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Pediatric Stroke Imaging

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Abstract

Background: Pediatric stroke is a distinct clinical entity as compared to that in adults due to its unique and diverse set of etiologies. Furthermore, the role and application of diagnostic imaging has specific constraints and considerations. The intention of this article is to review these concepts in a thorough manner to offer a pediatric stroke imaging framework that providers can employ when taking care of these patients.

Methods: A comprehensive primary and secondary literature review was performed with specific attention to the common causes of pediatric stroke, appropriate use of neuroimaging, specific imaging findings, and developing techniques which may improve our ability to accurately diagnose these patients.

Results: Findings from this literature review were synthesized and summarized in order to thoroughly review the aforementioned concepts and outline the current consensus-based approach to diagnostic imaging in pediatric stroke. Furthermore, imaging findings drawn from cases which presented to our institution are demonstrated to familiarize readers with pediatric stroke neuroimaging.

Conclusion: The challenges posed by pediatric stroke can be mitigated, in part by the thoughtful application of diagnostic imaging, with the ultimate hope of improving outcomes for these vulnerable patients.

Keywords

pediatric stroke; ischemic stroke; hemorrhagic stroke; MRI; CT

Introduction

Pediatric stroke is a distinct clinical entity with unique etiologies, diagnostic considerations, and other challenges compared to stroke in adults. United States' population-based studies estimate a stroke incidence of 4.6–6.4 per 100,000 children[1,2]. Furthermore, cerebrovascular

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disease ranks within the top 10 causes of childhood mortality for all age ranges[3,4]. Pediatric stroke is stratified based on age, with childhood stroke defined as occurring between 29 days of life and 18 years, and perinatal stroke arising prior to 29 days. The latter will not be addressed in this review.

Pediatric stroke is classified as either ischemic or hemorrhagic, with each category accounting for approximately half of pediatric strokes[5]. However, the pathophysiology underlying these conditions differ greatly from adults. Pediatric stroke is characterized by a wider variety of etiologies than in adults, and this may impede timely identification of at risk patients if they are not under the care of physicians familiar with this condition. This is further complicated by the non-specific signs and symptoms patients may present with, and the numerous prevalent stroke mimics in children, including epilepsy, intracranial infections, and focal lesions[6].

Effective diagnostic neuroimaging in children is uniquely challenging. Obtaining acute diagnostic neuroimaging in pediatric patients is inhibited by factors including high clinical thresholds for exposing children to ionizing radiation and the frequent necessity of sedation in this population. Furthermore, pediatric stroke cases are infrequently encountered by most practicing radiologists, resulting in reduced familiarity with the imaging manifestations of stroke in the developing brain.

To facilitate appropriate neuroimaging in these patients, there has been an effort to standardize the application of diagnostic imaging in suspected pediatric stroke. Specifically, the International Paediatric Stroke Study Neuroimaging Consortium in 2017 published a set of guidelines which are outlined in **Figure 1**. The application of these recommendations in specific subtypes of pediatric stroke will be explored in the following sections.

Arterial Ischemic Stroke

Arterial ischemic stroke (AIS) is defined as a presentation including a focal neurological deficit which corresponds to an identified region of ischemic brain. The two most common risk factors for AIS include cerebral arteriopathy and congenital cardiac disorders (i.e. thromboembolic disease)[5]. Cerebral arteriopathy itself is associated with approximately half of all AIS cases. Another important patient population to consider for AIS are patient's with sickle cell disease as > 10% of these patients have been found to suffer clinically apparent strokes before age 20, with a much higher percentage suffering silent cerebral infarcts, up to 37% in a study by Bernaudin *et al.* (2011)[7,8]. Other risk factors to consider include hematologic and solid tumor malignancies, prothrombotic conditions, acute systemic illnesses (e.g. sepsis), trauma, and many others[9].

Computed tomography (CT) head scans are an efficient modality for diagnosis, but have significant drawbacks including exposure of the developing brain to ionizing radiation, decreased sensitivity for acute infarcts, and an inability to differentiate infarcts from mimics (e.g. meningitis, posterior reversible encephalopathy syndrome). Magnetic resonance imaging (MRI) is the preferred modality for diagnosis, but in hospitals without readily available MRI or in unstable patients, CT may be necessary. Non-enhanced CT findings of

acute and subacute ischemic stroke are similar to those in adults, including loss of grey-white matter differentiation, hyperdense artery sign, and cerebral edema with associated mass effect. Further, non-enhanced CT will reliably rule out intracranial hemorrhage, allowing clinicians to safely administer tissue plasminogen activator (tPA) when appropriate[10]. If possible, simultaneous head and neck CT angiography (CTA) is a useful adjunct due to the large proportion of patients with underlying arteriopathy which may require intervention. CT perfusion imaging may also be employed in these patients, but widespread use of this technique has been limited by its lack of validation in this population, and the radiation dose these studies require.

To overcome some of the barriers precluding MRI's use as the first line diagnostic imaging modality, some institutions have developed shortened brain MRI protocols which include diffusion-weighted imaging (DWI), apparent diffusion coefficient (ADC) maps, hemorrhage specific sequences such as susceptibility-weighted imaging (SWI) or gradient echo (GRE), and time-of-flight (TOF) MR-angiogram (MRA)[5]. The interpretation of the images from these sequences mirrors that in adults with DWI demonstrating increased signal in an area of infarction, and corresponding signal loss in the same region on ADC maps. The latter sequence helps identify physiologic T2 prolongation as the cause of increased signal on DWI (i.e. T2 shine through), as opposed to true infarction related restricted diffusion. The SWI and GRE sequences are highly sensitive for blood products, which like CT allows patients to rule out hemorrhage and administer tPA. Furthermore, they can demonstrate blooming (i.e. hypointensity) at the site of a thrombosis, which is referred to as the "susceptibility vessel sign," (Figure 2), and can be corroborated with the TOF MRA[11]. Table 1 describes the commonly used MRI sequences performed in pediatric stroke and their intended clinical uses. Regardless of suspected stroke subtype, the decision to use MRI versus CT is influenced by institution-specific factors (e.g. ready availability of an MRI scanner and shortened protocols) and patient-specific factors (e.g. patient instability in setting of acute head trauma).

Vascular imaging is crucial in these patients due to the high likelihood of underlying arteriopathy, which may influence management. Furthermore, arteriopathy has been found to be a poor prognostic marker and is associated with recurrent cerebrovascular accidents. In one prospective study of 355 pediatric stroke patients, those diagnosed with arteriopathy compared to those without had a hazard ratio of 5.0 for stroke recurrence[15,16]. Again, MRA's of the head and neck are favored over CTA's and can facilitate the identification of conditions such as transient cerebral arteriopathy (TCA), arterial dissection, moyamoya disease, and fibromuscular dysplasia (FMD). MRA's with contrast enhancement are preferred over time-of-flight due to the latter's inability to reliably distinguish between some of these conditions. When initial MRA/CTA is negative or equivocal in patients where there is high suspicion for arteriopathy, conventional angiography becomes necessary[17]. In addition to vascular imaging on presentation, follow-up imaging is also required in the range of 6–12 weeks to monitor arteriopathy evolution, identify previously occult arteriopathy and/or new infarcts[5].

TCA is defined as a unilateral inflammatory arterial process which typically manifests initially as an occlusion or irregular stenosis in the distal internal carotid artery (ICA),

proximal middle cerebral artery, and/or proximal anterior cerebral artery. The progression of these filling defects on follow-up imaging demonstrate a common pattern: variable degrees of stenosis/occlusion acutely (including no visible filling defects), maximal stenosis/occlusion within 3 months, followed by varying degrees of recanalization by 6 months (Figure 3)[17,18].

The underlying pathophysiology of TCA is thought to be mediated in part by a post-infectious inflammatory process. Varicella infections in particular have been described as having a strong association with TCA, with a study by Danchaivijitr *et al.* (2006) finding that > 50% of the children with the aforementioned vascular imaging characteristics had a varicella infection within 1 year of presentation[19,20]. This significant association may be related to the varicella virus' predilection for infecting neuronal ganglia, and the possible presence of afferent nerve fibers between the trigeminal ganglia and middle cerebral artery. The latter would provide a route for infection spread to the intracranial vasculature[21]. Other associated infections which have been observed in a smaller number of patients include unspecified upper respiratory infections, Epstein-Barr virus, herpes simplex virus 1, and enterovirus[22–25]. Not all patients with TCA imaging findings can readily be identified to have had a prior infection, which implies that infections may not have been recognized by patients, caregivers, or providers, and/or that the pathophysiological processes underlying TCA are incompletely understood.

In the acute setting, TCA can be difficult to differentiate from intracranial carotid artery dissection (ICAD). ICAD presents on imaging with filling defects characterized by arterial wall irregularities, arterial stenosis or occlusion, and sometimes intimal flaps. MRI can also identify arterial wall hematomas. The imaging abnormalities of TCA can mimic these findings in the same distribution of vessels. Patient history can be especially useful in this context, with recent trauma suggestive of ICAD rather than TCA. Research also suggests TCA typically presents with infarcted territories restricted to the basal ganglia, whereas ICAD may additionally have cortical involvement. However, diagnostic uncertainty in equivocal cases should prompt more robust imaging modalities, including conventional angiogram and MRI vessel wall imaging/black blood imaging (to be discussed separately)[17]. Follow-up vascular imaging in the subacute and chronic stages after a stroke can also elucidate the underlying pathology as TCA filling defects evolve over time, while ICAD findings will not[17,18]. A similar rationale can be used to differentiate TCA from large vessel occlusion secondary to thromboembolic disease, as TCA can also present as complete occlusion on imaging depending on the scan's time-point in the diseases' progression.

Extracranial carotid and vertebral artery dissections have demonstrated increasing prevalence over the previous 10–15 years. This has been specifically reported in adult populations in the setting of trauma, as opposed to spontaneous dissections. The largest study by Harrigan *et al.* (2014), using the Nationwide Inpatient Sample, found among presenting blunt trauma cases, the incidence of diagnosed extracranial arterial dissection increased from 0.46% to 0.95% between the study's period of 2003–2010. However, the authors suggest more aggressive screening protocols following trauma may be driving this finding[26]. In children there has been limited research into extracranial carotid and vertebral artery dissections regarding the prevalence for traumatic/spontaneous dissections, and their

imaging characteristics. Spontaneous vertebrobasilar dissections, both intra/extracranial, were characterized in a case series of 29 pediatric patients by Songsaeng *et al.* (2010). The authors noted a male predominance in their sample, and a high propensity for associated subarachnoid hemorrhage. The latter occurred in all 14 patients with dissections which at least in part involved the basilar artery, possibly indicating an association between basilar artery dissections and subarachnoid hemorrhages. Furthermore, at least in spontaneous dissections, literature supports an increased risk of subarachnoid hemorrhage in children compared to adults, likely relating to the fact that adults have extradural vertebral artery dissections more commonly[27,28]. They also observed a subset of patients with spontaneous extradural vertebral artery dissections that all presented with ischemic stroke, which was thought to be related to false lumen thromboembolism or false lumen occlusion of communicating arterial branches/perforators[29]. Figure 4 demonstrates an analogous case in a 7 year old girl presenting with severe headaches and nausea/vomiting. There is very limited research to suggest MRA's may have decreased sensitivity for dissections, especially in the extracranial vessels, but this will need to be further evaluated before specific recommendations can be made[30].

Another form of arteriopathy, moyamoya disease, is defined as a progressive occlusive cerebrovascular disorder with bilateral narrowing of distal ICAs or their proximal branches, with subsequent growth of collaterals. This condition can develop in isolation (primary) or in association with known risk factors (secondary), including sickle cell disease, radiation therapy, and Down syndrome[31]. Moyamoya means "puff of smoke" in Japanese and describes the appearance of the extensive tangle of collaterals which develop distal to occluded vessels. Furthermore, Moyamoya often demonstrates the "ivy sign" where prominent leptomeningeal collaterals result in T2 FLAIR sulcal/subarachnoid hyperintensities which resemble growing ivy (Figure 5). "Possible moyamoya syndrome" has been proposed as a classifier for cases where patients have bilateral ICA stenosis/occlusion without collateral development or unilateral ICA stenosis/occlusion with collaterals, and is thought to represent early moyamoya syndrome[18,32].

FMD is a much less common etiology for AIS with a study by Kirton *et al.* (2013), which assessed FMD-related pediatric stroke, finding that the disease accounts for < 1% of reported strokes[33]. FMD almost exclusively involves extracranial cervical vasculature, including the proximal ICA and vertebral arteries[34]. The range of vascular imaging findings are variable with findings including unilateral and bilateral distributions, focal or segmental stenosis/occlusion, and the classic string-of-beads sign (Figure 6).

Patient's suffering from sickle cell disease are at significant risk of developing ischemic stroke during their lifetime, with a large proportion occurring during childhood[8]. Arteriopathy in sickle cell disease patients manifests as unilateral or bilateral stenosis/occlusion of the ICA and its branches. In a case series of sickle cell disease pediatric patients who suffered a stroke, vascular imaging demonstrated variable appearances of such filling defects, including stenosis/occlusions with smooth tapering, beading, and other irregular vessel contours[35]. Vasculature abnormalities detected by transcranial doppler are not sufficient, as this technique may identify factors that precede development of structural disease, such as elevated resistive indices[32].

Practical Tips –Arterial Ischemic Stroke
1. Pseudo-occlusion: a. This entity occurs when intracranial ICA occlusions manifest as extracranial ICA filling defects on CTA/MRA due to a sluggish column of blood proximal to the intracranial occlusion. The latter results in delayed contrast opacification of the proximal extracranial ICA, and thus absent opacification during the arterial phase. b. Only reliably identified with conventional angiograms, and to a lesser degree multiphase CTA[36,37]. 2. Distinguishing between congenitally hypoplastic vessels versus true flow-limiting arterial lesions a. Hypoplastic vessels have smooth contours as opposed to irregular on vascular imaging. b. Associated structures are smaller with hypoplastic vessels (e.g. supplied brain volume, vertebral foramina for vertebral arteries).

Hemorrhagic Stroke

Pediatric hemorrhagic stroke is defined as non-traumatic intraparenchymal and/or subarachnoid hemorrhage. Contrasting with adults, in whom major risk factors for hemorrhagic stroke primarily include hypertension and amyloid angiopathy, most hemorrhagic strokes in children are related to arteriovenous malformations (AVM), cavernous malformations (CM) or aneurysms. Less common risk factors include brain tumors and hematologic conditions, such as acute leukemia, thrombocytopenia and other coagulopathies[38].

Studies have demonstrated similar sensitivities of CT and MRI for acute intracranial hemorrhage, and superior sensitivity of MRI for chronic intracranial blood products[39,40]. At the time of presentation it is often not possible to differentiate patients likely to have ischemic versus hemorrhagic stroke. Therefore, in the context of MRI's higher sensitivity for hyperacute/acute ischemic infarcts, MRI remains the preferred first-line modality in patients with hemorrhagic stroke. However, the aforementioned barriers to its use, often compel providers to employ CT at least in the acute setting. A limitation of non-enhanced CT in hemorrhagic strokes is decreased sensitivity for underlying vascular malformations or aneurysms. Rare non-enhanced CT findings include focal calcifications which may be seen in both AVM's and CM's, and round hyperdense lesions which are often the manifestations of CM's[41].

The classic appearance for AVM's on T1-weighted imaging (T1WI) and T2-weighted imaging (T2WI) is a conglomeration of flow voids forming a "honeycomb" pattern with possible adjacent gliosis manifesting as increased T2 signal. In contrast, CM's appearance on T1W1 and T2WI is that of a "popcorn ball" with areas of low and high signal for blood products at varying stages of evolution, and associated GRE/SWI blooming. (Figure 7). Intracranial aneurysms in pediatric patients include saccular, fusiform, and giant aneurysms (i.e. greater than 2.5 cm in diameter). Flow voids on T1W1/T2WI will outline a focally dilated intracranial artery with a morphology corresponding to the aneurysm type (Figure 8). The presence of thrombus or turbulent flow in the aneurysm can result in significant variations from this appearance[42]. Furthermore, in the setting of a hemorrhagic stroke these findings can be obscured by the hematoma.

Vascular imaging is again of great importance in these patients either at the time of the initial study or subsequently. CM's are not typically identified on vascular imaging unless they are associated with another vascular malformation (e.g. developmental venous anomaly), but AVM's will demonstrate an abnormal collection of closely packed arteries and veins (Figure 9). MRA and CTA are first-line modalities, but have decreased sensitivity for smaller AVM's and aneurysms. Therefore, conventional angiography is indicated in children with hemorrhagic stroke, negative CTA/MRA, and absence of other risk factors that would account for intracranial hemorrhage. Again, in the setting of acute intracranial hemorrhage, vascular abnormalities may be obscured by blood products, which will necessitate follow-up vascular imaging if initial studies are negative. If an AVM is identified, even if resected, follow-up vascular imaging is necessary due to the risk of recurrence. A small retrospective cohort study of pediatric patients status-post AVM resection, found 14.3% of patients suffered a recurrence within 15 months of resection[43]. No consensus guidelines exist for the timing of follow-up vascular imaging in these patients. However, some institutions have developed timelines that call for repeat vascular imaging at one and three months following initial hemorrhagic stroke. In the absence of newly identified vascular abnormalities, or recurrence/progression of known abnormalities, some institutions will also repeat imaging at five or eighteen years of age[5].

Practical Tips –Hemorrhagic Stroke	
1.	"Spot sign" occurs with active bleeding, and is seen on contrast-enhanced imaging with contrast visualized within area of hemorrhage
	a. Predictive of hemorrhage progression[44]
2.	False negative non-contrast CT can occur with reduced hemorrhage density secondary to anemia
3.	Posterior fossa CT beam hardening artifact may obscure smaller hemorrhages in this region

Cerebral Sinovenous Thrombosis

Pediatric ischemic/hemorrhagic strokes related to sinovenous thrombo-occlusive disease have risk factors that overlap significantly with AIS, and broadly include infection, trauma, and hematologic disorders such as anemia, polycythemia, and hypercoagulable states. Thrombosis may involve the dural sinuses, cortical/deep cerebral veins, and can often span both systems. Ischemia related to sinus or venous thrombosis results from backup of arterial blood flow secondary to venous congestion and/or arterial compression[5].

Findings on CT/MRI that are observed in the setting of venous related ischemia include non-arterial distribution of infarction and simultaneous cytotoxic and vasogenic edema. It is important to note that hemorrhage is a common finding in intracranial venous thrombosis, and is related to diapedesis of blood from venous congestion (Figure 10). Frontal lobes, temporal lobes or bilateral thalami can be involved depending on the venous structure being thrombosed. Non-enhanced CT may demonstrate hyperattenuating thrombus within a cortical vein as the "cord sign". Sinovenous thrombus' appearance on MRI varies based on its age[5]. On T1WI the thrombus will be isointense (relative to brain) acutely, hyperintense subacutely, and isointense again chronically. On T2WI the thrombus will be hypointense

acutely, and hyperintense in the subacute and chronic phases. Importantly, acute hypointense thrombi on T2WI can mimic typical sinovenous flow voids.

With vascular imaging the thrombosis will manifest as filling defects of the cerebral sinuses or cerebral veins. Studies in adults have demonstrated magnetic resonance venograms and CT venograms have similar sensitivities for venous thrombosis[45]. Sinovenous thrombosis often presents as the classic “empty delta sign” where the thrombus manifests as a hypoattenuating triangle within the peripherally opacified straight or transverse sinus (Figure 11).

Practical Tips –Cerebral Sinovenous Thrombosis
1. False positive “cord sign” can occur with:
a. Diffusely increased attenuation of sinovenous blood from conditions such as dehydration or polycythemia
b. Diffuse cerebral edema resulting in relative hyperattenuation of normal sinovenous blood[46]
2. Sino venous thrombosis mimics on vascular imaging include:
a. Arachnoid granulations extending into lumen of dural Sinuses
b. Sinovenous hypoplasia or atresia (which are common anatomic variants)

Acute Management

The acute therapeutic management of pediatric ischemic stroke still varies considerably, with limited data to support specific therapies. The Thrombolysis in Pediatric Stroke (TIPS) trial attempted to determine the safety and efficacy of intravenous tPA in pediatric stroke, but was ultimately unsuccessful due to the small number of patients meeting enrollment criteria. The underlying issues cited by the study authors included diagnoses outside treatment window, and approximately 50% of screened patients having contraindications to thrombolytics[47].

Likewise, only a small number of investigations have examined mechanical thrombectomy in these patients. Recent large scale clinical trials, which demonstrated the benefit of mechanical thrombectomy in selected acute ischemic stroke patients, excluded patients < 18 years old, and only a small number of case reports and a case series have been published to evaluate this treatment modality’s efficacy in children[48–50]. These limited studies do indeed show clinical benefits to timely thrombectomy, but are far from providing definitive evidence of this therapy’s safety and effectiveness.

Currently the joint American Heart Association and American Stroke Association 2015 acute stroke therapy guidelines do not include any recommendations regarding management in pediatric patients related to the absence of robust clinical trial data. As cited by the guideline’s authors, and supported by the findings in the TIPS trial, only a small number of pediatric patients would meet inclusion criteria for such trials. Specifically, delayed diagnosis likely represents the largest barrier to increasing the number of patients eligible for stroke therapies[51]. A retrospective study of 209 children at a single tertiary center with AIS diagnosed via neuroimaging, found that the median time from symptom onset to diagnosis

was 22.7 hours (interquartile range 7.1–57.7 hours) which was largely related to in-hospital delays[52]. Therefore, significant improvements in time to diagnosis will likely need to be established before large clinical trials will become feasible. In the interim institutions and physicians must rely on the limited available data, clinical judgement, and patient/family wishes when deciding to pursue therapeutic interventions.

Emerging Imaging Techniques

Perfusion imaging has developed into an important tool in adult stroke for identifying patients with brain tissue that can be salvaged with intervention. However, perfusion imaging's use in pediatric patients is presently limited by many obstacles. Perfusion CT is not typically employed due to the radiation required for these studies. MRI perfusion techniques primarily include dynamic susceptibility contrast, a T2*-weighted sequence requiring contrast, and arterial spin labeling (ASL), a T1WI sequence which does not require contrast. While these sequences can yield cerebral perfusion data in children, its interpretation can be challenging. Cerebral perfusion and its complex set of underlying factors are known to vary throughout childhood which complicates the application of adult stroke MR perfusion parameters in pediatric patients[53]. Moreover, the effects of anesthesia and sedation, which is required in many of these patients, on cerebral perfusion may affect the perfusion measurements by CT and MRI.

ASL is the preferred MR perfusion technique in pediatric patients due to its use of blood as an endogenous tracer rather than contrast agents. The latter would require powered injection that is often incompatible with the smaller caliber of pediatric veins. In addition to the above confounding factors, many others influence ASL cerebral perfusion parameters in children, including the variable molecular composition of brain parenchyma which alters T1 relaxation, the increased T1 relaxation time of blood, and an increased blood-brain partition coefficient. Fortunately, these differences are thought to counteract each other resulting in cerebral blood flow values that in one series were found to only deviate by 5–10% from expected values when using an adult perfusion protocol[54]. Pseudo-continuous ASL is the more widely used ASL technique sub-type which was developed to mitigate some of the disadvantages of other ASL sequences, and is characterized by improved signal to noise ratio, and the ability to be performed with MRI systems that are not capable of continuous radiofrequency pulses[55]. Regardless, the application of ASL sequences in pediatric stroke is relatively new, and requires special expertise to account for some of the previously noted complexities[56].

Novel vascular imaging approaches are also starting to be applied in patients with pediatric stroke. Vessel wall imaging is a technique that acquires high resolution images of intracranial vessels and suppresses signal from flowing blood to better assess their walls. This sequence excels in differentiating the many causes of arteriopathy, something of critical importance in pediatric patients with AIS[57,58]. Black blood MRI is another method of obtaining more detailed images of the intracranial vasculature. For this technique T1W1 images with blood suppression pre- and post-contrast are obtained. Contrast enhancement of vessel walls on these images can help to identify and monitor active inflammatory vascular

changes which is again crucial in accurately diagnosing underlying arteriopathy in the setting of AIS (Figure 12)[5,59].

Conclusion

The step-wise imaging algorithm for pediatric stroke seen in Figure 1 should be the diagnostic imaging paradigm with which radiologists and clinicians alike approach these patients. MRI is the initial modality of choice, including shortened stroke protocols (e.g. DWI, ADC, and SWI/GRE), followed by vascular imaging to detect abnormalities which may underlie an identified stroke. These patients require follow-up vascular imaging at institution-specific intervals to identify previously occult vascular abnormalities, recurrence/progression of known lesions, and/or new infarcts. Evolving MR perfusion techniques are making progress in identifying salvageable brain parenchyma, and vascular imaging methods (e.g. vessel wall imaging and black blood MRI) are helping to recognize vascular abnormalities that so often underlie these cases. With early clinician suspicion for pediatric stroke and a robust diagnostic imaging toolset it should be possible to mitigate the acute and long-term morbidity and mortality seen in these patients.

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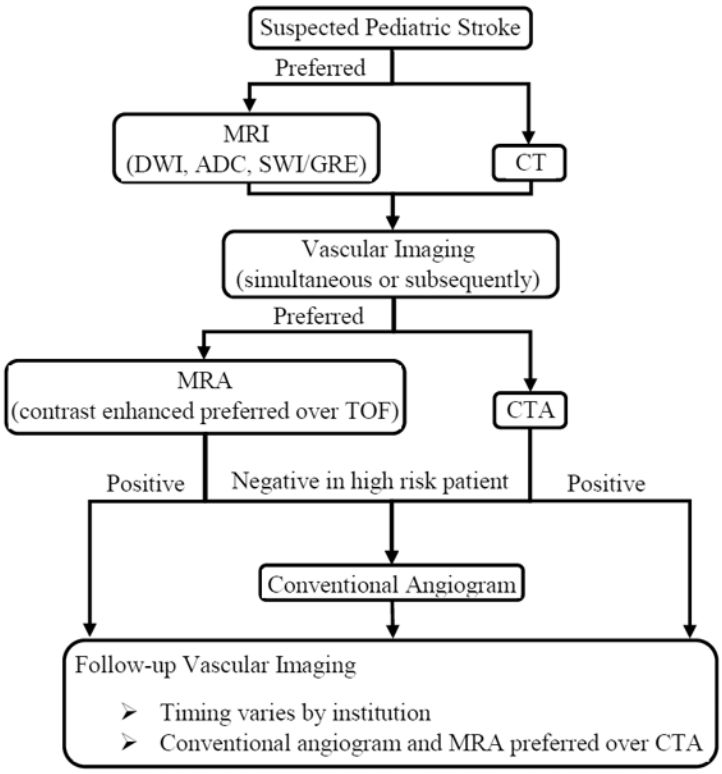

Figure 1:

Proposed imaging algorithm in pediatric patients with suspected stroke. Adapted from the Paediatric Stroke Study Neuroimaging Consortium[5]. Other MRI sequences including T1-weighted imaging, T2-weighted imaging, and arterial spin labeling perfusion techniques may also be applied to the above algorithm where appropriate. ADC = apparent diffusion coefficient; CT = computed tomography; CTA = computed tomography angiogram; DWI = diffusion weighted imaging; GRE = gradient echo; MRA = magnetic resonance angiogram; MRI = magnetic resonance imaging; SWI = susceptibility weighted imaging; TOF = time of flight.

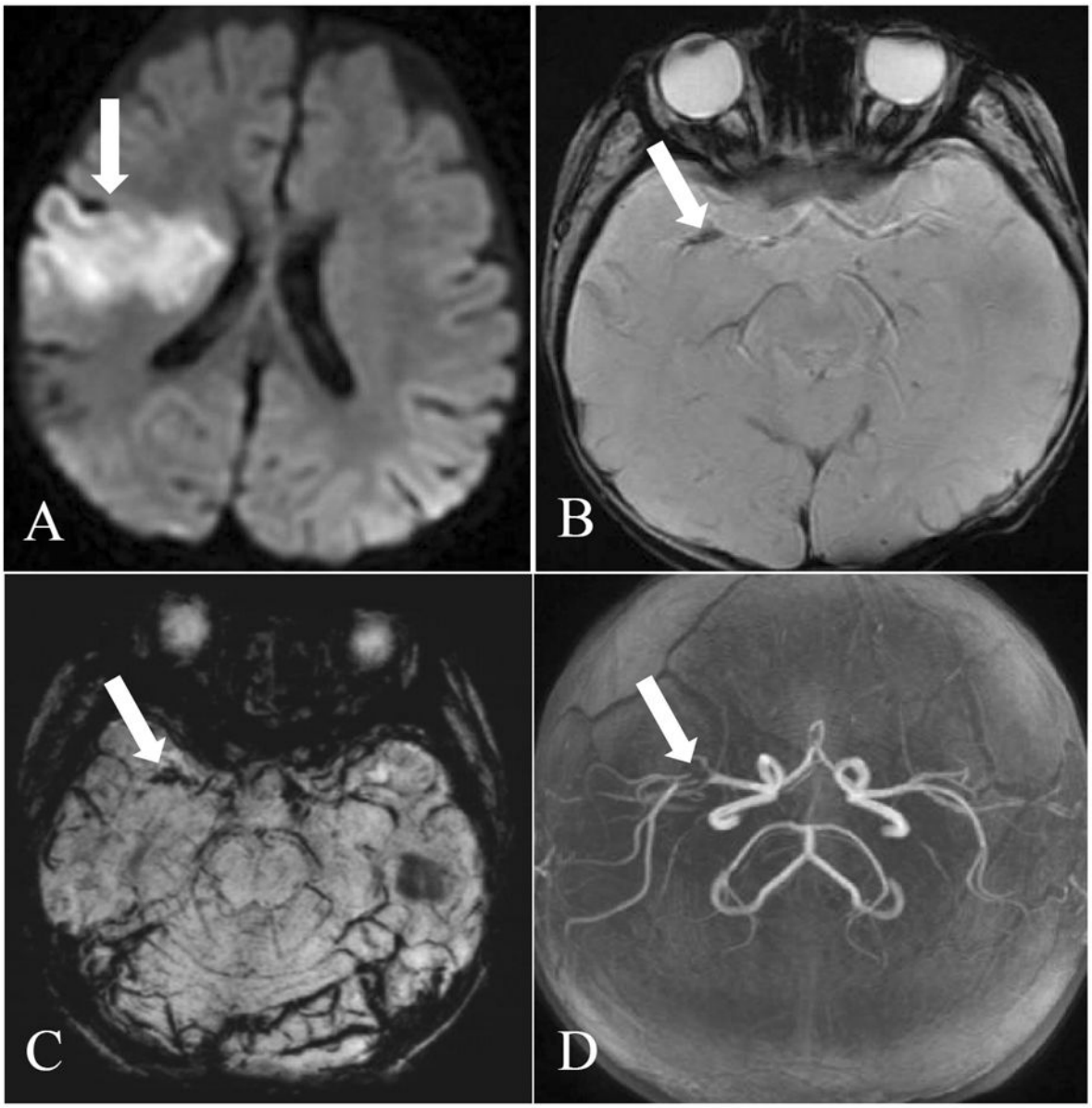

Figure 2:

MRI in 1 year old male with single ventricle congenital heart disease status-post Glenn Procedure presenting with cardioembolic large vessel occlusion. (A) Axial DWI sequence with hyperintensity centered in the right frontoparietal region, which along with corresponding ADC map hypointensity (not shown) is indicative of infarction. (B and C) Axial GRE and SWI sequences, respectively, demonstrating blooming (i.e. hypointensity) within region of the right MCA, which is representative of intraarterial thrombus (i.e. susceptibility vessel sign) (D) Axial maximum intensity projection MRA corroborating SWI/GRE findings with an associated filling defect in the right MCA. ADC = Apparent Diffusion Coefficient; DWI = diffusion weighted imaging; GRE = gradient recall echo; MCA = middle cerebral artery; MRA = magnetic resonance angiogram; MRI = magnetic resonance imaging; SWI = susceptibility weighted imaging.

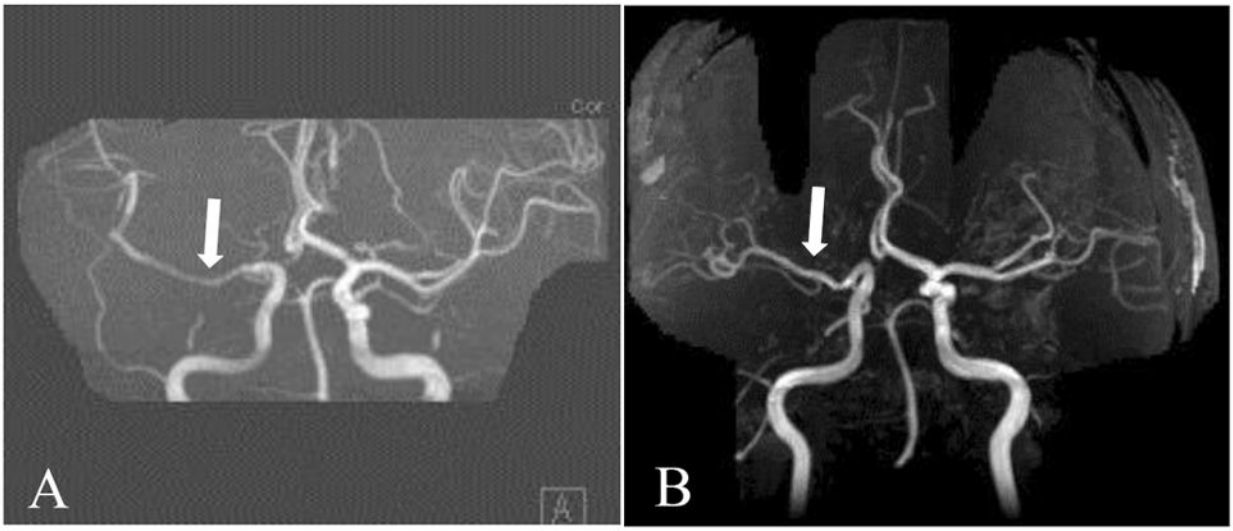

Figure 3:

MRI in pediatric patient found to have stroke within right MCA distribution. (A) Coronal maximum intensity projection MRA on day of presentation demonstrating irregularity and focal stenoses along the right M1 segment (B) Follow-up coronal maximum intensity projection MRA 1.5 years after initial presentation with resolution of MCA focal stenosis and irregularities. Patient was reported to have unspecified viral infection prior to initial presentation. These findings are suggestive of transient cerebral arteriopathy. MCA = middle cerebral artery; MRA = magnetic resonance angiogram.

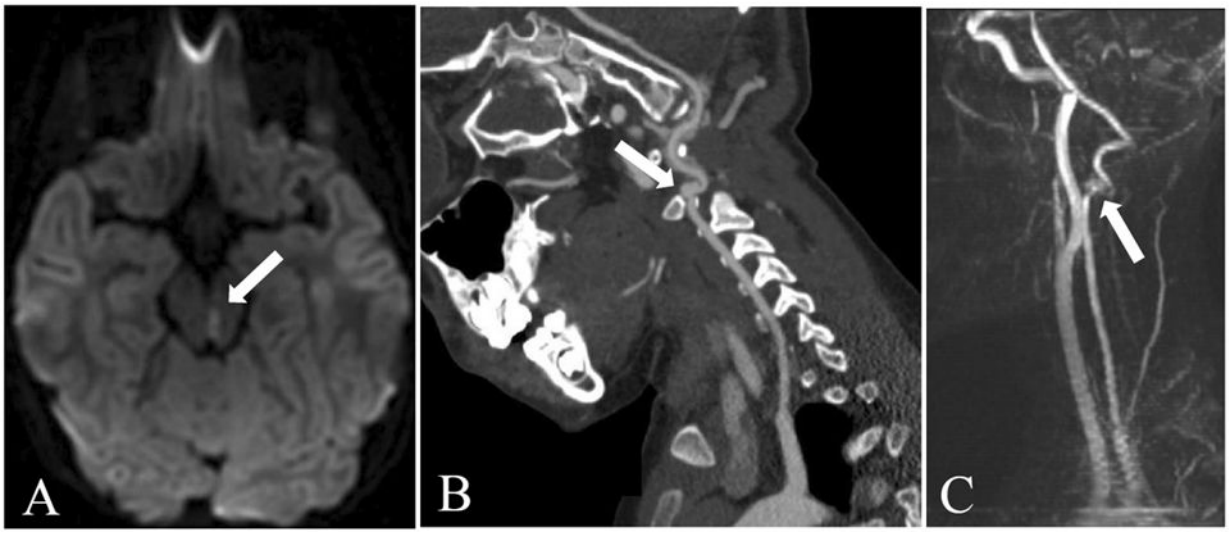

Figure 4:
MRI and CTA in 7 year old female with suspected stroke. (A) Axial DWI sequence demonstrating restricted diffusion (i.e. hyperintensity) within the left paramedian midbrain consistent with infarction (B) Subsequent sagittal CTA and 3D reconstruction showing focal stenosis with aneurysmal outpouching of the left segment 3 vertebral artery. Findings were consistent with spontaneous vertebral artery dissection with associated pseudoaneurysm. DWI = diffusion weighted imaging; CTA = computed tomography angiogram; MRI = magnetic resonance imaging.

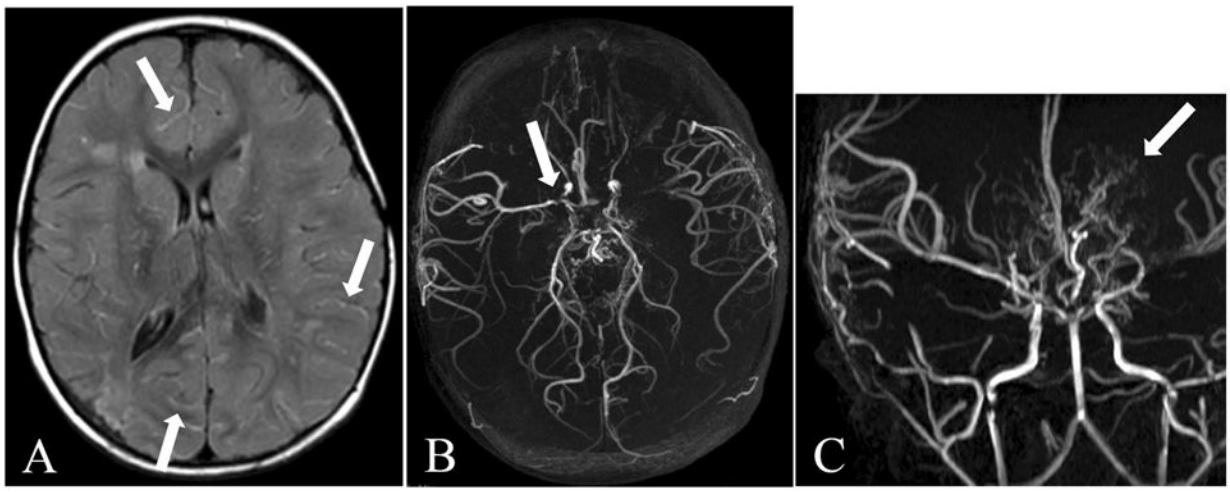

Figure 5:

Brain MRI in pediatric patient with moyamoya disease. (A) Axial T2 FLAIR sequence depicting “ivy sign” with sulcal/subarachnoid hyperintensities (arrows) reflective of leptomeningeal collaterals (B and C) Axial and coronal maximum intensity projection MRA: Axial view demonstrates filling defects in the left M1 and A1 segments and narrowing of the right proximal M1 segment (arrow). Coronal view demonstrates multiple collaterals appearing with classic “puff of smoke” appearance (arrow). MRA = magnetic resonance angiogram; magnetic resonance imaging = MRI.

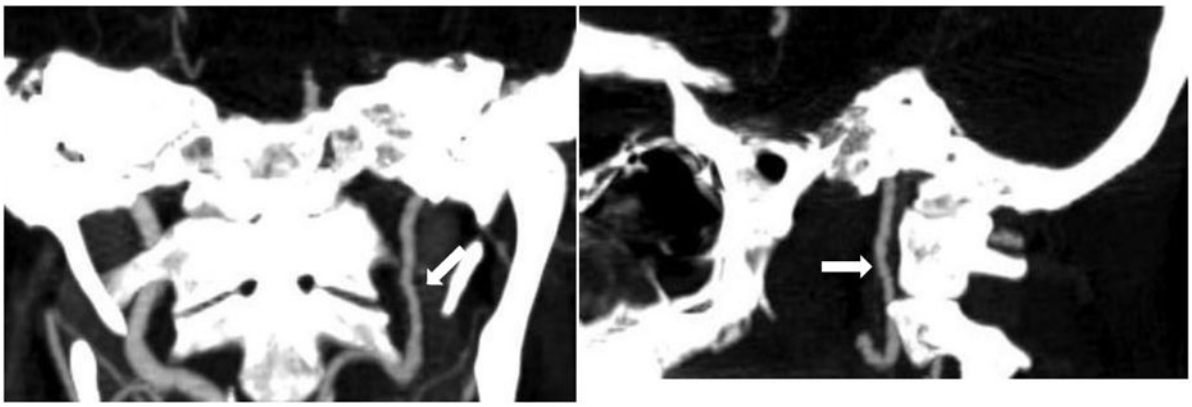

Figure 6:
Coronal and sagittal CTA maximum intensity projections in pediatric patient presenting with left-sided stroke. In both planes the left internal carotid artery demonstrates small continuous undulations representing the classic fibromuscular dysplasia string-of-beads sign. CTA = computed tomography angiogram.

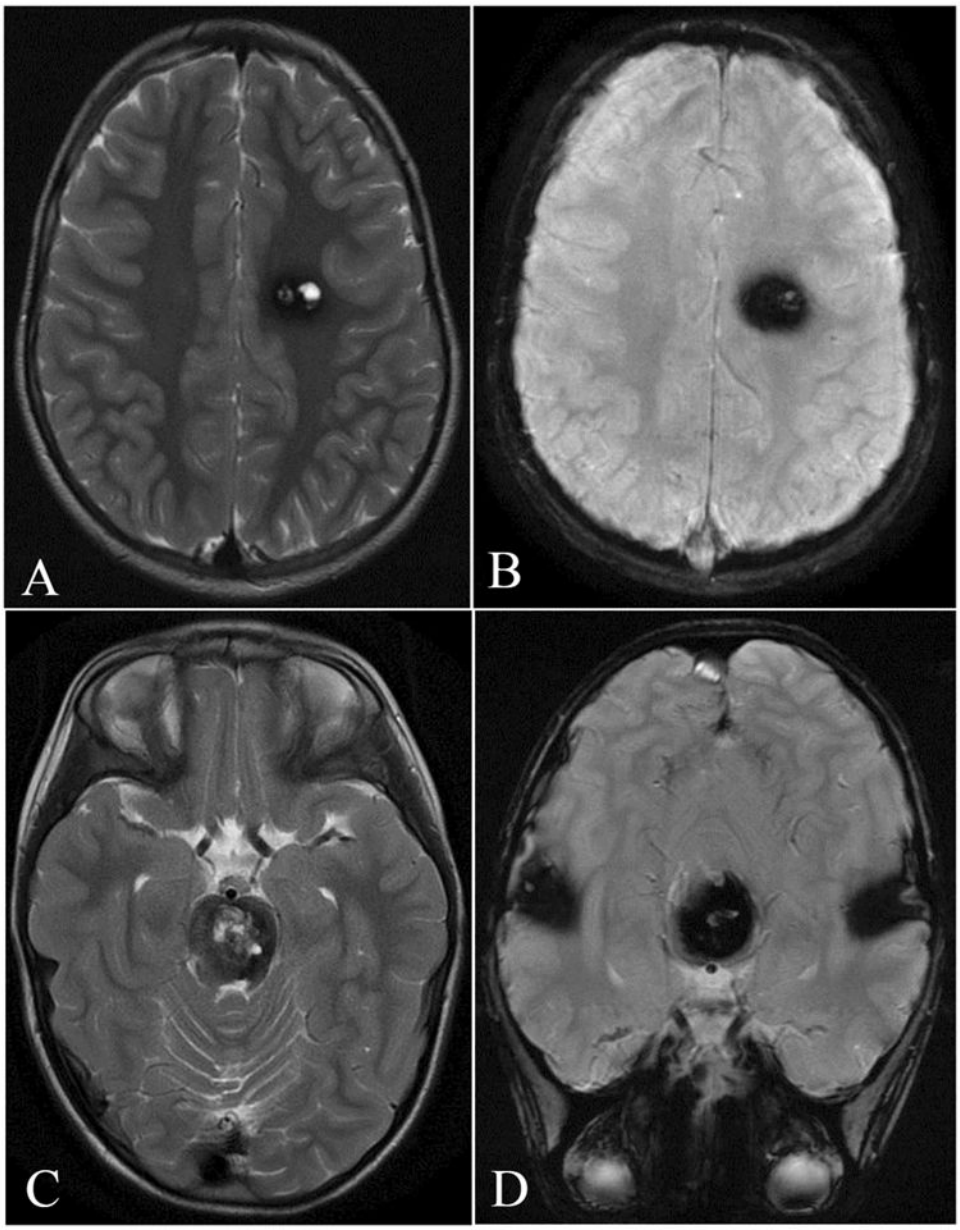

Figure 7:
MRI in 8 year old male with history of prior hemorrhagic strokes and multifocal cavernous malformations. (A) Axial T2WI demonstrating heterogenous T2 signal in the left centrum semiovale with “popcorn” morphology (B) Axial SWI sequence demonstrating blooming (i.e. hypointensity) in the same distribution reflective of blood products (C and D) Similar appearance of pontine cavernous malformation on T2WI and SWI, respectively. MRI = magnetic resonance imaging; SWI = susceptibility-weighted imaging; T2WI = T2-weighted imaging.

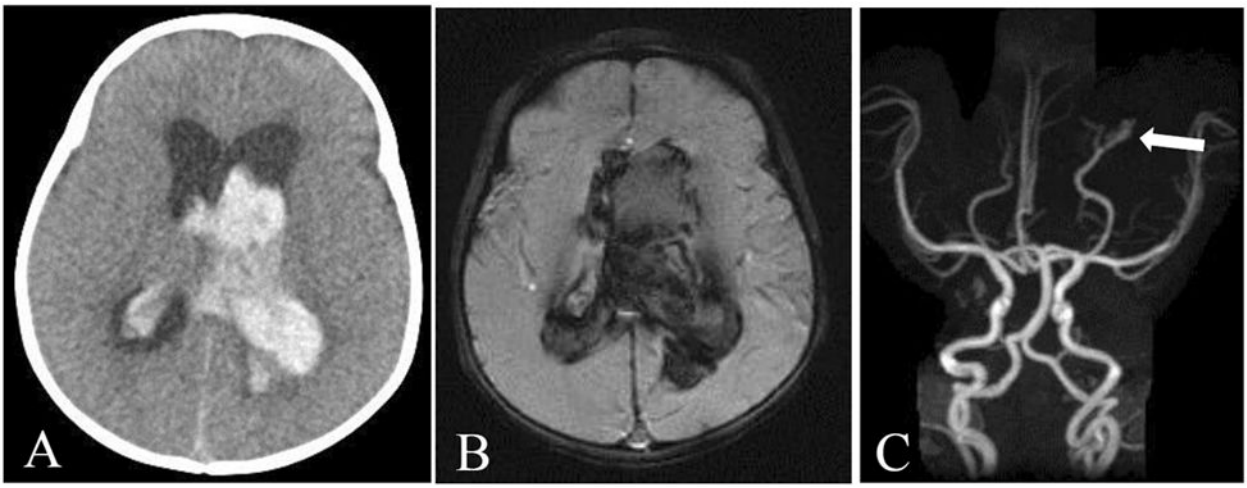

Figure 8:

9 month old female presenting with seizures found to have intraparenchymal and intraventricular hemorrhage. (A) Axial CT head demonstrating hyperintense acute blood within the ventricular system and left occipital lobe consistent with hemorrhage (B) Axial GRE sequence with blooming (i.e. hypointensity) within a similar distribution. (C) Coronal maximum intensity projection MRA showing fusiform dilation of distal left posterior cerebral artery consistent with fusiform aneurysm. CT = computed tomography; GRE = gradient recall echo; MRA = magnetic resonance angiogram; MRI = magnetic resonance imaging.

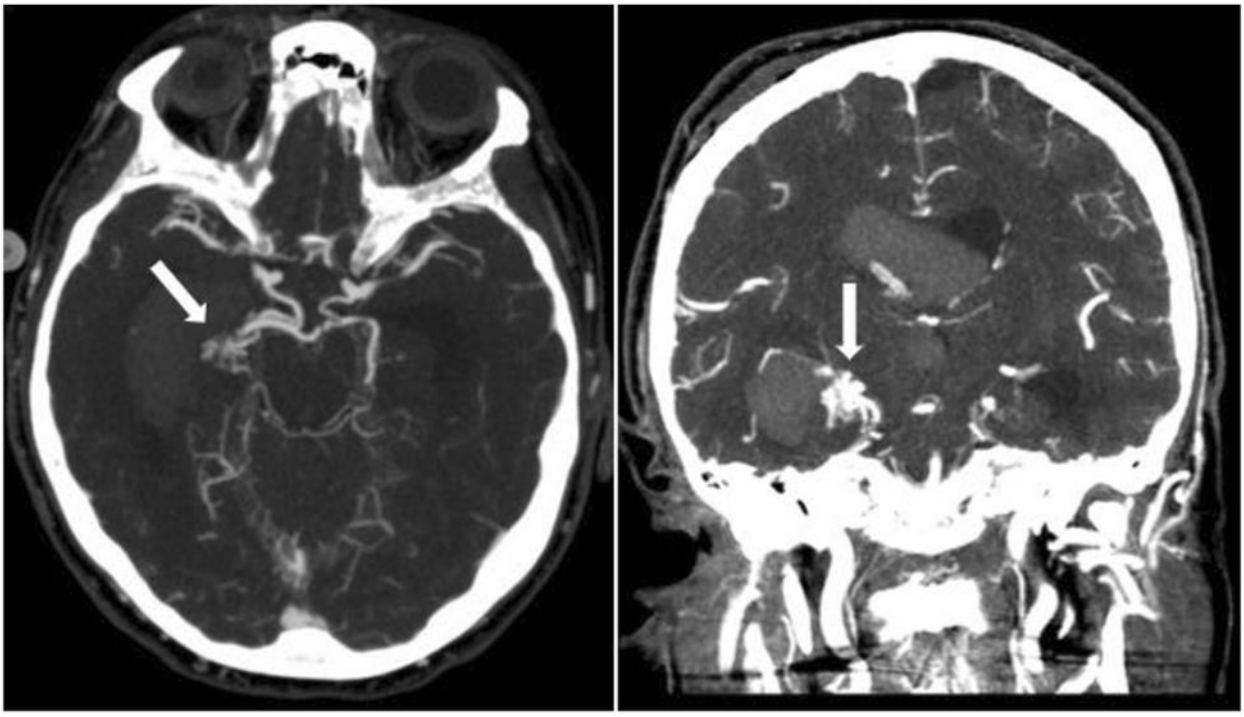

Figure 9:
Axial and coronal maximum intensity projection CTA in pediatric patient with history of hemorrhagic stroke demonstrating abnormal nidus of vessels (arrows) along the distribution of the right posterior cerebral artery. This finding is consistent with arteriovenous malformation. CTA = computed tomography angiography.

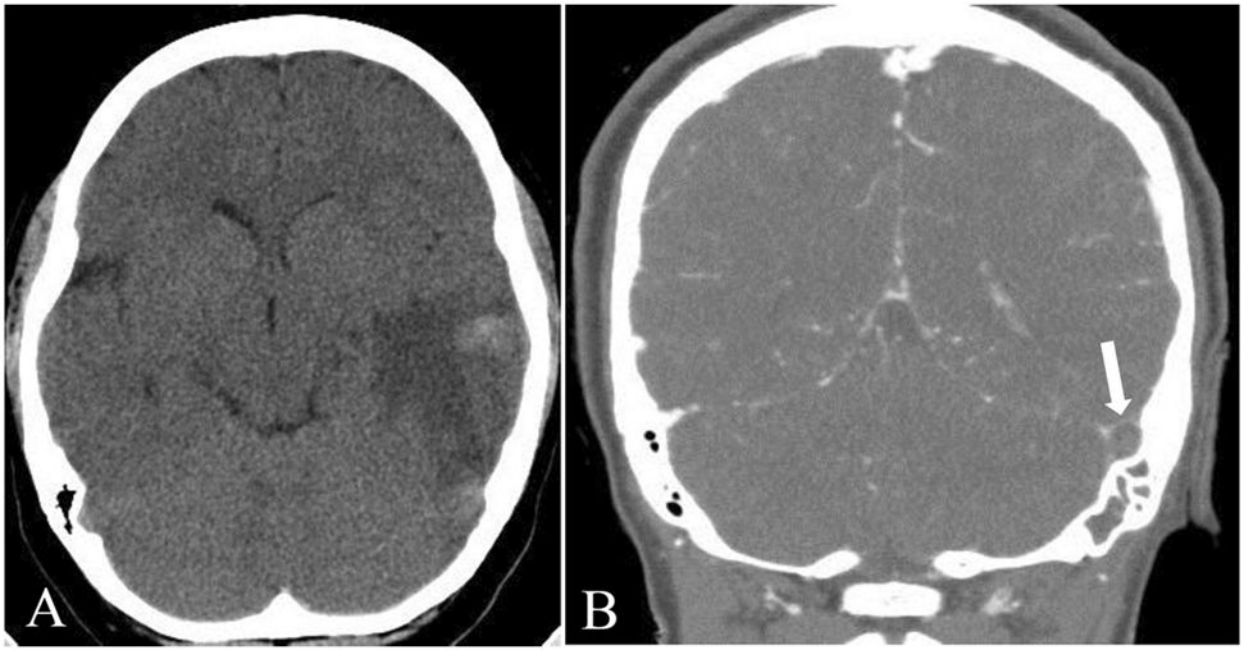

Figure 10:
CT in pediatric patient presenting with suspected stroke. (A) Axial non-enhanced CT with hypodense lesion centered in the left temporal lobe suggestive of infarct related cerebral edema. Regions of hyperdensity indicate associated hemorrhage. (B) Coronal contrast enhanced CT in the same patient with left-sided mastoid opacification and an adjacent filling defect in the transverse sinus (arrow) indicative of mastoiditis related transverse sinus thrombosis. CT = computed tomography.

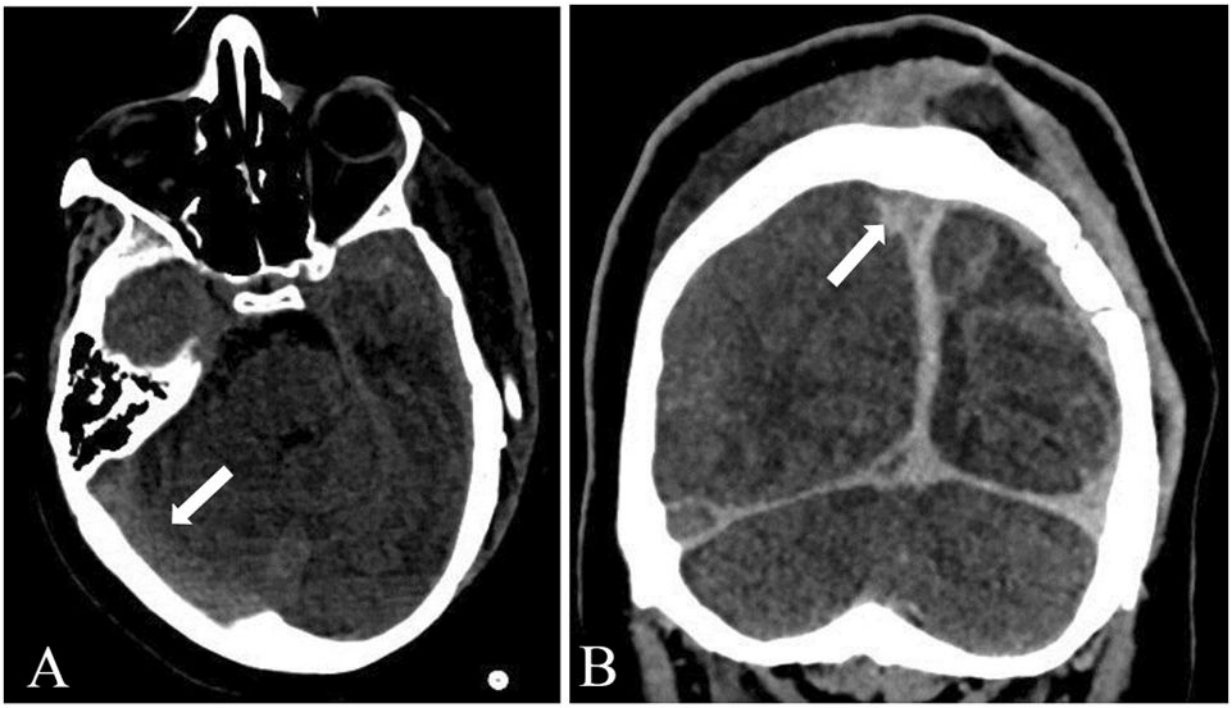

Figure 11:
CT of 15 year old male with history of Rasmussen's encephalitis status-post partial left hemispherectomy due to refractory seizures with recurrent dural venous sinus thrombosis. (A) Axial non-enhanced CT with curvilinear hyperdensity within distribution of right transverse sinus (arrow) representative of the "cord sign" seen with dural venous sinus thrombosis. (B) Coronal contrast-enhanced CT demonstrating abnormal hypodensities within the right transverse and straight sinuses. Hypodensity within the straight sinus (arrow) reflective of dural venous sinus thrombosis' "empty delta sign." CT = computed tomography.

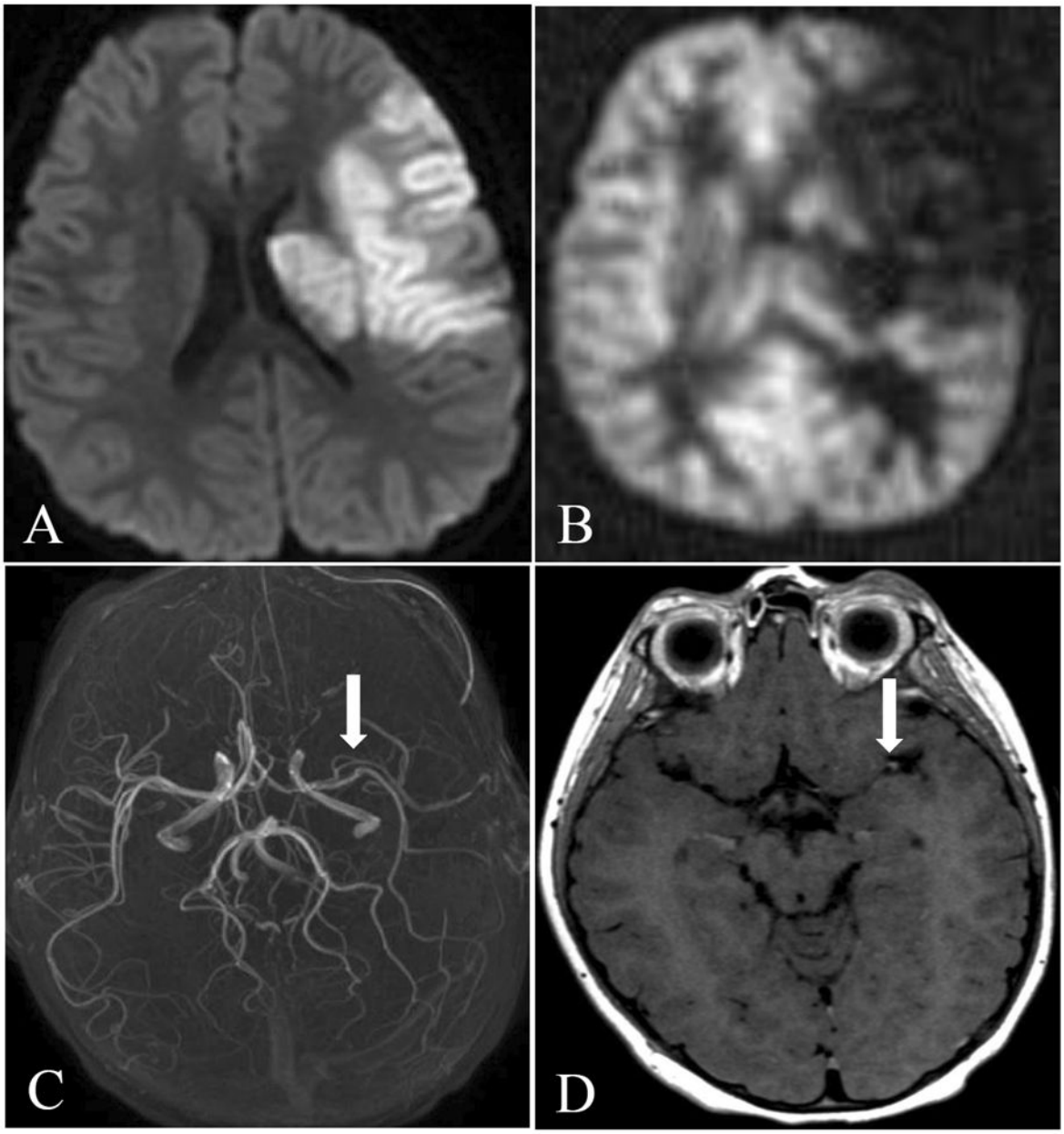

Figure 12:

MRI in a 5 year old male presenting with seizures and right sided weakness. (A) Axial DWI sequence with infarct in the left frontal lobe, insula, and basal ganglia (B) Axial ASL sequence with corresponding hypointensity reflective of decreased cerebral blood flow (C) Axial maximum intensity projection MRA illustrating diminutive left MCA and its branches (D) Axial black blood imaging post-contrast sequence with foci of enhancement in wall of the left MCA indicative of arterial wall inflammation. This finding was concerning for TCA.ASL = arterial spin labeling; DWI = diffusion weighted imaging; MCA = middle cerebral artery; MRA = magnetic resonance angiogram; TCA = transient cerebral arteriopathy.

Table 1:

Common MRI Sequences in Pediatric Stroke

Sequence	Clinical Utility
T1-weighted imaging (T1WI)	– Overall anatomic delineation – Utilized post-contrast to assess parenchymal enhancement, which in the context of stroke is related to disruption of the blood brain barrier
T2-weighted imaging (T2WI)	– Fluid sensitive sequence delineating ventricular system and fluid filled structures (e.g. cysts, abscesses)
Fluid attenuated inversion recovery (FLAIR)	– T2WI with suppression of fluid signal – High sensitivity for parenchymal edema which may be related to stroke
Diffusion weighted imaging (DWI)	– Highly sensitive for hyperacute and acute stroke[12]
Apparent diffusion coefficient (ADC) maps	– Confirmation that DWI hyperintense lesion is related to restricted diffusion rather than intrinsic tissue T2 signal
Gradient recall echo (GRE)	– Sensitive for blood products and calcifications, but is unable to distinguish between them
Susceptibility weighted imaging (SWI)	– Increased sensitivity for blood products and calcifications as compared to GRE, and utilizing specific techniques can be used to differentiate between them[13,14]

MRI = magnetic resonance imaging.