International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Website: https://ijmpr.in/

Systematic Review

Diffuse Axonal Injury in Young Neurotrauma Patients Complicated by Paroxysmal Sympathetic Hyperactivity Syndrome: A Case Series and Systematic Review

Dr Chandrakant Prasad[1], Dr Ankur Anand[2]

[1]Assistant Professor, Department of Critical Care Medicine, PMCH, Patna, Bihar, India.
[2]Assistant Professor, Department of Neurosurgery, SKMCH, Muzaffarpur, Bihar, India.

OPEN ACCESS

Corresponding Author:

Dr Chandrakant Prasad
Assistant Professor,
Department of Critical Care Medicine, PMCH, Patna, Bihar, India.

Received: 14-03-2026
Accepted: 06-04-2026
Published: 27-04-2026

ABSTRACT

Paroxysmal sympathetic hyperactivity (PSH) is a set of clinical conditions that occur after any form of acquired brain injuries viz trauma, meningitis, encephalitis, or stroke. This syndrome is characterized by paroxysmal and transient episodes of fever, tachypnea, tachycardia, hypertension, diaphoresis, hyperthermia and dystonia (all or in various combinations). Diffuse axonal injury (DAI) is also very common in brain trauma patients, especially in younger ones. We are highlighting here our experience in managing three cases of DAI in young neurotrauma patients complicated with PSH.

Keywords*: Paroxysmal sympathetic hyperactivity (PSH), Diffuse axonal injury (DAI), Traumatic brain injury (TBI), Autonomic dysfunction, Neurocritical care management.*

INTRODUCTION

Paroxysmal sympathetic hyperactivity (PSH) is a well-recognized but frequently underdiagnosed complication of severe acquired brain injury, particularly in patients with traumatic brain injury (TBI) (Baguley et al., 2014; Perkes et al., 2010). It is characterized by episodic and simultaneous increases in sympathetic and motor activity, manifesting as tachycardia, hypertension, hyperthermia, tachypnea, diaphoresis, and abnormal posturing (Perkes et al., 2010; Rabinstein, 2007). These paroxysmal episodes are typically triggered by external or internal stimuli and can significantly complicate the clinical course of critically ill neurotrauma patients.

Diffuse axonal injury (DAI), a common pathological substrate in high-velocity neurotrauma, is often associated with the development of PSH (Meyfroidt et al., 2017; Dolce et al., 2008). The shearing forces involved in DAI lead to widespread disruption of white matter tracts, particularly affecting the diencephalon, brainstem, and cortical-subcortical connections that regulate autonomic function (Meyfroidt et al., 2017). This structural disconnection is believed to result in loss of inhibitory control over sympathetic centers, giving rise to the exaggerated autonomic responses observed in PSH.

The reported incidence of PSH varies widely, ranging from 8% to 33% among patients with severe TBI, with higher rates observed in those with DAI (Perkes et al., 2010; Fernandez-Ortega et al., 2006). Young patients appear to be particularly susceptible, possibly due to higher exposure to high-impact trauma and differing neurophysiological responses. Despite its clinical significance, PSH remains under recognized, often misdiagnosed as sepsis, withdrawal syndromes, or uncontrolled pain, leading to delays in appropriate management (Godoy & Rabinstein, 2015; Hinson & Sheth, 2012).

Given the clinical burden and diagnostic challenges associated with PSH, especially in young patients with DAI, there is a need for better characterization of its clinical profile, triggers, treatment response, and outcomes. This case series, along

with systematic review of the literature, aims to enhance understanding of PSH in this specific population and contribute to improved recognition and management strategies in neurocritical care settings.

CASE 1

15 years male patient having history of road traffic accident presented in ER with GCS E2V2M4. NCCT brain (fig 1) did not reveal any major intracranial injury. Supportive managements were started in line with diffuse axonal injury. On Day 3rd of injury he started to develop episodic hypertension, tachycardia, spasticity of limbs associated with profuse sweating. He also had persistent low grade fever with episodic spikes of temperature. After excluding other reasons and considering PSH, he was started on broad spectrum beta blocker (Propranolol 20 mg twice daily) in addition to all other ongoing supportive cares. PSH episodes kept on increasing in initial 3 days thereafter started to decline and became only occasional by day 12th of injury. GCS also gradually improved with control in PSH incidences. He was discharged to home care on day 15th of injury at full GCS and hemodynamic stable conditions. Propranolol was gradually tapered off over next 15 days in follow up care.

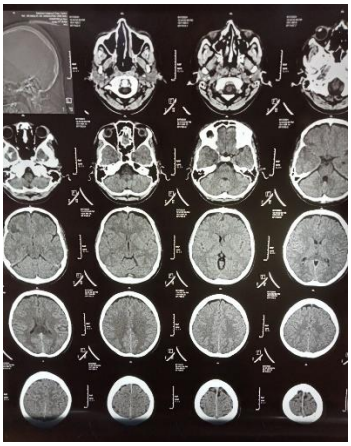

Fig 1 NCCT brain of case 1

CASE 2

19 years male patient was admitted in neuro-intensive care unit in altered sensorium state with history of RTA 11 hours ago. At admission GCS were E2V2M5 and blood pressure was on lower side. NCCT brain (fig 2) was not suggestive of any surgically correctable lesion and was not corresponding to his current sensorium. Conservative cares were tailored towards DAI management. Hemodynamics were improved on fluid correction. He started following commands on day 3rd of admission but again sensorium declined on day 4 with occurrence of frequent tachycardia, hypertension and limb spasticity. After ruling out seizures, sepsis and pain, propranolol 10 mg twice daily was added to the treatment regime considering PSH syndromes. Sympathetic episodes kept on increasing in spite of beta blocker, so infusion fentanyl at the rate of 30 mcg/hour was added on day 5th. Symptoms started to settle down by day 7th and became nil bothering by day 11th. Fentanyl was withdrawn on day 8th and beta blocker gradually stopped on day 25th of starting. He was discharged at GCS 15 on day 17th of admission.

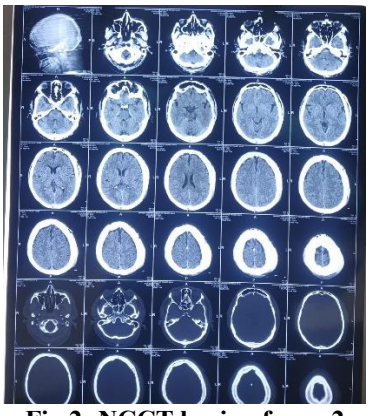

Fig 2: NCCT brain of case 2

CASE 3

26 years male patient was admitted in neuro ICU with alleged history of RTA 1 day ago with low GCS (E1V1M4) and pupils bilateral equal and reactive to light. After securing airway and stabilizing hemodynamic conditions, NCCT brain (fig 3) was done which showed right intra-ventricular bleed without hydrocephalus and small right deep temporal contusions. NCCT brain findings could not explain the decline in sensorium so, patient was kept on supportive cares keeping DAI as differential. Patient started regaining consciousness on day 3rd of injury (E2VtM5) but remained non

progressive till day 5th. On day 5th he started to trigger paroxysmal episodes of hypertension, tachycardia, tachypnea and bilateral upper limb straitening. Considering PSH, he was started on Propranolol 20 mg twice daily and supplemented with infusion dexmedetomidine 4 mcg/kg/hour for 48 hours taking refractory nature of PSH into account. Sympathetic episodes started to decrease by day 9th and almost vanished by day 14th. Sensorium was restored to E4VtM6 on day 9th and was weaned from ventilation followed by extubation of endotracheal tube on day 10th. He was sent for home care on days 17th at full GCS and hemodynamic stable conditions. Propranolol was tapered and stop gradually by day 29th of injury.

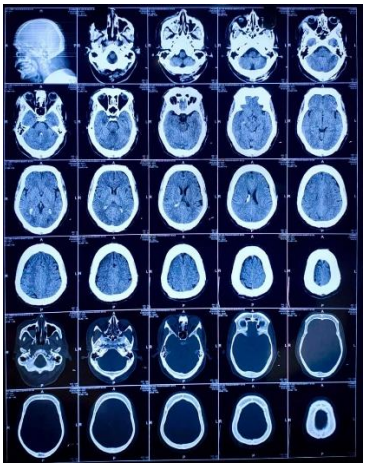

Fig 3: NCCT brain of case 3

DISCUSSION

Paroxysmal sympathetic hyperactivity (PSH) remains a challenging and often under recognized complication in patients with severe traumatic brain injury (TBI), particularly those with diffuse axonal injury (DAI). In the present case series focusing on young neurotrauma patients, PSH was observed as a significant contributor to secondary morbidity, consistent with prior literature (Baguley et al., 2014; Perkes et al., 2010).

Clinically, PSH is characterized by recurrent episodes of tachycardia, hypertension, hyperthermia, tachypnea, diaphoresis, and dystonic posturing. These features were consistently observed, often triggered by ICU stimuli. Diagnostic challenges persist due to overlap with sepsis and other conditions.

The pathophysiology of PSH is best explained by the "disconnection theory," which postulates an imbalance between excitatory and inhibitory autonomic pathways following brain injury. Loss of descending inhibitory control from cortical and subcortical centers leads to unchecked sympathetic outflow from hypothalamic and brainstem centers. Alternative hypotheses, including the excitatory:inhibitory ratio (EIR) model, further support this concept by emphasizing the role of impaired modulation of spinal and supraspinal reflexes.

In 2014, an international consensus group proposed standardized diagnostic criteria and the PSH Assessment Measure (PSH-AM), which includes a Clinical Feature Scale (CFS) and Diagnosis Likelihood Tool (DLT), significantly improving diagnostic consistency (Baguley et al., 2014). Early recognition and targeted management are crucial, as PSH is associated with prolonged mechanical ventilation, increased intensive care unit (ICU) stay, higher healthcare costs, and poorer neurological outcomes (Lv et al., 2010; Zheng et al., 2020).

Management of PSH remains largely supportive and symptomatic, involving multimodal pharmacotherapy aimed at reducing sympathetic overactivity. Commonly used agents include opioids, beta-blockers (e.g., propranolol), alpha-2 agonists (e.g., clonidine, dexmedetomidine), benzodiazepines, and gabapentin. However, there is no universally accepted treatment protocol, and current practices are largely based on observational studies and expert consensus. non-pharmacological strategies such as minimizing triggers and ensuring adequate analgesia are also important. Early recognition improves outcomes.

CONCLUSION

PSH in not very uncommon in young neurotrauma patients having DAI. This impairs the clinical improvement and increase the ICU/hospital stay of patients. Early clinical diagnosis and prompt management of sympathetic response along with primary injury remains the cornerstone of good patient outcome.

REFERENCES:

1. Baguley, I. J., Perkes, I. E., Fernandez-Ortega, J. F., Rabinstein, A. A., Dolce, G., & Hendricks, H. T. (2014). Paroxysmal sympathetic hyperactivity after acquired brain injury: Consensus on conceptual definition, nomenclature, and diagnostic criteria. Journal of Neurotrauma, 31(17), 1515–1520.

2. Dolce, G., Quintieri, M., Serra, S., Lagani, V., & Pignolo, L. (2008). Dysautonomia after traumatic brain injury. Journal of Neurology, Neurosurgery & Psychiatry, 79(6), 674–676.
3. Fernandez-Ortega, J. F., Prieto-Palomino, M. A., Munoz-Ledo, E., Lebron-Gallardo, M., Cabrera-Ortiz, H., & Quesada-Garcia, G. (2006). Prognostic influence and CT findings in dysautonomic crises after TBI. Journal of Trauma, 61(5), 1129–1133.
4. Godoy, D. A., & Rabinstein, A. A. (2015). Paroxysmal sympathetic hyperactivity following traumatic brain injury. Current Neurology and Neuroscience Reports, 15(6), 20.
5. Hinson, H. E., & Sheth, K. N. (2012). Manifestations of the hyperadrenergic state after acute brain injury. Current Opinion in Critical Care, 18(2), 139–145.
6. Lv, L. Q., Hou, L. J., Yu, M. K., Qi, X. Q., Chen, H. R., & Chen, J. X. (2010). Clinical features and prognosis of PSH after TBI. Journal of Clinical Neuroscience, 17(2), 161–165.
7. Meyfroidt, G., Baguley, I. J., & Menon, D. K. (2017). Paroxysmal sympathetic hyperactivity: The storm after acute brain injury. Lancet Neurology, 16(9), 721–729.
8. Perkes, I., Baguley, I. J., Nott, M. T., & Menon, D. K. (2010). A review of PSH after acquired brain injury. Annals of Neurology, 68(2), 126–135.
9. Rabinstein, A. A. (2007). PSH in neuro ICU. Neurological Research, 29(7), 680–682.
10. Zheng, R. Z., Lei, Z. Q., Yang, R. Z., Huang, G. H., & Zhang, G. M. (2020). Identification and management of PSH after TBI. Frontiers in Neurology, 11, 81.