

PAPER DETAILS

TITLE: Circulus Willis Anomalies Diagnosed with CT Angiography and Evaluation of Their Relations with Ischemic Stroke

AUTHORS: Halil Ay

PAGES: 1924-1929

ORIGINAL PDF URL: <https://dergipark.org.tr/tr/download/article-file/3460880>

Circulus Willis Anomalies Diagnosed with CT Angiography and Evaluation of Their Relations with Ischemic Stroke

BT Anjiyografi ile Teşhis Edilen Circulus Willis Anomalileri ve İskemik İnme ile İlişkilerinin Değerlendirilmesi

Halil AY¹

ABSTRACT

Our aim in this study is examining the frequency of posterior communicating artery (PCoM), Anterior Cerebral Artery A1 segment (ACA A1) and Fetal type posterior cerebral artery (FPCA) anomalies and their effects on ischemic stroke by retrospectively analyzing examinations of Computerized Tomography Angiography (CTA) taken in our hospital within 2017. 22 cases (26.5%) diagnosed with bilateral hypoplastic / aplastic PCoA anomaly, were diagnosed with anterior circulation infarct, 24 cases (28.9%) were diagnosed with posterior circulation infarct, but 37 cases (44.6%) were not diagnosed with ischemic stroke. 17 (37.8%) of the cases who have unilateral hypoplastic / aplastic PCoA anomaly, were diagnosed with anterior circulation infarct, 14 cases (31.1%) were diagnosed with posterior circulation infarct. 13 (26%) of 50 cases diagnosed with unilateral hypoplastic / aplastic ACA A1 segment, were diagnosed with anterior circulation infarct, 10 cases (20%) were diagnosed with posterior circulation infarct. 27 cases (54%) were not diagnosed with ischemic stroke. Both of 2 cases diagnosed with bilateral hypoplastic / aplastic ACA A1 segment, had anterior circulation infarct. 4 (30.8%) of 13 cases diagnosed with unilateral FPCA anomaly, were diagnosed with anterior circulation infarct, 3 cases (23.1%) were diagnosed with posterior circulation infarct, 6 cases (46.2%) did not have ischemic stroke symptoms. In conclusion, even though these variations' role in optimum stroke prophylaxis in individuals having one or more stroke risk factors, has not determined yet, there is no doubt that clinical researcher will eliminate these uncertainties in future studies.

Keywords: Anterior Cerebral Artery, Computerized Tomography Angiography, Stroke, Posterior Communicating Artery

ÖZ

Bu çalışmadaki amacımız Bilgisayarlı Tomografi Anjiyografi (BTA) incelemelerini retrospektif olarak analiz ederek Posterior Komünikan Arter (PCoM), Anterior Serebral Arter A1 segmenti (ACA A1) ve Fetal Tip Posterior Serebral Arter (FPCA) anomalilerinin sıklığını ve iskemik inme üzerindeki etkilerini incelemektir. Çalışma retrospektif olarak planlandı ve 2017 yılında BTA çekilen hastalar çalışmaya dahil edildi. Bilateral hipoplastik/aplastik PCoA anomalisi tanısı alan 22 olguya (%26,5) anterior sirkülasyon infarktı, 24 olguya (%28,9) posterior sirkülasyon infarktı tanısı konulmuştur, 37 olguya (%44,6) % iskemik inme tanısı konulmamıştır. Tek taraflı hipoplastik/aplastik PCoA anomalisi olan olguların 17'sine (%37,8) anterior sirkülasyon enfarktı, 14 olguya (%31,1) posterior sirkülasyon enfarktı tanısı konulmuştur. Tek taraflı hipoplastik/aplastik ACA A1 segmenti tanısı alan 50 olgunun 13'üne (%26) anterior sirkülasyon infarktı, 10 olguya (%20) posterior sirkülasyon infarktı tanısı konulmuştur. 27 olguya (%54) iskemik inme tanısı konulmamıştır. Bilateral hipoplastik/aplastik ACA A1 segmenti tanısı alan 2 olgunun her ikisinde de anterior sirkülasyon infarktı mevcuttu. Tek taraflı FPCA anomalisi tanısı alan 13 olgunun 4'üne (%30,8) anterior sirkülasyon enfarktı tanısı konulmuştur, 3 olguya (%23,1) posterior sirkülasyon enfarktüsü tanısı konulurken, 6 olguda (%46,2) iskemik inme semptomu saptanmamıştır. Sonuç olarak, bir veya daha fazla inme risk faktörüne sahip bireylerde bu varyasyonların optimum inme profilaksisindeki rolü henüz belirlenmemiş olsa da, klinik araştırmacıların gelecekteki çalışmalarda bu belirsizlikleri ortadan kaldıracığından şüphe yoktur.

Anahtar Kelimeler: Anterior Serebral Arter, Bilgisayarlı Tomografi Anjiyografi, İnme, Posterior Komünikan Arter.

Harran Üniversitesi etik kurul onayı alınmıştır. Bu çalışma VIII. Ulusal Beyin Damar Hastalıkları Kongresi'nde sözlü bildiri olarak sunulmuştur.

¹ Doç. Dr. Halil AY, Nöroloji, Gaziantep Üniversitesi ve Tıp Fakültesi, ayhalil27@hotmail.com, ORCID: 0000-0001-7233-7698

İletişim / Corresponding Author:
e-posta/e-mail:

Halil AY
ayhalil27@hotmail.com

Geliş Tarihi / Received: 26.09.2023
Kabul Tarihi/Accepted: 26.12.2023

INTRODUCTION

Circulus Willis (CW) which was identified by Thomas Willis in 1664, supplies collateral blood flow by many ways.¹⁻² CW is a circulation taking place between both carotid systems and vertebrobasilar system in intracranial area, when necessary. Anterior communicating arteries (AcoM) and posterior communicating arteries (PCoM) are component arteries of CW and they are defined as primary collateral way.³⁻⁴ The other way which is known as secondary collateral way is anastomosis located between internal maxillary artery which is section of external carotid artery, and ophthalmic artery, and leptomeningeal anastomosis.⁵⁻⁷

The efficiency of CW depends on its integrity, length and width of its arteries. Clinical importance of variations in CW morphology which causes redistribution of blood, has increasingly been becoming obvious. Hemodynamic analyses have shown that an occlusion in a artery redirects the blood flow to other arteries and this results in decreased collateral flow, and also these variations can cause measurable and clinically related ischemia, including stroke.⁸⁻¹⁰

Fetal type posterior cerebral artery (FPCA) is a common anatomical variation,

observed in CW and regardless of a small relation with basilar artery (BA), it is defined as posterior cerebral artery (PCA) originated from internal carotid artery (ICA).¹¹ In this case, instead of BA, ICA supplies blood to PCA.¹² It is reported that between 12% to 36% of cases within literature have FPCA.¹³ Some studies have found that there is a significant relation among FPCA and occipital lobe infarction, life threatening headache and White matter degeneration.¹⁴⁻¹⁵

Due to ever increasing role of CW morphology in cerebral hemodynamic and ischemia, it is possible that variations within CW morphologies would be an important risk factor for development of cerebrovascular disease (CVD). In many studies it has been found that healthy collateral circulation decreases the risk of temporary ischemic attack and fatal ischemic stroke.¹⁶⁻¹⁸

Our aim in this study is examining the frequency of posterior communicating artery (PCoM), Anterior Cerebral Artery A1 segment (ACA A1) and Fetal type posterior cerebral artery (FPCA) anomalies and their effects on ischemic stroke by retrospectively analyzing examinations of Computerized Tomography Angiography (CTA) taken in our hospital within 2017.

MATERIAL AND METHOD

Material

In 2017, 220 outpatients and inpatients, treated in Harran University Faculty of Medicine and examined with CT Angiography were scanned retrospectively. Clinical and detailed CTA reports of these patients were obtained from patients' files.

Method

In this study, 16-sliced spiral CT device (General Electric Revolution 64 Detector 256 sections) and 400 pressure injectors (MedradStellant) were used. Hypoplastic / Aplastic PCoA diagnosis was made by CTA and with less than 1 mm artery diameter or

without artery diameter, whereas Hypoplastic / Aplastic ACA A1 segment diagnosis was made with less than 1.5 mm artery diameter or without artery diameter. FPCA diagnosis was made with larger diameter of PCoA, located in the same side of P1 segment and dominant blood build-up of occipital lobes' being supplied by internal carotid artery which is in the same side. Before the beginning of study we got ethics committee approval from Harran University. In Figures 1 and 2, examples of CW anomalies within the CTA taken in our hospital, are given.

In the statistical method, frequency analysis of variables was performed.

Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

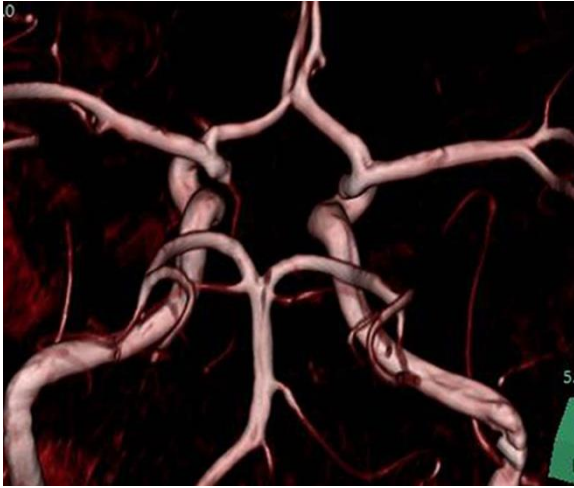


Figure 1. Bilateral Hypoplastic Pcoa Anomaly

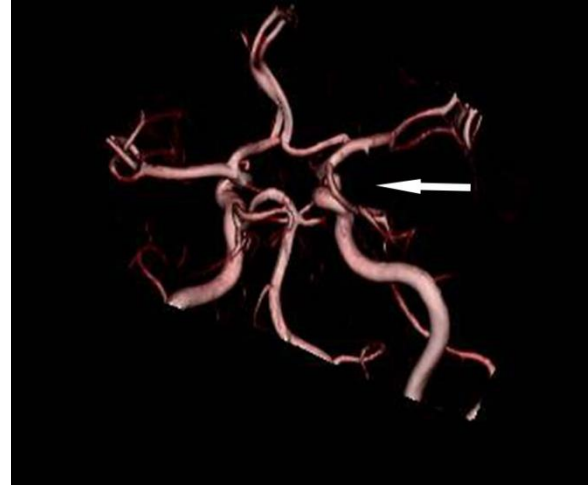


Figure 2. Left Fetal Type Posterior Cerebral Artery

RESULTS AND DISCUSSION

There were 220 cases in total, who had CTA scan, and 130 of them (59.1%) were male, 90 of them (40.9%) were female. Age average of male cases was 59.69 ± 15.06 . Age average of female cases was 60.38 ± 15.85 .

45 of 220 cases (20.5%) were diagnosed with unilateral hypoplastic/aplastic PCoA anomaly, 83 cases (37.7%) were diagnosed with bilateral hypoplastic/aplastic PCoA anomaly. 92 cases (41.8%) were not diagnosed with hypoplastic/aplastic PCoA anomaly. 22 cases (26.5%) diagnosed with bilateral hypoplastic/aplastic PCoA anomaly, were diagnosed with anterior circulation infarct, 24 cases (28.9%) were diagnosed with posterior circulation infarct, but 37 cases (44.6%) were not diagnosed with ischemic stroke. 17 (37.8%) of the cases who have unilateral hypoplastic / aplastic PCoA anomaly, were diagnosed with anterior circulation infarct, 14 cases (31.1%) were diagnosed with posterior circulation infarct. 14 cases (31.1%) cases were not diagnosed

with ischemic stroke. Only 5 (3.9%) of cases diagnosed with hypoplastic/aplastic anomaly, were diagnosed with unilateral ICA stenosis which was higher than 50%, and all had stroke symptoms, but this ratio was not statistically significant both in cases with PCoA anomaly and all cases who had stroke symptoms within this group ($p > 0.05$).

13 of 220 cases (5.9%) were diagnosed with FPCA anomaly, 1 case (0.5%) was diagnosed with FPCA anomaly. 206 cases (93.6%) were not diagnosed with FPCA anomaly. 4 (30.8%) of 13 cases diagnosed with unilateral FPCA anomaly, were diagnosed with anterior circulation infarct, 3 cases (23.1%) were diagnosed with posterior circulation infarct, 6 cases (46.2%) did not have ischemic stroke symptoms. Only one case diagnosed with bilateral FPCA anomaly, did not have ischemic stroke symptoms. Only 1 (7.1%) of cases diagnosed with FPCA anomaly, was diagnosed with unilateral ICA stenosis which was higher than 50%, and all

had stroke symptoms, but this ratio was not statistically significant both in cases with FPCA anomaly and all cases who had stroke symptoms within this group ($p>0.05$). In Table 1 and 2, frequency of CW anomaly and concomitant stroke ratio are given.

Table 1. Frequency of circulus willis anomaly

Circulus Anomalies	Willis	Number of Patients and Ratio
PCoA		128 (58.1%)
ACoA		52 (23.6%)
Fetal PCA		14 (6.3%)

In recent years there has been an increasing interest in CW morphology studies in order to reveal the relations between CW morphology and ischemic stroke. By referring to postmortem studies, normal CW prevalence can vary between 15% and 59%.¹⁹⁻²⁰ In imaging studies, in 27-90% of healthy individuals and in 18-55% of cerebrovascular diseases all CW variations were reported.²¹⁻²²

Much as digital subtraction angiography (DSA) is still the standard reference procedure for detection of intracranial vascular variations, it is an invasive technique which has potential complications.²³ Additionally, CTA has an increased sensitivity and freedom which is close to diagnostic accuracy of DSA (respectively 81-90% and 93%), it gives useful information about anatomical variations of cerebral circulation.²⁴ Therefore, in this study we aimed at evaluating the relations between Circulus Willis anomalies and ischemic stroke by using CTA technique, because this technique is less invasive.

AcoM and especially existence and diameter of PCoA show quite a vast variation in angiographic imaging of patients who have cerebrovascular diseases and in cadaver studies of individuals who are free from cerebral disease.²⁵

The average ratio of hypoplastic PCoA incidence (small lumen diameter) in angiographic studies and non-existence of PCoAs in cadaver studies is 47%. In the biggest CTA study till today, bilateral or

Table 2. Circulus Willis anomaly and frequency of stroke

Circulus Willis Anomalies	Frequency of Stroke	
	With stroke	Without stroke
PCoA (N:128)	77 (60.1%)	51 (39.9%)
ACoA (N:52)	25 (48.1%)	27 (51.9%)
Fetal PCA (N:14)	7 (50%)	7 (50%)

unilateral non-existence incidence of PCoAs are respectively 17% and 16%, and non-existence of AcoM is 1%.²⁶ In our study 128 of 220 cases (58.2%) were diagnosed with PCoA anomaly (unilateral in 45 cases, bilateral in 83 cases, respectively 35.1%, 64.9%). PCoA hypoplasia is recognized as a predisposing factor in carotid occlusive diseases, hemispheric low flow infarcts.²⁷ In a study which was carried by Yu-Ming et al, which covered 310 patients who had ischemic stroke, it was found that in hemispheric ischemic stroke patients PCoA hypoplasia incidence was 19.35% (60/310). Even though there is no concomitant ICA stenosis of PCoA hypoplasia, it was recommended that this should have been a risk factor for ischemic stroke.²⁸

ACA A1 segment hypoplasia is a rare and important variation of CW.²⁹ In general most of hypoplastic and aplastic A1 ACA are asymptomatic, and they are not directly related to any neurological diseases, but they can cause inter or intrahemispheric collateral circulatory failure.³⁰ This frequency of congenital variation is 1-13% as derived from angiograms and autopsy reports in various experimental studies.^{29,31} In our study 52 of 220 cases (23.6%) were diagnosed with hypoplastic/aplastic ACA A1 segment anomaly (unilateral in 50 cases, bilateral in 2 cases, respectively 96.1%, 3.9%). In a study carried by Chuang et al, the ratio of ACA A1 segment hypoplasia which can be detected with MR angiography in stroke patients, was found as 17.5%. In control group it was found as 4.28%.²⁹

In a study carried by Kovac et al, the ratio of ACA A1 segment hypoplasia in a series of 455 patients for which CTA technique was used, was found as 17.6%.³² However in our study this ratio was calculated as 23.6%. ACA A1 segment hypoplasia, which is an insufficient part of CW, is seen as a risk factor for acute ischemic stroke.²⁹ The frequency of ischemic stroke in cases who had ACA A1 segment hypoplasia, was determined as 48.1%.

Despite the fact that FPCA is recognized as a normal anatomical variant, its existence can change the distribution and severity of cerebral damage in thromboembolic events. Some studies were reported, in which some patients having acute ischemic stroke and concurrent or almost simultaneous PCA-ICA area infarction, were detected ipsilateral FPCA in paradoxical PCA occlusion.^{33,34} In

our study, 13 cases (5.9%) were diagnosed with unilateral FPCA anomaly, 1 case (0.5%) was diagnosed with bilateral fetal PCA anomaly. 206 cases (93.6%) were not diagnosed with FPCA anomaly. 4 (30.8%) of 13 cases having unilateral FPCA, were diagnosed with anterior circulation infarct, 3 cases (23.1%) were diagnosed with posterior circulation infarct, and in 6 cases (46.2%) there were no ischemic stroke indications. In total, in 53.8% of cases having FPCA anomaly, there were concurrent ischemic stroke indications. FPCA can increase the dimension and severity of anterior circulation strokes by providing additional infarction in PCA area. Therefore clinicians should carefully evaluate the stroke risk factors of individuals who have FPCA, such as ICA stenosis and atrial fibrillation.

CONCLUSIONS AND SUGGESTIONS

In our study, the lower number and statistical insignificance of concurrent severe ICA stenosis in cases who have ischemic stroke concomitant with FPCA, hypoplastic / aplastic PCoA and hypoplastic / aplastic ACA A1 segment variations, brings the question to mind whether these variations are independent risk factors or not. But still it is not determined that the variations in CW morphology can increase the general stroke risk, which is independent from other risk factors. In conclusion, even though these variations' role in optimum stroke prophylaxis in individuals having one or more stroke risk factors, has not determined

yet, there is no doubt that clinical researchers. will eliminate these uncertainties in future studies.

Conflict of Interest

Author Halil AY declares that he has no conflict of interest.

Thanks

I would like to thank the late Dr. Dilek Şen Dokumacı for her great efforts in this study, and I pray to God that she rests in peace.

REFERENCES

1. Willis, C. A. D. T. (1997). "The Origins of Clinical Neuroscience". Cambridge: Plumridge.
2. Krishnaswamy, A, Klein, J. P, and Kapadia, S. R. (2010). "Clinical cerebrovascular anatomy". Catheterization and Cardiovascular Interventions, 75(4), 530-539.
3. Mull, M, Schwarz, M, and Thron, A. (1997). "Cerebral hemispheric low-flow infarcts in arterial occlusive disease: lesion patterns and angiomorphological conditions". Stroke, 28(1), 118-123. <https://doi.org/10.1161/01.STR.28.1.118>
4. Miralles, M, Dolz, J. L, Cotillas, J, Aldoma, J, Santiso, M. A, Gimenez, A, ... and Cairois, M. A. (1995). "The role of the circle of Willis in carotid occlusion: assessment with phase contrast MR angiography and transcranial duplex". European Journal of Vascular and Endovascular Surgery, 10(4), 424-430.
5. Drayer, B. P, and Djang, W. T. (1990). "Physiological considerations in extra and intracranial vascular disease. Radiology-diagnosis-imaging-intervention". Philadelphia: JB Lippincott Company, 1-10.
6. Hedera, P, Bujdakova, J, Traubner, P, and Pancak, J. (1998). "Stroke risk factors and development of collateral flow in carotid occlusive disease". Acta Neurologica Scandinavica, 98(3), 182-186. <https://doi.org/10.1111/j.1600-0404.1998.tb07291.x>
7. van Everdinge, K. J, Visser, G. H, Klijn, C. J. M, Kappelle, L. J, and Van der Grond, J. (1998). "Role of collateral flow on cerebral hemodynamics in patients with unilateral internal carotid artery occlusion". Annals of Neurology, 44(2), 167-176. <https://doi.org/10.1002/ana.410440206>
8. Alnæs, M. S, Isaksen, J, Mardal, K. A, Romner, B, Morgan, M. K, and Ingebrigtsen, T. (2007). "Computation of hemodynamics in the circle of Willis". Stroke, 38(9), 2500-2505. <https://doi.org/10.1161/STROKEAHA.107.482471>

9. de Boorder, M. J, van der Grond, J, van Dongen, A. J, Klijn, C. J, Jaap Kappelle, L, Van Rijk, P. P, and Hendrikse, J. (2006). "Spect measurements of regional cerebral perfusion and carbondioxide reactivity: correlation with cerebral collaterals in internal carotid artery occlusive disease". *Journal of Neurology*, 253, 1285-1291.
10. Arteaga, D. F, Strother, M. K, Davis, L. T, Fusco, M. R, Faraco, C. C, Roach, B. A, ... and Donahue, M. J. (2017). "Planning-free cerebral blood flow territory mapping in patients with intracranial arterial stenosis". *Journal of Cerebral Blood Flow and Metabolism*, 37(6), 1944-1958. <https://doi.org/10.1177/0271678X16657573>
11. Arjal, R. K, Zhu, T, and Zhou, Y. (2014). "The study of fetal-type posterior cerebral circulation on multislice CT angiography and its influence on cerebral ischemic strokes". *Clinical Imaging*, 38(3), 221-225.
12. Lv, X, Li, Y, Yang, X, Jiang, C, and Wu, Z. (2012). "Potential proneness of fetal-type posterior cerebral artery to vascular insufficiency in parent vessel occlusion of distal posterior cerebral artery aneurysms". *Journal of Neurosurgery*, 117(2), 284-287.
13. Van Raamt, A. F, Mali, W. P, Van Laar, P. J, and Van Der Graaf, Y. (2006). "The fetal variant of the circle of Willis and its influence on the cerebral collateral circulation". *Cerebrovascular Diseases*, 22(4), 217-224.
14. Diogo, M. C, Fragata, I, Dias, S. P, Nunes, J, Pamplona, J, and Reis, J. (2017). "Low prevalence of fetal-type posterior cerebral artery in patients with basilar tip aneurysms". *Journal of NeuroInterventional Surgery*, 9(7), 698-701.
15. Kolukisa, M, Gürsoy, A. E, Kocaman, G, Dürüyen, H, Toprak, H, and Asil, T. (2015). "Carotid endarterectomy in a patient with posterior cerebral artery infarction: influence of fetal type PCA on atypical clinical course". *Case Reports in Neurological Medicine*, 2015.
16. Hoksbergen, A. W. J, Legemate, D. A, Csiba, L, Csati, G, Siro, P, and Fülesdi, B. (2003). "Absent collateral function of the circle of Willis as risk factor for ischemic stroke". *Cerebrovascular Diseases*, 16(3), 191-198.
17. Zhou, H, Sun, J, Ji, X, Lin, J, Tang, S, Zeng, J, and Fan, Y. H. (2016). "Correlation between the integrity of the circle of Willis and the severity of initial noncardiac cerebral infarction and clinical prognosis". *Medicine*, 95(10).
18. Harrison, M. J, and Marshall, J. O. H. N. (1988). "The variable clinical and CT findings after carotid occlusion: the role of collateral blood supply". *Journal of Neurology, Neurosurgery and Psychiatry*, 51(2), 269-272.
19. Alpers, B. J, and Berry, R. G. (1963). "Circle of Willis in cerebral vascular disorders: the anatomical structure". *Archives of Neurology*, 8(4), 398-402.
20. Puchades-Orts, A, Nombela-Gomez, M, and Ortuño-Pacheco, G. (1976). "Variation in form of circle of Willis: some anatomical and embryological considerations". *The Anatomical Record*, 185(1), 119-123.
21. Waaijer, A, Van Leeuwen, M. S, Van der Worp, H. B, Verhagen, H. J. M, Mali, W. P. T. M, and Velthuis, B. K. (2007). "Anatomic variations in the circle of Willis in patients with symptomatic carotid artery stenosis assessed with multidetector row CT angiography". *Cerebrovascular Diseases*, 23(4), 267-274. <https://doi.org/10.1159/000098326>
22. Wholey, M. W. A, Nowak, I, and Wu, W. C. L. (2009). "CTA and the Circle of Willis. Early use of multislice CTA to evaluate the distal internal carotid artery and the Circle of Willis and their correlation with stroke". *Endovascular Today*, 7, 33-44.
23. van der Lugt, A, Buter, T. C, Govaere, F, Siepmann, D. A. M, Tanghe, H. L. J, and Dippel, D. W. J. (2004). "Accuracy of CT angiography in the assessment of a fetal origin of the posterior cerebral artery". *European Radiology*, 14, 1627-1633. <https://doi.org/10.1007/s00330-004-2333-1>
24. Jayaraman, M. V, Mayo-Smith, W. W, Tung, G. A, Haas, R. A, Rogg, J. M, Mehta, N. R, and Doberstein, C. E. (2004). "Detection of intracranial aneurysms: multi-detector row CT angiography compared with DSA". *Radiology*, 230(2), 510-518. <https://doi.org/10.1148/radiol.2302021465>
25. Liebeskind, D. S, and Caplan, L. R. (2016). "Intracranial arteries-anatomy and collaterals". *Intracranial Atherosclerosis: Pathophysiology, Diagnosis and Treatment*, 40, 1-20. <https://doi.org/10.1159/000448264>
26. Zaninovich, O. A, Ramey, W. L, Walter, C. M, and Dumont, T. M. (2017). "Completion of the circle of Willis varies by gender, age, and indication for computed tomography angiography". *World Neurosurgery*, 106, 953-963.
27. Kluytmans, M, Van der Grond, J, Van Everdingen, K. J, Klijn, C. J. M, Kappelle, L. J, and Viergever, M. A. (1999). "Cerebral hemodynamics in relation to patterns of collateral flow". *Stroke*, 30(7), 1432-1439. <https://doi.org/10.1161/01.STR.30.7.1432>
28. Chuang, Y. M, Liu, C. Y, Pan, P. J, ve Lin, C. P. (2007). "Anterior cerebral artery A1 segment hypoplasia may contribute to A1 hypoplasia syndrome". *European Neurology*, 57(4), 208-211.
29. Gerstner, E, Liberato, B, ve Wright, C. B. (2005). "Bi-hemispheric anterior cerebral artery with drop attacks and limb shaking TIAs". *Neurology*, 65(1), 174-174.
30. Mäurer, J, Mäurer, E, ve Perneczky, A. (1991). "Surgically verified variations in the A1 segment of the anterior cerebral artery: report of two cases". *Journal of Neurosurgery*, 75(6), 950-953. <https://doi.org/10.3171/jns.1991.75.6.0950>
31. Kovač, J. D, Stanković, A, Stanković, D, Kovač, B, ve Šaranović, D. (2014). "Intracranial arterial variations: a comprehensive evaluation using CT angiography". *Medical Science Monitor: International Medical Journal of Experimental and Clinical Research*, 20, 420.
32. Yang, J. H, Choi, H. Y, Nam, H. S, Kim, S. H, Han, S. W, ve Heo, J. H. (2007). "Mechanism of infarction involving ipsilateral carotid and posterior cerebral artery territories". *Cerebrovascular Diseases*, 24(5), 445-451.
33. Eswaradass, P. V, Ramasamy, B, Ramadoss, K, ve Gnanashanmugham, G. (2015). "Can internal carotid artery occlusion produce simultaneous anterior and posterior circulation stroke?". *Indian Journal of Vascular and Endovascular Surgery*, 2(3), 130-132.