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Original Article

Intracranial arterial variations and their relation with cerebral aneurysms: Analysis of 640 patients

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Abstract

Aim: To evaluate prevalence of cerebral arterial variations in Turkish population and association of the variations with cerebral aneurysms

Materials and Methods: Digital subtraction angiography of 640 consecutive patients between January 2011-December 2013 were evaluated retrospectively. Patients with aneurysms were compared with patients without aneurysm to determine the effect of variations. Comparisons were made using Chi square or Fisher exact test for categorical variables and Student's t test or Mann Whitney U test for continuous variables.

Results: The most common variation in the anterior circulation was hypoplasia of anterior cerebral artery A1 segment and the most common variation in the posterior circulation was fetal origin of the posterior cerebral artery. Hypoplasia and aplasia of the anterior cerebral artery A1 segment was significantly more frequent in patients with anterior communicating artery aneurysms than the control group (p:<0.001). Prevalence of azygos anterior cerebral artery variation was higher in distal anterior cerebral artery aneurysms than the control group (p <0.001). There was no association between bihemispheric anterior cerebral artery and anterior communicating artery aneurysms (p = 0.453). Similarly, no significant association of fetal origin of posterior cerebral artery with posterior communicating artery aneurysms was found (p = 0.133).

Conclusion: Prevalence of cerebral arterial variations in this study were compatible with literature. Some variations may play a role in development of aneurysms by alterations in hemodynamics and increasing shear wall stress. Further larger studies are needed to clarify the relation between variations in cerebrovascular structure and aneurysm formation.

Keywords: Aneurysm; angiography; intracranial; variation

INTRODUCTION

The textbook circle of Willis is defined as a complete symmetrical polygon, consisting vessels of both anterior and posterior circulation with connecting communicating arteries. In practice, although asymptomatic, incomplete circle of Willis has been shown to be more frequent (1-3). As development of cerebral arteries is a complex process, consisting multiple steps, many kinds of variations could be encountered in circle of Willis.

Awareness of cerebrovascular variations is important and these variations should be detailed in preoperative imaging to determine strategies in endovascular or surgical treatment, to prevent complications that may occur during operation and to achieve success.

It is known that cerebrovascular variations coexist with various cerebrovascular pathologies such as aneurysm, arteriovenous malformation and stroke. In patients with cerebrovascular variations, hemodynamic stresses resulting from the variant anatomy may induce formation of an aneurysm or other vascular anomalies (4-7). Therefore, cerebrovascular variations can be considered as a risk factor.

In this retrospective study, the frequency of cerebrovascular variations in Turkish population and association of certain variations with cerebrovascular aneurysms were investigated.

MATERIALS and METHODS

This study was approved by local review board and informed consent was waived due to its retrospective design. Patients who underwent cerebral digital subtraction angiography in a training and research hospital between January 2011 and December 2013 were reviewed by two interventional radiologists. The angiographic images were obtained with the General Electric Innova 4100 IQ 2008

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monoplane angiography device and archival images were evaluated on the HP XW8400 model workstation.

A total of 921 patients underwent cerebral angiography during the study period. Patients with arteriovenous malformation, carotid-cavernous fistulas, intracranial mass lesions, severe carotid stenosis or occlusion, history of ischemic cerebrovascular accident and suboptimal image quality were excluded from the study. After exclusion, a total of 640 patients were included in the study.

Variations of the cerebral arteries were evaluated in two main groups. Anterior circulation variations were defined as variations of anterior cerebral artery (ACA), middle cerebral artery (MCA), internal carotid artery (ICA) and anterior communicating artery (AComA). Posterior circulation variations were defined as variations of vertebral artery, posterior cerebral artery (PCA), basilar artery and its main branches. Definition of the variations were as follows:

Hypoplasia: Diameter of the vessel less than half of the contralateral side and/or less than 1.5mm

Aplasia: Absence of the vessel

Azygos ACA: Merging of bilateral ACA A1 segments and forming a single central A2 segment perfusing both lobes

Bihemispheric ACA: Hypoplasia of one ACA A1 segment and perfusion of the both lobes from contralateral ACA A2 segment

Accessory ACA: Additional ACA A2 segment perfusion both lobes

Accessory MCA: Additional MCA M1 artery originating from ACA A1 segment

Duplication: Additional artery originating from the same vessel and perfusing the same segment

Fetal origin of PCA: Hypoplasia of the PCA P1 segment and supplement of PCA flow from ICA

Fenestration: Partial luminal duplication of the artery without distinct origin and with the fusion distally

Three hundred and seventy-three patients in whom intracranial aneurysms were detected were included in study group and 267 patients in whom there were no intracranial aneurysm were included in the control group.

Cerebrovascular variations and aneurysms detected in three-dimensional angiographic images were evaluated and the relationship between the frequency of variations and the presence of aneurysms were studied.

Statistical analysis of the data was done with SPSS 15 packet program. Descriptive statistics were expressed with mean, standard deviation, frequency and percentages. Categorical variables were compared with Chi square or Fisher exact test and Student's t test or Mann Whitney U test were used for intergroup comparison of continuous variables.

RESULTS

Incidence of variations

Mean age of the patient population was 53±14.3 and 47.6% of the patients were men. In the anterior circulation, ACA A1 segment hypoplasia was the most common variation (44 left, 43 right) and ACA A1 segment aplasia was detected in 20 cases (13 left, 7 right). Azygos ACA was observed in 8 patients. Bihemispheric ACA was reported in 24 patients (18 left and 6 right). ICA agenesis (1 right, 1left) was observed in 2 cases. In 1 case, ICA fenestration was seen on the right side. MCA fenestration was seen in only 2 cases (1 right, 1 left). Accessory ACA was detected in 2 cases. ACA A1 segment fenestration was observed in only 1 case. Anterior system variations were summarized in Table 1.

Table 1. Distribution of detected anterior system variations

Variable	n (%)
ACA A1 segment hypoplasia	87 (13.5)
ACA A1 segment aplasia	20 (3.1)
Bihemispheric ACA	24 (3.7)
Azygos ACA	8 (1.2)
MCA duplication	11 (1.7)
ICA agenesis	2 (0.3)
ICA hypoplasia	1 (0.15)
MCA fenestration	2 (0.3)
Accessory MCA	1 (0.15)
Accessory ACA	2 (0.3)
ACA A1 segment fenestration	1 (0.15)
ICA fenestration	1 (0.15)

ACA: Anterior cerebral artery; MCA: Middle cerebral artery; ICA: Internal carotid artery

In the posterior circulation, the most common variation was fetal origin of PCA (34 left, 86 right, 7 bilateral). PCA P1 segment hypoplasia in 41 patients and PCA P1 segment aplasia in 6 patients were associated with fetal origin of PCA. PComA infundibulum dilatation was observed in 35 patients (13 left and 22 right) and vertebral artery hypoplasia in 33 patients (11 left and 22 right) and duplication of the superior cerebellar artery in 24 patients (10 left, 13 right and 1 bilateral) were found. Basilar artery fenestration was detected in 8 patients, PICA ending of the vertebral artery was observed in 8 patients (5 right and 3 left). Vertebral artery fenestration was detected in 4 patients (1 right and 3 left), while the duplicated origin of the vertebral artery was detected in 2 patients. The posterior system variations were summarized in Table 2.

Persistent carotid-basilar anastomoses were detected in two cases, both being proatlantal intersegmental artery on the right side.

Aneurysms and their association with variations

In the second part of the study, relation between aneurysms and cerebral arterial variations was evaluated.

A total of 403 aneurysms were detected in 373 patients. The mean age of the group without aneurysm (n:267) was 50.3 ± 15.6 years and the mean age of the group with aneurysm (n:373) was 55,0 ± 13,0 years. In the aneurysm group 55.8% of the patients were male, whereas 41.8% were male in the control group. There was no significant difference between the study and control groups in terms of gender and age (p >0.05).

Table 2. Distribution of detected posterior system variations

Variable	n (%)
Fetal origin PCA	134 (20.9)
Infundibular Dilatation of PComA	35 (5.4)
Vertebral artery hypoplasia	33 (5.1)
SCA duplication	25 (3.9)
Arcus origin of left vertebral artery	18 (2.8)
Basilar artery fenestration	8 (1.2)
PICA termination of vertebral artery	8 (1.2)
Duplicate origin of the vertebral artery	2 (0.3)
Vertebral artery fenestration	4 (0.6)

PCA: Posterior Cerebral Artery; PComA: Posterior Communicating Artery; SCA: Superior Cerebellar Artery; PICA: Posterior Inferior Cerebellar Artery

Multiple aneurysms were detected in 19 patients. Five aneurysms detected in 1, 4 detected in 1, 3 detected in 6, and 2 detected in 11 patients. Distribution of aneurysms were presented in Table 3.

Table 3. Distribution of detected aneurysms according to localizations

Aneurysm Location	n (%)
AComA	101 (25)
MCA M1 Bifurcation	99 (24.5)
MCA M1 Trunkus	26 (6.4)
Proximal ACA	18 (4.4)
Distal ACA	8 (1.9)
ICA supraclinoid	59 (14.6)
ICA-PComA	31 (7.6)
ICA ophthalmic	20 (4.9)
ICA cavernous	17 (4.2)
Basillar artery	12 (2.9)
Other*	12 (2.9)

*** Other localizations: Posterior cerebral artery, anterior inferior cerebellar artery, superior cerebellar artery, posterior inferior cerebellar artery ACA: Anterior cerebral artery; AComA: Anterior communicating artery; MCA: Middle cerebral artery; ICA: Internal carotid artery; PComA: Posterior communicating artery**

In 87 patients with ACA A1 segment hypoplasia, 57 AComA, 20 MCA, 11 ACA, 15 ICA and 6 PComA aneurysms were associated. Twenty patients with ACA A1 segment aplasia presented with 15 AComA, 3 MCA, 8 ICA, 1 PComA and 1 ACA aneurysm. Eight patients with Azygos ACA variation presented with 4 distal ACA, 3 MCA and 1 ICA aneurysm. Of the 24 patients with bihemispheric ACA variation, 5 had AComA, 3 had MCA, 1 had ACA, 1 had PCA and 1 had an ICA aneurysm. In 2 patients with Accessory ACA, one AComA aneurysm was observed. There was 1 MCA aneurysm in a case of ACA A1 segment fenestration.

One of the two cases with ICA agenesis was accompanied by one ICA aneurysm on the opposite side and other case was presented with a basilar artery aneurysm. In the only case with ICA hypoplasia, there was an aneurysm in AComA and 1 in the ACA A1 segment. A case with ICA fenestration was accompanied by fusiform dissecting aneurysm in ICA supraclinoid segment.

In 11 patients with MCA duplication, 2 ICA aneurysms and 3 MCA aneurysms were detected. Sample case of MCA duplication associated with MCA aneurysm was presented in Figure 1. One of the 2 patients with MCA fenestration had one AComA aneurysm. There was no aneurysm in the only patient with Accesory MCA.

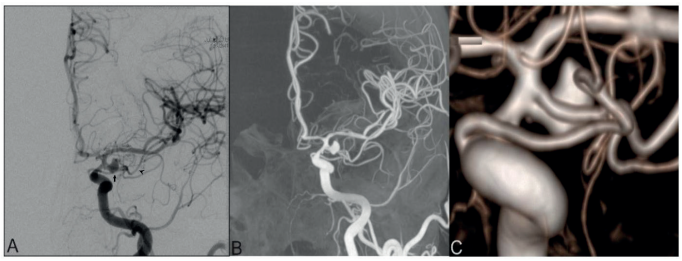

Figure 1. Left internal carotid arteriography shows duplication of the middle cerebral artery (black arrowhead) and associated middle cerebral artery M1 segment aneurysm (black arrow) (a). Maximum intensity projection and 3D volumetric reconstruction images depict the variation and aneurysm better (b,c)

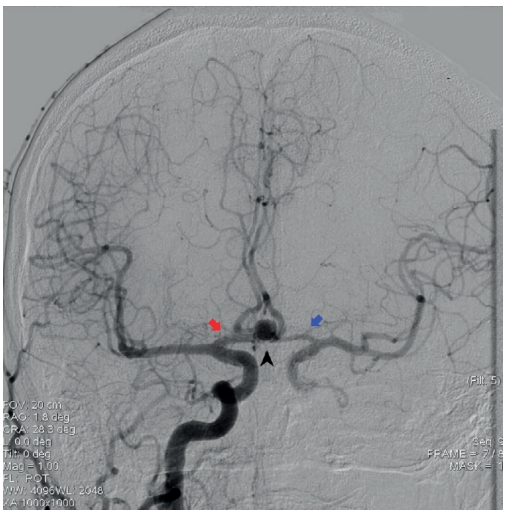

Figure 2. Right carotid angiography during compression to the left side shows hypoplasia of the left anterior cerebral artery A1 segment (blue arrow) defined as smaller in diameter than right anterior cerebral artery A1 segment (red arrow) and aneurysm of anterior communicating artery (black arrowhead)

ACA A1 hypoplasia and aplasia were observed in 72 (71.2%) of 101 patients with AComA aneurysm. In the control group, 35 of the 267 patients had A1 hypoplasia and aplasia. Compared with the control group, the A1 hypoplasia and aplasia were 16.46 times more frequent in AComA aneurysms (p <0.001; CI: 9.41-28.77). Sample case of ACA A1 hypoplasia with AComA aneurysm was presented in Figure 2.

Bihemispheric ACA was seen in 5% of patients with AComA aneurysm. In the control group bihemispheric ACA was observed in 7%. The odds ratio was 0.68 and there was no statistically significant difference (p = 0.453).

Azygos ACA were observed in 4 of 8 patients with distal ACA aneurysms while Azygos ACA variation was present in 4 of 267 patients in the control group. Compared with the control group, rate of Azygos ACA variation in the distal ACA aneurysms was 65.75 times higher (p <0.001; CI:11.99-360.52). Sample case of Azygos ACA with distal ACA aneurysm was presented in Figure 3.

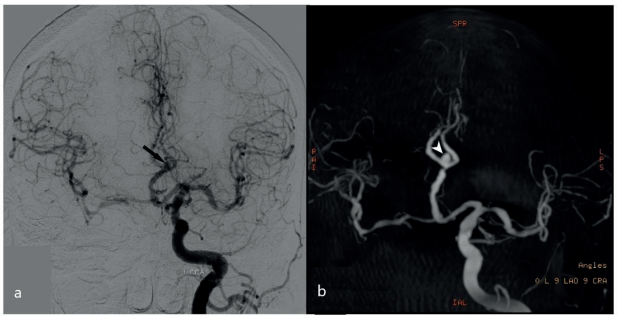

Figure 3. Left internal carotid angiogram shows single anterior cerebral artery A1 segment defined as azygos cerebral artery and associated aneurysm at A2-A3 segment (black arrow) (a). Maximum intensity projection reconstruction depicts the variation and aneurysm (white arrowhead) (b)

Eight patents with basilar fenestration showed 1 aneurysm of vertebrobasilar junction, 1 aneurysm in AComA, 1 in ICA and 1 aneurysm in AICA. Sample case of basilar artery fenestration associated with aneurysm was presented in Figure 4. A hundred and thirty-four patients with Fetal origin of PCA were accompanied by 18 AComA, 10 PComA, 25 MCA, 15 ICA, 7 PCA, 3 ACA and 2 basilar aneurysms.

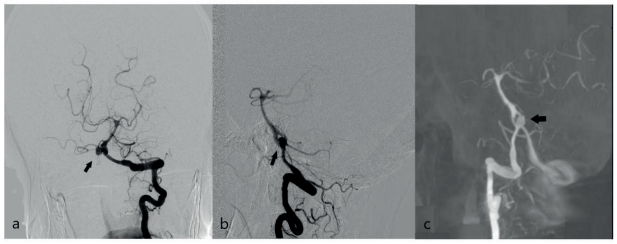

Figure 4. Anterior-posterior (a), lateral (b) view angiographies of left vertebral artery and oblique 3D reconstruction of angiograms (c) show fenestration of basillar artery with associated saccular aneurysm (black arrow)

In 32.3% of patients with PComA aneurysm, there was fetal PCA on the ipsilateral side. In the control group, fetal PCA was detected in 46.4%. There was no significant difference in the incidence of fetal PCA between control group and PComA aneurysms (p = 0.133). Sample case of fetal PCA with PComA aneurysm was presented in Figure 5.

Persistent proatlantal intersegmental artery was seen in two patients accompanied by one in PICA aneurysm in one patient and one in AComA and one ICA aneurysms in the other patient.

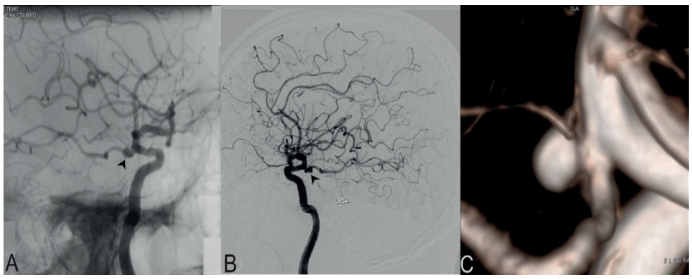

Figure 5. Oblique (a) and lateral (b) view angiography of carotid artery and 3D volumetric reconstruction show fetal type posterior cerebral artery with associated posterior communicating artery aneurysm (black arrowhead)

DISCUSSION

In this study, incidence of cerebral arterial variations and their association with cerebral aneurysms were evaluated. As previously reported, a complete and symmetrical circle of Willis seldomly detected in most patients and awareness of existing variations is important in treatment planning (1-3). Variations in cerebral arteries may have influence cerebral aneurysms formation with hemodynamic changes in the Willis polygon (4-7). The hypothesis that there may be a relationship between variations in the Willis polygon and the formation of aneurysms was first reported by Padget et al (8). They observed higher aneurysm rates in patients with variations than patients without variation. Kayembe et al also supported this hypothesis and proposed that variations play an important role in the formation of aneurysms (5).

The most common variation detected in the anterior circulation was ACA A1 segment hypoplasia. Although the frequency of ACA A1 segment hypoplasia in the anatomical studies is reported between 2-11.9% in the literature, in the imaging studies it was reported up to 21.07% (1,3,9,10). ACA A1 segment aplasia was reported in anatomical studies at a frequency of 1.8-2.1% and 2.4-19% in angiographies (1,3,9,11). In this study, ACA A1 segment aplasia was detected in 3.1% and ACA A1 segment hypoplasia was detected in 13.5% of the patients, which were compatible with literature.

Hypoplasia and aplasia of ACA A1 segment were found strongly related with AComA aneurysms in this study. In previous comparative studies by Krasny and Tarulli, ACA A1 hypoplasia was found to be significantly higher in AComA aneurysms than the group without aneurysms which supports findings of our study (12,13).

In addition to aneurysm formation, ACA A1 segment hypoplasia were reported to correlate with higher risk of rupture and recurrence after endovascular treatment of AComA aneurysms (4,14,15). Rinaldo et al found that the hypoplasia of ACA A1 segment results in larger diameter and decreased dome-neck ratio suggesting this variation has important effect on aneurysm morphology (16).

The incidence of azygos ACA reported in healthy population ranges from 0 to 5% and bihemispheric ACA has been reported to be 2-7% (17-20). In our study, the azygos ACA variation was found in 1.2% and bihemispheric ACA variation was found in 3.7% of the patients, which were compatible with the literature. In our study, azygos ACA variant was accompanied by distal ACA aneurysm in 50% of the cases. When compared to the control group, the association of azygos ACA variation in distal ACA aneurysms was significantly higher. However, the number of azygos ACA variations in our study group and the number of distal ACA aneurysms may be considered to be small, and it is clear that by evaluating a greater number of cases with distal ACA aneurysms will determine the relationship with azygos ACA variation more precisely.

Fetal PCA has been reported in the literature with an incidence ranging between 4-29% and it is bilateral in 1-9% (21,22). It is more common in females than in males. In general, this variation is associated with hypoplasia of ipsilateral PCA or, more rarely, with aplasia. In our study, consistent with literature, fetal PCA was detected in 20.1% and fetal PCA was the most frequent variation observed in the posterior system.

Fetal PCA is thought to contribute aneurysm formation in PComA due to increased flow-induced hemodynamic stress. In an MR angiography study performed by Horikoshi et al. in patients with known ICA aneurysm, the most frequent variation accompanying the aneurysms was found to be fetal-originated PCA in 47% of cases (23). Fetal originated PCA was observed in the aneurysmal side of 30 patients (11.9%) in the PComA aneurysm series of 273 cases reported by Zada et al. (24). In a CT angiographic study fetal PCA was reported 4.4 times more prevalent in patients with PComA aneurysms (25). Similarly, Kayembe and his colleagues in patients with PComA asymmetry, monitoring of the aneurysm more frequently in this region supports the hypothesis that hemodynamic stresses play a role in the formation of aneurysms (5). There was no significant difference in fetal PCA variation between patients with PComA aneurysm and the control group in our study. Although no difference was found between fetal PCA and PComA aneurysm, it should be noted that the statistical analysis of our study was performed on fewer PComA aneurysms than the reported series literature. Fetal origin PCA variation may be effective in the formation of aneurysms according to hemodynamic theory. However, we think that this variation is very common and the probability of incidentally detecting this variation in patients with PComA aneurysm is high.

LIMITATIONS

This study has some limitations to mention. Firstly, this study was designed as a retrospective cohort control study and patient selection bias could not be excluded. Secondly, other clinical variables that may contribute aneurysm formation were not included and studied which may preclude to make general statements. Since this is an angiographic study and mostly clinical indication was subarachnoid hemorrhage, our patient population may not present the general population precisely and vasospasm associated with subarachnoid hemorrhage may obscure some of the variations.

CONCLUSION

Knowledge of cerebrovascular anatomy, awareness of variations is of great importance in both surgical and endovascular treatment in terms of treatment planning and prevention of potential complications. Some variations are strongly associated with aneurysm in specific locations and may contribute to de novo aneurysm formation.

Conflict of interest : The authors declare that they have no competing interest.

Financial Disclosure: There are no financial supports.

Ethical approval: This study was approved by the local ethics committee of Okan University with the protocol number 204.01.07 / 22.

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