HEAT STROKE AMONG 11 PATIENTS IN BASRAH

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ABSTRACT

Heat stroke is a preventable medical emergency. The aim of this study was to study heat stroke in Basrah during the heat waves of 1998. We studied 11 patients with exertionl heat stroke. Those who survived were followed for 3 years. Five patients died within 48 hours of admission, six patients survived, of them five were left with permanent cerebellar signs. From this we conclude that irreversible brain dysfunction is an important sequel of heat stroke.

INTRODUCTION

Heat stroke is a medical emergency that occurs in two distinct settings[1-3]. Exertional heat stroke which occurs due to muscle exertion in hot, humid weather. Those affected are generally young and healthy. Predisposing factors include lack of acclimation, lack of cardiovascular conditioning, dehydration, the wearing of heavy clothing and excessive exertion. In hot humid air exertional heat stroke can develop even if sweating is normal and in the absence of exertion[4]. While the other type of heat stroke is classic heat stroke, which is more common in sedentary, elderly patients. In the classic heat stroke usually there is underlying disease that predisposes to heat stroke, like neurologic disease, cardiovascular disease, diabetes mellitus, thyrotoxicosis, psychiatric illness, drugs like anticholinergics, antidepressants, antihistamines, ß-blockers, diuretics, phenothiazine and ehanol[1]. Heat stroke susceptibility may be due to inherited abnormality in the skeletal muscles, similar to that of malignant hyperthermia[5]. The clinical features of heat stroke, include acute onset of hyperpyrexia, altered sensorum, hypotesion, tachycardia, and hyperventilation.[1-3]. Together with laboratory evidence of hemoconcetration, proteinuria, microscopic hematuria, abnormal liver enzymes and rhabdomyolysis[1,6-9]. Disseminated intravascular coagulation is common.[2,4,10]. Hypoglycemia is also reported with early respiratory alkalosis and hypokalemia[7]. Lactic acidosis with hyperkalemia occur later. Both hypo and hyperphosphatemia are reported, and serum calcium might be low[9].

PATIENTS & METHODS

During the heat waves of summer 1998,eleven patients were admitted to the military hospital in Basrah *(southern Iraq).* All patients fulfilled the criteria of heat stroke: exposure to high environmental temperature, hyperpyrexia, hot dry *skin (not necessary for diagnosis)* and central nervous system disturbances[1-4]. Temperatures were measured rectally. Patients were resuscitated by cooling rapidly using physical way by removing clothes, sponging with tepid water and fanning. Some were immersed in a large tube of ice water as practiced by others[11]. Intravenous fluid was given for all, which was 1 liter of normal saline or ringer lactate over one hour. Cooling time usually continued until the core body temperature dropped to 37Ċ. For all patients, baseline investigations were done including serum urea, serum glucose, serum creatinine, complete blood count, urine examination, and electrocardiography (ECG).

RESULTS

All of the patients were males and none of them had history of diseases or illnesses that predispose to heat stroke (all sustained exertional heat stroke). All except 2 had history of exposure to high environmental temperature in poorly ventilated rooms and high humidity.Two patients gave a history of direct exposure to sunlight. On admission, rectal temperature ranged from 40.6 Ċ to 42.7Ċ (Table-1). All patients were hypotensive with blood pressure ranging from 90/0-60/0 mm/Hg and pulse rate ranging from 120-180 beat per minutes. All were tachypneic with respiratory rate ranging from 30-40/minute. Evidence of pre renal azotemia was seen in all patients. Total number of patients were 11, five of them recovered after variable periods with bilateral cerebellar signs with subsequent partial improvement over years (Table-2). One patient recovered completely without neurological sequel, and five died within 48 hours of

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admission *(the mortality rate was 45%)*. Important complications are presented in Table-2. While cerebellar signs and postmortem findings are presented in Tables-3 and 4 respectively. Duration of follow up was three years.

Table 1. ***Patient's characteristics.***

Patient name and number	***TIF 1***	***ZMI 2***	***SMF 3***	***ZHA 4***	***HAA 5***	***MAJ 6***	***HJK 7***	***HKD 8***	***MDI 9***	***HAH 10***	***HAA 11***
Age (years)	18	18	22	28	30	20	24	19	23	24	19
Rectal temperature on admission (Ċ)	41	41	40.6	41.3	41,5	41.3	42.7	41.9	41.8	42.7	41.5
Direct sun exposure	+	_	_	_	_	+	_	_	_	_	_
Coma on admission	+	+	+	+	+	*Confusion*	+	+	+	+	+
Duration of coma (days)	4	3	14	3	4	_	_	_	_	_	_
Duration of hospitalization (days)	14	14	28	14	10	2	_	_	_	_	_

Table 2. ***Complications reported among patients with heat stroke.***

TIF 1	Status epilepticus, aspiration pneumonia, hepatitis, 9th and 10th cranial nerve palsies, dysphagia, irreversible cerebellar signs , leucocytosis
ZMI 2	Meningism and irreversible cerebellar signs
SMF 3	Irreversible cerebellar signs
ZHA 4	Irreversible cerebellar signs
HAA 5	Irreversible cerebellar signs, leucocytosis
MAJ 6	Full recovery within five hours and discharged after two days
HJK 7	Anuria,death (12 hour after admission)
HKD 8	Generalized bleeding tendency, elevated CSF protein, ST&T changes on ECG, death (30 hour after admission)
MDI 9	Status epilepticus, ST& T changes on ECG, death (48 hour after admission)
HAH 10	Death (12 hour after admission)
HAA 11	ST&T changes on ECG, death (12 hour after admission)

Table 3. ***Cerebellar signs in patients who survived after three years of follow up.***

Patient name and number	***TIF 1***	***ZMI 2***	***SMF 3***	***ZHA 4***	***HAA 5***
Disability	***Mild***	***Moderate***	***Mild***	***Moderate***	***Mild***
Dysarthria	+	+	+	+	+
Finger-nose impairment	+	+	+	+	+
Heel-shin impairment	+	+	+	+	+
Truncal ataxia	–	–	–	+	–
Impaired tandem gait	+	+	+	+	–
Jerky nystagmus	–	–	+	–	–

Table 4. ***Post mortum findings of patients who died.***

Patient name and number	***HJK 7***	***HKD 8***	***MDI 9***	***HAH 10***	***HAA 11***
Brain white matter petechial hemorrhage	+	+	–	–	–
Brain white and grey matter petechial hemorrhage	–	–	+	+	–
Subendocardial hemorrhage	+	+			
Acute myocardial infarction	–	+anterior	–	–	–
Gastroduodenal erosions	-	-	+	-	-
Kidneys oedema	–	–	–	–	+

DISCUSSION

The pathophysiology of heat stroke is not fully understood. Endotoxin and cytokines appear to play a major part in the central nervous system and in the peripheral tissues[12]. The basic pathology is widespread cellular damage of vital organs, as a result of high body temperature. Treatment of patients with heat stroke with interleukin-1 receptor antagonists seems to improve recovery and prognosis[13,14]. The arterial hypotension and cerebral ischemic damage are the main reasons of heat stroke syndromes[15]. Six of our patients recovered, one of them completely and five with variable cerebellar signs. This is a well known fact as severe cases of heat stroke may die suddenly or if they recover they my show lasting cerebellar or cerebral signs[16-20]. Cerbellar atrophy has been, reported two years after the heat stroke[16]. Heat stroke may cause cerebral hemorrhage and is a recognized cause of permanent cerebellar dysfunction with cerebellar atrophy on CT, years after the attack[16,21]. Heat stroke is also associated with transverse myelopathy[16]. Post mortum examination may show little abnormality in cases of rapid death in the from of degeneration

of purkinje cells and other large cells of the brain cortex[21]. Less rapid death may show oedema and petechial hemorrhages in the brain and some time in other tissues. All of our patients who died except one were having petechial hemorrhage in the brain distributed in the white matter and gray matter. Patients with prolonged hyperpyrexia due to heat stroke have poor outcome[11]. Thus, the most important feature in the management is rapid cooling like fan therapy, cold water immersion, iced baths, and evaporative cooling[1,11]. We have used fluid cautiously to avoid overloading of patients, as only one third of patients with heat stroke has low central venous pressure[22]. The mortality in the studied patients was 45%,while in other studies the mortality ranged from 10% to 70%[1,7,9]. This wide range difference depends on how rapid the cooling procedure took place[11]. All patients who died never regained consciousness because of irreversible heat induced brain injury[7]. The hepatic injury is nearly a constant event in the course of heat stroke, but rarely evolve to a severe liver failure[23]. Liver injury was seen in one patient, who survived, however we could not exclude liver injury in others because transaminases were not measured in all patients. Two of those who survived had a history of direct sun light exposure. Heat stroke due to direct sun light exposure is considered by some to be associated with better survival[24]. Cardiac complications of heat stroke include, arrhythmia, chamber dilatation, myocardial infarction with normal coronary arteries[6,25]. Only one of our patients had definite acute myocardial infarction proved by post mortum study, other patients showed electrocardio-graphic non-specific ST&T changes. Heat stroke is a preventable medical emergency[1]. Limited sun exposure, adequate fluid and electrolyte replacement and acclimatization are the key factors for prevention[1,26,27]

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