CASE REPORT

Beta-blockers in the management of posttraumatic paroxysmal sympathetic hyperactivity: A case report from western Cameroon

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Abstract

Traumatic brain injuries are the most common cause of paroxysmal sympathetic hyperactivity (PSH), a life-threatening autonomic dysregulation syndrome. Although PSH was first described in 1929, there remains a paucity of publications on this subject. Here, we report a case of posttraumatic PSH treated with beta-blockers. Two days after suffering a moderate traumatic brain injury, a 32-year-old man was admitted to our emergency department at a teaching hospital in western Cameroon. The patient experienced 16-minute episodes of hypertension, diaphoresis, and agitation, occurring 3 to 4 times daily. His symptoms resolved after treatment with oral atenolol 50 mg every 12 hours. Our experience corroborates the effectiveness of beta-blockers in the treatment of posttraumatic PSH.

Keywords: paroxysmal sympathetic hyperactivity, traumatic brain injury, beta-blockers, Cameroon

Introduction

Paroxysmal sympathetic hyperactivity (PSH) is a syndrome of hypertension, lacrimation, tachycardia, diaphoresis, and agitation, which was first described by Wilder Penfield in 1929.[1] Recent studies have shown the absence of electroencephalographic activity during PSH.[2] These findings have led to the rejection of the nomenclature suggested by Penfield, 'diencephalic seizure', along with other names proposed thereafter. In 2014, the International Brain Injury Association agreed on the name 'paroxysmal sympathetic hyperactivity' and established diagnostic criteria.[2]

PSH is secondary to severe acquired brain injury, such as that associated with traumatic brain injury (TBI), stroke, tumours, infection, spinal injury, or serotonin syndrome.[3] The prevalence of PSH varies from 8% to 33% of patients admitted to intensive care units.[4] Most PSH results from TBI (79.4%), with 80% associated with moderate to severe brain injury.[4],[5]

The pathophysiology of PSH is poorly understood, and the treatment is even less well established. Numerous drugs have been used alone or in combination—namely, benzodiazepines, opiates, morphine, gabapentin, baclofen, and beta-blockers.[6]

Beta-blockers act at the cardiac level to reduce heart rate, perfusion volume, and mean arterial pressure. This, in turn, reduces oxygen consumption and lowers the risk of myocardial infarction. At the nervous level, noncardioselective beta-blockers, due to their lipophilic nature, can cross the blood–brain barrier and reduce cerebral perfusion, thereby reducing glucose and oxygen consumption. These effects lower the basal metabolic rate by tempering the effects of catecholamines.[6]

This article reports on beta-blocker treatment of posttraumatic PSH at a teaching hospital in Bangangté, Cameroon.

Case presentation

Patient information

A 32-year-old man was brought to our emergency room with a headache and scalp wound sustained during a fight. The patient was hit on the right side of the head with a blunt object, causing an initial loss of consciousness lasting approximately 2 minutes. The patient had no relevant personal or family medical history.

[PAGE NUMBERS NOT FOR CITATION PURPOSES]

Table. Paroxysmal Sympathetic Hyperactivity Assessment Measure[2],[3] findings

Clinical Feature Scale (CFS)					
	0	**1**	**2**	**3**	**Score**
Heart rate, beats/min	100	100-119	**129-139**	≥140	**2**
Respiratory rate, breaths/min)	<18	18-23	**24-29**	≥30	**2**
Temperature, °C	<37.0	37.0-37.9	**38.0-38.9**	≥39	**2**
Systolic pressure, mmHg	140	140-159	160-179	**≥180**	**3**
Posturing during episodes	Nil	**Mild**	Moderate	Severe	**1**
Sweating	**Nil**	Mild	Moderate	Severe	**0**
Diagnosis Likelihood Tool (DLT)					
					Score[a]
1. Clinical features occur simultaneously					**1**
2. Episodes are paroxysmal					**1**
3. Sympathetic overactivity to normally nonpainful stimuli					**1**
4. Features persist >3 consecutive days					**1**
5. Features persist >2 weeks postinjury					**0**
6. Features persist despite treatment of differential diagnoses					**1**
7. ≥2 episodes daily					**1**
8. Medication administered to decrease sympathetic features					**0**
9. Absence of parasympathetic features during episodes					**1**
10. Absence of the other presumed cause of features					**1**
11. Antecedent acquired brain injury					**1**
Total			**19**		

[a]Scored as 1 if present, 0 if absent

Clinical observations

At admission, the patient presented with an altered state of consciousness, registering a Glasgow Coma Scale score of 10/15 (eye, 3/4; verbal: 3/5, motor, 4/6). He had elevated blood pressure (195/88 mmHg), tachycardia (133 beats/min), tachypnoea (28 breaths/min), and a temperature of 37.8 °C. He also exhibited agitation and diaphoresis.

The patient was admitted to the intensive care unit, and neuroimaging was ordered. Noncontrast computed tomography revealed no abnormalities.

Standard neurointensive care was administered. However, the patient experienced 16-minute episodes of agitation and diaphoresis, accompanied by elevated blood pressure, temperature, heart rate, and respiratory rate. These episodes occurred every 6 to 8 hours and persisted for 2 days, suggesting the possibility of PSH.

Diagnosis

The diagnosis was confirmed using the Paroxysmal Sympathetic Hyperactivity Assessment Measure (PSH-AM) scoring tool.[2],[3] The PSH-AM has 2 components: the CFS (Clinical Feature Scale), which assesses the severity of clinical symptoms, and the DLT (Diagnosis Likelihood Tool), which assesses the probability of PSH based on the presence and frequency of symptoms.[3] As there are no confirmatory laboratory or imaging techniques for PSH, the PSH-AM is the only available diagnostic tool.[2] Our patient's PSH-AM score was 19 (Table).

Treatment and follow-up

The patient received oral atenolol (50 mg every 12 hours). The results were almost immediate (Figure). The patient did not experience further episodes until the tenth day of admission, when treatment was discontinued.

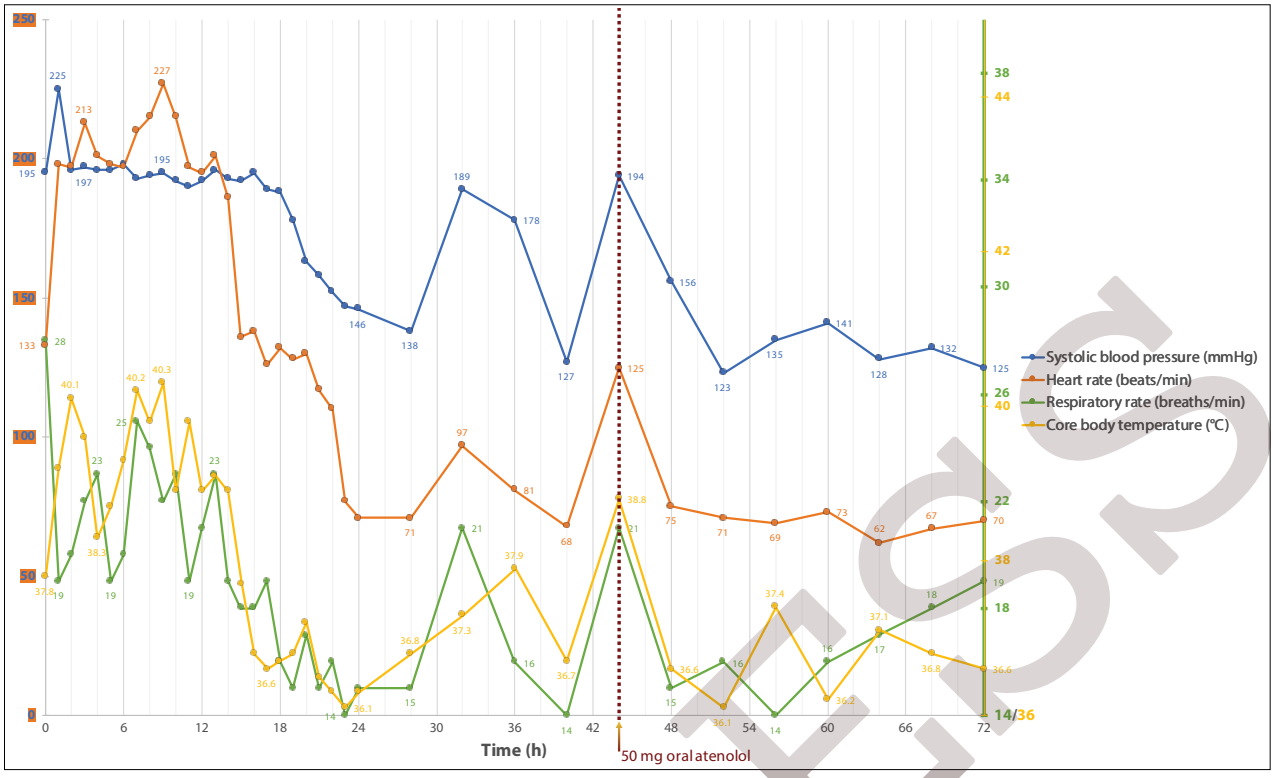

Figure. Core body temperature, pulse rate, respiratory rate, and systolic blood pressure during paroxysmal sympathetic hyperactivity episodes

Discussion

Beta-blockers are widely used in the treatment of PSH, either alone or in combination with other agents.[6] We achieved rapid symptom resolution for our patient by administering 50 mg of oral atenolol every 12 hours over 10 days.

Cotton et al.[7] found that patients with TBI who received beta-blockers for ≥2 days had a significantly lower mortality rate than those who did not receive beta-blockers. Schroeppel et al.[8] had similar findings. In a large prospective, multicentre study in the United States and Canada, head trauma patients who were treated with beta-blockers had a significantly lower 30-day mortality rate than those who did not receive beta-blockers.[9] In these 3 studies,[7]-[9] TBI patients treated with beta-blockers had significantly longer hospitalizations than those who did not receive beta-blockers; Schroeppel et al.[8] additionally found that propranolol administration was specifically associated with longer hospital stays than treatment with other beta-blockers.

Compared with other beta-blockers, noncardioselective agents, such as propranolol, could be more effective in TBI treatment due to their lipophilic nature, which enables them to cross the blood–brain barrier.[8] In the previously referenced study by Schroeppel et al.,[8] the mortality rate associated with propranolol treatment was significantly lower than that associated with other beta-blockers. A randomized, double-blind trial found that TBI patients with posttraumatic PSH who underwent a 7-day propranolol treatment regimen had significantly lower catecholamine levels and higher Glasgow Coma Scale scores than those who received placebo treatment.[10] In Iran, researchers determined a significantly lower in-hospital mortality rate among TBI patients treated with propranolol (20 mg orally every 12 hours up to 10 days or until discharge) relative to untreated individuals.[11] Other trialled propranolol treatment regimens for patients with TBI have included 1 mg intravenously every 6 hours[10],[12] and an initial oral dose of 20 mg 3 times daily adjusted to maintain a target heart rate of <100 beats per minute with increments up to a daily maximum of 640 mg.[13] The optimal dosage remains to be established.[9]

Notably, atenolol has been associated with reduced cardiac morbidity in TBI patients treated with 10 mg intravenously every 6 hours for 3 days, followed by oral administration of 100 mg daily for 4 days.[14]

PSH is an anxiety-inducing condition for patients and their families. The symptoms are dramatic, frequent, and unpredictable. In our case, the patient and his family were understandably worried about the potential permanence of PSH, a concern not alleviated by the current lack of a comprehensive understanding of its pathophysiology. While reassured by the patient's response to treatment, they understandably remained fearful of future episodes. This underscores the ongoing need for further research into the mechanisms underlying PSH and the development of regimented management strategies. Enhancing our comprehension of this condition will equip healthcare providers with reliable approaches to treatment, as well as provide clarity and comfort to those affected.

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