Case report

Autonomic dysreflexia-induced reversible posterior leukoencephalopathy syndrome in patients with spinal cord injury: Two case reports

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Background/objectives: To report two cases of reversible posterior leukoencephalopathy syndrome (RPLS) in patients with traumatic cervical spinal cord injury.

Design: Case report.

Setting: Rehabilitation Inpatient Unit, Pusan National University Hospital, Yangsan-si, Korea.

Results: Two men with spastic tetraplegia developed autonomic dysreflexia. Use of antihypertensive medication and Foley catheter insertion prevented further episodes of acute arterial hypertension and development of new symptoms.

Conclusion: RPLS can occur in the setting of autonomic dysreflexia in patients with traumatic cervical cord injury. The prompt recognition of this syndrome is of importance to prevent further morbidity and mortality in patients with spinal cord injury.

Keywords: Autonomic dysreflexia, Reversible posterior leukoencephalopathy syndrome, Spinal cord injuries, Spasticity, Tetraplegia, Bladder distension, Antihypertensives, Nifedipine, Nitroglycerin

Introduction

Reversible posterior leukoencephalopathy syndrome (RPLS) is identified on magnetic resonance imaging (MRI) by posterior-predominant white and gray matter lesions. The most common clinical symptoms of this syndrome are headaches, seizures, visual complaints, altered alertness ranging from drowsiness to stupor.¹ Autonomic dysreflexia (AD) is seen in patients with spinal cord injury (SCI) at the level of T6 or higher. It is caused by sudden reflex sympathetic activity uninhibited by supraspinal centers, resulting in systolic and diastolic hypertension.² Only one previous case report of RPLS resulting from AD exists.³ Here, we report two patients with spastic tetraplegia who developed

RPLS along with AD, the result of bladder overdistension.

Case 1

A 58-year-old man had a traumatic complete spinal cord lesion C5 American Spinal Injury Association Impairment Scale (AIS) A resulting from a C5-C6 fracture.⁴ The patient had a cystostomy and complained intermittently of a headache when his bladder became full after clamping of the urine bag. Basal blood pressure was 110/95 mmHg. At each episode of headache, the patient's blood pressure rose to 160/80 mmHg.

The patient was admitted to the emergency room due to an inability to be awakened during sleep. His blood pressure was 160/80 mmHg, and, as his respiration rate was low, a ventilator was applied. His bladder was emptied and nifedipine was given. Emergency cerebral MRI showed T2 hyperintensities in the subcortical

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Figure 1 (A,B) Initial FLAIR magnetic resonance imaging (MRI) of the brain showing T2 hyperintensities in the subcortical white matter of both parietooccipital lobes, temporal lobes, cerebellum, and basal ganglia. (C,D) Three months later, MRI showed decreased T2 hyperintensities in the subcortical white matter and increased bilateral periventricular leukoencephalopathy.

white matter of both parieto-occipital lobes, temporal lobes, cerebellum, and basal ganglia (Fig. 1A and 1B). He was diagnosed with PRES. After 5 days, the patient became fully alert and his respiration normalized, so he was removed from ventilation. His Glasgow coma scale (GCS) score was 13. Two months later, his blood pressure was 80/50 mmHg, and he was diagnosed with orthostatic hypotension and prescribed fludrocortisones.

Early in the morning, 3 months after admission, saturation of the patient decreased again. Emergency brain MRI showed decreased T2 hyperintensities in the subcortical white matter and increased bilateral periventricular leukoencephalopathy (Fig. 1C and 1D). After transfer to the intensive care unit (ICU), three tonic- and clonic-type seizures occurred. Lorazepam was given, but the patient's blood pressure fluctuated from 70/40 to 240/100 mmHg. Over 5

days in the ICU, the patient's mental status slowly improved. After tracheostomy and antibiotic therapy for pneumonia, the patient was again removed from ventilation.

Case 2

A 54-year-old man had a traumatic incomplete spinal cord lesion C6 AIS C resulting from a C6-C7 fracture. The patient, who had also been diagnosed with orthostatic hypotension and AD, complained intermittently of a headache believed to be due to AD, which resulted from an overactive bladder. The patient's basal blood pressure was 125/85 mmHg, which rose to 160/110 mmHg when he complained of headache. The patient developed unstable blood pressure caused by AD just before admission. Just before urethral catheterization to drain his bladder, the patient developed decreased mentality and seizure-like movement

Figure 2 (A,B) Initial MRI showed T2 hyperintensities in the subcortical white matter of both parietooccipital lobes and cerebellum. (C,D) MRI done 4 weeks later showed complete resolution of the T2 hyperintensities at both parietooccipital lobes and cerebellum.

accompanied by uncontrolled blood pressure with a systolic blood pressure of 180 mmHg. A lack of responsiveness and confused mentality were noted, the patient's GCS score was 5. Cerebral MRI showed T2 hyperintensities in the subcortical white matter of both parieto-occipital lobes and the cerebellum (Fig. 2A and 2B). Multiple hemorrhagic foci in the lesion were also observed. To control the AD, a Foley catheter was inserted and the patient's blood pressure was controlled by nifedipine. After 2 days of ICU, the patient's mentation fully recovered and his GCS score was 14. Urodynamic study showed overactive type bladder. The patient was given oxybutynin with straight intermittent catheterization every 4–6 hours.

A follow-up MRI 4 weeks later showed complete resolution of the T2 hyperintensities at both parieto-occipital lobes and the cerebellum (Fig. 2C and 2D). He did not show any neurologic or cognitive deficits resulting from RPLS.

Discussion

RPLS has many causes, including acute severe hypertension, preeclampsia and eclampsia, renal disease, or immunosuppressive and cytotoxic drugs.¹ Most authors state that hypertensive encephalopathy is the cause of this syndrome, which has been thoroughly investigated clinically and experimentally.⁵

AD is a composite of symptoms resulting from a sudden rise in blood pressure.² It is caused by a noxious, non-noxious, and visceral stimulus below the level of the injury. The most common source is from the bladder, either from overdistension or infection, followed by fecal impaction. The stimuli result in an exaggerated sympathetic activity below the lesion resulting in vasoconstriction. Symptoms of AD include hypertension, headache, profuse sweating, and cutaneous vasodilatation with flushing of the face, neck.⁶ Hypertension is defined as a rise of 20–40 mmHg above baseline.⁷ In these cases, both patients complained of a headache upon

bladder distension, and their blood pressures rose to the defined level of hypertension in AD. This hypertension was thought to cause RPLS in both patients. Patients with complete SCI (AIS A or AIS B)⁶ had a higher percentage of AD during their initial hospitalization than did patients with incomplete SCI. However, patients with motor-incomplete injuries also were at risk for developing AD, especially when the exposed to more noxious, non-noxious, and visceral stimulus.³

RPLS is typically characterized radiologically by focal regions of symmetric hemispheric edema. The parietal and occipital lobes are most commonly affected.⁵

Conclusion

RPLS is reversible when treatment is instituted early. The early recognition and treatment of high blood pressure and seizure is the mainstay of treatment. The treatment of acute AD must be prompt and efficient. Blood pressure should be lowered immediately with the use of fast-acting antihypertensive agents, usually nitroglycerin or nifedipine.⁷ The bladder should be checked for overdistension and emptied promptly. In these patients, the blood pressure responded promptly

to medication when AD was suspected as a cause of decreased mental acuity.

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