Relationship of A₁ segment hypoplasia to anterior communicating artery aneurysm morphology and risk factors for aneurysm formation

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OBJECTIVE Hypoplasia of the A₁ segment of the anterior cerebral artery is frequently observed in patients with anterior communicating artery (ACoA) aneurysms. The effect of this anatomical variant on ACoA aneurysm morphology is not well understood.

METHODS Digital subtraction angiography images were reviewed for 204 patients presenting to the authors' institution with either a ruptured or an unruptured ACoA aneurysm. The ratio of the width of the larger A₁ segment to the smaller A₁ segment was calculated. Patients with an A₁ ratio greater than 2 were categorized as having A₁ segment hypoplasia. The relationship of A₁ segment hypoplasia to both patient and aneurysm characteristics was then assessed.

RESULTS Of 204 patients that presented with an ACoA aneurysm, 34 (16.7%) were found to have a hypoplastic A₁. Patients with A₁ segment hypoplasia were less likely to have a history of smoking (44.1% vs 62.9%, $p = 0.0410$). ACoA aneurysms occurring in the setting of a hypoplastic A₁ were also found to have a larger maximum diameter (mean 7.7 vs 6.0 mm, $p = 0.0084$). When considered as a continuous variable, increasing A₁ ratio was associated with decreasing aneurysm dome-to-neck ratio ($p = 0.0289$). There was no significant difference in the prevalence of A₁ segment hypoplasia between ruptured and unruptured aneurysms (18.9% vs 10.7%; $p = 0.1605$).

CONCLUSIONS Our results suggest that a hypoplastic A₁ may affect the morphology of ACoA aneurysms. In addition, the relative lack of traditional risk factors for aneurysm formation in patients with A₁ segment hypoplasia argues for the importance of hemodynamic factors in the formation of ACoA aneurysms in this anatomical setting.

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KEY WORDS A₁ hypoplasia; anterior communicating artery aneurysms; aneurysm morphology; dome-to-neck ratio; vascular disorders

HYPOPLASIA of the A₁ segment of the anterior cerebral artery (ACA) is a common anatomical variant, occurring in approximately 2% to 22% of the general population.^{5,14,15,18,21} A number of studies have described an association between A₁ segment hypoplasia and the presence of saccular berry aneurysms along the anterior communicating artery (ACoA) complex.^{4,13,15,16,22} As within this anatomical context the contralateral A₁ perfuses bilateral ACA territories, this association is thought to arise from the increased hemodynamic stress that occurs with greater flow across the ACoA, which may predispose to aneurysm formation.^{3,21,25}

The interaction of A₁ segment hypoplasia with other known risk factors for aneurysm growth and rupture of ACoA aneurysms, however, is less well understood. More-

over, the effect of this anatomical variant on ACoA aneurysm morphology has not been established. Herein, we examined the relationship between A₁ segment hypoplasia and risk factors for aneurysm formation and rupture in a cohort of patients with ruptured and unruptured aneurysms. In addition, we compared the prevalence of A₁ hypoplasia in patients with ruptured and unruptured ACoA aneurysms.

Methods

Data

After obtaining approval from our institutional review board, we conducted a retrospective cohort analysis

ABBREVIATIONS ACA = anterior cerebral artery; ACoA = anterior communicating artery; DSA = digital subtraction angiography; mFS = modified Fisher scale; WFNS = World Federation of Neurosurgical Societies.

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through electronic chart review of our institutional data. Patients were included for analysis if they presented to our institution with either a ruptured or unruptured ACoA aneurysm and underwent digital subtraction angiography (DSA) between the years of 2003 and 2013.

Determination of A₁ Segment Hypoplasia

The width in pixels of bilateral A₁ segments was measured immediately after take-off from the terminus of the internal carotid artery. Observers were blinded to patient and aneurysm characteristics at the time measurements were made. The ratio of the width of the larger A₁ segment to the smaller A₁ segment was then determined. An A₁ segment was considered hypoplastic if its width was less than 50% of the width of the contralateral A₁. This particular cut-off has been used previously to define A₁ segment hypoplasia¹⁶ and was also chosen for this study because of computational data suggesting that hemodynamics within the ACoA are altered when the width of the dominant A₁ segment is more than twice that of the contralateral A₁.¹²

Covariates

The primary covariate of interest was the presence of A₁ segment hypoplasia. Depending on the analysis in question, A₁ segment hypoplasia was considered as a categorical or continuous variable (with the A₁ ratio constituting the continuous variable). Additional covariates included patient age, sex, smoking history, history of hypertension, family history of aneurysms, modified Fisher scale (mFS) grade,¹⁰ World Federation of Neurosurgical Societies (WFNS) score,²³ maximum aneurysm diameter, aneurysm dome-to-neck ratio, aneurysm aspect ratio, presence of a daughter sac, and treatment modality. Patients were considered to have a positive smoking history if they had previously smoked cigarettes and quit or were current smokers at the time of subarachnoid hemorrhage or at the time of initial presentation for evaluation of their unruptured ACoA aneurysm. Active smoking was also considered as a separate covariate. Patients with unruptured aneurysms were considered to be active smokers if they were currently smoking at the time of their initial presentation to our institution for evaluation of an unruptured aneurysm. Patients with ruptured aneurysms were considered to be active smokers if they were smoking at the time of subarachnoid hemorrhage. The mFS grade was dichotomized into grades of 1 to 2 and 3 to 4. Similarly, the WFNS score was dichotomized into scores of 1 to 3 and 4 to 5. Aneurysm size (calculated as maximum diameter), presence of a daughter sac, dome-to-neck ratio, and aspect ratio were determined through visualization of DSA images. The dome-to-neck ratio was defined as the ratio of aneurysm dome width to neck width, while the aspect ratio was defined as the ratio of aneurysm height to neck width (Fig. 1), as has been described previously.¹ Patients without DSA images adequate for obtaining the necessary measurements or those whose images were unavailable were excluded from analyses examining the relationship of A₁ ratio to dome-to-neck and aspect ratios ($n = 36$).

FIG. 1. Representative ACoA aneurysm. The dome-to-neck ratio was calculated as dome width (d)/neck width (n). The aspect ratio was calculated as aneurysm height (h)/neck width (n), as previously described.¹

Statistical Analysis

Descriptive statistics for continuous variables are reported as a mean (SD) and median (range), while categorical variables are reported as frequencies and percentages. Continuous variables were compared between patients with and without A₁ segment hypoplasia and with a ruptured or an unruptured ACoA aneurysm using the 2-sample Student *t*-test, while categorical variables were compared using the Pearson chi-square test. The association of A₁ ratio and continuous variables was evaluated using the Spearman rank correlation test. The likelihood of patient and aneurysm characteristics being present in patients with ruptured ACoA aneurysms was determined using multivariate logistic regression analysis. All statistical tests were 2-sided with the alpha level set at 0.05 for statistical significance.

Results

We studied 204 consecutive patients with an ACoA aneurysm who presented to our institution between 2003 and 2013 and underwent DSA. Of these 204 patients, 148 had ruptured aneurysms and 56 had unruptured aneurysms. The patients' characteristics are detailed in Table 1. Within our cohort, 34 patients (16.7%) were found to have a hypoplastic A₁ segment (Fig. 2). The mean A₁ ratio of patients with A₁ segment hypoplasia was 3.6 (SD 1.6, median 3.2, range 2.0–9.7, $n = 34$), compared with a mean ratio of 1.3 (SD 0.3, median 1.3, range 1.0–1.9, $n = 170$) in patients

TABLE 1. Patient and aneurysm characteristics

Variable	Symmetric A ₁ Segments (n = 170)	Hypoplastic A ₁ Segment (n = 34)	Total (n = 204)	p Value*
Age in yrs				
Mean (SD)	58.4 (13.1)	59.1 (12.8)	58.6 (13.0)	0.7753
Median	56.5	58.0	56.5	
Range	(27.0–89.0)	(34.0–80.0)	(27.0–89.0)	
Sex				
Female	87 (51.2%)	17 (50.0%)	104 (51.0%)	0.9003
Male	83 (48.8%)	17 (50.0%)	100 (49.0%)	
Family history of aneurysms				
No	146 (85.9%)	32 (94.1%)	178 (87.3%)	0.1887
Yes	24 (14.1%)	2 (5.9%)	26 (12.7%)	
Smoking history				
No	63 (37.1%)	19 (55.9%)	82 (40.2%)	0.0410
Yes	107 (62.9%)	15 (44.1%)	122 (59.8%)	
Current smoker				
No	92 (54.1%)	24 (70.6%)	116 (56.9%)	0.0767
Yes	78 (45.9%)	10 (29.4%)	73 (43.1%)	
Hypertension				
No	73 (42.9%)	14 (41.9%)	87 (42.7%)	0.8494
Yes	97 (57.1%)	20 (58.8%)	117 (57.4%)	
WFNS score†				
1–3	80 (70.2%)	19 (73.1%)	99 (70.7%)	0.7692
4–5	34 (29.8%)	7 (26.9%)	41 (29.3%)	
mFS grade‡				
1–2	25 (21.9%)	6 (23.1%)	31 (22.1%)	0.8988
3–4	89 (78.1%)	20 (76.9%)	109 (77.9%)	
Aneurysm size (mm)‡				
Mean (SD)	6.0 (3.2)	7.7 (4.0)	6.3 (3.4)	0.0084
Median	5.0	6.0	5.0	
Range	(1.5–23.0)	(2.0–18.0)	(1.5–23)	
Daughter sac				
No	136 (80.0%)	28 (82.4%)	164 (80.4%)	0.7524
Yes	34 (20.0%)	6 (17.7%)	40 (19.6%)	
Treatment				
Medical	16 (9.4%)	3 (8.8%)	19 (9.3%)	0.1438
Clipping	38 (22.4%)	13 (38.2%)	51 (25.0%)	
Coiling	116 (68.2%)	18 (52.9%)	134 (65.7%)	

* Boldface type indicates statistical significance.

† Applicable only to patients with ruptured aneurysms and information only available for 140 of 148 patients.

‡ Maximum diameter; information only available for 202 patients.

without A₁ segment hypoplasia (Fig. 3 upper). The mean A₁ ratio of patients with ruptured aneurysms was 1.8 (SD 1.2, median 1.3, range 1.0–9.7, n = 148), compared with a mean A₁ ratio of 1.5 (SD 0.7, median 1.3, range 1.0–5.7, n = 56; p = 0.1540) in patients with unruptured aneurysms (Fig. 3 lower).

The mean age of our patient cohort was 58.6 years, and

FIG. 2. DSA images obtained in a representative patient with a ruptured ACoA aneurysm and a hypoplastic right A₁ segment. **Left:** Injection of right internal carotid artery demonstrates filling of the right hypoplastic A₁ and minimal filling of the right anterior cerebral artery. **Right:** Injection of left internal carotid artery fills the bilateral A₂ segments and the ACoA aneurysm.

there was a slight female predominance (51.0%). There were no statistically significant differences in age or sex between patients with and without A₁ segment hypoplasia.

We then assessed the relationship of A₁ segment hypoplasia to known risk factors for aneurysm formation and rupture.¹¹ Patients with A₁ segment hypoplasia were significantly less likely to have a history of smoking (44.1% vs 62.9%, p = 0.0410). Similarly, fewer patients with A₁ segment hypoplasia were found to be active smokers (29.4% vs 45.9%, p = 0.0767), although this difference was not statistically significant. There was also a trend toward a lower prevalence of a family history of intracranial aneurysms in patients with A₁ segment hypoplasia (5.9% vs 14.1%, p = 0.1887). There was no significant difference in the incidence of hypertension between patients with and without A₁ segment hypoplasia (58.8% vs 57.1%, p = 0.8494; Table 1).

The effect of A₁ segment hypoplasia on ACoA aneurysm characteristics was also investigated. ACoA aneurysms occurring in the setting of A₁ segment hypoplasia were significantly larger than aneurysms occurring in patients without a hypoplastic A₁ (mean maximum diameter 7.7 vs 6.0 mm, p = 0.0084). There was no difference in the incidence of a daughter sac between patients with and without A₁ segment hypoplasia (17.7% vs 20.0%, p = 0.7524). More patients with A₁ segment hypoplasia underwent surgical clipping of their ACoA aneurysms compared with patients with symmetric A₁ segments (38.2% vs 22.4%, Table 1). When analysis was limited to patients who underwent either clipping or endovascular coil embolization, there was a marked trend toward a greater likelihood of treatment with clipping in patients with A₁ segment hypoplasia (OR 2.20, 95% CI 0.95–4.90, p = 0.0575).

FIG. 3. Distribution of A_1 ratios in patients with and without A_1 segment hypoplasia (**upper**) and in patients with ruptured and unruptured aneurysms (**lower**). Midline of box denotes median value. Upper and lower borders of box denote 75th and 25th percentiles, respectively. Error bars denote 95th and 5th percentiles.

Aneurysm morphology can influence the selection of a treatment modality.^{6–8} To assess for a relationship between A_1 segment hypoplasia and the geometrical properties of ACoA aneurysms, the dome-to-neck and aspect ratios were calculated. A total of 169 patients had DSA images adequate for the accurate measurement of these metrics. Of these patients, 28 (16.6%) had a hypoplastic A_1 segment; the average ACoA aneurysm dome-to-neck ratio for these patients was 1.5 (Table 2). To assess the relationship of A_1 segment hypoplasia to aneurysm morphology, the ratio of the widths of bilateral A_1 segments for each ACoA aneurysm was considered as a continuous variable and related to dome-to-neck and aspect ratios using the Spear-

man rank-correlation test. We found that increasing A_1 ratio was significantly associated with decreasing dome-to-neck ratio ($p = 0.0289$). The A_1 ratio was not found to be related to the aspect ratio (Table 2).

We then assessed for differences in demographic and aneurysm characteristics between the ruptured and unruptured aneurysm groups. Within our cohort, patients with ruptured aneurysms had a younger mean age (56.9 vs 62.7 years, $p = 0.0050$) and were more likely to be male (46.6% female vs 62.5% male, $p = 0.0429$). While there was no statistically significant difference in the prevalence of a previous history of smoking (61.5% vs 55.4%, $p = 0.4255$), patients with ruptured aneurysms were more likely to be active smokers at the time of presentation (49.3% vs 26.8%, $p = 0.0037$). A_1 segment hypoplasia was more common in patients with ruptured aneurysms than in those with unruptured aneurysms (18.9% vs 10.7%), but the difference was not statistically significant ($p = 0.1605$). The maximum diameter of the aneurysms was similar in ruptured and unruptured aneurysms (mean 6.1 mm vs 6.5 mm, $p = 0.4711$). Regarding aneurysm shape, while the mean dome-to-neck ratio was similar in ruptured and unruptured aneurysms (1.7 vs 1.5, $p = 0.1369$), the mean aspect ratio was significantly greater in ruptured aneurysms than in unruptured aneurysms (1.9 vs 1.5, $p = 0.0108$). Finally, there was no significant difference in the prevalence of a daughter sac between ruptured and unruptured aneurysms (20.3% vs 17.9%, $p = 0.6985$). All comparisons between ruptured and unruptured aneurysms are detailed in Table 3.

The relationship of patient and aneurysm characteristics to the ruptured or unruptured status of the ACoA aneurysm was then investigated using multivariate logistic regression analysis. In addition to the presence of A_1 segment hypoplasia, variables found to be significantly different between ruptured and unruptured aneurysms were included in the multivariate model. On adjusted analysis, A_1 segment hypoplasia was not predictive of ruptured status. Instead, younger age, absence of family history of intracranial aneurysms, and acute smoking were independently predictive of ruptured status. The full results of the multivariate analysis are presented in Table 4.

Discussion

A_1 segment hypoplasia is a common anatomical variant that is encountered frequently in patients with an ACoA aneurysm.^{4,13,15,16,22,26} In the present study, we compared demographic and aneurysm characteristics of patients with and without A_1 segment hypoplasia presenting with either a ruptured or an unruptured ACoA aneurysm. We found that patients with A_1 segment hypoplasia were less likely to have a history of smoking and had larger aneurysms with a broader neck (i.e., lower dome-to-neck ratio). These results suggest that the hemodynamic changes resulting from a unilaterally hypoplastic A_1 can precipitate aneurysm formation along the ACoA complex even in the absence of traditional risk factors.

In the present study, an A_1 segment was considered hypoplastic if its diameter was less than 50% of the width of the contralateral A_1 .^{12,16} On the basis of this definition, 34 (16.7%) of the 204 patients in our cohort were found to

TABLE 2. Association between A₁ ratio and aneurysm morphology*

Aneurysm Characteristic	A ₁ Ratio				Total (n = 169)	p Value†
	1–1.2 (n = 65)	1.3–1.5 (n = 49)	1.6–2 (n = 27)	>2 (n = 28)		
Dome-to-neck ratio						
Mean (SD)	1.7 (0.7)	1.6 (0.6)	1.5 (0.6)	1.5 (0.6)	1.6 (0.6)	0.0289
Median	1.5	1.5	1.4	1.4	1.5	
Range	(0.7–4.7)	(1.0–3.6)	(0.8–2.7)	(0.8–2.7)	(0.7–4.7)	
Aspect ratio						
Mean (SD)	1.8 (0.7)	1.8 (0.8)	1.7 (0.9)	1.7 (0.8)	1.7 (0.8)	0.1791
Median	1.7	1.5	1.5	1.5	1.6	
Range	(0.5–4.0)	(0.7–4.4)	(0.6–3.7)	(0.6–3.1)	(0.5–4.4)	

* Information only available for 169 patients.

† Spearman rank correlation test. Boldface type indicates statistical significance.

have A₁ segment hypoplasia. Previous studies have documented a prevalence of A₁ segment hypoplasia ranging from 24% to 90% in patients with ACoA aneurysms (mean 41.5%, n = 6),^{4,13,15,16,22,26} suggesting that our definition of A = segment hypoplasia was relatively less inclusive. Nevertheless, the selection of a physiologically relevant cut-off¹² was intended to maximize the hemodynamic differences between patients classified as having or not having a hypoplastic A₁ segment.

Cigarette smoking has been consistently associated with both formation and rupture of intracranial aneurysms.^{11,17,27,28} Indeed, 59.8% of patients within our cohort had a history of tobacco use. Patients with A₁ segment hypoplasia, however, were less likely to have a smoking history (44.1% vs 62.9%), suggesting that, in accordance with previous studies,^{4,13,15,16,22,26} these patients likely have a predilection for ACoA aneurysm formation even in the absence of known risk factors. In addition, we found that ACoA aneurysms occurring in the setting of A₁ segment hypoplasia were larger and had greater dome-to-neck ratios relative to aneurysms occurring in patients with symmetric A₁ segments, suggesting that the hemodynamic changes associated with a hypoplastic A₁ segment may influence both aneurysm size and shape. Cadaveric studies have found the ACoA to be enlarged in proportion to the difference in width between A₁ segments, representing the need of the dominant A₁ segment to supply bilateral ACA territories.²¹ In a modeling study examining the biophysical effects of flow across the ACoA, increased cross-flow was correlated to high levels of shear stress on the arterial wall.²⁵ In another computational study, the increase in wall shear stress rose dramatically when the difference between A₁ segment widths was greater than or equal to 50%,¹² which was the rationale for the definition of A₁ segment hypoplasia chosen in this study. These effects may explain the morphological differences in ACoA aneurysms between patients with and without a hypoplastic A₁ segment.² Case-control studies comparing ACoA aneurysm dimensions over time in patients with and without A₁ segment hypoplasia are needed to gain insight into the effect of a hypoplastic A₁ on ACoA aneurysm growth patterns.

Given the potential effects on arterial wall shear stress, it is possible that A₁ segment hypoplasia may also confer an increased risk of ACoA aneurysm rupture.^{3,19,20,29} Al-

though the proportion of patients with A₁ segment hypoplasia was greater in patients presenting with a ruptured ACoA aneurysm, the difference was not statistically significant in our cohort. It may be difficult to determine factors associated with rupture from a cross-sectional comparison of cases of ruptured and unruptured aneurysms. For example, while active smoking was more prevalent in patients with ruptured aneurysms, female sex and a family history of intracranial aneurysms, 2 known risk factors for subarachnoid hemorrhage,²⁴ were associated with unruptured aneurysm status. These latter findings may be explained by a lower threshold for pursuing angiography in patients with demographic characteristics associated with increased risk of aneurysm rupture. Indeed, patients with unruptured aneurysms selected for angiography likely had features suggestive of an increased risk for rupture visualized on noninvasive imaging, potentially confounding any interpretation of the differences between patients with ruptured and unruptured aneurysms in our cohort. As stated above, prospective studies will likely be needed to better understand which patient and aneurysm characteristics carry an increased risk of rupture.

We observed a trend toward a greater likelihood of treatment of ACoA aneurysms with clipping in patients with A₁ segment hypoplasia. Smaller aneurysm dome-to-neck ratios have been correlated to lower rates of successful endovascular coil embolization,^{6–8} prompting surgeons to favor surgical clipping of broad-necked aneurysms. In our cohort, we found the A₁ ratio to be negatively correlated with the dome-to-neck ratio, and this may have contributed to the increased frequency of treatment with clipping in patients with A₁ segment hypoplasia. An additional possibility is that a unilaterally hypoplastic A₁ made endovascular access to ACoA aneurysms more difficult. It is our experience, however, that the A₁ contralateral to the hypoplastic segment is often large in diameter, in fact facilitating access. Ultimately, the choice of treatment modality for intracranial aneurysms depends on a number of different factors, some of which are independent of aneurysm morphology, thus it is difficult to ascertain how much the presence of a hypoplastic A₁ segment influenced the choice of treatment modality. Whether A₁ segment hypoplasia affects the efficacy or complication rate of different modes of aneurysm treatment may warrant

TABLE 3. Comparison of demographic and morphologic characteristics of aneurysms in patients with unruptured and ruptured ACoA aneurysms

Variable	Unruptured (n = 56)	Ruptured (n = 148)	Total (n = 204)	p Value*
Age in yrs				
Mean (SD)	62.7 (11.5)	56.9 (12.8)	58.6 (13.0)	0.0050
Median	66	55	57	
Range	(37–83)	(27–89)	(27–89)	
Sex				
Female	35 (62.5%)	69 (46.6%)	104 (51.0%)	0.0429
Male	21 (37.5%)	79 (53.4%)	100 (49.0%)	
Family history of aneurysms				
No	42 (75.0%)	136 (91.2%)	178 (87.3%)	0.0012
Yes	14 (25.0%)	12 (8.1%)	26 (12.7%)	
Smoking history				
No	25 (44.6%)	57 (38.5%)	82 (40.2%)	0.4255
Yes	31 (55.4%)	91 (61.5%)	122 (59.8%)	
Current smoker				
No	41 (73.2%)	75 (50.7%)	116 (56.9%)	0.0037
Yes	15 (26.8%)	73 (49.3%)	73 (43.1%)	
Hypertension				
No	22 (39.3%)	65 (43.9%)	87 (42.7%)	0.5504
Yes	34 (60.7%)	83 (56.1%)	117 (57.3%)	
Hypoplastic A ₁				
No	50 (89.3%)	120 (72.6%)	170 (83.3%)	0.1605
Yes	6 (10.7%)	28 (18.9%)	34 (16.7%)	
Aneurysm size (mm)†				
Mean (SD)	6.5 (3.0)	6.1 (3.6)	6.3 (3.4)	0.4711
Median	6.0	5.0	5.0	
Range	(2.0–16.0)	(1.5–23.0)	(1.5–23)	
Dome-to-neck ratio‡				
Mean (SD)	1.5 (0.5)	1.7 (0.7)	1.6 (0.6)	0.1369
Median	1.4	1.5	1.5	
Range	(0.7–3.0)	(0.8–4.7)	(0.7–4.7)	
Aspect ratio‡				
Mean	1.5 (0.6)	1.9 (0.9)	1.7 (0.8)	0.0108
Median	1.4	1.7	1.6	
Range	(0.5–3.6)	(0.6–4.4)	(0.5–4.4)	
Daughter sac				
No	46 (82.1%)	118 (79.7%)	164 (80.4%)	0.6985
Yes	10 (17.9%)	30 (20.3%)	40 (19.6%)	
Treatment				
Medical	12 (21.4%)	7 (4.7%)	19 (9.3%)	0.0006
Clipping	9 (16.1%)	42 (28.4%)	51 (25.0%)	
Coiling	35 (62.5%)	99 (66.9%)	134 (65.7%)	

* Boldface type indicates statistical significance

† Information only available for 202 patients.

‡ Information only available for 169 patients.

TABLE 4. Multivariate analysis indicating likelihood of aneurysm having ruptured*

Variable	OR (95% CI)	p Value†
Age in yrs	0.96 (0.93–0.99)‡	0.0182
Female sex	0.52 (0.24–1.10)	0.0856
Family history of aneurysms	0.24 (0.08–0.66)	0.0056
Current smoker	2.61 (1.11–6.50)	0.0271
Hypoplastic A ₁	1.58 (0.56–4.99)	0.3999
Aspect ratio	1.46 (0.85–2.63)‡	0.1789

* Variables with significant differences between ruptured and unruptured aneurysms (apart from A₁ hypoplasia) were included in the multivariate model.

† Boldface type indicates statistical significance.

‡ Unit odds ratio, denotes change in likelihood for every 1 integer increase in variable of interest.

further investigation. Our study is limited by the relatively small number of patients found to have a hypoplastic A₁ segment (34 [16.7%] of 204); thus, the associations found in our analysis need to be confirmed. In addition, measurement of width based on pixels may be prone to more error than measurements of absolute width,⁹ allowing for the possibility that borderline cases may have been categorized incorrectly. Finally, in 17.2% of our cases (35 of 204), we did not have the angiograms to perform the necessary measurements, which may have influenced our results.

Conclusions

In the present study, we found that patients with an ACoA aneurysm and A₁ segment hypoplasia differed with respect to both demographic and aneurysm characteristics when compared with patients with bilaterally symmetric A₁ segments. Our results suggest that the presence of a hypoplastic A₁ segment not only predisposes to ACoA aneurysm formation but may also influence aneurysm size and shape.

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Disclosures

Dr. Lanzino reports a consultant relationship with Covidien/Medtronic.

Author Contributions

Conception and design: Lanzino, Rinaldo, McCutcheon, Murphy. Acquisition of data: Rinaldo. Analysis and interpretation of data: Rinaldo. Drafting the article: Rinaldo. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Lanzino. Statistical analysis: Rinaldo.

Supplemental Information

Previous Presentations

A modified version of this study has been presented in oral form at the 2016 CNS Annual Meeting, September 24–28, in San Diego, California.

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