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Original Article

Intracranial aneurysms and abducent nerve palsy

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ABSTRACT

Background: Cranial nerve (CN) palsy may manifest as an initial presentation of intracranial aneurysms or due to the treatment. The literature reveals a paucity of studies addressing the involvement of the 6th CN in the presentation of cerebral aneurysms.

Methods: Clinical patient data, aneurysmal characteristics, and CN 6th palsy outcome were retrospectively reviewed and analyzed.

Results: Out of 1311 cases analyzed, a total of 12 cases were identified as having CN 6th palsy at the presentation. Eight out of the 12 were found in the unruptured aneurysm in the cavernous segment of the internal carotid artery (ICA). The other four cases of CN 6th palsy were found in association with ruptured aneurysms located exclusively at the posterior inferior cerebellar artery (PICA). For the full functional recovery of the CN 6th palsy, there was 50% documented full recovery in the eight cases of the unruptured cavernous ICA aneurysm. On the other hand, all four patients with ruptured PICA aneurysms have a full recovery of CN 6th palsy. The duration for recovery for CN palsy ranges from 1 to 5 months.

Conclusion: The association between intracranial aneurysms and CN 6th palsy at presentation may suggest distinct patterns related to aneurysmal location and size. The abducent nerve palsy can be linked to unruptured cavernous ICA and ruptured PICA aneurysms. The recovery of CN 6th palsy may be influenced by aneurysm size, rupture status, location, and treatment modality.

Keywords: Abducent nerve, Cranial nerve palsy, Intracranial aneurysms

INTRODUCTION

Cranial nerve (CN) palsy can be observed in patients with intracranial aneurysms as an initial disease manifestation or a result of the treatment. As a presenting finding, CN palsy can be a component of the constellation of symptoms encountered in patients with cerebral aneurysms.[5] They can be detected at the initial assessment of patients with either unruptured or ruptured intracranial aneurysms. In unruptured aneurysms, CN palsy may denote an underlying aneurysmal growth that stretches the surrounding neuronal tissues and is not uncommon to be the sole finding in this set of patients. On the other hand, in ruptured aneurysms, CN palsy can result directly from the hemorrhage or indirectly from increased intracranial pressure (ICP). The CN dysfunction can be attributed to either affection of the nerve, its blood supply, or related brainstem structures.[7] This loss of function can be partial or complete, temporary, or

permanent, and unilateral or bilateral, based on the location, size, rupture status, and other characteristics related to the involved aneurysm.[7]

The anatomical proximity forms the background for the association between a CN palsy and an aneurysm. Classic examples include oculomotor nerve palsy associated with a posterior communicating artery aneurysm.[2] The abducent nerve dysfunction can be attributed to various etiologies, including infarction, tumors, demyelination, infections (specifically tuberculosis), idiopathic intracranial hypertension (HTN), Gradenigo syndrome, Tolosa-Hunt syndrome, clivus tumors, and aneurysms are among the described causes in literature.[4,6,16,18]

The abducent nerve follows a peculiar intracranial pathway and is susceptible to being affected by an enlarged aneurysm along the distance from the brainstem to the orbit. There are potentially vulnerable compression/entrapment points for the abducent near the petrous apex posteriorly and in the lateral wall of the cavernous sinus anteriorly. Thus, CN 6th palsy can be linked to aneurysms originating from arteries with close anatomical proximity.[1] Existing literature reveals a paucity of studies addressing the involvement of the 6th CN in cerebral aneurysms. Here, we share our experience with 6th nerve palsy as a presenting feature for intracranial aneurysms, along with an analysis of associated aneurysmal characteristics and the CN's follow-up and functional outcome.

MATERIALS AND METHODS

A retrospective chart review was conducted at the University of Pittsburgh Medical Center from June 2016 to July 2023, with an emphasis on CN 6th palsy at presentation while excluding cases of postprocedural CN 6th palsy. The study only included newly diagnosed aneurysms ruptured or unruptured aneurysms undergoing initial treatment; previously treated/retreated aneurysms were excluded from the study. Patients with mycotic aneurysms were also excluded from the study. The study period, inclusion, and exclusion criteria were defined as per this objective. The following variables were extracted and analyzed: demographics (age and sex), clinical characteristics (ruptured status, HTN, diabetes mellitus [DM], and CN 6th palsy), aneurysm characteristics (location, size categorized as small: 0–4.9 mm, medium: 5–9.9 mm, large: 10–24.9 mm, and giant: 25+ mm), for ruptured aneurysms (Hunt and Hess score categorized as score 1: minimal symptoms, score 2: moderate-to-severe headaches, score 3: confusion or mild neurological issues, score 4: stupor and possible weakness, and score 5: deep coma, and subarachnoid hemorrhage [SAH] severity score characterized as score 0: no blood, score 1: small amount of blood, score 2: moderate amount of blood, score 3: completely filled with blood) treatment modalities (clipping, coiling, pipeline embolization device [PED], and other treatments as applicable), and outcome variables for CN resolution status, follow-up duration of the CN 6th palsy, and functional outcome of the patient modified Rankin scale (mRS) categorized as a good outcome for the scores of 0–2, while a poor outcome for 3–6 scores. Data were collected from electronic health records, and the variables were included in the study. Any discrepancies in the data were resolved through a thorough review of the medical records. Retrospective data collection was approved by the University of Pittsburgh's Institutional Review Board. All patients underwent standard surgical consent.

Descriptive statistics were first employed to summarize the continuous and categorical variables, including frequencies and percentages for sex, DM, ruptured status, CN 6th palsy, location of the aneurysm, size, and treatment modalities, as well as the mean and standard deviation (SD) for age. Subsequently, CN 6th palsy and aneurysm location are associated with categorical variables such as sex, ruptured status, DM, mRS, and aneurysm Size were explored using Chi-square tests. Finally, to investigate the factors associated with CN 6th palsy in aneurysm patients, univariate logistic regression analyses were performed. The predictors analyzed were sex, ruptured status, DM, aneurysm location, size, and treatment. $P < 0.05$ is considered significant. IBM Statistical Package for the Social Sciences Statistics version 25 software was used in the analysis.

RESULTS

In this study, 1311 patients with previously untreated intracranial aneurysms who were treated at the University of Pittsburgh Medical Center from June 2016 to July 2023 were included in the study. The general patient characteristics were as follows: 564 cases accounted for ruptured (43.0%), and 747 cases for unruptured aneurysms (57.0%). The mean age was 58.6 years (SD = 13.1), with 71.6% females. The most frequently identified aneurysm location was the anterior communicating artery (Acom) at 23.0%. The cavernous internal carotid artery (ICA) represented 1.7% (22 cases), and the posterior inferior cerebellar artery (PICA) represented 5.1% (67 cases). About 46.1% were treated with microsurgical clipping, and 53.9% had neuroendovascular treatment.

In our study, CN 6th palsy was observed in 0.9% (12 cases) of the total 1311 cases. Further, analysis of the abducent nerve revealed that in the unruptured category, all the aneurysms were located in the cavernous ICA. Among the 22 cases of treated cavernous ICA aneurysms, 8 individuals (36.4%) presented with CN 6th palsy, while 14 patients (63.6%) did not. Aneurysm size displayed differences, with the CN 6th palsy group having a higher proportion of giant-sized aneurysms in 3 cases (13.6%) compared to the CN 6th non-palsy group in 2 cases (9.1%). Conversely, the CN 6th non-palsy group had a higher number of large-sized aneurysms, with

8 cases (36.4%), compared to the CN 6th palsy group with 2 cases (9.1%). Furthermore, in the analysis of this group, the aneurysm size showed significant associations with CN 6th palsy ($P < 0.001$) [Table 1].

The analysis of abducent nerve palsy at presentation revealed that in the ruptured category, all the aneurysms were PICA (four cases). Those four cases represent 6.0% of total PICA (67 cases) and 6.8% of ruptured PICA patients. For aneurysm size, in the CN 6th palsy group, small-sized aneurysms were found in three cases, and one patient had a large-sized aneurysm. In our statistical analysis of the PICA aneurysm group, no statistically significant associations were found regarding the factors in the study [Table 1].

Logistic regression confirmed that the presence of a cavernous ICA aneurysm is associated with an increased likelihood of abducent palsy (odd ratio [OR] = 183.4, P <0.001), as did PICA aneurysm (OR = 9.8, $P < 0.001$). In addition, aneurysmal size (OR = 2.8, $P = 0.005$) significantly correlated with abducent nerve palsy. However, factors such as sex (OR = 4.3, $P = 0.157$), rupture status (OR = 0.6, $P = 0.499$), and the presence of DM (OR = 0.000, $P = 0.996$) did not yield statistically significant correlations with CN 6th palsy [Table 2].

For treatment strategies and outcomes, in the ruptured/PICA cases, three out of four patients underwent clipping, while one patient underwent coiling. Resolution of the CN 6th palsy was seen within 1–3 months in all ruptured cases with a 2-month average recovery time. All had good functional outcomes (mRS 0–2) at follow-up. In unruptured/cavernous ICA cases, all eight patients underwent coiling and PED placement. A median follow-up period was 15.5 months, ranging from 4 to 24 months. The mRS of this group revealed that six patients had good outcomes, and two had poor ones. This cavernous ICA group has a 50.0% resolution rate (four cases) with a 2.7-month average recovery time and an equal number of unresolved instances (four cases) of CN 6th palsy.

Table 1: The clinical characteristics of CN 6th palsy at the presentation in cases of unruptured (Cavernous ICA) and ruptured (PICA) aneurysm.

Parameters	Unruptured/Cavernous ICA aneurysm cases (*n*=22)		*P*-value*	Ruptured/PICA aneurysm cases (*n*=67)		*P*-value*
	With CN 6th palsy (*n*=8) (%)	Without CN 6th palsy (*n*=14) (%)		With CN 6th palsy (*n*=4) (%)	Without CN 6th palsy (*n*=63) (%)	
Age						
21–40	-	2 (14.3)	0.42	-	4 (6.5)	0.49
41–60	-	3 (21.4)		2 (50)	33 (52.4)	
61–80	7 (87.5)	8 (57.4)		2 (50)	22 (34.9)	
81–100	1 (12.5)	1 (7.1)		-	4 (6.5)	
Sex						
Male		3 (21.4)	0.49	1 (25)	18 (28.6)	0.90
Female	8 (100)	11 (78.6)		3 (75)	45 (71.4)	
Diabetes mellitus						
Absent	8 (100)	14 (100)	1.00	4 (100)	55 (87.3)	0.35
Present	-	-		-	8 (12.7)	
Size*						
Small	-	1 (7.1)	**<0.001**	3 (75)	22 (34.9)	0.60
Medium	3 (37.5)	3 (21.4)		-	34 (54)	
Large	2 (25)	8 (57.4)		1 (25)	7 (11.1)	
Giant	3 (37.5)	2 (14.3)		-	-	
Treatment						
Clipping	-	1 (7.1)	**0.002**	3 (75)	30 (47.6)	**0.03**
Coiling	-	3 (21.4)		1 (25)	29 (46)	
Coil+PED	8 (100)	10 (71.4)		-	4 (6.5)	
mRS						
0	2 (25)	11 (78.6)	0.16	3 (75)	58 (92.1)	0.78
1	3 (37.5)	1 (7.1)		1 (25)	-	
2	1 (12.5)	2 (14.3)		-	-	
4	1 (12.5)	-		-	-	
6	1 (12.5)	-		-	5 (7.9)	

Size categorized as small: 0–4.9 mm, medium: 5–9.9 mm, large: 10–24.9 mm, and giant: 25+mm. CN: Cranial nerve, ICA: Internal carotid artery, PED: Pipeline embolization device, PICA: Posterior inferior cerebellar artery, mRS: modified Rankin scale. **P*-value based on the Fisher test is significant If $P \leq 0.05$ (Bold)

In resolved cases, all associated aneurysms were classified as medium or large, with two cases falling into each category. Conversely, among the unresolved cases, three of these cases were with giant-sized aneurysms, while only one showed an aneurysm of medium size. For aneurysms, a similar distribution of outcomes was observed in unresolved cases [Table 3]. All CN 6th palsy cases were ipsilateral except for one bilateral instance.

Table 2: Risk factors associated with abducent nerve palsy at the presentation in the setting of intracranial aneurysms.

Predictors	Univariant OR (95% CI)	*P*-value*
Sex	4.3 (0.5–34.1)	0.16
Rupture status	0.6 (0.1–2.2)	0.50
Diabetes mellitus	0.0 (0.0–1)	1.00
PICA	9.8 (2.8–33.4)	**<0.001**
Cavernous	183.4 (49.4–680.2)	**<0.001**
Size	2.8 (1.3–5.8)	**0.005**
Treatment modality	2.1 (1.3–3.3)	0.002
mRS	1.3 (1.0–1.7)	0.02

PICA: Posterior inferior cerebellar artery, mRS: modified Rankin Scale, OR: Odd ratio. **P*-value based on the Fisher test is significant If $P \leq 0.05$. CI: Confidence interval

DISCUSSION

The abducent nerve emerges from the pontomedullary junction and proceeds to enter the prepontine cistern. It subsequently follows a relatively vertical course along the dorsal surface of the clivus adjacent to the petrous apex. Continuing its course, the nerve gains access to the cavernous sinus through Dorello's canal, eventually entering the orbit through the superior orbital fissure.[9] Throughout its path, the abducent nerve maintains anatomical relations with the anterior inferior cerebellar artery, the dorsal surface of the clivus, Dorello's canal, and the cavernous sinus. Its cisternal course over the clivus and the sharp ridges of the petrous temporal bone render it vulnerable to injury from various traumatic forces or compressive vascular pathologies.[6] A range of aneurysms, including those affecting the PICA, ICA, Acom, posterior communicating artery, and middle cerebral artery aneurysms, hold the potential to impact the abducent nerve function.[1,3,5,10]

According to the findings of our study, a noteworthy association has been established between the abducent nerve and ruptured PICA aneurysms. While, in the context of

Table 3: Patients with intracranial aneurysm and abducent nerve palsy at presentation with clinical, treatment, and follow-up data.

Case No.	Age (years), Sex	Rupture Status	Location	Size (mm)*	Admission H&H	SAH (0–2)	CN 6th palsy laterality	Treatment	CN functional outcome (months)	mRS
1	55, F	+	L. PICA	Small (3)	3	2	Ipsilateral	Clipping	Completely resolved (2)	0
2	65, F	+	R. PICA	Small (2)	3	2	Bilateral	Clipping	Completely resolved (3)	1
3	63, M	+	R. PICA	Small (2)	2	2	Ipsilateral	Clipping	Completely resolved (2)	0
4	56, F	+	R. PICA	Large (10)	3	2	Ipsilateral	Coiling	Completely resolved (1)	0
5	70, F	_	L. Cavernous ICA	Medium (9)	-	-	Ipsilateral	Coiling, PED	Unresolved (19)	1
6	65, F	_	R Cavernous ICA	Medium (5)	-	-	Ipsilateral	Coiling, PED	Completely resolved (3)	4
7	82, F	_	R Cavernous ICA	Large (18)	-	-	Ipsilateral	Coiling, PED	Completely resolved (1)	1
8	64, F	_	L. Cavernous ICA	Giant (25)	-	-	Ipsilateral	Coiling, PED	Unresolved (15)	1
9	71, F	_	L. Cavernous ICA	Giant (30)	-	-	Ipsilateral	Coiling, PED	Unresolved (24)	6
10	75, F	_	L. Cavernous ICA	Large (14)	-	-	Ipsilateral	Coiling, PED	Completely resolved (5)	0
11	79, F	_	L. Cavernous ICA	Giant (25)	-	-	Ipsilateral	Coiling, PED	Unresolved (4)	2
12	65, F	_	R. Cavernous ICA	Medium (9)	-	-	Ipsilateral	Coiling, PED	Completely resolved (2)	0

*Size categorized as small: 0–4.9 mm, medium: 5–9.9 mm, large: 10–24.9 mm, and giant: 25+mm. CN: Cranial nerve, ICA: Internal Carotid artery, F: Female, H&H: Hunt and Hess, M: Male, PED: Pipeline embolization device, PICA: Posterior inferior cerebellar artery, mRS: modified Rankin Scale, L: Left, R: Right

unruptured aneurysms, a statistically significant correlation has been identified between the abducent nerve palsy and factors such as the cavernous location, and dome size. An anatomical understanding of the aneurysm locations in our study that presented with 6th CN palsy is described in [Figure 1].

Correlation between abducent nerve palsy and unruptured intracranial aneurysms

In our study, eight cases with abducent nerve palsy were found to be associated with unruptured cavernous ICA aneurysms with 75% related to giant dome size. In a comparable study conducted by Durner *et al*., a total of 36 unruptured giant cavernous ICA aneurysms were examined within a cohort of 34 patients, 25 individuals had CN dysfunction. The findings indicated that abducent nerve palsy manifested in 84% of the patients. Among these cases, 6th nerve palsy exclusively presented in 44% of the patients as the sole CN impairment.[3] Koskela *et al*. reported potential locations for unruptured aneurysms with abducent nerve palsy at presentation, including Acom, posterior communicating artery, and middle cerebral artery aneurysm, with a single case for each location.[10] However, no other significant report is suggestive of a more consistent correlation and our series had no unruptured aneurysms in a location other than cavernous ICA.

The potential mechanisms by which an aneurysm can result in dysfunction of the abducent nerve encompass direct compression due to the nerve's proximity to the ICA within the cavernous sinus. In addition, occlusion of the feeding vessels supplying the structures of the cavernous sinus may occur due to either compression by the aneurysm or thrombosis and may cause an acute simultaneous ischemic lesion of the CNs.[13]

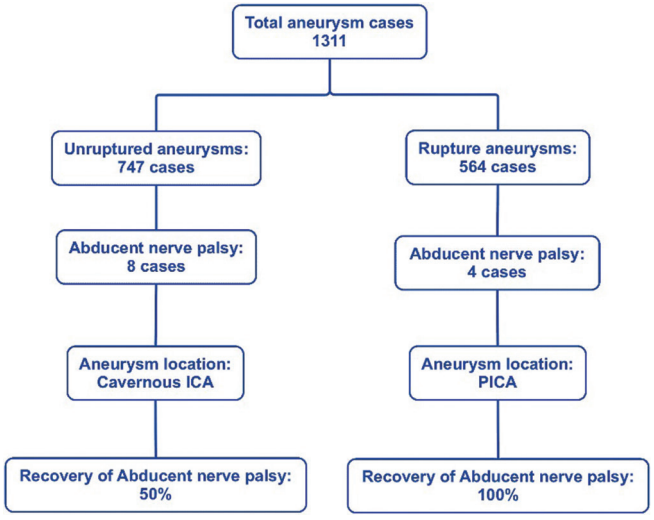

Figure 1: Summary of cases with abducent nerve palsy at presentation in the setting of intracranial aneurysms. ICA: Internal carotid artery, PICA: Posterior inferior cerebellar artery.

Correlation between abducent nerve palsy and ruptured intracranial aneurysms

Four cases in the ruptured aneurysm category were found to have CN 6th palsy at presentation, with all located at the PICA junction with vertebral artery (VA). This aligns with the work of Burkhardt *et al*., who investigated 13 patients with 6th nerve palsy from a broader cohort of 106 patients displaying PICA aneurysms.[1] The researchers suggest the abducent nerve palsy as a potential localizing sign for a ruptured PICA aneurysm. However, considering that the sample size could pose limitations to drawing definitive conclusions. Our study presents a comparable observation involving 93 PICA aneurysms and four instances of abducent nerve palsy. However, we remain cautious in reaching definitive inferences regarding a particular localizing sign.

Due to the close anatomical proximity of the abducent nerve's origin at the pontomedullary junction to that of the PICA from the VA, a PICA aneurysm holds the potential to serve as a compressive element affecting the 6th CN at the root exit zone.[1] We notice a variation in the PICA take-off from the VA and a unique horizontal segment of VA from the origin of PICA to union with the other VA to form the basilar artery [Figures 2 and 3]. However, this anatomical configuration cannot be addressed as a consistent anatomical variation due to the limited number of patients. Within the context of rupture status, the subsequent SAH can contribute to the elevation of ICP, thereby indirectly influencing the functionality of the CN.[7] Furthermore, direct contact between the blood within the subarachnoid space and the CN may initiate a series of reactions, culminating in secondary spasms and compromising CN function.[5,7,12]

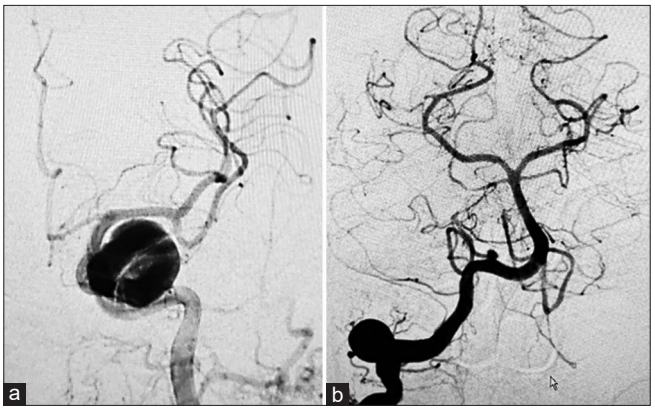

Figure 2: Example of catheter cerebral angiography for intracranial aneurysm cases that were presented with abducent nerve palsy. (a) Left cavernous internal carotid artery aneurysm. (b) Right posterior inferior cerebellar artery aneurysm.

Laterality of abducent nerve palsy associated with intracranial aneurysms

The distinction between bilateral and unilateral occurrences of abducent palsy hinges on a multitude of factors. Unilateral palsy primarily arises due to direct compression of the aneurysm on the neuronal fibers, with the resulting condition being either ipsilateral or contralateral to the affected side and this can be applied regardless of the rupture status of the aneurysm.[1] For ruptured aneurysms, bilateral abducent palsy has been associated with elevated ICP. However, certain reports propose that hemorrhage can also precipitate this condition and may be a major cause in ruptured cases.[7] Furthermore, the specific aneurysms that exhibit dome projection and size characteristics may lead to the presentation of bilateral 6th nerve palsy due to direct compression, thereby constituting a third plausible mechanism.[1] However, bilateral abducent nerve involvement is very rare in the setting of intracranial aneurysms as compared to traumatic brain injury for example.[15] Within the context of our study, it was observed that all abducent palsies displayed an ipsilateral relationship to the aneurysm, except for a singular case involving a ruptured PICA aneurysm where bilateral palsy manifested.

Functional recovery of abducent nerve palsy in the setting of intracranial aneurysms

CNs typically require a certain duration to regain their functionality following the treatment of the underlying cause. Surgical or endovascular interventions targeted at treating caused aneurysm, offer the potential for recovery of nerve function. Nonetheless, the scope and speed of recuperation can exhibit significant variability among patients. The literature showed quite a variability in the period of regaining function of the CN palsy in the setting of intracranial aneurysms. This is because the factors contributing to the nature of recovery encompass the degree of nerve compression, the duration of symptoms before treatment, and the efficacy of the intervention in alleviating aneurysm-induced pressure on the nerve.[8,11,17]

The general anticipation of the functional recovery in aneurysm-induced-CN palsy is controversial with the actual impact of treatment modality on the CN palsy outcome is difficult to be precisely determined using a small sample size. While some patients may encounter full or nearly complete restoration of function, others could be left with residual deficits.[5,11] Our focus in this study is on the 6th CN palsy at the presentation, as the CN palsy after treatment may reflect a potential iatrogenic nerve injury related to the endovascular procedure or microsurgical management.[14] Notably, there exists no established range of recovery duration for this condition within the existing literature. However, in the case of CN 6th palsy, the regression of nerve damage is notably high (89%) and tends to occur with remarkable rapidity within a month in 75% of patients.[12] The duration of recovery spans from 3 days to 3 months, as documented in numerous cases described in the literature.[1,5,8,12] In our study, 100% of ruptured aneurysms patients are PICA aneurysms, all of which exhibited a resolution of 6th nerve palsy. Conversely, only 50% of patients with unruptured/cavernous ICA aneurysms experienced resolution. The cases of resolution observed in ruptured/PICA aneurysms spanned 1–3 months. While within the context of unruptured/cavernous ICA aneurysms, the resolved cases exhibited a recovery period ranging from 4 to 24 months. These findings are consistent with the broader understanding of CN functional recovery.

The variation observed in CN recovery between ruptured and unruptured cases could be attributed to prolonged compression by the aneurysm within confined spaces, rendering the CN less prone to recovery. This notion is substantiated by the size of the aneurysms in unresolved cases, with 75% of them being classified as giant aneurysms. As previously elucidated, the plausible mechanism underlying this phenomenon involves the occlusion of feeding vessels supplying the abducent nerve, thereby inducing acute ischemic lesions within the nerve simultaneously. Furthermore, the type of treatment can potentially influence the functional regaining of CN palsy.

Treatment options may directly impact the recovery of 6th nerve palsy; it is noteworthy that surgical clipping offers prompt relief from compression, regardless of the specific surgical technique employed, whether involving clipping alone or in conjunction with aneurysm sac decompression. Endovascular coiling may not alleviate the entirety of the mass effect, some studies mentioned that the non-compressible coil mass might potentially contribute to an increase in pressure. However, recent studies mentioned that the mass effect remains after endovascular packing, and CN palsies improve comparably to the recovery observed after clipping.[11,17]

A study focused on the long-term functional outcomes of CN palsy underscores the observation that endovascular treatment of cavernous ICA aneurysms tends to render the abducent palsy less likely to resolve.[11] In our study, within the subset of four ruptured cases, three cases underwent clipping, and one was treated with endovascular coiling, leading to the complete resolution of abducent palsy. Among the unruptured cases, eight were subjected to coiling and a PED; this resulted in 50% of instances where resolved, and the other 50% of instances remained unresolved during a follow-up period ranging from 4 to 24 months. CN 6th supplies a single muscle, which results in a limitation in providing detailed assessments of the extent of palsy. Moreover, the

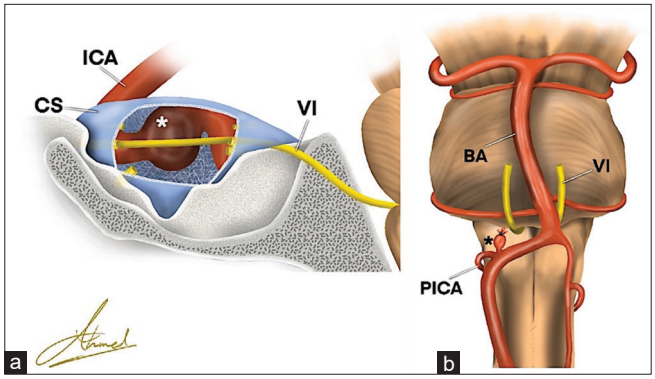

Figure 3: Illustrative depiction of the abducent nerve relation to various aneurysms: (a) cavernous ICA aneurysm (white asterisk) and (b) PICA aneurysm (black asterisk). BA: Basilar artery, VI: Abducent nerve, PICA: Posterior inferior cerebellar artery, ICA: Internal carotid artery, CS: Cavernous sinus. Illustration prepared by Ahmed Muthana and courtesy of Samer Hoz.

recovery associated with CN 6th palsy is more qualitative than quantitative when compared to other CNs, such as the oculomotor nerve, where both partial and complete palsy are distinctly evident and can be assessed in various clinical settings. In addition, CN 6th palsy resulting from increased ICP presents a challenging entity due to the frequent overlap with aneurysmal compression or hemorrhagic causes, making them potential underlying causes.

In managing persistent symptoms after a significant duration, usually more than 3 months, and ameliorating functional outcomes, therapeutic approaches such as eye patching, prismatic glasses or botulinum toxin injections can be employed.[17]

Based on the observations, CN 6th palsy at the time of presentation is linked to cavernous ICA aneurysms in unruptured cases, while in ruptured cases, it is associated with PICA aneurysms. A discernible pattern emerges, indicating a greater likelihood of CN function recovery in ruptured aneurysm cases than in unruptured ones.

While this study effectively explores the link between CN palsy and aneurysms, the multifactorial etiology of CN palsy could not be exhaustively addressed. This condition often arises from a complex interplay of anatomical, physiological, and potentially genetic factors, extending beyond aneurysm presence alone.

The relationship between CN 6th palsy and treatment or mRS cannot be directly established, as these CNs were assessed at the time of presentation and could not be tested within the scope of our study regarding treatment and outcomes. This applies even if there is statistical significance; it does not necessarily imply strong correlations. Instead, it serves as an indicator of a correlation, irrespective of its level of significance.

Limitations

This study possesses certain limitations that warrant consideration when interpreting the results. The sample size comprised 12 patients with CN 6th palsy, in the form of four patients out of 67 with PICA and eight patients out of 22 with cavernous ICA aneurysms, which, while substantial, might not fully represent the diverse spectrum of patient and aneurysm characteristics present in the broader population. Consequently, the generalizability of the findings to larger and more varied patient groups could be limited.

CONCLUSION

There is a significant association between the abducent nerve and intracranial aneurysms, illuminating distinct patterns concerning the location and size of the aneurysm. A robust connection has been observed between the abducent nerve and ruptured PICA aneurysms. In addition, within the realm of unruptured aneurysms, there are compelling correlations, particularly about the cavernous location and size of the aneurysm.

Disclosures

The data and analytic code used in this study are available from the corresponding authors upon reasonable request.

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Ethical approval

The research/study was approved by the Institutional Review Board at UPMC, number 2210096, dated 2023.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

Dr. Gross is a consultant for Medtronic, Stryker, and MicroVention.

Dr. Nogueira reports consulting fees for advisory roles with Stryker Neurovascular, Cerenovus, Medtronic, Phenox, Anaconda, Genentech, Biogen, Prolong Pharmaceuticals, Imperative Care, and stock options for advisory roles with

Brainomix, Viz-AI, Corindus Vascular Robotics, Vesalio, Ceretrieve, Astrocyte, and Cerebrotech.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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