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Twig-like middle cerebral artery misdiagnosed as Moyamoya: three cases and a literature review

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ABSTRACT

Introduction: The twig-like middle cerebral artery (TL-MCA) constitutes an uncommon vascular anomaly characterized by the substitution of the M1 segment with a plexiform network of diminutive, delicate vessels. Due to its rarity and similarities in radiological features, it is frequently misdiagnosed as Moyamoya disease or arteriovenous malformation. This publication presents three cases of TL-MCA identified at a single center, providing detailed insights into the radiological and clinical findings, and discussing management strategies through illustrative cases.

Material and methods: We conducted a retrospective review of the records of patients referred for the management of Moyamoya disease. A total of 24 patients diagnosed with either Moyamoya disease or syndrome were included, with assessments encompassing clinical presentation, symptoms at the initial consultation, and findings from angiography and MRI.

Results: Between 2019 and 2022, a total of 24 patients referred for Moyamoya disease were evaluated. Of these, three patients exhibited the terminal segment of the middle cerebral artery (TL-MCA). The cohort included two females and one male, aged between 20 and 54 years, with a mean age of 41 years. Clinical presentations varied: two patients experienced only headaches, while one patient reported dysarthria and hemiparesis. Angiographic assessment revealed that the TL-MCA was on the left side in two patients and on the right side in one patient. In one patient, a distinguishable MCA trunk was observed, whereas in the other two patients, no trunk was identified; instead, a web-like plexiform structure was present in all cases.

Discussion: Moyamoya and TL-MCA are two distinct entities that are frequently conflated owing to their comparable radiological characteristics. Since Moyamoya disease and TL-MCA both cause hypoperfusion of cerebral tissue, consideration should be given to direct extra-intracranial revascularization through bypass surgery, supplemented by indirect techniques.

Conclusion: TL-MCA and Moyamoya disease differ in their pathophysiology and radiological criteria. However, they share similarities in inducing cerebral hypoperfusion and clinical presentation. Consequently, decision-making and management strategies for Moyamoya are also applicable to TL-MCA, and bypass surgery is recommended in specific situations.

Keywords
twig-like middle cerebral artery,
moyamoya,
brain hypoperfusion,
vascular malformation



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INTRODUCTION

The twig-like middle cerebral artery (TL-MCA) is a rare vascular anomaly characterized by the replacement of the M1 segment with a plexiform network of small, fragile vessels (1). Due to its rarity, it is frequently misdiagnosed as Moyamoya disease or arteriovenous malformation, given the similarity in key radiological findings.

Literature regarding TL-MCA is limited to case reports, which constitute approximately 0.08% to 0.45% of all diagnostic angiographies (2,3). In 2005, Cekirge *et al.* documented a case involving an occluded origin of the MCA trunk with a collateral plexiform network of small arteries extending beyond, attributing the anomaly to a failure in the embryonic fusion of the MCA (4). In the same year, Liu *et al.* reported two similar cases and designated them as twig-like MCA (5). Both authors described the anomaly as being associated with aneurysms.

In this publication, we present three cases of TL-MCA identified at a single center (Al Azhar Clinic, Algiers, Algeria, North Africa), which were initially misdiagnosed and referred for management as Moyamoya disease of the middle cerebral artery.

The radiological and clinical findings are detailed, and the approach to management is discussed through illustrative cases.

MATERIAL AND METHODS

We conducted a retrospective review of the records of patients referred for the management of Moyamoya disease. As a national reference center for Moyamoya, we received a total of 24 patients diagnosed with either Moyamoya disease or syndrome. The TL-MCA was initially misdiagnosed as Moyamoya disease by their referring physicians; however, the diagnosis was subsequently corrected. The report includes the clinical presentation, symptoms at the initial consultation, and findings from angiography and MRI.

RESULTS

Between 2019 and 2022, a total of 24 patients referred for Moyamoya disease were evaluated (see Table 1). Of these, three patients exhibited the terminal segment of the middle cerebral artery (TL-MCA). The cohort included two females and one male, aged between 20 and 54 years, with a mean age of 41 years. Clinical presentations varied: two patients experienced only headaches, while one patient reported dysarthria and hemiparesis graded 3/5; the neurological symptoms had an onset 18 months prior, and the patient has since fully recovered from the motor impairment. Angiographic assessment revealed that the TL-MCA was on the left side in two patients and on the right side in one patient. In one patient, a distinguishable MCA trunk was observed, whereas in the other two patients, no trunk was identified; instead, a web-like plexiform structure was present in all cases.

Table 1. Pooled clinical and imaging features.

Patient	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 prior to consultation with right hemiparesis rated 3/5 and dysarthria. The patient underwent magnetic resonance imaging (MRI), which revealed an ischemic stroke in the frontal region (indicated by a blue arrow in image 1) and signs of hypoperfusion in junctional areas (indicated by a red arrow in image 1). Perfusion sequences demonstrated a decrease in

cerebral blood volume (CBV), particularly in the region surrounding the ischemic stroke.

Cerebral angiography showed an aplastic left middle cerebral artery (MCA) with an absent trunk and a plexiform arterial network (indicated by a green arrow in images 3 and 3,4).

The patient experienced improvement in her motor deficits; however, she continues to have mild dysarthria. The clinical decision was to consider Moyamoya disease, as indicated by the absence of a

mismatch between perfusion and diffusion sequences. Consequently, surgical intervention was not recommended.

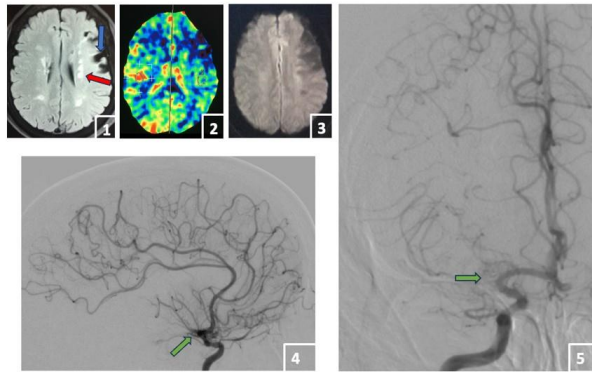


Figure 1. Case 1. (Image 1) Brain axial MRI, T2 FLAIR sequences display ischemic stroke in the left frontal region (blue arrow), with hypersignal paraventricular lesions corresponding to white matter ischemia in junctional areas (red arrow). (Image 2) Perfusion MRI sequences, demonstrating hypoperfusion and decreased CBV in the blue penumbra regions. (Image 3) Diffusion sequence MRI demonstrating a left frontal ischemic stroke. (Image 4) Lateral cerebral angiography demonstrates the absence of a middle cerebral artery (MCA) trunk and the presence of a plexiform vascular network of small arteries (green arrow). The carotid artery and anterior cerebral artery are within normal limits. (Image 5) Frontal cerebral angiography demonstrates no discontinuity in the carotid artery, with the absence of the MCA replaced by a mildly dense plexiform vessel network (green arrow). The inferior parietal region exhibits poor cortical perfusion.

Case 2

A 20-year-old woman presented with a long-standing history of headaches unrelieved by standard analgesics. She consulted a neurologist who ordered an MRI scan.

The MRI revealed lesions in the right basal ganglia that appeared hyperintense on T2-weighted images. Some lesions, centered around vessels (white arrow, Image 6), appeared hypointense on T1-weighted images (white arrow, Image 7). The radiologist suspected the characteristic 'puff of smoke' appearance associated with Moyamoya disease or a nidus of arteriovenous malformation (AVM), prompting an angiographic assessment.

Cerebral angiography demonstrated a short primitive trunk of the middle cerebral artery (MCA) measuring a few centimeters with an early bifurcation (purple arrow), followed by a plexiform vascular network at the distal M1 segment (yellow

arrow). Additionally, there was evidence of reduced blood flow in the distal cortical branches of the MCA (grey arrows, Images 8 and 8,9).

Perfusion and diffusion studies appeared normal. The patient responded favorably to migraine pharmacotherapy. Conservative management was recommended.

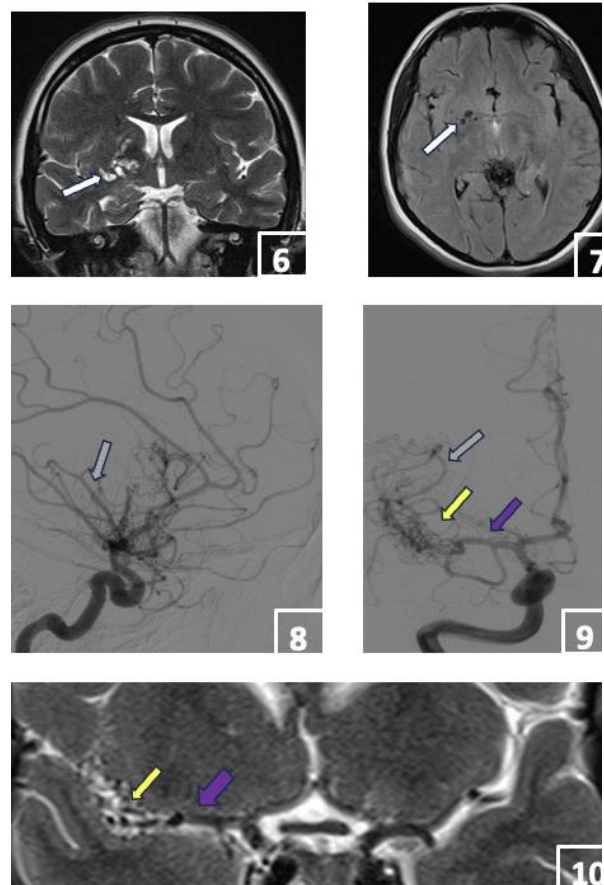


Figure 2. Case 2. (Image 6) Coronal T2-weighted MRI sequence illustrating hyperintense lesions in the basal ganglia, centered around a vessel (white arrow). (Image 7) Axial Flair MRI sequence, exhibiting multiple hypointense circular lesions of varying sizes in the right basal ganglia (white arrow). (Image 8) Cerebral angiography profile demonstrating a plexiform network surrounding the MCA bifurcation, with small distal cortical MCA arteries indicated by the grey arrow. (Image 9) Frontal cerebral angiography demonstrating right carotid enhancement. The un M1 trunk measures 24 mm in length, with pre-bifurcation middle cerebral artery (MCA) branches emanating from the midsection of the trunk (purple arrow). A plexiform network of small vessels is observed beyond the trunk (yellow arrow), followed by distal cortical branches of the MCA (grey arrow). (Image 10) Coronal T2-weighted MRI sequence illustrating the M1 trunk (indicated by the purple arrow), followed by a plexiform network (indicated by the yellow arrow).

Case 3

A 54-year-old man with no prior medical history presents with a longstanding history of headaches. An MRI with contrast revealed the absence of a segment of the left middle cerebral artery (MCA) (see image 14, indicated by the yellow arrow), exhibiting a plexiform pseudo-network. T2-weighted MRI sequences demonstrated the presence of the M1 trunk and MCA bifurcation (indicated by the blue arrows in images 11 and 12). Cerebral angiography revealed an abnormal appearance of the left MCA with an amputation at its origin (blue arrow, image 13) and identified a plexiform network (yellow arrow, image 15), followed by cortical branches (grey arrow, image 15). The patient was managed with migraine medications, which alleviated the headache symptoms. Given that perfusion diffusion studies were within normal limits, a conservative management approach was adopted.

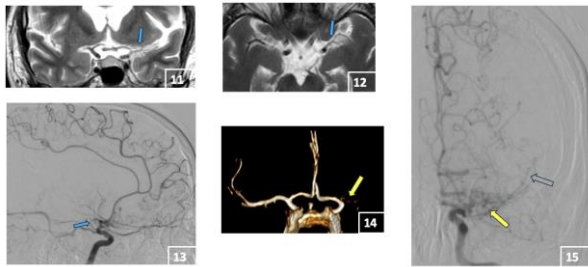


Figure 3. Case 3. (Image 11) Coronal T2-weighted MRI sequence illustrating the M1 trunk (indicated by the blue arrow), followed by a bifurcation of the middle cerebral artery (MCA). (Image 12) Axial T2-weighted MRI sequence depicting the M1 trunk (indicated by the blue arrow), followed by a bifurcation of the middle cerebral artery (MCA). (Image 13) Lateral cerebral angiography demonstrates the absence of an MCA trunk and the presence of a plexiform vessel network of small arteries (blue arrow). A normal carotid artery and anterior cerebral artery are also observed. (Image 14) Three-dimensional reconstructions of an angiographic magnetic resonance imaging (MRI), illustrating the absence of the left middle cerebral artery (MCA), which has been substituted by a small vascular network (yellow arrow). (Image 15) Frontal cerebral angiography demonstrates continuity in the carotid artery, with an absent MCA replaced by a mildly dense plexiform vascular network (yellow arrow). Cortical distal MCA branches are indicated by the grey arrow.

DISCUSSION

TL-MCA refers to congenital anomalies resulting from interference in the process of fusion and regression during the development of the embryonic

MCA (6). The angiographic architecture is characterized by a plexiform fetal arterial network replacing a steno-occlusive MCA trunk.

In our series, diagnosis via MRI and CT scan is often challenging, with occasional misdiagnosis, including unilateral Moyamoya in two patients, and MCA arteriovenous malformations in one patient. Cerebral angiography remains the gold standard.

Ota *et al.* summarized angiographic findings in TL-MCA (7); comparing the common features reported across cases, the following are noted: 1) a steno-occlusive lesion at the unilateral MCA (1,5,8), except for one bilateral case (9); 2) a plexiform arterial network replacing the proximal MCA trunk; 3) lenticulostriate arteries (LSAs) arising from this plexiform arterial network; 4) normal cortical branches beyond the plexiform network; 5) compromised blood flow to the distal MCA with contributory leptomeningeal collaterals (6).

In our series, the diagnosis of TL-MCA was established based on these radiological features; however, some discrepancies and unique observations were noted:

In the first and third illustrative cases, the patients exhibited a direct continuation of the internal carotid artery with the anterior cerebral artery, absent any apparent bifurcation or steno-occlusive trunk. Conversely, in the second illustrative case, a primitive M1 trunk measuring 24 mm was present. All patients demonstrated a plexiform vessel network and distal MCA branches; LSAs were developed but were often mistaken as part of the plexiform network, making the distinction challenging.

In the final illustrative case, angiography suggested a TL-MCA diagnosis due to the absence of the MCA, presence of a plexiform network, and distal MCA branches. However, T2 MRI sequences showed the anatomical presence of M1 bifurcations and M2 segments (images 11,12).

Angiography permits tracking dye progression and evaluating vascular patency, but does not provide information about anatomical variations, such as when a vessel exists but fails to fill. Stenotic or spastic vessels cannot be visualized through angiography.

Complementary MRI sequences should be employed to support diagnosis and inform decision-making.

A normally functioning MCA can support pressure transmission from the ICA (2). However,

fetal arterial branches are typically fragile, and the primitive arterial walls of the plexiform network are vulnerable to hemodynamic stress, potentially leading to aneurysm formation and rupture (10).

In our series, two patients experienced headaches, and one had a neurologic deficit 18 months prior, caused by an ischemic stroke. Contrary to the existing literature, no hemorrhagic strokes were reported.

Since Moyamoya disease and TL-MCA both induce hypoperfusion of brain tissue, direct extra-intracranial revascularization via bypass surgery, combined with indirect techniques, should be considered.

In decisions regarding intervention, as with Moyamoya disease, brain perfusion guides the choice. Perfusion and diffusion MRI sequences are critical; a mismatch between these sequences indicates an ischemic penumbra that could benefit from bypass surgery. In the absence of mismatch or hypoperfusion, patients should be informed of potential risks and monitored through periodic imaging.

Since the cases reported in the literature highlighted no progression of the steno-occlusive lesion, invasive assessment with angiography has been replaced by periodic perfusion MRI.

In our patients, we perform MRI perfusion and diffusion sequences. A conservative treatment was decided based on the absence of a mismatch in one patient and normal brain perfusion in the other two patients.

Antiplatelet therapy should be used during the stroke period. However, since the increased risk of hemorrhage, we avoided a long-term continuation of dual-antiplatelet therapy.

CONCLUSION

TL-MCA and Moyamoya disease differ in their physiopathology and radiological criteria. However, they share similarities in inducing brain

hypoperfusion and clinical presentation. Consequently, decision-making and management strategies for Moyamoya are also applicable to TL-MCA, and bypass surgery is recommended in specific situations.

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