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# Paroxysmal Sympathetic Hyperactivity (PSH) After Non-Traumatic Brain Injury

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## ABSTRACT

Paroxysmal sympathetic hyperactivity (PSH) is a hyperadrenergic syndrome that may follow acute brain injury. Although it is relatively a common complication following acute traumatic brain injury but not commonly seen in non-traumatic brain injury cases. It is presumed to be caused by loss of cortical inhibitory modulation of diencephalic and brain stem centres and possible additional maladaptive changes in the spinal cord that combine to produce exaggerated sympathetic responses to stimulation. Failure to timely diagnose PSH lead to worse neurologic outcomes, prolong hospital stays, more complications and delayed recovery.

Here we are reporting a case of Paroxysmal sympathetic hyperactivity following non-traumatic brain injury secondary to stroke. With early recognition and prompt intervention he showed good recovery and shortened hospital stay.

**Keywords:** Paroxysmal sympathetic hyperactivity; Acquired brain injury; Fever

## CASE REPORT

A 33 year old previously healthy gentleman admitted in January 2020 through emergency when found by family on bed with right dense hemiplegia, aphasia, eyes deviated to left, and right sided up going planter. Initial GCS was 13 which improved gradually. CT head confirmed left pontine and midbrain bleed while no cerebral aneurysm was found on CT angiogram. Patient did not required neuro-surgical intervention and therefore repatriated from university hospital to stroke unit of this regional hospital. His was hemodynamically stable with normal vitals, a febrile and oxygen saturation of 97% on room air. He was following commands, alert and oriented with GCS of 15 and chest was clear to auscultation. However, neurological deficits remain unchanged with dense right hemiplegia and aphasia. He was started on physiotherapy and routine medications.

On day 30 patient developed fever (40.1 degree Celius), Tachycardia (Heart rate 110-130 /min), BP: 125-140/75-80 Tachypnea (Respiratory rate 35-40/min) and was diphoretic.

He was pan-cultured including blood, urine, stool and sputum cultures, however, all came out to be negative for any growth. We changed IV lines sites and Foley's catheter. WBC, renal and liver functions were normal and inflammatory markers were also negative. CXR, CT Chest, Abdomen, pelvis, Echocardiogram and Ultrasound Doppler of legs were all unremarkable. Repeat MRI of brain showed resolution of bleed and no new lesion found.

Patient continued to have episodic high grade fever, tachycardia, tachypnea and diaphoresis at least 2-3 times every day despite on broad spectrum antibiotics, therefore after discussing with infectious diseases, the antibiotics were discontinued.

The diagnosis of Paroxysmal sympathetic hyperactivity (PSH) was then considered and started propranolol and lorazepam. Gradually over next few days patient's symptoms resolved completely and he became asymptomatic. He was then transferred to rehabilitation unit where he remained for 4 weeks after which discharged home with home care. He has slight improvement in his speech with dysarthria and minimal improvement in weakness.

## DISCUSSION

### Introduction

First described by Wilder Penfield in 1929 as 'mesencephalic seizures[1] Paroxysmal sympathetic hyperactivity (PSH) was then time to time referred as autonomic storms, sympathetic storms, hypothalamic dysregulation syndrome, dysautonomia, paroxysmal autonomic instability with dystonia, and even diencephalic autonomic epilepsy (although it is not epileptic in nature)[2]. Paroxysmal sympathetic hyperactivity (PSH) was the term introduced by Rabinstein in 2007[3] since then this term is being considered most clinically accurate.

### Pathophysiology

It is proposed that that following brain injury there is a disconnection between cortical inhibitory areas and lower sympathetic centers in the diencephalon (especially the hypothalamus), brainstem, and spinal cord leading to release of higher inhibitory drive in the central nervous system resulting in increased excitatory interneuronal activity that is hyper sympathetic state[4,5].

### Epidemiology

About 80% followed by traumatic brain injury (TBI), 10% followed by anoxic brain injury, 5 % followed by stroke and the remaining 5% occurred in association with hydrocephalus, tumours, hypoglycaemia, autoimmune encephalitis, cerebral fat embolism syndrome, fulminant multiple sclerosis, vasculitis, postpartum vasoconstriction, infections including bacterial and tubercular meningitis, viral encephalitis, or unspecified

causes[1,2,3,6].Regardless of underlying diagnosis, the reported prevalence of patients with PSH in other studies from various countries ranges from 8% to 33%[1,2,3,4,5].

**Clinical Features and Diagnosis**

PSH is a clinical diagnosis without any confirmatory test. Rabinstein[3]had defined it as transient presence of at least 4 out of following 6 criteria[1,3,6].

- Fever (> 38.3 degree Celius, at least one measurement for 2 consecutive days),
- Tachycardia with heart rate > 100/min,
- Hypertension (SBP> 140 mmHg or pulse pressure > 80 mmHg),
- Tachypnea (respiratory rate > 18/min),
- Diaphoresis and
- Extensor or dystonic posturing is only observed with the most severe episodes and can be mistaken for a tonic seizure.

Parasympathetic signs (bradycardia, hypotension) are characteristically absent during the episode. The episodes are intermittent with sudden onset[1]and can last for up to 20 to 30 minutes. PSH may present as early as within the first day after brain injury, or may first be identified later during rehabilitation[2,5,6].

The most commonly considered differential diagnoses include[6]Pulmonary embolism (PE), Sepsis, Elevated intracranial pressure, Tonic seizures, Acute painful episodes and Alcohol withdrawal.

**Treatment**

Goal of the treatments is to decrease the frequency and intensity of episodes. Medications include benzodiazepines, opiods, non-cardio-selective β-blockers are more effective as compared to cardio-selective beta blockers, alpha adrenergic blockers like clonidine reduces heart rate and blood pressure but less effective in controlling temperature, however, combination of alpha adrenergics and beta adrenergic blockade has also shown effective in controlling paroxysms. GABA agonists like baclofen has been used to treat refractory PSH including intra-thecalinjections[1].

The use of psychoactive medications typically prescribed for agitation is discouraged. Dantrolene can be reserved only for patients with refractory posturing resulting in muscle contractures and in malignanthyperthermia[1].

## SUMMARY

In our patient, we identified Paroxysmal sympathetic hyperactivity (PSH) after excluding other causes. Although less common in non-traumatic brain injury the diagnosis for PSH requires high suspicion. The early recognition of PSH could spare patients from many invasive and expensive procedures.

## DECLARATION OF CONFLICT OF INTEREST AND FUNDING INFORMATION

Author declares that there are no conflict of interest involved and no funding.

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