

Blindness Caused by Wrapping of the ICA Aneurysm

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The incidence of blindness after aneurysm surgery is very rare. We experienced a case of unilateral blindness after internal carotid artery(ICA) aneurysm wrapping. A 43-year-old male immediately developed ipsilateral ocular pain and visual loss in his left eye after the treatment of a lateral ICA aneurysm by wrapping with muscle pieces. He had also multiple aneurysms, which were multilobulated anterior communicating artery (A-com), middle cerebral artery(MCA) and posterior communicating artery (P-com) aneurysms. Coilings were done for a part of A-com artery aneurysm and P-com artery aneurysm on admission. The remaining A-com artery aneurysm was clipped and ICA aneurysm was wrapped with temporal muscle piece. A retrobulbar optic neuropathy might have resulted from either direct injury or damage to small dural vessels of the posterior optic nerve. Actually, the optico-carotid space was tight and the optic nerve was compressed by swollen muscle piece. Despite releasing of compression of the optic nerve on second day, his visual loss was irreversible.

KEY WORDS : Blindness · Wrapping · ICA aneurysm.

Introduction

Aneurysms of the internal carotid artery(ICA) proximal to the posterior communicating(P-com) artery usually arise near to the optic nerve and the origin of the ophthalmic artery. These aneurysms are relatively uncommon, accounting for approximately 5% of all intracranial aneurysms^{1,2}. Despite significant technical advances, postoperative blindness occasionally has been reported. If it happens, its etiologies may include the direct injury of optic nerve, increased intracranial pressure(ICP), compromised ophthalmic arterial or venous circulation, or impaired orbital venous outflow^{2,11,18,19}. Rizzo¹⁶ reported 3 cases of unexplained visual loss including 1 case; compromise of large draining vein and 2 cases; direct nerve injuries including damage to small dural vessels of the posterior optic nerve after clipping of paraclinoid aneurysms. We report one patient who had acute unilateral visual loss after the wrapping of ICA aneurysm.

Case Report

A forty three-year-old man had severe headache on March 2005. No focal neurologic deficits were noted. His bilateral visual acuity was 0.6. His clinical condition was Hunt-Hess

grade II. A cerebral angiography revealed multiple aneurysms, which were located on lateral side of ICA proximal to P-com artery, P-com artery portion of ICA, MCA and A-com artery. We supposed that the lateral directed aneurysmal sac of bilobulated A-com artery aneurysms was ruptured and bleeding focus.

On the first day we tried to treat them with Guglielmi Detachable Coils(GDC) embolization but coilings were limited to obliterate the lateral directed A-com aneurysm, which was ruptured, and P-com artery aneurysms. So the medial directed of A-com artery aneurysm, one broad neck aneurysm of ICA, and small MCA aneurysm inevitably were decided to surgery (Fig.1).

The ICA aneurysm was decided to wrap with temporal muscle piece. Surgicel (Ethicon, Somerville, NJ), Gelfoam (Pharmacia and Upjohn, North Peapack, NJ) and Greenplast (Green cross) also were used to provide additional support.

Ten hours later, we found visual loss with moderate dilated pupil size on the ipsilateral side of the operated site. He also complained frontal and orbital pain with mild conjunctival edema but extraocular muscle movements were preserved completely. Postoperative computerized tomography(CT) revealed diffuse swelling on brain without the evidence of intra-orbital swelling. On ophthalmoscopic examination, the patient

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had loss of vision in the left eye and the left pupil did not respond to light with consensual afferent defect. The ophthalmologist described the retrobulbar optic neuropathy with no pulsation on optic disc, relative afferent papillary deficit and dilated pupil size. About 15 hour later, the operation site was revised and optic nerve was compressed by the wrapped muscle

pieces of ICA aneurysm. After removal of them, we observed the initial tight optico-carotid space was more or less widened by swollen muscle piece that encircled ICA crossing over the optic nerve (Fig. 2). There was no significant improvement in visual acuity. Visual evoked potential(VEP) was not obtained on left side (Fig. 3). The patient had no visual improvement 10 months later.

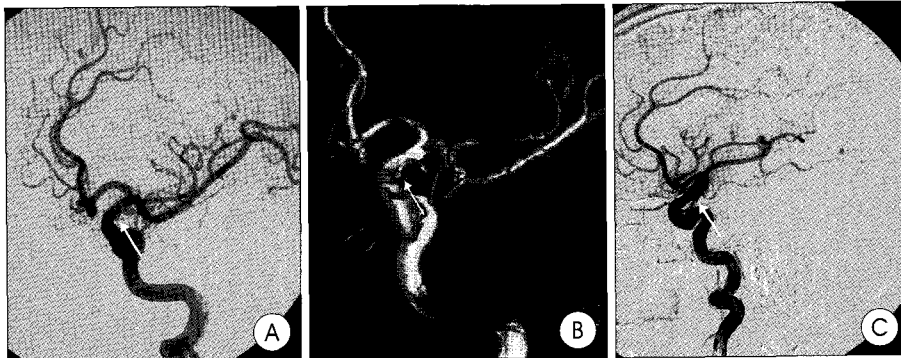


Fig. 1. A, B : Preoperative angiography shows left internal carotid artery(ICA) aneurysm (arrow). C : A part of A-com and P-com artery aneurysms were coiled but ICA aneurysm (arrow) was still remained on post coiling angiography.

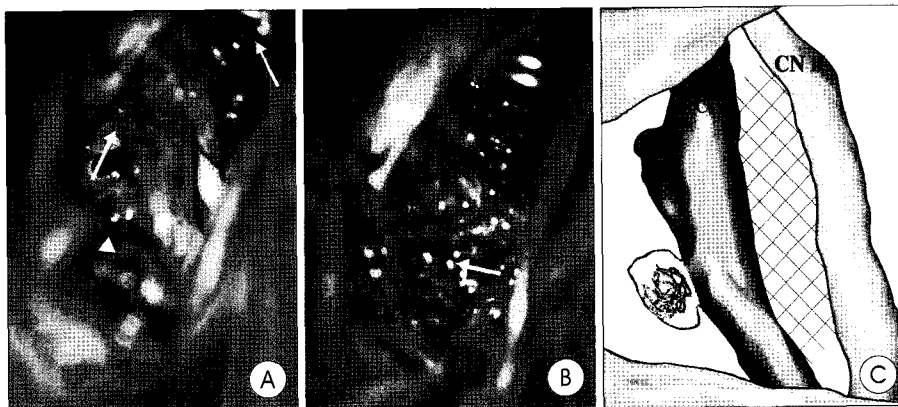


Fig. 2. A : Intraoperative photograph demonstrating the tightness of optico-carotid space due to internal carotid artery(ICA) tortuosity (yellow arrow : ICA aneurysm, white arrow head : Guglielmi detachable coils(GDC) embolization of P-com artery aneurysm, white arrow : clip of A-com artery aneurysm). B : ICA aneurysm was wrapped with temporal muscle piece (White arrow: temporal muscle piece, Red line : ICA). C : After releasing the optic nerve, intraoperative illustration shows widened optico-carotid space (CN II : Cranial Nerve II).

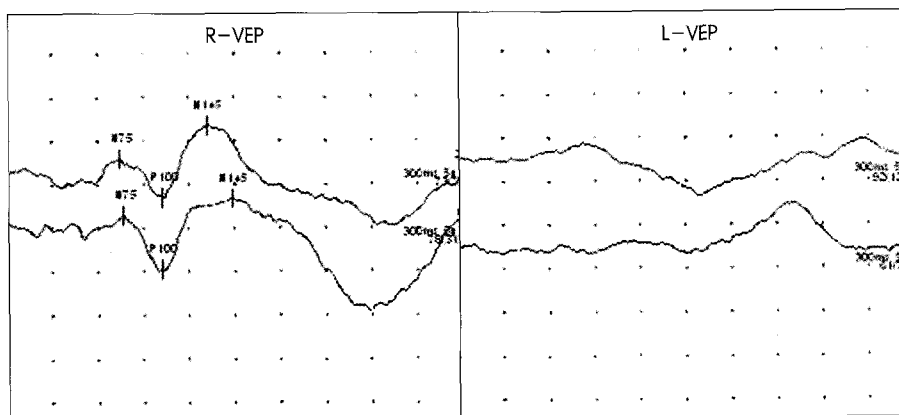


Fig. 3. Postoperative visual evoked potentials(VEP) indicated conduction defect of left VEP pathways.

Discussion

This patient illustrates a visual complication after ICA aneurysm combined with tight optico-carotid space was wrapped with muscle pieces. According to some literatures, some patients have been reported with this complication since the advent of microsurgery^{2,4,7,10,12,14}.

In case of paraclinoid aneurysms, there was posterior optic neuropathy or orbital syndrome with compromise of ophthalmic artery. Rizzo¹⁶ explained that two clinical patterns were evident in three patients. Two patients among them had a fulminant orbital syndrome with marked proptosis, periocular swelling, and compromise of retinal and choroidal perfusion. The explosive onset of orbital congestion probably could be explained occlusion of the superior ophthalmic vein, which courses near the surgical field where it would be vulnerable to inadvertent trauma, postoperative inflammation, and possibly thrombosis and secondary to packing of the anterior cavernous sinus to gain hemostasis. Another optic neuropathy developed in the third case without edema of the retina or nerve during the acute phase. This suggests that damage occurred along posterior segment of optic nerve. In the absence of systemic hypotension or marked blood loss, local factors at the perioperative site most likely suggest the neural damage. This patient who had suspicious direct neural damage had moderate visual recovery several

weeks later. He also mentioned other etiologies for visual loss including vasospasm, ischemia secondary to either mechanical or inflammatory damage to the vasa nervosa, and sometimes direct neural trauma in a blind technique. Actually, his second cases had postoperative spasm around ophthalmic artery.

Guidetti and La Torre⁹⁾ attributed carotid-ophthalmic aneurysm cases to inadvertent closure of the ophthalmic artery. Indirect surgery (common carotid artery ligation) did not prevent the aneurysm from growing or restore useful vision. On the other hand, a series of 16 cases treated by direct approach with ligation of the aneurysmal neck or local trapping, where necessary, followed by removal of the aneurysm resulted in marked improvement of impaired vision. Almeida et al.¹⁾ described that the causes of visual loss could be the excessive manipulation of the optic nerve, injury of parachiasmatic vessels and a thrombosis of the ophthalmic artery. Fox⁸⁾ suspected retinal ischemia associated with temporary occlusion of proximal arteries (i.e., "trapping" procedure).

Generally, most of neurosurgeon described that visual impairment by simple compression to the optic nerve had relatively good prognosis but microvascular insults related to the optic nerve occurred compound and persistent visual loss⁶⁾.

According to some literatures visual loss also has been ascribed to the use of muslin gauze wrapping although these patients differed from the delay in onset of symptoms up to several months, sometimes over 1 year postoperatively. Muslin-induced optic arachnoiditis ('Gauzoma'), optochiasmatic arachnoiditis and muslin induced granuloma had been reported^{2,4,7,12)}.

These impacts pressed us to use temporal muscle piece to wrap the aneurysm of ICA. The optic nerve might be directly injured by tough wrapping with muscle piece. We supposed that it seemed to be happened microvascular insufficiency of posterior optic nerve and compression of the volume of muscle because the initial tight optico-carotid space was exceedingly displaced. The disappearance of the optic disc pulsation on fundoscope also might support ophthalmic artery insufficiency but it made us not define the blockage of ophthalmic artery because we did not have the postoperative angiography. In case of acute obstruction of the retinal and choroidal circulations, ophthalmoscopic features included (1) intense opacification of the retina in the posterior pole, (2) variable absence of a cherry red spot, and the subsequent development of (3) optic atrophy and (4) pigment epithelial changes. Fluorescein angiographic signs encompassed (1) delayed retinal and choroidal filling, as well as (2) leakage at the level of the retinal pigment epithelium. Electroretinography disclosed absence of both the a- and b-waves³⁾. Unfortunately, although we did not have any ophthalmologic study except fundoscope because the second operation had done urgently, fundoscopic finding of our case was completely not consistent to acute obstruction

of ophthalmic arterial circulation. Resultingly, we think that visual loss of our case might be caused by direct compression and microvascular insufficiency of the retrobulbar optic nerve.

Conclusion

We suggest that the extent of optico-carotid space should be considered at the time of wrapping of the ICA aneurysm because optic nerve particularly seems to be vulnerable to direct damage and microvascular insufficiency, especially in case of ICA aneurysms.

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