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Acute Angle-Closure Glaucoma After Successful Embolization of Traumatic Carotid-Cavernous Sinus Fistula

—Case Report—

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Abstract

A 69-year-old woman developed acute angle-closure glaucoma 2 weeks after successful transvenous coil embolization of a traumatic carotid-cavernous sinus fistula. The angle-closure glaucoma was precipitated by oculomotor palsy caused by transvenous coil packing of the cavernous sinus. Emergency iridotomy resulted in normalization of the intraocular pressure and restoration of vision. Acute angle-closure glaucoma may develop in association with an oculomotor palsy caused by any etiology, including neurointervention.

Key words: carotid-cavernous sinus fistula, embolization, glaucoma, oculomotor palsy

Introduction

Aqueous humor of the eye is produced by the ciliary body epithelia and drained in the anterior chamber angle through the trabecular meshwork. A narrow angle causes the iris to approach close to the trabecular meshwork. Angle-closure glaucoma is caused by the disturbance of outflow due to the obstructed angle. Acute angle-closure glaucoma occurs frequently in darkness, which causes the pupil to dilate, thus further narrowing the angle. Oculomotor palsy due to any etiology will also cause pupil dilation, and may induce angle-closure glaucoma.[9,12] We treated a patient with traumatic carotid-cavernous sinus fistula (CCF) by transvenous coil embolization, but the patient developed acute angle-closure glaucoma 2 weeks later.

Case Presentation

A 69-year-old woman suffered severe craniofacial trauma caused by a traffic accident. The patient was transferred immediately to our department. On admission, she was drowsy with a Glasgow Coma Scale score of 10 (E2, V2, M6). Her pupils were isocoric and reactive to light. No apparent motor weakness was observed. Computed tomography of the brain showed no abnormality. Since nasal packing could not control profuse nasal bleeding, leading to hypovolemic preshock, endovascular treatment was indicated. Digital subtraction angiography (DSA) showed multiple extravasations of contrast material from the bilateral distal internal maxillary arteries as well as the left middle meningeal artery, but no extravasation from the bilateral internal carotid arteries. Arteriovenous fistulas were located in the bilateral internal carotid arteries (C_4–C_5 portions), larger on the left than on the right (Fig. 1A). Transarterial coil embolization with polyvinyl alcohol particles and platinum coils controlled the extravasation and resulted in rapid stabilization of the systemic blood pressure. Postoperatively, extreme facial swelling was observed.

The patient gradually became conscious. Right peripheral mild facial palsy and right abducens palsy became apparent. Follow-up DSA at 1 month showed that the right CCF had spontaneously disappeared, but the left CCF still persisted. About 6 weeks after the initial trauma, her corrected visual acuity was 20/25 on both sides and the intraocular pressures (IOPs) were 7 mmHg on the right and 9 mmHg on the left. Two months after the trauma, the abducens palsy disappeared and the facial palsy

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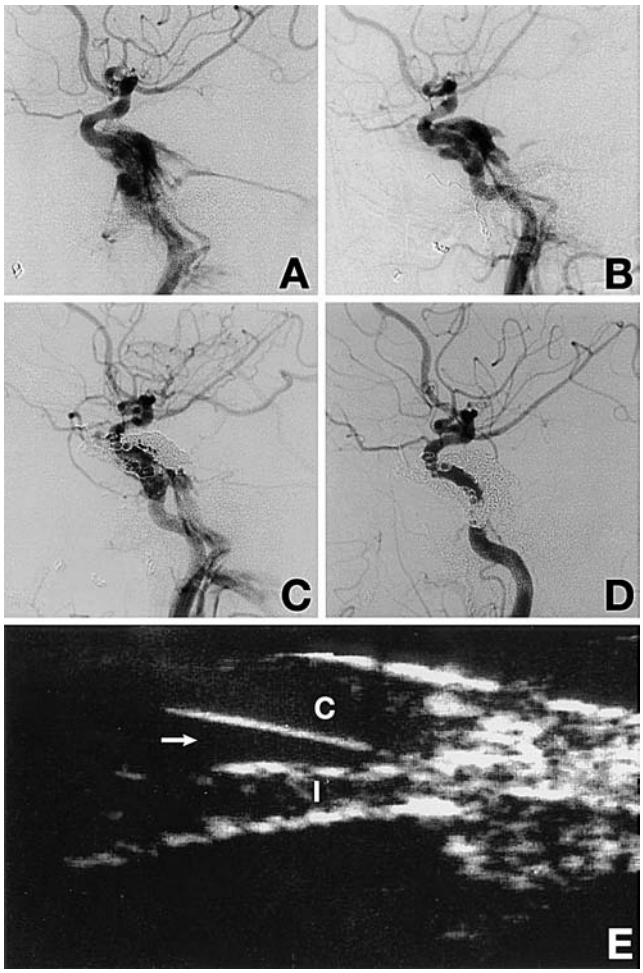

Fig. 1 **A: Left internal carotid angiogram, lateral view, on admission showing an arteriovenous fistula (AVF) at the posterior cavernous sinus. B: Left internal carotid angiogram, lateral view, 2 months after trauma showing a persistent AVF at the cavernous sinus. C: Left internal carotid angiogram, lateral view, showing partial occlusion of the carotid-cavernous sinus fistula (CCF) after the initial transvenous embolization from the contralateral inferior petrosal sinus. Oculomotor palsy developed after this coil embolization. D: Left common carotid angiogram, lateral view, showing the absence of any arteriovenous shunt after complete occlusion of the CCF with coils at the second embolization. E: Orbital echogram showing a narrow angle (*arrow*) of the left eye. The intraocular pressure was 49 mmHg. C and I indicate cornea and iris, respectively.**

had become minimal. Treatment of the left CCF was scheduled (Fig. 1B).

Since we thought that the intracavernous internal carotid artery had a small laceration, which would not accept a deflated detachable balloon, transfemoral transvenous embolization was performed under local anesthesia. The guiding catheter did not enter the left internal jugular vein, so we navigated the microcatheter from the right inferior petrosal vein to the left cavernous sinus crossing the basilar plexus. During the coil embolization lasting 3 hours, the patient complained of intractable fatigue. Thus, embolization was immediately terminated. The CCF was partially occluded with platinum coils (total length 269 cm). After this embolization, this patient developed left oculomotor palsy manifesting as ptosis, pupil dilation, sluggish light reflex, and limited extraocular motilities of 2/5 except for abduction, which began to improve gradually in several days. Six days after the first intervention, second embolization was performed under general anesthesia (Fig. 1C). This time, the microcatheter could be navigated through the left internal jugular vein and left inferior petrosal sinus to the left cavernous sinus. Coil packing of the cavernous sinus required additional platinum coils (length 152 cm), and resulted in complete disappearance of the CCF (Fig. 1D).

Two weeks after the second intervention, the patient complained of left retro-orbital pain at night, which was controlled with analgesics. The patient did not complain of visual disturbance due to left ptosis at that time. Next morning, left orbital pain increased and visual loss (light perception) was discovered when the ptotic upper eyelid was elevated. The cornea was cloudy, and the pupil was more dilated than before and not reactive to light. Emergency ophthalmologic examination revealed a shallow angle and elevated IOP to 49 mmHg on the left compared to 10 mmHg on the right. The diagnosis was acute angle-closure glaucoma (Fig. 1E). There was no choroidal effusion. Intravenous administration of acetazolamide and glycerin resulted in reduction of the IOP on the left to 16 mmHg. Laser iridotomy was then performed under topical anesthesia on the same day, which further reduced the IOP to 8 mmHg. Within several days, the patient's left vision slowly improved and the IOP remained below 10 mmHg. She was discharged 6 days after the iridotomy. Prophylactic laser iridotomy was performed on the right unaffected eye 19 days later. At the last follow up, 3 months after the glaucoma attack, her corrected visual acuity was 20/15 on the right and 20/20 on the left, and the IOP was 16 mmHg on the right and 12 mmHg on the left. Motility of both eyes was full in all directions.

Discussion

The episcleral veins drain the aqueous humor from the anterior chamber via Schlemm's canal. Normal IOP is maintained by the pressure gradient between the anterior chamber and episcleral veins. IOPs are usually elevated in patients with CCFs due to the elevated episcleral venous pressure from the CCF, which decreases the outflow of aqueous humor.[6–8,11,14] Although the IOP is difficult to control without closure of the CCF, the IOP usually decreases to within the normal range after treatment of the CCF.

Angle-closure glaucoma is commonly observed in Orientals, hyperopes, and elderly women. This type of glaucoma occurs in patients with a narrow angle. The symptoms include severe headache, visual decline, orbital pain, nausea, vomiting, and halos or rainbows around objects. The affected eye becomes red, and the cornea swells and clouds. Red eye caused by conjunctival venous congestion could be misunderstood as a symptom of CCF. Acute angle-closure glaucoma is reliably identified by a narrow angle as measured by gonioscopy. Tonometry is used to measure the elevated IOP, which occasionally reaches 100 mmHg.

Choroidal detachment may develop in patients with CCFs because of choroidal transudation caused by the venous stasis in the vortex veins.[3,4,7,8,10,12] Choroidal detachment and/or elevated venous pressure due to CCF may cause angle-closure glaucoma because the lens and iris are rotated anteriorly, occluding the trabecular meshwork.[2,4–6,16]

Oculomotor palsy may precipitate a decrease in the outflow of aqueous humor, resulting in glaucoma in patients with narrow angles.[13,17] Oculomotor palsy is caused by various etiologies, such as posterior communicating artery aneurysm, diabetic neuropathy, skull base tumor involving the cavernous sinus, CCFs, brain stem infarction, and hemorrhage.[11,18] Oculomotor palsy also occurs pharmacologically and iatrogenically during neurosurgical and/or neurointerventional procedures. Mid-dilation of the pupil most likely precipitates angle-closure glaucoma because the outflow of aqueous humor is blocked maximally.[13] The delayed manifestation of glaucoma in our patient could be explained by the mid-dilation of the pupil during the course of improving oculomotor palsy. A patient with oculomotor palsy and ptosis may fail to notice the visual decline, and only complains of headache as happened in our case. Atropine, which is frequently used in the neurointerventions, especially in carotid stenting, dilates the pupils and may also cause acute angle-closure glaucoma.

Oculomotor, trochlear, abducens, and/or trigeminal nerve paresis may develop in patients with CCFs, probably due to the increased mechanical compression on these nerves in the cavernous sinus or ischemia caused by the arteriovenous fistulas.[9] These nerve pareses could also be caused by transvenous embolization of the cavernous sinus due to compression of the nerves by the embolic materials or thrombus, or increased intrasinusoidal pressure.[1,15] Therefore, oculomotor palsy may develop spontaneously or iatrogenically in patients with CCFs.

Acute angle-closure glaucoma is an emergency condition which, if left untreated, will cause irreversible loss of vision within a few hours. Laser iridotomy is indicated for acute angle-closure glaucoma after the IOP is controlled to a safe level with topical, oral, or intravenous medication. Laser iridotomy is used to make small opening in the iris to facilitate the flow of aqueous humor. This procedure may be performed prophylactically on the opposite unaffected eye because both eyes commonly have narrowed angles and are predisposed to develop acute angle-closure glaucoma.

In conclusion, acute angle-closure glaucoma may develop in association with oculomotor palsy caused by any etiology, including packing of the cavernous sinus during the treatment of CCFs.

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