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UNCOMMON AND/OR UNUSUAL HEADACHES AND SYNDROMES (J AILANI, SECTION EDITOR)

Headache Attributed to Autonomic Dysreflexia: Clinical Presentation, Pathophysiology, and Treatment

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Abstract
A patient presenting with marked elevation in blood pressure and concurrent headache often presents a diagnostic challenge for even the most seasoned clinician. When marked hypertension and headache occur in a patient with a history of upper spinal cord injury, the patient should be presumed to have autonomic dysreflexia until proven otherwise. Autonomic dysreflexia can at times trigger headaches, hypertension, and variations in pulse, as well other autonomic signs and symptoms. Autonomic dysreflexia is a medical emergency for which appropriate treatment may be life-saving. In this review, we address the historical origins, risk factors, pathophysiology, diagnostic criteria, clinical presentation, differential diagnosis, and treatment of headache attributed to autonomic dysreflexia. Included are two case presentations from the authors' clinic, which illustrate the diagnosis and treatment of headache attributed to autonomic dysreflexia.

Keywords Headache · Autonomic dysfunction · Autonomic dysreflexia

Introduction

Patients presenting with both severe headache and hypertension are often diagnostically challenging. Not only is there a question about whether the hypertension is the cause or the effect of the headache pain, but there are multiple conditions affecting the central nervous system that can contribute to both hypertension and headache, often in the context of autonomic dysregulation. In the absence of a known catastrophic neurologic event, such as stroke, intracranial hemorrhage, traumatic brain injury, or neuroleptic malignant syndrome, a patient presenting with abrupt onset headache and an acute pressor response (diastolic blood pressure increase > 25 mmHg) should raise concern for hypertensive crisis, hypertensive encephalopathy, and pre-eclampsia/eclampsia (in a patient who is pregnant/postpartum). When a patient presents repeatedly with abrupt onset headaches and labile hypertension, this should raise suspicion for a process contributing to paroxysmal autonomic dysregulation, such as a pheochromocytoma or autonomic dysreflexia. Although a well-known clinical entity among specialists treating spinal cord injuries (SCI), "Headache Attributed to Autonomic Dysreflexia" was only recently included in the International Classification of Headache disorders 3rd edition (beta version) in 2013 [1]. Many clinicians are unaware of this entity, and a lack of awareness can result in a delay in the diagnosis and appropriate management of a potential medical emergency. For this reason, we will review the clinical presentation, pathophysiology, and management options for this unique syndrome.

This article is part of the Topical Collection on *Uncommon and/or Unusual Headaches and Syndromes*

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Case 1: Headache Attributed to Autonomic Dysreflexia

A 56-year-old man with a history of quadriplegia following a remote C5–6 traumatic spinal cord injury suddenly awoke at 4 a.m. with a thunderclap headache, which had a 10/10 in intensity. He described the pain as severe, bilateral, and sharp/stabbing in quality. He reported some nausea without vomiting. He denied photophobia, phonophobia, or neck stiffness. There were no focal neurologic deficits or aura-like symptoms. Systolic blood pressure at the time of headache onset was 235 mmHg. He also noted abdominal spasm, generalized body pain, and flushing of his face and neck with pallor of the lower extremities. He was evaluated in the ED and noted to have a urinary tract infection associated with a clogged Foley catheter. His blood pressure spontaneously lowered after removal of the clogged Foley catheter and his headache subsequently resolved.

Historical Origins

Autonomic dysreflexia was first described by Hilton in 1860 [2]. It was later described by Head and Riddoch in 1917, who noted episodes associated with bladder distention, catheter blockages, and enema administration in soldiers who had sustained spinal cord injuries during World War I. They described a syndrome "manifested by excessive sweating, flushing of the face, congestion of the nasal passages, pilomotor erection, sometimes shivering, and a feeling of dullness in the head, which may progress to a severe, throbbing, bitemporal, occipital or frontal headache" sometimes associated with blurred vision [3]. Over the years, autonomic dysreflexia has been described in the literature with several different names including autonomic headache, autonomic or sympathetic hyperreflexia, spinal poikilopiesis, autonomic spasticity, autonomic reflex, mass reflex, neurovegetative syndrome of bladder vesicle distension, paroxysmal hyperactive autonomic reflexes, paroxysmal hypertension, hyperactive autonomic reflexes, and paroxysmal neurogenic hypertension [3, 4, 5•, 6–10].

Risk Factors

Autonomic dysreflexia may occur in complete cord injuries (complete loss of function below the level of the injury) as well as in incomplete cord injuries (some preservation of function below the level of injury), and the severity and frequency of episodes often correlate with the completeness of injury [11, 12]. Individuals with injury at or above the level of T6 are at the highest risk for this condition, although cases involving injury as low as T8 have been described [13, 14]. Additionally, cervical injuries appear to have higher occurrence of autonomic dysreflexia than high thoracic lesions [15]. Autonomic dysreflexia is rarely seen in myelopathy secondary to cancer or autoimmune etiologies such as multiple sclerosis, but the prevalence is unknown [16•]. The prevalence of autonomic dysreflexia in cervical or upper thoracic spinal cord injury has been variably reported to be between 20 and 90%. However, most authors speculate that some degree of autonomic dysreflexia occurs in the majority of patients with upper cord injury [17–19].

Pathophysiology

The principle mechanism for autonomic dysreflexia results from failure of neuronal regulation. Parasympathetic excitation is predominantly occurring above the level of injury and sympathetic excitation occurs below the level of injury. Episodes are triggered by unregulated afferent stimuli that reach sympathetic preganglionic fibers. Most often these triggers are urologic in origin and include urinary tract infection, bladder distension, and/or a clogged Foley catheter [17]. Other inciting conditions include bowel distension/impaction, overgrown toenails, tight clothing, pressure ulcers, pregnancy/labor, psychological stress, sexual intercourse, and injuries such as fractures or burns [8, 20]. Because this patient population often has reduced to absent sensation below the level of their injury, they may be unaware of the painful stimulus below the waist that is contributing to their symptoms.

These noxious stimuli trigger a sympathetic response resulting in vasoconstriction of the muscular, splanchnic, and cutaneous vascular beds above the level of injury causing severe paroxysmal hypertension. Baroreceptors on the great vessels and heart detect hypertension and induce reflex bradycardia with subsequent vasodilatation via descending inhibitory parasympathetic signals. In cases of autonomic dysreflexia, a lack of upper thoracic splanchnic innervation does not allow the autonomic nervous system to provide compensatory regulation of the two systems at the level of cord injury. As such, continuous vasodilation, flushing, and sweating of the head and neck continue above the level of injury where the parasympathetic nervous system is attempting to regulate the massive sympathetic response. Below the level of the lesion, the sympathetic response simultaneously continues, resulting in vasoconstriction with cold/pale extremities [16•, 21]. Figure 1 provides a visual representation of the pathophysiology and some of the clinical manifestations of a patient with autonomic dysreflexia.

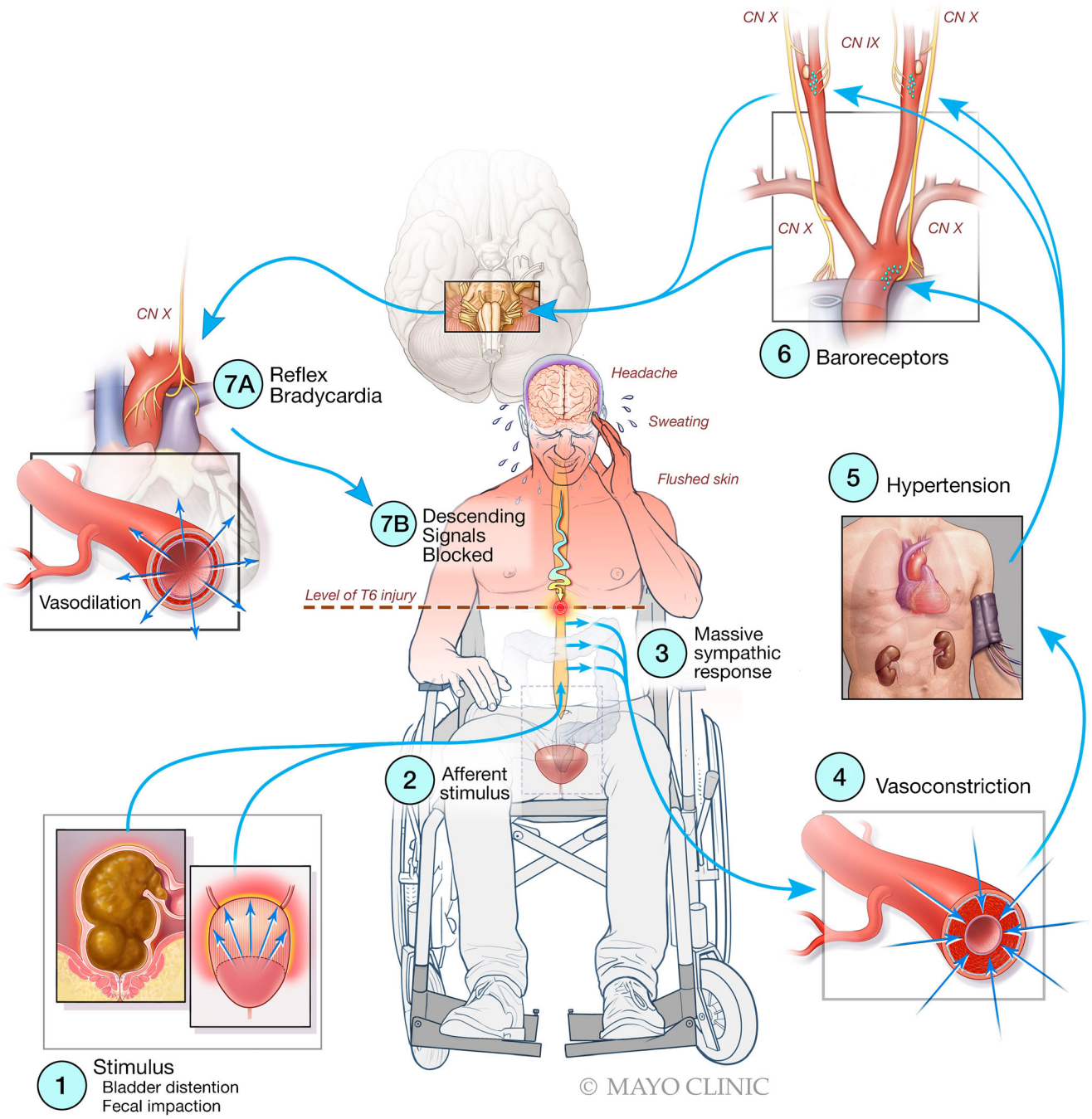

Fig. 1 Mechanism of autonomic dysreflexia in a person with spinal cord injury (at the level of T6 or above). An afferent stimulus (e.g., distended bladder or fecal impaction) triggers a peripheral sympathetic response that results in widespread vasoconstriction and subsequent hypertension. Baroreceptors in blood vessels detect this hypertensive crisis and signal the brain via cranial nerve IX and X. Descending inhibitory signals normally respond to counteract the rise in blood pressure with slowed heart rate and vasodilation. However, vasodilation is blocked at the level of the spinal cord injury. This results in unregulated hypertension, flushed/warm skin, and sweating above the level of the lesion. Below the level of the lesion, cold/pale extremities and piloerection may be seen. Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved

Clinical Presentation

Eighty-five percent of patients with autonomic dysreflexia presents with a triad of headache, hyperhidrosis, and cutaneous vasodilation [12]. Most of these individuals (56–89%) present with severe headache at onset [12, 17]. Individuals with cervical or upper thoracic spinal cord lesions who complain of new headache should have their blood pressure checked, looking for any increase in systolic pressure more than 30 mmHg or diastolic pressure more than 20 mmHg

above their baseline. If blood pressure is elevated significantly above their baseline, they should be treated as presumed autonomic dysreflexia until proven otherwise.

The headache encountered by these patients tends to be sudden onset, often reaching maximal intensity in less than 1 min. The pain is most commonly described as severe pounding pain, although explosive or throbbing characteristics are frequently reported. Pain can be unilateral, bilateral, or generalized often with radiation to the neck. The duration of pain is variable and dependent on trigger removal with a mean duration of 30 min. The headache is generally associated with diaphoresis/flushing cranial to the level of SCI, a sudden rise in blood pressure, and heart rate alterations including initial bradycardia often followed by tachycardia later in the episode [20, 22, 23]. Other common manifestations of autonomic dysreflexia include dry and pale skin ± piloerection below the level of injury, visual disturbances, nasal stuffiness, anxiety, feelings of doom, and nausea/vomiting [1, 5•, 17, 23–25]. Table 1 provides a summary of the signs and symptoms that may be present in patients with autonomic dysreflexia. The diagnostic criteria for headache attributed to autonomic dysreflexia (10.3.5) according to the International Classification of Headache Disorders 3rd Edition [26] are listed in Table 2.

Table 2 Headache attributed to autonomic dysfunction (10.3.5) according to the International Classification of Headache Disorders 3rd Edition

A. Headache of sudden onset, fulfilling criterion C

B. Presence of spinal cord injury and autonomic dysreflexia documented by a paroxysmal rise above baseline in systolic pressure of ≥ 30 mmHg and/or diagnostic pressure of ≥ 20 mmHg.

C. Evidence of causation demonstrated by at least two of the following:
1. Headache has developed in temporal relation to the rise in blood pressure.
2. Either or both of the following:
   a. Headache has significantly worsened in parallel with increased blood pressure.
   b. Headache has significantly improved in parallel with decrease in blood pressure.
3. Headache has at least two of the following four characteristics:
   a. Severe intensity
   b. Pounding or throbbing (pulsating) quality
   c. Accompanied by diaphoresis cranial to the level of the spinal cord injury
   d. Triggered by bladder or bowel reflexes

D. Not better accounted for by another ICHD-3 diagnosis.

Case 2: Headache Attributed to Autonomic Dysreflexia

A 38-year-old woman with incomplete tetraparesis related to a remote history of traumatic C5 burst fracture with retropulsion presented to the ED with an acute onset 10/10 headache that occurred spontaneously while riding in the car. Within 10 min, she was vomiting and noted right-sided weakness. Upon presentation to the ED, she was intubated in order to protect her airway, and blood pressure was noted to be elevated at 192/110. CT head revealed left basal ganglia intracerebral hemorrhage, and she was briefly on IV nicardipine. She was extubated on day 3 of hospitalization and was then noted to have continuous severe headache and several spells of "altered behavior" by her husband. An MRI of the brain was obtained and demonstrated bilateral anterior cerebral artery territory infarctions. Digital subtraction angiogram was performed revealing areas of narrowing consistent with reversible cerebral vasoconstriction syndrome. She was subsequently started on verapamil, and her headache improved within 2 days.

It was later revealed that the patient had just recently been hospitalized for fecal impaction, raising the question of autonomic dysreflexia as a cause of her thunderclap headache and acute pressor response.

Autonomic dysreflexia is a medical emergency given the potentially devastating complications that can arise from hypertensive crisis. Potentially devastating complications described in the literature include intracranial hemorrhage, reversible cerebral vasoconstriction syndrome, retinal detachments, pulmonary edema, seizures, coma,

Table 1 Signs and symptoms that may be present in autonomic dysreflexia

General	Above lesion	Below lesion
• Hypertension • Bradycardia, often followed by tachycardia • EKG changes (e.g., atrial fibrillation, bigeminy, premature atrial and ventricular contractions, prominent T waves) • Change in level of consciousness • Headache • Aphasia • Anxiety, irritability, and feelings of doom • Nausea and vomiting • Chest tightness	• Flushing head/neck • Sweating head/neck • Splotches of face and neck • Congestion of mucous membranes • Visual field defects • Ocular findings (e.g., lid lag, mydriasis, conjunctival congestion, Horner's oculosympathetic spasm)	• Pallor of abdomen and lower extremities • Piloerection • Cold distal legs • Increased spasticity • Penile erection and seminal fluid emission • Bladder distension • Fecal impaction

Table 3 Differential diagnosis of headache attributed to autonomic dysreflexia

	typical presentation	Provoking factors for symptoms	Common causes
Autonomic dysreflexia	Refer to Table 1 for complete list -Thunderclap headache -Hypertension -Bradycardia* -Flushing/diaphoresis above the lesion -Pallor/cold extremities below the lesion	-Stimulus below level of spinal cord injury	Cord pathology above T6 -Spinal cord injury -Multiple sclerosis -Malignancy
Baroreflex failure	-Thunderclap headache -Diaphoresis -Hypertension -Head/neck flushing -Splotches on face -Agitation/ emotional lability -Palpitations -Tachycardia due to lack of reflex bradycardia	-Stress -Exercise -Arousal; even minor mental arousal (e.g., mental-arithmetic calculations) -Pain	*Transient* -Post carotid endarterectomy -Takayasu arteritis -Postsurgical deafferentation of carotid baroreceptors *Chronic* -Bilateral lesions of the nucleus of the solitary tract (brainstem stroke) -Familial paraganglioma syndrome -Surgical resection of the glossopharyngeal nerves -Idiopathic (Page syndrome) -Radiation therapy to neck for head/ neck malignancies
Reversible cerebral vasoconstriction syndrome	-Thunderclap headache (can be recurrent) -Hypertension -Blurred vision -May develop focal neurologic deficits related to brain edema, stroke or seizure (hemiplegia, tremor, hyperreflexia, ataxia, aphasia, visual deficits) -Most often monophasic	-Orgasm -Physical exertion -Acute stressful or emotional situations -Valsalva -Bathing, swimming	Medications/supplements, Illicit drugs Vasoactive secreting tumors Subarachnoid hemorrhage Neurosurgical procedures Postpartum Pheochromocytoma (see below)
Cushing response	-Thunderclap headache -Bradycardia -Respiratory depression -Focal neurologic deficits of herniation syndromes	-Valsalva maneuvers	Increased intracranial pressure - Cerebral hematomas -Posterior fossa tumors -Edematous cerebellar infarct -Basilar artery aneurysm
Pheochromocytoma	-Thunderclap headache -Diaphoresis -Hypertension -Palpitations -Anxiety/sense of doom -Tachycardia -Tremors -Paleness in the face -Dyspnea	-Commonly unprovoked or -Physical exertion -Anxiety/stress -Change in body position -Labor and delivery -Surgery and anesthesia -Tyramine-containing foods -Medications	Tumor develops in chromaffin cells in the adrenal gland, releasing hormones (epinephrine and norepinephrine) Rare inherited disorders -Multiple endocrine neoplasia, type I -Von Hippel-Lindau disease -Neurofibromatosis 1 -Hereditary paraganglioma syndromes
Hypertensive encephalopathy	-Thunderclap headache -Restlessness -Visual disturbances -Hyperreflexia -Restlessness/ agitation -Seizure -Confusion/altered consciousness -Papilledema or retinal hemorrhage	-Any factors that would result in uncontrolled hypertension including stress or anxiety	Acute or chronic renal diseases Vasculitis Eclampsia Thrombotic thrombocytopenic purpura Medications/immunosuppressants Sepsis Transplantation
Venous sinus thrombosis [36]	-Headache with or without vomiting -Visual problems -Behavioral disturbances -Confusion/altered consciousness -Seizures -Language deficits -Papilledema	-Valsalva -Supine positioning	Oral contraceptives Pregnancy and the puerperium Malignancy Head injury and mechanical precipitants Infection Other genetic or acquired prothrombotic conditions

*Bradycardia may be followed by tachycardia

myocardial infarction, atrial fibrillation, and even death [27–32]. The combination of severe hypertension with concomitant cerebral vasodilation places patients at high risk for potentially fatal hemorrhagic stroke [32–35], highlighting the importance of prompt recognition and treatment of this syndrome.

Differentiating headache attributed to autonomic dysreflexia from other abrupt onset headaches associated with acute pressor response and autonomic disturbance is essential in instituting appropriate prompt treatment. Table 3 summarizes the major mimickers that should be considered in cases of headache attributed to autonomic dysreflexia.

Treatment/Management

The primary management for autonomic dysreflexia involves prevention of these crisis episodes, including strict adherence to bowel, bladder, and skin care regimens. It is important that patients and their caregivers are educated about this condition, its potential dangers, and the importance of prompt recognition for institution of appropriate treatment. It is recommended that patients with spinal cord injury at risk for autonomic dysreflexia carry a card in their wallet/purse identifying them as having this problem as well as emergency management instructions for autonomic dysreflexia, as medical personnel in an acute setting outside of a spinal cord injury center may not be aware of this condition [4].

Because patients with spinal cord injury often have baseline systolic blood pressure in the 90–110-mmHg range, an elevation in pressure 20–40 mmHg above their baseline is a red flag. Once a patient presents in the setting of symptomatic autonomic dysreflexia, lowering blood pressure is the first step in treatment. The Clinical Practice Guidelines of the Consortium for Spinal Cord Medicine provides detailed recommendations for acute management of autonomic dysreflexia and recommends rechecking the patient's blood pressure and heart rate after each intervention step [37••].

The Guidelines recommend raising the head of the bed to 90° and loosening any clothing or constrictive devices. If evident, removal of the noxious stimulus is recommended, provided such removal would not result in further sympathetic stimulation [22, 32]. Patients with indwelling urinary catheters should be checked for correct placement and/or blockage, and problems should be corrected immediately. Urologic consultation should be considered if difficulties arise in catheter placement. It is recommended to limit patient manipulation as much as possible. If the noxious stimulus triggering the autonomic dysreflexia is not immediately identified (e.g., pinched skin, tight clothing), then blood pressure should be controlled using systemic medications while the patient is further evaluated. It is preferred to start with an antihypertensive with rapid onset, but short duration (e.g., nifedipine or nitrates) [5•, 37••] given risk for sudden hypotension if autonomic dysreflexia subsides. Evaluation for fecal impaction should not be attempted unless or until blood pressure has been lowered to below 150 mmHg systolic and should be stopped if autonomic dysreflexia becomes worse during manual evacuation [37••].

Conclusion

When evaluating a patient with severe headache and markedly elevated blood pressure, secondary causes should be considered including stroke, intracranial hemorrhage, traumatic brain injury, venous sinus thrombosis, reversible cerebral vasoconstriction syndrome, or pre-eclampsia/eclampsia (in pregnancy). If initial imaging is negative, and there is no history of trauma or pregnancy, consider alternative causes of labile blood pressure and headache such as hypertensive encephalopathy, Cushing response, baroreflex failure, and pheochromocytoma.

In cases of patients with cervical or upper thoracic spinal cord injury with severe headache and markedly elevated blood pressure, autonomic dysreflexia should be the highest on the differential. It is important to remember that autonomic dysreflexia can occur not only in complete spinal cord injury but also in incomplete injuries with partial preservation of function below the lesion. Therefore, this should be considered in the differential even when motor weakness is not readily apparent. Treatment should be instituted emergently while simultaneously excluding alternative diagnoses and or potential complications of autonomic dysreflexia. An understanding of this condition as well as other disorders associated with paroxysmal dysautonomia may prevent devastating complications and in some cases may even be life-saving.

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Compliance with Ethical Standards

Conflict of Interest Jaclyn R. Duvall declares no conflict of interest. Paul Mathew is on the Advisory Board for Allergan, Amgen, Biohaven, Cowen, Eli-Lilly, Promius, Revance, Satsuma, Stealth BioTherapeutics, Supernus, and Takeda. Carrie E. Robertson receives honoraria from UpToDate as author and she is also on the Advisory Board for Alder and Eli-Lilly.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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