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ANTERIOR CEREBRAL CIRCULATION: A LITERATURE REVIEW

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ABSTRACT

Anterior cerebral circulation consists of the anterior cerebral artery and middle cerebral artery of the circle of Willis. The anterior cerebral artery's course is classified into two main segment classifications constructed by Fischer and Osburn et al. The perfusion area of the anterior cerebral artery extends medially over the entire frontal and parietal lobes, the septum, and the basal forebrain structures such as the hypothalamus, hypophysis, and optic chiasm. The anterior cerebral artery also provides blood to the rostrum, genu, and body of the corpus callosum. The anterior cerebral artery shows some anatomical variations throughout its course. Aplasia or hypoplasia are the common variants in the A1 segment, whereas fenestration occurs in rare cases. Azygos artery formation is a rare variant of the A2 segment, and recurrent artery of Heubner is an important branch arising from A1 and A2 segments, which is one of the main perforators of structures such as but not limited to the anterior parts of the internal capsule and the lentiform nucleus. Common variations of the middle cerebral artery comprise accessory artery formation or duplication, early branching, and also fenestration. Although cerebral circulation is complex with many collaterals and variations, pathological disturbances in the blood supply can still occur. Anatomical variations, cardiac problems, ethnicity, age, and physical exercise are some of the many risk factors that account for pathological cases such as aneurysms, occlusions, dissections, infarctions, and stroke. Disturbances in the blood supply of such a crucial region may lead to severe disabilities, if not death. Middle cerebral artery syndrome is one of the most important pathologies of the brain, where the outcome is stroke. Whether the underlying etiology is stenosis or obstruction, two main mechanisms can be categorized as atherosclerotic and non-atherosclerotic. Atherosclerotic causes can be thrombus or emboli origin, and non-atherosclerotic causes can be due to hemodynamic compromise, vasculitis, arterial wall dissections, and moyamoya disease. **Keywords:** Anterior cerebral artery, middle cerebral artery, aneurysm, infarction

INTRODUCTION

The brain is one of the most complex organs of the human body. Therefore it is highly perfused compared to other organs. Even though the brain is not the heaviest in mass compared to other organs, it still requires the highest energy to carry out its vital functions for the body (1). Despite representing merely 2% of the body mass, the brain uses approximately 50% of the total body glucose (1). The source of glucose, the fuel that the brain runs on, is the blood circulation, hence the reason why two different arterial systems supply it: anterior cerebral circulation and posterior cerebral circulation. Despite serving the same purpose, these two sets of circulation arise from the different branches of the aorta (2).

The aorta is divided into three sections after it courses out of the heart and into the middle mediastinum (2). These parts are the ascending aorta, aortic arch, and descending aorta. Only the aortic arch supplies the brain. The aortic arch has three branches: the brachiocephalic trunk, left common carotid artery, and left subclavian artery, from proximal to distal (2). While the brachiocephalic trunk does not directly contribute to the brain's blood supply and brainstem, the latter further gives off branches to supply the neuraxis.

The left common carotid artery bifurcates into the internal and external carotid arteries at the level of T4 (3). The internal carotid artery (ICA) runs superiorly into the brain to form the anterior cir-

culation of the brain. On the other hand, the subclavian arteries of both sides give off vertebral arteries that run on both sides of the vertebra through the foramen transversarium and enter the brain through the foramen magnum to form the posterior circulation of the brain and the spinal cord (3).

The major suppliers of the anterior cerebral circulation are the anterior cerebral artery (ACA) and middle cerebral artery (MCA) (3). Knowing its distinct and precise anatomy, pathological variations, and possible pathological outcomes of the anterior cerebral circulation is essential for surgical interventions and postoperative follow-ups. Successful surgery without incurring significant neurovascular morbidity in this region depends on the detailed knowledge of vascular anatomy and the relevant pathologies (3).

ANATOMY OF THE ANTERIOR CEREBRAL CIRCULATION

Anterior Cerebral Artery

The knowledge of anatomical variants is crucial for therapeutic success. In their article Sañudo et al. (4) demonstrated that the lack of anatomical knowledge caused around 10% of all medical errors. A certain degree of asymmetry was detected between anterior arteries in 80% of patients in a study conducted by Given et al. (5).

Segmentation and Course of Anterior Cerebral Artery

The ACA arises from the termination point of the ICA and is described as a part of the circle of Willis. There are two main classification schemes for the segments of the ACA. The first, and to our knowledge the oldest classification, Fischer described in 1938 (6). In Fischer's classification, the ACA is divided into five segments: 1 the pre-communicating (A1), 2 below the genu of the corpus callosum (A2), 3 around the genu of the corpus callosum (A3), 4 the terminal branch of the A4, and 5 the terminal branch of the A5 (6). Fischer's classification was further examined and modified by several authors. In the recent modifications to Fischer's classification, the ACA was divided into the pre-communicating segment (A1) and post-communicating segment, which was further divided into infracallosal (A2), precallosal (A3), supracallosal (A4), and posterocallosal (A5) segments (7). In another classification developed by Osborn et al. (8), the ACA was divided into three segments: the segment which runs over the optic chiasm, the segment running vertically and entering into interhemispheric fissure after crossing the anterior communicating artery (ACoA), and a group of terminal segments (8, 9).

The perfusion area of ACA is considered the most variable when compared to other essential blood sources, such as the MCA and posterior cerebral artery (10). The perfusion area extends medially over the entire frontal and parietal lobes, the septum, and the basal forebrain structures such as the hypothalamus, hypophysis, optic chiasm. ACA also provides blood to the rostrum, genu, and body of the corpus callosum. From the perspective of functional centers, ACA thus supplies blood to a large part of the prefrontal and premotor cortex. Through the recurrent artery of Heubner (RAH), blood is perfused to the internal capsule, from the anterior to the beginning of the posterior crus, and the anterior part of the striatum (11).

Anatomical variations and measurements of ACA segments have been studied by magnetic resonance or computed tomography angiography (MRA/CTA) and cadaveric methods. In studies that we reviewed, the number of hemispheres examined was lower in cadaveric studies than in MRA/CTA studies. On the other hand, in MRA/CTA studies, distal branches or low diameter branches could not be examined thoroughly.

Variations of A1 and A2 Segments

Aplasia or hypoplasia of the A1 segment are common anatomical variants. Hypoplasia (in most of the studies defined as a diameter of A1 less than 1.5 mm) of the A1 segment was detected in 10% of cadavers in post-mortem examinations run by Perlmutter et al. (12). In the same study, the rate of aplasia was 2% (12). In cases with hypoplasia or aplasia of the A1 segment, the contralateral ACoA was dilated, allowing the contralateral artery to supply blood to both sides. These variants increase the risk and extent of neural tissue ischemia in the frontal lobe region during intravascular procedures in the area of ACoA or during ischemic episodes (13). Another common variation of the ACA is fenestration. Its prevalence in the A1 region was 0-4% in anatomical studies and 0-2% in MRA/CTA studies (14-17).

A rare variant found in the A2 segment is the azygos-ACA, in which two A1 segments of both hemispheres form a single A2 truck. As a result, an ACoA could not be found in patients with azygos-ACA. This variant was found in around 1.5% of the cases studied by Auguste et al. (18). Azygos-ACA is considered an essential predictor of bilateral frontal strokes, and saccular azygos-ACA aneurysms are relatively common with a prevalence between 13-71% (19).

Variations of Anterior Communicating Artery

A further variation involving the ACA is the accessory anterior cerebral artery. It describes a small, additional artery (so-called median artery of the corpus callosum) branching from the ACoA in addition to two A2 segments of both hemispheres. Its prevalence was around 8% in the studies reviewed by Dimmick et al. (13). In another MRA study conducted by Uchino et al. (14), its frequency was reported as 3%. The description of this variation is relevant before the clipping of ACoA aneurysms.

Due to its short length, fenestration and duplication of ACoA are challenging to differentiate. Thus, they are often grouped in studies. Although it is found frequently in cadaveric studies (20-30%), it is not a common finding in angiographic studies (0-10%) (12, 15, 16, 20). Uchino et al. (20) stated in their case report that improved image quality of MRA can make the accurate diagnosis of tiny variations of ACoA possible, including fenestrations and duplications.

An overview of different variations of A1 and A2 based on several studies can be seen in Table 1 (12, 14-16, 21-23).

Recurrent Artery of Heubner

Otto Heubner first described the recurrent artery of Heubner in 1872 (24). RAH is also called the median striatal artery and is one of the main perforators of structures such as the anterior parts of the internal capsule and the lentiform nucleus, and the head of the caudate nucleus (24). Embryologically, it is formed by the fusion of several smaller arteries. It arises from the A1 or A2 segment of the anterior cerebral artery, mainly in the area of the ACoA. In the cadaveric study conducted by Loukas et al. (25), the frequency of the branching of this artery from the ACoA was found to be 62.3%. The branching followed this from the proximal A2 with 23.3%, and the branching from the A1 segment with 14.3%. In the same study, the diameter of the same artery was found to be approximately 0.8 mm (25). Results, including the site of origin, mean diameter, and length of the RAH of several reviewed studies, are illustrated in Table 2 (12, 24-30).

Other Rare Variants

A rare variation considering the course of the proximal part of the ACA is its infraoptic course. The infraoptic course arises from the ICA at the level of the ophthalmic artery and terminates in the proximal segments of the ACA, thereby constitutes an anastomosis between the anterior cerebral circulation and the ICA (31).

Another rare variant with an abnormal anterior cerebral artery course is the persistent primitive olfactory artery (PPOA). Embryologically, the primitive olfactory artery (POA) is considered to be a rostral division of the primitive internal artery. The proper ACA develops from the POA. In the case where POA keeps its embryological course along with the olfactory bulb, it is described as PPOA. The characteristic course of this artery is associated with an increased risk of aneurysms (32).

Middle Cerebral Artery

Common variations of the middle cerebral arteries (MCAs) comprise accessory or duplicated MCA, early branching, and fenestrations. There are different classifications of additional MCAs.

Teal et al. (33) classified the additional MCAs into two major groups in 1973: duplicated and accessory MCAs. The accessory MCA, arising from the ACA in the A1 segment, was documented in 0.4% of the patients in the study conducted by Loukas et al. (25) and 3% of the post-mortem examinations conducted by Jain (34).

Duplicated MCAs, on the other hand, branch from the distal internal carotid arteries and were found to be present in about 2% of patients in the same study. In another study, conducted by Komiyama et al. (35), the prevalence of both arteries was found to be 0.4% and both accessory and duplicated MCAs supplied either the anterior temporal lobe or the anterior frontal lobe.

Another common categorization of the additional MCAs is the Manelfe classification (36). Manelfe grouped accessory MCAs in 3 different sub-categories. Type 1 MCA is the typical duplicated MCA, type 2 branches from the proximal anterior cerebral artery as an accessory MCA, and type 3 arises from the distal anterior cerebral artery as another form of accessory MCA (36).

Table 1: A review of several studies that examined the proximal part of ACA (12, 14-16, 21-23).

Study, year	Study method	Number of hemispheres (n)	Mean A1 diameter, length (mm)	Anatomical variations of A1 and A2 (%)	Mean ACoA diameter, length (mm)	Anatomical variations of ACoA (%)
Perlmutter et al., 1976 (12)	Cadaveric	100	2.6, N/A	Hypoplasia: 10 Aplasia: 1-2 Fenestration: 4	1.5, N/A	Duplication-Fenestration: 30%
Uchino et al., 2006 (14)	MRA	1782	N/A	Unilateral Aplasia: 5-6 Fenestration: 1.2 Azygos ACA: 2.0	N/A	N/A
Shatri et al., 2019 (15)	MRA	1026	2.05, 14-1	Unilateral Agenesis: 5.65 Fenestration: 0.58	1.16, 2-99	Hypoplasia or Aplasia: 15.66% Fenestration: 3.89% Duplication: 0.6%
Şahin et al., 2018 (16)	CTA	1502	N/A	Hypoplasia: 14.6 Aplasia: 2.53 Fenestration: 1.06 Trifurcation: 4.53 Azygos ACA: 1.46	N/A	Fenestration-Duplication: 10.12%
Karatas et al., 2016 (21)	Cadaveric	200	1.87(R)-1.96(L), 14.44(R)-13.72(L)	Hypoplasia: 2 Aplasia: 1	1.43(R)-1.95(L), N/A	Hypoplasia: 20% Aplasia: 1%
Shatri et al., 2017 (22)	MRA	266	2.09, 13.96	N/A	1.5, 2-74	N/A
Yeniceri et al., 2017 (23)	MRA	768	1.58(R)-1.60(L), N/A	N/A	N/A	N/A

A1 and A2: Proximal segments of anterior cerebral artery, **ACA:** Anterior cerebral artery, **ACoA:** Anterior communicating artery, **CTA:** Computed tomography angiography, **MRA:** Magnetic resonance angiography, **R:** Right, **L:** Left, **N/A:** Not available

Table 2: A Review of several studies that examined the recurrent artery of Huebner (12, 24-30).

Study, year	Study method	Number of hemispheres (n)	Site of origin			Mean RAH diameter, length (mm)
			A1 (%)	A2 (%)	J* (%)	
Perlmutter et al., 1976 (12)	Cadaveric	100	14	78	8	1.0, N/A
Maga et al., 2013 (24)	Cadaveric	140	26.2	33.8	40	1.0, 25.2
Loukas et al., 2006 (25)	Cadaveric	69	14.3	23.3	62.3	0.8, 24
Gomes et al., 1984 (26)	Cadaveric	60	8	57	35	0.8, 23.4
Avci et al., 2003 (27)	Cadaveric	62	8	64	29	N/A
Uzun et al., 2009 (28)	Cadaveric	108	6.2	14.6	79.2	0.67, N/A
Zunon-Kipré et al., 2012 (29)	Cadaveric	40	30	58	12	0.7, 24
Najera et al., 2019 (30)	Cadaveric	50	41	31.5	27.3	0.4, 13

RAH: Recurrent artery of Huebner, **A1 and A2:** Proximal segments of the anterior cerebral artery, **J:** Junction, **N/A:** Not available
*At the level of the anterior cerebral artery and anterior communicating artery junction.

PATHOLOGIES OF ANTERIOR CEREBRAL CIRCULATION

Anterior Cerebral Artery

Knowing how crucial the cerebral blood supply is, it undoubtedly concerns if any of the blood vessels do not function properly. From cerebral aneurysms to cerebral occlusions and dissections, any change in the cerebral circulation may lead to a stroke. Although ACA infarction is known to account for only 0.5-3% of ischemic strokes, its severe clinical outcomes such as death, should not be overlooked (37, 38).

Anterior cerebral artery infarcts occur predominantly on the left hemisphere, while bilateral ACA infarcts only account for 0-9% of ACA infarcts (39). Studies on Western populations suggest that ACA infarction rarely stems from ACA atherosclerosis (40). However, embolism from either ICA or the heart is considered the primary reason, given the approximate rate of 63% among ACA infarction patients (40). Studies on Asian populations, on the other hand, suggest different percentages for the listed possible reasons for ACA infarction. In Asian patients, the primary reason is ACA internal atherosclerosis with a large portion of 59%, with the atherosclerotic change mostly occurring in the A2 region of the ACA (40). Large atherosclerotic cerebral infarction occurs later in life, whereas cardioembolic cerebral infarction can occur at any age (41). Although intracranial dissections are less common than extracranial dissections in Western populations, ACA is not the affected region in most cases (38). However, in Asian populations, ACA dissection accounts for 8% of intracranial dissections (38). Cerebral infarction originating from atherothrombosis mainly affects the right hemisphere, and it is not bilateral unless the reasons are chronic atherosclerosis and acute cardioembolism (41).

Major risk factors for ACA infarction are age, heart disease, high blood pressure, and a history of strokes (38). In patients with ACA infarction stemming specifically from dissection, these factors were different: younger age, lower blood pressure, and no underlying heart diseases (38). In dissection cases, stroke develops more suddenly and more commonly after physical exertion, such as defecation, singing, or heavy exercise (38).

Anterior cerebral artery infarction causes clinical symptoms such as neurological deficits, accompanied by headache and physical exertion at the onset (38). It causes weakness on the limbs, contralateral hemiparesis, or monoparesis that generally affect the legs, sometimes accompanied by weakness on the shoulders and arms (39). The arm and face weakness is mainly associated with Heubner's artery and the medial striate arteries (39). In the affected limbs, sensory dysfunction may also appear (37). Headache is also an essential sign of this condition. Although headache is not considered a typical symptom, approximately 30% of the patients experience it and is defined as a "non-throbbing" headache (39). Patients with dissection have an earlier onset of a headache than patients without dissection (38). Along with these major symptoms, some patients may also present speech disturbances such as aphasia and dysarthria, decreased level of consciousness, grasp reflex, and urinary incontinence (39, 42, 43).

Stroke led by ACA infarction results in 7.8% in-hospital mortality (42). Even if the patients survive, only 9.8% of patients are symptom-free at hospital discharge. The remaining majority is unfortunately left with different levels of disability (42).

Another pathology that needs to be elucidated is ACA aneurysms. The vast majority of ACA aneurysms are located at or adjacent to the ACoA (44). ACA aneurysms that occur distal to the ACoA (A2:vertical or post-communicating portion) are called distal ACA aneurysms, and they are not as common (44). Proximal ACA (A1) is considered a rare location for aneurysms to develop, as they only account for 0.59-4% of intracranial aneurysms (44, 45). For an A1 aneurysm to develop, vascular abnormalities such as hypoplasia, aplasia, or fenestration of the ACA need to be present (45). Sixty percent of A1 aneurysms remain unruptured (45). Distal anterior cerebral artery (DACA) aneurysms, which develop on the A2-A5 segments, account for 2-9% of intracranial aneurysms (46). DACA aneurysms stem from various ACA abnormalities that increase the blood flow and shear stress in DACA and cause aneurysms at the bifurcations (47).

Although rare and mostly asymptomatic, ACA aneurysms might cause symptoms if they are ruptured (48). Ninety percent of ACA aneurysms are asymptomatic and are discovered incidentally (48). Unruptured ACA aneurysms might present with vague or non-specific symptoms such as headache, dizziness, or loss of consciousness depending on the severeness of the situation (47, 48). The risk of rupture depends on several risk factors such as aneurysm size, specific location, female gender, older age (mostly over 60 years), hypertension, and smoking (48). With the present risk factors, physical exercise that increases intracranial pressure may cause the aneurysm to rupture (48). In addition to physical exercise, sexual intercourse, defecation, micturition, excessive caffeine intake, and excessive anger are among the risk factors that may increase intracranial pressure (48). Ruptured aneurysms account for 85% of subarachnoid hemorrhages, which lead to death in 35-39% of the cases (48). Subarachnoid hemorrhages from an aneurysm rupture present with a sudden and severe headache that reaches its maximum intensity within seconds (48). Apart from the particular headache defined as "the worst headache of my life" by many patients, vomiting is another sign that presents approximately half of the patients (48).

A1 segment of ACA is the principal source of the blood supply for the circle of Willis (49). Not only is the ACA a principal blood supply, but it also branches to the striate arteries that supply blood to a broad range of crucial areas: anterior hypothalamus, septum pellucidum, and the anterior and inferior portions of corpus striatum (49). The hypoplastic A1 segment is an uncommon congenital variation with a frequency of 1-13% (49). A1 segment is considered hypoplastic if its width is less than 50% of the contralateral A1 segment or if its diameter is less than 1 mm (45, 49). In some cases, if the A1 segment is hypoplastic or aplastic, the ACoA supplies the territory (39). However, the absence of such a large blood supply can still be an issue. Hypoplasia or aplasia contributes to an increase in local hemodynamic forces on the contralateral side, and this increases wall shear stress (32, 45). A1 segment hypoplasia is classified as a predisposing factor for occlusion pathologies (49). Therefore, disturbances of the supply stemming from hypoplasia or aplasia of the ACA lead to ischemic strokes of the anterior cerebral area (49). Around 83.3% of A1 hypoplasia-related ischemic strokes were thought to stem from occlusions, especially within the striate arteries (49). In 76.2% of ipsilateral hemispheric ischemia, A1 segment hypoplasia, mostly on the right side, was associated (49). A1 hypoplasia was also associated with 19.2% of hemispheric infarction cases and 4.8% of brainstem or cerebellar ischemic stroke cases (49). 41.9% of patients with A1 segment aneurysms had vascular abnormalities such as fenestrations, hypoplasia, and aplasia on the contralateral side (45). Although many agree that A1 segment hypoplasia has the potential to cause important clinical presentations, there is evidence that it might even be asymptomatic if its collaterals compensate for the disturbances (49).

Middle Cerebral Artery

The MCA has an extensive vascular territory with an elaborate course contributing to the arterial supply of the anterior circulation of the cortex. However, due to its long course, structure, and effects of systemic diseases, many pathologies associated with various clinical presentations have been documented in the literature. For a more focused look, this part will include the MCA syndrome only.

Middle Cerebral Artery Syndrome (Disease)

Restriction of blood supply to the MCA is defined as MCA syndrome or MCA disease. The outcome of this blood flow restriction is stroke (50). Bogousslavsky et al. (51) state that stroke results from either stenosis or occlusion of the MCA. Factors resulting in cerebral infarction are categorized by Wong et al. (52) categorised factors resulting in cerebral infarction as embolism, thrombosis, hemodynamic compromise, local occlusion of branches, and a mixture of these etiologies. In terms of conditions contributing to the development of stroke, the aetiologies can be grouped into two main categories: atherosclerotic MCA syndrome and non-atherosclerotic MCA syndrome.

According to the study conducted by Ahn et al. (53), the origin of MCA disease in 72.6% of their patients was found to be atherosclerosis. Another study found that only in 12.5% of the patients, the MCA obstruction was due to local thrombosis, whereas in 40% of the patients it was due to embolism of the heart and the ICA, and in 47.5% of the patients its origin was undetermined (54). Other studies found out that one-third is due to embolism of all cases of MCA syndrome (51, 55). In their study, Lee et al. (56) found that 76 out of 107 patients had atherosclerosis and 31 patients suffered from cardiac embolism. Whether atherosclerotic or non-atherosclerotic, the infarcts are categorized as cortical infarcts, border zone infarcts, and penetrating artery infarcts. In their study, Kim et al. (40) found that the non-atherosclerosis group was younger when compared to the atherosclerosis group (40.1 ± 10.5 years, 45.7 ± 8.9 years respectively), and the women were more affected than men (58.3% to 44.4%).

Cortical Infarcts

In their study, Wong et al. (52) defined cortical infarcts as smaller than 5 mm in diameter. They also found no cortical infarct was isolated and suggested that cortical infarcts usually are asymptomatic and co-exist with other types of infarcts (border zone and penetrating artery).

Border Zone Infarcts

Wong et al. (52) found that border zone infarcts can exist as both single and multiple infarcts, where the latter is more common (73.3% multiple, 33.3% single). They describe the underlying pathology as cholesterol crystals (52, 55). However, cholesterol crystals are not the sole pathology in border zone infarcts, since Torvik (57) and Beal et al. (58), in their studies, reported platelet-emboli-occluded leptomeningeal arteries where border zone infarcts were seen.

Penetrating Artery Infarcts

Penetrating artery infarcts (PAI) can be seen as both single and multiple. In their study, Wong et al. (52) found no PAI larger than 15 mm. They found that the lesions were identical to that of lacunar infarcts where the lesion is due to lipohyalinosis. The underlying mechanism in this type of infarct is the occlusion of MCA in the origin of a penetrating artery that results in lacuna-like infarcts (59). Occasionally, small embolisms may also cause the block leading to PAI.

Atherosclerotic Causes

Atherosclerotic MCA syndromes result from either intracranial atherosclerosis or extracranial atherosclerosis (37). Intracranial atherosclerosis arise within the MCA, and extracranial atherosclerotic causes of infarctions are due to embolism that arises in the proximal ICA, heart chambers, and aorta (52, 54). Lee et al. (56) stated although rare in Caucasians, atherosclerosis of MCA is more common in Asians, and therefore should be included in the differential diagnosis of ischemic strokes (51). In a study conducted by Kim et al. (37), ischemic strokes due to atherosclerosis were found to be in MCA in 34% of the cases. They also found that most cases were due to intracranial atherosclerosis.

One other cause of MCA syndrome is artery-to-artery embolism. When small embolisms are released, there exist two possible results: total clearance and ischemia. The latter is, as presumed by Wong et al. (52), the low risk regarding the ability of cerebral circulation in terms of arterial anastomoses and collateral supply, which prevents the formation of infarct and doles out adequate blood flow that prevents the formation of large ischemic area. These infarcts caused by small emboli usually result in cortical infarcts that appear to be less harmful, as they are more benign and affect a smaller area. Wong et al. (52) state that this is due to anastomoses formed during a rather long process of MCA stenosis caused by atherosclerosis. As mentioned above, the common pathology underlying artery-to-artery embolism is cholesterol crystals (52). Atheroma in MCA can cause occlusion and lead to PAI when the block is formed in the origin of a penetrating artery (59).

Non-atherosclerotic Causes

Although atherosclerotic lesions are the most common causes of MCA strokes, many non-atherosclerotic causes also play a role in the development of infarctions. Kim et al. (60) state that moyamoya disease (MMD), arterial wall dissections, and vasculitis are some of these etiologies and these etiologies are the underlying lesions for younger individuals.

In their study, Kim et al. (60) found that 28 patients had concentric stenosis with a smaller diameter. MMD is typically symptomatic from the midst of the 3rd decade to the 4th decade of life (60). Scott et al. (61) state that 2/3 of the affected population are female. Kim et al. (60) state that hemodynamic compromise may exist along MMD and when affected unilaterally, MMD on its own may not cause any symptoms by hemodynamic stability and collateral perfusion.

CONCLUSION

With the brain being one of the most complex organs in the human body, it undoubtedly has crucial tasks. In order to maintain its vital functions, the brain must be sufficiently perfused. Accordingly, it is supplied by two distinct arterial systems: the anterior cerebral circulation and the posterior cerebral circulation. The major suppliers of the anterior cerebral circulation are ACA and MCA. Having discussed its distinct and precise anatomy, it is essential to know the variations that might lead to pathologies. Besides its anatomy, various pathological cases should be kept in mind, essentially for surgical interventions and postoperative follow-ups. Successful surgery without incurring significant neurovascular morbidity in this region depends on the detailed knowledge of its vascular anatomy and pathologies.

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