

Case Report

Acute Onset of Intracerebral Hemorrhage due to Autonomic Dysreflexia

Amber Eker, M.D.,¹ Pembe Hare Yigitoglu, M.D.,² H. Ilker Ipekdal, M.D.,¹ Aliye Tosun, M.D.²

Departments of Neurology,¹ Physical Medicine and Rehabilitation,² Near East University Faculty of Medicine, Nicosia, Cyprus

Autonomic dysreflexia is a clinical emergency syndrome of uncontrolled sympathetic output that can occur in patients who have a history of spinal cord injury. Despite its frequency in spinal cord injury patients, central nervous system complications are very rare. We report a man with traumatic high level incomplete spinal cord injury who suffered hypertensive right thalamic hemorrhage secondary to an episode of autonomic dysreflexia. Prompt recognition and removal of the triggering factor, the suprapubic catheter obstruction which led to hypertensive attack, the patient had a favorable functional outcome after the resorption of the hematoma and effective rehabilitation programme.

Key Words : Spinal cord injury · Intracerebral hemorrhage · Autonomic dysreflexia · Urinary tract obstruction.

INTRODUCTION

Autonomic dysreflexia (AD) is a well known condition of an uncontrolled sympathetic output, that generally occurs in patients with spinal cord injury (SCI) who have a lesion above T6 spinal level¹. AD is caused by spinal reflex mechanisms that remain intact despite the injury of the spinal cord. In SCI patients, blood pressure levels can fluctuate easily. Orthostatic hypotension and hypertensive attacks influence greatly the quality of life of these patients. In addition, the hypertensive attacks can be life-threatening by causing end organ damage¹⁴. Central nervous system complications are uncommon but it may be the most common reason of morbidity and mortality. AD is an important clinical diagnosis that requires prompt recognition and treatment in order to avoid this devastating complications.

AD is often triggered by noxious stimulus below the level of the SCI. Most important aspect of the management of AD is prevention, early recognition and removing of the triggering factors. In particular, the urinary tract obstruction and fecal impaction, which are the most common triggers of AD, must be checked out urgently and regularly.

Here, we report a SCI patient who had a rare hypertensive cerebral hemorrhage complication during an AD episode and we discuss the pathophysiology and the treatment methods of the AD.

CASE REPORT

The patient was a 43-year-old man who had a American Spinal Injury Association Impairment Scale-B (ASIA-B) type incomplete SCI at level C6 secondary to a traffic accident for three years (Fig. 1). He was under a physiotherapy programme for his quadriparesis. He had been diagnosed with autonomic dysreflexia due to the hypertensive attacks which were followed with hypotension and bradycardia. His neurogenic bladder was initially managed with an indwelling foley catheter and then clean intermittent catheterisation for two years. Because of persistent attacks, suprapubic catheterization with cystofix has been performed one year ago. The patient admitted to our emergency department with letargy, left sided facial paresis and worsening in his paresis in the left upper extremity. His family described the history of cystofix dysfunction with abdominal distention and increasing in blood pressure up to 220/100 mm Hg right before these symptoms. His cranial CT revealed right thalamic and intraventricular hematoma with 21 mm³ in volume (Fig. 2). He has been followed-up with medical treatment and his cystofix catheter had been replaced with a wider one. Afterward, there were no additional AD attacks. He became alert and his nascent motor deficit on his upper extremity improved completely.

• Received : March 28, 2013 • Revised : September 9, 2013 • Accepted : May 15, 2014

• Address for reprints : Amber Eker, M.D.

Department of Neurology, Near East University Faculty of Medicine Hospital, Near East Avenue, Nicosia 922022, Cyprus

Tel : +90-392-675-1000-1371, Fax : +90-392-675-1090, E-mail : ambereker@yahoo.com

• This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

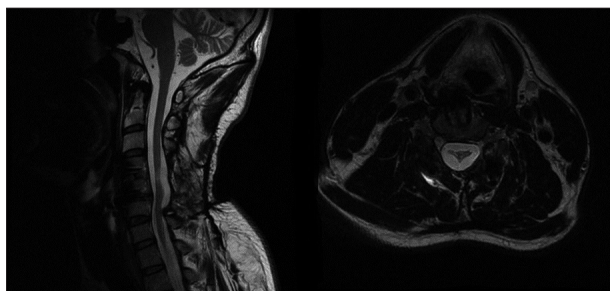


Fig. 1. Cervical MRI demonstrates the atrophy of the spinal cord below the level of the trauma.

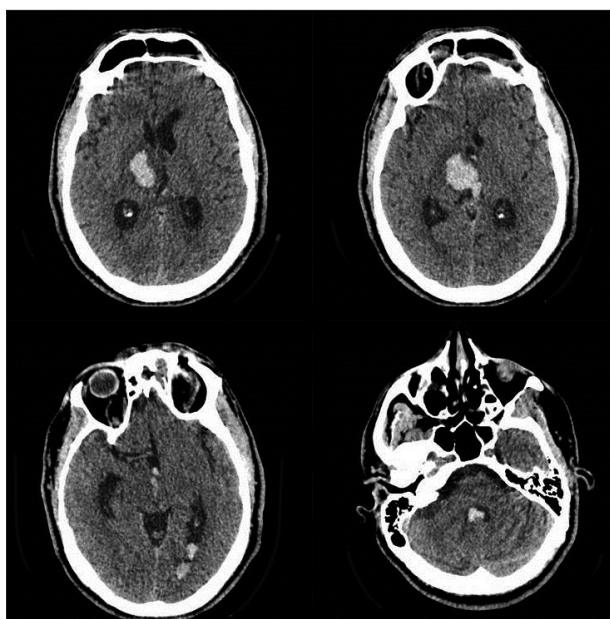


Fig. 2. Cranial CT scan demonstrates hemorrhage in the right thalamic nucleus and also in the third, fourth and lateral ventricles.

DISCUSSION

AD is a common complication of high level spinal cord injury. Although it is common in SCI, non-traumatic diseases such as multiple sclerosis and spinal cord tumor can also be the cause of AD². AD mainly occurs in chronic stage of disease both in the complete and the incomplete injury group¹.

The pathophysiology of AD is related with the disconnection of the spinal sympathetic centers from supraspinal control. Normal regulation of sympathetic output from the spinal cord is modulated by input from the higher centers. After SCI, this system is no longer retain by descending control¹. The development of AD after injury is a multifactorial complex issue. Degenerative and plastic changes within the injured cord play the main roles. These changes are thought to occur because of the synaptic re-organisation and reduction in gliosis around the pre-synaptic sympathetic neurones^{2,5,14}. As a result of this process, exaggerated reaction occurs within the sympathetic pre-ganglionic neurones as a response to the afferent stimulus. The reason that AD is a feature of lesions at or above the T6 level is related

with splanchnic circulation response to this sympathetic over-activity. Overactivity below the injury level results with splanchnic and peripheral vasoconstriction and causes hypertension. The excessive parasympathetic output (and lack of sympathetic tone) above the level of the lesion results with peripheral vasodilation. Hence, patients experience sweating, nasal congestion, flushing and bradycardia. In brief, AD result from the parasympathetic excitation above the level of injury, and sympathetic excitation below the level of injury and clinically it is characterized by an acute increase in blood pressure, headache, sweating, facial flushing and bradycardia^{1,2,5,11}. We must always be aware of and search these features during the SCI patients follow up.

This AD crisis may lead to injuries in cardiac, pulmonary, retinal and renal tissues¹⁶. Uncommonly, this hypertensive crises may also effect central nervous system by causing cerebral hemorrhage^{10,12-16} and more rarely posterior reversible encephalopathy syndrome (PRES) or reversible cerebral vasoconstriction syndrome^{3,7,13}. Central nervous system involvement is very rare and if occurs, it is most commonly asymptomatic. But, it can also result in altered mental status, local neurologic deficits and seizures^{4,16}. Intracranial hemorrhage is an uncommon complication of AD but fatal cases with massive bleeding have also been reported^{6,8,10,13,17}. Here, we report a rare symptomatic cerebral hemorrhage which is result from an AD attack.

Physicians should repeatedly check and note the neurologic status of every SCI patient even during chronic stage because it may be difficult to distinguish the new neurologic findings in this tetraparetic or tetraplegic high level SCI patients.

The main triggering factor of AD is related with the urinary tract. Blocked or kinked indwelling urinary catheters are one of the most common causes. Choosing the safest and appropriate method for the patients and family education for controlling the function of the catheter are the important issues. The goal in all bladder treatment is the creation of a balanced bladder, with low intravesical pressure that is drained totally at regular intervals and is free of urinary tract infections. In patients with SCI, clean intermittent catheterization has been recommended rather than indwelling urinary catheters if the patient and family can adjust⁹.

There are also many other urological causes for AD like bladder distension, urinary tract infection, bladder or kidney stones, urologic procedures, detrusor sphincter dysinergia, epididymitis or scrotal compression. The second most common causes of AD due to SCI are gastrointestinal problems, including faecal impaction and rectal distension also anorectal conditions like haemorrhoids and fissures. Orthopedic conditions such as fractures, dislocation, heterotopic ossification and general surgical conditions of the abdominal organs, such as appendicitis, gastro-oesophageal reflux, and peptic ulceration may presipitate the AD. Bed wounds, skin ulceration or ingrowing toenails may be the dermatologic triggering factors. Female patients also have higher risk for developing AD during pregnancy and labour^{1,2,5}. All of these triggering factors must be kept in mind

during evaluation of a patient with AD.

The first step of the treatment of AD, regardless of the cause, is to make patient sit in an upright position to maintain an orthostatic decrease in the blood pressure. Main treatment is removal of the triggering factors. Antihypertensive drugs are rarely needed. If needed, the calcium antagonists or adrenoceptor blockers may be beneficial in the treatment of AD^{1,2,5}.

This life-threatening complication should be kept in mind in the patients, especially with upper SCI. The medical team who are dealing with SCI patients as well as the SCI patients themselves and their relatives should be aware of this disorder and appropriate bladder care, bowel exercises and skin care should be provided.

CONCLUSION

This case has been reported to emphasises the rare hypertensive cerebral hemorrhage complication during common AD episodes in SCI. Clinicians should always closely monitor the patients with SCI, especially those in the high spinal levels. These patients can easily develop AD which is triggered by the stimulation of lower parts of the body. Our patient had a common triggering factor; urinary drainage malfunction. Prompt removal of the triggers and choosing the safest urinary drainage method are very important to save the patients from the complications of AD.

References

- Blackmer J : Rehabilitation medicine : 1. Autonomic dysreflexia. *CMAJ* 169 : 931-935, 2003
- Bycroft J, Shergill IS, Chung EA, Arya N, Shah PJ : Autonomic dysreflexia : a medical emergency. *Postgrad Med J* 81 : 232-235, 2005
- Chaves CJ, Lee G : Reversible posterior leukoencephalopathy in a patient with autonomic dysreflexia : a case report. *Spinal Cord* 46 : 760-761, 2008
- Colachis SC, Fugate LP : Autonomic dysreflexia associated with transient aphasia. *Spinal Cord* 40 : 142-144, 2002
- Dincer U : Autonomic dysfunction and their treatment in spinal cord injury. *Turk J Phys Med Rehab* 56 Suppl 2 : 93-98, 2010
- Dolinak D, Balraj E : Autonomic dysreflexia and sudden death in people with traumatic spinal cord injury. *Am J Forensic Med Pathol* 28 : 95-98, 2007
- Edvardsson B, Persson S : Reversible cerebral vasoconstriction syndrome associated with autonomic dysreflexia. *J Headache Pain* 11 : 277-280, 2010
- Eltorai I, Kim R, Vulpe M, Kasravi H, Ho W : Fatal cerebral hemorrhage due to autonomic dysreflexia in a tetraplegic patient : case report and review. *Paraplegia* 30 : 355-360, 1992
- Furusawa K, Tokuhiko A, Sugiyama H, Ikeda A, Tajima F, Genda E, et al. : Incidence of symptomatic autonomic dysreflexia varies according to the bowel and bladder management techniques in patients with spinal cord injury. *Spinal Cord* 49 : 49-54, 2011
- Pan SL, Wang YH, Lin HL, Chang CW, Wu TY, Hsieh ET : Intracerebral hemorrhage secondary to autonomic dysreflexia in a young person with incomplete C8 tetraplegia : a case report. *Arch Phys Med Rehabil* 86 : 591-593, 2005
- Park KS, Lee YW : Cardiovascular response to experimental spinal cord trauma in cats. *J Korean Neurosurg Soc* 10 : 419-426, 1981
- Vallès M, Benito J, Portell E, Vidal J : Cerebral hemorrhage due to autonomic dysreflexia in a spinal cord injury patient. *Spinal Cord* 43 : 738-740, 2005
- Vallès-Casanova M, Benito-Penalva J, Portell-Soldevila E, Vidal-Samsó J : [Cerebral hemorrhage secondary to autonomic dysreflexia in a spinal cord injury]. *Med Clin (Barc)* 127 : 358, 2006
- Weaver LC, Marsh DR, Gris D, Brown A, Dekaban GA : Autonomic dysreflexia after spinal cord injury : central mechanisms and strategies for prevention. *Prog Brain Res* 152 : 245-263, 2006
- Yamashita T, Hiramatsu H, Sakai N, Namba H : Cerebral hemorrhage due to posterior reversible encephalopathy syndrome associated with autonomic dysreflexia in a spinal cord injury patient. *Neurol Med Chir (Tokyo)* 52 : 640-643, 2012
- 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* 56 : 659-661, 2010
- 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* 76 : 554-558, 2010