Heat Stroke and Cocaine in an inner-city New York hospital

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Citation

Abstract

Background: The 2006 summer heat wave killed or contributed to the death of about 40 people in New York City.

Methods: A retrospective review of patients admitted with heat stroke during the summer of 2006 and the association with toxic drugs.

Results: Four patients were admitted with heat stroke. All of them had a temperature of more than 40 °C. Three of the patients were admitted with respiratory failure and were found to have used cocaine. Renal insufficiency, respiratory failure and rhabdomyolysis were common in our group. The Mean SAPS II score at admission was 57. All the four patients survived to hospital discharge.

Conclusions: The use of cocaine could decrease the threshold and predispose otherwise healthy patients during heat waves to develop heat stroke and multiorgan dysfunction. Urine toxicology and search for a toxic drug should be done routinely in patients with heat related illness presenting to the hospital.

INTRODUCTION

A heat wave is among the major natural events that can lead to deaths of hundreds of people within a few days or weeks (1,2,3). During the extreme heat wave in New York in August 2006, the ambient maximum daily temperature reached 102°F degrees and the minimum nocturnal temperature was in the high 90°F degrees for five consecutive days. Heat stroke claimed 40 lives in early August, the most in any heat wave since 1952.

There are several reports linking heat related illness and toxic drugs, especially illicit drugs like cocaine and opioids (2,3). The lethal effect of cocaine is unique among other illicit drugs because it is related not only to dose but also to cocaine's propensity to cause hyperthermia. Mortality rates for cocaine overdose increase substantially in hot weather (2,3).

The goal of our study was to evaluate all patients admitted with the diagnosis of heat stroke to the intensive care unit (ICU) and see the relationship with use of illicit drugs.

Methods: This was a retrospective review of all medical records of all patients admitted to the ICU with heat stroke during the summer of 2006. Baseline characteristics of patients and their predicted ICU mortality was compared using the new simplified Simplified Acute Physiology Score II (SAPS II), which is a standardized scoring system to predict ICU mortality based on 17 variables which includes 12 physiology variables, age, type of admission (scheduled surgical, unscheduled surgical, or medical), and three underlying disease variables.

Heatsroke is defined as hyperthermia of >40.5°C, neurologic signs and most of the time anhidrosis, without any other causes of fever—especially infections or endocrine disorders— other than the ambient heat.

RESULTS

During the summer of 2006, 468 patients were admitted to the ICU; four patients with heat stroke were identified, three males and one female. All of them had a temperature above 40 °C. Three of the patients were admitted with respiratory failure and were found to have used cocaine. Multiorgan dysfunction with renal insufficiency, respiratory failure and rhabdomyolysis were common in our group. The Mean SAPS II score at admission was 57. All the four patients
survived to hospital discharge.

The demographic characteristics and presentation can be seen in table 1.

**Figure 1**

Table 1: Characteristics and presentation of patients with heat stroke.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age &amp; Sex</th>
<th>Location</th>
<th>Body temperature</th>
<th>Peak Heat index</th>
<th>Implicated drug(s)</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>61 yrs</td>
<td>Galveston</td>
<td>103°F (40°C)</td>
<td>104°F (40°C)</td>
<td>Cocaine, Methadone</td>
<td>AMI, Respiratory failure</td>
</tr>
<tr>
<td>2</td>
<td>56 yrs</td>
<td>Closed car in the street</td>
<td>103°F (43°C)</td>
<td>104°F (41°C)</td>
<td>Cocaine, Methadone</td>
<td>AMI, Respiratory failure</td>
</tr>
<tr>
<td>3</td>
<td>68 yrs</td>
<td>Apartment, No air conditioning</td>
<td>103°F (41°C)</td>
<td>104°F (41°C)</td>
<td>None</td>
<td>AMI, Acute renal failure</td>
</tr>
<tr>
<td>4</td>
<td>41 yrs</td>
<td>Subway</td>
<td>103°F (41°C)</td>
<td>104°F (41°C)</td>
<td>Methadone, Cocaine</td>
<td>AMI, Respiratory failure</td>
</tr>
</tbody>
</table>

AME = alert mental status

Our four patients presented with a Glasgow Coma Scale (GCS) of less than 10, three of which required emergency endotracheal intubation for acute respiratory failure in the emergency department (ED). All patients were found to have acute renal insufficiency and two of three patients using cocaine developed ischemic hepatopathy.

All patients received treatment with external and internal cooling techniques in the ED. They were removed from the heat and placed in a cool area with air conditioning as well as aggressive fluid resuscitation and gastric lavage with cold water. The complications, management and outcome data of the patients can be seen in table 2.

**Figure 2**

Table 2: Evolution and management of patients with heat stroke.

<table>
<thead>
<tr>
<th>Patient</th>
<th>50% 1yr mortality (%)</th>
<th>Co existing conditions</th>
<th>Complications</th>
<th>Management</th>
<th>Hospital stay</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>56.4%</td>
<td>Fever, hypertension C.</td>
<td>Rhematoid arthritis, Respiratory failure</td>
<td>Cooling, IV Fluids, Alkalization, Hemodialysis</td>
<td>6 days</td>
<td>Survived</td>
</tr>
<tr>
<td>2</td>
<td>56%</td>
<td>None</td>
<td>Rhematoid arthritis, Respiratory failure, Shock, Ischemic Hepatopathy, GI bleeding, DIC, Bilirubin DVT</td>
<td>Cooling, IV Fluids, Alkalization, Blood products</td>
<td>16 days</td>
<td>Survived</td>
</tr>
<tr>
<td>3</td>
<td>55.3%</td>
<td>Hypertension, CAD</td>
<td>Rhematoid arthritis, HSTMT</td>
<td>Cooling, IV Fluids</td>
<td>9 days</td>
<td>Survived</td>
</tr>
<tr>
<td>4</td>
<td>65%</td>
<td>None</td>
<td>Rhematoid arthritis, Ischemic Hepatopathy</td>
<td>Cooling, IV Fluids</td>
<td>22 days</td>
<td>Survived</td>
</tr>
</tbody>
</table>

All patients survived and were discharged home without any neurological sequelae and without need for long-term dialysis.

**DISCUSSION**

From 1979 to 2003, excessive heat exposure caused 8,015 deaths in the United States. In urban areas of USA during warm weather the incidence of heat stroke has been reported as high as 20/100,00. The Heat index (HI) combines air temperature and relative humidity to determine an apparent temperature — how hot it actually feels. With HI higher than 130 °F there is a higher risk for heat stroke in high risk groups.

Heat illness is caused by an inability to maintain normal body temperature because of excess heat production or decreased heat transfer to the environment. Heat stroke arises when cellular injury is caused by excess body temperature. If the core temperature rises above 105.8°F (41°C) for more than a short time, thermal injury results. Proteins are denatured, and injured cells undergo apoptosis (programmed cell death) or necrosis. Even before injury takes place, an individual may suffer transient mental and physical impairment, which is called heat exhaustion.

It is important to recognize the difference between fever and heat stroke. Fever is a normal response, during which the core temperature remains under the control of the central thermoregulatory centers that reside in the hypothalamus and brainstem. When a pyrogenic stimulus is received, core temperature is elevated rapidly to a new set point that is regulated by normal mechanisms. Maximum febrile temperatures rarely exceed 105.8°F (41°C). In contrast, during heat illness, normal heat transfer mechanisms are overwhelmed and central thermoregulatory control is ineffective. Consequently, the core temperature can rise quickly to injurious levels. Some of the reported risk factors for heat related illness are:

- Increased endogenous heat production: exercise, febrile illness and drugs.
- Impaired heat dissipation: clothing, obesity, dehydration, extreme of ages, skin disorders and cardiovascular diseases.

The heat related illness ranges from heat exhaustion to heat stroke. Unlike heat exhaustion, heat stroke is a medical emergency and is usually classified as “classical” versus “exertional” depending of the demographics group affected.

It is due to impaired heat dissipation and commonly
associated with altered mental status (AMS) and temperatures above 41°C.

The “classical” type occurs in high-risk groups during periods of excessive hot temperatures. The “exertional” type usually occurs in healthy individuals during exercise in very hot and humid environment that results in heat production that can not be dissipated by sweating. Heat stroke and the relationship to medications or illicit drugs have been reported (1, 2). The most common culprits are cocaine, tranquilizers, alcohol, anti-cholinergic drugs, beta-blockers and diuretics. Three of our four patients were using cocaine. The underlying mechanisms mediating cocaine-induced hyperthermia are unclear and they have been attributed to:

- Hypermetabolic state (agitation with increased locomotor activity) that increases heat production
- Impaired heat dissipation by decreasing skin blood flow and sweating
- Decrease in heat perception
- Acute intoxication with cocaine may cause serotonin syndrome.

In most cases of serotonin syndrome, patients present with the triad of AMS, autonomic instability, and abnormal neuromuscular activity. Most cases are mild and self-limited (1, 2, 3). The clinical presentation of fatal intoxications is characterized by diaphoresis, tachycardia, muscle rigidity, rhabdomyolysis, metabolic acidosis, seizures, hyperkalemia, coagulopathy, and hyperpyrexia, with temperatures as high as 43.9°C like our three patients that were using cocaine(4).

Mortality correlates with core body temperatures, greater than 41.5°C results in a fatality greater than 50% (4). Although severe hyperthermia represents the most serious manifestation of serotonin syndrome, it is reported in about a third of cases (5).

The mechanism of serotonin syndrome is complex and involves interaction between the environment, the central catecholamine release, the hypothalamic-pituitary-thyroid-adrenal axis, the sympathetic nervous system, and skeletal muscle. Some studies have shown that elevating ambient temperature increases the toxicity of cocaine; this is supported by the finding that deaths from cocaine are more frequent during months with elevated ambient temperatures (6). Martinez et al reported eight patients with drugs associated heat stroke, 50% of them were using cocaine, 5% of them had rhabdomyolysis and the mortality was 25% (7).

The management of heat stroke includes removing the patient from the hot environment and reducing the temperature to 39°C as soon as possible. More rapid the cooling - the lower the mortality. Some cooling techniques include ice water soaks, immersion in ice bags, cooling blankets and evaporative techniques.

In addition, prevention /avoidance of organ dysfunction and airway and circulatory stabilization are paramount. Basics recommendations include:

- Airway protection
- Shivering & Seizures- Benzodiazepines
- Hypotension- fluids, pressors if needed.
- Exclude infectious causes for fever
- Cerebral edema- Mannitol, Dexamethasone
- Rhabdomyolysis /renal failure - Alkalinization /dialysis.
- Gastrointestinal & deep venous thrombosis prophylaxis

Medications have little efficacy in treating heatstroke. Muscle relaxants such as benzodiazepines and neuroleptic agents such as chlorpromazine have been used to inhibit shivering and as prophylaxis against seizures, but clinical trials are lacking.

Cooling usually is discontinued once the core temperature has reached 38°C (100.4°F) (9).

The reported mortality in heat stroke ranges from 15- 70% with a morbidity of up to 33% due to neurological sequelae (10).

In a large series reported by Misset et al. that included 345 patients with heat stroke admitted to ICU during the heat wave of 2003 in France, the mortality was 62.6% and was associated with occurrence of heatstroke at home or in a healthcare facility, a high SAPS II score, high body temperature, prolonged prothrombin time, use of vasoactive drugs within the first day, and patient management in an ICU without air conditioning (10). LoVecchio et al evaluated the outcome of 52 patients presenting to ED for hyperthermia; 18 (35%) were found to have ethanol /illicit
drugs present. Poor prognostic factors in this group were a low GCS, hyperthermia and hypotension. All our patients survived despite the presence of predictors for high mortality; in addition the Mean SAPS II score was 57 (expected mortality of 68%).

CONCLUSIONS

Projections over the next 100 years include higher maximum temperatures and heat indexes. Heat waves such as those observed in 2003 in Europe and in 2006 in New York are likely to recur. Our hospital is an 858-bed, acute-care facility, which serves the South and Central Bronx population of more than half a million people. High poverty levels, alcohol and intravenous drug abuse are common in this population. Early identification in the ED and aggressive management ideally in an intensive care unit is paramount. Recognizing the relationship between heat stroke and use of toxic drugs in our communities is important because this could a contributing factor for the development of multi-organ dysfunction.

The presentation of an otherwise healthy individual, without the usual risk factors for heat stroke should prompt the physician to look for use of drugs or illicit substances that decrease the threshold for hyperthermia related disorders. Heatstroke should be considered a multisystem disorder with a potential mortality as high as sepsis and septic shock.

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References

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