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.

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Major arteries supplying the brain, paired internal carotid (ICA) and vertebral arteries, form a unique anastomosis called the *Circle of Willis* (COW), named after Dr. Thomas Willis, who first accurately described it and provided its physiological significance in 1664.[1] Even then, Dr. Willis surmised its importance as seen in two clinical circumstances (ie, incidental detection of occlusion of major arteries in asymptomatic cases and when surgical occlusion of a major vessel is considered).[1]

There have been several articles that addressed the anatomical features of the COW using digital subtraction angiography and magnetic resonance angiography (MRA).[2,3] However, little has been written with the use of high-speed multidetector computed tomographic angiography (CTA). Also, there have been interesting articles regarding the variants in the COW, namely the presence of the primitive or fetal posterior cerebral arteries as well as the absence of certain segments, such as the posterior communicating artery (PCOM) and its effect in potential strokes.[4-7] But there has been little published with newer multislice CTA and how it pertains to stroke potential, which this article attempts to study. There has been some discussion with the role of intracranial atherosclerosis as detected with CTA, with little written on its role in predicting stroke.[8]

With the advent of carotid stenting and the recent development of embolic protection devices that temporarily occlude flow to the cerebral circulation, there is renewed interest in the COW. These new carotid embolic protection devices can filter, reverse, and arrest flow in the distal common carotid and external carotid arteries while intervention is performed on the cervical ICA. They have been very helpful in reducing major neurologic events associated with stenting, but there is an intolerance in 5% to 23% of patients in those devices that arrest or reverse carotid flow.[9,10]

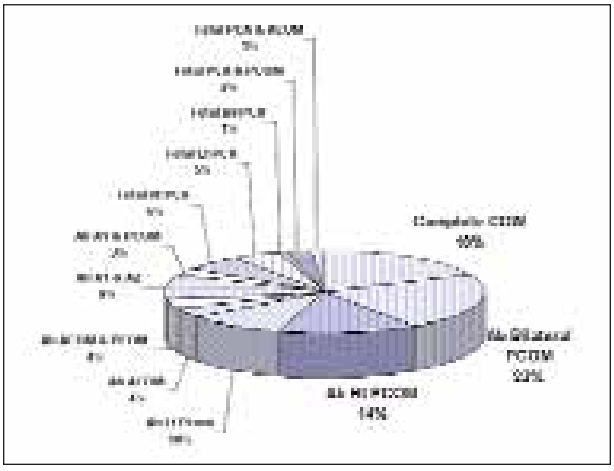

Figure 1. CTA findings of Circle of Willis and variants, n = 212 patients. Abbreviations: Ab, absent; PCOM, posterior communicating artery; ACOM, anterior communicating artery; A1, first segment anterior cerebral artery; A2, second segment anterior cerebral artery; fetal rt/lt PCA, fetal or primitive right or left posterior cerebral artery.

Symptoms of cerebral hypoperfusion during temporary occlusion of the ICA include dysarthria, agitation, decreased level of consciousness, and focal hemispheric deficit.[8,9]

METHODS

During the past 2 years, patients with suspected cervical carotid arterial disease were referred to our institution for 64-slice CTA carotid angiograms including postcontrast brain images. Most patients had a history of coronary artery disease, peripheral vascular disease, or known carotid disease. Carotid ultrasound exams had been performed on many of the patients. Of the total 212 patients, 119 of the carotid Doppler studies were performed beforehand.

TABLE 1. LOCATION OF CTA COW VARIANTS IN RELATIONSHIP TO STROKE AND NONSTROKE PATIENT POPULATIONS

CTA Findings	Subcategory	Nonstroke	Stroke	*P* Value
Complete COW				
		25 (18%)	13 (19%)	.85
Absent PCOM				
	Absent bilateral PCOM	34 (24%)	14 (20%)	.33
	Absent right PCOM	24 (17%)	6 (9%)	.14
	Absent left PCOM	12 (9%)	9 (13%)	.28
	Absent PCOM and ACOM	4 (4%)	4 (6%)	1
	Absent A1 and PCOM	1 (1%)	5 (7%)	.017
	Total	75 (53%)	38 (53%)	1
Absent ACOM				
	Absent ACOM	7 (5%)	1 (1%)	.2
	Absent PCOM and ACOM	4 (4%)	4 (6%)	1
	Absent ACOM and patent fetal PCA	2 (1%)	1 (1%)	1
	Absent PCOM and ACOM	4 (4%)	1 (1%)	1
	Total	17 (12%)	7 (10%)	.82
Patent Fetal PCA				
	Patent fetal right PCA	10 (7%)	3 (4%)	.55
	Patent fetal left PCA	7 (5%)	3 (4%)	1
	Patent fetal bilateral PCA	0	2 (3%)	.11
	Absent PCOM and patent fetal PCA	4 (3%)	1 (1%)	.66
	Absent ACOM and patent fetal PCA	2 (1%)	1 (1%)	1
	Total	23 (16%)	10 (14%)	.84
Absent A1/A2				
	Absent A1	8 (6%)	8 (11%)	.17
	Absent A2	3 (2%)	1 (1%)	1
	Absent A1 and PCOM	1 (1%)	5 (7%)	.017
	Total	12 (8%)	14 (20%)	.05
Total		**141**	**71**	

Two-tailed Fisher's Exact Test used to provide P values.

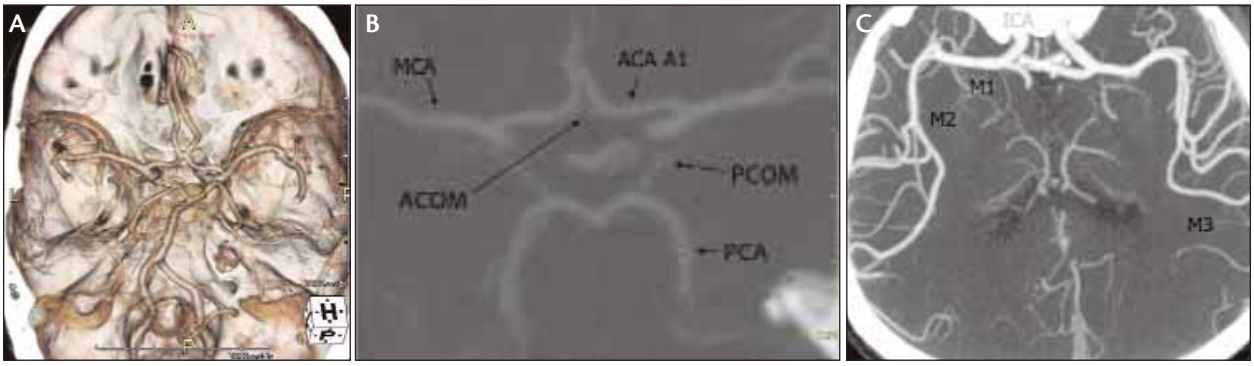

Figure 2. Three-dimensional reconstruction of the COW, which is complete in this patient (A). CTA multiplanar reconstruction axial (MPR) image axial presentation with labels provided of the vessels (B). MPR axial images showing the divisions of the MCA (C).

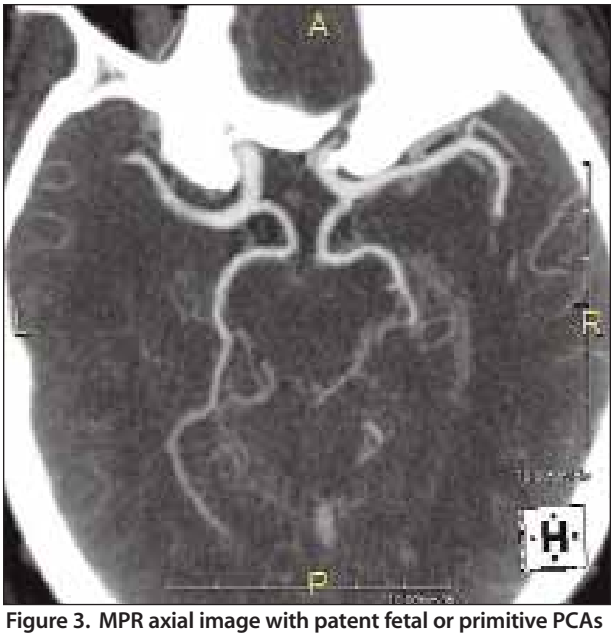

Figure 3. MPR axial image with patent fetal or primitive PCAs bilaterally.

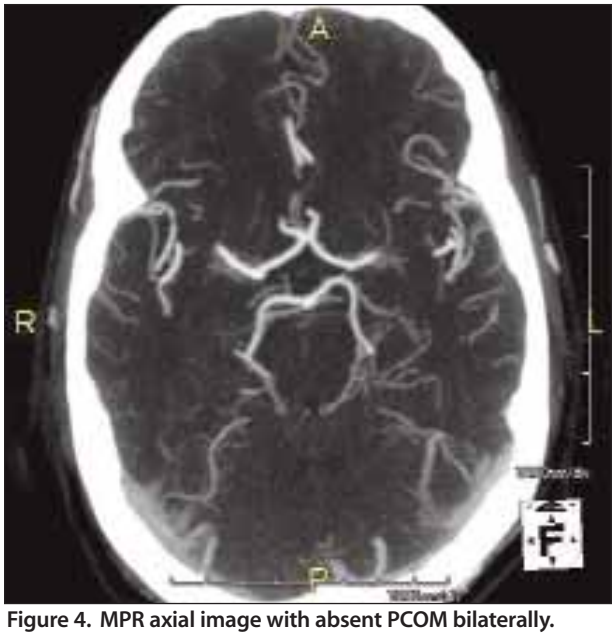

Figure 4. MPR axial image with absent PCOM bilaterally.

A Philips 64-slice CT scanner (Philips Medical Systems, Bothell, WA) was used to scan approximately 212 patients for carotid CTA. The protocol for carotid CTA included 0.45-mm slice thickness cuts obtained with a tracking marker placed upon the aortic arch. We used a field of view of 180 mm with 0.9- X 0.625-mm collimation. Usually 75 mL of nonionic contrast media was injected at 5 mL/sec through a large-bore intravenous access in the antecubital fossa. Radiation parameters were performed at 120 kVp (peak kilovoltage) at 300 mAs (milliampres). After injection, routine brain and bone windows were obtained after a 3-minute delay. Reconstructions of the raw data (512 X 512 matrices) were performed using the Philips Brilliance Extended workstation and the TeraRecon (San Mateo, CA) workstations. Axial images as well as the reconstructed multiplanar (MPR) and maximum intensity projections (MIP) were used primarily for diagnostic purposes. Three-dimensional imaging was also used in helping to diagnose patients.

Two radiologists interpreted all CTA scans. After each scan, the postcontrast CT scan of the brain was reviewed for signs of cerebral infarcts, excluding hypertensive-related infarcts. Next, the carotid and intracranial CTA images were reviewed. The COW was reviewed for complete communication or for incomplete COW. Absence or hypoplasia of the arteries of the COW was then recorded.

Patients were divided into nonstroke and stroke groups. The nonstroke group had no history of stroke or findings of cerebrovascular accident (CVA) on CT postcontrast. Patients diagnosed with previous or recent strokes as well as recent CVA diagnosed by CT were included in the stroke group.

TABLE 2. INCIDENCE AND LOCATION OF DISTAL ICA ATHEROSCLEROSIS AND OTHER INTRACRANIAL PATHOLOGY (N = 212)

CTA Findings	Number (%)
No significant atherosclerosis	100 (47)
Mild distal ICA atherosclerosis	66 (31)
Moderate distal ICA atherosclerosis	22 (10)
Severe distal ICA atherosclerosis	8 (4)
A1/A2 atherosclerosis	7 (3)
M1/M2 atherosclerosis	6 (3)
Basilar atherosclerosis	3 (1)
Total	**212 (100)**
Cerebral aneurysms	6 (3)
Carcinoma	1 (< 1)

The presence of atherosclerotic plaque in the petrous and cavernous segments of the distal ICA was recorded as mild (1%–49% stenosis), moderate (50%–69% stenosis), or severe (> 70% stenosis). Atherosclerosis was also recorded for the M1/M2 segments of the middle cerebral artery (MCA), the A1/A2 segments of the anterior cerebral artery (ACA) as well as in the basilar artery after the formation with the vertebral arteries. Finally, intracranial aneurysms and cerebral tumors were also noted.

Statistical analyses were completed using two-tailed Fisher's Exact Test Calculator for 2 X 2 Contingency Tables from Graphpad.com. The study was limited in being a retrospective study in a biased population of patients with peripheral and cardiovascular disease with suspected carotid occlusive disease. It was a variation to include CVA patients and those with "silent" CVA per CT in the same stroke group. *P* values of .05 or less are considered statistically significant.

RESULTS

There were 212 consecutive patients who underwent multislice CTA at our institution in the past 2 years (2006–2008). Patient demographics included 103 women and 109 men with a mean age of 67.6 that ranged from 38 to 92 years. Of the total 212 patients, 119 of the carotid Doppler studies were performed beforehand. Clinical suspicion for significant carotid disease was the major factor for ordering carotid CTAs despite absence of carotid Doppler or even with mild and moderate ultrasound results.

Of the 212 patients, 38 (18%) had a complete COW. The most common variant was the absence or hypoplasia of the PCOM, which occurred in 99 patients (47%) (Figure 1). Among these, the most common was the absence or hypoplasia of bilateral PCOMs in 48 patients (23%), the absence or hypoplasia of the right PCOM in 30 (14%), and the absence or hypoplasia of the left PCOM in 21 (10%). The next most common variant was the presence of a fetal or primitive posterior cerebral artery (PCA) in 33 patients (16%), which arises directly off an ICA. Absent/hypoplastic anterior communicating artery (ACOM) alone or combined with absent/hypoplastic PCOMs occurred in 16 patients (8%). Finally, absent/hypoplastic A1 and/or A2 segments of the ACA occurred in 26 cases (12%).

Of the 212 patients, 141 were included in the nonstroke group without history of CVA and with a normal CT brain postcontrast. There were 71 patients included in the stroke group with a history of recent and remote stroke and/or findings of stroke on CT scan. As Table 1 shows, 25 patients in the nonstroke group and 13 in the stroke group had a complete COW ($P = .85$) (Figure 2).

Hypoplasia or absence of the ACOM occurred in 17 patients (12%) in the nonstroke group and in 7 (10%) in the stroke group ($P = .82$). The next most prevalent finding was the presence of a fetal or primitive PCA with 23 (16%) in the nonstroke group and 10 (14%) in the stroke group ($P = .84$) (Figure 3). Absent/hypoplastic PCOM occurred in 75 (53%) in the nonstroke group and in 38 (53%) when compared to those in the stroke group ($P = 1.0$) (Figure 4). The absence of the A1 and/or A2 segments of the ACAs occurred in 12 (8%) and in 14 (20%) in the stroke group ($P = .05$); the most significant *P* value within this group was of patients with absent/hypoplastic A1 and PCOM with $P = .017$ (Figure 5). Although the *P* value was .11 and considered not significant, it was approaching statistical significance.

Of the 212 patients, the presence of atherosclerotic plaque in the petrous and cavernous segments of the distal ICA was recorded as mild in 66 patients (31%) (Figure 6). It was moderate in 22 (10%) and severe in eight (4%) patients (Figure 7). Atherosclerosis was also seen in seven (3%) A1/A2, six (3%) M1/M2, and three (2%) basilar artery patients (Table 2). Unsuspected cerebral aneurysms were

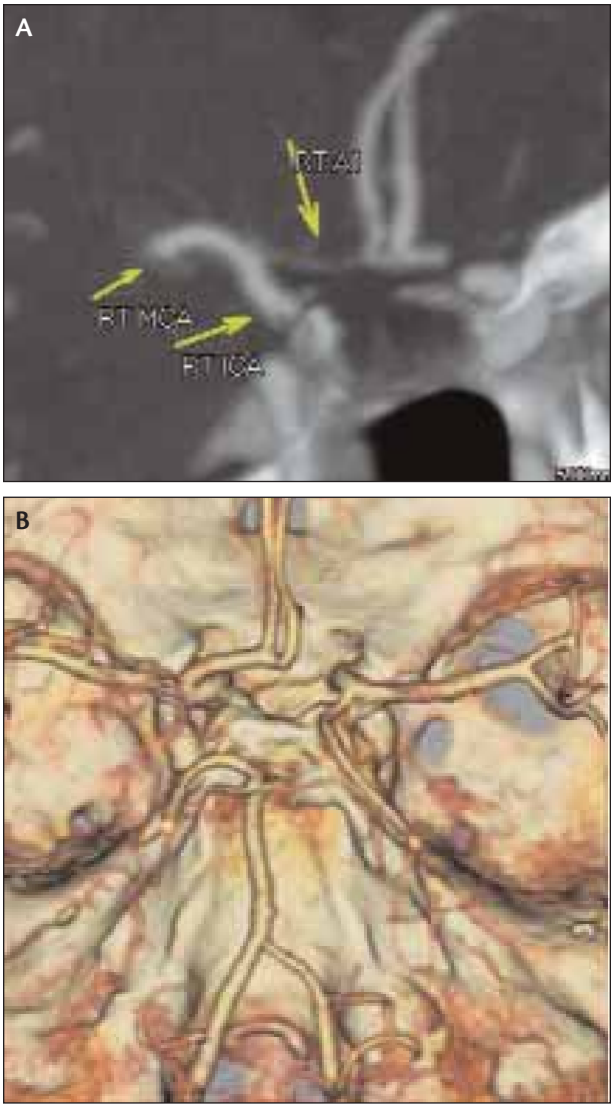

Figure 5. Two different patients: MPR coronal image revealing a hypoplastic right A1 ACA (A); three-dimensional image showing absent left A1 segment of the ACA with a fetal left PCA (B).

found in six patients (3%), two of which were greater than 8 mm. One patient was found to have intracranial carcinoma.

The intracranial atherosclerosis findings were divided into the two groups of nonstroke and stroke. Overall, the *P* value was significant of patients with intracranial atherosclerosis with *P* = .057, which was approaching statistical significance. The degrees of intracranial atherosclerosis were noted as follows. In patients with mild atherosclerosis, the *P* value was .12. Patients with moderate distal intracranial atherosclerosis had a *P* value of .82, and patients with severe intracranial atherosclerosis had a *P* value of .27.

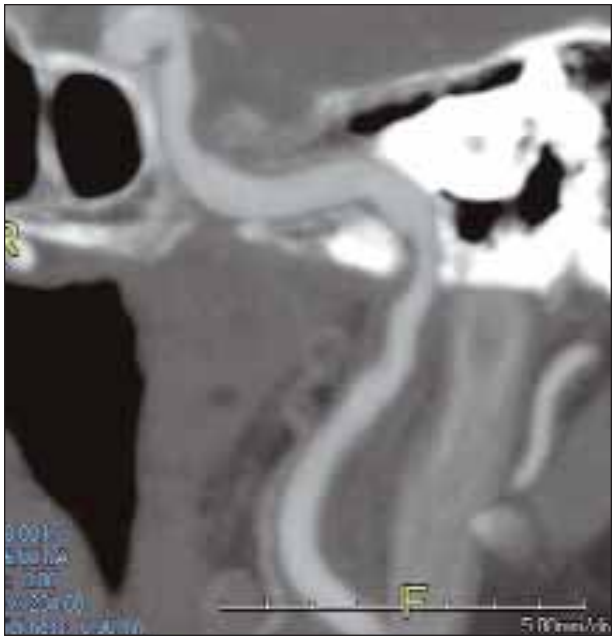

Figure 6. Sagittal MPR image the presence of atherosclerotic plaque in the petrous and cavernous segments of the distal ICA.

DISCUSSION

There are different variations in the components of the COW as described in the literature. Our results are similar to the published results in which the COW is complete in 18% to 41%.[10,11] Other variations include hypoplasia of one or both PCOMs (34%), which was slightly less than our 47%.[10,11] There was a hypoplastic/absent A1 segment of ACA (17%) compared to our 12%.[10,11] Primitive or fetal PCA was found in 16% in our series compared to 15% in other series.[12]

There have been several publications that addressed the relationship between the COW and its variations and propensity for stroke—particularly the presence of the fetal PCA. In a fetal-type PCA, a larger area is thus dependent on the ICA; leptomeningeal vessels cannot develop between the anterior and posterior circulation.[13] The tentorium namely prevents cerebellar vessels from connecting to the PCA territory.[13] Therefore, patients with a fetal PCA could be more prone to develop vascular insufficiency.[13] Whether patients with the anomaly of the fetal PCA have a higher risk of ischemic stroke in the territory of the PCA is not known.[6] This question was first reviewed by de Monye et al who did not find enough support in their study of 84 patients.[4] In our series of 33 patients with a fetal PCA, there was little significance with the fetal PCA and a propensity for stroke, although those patients (n = 2) with bilateral PCAs had a slightly higher chance with *P* = .11; this was too small a number for conclusions.

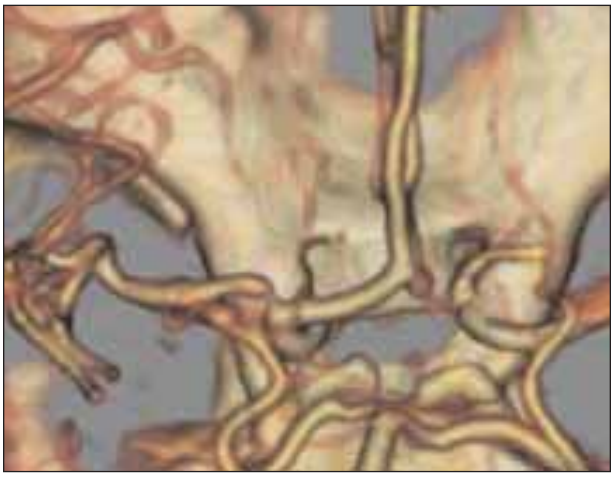

Figure 7. Axial three-dimensional image showing severe right MCA M1 stenosis with absent or hypoplastic left ACA A1 segment.

Another anomaly that has drawn attention has been hypoplastic/absent PCOM. Chuang et al examined 310 acute ischemic stroke patients with MRA performed within 72 hours of ischemic stroke onset.[14] They found PCOM hypoplasia appears to be a contributor to the risk of ischemic stroke, even in the absence of ICA occlusion.[14] We found in our series that the absence/hypoplasia of PCOM arteries did not correlate with risk of stroke ($P = 1.00$), except for cases of combined PCOM and A1 absence.

The absence/hypoplasia of A1 and/or A2 had a P value of .05, which is statistically significant. As stated, the absent/hypoplastic A1 and PCOM had a significant $P = .017$, which has not been recorded before; however, this is only with six patients, which is a small series to study.

The advent of carotid stenting has introduced the potential for proximal and distal occlusion, but with this technology comes the potential of patients' intolerance for temporary occlusion. There has been concern that patients with an incomplete COW may suffer from this temporary occlusion. Our anecdotal experience of 45 PercuSurge (Medtronic, Inc., Minneapolis, MN) cases was the intolerance of approximately three patients, one having had a complete COW, which provided occlusion of the ICA flow distal to the lesion, was the intolerance of approximately three patients (7%), one having had a complete COW. There is still not enough known to state which carotid artery stenting candidates with COW variants are at risk. In our experience, we found that

patients with hypoplastic or absent A1 or A2 segments had a higher risk of stroke. Risk was slightly higher in those with hypoplastic/absent PCOMs. Risk was also present with those with intracranial atherosclerosis. Hence, these patients may be considered at risk for temporary occlusion, but this remains only a hypothesis.

The presence of intracranial atherosclerosis has been discussed in previous literature with CTA being one of the best image modalities to view it.[8,15] CTA can give excellent anatomic visualization of intracranial atherosclerosis. Spatial resolution may occasionally limit our ability to distinguish very severe stenosis from occlusion compared with digital subtraction angiography.[16] Calcium might also occasionally cause overestimation of a stenosis.[15] Compared to digital subtraction angiography, CTA has high sensitivity and specificity for detecting ≥ 50% stenosis of large intracranial arterial segments. CTA is minimally invasive and may be a useful screening tool for intracranial arterial disease and occlusion.[16]

As we found in our study, the presence of intracranial atherosclerosis in the petrous-cavernous segment was surprisingly high; 51% of the patient population had some form of intracranial atherosclerosis. The majority was mild (31%) with minimal narrowing. Overall, there was little relationship with patients' stroke history. As Cloft stated, there is certainly confusion with intracranial atherosclerosis not only in its diagnosis but in its management as well—the latest results of the WASID (Warfarin versus Aspirin Symptomatic Intracranial Disease) trial indicate that warfarin offers no benefit over aspirin.[15,17] Nevertheless, we should strive to keep the art of diagnostic imaging ahead of the art of therapy.[17] Treatments targeted to a patient's specific disease can be developed only if we can reliably diagnose that disease.[17]

CONCLUSION

We evaluated 212 patients to reveal the pattern of the COW and its variants using 64-slice CTA technology. We were able to easily determine the COW and its variants. When we compared the COW and its variants to the patients in the stroke and nonstroke groups, the only variable that proved positive included the absent/hypoplastic A1/A2 and PCOM (especially bilateral PCOM). Another COW variant, hypoplastic/absent PCOM, had a slight risk but still not significant. Other variants including absent/hypoplastic ACOM and fetal or primitive PCA did not have significant correlation with stroke despite other publications. In a separate subset, the presence of intracranial atherosclerosis proved quite high. Overall, there was an increased incidence of stroke among these people. There is still not enough information to accurately predict who is at greater risk for stroke as well as who may not tolerate new carotid stent procedures requiring temporary flow occlusion. ■

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