Resolution of Third Nerve Paresis after Endovascular Management of Aneurysms of the Posterior Communicating Artery

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Summary: The effect of endovascular treatment on the recovery of neural function in patients with third nerve palsy caused by an aneurysm of the posterior communicating artery is poorly documented. We report three cases in which third nerve paresis resolved completely within 2 to 3 weeks of endovascular occlusion of a posterior communicating artery aneurysm.

The recovery of neural function after surgery in patients with oculomotor nerve palsy caused by an aneurysm of the posterior communicating artery is well documented (1), but the effect of endovascular management of such patients on third nerve function is less clear. We report three cases in which posterior communicating artery aneurysms were treated with endovascular coil packing, and describe the effect of treatment on concomitant third nerve palsy.

Case Reports

Case 1

A 90-year-old woman had a 1-day history of occipital headache, and on admission was alert with no neurologic signs. A CT study confirmed the presence of subarachnoid blood within the right sylvian fissure and ambient cistern, and the patient was managed conservatively owing to her age and wishes. Ten days after admission, she experienced increasing right-sided craniofacial pain in association with the development of ptosis and a dilated, poorly responsive pupil on the right. As her pain was refractory to narcotic analgesia, interventional treatment was accepted. Angiography revealed a bilobular 6-mm-diameter aneurysm of the right posterior communicating artery, with 95% occlusion subsequently achieved with endovascular coiling (five coils deployed [GDC-10, FasTracker 10 microcatheter system, Target Therapeutics, Fremont, CA]). Improvement in third nerve function was noted 3 days later, with complete resolution of signs and of craniofacial pain by 18 days after the procedure. At the time of discharge, the patient was well and fully mobile.

Case 2

A previously well 84-year-old woman had a 5-day history of headache and photophobia. On admission, she was alert

with no deficit other than a right-sided ptosis with an associated fixed dilated pupil. A CT scan showed subarachnoid hemorrhage within the right sylvian fissure and ambient cistern. Angiography confirmed the presence of a bilobular aneurysm of the right posterior communicating artery, with the superior and inferior lobules measuring 2 mm and the base 4 mm in diameter. The aneurysm was managed with endovascular coiling (four coils deployed [GDC-18, FasTracker 18 microcatheter system]) the day after admission (6 days postictus), with 100% occlusion achieved. The patient remained well after the procedure, with resolution of the ptosis and recovery of pupillomotor function after a further week. At the time of discharge 3 weeks after admission, she was fully independent with no residual neurologic deficit.

Case 3

A 78-year-old woman had a 1-day history of severe occipital headache. Over the previous 3 weeks she had experienced right-sided craniofacial pain and blurred vision. On admission, she was drowsy and had ptosis on the right in association with a dilated, poorly responsive pupil and with paresis of third nerve–supplied extraocular muscles. A CT scan showed subarachnoid blood within the basal cisterns, with early hydrocephalus. Angiography confirmed the presence of a 5-mm-diameter multilobular aneurysm of the right posterior communicating artery, which was 95% occluded after endovascular coil placement (two coils deployed [GDC-18, FasTracker 18 microcatheter system]) 3 days after admission. The postprocedural course was complicated by an acute myocardial infarction and left ventricular failure, from which a gradual recovery was made. The third nerve palsy had completely resolved 2 weeks after endovascular treatment, and there was concomitant resolution of associated craniofacial pain. The patient was subsequently discharged with home support.

Discussion

The association between rupture of aneurysms of the posterior communicating artery and onset of isolated oculomotor nerve palsy is well established, with a reported frequency of third nerve paresis of up to 34% to 56% (1–3). Impairment of oculomotor nerve function has been attributed to the direct compressive effect of the enlarging aneurysmal sac within the suprasellar cistern (4) and to the irritant effect of an adherent aneurysm and associated subarachnoid blood (5). Associated factors include the presence of larger multilobular aneurysms and female gender (1). In addition, oculomotor nerve palsy, particularly in association with ipsilateral orbitofacial pain, is a well-recognized warning sign, and may indicate impending aneurysmal rupture (6).

Third nerve palsy resulting from aneurysmal disease is complete in approximately 46% of patients at 24 hours, with 66% experiencing complete palsy by 1 week (1). Aneurysm-associated incomplete third nerve palsy usually is seen in the absence of ptosis or extraocular muscle paresis. Sparing of the pupillary responses is less common but may occur with lesions compressing only the inferior portion of the nerve, while the more superiorly placed pupillomotor fibers remain relatively unaffected (7). Third nerve palsy is frequently associated with ipsilateral facial pain, occurring in 64% of cases in one series (1), and has been attributed to the compression of pain sensory afferent fibers recruited from the ophthalmic division of the trigeminal nerve and lying within the periphery of the oculomotor nerve (7).

Improvement in neurologic status after surgical clipping of posterior communicating artery aneurysms has been well documented. Soni (1) reported a series of cases in which all patients operated on within 10 days of onset of signs of third nerve palsy experienced complete recovery of oculomotor nerve function within 3 months of surgery. Conversely, in patients in whom a preoperative palsy had been present for longer than 10 days, recovery was delayed and in no case complete, with 57% experiencing gross residual defect. Giombini et al (4) similarly noted that early surgery resulted in more complete recovery of neural function, but reported that 38% of patients operated on within 14 days of onset of signs still failed to make a complete recovery. By contrast, Kyriakides et al (8), in their review, concluded that the major factor determining postoperative recovery of third nerve function is the degree of preoperative deficit; only 27% of patients with preexisting complete third nerve palsy were found to have made a total recovery. Other early reports have described the effect of carotid ligation on recovery of oculomotor nerve paresis, with residual deficits present in all patients studied (9).

The return of third nerve function after surgical treatment of aneurysms of the posterior communicating artery follows a predictable course: ptosis is frequently the first ocular sign to subside, with recovery generally beginning within the first month after surgery, and full recovery taking several months (1, 4, 8, 10). Resolution of ptosis is usually complete, whereas extraocular muscle function frequently remains impaired, probably as a result of aberrant regeneration (1). In addition, pupillary abnormalities are frequently persistent. Aberrant regeneration may occur in association with nerve recovery months to years after the initial presentation and management of such patients, and is most commonly characterized by abnormal lid movements during movement of the globe and by adduction of the eye on attempted elevation (11). The reported frequency of aberrant regeneration varies greatly, with reported prevalences of 5% to 80% (12, 13), although in one series aberrant regeneration was observed only in patients in whom surgery had been delayed and occurred after an interval of not less than 4 years after surgery (1).

The effect of endovascular management of posterior communicating artery aneurysms on the recovery of concomitant third nerve function is not well documented. In the three cases described in this report, the third nerve paresis was painful in two patients (cases 1 and 3) and complete in one (case 3). The paresis preceded the presenting subarachnoid hemorrhage in one case, was concomitant with hemorrhage in another, and occurred subsequent to the initial hemorrhage in the third case. In each case, the causative posterior communicating artery aneurysm was demonstrated by angiography to be multilobular, and was successfully treated with endovascular coil packing. Recovery of third nerve function was complete in all three cases, including the patient in whom third nerve palsy had probably been present for 3 weeks prior to treatment. Time taken for complete recovery of third nerve function ranged from 1 week to 18 days.

The mechanism of recovery of neural function following endovascular aneurysmal occlusion is unclear. In our patients, the rapid resolution of symptoms and signs of third nerve palsy following coil occlusion suggests that factors other than reduction in the aneurysm's size may play a role in recovery of neurologic function. It has been well documented that giant aneurysms can induce adjacent cerebral edema (14), which may resolve rapidly after endovascular aneurysmal occlusion (15). These observations may reflect the degree of pulsatility within the aneurysmal sac, and it seems likely that decrease of aneurysmal pulsatility is responsible for the dramatic improvement in the neurologic status of the patients included in our report.

The results in these reported cases are noteworthy for two reasons. First, recovery of neural function was complete within 18 days, whereas full recovery following aneurysmal clipping generally takes months to occur. This is probably a reflection of the relatively atraumatic approach associated with endovascular management as opposed to craniotomy, in which intracranial traction may potentially induce neurapraxia of the oculomotor nerve. Second, in one case, oculomotor nerve palsy was probably present for 3 weeks prior to treatment and the full recovery observed in this patient contrasts with the relatively poor postsurgical recovery seen in patients with palsies of longer than 8 days' duration. This observation may similarly reflect the intracranial trauma associated with craniotomy and aneurysmal clipping.

Conclusion

These cases indicate that endovascular management of posterior communicating artery aneurysms may produce effective recovery of associated oculomotor nerve dysfunction. Neural recovery after

endovascular treatment was complete in all cases, and appears to be more rapid than that observed after craniotomy.

References

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