing factors leading to heat stroke are many (2-10). Febrile illnesses, the result of upper respiratory tract infections approximately a week or two prior to admission was relatively common being found in a quarter of the cases admitted. A history of a diarrhoeal illness before the collapse was elicited in only 7.4% of cases. These illnesses may in conjunction with other factors such as poor physical condition, lack of acclimatization and hot, humid climatic conditions brought about heat disorders.

Neurological manifestations were present in all cases. Direct thermal effects compounded by ischaemia and metabolic derangements were responsible. Although a large percentage of cases (70.4%) presented in coma III or IV, most recovered. More significantly, of the three who were in coma for more than forty-eight hours despite adequate cooling, two eventually developed persistent neurological deficits. The neurological manifestations of heat stroke can be divided into 3 categories depending upon the time of occurrence. At the acute stage, disturbances of consciousness, convulsions and profound mental changes are the predominant abnormalities. In the convalescent stage, irritability, lack of concentration, delusions and disorientation are common. Neurological deficits that appear late are uncommon and may be permanent (14). Prolonged coma despite adequate cooling is a poor prognostic sign suggesting more severe damage to the cerebrum and this observation was similar to those of Shibolet (16). Tissue injury occurs in direct relationship not only to the level but the duration of exposure to hyperpyrexia (15). Degenerative cerebral neuronal change was a constant finding in cases of heat stroke and have been attributed to hyperthermia whereas the haemorrhages, congestion and oedema are mainly secondary phenomena coincident with shock. Changes in the cerebellum were said to be more consistent and occurred more rapidly than other parts of the brain (15, 16). Behavioural abnormalities and convulsions observed in our series appeared to be relatively benign with no sequelae and did not indicate grave prognosis.

Vomiting and diarrhoea were relatively uncommon gastrointestinal manifestations seen in our series occurring in 7.4% of cases. Khogali (13) noted diarrhoea to be common in his study. In concurrence with his observation, our stool cultures have not isolated any pathological microorganism. The cause of the diarrhoea had been postulated to be the result of thermal alteration of gut flora and/or the massive exudation of fluid into the hollow viscus as a result of thermal damage to the mucosal lining of the gut. In our experience, the episodes of diarrhoea were mild and resolved spontaneously with only simple supportive measures.

Various patterns of acid-base disturbances had been observed in heat stroke (12, 17). Of the fifteen cases in our series where blood gas estimations were carried out, fourteen patients (93.3%) had metabolic acidosis while one had normal acid base status. Compensation had occurred in five of the fourteen cases. Metabolic acidosis in exertional heat stroke was also observed by others (18, 19). Lactic acid formation contributed by physical exertion, dehydration, hypotension and tissue anoxia was thought to be responsible. Levels in the range of 10-20

mmol/L were said not to be unusual. Costrini (20) in his study of thirteen marine recruits saw no clear evidence that blood lactate correlated with prognosis in men with exertional heat stroke.

Severe metabolic disturbances were uncommon. The mean values in our series on serum sodium and potassium were within normal limits. There were, however, two patients (7.1%) with hyponatraemia and three (11.1%) with hypokalaemia. Excessive sweating during exertion in the hot climatic conditions or rapid dehydration with intravenous fluid during resuscitation could account for these abnormalities. Fluid loss leading to hypovolaemia could further contribute to potassium loss through the Renin-Angiotensin Mechanism. Hyperglycaemia (blood glucose > 180 mg/dl) seen in five cases (20%) probably reflected the state of dehydration and increased catecholamine activity in response to stress (21). The only case of hypoglycaemia seen could be explained by plasma dilution following rapid intravenous fluid replacement. The biochemical derangements as a whole were easily correctable and none of our cases had complications resulting from rapid replacement with, quite frequently, large volumes of intravenous fluids.

Four patients (14.8%) in our series developed oliguric renal failure. Management of these cases was entirely conservative. Dialysis was not required and no deaths resulted from renal failure. Acute tubular necrosis complicates heat stroke in 2 to 9% of cases in other studies (15, 23) and its incidence appear related to the degree of hyperpyrexia and prolonged shock.

In his report on 125 fatal cases of heat stroke, Malamud (15) found 60% of the cases had hypotension and that fatality correlated best with the incidence of shock. All four cases with oliguric renal failure in our series had prolonged pyrexia beyond eight hours and and three of the four cases were hypotensive on admission to hospital. Fortunately, no mortalities were recorded in our series. The hypotension was rapidly corrected mainly through the infusion of fluids. Apart from direct thermal injury, acidosis, hyperuricaemia and myoglobinuria (22) were other possible contributory factors leading to renal dysfunction. Mortality from azotaemia complicating heat stroke was as high as 31% (23) and death was mainly from hyperkalaemia.

The mean plasma enzyme (creatinine phosphokinase, aspartate amino transferase and lactate dehydrogenase) levels were elevated. In five cases (20%), there was a dramatic rise in the creatinine phosphokinase to values beyond 10,000 U/L. These high values indicated the presence of rhabdomyolysis which is a feature of exertional heat stroke. It would be of interest to postulate the susceptibility of muscles in the context of exertional heat stroke to rhabdomyolysis. A combination of factors is likely. Part of which could be due to the summation of metabolic heat in exercising muscles compounding the environment heat load. Acidosis from local lactate formation and other local metabolic changes, though physiological, may exacerbate and hence precipitate muscle injury. DIVC and ischaemia further adds to the list of initiating events. SGOT over 1,000 U/L during the first 24 hours usually indicated a severe case of tissue injury (16).

Haemorrhagic abnormalities in severe heat stroke were found to be common. They include petechial haemorrhages, ecchymosis, epistaxis and haemetemesis (24). The haematological abnormalities noted in our series were not clinically overt except for three cases (11.1%) of disseminated intravascular coagulation (DIVC). Laboratory findings of transient mild prolongation of the prothrombin time were common, seen in ten cases (37.0%). In fatal cases of DIVC, widespread microthrombus formation were seen in various organs supporting the theory of DIVC being secondary to endothelial damage (25). The resultant occlusion of small vessels led to

ischaemic injury and necrosis of organs.

Management of all cases of heat stroke could be categorised into 3 stages. First and foremost is resuscitation and rapid lowering of an elevated body temperature. Lind (26) felt that early detection and treatment of heat disorders could prevent morbidity and mortality. Fourteen cases (60.9%) in our series attained the target rectal temperature of 37.5°C within four hours of "collapse" but nine (39.1%) exceeded that time period. All the four cases of oliguric renal failure, two out of three cases of DIVC and the two cases of persistent neurological dysfunction were found in this latter group.

The second stage in management included the treatment of complications, eg renal failure and infections. Lastly, rehabilitation particularly for those who have sustained long term neurological disability is equally important.

In summary, heat stroke is a multi system disorder. Complications affecting the blood, kidneys and the brain are serious and life-threatening. Treatment of heat stroke should be prompt as the incidence of severe complications is related to the degree of hyperpyrexia and duration of exposure to hyperpyrexia and shock.

ACKNOWLEDGEMENT

REFERENCES

1. WHO (1977). Manual Of Intern. Statistical Classification Of Diseases, Injuries And Causes Of Death. 9th Revision. WHO, Geneva.
2. Gottschalk PG, Thomas JE. Heat Stroke. Mayo Clin. Proc. 1966; 41: 470-82.
3. Levine JA. Heat Stroke In The Aged. Am. J. Med. 1969; 47: 251-8.
4. King K, Negus K, Vance JC. Heat Stress In Motor Vehicles: A Problem In Infancy. Pediatrics 1981; 68: 579-82.
5. Sprung CL. Heat Stroke: Modern Approach To An Ancient Disease. Chest 1980; 77: 461-2.
6. Wyndham CH, Strydom NB, Morrison JF. et al. Criteria For Physiological Limits For Work In Heat. J. Appl. Physiol. 1965; 20: 37-45.
7. Minard D, Belding HS, Kingston JR. Prevention Of Heat Casualties. JAMA 1957; 165: 1813-8.
8. Bartley JD. Heat Stroke: Is Total Prevention Possible? Milit. Med. 1977; 142: 528-35.
9. Barry ME, King BA. Heat Stroke. S. African Med. J. 1962; 36: 455-61.
10. Shibolet S, Coll R, Gilat T, Sohar E. Heat Stroke: Its Clinical Picture And Mechanism In 36 Cases. Q. J. Med. 1967; 36: 525-48.
11. Kilbourne EM, Choi K, Jones S. et al. Risk Factors For Heat Stroke. JAMA 1982; 247: 3332-6.
12. Hart GR, Anderson RJ, Crumpler CP. et al. Epidemic Classical Heat Stroke: Clinical Characteristics And Course Of 28 Patients. Medicine 1982; 61: 189-97.
13. Al-Khawashki MI, Mustafa MKY, Khogali M. et al. Clinical Presentation Of 172 Heat Stroke Cases Seen At Mina And Arafat — September 1982 In "Heat Stroke And Temperature Regulation" (M. Khogali and J.R.S. Hales, eds.), pp 99-108. Academic Press: Sydney 1983.
14. Mehta AC, Baker RN. Persistent Neurological Deficits In Heat Stroke. Neurology 1970; 20: 336-40.
15. Malamud N, Haymaker W, Custer RP. Heat Stroke: A Clinico-Pathologic Study Of 125 Fatal Cases. Milit. Surg. 1946; 99: 397-449.
16. Shibolet S, Lancaster MC, Danon Y. Heat Stroke: A Review. Aviat. Space & Environ. Med. 1976; 47: 280-301.
17. Gumaa K, El-Mahrouky SF, Mahmoud N, Mustafa MKY, Khogali M. The Metabolic Status Of Heat Stroke Patients: The Makkah Experience In "Heat Stroke And Temperature Regulation" (M. Khagoli and J.R.S. Hales, eds.), pp 157-69. Academic Press: Sydney 1983.
18. Hanson PG, Zimmerman SW. Exertional Heat Stroke In Novice Runners. JAMA 1979; 242: 154-7.
19. Ruppert RD, Newman A, Scarpelli DG, Weissler AM. The Mechanisms Of Metabolic Acidosis In Heat Stroke. Clin. Res. 1964; 12: 356.
20. Costrini AM, Pitt HA, Gustafson AB, Uddin DE. Cardiovascular And Metabolic Manifestations Of Heat Stroke And Severe Heat Exhaustion. Am. J. Med. 1979; 66: 296-302.
21. Escourrou P, Freund-PR, Rowell LB, Johnson DG. Splanchnic Vasoconstriction In Heat-Stressed Men: Role Of Renin-Angiotensin System. J. Appl. Physiol. 1982; 52: 1438-43.
22. Carson J. Heat Stroke With Left Hemiplegia, Acute Tubular Necrosis, Hypertension And Myocardial Damage. Proc. Roy. Soc. Med. 1972; 65: 752-3.
23. Austin MG, Berry JW. Observations On One Hundred Cases Of Heat Stroke. JAMA 1956; 161: 1525-9.
24. Knochel JP, Beisel WR, Herndon EG. et al. The Renal, Cardiovascular, Haematologic And Serum Electrolyte Abnormalities Of Heat Stroke. Am. J. Med. 1961; 30: 299-309.
25. Chao TC, Sinniah R, Pakiam JE. Acute Heat Stroke Deaths. Pathology 1981; 13:145-56.
26. Lind AR. Pathophysiology Of Heat Exhaustion And Heat Stroke (1983) In "Heat Stroke And Temperature Regulation" (M. Khogali and J.R.S. Hales, eds.), pp 179-88. Academic Press: Sydney 1983.