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 Liliequist's membrane provides clinical improvement due to CSF release.

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

Introduction

Abducens nerve originates from pontomedullary junction. It crosses between anterior inferior cerebellary artery (AICA) and labyrinthine artery and passes through the pre-pontine 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 disorders. About 10% of patients with sixth nerve paresis have bilateral involvement. 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 substraction angiography (DSA) (Figure 1 B), was clipped succesfully 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 intraparanchymal hemorrhage located in basel cisterns, anterior interhemisféric fissure and right frontal lobe (Fischer grade IV) (Figure 3 A) and cerebral angiography revealed ACoA aneurysm (Figure 3 B). Microsurgical clip-
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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 nervew 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 4A, 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%. Isolated or combined palsies of the sixth nerve had been reported in various aneurysm localisations, including: infracranial internal carotid, intracavernous carotid, anterior communicating, basilar, superior cerebellar, vertebral and posterior inferior cerebellar arteries. 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 especially. 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 interpeduncular cistern are in apposition - sometimes causing a single membrane (Liliequist'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 interpeduncular and prepontine area. Opening of this membrane at operation almost always results in the escape of some CSF, even in the profuse lumbar drainage.

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 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.

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.

References


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