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ORIGINAL RESEARCH

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Recovery of Posterior Communicating Artery Aneurysm-Induced Oculomotor Palsy after Coiling

BACKGROUND AND PURPOSE: Recovery of oculomotor (cranial nerve [CN] III) palsy after surgery of posterior communicating artery (PcomA) aneurysms has been well documented, but recovery after coiling is poorly understood. In this study, we report the recovery after coiling of PcomA aneurysm-induced CN III palsy in 21 patients at follow-up of 1 to 7 years.

MATERIALS AND METHODS: Of 135 patients with a PcomA aneurysm treated with coils between January 1997 and December 2003, there were 21 patients with initial CN III dysfunction who were selected and reevaluated. There were 2 men and 19 women with a mean age of 54.9 years. In 17 patients, CN III palsy was associated with subarachnoid hemorrhage (SAH). Timing of treatment after onset of symptoms was 1 to 3 days in 5 patients, 4 to 14 days in 13, and more than 14 days in 3. Mean size of the aneurysm was 9 mm. Initial CN III palsy was complete in 15 patients and partial in 6. Mean follow-up after coiling was 3.7 years (range, 1–7 years).

RESULTS: Of 15 patients with initial complete CN III palsy, recovery was complete in 3 and partial in 10. In 2 patients, complete CN III palsy was unchanged. Of 6 patients with initial partial CN III palsy, recovery was complete in 5 and partial in 1. Initial partial CN III palsy was the only predictor of complete recovery at follow-up.

CONCLUSION: PcomA aneurysm-induced CN III palsy improves or cures after coiling in most patients. Complete recovery is more likely with initial partial dysfunction of the nerve.

Oculomotor nerve (cranial nerve [CN] III) palsy from posterior communicating artery (PcomA) aneurysms is a well-known clinical entity. Direct mechanical compression of the nerve by the aneurysm may result in partial or complete dysfunction of CN III. Onset may be acute by sudden increase in aneurysm volume as a result of aneurysmal wall dissection with or without accompanying subarachnoid hemorrhage (SAH). In a minority of patients, CN III palsy is the only clinical presentation of a PcomA aneurysm.

Complete or partial recovery of CN III palsy after surgical treatment of the aneurysm has been reported to occur in most patients.^{1–10} Recovery after coiling has been documented in case reports or in small case series only.^{11–13} In our study, we want to report the recovery after coiling of PcomA aneurysm-induced CN III palsy in 21 patients at follow-up of 1 to 7 years.

Patients and Methods

The Institutional Review Board approved our study, and we obtained written informed consent from all patients.

Between January 1997 and December 2003, a total of 135 patients with a PcomA aneurysm were treated with coils. In June 2005, a written request to cooperate with this follow-up study was sent to 115 surviving patients, and 63 patients responded with written consent. The hospital records of these 63 patients were reevaluated on the basis of CN III symptoms at presentation. To verify hospital recordings and identify patients with initial CN III symptoms that were not noted in the records, we interviewed the 63 patients by telephone with a standardized questionnaire about ocular symptoms at the time of treatment of the aneurysm. Of 63 patients, 21 were found to have a history

of CN III symptoms and were reevaluated at the outpatient clinic. Next to a detailed retrospective and current anamnesis and reevaluation of the medical records, complete neuro-ophthalmologic examination was performed.

The following parameters were assessed: initial degree of CN III palsy, sex, age, aneurysmal size and rupture status, Hunt and Hess (HH) grade at the time of treatment, interval between onset of symptoms, and treatment and duration of follow-up.

Complete CN III palsy was defined as diplopia, ptosis, ophthalmoplegia, and pupillary dysfunction. Partial CN III palsy was defined as incomplete ptosis associated with partial ocular movement in upward, medial, and downward gazes, or pupillary sparing.

The criteria for complete recovery of CN III palsy were no diplopia in all direction of gazes, complete resolution of ptosis, and partial or complete recovery of pupillary reaction. Partial recovery of CN III palsy was defined as residual diplopia in upward, downward, or medial gaze with or without normal primary gaze, residual ptosis, and pupillary dysfunction.

Statistical Analysis

The Fisher exact test was used to establish the relationship between complete recovery of CN III palsy at follow-up and the following 3 parameters: initial complete or partial CN III palsy, timing of treatment dichotomized as 1 to 14 days and 14 days or more, and aneurysmal size dichotomized as less than or equal to 10 mm or more than 10 mm.

Results

Patients

Of 21 selected patients with a PcomA aneurysm and CN III palsy treated with coils, 2 were men and 19 were women with a mean age at the time of treatment of 54.9 years (median age 54 years; age range, 33–76 years). There were 17 patients who presented with both SAH and CN III palsy as symptoms and 4 patients with CN III palsy as the only symptom. HH grade at

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Table 1: Relationship of initial degree of CN III palsy and recovery at follow-up in 21 patients with a coiled posterior communicating artery aneurysm

Initial CN III Palsy	n	Complete Recovery	Partial Recovery	No Recovery
Partial	6	5	1	0
Complete	15	3	10	2
Total	21	8	11	2

Table 2: Relationship of initial degree of CN III palsy, treatment interval, and recovery at follow-up in 21 patients with a coiled posterior communicating artery aneurysm

	n	Complete Recovery	Partial Recovery	No Recovery
Total group				
1–3 days	5	4	1	0
4–14 days	13	4	9	0
>14 days	3	0	1	2
Initial partial CN III palsy				
1–3 days	3	3	0	0
4–14 days	3	2	1	0
>14 days	0	0	0	0
Initial complete CN III palsy				
1–3 days	2	1	1	0
4–14 days	10	2	8	0
>14 days	3	0	1	2

the time of treatment in the 17 patients with a ruptured aneurysm was HH I-II in 13, HH III in 3, and HH IV-V in 1 patient. Timing of treatment after onset of symptoms was 1 to 3 days in 5 patients, 4 to 14 days in 13, and more than 14 days in 3. Mean aneurysmal size was 9 mm (median, 8 mm; range, 5–20 mm). Initial CN III palsy was complete in 15 patients and partial in 6. Mean follow-up after coiling was 3.7 years (median, 3 years; range, 1–7 years).

Neuro-Ophthalmologic Follow-Up

Results of neuro-ophthalmologic follow-up are summarized in Table 1. Of 15 patients with initial complete CN III palsy, recovery was complete in 3 and partial in 10. In 2 patients, complete CN III palsy was unchanged. Of 10 patients with partial recovery of CN III palsy, primary gaze diplopia was present in 2, and in 8 patients, residual diplopia was limited to upper or medial gaze. In all patients with partial recovery, the ptosis resolved completely.

Of 6 patients with initial partial CN III palsy, recovery was complete in 5 and partial in 1 (residual upper gaze diplopia).

Most patients reported recovery of ptosis first (within 3 days to 3 months). Recovery of diplopia generally took longer but was completed to end stage within 1 year in all patients.

Statistical Analysis

Complete recovery of CN III palsy was significantly higher in patients with initial incomplete palsy versus patients with initial complete palsy (5/6 vs 3/15; $P = .014$). There was no relationship between complete recovery of CN III palsy and treatment interval (Table 2) and aneurysmal size.

Discussion

In our study, we found that recovery of CN III palsy after coiling of a PcomA aneurysm occurred in 21 (91%) of 23

patients. Recovery was complete in 8 and partial in 13 patients. Most patients with partial recovery had residual diplopia in upward or medial gaze only. The only predictor for complete recovery was partial CN III palsy from the onset; other possible factors such as aneurysmal size and treatment interval had no predictive value in our limited patient group.

A limitation of our study was the introduction of a selection bias toward patients who were willing to participate in this study that may have influenced results. Another limitation was that hospital records were not always complete, and the initial degree of CN III palsy had to be assessed from retrospective anamnesis in some patients. It is likely that subtle manifestations of initial CN III dysfunction (eg, pupillary function) were missed, both at initial assessment and from retrospective anamnesis.

Outcome of CN III function after surgery has been well documented. Overall, the rate of complete recovery after clipping has ranged between 40% and 60%.^{1–7,10} When surgery has been performed in the first days after onset of CN III palsy, the rate of full recovery has been reported to be as high as 80% to 90%.^{8,9} Patients with initial partial CN III palsy more often had complete recovery than patients with initial complete palsy.

Recovery of CN III palsy after coiling has been documented only in case reports or small case series.^{11–13} Unlike surgical clipping, coiling does not immediately resolve the mass effect of an aneurysm. However, the loss or reduction of aneurysmal pulsatility followed by some shrinkage of the aneurysm may be more important in nerve recovery than surgical anatomic detachment of the nerve from an adjacent and adherent aneurysm. In a prospective study by Stiebel-Kalish et al¹¹ of 11 patients with complete CN III palsy, recovery was partial after coiling in all 11 patients. However, residual oculomotor nerve deficits did not cause diplopia with primary gaze for 10 of 11 patients, and clinically significant ptosis did not persist for any of the patients. The degree of nerve recovery correlated with aneurysmal size, duration of CN III palsy before coiling, age, and the presence of other microvascular risk factors.

Recently, outcome of CN III function after surgery and coiling has been directly compared in 2 studies of 17 and 13 patients. Ahn et al¹⁴ and Wong et al¹⁵ compared 10 patients after coiling with 7 patients after clipping and found no difference in complete CN III recovery versus partial recovery with both treatment modalities. Significant factors of recovery after coiling were the degree of CN III palsy at presentation and interval between onset and treatment. Chen et al¹⁶ compared 6 patients after coiling with 7 patients after surgery and found a higher probability of complete CN III recovery after surgery (6/7 vs 2/6). Patients with initial partial CN III palsy more often had complete recovery.

In general, both after surgery and coiling, functional recovery was noted first in the levator palpebrae muscle, followed by the medial rectus muscle, superior rectus muscle, constrictor muscles of the iris, and ciliary muscle. Patients with incomplete recovery often had residual diplopia in upward gaze and pupillary dysfunction. Nerve recovery was invariably within the first year after treatment.

Overall, our findings of CN III recovery after coiling are in concordance with those of other studies regardless of treatment method. Small differences in study results may be ex-

plained by limited numbers of evaluated patients and differences in methodology (eg, in inclusion of proportion of patients with initial partial or complete nerve dysfunction). From the limited available data, there is no evidence of better outcome with any of the 2 therapies.

Initial partial CN III palsy is a solid predictor for complete recovery in both therapies. A nerve in partial palsy from compression is likely in a neurapraxic phase, a reversible conduction block. Early therapy is likely to improve prognosis, both after surgery and after coiling.

Conclusion

PcomA aneurysm-induced CN III palsy improves or cures after coiling in most patients. Complete recovery is more likely with initial partial nerve dysfunction. Early therapy is likely to improve prognosis.

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