# Posterior communicating artery aneurysm oculomotor nerve palsy

Oculomotor nerve palsy (ONP) is often the presenting symptom in patients with posterior communicating artery aneurysms with variable recovery of oculomotor nerve function following treatment.

Unruptured posterior communicating artery (PCoA) aneurysms with oculomotor nerve palsy (ONP) have a very high risk of rupture.

ONP can occur with PCOMM aneurysm with or without subarachnoid hemorrhage (SAH) [1].

## Epidemiology

It has been estimated that oculomotor nerve palsy (OMNP) occurs in up to one-third of patients with posterior communicating artery aneurysms due to a mass effect on the oculomotor nerve [2].

## Outcome

The prognosis of oculomotor palsy mainly depends on the interval between the onset of palsy and the time of operation, and furthermore on the degree of preoperative deficit and the development of the cranial nerve lesion. The incidence of ultimately complete or incomplete palsy is the same in cases with subarachnoid haemorrhage and without rupture (“warning symptom”).

In many cases, an initially incomplete paresis develops to a complete ocular palsy within eight days. Ptosis is generally the first symptom, and it frequently shows the earliest recovery of all other disturbed oculomotor functions after surgery. Full recovery of oculomotor palsy occurs usually only in those patients who undergo early clipping of an aneurysm, i.e. mainly within 10 days after onset of ocular palsy. Complete restitution after carotid ligation is possible, but rare. In cases with full recovery, restitution occurs mostly within three months, sometimes even within a few weeks. An improvement in oculomotor palsy is still possible after a year, but ultimately in these patients recovery remains always more or less incomplete. Incomplete restitution of a third cranial nerve lesion is very often associated with aberrant regeneration and subsequent synkinetic ocular movement. The restitution of the single ocular muscle functions shows a fairly constant course: the levator palpebrae muscle and the M. rectus medialis show rapid recovery. The parasympathetic fibres follow next, but normal function of elevation and depression of the ocular bulb (M. rectus sup., M. obliquus inf. and M. rectus inf.) is often delayed [3].

Patients with ONP secondary to PCoA aneurysms treated with clipping showed higher rates of full ONP resolution than patients treated with coil embolization. Larger prospective studies are needed to determine the true potential of recovery associated with each treatment [4].

Eleven relevant studies involving a total of 384 patients with third nerve palsy due to PCoA aneurysms at baseline, of whom 257 (67.0%) were treated by clipping and 127 by coiling (33.0%), were included in a meta-analysis. Pooled Odds Ratios of the impact of clipping or coiling on complete ONP recovery, lack of ONP recovery and procedure-related death were calculated. The overall complete ONP

recovery rate was 42.5% in the coiling group compared to 83.6% in the clipping group. The increase in complete ONP recovery in the clipping group corresponds to an overall pooled Mantel-Haenzel odds ratio of 4.44 (95% CI 1.66-11.84). Subgroup analysis revealed a clear benefit of clipping over coiling in patients with ruptured aneurysms, but not in unruptured aneurysms. None of the eleven studies reported any procedure-related death.

Surgical clipping of PCoA aneurysms causing third nerve palsy achieves better ONP recovery than endovascular coiling. This result could be particularly true in the case of ruptured aneurysms. In view of the purely observational data, statements about this effect should be made with great caution. A randomized trial would address the therapeutic dilemma involved better, but pending the results of such a trial, we recommend treating PCoA artery aneurysms causing ONP with surgery [5].

Simultaneous elimination of 2 injury mechanisms, compression and pulsation, when treating the oculomotor nerve by surgical clipping may be more advantageous than endovascular embolization [6].

## Mecobalamin treatment

27 patients were given embolization treatment and 28 received embolization + mecobalamin treatment. The recovery condition of ONP were followed and compared one year after the treatment.

All patients were followed up for more than a year. And 31 patients (56.4%) out of 55 achieved complete recovery, 19 (34.5%) attained partial recovery and 5 (9.1%) had no recovery from ONP. Whereas, 20 patients (71.4%) in the embolization + mecobalamin treatment group achieved complete recovery and 11 (40.7%) in the embolization treatment group achieved partial recovery, and the comparative difference was statistically significant ($p < 0.05$).

Endovascular is highly efficacious treatment for ONP-inducing PcomA and can promote the recovery of oculomotor nerve palsy after embolism [7].

## Systematic reviews

A meta-analysis of studies that compared surgical clipping with endovascular coiling was conducted by searching the literature via Pubmed, Embase and Cochrane Library databases without restricting the publication year. We extracted the following information: author names and publication year; clinical outcome (number of complete and incomplete recovery of ONP); perioperative data (number of pre-operatively complete or incomplete ONP, subarachnoid hemorrhage or not, number of complications (hydrocephalus, recurrence of PcomAA)). Except for author names and publication year, the data was pooled to perform a mean effect size estimate. The effects of two treatment modalities were then analyzed.

Nine published reports of eligible studies involving 297 participants met the inclusion criteria. Overall, compared with endovascular coiling, surgical clipping had no statistically significant difference on the complete recovery of ONP, although there was an obvious trend in favor of clipping [RR=1.48, 95%CI (0.95, 2.29), p=0.08]. There was no significant difference in the total efficiency (any degree of change) on ONP [RR=1.08, 95%CI (0.94, 1.25), p>0.05], the overall complications [RR=0.60, 95%CI (0.33, 1.10), p>0.05], the efficacy on the complete recovery of ONP in patients without SAH [RR=0.83, 95%CI (0.53, 1.31), p>0.05], the effect on the complete recovery of ONP in patients with pre-operatively complete or incomplete ONP [RR=1.12, 95%CI (0.68, 1.85), p>0.05], [RR=1.12,

95%CI (0.79, 1.59), p>0.05]. In a comparison of a small cohort of patients that had suffered an SAH (17 vs. 22) there was a significant difference on the effect on complete recovery of ONP between clipping and coiling [RR=1.70, 95%CI (1.08, 2.67), p<0.05].

A superiority of clipping over coiling for the complete recovery of oculomotor nerve palsy in patients that had suffered an SAH from a ruptured aneurysm of the posterior communicating artery was found in the present meta-analysis. Limited by the relatively small sample sizes included, there were no significant differences observed in the clinical outcome between coiling and clipping in the treatment of unruptured PcomAA causing ONP. More evidence from advanced multi-center studies of large scale is needed to provide insight into the optimal treatment for outcome of ONP caused by PcomAAs [8].

## Case series

### 2017

An analysis of the clinical data of 52 enrolled PcoAA patients with ONP who had treatment in the Department of Neurosurgery in Anhui Provincial Hospital from January 2011 to June 2015 was conducted. There were 23 patients among a total underwent surgical clippings and others 29 patients received endovascular embolization treatment. Then, the age, gender, aneurysm size and rupture status, onset duration, preoperative ONP severity and postoperative recovery degree of ONP of patients in the two groups were compared.

The final ONP outcomes of the 52 PcoAA patients consisted of 27 full recovery patients (51.9%), 21 partial recovery patients (40.4%), and 4 no recovery patients (7.7%). (1) Within the 23 patients in the surgical clipping group, subarachnoid hemorrhage (SAH) occurred in 16 patients, and no SAH occurrence in the other 7 patients; the final ONP evaluation showed 18 patients fully recovered (78.3%) and 5 patients partially recovered (21.7%). Within the 29 patients in the endovascular embolization group, SAH occurred in 18 patients, and no SAH occurrence in the other 11 patients; the final ONP evaluation showed 9 patients fully recovered (31%), 16 patients partially recovered in 16 patients (55.2%) and 4 no recovery patients (13.8%). (2) The postoperative ONP recovery was analyzed with multivariate logistic regression, and the treatment method was an independent factor for ONP recovery (OR = 0.041, 95% CI: 0.007-0.261, p < 0.01).

When compared with the endovascular embolization, the surgical clipping showed a better efficacy in the recovery from PcoAA related ONP [9].

### 2016

Fourteen unruptured PCoA aneurysms with ONP, 33 ruptured PCoA aneurysms, and 21 asymptomatic unruptured PCoA aneurysms were included in a study. The clinical, morphological, and hemodynamic characteristics were compared among the different groups.

The clinical characteristics did not differ among the 3 groups (p > 0.05), whereas the morphological and hemodynamic analyses showed that size, aspect ratio, size ratio, undulation index, nonsphericity index, ellipticity index, normalized wall shear stress (WSS), and percentage of low WSS area differed significantly (p < 0.05) among the 3 groups. Furthermore, multiple comparisons revealed that these parameters differed significantly between the ONP group and the asymptomatic unruptured group and between the ruptured group and the asymptomatic unruptured group, except for size, which

differed significantly only between the ONP group and the asymptomatic unruptured group (p = 0.0005). No morphological or hemodynamic parameters differed between the ONP group and the ruptured group.

Unruptured PCoA aneurysms with ONP demonstrated a distinctive morphological-hemodynamic pattern that was significantly different compared with asymptomatic unruptured PCoA aneurysms and was similar to ruptured PCoA aneurysms. The larger size, more irregular shape, and lower WSS might be related to the high rupture risk of PCoA aneurysms [10].

### 2015

230 PCOMM aneurysm endovascular coilings between the years 2006 and 2011, of which 20 cases presented with ONP. Sheehan et al. recorded the degree of nerve recovery - complete, partial or none - while also documenting other predictive factors, such as degree of pre-intervention nerve deficit, presence of subarachnoid haemorrhage (SAH), size and location of the PCOMM aneurysm and length of follow-up.

Of the 20 patients, 9 (45%) presented with complete ONP and 11 (55%) with partial ONP. After an average follow-up period of 16 months, all patients achieved oculomotor nerve recovery; 9 (45%) patients had complete recovery and 11 (55%) patients had partial recovery. Of the 9 patients who presented with complete ONP, 5 (56%) patients made a complete recovery and 4 (44%) made a partial recovery. Of the 11 patients who initially presented with partial ONP, 4 (36%) made a complete recovery and 7 (64%) made a partial recovery. 7 (35%) patients also had a SAH, of whom 3 (43%) made a complete recovery with 4 (57%) making a partial recovery [11].

### 1974

One hundred and seventy-four patients with a posterior communicating aneurysm were seen over a 21 year period. There was a ratio of four females to one male and women were on average five years older. Fifty-nine (34%) had an oculomotor paresis. This group had up to four attacks of localized headache, large multiloculated aneurysms, and a greater time lapse from the onset of symptoms to surgery compared with those patients without oculomotor palsy. Delay in treatment allowed further attacks to occur which increased the mortality rate and decreased the chance that the eye would recover. Eighteen people who had had a palsy before craniotomy two to 18 years previously were examined. In four (22%) the paralysis had recovered completely, 14 (78%) had greatly reduced oculomotor function, and nine (50%) showed aberrant regeneration of the nerve. Nine of 62 patients, seven of whom were seen, developed a palsy after craniotomy and in five the eye had returned to normal [12].

### 1947

A paper is concerned with 55 aneurysms out of a total of 158 that caused isolated paralysis of the oculomotor nerve [13].

## Case reports

### 2016

Binyamin et al report on two cases of resolution of third nerve palsy after flow diversion embolization of large and giant PCOM aneurysms without adjuvant coil placement. The resolution of third nerve palsy was not preceded by significant shrinkage of the aneurysmal sac on MRI. However, one patient showed resolution of T2-weighted signal abnormalities in the midbrain and mesial temporal lobe despite a similar size of the aneurysm. Therefore, flow diversion embolization of a PCOM aneurysm may resolve oculomotor nerve palsies through decreasing arterial pulsations against the nerve or midbrain [14)].

### 1975

A patient had pupillary sparing the absence of subarachnoid bleeding. A few similar cases have appeared in the literature. The mechanism of pupillary sparing appears to be based on the position of the parasympathetic pupilloconstrictor fibers within the subarachnoid portion of the third nerve and on the anatomic relationship between the third nerve and the junction of the carotid and posterior communicating arteries [15)].

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