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## Case Report

# Advanced multimodality neuroimaging of a giant, thrombosed MCA aneurysm complicated by an acute stroke in a pediatric patient

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### ABSTRACT

A 17-year-old boy presented to our quaternary hospital because of acute mental status changes following prolonged gastrointestinal illness resulting in dehydration. Neuroimaging studies with computed tomography and magnetic resonance imaging (MRI) revealed a giant thrombosed aneurysm of the left middle cerebral artery (MCA) with acute left MCA stroke. An ischemic penumbra was identified based upon the mismatch between diffusion weighted (DWI) and susceptibility weighted (SWI) MRI matching with the perfusion weighted imaging (PWI). On follow-up MRI, the core of ischemia as identified by DWI progressed into the ischemic penumbra identified by SWI. The patient had permanent moderate right hemiparesis and aphasia on last follow-up. In conclusion, thrombosis is a rare complication of a giant aneurysm in children. Advanced neuroimaging using the combination of DWI and noncontrast enhanced SWI is a valuable alternative or possibly adjunct to PWI to identify tissue at risk for progressing stroke.

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## Introduction

Pediatric intracranial aneurysms are not as common as in adults. The etiology, demographics, aneurysm location,

morphology, clinical presentation, and outcomes are different for pediatric intracranial aneurysms compared to adults [1–3]. Among all reported intracranial aneurysms less than 5% are seen in children [4]. The incidence of giant aneurysms are reported higher in children than in adults [4]. Middle cerebral

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artery (MCA) aneurysms in children are often fusiform, giant, and incorporate the origins of distal branches [5]. A spontaneous thrombosis of the aneurysm may consequently be complicated by an ischemic stroke.

Diffusion weighted imaging (DWI) allows to identify an ischemic stroke within minutes while contrast enhanced perfusion weighted imaging (PWI) allows to identify the area of hypoperfusion. Matching areas of DWI and PWI abnormalities are known to represent the irreversible core of infarction while the “mismatch” represents the ischemic penumbra, respectively tissue at risk for stroke progression. Susceptibility weighted imaging (SWI) may serve as a noncontrast alternative imaging approach in which the draining veins of hypoperfused tissue appear SWI-hypointense due to the relative increase of deoxygenated blood. SWI is increasingly used in pediatric and neonatal neuroimaging and can give valuable information if used in combination with multiple advanced magnetic resonance imaging (MRI) techniques [6–8].

We (a) present the acute and follow-up imaging findings of a giant thrombosed left MCA aneurysm complicated by an acute stroke and (b) discuss the value of noncontrast SWI combined with DWI to identify the ischemic penumbra. PWI served as reference for the SWI.

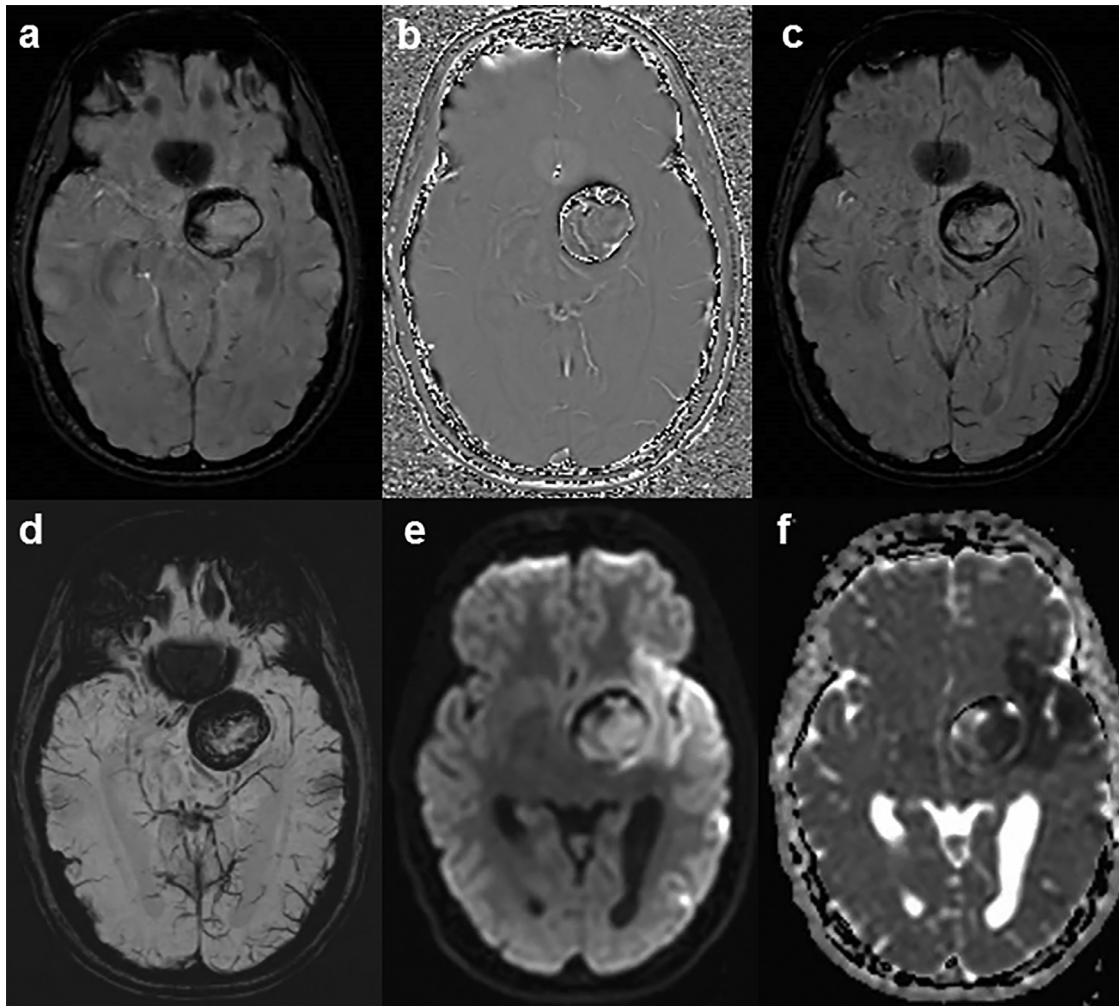
## Case report

A previously healthy 17-year-old right-handed male with no prior medical history was admitted to our quaternary children’s hospital because of an acute mental status change following a camping trip of several days. There was a gastrointestinal illness causing nausea, vomiting, and diarrhea amongst the children on the trip. The patient suffered from episodes of diarrhea and nonbloody, nonbilious emesis with resultant dehydration a day before his presentation. Parents noted he seemed neurologically normal when he went for sleeping at 23:00 after vomiting. At night, he was found in the bathroom with slurred speech, right-sided weakness, and altered gait at 1:00 am. He was initially admitted to an outside hospital where emergency head computed tomography (CT) was performed within 1-2 hours after he was found in the bathroom. CT showed a large well-circumscribed, hyperdense focal lesion along the expected course of the left MCA (Fig. 1). In addition, the MCA distal to the focal lesion appeared hyperdense compatible with a “hyperdense artery sign.” He was subsequently transferred to our quaternary children’s hospital because of progressive right hemiparesis and aphasia. Follow-up CT confirmed the hyperdense lesion along the left MCA measuring  $3.1 \times 3.3 \times 3.5$  cm (AP by TR by CC) compatible with a thrombosed aneurysm of the proximal left M1 segment of the MCA as well as a large acute left MCA stroke. Subsequent MRI was performed on a 1.5 T clinical MRI scanner (Avanto; Siemens, Erlangen, Germany) including 3D isotropic T1 weighted imaging, axial T2WI, Fluid-Attenuated Inversion Recovery (FLAIR) images, diffusion tensor imaging (DTI), SWI, PWI, and magnetic resonance angiography (MRA). Trace of diffusion and apparent diffusion coefficient (ADC) maps were automatically calculated by the vendor-specific softwares. For the SWI sequence minimum intensity projection (mIP)



**Fig. 1** – Initial axial CT performed at outside hospital within 1-2 hours from initial presentation identifies a well circumscribed hyperdense thrombosed giant left middle cerebral artery aneurysm. The arrow points out to the thrombosed distal MCA branch which is referred to as a “dense dot” or “hyperdense artery” sign. Ill-defined white matter hypodensity is noted in the left insular and temporal white matter consistent with ischemic injury.

images were automatically reconstructed. An effective mIP thickness of 16 mm was used. SWI showed the thrombosed aneurysm with a SWI-hypointense signal along the periphery of the aneurysm while the center appeared SWI-hyperintense (Fig. 2a-d), DTI images acquired at the same level showed restricted diffusion (DTI-hyperintense, ADC-hypointense) within the majority of the thrombus as well as restricted diffusion in the adjacent insular and fronto-opercular and temporo-opercular brain parenchyma (Fig. 2e and f). Additional areas of restricted diffusion are noted affecting the left caudate nucleus as well as adjacent white matter and cortical gray matter superior to the aneurysm (Fig. 3a and b). The corresponding SWI slice shows a prominent SWI-hypointensity of the intramedullary veins within the left hemispheric white matter which drain superficially into SWI-hypointense sulcal veins as well as deep into similarly SWI-hypointense subependymal veins including the anterior caudate vein and terminal vein. These veins are significantly more prominent and hypointense compared to the matching contralateral veins. The area with SWI-hypointense veins is matching the area of altered perfusion on the PWI map (Fig. 3c and d). The mismatch between DWI and SWI/PWI indicates a large area at risk for infarct progression (ischemic penumbra) surrounding



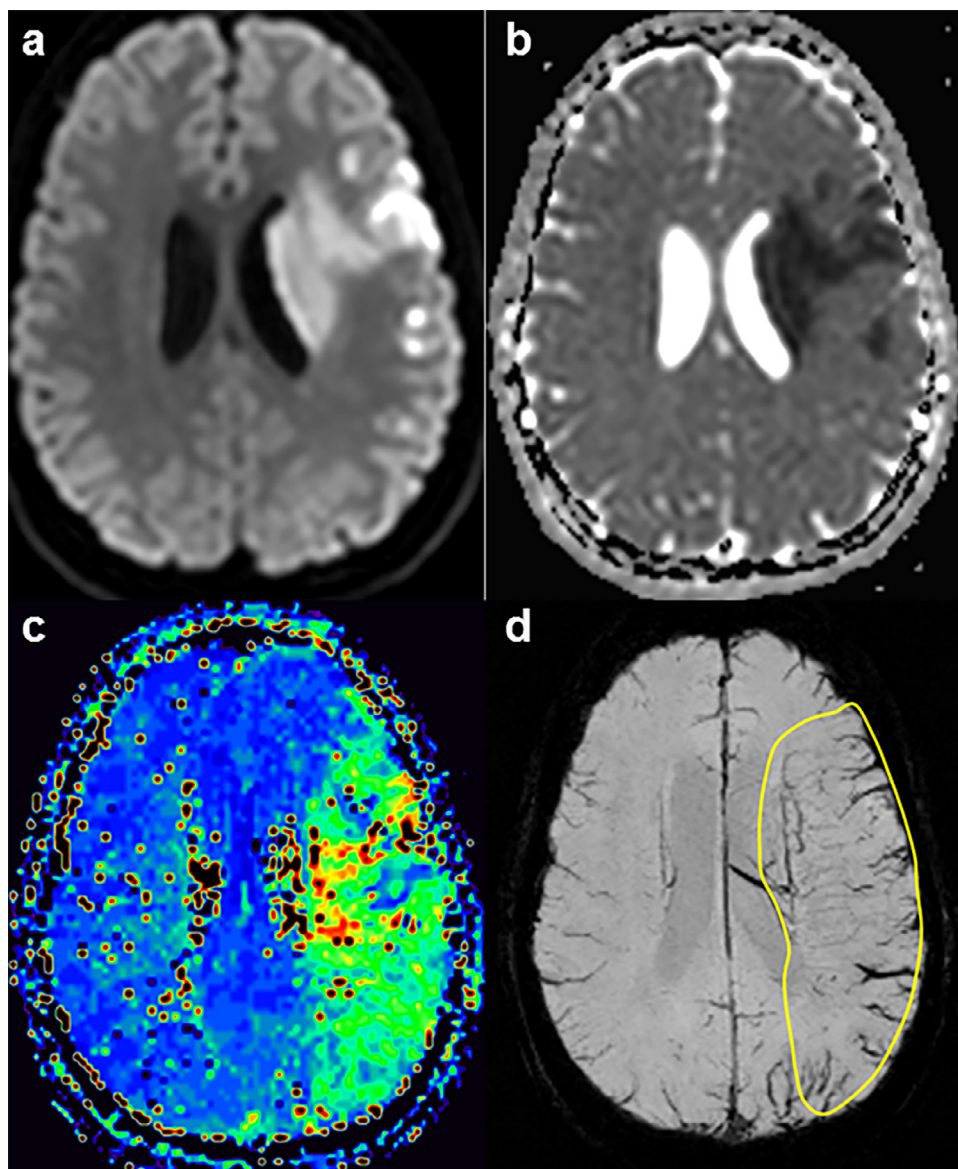
**Fig. 2 – Acute MRI performed at our institution after 8-9 hours from initial presentation: Axial SWI magnitude image (a), SWI phase map (b), processed SWI magnitude image (c) and minimum intensity projection (minIP) SWI image (d) characterize the thrombosed aneurysm. The periphery is SWI-hypointense, likely due to T2\*-dephasing from the superparamagnetic effects of accumulating ferritin/hemosiderin; the center is SWI-hyperintense due to the long T2-relaxation of dissolving hemoglobin. Trace of diffusion (e) and matching ADC map (f) identify restricted diffusion inside the thrombosed aneurysm and adjacent brain insular and temporo-opercular brain parenchyma compatible with acute ischemic stroke.**

the core of infarction. Catheter angiography confirmed a completely thrombosed left MCA aneurysm (Fig. 4a and b). The distal MCA branches appeared to originate from the aneurysm and it was consequently decided that based upon the specific architecture of the aneurysm no recanalization could be performed. Because of the uncertain timing and the size of the aneurysm, it was decided not to pursue endovascular therapy with intravenous tissue plasminogen activator. Therefore, decompressive left craniotomy with aneurysm clipping was performed. Follow-up MRI 10 days later showed progression of the core of infarction into the ischemic penumbra despite decompressive surgery (Fig. 5a and b). Late follow-up imaging showed expected chronic evolution of the infarction within the left MCA territory (Fig. 6a-c). Patient exhibited moderate right hemiparesis and aphasia on the last follow-up visit at the time of last follow-up MRI.

## Discussion

Pediatric patients with intracranial aneurysms most commonly present with subarachnoid hemorrhage [1]. This particular case is unique because the aneurysm did not primarily present with a hemorrhage but with an ischemic stroke. The thrombosis of the aneurysm was likely facilitated due to patient's dehydration and diarrhea.

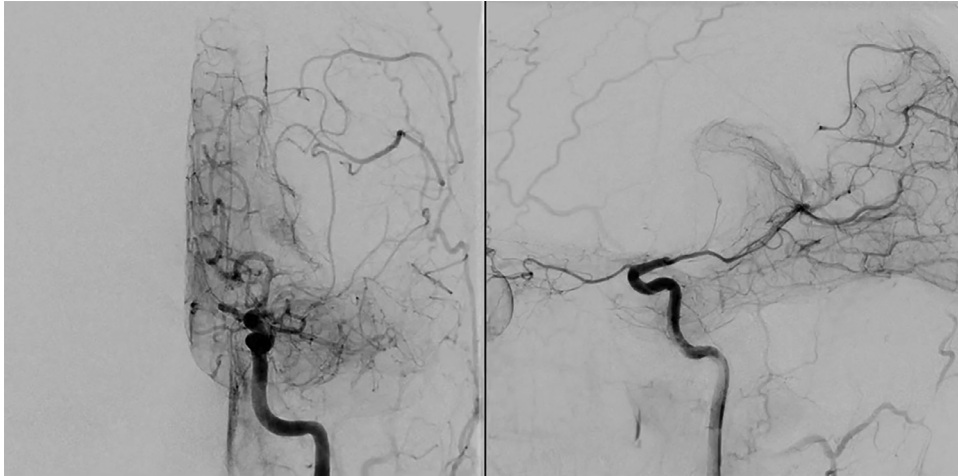
The unique characteristics of the imaging in this particular case are: (1) The distal MCA appeared to originate from the aneurysm sac. (2) The neuroimaging confirmed a significant mismatch between DTI and SWI/PWI on the acute imaging identifying the ischemic penumbra adjacent to the core of ischemia. Ischemic penumbra is characterized by hypoperfused brain tissue with the potential for functional recovery without morphologic damage. This is possible if



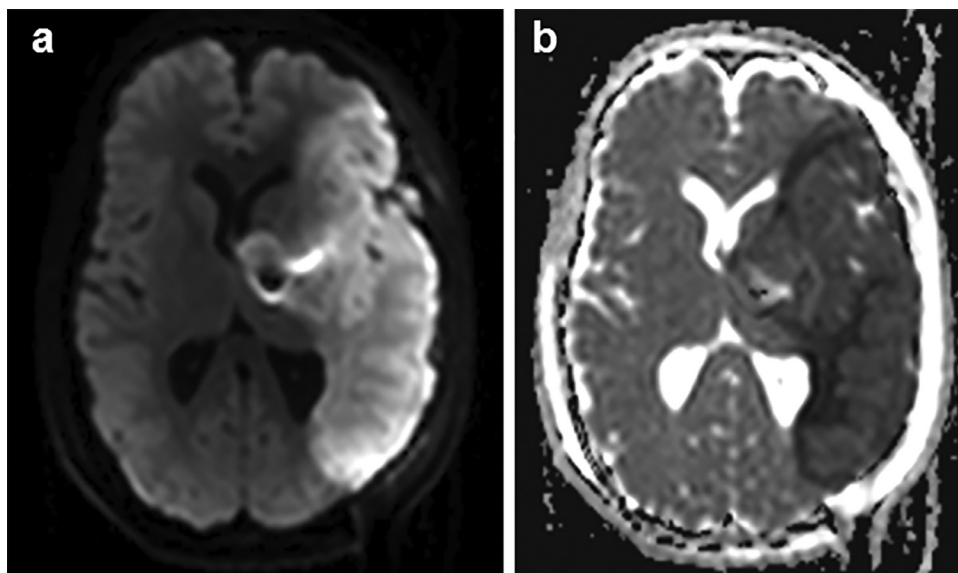
**Fig. 3 – Acute axial trace of diffusion MR image (a) and matching ADC map (b) performed at our institution after 8-9 hours from initial presentation show a geographic area of restricted diffusion compatible with a region of cytotoxic edema secondary to an ischemic stroke. The PWI map (c) as well as the minIP SWI image (d) show an area of impaired perfusion which is substantially larger than the area of restricted diffusion. The area of restricted diffusion (a, b) represents the core of infarction, the area on c and d that exceeds the area of restricted diffusion is known as the ischemic penumbra.**

local blood flow can be restored before infarct progression occurs. Identification of the ischemic penumbra is important because it represents tissue that can potentially be salvaged with thrombolytic therapy or neurovascular interventions. Because of the uncertain timing and the unique angioarchitecture of the aneurysm, it was decided not to move forward with thrombolytic therapy in this case; follow-up MRI showed progressive infarction into the ischemic penumbra. (3) SWI proved to render hemodynamic information similar to PWI, in concordance with the previous literature [6,7]. PWI may be limited because of the low signal to noise ratio and limited spatial resolution. SWI is a valuable high-resolution alterna-

tive to PWI which does not require injection of intravenous contrast agents. Critical hypoperfusion typically results in increased blood oxygen extraction fraction resulting in a more prominent SWI-hypointensity of draining veins. This shift in SWI signal intensity allows to identify hypoperfused tissue similar to PWI as demonstrated in this particular patient. On the other hand, an increased SWI signal intensity of draining veins suggests areas of hyperperfusion after an ischemic event known as luxury perfusion increasing the risk for reperfusion injury or postischemic malignant edema [7,8]. SWI renders however multiple additional, important data. SWI allows to detect hemorrhagic components within infarcted



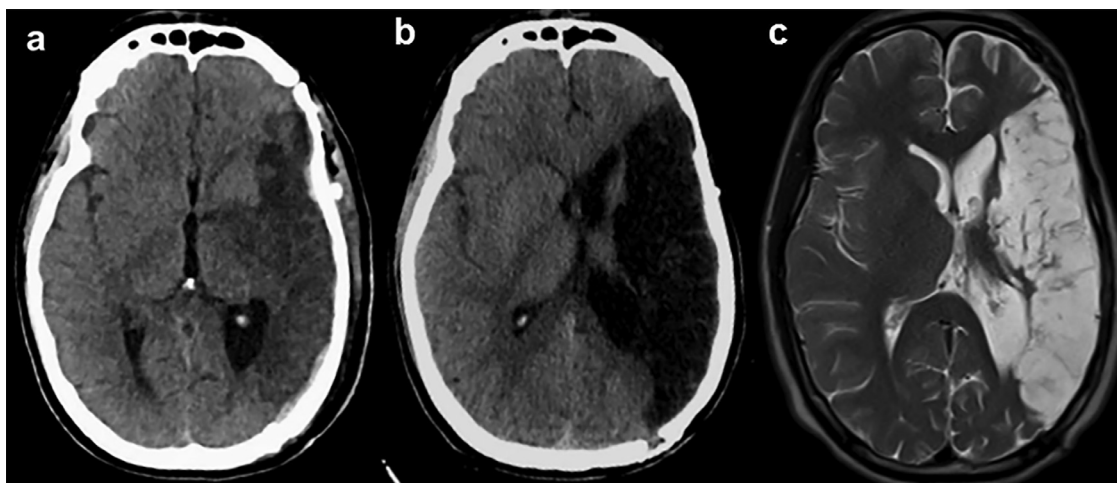
**Fig. 4 – Catheter digital angiography (AP and lateral projection) performed after 10 hours from initial presentation shows the lack of contrast opacification of the left middle cerebral artery secondary to thrombosis of the aneurysm and distal MCA branches.**



**Fig. 5 – Follow-up DWI (a) and ADC (b) MRI 10 days after decompressive surgery show progression of ischemia into the previously identified ischemic penumbra.**

tissue with higher sensitivity than other MRI sequences, can detect acute occlusive arterial thromboemboli, quantify microhemorrhages, and predict hemorrhagic transformation before thrombolytic therapy is initiated; and can detect early hemorrhagic complications after intra-arterial thrombolysis [7,8].

As a conclusion, in this article, we have reported a pediatric case of giant MCA aneurysm, highlighting its rare and unique presentation along with its advanced imaging features, in particular emphasizing the value of SWI as a valuable alternative or possibly adjunct to PWI to identify the ischemic penumbra.



**Fig. 6 – Follow-up CT after 1 month (a) and 6 months (b), and axial T2-Weighted MRI after 9 months (c) confirm chronic evolution of the infarcted tissue.**

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