

CASE REPORT

A Rare Case of Heat Stroke Complicated by Psychotropic-Induced Impaired Thermoregulation

Uvesh M. Vaja¹, Tushau Prasad²

HOW TO CITE THIS ARTICLE:

Uvesh M. Vaja, Tushau Prasad. A Rare Case of Heat Stroke Complicated by Psychotropic-Induced Impaired Thermoregulation. Ind J Emerg Med. 2025; 11(4): 263-266.

ABSTRACT

Background: Heat stroke is a medical emergency marked by core temperature $>40^{\circ}\text{C}$ and neurological dysfunction.¹ Individuals on psychotropic medications are especially vulnerable due to impaired thermoregulation and altered behavioural heat responses.^{3,7} Recognition and rapid intervention are critical to prevent morbidity and mortality.⁵

Case Presentation: A 28-year-old male with major depressive disorder on multiple psychotropics developed classical heat stroke while working outdoors. He presented with hyperthermia (106°F), seizure like activity, aspiration pneumonia, altered mental status, and respiratory failure. Prompt emergency management included airway protection, rapid cooling, and ventilator support. Imaging revealed aspiration pneumonia, and labs indicated early acute kidney injury and mild rhabdomyolysis. With intensive multidisciplinary care, including psychiatry input, the patient fully recovered and was discharged in stable condition.

Conclusion: This case underscores the compounded risks of exertional heat stroke in psychiatric patients and highlights the importance of rapid recognition, aggressive supportive care, and coordinated management in such high-risk populations.⁶

KEYWORDS

• Heat stroke • Aspiration Pneumonia • Antipsychotic drugs • Thermoregulation
Psychiatric polypharmacy • Emergency Medicine

INTRODUCTION

Heat stroke is a life-threatening condition characterized by core body temperature above

40°C (104°F) and central nervous system dysfunction. It is classified into exertional and non-exertional types, with the former often

AUTHOR'S AFFILIATION:

¹ Associate Consultant, Department of Emergency Medicine, Reliance Foundation Hospital, DAOH, Gujarat, India.

² Head of Department, Department of Emergency Medicine, Reliance Foundation Hospital, DAOH, Gujarat, India

CORRESPONDING AUTHOR:

Uvesh M. Vaja, Associate Consultant, Department of Emergency Medicine, Reliance Foundation Hospital, DAOH, Gujarat, India.

E-mail: uveshvaja@gmail.com

➤ Received: 01-07-2025 ➤ Accepted: 13-08-2025



Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (<http://www.creativecommons.org/licenses/by-nc/4.0/>) which permits non-Commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the Red Flower Publication and Open Access pages (<https://www.rfppl.co.in>)

affecting young individuals under physical stress. Psychotropic medications can impair thermoregulation, increasing susceptibility to heat-related illnesses. We report a case of severe heat stroke in a young male with major depressive disorder on psychotropic polypharmacy.

Case Presentation

A 28-year-old male with a history of major depressive disorder on multiple antidepressants and antipsychotic medications was brought to the Emergency Department after collapsing at work. He developed sudden uneasiness followed by shivering and tonic-clonic movements of all four limbs, suggestive of seizure activity. He had an episode of vomiting followed by aspiration and was initially taken to the occupational health center, where he became drowsy.

On arrival at our hospital, he was unresponsive, febrile, and gasping. Immediate intubation was done in the ER using a 7.5 mm endotracheal tube, and Ryle's tube and Foley catheter were inserted. Initial vitals: BP 130/90 mmHg, HR 190/min, Rectal Temp 106°F, RR 8/min, SpO₂ 70% on room air, RBS 100 mg/dL. CNS exam showed post-ictal state with pinpoint pupils not reacting to light. Chest auscultation revealed bilateral crepitations.

Rapid temperature control measures were initiated: cold saline boluses, ice packs, and cooling blanket. Initial investigations showed: Potassium 3.1 mmol/L, Creatinine 1.8 mg/dL, Platelets 19,000, CPK 580 U/L. NCCT Brain was normal. HRCT Chest showed bilateral aspiration pneumonia. 2D Echo was normal (LVEF 60%).

The patient was shifted to the ICU, where temperature normalized to 98°F within a few hours. He was awake on ventilator support and managed with sedation, broad-spectrum antibiotics, antiepileptics, and supportive care. Blood cultures showed no growth. Gradual clinical improvement was observed: he was weaned off the ventilator, transitioned to non-invasive ventilation (NIV) and then to nasal prongs with SpO₂ maintained between 96–98% on 1 L/min oxygen. Awake proning sessions were performed to optimize ventilation.

Renal Function: Initial serum creatinine at the time of ER admission was elevated at 1.80 mg/dL, suggestive of early acute kidney injury (AKI), likely secondary to dehydration and

heat-related volume depletion. With careful fluid resuscitation (guided by IVC collapsibility on point-of-care ultrasound), serum creatinine levels improved steadily:

- 12 hours: 1.41 mg/dL
- 48 hours: 1.33 mg/dL
- 72 hours: 1.13 mg/dL

Trend of Renal Function

Timepoint	Serum Creatinine (mg/dL)
Admission (Day 0)	1.80
12 hours	1.41
48 hours	1.33
72 hours	1.13

***Table X:** Trend of serum creatinine showing gradual improvement with IV fluid therapy*

Arterial Blood Gas (ABG) Trend and Comparison

The patient's ABG parameters were closely monitored across ICU stay and showed progressive improvement. Below are representative ABG reports from Day 0 (on admission), Day 1 (after 24 hours), and Day 3 (after 72 hours).

Test Name	Result	Unit	Reference Interval
LAB-BC021 (Result Provisional)			
Sample Type (Result Finalized)	Arterial blood		
B Eact (Result Finalized)	-11	mmol/L	-2 to +3
B Eact (Result Finalized)	-12.2	mmol/L	-2 to +3
CA++ (Result Finalized)	1.09	mmol/L	1.15-1.33
Cl- (Result Finalized)	115	mmol/L	98-107
COHb (Result Finalized)	-0.2	%	0.0-3.0
HCO3 (Result Finalized)	14.60	mmol/L	18-23
K+ (Result Finalized)	2.9	mmol/L	3.5-5.1
METHb (Result Finalized)	0.7	%	0.01-5
Na+ (Result Finalized)	138	mmol/L	136-145
pCO2 (Result Finalized)	31.00	mmHg	32-48
Ph (Result Finalized)	7.28		7.35-7.45
p O2 (Result Finalized)	193.00	mmHg	83-108
sO2 (Result Finalized)	98.30	%	94-98
sT. HCO3- (Result Finalized)	15.9	mmol/L	22-24
t HB (Result Finalized)	12.6	g/dl	11.5-17.8

Test Name	Result	Unit	Reference Interval
Lactate (Result Finalized)	2.00	mmol/L	Arterial blood: <1.3 Venous Blood: = 1.7

Table 1: ABG on Day 0 – Severe metabolic derangement and lactic acidosis

Test Name	Result	Unit	Reference Interval
LAB-BC021 (Result Provisional)			
B Eact (Result Finalized)	-8.4	mmol/L	-2 to +3
B Eact (Result Finalized)	-9.5	mmol/L	-2 to +3
Ca++ (Result Finalized)	1.07	mmol/L	1.15-1.33
Cl- (Result Finalized)	113	mmol/L	98-107
HCO ₃ (Result Finalized)	16.20	mmol/L	18-23
K+ (Result Finalized)	4.3	mmol/L	3.5-5.1
Na+ (Result Finalized)	138	mmol/L	136-145
pCO ₂ (Result Finalized)	30.20	mmHg	32-48
pH (Result Finalized)	7.33		7.35-7.45
pO ₂ (Result Finalized)	72.50	mmHg	83-108
sO ₂ (Result Finalized)	93.70	%	94-98
sT. HCO ₃ - (Result Finalized)	17.7	mmol/L	22-24
t HB (Result Finalized)	12.2	%	11.5-17.8
Lactate (Result Finalized)	1.50	mmol/L	Arterial blood: <1.3 Venous Blood: = 1.7

Table 2: ABG on Day 1 – Improving metabolic profile with mild acidosis

Test Name	Result	Unit	Reference Interval
LAB-BC021 (Result Provisional)			
pH (Result Finalized)	7.43		7.35-7.45
pCO ₂ (Result Finalized)	33.70	mmHg	32-48
pO ₂ (Result Finalized)	56.30	mmHg	83-108
ctHb (Result Finalized)	10.8	g/dl	11.5-17.8
sO ₂ (Result Finalized)	88.00	%	94-98
FO ₂ Hb (Result Finalized)	87.1	%	
FCO ₂ Hb (Result Finalized)	0.4	%	0.5-1.5
FHHB (Result Finalized)	11.9	%	
FMetHb (Result Finalized)	0.6	%	0.0-1.5

Test Name	Result	Unit	Reference Interval
cK+ (Result Finalized)	3.5	mmol/L	3.5-4.5
cNa+ (Result Finalized)	138	mmol/L	136-145
cCa ²⁺ (Result Finalized)	1.13	mmol/L	1.15-1.33
cCl- (Result Finalized)	107	mmol/L	98-107
cGlu (Result Finalized)	88	mg/dL	65-95
cLac (Result Finalized)	1.20	mmol/L	Arterial blood: 0.3-0.7
ctBil (Result Finalized)	1.8	mg/dL	
cBase(B)c (Result Finalized)	-1.4	mmol/L	
cBase(Ecf)c (Result Finalized)	-1.9	mmol/L	
cHCO ₃ - (P)c (Result Finalized)	22.40	mmol/L	22-28
cHCO ₃ -(P,st)c (Result Finalized)	23.1	mmol/L	
AnionGapc (Result Finalized)	9	mmol/L	8-16
AnionGap,k+c (Result Finalized)	12.6	mmol/L	
ctCo ₂ (P)c (Result Finalized)	52.6	%	

Imaging Findings

High-resolution computed tomography (HRCT) of the chest revealed bilateral lower lobe consolidations with air bronchograms, suggestive of aspiration pneumonia (see Figure 1). These findings correlated with the patient's clinical course of vomiting, reduced consciousness, and hypoxia on admission.

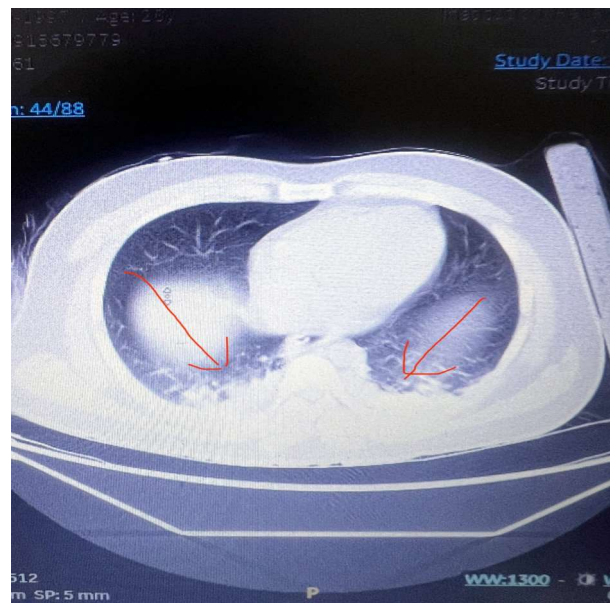


Figure 1: HRCT Chest showing bilateral lower lobe infiltrates consistent with aspiration pneumonia

DISCUSSION

Heat stroke is a medical emergency^{1,5} with multi-organ involvement. CNS dysfunction and hyperpyrexia are hallmark features.¹ Patients on antipsychotic or antidepressant medications have impaired thermoregulation^{3,7}, reduced thirst perception, and decreased sweating predisposing them to heat-related illnesses.

In this case, the patient developed classical heat stroke with an initial core temperature of 106°F, unresponsiveness, and seizures. Neurologically, the presentation mimicked a seizure disorder, but the context and rapid fever reduction pointed toward hyperthermia-induced encephalopathy.

Additionally, the patient demonstrated early signs of renal injury, evidenced by an elevated serum creatinine of 1.80 mg/dL on admission. Heat stroke can cause renal dysfunction via direct thermal injury, hypoperfusion, and rhabdomyolysis.⁵ However, mildly elevated CPK (580 U/L) and rapid improvement with fluid therapy suggested pre-renal azotemia.

Aspiration pneumonia further complicated the course, likely due to seizure and vomiting. Broad-spectrum antibiotics and ventilatory support were initiated. HRCT confirmed bilateral lower lobe involvement. Multidisciplinary management, including psychiatric and neurologic input, was critical in recovery.

CONCLUSION

This case highlights the need for heightened clinical vigilance for heat stroke in patients on antipsychotic medications. Timely diagnosis, aggressive temperature control, and supportive ICU care can result in complete recovery, even in severe presentations.

REFERENCES

1. Bouchama A., Knochel J.P. Heat stroke. *N. Engl J Med.* 2002; 346(25): 1978–88.
2. Leon L.R., Helwig B.G. Heat stroke: Role of the systemic inflammatory response. *J Appl Physiol.* 2010; 109(6): 1980–8.
3. Raza M., Qadir T., Khan S. Antipsychotic drugs and thermoregulation: implications for emergency care. *Emerg Med Int.* 2014; 2014: 123091.
4. Sawka M.N., Wenger C.B., Pandolf K.B. Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In: *Handbook of Physiology: Environmental Physiology.* 1996.
5. Epstein Y., Yanovich R. Heatstroke. *N. Engl J Med.* 2019; 380(25): 2449–59.
6. House M.M., Wertheimer A.C. Emergency department management⁶ of heat stroke: a review. *Emerg Med Pract.* 2018; 20(7): 1–23.
7. Bassi G., DiGiovanni C.M. Psychotropic medications and heat illness risk: a narrative review. *J Psychopharmacol.* 2021; 35(9): 1023–32.