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Paroxysmal Sympathetic Hyperactivity Following Severe Traumatic Brain Injury : A Case Report

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Introduction

Paroxysmal sympathetic hyperactivity (PSH) complicates severe traumatic brain injury (TBI) recovery. It manifests as transient sympathetic overactivity, including tachycardia, hypertension, hyperthermia, and abnormal motor posturing. It stems from lost inhibitory control over spinal sympathetic circuits following cortical or subcortical damage. Often misdiagnosed as sepsis or seizures, PSH prolongs hospitalizations and testing. We report a severe TBI case, highlighting the PSH assessment measure (PSH-AM) for diagnosis and multimodal management targeting sympathetic and nociceptive triggers.

Case report

A healthy 16 years old male presented comatose after a pedestrian accident on October 10, 2025. Brain CT showed intracerebral hemorrhage in the left frontal and right temporal lobes with subarachnoid hemorrhage; MRI confirmed diffuse axonal injury (Fig. 1). During his intensive care unit stay for acute neurosurgical management, he remained in a semi-comatose state. Persistent fever (>38°C) was unrelieved despite empirical antibiotics for suspected infection.

Upon transfer to rehabilitation medicine on October 27, 2025 his consciousness was severely impaired, presenting as unresponsive wakefulness syndrome (JFK coma recovery scale-revised total score: 7). Persistent fever, tachycardia, and increased spasticity, with low infection probability on labs, prompted PSH suspicion.

PSH-AM evaluation yielded a clinical feature scale (CFS) score of 7 and a diagnostic likelihood tool (DLT) score of 7, totaling 14 (possible PSH) (Table 1). Intervention prioritized pain control and sympathetic modulation. A fentanyl patch was applied, with intermittent intravenous pethidine for breakthrough surges. Baclofen was initiated to manage spasticity, and propranolol for sympatholysis. Autonomic instability initially improved.

However, on November 15, 2025 fever, tachycardia, and rigidity resurged, although he was receiving antibiotics for a urinary tract infection and inflammatory markers were improving. CT ruled out new infections but revealed significant fecal stasis, suggesting that the exacerbation was PSH triggered by visceral nociception from constipation. Following aggressive bowel management (rectal tube decompression), the sympathetic storm resolved, and the CFS score dropped to 0–1. No further autonomic instability occurred. Residual spasticity was attributed to primary brain injury; thus, baclofen was maintained, while propranolol and the fentanyl patch were tapered and discontinued.

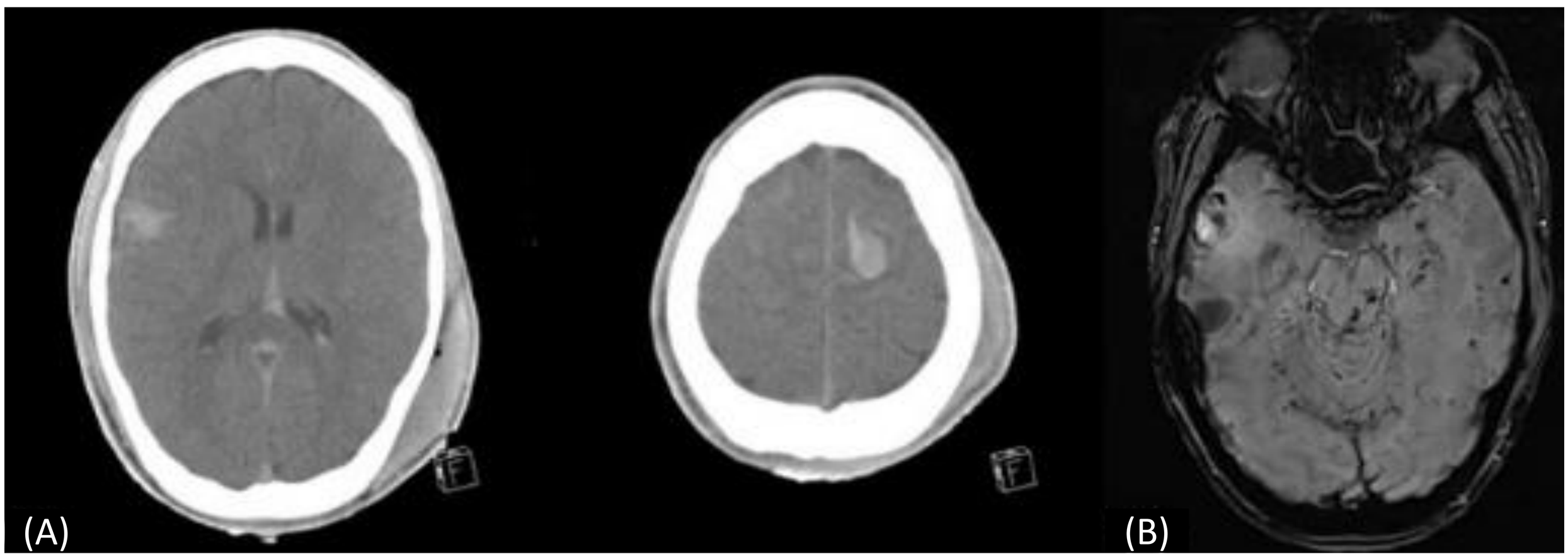

Figure 1. (A) Initial brain CT showing traumatic subarachnoid hemorrhage, (B) Susceptibility-weighted MRI showing diffuse microbleeds, consistent with diffuse axonal injury

Table 1. PSH assessment measure

DLT	CFS					
Clinical features occur simultaneously		0	1	2	3	0
Episodes are paroxysmal in nature	Heart rate (bpm)	<100	100-119	120-139	≥140	<100
Sympathetic over reactivity to normally non painful stimuli	Respiratory rate (breaths/min)	<18	18-23	24-29	≥30	<18
Features persist ≥3 consecutive days	Systolic blood pressure (mmHg)	<140	140-159	160-179	≥180	<140
Features persist ≥2 weeks post brain injury	Temperature (°C)	<37	37.0-37.9	38.0-38.9	≥39	<37
Medication administered to decreased sympathetic features	Sweating	Absent	Mild	Moderate	Severe	Absent
≥2 episodes daily	Posturing during episode	Absent	Mild	Moderate	Severe	Absent
Absence of parasympathetic features during episodes						
Absence of other presumed cause of features						
A score of 1 is given for each criterion met.						
PSH-AM score (CSF subtotal + DLT subtotal)						
<8	PSH unlikely					
8-16	PSH possible					
≥17	PSH probable					

PSH-AM; parosxymal sympathetic hyperactivity assessment measure, DLT; diagnostic likelihood tool, CFS; clinical feature scale.

Conclusion

In conclusion, PSH must be strongly considered in the differential diagnosis for severe TBI patients presenting with unexplained fever, tachycardia, and spasticity throughout acute and subacute recovery. Early recognition using the PSH-AM and multimodal management of noxious stimuli are crucial. Maintaining high clinical suspicion ensures timely intervention, preventing unnecessary workups, stabilizing vital signs, and maximizing clinical benefit for the patient.

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