Case Report

Autonomic dysreflexia associated with transient aphasia

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Study design: Case report of autonomic dysreflexia presenting with transient aphasia in a subject with C4 tetraplegia.

Objectives: To report a rare case of autonomic dysreflexia.

Setting: Rehabilitation Service, The Ohio State University, USA.

Case report: A 21-year-old man with a C4 spinal cord injury (ASIA B) developed aphasia associated with autonomic dysreflexia. He was treated with an adrenergic blocking agent.

Conclusion: Autonomic dysreflexia manifested by a transient aphasia and seizures is uncommon.

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Introduction

Autonomic dysreflexia is a condition of massive paroxysmal reflex sympathetic response to noxious stimuli occurring in persons with tetraplegia and paraplegia with spinal cord injuries (SCI) above the major splanchnic sympathetic outflow.^{1–3} Noxious stimuli with root entry zones more distal to the site of neurologic injury (eg S2–S4) illicit the most profound sympathetic adrenergic and cholinergic responses⁴ accounting for why the majority of episodes are attributed to stimulation from the urinary and alimentary tracts.^{3,5} Clinical features are varied, but depend in large part upon the resultant reflex sympathetic adrenergic and cholinergic activity. These include cardiac chronotropic and inotropic effects, sudomotor and pilomotor responses, cephalgia, chest pain, visual disturbances, and paroxysmal hypertension.^{2–11} The resulting hypertension can be severe with systolic and diastolic recordings two to three times greater than the patient's baseline values.^{1,3,7,9} A hypertensive crisis, if untreated, can result in seizures,^{5,6,8–12} hemorrhages (cerebral, retinal, subarachnoid),^{6,12,13} stroke,^{12,14} coma,^{5,12} and death.^{5,12,13} Removal of the noxious stimuli results in abrupt resolution of the autonomic response.

We present an unusual case of autonomic dysreflexia presenting with transient aphasia in an individual with C4 tetraplegia which illustrates the profound paroxysmal effects of the sympathetic nervous system

on patients at risk for this reflex phenomenon. We are unaware of a similar presentation in the literature.

Case report

A 21-year-old white male with no significant past medical history sustained an acute SCI with immediate C4 motor complete, sensory incomplete tetraplegia (ASIA B) after 'belly flopping' into a baby pool on 31 July 1999. Evaluation at that time revealed C5 and C6 vertebral body fractures, and C5 laminar fractures with canal compromise. There was no evidence of a traumatic brain injury. He underwent anterior decompression, fusion, and fixation on 3 August 1999. His acute hospital course was complicated by recurrent mucous plugging requiring repeated bronchoscopy, aspiration pneumonia, and sepsis. He subsequently underwent placement of a tracheotomy tube and a feeding percutaneous endoscopic gastrostomy tube.

He was admitted to our SCI rehabilitation service on 20 September 1999 for continued inpatient rehabilitation. Over the next several days his medical condition improved considerably. His upper limb motor function began to return and his vital capacity improved to over 2200 ml allowing the tracheotomy tube to be downsized in anticipation of decannulation. He was out of the spinal shock phase of his injury and was developing increased spasticity. During this time period, he developed severe central pain involving his lower limbs which was treated successfully with gabapentin, amitriptyline, and mild narcotic analgesics. He was also noted to have a mildly elevated

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blood pressure for brief periods associated with flushing. His neuropathic bladder dysfunction was managed with a program of reflex voiding and condom catheter drainage at his request.

Seven days after admission, while receiving a range of motion exercises in bed, he exclaimed that he didn't feel well, and abruptly became unresponsive with seizure-like activity and spontaneous raising of his upper limbs and drooling for approximately 1 min. When he awoke he appeared to be in a post-ictal state with confusion and emotional lability which shortly thereafter resolved. During the event his blood pressure was 180/110 mmHg with a pulse of 86 beats per min and respirations of 20 per min. Pulse oxymetry was 98% saturation. His heart and lung examinations were unremarkable, profuse flushing of his face was noted.

He was transferred urgently to the acute care hospital for further evaluation and management. During that initial evaluation it was noted that he was developing word-finding difficulties and aphasia in the absence of clinical seizure activity and the possibility of a stroke in evolution was entertained. The aphasia resolved subsequently. Extensive evaluation over the next 48 h including CT and MRI scans of his head, an electroencephalogram, electrocardiogram, carotid dopplers, cardiac echo, laboratory studies, and repeat lower limb duplex ultrasonography failed to identify an etiology for the episodes. These episodes were associated with elevated blood pressure and ultimately believed to be of autonomic origin. Upon readmission to our facility 2 days later, he was placed initially on clonidine, which was later changed to doxazosin with improved bladder emptying, and resolution of hypertension associated with autonomic dysreflexia. He has had no recurrence of mental status changes, aphasia, or seizures since.

Discussion

The incidence of seizures associated with autonomic dysreflexia appear to be uncommon, and transient aphasia in the absence of stroke or hemorrhage is rare. The clinical features of autonomic dysreflexia in our patient were quite unusual and we are unaware of a similar presentation. Prior authors have reported aphasia in the presence of hemorrhage,¹² in one other description, clarification was not given.⁵ We have treated three other patients who experienced seizures associated with autonomic dysreflexia over the past several years. None were associated with aphasia. In all cases, there was a period of time ranging from a few minutes to over an hour where classic symptoms (eg flushing, sweating, cephalgia, etc.) were observed before the onset of seizures, unlike this patient's presentation. Although prior observations indicate the clinical symptoms appear to precede seizures, descriptions of duration of those symptoms are not well characterized.¹⁰⁻¹² In one of the earliest descriptions of hypertension associated with autonomic dysreflexia,

Thompson and Withan¹⁰ induced, with bladder distention in a patient with spinal cord injury symptoms of autonomic dysreflexia followed by a seizure which resolved with bladder emptying.

In the present case the seizure onset was near immediate raising the possibility of another serious etiology such as intracranial hemorrhage or severe hypoxia. No intracranial abnormalities were identified and oxygen saturation was normal. The transient aphasia observed later was associated with an elevated blood pressure 50% above his baseline but not with alterations in his level of consciousness or clinical seizure activity. This was initially felt to represent cerebral vascular insufficiency and a stroke in evolution but resolved shortly thereafter. We currently believe that the seizure was a result of heightened sympathetic activity with hypertension, and the aphasia a possible manifestation of cerebral vascular insufficiency.

The present case illustrates several features of this condition. Our patient sustained a SCI with C4 incomplete tetraplegia, above the level of the major splanchnic sympathetic outflow (ie T4-T6), placing him at risk for autonomic dysreflexia.¹⁻³ It is only rarely that reports have appeared in the literature describing this phenomenon in patients with spinal cord injuries below T4-T6, and in our experience, those patients with lower injury levels experiencing these episodes give a history of neurologic improvement from an original injury level above T4-T6. Autonomic dysreflexia occurs in both complete and incomplete injuries after resolution of the spinal shock phase of their injury¹ as in our patient.

Although the onset of this unusual presentation of autonomic dysreflexia was associated with a range of motion exercises, he experienced more episodes with reflex voiding and condom catheter drainage. The range of motion exercises have been associated with autonomic dysreflexia,¹⁵ but stimulation from the urinary tract is much more common.^{3,5} The range of motion exercises in this instance have resulted in increased intravesicular bladder pressure providing the needed noxious stimulation. Resolution of the episodes with the use of an adrenergic blocking agent (eg doxazosin) with improved bladder drainage and blockage of reflex sympathetic activity peripherally points to this etiology for this phenomenon. His neuropathic bladder dysfunction is currently managed with a program of clean intermittent catheterization.

The medical literature is limited in regard to long-term outcomes in patients experiencing seizures attributed to autonomic dysreflexia. One of three patients reported by Kursh *et al*¹² died shortly after a seizure associated with intracerebral hemorrhage. Other reports reviewed do not detail long term outcomes.^{5,6,8-12} We believe that patients who have experienced a seizure associated with autonomic dysreflexia are at increased risk for other serious sequelae and close monitoring and treatment of autonomic dysreflexia is very important. The sympa-

thetic stimulation sufficient to produce such a response in these individuals is profound and may well place them at increased risk for subsequent serious complications.

Two of our four patients who have experienced seizures have since died. The first patient sustained a massive intracerebral hemorrhage and was reported previously.¹ The second patient was 17 at the time of his SCI and after discharge from rehabilitation had a seizure during an autonomic episode with hypertension. During his subsequent care, he was noted to have a small cerebral infarct without clinical symptoms or signs. His death occurred approximately 16 months later and was not believed to be related to autonomic dysreflexia. Our patient in the present case is doing well without significant recurrences. The fourth patient has had no further seizures but continues to have significant difficulty with vasomotor instability with hypotension and intermittent autonomic dysreflexia for the past several years.

Conclusion

Autonomic dysreflexia manifested by transient aphasia and seizures is uncommon. This small population of spinal injured patients may be at increased risk of serious sequelae and should be monitored closely. We believe that long-term follow-up studies are clearly warranted to further assess the risk. The current availability of shared data bases (eg Model Systems) make such evaluation possible.

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