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Transient Monocular Blindness during Manual Carotid Compression for Carotid-cavernous Sinus Fistulas

—Two Case Reports—

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Abstract

Transient monocular blindness occurred during manual carotid compression in two patients with a traumatic direct and a spontaneous dural arteriovenous carotid-cavernous sinus fistula. Ocular complication may occur during diagnostic or therapeutic carotid compression for carotid-cavernous sinus fistulas, so patients should be informed of this possibility. Ocular examination should be repeated during carotid compression to prevent sequelae of ischemic oculopathy.

Key words: carotid-cavernous sinus fistula, carotid compression, monocular blindness

Introduction

Manual carotid compression is often used for therapeutic purposes in patients with dural carotid-cavernous sinus fistulas (CCFs),[7] and for diagnostic purposes in many neurosurgical settings, such as possible internal carotid artery occlusion, to evaluate the adequacy of the intracranial collateral pathways.[11] We experienced temporary monocular blindness during diagnostic or therapeutic manual carotid compression in two patients with CCFs, and discuss the possible mechanisms and pathophysiological significance of this phenomenon.

Case Reports

Case 1: A 45-year-old female was referred to us for endovascular treatment of a traumatic right CCF about 1 month after the head trauma occurred. On admission, examination found right oculomotor nerve paresis and proptosis on the right side. Bruit synchronous with the heart beat was heard over the right eye.

Angiography revealed the high flow, direct CCF located at the C_4 portion of the right internal carotid artery (Barrow type A[1]). Right carotid angiography detected no intracranial arterial trees. Drainage from the cavernous sinus was toward the right superior orbital vein, the cortical veins, the right pterygoid plexus, and the right inferior petrosal sinus (Fig. 1 *left*).

Endovascular treatment using a detachable bal-

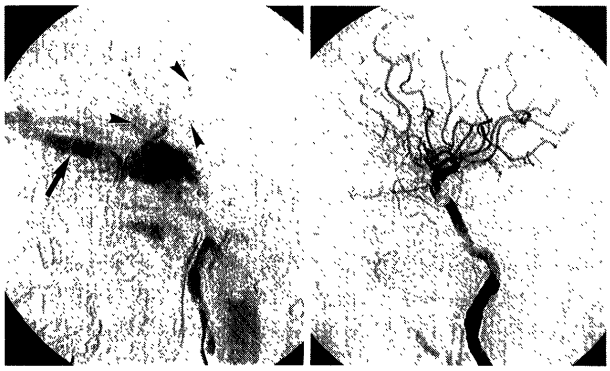

Fig. 1 Case 1. *left*: Right internal carotid angiogram showing the high flow, direct shunt from the internal carotid artery to the cavernous sinus without intracranial arterial trees. Drainage is toward the superior orbital vein (*arrow*), the middle cerebral veins (*arrowheads*), the pterygoid plexus, and the inferior petrosal sinus. *right*: Right internal carotid angiogram after balloon occlusion of the fistula showing the absence of arteriovenous fistula.

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loon was scheduled. Manual carotid compression was carried out to investigate her tolerance to occlusion of the internal carotid artery. Soon after the compression of the cervical carotid artery, she complained darkness in the visual field and then blindness in her right eye within about 10 seconds, which was relieved within 20 seconds of relief of carotid compression. Examination confirmed that her right eye became blind temporarily. This phenomenon was repeatedly observed. No other neurological deficits including hemiparesis occurred.

The following day, the traumatic CCF was occluded with a detachable balloon with preservation of the carotid flow (Fig. 1 *right*). She recovered completely from the right ocular symptoms in 3 months. Manual carotid compression at that time did not cause monocular blindness. She was followed up for 4 years without neurological deficits.

Case 2: A 50-year-old male suddenly developed diplopia. On admission 1 week later, right oculomotor nerve paresis and mild proptosis on the right side were observed. Optic fundi were normal, and no chemosis or bruits were observed.

Angiography revealed a dural CCF (Barrow type D[1]) on the right side. The CCF was fed by branches from the bilateral internal and external carotid arteries. There was no obvious drainage to the right superior orbital vein or cortical veins, only to the right inferior petrosal sinus (Fig. 2 *left*). Normal venous flow collecting the blood from the right cerebral hemisphere drained to the right cavernous sinus, then to the right inferior petrosal sinus as well as the right pterygoid plexus (Fig. 2 *right*).

Ten days after the onset, therapeutic manual carotid-jugular compression was attempted. During compression, he complained that the visual field of the right eye became gradually dark, and then the right eye became nearly blind after about 1 minute. When the compression was stopped, his vision returned to normal gradually over 1–2 minutes. Examination confirmed that his right eye became nearly blind temporarily. This phenomenon was repeatedly observed. No other neurological deficits including hemiparesis occurred. Therefore, therapeutic carotid compression was abandoned.

Transarterial embolization using polyvinyl alcohol particles was carried out in the bilateral middle meningeal arteries and the distal branches of the internal maxillary arteries. After 1 month, his ocular symptoms disappeared completely but the subjective bruits remained. Ten months later, follow-up angiography revealed the residual right dural CCF with almost the same angioarchitecture as previously, but less prominent shunting than before. Manual carotid-jugular compression did not cause any change in his vision.

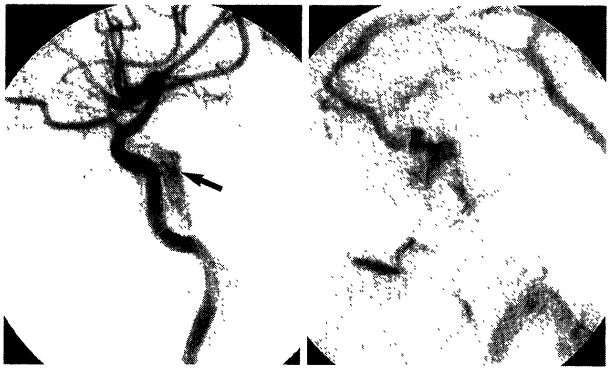

Fig. 2 Case 2. *left*: Right internal carotid angiogram showing the dural CCF located at the posterior cavernous sinus (*arrow*). Drainage is to the right inferior petrosal sinus. *right*: Late phase of the right internal carotid angiogram showing the normal venous return from the right cerebral hemisphere to the cavernous sinus through the sylvian vein and then to the right inferior petrosal sinus and right pterygoid plexus.

Discussion

The retina, the choroidea, and the anterior optic nerve are supplied by the central retinal artery and the posterior ciliary arteries, which originate from the ophthalmic artery, which in turn is supplied by the internal carotid artery. The retina, the choroidea, and the anterior optic nerve drain into the central retinal vein and the vorticose veins. Both venous routes are connected to the superior or inferior orbital veins, which are further connected to the cavernous sinus posteriorly and the superficial facial veins anteriorly.[13]

Visual acuity depends upon the perfusion pressure in the retina, the choroidea, and the anterior optic nerve. The perfusion pressure in these structures is grossly dependent upon the pressure difference between the pressure of the internal carotid artery and the cavernous sinus, and the intraocular pressure. A significant decrease in retinal artery pressure is observed when stenosis of the internal carotid artery is over 80%.[9] Elevated intraocular pressure contributes to the reduction of the perfusion pressure of the retina.[6] Occlusion or stenosis of the internal carotid artery or the common carotid artery may cause ischemia in the ophthalmic artery and sometimes results in decreased ipsilateral ocular acuity.[2,4,5,15] Our two patients did not develop monocular blindness during manual carotid compression after treatment, suggest-

ing that the mechanism was not simply reduction of the blood flow in the ophthalmic artery.

Occlusion of the central retinal vein can cause either "venous stasis retinopathy" or "hemorrhagic retinopathy." Venous stasis retinopathy is simple occlusion of the central retinal vein, for which a good ocular prognosis can be expected. Hemorrhagic retinopathy is occlusion of the central retinal vein with retinal ischemia, which usually has a poor ocular prognosis due to retinal hemorrhage.[6] Hemorrhagic retinopathy is often associated with dural CCF, without ischemia of the ophthalmic artery, but with reduced perfusion pressure in the retina due to increased pressure in the cavernous sinus and increased intraocular pressure.[10,12] This mechanism can cause total monocular blindness in patients with traumatic CCF, but one such patient recovered completely from total monocular blindness after balloon occlusion of the fistula.[3]

Transient monocular blindness has occurred during the balloon occlusion test of the internal carotid artery.[8,14] The causative mechanisms were ischemia in an anomalously proximal origin of the ophthalmic artery[8] and the replacement of oxygenated blood by saline administered in the carotid artery during the temporary balloon occlusion.[14] These mechanisms are different from those in our series, which have not been described before.

Manual carotid-jugular compression is an established treatment for dural CCFs that do not require urgent therapy.[7] Carotid-jugular compression can cause reduction of the pressure in the internal carotid artery, and compression of the jugular vein may result in elevation of the jugular venous pressure, resulting in indirect elevation of the pressure in the cavernous sinus. Therefore, carotid-jugular compression can reduce the perfusion pressure in the retina, the choroidea, and the anterior optic nerve, which leads to temporary visual decline.

Carotid-jugular compression may be carried out by patients themselves under supervision. Patients who undergo this maneuver should be informed of the possible ocular complication of temporary monocular blindness. Ocular examination should be repeated to prevent sequelae of ischemic oculopathy.

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