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Refractory Central Hyperthermia after Pontine Hemorrhage : Rhabdomyolysis and AKI

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BACKGROUND

Central fever is a noninfectious hyperthermic response after acute brain injury, attributed to disruption of hypothalamic or brainstem thermoregulatory pathways. Severe dysregulation may cause extreme hyperthermia and life-threatening systemic complications. We report a case of pontine intracerebral hemorrhage (ICH) complicated by refractory hyperthermia leading to rhabdomyolysis and acute kidney injury (AKI) requiring intensive care.

CLINICAL COURSE

A 41-year-old woman with acute dorsal pontine ICH (4.6 mL) with fourth-ventricle extension was transferred to the Department of Rehabilitation Medicine (***Figure 1***). On HD 11, she developed hyperthermia (Tmax 39.0°C) with only mild inflammatory marker elevation, suggesting central fever. TTM targeting 37°C was applied from HD 13 to HD 20. After discontinuation, low-grade fever persisted and intermittent spikes were treated symptomatically. On HD 46, increased sputum production and oxygen demand led to antibiotic treatment for suspected pneumonia. Despite this, on HD 51–52 she developed refractory hyperthermia up to 42.0°C with prolonged shivering, followed by oliguria, dark urine, marked CK and myoglobin elevation, and azotemia, consistent with rhabdomyolysis-associated AKI (***Figure 2***). She required ICU care, reinitiation of TTM, and supportive management. After 4 weeks, she stabilized without recurrent fever and was discharged after rehabilitation (***Figure 3***).

HD 11	Hyperthermia with mild inflammation → **central fever suspected**
HD 13–20	TTM was initiated and maintained (target 37°C)
HD 46	Respiratory worsening → **suspected pneumonia**
HD 51–52	**Refractory hyperthermia**, prolonged shivering, **rhabdomyolysis/AKI**
ICU / recovery	**TTM restarted** : stabilized without recurrent fever

CONCLUSION

Pontine hemorrhage likely disrupted descending thermoregulatory pathways, resulting in refractory hyperthermia despite minimal inflammatory response. Prolonged extreme hyperthermia with sustained shivering probably triggered massive rhabdomyolysis and subsequent AKI. Early recognition of central thermoregulatory failure and timely initiation or reinitiation of TTM may help prevent catastrophic systemic complications in similar patients.

Key message: Central hyperthermia, leading to rhabdomyolysis and AKI. Early recognition and timely targeted temperature management may prevent severe systemic complications.

FIGURES

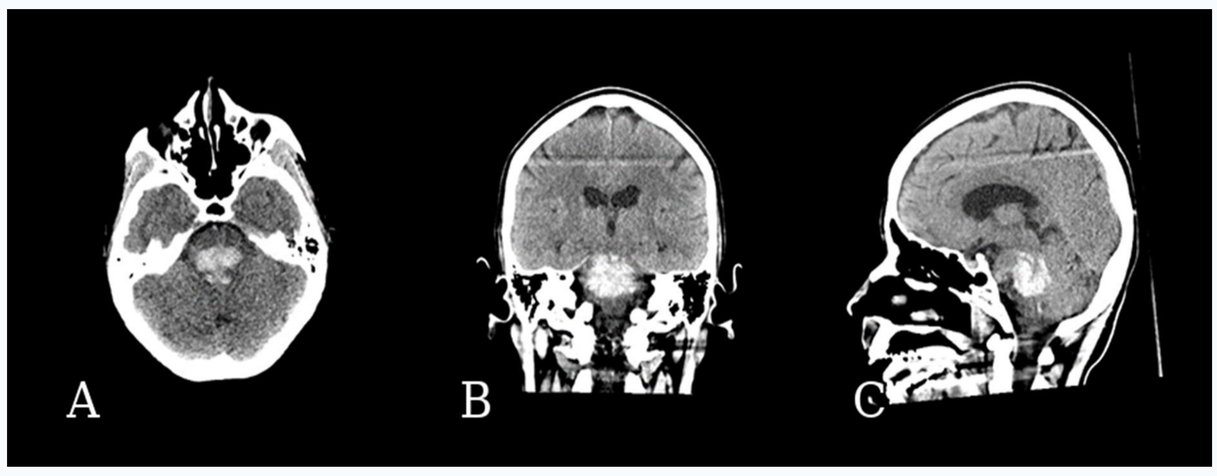

Figure 1. *non-contrast Brain CT images CT (A) Axial, (B) Coronal, and (C) Sagittal*

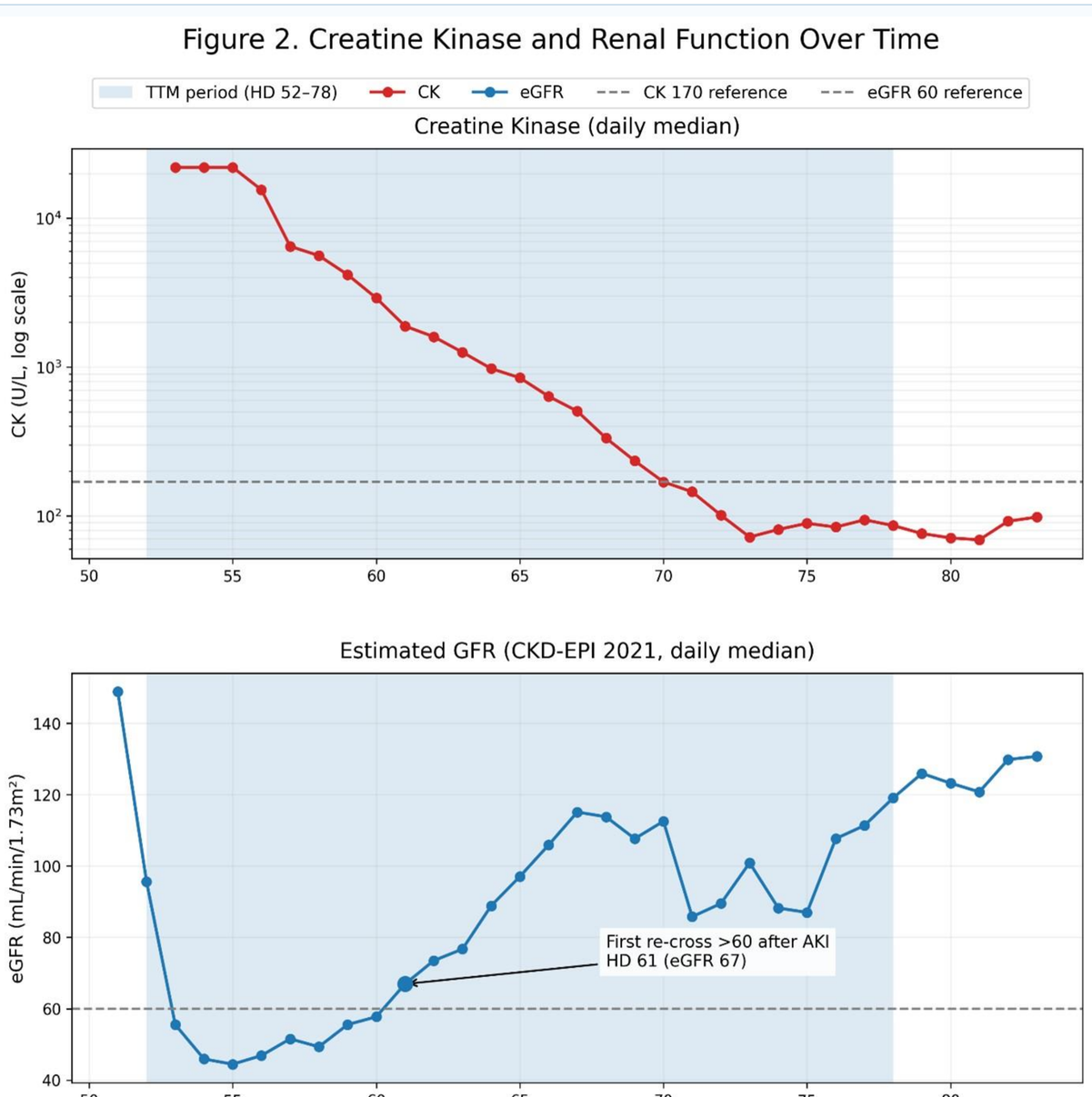

Figure 2. *CK and renal function over time Daily median serum creatine kinase and eGFR. CK peaked above 22,000 U/L and renal function transiently declined after extreme hyperthermia, then improved during TTM.*

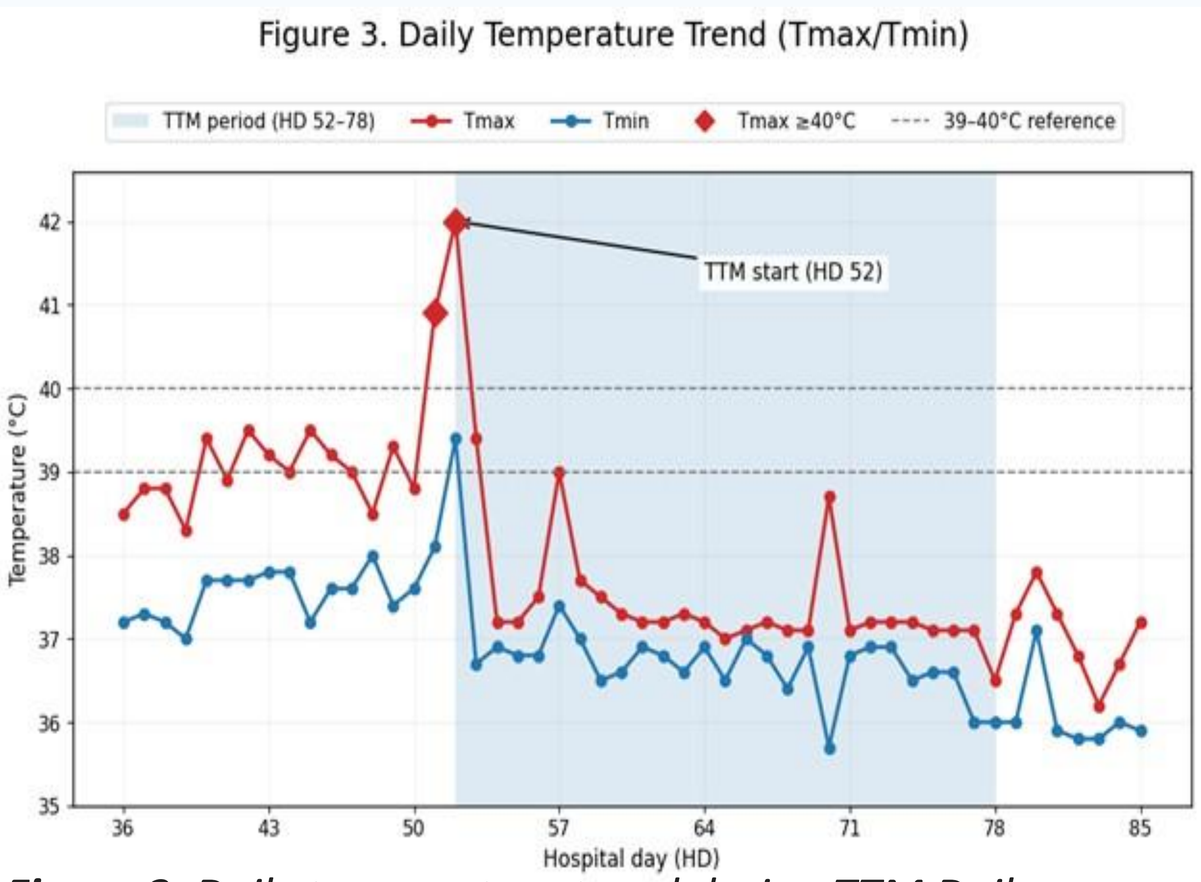

Figure 3. *Daily temperature trend during TTM Daily maximum (Tmax) and minimum (Tmin) body temperatures are plotted by HD. Temperature normalized after TTM was restarted on HD 52.*