Recovery of Third Nerve Palsy after Endovascular Packing of Internal Carotid-Posterior Communicating Artery Aneurysms

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Summary

Endovascular packing of intracranial aneurysm with preservation of the parent vessel has become in many cases a valid alternative to surgical clipping. Regression of oculomotor disorders after clipping of internal carotid-posterior communicating artery (ICA-PCoA) aneurysms has been well assessed. This report focuses on the reversal of third nerve palsy after endovascular packing of ICA-PCoA aneurysms. To this end, clinical appearances, neuroradiological features, and endovascular interventional procedures of six treated patient are reported and discussed in the light of the very few previous case observations found in the literature. Results indicate that endovascular packing of ICA-PCoA aneurysms may produce effective recovery of correlated third nerve dysfunction.

Introduction

Intracranial aneurysms are the commonest cause of isolated nontraumatic third nerve palsies, with a percentage ranging from 18 to 40%¹⁻⁴. Internal carotid-posterior communicating artery (ICA-PCoA) aneurysms are more frequently linked with such a nerve dysfunction up to a rate of 89%⁵, whereas aneurysmal in-

volvement at the carotid cavernous, carotid bifurcation, and at the junction of the basilar artery with the superior cerebellar artery or with the posterior cerebral artery has rarely been observed⁵⁻⁷. Reversal of oculomotor disorders after surgical management of correlated intracranial aneurysms has also been well established⁴⁻⁹. Conversely, third nerve function improvement after endovascular treatment of correlated intracranial aneurysms has been poorly documented, only a series of three cases having been reported in the relevant literature¹⁰. In addition, four case observations were preliminarily communicated at the 15th Congress of the Italian Association of Neuroradiology¹¹. With this background, we report our experience of six patients who had full recovery of third nerve paresis after endovascular treatment of ICA-PCoA aneurysm with Guglielmi detachable coils (GDCs)^{12,13}.

Material and Methods

In the period 1996-1999, a consecutive series of 15 patients with ICA-PCoA aneurysms were treated by means of endovascular packing with GDCs. In six cases (40%), oculomotor nerve palsy existed. In all cases but one of this group the deficit was complete. There were five berry

and one large ICA-PCoA aneurysms. In one patient only (case 2), there was no subarachnoid haemorrhage (SAH). The ICA-PCoA aneurysm origins were classified according to Pia¹⁴. The projection of the aneurysmal fundus was therefore recognized according to the Yasargil categories¹⁵ (in all our cases the direction of the fundus was posterolateral-inferior). All interventional neuroradiological procedures were performed using live simultaneous biplane roadmapping, under general anesthesia, and systemic heparinization with a 1000-unit bolus (5000-unit bolus in the case of unruptured aneurysm) at the time of the first coil deployment and 1000 units of heparin per hour until completion of the procedure. Transfemorally inserted 6F guiding catheters were positioned in the internal carotid artery. The GDC-10, the GDC-14, and the GDC-18 microcatheters were used, in combination with the 10-Transend Ex Platinum or the 12/16-Terumo GT guidewires, for endosaccular packing. At the end of the procedure, all patients were transferred to the Neurointensive Care Unit for observation. The time of stay depended on the course and severity of the disease. The patient with the unruptured aneurysm was discharged within 42 hours. In SAH patients, Transcranial Doppler (TCD) was carried out to monitor any vasospasm. For this purpose, Nimodipine therapy (10 ml/h) was administered. Digital subtraction angiography (DSA) and magnetic resonance angiography (MRA) controls were scheduled at three months and one year (MRA only) after embolisation.

Reports

Case 1

This 67-year-old woman complained of transient episodes of diplopia and right orbital pain. She was then admitted owing to onset of acute headache, photophobia and right ptosis. Signs of meningeal irritation and complete right third nerve palsy were observed at neurological examination. A diffuse SAH, with blood prevalence in the right basal cisternae and sylvian fissure, was demonstrated by cranial CT scan (Fisher grade 2). DSA revealed a 13-mm-diameter aneurysm of the right ICA-para PCoA. Embolisation, two days later, was performed with subtotal aneurysmal occlusion. Two days after treatment, ptosis improved. At

two months, the patient is well and the oculomotor deficit has completely recovered.

Case 2

A 70-year-old woman had a six month history of diplopia and right orbital pain. Ophthalmologic evaluation showed a slight right pupil enlargement and incomplete rectus medialis muscle deficit, without ptosis. Rectus medialis deficit was confirmed at Lancaster-Hess test. Cranial and orbital CT scans were negative. Finally, an unruptured 4-mm-diameter ICA-para PCoA (fetal type posterior cerebral artery) aneurysm was diagnosed at DSA. Embolisation was carried out and subtotal aneurysm occlusion was achieved. The oculomotor deficit fully recovered in 14 days.

Case 3

A 59-year-old woman presented with acute headache, right hemiparesis, dysphasia, and complete left third nerve palsy due to SAH (Fisher grade 4 at CT scan). DSA and MRA revealed a 6-mm-diameter aneurysm located at left ICA-supra PCoA and a 2-mm-diameter aneurysm at left middle cerebral artery bifurcation (fig. 1 A, B). Rebleeding occurred five days later and neurological status dropped to grade 4 of the Hunt-Hess SAH classification. Endovascular management with GDCs was delayed to 35 days after SAH and onset of symptoms, when the patient's conditions improved and vasospasm subsided. Total obliteration of both aneurysms was achieved with embolisation (figure 1C). Two days later, ptosis improved. Twenty days after treatment, complete regression of hemiparesis, dysphasia, and third nerve palsy were observed. At one year the patient is well and MRA control confirms full obliteration of the aneurysms (figure 1D).

Case 4

A previously healthy 56-year-old woman was admitted with clinical and neuroradiological findings of SAH (Fisher grade 3). She was alert, with no deficit other than complete right third nerve palsy with orbital pain. DSA demonstrated a 4-mm-diameter ICA-origin of PCoA (aplasia of PCoA) aneurysm. Endovascular management was delayed due to the presence of angiographic and TCD vasospasm. Twenty-two days after onset of SAH and oculomotor deficit, subtotal occlusion of the

Table 1 Clinical features, neuroradiological appearances, endovascular treatment, and clinical results of ICA-PCoA aneurysms with third nerve palsy

N	Sex Age	Oculomotor deficit	SAH	Hunt- Hess grading	Origin and diameter of aneurysm	Aneurysmal fundus direction	Endovascular packing time after onset of symptoms	Occlusion of the aneurysm	Glasgow Outcome Scale	Recovery time of III nerve after packing
1	F 67	Complete	Yes	2	Right 13-mm ICA-para PCoA	Posterolateral inferior	2 days	Subtotal	5	Full 60 days
2	F 70	Partial	No	0	Right 4-mm ICA-para PCoA (fetal type posterior cerebral artery)	Posterolateral inferior	6 months	Subtotal	5	Full 14 days
3	F 59	Complete	Yes	3 worsened to 4	Left 6-mm ICA-supra PCoA and left 2-mm middle cerebral artery bifurcation	Posterolateral inferior	35 days	Total	5	Full 20 days
4	F 56	Complete	Yes	2	Right 4-mm ICA-origin of PCoA (aplasia of PCoA)	Posterolateral inferior	22 days	Subtotal	5	Full 10 days
5	F 77	Complete	Yes	2	Left 5-mm ICA-para PCoA	Posterolateral inferior	3 days	Total	5	Subtotal (anisocoria) 30 days
6	F 29	Complete	Yes	2	Right 7-mm ICA-para PCoA	Posterolateral inferior	2 days	Total	5	Full 35 days

aneurysm was achieved by means of GDCs. At discharge, ten days after embolisation, the patient was well and the oculomotor deficit and orbital pain had completely disappeared.

Case 5

A 77-year-old woman was admitted for acute headache, vomiting, and complete left third nerve palsy. CT showed diffuse SAH (Fisher grade 2). Angiography diagnosed a left 5-mm-diameter ICA-para PCoA aneurysm. Total occlusion of the aneurysm was obtained with embolisation, performed three days later. Ptosis improved the day after embolisation, and one month later the third nerve palsy had subsided completely except for a slight left pupil enlargement, which, however, did not affect her visual function.

Case 6

This 29-year-old woman presented with an episode of transient diplopia. She was then ad-

mitted ten days later because of acute headache and right ptosis. Neurological examination indicated rigor nuchalis and complete right third nerve palsy. CT scan revealed subarachnoid blood effusion in the basal cisternae and in the fourth ventricle (Fisher grade 4). DSA demonstrated a right 7-mm-diameter ICA-para PCoA aneurysm (figures 2A, 2B). Endovascular packing was performed within 24 hours after haemorrhage and total obliteration of the aneurysmal sac was therefore obtained (figures 2C, 2D). At discharge, eight days after the packing, the patient was well and oculomotor function has partially recovered. Complete reversal of the oculomotor deficit was observed thirty-five days later.

Result

Overall clinical data, neuroradiological findings, times of treatment and clinical results are summarized in table 1. In all six reported cases,

Figure 1 Case 3. Left internal carotid angiography (A) and magnetic resonance angiography (B) in the oblique projection give evidence of two aneurysms at ICA-supra PCoA artery and middle cerebral artery bifurcation. Total obliteration of aneurysms was possible through endovascular treatment, as shown at angiographic (C) and MRA (D) controls.

total (three cases) or subtotal occlusion of the aneurysm was obtained (subtotal = 95-99% occlusion of aneurysm, according to the occlusion grading system of Cognard¹⁶). All patients are well at one year follow-up on average. In three cases of SAH patients, treatment was performed within 72 hours after onset of deficit and in the other two cases after 20 days and one month respectively. In the only patient with unruptured aneurysm, treatment was performed at six months after onset of deficit,

which, however, was not complete. In all six cases, no improvement of the third nerve deficit was observed before the endovascular treatment. Following endovascular packing, full recovery of the third cranial nerve function was obtained in all patients, within a period ranging from ten days and two months. Only in one case with early treatment, does a modest anisocoria remain, which, however, is irrelevant to vision quality. The total recovery times, from onset of deficit to recovery, for the early treat-

Figure 2 Case 6. Lateral (A) and oblique (B) right carotid angiography indicates a right ICA-para PCoA aneurysm. Angiographic controls (C, D) after endovascular packing demonstrate total aneurysm occlusion.

ed (within 72 hours) patients were 62, 33, and 37 days (cases 1, 5, and 6), respectively, while for delayed treatment patients (cases 2, 3 and 4) the total recovery times were 196, 55, and 32 days. Conversely, we have noticed that the deficit regression obtained from the day of endovascular packing in the patients with delayed treatment was achieved in less time, respectively at 10, 14, and 20 days, than in the cases treated during the early phase, who recovered at 30, 35, and 60 days, respectively.

Discussion

The first autopsy finding of an intracranial aneurysm correlated to the clinical observation of a third nerve palsy dates back to France¹⁷ in 1846. Hutchinson¹⁸ in 1875 recognized the presence of an ICA aneurysm in a patient with a third nerve palsy. Since then, the association between ICA-PCoA aneurysms and third nerve palsy has been well documented in many reports, with an incidence ranging from 20% to

61% of all ICA-PCoA aneurysms^{2,4-8,10}. According to Soni⁴, 50% of ICA-PCoA aneurysms associated with oculomotor palsy are larger than 11 mm. Other authors^{1,15} have stressed that the posterolateral-inferior direction of the aneurysmal fundus was the most frequently encountered in the presence of oculomotor deficit. On the other hand, Giombini⁷ observed, by analyzing the pertinent literature, that there is no significant relationship between the radiological measurement, morphology or direction of ICA-PCoA aneurysms and the clinical picture. In our series, we have no significant data to support these hypotheses, even if the aneurysm was larger than 11 mm in only one of our six cases, while all aneurysms pointed posterolateral-inferiorly. In another review of the relevant literature, Leivo⁵ found that SAH was present in 55% of ICA-PCoA aneurysms with third nerve palsy. Surely, oculomotor palsy is often a "warning symptom" of an enlarging ICA-PCoA aneurysm or of minor aneurysmal bleeding^{1,19}. The anatomic damage to the nerve may be caused by the pulsing and mass effect of the sac, as well as by the ruptured aneurysm with pressure arterial bleeding. During the early neurovascular conflict, a conduction block takes place, while, after longer periods, continuous pulsing or mass effect are likely to affect intraneural circulation thus leading to nerve ischemia. If these phenomena become chronic, the occurrence of fibrosis, scarring, and necrosis is likely. When SAH is present, there may be several phases of neural damage, from minor axonolysis, to direct damage to the nerve when disastrous bleeding occurs⁷. The clinical deficit may either be partial or incomplete, in correlation with the anatomic damage. Dilatation of the pupil, palpebral ptosis and, finally, muscular impairment of upper, lower and medial recti may occur, here listed in progressive order. With a full deficit, the eye is totally closed, the mydriatic pupil fails to react to direct and consensual light stimuli, the eyeball is deviated laterally and movements of elevation, adduction and depression are impossible. Complete palpebral ptosis prevents diplopia. The association with orbital pain has a frequency of up to 66%⁵, which, according to an autoptic study¹⁹, is due to the involvement of sensory fibers of the third cranial nerve originating from the ophthalmic division of the fifth nerve and joining the third nerve at the level of the lateral wall of

the cavernous sinus. The first major study on recovery of third nerve function after surgical management of ICA-PCoA aneurysms was published by Botterell²⁰ in 1962. Furthermore, other reports have documented third nerve paresis regression after clipping of intracranial aneurysms, the times of recovery having been correlated to early or delayed surgery^{1,4-8}. The best results are achieved when early surgery (within 72 hours from the onset of deficit) is performed, while two thirds of patients reported from literature had complete recovery when operated on within two weeks⁵. Improvement prospects drop when surgery is performed later than one month after onset of deficit. The advent and improvement of endovascular packing techniques of intracranial aneurysms in recent years has considerably changed the approach to this problem. Safety and efficacy of packing with GDCs of unruptured or acutely ruptured intracranial aneurysms has been well established. In Birchall's report¹⁰, the three cases of very elderly patients, respectively 90, 84, and 78 years, with ruptured ICA-PCoA aneurysm associated with third nerve dysfunction recovered 7, 14, and 18 days after embolisation. Even the results on the times of improvement and recovery following endovascular packing of our six cases are really short. In any case, the number of reported aneurysmal patients with third nerve palsy involved in endovascular management is too small, and no random or even prospective assignment may be made. We are aware that any initial comparison between the results of surgical and endovascular treatment must be very cautious, even if it certainly appears that GDC treatment is compatible with a very good recovery of third nerve function. The clinical improvement mechanism after embolisation is still unclear, especially in ruptured aneurysms. Important factors in deficit improvement following embolisation have been correlated to the regression in aneurysm pulsation and to mass effect resolution^{21,22}. The mass effect of the sac is also well relieved when clipping the aneurysm with correlated nerve deficit. However surgery, with respect to a minimally traumatic procedure such as endovascular packing, is likely to cause direct compression or traction, thus offsetting the more rapid reduction in pulsatility and mass effect that it provides. The recovery of nerve function begins with improvements in

eyelid elevation, followed by pupil contraction, and finally by the resumption of intrinsic and extrinsic ocular movements. In our limited number of cases, while the total recovery times between the two groups of patients (early and delayed treated) essentially overlapped, the recovery times from the day of treatment were longer in the three cases who underwent early endovascular occlusion than in the three delayed cases. Probably, unlike previous reports, the recovery times are not related only to the precocity of treatment, but also to the natural history which primarily affects patient selection and timing of treatment, and, as reported else-

where^{1,2,4,7}, other factors such as disease severity, extent of anatomical damage to the nerve, deficit degree, and aberrant regeneration phenomena, are likely to influence the times and speed of reversibility and deficit recovery.

Conclusions

By analyzing our results it is clear that endovascular packing of ICA-PCoA aneurysm may lead to satisfactory recovery of correlated third nerve palsy, even if additional prospective trials on larger population series are required to collect more information on this issue.

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