

Prevalence and Pattern of Cerebral Variations during Endovascular Diagnostic Angiography in Saudi Patients: A Retrospective Observational Study

Ibrahim A. Almulhim¹, Bothaina Mohammed Abdulshakour², Atif Hussian Alkhlaqi³, Omar Ahmed Mohammed Alkhairi⁴, Hatim Mustafa Almasri⁵, Basem Mohammad Almasri⁶, Basem Hamid Almalki⁶, Dania Mohammed Ali Alharbi⁷, Amani Amjad kemawi⁷, Dalal Abdullah Hawsawi⁸, Anwaar Mosa Alhawsawi⁹, Waleed Ibrahim Sendi¹⁰, Daniah Ibrahim Bakhsh⁶, Afnan Saleh Hawsawi¹¹

¹Neuroradiology consultant, King Fahad Medical City- Riyadh, Saudi Arabia.

²Radiology Consultant, Makkah Health Cluster, Saudi Arabia.

³Medical physics specialist, Radiology Department, M. K. F. H, Saudi Arabia.

⁴Specialist - radiological technology, medical services outputs department - Directorate of Health Affairs Makkah Region, Saudi Arabia.

⁵Technician - radiological technology, medical services outputs department - Directorate of Health Affairs Makkah Region, Saudi Arabia.

⁶Radiological Technician, Makkah Health Cluster, Saudi Arabia.

⁷Radiology, Al-Azizia Children Hospital, Saudi Arabia.

⁸Radiology, Polyclinic Alawali Center, Makkah, Saudi Arabia.

⁹Radiology, King Fahad general hospital, Saudi Arabia.

¹⁰Radiological Technician, MOH MAKKAH, Saudi Arabia.

¹¹Radiographer, King Fahd Hospital in Almdinah Almonorh, Saudi Arabia.

Abstract

Background: Previous studies detected large number of variations in circle of Willis in normal populations. There is limited data for the effect and correlation of normal varieties and pathologic vascular anomalies

Purpose: Prevalence of normal variants and impact of its presence in decision making process.

Patients and methods: Ninety patients' records underwent therapeutic angiography were retrospectively reviewed and reallocated into two equal groups according to pathology (group 1=vascular malformation group, group 2= ischemic insults). Age, sex, type of pathology, site of normal variants and finally their impact on decision making process were calculated.

Results: The mean and standard deviation of age in our study was found to be (40.18 years \pm 15.6), most of patients were between 30-40 years old. The total number of normal variants that were detected in our study either at extra- or intracranial vascular tree was 46 variants. Forty-nine cases were "ignored" or added no impact on the proposed plan during neurointervention. The sensitivity of normal variants in changing decision making process was seen in 15% of cases while the specificity was 100%.

Conclusion: Normal variants were detected accidentally through DSA for therapeutic purposes in 47% of cases. The term normal vascular variation is simple to be defined per se. However, when additional vascular event (aneurysm or AVM) was detected, the neurointerventionist should be aware of its presence, possible complications and how to proceed to the target without additional damage. These variations should be reported and explained to the patient and/or family especially when they interfere with operative plan or decision to avoid medicolegal consequences.

Keywords: DSA, normal variations, variants, aneurysm, COW

Introduction

Since the description of the arterial anatomy by Thomas Willis in 1664, many variations were described in the literature (1–4). The classical circle of Willis (COW) was seen only in less than 40% of populations (5–7). Vessels are known to exhibit anatomical variations more frequently than bone, ligaments, nerves and muscles (8,9). Different patterns of variations were described. These included aplasia, hypoplasia, duplication, fenestration and trifurcation(10). These normally found variations are benign course in its own. They may detected simultaneously with AVM or aneurysm in the same territory or apart from it. For example, presence of aplasia or hypoplasia of a parent vessel may produce a hemodynamic stress on an aneurysm which is already detected or produce an ischemic attacks thereafter (5,11,12).

There are two important questions were raised by authors. First; are these normal variations associated with vascular malformations (AVM or aneurysms). Second, could these normal variations change the decision of the interventionists by trying different route, different instrumentations or change the length of the procedure?

Patients and Method

Study Population and Sample Size

It is a retrospective, observational and controlled study started from July 2020 to November 2020. This study included 90 patients undergoing diagnostic and therapeutic catheter angiography complaining of symptoms and signs of, ICH or manifestations due to aneurysm or arteriovenous malformation (AVM) referred from ER or outpatient clinic for doing urgent angiography at our institute. After approval of the ethics committee of researches and obtaining written consent from all patients scheduled for digital subtraction angiography.

Study groups

All patients were reallocated within group 1 or group 2 as follows:

1. Group 1: 45 patients with AVM and aneurysm with inclusion and exclusion criteria described below.
2. Group 2: 45 patients with known ischemia or TIA with no aneurysm or AVM during routine pre-angiographic tests.

The first group is representative of cases group while the second one is the representative of control group.

Inclusion and exclusion criteria

The main inclusion criteria were; age above 18 years and presence of symptoms and signs of chronic ischemia, ICH, fits or disturbance of conscious level that indicate performing DSA later on. History of recent trauma and/or neurosurgical procedure were regarded as exclusion criteria.

Techniques

Conventional endovascular diagnostic angiography should be performed to all patients referred from neurology or neurosurgery ER/outpatient clinic.

Definition of terms

There are two important definitions; normal variations and change of decision. The definition of normal variation is any change in the anatomical architecture of the cerebral vessels that carry no hazards to the neural tissue. Change in the decision is defined as any deviation in time, change of procedure and usage of different instrumentations.

Statistical analysis

The statistical analysis was done by the Statistical Package of Social Sciences version 25 (Chicago, IL, USA). Categorical data were presented as percentages and compared using Chi-square t-test. Numerical data were presented as mean and standard deviation and compared by using Student's t-test. P value below 0.05 were regarded statistically significant.

Results*Patients' Criteria*

The mean and standard deviation of age in our study was found to be (40.18 years \pm 15.6), most of patients were between 30-40 years old (Figure 1). Males constituted 42.2% (38) pf patients. Patients' criteria are well plotted in Table 1. There was no statistically significant difference between groups as regard age (p=0.145). Aneurysms was detected in 24 patients (53.3%) while AVM were detected in 21 (46.7%) of patients in group 1.

Table 1: Patients' criteria

		Groups				Total		Chi-square	
		Group 1		Group 2					
		N	%	N	%	N	%	X ²	P-value
Age	<30	15	33.3%	8	17.8%	23	25.6%	3.378	0.497
	30-40	13	28.9%	13	28.9%	26	28.9%		
	40-50	8	17.8%	11	24.4%	19	21.1%		
	50-60	5	11.1%	7	15.6%	12	13.3%		
	>60	4	8.9%	6	13.3%	10	11.1%		
Sex	Female	24	53.3%	28	62.2%	52	57.8%	0.730	0.393
	Male	21	46.7%	17	37.8%	38	42.2%		
presentation	ICH	29	64.4%	18	40.0%	47	52.2%	22.823	*0.001

	Ischemia	2	4.4%	7	15.6%	9	10.0%		
	MASS	11	24.4%	6	13.3%	17	18.9%		
	Vasculitis	0	0.0%	9	20.0%	9	10.0%		
	Incidental	1	2.2%	1	2.2%	2	2.2%		
	CCF	1	2.2%	0	0.0%	1	1.1%		
	Other	1	2.2%	4	8.9%	5	5.6%		
Side	Right	1	2.2%	2	4.4%	3	3.3%	-	-
	Lift	1	2.2%	4	8.9%	5	5.6%	-	-
	Both	0	0.0%	1	2.2%	1	1.1%	-	-
DM		1	2.2%	6	13.3%	7	7.8%	4.264	0.039
CVD		0	0.0%	2	4.4%	2	2.2%	2.818	0.093
HTN		7	15.6%	8	17.8%	15	16.7%	0.080	0.881
Smoking		0	0.0%	0	0.0%	0	0.0%	-	-
SCD		0	0.0%	2	4.4%	2	2.2%	2.818	0.093

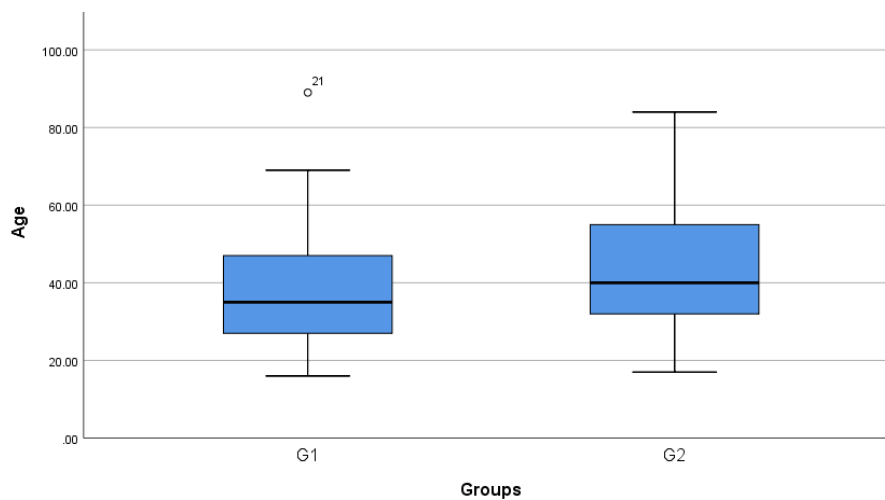


Figure 1: Boxplot graph for comparing mean of age at both groups.

Normal variants: incidence and types

The total number of normal variants that were detected in our study either at extra- or intracranial vascular tree was 58 variants in 43 patients (47%). The distribution of these variants are plotted in Figure 2. The distribution of intracranial variants is shown in Table 2 and Figure 3. By comparing events of both groups with Mann-Whitney test, it has been found that

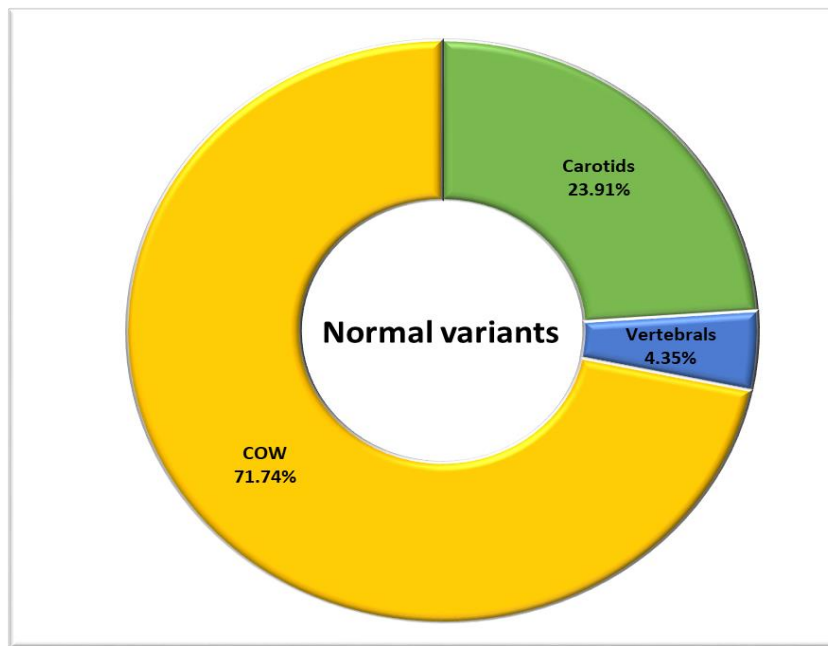


Figure 2: Normal variations in our study according to area.

There was a statistically significant difference in events between two groups ($p=0.0001$) with superior results to group 1 (vascular malformation group). The most common variant was fetal origin of PCA followed by hypoplastic A1.

Table 2: Frequency of normal variants in both groups.

	Groups				Total		Chi-square	
	G1		G2		N	%	X ²	P-value
	N	%	N	%				
Presence of NV	43	95.6%	15	33.3%	58	64.4%	43.497	0.0001*
Fetal Origin of PCA	7	15.6%	6	13.3%	13	14.4%	0.090	0.764
Hypoplastic A1	6	13.3%	2	4.4%	8	8.9%	2.288	0.130
Vertebral artery Fenestration	2	4.4%	0	0.0%	2	2.2%	2.818	0.093
Hypoplastic P1	1	2.2%	2	4.4%	3	3.3%	0.351	0.553
Persistent trigeminal	1	2.2%	0	0.0%	1	1.1%	1.398	0.237
Persistent Hypoglossal	0	0.0%	2	4.4%	2	2.2%	2.818	0.093
ICA origin accessory middle meningeal artery	1	2.2%	0	0.0%	1	1.1%	1.398	0.237
Hypoplastic vertebral terminating as PICA	0	0.0%	1	2.2%	1	1.1%	1.398	0.237
Origin of vertebral artery from the CCA	0	0.0%	2	4.4%	2	2.2%	2.818	0.093

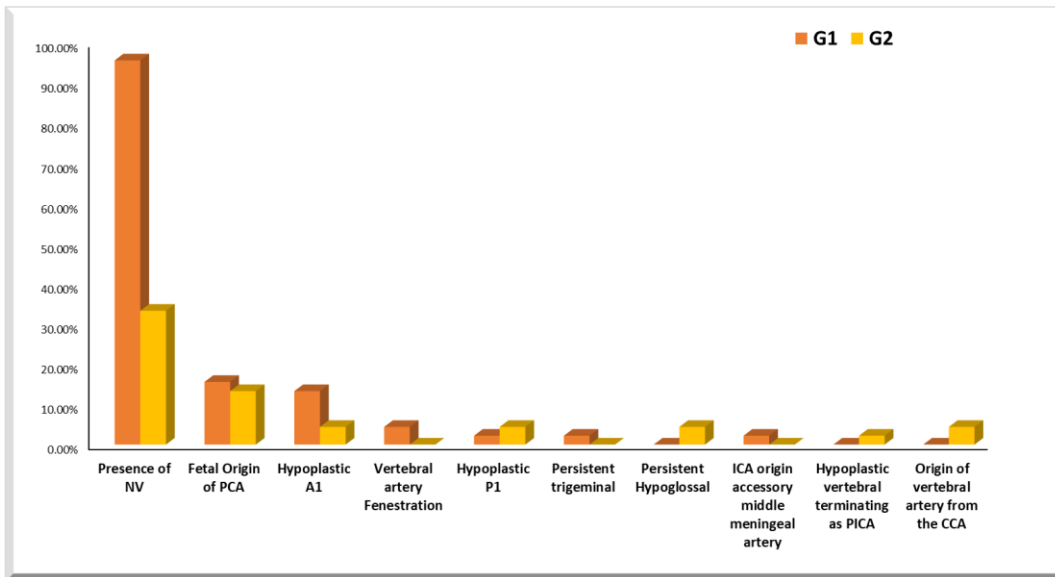


Figure 3: Bar chart of types of anomalies in our study.

By comparing the following variables (sex and age) versus presence of normal variants detected , it was found that neither age nor sex were of statistically significant correlation with the event ($p=0.828, 0.124$) respectively (see Figure 4 & 5).

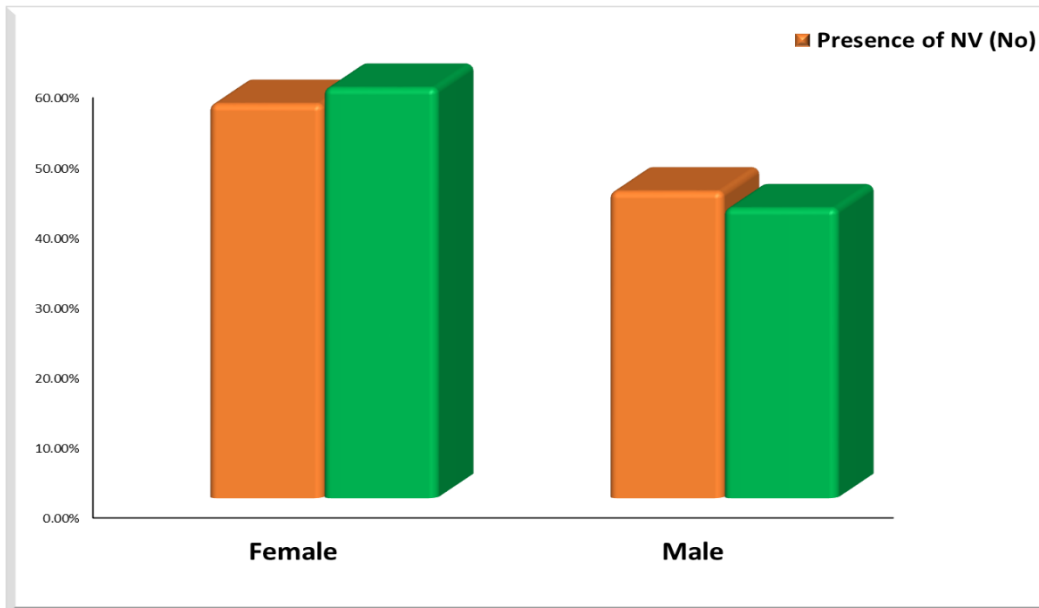


Figure 4: Impact of sex on normal variants.

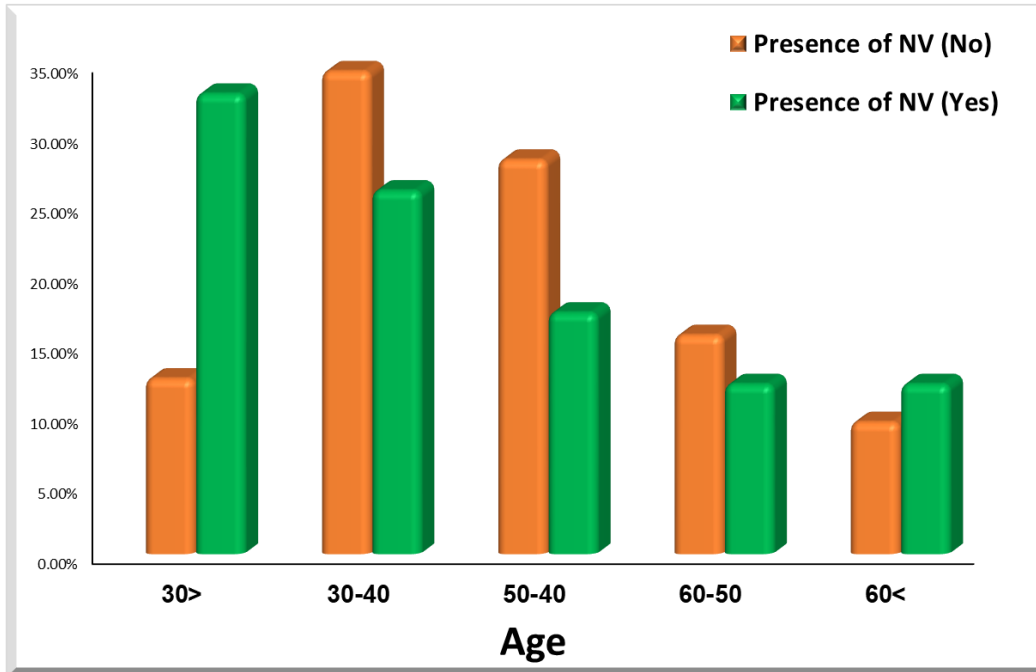


Figure 5: Age distribution in normal variants cases and no variants.

Impact of normal variants' presence on decision making process

The presence of a normal variations during diagnostic angiography changed the pathway of a therapeutic angiography in minority of cases (9 cases) as shown in Table 3 and Figure 6. However, 49 cases were "ignored" or added no impact on the proposed plan during neurointervention. In Table 4, the diagnostic indexes of the normal variants' presence were plotted, the sensitivity of normal variants in changing decision making process was seen in 15% of cases while the specificity was 100%.

Table 3:

			Presence of NV		Total	Chi-square	
			No	Yes		X ²	P-value
Decision	No	N	32	49	81	8.452	<0.001
		%	35.6%	54.4%	90.0%		
	Yes	N	0	9	9		
		%	0.0%	10.0%	10.0%		
Total		N	32	58	90		
		%	35.6%	64.4%	100.0%		

Table 4: Diagnostic index of normal variants presence in decision making process of therapeutic angiography.

Statistic	Value	95% CI
Sensitivity	15.52%	7.35% to 27.42%

Specificity	100.00%	89.11% to 100.00%
Disease prevalence (*)	51.00%	
Positive Predictive Value (*)	100.00%	
Negative Predictive Value (*)	53.21%	50.46% to 55.94%
Accuracy (*)	45.56%	38.45% to 52.67%

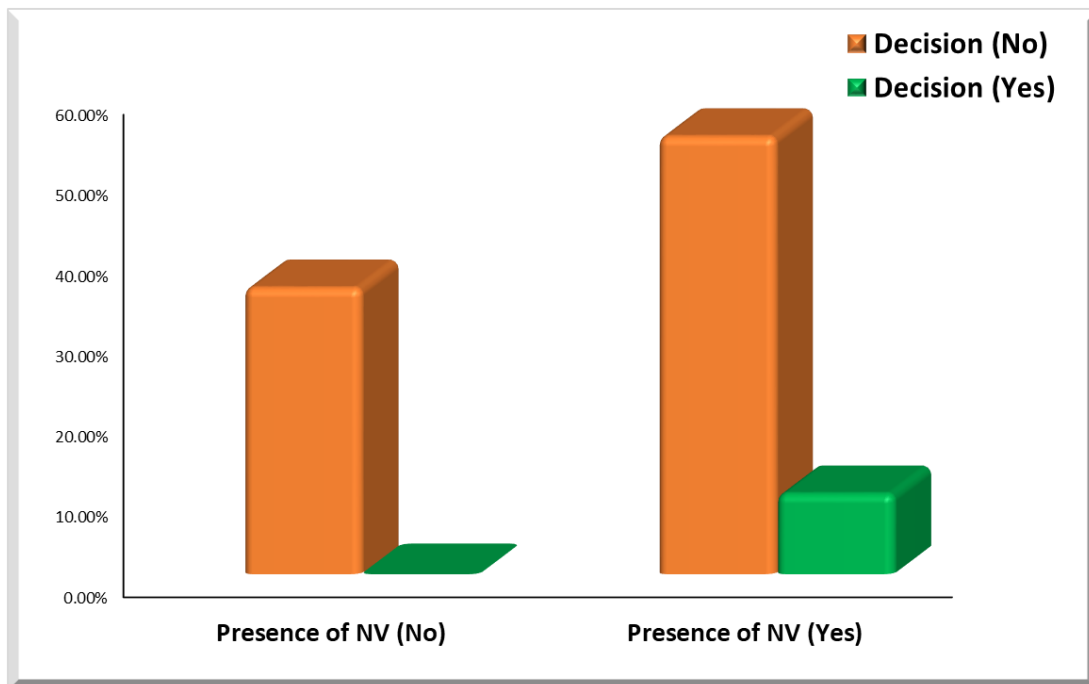


Figure 6: Bar chart of decision taken in cases of normal variation.

Discussion

Prevalence of normal variations in our study at both extra- and intracranial vasculature levels were found in 58 in 43 patients (47%). Most of these findings were suffering from acute adverse event of a vascular malformation disease (AVM or aneurysm). Indeed, our study showed that a presence of a normal variant may or may not abrupt the plan of therapeutic angiography (only 9 cases). In our study, the complete pattern of circle of Willis was found in 32 patients (35.6%). Hashemi et al found a complete circle in around 35% of cases (13). Klimek-Piotrowska and colleagues surveyed 250 patients with a high resolution CT angiography and they found complete circle in less than 20% of cases (14). In contrast, anatomical study did by Sinha and coworkers found a complete, normal caliber and symmetrical circle in 77.5% of specimens (15). Therefore, the pattern of complete circle may be geographically varied. In many literatures, the most common variant was a hypoplastic P1 (4,7,10,16), however, A1 hypoplasia following fetal PCA were the most common variants. In Kovac et al study, the most common location was the vertebrobasilar system with 5 (1.1%) fenestrations (8). The incidence of A1 hypoplasia was found to be 36% of cases (17). A study on anterior cerebral artery by using magnetic resonance angiography (MRA) showed that aplasia rate was 5.6% (18). In contrast, Iqbal et al (19) stated a hypoplasia in PcomA rate of 24% that was followed by hypoplasia in the P1, A1 and AcomA segments. Similarly, Karatas et al.(20) found hypoplasia most commonly in the PcomA. Certain types of vascular malformations may encountered with normal variant presence, for example AcomA aneurysm (17). This finding was also recorded by Suzuki (21) and van Rooij (22). In a study that assessed anatomic variations of patients undergoing coil-embolization with aneurysms, A1 dominance on one side with AcomA aneurysms was found up to 70%. A1 dominant flow was shown to act in aneurysm formation, growth and instability after coil embolization treatment (18,23–25). Kryzewski et al. (26) suggested that A1 and A2 segment anomalies of the anterior cerebral artery may potentially be associated with aneurysm formation. Although the clinical significance of ACA variations is usually minor, an associated aneurysm is found relatively frequently(9,26–28). Till this moment, many articles discussed the impact of a single variant on the clinical outcome of a specific type of events (e.g. aneurysm). In our study, the fetal origin of PCA was detected in 14.4% of cases. Normally, the PCA is origination from the bifurcation of basilar artery (posterior circulation). In fetal type, the PCA is origination from internal carotid artery instead (29). Fetal origin of PCA was regarded as risk factor for poor outcome in Pcom aneurysm natural history (30). Zanaty and colleagues reported a case of failure pipeline embolization due to fetal PCA (31). Another variant is hypoplasia of A1, in our study, the prevalence of this variation was seen in 8.9% of cases. This finding is used to be harmful when coincide with Acom aneurysm due to three causes. First, hypoplasia of A1 produces asymmetry in vascular tree which in turn exhibit a hemodynamic stress on the Acom artery (32,33).

Experimental ligation of common carotid artery in hypertensive rats produces Acom aneurysm (34). Second, the growth of the aneurysm. Many literatures found a linkage between AcomA and A1 hypoplasia (35–37). Third, treatment outcome. the possible role of A1 hypoplasia in the treatment success of Acom aneurysm has also been debated (35,38,39). In particular, A1 hypoplasia carriers show an increased risk of aneurysm recurrence following coil embolization (36,37). In support of the clinical importance of A1 hypoplasia for the endovascular treatment of Acom aneurysms, there was a higher risk of intra- procedural complications during Acom aneurysm coiling in SAH patients with A1 hypoplasia (32).

Conclusion

Normal variants were detected accidentally through DSA for therapeutic purposes in 47% of cases. The term normal vascular variation is simple to be defined per se. However, when additional vascular event (aneurysm or AVM) was detected, the neurointerventionist should be aware of its presence, possible complications and how to proceed to the target without additional damage. These variations should be reported and explained to the patient and/or family especially when they interfere with operative plan or decision to avoid medicolegal consequences.

Conflict of interest

There is no conflict of interest.

References

1. Vishruth R, Chennamaneni V, Ramesh T. Incidence of Normal variants of the cerebral circulation at 128 slice computed tomography angiography. *Perspectives in Medical Research*. 2015;(July):17–21.
2. Akgun V, Battal B, Bozkurt Y, Oz O, Hamcan S, Sari S, et al. Normal anatomical features and variations of the vertebrobasilar circulation and its branches: An analysis with 64-detector row CT and 3T MR angiographies. *The Scientific World Journal*. 2013;2013.
3. Bastos Soledade LE, Masuda H. Normal Variants of the Cerebral Circulation at Multidetector CT Angiography. *RadioGraphics*. 2009;29(212):1027–43.
4. Karatas A, Coban G, Cinar C, Oran I, Uz A. Assessment of the Circle of Willis with Cranial Tomography Angiography. *Medical science monitor: international medical journal of experimental and clinical research*. 2015;21:2647–52.
5. Setacci C, Sirignano P, de Donato G, Setacci F. Anomalies and Variant Anatomy of the Aorta and the Supra- aortic Vessels. *Thoraco-Abdominal Aorta*. 2011;585–93.
6. Alawad AH, Hussein MA, Hassan MA. Morphology and normal variations of the Cerebral Arterial Circle “of Willis” in Khartoum Diagnostic Centre. *Khartoum Medical Journal*. 2012;2(2):215–8.
7. Saikia B, Handique A, Phukan P, Lynser D, Sarma A. Circle of Willis: Variant Forms and Their Embryology Using Gross Dissection and Magnetic Resonance Angiography. *Int J Anat Res*. 2014;2(2):344–53.
8. Kovač JD, Stanković A, Stanković D, Kovač B, Šaranović D. Intracranial arterial

- variations: A comprehensive evaluation using CT angiography. *Medical Science Monitor*. 2014;20:420–7.
9. Machasio RM, Nyabanda R, Mutala TM. Proportion of Variant Anatomy of the Circle of Willis and Association with Vascular Anomalies on Cerebral CT Angiography. *Radiology Research and Practice*. 2019;2019:1–7.
 10. Dimmick SJ, Faulder KC. Normal variants of the cerebral circulation at multidetector CT angiography. *Radiographics*. 2009;29(4):1027–43.
 11. Alawad A, Hussein M, Hassan M. Morphology and normal variations of the cerebral arterial circle “of Willis” in Khartoum Diagnostic Centre. *Khartoum Medical Journal*. 2012;2(2):215–9.
 12. Tapia GP, Zhu X, Xu J, Liang P, Su G, Liu H, et al. Incidence of branching patterns variations of the arch in aortic dissection in Chinese patients. *Medicine*. 2015;94(17):e795.
 13. Hashemi SM, Mahmoodi R, Amirjamshidi A. Variations in the Anatomy of the Willis' circle: A 3-year cross-sectional study from Iran (2006-2009). Are the distributions of variations of circle of Willis different in different populations? Result of an anatomical study and review of literature. *Surgical Neurology International*. 2013;4(1):65.
 14. Klimek-Piotrowska W, Kopeć M, Kochana M, Krzyżewski RM, Tomaszewski KA, Brzegowy P, et al. Configurations of the circle of Willis: a computed tomography angiography based study on a Polish population. *Folia Morphologica*. 2013 Dec 4;72(4):293–9.
 15. Sinha I, Ghosal AK, Basu R, Dutta I. Variation in the pattern of circle of willis in human brain –A morphological study and review. *Al Ameen J Med Sc*. 2014;7(1):4–1.
 16. Bahaddur A, Chandan G. Anatomical Variants of Circle of Willis in South Indian Population: A Study by Using Magnetic Resonance Angiography. *International Journal of Science and Research (IJSR) ISSN (Online Index Copernicus Value Impact Factor)*. 2013;14(5):2319–7064.
 17. Orakdogan M, Emon ST, Somay H, Engin T, Is M, Hakan T. Vascular variations associated with intracranial aneurysms. *Turkish Neurosurgery*. 2017;27(6):853–62.
 18. Uchino A, Nomiyama K, Takase Y, Kudo S. Anterior cerebral artery variations detected by MR angiography. *Neuroradiology*. 2006 Sep 20;48(9):647–52.
 19. Iqbal S. A comprehensive study of the anatomical variations of the circle of Willis in adult human brains. *Journal of Clinical and Diagnostic Research*. 2013;7(11):2423–7.
 20. Karatas A, Yilmaz H, Coban G, Koker M, Uz A. The anatomy of circulus arteriosus cerebri (circle of willis): A study in Turkish population. *Turkish Neurosurgery*. 2016;26(1):54–61.
 21. Suzuki M, Onuma T, Sakurai Y, Mizoi K, Ogawa A, Yoshimoto T. Aneurysms arising from the proximal (A1) segment of the anterior cerebral artery. *Journal of*

- Neurosurgery. 1992 Mar;76(3):455–8.
22. van Rooij SBT, Bechan RS, Peluso JP, Sluzewski M, van Rooij WJ. Fenestrations of Intracranial Arteries. *American Journal of Neuroradiology*. 2015 Jun;36(6):1167–70.
 23. Tanaka H, Fujita N, Enoki T, Matsumoto K, Watanabe Y, Murase K, et al. Relationship between variations in the circle of Willis and flow rates in internal carotid and basilar arteries determined by means of magnetic resonance imaging with semiautomated lumen segmentation: Reference data from 125 healthy volunteers. *American Journal of Neuroradiology*. 2006;27(8):1770–5.
 24. Saikia B, Handique A, Phukan P, Lynser D, Jamil M. Study of anomalies in the circle of Willis using magnetic resonance angiography in north eastern India. *Journal of the Anatomical Society of India*. 2014 Jun;63(1):67–73.
 25. Saha A, Bhagyalakshmi B, Mandal S, Banopadhyaya M, Medinipur P, Bengal W, et al. Variation of Posterior Communicating Artery in Human Brain: a Morphological. 2013;11(1).
 26. Krzyzewski RM, Tomaszewska IM, Lorenc N, Kochana M, Goncerz G, Klimek-Piotrowska W, et al. Variations of the anterior communicating artery complex and occurrence of anterior communicating artery aneurysm: A2 segment consideration. *Folia medica Cracoviensia*. 2014;54(1):13–20.
 27. Lee JH, Choi CG, Kim DK, Kim GE, Lee HK, Suh DC. Relationship between Circle of Willis Morphology on 3D Time-of-Flight MR Angiograms and Transient Ischemia during Vascular Clamping of the Internal Carotid Artery during Carotid Endarterectomy. *American Journal of Neuroradiology*. 2004;25(4):558–64.
 28. Makowicz G, Poniatowska R, Lusawa M. Variants of cerebral arteries - Anterior circulation. *Polish Journal of Radiology*. 2013;78(3):42–7.
 29. Bisaria KK. Anomalies of the posterior communicating artery and their potential clinical significance. *Journal of Neurosurgery*. 1984 Mar;60(3):572–6.
 30. Wentland AL, Rowley HA, Vigen KK, Field AS. Fetal Origin of the Posterior Cerebral Artery Produces Left-Right Asymmetry on Perfusion Imaging. *American Journal of Neuroradiology*. 2010 Mar;31(3):448–53.
 31. Zanaty M, Chalouhi N, Starke RM, Jabbour P, Ryken KO, Bulsara KR, et al. Failure of the Pipeline Embolization Device in Posterior Communicating Artery Aneurysms Associated with a Fetal Posterior Cerebral Artery. *Case Reports in Vascular Medicine*. 2016;2016(Figure 1):1–4.
 32. Jabbarli R, Reinhard M, Roelz R, Kaier K, Weyerbrock A, Taschner C, et al. Clinical relevance of anterior cerebral artery asymmetry in aneurysmal subarachnoid hemorrhage. *Journal of Neurosurgery*. 2017;127(5):1070–6.
 33. Eissa MA, Ganna A, Amer M, Shakal A. Angiographic Study of the Anatomical Variations of the Anterior Communicating Artery Complex. 2020;32(20):97–101.
 34. Hashimoto N, Handa H, Nagata I, Hazama F. Experimentally induced cerebral

- aneurysms in rats: Part V. Relation of hemodynamics in the circle of Willis to formation of aneurysms. *Surgical neurology*. 1980 Jan;13(1):41–5.
35. Songsaeng D, Geibprasert S, Willinsky R, Tymianski M, TerBrugge KG, Krings T. Impact of anatomical variations of the circle of Willis on the incidence of aneurysms and their recurrence rate following endovascular treatment. *Clinical Radiology*. 2010 Nov;65(11):895–901.
 36. Weil AG, Bojanowski MW, Scholtes F, Darsaut TE, Signorelli F, Weill A. Angiographic Pitfall: Duplicated Tapered A1 Segment of the Anterior Cerebral Artery Mimicking an Anterior Communicating Artery Aneurysm. *Interventional Neuroradiology*. 2011 Jun 1;17(2):179–82.
 37. Lazzaro MA, Ouyang B, Chen M. The role of circle of Willis anomalies in cerebral aneurysm rupture. *Journal of NeuroInterventional Surgery*. 2012 Jan;4(1):22–6.
 38. Uemura A, Kamo M, Matsukawa H. Angiographic outcome after endovascular therapy for anterior communicating artery aneurysms: correlation with vascular morphological features. *Japanese Journal of Radiology*. 2012 Oct 5;30(8):624–7.
 39. Gonzalez N, Sedrak M, Martin N, Vinuela F. Impact of Anatomic Features in the Endovascular Embolization of 181 Anterior Communicating Artery Aneurysms. *Stroke*. 2008 Oct;39(10):2776–82.