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Twig-like middle cerebral artery: a rare and frequently misdiagnosed vascular anomaly

L. Houari[1], D. Ait Ali[2], G. T. Houari[3]

[1] Clinic Fatema Al Azhar, Algiers, Algeria
[2] Department of Natural and Life Sciences, Targa Ouzemmour, University of Bejaia, Algeria
[3] Santa Monica College, United States

Keywords
Twig-like middle cerebral artery, Moyamoya disease, Vascular anomaly, Cerebral angiography, Congenital cerebrovascular malformation

Abstract

Objective: The twig-like middle cerebral artery (TL-MCA) is a rare congenital vascular anomaly in which the M1 segment is replaced by a plexiform network of primitive arterial twigs. Because its angiographic appearance overlaps with Moyamoya disease and arteriovenous malformations, TL-MCA is frequently misdiagnosed. We report three cases initially referred as Moyamoya disease and provide an expanded review of diagnostic criteria, embryological mechanisms, and management strategies.

Methods: A retrospective review of all patients referred to Al Azhar Clinic (Algiers, Algeria) between 2019 and 2024 for presumed Moyamoya disease was conducted. Among 24 patients diagnosed with Moyamoya disease or syndrome, three were ultimately identified as having TL-MCA. Clinical presentation, MRI findings, angiographic characteristics, and management decisions were analyzed.

Results: The three patients (two females, one male; mean age 41 years) presented with headaches (n=2) or transient neurological deficits (n=1). TL-MCA was left-sided in two cases and right-sided in one. An MCA trunk was present in one patient and absent in two. All demonstrated a plexiform arterial network replacing the M1 segment. Perfusion imaging was normal or showed no mismatch, and conservative management was favored.

Conclusions: TL-MCA is a non-progressive congenital anomaly that mimics Moyamoya disease radiologically and clinically. Accurate differentiation is essential to avoid unnecessary revascularization surgery. Combined MRI-angiography assessment is critical for diagnosis and management. Treatment should be individualized, with revascularization reserved for patients demonstrating perfusion-diffusion mismatch or progressive ischemia.

Introduction

Twig-like middle cerebral artery (TL-MCA) is a rare congenital vascular anomaly characterized by replacement of the M1 segment by a plexiform network of small, primitive arterial twigs [1]. Because of its rarity and its angiographic resemblance to Moyamoya disease or arteriovenous malformations (AVMs), TL-MCA is frequently misdiagnosed. The reported prevalence ranges from 0.08% to 0.45% across diagnostic angiographies [2,3]. The anomaly was first described by Cekirge et al. in 2005, who reported

Corresponding author:
Loucif Houari

Clinic Fatema Al Azhar, Algiers, Algeria

lhouari1@jh.edu

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an occluded MCA origin supported by a collateral plexiform network, attributed to incomplete embryonic fusion of the MCA [4]. Liu et al. subsequently coined the term "twig-like MCA" and emphasized its association with aneurysm formation [5]. This study presents three cases of TL-MCA initially misdiagnosed as Moyamoya disease. We expand on the radiological criteria, embryological basis, and clinical implications, and propose a structured diagnostic and management approach relevant for neurosurgeons.

Material and Methods

A retrospective review of medical records was conducted for patients referred for treatment of Moyamoya disease. As a national reference center, we documented 24 patients diagnosed with Moyamoya disease or syndrome between 2019 and 2023. Three patients with TL-MCA were found to have been mistakenly diagnosed with Moyamoya disease by referring physicians. We report their clinical presentation, initial symptoms, and findings from angiography and MRI.

Between 2019 and 2022, 3 of the 24 patients referred for Moyamoya had TL-MCA. The group consisted of two females and one male, aged 20-54, with a mean age of 41. Two patients experienced only headaches, while one had dysarthria and hemiparesis rated 3/5. This patient's neurological symptoms had emerged 18 months earlier, but she recovered from the motor deficit.

Regarding the angiographic findings, TL-MCA was on the left side in two patients and on the right side in one. An MCA trunk was seen in one patient, but the other two had no trunk despite all having a plexiform web (Table 1).

Table 1. Clinical and imaging characteristics of the three patients with TL-MCA. TL-MCA: twig-like middle cerebral artery; MCA: middle cerebral artery.

	Age (years)	Sex	Symptoms at onset	Symptoms at 1st consultation	Side	MCA trunk	Plexiform network
1	20	Female	Headaches	Headaches	Right	Yes	Yes
2	54	Male	Headaches	Headaches	Left	No	Yes
3	51	Female	Dysarthria, hemiparesis	Slight dysarthria	Left	No	Yes

Illustrative Cases

Case 1

A 51-year-old woman presented 18 months earlier with a 3/5 right hemiparesis and dysarthria. MRI revealed a frontal ischemic stroke and signs of hypoperfusion in junctional areas. Perfusion imaging showed reduced cerebral blood volume (CBV), particularly around the ischemic region. Cerebral angiography demonstrated an aplastic left middle cerebral artery (MCA) lacking a trunk and displaying a plexiform arterial network (Figure 1).

The patient's motor deficit improved, although mild dysarthria persists. The treatment approach was similar to that for Moyamoya disease; because there was no mismatch between perfusion and diffusion sequences, surgical intervention was not recommended.

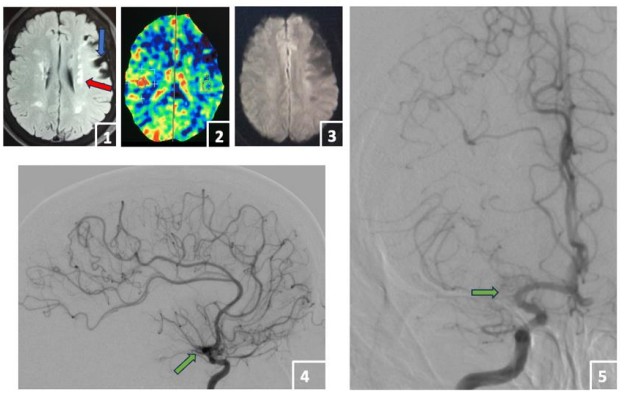

Figure 1. Case 1. Image 1: Axial brain MRI, T2 FLAIR sequence, showing an ischemic stroke in the left frontal area (blue arrow), with hypersignal paraventricular lesions indicating white matter ischemia at junctional zones (red arrow). Image 2: Perfusion MRI showing hypoperfusion and decreased CBV in the penumbra regions. Image 3: Diffusion MRI showing an ischemic stroke in the left frontal region. Image 4: Lateral cerebral angiography showing absence of an MCA trunk with a plexiform vessel network of small arteries (green arrow), and a normal carotid and anterior cerebral artery. Image 5: Frontal cerebral angiography showing no discontinuity in the carotid artery and absence of the MCA, replaced by a mildly dense plexiform vessel network (green arrow), with poor cortical perfusion in the inferior parietal area.

Case 2

A 21-year-old woman with persistent headaches unrelieved by common analgesics consulted a neurologist. MRI revealed lesions in the right basal ganglia that appeared hyperintense on T2-weighted images. Some lesions were vessel-centered and showed hypointensity on T1-weighted images. The radiologist described a "cheese-like" appearance, raising suspicion for Moyamoya disease or a vascular malformation nidus, prompting angiography.

Cerebral angiography showed a short trunk of the MCA with early bifurcation and a plexiform vascular network at the distal M1 segment, along with reduced blood flow in the distal cortical branches of the MCA (Figure 2).

Perfusion and diffusion studies were normal, and the patient responded well to migraine medication. Conservative management was recommended.

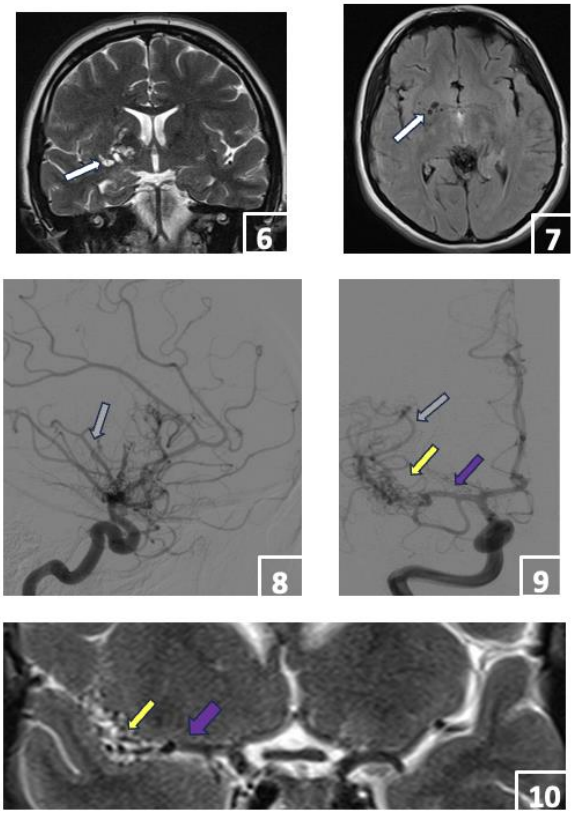

Figure 2. Case 2. Image 6: Coronal T2-weighted MRI showing hyperintense lesions in the basal ganglia, centered around a vessel (white arrow). Image 7: Axial FLAIR MRI showing multiple circular hypointense lesions of different sizes in the right basal ganglia (white arrow). Image 8: Profile cerebral angiography showing a plexiform network around the MCA bifurcation, with small distal cortical MCA arteries marked by the grey arrow. Image 9: Frontal cerebral angiography with right carotid contrast showing an M1 trunk measuring 24 mm, a pre-bifurcation MCA branch arising from the middle of the trunk (purple arrow), a plexiform network of small vessels beyond the trunk (yellow arrow), and distal MCA cortical branches (grey arrow). Image 10: Coronal T2-weighted MRI showing the M1 trunk (purple arrow) followed by a plexiform network (yellow arrow).

Case 3

A 54-year-old man with no relevant medical history had ongoing headaches for years. Contrast-enhanced MRI revealed a missing segment of the left MCA and a plexiform pseudo-network. T2-weighted MRI sequences confirmed the presence of an M1 trunk and MCA bifurcation.

Cerebral angiography showed a TL-MCA appearance with an atretic MCA segment at its origin and a plexiform network extending to cortical branches (Figure 3).

The patient received migraine medication that alleviated the headaches. Given normal perfusion and diffusion studies, a conservative management plan was recommended.

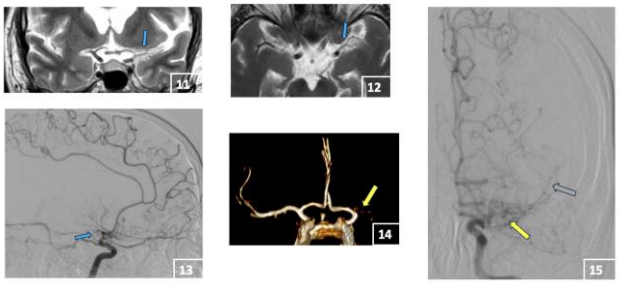

Figure 3. Case 3. Image 11: Coronal T2-weighted MRI showing the M1 trunk (blue arrow) and the bifurcation of the MCA. Image 12: Axial T2-weighted MRI showing the M1 trunk (blue arrow) alongside the MCA bifurcation. Image 13: Lateral cerebral angiography showing no MCA trunk, with a plexiform network of small arteries (blue arrow). Image 14: 3D reconstruction from angio-MRI showing absence of the left MCA, replaced by a small vessel network (yellow arrow). Image 15: Frontal cerebral angiography showing an intact carotid artery without discontinuity; the MCA is absent and replaced by a mildly dense plexiform vessel network (yellow arrow), with cortical distal MCA branches marked by a grey arrow.

Discussion

The embryological basis of TL-MCA is increasingly understood to result from a failure of fusion and regression of the primitive arterial twigs that normally coalesce into the definitive M1 segment between the 5th and 7th gestational weeks [6].

Recent research on vascular development indicates that incomplete fusion of the lateral striate arterial plexus leads to a persistent fetal arterial network. This network lacks a fully developed internal elastic lamina and exhibits reduced smooth muscle differentiation, explaining why the vessel walls are fragile and more susceptible to flow-related aneurysms [7,8].

This congenital persistence is markedly different from Moyamoya disease, a progressive vasculopathy caused by inflammation. It features fibrocellular

hyperplasia of the terminal ICA and endothelial dysfunction associated with RNF213 [1,9].

From a radiological perspective, shown in Table 2, MRI alone is insufficient for diagnosis because TL-MCA can mimic various conditions. These include basal ganglia hyperintensities, vessel-centered lesions, and watershed ischemia, which may resemble early Moyamoya disease, vasculitis, or concealed AVMs [10]. High-resolution vessel-wall MRI has recently demonstrated effectiveness in differentiating TL-MCA from Moyamoya disease by showing the absence of concentric ICA wall thickening, a feature typically seen in Moyamoya vasculopathy [11].

Therefore, angiography remains the gold standard, as supported by the five diagnostic criteria outlined by Ota et al. [12], which remain the most dependable framework, although newer series have highlighted that the MCA trunk might be partially present or hypoplastic rather than entirely absent [13].

Clinically, the primitive arterial twigs of TL-MCA are hemodynamically inefficient, producing low-flow states that may manifest as headaches, transient ischemic symptoms, or ischemic stroke, while hemorrhage — though rare — has been reported in cases with associated aneurysms [2,7,14].

Table 2. Radiological distinction between twig-like middle cerebral artery and Moyamoya disease.

Feature	TL-MCA	Moyamoya disease
Laterality	Typically unilateral	Often bilateral
M1 segment	Absent or replaced by a plexus	Stenotic or occluded
ICA involvement	Normal	Progressive stenosis
Collaterals	Localized plexiform network	Basal "puff-of-smoke" collaterals
Natural history	Non-progressive	Progressive vasculopathy

Decision Making and Management

Since Moyamoya disease and TL-MCA both cause reduced cerebral blood flow, it is reasonable to consider direct extracranial-to-intracranial revascularization via bypass surgery, combined with indirect techniques, in appropriately selected patients.

For Moyamoya disease, the choice to perform extracranial revascularization of the middle cerebral artery depends on imaging results. Perfusion and diffusion MRI sequences are crucial for surgical planning. A mismatch between these modalities suggests a penumbra region that could benefit from bypass. If no mismatch or signs of hypoperfusion are observed, the patient should undergo regular imaging follow-up as scheduled [5].

In cases reported in the literature where no progression of the steno-occlusive lesion was observed, invasive angiography was replaced with routine MRI monitoring. MRI perfusion and diffusion sequences were used, and a conservative approach was adopted, based on the absence of mismatch in one patient and normal cerebral perfusion in the others.

Antiplatelet therapy is recommended during the stroke period. However, long-term dual antiplatelet treatment was avoided due to bleeding risk.

A schematic overview of the decision-making process and management strategies based on clinical presentation is shown in Figure 4 (STA: superficial temporal artery; MCA: middle cerebral artery).

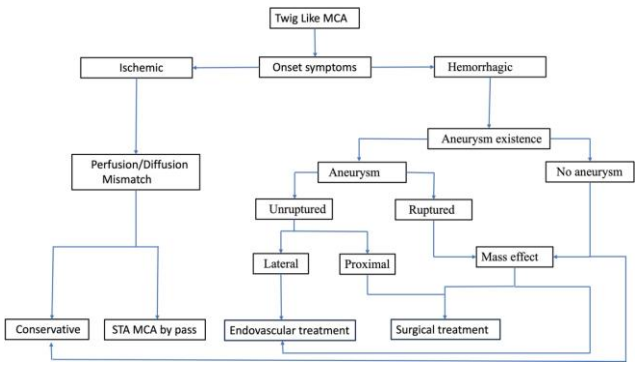

Figure 4. Schematic illustration of twig-like MCA management depending on symptom onset. STA: superficial temporal artery; MCA: middle cerebral artery.

Since TL-MCA is a non-progressive congenital condition, its treatment approach is fundamentally different from that of Moyamoya disease. The main principles involve avoiding unnecessary revascularization surgery — often performed in Moyamoya disease but seldom helpful in stable TL-MCA — and remaining alert for aneurysm formation due to the inherent fragility of the plexiform arterial network [9].

Treatment should be tailored based on symptoms and vascular risk. In our cases, management ranged from conservative observation to specific endovascular procedures when prevention of aneurysms was necessary. Notably, unlike Moyamoya disease, TL-MCA shows long-term stability; serial imaging has shown no progression of stenosis or collateral development over years of

follow-up [3,15]. These differences have significant therapeutic implications. While both conditions can be associated with decreased cerebral perfusion, revascularization in TL-MCA should be limited to patients with a definite perfusion-diffusion mismatch or recurrent ischemic events. Performing extracranial-intracranial bypass in a non-progressive congenital anomaly may provide no physiological benefit and could subject patients to procedural risks [15,16].

Conversely, when cerebral perfusion remains intact, conservative management — including antiplatelet therapy, migraine treatment, and ongoing monitoring for aneurysm development — is suitable. Therefore, a well-structured diagnostic and therapeutic approach that combines MRI, MRA, angiography, and perfusion imaging is crucial to prevent unnecessary surgery and to provide proper long-term surveillance of this rare yet clinically important vascular anomaly.

Conclusion

TL-MCA is a rare congenital vascular anomaly frequently mistaken for Moyamoya disease. Although both conditions may present with similar symptoms and reduced cerebral perfusion, their pathophysiology and natural history differ significantly. Accurate diagnosis relies on combined MRI and angiography. Management should be individualized, with revascularization reserved for patients demonstrating perfusion deficits. Most TL-MCA cases can be safely managed conservatively with clinical and radiological follow-up.

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