Case Report

Brainstem Haemorrhage due to Autonomic Dysreflexia in a Person with C6 Tetraplegia

Swapna Patil[1], Raji Thomas[2]

Abstract

Autonomic dysreflexia is an important clinical complication occurring in patients with high levels of spinal cord injury. If untreated, the acute rise in blood pressure can cause end organ damage, including intracerebral haemorrhage. Though unusual, it can be fatal with large haemorrhages causing brain herniation syndromes. Here we report the case of a patient with C6 complete tetraplegia patient who developed brainstem haemorrhage during an episode of autonomic dysreflexia. The pathophysiology and treatment methods of this condition are discussed, highlighting the importance of preventive measures to avoid the same.

Key words: **Autonomic dysreflexia, spinal cord injury, cerebral haemorrhage, hypertensive encephalopathy.**

Introduction:

Autonomic dysreflexia is an acute medical emergency occurring in patients with spinal cord injury. Patients with tetraplegia and high paraplegia (at or above T6 level) are at risk of developing this complication[1]. In these patients, there is disconnection between the spinal sympathetic centres and the supraspinal sympathetic centres leading to loss of organised/ controlled sympathetic outflow below the level of the spinal lesion[2]. It is often triggered by non-specific stimuli below the level of lesion, bladder and bowel distension being the most common causes[1,2]. Clinical features frequently include sudden rise in blood pressure, headache, sweating, and flushing[2]. Early recognition and removal of the triggering factors leads to spontaneous resolution of the crisis. In case of inappropriate or delayed treatment, the sudden rise in blood pressure can cause end organ damage, including damage to the central nervous system leading to hypertensive encephalopathy or rarely intracerebral haemorrhage[1, 2].

Literature review shows few case reports of intracranial haemorrhage associated with autonomic dysreflexia. There are no previous reports of brainstem haemorrhage associated with this condition. Here we report the case of a patient with C6 tetraplegia who developed brainstem haemorrhage secondary to autonomic dysreflexia.

Case Report:

A 30-year-old male presentedwith history of weakness and loss of sensation in both lower limbs and upper limbs following fall from height in August 2010. He was diagnosed to have fracture of the C6 vertebral body with C6-C7 dislocation (Fig 1) and was managed with cervical traction in the Spinal Disorders Unit. Thereafter he was bedridden, developed pressure sores and was on indwelling urethral catheter.

Eight months after the event, he was referred to us for rehabilitation. On examination he was found to have C6 complete tetraplegia with pressure sores and chronic collapse of the left lung secondary to a mucus plug in the bronchial tree.The pressure sores were managed conservatively. Left lung collapse was managed with chest physiotherapy. With 6 weeks of rehabilitation,he was discharged to be partially independent from a wheelchair and was on an indwelling catheter (clean

Authors' affiliation:

[1] *MBBS, MD, Assistant Professor*
[2] *MBBS, MD, DNB, Professor*
Department of Physical Medicine and Rehabilitation, Christian Medical College, Vellore, Tamil Nadu.

Cite as:

Swapna Patil, Raji Thomas. Brain stem Haemorrhage due to Autonomic Dysreflexia in a Person with C6 Tetraplegia. IJPMR, December 2015; Vol 26(4) : 114-16

Correspondence:

Dr. Swapna Patil, Department of Physical Medicine and Rehabilitation, Christian Medical College, Vellore, Tamil Nadu. dr.swapnapatil@gmail.com[1], rajithomas@cmcvellore.ac.in[2]

Received on 20/10/2015, Accepted on, 11/12/2015

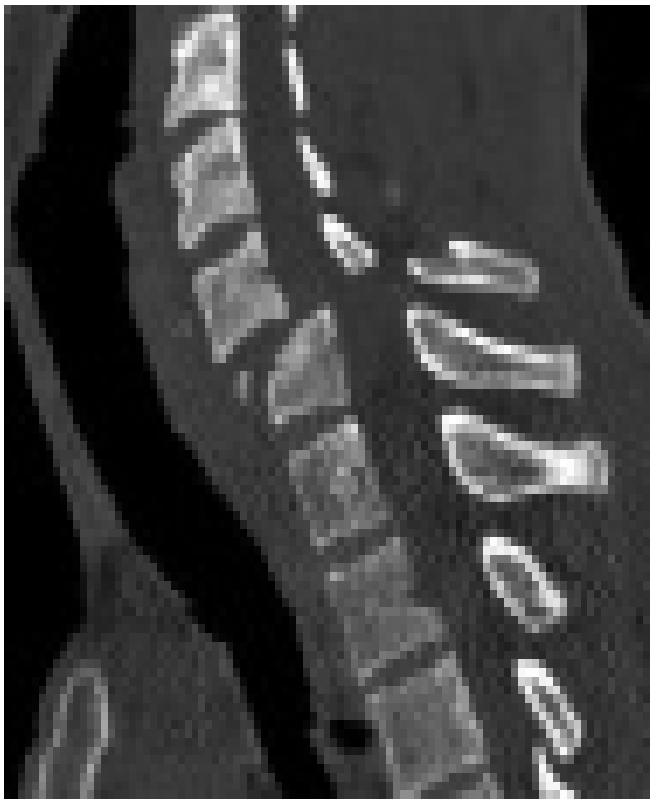

Fig 1: CT Cervical Spine Sagittal Image Showing Fracture of the Spinous Process of C6 Vertebra, Anterolisthesis of C6 over C7 and Compression Fracture of Anterosuperior Border of C7

intermittent catherisation was not considered in view of poor hand functions), engaging in a meaningful vocation. Two months following discharge, he again presented withpounding headache,right facial paralysis and double vision of three days duration. He gave history of an episode of bladder distension with an increase in blood pressure to 180/110 mm Hg associated with the onset of these symptoms. The blood pressure hadcome down over few hours after bladder evacuation. On examination,he had right sided partial ptosis, right lateral rectus palsy, right sided gaze evoked nystagmus, right sided LMN facial palsy and impaired gag reflex. MRI brain showed a 10x12x15 mm lesion situated inferomedial to the right middle cerebellar peduncle and posterolateral to the medulla. This was isointense to the gray matter and non-enhancing. Marked blooming was seen on SWI images (Fig 2). Features were suggestive of subacute haematoma.

During the hospital stay, he continued to have episodic headaches which were associated with sudden rise in blood pressure. He was started on amlodipine 2.5 mg once daily with which blood pressure normalised. After the acute phase settled he was continued on the

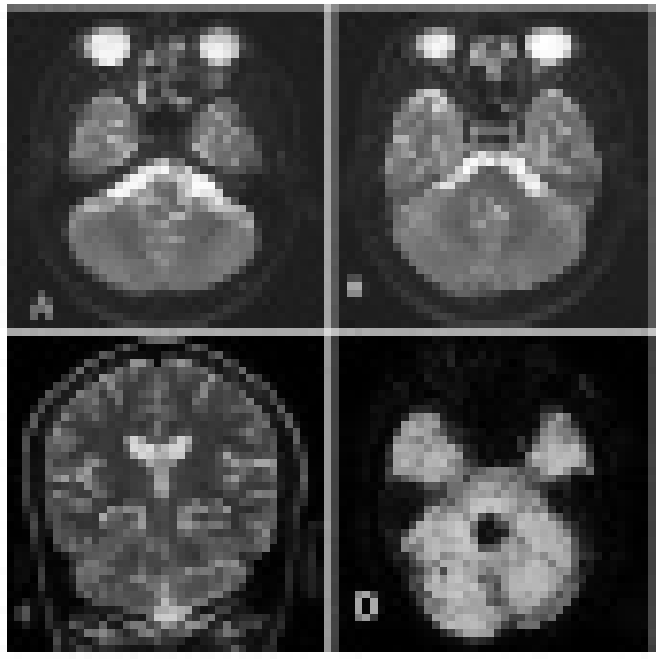

Fig 2 : MRI Brain T2 Axial (A and B) and Coronal (C) Images Showing Isointense Lesion with Surrounding Area of Hyperintensity Inferomedial to the Right Middle Cerebellar Peduncleand Posterolateral to the Medulla. SWI (D) Image Shows Marked Blooming of the Lesion

rehabilitation programme.Later amlodipine was stopped and suprapubic catheterisation was done. There were no further episodes of autonomic dysreflexia.

Discussion:

Autonomic dysreflexia (AD) was first observed by Anthony Bowlby in 1890, and described by Guttmann and Whitteridge in 1947. It is seen in patients with cervical or high thoracic cord injury (at or above T6 level below which the main sympathetic outflow exits from the spinal cord), occurring in up to 50 - 90% of these patients. AD increases with ascending level and completeness of SCI/ injury severity. It is 3 times commoner after complete injuries than after incomplete injuries[1-4].

AD after spinal cord injury is due to changes in the spinal and peripheral autonomic circuits, occurring during the acute and chronic stages of SCI. The mechanisms involved include loss of supraspinal inhibitory control due to destruction of the descending vasomotor pathways, plasticity occurring within the spinal cord mainly at the level of the spinal sympathetic neurons and the primary afferents and changes in the sensitivity of peripheral adrenergic receptors[2-4]. It usually develops weeks to months after the SCI, but can occur as early as 4 days after severe cervical cord injuries[2, 4, 5].

Noxious and non-noxious stimuli can trigger this condition, with common stimuli being bowel and bladder distension, spasms, and pressure sores. Other triggers include urethral catheterisation, bladder percussion, urinary tract infections, cystoscopy, cystometry, and electrical stimulation of muscles[2-5].

AD is characterised by episodes of extreme hypertension (systolic blood pressure up to 300 mmHg). As spinal cord injury patients usually have low systolic BP (90 to 110 mmHg range or 15 to 20 mmHg lower than normal people), an increase in blood pressure by 20 to 40 mmHg above the baseline can also be considered as a feature of AD, if associated with other features. Intensity of these episodes varies from being asymptomatic, or associated with mild discomfort and headache to a life threatening hypertensive emergency/ encephalopathy[1-6].

Clinical features include hypertension, bradycardia, pounding headache, increased spasticity, sweating, blurred vision, nasal congestion, cutis anserine, piloerection, upper body flushing, and apprehension. If untreated, these episodes can cause intracranial haemorrhage with risk of brain herniation, cardiac complications, retinal detachment, seizures and death[1-6].

Previous reported cases of intracerebral haemorrhage following autonomic dysreflexia were in the left putamen[7], left basal ganglia and thalamus leading to death[5], left occipital region[8], right thalamus[9], cerebellum[10] and right putamen with extrinsic compression of the lateral ventricle[1]. Massive right cerebral haemorrhage with rupture into the lateral, third and fourth ventricles with bilateral uncal, trans-tentorial and cerebellar tonsillar herniation has also been reported[6].

Our patient had C6 complete tetraplegia and he developed features of AD related to bladder distension 10 months after the injury. During this episode he had sudden increase in blood pressure and subsequently brainstem haematoma. A literature search could not find any previous reports of brainstem haemorrhage following autonomic dysreflexia. The LMN facial palsy could have resulted from involvement of the facial nucleus in the pons;due to extension from the dorsolateral medullary lesion. This episode subsided with bladder evacuation, but produced new neurological deficits in addition to the old deficits. On follow-up there was improvement in the diplopia and ptosis but facial palsy persisted.

Conclusions:

Brainstem haemorrhage, though a rare complication of autonomic dysreflexia, due to its potential morbidity and mortality becomes significant in patients with spinal cord injury. Preventive measures, early diagnosis and adequate treatment would avoid life threatening complications related to AD.

References:

1. Pan SL, Wang YH, Lin HL, Chang CW, Wu TY, Hsieh ET. Intracerebralhemorrhage secondary to autonomic dysreflexia in a young person with incomplete C8 tetraplegia: a case report. *Arch Phys Med Rehabil* 2005; **86:**591-3.
2. Krassioukov A. Autonomic dysreflexia: current evidence related to unstable arterial blood pressure control among athletes with spinal cord injury. *Clin J Sports Med* 2012; **22:**39–45.
3. Popa C, Popa F, Grigorean VT, Onose G, Sandu AM, Popescu C, *et al*. Vascular dysfunctions following spinal cord injury. *J Med Life*. 2010; **3:** 275-85.
4. Krassioukov A, Blackmer J, Teasell RW, Eng JJ. Autonomic dysreflexia following spinal cord injury. In: Eng JJ, Teasell RW, Miller WC, Wolfe DL, Townson AF, Hsieh JTC, editors. Spinal Cord Injury Rehabilitation Evidence. 2012. Version 4.0. Vancouver: 1-31.
5. Yoo KY, Jeong CW, Kim WM, Lee HK, Kim SJ, Jeong ST, *et al.* Fatal cerebral hemorrhage associated with autonomic hyperreflexia during surgery in the prone position in a quadriplegic patient: a case report. *Minerva Anestesiol* 2010; **76:** 554-8.
6. Eltorai I, Kim R, Vulpe M, Kasravi H, Ho W. Fatal cerebral haemorrhage due to autonomic dysreflexia in a tetraplegic patient: case report and review. *Paraplegia* 1992; **30:**355-60.
7. Tadashi S. Hypertensive intracerebral hemorrhage due to autonomic dysreflexia in a young man with cervical cord injury. I *UOEH* 06/2013; **35:**159-64.
8. Vallès M, Benito J, Portell E, Vidal J. Cerebral hemorrhage due to autonomic dysreflexia in a spinal cord injury patient. *Spinal Cord* 2005; **43:**738–40.
9. Eker A, Yigitoglu PH, Ipekdal HI, Tosun A. Acute Onset of Intracerebral Hemorrhage due to Autonomic Dysreflexia. *Korean Neurosurg Soc* 2014; **55:**277-9.
10. Yokomizo Y, Goubara A, Tanaka K, Yokoyama O. A case of cerebellar hemorrhage secondary to autonomic dysreflexia (AD) in a patient with cervical spinal cord injury. *Hinyokika Kiyo* 2010; **56:**659–61.