Case Report

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Heat Stroke in a COVID-19 Field Hospital

Farzin Vajifdar[1], Renaldo Pavrey[2]

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Author's Affiliation: [1]Associate Consultant, Department of Emergency Medicine, Holy Family Hospital, Mumbai 400050, Maharathra, India, [2]Lead Consultant & Head, Department of Emergency Medicine, Prince Aly Khan Hospital, Mumbai 400010, India.

Corresponding Author: Renaldo Pavrey, Lead Consultant & Head, Department of Emergency Medicine, Prince Aly Khan Hospital, Mumbai 400010, India.

E-mail: renaldo.pavrey@gmail.com

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Abstract

Heatstroke is a medical emergency warranting critical, time sensitive interventions. Due to its infrequent occurrence inside medical facilities, and symptoms overlapping with other commonly seen conditions in an emergency setting, the recognition of heatstroke is often undermined.

With the ongoing SARS CoV-2 pandemic, Several makeshift field hospitals have been set up in Mumbai, India. Heatstroke is not a common occurrence in patients infected by the SARS CoV-2 virus. More so, it is almost never anticipated in hospitalized individuals.

Our case report aims to highlight the unusual presentation of a heat illness occurring inside a field hospital dedicated to managing Coronavirus disease (COVID-19). We hope to sensitize the clinician to this rare event, and subsequently aim towards preparedness and mitigation for the future strategizing of such field hospitals.

Keywords: Heatstroke, COVID-19, field hospital

Introduction

Heat illnesses are a continuum of diseases ranging from mild to severe, with heatstroke being at the extreme end of the spectrum. Heatstroke is an acute, life threatening emergency, which left untreated, is universally fatal[1]. It is characterized by hyperthermia (core body temperature exceeding 40° C or 104° F) and accompanying CNS dysfunction.

There are no diagnostic tests for heatstrokes, and the list of differential diagnoses is extensive. Diagnosis is therefore established by history, clinical examination, and keeping a high index of suspicion. Mainstay of treatment is early and aggressive cooling[2]. Delay in diagnosis causes a significant increase in mortality, thereby making it imperative for clinicians to familiarize themselves with the signs, symptoms and predisposing factors for heatstrokes.

With the ongoing COVID-19 pandemic, multiple makeshift hospitals have been set up to accommodate the exponential rise in cases. While these hospitals are well equipped in terms

of modalities of treatment, it is important to bear in mind that these makeshift structures are devoid of certain attributes, thereby causing impediments specific to these sites. The harsh ambient temperatures in summer, coupled with the tented structure and inadequate cooling against the background of a viral illness in elderly patients, are ideal precipitants for heat illness.

Case presentation

A 64-year-old man was shifted to the Emergency Care Unit (ECU) of a COVID-19 field hospital in view of persistent tachypnea and high-grade fever. He had been admitted to the non-oxygen ward of the COVID-19 field hospital two days ago, after testing positive for SARS CoV-2.

The patient was hypertensive, controlled with Amlodipine 5 mg daily.

On admission to the ward, symptomatic treatment had been initiated for his mild illness. His fever, however, had not responded to intravenous Paracetamol, and remained sustained at Day 2 of admission, shortly before the tachypnea set in.

On initial evaluation in the ECU, his vital signs were:

Heart rate (HR) – 140 beats/minute and regular, blood pressure (BP) – 110/60 mmHg, respiratory rate (RR) – 44 breaths/minute, SpO2 – 92 % on room air, random blood glucose – 142 mg/dl, body temperature – 105° F, Glasgow Coma Scale E3 V4 M6 (GCS – 13).

Fig. 1

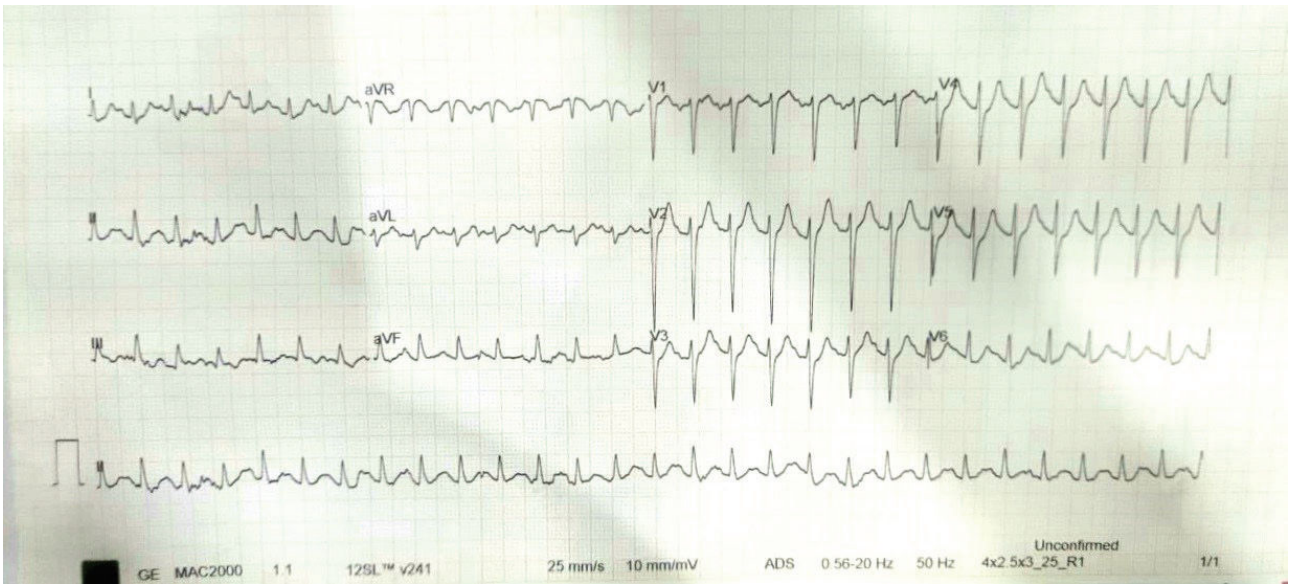

Table 1

Test	Value
pH	7.513
pCO_2	16.6
pO_2	67.3
BEecf	9.9
BEb	41.3
HCO_3	13
TCO_2	11.1
SO_2 %	93.8
Na^+	128.6
K^+	3.96
Cl^+	92.1
Ionised Ca^{++}	0.627
Lactate	8.73

On physical examination, the patient was profusely diaphoretic and flushed, with tongue and oral mucosa evidently dry. The remainder of the examination proved to be grossly normal.

Investigations

A 12-lead electrocardiogram (Figure 1) showed a sinus rhythm with tachycardia.

An arterial blood gas analysis (Table 1) done on room air, revealed type 1 respiratory failure with primary respiratory alkalosis and concomitant metabolic acidosis. The serum lactate was 8.73 mmol/L.

Bedside ultrasound showed a collapsed inferior

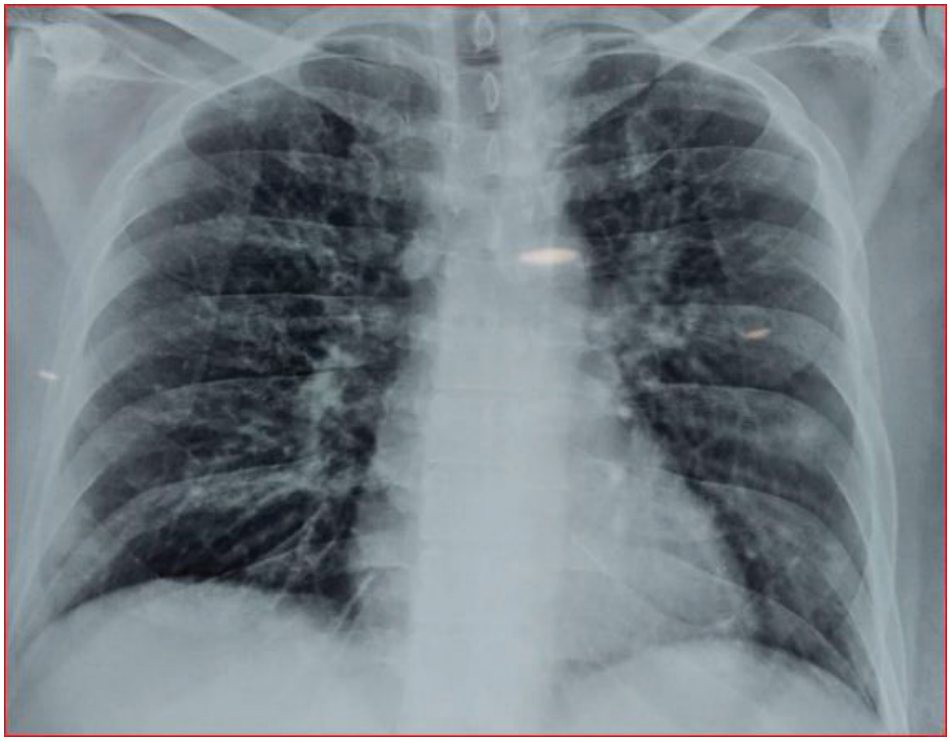

Fig. 2

vena cava. The Troponin-I was within the normal range. His complete blood count was: hemoglobin 11.2 g%, white cell count 10,400 and platelet count 2,73,000. Tests for malaria and dengue were both negative. His creatinine was 1.32 g%; electrolytes were sodium 129.6, and potassium 4.2 mmol/L. SGOT and SGPT were elevated at 187 and 193 respectively. The creatine kinase (CK) level was normal at baseline. Urinalysis was negative for ketones and myoglobin. His X-ray Chest (Figure 2) did not reveal consolidation or interstitial fluid.

Differential diagnosis

- COVID-19 disease progression to severe category of illness.
- Cerebral malaria.
- Sepsis.
- Pulmonary embolism.
- Malignant hyperthermia.
- Neuroleptic malignant syndrome.
- Encephalitis
- Impending heatstroke.

Treatment

The patient was attached to a telemetry monitor and started on high flow nasal oxygenation (HFNO) therapy with FiO2 of 35% and flow rate at 45 liters/minute. Fluid resuscitation was initiated with 0.9% saline via two l6 gauge peripheral IV lines. Blood cultures were collected, and antibiotics escalated.

Patient's clothing was removed, and rapid cooling measures were initiated by placing ice packs in the axillae and groin, covering the patient with a cold water-soaked bed sheet, and placing portable fans adjacent to the bed.

Outcome

The patient had transient hypotension in the ECU, which responded to crystalloid resuscitation and rapid, aggressive cooling measures.

After half an hour, the patient's parameters were:

HR – 108 beats/minute, BP – 100/60 mmHg, RR – 32 breaths/minute, SpO2 – 98% on HFNO, body temperature – 100.4° F, GCS – 15. Cooling measures and intravenous fluids were continued.

Follow-up

The patient was shifted to the High Dependency Unit (HDU) for further management.

Serial renal biomarkers showed recovering kidney injury and progressively resolving

hypovolemic hyponatremia. An arterial blood gas after 6 hours showed improving metabolic acidosis, with a reduction in lactate levels (Lactate 4.6 mmol/L).

He was weaned off HFNO within 24 hours, and kept on supplemental oxygen at 2 liters/minute via nasal cannula. Blood culture at 48-hours was negative for the growth of any pathogenic organism.

Discussion

We describe the case of a 64-year-old man positive for SARS CoV-2 who presented to us on Day 6 of illness, and Day 3 of hospitalization, with hyperthermia and tachypnea. His laboratory results and clinical findings were consistent with multi-organ affliction.

The patient's chest X-ray was grossly normal, without any evidence of progression of COVID-19 disease. The proportion of Troponin-I was not high; thus, we did not suggest coronary syndrome. A bedside transthoracic echocardiogram ruled out a massive / sub-massive pulmonary embolism.

Several other differential diagnoses, such as malignant hyperthermia, malignant syndrome, and rhabdomyolysis, were considered. As the patient was not taking any anti-psychotic drugs, malignant hyperthermia and neuroleptic malignant syndrome were ruled out. Rhabdomyolysis was also subsequently ruled out, owing to normal CK levels and absence of myoglobinuria.

Our patient presented with acute altered consciousness, tachypnea and hyperthermia with a body temperature >104° F. These are cardinal symptoms consistent with a heatstroke. Classic, non-exertional heatstrokes occur during environmental heat waves, and are more commonly seen in extremes of age and chronically ill individuals.[3]

Clinically, while anhidrosis is generally present, sweating occurs in over half the patients with heatstroke;[4] some patients may actually present with profuse diaphoresis. When present though, anhidrosis is often a late sign in heat stroke. Heart rate at or above 140 beats/minute is frequently seen in heatstrokes. This is because the cardiac output increases by 3L/minute for each 1°Celsius elevation in core body temperature.[3]

Antipyretics do not have a role in the treatment of heatstrokes since the thermoregulatory mechanism in the anterior hypothalamus is overwhelmed.[2] Instead, conduction and evaporative cooling mechanisms are employed like placing ice packs in the axillae and groin, covering the patient in ice water-soaked sheets, spraying the patient with cold water and placing fans adjacent to the patient. Retroactively, we can state that this was not only evident in our case, but also therapeutically successful.

Tachypnea in heatstrokes is accompanied by a respiratory alkalosis (hyperthermia induced hyperventilation) with PaCO2 often <20 mmHg, and lactic acidosis on the blood gas[5]. Common complications include hypoglycemia, elevated liver enzymes and renal failure; the latter usually occurring secondary to hypovolemia, seen in nearly one third of the cases.[3] Adult respiratory distress syndrome may be seen due to direct lung injury from the heat.

To the best of our knowledge, there are no case reports highlighting the manifestation of heatstroke in a case of COVID-19 disease in the presence of environmental factors that make such patients highly susceptible to heat illness.

Conclusion

Identifying heatstroke in a patient with COVID-19 disease is challenging. This is because the pathognomonic signs seen in heatstroke overlap with those seen in COVID-19 disease. On a microvascular level, progression of a heatstroke resembles sepsis. This makes it especially conducive to skip the diagnosis of heatstroke in favour of sepsis / septic shock.

Along with the clinical profile of the patient, environmental factors must be taken in toconsideration when arriving at a diagnosis. Our patient was admitted in a field hospital at the peak of summer (May 2021), when the average daily temperature was 91° F with a humidity of 74%. Cooling, understandably posed a challenge. The inherent properties of the tented structure meant that it not only heated up significantly during day time, but also retained the heat for a longer duration. Since the heat could not be effectively dissipated, the temperature in the wards remained elevated for a prolonged duration.

Portable fans were provided in the wards to combat the heat; however, these were shared between patients, and there were further disadvantages, like limited access to electrical power points and extension cords. The internal environment in the wards juxtaposed against the patient's clinical profile, providing an archetypal setting for a heatstroke. Prompt recognition and

rapid cooling in the Emergency Care Unit ensured a favourable outcome in this case.

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