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Case Report

Exercise-Related Heatstroke in the Intensive Care Unit: A Case Report

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ABSTRACT

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As climate changes accelerate, heatstroke becomes more and more often; both in its classical (non-exertional) form and in its exertional form. We hereby present a case of exertional heatstroke in a young man that was hospitalized in intensive care unit and discuss the relevant literature.

Keywords: Heatstroke, Excersise, Critical care

INTRODUCTION

During the last decade, Europe became known as a major climatic hotspot[1], due to the world's highest increase in average temperature; thus, associated with an increase in the frequency and intensity of heat waves and hot summers[2]. Furthermore, climate future projections reveal a tendency towards more increased average temperatures and intense weather conditions that will affect even more people[3].

Heat poses a major contributing morbidity and mortality factor to high-risk populations in Europe and worldwide. Almost 71,500 additional deaths occurred in Europe during the summer 2003[4]. A more recent study estimated that al-

most 62000 heat-related deaths in Europe between 30 May and 4 September 2022[5].

The most dangerous form of heat-related illness is heatstroke. Heat stroke is a life-threatening condition that occurs when thermorelatury fails. It is divided into exertional and non-exertional (classical) heat stroke. While classical heat stroke victims are usually elderly and vulnerable persons, exertional heat stroke, emerges in previously healthy young people exercising, usually in hot and humid climates, probably without being acclimatized. In the present article we present the management of a patient with severe exercise-related heatstroke and we discuss the implication of critical care and anesthesia in similar cases.

CASE REPORT

A 39-year-old man (body weight: 72kg, height – 178 cm) was transported comatose to a regional hospital. The man was participating in a marathon (41km) race and lost consciousness 3km before the finish line. At the time, ambient temperature (t^o) was about 32^{o}C, ambient moisture 49% with South-East light wind of about 2-3 BFT (Beaufort scale). Patient's medical history was free.

At Emergency Department (ED), his vitals were: heart rate (HR) 110bpm, blood pressure (BP) 140/70 mmHg, t^o – 38.8^{o}C, Glasgow Coma Scale (GCS)– E1/V1/M5, pupils -equal, reactive yet with mydriasis, and with increased muscle rigidity. Initially GCS improved to E4/V4/M5 with normalization of pupils' size. Yet soon after, local epileptic activity emerged with concomital neck rigidity, and eyeballs' left deviation. Initial management with fluid (Ringer's lactate 2lt), Diazepam 10 mg iv, paracetamol 1gr, levetiracetam 1500mg iv, a loading dose of 1200mg of sodium valproate over 30 min, followed by continuous infusion of 1200 mg/24h seized epileptic activity, but CGS decreased to E3/V1/M5. Diagnostic spinal puncture and brain computer tomography did not reveal any abnormalities. Emergency cardiological evaluation did not found major abnormalities: electrocardiography (ECG)-sinus rhythm (SR) (105 bpm) with right-bundle branch block (RBBB), transthoracic echocardiography (TTE)–ejection fraction 50% with no regional wall abnormalities, no valvular diseases or pericardial effusion. However, 2 consecutive troponin (Tn) measurements were abnormally increased (612 and 516 ng/ml)

The patient was intubated and transferred to our intensive care unit. He was presented with metabolic acidosis (pH -7.22, BE -2.7, $PaCO_2$ – 50.1 mmHg, Lactate – 3.05 mmol/lt, blood glucose 100 mg/dl) and good oxygenation status (PaO_2/FiO_2 ratio 410); but with leucocytosis, thrombocytopenia, acute kidney injury, acute liver injury, rhabdomyolysis, and elevated lactate dehydrogenase, troponin ,β-natriuretic peptid (BNP), procalcitonin (PCT); and a mean t^o of 37.8 (pick 39^{o}C) for the first

24h (Table 1). There was minimal need for hemodynamic support (noradrenaline civ 0.1 μγ/kg/min). On admission, APACHE II score was 23.

	ED	ICU day 1	ICU day 2	ICU day 3	ICU day 4	ICU day 5
WBC	17.569	19.780	16100	14630	10100	9460
PLT	222	111	100	98	81	95
INR	1.14	1.4	1.53	1.43	1.12	1.03
Fib		180	165	338	445	452
Cr	64	55	50	50	33	20
Ur	2.02	1.96	1.44	1.29	1.14	0.96
SGOT	51	161	284	342	252	92
SGPT	27	54	161	202	206	33
LDH	371	289	380	512	521	336
CPK	992	4630	4648	4590	3798	2725
Chol		0.9	1.3	1.7	0.61	0.5
Trop	612	522	93	49	22	9
BNP		154			40	
PCT		8.18	10.4	5.64	2.17	0.17
CRP	0.13	0.3	1	4.7	4.2	2.3

WBC-white blooc cells (k/ml), PLT- platelets (k/μl), INR-international normal ratio, Fib-fibrinogen (mg/dl), Cr-creatinine (mg/dl), Ur-urea (mg/dl), SGOT-aspartate aminotransferase (U/l), SGPT-alanine aminotransferase (U/l), LDH-lactade dehydrogenase (U/l), CPK–creatine phosphokinase (U/l), Chol-cholerythrin total (mmol/l), PCT procalcitonin (μg/l), CRP-C reactive protein (mg/dl).

Table 1. Course of selected laboratory parameters during ICU stay

However, his clinical status improved relatively fast. Thus, he was transferred to ward after 5 days and was discharged from the hospital 7 days later without any sequelae.

DISCUSSION

Heat stroke (HS) is the most severe form of heat-related illness. The other two more mild and frequent forms are heat fatigue and heat exhaustion. To date, no universally accepted definition of heat stroke exists. The most popular definition is the Bouchama's definition: core body temperature that rises above 40 C, accompanied by hot dry skin and central nervous system abnormalities, such as delirium, convulsions, or coma[6]. The same author proposed and a pathophysiology -based definition: heat stroke is a form of hyperthermia associated with a systemic inflammatory response that leads to a syndrome of multiorgan dysfunction, predominantly encephalopathy. Other authors, such as Pease et al or Misset et al , described different cut-off temperature limit (40.6^{o}C and 40.5^{o}C respectively) with including or omitting the "core" for the core body temperature.

The fullest definition comes from the work of Japanese Association for Acute Medicine (JAAM) and JAAM Heat Stroke Committee working group (HS-WS) in 2015-2016[7] (Table 2).

	JAAM	JAAM-HS
Environment	Exposure to high environmental temperature	Exposure to high environmental temperature
Nervous system	Impaired consciousness JCS ≥2, cerebellar symptoms, convulsive seizures	GCS score ≤ 14
Coagulopathy	Diagnosed as DIC by JAAM	JAAM DIC score ≥ 4
Renal Hepatic dysfunction	Follow-up after admission to hospital, hepatic or renal impairments requiring inpatient hospital care	Creatinine or total bilirubin levels ≥ 1.2 mg/dL

GCS Glasgow Coma Scale, JAAM Japanese Association of Acute Medicine, JAAM-HS-WG Japanese Association of Acute Medicine heat stroke committee working group, JCS Japan Coma Scale, DIC disseminated intravascular coagulation.

Table 2. JAAM Heat stroke definition criteria

The body temperature was not included in these diagnostic criteria because of several fatal cases of patients whose body temperatures were below 40°C that were observed in clinical practice. Examples of such cases are a report f Giersky et al. of a EHS with consequent liver failure in a 31-year-old male after running 5km at 21^{o}C at Norway, and of fatal EHS due to DIC and intracerebral haemorrhage in a 20-year-old male after 3h mountain biking in 26^{o}C heat in forest at Netherlands[7].

In general, there are two forms of HS: classical (induced by heat exposure in the absence of physical) exertional (EHS). EHS s induced by vigorous physical activity performed normally, but not always, in hot or humid environments. EHS is the third leading cause of mortality in athletes during physical activity with epidemiological data showing a mortality rate of upto 27% with survivors display long term negative health consequences ranging from neurological to cardiovascular dysfunction[8].

Several risk factors, such as dehydration, electrolyte imbalance, sex, body composition, age and previous clinical status are implicated in EHS; yet no definite indication exists that they increase EHS predisposition. Dehydration may lead to greater blood viscosity from decreased plasma volume which causes cardiac drift, leading to greater cardiac strain[9]. Obesity and even greater adipose tissue body probably contribute to an increased core temperature and decreased heat loss. They also cause chronic inflammation and metabolic disease, predisposing for higher EHS incidence[9]. In our case, we do not have information about the hydration status before the incidence, nor the pre-event physical activity status.

Differential diagnosis includes a plethora of conditions, such as meningitis, encephalitis, malaria, malignant neuroleptic syndrome, hy-

ponatremia, septic shock, thyroid crisis, acute myocardial infarction, malignant hyperthermia, and drug use or reactions. There is no single laboratory test that can confirm or exclude the diagnosis of heatstroke. In our case, laboratory features that support the diagnosis of heatstroke include leucocytosis, increased urea and creatinine, increased serum transaminases, increased creatinine kinase, coagulopathy, metabolic acidosis and rhabdomyolysis[10]. High serum PCT levels can be observed in heatstroke without any concomitant documented bacterial infection. The PCT is not a valid mortality predictor in heatstroke but could be an indicator of the severity of illness[11].

CONCLUSION

EHS consist of an emergency life-threatening condition with complex pathophysiology that may involve different organs and system. Rapid evaluation and early management are critical for successful outcomes. A high suspicion is also needed, event in the cases in which enviromental conditions look “safe”.

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