Pediatric Moya-Moya disease and syndrome: experience in our pediatric hospital

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Learning objectives

- To describe the general features of moyamoya vasculopathy and the special features of pediatric disease.

- To explain the difference between moyamoya disease and moyamoya syndrome.

- To mention the diagnostic criteria and the special considerations for pediatric patients.

- To discuss the imaging findings of moyamoya vasculopathy and the different imaging techniques to acquire them.

- To describe the treatment and the role of imaging techniques in the pre and post-surgical management.
Background

Moyamoya disease (MMD) is characterized by progressive narrowing (and ultimately, occlusion) of the terminal internal carotid artery (ICA) and proximal middle and anterior cerebral arteries (MCA and ACA).

Takeuchi and Shimizu were the first to describe this disease in Japan in 1957 as "hypoplasia of the bilateral internal carotid arteries". It was called "moyamoya disease" for the first time by Suzuki and Takaku in 1969.

The term, "moyamoya," is a Japanese expression signifying "something hazy, like a puff of cigarette smoke drifting in the air," and is used because of the distinctive angiographic appearance of the collateral network. These arteries, called moyamoya vessels, provide blood flow to areas of hypoperfused brain in response to ICA stenosis.

Moyamoya disease typically refers to the idiopathic form of the arteriopathy, usually bilateral, whereas moyamoya syndrome (MMS) represents cases in which the characteristic angiographic findings occur in association with other pathological processes and are usually unilateral. Nevertheless, their angiographic and clinical courses are nearly identical.

EPIDEMIOLOGY

The highest known prevalence of MMD is in Japan, however the disease occurs worldwide.

Epidemiological studies found a MMD prevalence of 3.16-10.5/100,000 and an incidence of 0.35-1.13/100,000/year in Japan. The incidence in Europe is one-tenth the incidence in Japan.

A familial history is noted in 10% to 15% of cases in Japan.

It is more prevalent in women than men (2.18:1).

There is a bimodal age distribution, with patients typically presenting either in the 1st or 4th decade of life.

ETIOLOGY
Pathogenesis and etiology of MMD remain unknown.

The strong prevalence of MMD in Japan suggests a genetic trait associated with the disease and some studies have described an association between MMD and several chromosomes like 17q25.

Despite the genetic features of the disease, sporadically occurring MMD is still the most common form.

A crucial step in the examination of patients who have moyamoya angiopathy is the research of underlying condition or neurological and extra-neurological signs that would suggest MMS (table 1).

**CLINICAL PRESENTATION**

MMD typically presents acutely with cerebrovascular events:

- Ischemic events (transient ischemic attack (TIA), strokes): More common in children. MMD represents 6% of strokes in children. 80% of children with MMD presents with evidence of cerebral ischemia.

Ischemic symptoms are often instigated by crying, coughing, blowing or hyperventilation. In these patients, cortical vessels are most often maximally dilated to compensate for chronic cerebral ischemia and such activities lead to carbon dioxide CO2 decrease which induces vasoconstriction resulting in reduced cerebral perfusion.

Symptoms related to posterior circulation ischemia (visual field defects, decreased visual acuity, transient blindness, scintillating scotomas, diplopia, ataxia, vertigo...) are uncommon presenting features in both children and adults. However, when they do occur, they are observed more often in children.

- Intracranial hemorrhage: More characteristic of adult onset.

- Epileptic seizures: More often in children younger than 10 years of age. Related to cortical ischemia.

- Headaches: Likely resulting from dilation of meningeal and leptomeningeal collateral vessels.

There are also asymptomatic cases in which MMD is found incidentally.
NATURAL HISTORY

The rate of disease progression is variable but despite the course, the disease inevitably progresses in untreated patients, with angiographic and clinical worsening over time that can lead to irreversible neurological deficits.

Progression of occlusion is more common and quick in children than adults (54% of pediatric patients over a mean period of 5.4 years and in 23.8% of adult patients over a mean period of 6.1 years). This is especially true in patients younger than 2 years of age, accounting for their poor prognosis.

Several factors determine the overall prognosis of patients with moyamoya disease:
- the rapidity and extent of vascular occlusion
- the ability to develop effective collateral circulation
- the age at onset of symptoms
- the severity of presenting neurological deficits and degree of disability

Although most MMD patients present with bilateral involvement, up to 18% of patients have angiographically documented unilateral involvement. The progressive bilateralization is more frequent in pediatric patients.

PATHOLOGY

In MMD, stenotic changes initially appear in the intracranial ICAs distally at the level of their bifurcation. Later, the stenosis progresses to involve the proximal ACAs and MCAs. During subsequent stages, the posterior circulation may also become involved.

Histological changes of the stenotic arteries:
- Endothelial hyperplasia and fibrocellular thickening of the intima
- Tortuosity or undulation and sometimes duplication of the internal elastic lamina
- Attenuation of the media
The fibrocellular intimal thickening noted in the intracranial arteries is also observed in other arteries of patients with MMD (extracranial arteries, pulmonary arteries, renal arteries, hepatic arteries and coronary arteries).

The compensatory recruitment of new vessels provides collateral blood flow to areas of hypoperfused brain distal to the narrowed vessels. There are three basic collateral pathways:

- Moyamoya vessels from parenchymal perforators
- Leptomeningeal collateral vessels from the posterior cerebral artery
- Transdural collateral vessels from the external carotid

As posterior cerebral artery stenosis advances, the leptomeningeal vessels from posterior to anterior circulation decreases.

Histological changes of the moyamoya vessels:

- Thin walls
- Mural fibrin deposits
- Fragmentation of the elastic lamina
- Microaneurysm formation

Cerebral aneurysms have been associated with MMD but in children are less frequent (1%) than in adults (6%).

Arteriovenous malformations (AVMs) can occur, typically in the MCA territory but the relationship between AVMs and moyamoya disease remains unknown.
MOYAMOYA SYNDROME

- Prior cranial irradiation
- Skull base tumor
- Genetic disorders (Down syndrome, neurofibromatosis type 1, tuberous sclerosis, Alagille syndrome, Hirschsprung syndrome ...)
- Hematologic conditions (Fanconi anemia, sickle cell anemia, homocystinuria, essential thrombocythemia...)
- Autoimmune diseases (Graves, Systemic Lupus Erithematos...)
- Collagen vascular disorders (Marfan syndrome, Ehler-Danlos...)
- Congenital cardiac disease
- Renal artery stenosis
- Infections (tuberculous meningitis, leptospirosis...)
- Atherosclerosis
- Fibromuscular dysplasia
- Cranial trauma

Table 1: Moyamoya syndrome causes

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Findings and procedure details

**DIAGNOSIS** *(table 2)*

**Conventional digital subtraction angiography:**

Remains the gold-standard imaging technique to demonstrate the characteristic features of this angiopathy. These features are not pathognomic because the stenotic lesions themselves may be indistinguishable from other cerebrovascular angiopathies.

The prominent collateral vascular network results in the dense angiographic blush occurring in the arterial phase, which is responsible for the "puff of smoke" description *(Figure 1)*.

There are 6 angiographic stages of MMD that appear with progression of the disease *(table 3)*.

Because of the invasivity of the technique, is realized in limited circumstances:

- Uncertain diagnosis or progression of the angiopathy during follow-up with non-invasive techniques.

- Pre-surgical management (because is the most accurate procedure to obtain a detailed mapping of collateral networks, which is essential for planning surgical treatment and avoiding disrupting existing collaterals during revascularization procedure) and sometimes evaluation after surgical revascularization *(Figure 2)*.

**Computed tomography (CT):**

CT is rapid and easily available. Its main disadvantage is the use of ionizing radiation, which is a point to keep in mind always in the pediatric population.

It frequently shows *(figure 3)*:
- Areas of hypodensity (infarct in cortical watershed zones, basal ganglia, deep white matter, or periventricular regions).

- Hemorrhage (rare in children. The most common sites are the basal ganglia, ventricular system, medial temporal lobes and thalamus).

- Atrophy of the affected hemisphere (frequently seen in patients who have had severe stroke).

- Ventricular dilatation and subarachnoid space widening (resulting from chronic ischemic lesions).

- Gyral enhancement after contrast administration.

- Punctate enhancement in the basal ganglia after contrast administration (enlarged lenticulostriate artery collaterals).

- Arterial narrowing and collateral vessels at the base of the brain on angio-CT.

**Magnetic resonance imaging (MRI) and MR angiography (MRA):**

Because of its excellent diagnostic yield and noninvasiveness, may be used as a surrogate for conventional angiography in children (table 4).

This technique provides information of cerebral arteries, brain parenchyma, and cerebral perfusion, so it is a good method to detect MMD, especially when it is in Stage 3 or 4.

The increased signal gained with stronger 3-T magnets provides significantly greater angiographic detail that has been used for the better selection of bypass arteries for revascularization surgery.

Also, can be used postoperatively to determine the state of collateral flow and patency of extracranial-intracranial bypass grafts (Figure 4).

Disadvantages:
- It is not very effective as a means of staging the disease because it is limited in spatial resolution and flow direction evaluation and often overestimates degree of stenosis.

- It poorly visualize smaller moyamoya vessels.

MRI shows:

- Stenosis or occlusion of the distal internal carotid artery: T2-weighted images allow to visualize the stenotic arteries directly as diminished flow voids in the ICA, MCA, and ACA (Figure 5 and 6).

- Moyamoya vessels: Multiple punctate flow voids representing enlarged collaterals arteries in the basal ganglia are seen in T1-weighted images. They enhance in contrast-enhanced T1-weighted images. FLAIR images show punctate hyperintensities that represent slow flow in enlarged lenticulostriate collaterals (Figure 7 and 8).

- Hemorrhagic lesions (their behavior in both T1- and T2-weighted images varies according to the chronology of bleeding).

- Ischemic lesions: White matter hyperintensities located in the distal vascular bed supplied by penetrating branches of MCA/ACA and related to border-zone infarcts are frequently seen in T2-weighted imaging. FLAIR images are better for showing subtle parenchymal changes and show parenchymal hyperintensity sooner than other sequences. Small or/and acute infarcts are well seen using diffusion or perfusion MR techniques. Chronic infarcts are better delineated with T1- and T2-weighted imaging (Isointense to cerebrospinal fluid (CSF) (Figure 9, 10 and 11).

- Microbleeds: Reported to be a predictor for hemorrhage. They are seen in T2* GRE and SWI sequences.

- Ivy sign: Linear hypersignals that follow a sulcal pattern on FLAIR sequences and marked leptomeningeal enhancement in Contrast-enhanced T1-weighted images (that may be better than FLAIR images for depicting it). It represent a retrograde slow flow of engorged
pial vasculature toward an ischemic area via leptomeningeal collaterals. It is seen in hemispheres with diminished cerebrovascular reserve. Relatively common and characteristic (Figure 12 and 13).

- Brush sign: Prominent hypointense signals seen in T2* GRE and SWI in the draining veins found in patients with TIA and infarct that indicate increased oxygen extraction or venous stasis, implying impaired perfusion in this area (Figure 14).

- Others: Gliosis, atrophy, ventriculomegaly.

- MR findings suggesting underlying disease (Figure 15).

MR angiography shows (Figure 16,17 and 18):

- Stenosis or occlusion of arteries.

- Moyamoya vessels: unusual gangliobasal vessels.

Hemodynamic and metabolic studies:

- Single Photon Emission Computed Tomography (SPECT) (Figure 19)

- Perfusion Computed Tomography

- Xenon-enhanced CT

- Positron Emission Tomography (PET)

- MRI-based perfusion methods: Dynamic Susceptibility Contrast (DSC), Dynamic Contrast-Enhanced (DCE) and Arterial Spin Labeling (ASL)

Perfusion MRI:

MRI perfusion sequences allow the estimation of the degree of cerebral hypoperfusion.
DSC: blood oxygen level-dependent MR imaging used to map cerebrovascular reactivity in cases with arterial stenoocclusive disease. This is a contrast-enhanced dynamic susceptibility imaging based on the decrease in T2 signal that occurs with an increased concentration of intravoxel deoxyhemoglobin, which is related to either reduced perfusion or increased metabolic activity. It has been shown to correlate well with PET.

DCE: analyzes the temporal enhancement pattern of a tissue following the introduction of a paramagnetic contrast agent (CA) into the vascular system. During the first-pass of the CA through the circulation (typically 45-60 s after injection), CA is predominantly intravascular allowing evaluation of perfusion (i.e. blood flow per unit volume or mass of tissue), relative blood volume (rBV) and mean transit time. During the subsequent 2-10 min, there is increasing passage of CA into the extravascular space, and imaging during this delayed phase enables measurement of vascular permeability and relative extravascular volume.

ASL: alternative noncontrast, noninvasive perfusion technique, so it represents an ideal tool for the assessment of cerebral perfusion in children. It is based on the T1 magnetization state of electromagnetically tagged, freely diffusible arterial water. Blood flow measurements with ASL have been shown to correlate well with those from DSC imaging.

All the three are limited by the prolonged transit times in MMD, which stretches the sensitivity of these techniques, resulting in greater artifact.

Patterns observed in pediatric patients with MMD are (Figure 20,21 and 22):

- Decrease of global cerebral blood flow (CBF) with a predominant posterior CBF distribution.
- Impaired cerebrovascular reactivity to acetazolamide or carbon dioxide in the ICA territory, suggesting reduced cerebrovascular reserve (CVR).
- Compensatory increase of cerebral blood volume (CBV) by cerebral vessels vasodilation, and increase of oxygen fraction extraction (OEF).
- Increase of Mean Transit Time (MTT) and Time To Peak (TTP) due to the presence of collateral vessels that introduces large delays and is likely to disperse contrast agent.

These modifications are usually more prominent in pediatric than adult patients.
Electroencephalography:

50% of pediatric patients show a hyperventilation-induced diffuse pattern of monophasic slow waves (buildup) followed by a characteristic "re-buildup" phenomenon (slow waves after hyperventilation has stopped). This electroencephalography finding is characteristic of moyamoya disease and is thought to represent cerebral ischemia due to vasoconstriction of dilated normal cerebral vessels as a consequence of decreased arterial CO2 tension (Figure 23).

Transcranial Doppler:

Measures blood flow velocity in large intracranial vessels at the circle of Willis but cannot be a sufficient tool for evaluation and follow-up in moyamoya angiopathy.

TREATMENT

Non-ischemic symptoms such as headache and seizure are treated symptomatically with antiepileptic drugs and analgesics.

Ischemic symptoms are treated with acetylsalicylic acid at the dose of 50-100 mg daily, to prevent thrombosis and thromboembolism at sites of arterial stenosis. This may be the sole treatment when surgery revascularization is postponed because of recent infarction, or when surgical bypass is not indicated because the patient has relatively mild disease or good spontaneous revascularization.

Common general measures are recommended in order to prevent deterioration of cerebral hemodynamics, such as avoidance of hypotension, dehydration, and hyperventilation, because of these patient's deficit in cerebrovascular reserve.

Revascularization should be offered to children with evidence of moyamoya, including ongoing ischemic symptoms and/or evidence of compromised blood flow or cerebral perfusion reserve.

Therefore, the evaluation of cerebral hemodynamic status is one of the most important factors to consider when deciding surgical indication.
The revascularization goal is to improve cerebral blood flow and to reduce moyamoya collateral network, thus reducing the risk of new ischemic or hemorrhagic events. There are no firm recommendations on specific timing for surgery, although the general principle of minimizing the time between diagnosis and revascularization is supported.

Surgical revascularization procedures can be divided into:

- Direct procedures: direct anastomosis of arteries between the extracranial and intracranial circulations. Most commonly, the superficial temporal artery is anastomosed to a cortical branch of the middle cerebral artery.

- Indirect procedures: placing of the superficial temporal artery or vascularized tissues such as the temporalis muscle, dura mater, or omentum directly on the brain surface to promote collateral vessel formation. The name of the operation depends on the tissue inserted: encephaloduroarteriosynangiosis (EDAS), encephalogaleo(periosteos)ynangiosis, encephalomyosynangiosis, or encephaloduroarteriomyosynangiosis.

EDAS is the most used indirect procedure. After surgery the superficial temporal artery and/or an adjacent middle meningeal artery participate in neoangiogenesis, which can be detected by using conventional angiography or MR angiography.

The pediatric population is typically treated with indirect revascularization because the likelihood of angiogenesis is higher in children than in adults and a direct bypass is frequently technically not feasible. In addition, performing an anastomosis with very small vessels carries a greater risk of thrombosis of the donor graft or recipient artery.

However, collateral formation and angiogenesis take longer to develop after indirect revascularization (1 or 2 weeks are required for stabilization of symptoms), compared with direct revascularization.

Perfusion MR imaging has been widely used for postoperative follow-up imaging of children with moyamoya disease.

- Reduction of TTP and CBV (because of rapid parenchymal perfusion after revascularization surgery and decrease of collateral vessel formation) can depict hemodynamic status after revascularization. Changes in TTP values can be used to predict clinical outcomes.
**Diagnostic criteria of MMD**

**A. Cerebral angiography is indispensable for the diagnosis and should present at least the following findings:**
1. Stenosis or occlusion at the terminal portion of the ICA and/or at the proximal portion of the ACAs and/or the MCAs.
2. Abnormal vascular networks in the vicinity of the occlusive or stenotic lesions in the arterial phase.
3. These findings should present bilaterally.

**B. When MR imaging and MR angiography clearly demonstrate all the subsequently described findings, conventional cerebral angiography is not mandatory:**
1. Stenosis or occlusion at the terminal portion of the ICA and at the proximal portion of the ACAs and MCAs on MR angiography.
2. An abnormal vascular network in the basal ganglia on MR angiography. Note that an abnormal vascular network can be diagnosed when more than 2 apparent flow voids are observed in 1 side of the basal ganglia on MR imaging.
3. (1) and (2) are observed bilaterally.

**C. Because the origin of this disease is unknown, cerebrovascular disease with an underlying diseases or conditions should thus be eliminated:**

**Diagnosis:**
Definite case: One which fulfills either criteria A or B, and C.
In children, however, a case that fulfills A-1 and A-2 (or B-1 and B-2) on 1 side and with remarkable stenosis at the terminal portion of the ICA on the opposite side is also included.

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**Table 2:** Diagnostic criteria of MMD

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**Fig. 3:** 5 years old caucasian male with MMD. Axial CT shows bilateral postoperative changes, atrophy of both hemispheres, ventricular dilatation and subarachnoid space widening.

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### Table 3: Angiographic stages of MMD

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Diagnostic criteria utilizing MR imaging and angiography

A. When MR imaging and MR angiography clearly demonstrate all the findings described below, conventional cerebral angiography is not mandatory:
1. Stenosis or occlusion at the terminal portion of the intracranial ICA and at the proximal portion of the ACAs and MCAs.
2. An abnormal vascular network in the basal ganglia.
3. 1) and 2) are seen bilaterally.

B. Imaging methods and judgment:
1. More than a 1.0-T magnetic field strength is recommended.
2. There are no restrictions regarding MR angiography imaging methods.
3. The imaging parameters, such as the magnetic field strength, the imaging methods, and the use of contrast medium, should be clearly documented.
4. An abnormal vascular network can be diagnosed when more than two apparent flow voids are observed on 1 side of the basal ganglia on MR imaging.
5. Either an over- or underestimation of the lesion could be made according to the imaging conditions. To avoid a false positive diagnosis, only definite cases should thus be diagnosed on the MR imaging and MR angiography findings.

C. Because similar vascular lesions secondary to other disorders are sometimes indistinguishable from this disease in adults, a diagnosis based on MR imaging and MR angiography without conventional angiography is thus only recommended in pediatric cases.

Table 4: Diagnostic criteria utilizing MR imaging and angiography

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Fig. 1: 15 years old caucasian male with MMS secondary to neurofibromatosis type 1. Angiographic findings include occlusion at the proximal portion of the left MCA (blue arrow) with collaterals vessels suppling the ischemic brain (orange arrow). Suzuki stage 2.

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Fig. 2: 15 years old caucasian male with MMS secondary to neurofibromatosis type 1. Angiographic findings include postsurgical changes with permeable synangiosis (arrow).

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Fig. 4: 5 years old caucasian male with MMD after bilateral direct and indirect surgical revascularization. TOF 3D angio-MR (left) and angio-CT (right) show permeable bilateral superficial temporal artery-MCA anastomosis.

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Fig. 5: 5 years old caucasian male with MMD. Brain MRI. T2-weighted image demonstrating diminished flow voids in both MCA.

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**Fig. 6:** 15 years old caucasian male with MMS secondary to neurofibromatosis type 1. Brain MRI. T2-weighted image demonstrating diminished flow void in left MCA.

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**Fig. 7:** 5 years old caucasian male with MMD. Brain MRI. T1-weighted images (left) show multiple punctate flow voids representing enlarged collaterals arteries in the basal ganglia are seen. They enchance in contrast-enhanced T1-weighted images (right).

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Fig. 8: 5 years old caucasian male with MMD. Brain MRI. FLAIR sequence shows punctate hyperintensities in the basal ganglia representing slow flow in enlarged lenticulostriate collaterals.

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**Fig. 9:** 14 years old african female with MMS secondary to sickle cell disease. Brain MRI. T2-FLAIR weighted images. Chronic cerebral infarction in right MCA-ACA and left ACA territories. Low signal in encephalomalacic areas (green arrows) with hyperintense gliotic white matter at margins (orange arrows). Cortico- subcortical atrophy (blue arrow) with enlargement of the right lateral ventricle. Bone marrow hyperplasia affecting the cranial vault secondary to her underlying disease.

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Fig. 10: 5 years old caucasian male with MMD. Brain MRI. Acute right cortico-subcortical frontal ischemic lesion, hyperintense in T2-FLAIR weighted sequence (left image) and diffusion weighted sequence (middle image) with low signal on ADC maps (right image).

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**Fig. 11:** 9 years old caucasian male with MMS secondary to neurofibromatosis type 1. Brain MRI. T2-weighted image demonstrating ischemic left semioval center injury.

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**Fig. 12:** 13 years old caucasian female with MMD. Brain MRI. T2-FLAIR weighted sequence. Right leptomeningeal collaterals seen as linear hypersignals that follow a sulcal pattern (Ivy sign).

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**Fig. 13:** 5 years old caucasian male with MMD. Brain MRI. T2-FLAIR weighted sequence. Bilateral leptomeingeal collaterals seen as linear hypersignals that follow a sulcal pattern (Ivy sign).

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Fig. 14: 5 years old caucasian male with MMD. Brain MRI. Susceptibility weighted imaging. Moderate hypointense signals in the draining veins (Brush sign).

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Fig. 15: 5 years old caucasian male with MMS secondary to neurofibromatosis type 1. Brain MRI. T2-FLAIR weighted sequences show UBOs (unidentified bright objects) involving globus pallidus and cerebellum. They are areas of increased signal intensity that occur in 43% to 93% of children with neurofibromatosis type 1, suggestive of vacuolization or hamartomatous lesions. It is important not to confuse them with ischemic lesions.

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**Fig. 16:** 5 years old caucasian male with MMD. TOF 3D angio-MR shows occlusion of the supraclinoid portion of both ICA (red arrows), with significant MCAs (blue arrows), ACAs (purple arrows) flow reduction, being more severe on the right side. Prominent collateral vascular network that gives the typical appearance of puff of smoke (green arrows). Suzuki-Takaku grade 3.

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**Fig. 17:** 13 years old caucasian female with MMD. TOF 3D angio-MR demostrating stenosis at the terminal portion of the right intracranial ICA and at the proximal right MCA (orange arrow), with abnormal vascular network in the basal ganglia (green arrow). Distal MCA-dependent vessels are reduced in size. Suzuki stage 2.

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Fig. 18: 14 years old african female with MMS secondary to sickle cell disease. TOF 3D angio-MR demonstrating occlusion of the right ICA, right MCA and bilateral ACAs (stars) with abnormal vascular network. Suzuki grade 3.

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**Fig. 19:** 13 years old caucasian female with MMD. Basal brain perfusion SPECT (left) shows decreased CBF in the right temporal lobe. Acetazolamide stress brain perfusion SPECT (right) shows improvement of CBF. This implies a negative response to acetazolamide, so the cerebrovascular reserve is not reduced.

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Fig. 20: 13 years old caucasian female with MMD. Brain MRI. ASL image (left) demonstrates a slow flow in the superficial territory of right MCA. DSC images (right) show an asymmetric MTT.

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Fig. 21: 14 years old african female with MMS secondary to sickle cell disease. Brain MRI. ASL images demostrate low right frontoparietal and left frontal CBF.

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**Fig. 22:** 15 years old caucasian male with MMS secondary to neurofibromatosis type 1. Brain MRI. DSC image shows a reduced temporo-parietal CBV.

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Fig. 23: Electroencephalogram shows slow waves after hyperventilation has stopped (Re-buildup phenomenon).

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Conclusion

Moyamoya vasculopathy should be considered in any child who presents with symptoms of cerebral ischemia.

MRI and MRA have greater sensitivity for detecting the image features of this disease so may be used as a surrogate for conventional angiography in pediatric diagnosis. In addition, MR specially perfusion-MR, has an important role in pre and post-surgical management.
References


