

Bilateral abducens nerve palsies and urinary retention caused by the rupture of a vertebral artery aneurysm

A case report and literature review

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Abstract

Rationale: Bilateral abducens nerve deficits caused by vertebral artery (VA) aneurysm have been reported, but there have been no reports regarding accompanying urinary retention.

Patient concerns: In this report, we describe an unusual case with bilateral abducens nerve palsy and urinary retention due to rupture of a vertebral aneurysm.

Diagnoses: Subarachnoid hemorrhage caused by the rupture of a left VA aneurysm.

Interventions: The VA aneurysm was successfully controlled by coil embolization.

Outcomes: Urinary retention was improved after embolization and recovered by the day the patient left hospital. The bilateral abducens nerve palsy gradually recovered 6 months later.

Lessons: This case emphasizes the importance of maintaining a broad, open mind in approaching the diagnosis and management of urinary retention associated with cranial nerve symptoms and reacting quickly to the clinical developments.

Abbreviations: CSF = cerebral spinal fluid, CT = computed tomography, ICP = intracranial pressure, VA = vertebral artery.

Keywords: abducens nerve palsy, urinary retention, vertebral artery aneurysm

1. Introduction

Bilateral involvement of the abducens nerve as a direct or indirect result of intracranial aneurysm is not well known, as most cases are due to compression of the third to sixth cranial nerves of the cavernous sinus.^[1] Moreover, bilateral abducens nerve deficits accompanied with urinary retention are especially rare. Dailey et al^[2] reported on 65 patients with ocular signs due to verified aneurysms, but no patients presented with isolated abducens nerve palsy. Indeed, little is known about the pathophysiology or prognosis of abducens nerve palsy and/or urinary retention after rupture of an intracranial aneurysm.^[3]

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The abducens nerve originates from the pontomedullary junction. It crosses between the anterior inferior cerebellar artery and the labyrinthine artery, before passing through the prepontine cistern, Dorello's canal, cavernous sinus, superior orbital fissure, and the orbit. Due to its extremely long extracerebral intracranial course, the abducens nerve is the most frequently involved cranial nerve in many disorders.^[4] Approximately 10% of patients with sixth nerve paresis show bilateral involvement.^[5] However, bilateral abducens nerve palsy and urinary retention associated with intracranial aneurysm has rarely been reported. Here, we report a case exhibiting bilateral sixth nerve palsy and urinary retention due to rupture of a dissecting vertebral artery (VA) aneurysm, and also discuss the possible mechanism.

2. Case presentation

A 66-year-old male patient had suffered from a slight headache 1 day before admission to our emergency department. He also complained of double vision and urinary retention, and was transferred to the neurology department. He currently smoked (20 cigarettes/day for 35 years) and drank alcohol (ethanol 30 g/day for 20 years), but had no history of medication. Physical examination showed blood pressure of 144/88 mm Hg and body temperature of 38°C. He was alert and followed some commands with slight nuchal rigidity and bilateral abducens nerve palsy (Fig. 1A). He also had marked urinary retention but without lower abdominal pain. Urinary catheterization revealed a residual volume of 600 mL.

No abnormality was found in routine laboratory studies. Chest x-ray, electrocardiography, and carotid ultrasonography were

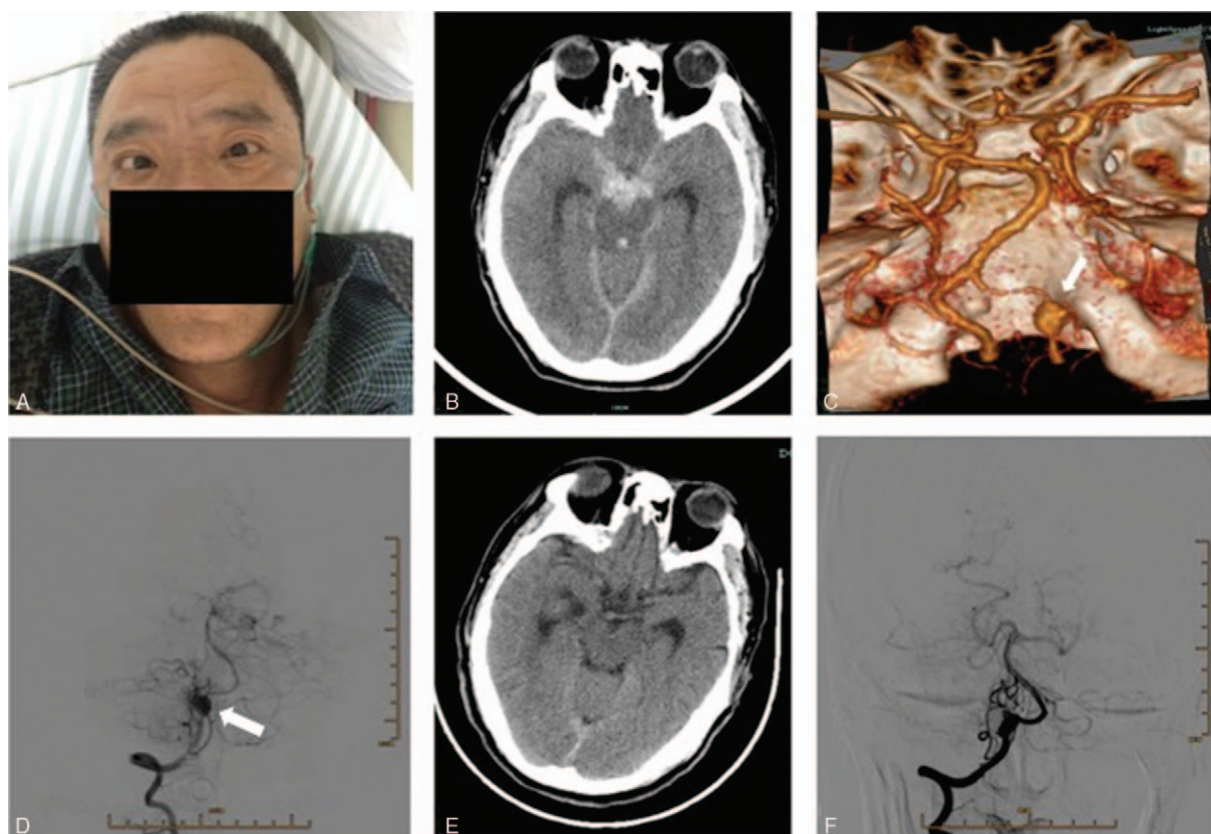


Figure 1. (A) Initial photograph disclosing the bilateral sixth nerves palsy. (B) Emergent CT scan demonstrating thick cisternal hemorrhage, in particular preoptine cistern without ventriculomegaly. (C) Emergent CT angiography demonstrating the aneurysm on the left VA (white arrow). (D) Left vertebral angiography showing a dissecting aneurysm of the VA (white arrow). (E) Resolution of hemorrhage is noted in the preoptine cistern. (F) Postdeployment and coil embolization, DSA revealing complete occlusion of the left vertebral aneurysm. CT=computed tomography, VA=vertebral artery.

unremarkable. To exclude intracranial infection, a lumbar puncture was performed on the same day and revealed a bloody cerebral spinal fluid (CSF) sample with normal intracranial pressure (ICP), indicative of a subarachnoid hemorrhage. Emergent computed tomography (CT) confirmed a subarachnoid hemorrhage filling the anterior supratentorial, preoptine, and perimesencephalic cisterns (Fig. 1B). Emergency CT angiography demonstrated a dissecting aneurysm on the left VA (Fig. 1C). The patient received a digital subtraction angiography the next day and the aneurysm was embolized with a coil (Fig. 1D). There was no evidence of thrombosis or distal artery cutoff. The patient was transferred to the intensive care unit, stable, and intubated. He was extubated the following day and by postoperative day 3 was doing well: neurologically alert, verbal without aphasia, and following commands. Four weeks later, another angiography found that compared with previous images and CT scans, the vertebrobasilar artery showed hemorrhage resolution in the preoptine cistern (Fig. 1E). And his urinary retention improved after temporary cystostomy and had completely recovered by the day he left hospital. His bilateral abducens nerve palsy gradually recovered and 6 months later he had normal ocular movements. Meanwhile, follow-up angiography found complete occlusion of the vertebral aneurysm (Fig. 1F).

3. Discussion

To our knowledge, there has been no previous reported case of aneurysm with bilateral abducens nerve palsy and urinary

retention. The abducens nerve is very sensitive to any kind of brain impairment, probably because of its extremely long extracerebral intracranial course. Lesions causing abducens nerve palsy may be located in the brain stem, subarachnoid space, petroclival region, cavernous sinus, or in the orbit along the course of the nerve. Thus, differential diagnosis of abducens paresis is broad, with several underlying diseases known, although its etiology remains unclear in more than two-thirds of cases.^[6] Isolated or combined abducens nerve palsy may be seen with aneurysms of different arteries, including the infraclinoid internal carotid, intracavernous carotid, anterior communicating, basilar, superior cerebellar, vertebral, and posterior inferior cerebellar arteries.^[7] Abducens nerve palsy caused by VA aneurysm has rarely been reported.^[8,9] Patients usually present with gaze palsy that is bilateral or ipsilateral to the pathological lesion. Possible mechanisms of isolated nerve palsy related to VA aneurysm can be divided into several groups: direct compression by the aneurysm, brain stem, or nerve compression due to cisternal hematoma (especially the preoptine cistern),^[10] stretch nerve injury on the petrous apex triggered by increased ICP,^[11] and vascular insufficiency due to vasospasm of abducens nuclei.^[10]

For our case, 2 possible mechanisms were considered, direct compression of the cisternal hematoma and increased ICP. Intracranial CT scans showed dense hemorrhage in the preoptine cistern, which, considering the preoptine segment of the abducens nerve, suggested the possibility that nerve paresis was caused by direct compression of the cisternal hematoma.

Morioka et al^[9] also reported 2 cases of bilateral isolated sixth nerve palsy compressed by cisternal hematoma. Abducens nerves are anchored in Dorello's canal, making it prone to pressure and stretching. Increased ICP resulting from an aneurysm rupture was reported to be related to bilaterally isolated abducens palsy.^[12] However, in the current patient no prominent brain swelling or acute hydrocephalus was observed on brain CT, and CSF showed normal ICP. Thus, increased ICP resulting from aneurysmal subarachnoid hemorrhage could be excluded as a candidate etiology in this patient. Regarding the vasospasm, signs of neurological deficits associated with facial nuclei or the medial longitudinal fasciculus were not noted, so it was not considered a candidate etiology.

Acute urinary retention is a common presentation in the emergency setting. Irrespective of etiology, initial management usually involves bladder decompression, typically by catheterization. Prompt identification of the underlying cause is essential in directing further management. In our case, urinary retention may have been due to sacral nerve compression caused by a blood clot that accumulated in the subarachnoid space.

Prospective data from the International Subarachnoid Aneurysm Trial suggest that patients with endovascularly treated subarachnoid hemorrhage have better outcomes than those managed surgically.^[13] The complicated cerebrovascular anatomy presents technical challenges and requires more innovative approaches to treatment strategies. VA aneurysms presenting with sixth nerve palsy are always treated by clip placement. Opening of the Liliequist membrane and cisternal irrigation of the hematoma may be effective for clinical improvement.^[8,9] In this case, although the patient did not receive surgical clipping and hematoma cleaning, the coil embolism was successful and he gradually recovered after several weeks of treatment.

4. Conclusion

We report a patient who exhibited bilateral abducens nerve paralysis and urinary retention caused by a ruptured vertebral aneurysm. We suspect that the bilateral sixth nerve palsy and urinary retention were due to direct compression of the blood clot on the related nerves. This case emphasizes the importance of maintaining a broad, open mind in approaching the diagnosis

and management of urinary retention associated with cranial nerve symptoms and reacting quickly to clinical developments.

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