

Bilateral abducens nerve palsy following ruptured anterior communicating artery aneurysm: report of 2 cases

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Summary

Isolated abducens nerve palsies associated with intracranial aneurysms have rarely been reported. Their association with anterior communicating artery (ACoA) is even rarer. Intracisternal clot formation and elevated intracranial pressure has been proposed to be the responsible mechanisms.

Herewith, we report two cases of bilateral abducens palsies following ruptured ACoA aneurysms and speculated the possible mechanisms.

Opening of Lilliequist's membrane provides clinical improvement due to CSF release.

KEY WORDS: Abducens nerve palsy. Lilliequist's membrane. Aneurysm.

Parálisis bilateral del nervio abductor después de la ruptura de un aneurisma de la arteria comunicante anterior. Presentación de dos casos

Resumen

Rara vez se ha publicado un caso de parálisis aislada del nervio abductor craneal, asociada a un aneurisma. Su asociación con un aneurisma de la arteria comunicante anterior (ACoA) es todavía más rara. Los mecanismos propuestos son la formación de un coágulo intracisternal y la hipertensión intracraneal

Aportamos dos casos de parálisis bilateral del sexto par, después de la ruptura de aneurismas de la ACoA y discutimos los mecanismos posibles.

La apertura de la membrana de Lilliequist da lugar a una mejoría clínica, debido a la liberación de l.c.r.

PALABRAS CLAVE: Parálisis nervio abductor. Aneurisma. Membrana de Lilliequist

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Introduction

Abducens nerve originates from pontomedullary junction. It crosses between anterior inferior cerebellary artery (AICA) and labyrinthine artery and passes through the prepontine cistern, Dorello's canal, cavernous sinus, superior orbital fissure and orbita respectively. Due to its extremely long extracerebral intracranial course the abducens nerve is the most frequently involved cranial nerve in a number of disorders³. About 10% of patients with sixth nerve paresis have bilateral involvement². Isolated abducens nerve palsies associated with intracranial aneurysms have rarely been reported. Their association with anterior communicating artery (ACoA) is even rarer.

Cases

Case 1

A-51-year old woman was admitted with sudden onset of headache, vomiting and diplopia. Neurologic examination showed bilateral sixth nerve palsy (Hunt-Hess grade II). Computed tomography (CT) revealed subarachnoid hemorrhage (Fischer grade II) (Figure 1 A). ACoA aneurysm, determined with digital subtraction angiography (DSA) (Figure 1 B), was clipped successfully on the sixth day of her admission. Seven days after the operation her left side abducens palsy recovered gradually and one month later she had normal ocular movements.

Case 2

A-53-year old woman suffering from sudden headache, nausea and temporary disturbance of consciousness was admitted to our clinic. Bilateral abducens nerve palsies were determined on neurologic examination (Hunt-Hess grade II) (Figure 2 A, B). CT scan showed subarachnoid and intraparenchymal hemorrhage located in basal cisterns, anterior interhemispheric fissure and right frontal lobe (Fischer grade IV) (Figure 3 A) and cerebral angiography revealed ACoA aneurysm (Figure 3 B). Microsurgical clip-

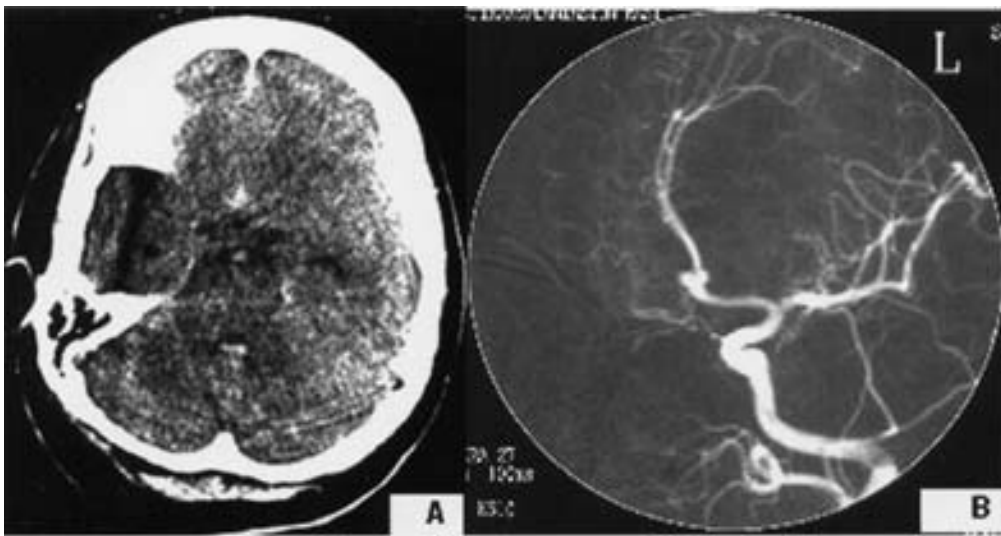


Figure 1. A: cranial CT showing subarachnoid hemorrhage Fisher Grade II. B: Cerebral angiography revealing ACoA aneurysm.

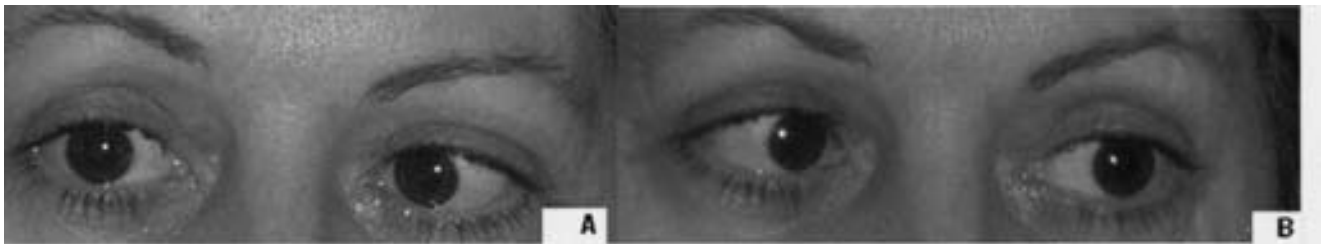


Figure 2. A, B: photograph of the patient showing bilateral abducens nerve palsy.

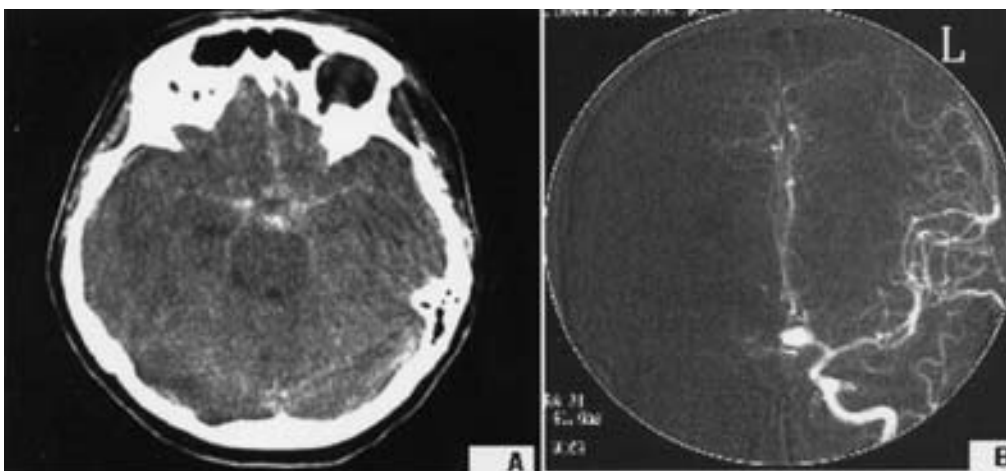


Figure 3. A: Cranial CT showing subarachnoid and intracisternal hemorrhage and frontal intracerebral hematoma (Fisher Grade IV). B: cerebral angiography revealing ACoA aneurysm.



Figure 4. A, B: photograph of patient showing normal lateral ocular movements.

ping of ACoA aneurysm was performed on the 15th day of her admission. Postoperatively bilateral sixth nerve paresis improved partially and one month later the neuroophthalmologic examination was normal (Figure 4 A, B)

In both cases, Lilliequist's membrane and prepontine cistern were opened and meticulous cisternal toilette of the subarachnoid clot was performed during the operation. In the first case high pressure cerebrospinal fluid (CSF) drainage was observed after opening the membrane.

Discussion

The frequency of abducens nerve palsies associated with intracranial aneurysms is between 3.3% and 3.6%^{1,4}. Isolated or combined palsies of the sixth nerve had been reported in various aneurysm localisations, including; infraclinoid internal carotid, intracavernous carotid, anterior communicating, basilar, superior cerebellar, vertebral and posterior inferior cerebellar arteries^{1,4,5,7}. Several mechanisms have been proposed to explain the bilateral involvement of the abducens nerve after an aneurysm rupture such as: direct compression of the aneurysm, elevated intracranial pressure, vasospasm of the pontine branches of the basilar artery affecting the abducens nuclei and direct compression of the clot on the nerve in the prepontine cistern especially⁷. Because of far way from the anterior communicating artery the mechanism of bilateral sixth nerve involvement following aneurysm rupture has been debated.

The inferior part of the carotid cistern and superior part of the interpedicular cistern are in apposition - sometimes causing a single membrane (Lilliequist's membrane, 1959), which may be thick or thin, but normally forming two separate layers.

Following subarachnoid hemorrhage, this arachnoid membrane may become thickened and create a loculation of CSF in the interpedicular and prepontine area. Opening of this membrane at operation almost always results in the escape of some CSF, even in the profuse lumbar drainage⁶.

In the literature two cases of bilateral abducens palsies following ruptured ACoA aneurysm had been reported. In the first case, reported by Nathal et al. (1992) the mechanism of the palsy was explained with the vasospasm of the pontine branches of the basilar artery. and the deficits had resolved completely three months after admission⁵. In the second case, Ziyal et al. (2003) speculated that direct compression of both abducens nerves was caused by the extensive clot in the basal cisterns and bilateral palsies fully recovered on the third postoperative day⁷.

In our first case both abducens nerves were probably affected from the intracisternal entrapment of CSF. In our second case the nerves were affected due to either the intracisternal clot formation as Ziyal et al. speculated or high intracranial pressure caused by the intracerebral hematoma

and SAH. In all mechanisms, the direct compression of the nerve is the main cause. We cannot explain the mechanism of the palsy with vasospasm of the pontine branches of the basilar artery because there was neither clinical nor radiological vasospasm.

We believe that the most important physiopathologic mechanism of the bilateral sixth nerve palsies following ACoA aneurysm rupture is the direct compression of the nerve by blood or CSF. Recovery occurs usually 3-8 weeks to literature^{1,5}.

Conclusion

In this report, we speculated that bilateral sixth nerve palsy can occur in the presence of intracisternal CSF entrapment, clot formation and elevated intracranial pressure. Opening of the Lilliequist's membrane which provides CSF release and extensive cisternal irrigation of the subarachnoid clot, seems to be an effective surgical maneuver.

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