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Comparison of surgical clipping and endovascular coiling in the treatment of oculomotor nerve palsy caused by posterior communicating artery aneurysm

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Abstract

Oculomotor nerve palsy (ONP) caused by posterior communicating aneurysm (PcomAA) is mainly treated by surgical clipping or endovascular coiling. However, there are still some controversies about which treatment method could provide the more beneficial prognosis. This study aimed to compare ONP recovery rate between surgical clipping and endovascular coiling in patients diagnosed as PcomAA combined with ONP, and explore the potential risk factors of ONP recovery.

The clinical data of 152 patients with ONP caused by PcomAA were retrospectively analyzed. Diameter of aneurysm, different treatment methods (surgical clipping or endovascular coiling), subarachnoid hemorrhage (SAH), degree of preoperative ONP, time from ONP onset to treatment, as well as degree of ONP symptom recovery were collected from medical records. All patients were followed up for at least 1 year.

One hundred twelve patients underwent surgical clipping and 40 patients received endovascular coiling. There were no significant differences in age, gender, aneurysm diameter, hypertension, dyslipidemia, time from ONP symptom onset to treatment, SAH, and preoperative ONP degree between the 2 groups (all $P > .05$). Time to complete or partial recovery was 86.7 ± 35.7 days for patients receiving surgical clipping and 132.6 ± 37.5 days for patients receiving endovascular coiling, respectively (Log rank test, $P < .001$). The recovery rate was 94.6% in the surgical clipping group and 65.0% in the endovascular coiling group. The difference between the two groups was statistically significant ($P < .001$). Postoperative ONP recovery in the surgical clipping group was significantly superior to that of patients in the endovascular coiling group (HR, 2.625; 95% CI: 1.423–4.841; $P = .002$). Time from ONP symptom onset to treatment exerted the obvious effect on the ONP prognosis (HR, 0.572; 95% CI: 0.384–0.852; $P = .006$). In addition, the ONP recovery in patients with SAH before surgery was also independently associated with ONP prognosis (HR, 1.276; 95% CI, 1.043–1.562; $P = .018$). There was no treatment-related death in either group, and postoperative complications were within the manageable range.

The recovery rate and recovery degree of ONP after surgical clipping was significantly better than that of endovascular coiling in PcomAA patients combined with ONP. The postoperative ONP recovery was associated with preoperative spontaneous SAH and time from ONP onset to treatment.

Abbreviations: CI = confidence interval, CTA = computed tomography angiography, DSA = digital subtraction angiography, HR = hazards ratio, ONP = oculomotor nerve palsy, PcomAA = posterior communicating artery aneurysm, SAH = subarachnoid hemorrhage, SD = standard deviation.

Keywords: endovascular coiling, oculomotor nerve palsy, posterior communicating aneurysm, surgical clipping

Editor: Danny Chu.

JL and CP have equal contribution on this work as the co-first authors.

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The authors have no funding and conflicts of interest to disclose.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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How to cite this article: Liu J, Peng C, Zhu G, Sheng C, Song S, Cheng Z, Zhu J. Comparison of surgical clipping and endovascular coiling in the treatment of oculomotor nerve palsy caused by posterior communicating artery aneurysm. Medicine 2020;99:47(e22969).

Received: 9 September 2019 / Received in final form: 14 September 2020 / Accepted: 29 September 2020

http://dx.doi.org/10.1097/MD.0000000000022969

1. Introduction

Intracranial aneurysm is the pouch like dilatation in the intracranial artery wall due to the multi-pathological causes. Intracranial aneurysms always occur in the bifurcations of the arteries that formed the circle of Willis, where the vessel wall is mostly affected by hemodynamic force.[1] Among them, posterior communicating artery aneurysms (PcomAA) account for about 45.9% of all intracranial aneurysms, which is a common type of intracranial aneurysms.[2] Spontaneous subarachnoid hemorrhage (SAH) and oculomotor nerve palsy (ONP) are common complications of PcomAA due to the adjacent anatomical relationships. The incidence of ONP symptoms is about 34% to 50%.[3] Patients usually have no obvious clinical symptoms or only mild headache before the aneurysms are ruptured. The size of PcomAA gradually increases with disease progression, and the function of the oculomotor nerve could be influenced by the pulsation of the aneurysm, resulting ONP occurrence. ONP could also be the first symptom of the patients and the main reason for hospital admission.

Recovery from ONP after treatment and the degree of ONP recovery often exert the profound impact on postoperative physical and psychological health of the patients. Currently, ONP caused by PcomAA is mainly treated by surgical clipping or endovascular coiling. However, there are still some controversies about which treatment method provide the most favourable patient outcome.[4] Symptoms of ONP induced by PcomAA could be relieved after early surgical clipping, which has become widely accepted.[5,6] Studies have shown that interventional embolization of ONP caused by PcomAA could also be performed effectively and safely in recent years.[7,8]

In this study, we retrospectively analyzed the clinical data of patients who were diagnosed as PcomAA combined with ONP, aimed to compare ONP recovery rate between surgical clipping and endovascular coiling and explore the potential risk factors of ONP recovery.

2. Materials and methods

2.1. Patients

This was a single-center retrospective cohort. The clinical data of patients with ONP caused by PcomAA treated in the Department of Neurosurgery, The Second Affiliated Hospital of Nanchang University between May 2014 and July 2018 were retrospectively analyzed. Inclusion criteria:

All PcomAA patients were confirmed by preoperative head computed tomography angiography (CTA) and/or digital subtraction angiography (DSA), and ipsilateral ONP symptoms had the definite correlation with PcomAA.

The patient received treatment of surgical clipping or endovascular coiling.

Exclusion criteria:

Tolosa-Hunt Syndrome;

Diabetic ophthalmoplegia;

Follow-up time less than 12 months.

The study was approved by the ethics committee of The Second Affiliated Hospital of Nanchang University and the informed consent of patient was waived by the ethics committee due to the retrospective nature of the study design.

2.2. Treatment method

Treatments were performed on the basis of standards recommended by the International Study of Unruptured Intracranial Aneurysms (ISUIA).[9] The difficulties and risks of surgical clipping and embolization coiling were evaluated by the neurointerventional physicians and neurosurgeons, as well as the characteristics of patients. All patients and/or their family members then chose the surgical strategy after explanation of the surgical indications and procedures. Treatment regimen of all patients with preoperative SAH consisted of 50 mg nimodipine intravenously twice daily, and 2000 mg tranexamic acid intravenously twice daily throughout the time from admission to surgery.

2.3. Data collection

The potential risk factors that may influence the ONP recovery, such as age, patients' comorbidities, different treatment methods, SAH before surgery, degree of preoperative ONP, time from ONP onset to treatment, aneurysm diameter, degree of ONP symptom recovery and time from treatment to obtain complete or partial recovery (days) were collected from medical records. Hypertension was defined as systolic blood pressure (SBP) $\geq$ 140 mmHg and/or diastolic blood pressure (DBP) $\geq$ 90 mmHg, or currently taking antihypertensive medication prescribed by a physician. Dyslipidemia was defined as a lipid metabolic disorder characterized by an increase or decrease in lipid fraction in plasma.[10]

2.4. Postoperative follow-up

All patients were performed clinical follow up after surgery. For patients receiving surgical clipping, cranial CTA was detected on the first day after surgery and before discharge to observe the condition of aneurysm clipping. For patients who received endovascular coiling treatment, DSA was conducted immediately after surgery. All patients were followed up at 1, 3, 6 and 12 months after surgery to understand the ONP recovery. CTA or DSA was performed after postoperative 6 and 12 months. The criteria for judging the preoperative ONP and postoperative recovery of ONP was listed as below:[11,12]

1) blepharoptosis;
2) external ophthalmoplegia;
3) diplopia;
4) dilated pupil and disappearance of direct and indirect light reflex in the affected side.

Complete ONP recovery was defined as complete resolution of all symptoms. ONP was defined as partial when one of the cited symptoms was absent. Partial ONP recovery was defined if one or more symptoms, even subtle subjective diplopia, remained.

2.5. Statistical analysis

The distribution of continuous data was assessed using the Kolmogorov-Smirnov test. Normally distributed continuous data were expressed as mean $\pm$ standard deviation (SD) and compared using the Student t test. Non-normally distributed data were presented as median (range) and analyzed with the Mann-Whitney U test. Categorical data were presented as proportion and analyzed with the chi-square test or Fisher exact test, as appropriate. Kaplan-Meier curve and Log-rank test were performed to assess postoperative recovery of ONP between surgical clipping group and endovascular coiling group. Cox proportional hazards model was further conducted to analyze the

potential risk factors (Enter method). A 2-sided $P < .05$ indicated statistical significance.

3. Results

3.1. Baseline data and aneurysm characteristics in patients receiving surgical clipping or endovascular coiling treatment

One hundred fifty-two patients with PcomAA and ONP symptoms who met the inclusive and exclusive criteria were finally analyzed. Among them, 112 patients underwent surgical clipping and 40 patients received endovascular coiling. All patients were followed up no less than 1 year. There were no statistically differences in age, gender, aneurysm diameter, hypertension or dyslipidemia, time from ONP symptom onset to treatment, SAH before surgery, and preoperative ONP degree between the 2 groups (all $P > .05$). The mean PcomAA diameter was 7.91 ± 1.23 mm in surgical clipping group and 7.65 ± 1.58 mm in endovascular coiling group, respectively. The difference did not show statistically significant between the 2 groups ($P = .347$). Fifty-six (50%) and 21 (52.5%) patients had SAH before surgery in the surgical clipping group and the endovascular coiling group, respectively, and no significant difference between the 2 groups was observed ($P = .855$). The baseline data between the 2 groups were comparable, as shown in Table 1.

3.2. Postoperative follow-up

There were 75 patients with complete ONP (57 cases in the surgical clipping group and 18 cases in the endovascular coiling group), 77 cases with incomplete ONP (55 cases in the surgical clipping group, and 22 cases in the endovascular coiling group) before treatment. No difference of preoperative ONP degree was observed between the two groups ($P = .522$). According to the recovery time after treatment, time to obtain complete or partial recovery was 86.7 ± 35.7 days for patients receiving surgical clipping and 132.6 ± 37.5 days for patients receiving endovascular coiling, respectively ($P < .001$). The recovery rate was 94.6% in surgical clipping group and 65.0% in endovascular coiling group ($P < .001$). During the postoperative 12-month follow up, 132 patients recovered from the preoperative ONP symptoms, including 108 cases of complete recovery (97 cases in surgical clipping group and 11 cases in endovascular coiling group), and 24 cases of partial recovery (9 cases in surgical clipping group and 15 cases in endovascular coiling group). The ONP did not recover in 20 cases (6 cases in surgical clipping group and 14 cases in endovascular coiling group). Postoperative ONP recovery was shown in Table 2. Survival analysis showed that the recovery rate of surgical clipping group and endovascular coiling group was similar during the initial recovery period. Since approximate 50 days after surgery, the cumulative recovery rate of surgical clipping group at any time was significantly higher than that of endovascular coiling group at any time point (Log rank test, $P < .001$). The result was shown in Figure 1.

3.3. Multivariate analyses

Based on the results of univariate analyses, time from ONP symptom onset to treatment (days) and treatment strategy had significant influences on the postoperative recovery of ONP induced by PcomAA, and the results were shown in Table 3. Potential risk factors, including age, gender, aneurysm diameter, combordity (hypertension or dyslipidemia), degree of preoperative complete ONP, SAH before surgery, time from ONP symptom onset to receiving treatment (days) and treatment

Table 1

Baseline data and aneurysm characteristics of patients in the surgical clipping group and endovascular embolization group.

Variables	Endovascular coiling group (n = 40)	Surgical clipping group (n = 112)	*P* value
Age (yr), mean ± SD	55.6 ± 7.9	56.2 ± 8.2	.684
Gender, male n (%)	20 (50.0%)	50 (44.6%)	.560
Aneurysm diameter (mm), mean ± SD	7.65 ± 1.58	7.91 ± 1.23	.347
Hypertension n (%)	21 (52.5%)	54 (48.2%)	.642
Dyslipidemia n (%)	17 (42.5%)	50 (44.6%)	.815
Time from ONP symptom onset to treatment (days), mean ± SD	11.27 ± 8.47	10.92 ± 9.61	.829
SAH n (%)	21 (52.5%)	56 (50%)	.855

ONP = oculomotor nerve palsy, SAH = subarachnoid hemorrhage, SD = standard deviation.

Table 2

Comparison of preoperative ONP and postoperative ONP recovery in the surgical clipping group and endovascular coiling group.

Variables	Endovascular coiling group (n = 40)	Surgical clipping group (n = 112)	*P* value
Preoperative ONP degree			
Complete ONP	18 (45%)	57 (50.9%)	.522
Incomplete ONP	22 (55%)	55 (49.1%)	
Post-treatment recovery time (days), mean ± SD	132.6 ± 37.5	86.7 ± 35.7	<.001
Recovery rate n (%)	65.0%	94.6%	<.001
Complete recovery	11	97	
Partial recovery	15	9	
No recovery	14	6	

ONP = oculomotor nerve palsy, SD = standard deviation.

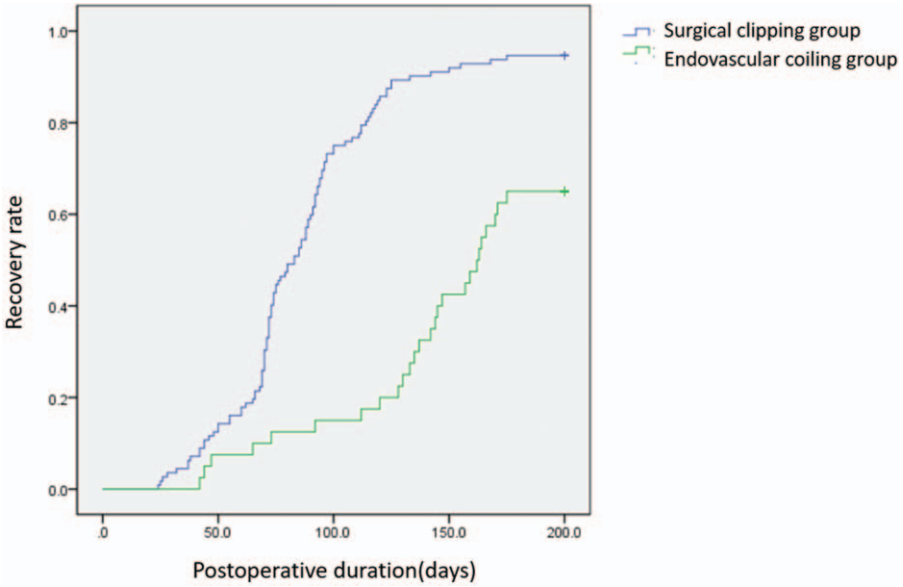

Figure 1. Comparison of postoperative ONP recovery rate between the surgical clipping and the endovascular coiling group.

strategies, were entered into the multivariate regression model. The results revealed that the postoperative ONP recovery in the surgical clipping group was significantly better than that in the endovascular coiling group (HR, 2.625; 95% confidence interval [CI], 1.423–4.841; $P=.002$). Time from ONP symptom onset to treatment exerted the obvious effect on the ONP prognosis (HR, 0.572; 95% CI: 0.384–0.852; $P=.006$), suggesting earlier intervention could provide better ONP recovery. In addition, the ONP recovery in patients combined with SAH before surgery was also independently associated with ONP prognosis (HR, 1.276; 95% CI, 1.043–1.562; $P=.018$).

3.4. Postoperative complications

There was no treatment-related death in either group, and postoperative complications were within the manageable range. In the surgical clipping group, there were 3 cases of obvious cerebral vasospasm, one case of partial hemiplegia, and seven cases of hydrocephalus after surgery. In the endovascular coiling group, there were 2 cases of obvious cerebral vasospasm, 1 case of aphasia, and 5 cases of hydrocephalus after surgery. After multiple lumbar punctures to release bloody cerebrospinal fluid, 1 patient of either group underwent ventriculo-peritoneal shunt, and the remaining complications were cured. The postoperative complications of the 2 groups were shown in Table 4.

Table 3

Cox regression analysis of influencing factors for postoperative ONP recovery.

Risk factors	Adjusted HR	95% CI	*P* value
Age (yr)	1.125	0.909–1.392	.278
Gender	0.731	0.420–1.271	.267
Aneurysm diameter (mm)	0.832	0.657–1.054	.127
Hypertension	0.473	0.164–1.361	.165
Dyslipidemia	0.567	0.242–1.327	.191
Preoperative complete ONP	1.365	0.788–2.365	.267
Preoperative SAH	1.276	1.043–1.562	.018
Time from ONP symptom onset to treatment (days)	0.572	0.384–0.852	.006
Treatment strategy (surgical clipping versus embolization coiling)	2.625	1.423–4.841	.002

ONP = oculomotor nerve palsy, HR = hazards ratio, CI = confidence interval.

4. Discussion

The results of this study revealed that postoperative ONP recovery in the surgical clipping group was significantly superior to that of patients in the endovascular coiling group. In addition, time from ONP symptom onset to treatment and patients combined with SAH before surgery were independently associated with ONP prognosis.

Endovascular coiling and surgical clipping had their own pros and cons for the treatment of PcomAA combined with ONP.[12–15] Although the trauma was relatively large, surgical clipping could effectively block the spread of continuous pulsation, reduce the compression of aneurysm to oculomotor nerve, prevent aneurysms rebleeding, and improve ONP symptoms.[16] Meanwhile, ONP could also be restored by endovascular coiling to some extent. The possible reason may be that the pulsation of the aneurysm could be weakened after interventional embolization, which also played a certain role in ONP recovery. Both thrombosis formation inside the aneurysm cavity and the

Table 4

Postoperative complications in the 2 groups of patients.

Complication	Endovascular coiling group (n = 40)	Surgical clipping group (n = 112)
Significant postoperative cerebral vasospasm	2	3
Partial hemiplegia	0	1
Aphasia	1	0
Hydrocephalus	5	7
Ventriculo-peritoneal shunt	1	1

shrinkage of the tumor wall weakened the pulsation of the aneurysm, thereby eliminating the damage of aneurysm pulsation on the oculomotor nerve.[13,15,17–19] Some scholars considered that aneurysm decompression during surgical clipping did not promote the recovery of ONP symptoms.[20] Even in PcomAA patients with residual aneurysm neck, the symptoms of ONP could be completely recovered.[21] However, other study indicated that interventional embolization could not eliminate the damage caused by the above-mentioned mechanical factor to the oculomotor nerve. On the contrary, excessive and too dense fillings of coils could enlarge the aneurysm size and aggravate the compression of the oculomotor nerve, which made the ONP symptoms worsen.[3]

Our study suggested surgical clipping was superior to endovascular coiling in the aspect of ONP improvement. Surgical clipping could not only clip the aneurysm to prevent it from bleeding again, but also remove the intracranial hematoma, and rinse the blood on the surface of brain tissue to prevent cerebral vasospasm induced by postoperative blood accumulation. However, intracranial hematoma in patients receiving endovascular coiling could only be absorbed by themselves. After intracranial aneurysm was clipped, blood could be completely blocked to flow into the tumor capsule. Nevertheless, the blood could still be extravasated through the interval space of the coils after interventional embolization, and continue to stimulate the oculomotor nerve, which may be due to inability to densely embolize the aneurysm. Surgical clipping could decompress the aneurysm directly, eliminate mechanical damage of the aneurysm to the oculomotor nerve, while the aneurysm may continue to expand after endovascular coiling. In our study, only the neck was clipped, the capsule was not forcibly separated from the oculomotor nerve, and the part of the aneurysm wall tightly attached to the nerve should be retained, in order to avoid the iatrogenic damage on the nerve during surgical clipping and achieve decompression effects, which was consistent with the opinions of previous studies.[6,14] Moreover, cerebral vasospasm was prone to be observed in patients receiving endovascular coiling, especially those who underwent treatment at early stay of bleeding, which may be associated with hemorrhage or intraoperative stimulus from guidewire, catheter and contrast agent in the vessels. In addition, endovascular coiling could only embolize aneurysm, but could not effectively reduce intracranial pressure for patients with a large amount of bleeding that led to intracranial hematoma or high intracranial pressure with the possibility of brain hernia.[12,22]

Our study showed that time from ONP symptom onset to treatment and treatment method had the significant effects on ONP recovery, and the results were in line with previous findings.[5,23] It was speculated that oculomotor nerve function could be in the status of reversible conduction block and neurapraxia for patients who were in the early stage of aneurysm rupture or partial ONP before treatment. At this time, the damage of oculomotor nerve was relatively mild, timely surgery could avoid the continuous stimulation and injury of the aneurysm, and oculomotor nerve function could also be improved quickly. Moreover, early surgical intervention could also prevent cerebral vasospasm caused by the aneurysm re-rupture and hemorrhage.

The results of this study indicated that ONP recovery rate was significantly higher in patients combined with SAH than that of those without ruptured aneurysms, which was similar with the results of previous studies.[21,24] It was speculated that for part of patients with ruptured aneurysm, the occurrence of ONP may be induced by hemorrhage stimulation and blood clot compression. With blood and blood clots absorbed, the stimulation and compression were relieved, then the recovery of ocular nerve function could be conducted smoothly. Moreover, patients with SAH were often admitted to hospital due to sudden and severe headaches. The treatments of these patients were prompt, the duration of oculomotor nerve stimulation by hemorrhage was short, and the possibility of ONP recovery was relatively high. However, in patients with unruptured aneurysms, ONP was often caused by space-occupying compression or pulsatile stimulation, and these patients often admitted due to symptoms of diplopia or blepharoptosis. Although there was no SAH stimulation, the disease course of these patients was generally longer. At this moment, the oculomotor axons had been degenerated, and the nerve conduction function had also undergone irreversible damage.

Generally, the degree of nerve damage could influence the recovery time of ONP to certain extent. For most patients, the symptoms of ONP began to recover within one month after surgery, and could be fully recovered after 3 to 4 months. Some scholars believed that oculomotor function usually could not be fully recovered, and some symptoms of ONP were still remained, if the disease course of patient's ONP recovery lasted for more than 1 year.[20] However, other study had indicated that these patients should be followed up for a longer period to determine whether the symptoms could be fully recovered. The ONP symptoms of a small population could be completely recovered within 2 years after surgery.[24]

Our retrospective study had several limitations worth noting. First, this was a single-center study and the patient population is limited. The results could not be generalized to other cohorts. Second, the selection of treatment strategy was based on surgeons' experience and preference, rather than randomization. Therefore, selection biases and information bias were unavoidable due to retrospective design. Third, direct nerve damage caused by hemorrhagic stimulation due to aneurysm rupture was one of the possible reasons of ONP, and patients with such trauma had the relatively low probability of obtaining complete recovery. However, it was difficult to distinguish the damage mechanisms from aneurysmal mass compression, aneurysm pulsation effect and aneurysm rupture impact. In addition, other anatomical characteristics of the aneurysms such as morphology and aneurysm neck were not assessed in our retrospective analysis. Fourth, due to majority of patients with unruptured aneurysms in our study, the hemorrhagic impact effect of aneurysm rupture should not be considered as the main cause of ONP.

5. Conclusion

The recovery rate and recovery degree of ONP after surgical clipping was superior to that of endovascular coiling in PcomAA patients with ONP. The postoperative ONP recovery was associated with preoperative spontaneous SAH and time from ONP onset to treatment. Early surgical treatment was particularly important for the recovery of oculomotor nerve function.

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References

[1] Texakalidis P, Sweid A, Mouchtouris N, et al. Aneurysm formation, growth and rupture: the biology and physics of cerebral aneurysms. World Neurosurg 2019.
[2] Golshani K, Ferrell A, Zomorodi A, et al. A review of the management of posterior communicating artery aneurysms in the modern era. Surg Neurol Int 2010;1:88.
[3] Hall S, Sadek AR, Dando A, et al. The resolution of oculomotor nerve palsy caused by unruptured posterior communicating artery aneurysms: a cohort study and narrative review. World Neurosurg 2017;107:581–7.
[4] Falk Delgado A, Andersson T, Falk Delgado A. Ruptured carotid-ophthalmic aneurysm treatment: a non-inferiority meta-analysis comparing endovascular coiling and surgical clipping. Br J Neurosurg V 31 2017;345–9.
[5] Tan H, Huang G, Zhang T, et al. A retrospective comparison of the influence of surgical clipping and endovascular embolization on recovery of oculomotor nerve palsy in patients with posterior communicating artery aneurysms. Neurosurgery 2015;76:687–94. discussion 694.
[6] McCracken DJ, Lovasik BP, McCracken CE, et al. Resolution of oculomotor nerve palsy secondary to posterior communicating artery aneurysms: comparison of clipping and coiling. Neurosurgery 2015;77: 931–9. discussion 939.
[7] Zu QQ, Liu XL, Wang B, et al. Recovery of oculomotor nerve palsy after endovascular treatment of ruptured posterior communicating artery aneurysm. Neuroradiology 2017;59:1165–70.
[8] Sheehan MJ, Dunne R, Thornton J, et al. Endovascular repair of posterior communicating artery aneurysms, associated with oculomotor nerve palsy: A review of nerve recovery. Interv Neuroradiol 2015;21:312–6.
[9] Connolly ES. International study of unruptured intracranial aneurysms. J Neurosurg 2014;121:1022–3.
[10] Nelson RH. Hyperlipidemia as a risk factor for cardiovascular disease. Prim Care 2013;40:195–211.
[11] Yanovitch T, Buckley E. Diagnosis and management of third nerve palsy. Curr Opin Ophthalmol 2007;18:373–8.
[12] Zheng F, Dong Y, Xia P, et al. Is clipping better than coiling in the treatment of patients with oculomotor nerve palsies induced by posterior communicating artery aneurysms? A systematic review and meta-analysis. Clin Neurol Neurosurg 2017;153:20–6.
[13] Ahn JY, Han IB, Yoon PH, et al. Clipping vs coiling of posterior communicating artery aneurysms with third nerve palsy. Neurology 2006;66:121–3.
[14] Chen PR, Amin-Hanjani S, Albuquerque FC, et al. Outcome of oculomotor nerve palsy from posterior communicating artery aneurysms: comparison of clipping and coiling. Neurosurgery 2006;58:1040–6. discussion 1040-1046.
[15] Patel K, Guilfoyle MR, Bulters DO, et al. Recovery of oculomotor nerve palsy secondary to posterior communicating artery aneurysms. Br J Neurosurg 2014;28:483–7.
[16] Gaberel T, Borha A, di Palma C, et al. Clipping versus coiling in the management of posterior communicating artery aneurysms with third nerve palsy: a systematic review and meta-analysis. World Neurosurg 2016;87:498–506. e494.
[17] Inamasu J, Nakamura Y, Saito R, et al. Early resolution of third nerve palsy following endovascular treatment of a posterior communicating artery aneurysm. J Neuroophthalmol 2002;22:12–4.
[18] Mavilio N, Pisani R, Rivano C, et al. Recovery of third nerve palsy after endovascular packing of internal carotid-posterior communicating artery aneurysms. Interv Neuroradiol 2000;6:203–9.
[19] Brigui M, Chauvet D, Clarençon F, et al. Recovery from oculomotor nerve palsy due to posterior communicating artery aneurysms: results after clipping versus coiling in a single-center series. Acta Neurochir 2014;156:879–84.
[20] Hanse MC, Gerrits MC, van Rooij WJ, et al. Recovery of posterior communicating artery aneurysm-induced oculomotor palsy after coiling. AJNR Am J Neuroradiol 2008;29:988–90.
[21] Mansour N, Kamel MH, Kelleher M, et al. Resolution of cranial nerve paresis after endovascular management of cerebral aneurysms. Surg Neurol 2007;68:500–4. discussion 504.
[22] Smith TR, Cote DJ, Dasenbrock HH, et al. Comparison of the efficacy and safety of endovascular coiling versus microsurgical clipping for unruptured middle cerebral artery aneurysms: a systematic review and meta-analysis. World Neurosurg 2015;84:942–53.
[23] Zhong W, Zhang J, Shen J, et al. Posterior communicating aneurysm with oculomotor nerve palsy: Predictors of nerve recovery. J Clin Neurosci 2019;59:62–7.
[24] Kassis SZ, Jouanneau E, Tahon FB, et al. Recovery of third nerve palsy after endovascular treatment of posterior communicating artery aneurysms. World Neurosurg 2010;73:11–6. discussion e12.