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CASE REPORT
Korean J Neurotrauma 2012;8:146-148 ISSN 2234-8999

Unilateral Abducens Nerve Palsy Associated with Ruptured Anterior Communicating Artery Aneurysm

Yeon Joon Kim, MD, Cheol Wan Park, MD, PhD, Chan Jong Yoo, MD,
Eun Young Kim, MD, Jae Myoung Kim, MD and Woo-Kyung Kim, MD

Department of Neurosurgery, Gil Hospital, Gachon University of Medicine and Science, Incheon, Korea

Isolated unilateral abducens nerve palsies associated with spontaneous subarachnoid hemorrhage have rarely been reported, and their association with anterior communicating artery is even rarer. We report two cases of unilateral abducens nerve palsies following rupture of anterior communicating artery aneurysms. The aneurysms were successfully clipped, and abducens nerve palsies were gradually recovered.

(Korean J Neurotrauma 2012;8:146-148)

KEY WORDS: Abducens nerve palsy · Anterior communicating artery aneurysm · Subarachnoid hemorrhage.

Introduction

It is well known that abducens nerve paresis is caused by several neurosurgical conditions such as aneurysms in the cavernous sinus, chronic intracranial hypertension, certain intracranial masses and some traumatic brain lesions. It has been reported that subarachnoid hemorrhage (SAH) also causes abducens nerve palsies. Isolated abducens nerve palsies associated with intracranial aneurysms have rarely been reported. Their association with anterior communicating artery (ACoA) aneurysm is even rarer. We present here interesting cases in which isolated unilateral abducens nerve palsy was one of symptoms for ruptured aneurysm of the ACoA.

Case Report

Case 1

A 45-year-old woman with sudden severe headache and drowsy mentality (Hunt and Hess grade 3) was referred to our emergency department. On admission her Glasgow Coma Scale (GCS) score was 13. She did not show any focal neurologic deficit on neurologic examination. She had a history of systemic lupus erythematosus, otherwise her medical history was unremarkable. The routine hematologic and serum biochemistry tests were all within normal limit. Initial head computed tomographic (CT) scan revealed Fisher's grade 3 SAH (Figure 1A). And a cerebral CT angiography revealed ACoA aneurysm and right middle cerebral artery bifurcation (MCAb) aneurysm (Figure 1B). Neurosurgical clipping for aneurysms of ACoA and MCAb aneurysms was performed via a right pterional approach on the day of admission. In the operation, it was confirmed that ACoA aneurysm was ruptured one and MCAb aneurysm was unruptured. She complained of diplopia when she gazed to the left side from the postoperative 3rd day. There was no evidence of brain stem lesion on magnetic resonance images on the day 30 (Figure 1C). On the postoperative transfemoral cerebral angiogram which was performed to confirm the improvement of vasospasm on the day 36, there was no significant vasospasm (Figure 1D). Left abducens nerve palsy gradually improved during out-patient follow-up. She had normal ocular movements after 3 months.

Case 2

A 52-year-old woman visited emergency department with the chief complaint of sudden burst headache. On ad-

Received: May 7, 2012 / **Revised:** May 31, 2012
Accepted: June 5, 2012
Address for correspondence: Cheol Wan Park, MD, PhD
Department of Neurosurgery, Gil Hospital, Gachon University of Medicine and Science, 1198 Guwol-dong, Namdong-gu, Incheon 405-760, Korea
Tel: +82-32-460-3304, Fax: +82-32-460-3899
E-mail: cwpark@gilhospital.com

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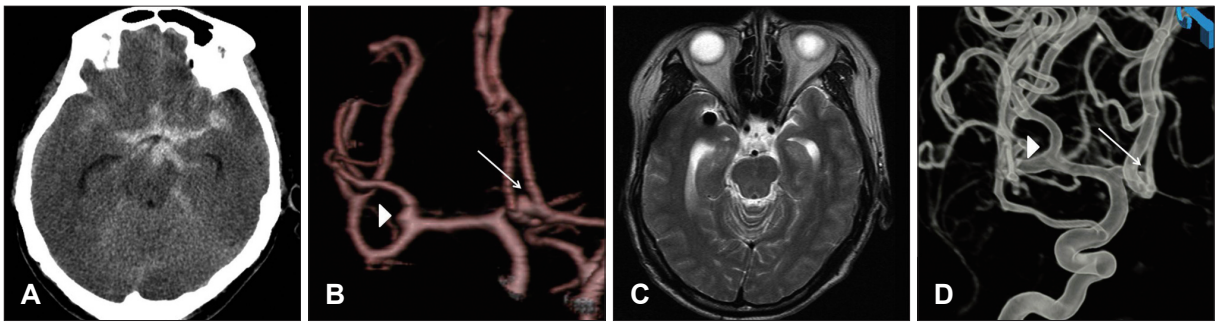

FIGURE 1. Radiological findings of case 1. A: Head computed tomography (CT) showing subarachnoid hemorrhage of Fisher's grade 3. B: Cerebral CT angiography. Arrows indicate anterior communicating artery and arrow head middle cerebral artery aneurysms, respectively. C: Brain magnetic resonance image demonstrating no definitive abnormal lesion in the brain stem. D: Postoperative cerebral angiography indicating complete disappearance of aneurysms and no vascular abnormality.

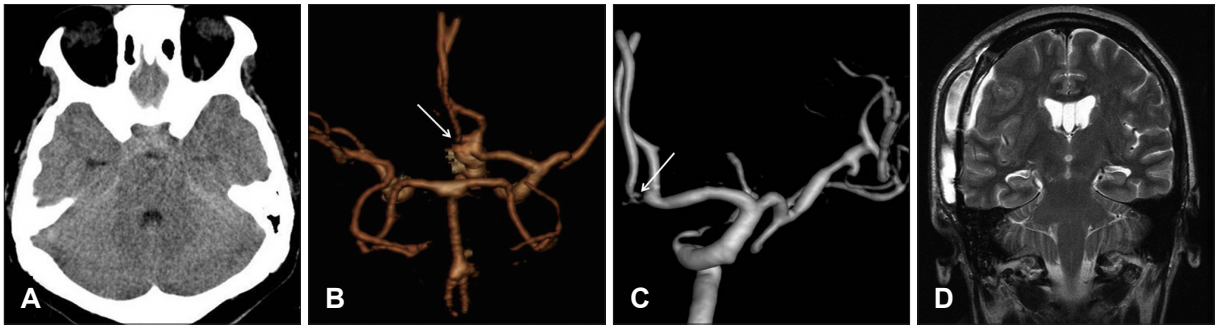

FIGURE 2. Radiological findings of case 2. A: Head computed tomography (CT) showing subarachnoid hemorrhage of Fisher's grade 2. B: Cerebral CT angiography. Arrow indicates anterior communicating artery aneurysm. C: Postoperative cerebral angiography indicating no residual aneurysmal sac or vascular abnormality. D: Brain magnetic resonance image demonstrating a small amount of fluid collection over the right frontotemporal convexity with unremarkable brain stem area.

mission her neurologic examination resulted in Hunt and Hess grade 2. There was no focal neurologic deficit. She had a history of essential hypertension with antihypertensive medications. Her head CT scan revealed SAH of Fisher's grade 2 (Figure 2A). A CT angiography showed ruptured ACoA aneurysm (Figure 2B). Neurosurgical clipping was performed via a right pterional approach on the same day of admission.

On the day 5, right abducens nerve palsy was apparent on neurologic examination. Postoperative transfemoral cerebral angiogram was performed to confirm the improvement of vasospasm on the day 14, and there was no definitive evidence of vasospasm (Figure 2C). Magnetic resonance imaging on the day 18 showed fluid collection in right frontotemporal convexity, and was otherwise unremarkable (Figure 2D). Abducens nerve palsy gradually improved during clinic follow-up and was fully recovered at 4 months after SAH.

Discussion

The known frequency of abducens nerve palsies associated with intracranial aneurysms is about 3%.[2,4] Isolated or combined palsies of the abducens nerve with other cranial nerves has been reported in various aneurysm locations including infraclinoid internal carotid, intracavernous carotid, anterior communicating, basilar, superior cerebellar, vertebral and posterior inferior cerebellar arteries.[2,4,5,9] Although the most common cause of acquired, isolated sixth cranial nerve palsy is vascular disorders,[1] a ruptured aneurysm is distinctly uncommon as a cause of acquired, isolated sixth abducens nerve paresis, and this accounted for only 3.6% and 6.0% of all the sixth cranial nerve palsies.[1,7]

Several mechanisms have been suggested to explain the involvement of the abducens nerve after an aneurysm rupture such as: direct compression of sixth cranial nerve by the aneurysm, elevated intracranial pressure, vasospasm of the pontine branches of the basilar artery affecting the abducens nuclei and direct compression of sixth cranial nerve by the dense clot in the prepontine cistern, especially.[9]

In our patients, location of the aneurysm indicates that the palsies could not have been caused from direct pressure by the aneurysm on the abducens nerve because ACoA is located far away from the abducens nuclei or course of abducens nerves.

We postulate several potential etiologies for abducens

palsies that alone or in combination may explain our patients' abducens nerve palsies. The first potential pathogenesis is compression of nerve due to increased intracranial pressure (IICP). Dysfunction of all the cranial nerves has been reported in association with IICP in the presence or absence of herniation.[6] The abducens nerve has a long intracranial course at the base of the brain, and is consequently vulnerable to injury including IICP, especially where it crosses the petrous apex and traverses the sharp edge of the petrous temporal bone.[5] In our patients, however, clinically estimated ICP seemed to be not that high to precipitate abducens nerve palsy. The second plausible mechanism is believed to be direct injury of the abducens nerve by aneurysmal rupture. It probably results from the filling of basal cisterns with blood and/or the direct shock to the brainstem caused by aneurysmal rupture, followed by stretching of the structures lying between the brain and the skull in the subarachnoid space. Rupture of an ACoA aneurysm is well known to cause marked filling of the basal cisterns with blood, sometimes expanding into the posterior fossa, particularly when the aneurysm is directed inferiorly.[5] As other possible mechanism of abducens palsies, cranial nerve palsy may result from the increased osmotic tonicity of the surrounding blood, the deleterious effects of the concentrated blood breakdown products or ischemia by the compression of small nutrient arterioles of the nerve.[3] For our patients, dense blood clot in prepontine cistern, which was revealed on the initial CT scans, was large and thick enough to warrant consideration of this mechanism as the cause of sixth cranial nerve palsies. Finally, the unilaterality of abducens nerve palsy in both of our patients could be explained by the laterality of the approach. We approached both of our cases through the right pterional craniotomy. And we speculated that the blood clots within the right basal cisterns were easier to remove than those of the left side basal cisterns, intraoperatively. Thus, left abducens nerve might have a chance to be exposed to much concentrated blood breakdown products, postoperatively.

Vasospasm of the pontine branches of the basilar artery supplying the pontine abducens nuclei is also a possible cause of abducens nerve palsy. Suzuki and Iwabuchi[8] speculated that the abducens nerve palsies associated with aneurysmal SAH was caused by vasospasm of the pontine branches of the basilar artery, rather than mechanical pressure on the nerve. However, angiographic evidence of vasospasm had not been demonstrated in our patients. If vasospasm of the pontine branches of the basilar artery affected the abducens nerve nuclei, gaze paresis and facial paresis should coexist and be apparent because of the close anatomical relationships between abducens nuclei, the medial longitudinal fasciculi and the facial nuclei.[5] In our cases, no other accompanying signs except abducens palsies was apparent.[8] The intracisternal part of the abducens nerve is supplied by branches of the same artery that supplies the third and fourth cranial nerve. If vasospasms of these branches were responsible for abducens nerve palsies, third and fourth cranial nerve paresis should have been seen in association.

Generally, recovery of abducens nerve palsies may occur usually within 3−8 weeks, or after 3 months.[9] Our patients' symptoms were also resolved during the 3 months and 4 months of follow-up period, respectively.

Conclusion

We describe two patients who suffered SAH after the rupture of anterior communicating artery aneurysms, with the clinical course consisting of isolated unilateral abducens nerve palsy. Elevated intracranial pressure without herniation, direct nerve compression or shock by large subarachnoid blood clots alone or in combination have all been suggested as possible mechanisms for this type of palsy.

■ The authors have no financial conflicts of interest.

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