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Case Report

Cerebral hemorrhage due to autonomic dysreflexia in a spinal cord injury patient

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Study design: Case report.
Objective: To report an uncommon case of cerebral hemorrhage due to autonomic dysreflexia (AD) in a spinal cord injury (SCI) patient.
Setting: Institut Guttmann, Neurorehabilitation Hospital in Barcelona, Spain.
Case report: An SCI patient developed AD due to urinary tract infection after surgery for a pressure sore. The hypertension was difficult to control and the case progressed to hypertensive encephalopathy. MRI of the brain was performed showing a hemorrhagic lesion on the left occipital area. The hypertension was finally controlled and the neurological status improved although with some cognitive deficits.
Conclusion: This is an uncommon case of cerebral hemorrhage due to AD, showing the importance of an adequate diagnosis and treatment of AD to avoid this life-threatening complication.
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Keywords: cerebral hemorrhage; hypertensive encephalopathy; autonomic dysreflexia; spinal cord injury

Introduction

Autonomic dysreflexia (AD) is a known complication in patients with spinal cord injury (SCI) above T6. It is a common problem in many patients with SCI and can be very dangerous due to a significant rise in blood pressure resulting in a hypertensive emergency.[1] Hypertension may affect, among others, the central nervous system (CNS), causing hypertensive encephalopathy, and in rare cases cerebral hemorrhage.

We present a patient with SCI and a history of AD resulting in hypertensive encephalopathy in the past, who had a cerebral hemorrhage during a new episode of AD.

Case description

The patient is a 48-year-old male with T4 American Spinal Injury Association Impairment Scale A paraplegia due to motor vehicle accident in 1979. He manages his bladder with an indwelling foley catheter. He was known to have a small bladder capacity and a history of recurrent urinary tract infection (UTI). He has a history of AD episodes, most commonly related to his neurogenic bladder.

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In 2002, the patient was admitted at another hospital with the diagnosis of hypertensive encephalopathy. This presented in the context of a UTI. During that hospitalization, he had a normal MRI of the brain, and normal laboratory work up. He was discharged home and a posterior rhizotomy was recommended at that time with the goal of deafferentization of the bladder. The patient refused the intervention.

In January of 2004, the patient was admitted to our hospital for the surgical treatment of a nonhealing pressure ulcer over the left ischium. His baseline blood pressure was 125/80 mmHg. He underwent plastic surgery with a gluteus rotational flap. At 3 days after surgery, he presented with fever and hematuria, and was started on antibiotics for probable UTI. After a day, he developed sustained hypertension up to 220 mmHg systolic and 120 mmHg diastolic with confusion. The hypertension did not respond to standard treatment including oral nifedipine, or sublingual nitroglycerine, and he was started on intravenous nitroprusside and later on intravenous labetalol. Since the severe hypertension was thought to be related to noxious stimuli below his level of SCI, the patient was started on bupivacaine by epidural infusion pump, with a better control of the blood pressure. The patient was diagnosed as having hypertensive encephalopathy due

to sustained hypertension due to AD due to a UTI. The neurological exam showed a somnolent patient, with eye opening to verbal stimuli, spontaneous movement of the upper extremities, aphasia, pupils equally round and reactive to light, and eye exam without signs of papilledema.

Once the patient was stabilized, an MRI of the brain was performed which showed a hemorrhagic lesion in the left occipital area measuring $4.5 \times 5 \times 5$ cm with moderate peri-lesional edema, a small midline deviation, tentorial herniation and mesencephalic distortion (Figure 1). Neurosurgery was consulted and but surgical option was not indicated at that time.

Over time his neurological status improved, and he was able to maintain a normal blood pressure, which allowed discontinuing the antihypertensive treatment. MRI was performed 10 days later showing a reduction of the lesion size and reabsorption of the blood.

Discussion

AD may occur in persons with SCI above T6, although it has been described in patients with lesions as low as T10. Noxious stimuli below the level of the lesion may activate autonomic reflexes. The sympathetic inhibitory impulses originated above T6 are blocked due to the injury. This mechanism may produce a clinical picture, which most commonly includes pounding headache accompanied by an increase in blood pressure of as little as 20–40 mmHg above baseline or significantly greater. Other symptoms may include sweating, goose flesh, flushing of the skin above the SCI level, blurred vision, appearance of spots in the patient's visual fields, nasal congestion, feelings of apprehension or anxiety or cardiac arrhythmias. Occasionally a patient will have an elevated blood pressure without any symptoms. The severity and constellation of symptoms may vary.[1]

Any stimuli below the level of the lesion may be the triggering factor, the most frequent being urological causes including bladder distension, UTI, bladder or kidney stones, urologic procedures, detrusor sphincter dysinergia, epididymitis or scrotal compression.[1,2]

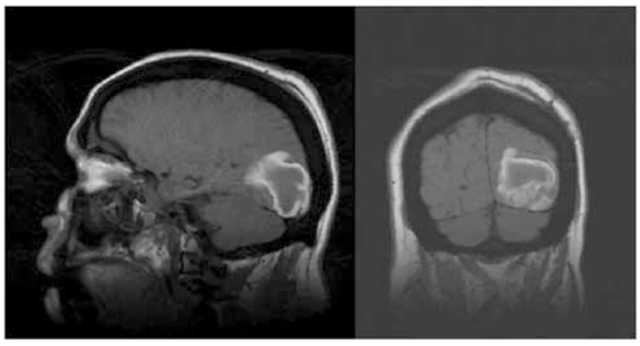

Figure 1 MRI of the brain with hemorrhagic lesion in the left occipital area

After an episode of AD has resolved and once the originating factor has been corrected, the patient may have a predisposition to experience a new episode for about 7–10 days. Some authors have called it status dysreflexia,[3–5] which indicates an easily excitable autonomic nervous system. In our case, the patient maintained difficult to control high blood pressure for a number of days even after appropriate treatment.

Elevated blood pressure during AD can lead to end-organ damage, as in this case. Other organs in addition to the CNS that can be affected are the retina (hypertensive retinopathy), heart (arrhythmia, cardiac failure, pulmonary edema) and kidney (hematuria). If the CNS is affected the patient may experience hypertensive encephalopathy, and even cerebral hemorrhage.

Hypertensive encephalopathy is an acute situation with severe hypertension and neurological signs of diffuse brain injury (headache, nausea, vomiting, seizure, confusion, somnolence and coma). It also may present with focal neurological signs, although this is less frequent. Lowering the blood pressure will usually result in resolution of symptoms in one or two days, but if the blood pressure remains uncontrolled the results may be fatal. In our case, the previous episode of hypertensive encephalopathy resolved without consequences for the patient.

The pathophysiology of this syndrome is an increase of the blood pressure above the levels of auto regulation of the brain blood flow, producing areas of vasodilatation, with increase in blood flow and capillary permeability, causing edema and in some rare occasion areas with localized ischemia, micro infarcts and/or petechial hemorrhages. The brain areas more commonly affected are the occipito-parietal lobes,[6] probably due to the heterogeneity of the sympathetic innervation of the brain.[7] The pathological exams in patients who died from hypertensive encephalopathy showed diffuse cerebral edema.[8] Hypertensive encephalopathy may led to cerebral hemorrhage in rare cases, especially if there are predisposing factors like thrombocytopenia.[6]

Cases of cerebral hemorrhage are probably due to the fast increase in hydrostatics forces that overcome the auto regulation mechanisms of cerebral flow, producing a rupture of the blood vessels located in areas with previous pathologic changes. The mortality rate of the cerebral hemorrhage is high, being around 43%. The treatment is based in basic measures of vital support, bed-rest and treatment of the secondary complications (seizures, cerebral edema). The surgical option in cases of lobe hematoma is reserved for selected patients (Glasgow coma scale 6–12, evolution less than 24 h, less than 70 years old and hematoma diameter 3–5 cm).[9] In the case of our patient he did not meet criteria for surgery because the hematoma evolution was more than 24 h.

In SCI patients, episodes of AD with an increase in blood pressure are not uncommon, and the literature includes cases of hypertensive encephalopathy related to AD. Yarkoni *et al*,[10] describe three cases of seizures

secondary to AD that are compatible with hypertensive encephalopathy. They also found 10 cases in the literature related to this pathology. We have found five references of cerebral hemorrhage,[11–14] with two of them resulting in death.[12,14]

Conclusion

This is an unusual case of cerebral hemorrhage due to AD in an SCI patient, showing the seriousness of this syndrome. A correct diagnosis and adequate treatment may stop the progression of the AD and avoid its life threatening complications.

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