Original Article

Heat Stroke at Karachi

A Clinical Experience of Patients with Heat Stroke at Karachi During a Devastating Heat Wave in 2015

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ABSTRACT

Objectives: To review the clinical presentations, management and outcomes of heat stroke patients presented to Hamdard University Hospital, Karachi in summer 2015.

Study Design: Observational / descriptive study.

Place and Duration of Study: This study was conducted at the Hamdard University Hospital (Taj Medical Complex), Karachi from 15th to 30th June 2015.

Methods: A retrospective analysis of database of 51 patients presented with high grade fever (>1040F) and altered sensorium was performed. All data were transferred to proforma which included patient's demographic features (name, age and sex), clinical and laboratory parameters, treatment given, duration of hospital stay, outcomes (death or alive) and reasons of mortality. The SPSS version 19 was used for statistical analyses.

Results: Majority of the patients (63%) were between 61-80 years of age group with mean±SD age was 69.24±11.28 years. Males were affected more than females (60.7% vs. 39.3% respectively). Out of 51, 41 (80.4%) had co-morbidities and were on regular medications. The mean±SD Glasgow Coma Scale at the time of presentation was 10.29±4.33. The major laboratory derangements were hyponatremia (68.6%), elevated blood urea (52.9%), serum creatinine (41.2%) and alanine transferase (15.6%). Standard treatment strategies were provided to all patients. Out of 51, 19 (37.3%) patients were expired as a result of multi-organ failure, shock, arrhythmias and rhabdomyolysis.

Conclusion: Heat stroke is common in older males especially those who had co-morbidities. It carries a significant mortality due to multiorgan failure and shock.

Key Words: Heat stroke, Heat wave, Mortality.

Citation of article: Yahya M, Lal K, Khan MKH, Inam U. A Clinical Experience of Patients with Heat Stroke at Karachi During a Devastating Heat Wave in 2015. Med Forum 2016;27(6):32-35.

INTRODUCTION

Heat-related illnesses are group of disorder characterized by heat exhaustion and heat stroke, and if untreated, may lead to mortality as a consequence of multiorgan dysfunction syndrome.[1] It has a substantial effect on million of peoples worldwide. According to World Health Organization (WHO) statistical report, extreme temperature events contributed 23.8% of all disasters in 2012.[2] Hence, it is one of the foremost environmental hazards that requires well-timed reporting of its occurrence, management and complications. The classical heat stroke is manifested as an elevated core body temperature (>40^{0}C) associated with central nervous system (CNS) abnormalities like delirium, coma, seizures due to exposure of heat waves.[3] Elderly population with pre-existing co-morbidities, children, outdoor labors, athletes and those who are on medications that impair temperature homoestasis have been identified as most vulnerable cohorts in literature.[4-6] In urban areas, both heat island effect and higher population density have been considered as contributing factors of heat events.[7,8] In addition, rising trends in global climate change has been noticed and are anticipated to progress in future.[9] According to technical report of Pakistan Meteorological Department, the frequency of heat waves has been continuously increasing in Pakistan for past five decades.[10]

In June 2015, Karachi, a metropolitan city of Pakistan, experienced a heat wave for period of 5-days from 19th to 23rd June with the reported highest temperature 44.8^{0}C on 20th June.[13] Although, more than 1000 deaths have been noticed during this time period,[14] the exact mortality is seem to be under-reported by reason of lesser contribution of various centers publishing their statistics on heat stroke patient's management and outcomes. The purpose of this study was to review the clinical presentations, management and outcomes of heat stroke patients presented to Hamdard University Hospital, Karachi in summer 2015.

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Received: March 10, 2016; Accepted: April 29, 2016

MATERIALS AND METHODS

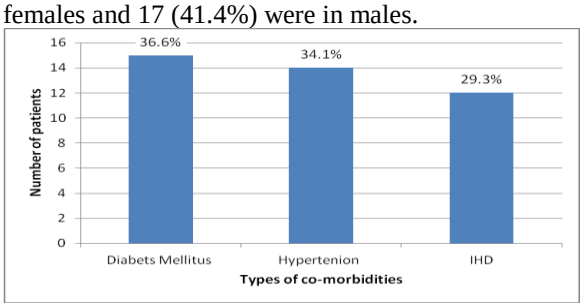
A retrospective review of database of all patients having body temperature >104^{0}F with altered sensorium presented at Hamdard University Hospital (Taj Medical Complex), Karachi from 15th to 30th June 2015 after heat wave exposure was accomplished. Clinical information was retrieved and those who had other causes of high grade fever like malaria, dengue, typhoid, meningitis, urinary tract infection and pneumonia were excluded from the study.

All clinical records were updated into proforma designed for the study. It included patient's demography (name, age, and sex), co-morbidities, drug history, Glasgow Coma Scale (GCS), pulse rate (in beat/min) and temperature (in ^{0}F), laboratory parameters [Complete blood count (CBC), blood urea and serum creatinine (in mg/dL), serum electrolytes (in mEq/L), and liver function test (LFTs) and creatinine phosphokinase (CPK)], electrocardiogram (ECG), chest X-ray and CT scan brain findings, outcomes (alive or death), reasons of death, management and duration of stay (in days).

Statistical analyses were conducted by SPP version 19. Qualitative data were represented by frequencies and percentages. Mean and standard deviation was employed for quantitative data.

RESULTS

A total of 51 patients having high grade fever (>104^{0}F) with altered level of consciousness were presented during study period. Mean±SD fever was 105.24+0.86^{0}F. The age of patients ranged from 40 to 87 years with mean±SD age was 69.24±11.28 years. Among these, 22% patients were in 40-60 years, 63% in 61-80 years, and 15% were in age group above 80 years. Of these 51 patients, 31 (60.7%) were males and 20 (39.3%) were females. The co-morbidities were noticed in 41 (80.4%) patients; 24 (58.6%) were in females and 17 (41.4%) were in males.



(IHD=Ischemic heart disease)

Figure No.1: Types of co-morbidities

Figure 1 shows the types of co-morbidities encountered in this study. All of these patients were on regular medications. These included oral hypoglycemics (29.5%), insulin (7.3%), calcium channel blockers (17%), beta blockers (9.7%), diuretics (4.9%), anti-platelets (7.3%), anti-hyperlipidemics (7.3%) and proton pump inhibitors (17%).

At the time of arrival, pulse was impalpable in 11 (21.6%) patients. 29 (56.8%) patients had a pulse rate between 120-130 beats/min and 11 (21.6%) had a rate between 131-140 beats/min. The mean±SD pulse rate was 128.80±7.55 beats/min. The GCS at the time of presentation were ranged from 6 to 14 with mean±SD GCS was 10.29±4.33. Table 1 shows the laboratory parameters of all patients. The hyponatremia (serum sodium <135 mEq/L), and elevated blood urea (>40 mg/dL), serum creatinine (>1.4 mg/dL) and alanine transferase (>36 U/L) were observed in 35 (68.6%), 27 (52.9%), 21 (41.2%), and 8 (15.6%) patients respectively. ECG was performed in 28 (54.9%) patients. Out of these 28, ventricular tachycardia was demonstrated in 08 (28.6%) patients. Chest X-rays was done in 44 (86.2%) patients. Of these 44, bronchitic changes due to age or smoking was noticed in 16 (36.4%) and cardiomegaly was observed in 04 (9.1%) patients. CT scan was advised on the basis of clinical parameters. In this study, 12 (23.5%) patients were subjected to CT scan brain; amongst them, cerebral edema was encountered in 04 (33.3%) patients.

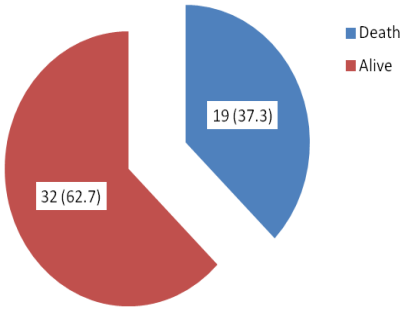
Table No.1: Laboratory parameters

Laboratory parameters	Mean	SD
Hemoglobin (gm/dL)	11.84	1.59
WBC (mm^3)	9357.14	4062.22
Platelets (per HPF)	216448.98	86101.21
Urea (mg/dL)	57.69	33.22
Creatinine (mg/dL)	2.07	1.21
Sodium (mEq/L)	125.69	12.96
Potassium (mEq/L)	3.32	0.54
Chloride (mEq/L)	92.67	9.41
Bicarbonate (mEq/L)	21.98	2.66
Total bilirubin (mg/dL)	1.19	0.77
Alanine transferase (U/L)	78.98	142.82
Alkaline phosphatase (U/L)	93.90	37.71
Creatinine phosphokinase (U/L)	897.56	913.41

SD=Standard deviation

Standard treatment of heat stroke was employed in all patients. These included cooling of patient by applying ice, maintenance of adequate hydration by intravenous fluids, cardiovascular monitoring, and administration of sedatives and analgesics. In addition to these, treatment of complications due to heat stroke was also started. Cardioversion and anti-arrhythmic medications were instituted in patients who developed ventricular tachycardia. The intravenous mannitol was given to two (3.9%) male patients who developed rhabdomyolysis. Later these patients required hemodialysis. Ventilatory support was started in 03 (5.9%) patients who had GCS less than 08.

The duration of stay ranged from 1 to 13 days with mean+SD stay was 6.61+2.87 days. The mortality was noticed in 19 (37.3%) patients in this study (Figure 2). Of these 19, 09 (47.4%) were females and 10 (52.6%) were males. The reasons of death were multiorgan failure, shock, arrhythmias and rhabdomyolysis (Table 2).



Data are shown in numbers followed by percentages in figure

Figure No.2: Outcomes

Table No.2: Reasons of mortality

Reasons of mortality	Male n=10	Female n=09
Multi-organ failure	03 (30)	02 (22.2)
Shock	03 (30)	03 (33.3)
Cardiac arrhythmias	01 (10)	00 (0)
Rhabdomyolysis	02 (20)	00 (0)
Shock and cardiac arrhythmias	01 (10)	04 (44.5)

Data are shown in numbers followed by percentages in parentheses

DISCUSSION

Heat stroke is the devastating public health problem. It commonly involves extreme of ages and especially those who have previous co-morbidities.[4-6] In this study, heat stroke afflicted 63% of patients between 61 to 80 years of age group with average age of 69 years. The males were affected more than females. In a large retrospective study, Piver et al observed older male population >65 years of age as an independent predictor of heat stroke.[15] Similarly, Kalaiselvan et al also encountered older age males in their case series who were affected by heat-related illnesses.[16] Most of them had diabetes mellitus and hypertension as co-morbidities.[16] In this study, large proportion of patients also had diabetes mellitus and hypertension.

In literature, associations of certain drugs with heat stroke have been documented.[5] It has been suggested that certain drugs like vasoconstrictors, calcium channel blockers and beta blocker are responsible for alteration in thermoregulatory mechanism of the body resulting in increase susceptibility to heat stroke.[17] In this study, significant proportion of patients (26.7%) were already taking calcium channel blockers and beta blockers.

In heat stroke, abnormalities are frequently noticed in serum electrolytes, blood urea, serum creatinine, coagulation profiles, LFTs and creatinine phosphokinase. In this study, hyponatremia, and elevated blood urea and creatinine were encountered in 68.6%, 52.9% and 41.2% patients respectively. Kalaiselvan et al in their case series found hyponatremia in 73% and raised serum creatinine in 57% patients, which is nearly comparable to this study.[16] The derangements in liver enzymes in heat stroke were first reported by Kew et al in 1970.[18] Arguad et al reported 40% elevation of alanine transferase levels in their case study.[19] In contrast to results of Arguad et al, 15.6% patients had elevated alanine transferase in this study.

The role of hyperthermia in causation of cerebral edema has been argued in literature. Sharma mentioned that the alteration in blood brain barrier resulting in vasogenic edema is the main etiologic factor of heat-related illness.[20] In addition, edema secondary to hyponatremia and direct effect of elevated temperature on neurons of basal ganglia, hypothalamus and cerebellum have been stated in literature as well.[16] Although, variations in neurological symptoms were noticed in this study, cerebral edema was noticed in four patients on CT scan brain.

Early recovery of hyperthermia, fluid and electrolyte management, cardiovascular monitoring, sedation and analgesia, and prompt recognition and treatment of complications are the standard care of heat stroke patients.[17] In this study, all patients were treated on the same line. Rhabdomyolysis was treated initially with intravenous mannitol administration and adequate hydration, and later by hemodialysis. Trujillo and Fragachan also adopted similar management strategies in their patient.[21]

There are wide ranges of mortality reported in literature. Misset et al reported 62.6% in-hospital mortality in France.[22] The 43.1% estimated mortality rate was documented in 2014 Ahmedabad heat wave event by Azhar et al.[23] In this study, 37.3% deaths were noticed which is comparable to the case study by Kalaiselvan et al[16]. A number of complications have been attributable to mortality after heat stroke viz. renal failure, hemodynamic shock, arrhythmias, rhabdomyolysis, and multi-organ failure.[17,24] In this study, mortality was related to shock and multiorgan failure.

The morbidity and mortality related to heat stroke can be preventable by appropriate diagnosis and institution of prompt treatment. The diagnosis is based on identification of precipitating factors with presence of hyperthermia and CNS dysfunction.[11] Early reversal of hyperthermia by cooling, cardiovascular monitoring, and sedation and analgesia are the essential treatment, which in turn, prevent multiorgan failure syndrome.[11] It has been suggested that with appropriate treatment, survival can approach 100%.[12]

CONCLUSION

Heat stroke is the major disaster which involves mainly older males with co-morbidities. It is associated with

significant derangements in laboratory parameters and carries significant mortality as a consequence of multi-organ failure. To prevent such disasters in future and to establish a uniform strategy we need further and more extensive studies.

Conflict of Interest: The study has no conflict of interest to declare by any author.

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