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Topical Review

Craniocervical Arterial Dissection in Children: Pathophysiology and Management

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ABSTRACT

Background: Craniocervical arterial dissection is a commonly reported arteriopathy associated with stroke in children. It is characterized by a high stroke recurrence rate and variable outcomes. Here we review the pathophysiology, clinical presentation, and diagnostic neuroimaging approaches that are helpful in accurate diagnosis and follow-up of children with arterial dissection.
Methods: MEDLINE searches (2000 to 2018) for articles that contained patients aged less than 18 years with craniocervical arterial dissection was performed, with the goal of analyzing their presenting features, pathophysiological mechanisms, and imaging characteristics and interventions.
Results: Sixteen articles met the study criteria and reported 182 cases of craniocervical arterial dissection, 68% male, average age 8.6 years. Dissection was associated with head and neck trauma in 56% of the cases and frequently involved the posterior (61%) and extracranial locations (64%); the vertebral artery was the most commonly involved artery (60%). The most common clinical presentation was hemiparesis (80/160, 50%), followed by headache (64/164, 39%). Magnetic resonance imaging was the preferred neuroimaging method, followed by cerebral catheter angiography as a gold standard definitive neurovascular imaging modality when the initial vascular imaging revealed nondiagnostic findings.
Conclusions: The diagnosis of arterial dissection requires a high index of suspicion and consideration for detailed neurovascular imaging, including both the cranial and cervical regions. Neurovascular imaging challenges, especially visualization of arterial abnormalities, highlight the importance of appropriate and timely use of specific neurovascular imaging techniques. Magnetic resonance imaging appears to be the preferred neurovascular imaging modality in children with arterial dissection and may obviate the need for invasive cerebral catheter angiography.

Introduction

Craniocervical arterial dissection (CCAD) is among the most commonly reported cerebral arteriopathies associated with arterial ischemic stroke (AIS) in both children and young adults. In adults an annual incidence for CCAD is estimated at 2.5 to 3 per 100,000 persons per year.[1] In children the exact incidence of CCAD is not known and tends to be under-reported, largely owing to the missed and inaccurate diagnoses.[2,3] CCAD accounts for 7.5% to 20% of all childhood AIS cases,[4-7] with an annual incidence of all AIS estimated at 2.5 to 8 per 100,000 children per year.[8-11]

Head and neck trauma accounts for more than half of all pediatric dissection cases, but in the remainder, dissection is classified as spontaneous with no obvious trauma.[5,6,12,13] The risk of traumatic CCAD increases with the severity and type of trauma; it frequently occurs in the context of major mechanical, whiplash, and penetrating injuries. A few cases of CCAD (up to 2%) are also reported in association with blunt injuries.[14,15] Interestingly, in up to 25% of childhood CCAD cases, trauma can be minimal, such as neck hyperextension or manipulation, physical effort, or participation in contact sports.[16-18] With spontaneous CCAD, at least 5% to 20%

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children have an underlying risk factor.[17,19-23] These commonly include connective tissue diseases, genetic disorders, anatomic bony and vascular variations, and familial segregation.[19,22,24] In addition, ultrastructural aberrations of dermal collagen or elastic fibers and neuroimaging findings of tortuous cervicocephalic arteries are reported in 19% to 68% of both pediatric and adult spontaneous CCAD cases, suggesting another vascular association.[25-27]

The clinical presentation of CCAD is highly variable depending on the location and the extent of arterial injury and is non-differentiating from other causes of AIS aside from a history of head and neck trauma or pain or the presence of cranial nerve palsies.[13,17,19] In the context of recent trauma, physicians often suspect CCAD; however, the diagnosis is either not confirmed or remains presumed because of the difficulties in demonstrating diagnostic neuroimaging features of CCAD.

Children with CCAD require an accurate diagnosis and prompt treatment along with close monitoring, as they carry a notably high recurrence rate for subsequent cerebral ischemic events[5,12,28-31] and their outcomes can be highly variable, ranging from a complete recovery to moderate neurological deficits.[5,6,12,17,21] The use of appropriate neurovascular imaging techniques is necessary to make a definitive diagnosis of CCAD. Owing to its high sensitivity and specificity, magnetic resonance imaging (MRI) has become a preferred neurovascular imaging modality in children with suspected dissection.[19]

The objective of the present article is to understand the pathophysiology and clinical presentation of CCAD (mainly carotid and vertebral artery) and to review the current diagnostic approaches, in particular specific and advanced neuroimaging techniques that are helpful in the timely and accurate diagnosis and follow-up of children with CCAD. This study was compared with a previous study that reviewed 35 years of pediatric stroke cases and identified 118 children with 132 CCADs,[17] attempting to identify any changes that may have occurred since the earlier study was published in 2001.

Materials and Methods

A comprehensive review of the literature was undertaken using the MEDLINE database PubMed. Keywords used to obtain all available literature included *craniocervical arterial dissection, craniocervical dissection, dissection, cervical dissection, vertebral dissection, pediatric(s) and stroke, craniovertebral, craniocephalic*. The literature search was conducted using an 18-year limit (2000 to 2018) with the goal to obtain case series articles that had greater than or equal to three CCAD cases reported and presented information on age, sex, underlying medical conditions, presence of trauma or other injuries, presenting symptoms, neuroimaging findings, and treatment. Articles were excluded if they contained adult patients, dissection in the perinatal period,[32,33] and contained only radiological reports.

The initial literature search identified a total of 135 articles, of which 71 articles did not meet the inclusion criteria. Of the remaining 64 articles, 48 articles describing only one or two cases were excluded, resulting in 16 articles with 182 cases to be analyzed and discussed in this article.[5,6,12,13,16,18,20,21,24,34-40]

Results

Of the 182 CCAD cases reviewed, the average age at presentation was 8.6 years (range six months to 17 years), with male predominance (68%) (Table 1). Fullerton et al.[17] noted a similar male predominance (more than 80% males with CCAD), with an average age at initial presentation of 10.4 years for the anterior circulation dissection and 8.6 years for the posterior circulation dissection. The most commonly suggested explanation of the male predominance is that males are commonly experience trauma and hence more likely to have secondary dissection[12,17,19,35,41] (Table 2).

Pathophysiology and risk factors

The underlying mechanism for CCAD involves tear or separation of the tunica intima layer of the artery because of the arterial injury, often in association with traumatic or vascular risk factors that predispose the artery to dissection, but can be spontaneous. The damage to the arterial endothelium results in collagen exposure and tissue factor activation leading to the platelet adhesion and aggregation, followed by fibrin and thrombus generation and deposition at the site of injury. Depending on the size and placement of the thrombus formation, an occlusion or stenosis at the site with or without thrombus dislodgement may occur resulting in AIS

TABLE 1.
Demographic and Risk Factor Data in Children With Craniocervical Arterial Dissection

Articles Analyzed	Total Cases	Gender		Average Age	Spontaneous Dissection or Cause Not Stated	Traumatic Dissection	Type of Trauma						
		Male	Female				Fall	Contact Sports	Not Reported	Neck Stretch or Hyperextension	Remote Trauma	Motor Vehicle Accident	Skull and/or Spine Fracture
Camacho et al., 2001[18]	5	2	3	10.8	0	5		5					
Cushing et al., 2001[24]	9	6	3	9.4	0	9		4	3	1	1		
Ganesan et al., 2002[13]	10	10	0	6.7	5	5		1	3	1			
Chabrier et al., 2003[12]	12	8	4	9.2	7	5			4	1			
Rafay et al., 2006[5]	16	8	8	9.5	8	8		6				2	
Salih et al., 2006[37]	3	2	1	3.8	0	3	2				1		
Tan et al., 2009[39]	13	10	3	7.5	8	5		2		2		1	
Lee et al., 2010[21]	9	7	2	10.7	5	4			2	2			
Mackay et al., 2010[34]	3	3	0	9.2	1	2			2				
Sepelyak et al., 2010[38]	3	3	0	9.6	0	3		3					
Songsaeng et al., 2010[20]	29	22	7	8.3	25	4		2	1	1			
Dlamini et al., 2011[40]	4	1	3	9.7	3	1	1						
Orman et al., 2014[36]	5	3	2	3.4	0	5	1	1	1			1	1
Rollins et al., 2013[16]	4	2	2	9	1	3			3				
Pandey et al., 2015[6]	42	24	18	14.8	8	34			21				13
McCrea et al., 2016[35]	15	13	2	6.5	9	6		0	6				
TOTAL cases	182	124	58	8.6	80	102	4	24	46	8	2	4	14
Percentages		68.2	31.8		44	56	4	23.5	45	7.8	2	4	13.7

TABLE 2.
Presenting Features of Children With Craniocervical Arterial Dissection

Case Studies	Total Cases	Hemiparesis	Facial Weakness	Headache	Neck Pain	Altered Consciousness	Vomiting	Dizzy	Seizure	Ataxia	Vision Change	Speech/Swallow Problem
Camacho et al., 2001[18]	5	4	2	4	0	0	0	1	0	2	1	2
Cushing et al., 2001[24]	9	6	6	6	1	0	3	3	0	5	3	2
Ganesan et al., 2002[13]	10	0	0	4	1	0	0	0	0	0	0	0
Chabrier et al., 2003[12]	12	12	0	9	0	8	0	0	3	0	0	0
Rafay et al., 2006[5]	16	9	2	9	1	4	3	2	2	2	4	6
Salih et al., 2006[37]	3	3	3	0	0	0	0	0	2	0	0	0
Tan et al., 2009[39]	13	8	0	6	2	6	4	2	2	3	2	1
Lee et al., 2010[21]	9	9	6	2	1	0	0	0	0	1	1	3
Mackay et al., 2010[34,^]	3											
Sepelyak et al., 2010[38]	3	3	2	0	0	0	1	0	0	2	1	1
Songsaeng et al., 2010[20]	29	10	0	14	3	0	3	4	1	2	4	0
Dlamini et al., 2011[40,≠]	4			3								
Orman et al., 2014[36]	5	2	0	0	0	1	0	0	1	0	0	1
Rollins et al., 2013[16]	4	1	0	3	0	0	0	1	2	1	2	1
Pandey et al., 2015[6]	42	13	0	4	0	5	0	0	0	1	2	3
McCrea et al., 2016[35,*]	15											
TOTAL cases	182	80	21	64	9	24	14	13	13	19	20	20

A "0" indicates that the clinical feature was either absent or else not reported in the case series.
^ No presenting features described.
≠ Focal neurological deficits were present in all four patients, but details not described.
* Presenting features combined with other causes of posterior circulation stroke.

or risk for AIS. A proportion of patients also develop hemorrhagic stroke, mainly subarachnoid hemorrhage, if there is complete tear of the vessel wall. Dissection of the artery within the cavernous sinus or near the vertebral venous plexus may result in an arteriovenous fistula or a perivascular extramural hematoma development.[19,42]

In our review of case studies, risk factor data for CCAD were available for all 182 cases (Table 1). Of these, 102 (56%) cases experienced concurrent or preceding trauma as the risk factor for dissection. Remote trauma was listed in 2% (n = 2) of cases. Some type of contact sport or physical activity before the onset of symptoms, including playing football, tennis or judo, diving, wrestling, bicycling, tobogganing, diving, or riding a roller coaster was reported in 24% (n = 25) of cases. Skull or spine fracture(s) was listed as a risk factor in 14%, stretches or hyperextension of the neck in 7%, fall in 4%, and a motor vehicle accident in 4% of cases. No information was available regarding the circumstances leading to the fractures, and severity or impact of motor vehicle injury (Table 1). Among the spontaneous dissection cases risk factors included one case hereditary exostosis,[35] one case of athetoid cerebral palsy and two cases of high homocysteine levels,[13] one case each with collagen vascular disease, factor IX deficiency, and trisomy 21,[42] and eight cases with aberrant vertebral arcuate foramina.[24]

The commonest site of arterial injury in CCAD is reported at the C1-C2 vertebral body level,[6,17,19,20,39,42] although any cervical arterial segments can be affected. Our review revealed similar results. The vertebral artery was involved at the C1-C2 level in up to 53% of extracranial dissection (ECD) cases,[6,17,19,39,42] whereas with internal carotid artery the most common location of injury was 2 to 3 cm above the carotid bulb (at the C1-C2 level).[5,18]

Certain regions are more susceptible to dissection, largely because of the anatomy and biomechanics of the cervical bony and arterial structures. The vertebral artery after its origin (V1, prevertebral segment) passes through the transverse foramina of the C6-C2 vertebrae (V2, cervical segment), takes a tortuous course from C2 to the suboccipital triangle between the C1 and the occiput, and then travels across the posterior arch of the atlas (V3, atlantoaxial segment) in proximity to the atlantoaxial (C1-C2) joint to enter the skull through the foramen magnum (V4, intracranial segment). The complex cervical spine movement, consisting of rotation and extension at the atlantoaxial junction coupled with lateral flexion predisposes the V3 segment to stretching and injury against the atlas or the atlantooccipital membrane as it courses through the C1-C2 vertebrae, resulting in narrowing of the artery lumen and slow and turbulent blood flow with a potential risk for developing an intra-arterial thrombus. Similarly, neck extension and lateral flexion can cause the C1 segment of the internal carotid artery to be stretched or compressed against the transverse process of C2 or C6 vertebra as it courses through the cervical spine or against the petrous bone as it enters the skull.[42] Furthermore, anatomically before entering the skull, proximal internal carotid artery travels close to oropharyngeal structures where it is predisposed to injury by direct oral trauma (e.g., injury from pencil or tonsillar region surgery).[11]

Types of craniocervical arterial dissection

CCAD can be categorized based on the location of dissection (extracranial versus intracranial), circulation involved (anterior versus posterior circulation), extent of injury (intramural versus transmural), presence or absence of associated head and neck trauma (traumatic versus spontaneous), and the acuteness of dissection (acute versus chronic).[17,19,20,36] Dependent on the aforementioned categorization, the etiopathogenesis, clinical presentation, and medical management of CCAD differ. ECD is common in children, accounting for 55% to 75% of CCAD cases.[19] Posterior circulation, mainly the vertebral artery, is more frequently affected.[17] Presentation is often preceded by head and neck trauma, headache, or neck pain[5,6,13,17,19]. Rarely, the extracranial cervical dissection may extend to involve the cephalic portion of the artery, less often with internal carotid artery possibly because of the restrictive anatomic course of the internal carotid artery within the petrous canal compared with vertebral artery in the large foramen magnum as these enter the skull.[42] Intracranial dissection (ICD), recently labeled as focal cerebral arteriopathy—dissection type, is noted in up to 25% of CCAD cases, although exact prevalence is likely not known owing to the neuroimaging challenges in differentiating it from other focal cerebral arteriopathies, mainly the focal cerebral arteriopathy—inflammatory type.[40,43,44] Contrary to ECD, anterior circulation (mainly internal carotid artery) is more commonly affected with ICD.[17,19,45]

The present review of childhood CCAD cases revealed results consistent with previously reported literature. The arterial dissection frequently involved posterior circulation (61%) and extracranial location (64%), with vertebral artery among the most commonly involved artery (60%). ICD accounted for 36% of cases. Some articles provided more detailed information about the specific artery and the side affected by dissection (Table 3). It appears that the left side dissected more commonly with the ICD; however, the small number of cases and the lack of consistent data across the studies do not allow for any meaningful conclusion.

Clinical presentation

The clinical features of CCAD are dependent on the location of the dissecting artery and the arterial territory affected by the ischemic infraction. Transient ischemic attacks and nonspecific symptoms preceding the index stroke event are common in children with CCAD.[5,12] It can also be identified on screening neuroimaging, such as with trauma.[17,19,38]

Children with anterior circulation dissection present typically with hemiparesis, and/or facial weakness, speech difficulties, and seizures, which are difficult to distinguish from stroke caused by other etiologies. In children with posterior circulation involvement, nonspecific clinical features, such as headache, dizziness, vomiting, altered consciousness, gait unsteadiness, and double vision,[5,6,17,19,21,38] are noted. However, a prominent headache and neck pain, tinnitus, and Horner syndrome (triad of miosis, ptosis, and anhidrosis) are considered specific presentations of posterior circulation dissection.[5,6,17,19,21,38] The present review revealed similar results, with hemiparesis (80/160, 50%) and headache (64/164, 39%) as the two most common presenting features of CCAD in children. A small proportion ($n = 9$) of the CCAD patients had neck pain, confirming the previously noted difference in clinical presentation of children with CCAD when compared with adults (who commonly present with neck pain).[17,19] The next most common presenting symptom was alteration in consciousness, followed by speech and swallowing difficulties and ataxia. Seizures occurred in only 13 children with CCAD and more often affected younger children (Table 2).

Diagnosis

The diagnosis of CCAD is based on clinical suspicion and the demonstration of specific neurovascular imaging findings, which require use of specific neuroimaging modalities and techniques. Furthermore, a personal and family history of comorbid diagnoses, such as connective tissue disease, also raises suspicion for CCAD. In addition, in otherwise healthy children with AIS involving posterior circulation dissection is among the commonest cause of AIS, and hence must be strongly suspected in cases with a clinical presentation consistent with posterior circulation AIS.[5,17,19,41]

On the basis of the recent CASCADE (Childhood AIS Standardized Classification and Diagnostic Evaluation) classification for stroke subtypes in children with AIS,[46] the International Pediatric Stroke study confirms the diagnosis of CCAD when one of the following three features is noted[47]:

1. Angiographic findings of a double lumen (a true and a false lumen created by an elongated arterial tear), intimal flap (a flap created by the tearing of vascular intimal layer), or pseudoaneurysm (blood contained perivascular cavity because of the leakage of blood from the injured artery into the surrounding perivascular tissues with a persistent direct communication with the artery), or on axial T1 fat-saturated MRI demonstration of intramural hematoma visualized as a “bright crescent sign” in the arterial wall (blood within the false lumen surrounded by a crescent-shaped intramural thrombus with or without annular enhancement of the *vasa vasorum*).
2. The presence of cervical or cranial trauma, neck pain, or headache less than six weeks preceding angiographic findings of segmental stenosis or occlusion of the cervical arteries.
3. Angiographic findings of segmental stenosis or occlusion of the vertebral artery at the level of the C2 vertebral body, even without known history of craniocervical trauma.

In children, published literature consists of a few case series that include information on specific diagnostic imaging findings of dissection. Furthermore, even when the neurovascular features are reported, details are not always available for each case in the series (Table 3). Double lumen (barrel gun sign) and intimal flap, although very specific luminal features of CCAD, are often difficult to visualize. Adult CCAD literature reports double barrel lumen and intimal flap in approximately 10% of cases, often with internal carotid artery dissection.[48-51] In pediatric literature the presence of double lumen appears to be one of the least frequently reported finding in children with CCAD,[40,48,49] whereas an intimal flap on neurovascular imaging was found most frequently in up to 20% of CCAD cases,[5,35,36] except in one pediatric case series where 46% demonstrated evidence of an intimal flap on conventional catheter angiography[39] (Table 3). Similar to adults, pseudoaneurysm was identified on vascular imaging in 7.6% to 33% of childhood CCAD cases.[5,12,13,18,35,39,45,48,51] Bright crescent sign, a commonly reported neurovascular imaging feature of CCAD in adults (seen in 76% to 91% of cases),[49,52] was not as frequently reported in children, seen in 6% to 40 % of pediatric CCAD cases.[5,35,36,39] Other arterial abnormalities indicative of stenosis or occlusion on neurovascular imaging include (1) focal narrowing with distal dilatation (*string and pearl sign*), (2) segmental smooth long tapering stenosis (*string sign*), or (3) short smooth tapering stenosis (*flame or rat tail* sign) (Figure). These arterial abnormalities are common findings in both adult and pediatric cases of CCAD, with over one third of both adult[45,48,51] and pediatric cases demonstrating one or more of these abnormalities (Table 3). However, these arterial abnormalities are only considered diagnostic of dissection when coupled with a preceding history of head and neck trauma or neck pain, or when located at the level of C2 vertebral body even without known history of head and neck trauma.

For demonstration of ischemic changes, computed tomography (CT) and MRI are the available neuroimaging modalities. Diffusion-weighted MRI images remain the most powerful brain parenchymal technique compared with CT.[3,19] In children, although not always readily available, MRI is considered as the preferred imaging modality for demonstration of parenchymal findings, because of the existence of several ischemic stroke mimics that are common than stroke.[2,53] Furthermore, CT is known to miss or underestimate AIS in at least 15% of children with AIS.[2,3] Dedicated neurovascular imaging of both cranial and cervical regions, including certain specific imaging techniques, is required to make or refute a diagnosis of craniocervical dissection with certainty. However, these imaging sequences, in particular cervical imaging, are not always included as part of the initial neuroimaging study obtained for acute stroke presentation. Even if the cervical imaging is performed, the images obtained may not be optimal to visualize the arterial segments at the C1-C2 location where most dissections tend to occur.

In the current cohort, MRI and magnetic resonance angiogram (MRA) of the brain and neck appeared to be the preferred neurovascular imaging modality, followed by invasive cerebral catheter

TABLE 3.
Neurovascular Imaging Features of Craniocervical Arterial Dissection in Children

Case Studies	Total Cases	Posterior/ Anterior Dissection	ECD/ICD	Vertebral Artery		Internal Carotid Artery		Basilar Artery	Angiographic Findings			
				Left	Right	Left	Right		DUS	CTA	MRA	DSA
Camacho et al., 2001[18]	5	1/4	4/1		1	2	2		4 DUS 3 Normal 1 Thrombus		4 MRA 2 Intramural clot 1 String sign 1 Thrombotic occlusion	2 DSA 1 Flame occlusion at C2 Others no details
Cushing et al., 2001[24,*]	9	9/0	8/1	4	5					1 Intimal flap, 1 pseudoaneurysm, 7 arterial abnormalities/occlusion at C1-C2 level		
Ganesan et al., 2002[13,†]	10	10/0	NA	NA	NA	NA	NA				10 MRA 2 Intramural hematoma and arterial abnormalities/ occlusion at C1-C2 level 8 Nondiagnostic abnormalities	8 DSA 5 Aneurysmal dilatation 3 Tapering stenosis/ occlusion, often at C1-C2
Chabrier et al., 2003[12]	12	2/10	5/7	NA	NA	NA	NA				12 MRA ECD: 2 elongated occlusion, 1 string sign, 2 short tapering stenosis, and aneurysmal dilatation ICD: 7 elongated and irregular tapering stenosis or occlusion	
Rafay et al., 2006[5]	16	7/9	12/4	5	3	4	5				11 MRA 9 Occlusion (2 at C1-C3) 2 Irregular stenosis at C1-C2	16 DSA 2 Pseudoaneurysm 3 intimal flap 1 String sign/tapering stenosis 9 Irregular stenosis/ occlusion at C1-C3
Salih et al., 2006[37]	3	0/3	3/0			1	1		2 DUS 1 Occlusion 1 Normal		3 MRA 2 Stenosis/occlusion 1 Nondiagnostic	
Tan et al., 2009[39]	13	9/4	10/3	6	2	2	2				5 MRA 2 Occlusion (1 at C1-C2) 2 Segmental narrowing (1 at C1-C2) 1 Mural hematoma	13 DSA 1 Pseudoaneurysm 6 Intimal flap 1 String sign 1 Segmental stenosis at C1-C3 2 Occlusion at C1-C2 3 Mural hematoma
Lee et al., 2010[21,‡]	9	3/6	9/1	2	0	4	2	1			8 MRA 1 Eccentric arterial enhancement Others no details	6 DSA 2 Dissecting aneurysms in one case Others no details
Mackay et al., 2010[34]	3	3/0	3/0	1	2	NA	NA				3 MRA 1 Stenosis/occlusion 2 Normal	2 DSA 1 Pseudoaneurysm 1 Dissection Others no details
Sepelyak et al., 2010[38]	3	2/1	2/0			NA	NA			1 CTA: normal	2 MRA: normal	2 DSA 1 Normal 1 Occlusion with intraluminal clot at C1-C2
Songsaeng et al., 2010[20]	29	29/0	11/18	10	13	NA	NA	9			16 Stenosis: 11 segmental stenosis (8 at C2-C3), 1 pearl, and string sign. Four tapering stenosis, 1 double lumen, 14 fusiform, and 2 saccular aneurysm, 1 intraluminal clot, 1 eccentric enhancement	
Dlamini et al., 2011[40,§]	4	0/4	0/4	0	0	2	2				4 MRA 3 Irregular stenosis or occlusion 1 Intramural hematoma	1 DSA: contrast filling defect suggestive of recanalizing thrombus or double lumen
Orman et al., 2014[36]	5	0/5	2/3			4	1			2 CTA 2 Tapering stenosis	5 MRA 1 Eccentric arterial wall enhancement 1 Intimal flap 3 Intraluminal clot with occlusion 5 Narrowing/occlusion	

(*continued on next page*)

TABLE 3. (*continued*)

Case Studies	Total Cases	Posterior/ Anterior Dissection	ECD/ICD	Vertebral Artery		Internal Carotid Artery		Basilar Artery	Angiographic Findings			
				Left	Right	Left	Right		DUS	CTA	MRA	DSA
Rollins et al., 2013[16,‖]	4	4/0	NA	0	0	¶	¶				4 MRA 1 irregular stenosis Others no details	4 DSA Vertebral artery dissection, no details provided
Pandey et al., 2015[6,*]	42	17/25	23/19[#]	¶	¶	¶	¶			4 Stenosis, 17 occlusion (10 internal carotid and 7 vertebral artery), 1 bilateral internal carotid and a vertebral artery occlusion, 9 pseudoaneurysms		
McCrea et al., 2016[35,‖]	15	15/0	15/0	NA	NA	NA	NA			1 CTA: details not available	15 MRA CCAD diagnosed in 6 as per CASCADE 1 V2-V4 irregularity	6 DSA 1 Intimal flap 1 Mural thrombus 1 V3 irregularity
TOTAL cases	182	111/71	107/61	28	26	13	7	10				

Abbreviations:
CTA = Computed tomography angiogram
DSA = Digital subtraction catheter angiogram
DUS = Doppler ultrasound
ECD =Extracranial dissection
ICD = Intracranial dissection
MRA = Magnetic resonance angiogram
NA = Not available
[*] Method of angiography not described in all.
[†] Limited details to classify as extra or intracranial dissection. One patient had more than one dissection in this series.
[‡] All with stenosis or occlusion at C1-C2, except one internal carotid artery occlusion at C3 without trauma.
[§] Three diagnosed on postmortem studies.
[‖] Case study with limited vascular imaging details available, dissection diagnosed as per CASCADE.
[¶] Vertebral artery dissection, side not specified.
[#] Numbers are estimated based on details provided.

angiography. One case series published since the development of recent stroke subtype criteria[35] used the CASCADE criteria for confirming a diagnosis of CCAD.[43,46] Only two case series reported using Doppler ultrasound of the neck, with both studies reporting negative results in the presence of positive findings on either cerebral MRA or digitial subtraction catheter angiography (DSA).[18,37] Interestingly, although CT was the initial brain imaging modality in a majority, CT tomography angiogram (CTA) was not included with the initial imaging and was only reported to be completed in three of 16 case studies (18.7%).[35,36,38] It is possible that in a few case studies CTA was done but results were not reported. In all case studies, MRI and MRA of the brain were completed as the preferred neurovascular imaging modality, both initially and after the initial nonenhanced cranial CT. Cerebral DSA was reported in all except two case studies (although it may have been performed, but not reported in the case article),[36,54] as the gold standard definitive neurovascular imaging modality, especially when the initial vascular imaging revealed nondiagnostic findings for arterial dissection.

The following section describes the available neurovascular imaging methods for demonstrating the diagnostic features of CCAD in children. A comparison of CTA, MRA, and DSA is presented in Table 4.

Color Doppler ultrasound

Although an inexpensive and radiation-free bedside imaging modality, color Doppler ultrasound has several challenges, mainly because of its huge reliance on the operator and radiographer. In adults, it is reported to be helpful in recognizing and monitoring evolution of dissection, with wide variation in reported sensitivities, ranging from 66% to 96%.[55-58] In children, Doppler ultrasound is not a favored cervical imaging modality because of the challenges related to the pediatric small neck size, lack of experience for evaluating dissected arteries, and the presence of little, if any, systematic data for its utility.[18,19]

CT and CT angiogram

Over the last decade, both precontrast and postcontrast enhanced craniocervical CT and CTA has become a quite sensitive imaging method in adults, in particular a multidetector and multilayer CT and CTA. The technique provides thinner sections than regular CT, with high spatial resolution images and ability to create multiplanar and volume reconstructs.[33,52] In adult CCAD cases, the multidetector CT and CTA reportedly has more than 98% sensitivity and specificity, when compared with Doppler ultrasound,[33] MRI and MRA,[52,59] and cerebral catheter angiogram.[60] It has the benefit of rapid imaging time (often obviating the need for sedation), fewer contraindications, and wide and rapid availability as part of the trauma protocol, especially during after-hours. However, CT has the drawback of radiation exposure (higher with newer techniques) and a lack of imaging studies using multidetector CT and CTA in children.

MRI and MR angiogram

Compared with CT, precontrast and postcontrast enhanced MRI and MRA of the head and neck has remarkably high resolution and sensitivity for parenchymal and vascular imaging[61,62] and hence greater ability to accurately diagnose both stroke and dissection simultaneously. However, specific MRI sequences must be obtained for demonstration of vascular findings specific to dissection. Gadolinium-enhanced MRI and MRA is reportedly superior then nonenhanced MRI and time of

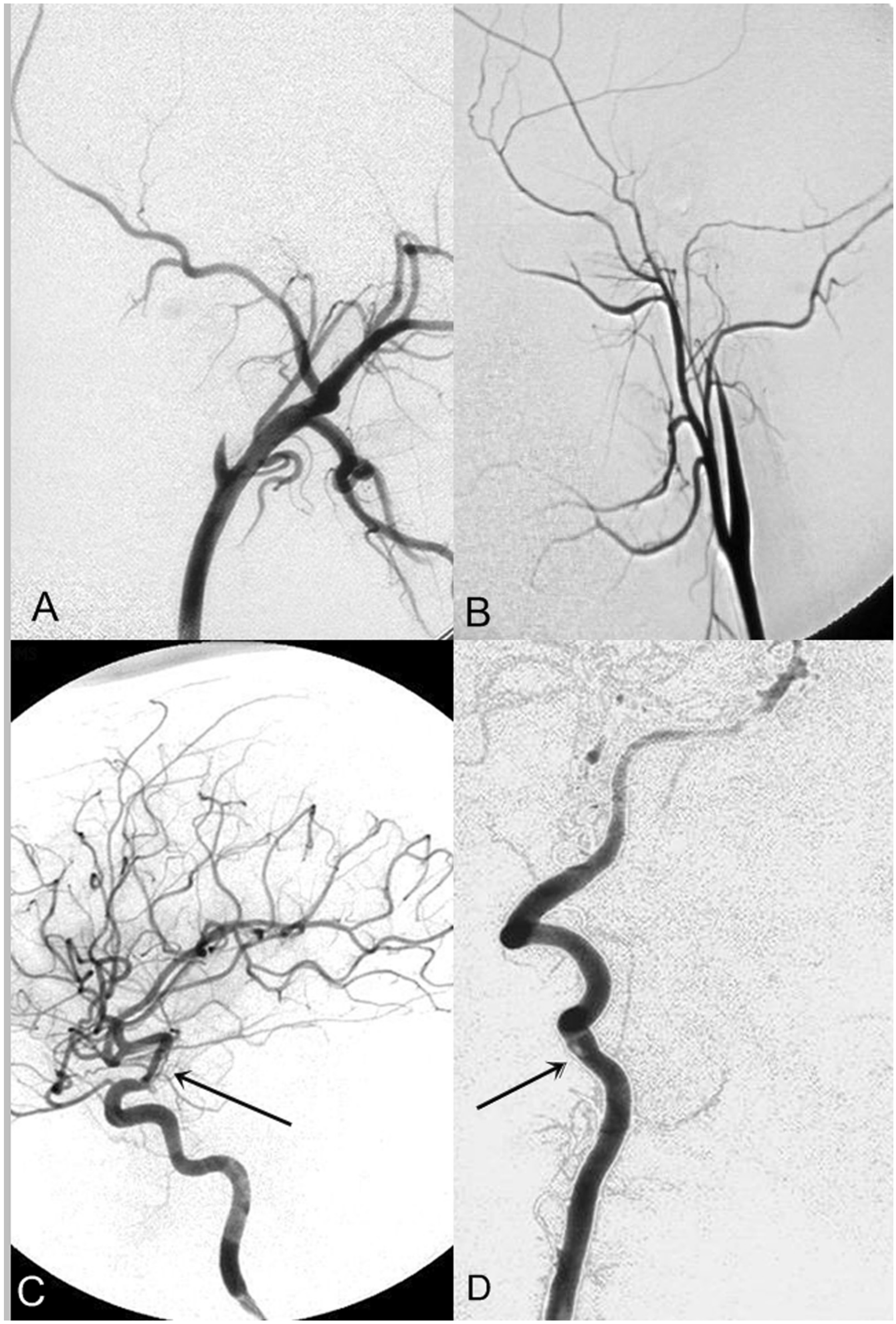

FIGURE. Radiological features of craniocervical arterial dissection in children. (A) Cerebral catheter angiography in a 13-year-old girl with motor vehicle accident and right middle cerebral artery territory infarction and evidence of short tapering stenosis (rat tail sign) in the right proximal internal carotid artery; (B) 13-year-old girl with left middle cerebral artery territory infarction showing tapering stenosis (string sign) of the left proximal internal carotid artery on cerebral catheter angiography; (C) nine-year-old boy with traumatic intracranial dissection of left internal carotid artery and evidence of luminal irregularity and a filling defect (suggestive of intramural flap or clot) in the supraclinoid portion of the left distal internal carotid artery on cerebral catheter angiography (arrow); and (D) cerebral catheter angiography in a nine-year-old boy with spontaneous dissection of the right vertebral artery demonstrating right vertebral artery luminal irregularity and aneurysmal dilatation (arrow) at the level of C2-C3 vertebral bodies.

flight MRA in both children and adults (detection rate 89% versus 50%).[39,63] High-resolution, axial fat-saturated T1 MRI brain imaging sequence is highly sensitive and is currently the best MRI technique for detecting intramural arterial clot. Images generated from this technique have the ability to demonstrate varying stages of aging blood clot.[49,52,64] Another evolving new

TABLE 4.
Comparison of Neurovascular Imaging Modalities

Comparative Features	CTA	MRA	DSA
Overall sensitivity	75-100%	89-100%	98-100%
Overall specificity	50-100%	29-100%	98-100%
Scope	Parenchyma, vessel lumen, and wall imaging	Parenchyma, vessel lumen, and wall imaging	Luminogram only
Findings			
Ischemic changes	75-85%	100%	NA
Intramural hematoma “bright crescent sign”	60-98%	98-100%	0%
Double lumen, intimal flap	90-100%	Up to 89%	98-100%
String and pearl sign/ pseudoaneurysm	Up to 98%	Up to 98%	98-100%
Short or long tapering stenosis/ occlusion/enlargement of the vessel	98-100%	98-100%	98-100%
Disadvantages/issues	High radiation burden Timely contrast bolus/high injection rate Contrast related contraindications Multidetector/multiplanar CTA not studied well in children	Need for sedation Lack of rapid availability at night Presence of few magnet related contraindications	Invasive/need for sedation Potential for complications in 1% radiation exposure Contrast related contraindications

Abbreviations:
CTA = Computed tomography angiogram
DSA = Digital subtraction catheter angiogram
MRA = Magnetic resonance angiogram

MRI technique is the arterial wall imaging, which has higher sensitivity than CT and uses both pre-gadolinium and post-gadolinium–enhanced, high quality images in multiplanar 2D and 3D, T1, and fluid-attenuated inversion recovery fast-spin echo sequences.[65-67]

Digital subtraction catheter angiography

Cerebral DSA is mainly a vascular luminogram that provides high-resolution images of vascular lumen and contour, and does not provide any brain parenchymal and perivascular tissue information. It is superior to CT and MRI in demonstrating intraluminal arterial findings, and hence remains a gold standard test, especially in patients with nondiagnostic CTA and MRA.[19,68] DSA does not demonstrate thickness and contents of the arterial wall, which are required to demonstrate specific mural signs of dissection. In addition, it is an invasive procedure with risk for complications.[19,69]

Treatment and follow-up

Evolution of arterial abnormalities over time and high stroke recurrence rates are two main reasons for the treatment and close

TABLE 5.
Treatment of Children With Craniocervical Arterial Dissection

Case Studies	Total Cases	No Treatment	Anticoagulant Agent (Heparin/ Warfarin)	Antiplatelet Agent (Aspirin)	Antithrombotic Treatment Not Specified	Surgical Intervention*
Camacho et al., 2001[18,†]	5		3	2		
Cushing et al., 2001[24,‡]	9			6		4
Ganesan et al., 2002[13]	10	1	9			
Chabrier et al., 2003[12]	12	1	2	9		
Rafay et al., 2006[5]	16	1	14	1		
Salih et al., 2006[37,§]	3					
Tan et al., 2009[39,§]	13					
Lee et al., 2010[21]	9	2	1	5		1
Mackay et al., 2010[34,§]	3					
Sepelyak et al., 2010[38]	3		3			
Songsaeng et al., 2010[20]	29	2			18	9
Dlamini et al., 2011[40]	4				4	
Orman et al., 2014[36,‡]	5	2		2		
Rollins et al., 2013[16]	4				4	1
Pandey et al., 2015[6]	42	11			22	9
McCrea et al., 2016[35,‖]	15	1	12	14		
TOTAL cases	182	20	43	39	48	24

* Endovascular occlusion or stent placement, aneurysm repair or coiling, and vertebral arcuate foramina repair.
† Recombinant prourokinase in 1.
‡ One case treated with both anticoagulant and antiplatelet agents.
§ No treatment listed in the article.
‖ Numbers do not add up in the article to 15 cases—details not available. Six cases received both anticoagulation and antiplatelet agents after stroke recurrence.

clinical and neurovascular imaging follow-up of children with CCAD.

Development of dissection specific of vessel wall abnormalities, such as pseudoaneurysm or segmental arterial dilatation, is not always evident on initial vascular imaging and is only visualized on subsequent imaging.[5,31] In addition, dissected and thrombosed artery heals over time and recanalizes, either partially or completely, in two thirds of childhood CCAD cases.[5,31] Arterial dissection may also recur in 1% of cases, especially in whom there is a personal or family history of connective tissue disorders or arterial dissection.[11,17,42] As a result, patients with CCAD are at increased risk for ischemic stroke recurrence. Recurrent cerebral ischemic events, after the index stroke or dissection, are reported in 12% to 19% of childhood CCAD cases.[3,11,12,28,30,39] Therefore follow-up neurovascular imaging is recommended to be performed within 3 to 6 months after the initial presentation in both adult[42] and childhood cases of CCAD,[11] specifically when initial vascular imaging revealed nondiagnostic results.

Antithrombotic treatment of patients with AIS due to arterial dissection has long been subject to controversy. The reason for this is evident from the current literature review, which clearly indicates a lack of a consistent approach in treating children with CCAD (Table 5). The major challenge is related to the underlying pathophysiological mechanisms that involve arterial tear and development of a fibrin-rich thrombus, thereby making anticoagulation a more appropriate antithrombotic treatment to choose in patients with CCAD, specifically in children with healthy arteries before dissection. A pilot study in adults, the Carotid Artery Dissection in Stroke study, demonstrated no difference in the efficacy of either antiplatelet or anticoagulant drugs in patients with AIS because of cervical dissection, but the stroke recurrence was rare in both treatment groups.[70] A slight increase in hemorrhages was noted in the group treated with anticoagulant compared with antiplatelet therapy. This trial led the adult stroke experts to conclude that antiplatelet treatment is safer, more convenient, and less costly default treatment for CCAD, but efforts to understand the pathophysiology of dissection and to better characterize the patients who carry a greater risk for stroke recurrence should be continued.[71]

Considering these challenges, the 2018 American Heart Association and American Stroke Association management guidelines for adult patients with AIS recommended that for extracranial carotid or vertebral arterial dissection either approach is reasonable for three to six months (class IIb, evidence level B-R), leaving both options open for the treating physicians.[72] The American Heart Association and American Stroke Association management guidelines for the management of children with AIS were last published in 2008.[11] According to these guidelines, it is reasonable to begin anticoagulation with either intravenous unfractionated or subcutaneous low-molecular-weight heparin as a bridge to oral anticoagulation for a period of three to six months in children with ECD (class IIa, evidence level C). Treatment with anticoagulation is not recommended in children with ICD (class III, evidence level C).[11] However, this recommendation requires further discussion in light of the recent adult Carotid Artery Dissection in Stroke study trial.[70] All guidelines recommend institution of acute neuroprotective measures and age-appropriate rehabilitation and therapy programs (class I, evidence level C).[11,72]

Conclusions

The diagnosis of CCAD requires high index of suspicion along with consideration for both personal and familial risk factors, including acquired or inherited predisposition and bony and vascular anatomic susceptibility. Furthermore, use of specific and sensitive neurovascular imaging techniques is paramount in making a timely and accurate diagnosis of CCAD. Although CT remains the preferred first line imaging modality in many pediatric centers, MRI appears superior to CT in demonstrating ischemic infarction and vessel wall abnormalities in children with CCAD. With advancements in arterial wall imaging, MRI may supersede and eventually replace invasive cerebral catheter angiography, which is currently considered a gold standard vascular imaging modality in children.

References

1. Schievink WI, Mokri B, Whisnant JP. Internal carotid artery dissection in a community. Rochester, Minnesota, 1987-1992. Stroke. 1993;24:1678–1680.
2. Gabis LV, Yangala R, Lenn NJ. Time lag to diagnosis of stroke in children. Pediatrics. 2002;110:924–928.
3. Rafay MF, Pontigon AM, Chiang J, et al. Delay to diagnosis in acute pediatric arterial ischemic stroke. Stroke. 2009;40:58–64.
4. Wintermark M, Hills NK, deVeber GA, et al. Arteriopathy diagnosis in childhood arterial ischemic stroke: results of the vascular effects of infection in pediatric stroke study. Stroke. 2014;45:3597–3605.
5. Rafay MF, Armstrong D, deVeber G, Domi T, Chan A, MacGregor DL. Craniocervical arterial dissection in children: clinical and radiographic presentation and outcome. J Child Neurol. 2006;21:8–16.
6. Pandey AS, Hill E, Al-Holou WN, et al. Management of pediatric craniocervical arterial dissections. Childs Nerv Syst. 2015;31:101–107.
7. Mackay MT, Wiznitzer M, Benedict SL, Lee KJ, deVeber GA, Ganesan V. Arterial ischemic stroke risk factors: the International Pediatric Stroke Study. Ann Neurol. 2011;69:130–140.
8. deVeber G, Kirton A, Booth FA, et al. Epidemiology and outcomes of arterial ischemic stroke in children: the Canadian Pediatric Ischemic Stroke Registry. Pediatr Neurol. 2017;69:58–70.
9. Giroud M, Lemesle M, Gouyon JB, Nivelon JL, Milan C, Dumas R. Cerebrovascular disease in children under 16 years of age in the city of Dijon, France: a study of incidence and clinical features from 1985 to 1993. J Clin Epidemiol. 1995;48:1343–1348.
10. Lynch JK, Hirtz DG, deVeber G, Nelson KB. Report of the National Institute of Neurological Disorders and Stroke workshop on perinatal and childhood stroke. Pediatrics. 2002;109:116–123.
11. Roach ES, Golomb MR, Adams R, et al. Management of stroke in infants and children: a scientific statement from a Special Writing Group of the American Heart Association Stroke Council and the Council on Cardiovascular Disease in the Young. Stroke. 2008;39:2644–2691.
12. Chabrier S, Lasjaunias P, Husson B, Landrieu P, Tardieu M. Ischaemic stroke from dissection of the craniocervical arteries in childhood: report of 12 patients. Eur J Paediatr Neurol. 2003;7:39–42.
13. Ganesan V, Chong WK, Cox TC, Chawda SJ, Prengler M, Kirkham FJ. Posterior circulation stroke in childhood: risk factors and recurrence. Neurology. 2002;59:1552–1556.
14. Schneidereit NP, Simons R, Nicolaou S, et al. Utility of screening for blunt vascular neck injuries with computed tomographic angiography. J Trauma. 2006;60:209–215.
15. Berne JD, Reuland KS, Villarreal DH, McGovern TM, Rowe SA, Norwood SH. Sixteen-slice multi-detector computed tomographic angiography improves the accuracy of screening for blunt cerebrovascular injury. J Trauma. 2006;60:1204–1209.
16. Rollins N, Pride GL, Plumb PA, Dowling MM. Brainstem strokes in children: an 11-year series from a tertiary pediatric center. Pediatr Neurol. 2013;49:458–464.
17. Fullerton HJ, Johnston SC, Smith WS. Arterial dissection and stroke in children. Neurology. 2001;57:1155–1160.
18. Camacho A, Villarejo A, de Aragon AM, Simon R, Mateos F. Spontaneous carotid and vertebral artery dissection in children. Pediatr Neurol. 2001;25:250–253.
19. Stence NV, Fenton LZ, Goldenberg NA, Armstrong-Wells J, Bernard TJ. Craniocervical arterial dissection in children: diagnosis and treatment. Curr Treat Options Neurol. 2011;13:636–648.
20. Songsaeng D, Srivatanakul K, Krings T, Geibprasert S, Ozanne A, Lasjaunias P. Symptomatic spontaneous vertebrobasilar dissections in children: review of 29 consecutive cases. J Neurosurg Pediatr. 2010;6:233–243.
21. Lee YY, Lin KL, Wang HS, et al. Craniocervical arterial dissection: a cause of childhood arterial ischemic stroke in Taiwan. J Formos Med Assoc. 2010;109:156–162.
22. Brandt T, Orberk E, Weber R, et al. Pathogenesis of cervical artery dissections: association with connective tissue abnormalities. Neurology. 2001;57:24–30.
23. Brandt T, Grond-Ginsbach C. Spontaneous cervical artery dissection: from risk factors toward pathogenesis. Stroke. 2002;33:657–658.
24. Cushing KE, Ramesh V, Gardner-Medwin D, et al. Tethering of the vertebral artery in the congenital arcuate foramen of the atlas vertebra: a possible cause

of vertebral artery dissection in children. Dev Med Child Neurol. 2001;43: 491–496.
25. Brandt T, Hausser I, Orberk E, et al. Ultrastructural connective tissue abnormalities in patients with spontaneous cervicocerebral artery dissections. Ann Neurol. 1998;44:281–285.
26. Hausser I, Muller U, Engelter S, et al. Different types of connective tissue alterations associated with cervical artery dissections. Acta Neuropathol. 2004;107:509–514.
27. Wei F, Diedrich KT, Fullerton HJ, et al. Arterial tortuosity: an imaging biomarker of childhood stroke pathogenesis? Stroke. 2016;47:1265–1270.
28. Amlie-Lefond C, Bernard TJ, Sebire G, et al. Predictors of cerebral arteriopathy in children with arterial ischemic stroke: results of the International Pediatric Stroke Study. Circulation. 2009;119:1417–1423.
29. Frosk P, Salman MS, Wrogemann J, Shah N, Rafay MF. Recurrent posterior circulation stroke in an infant with basilar artery aneurysm. J Child Neurol. 2009;24:1019–1020.
30. Fullerton HJ, Wintermark M, Hills NK, et al. Risk of recurrent arterial ischemic stroke in childhood: a Prospective International Study. Stroke. 2016;47:53–59.
31. Tan MA, Armstrong D, MacGregor DL, Kirton A. Late complications of vertebral artery dissection in children: pseudoaneurysm, thrombosis, and recurrent stroke. J Child Neurol. 2009;24:354–360.
32. Fluss J, Garcia-Tarodo S, Granier M, et al. Perinatal arterial ischemic stroke related to carotid artery occlusion. Eur J Paediatr Neurol. 2016;20:639–648.
33. Pugliese F, Alberghina F, Meijboom WB, Malago R, Gopalan D, de Feyter PJ. Post-processing using multislice computed tomography coronary angiography improves image interpretability in patients with fast heart rates and heart-rate variations. J Cardiovasc Med (Hagerstown). 2007;8:1088–1090.
34. Mackay MT, Prabhu SP, Coleman L. Childhood posterior circulation arterial ischemic stroke. Stroke. 2010;41:2201–2209.
35. Mccrea N, Saunders D, Bagkeri E, Chitre M, Ganesan V. Diagnosis of vertebral artery dissection in childhood posterior circulation arterial ischaemic stroke. Dev Med Child Neurol. 2016;58:63–70.
36. Orman G, Tekes A, Poretti A, Robertson C, Huisman TA. Posttraumatic carotid artery dissection in children: not to be missed!. J Neuroimaging. 2014;24: 467–472.
37. Salih MA, Al-Jarallah AA, Al-Salman MM, Alorainy IA, Hassan HH. Stroke from cervicocephalic arterial dissection in Saudi children. Saudi Med J. 2006;27 Suppl 1:S103–S107.
38. Sepelyak K, Gailloud P, Jordan LC. Athletics, minor trauma, and pediatric arterial ischemic stroke. Eur J Pediatr. 2010;169:557–562.
39. Tan MA, deVeber G, Kirton A, Vidarsson L, MacGregor D, Shroff M. Low detection rate of craniocervical arterial dissection in children using time-of-flight magnetic resonance angiography: causes and strategies to improve diagnosis. J Child Neurol. 2009;24:1250–1257.
40. Dlamini N, Freeman JL, Mackay MT, et al. Intracranial dissection mimicking transient cerebral arteriopathy in childhood arterial ischemic stroke. J Child Neurol. 2011;26:1203–1206.
41. Amlie-Lefond C. Dissecting etiologies of posterior circulation stroke. Dev Med Child Neurol. 2016;58:10–11.
42. Biller J, Sacco RL, Albuquerque FC, et al. Cervical arterial dissections and association with cervical manipulative therapy: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45:3155–3174.
43. Bernard TJ, Manco-Johnson MJ, Lo W, et al. Towards a consensus-based classification of childhood arterial ischemic stroke. Stroke. 2012;43:371–377.
44. Tolani AT, Yeom KW, Elbers J. Focal cerebral arteriopathy: the face with many names. Pediatr Neurol. 2015;53:247–252.
45. Schievink WI, Mokri B, Piepgras DG. Spontaneous dissections of cervicocephalic arteries in childhood and adolescence. Neurology. 1994;44:1607–1612.
46. Bernard TJ, Beslow LA, Manco-Johnson MJ, et al. Inter-rater reliability of the CASCADE criteria: challenges in classifying arteriopathies. Stroke. 2016;47: 2443–2449.
47. Internet Communication Accessed November 30 2h, clinicaltrials. Database for Stroke and Infants; 2018 gov/ct2/show/NCT00084292?cond=pediatric+stroke&rank=5.
48. Houser OW, Mokri B, Sundt Jr TM, Baker Jr HL, Reese DF. Spontaneous cervical cephalic arterial dissection and its residuum: angiographic spectrum. AJNR Am J Neuroradiol. 1984;5:27–34.
49. Oelerich M, Stogbauer F, Kurlemann G, Schul C, Schuierer G. Craniocervical artery dissection: MR imaging and MR angiographic findings. Eur Radiol. 1999;9:1385–1391.
50. Rodallec MH, Marteau V, Gerber S, Desmottes L, Zins M. Craniocervical arterial dissection: spectrum of imaging findings and differential diagnosis. Radiographics. 2008;28:1711–1728.
51. Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med. 2001;344:898–906.
52. Vertinsky AT, Schwartz NE, Fischbein NJ, Rosenberg J, Albers GW, Zaharchuk G. Comparison of multidetector CT angiography and MR imaging of cervical artery dissection. AJNR Am J Neuroradiol. 2008;29:1753–1760.
53. Shellhaas RA, Smith SE, O'Tool E, Licht DJ, Ichord RN. Mimics of childhood stroke: characteristics of a prospective cohort. Pediatrics. 2006;118: 704–709.
54. Chabrier S, Sebire G, Fluss J. Transient cerebral arteriopathy, postvaricella arteriopathy, and focal cerebral arteriopathy or the unique susceptibility of the M1 segment in children with stroke. Stroke. 2016;47:2439–2441.
55. Arnold M, Baumgartner RW, Stapf C, et al. Ultrasound diagnosis of spontaneous carotid dissection with isolated Horner syndrome. Stroke. 2008;39:82–86.
56. Benninger DH, Georgiadis D, Gandjour J, Baumgartner RW. Accuracy of color duplex ultrasound diagnosis of spontaneous carotid dissection causing ischemia. Stroke. 2006;37:377–381.
57. Dittrich R, Dziewas R, Ritter MA, et al. Negative ultrasound findings in patients with cervical artery dissection. Negative ultrasound in CAD. J Neurol. 2006;253: 424–433.
58. Pugliese F, Crusco F, Cardaioli G, et al. CT angiography versus colour-Doppler US in acute dissection of the vertebral artery. Radiol Med. 2007;112:435–443.
59. Elijovich L, Kazmi K, Gauvrit JY, Law M. The emerging role of multidetector row CT angiography in the diagnosis of cervical arterial dissection: preliminary study. Neuroradiology. 2006;48:606–612.
60. Chen CJ, Tseng YC, Lee TH, Hsu HL, See LC. Multisection CT angiography compared with catheter angiography in diagnosing vertebral artery dissection. AJNR Am J Neuroradiol. 2004;25:769–774.
61. Hanning U, Sporns PB, Schmiedel M, et al. CT versus MR techniques in the detection of cervical artery dissection. J Neuroimaging. 2017;27:607–612.
62. Chalela JA, Kidwell CS, Nentwich LM, et al. Magnetic resonance imaging and computed tomography in emergency assessment of patients with suspected acute stroke: a prospective comparison. Lancet. 2007;369:293–298.
63. Rizzo L, Crasto SG, Savio D, et al. Dissection of cervicocephalic arteries: early diagnosis and follow-up with magnetic resonance imaging. Emerg Radiol. 2006;12:254–265.
64. Levy C, Laissy JP, Raveau V, et al. Carotid and vertebral artery dissections: three-dimensional time-of-flight MR angiography and MR imaging versus conventional angiography. Radiology. 1994;190:97–103.
65. Dlamini N, Yau I, Muthusami P, et al. Arterial wall imaging in pediatric stroke. Stroke. 2018l;49:891–898.
66. Mandell DM, Han JS, Poublanc J, et al. Quantitative measurement of cerebrovascular reactivity by blood oxygen level-dependent MR imaging in patients with intracranial stenosis: preoperative cerebrovascular reactivity predicts the effect of extracranial-intracranial bypass surgery. AJNR Am J Neuroradiol. 2011;32:721–727.
67. Oppenheim C, Naggara O, Touze E, et al. High-resolution MR imaging of the cervical arterial wall: what the radiologist needs to know. Radiographics. 2009;29:1413–1431.
68. Hakimi R, Sivakumar S. Imaging of carotid dissection. Curr Pain Headache Rep. 2019;23:2.
69. Wolfe TJ, Hussain SI, Lynch JR, Fitzsimmons BF, Zaidat OO. Pediatric cerebral angiography: analysis of utilization and findings. Pediatr Neurol. 2009;40: 98–101.
70. Markus HS, Hayter E, Levi C, Feldman A, Venables G, Norris J. Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): a randomised trial. Lancet Neurol. 2015;14:361–367.
71. Kasner SE. CADISS: a feasibility trial that answered its question. Lancet Neurol. 2015;14:342–343.
72. Powers WJ, Rabinstein AA, Ackerson T, et al. 2018 Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2018;49:e46–e110.