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Case Report

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Vertebral Artery Dissection in a 7-year-old girl: A case report, pictorial review, and review of literature

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Abstract

Vertebral artery dissection (VAD) is a not uncommon cause of arterial ischemic stroke in the pediatric population. Though it is frequently overlooked, and resources help in diagnosis often a concern, the establishment of diagnostic criteria is very crucial for treatment and prognosis. Here, we present a case of VAD with a pictorial and literature review, in addition to imaging recommendations.

Keywords: Vertebral Artery Dissection, Craniocervical Artery Dissection, Pediatric Stroke, Posterior Circulation Stroke, Dissection Imaging, Antiplatelet, Anticoagulation.

Introduction

Vertebral Artery Dissection (VAD) is a frequently overlooked and underdiagnosed cause of stroke in the pediatric age group [1]. It occurs due to a tear in the intimal layer after trauma or spontaneously in the presence of underlying risk factors for vascular pathology, resulting in stenosis or thrombosis, with possible embolization [2, 3].

300 cases of craniocervical artery dissection (CCAD) in the pediatric age group (<18years) have been reported in the past 54 years since 1966, 158 of which involved posterior dissections with a mean age of 8.6 years [2, 4].

The incidence of arterial ischemic stroke (AIS) in the pediatric population is estimated to be 2.5 to 8 per 100,000 people/year. Up to 20% of which is reported to be due to CCAD, with VAD occurring in 60% of CCADs [2]. Thereby, the incidence of VAD in pediatrics is lower than the incidence in adults of 1-1.5 per 100,000; which, in contrast, along with carotid artery dissection, accounts for 2% of adult ischemic stroke cases [5].

Spontaneous and traumatic cases have been reported previously with a predominance of head and neck trauma, due to mechanisms ranging from minimal trauma encountered during sudden cervical movements to mechanical and penetrating trauma [2, 4, 6]. It has been reported predominantly in males, mainly attributed to higher susceptibility to trauma [2, 4, 7, 8]. However, spontaneous VAD may occur in the context of underlying risk factors or vascular pathologies such as connective tissue disease, fibromuscular dysplasia, or anatomical defects [2, 4, 9]. Most commonly, post-traumatic cases involve the posterior circulation, with dissection of the extracranial portion of the vertebral artery at the atlantoaxial C1/C2 region [2, 4, 7].

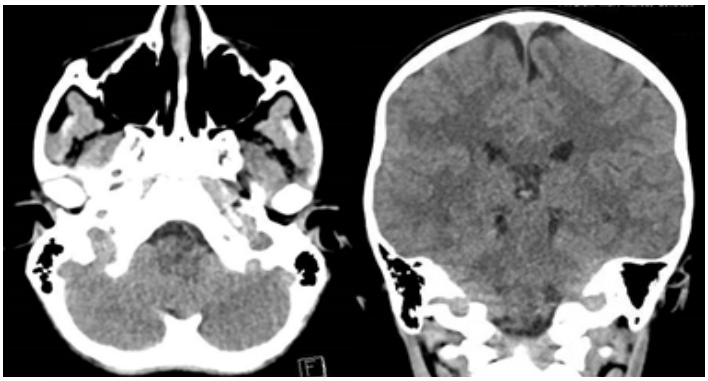
Due to the mild clinical picture in the early stages, early diagnosis is challenging. Neurodiagnostic imaging and expertise are critical in detecting these cases, which greatly influence immediate management decisions and hence determine outcomes ranging from full recovery to residual neurological deficits [2, 4, 10].

Case Presentation

A 7-year-old girl was transferred to our pediatric intensive care facility from a nearby general hospital as a case of unexplained acute encephalopathy with rapid deterioration and subsequent loss of consciousness within hours. She was initially admitted to the general hospital with complaints of sudden onset dizziness, decreased movement, fluctuating alertness, and rolling neck movements. These were explained as a possible extrapyramidal

adverse reaction secondary to an IM injection of metoclopramide received earlier at a clinic for vomiting.

Previously in the afternoon, she was reported to be well and was playing with her friends at the playground until the evening when she started feeling dizzy and vomited repeatedly. She was taken to a nearby clinic where she received an IM injection of metoclopramide. She was reported to have intact sensorium and was fighting against the injection. Soon at home, she had rolling movements of her head, with subsequently decreased movement and awareness. She was then taken early in the morning to the hospital with these presenting symptoms, with concerns of a possible adverse reaction. There was no history of headache, irritability, fever, or respiratory illness. She had no history of sick contact, and her vaccinations were up to date. She was a product of a twin IVF pregnancy, and her twin sister had no similar condition, with insignificant personal medical or surgical history. No family history of hypercoagulation, connective tissue disease, or neurologic disease. Her parents were not consanguineous. On examination, she was vitally stable, responding intermittently with some verbalization. She had abnormal movements of the upper and lower limbs. Her GCS was 10. Cranial nerve examination was unremarkable. There were no marfanoid aspects, and she had a normal upper to lower segment ratio. She continued to have fluctuations in the level of consciousness. Given the antiemetic history, it was suspected to be an acute dystonic reaction, and a dose of promethazine was given accordingly. Liver function tests and blood gases were normal. Her condition was not improving, and an unenhanced brain CT was performed (Figure 1) (9hrs after symptoms onset) which was reported to be normal. A following brain MRI without contrast was performed (Figure 2 (a)) (12hrs after symptoms onset) and was also reported to be normal. At noon, acute deterioration of her mental status and respiratory compromise necessitated intubation and mechanical ventilation. Empirical therapy with IV Ceftriaxone and Acyclovir was started.



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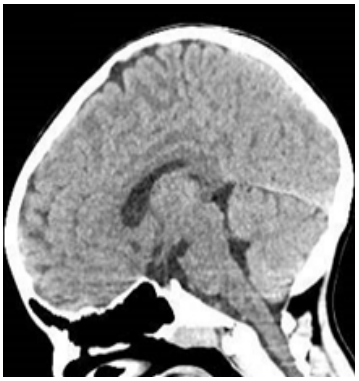

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Figure 1: (a) Axial, **(b)** coronal, and **(c)** sagittal reconstructions of helical acquisition of unenhanced brain CT. Multiple streaks artifacts (hardening artifacts) causing significant limitation in evaluating the posterior fossa structures, particularly the medulla oblongata; however, showing a subtle anterior triangular left paramedian hypodensity

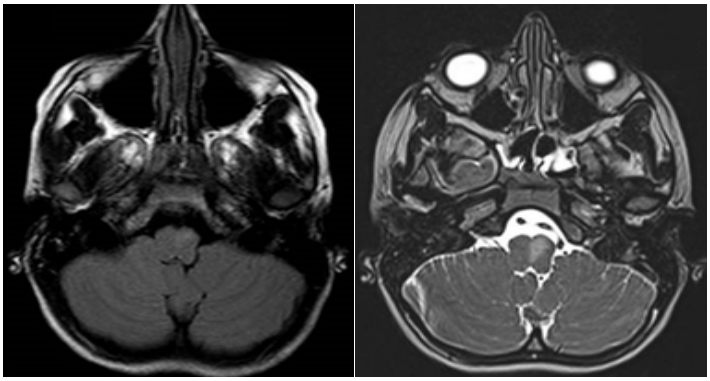
She was referred to our intensive care facility on the same evening with possibilities of dystonic reaction to metoclopramide, viral encephalitis, or acute disseminated encephalomyelitis (ADEM). On arrival, the patient was under sedation. Her examination showed a GCS of 3. Her temperature was 37.5, heart rate was 90, BP was 112/70 and SpO2 was 100%, normal capillary perfusion, no skin rash, jaundice, or pallor. Capillary blood gases showed mild respiratory acidosis. Her WBC, Hb, Plt, PT, PTT, INR, Electrolytes, CRP, PCT, ESR, Ammonia, Lactate, LFT, and RFT were all normal. Mycoplasma IgM was negative. Her chest x-ray was normal. After withdrawal of sedation, a neurological examination on the following day showed a GCS of 11/15. Reflexes were absent in the lower limbs but decreased in the upper limbs. Power was 2/5 in all her limbs. On funduscopic examination, there was no sign of papilledema.

CSF routine studies were normal and negative for Latex antigen, HSV, NMDA antibody, and oligoclonal bands. Blood was negative for aquaporin 4. Urine drug & toxicology screening were also negative. EEG showed diffused moderate slowing, with no epileptic activity. A second brain MRI without and with IV contrast (Figure 2(c-f)) (39hrs after symptom onset) demonstrated abnormal signal intensity in the upper cervical spine and lower medulla oblongata. No definite abnormal enhancement noted. No cord swelling noted. The radiology report proposed possible brainstem encephalitis or transverse myelitis.

The following day her condition improved and she was extubated. Upon a second reading of the performed CT and MRI exams by a neuroradiologist, an ischemic insult with suspicion of posterior CCAD was noted, and a brain and neck MRA was advised. In light of the suspicion of VAD by the neuroradiologist, the family

was inquired about the history of trauma. It was revealed that the girl was swung around by both arms and slipped off, resulting in hyperflexion of her neck.

An MRA of the brain and cervical spine performed (Figure 2(b) and Figure 3) (92hrs after symptom onset) confirmed a large ischemic infarct involving the left anterior paramedian rostral and midmedulla oblongata and the bilateral anterior paramedian caudal medulla oblongata extending around the ependymal canal in the upper cervical spinal cord downward to C2-C3 disc level in the proximal territory of the anterior spinal artery. Additionally, a highly conspicuous intraluminal flap is noticed in the cranial left V2 segment extending to the V4 segment of the vertebral artery.



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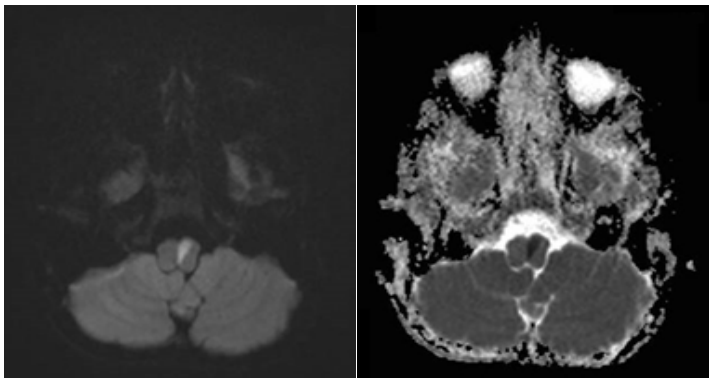

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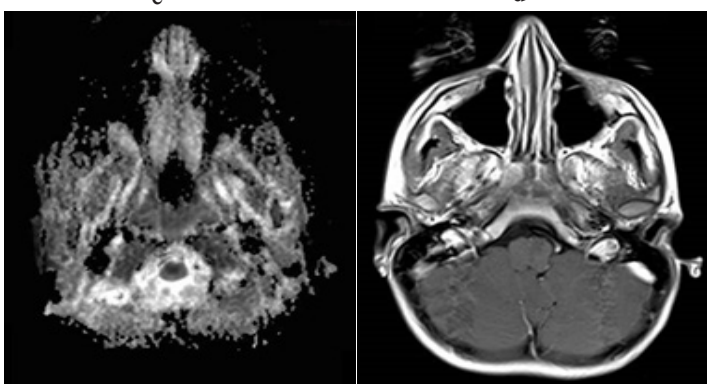

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Figure 2: Minimal FLAIR hyperintense signal **(a)** corresponding to a bright signal abnormality seen on T2 pulse sequence **(b)** and diffusion B1000 signal **(c)** with restricted (hyposignal) apparent diffusion coefficient (ADC) **(d)** noted in the left anterior paramedian rostral and mid medulla oblongata and the bilateral anterior paramedian caudal medulla oblongata extending around the ependymal canal in the upper cervical spinal cord downward to C2-C3 disc level **(e)**. Subtle T1 low signal abnormality is noted on the T1 pulse sequence with no significant enhancement after Gadolinium **(f)**

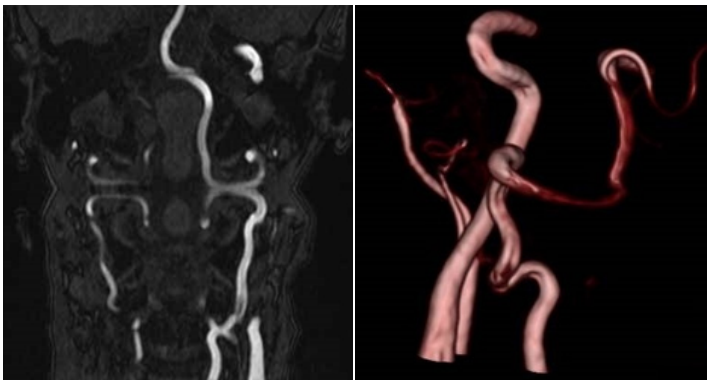

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Figure 3: A very specific feature of left vertebral dissection with a double lumen and intimal flap; although, are often difficult to visualize. The intraluminal flap is extending from the distal left V2 segment to the V4 segment seen on the stretched MIP reconstruction **(a)** and the volume rendering reconstruction **(b)**

As per the radiological findings, the patient was commenced on aspirin, with discontinuation of Ceftriaxone and Acyclovir. Physiotherapy and speech therapy were started. Gabapentin and clonidine were later on added for neuropathic pain. The patient was transferred to the ward 6 days after admission, and follow up cervical spinal cord MR and MRA were performed 3 weeks later, which demonstrated ischemic sequela in the medulla oblongata and the proximal cervical cord with no significant enhancement and a persistent flap between patent false and true channels.

Discussion and Review of Literature

VADs commonly present with features of AIS (Arterial Ischemic Stroke), however, may only be suspected if in the context of recent trauma; although, many cases occur with trivial or no history of trauma [2, 4]. This girl had a history of mild trauma, however, the IM metoclopramide misled the initial focus for investigations, until her condition progressed to more serious clinical manifestations. This emphasizes the importance of obtaining a detailed initial history and employing appropriate history documentation when transferring between healthcare facilities.

The initial suspicion of an acute dystonic adverse reaction to metoclopramide may have been appropriate given the temporal relationship of onset of her symptoms after the injection since

reactions have been reported even at therapeutic levels. However, these symptoms most likely appear within 24 to 72 hours of metoclopramide administration and involve characteristic extrapyramidal features such as acute torticollis, retrocollis, facial grimacing, and oculogyric crises [11].

Encephalitis in the pediatric age group is caused by several infectious, autoimmune, and toxic etiologies [12]. Rhomboencephalitis, encephalitis of the brainstem, was one of the early suspicions due to the presenting clinical features of posterior fossa involvement. The priority was to rule out infectious causes of rhomboencephalitis, which were investigated thoroughly in this case, and acyclovir and broad-spectrum antibiotics were empirically initiated. The normal CSF findings, negative cultures, and PCR makes this diagnosis less likely [13]. Moreover, the negative NMDR antibodies, oligoclonal bands, and aquaporin-4 rule out autoimmune or demyelinating causes of rhomboencephalitis and transverse myelitis. Although the clinical presentation of this case could have been an acute manifestation of ADEM, it would classically lead to multiple supratentorial asymmetrical lesions of the deep and subcortical white matter on MRI, and these radiographic findings are absent in our case [12].

This case occurred in a girl, contrary to the male predominance of CCAD in pediatrics, particularly posterior CCAD [2, 4, 8]. A case series of VAD has shown a male to female ratio of 6.6:1 [7]. Certain risk factors and underlying conditions predispose to dissections in the pediatric age group, including fibromuscular dysplasia, connective tissue diseases (including Ehlers Danlos type IV, Marfan Syndrome, and Osteogenesis Imperfecta type I), cervical spine anomalies, and vascular malformations [7, 14]. Nevertheless, the majority of cases that present with spontaneous dissection lack any underlying vascular pathology (vasculopathy) [4, 7, 14]. The absence of clinical features and negative family history of connective tissue disorders may minimize suspicion of this type of risk factor; however, absence of skin clinical manifestations cannot completely exclude possible connective tissue vasculopathy [15]. Fibromuscular dysplasia would have characteristic "string of beads" radiographic findings that are absent in this patient.

As reported in previous literature, the most commonly involved region in VAD is at the level of C1-C2 [2, 4, 7]. This anatomical predisposition is greatly due to the close proximity of the V3 segment to the atlantoaxial junction and lateral masses of C1 and C2, which predisposes the V3 segment to injury against these bony prominences, and the high V2 segment where it is susceptible to stretching against the transverse foramina of C1 and C2 [2, 14]. However, dissections can extend proximally or distally from that point [7]. In this case, the dissection involved the high V2 segment to the proximal V4 segment of the vertebral artery, extending and leading to the occlusion of the anterior spinal artery, with consequent spinal cord infarction at the C1 and C2 levels, an uncommon complication of VADs [16].

The presenting clinical features vary according to the affected portion of the vertebral artery and its branches, with the evolution of the dissection leading the progression of clinical features. In this case, the child first developed vomiting and dizziness, which later on progressed to weakness and reduced responsiveness. Previous studies have shown that VADs commonly present with vague early symptoms, including headache, nausea, vomiting, dizziness, diplopia, and altered level of consciousness, mimicking other neurologic conditions. These commonly progress later to hemiparesis, ataxia, and nystagmus. In addition, neck pain or stiffness may be one of the specific symptoms to posterior than anterior CCAD; however, it is less commonly found in pediatrics compared to adults [2, 4, 7, 10]. It is noteworthy that the rolling neck movements in our case may have been due to neck stiffness or pain. The involvement of the anterior spinal artery explains the radiographic findings of bilateral paramedian central cervical cord abnormalities and the clinical picture of bilateral hemiparesis.

Involvement of the vertebral artery, which supplies the medulla and cervical spine, leads to medial medullary syndrome (Dejerine syndrome), resulting in contralateral body weakness, loss of vibration & position sense, and ipsilateral tongue paralysis. The lateral extension into the intracranial portion to involve the PICA, which supplies the posterior inferior cerebellar hemisphere, vermis, and medulla; would lead to Lateral medullary syndrome (Wallenberg syndrome) presenting with nausea, vomiting, vertigo, dysmetria, ataxia, crossed sensory findings, nystagmus, Horner syndrome, and dysphonia. While the involvement of the PICA territory classically presents with lateral medullary syndrome (Wallenberg syndrome) in adults, it is uncommon in the pediatric age group. Further involvement of the anterior spinal artery would result in quadriparesis, hyperreflexia, bilateral loss of pain & temperature sensation, and autonomic instability [14, 17]. In accordance with these clinical features, bradycardia was noted on several occasions at nighttime during the patient's stay in the ward. The majority of reported cases had a variable combination of clinical features of these syndromes, which can be explained by the presence of collaterals, and the intermittent nature of flap occlusion and embolism in dissections; yet the non-specific symptoms (headache, dizziness, vertigo, ataxia) are all related to stroke of the posterior circulation.

These vague symptoms form a major challenge to the early clinical suspicion and diagnosis of VAD, in the absence of a history of trauma, or even stroke. Low clinical suspicion has been shown to contribute to the delay in stroke diagnosis in pediatrics, with a median time from symptom onset to diagnosis of AIS being 25 hours [18]. Besides, the consistent presence of ischemia at the time of diagnosis signifies failure in the diagnosis of dissections before ischemia ensues [4]. The time for our patient's symptom onset to radiological diagnosis was 92 hours, which was attributed

to several factors including history, delayed neuroradiological diagnosis & referral to another facility. Furthermore, retrospective analysis of the initial CT scan and MRI performed earlier demonstrated the ischemic insult in the vertebrobasilar circulation. Particularly, MRI diffusion-weighted images had clear evidence of ischemia, indicating a systematized stroke territory of the posterior fossa, which would be highly suspicious of VAD. These findings were overlooked and possibly misinterpreted as artifacts commonly seen in the posterior fossa. This highlights the need for neuroradiological review. In addition, in cases with evidence of systematized stroke involvement along one arterial territory, vascular assessment is required starting by brain and neck MRA or CTA; and eventually, a digital subtracted angiography in case of negative results.

The diagnosis of VAD and the subsequent prognosis is dependent on appropriate initial clinical suspicion and the results of the initial findings using specific neuroimaging modalities. Clinicians have to raise suspicion of VAD in the setting of posterior circulation stroke, as it has been shown to be a common etiology of posterior AIS [19]. The best imaging tool has to be selected first as per the well-established and evidence-based American College of Radiology (ACR) appropriateness criteria [20]. A CT scan is useful in settings where MRI services are not available to exclude hemorrhagic changes and mass effects. A normal unenhanced brain CT scan should not rule out the suspicion of VAD, as CT is known to miss or underestimate arterial ischemic stroke in at least 15% of children [1]. CTA of the brain and neck is required in the same settings to evaluate the anterior and posterior arterial circulation, but this will significantly increase the radiation dose, particularly to the lenses and the thyroid gland. The presence of oral metallic implants is a well-known generator of artifacts in CT, CTA, MRI, and MRA. Some CT scan technological advances (Dual Energy CTA) could be an excellent modality in the case of such implants. Additionally, the DE CTA has a low radiation dose and requires a lower dose of IV iodine contrast injection.

The brain MRI followed by brain and neck MRA is a non-radiating highly sensitive exam for strokes and represents a very good exam to demonstrate the extent of the ischemic insult in order to evaluate the prognosis and analyze the involved arterial structure needed eventually for immediate case management. The non-radiating MRI brain is superior to CT, particularly in demonstrating posterior fossa ischemic changes. The diffusion-weighted imaging MRI is the most powerful pulse sequence to diagnose the ischemic changes in comparison with T2 and FLAIR, particularly in the early stages. The brain MRA performed without IV contrast injection is useful to assess the patency of the Circle of Willis segments as well as the vertebrobasilar circulation patency. Before the IV injection, an axial T1 pulse sequence centered on the neck with triple saturation (arterial, venous, and fat saturation) is needed to identify arterial wall hematoma visualized as a "bright crescent sign" in the arterial wall, which is one of the characteristic dissection manifestations. Therefore, the diagnosis of CCAD relies heavily on MRI and MRA, which have become preferred modalities in neurovascular imaging [2, 9].

Missing the diagnosis of CCAD in MRA has been attributed to failure to scan the full extent of head and neck vasculature, diagnostic error, and suboptimal technique [21]. In the case of negative MRA, a DSA should be ordered. DSA is the gold standard of CCAD evaluation and is superior to CT and MRI in demonstrating intraluminal arterial findings.

The CASCADE criteria, followed by the International Pediatric Stroke Study, states that [22]: "Confirmation of the diagnosis of intracranial or cervical arterial dissection requires CTA, MRI/MRA or CA with one of the following three patterns:

1. Angiographic findings of a double lumen, intimal flap, or pseudo aneurysm, or, on axial T1 fat saturation MRI images, a "bright crescent sign" in the arterial wall;

2. The sequence of cervical or cranial trauma, or neck pain, or head pain less than 6 weeks preceding angiographic findings of segmental arterial stenosis (or occlusion) located in the cervical arteries;

3. Angiographic segmental stenosis (or occlusion) of the vertebral artery at the level of the C2 vertebral body, even without known traumatic history. (adapted from Sebire et al., 2004)"

The management approach to pediatric VAD and CCAD, in general, remains controversial, with studies reporting mixed outcomes using either antiplatelet or anticoagulation therapies [2, 4, 7, 10]. However, in adults, it has been shown that both have similar efficacy in reducing stroke recurrence and death [23]. Perhaps, the absence of therapeutic guidelines in VAD is mostly related to the wide range of presentation from wall hematoma to complete occlusion of the involved vessel. In our case, as the true and false channels were patent, antiplatelet therapy with aspirin was started. The use of either therapy depends on the occlusion size, severity, presence of thrombus, and extension of the dissection [17]. Although, based on the proposed mechanisms of thromboembolism in CCAD, it has been suggested that anticoagulation would be better suited, yet it still carries the risk of hemorrhage, especially with intracranial extension [17, 23]. The most recent guidelines set by the 2008 AHA/ASA recommended the following for childhood AIS, yet these have not been validated by controlled trials or prospective studies (Table 1) [24]:

Table 1: Management Recommendations for CCAD in Children

Class II Recommendations	
In children with extracranial CCAD, it is reasonable to begin either UFH or LMWH as a bridge to oral anticoagulation.	(Class IIa, Level of Evidence C)
It is reasonable to treat a child with an extracranial CCAD with either subcutaneous LMWH or warfarin for 3 to 6 months. Alternatively, an antiplatelet agent may be substituted for LMWH or warfarin. Extending anticoagulant therapy beyond 6 months is a reasonable option for individuals who develop recurrent symptoms. It is reasonable to continue antiplatelet agents beyond 6 months, especially when there is radiographic evidence of a residual abnormality of the dissected artery.	(Class IIa, Level of Evidence C)
In patients who continue to have symptoms from a CCAD despite optimal medical therapy, surgical procedures may be considered.	(Class IIb, Level of Evidence C)
Class III Recommendations	
Anticoagulation is not recommended for children with an intracranial dissection or those with SAH resulting from CCAD.	(Class III, Level of Evidence C)

The importance of rehabilitation in the post-stroke period using a multi-disciplinary team has been highlighted by the class I recommendation of age-appropriate rehabilitation and therapy programs in the 2008 and 2019 AHA/ASA scientific statements [24, 25]. Besides, follow-up neurovascular imaging has been recommended within 3-6 months, due to possible recurrence of dissection or ischemic events [2]. Partial or complete recanalization has been shown to occur in 50-67% of childhood CCAD, occurring more commonly in VAD than CAD [2, 4, 8, 10, 14].

Concluding Key Points

- VAD should be suspected in case of any posterior neurological symptoms even in absence of suggestive history
- MRI (including particularly diffusion-weighted) and MRA of the head and neck should be preferred for the diagnosis of any child presenting with neurologic deficit.
- Neuroradiological review is advised due to modality inherent technical limitations as well as common general radiologist interpretation limited skills; therefore, a dedicated stroke center referral is essential when available.
- In cases with evidence of systematized stroke along one territory, vascular causes have to be investigated.
- DSA should be performed in case of positive MRI but with negative brain and neck MRA exams. It may be used initially in patients having a suspicion of fibromuscular dysplasia or any anatomical vascular defect or abnormality.
- Patency of the dissected vessel can guide the management and intervention modality.

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