ANATOMICAL VARIATIONS IN THE COWS & THEIR ASSOCIATION WITH PATTERNS OF BRAIN INFARCT

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ABSTRACT

BACKGROUND

The COWs is a vascular anastomosis situated near the base of the brain.The association between structural changes in the COW& cerebral infarction is contentious. Aim of our study was “to evaluate the association between anatomical variations in the COWs & occurrence of different patterns of brain infarct”.

MATERIAL & METHODS

The present cross-sectional study was conducted at Department of Radiology of a tertiary care center among 500 patients of suspected cerebrovascular accidents during the study period of one year.Complete history of patients was taken. The following variations in the COW on MRA were recorded: pfPCA, cfPCA& aplasia or hypoplasia of PCoA.The statistical analysis was conducted using SPSS version 25.0.

RESULTS

240 (48%) had infarction while 260 (52%) patients did not have infarction.fPCA was present in 50 (20.8%) patients while aplasia or hypoplasia of communicating artery was present in 182 (75.8%) of patients.

Subgroup analysis of the infarcted territory indicated no significant difference between those with & without fPCA& hypoplasia of PCoA.No significant association was found between fPCA& hypoplasia of PCoA& clinical danger factors of CVD in patients with infarction.

CONCLUSION

This study demonstrated no significant relationship between particularchanges in the COW but gives a sign of vulnerability to silent cerebral vascular infarction.

KEYWORDS: Brain Infarction, Cerebral Artery, Cerebrovascular Disease,COWs, Communicating Artery, MRAngiogram.

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INTRODUCTION

The human brain with its intricate circulatory system might be seen as a "three-pound biological computer". It is abundantly vascularized, receiving oxygen & other vital nutrients.[1]The COWs (COW) is a vascular anastomosis system that links the internal carotid arteries & the vertebral arteries. It is situated near the brain's base & constitutes the principal intracranial collateral circulation. The COW is an anatomically variable structure. Anatomical variations may not directly affect brain perfusion; nonetheless, they may impact collateral capacity & heighten susceptibility to alterations in cerebral blood flow.[2-4]

Infarction in brain is a primary contributor to disability & the second leading cause of mortality globally.[5] Seventy-five percent of all fatalities & 81% of the total disability-adjusted life years lost owing to infarct transpire in developing nations.[6,7] Risk factors for infarct encompass age, sex, race, hypertension, diabetes, hyperlipidemia, dietary habits, tobaccouse, & alcohol consumption.[8] Common variations in the COWs include hypoplasia or aplasia of one or both posterior communicating arteries (PCoA) (34% to 68%), hypoplasia or aplasia of the A1 segment of the anterior cerebral artery (ACA) (4% to 10%), absence or fenestration of the anterior communicating artery (ACoA) (12% to 21%), persistent fetal origin of the posterior cerebral artery (fPCA) (4% to 26%), & infundibular dilatation or widening of the PCoA (7% to 15%).[9,10]

While prior studies have demonstrated a correlation between incomplete COW variations & ischemic stroke,[11] the association between COW variation & covert vascular brain injury remains inadequately clarified. Numerous studies indicate that an incomplete COWs (COW) correlates with more severe white matter hyperintensities (WMHs),[12-16] but others have not established a connection.[17-19]A recent meta-analysis indicated that patients with any anatomical variation in the COWs were 1.38 times more likely to experience ischemic stroke compared to those with a complete & intact COWs, concluding that there exists a positive association trend between variations in the COWs & ischemic stroke.[11] Limited research has examined the correlation between COW variations &cerebral microbleeds (CMBs) or enlarged perivascular spaces(EPVS).[18]

The aim of our research was “to evaluate the association between anatomical variations in the COWs & occurrence of different patterns of brain infarct”.

MATERIAL & METHODS

The present cross-sectional research was conducted at “Department of Radiology of a tertiary care center” among patients of suspected cerebrovascular accidents during the study period of one year. Before the study started, institutional ethics committee approval was obtained. After being fully informed about the procedure, patients were requested to sign an informed permission form.

Through consecutive sampling a total of 500 patients were selected onthe basis of inclusion & exclusion criteria.

Inclusion Criteria

1. Patients who are older than eighteen.

2. Individuals who had brain MRIs & MRAs to check for possible cerebrovascular accidents.
3. Patients who are open to taking part in the research.

Exclusion Criteria

1. Patients under the age of eighteen.
2. Individuals who have experienced head trauma, vasculitis, craniotomy or craniectomy pregnancy, vascular malformation, hemorrhagic infarction, significant stenosis or occlusion of the ICA, or significant stenosis or occlusion of the BA or its major branches.
3. Patients who refuse to take part in the research.

From the patients' computerized medical records, demographic information, current symptoms, & medical history were reviewed.A Siemens Magnetom Sempra 1.5-T was used for the scans. The examination, which included diffusion-weighted images (DWI), FLAIR, T1-weighted pictures, & T2-weighted images, was performed without the use of intravenous contrast.Images were obtained using the three-dimensional time-of-flight MRA method in three slabs, each including thirty slides, in order to evaluate the COWs. A maximum intensity projection technique was used to recreate MRA pictures in transverse oblique planes.

Variations in the COWs on MRA were noted as follows: PCoA aplasia or hypoplasia, as well as pfPCA&cfPCA.Based on MRI findings & clinical history, the injured side, vascular area, & age of the infarct were recorded. Based on the evaluating radiologists' agreement, the areas of diminished diffusion were qualitatively evaluated & categorized as acute or subacute infarct. Chronic infarcts were defined as areas of encephalomalacia or gliosis that showed volume loss & no concurrent restricted diffusion. When acute & chronic infarcts occurred at the same time, the acute infarct area was given priority. Anterior, posterior, thalamic, & watershed regions were used to classify the infarct's vascular distribution.

The findings were summarized by absolute frequencies & percentages for categorical variables & expressed as mean ± standard deviation (SD) for quantitative variables. The Chi-square was used to examine categorical variables when more than 20% of cells had an expected count of less than 5. The Mann-Whitney U test for nonparametric data & the Student's T test for parametric data were used to examine quantitative variables. SPSS version 25.0 for Windows was used to do the statistical analysis (SPSS Inc., Chicago, IL). P values were considered statistically significant if they were 0.05 or less.

RESULTS

Out of 500 patients 240 (48%) had infarction while 260 (52%) patients did not have infarction as shown n figure 1.

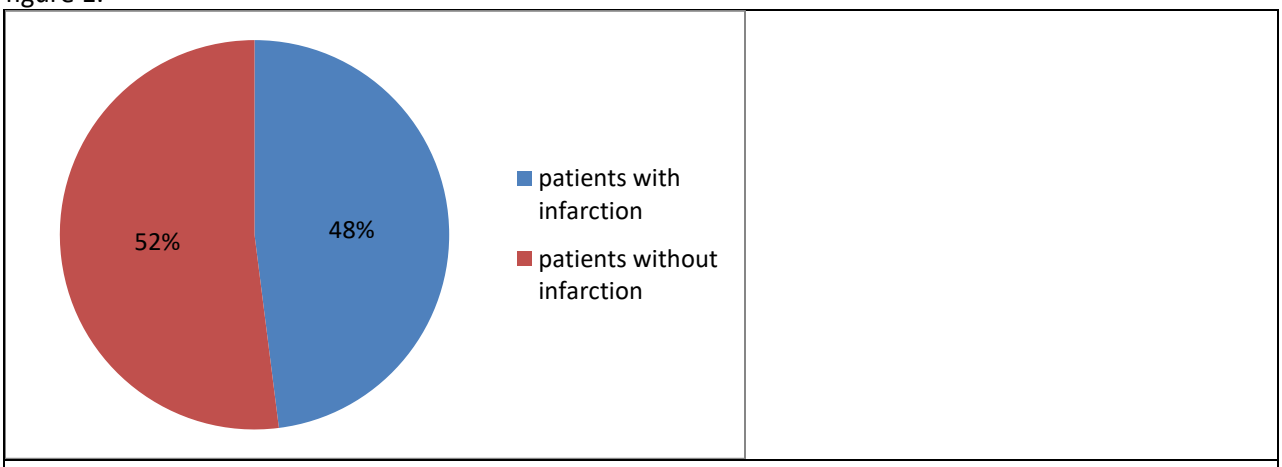

Figure 1: Prevalence of infarction in patients

In the infarction cohort, 108 (45%) experienced right-sided infarcts, while 132 (55%) suffered left-sided infarcts. Acute or subacute infarcts were observed in 180 (75%) of patients, while chronic infarcts, as the sole kind, were identified in 60 (25%) of patients as shown in figure 2.

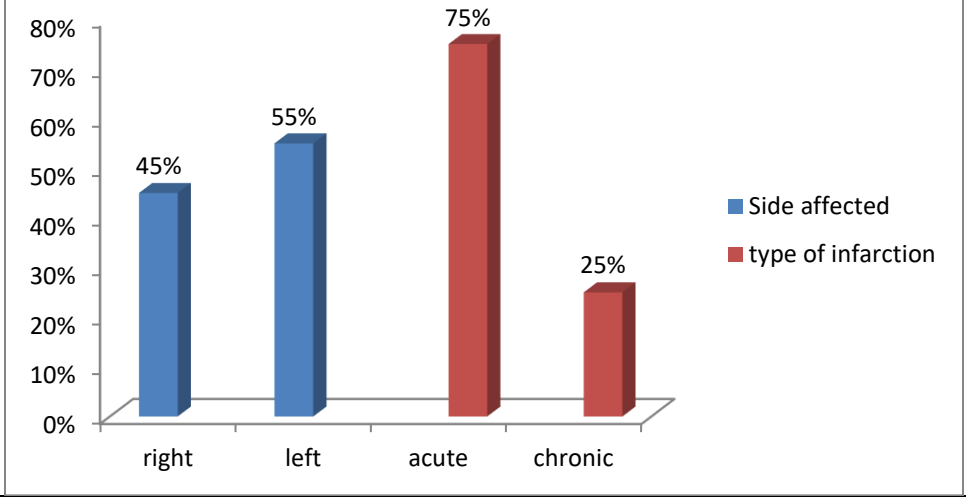

Figure 2: Distribution of patients on the basis of type of infarction & side affected

The mean age of patients with infarction was 65.2 years & those without infarction were 53.3 years. Numbers of male patients were higher than female patients in both the groups. Patients with infarction had higher rate of medical history as shown in Table 1.

Parameter	With infarction(n=240)	Without infarction(n=260)	P value
Mean age (years)	65.2±12.3	53.3±15.4	<0.001
Male	130 (54.1)	140 (53.8)	0.156
Female	110 (45.9)	120 (46.2)	0.158
Hypertension	175 (73)	124 (47.6)	<0.001
Diabetes mellitus	96 (40)	62 (23.8)	0.001
Hyperlipidemia	24 (10)	24 (9.2)	0.654
Smoking	36 (15)	24 (9.2)	0.035
Ischemic heart disease	72 (30)	42 (16.1)	0.007

Table 1: Distribution of CRF in patients with & without infarction

fPCA was present in 50 (20.8%) patients while aplasia or hypoplasia of posterior communicating artery was present in 182 (75.8%) of patients. 15 patients had ipsilateralfPCA whereas 35 patients had without ipsilateralfPCA& 110patients hadipsilateral hypoplasia of posterior communicating artery & 72 had without ipsilateral hypoplasia of posterior communicating artery as shown in figure 3.

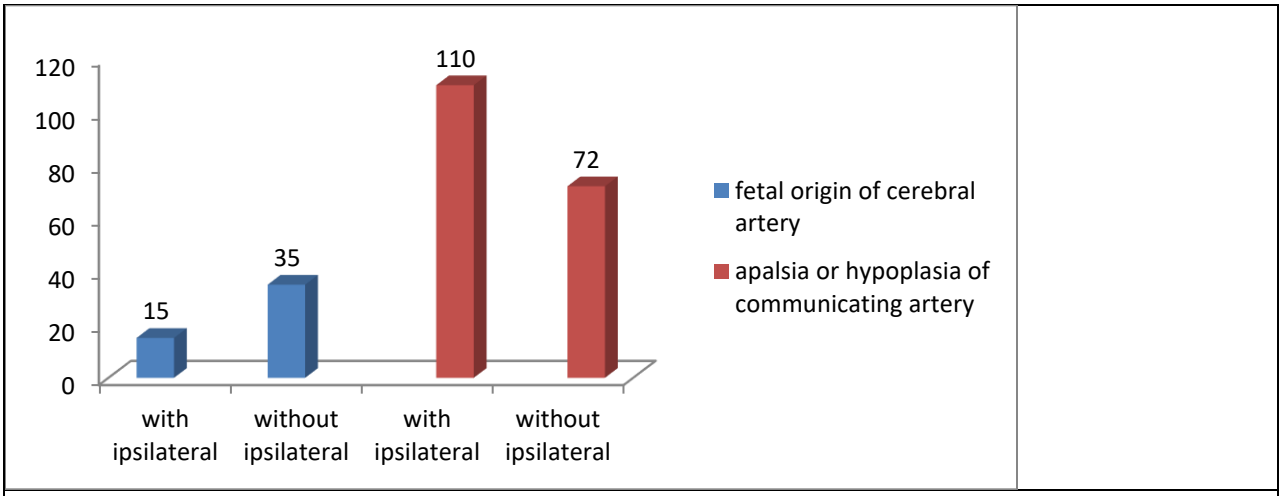

Figure 3: Distribution of patients on the basis of anatomic variation found

In the cohort of patients with infarction, a subgroup analysis of the infarcted territory indicated no significant difference between those with & without fPCA, as outlined in Table 2. Unsignificantassociation was found between fPCA& clinical threatfactor of CVD in patients with infarction.

IVT	**With ipsilateralfPCA(n=15)**	**Without ipsilateralfPCA (n=35)**
Anterior	8 (56)	20 (56.5)
Posterior	4 (24)	7 (19.2)
Thalamus	2 (14)	5(13.7)
Watershed	1 (6)	3(10.4)

Table 2: Participation of various regions in groups with & without variations in fPCA

Only thalamic infarcts (p=0.014) showed a statistically important difference in infarct frequency between individuals with & without PCoA hypoplasia/aplasia, as indicated in table 3. The association between PCoA hypoplasia/aplasia & clinical risk variables was examined in order to address the possibly confusing clinical risk factors in patients with anatomical abnormalities; however, no significant correlation was identified.

Infarcted vascular territory		**With ipsilateral(n=110)**	**Without ipsilateral(n=72)**
Anterior	ACA	56 (51.04)	12 (16.6)
	MCA	7 (6.7)	43 (60.4)
Posterior	PCA	6 (6.25)	5 (6.25)
	SCA	14 (11.4)	8 (12.5)
Thalamus		15 (14.0)	2 (2.08)
Watershed		12 (10.4)	2 (2.08)

Table 3: Territories' involvement in groups with & without ipsilateralhypoplasticPCoA variation

DISCUSSION

The COWs & its branches exhibit several variants. The variances differ not just across individuals but also between the right & left sides of the same individual. The function of the arterial circle is to equilibrate pressure, & under normal circumstances, minimal blood interchange occurs along the anastomotic channel due to pressure equilibrium. In the event of obstruction, the arterial circle functions

to equalize pressure, thereby preserving circulation.

The incidence of the standard "textbook type" polygon varies from 5% to 72%[20]. The extensive variety arises from the diversity in the criteria employed to designate hypoplastic vessels.The aim of our study was “to evaluate the association between anatomical variations in the COWs & occurrence of different patterns of brain infarct”.

In our study 48% patients had infarction while 52% patients did not have infarction. fPCA was present in 50 (20.8)% patients while aplasia or hypoplasia of posterior communicating artery was present in 182 (75.8%) of patients. Two investigations including over 1,000 participants were undertaken in Norwegian & male Chinese populations, revealing complete COW prevalence rates of 11.9%[3] & 12.24%,[4] respectively. The predominant COW variations in the anterior circulation were the absence of AcomA, absence/hypoplasia of A1 segment & in the posterior circulation were the absence/hypoplasia of unilateral/bilateral PcomAs. These results align with the aforementioned large-scale research.[3,4] Despite significant variation in COW structure within the general population, the prevalence of a complete COW & its most prevalent variant remained consistent across populations.

Our findings did not demonstrate a statistically significant association between fPCA& infarcts in various vascular areas. A study by de Monye et al[21] indicated that the risk of ischemic infarct or transient ischemic attack was not elevated with fPCA, corroborating our findings. Conversely, Arjal et al.[22] observed elevated rates of ischemia infarction in partial fetal PCA compared to total fetal PCA, contrary to the intuitive notion that complete fetal PCA would entail a greater stroke risk. Shaban et al[23] shown that the prevalence of pfPCA&cfPCA in patients with acute ischemic stroke did not exceed that of the general population; however, this finding requires validation through larger sample sizes.

Although there was no discernible correlation between PCoA variation & an increased risk of brain infarction, subgroup analysis showed that subjects with absent or hypoplasticipsilateralPCoA had a significantly higher incidence of infarction in the thalamic territory than patients without PCoA hypoplasia/aplasia. Cheung et al,[24] have indicated that PCoA hypoplasia contributes to the risk of ischemic stroke, especially in strokes affecting the arteries that pierce the thalamus. Zelante et al,[25] documented a case of Artery of Percheron infarct in a patient lacking bilateral posterior communicating arteries (PCoA).

Our research has inherent limitations. We classify hypoplasia/aplasia as a vascular diameter of lesser than 1 mm or absence to offset this inaccuracy, however MRI's poor resolution may cause PCoA to be non-visualized when it is extremely narrow. Other limitations include a small sample size & a limited number of patients with both A1 &PCoA hypoplasia/aplasia at the same time, which limits our ability to draw any conclusions about their combined impact.

CONCLUSION

This study demonstrated no significant relationship between particular changes in the COW but gives a sign of vulnerability to silent vascular cerebral infarction. Consequently, vigilant observation may be essential for individuals with variant COW anatomy through imaging assessments.

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