

Case Report

Unusual early autonomic dysreflexia in acute transverse myelitis – A case report

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ABSTRACT

Autonomic dysreflexia (AD) is a well-recognised event occurring most often during the late phase after recovery from spinal shock in patients with spinal cord injury. AD occurring soon after spinal cord dysfunction is uncommon, and that too in spinal cord disorders such as acute transverse myelitis (ATM). We present such a case of early onset AD occurring on day 7 of ATM at the thoracic level and highlight the clinical significance of recognising AD even at this early stage, as timely management of this potentially fatal condition is crucial.

Keywords: Acute transverse myelitis, Early autonomic dysreflexia, Spinal cord disorder

INTRODUCTION

Acute transverse myelitis (ATM) is an acquired focal inflammatory disorder that typically presents with the sudden onset of weakness, sensory deficits and bowel or bladder dysfunction, affecting the spinal cord at any level. Based on aetiology, it can be categorised into post-infectious, inflammatory, demyelinating and idiopathic, with idiopathic being the most common.^[1]

Autonomic dysreflexia (AD) is an uncommon but potentially life-threatening condition triggered by noxious stimuli arising below the level of spinal cord injury. These stimuli may originate from the skin, abdominal or pelvic viscera, skeletal muscles or most commonly, bladder distension-accounting for approximately 85% of cases. Faecal impaction is another frequent cause.^[2] Such stimuli elicit an exaggerated sympathetic response due to disrupted descending parasympathetic pathways, resulting in widespread vasoconstriction involving the splanchnic, muscular, vascular and cutaneous circulations.^[3]

In individuals with an intact autonomic system, this sympathetic surge activates baroreceptors in the carotid sinus and aortic arch, initiating a compensatory parasympathetic response via the vagus nerve, leading to generalised vasodilation. However, in patients with AD, this regulatory mechanism is impaired, potentially causing a sudden and severe elevation in blood pressure (BP) accompanied by reflex bradycardia, cardiac arrhythmias, headache, facial flushing and sweating above the level of injury. Clinical presentations can vary from asymptomatic to severe cardiovascular compromise.^[4,5]

Autonomic complications are well-documented among individuals with traumatic spinal cord injuries. However, the literature reveals only a few case reports highlighting early AD in patients with non-traumatic spinal cord disorders. Our case report, therefore, is pertinent as it highlights an early presentation of AD in a patient with ATM.

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CASE REPORT

A 25-year-old male not known to have any medical illness developed a sudden onset of progressive paraplegia, profound hypoesthesia with band-like sensation in the abdomen and urine incontinence for 3 days before presentation. There was no history of recent trauma, vaccination, dog bite, injectable drugs, preceding febrile illness and no family history of autoimmune diseases or similar illnesses. Upon examining the patient, he was conscious and oriented with a blood pressure of 110/70 mmHg, heart rate of 72 bpm and oxygen saturation of 98% on room air. A detailed neurological evaluation revealed bilateral lower limb hypotonia with a power of 0/5. Deep tendon reflexes were absent in both lower limbs, the abdominal reflex was absent, and the plantar reflex showed a bilateral mute response. In contrast, the tone and power in the upper limbs were normal. There was a complete sensory loss at a level of T6. Higher mental functions, cranial nerve and upper limb examinations were unremarkable. The patient had urinary retention, which required indwelling catheterisation at admission.

On admission, complete blood counts, electrolytes, renal and liver profiles and coagulation profile were within normal levels. The electrocardiogram (ECG) showed a normal sinus rhythm. A lumbar puncture was done, revealing cell count (10 cells with 60% lymphocytic predominance), protein (50 mg/dL) and glucose (44 mg/dL) within the normal range. The samples were sent for cell-based assay as well as serum antibody titres. Cerebrospinal fluid oligoclonal bands, neuromyelitis optica panel, culture and viral polymerase chain reaction were negative. Serum panel was unremarkable for antinuclear antibodies, neutrophil cytoplasmic antibodies and extra neutral alcohol. Fundus examination was unremarkable. Magnetic resonance imaging spine showed a central T2 hyperintense signal involving more than 50% cross-sectional area of the spinal cord at the T4 level [Figure 1]. A diagnosis of ATM with spinal shock was made, and the patient was started on pulse steroid therapy.

On day 7 of illness, the patient was found to be in asystole in the early morning hours and required immediate advanced cardiovascular life support. Return of spontaneous circulation was achieved after one cycle of cardiopulmonary resuscitation. Post-resuscitation, the patient remained unconscious, necessitating endotracheal intubation and initiation of mechanical ventilation. He was subsequently transferred to the intensive care unit the same day for intensive monitoring and critical care management.

Prior ECGs had demonstrated normal sinus rhythm; however, the ECG during the event revealed ventricular tachycardia, which later reverted to normal sinus rhythm in subsequent tracings. Serum electrolytes and arterial blood gas analyses were within normal limits. Throughout the earlier course of

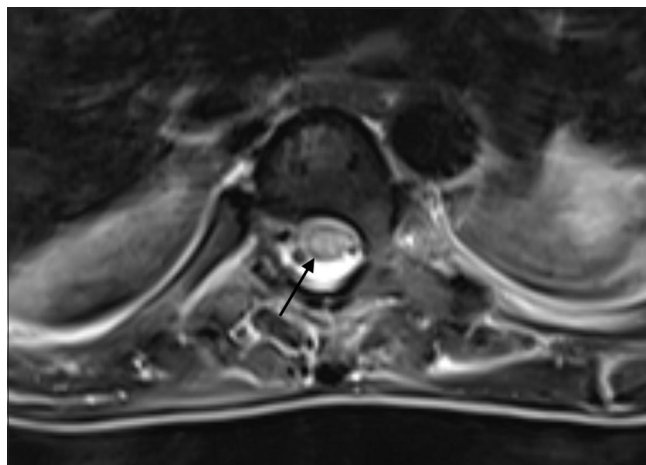


Figure 1: A twenty-five-year-old male presented with an acute onset, rapidly progressing paraparesis, sensory deficits with a level at T6, and bladder dysfunction, features consistent with transverse myelitis. Magnetic resonance imaging spine showed a central T2 hyperintense signal involving more than 50% cross-sectional area of the spinal cord at T4 level (arrow).

hospitalisation, the patient's blood pressure and heart rate had remained stable within normal ranges, without significant variability. Post-event, however, he exhibited marked haemodynamic fluctuations, with systolic blood pressure ranging from 120 to 160 mmHg, diastolic pressure from 70 to 110 mmHg and heart rate varying between 60 and 110 bpm [Figure 2].

A screening 2D echocardiogram showed preserved left ventricular ejection fraction with no evidence of valvular pathology, regional wall motion abnormalities or chamber dilatation. Subsequently, on enquiring, the patient's family reported that he had no complaints upon awakening, but lost consciousness suddenly while receiving leg massages from relatives. Hence, the possibility of AD precipitated by stimulation of calf muscles was kept as the patient met the criteria for AD.

Despite intensive care, the patient suffered a second cardiac arrest later that night and ultimately succumbed to his underlying condition.

Criteria of AD

Patients with paraplegia or quadriplegia with severe headache or found unconscious with Systolic blood pressure >150 mmHg or >40 mmHg above baseline should be considered highly suggestive of AD.^[3]

DISCUSSION

This case report presents an atypical manifestation of suspected ATM with possible early onset of AD. The patient exhibited an acute, rapidly progressive paraparesis, sensory

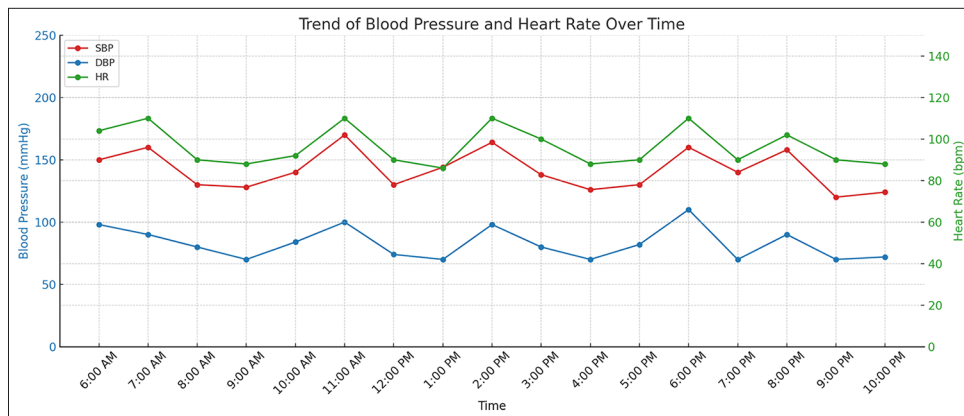


Figure 2: Graphical representation of blood pressure and heart rate variability over time following the suspected autonomic event.

deficits with a sensory level at T6, and bladder dysfunction—findings consistent with transverse myelitis. On the 7th day of illness, the patient developed clinical features raising the suspicion of AD.

Distinctive features that rendered this case unique included:

- Autonomic instability in the form of fluctuating heart rate and blood pressure emerged, which had not been observed earlier during the hospital stay
- AD is conventionally observed during the chronic phase of high cervical spinal cord injuries (above T5) as patients recover from spinal shock, given that autonomic activity is typically suppressed during the initial stage of spinal shock.^[2] However, in our patient, AD manifested acutely within 7 days of illness, an occurrence that is exceedingly rare according to existing literature
- The majority of reported cases of autonomic dysfunction, including AD, are secondary to traumatic spinal cord injury. However, only a limited number of cases have been documented in the context of non-traumatic aetiologies such as transverse myelitis, as observed in our patient.

A study by Cragg and Krassioukov highlights this anomaly, noting that the incidence of AD is about 7.5% in the acute phase compared to 90% in the chronic phase, with the majority of cases following traumatic spinal cord injury.^[4] Another study by Krassioukov *et al.* involved 58 individuals with acute traumatic spinal cord injuries, and the incidence of early AD was 5.2%. Every affected patient exhibited an elevated systolic blood pressure above baseline. Intriguingly, the earliest episode of AD emerged as soon as the 4th day post-injury.^[6] Similarly, as reported by Silver, in a series of 4 patients with acute cord transections accompanied by spinal shock, acute AD was noted between 7 and 31 days of presentation. Two of these cases showed a severe rise in blood pressure, and the other 2 presented with an over-distended bladder in response to traumatic catheterisation.^[2]

However, all these studies involved patients with acute spinal cord injuries. There are isolated case reports describing AD associated with ATM, as observed in our patient. In a case reported by Moreno-Escobar *et al.*, a 41-year-old man presented with transverse myelitis secondary to COVID infection and developed labile blood pressure and tachyarrhythmias on day 4, confirming a dysautonomic state. He was managed conservatively and improved with treatment.^[7] A similar case reported by Furlan, a 60-year-old female, developed cardiovascular AD when diagnosed with acute onset quadriplegia due to neuromyelitis optica spectrum disorder, with a potential trigger identified to be faecal impaction.^[8]

Given the potentially fatal nature of AD, it is imperative to avoid potential triggers and ensure diligent monitoring of the patient's vital signs. Early recognition and appropriate management of AD episodes are crucial to prevent severe complications and associated mortality. As AD can be challenging to identify for patients, caregivers and healthcare professionals, there is a prompt need for increased awareness regarding this condition, even in early stages.

CONCLUSION

This case underscores the clinical importance of recognizing autonomic dysreflexia as a potential early complication of acute transverse myelitis, even within the first week of illness. Although AD is classically associated with chronic cervical spinal cord injuries, its occurrence in non-traumatic spinal cord disorders such as ATM, though rare, highlights the need for heightened vigilance. Timely identification, avoidance of triggers, and prompt management are essential to prevent life-threatening complications.

Ethical approval: Institutional Review Board approval is not required.

Declaration of patient consent: The authors certify that they have

obtained all appropriate patient consent forms. In the form, the patients have given their consent for their image and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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