Factors Predicting the Oculomotor Nerve Palsy following Surgical Clipping of Distal Vertebrobasilar Aneurysms: A Single-Institution Experience

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Abstract

Background The aim of our study was to identify various clinical and radiologic factors that correlate with the oculomotor nerve palsy following clipping of distal vertebrobasilar aneurysms.

Methods A total of 48 patients with 51 aneurysms were included in this retrospective study. Patient's age, gender, size, location, and projection of the aneurysm, preoperative Hunt and Hess (H&H) grade, presence of subarachnoid hemorrhage (SAH), temporary clipping, preoperative third nerve palsy, and Glasgow Outcome Scale were included in the model for analysis.

Results A total of 15 patients (31.25%) developed oculomotor nerve palsy following clipping of basilar apex aneurysms. 38 patients (79.2%) presented with SAH and 35 patients (72.9%) had poor H&H grades at presentation. The size of the aneurysm ($p = 0.03$), preoperative H&H grade ($p = 0.04$), preoperative oculomotor nerve dysfunction ($p = 0.007$), and projection of an aneurysm ($p = 0.004$) had shown a significant correlation with the oculomotor nerve palsy. The size of the aneurysm ($p = 0.030$, odds ratio: 0.381; 95% confidence interval, 0.175–0.827) was an independent predictor of postoperative nerve dysfunction.

Conclusion The size of the aneurysm, clinical grade at presentation, and projection of the aneurysm correlated with the oculomotor nerve dysfunction following clipping. These clinical and radiologic parameters can be used to predict the oculomotor nerve outcome.

Keywords

- oculomotor nerve
- palsy
- clipping
- vertebrobasilar
- aneurysms

Introduction

Oculomotor nerve palsy (third cranial nerve palsy) is an accepted complication associated with the surgical clipping of distal vertebrobasilar aneurysms. Unfortunately, the incidence of oculomotor nerve palsy is not as well reported as the surgical experience of clipping posterior circulation aneurysms. This might be attributed to the fact that oculomotor nerve palsy is

not a life-threatening complication and most patients exhibit spontaneous recovery within several months.^{1–5} Even though the occurrence of oculomotor nerve palsy following clipping is not a serious complication, it is a cause of significant morbidity for the patients in the postoperative period and therefore cannot be neglected.^{6–8} The incidence of oculomotor nerve palsy following clipping of the distal basilar artery aneurysms ranges from 25 to 80% in the literature.^{1,4,8}

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With the introduction of endovascular coiling as an alternative treatment option for vertebrobasilar aneurysms, there has been a decline in the indications for open microsurgical clipping.^{9–12} Despite this decrease, it is important to understand the morbidities associated with the surgical clipping of distal basilar aneurysms. Understanding the incidence and the factors affecting these morbidities can help in planning and improving the surgical outcomes.

We present a retrospective review of 48 patients who underwent surgical clipping of distal vertebrobasilar aneurysms at our institution. The aim of our study was to identify various clinical and radiologic factors that correlate with the oculomotor nerve palsy following clipping of distal vertebrobasilar aneurysms. The incidence and factors affecting the onset of oculomotor nerve palsy obtained in this review are discussed and compared with the literature.

Clinical Materials and Methods

Clinical Data

We performed a retrospective review of distal vertebrobasilar aneurysms that underwent surgical clipping at our institution. This study was approved by the institutional review board at Louisiana State University and was in accordance with patient privacy rights protection as enforced by the Health Insurance Portability and Accountability Act.

A total of 75 patients with 87 vertebrobasilar aneurysms were identified from the database from 1995 to 2012. Of these, 48 patients with 51 distal basilar aneurysms who were clipped by the senior author (A.N.) were selected. Among them, 48 patients had basilar artery apex aneurysms, 2 patients had aneurysms of the superior cerebellar artery, and 1 had an aneurysm of the posterior cerebral artery. We had retrospectively reviewed the inpatient records, radiographic data, and operation notes of all the selected patients. All charts were studied for patient demographics, clinical grade at presentation, angiographic details, temporary clipping of the parent artery, postoperative complications, and follow-up. Specific data pertaining to the aneurysm such as size, location, and projection were also recorded from the database. The Hunt and Hess (H&H)¹³ scale was used in the preoperative evaluation of the patients and dichotomized into good (grades 1 and 2) and poor (grades 3, 4, and 5). Aneurysms were also classified according to their sizes as small (< 10 mm) and large ($\geq$ 10 mm) for analysis. Patients who underwent endovascular treatment or those with insufficient data were excluded from the study.

Outcome

Oculomotor nerve palsy was defined as an impaired motor function of the nerve following the procedure and at 6-month follow-up. Patients with nerve palsy due to other causes and those with isolated pupillary abnormalities were not considered in the analysis. In concordance with a previous study, good oculomotor nerve function was considered only after evaluation at 6-month follow-up.¹ The Glasgow Outcome Scale (GOS)¹⁴ was used to assess the clinical outcome at the last follow-up. Good outcome was defined as a GOS score of 4

and 5 and poor outcome as a GOS score of 1, 2, and 3. At 6-month follow-up, every patient had an evaluation of their outcome status, GOS, and oculomotor nerve function. The factors that might influence the incidence of postoperative oculomotor nerve palsy were included in the model for analysis (patient's age, gender, size, location and projection of the aneurysm, preoperative H&H grade, presence of subarachnoid hemorrhage [SAH], use of temporary clipping, preoperative third nerve palsy, and GOS at discharge and at 6-month follow-up).

Surgical Approach

The Pterional transsylvian approach was the most frequently used approach for clipping the basilar apex aneurysms at the normal anatomical level. Orbitozygomatic extension to the standard pterional approach was used in high basilar apex aneurysms.

Statistical Analysis

Statistical analysis was performed using SPSS v.20 (IBM, Inc., Chicago, Illinois, United States) and Microsoft Excel. The chi-square or Fisher test was used to compare the two groups on categorical variables. The Wilcoxon rank sum test was used to compare them on continuous or numeric variables. The independent *t* test was used to compare the means. Comparisons were considered significant only if the *p* value was <0.05. Univariate analysis was used to identify the covariates that might affect the outcome. Binary logistic regression analysis was used to identify the independent predictors of the postoperative oculomotor nerve palsy.

Results

A total of 48 patients with 51 distal vertebrobasilar aneurysms who were clipped by the senior author (A.N.) were included in this study.

Patient and Aneurysm Characteristics

The demographic data, clinical presentation, aneurysm characteristics (size, location, and projection), and outcomes are listed in ►Table 1. The mean age at the time of surgery was 50.18 ± 11.10 years (range: 26–77 years). Most patients were women ($n = 38$ [79.16%]). The mean size of the aneurysm was 13.58 ± 15.87 mm.

A total of 38 patients (79.2%) presented with SAH, and 35 patients (72.9%) had poor H&H grades at presentation. 94.11% ($n = 48$) of the aneurysms were located at the basilar apex, and 64.58% ($n = 31$) of these basilar apex aneurysms had an anterior projection on preoperative imaging. Four patients (8.3%) with basilar apex and none with superior cerebellar artery/posterior cerebral artery (SCA/PCA) aneurysms had the oculomotor nerve palsy at presentation, and all of these patients harbored large (> 10 mm) aneurysms. Intraoperative rupture was encountered in three cases (6.3%), and temporary clipping was used in 7 patients (14.6%). Overall, 68.8% ($n = 33$) of our patients had a favorable outcome (at 6-month follow-up) with an overall mortality rate of 8.3% ($n = 4$).

Table 1 Characteristics of the 48 patients with distal vertebrobasilar aneurysm

Characteristics	Present study (N = 48)
Mean age $\pm$ SD, y	50.18 $\pm$ 11.10
Female-to-male ratio	38:10
Mean size of the aneurysm, mm	13.58 $\pm$ 15.87
Hunt & Hess grade, n (%)	
Grades 1 and 2	13 (27.1)
Grades 3, 4, and 5	35 (72.9)
Location of the aneurysm (N = 51) (%)	
Basilar apex	48 (94.11)
SCA	2 (3.92)
PCA	1 (1.96)
Projection, basilar aneurysms only, (%)	
Anterior	31 (64.58)
Posterior	17 (35.41)
Preoperative SAH (%)	38 (79.2)
Preoperative third nerve palsy (%)	4 (8.3)
Intraoperative rupture (%)	3 (6.3)
Intraoperative temporary clipping done (%)	7 (14.6)
Postoperative third nerve palsy (%)	15 (31.25)
GOS at discharge (%)	
Good outcome	31 (64.58)
Poor outcome	17 (35.41)
GOS at 6-mo follow-up (%)	
A good outcome	33 (68.8)
Poor outcome	15 (31.3)

Abbreviations: GOS, Glasgow Outcome Scale; PCA, posterior cerebral artery; SAH, subarachnoid hemorrhage; SCA, superior cerebellar artery; SD, standard deviation.

Incidence of Oculomotor Nerve Palsy

Postoperative oculomotor nerve palsy was diagnosed in 15 patients (31.25%) with basilar apex aneurysms. None of the patients with SCA/PCA aneurysm exhibited this complication following clipping. Of 15 patients with oculomotor nerve palsy, complete paresis was noted in 6 (40%) and partial paresis in 8 patients (53.33%), whereas in 1 patient (6.66%) it was not possible to precisely evaluate the ocular movements because of a disturbance in consciousness after surgery.

Outcome

—**Fig. 1** and —**Tables 1** and **2** show the outcome of the study. Although the incidence of postoperative oculomotor nerve palsy was higher in elderly patients (> 60 years of age) as compared to younger patients, this difference did not reach statistical significance ($p = 0.183$). Similarly, gender did not

show a significant difference between the groups with and without oculomotor nerve palsy. Aneurysms with sizes > 10 mm were more likely to exhibit postoperative oculomotor nerve dysfunction ($p = 0.03$). Similarly, poor H&H grades and oculomotor nerve dysfunction at presentation were significantly correlated with adverse oculomotor nerve outcome ($p = 0.04$ and $p = 0.007$, respectively). A total of 58.8% of the basilar apex aneurysms with posterior projection exhibited postoperative oculomotor nerve palsy as compared to 16.12% of the aneurysms with an anterior projection ($p = 0.004$). Location of an aneurysm and presence of preoperative SAH did not show significant correlation with the postoperative oculomotor nerve palsy ($p > 0.05$). Similarly, intraoperative events such as aneurysmal rupture or application of the temporary clip were not associated with a higher incidence of nerve dysfunction ($p = 0.911$ and 0.183 , respectively). Finally, the GOS score also did not show significant correlation with the postoperative oculomotor nerve dysfunction ($p > 0.05$).

At 6-month follow-up, of 15 patients with postoperative oculomotor nerve palsy, complete recovery was seen in 11 patients (73.33%) (excluding the patient who died). Another three patients had persistent partial oculomotor nerve palsy at 12-month follow-up. These three patients had complete oculomotor nerve palsy with poor GOS in the immediate postoperative period and at 6-month follow-up.

Correlation of Factors and Regression Model

Of the various examined variables, the following factors showed a significant correlation with the postoperative oculomotor nerve palsy: size of the aneurysm ($p = 0.03$), preoperative H&H grade ($p = 0.04$), preoperative oculomotor nerve dysfunction ($p = 0.007$), and projection of an aneurysm (basilar apex) ($p = 0.004$). Binary logistic regression analysis was used to identify the independent predictors of postoperative oculomotor nerve palsy. Using this model, the size of the aneurysm ($p = 0.030$, odds ratio [OR]: 0.381, 95% confidence interval [CI]: 0.175–0.827) was identified as an independent predictor of postoperative nerve dysfunction.

Discussion

With the advances in microneurosurgical techniques, neuroanesthesia, innovative skull base approaches, and a better understanding of microsurgical anatomy, particularly brainstem perforators, there has been a significant improvement in the surgical outcomes of vertebrobasilar aneurysms.^{15–22} Considering the deeper location of these aneurysms and their intimate relationship to the brainstem perforators, surgical outcomes were measured in terms of mortality and major morbidities in most of the published series.^{16,20–23} Few studies had focused on oculomotor nerve outcome and factors associated with it following clipping of distal vertebrobasilar aneurysms.^{1,4,8}

Direct mechanical damage and vascular injury are the two possible mechanisms of postoperative oculomotor nerve palsy.^{1,4} Other rare causes of postoperative oculomotor nerve palsy are vasospasm, direct injury from a malpositioned clip,

Fig. 1 Bar chart showing the correlation between various clinicoradiologic factors and the oculomotor nerve palsy. Data are shown as percentage of patients who developed oculomotor nerve palsy following clipping in various categories. GOS, Glasgow Outcome Scale; H&H, Hunt and Hess grade; PCA, posterior cerebral artery; SCA, superior cerebellar artery.

and an inflammatory reaction to the muslin gauze wrapping resulting in a foreign body granuloma.^{24–26}

In our series, oculomotor nerve dysfunction occurred more frequently in patients with large basilar tip aneurysm compared with the smaller ones (58.33% versus 22.22%; $p = 0.03$), which is in concordance with the previously reported series.⁴ Horikoshi et al reported this complication in 47% of their patients with large distal basilar aneurysms (>10 mm) compared with 24% of patients with smaller aneurysms (< 10 mm).⁴ Contrary to this, Al-Khayat et al found no correlation between the oculomotor nerve dysfunction and size of the aneurysm; however, they attributed this to the smaller sample size of aneurysms >15 mm (14.8% of all distal basilar aneurysms).¹ Large size of an aneurysm is a well-accepted factor that increases surgical morbidity.^{8,20,27} The increased incidence of oculomotor nerve palsy following clipping of large distal basilar aneurysms can be attributed to the increased manipulation of nerve and injury to the brainstem perforators. In our study, the age of the patient did not show significant correlation with the postoperative oculomotor nerve palsy (26.82% versus 57.14%; $p = 0.183$). However, Al-Khayat et al and Horikoshi et al reported increased incidence of oculomotor nerve dysfunction in younger patients, which can be attributed to the narrow surgical corridor in these patients resulting in increased manipulation of the surrounding neurovascular structures.^{1,4}

In our series, complete resolution of oculomotor nerve palsy occurred in 73.33% of patients within 6 months. Al-Khayat et al and Solomon et al reported complete recovery of oculomotor nerve function in 86% and 95% of their patients within 9 and 6 months, respectively.^{1,28} Cruciger et al² described three groups of patients who developed oculomotor nerve palsy following clipping of basilar bifurcation aneurysms. Of 31 patients in his series, 67.7% ($n = 21$) patients developed oculomotor nerve palsy. Of these 21

patients, 9 (42.8%) developed unilateral palsy, 3 (14.28%) had bilateral palsy, and another 9 (42.8%) had oculomotor nerve palsy accompanied by hemiparesis. They concluded that the recovery from isolated oculomotor nerve palsy was excellent (eight of nine patients), whereas recovery was incomplete in patients with bilateral palsy or those with brainstem signs (half of the patients). Neuropraxia occurs due to mild to moderate mechanical trauma and usually resolves completely within 2 to 6 months. Whereas oculomotor axonotmesis that occurs due to severe injury to the nucleus/nerve fascicles within the brainstem or due to vascular injury often takes a longer time to recover or sometimes may not recover at all.^{1,2,29} Kerns et al showed that the reduction in the number of fibers (49%), the transverse area (62%), and the mean diameter of myelinated axons (23%) was responsible for the partial return of oculomotor nerve function following injury.⁷ Moreover there can be an aberrant regeneration of the oculomotor nerve following clipping of a basilar artery aneurysm that can result in ipsilateral abduction defects.⁶

Posteriorly projecting basilar apex aneurysms were more commonly associated with oculomotor nerve dysfunction in various published series.^{1,4} In our study, 58.8% of the posteriorly projecting aneurysms developed oculomotor nerve palsy as compared with 16.12% of the aneurysms with an anterior projection ($p < 0.05$). This may be attributed to the fact that posteriorly projecting basilar apex aneurysms were in close proximity to the oculomotor nerve as well as to the interpeduncular perforating vessels, which increases the probability of both mechanical and vascular injuries during dissection of the aneurysmal sac. In our series, oculomotor nerve palsy was encountered more frequently in patients with poor clinical grades (H&H) at presentation ($p = 0.04$), which is in concordance with the previously reported series.^{1,4} However the presence of SAH, application of a

Table 2 Correlated clinical characteristics of the patients with oculomotor nerve palsy following clipping of distal vertebrobasilar artery aneurysms

Characteristics	Patients without third nerve palsy (N = 33)	Patients with third nerve palsy (N = 15)	Significance p value
Age of the patient (%)			
≤ 60 y	30 (90.1)	11 (73.3)	0.183
> 60 y	3 (9.09)	4 (26.67)	
Female-to-male ratio n	25:8	13:2	0.472
Size of the aneurysm n (%)			
≤ 10 mm	28 (84.84)	8 (53.33)	0.031
> 10 mm	5 (15.15)	7 (46.66)	
Hunt & Hess grade n (%)			
Grades 1 and 2	12 (36.36)	1 (6.67)	0.040
Grades 3, 4, and 5	21 (63.63)	14 (93.33)	
Location of the aneurysm (%)			
Basilar apex	31 (93.93)	15 (100)	0.325
SCA/PCA	3 (9.09)	–	
Projection, Basilar aneurysms only (%)			
Anterior	26 (78.78)	5 (33.33)	0.004
Posterior	7 (21.21)	10 (66.67)	
Preoperative SAH (%)	24 (72.72)	14 (93.33)	0.140
Preoperative third nerve palsy (%)	–	4 (26.67)	0.007
Intraoperative rupture (%)	2 (6.06)	1 (6.67)	0.911
Intraoperative temporary clipping done (%)	3 (9.09)	4 (26.67)	0.183
GOS at discharge (%)			
Good outcome	24 (72.72)	7 (46.67)	0.098
Poor outcome	9 (27.27)	8 (53.33)	
GOS at 6-mo follow-up (%)			
Good outcome	25 (75.75)	8 (53.33)	0.180
Poor outcome	8 (24.24)	7 (46.67)	

Abbreviations: GOS, Glasgow Outcome Scale; PCA, posterior cerebral artery; SAH, subarachnoid hemorrhage; SCA, superior cerebellar artery.

temporary clip, and intraoperative rupture of the aneurysm did not correlate with the oculomotor nerve dysfunction in our study. Al-Khayat et al reported the higher incidence of this complication in patients with preoperative SAH and those who required the use of temporary clipping.¹ They postulated that the application of the temporary clip further reduces the already confined space around the aneurysm and thereby increases the chances of nerve injury.

Pterional approach alone or with the orbitozygomatic extension was used in all patients in our series. Heros et al reported the lower incidence of oculomotor nerve palsy using a combined pterional and anterior temporal approach as compared with the subtemporal approach.³ In subtemporal approach to the basilar tip aneurysms, Drake et al and Peerless et al proposed that the dissection of the arachnoid membrane around the oculomotor nerve should be limited to the medial side so that the nerve can be retracted laterally

with the uncus and its arachnoid.^{22,23} The vascularity of the oculomotor nerve must be preserved during dissection to prevent the ischemic injury.³⁰ Endovascular coil embolization is an established treatment option for patients with ruptured or unruptured basilar tip aneurysms.^{31–33} The incidence of oculomotor nerve palsy following endovascular embolization is reported as 20.8% with few studies focusing on this complication.^{5,34,35} The occurrence of oculomotor nerve palsy following endovascular treatment can be attributed either to the occlusion of the perforating vessels resulting in brainstem infarction or to the regrowth of the aneurysm.^{1,5} Bhatti et al reported worsening of the oculomotor nerve palsy 15 months following a coil embolization of the basilar artery aneurysm due to regrowth of the aneurysm.³⁴ Therefore, appropriate selection of either of the treatment modalities for patients with a distal basilar artery aneurysm is essential to avoid this complication.

Conclusion

Our study had shown that the oculomotor nerve palsy occurs in a significant number of patients undergoing clipping of distal basilar artery aneurysms. Various factors such as the size of the aneurysm, clinical grade (H&H) of the patient at presentation, and projection of the aneurysm correlated with the oculomotor nerve dysfunction following clipping. Of these, the size of the aneurysm was the most significant and independent predictor of oculomotor nerve palsy. A thorough understanding of the microsurgical anatomy and meticulous operative technique is essential to minimize this complication.

Because the incidence of this complication is based on the surgical technique and experience of the surgeon, our results cannot be extrapolated to other institutions. Therefore, each center should know the incidence of this complication in their set-up, and specific measures should be taken to avoid it. Moreover, surgeons can use these clinical and radiologic parameters to predict a patient's oculomotor nerve outcome, which can help with preoperative counseling and choosing treatment modalities.

Disclosures

The authors have no conflicts of interest or financial disclosures.

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