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Heat Stroke: A Diagnosis to Consider

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Abstract

Introduction: Differential diagnosis of hyperthermia can sometimes be challenging (taking into account infectious, endocrine, central nervous system, toxic and oncologic etiologies). Moreover, due to its low incidence, we are not aware of its implications, and do not always take into account every possibility.

Case presentation: We present a case of non-exertional heat stroke in a 65-year old Caucasian female, admitted to our intensive care unit with a four-day long altered level of consciousness. Diagnosis of heat stroke was yielded after performing different laboratory and imaging tests. Albeit prompt treatment, multiple organ failure followed by death occurred after 12 hours of admission.

Conclusion: This case highlights the importance for emergency physicians and intensivists to take into consideration heat stroke diagnosis when a patient presents with hyperthermia and neurological impairment, being able to distinguish it from other diseases based upon history and physical examination.

Keywords

hyperthermia; body temperature regulation; heat stroke; systemic inflammatory response syndrome; multiple organ failure; intensive care units

Introduction

Heat stroke is a life-threatening condition, with an extremely high mortality if not treated in an early manner. Heat stroke implies high temperature (at least 40ºC), and organic dysfunction (usually central nervous system involvement, especially cerebellum, and liver damage). However, development of severe multiple organ dysfunction, with acute renal failure, acute respiratory distress syndrome, disseminated intravascular coagulopathy and rhabdomyolisis can be seen [1-5]. However, due to its low incidence, critical care clinicians are generally less aware of its implications, and usually do not take this diagnosis into account when evaluating a patient presenting with hyperthermia and shock.

Case Presentation

A 65 year-old Caucasian female with a poor socioeconomic background and no family relatives was admitted in August(having being exposed to high temperatures at home)to our intensive care unit due to a four-day long altered level of consciousness. Her past medical history included allergy to amoxicillin, smoking, alcohol abuse, chronic hypertension (under treatment with atenolol and ramipril) and severe pulmonary obstructive disease (requiring home oxygen). On arrival to the emergency

department, the patient was diaphoretic with important lividness, with Glasgow score of 3 and arreactive pupils. Vital signs indicated high temperature (43.2ºC), heart rate of 160 bpm (atrial fibrillation), blood pressure of 99/57 mmHg and important desaturation (SpO_2 60% at room air). After endotracheal intubation, the performance of both head CT scan and chest X-ray revealed no significant findings.

On admission, the patient underwent femoral vein and artery cannulation, followed by body cooling placing ice-packs on neck, armpits and groins (monitoring her temperature with an esophageal thermometer). Vasoactive support with noradrenaline, along with fluid resuscitation with normal saline and sedoanalgesia (midazolam and fentanyl), were also initiated. Severe left ventricular dysfunction was observed at bedside echocardiography. Laboratory blood tests and cultures were obtained (blood, endotracheal aspirate, urine and lumbar puncture after 1000 ml of plasma transfusion):negative cultures, thrombocytopenia, severe coagulopathy, hepatocellular cytolysis and acute kidney injury were identified in the next hours of admission (Table 1), requiring initiation of continuous renal replacement therapy. Ammonia measurements were not performed.

Even though aggressive treatment was started from the very first moment, the patient developed refractory shock to high-dose vasopressors (noradrenaline and dobutamine, both removed after 30 minutes due to sustained ventricular tachycardia, followed by adrenaline perfusion, also with no response). Temperature control was not reached and, due to multiple organ failure, death occurred 12 hours after admission.

Discussion

Hyperthermia, defined as temperature greater than 38.3ºC, is due to failed thermoregulation, which differentiates from fever, where the body's temperature set point remains unchanged. Heat-related illness encompasses any pathological process caused by an acute increase in core temperature. Heat exhaustion is a lesser degree of heat stroke; its associates fatigue and tachycardia, without CNS symptoms.

Heat stroke takes place when the core body temperature rises surpassing the compensatory limits of thermoregulation. There are two types described in the literature: exertional heat stroke (occurring in a previously healthy young patient exercising in hot environments) and classical heat stroke (exposure of vulnerable patients –such as infants, elderly or patients with medical comorbidities or risk of social exclusion– to a high environmental temperature). Heat stroke pathophysiology is not completely elucidated, but could be defined as a form of thermoregulatory failure associated with a systemic inflammatory response with altered expression of heat-shock proteins, leading to a multiple organ dysfunction [1].

The two cardinal features of heat stroke are raised body temperature and neurological dysfunction (from mild neurological impairment to coma and seizures). Patients can present with multiple organ dysfunction, cardiovascular collapse, acute distress respiratory syndrome, acute respiratory alkalosis as mechanism of compensation of lactic acidosis, acute kidney failure, severe liver damage, hemorrhagic complications due to consumption coagulopathy that may be further compounded by hepatocellular damage. Neurological injury may not be transient: mental status changes may vary from impaired judgment and memory loss to delirium, seizures and coma; moderate to severe

impairment of neurological function at discharge from the hospital can reach 33%, with substantial functional impairment at discharge persisting 1 year after [2,6].

Mortality has been reported to be as high as 80% if not identified and treated early.

Management starts with rapid cooling and supportive care. Decreasing the body temperature below 38.9ºC within 30 minutes of presentation improves survival [6]. There are several ways to achieve it: increasing temperature gradient between the skin and the external environment (applying cold packs on the skin, especially on the neck, armpit and groin), increasing the water vapor pressure gradient (spraying the skin with cold water) and increasing conduction (fanning). In the last years newer technologies have been developed, such as Artic Sun Temperature Management System, Coolgard 300 Catheter Thermal Regulation System, or Rhino Chill Intra Nasal Cooling System. These devices have proved effectiveness when used in different scenarios, such as control of severe intracranial hypertension or targeted temperature management as part of the post-resuscitation care after cardiac arrest. It is relevant to point out that, in heat stroke, pharmacological agents that accelerate cooling are not helpful. Previous publications have shown that dantrolene is not useful, unless the patient has a past medical history or genetic predisposition to malignant hyperthermia [7,8]. Cooling therapy is usually stopped before the patient is normothermic. However, one of the risks of temperature management is hypothermia or rebound hyperthermia.

Tissue injury can continue to develop after cooling to normal body temperature in 25% patients (as cooling of the body to normal temperature does not result in suppression of pro-inflammatory mediators, such as cytokines and activated coagulation factors). Therefore, supportive care is crucial. Hemodynamic status is an important determination of initial fluid resuscitation. Administration of cold intravenous fluids is not usually recommended, although some centers have published good outcomes in patients admitted with heat stroke and treated with therapeutic hypothermia protocol used for cardiac-arrest patients, using cold fluids as the initial [4]. It is also unclear whether aggressive volume resuscitation is necessary (pulmonary edema as a known complication of fluid overload in these patients). Finally, initial permissive hypotension during cooling may be advisable, allowing gradual peripheral vasoconstriction and redistribution of the circulating volume centrally.

A case of classic heat stroke with a fatal denouement has been presented. Adverse reactions to drugs, as well as infectious diseases (negative cultures), neurologic conditions (such as intracranial hemorrhage) and endocrine derangements were discarded. Being an infrequent clinical presentation, there are no specific treatment guidelines. However, given its prognostic implications, general practitioners, emergency physicians and critical care specialists should bear in mind this possible diagnosis, since its prompt identification and management is essential to maximize a satisfactory outcome.

Table

Table 1: Changes in laboratory tests

Laboratory variables	On admission	4 hours after	10 hours after
White blood cell count (/mm3)	10,720	18,250	22,620
Hematocrit (%)	46.4	41.7	49.6
Platelets (/mm3)	205,000	143,000	55,000
Glucose (mg/dl)	232	83	52
Sodium (mmol/L)	134	137	135
Potassium (mmol/L)	6.9	5.4	5
Chloride (mmol/L)	98	103	102
Urea (mmol/L)	29	46	58
Creatinine (mmol/L)	1.27	2.36	4.02
pH (arterial blood gas)	7.09	7.22	6.8
pCO2 (arterial blood gas, mmHg)	86.1	41.8	36.2
pO2 (arterial blood gas, mmHg)	45	86.6	107
Bicarbonate (mmol/L)	17.3	18.3	5.9
Lactate (mmol/L)	5	2.9	10.6
Total creatinine kinase (U/L)	134	2690	13,760
Troponine (µg/L)	1.28	5.32	24.18
Total bilirubin (mg/dl)	0.8	0.9	2.2
ALT (U/L)	94	2440	11,580
AST (U/L)	156	5660	11,234
GGT (U/L)	48	64	97
Protrombine time (INR)	2.75	33.9	> 120
APTT (sec)	55.8	67.6	< 10
Fibrinogen (mg/dl)	544	447	66

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