Childs Nerv Syst (2011) 27:2045–2056
DOI 10.1007/s00381-011-1409-x

REVIEW PAPER

Pediatric traumatic carotid, vertebral and cerebral artery dissections: a review

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Received: 14 January 2011 / Accepted: 27 January 2011 / Published online: 12 February 2011

Abstract Traumatic cerebral dissections are rare but potentially dangerous conditions that through improved diagnostics have recently gained increased interest. However, there is still a significant lack of knowledge on the natural history, as well as on the best treatment options. Most of the literature on this topic consists of case reports and retrospective studies with no prospective randomized controlled studies. In our review, we highlight the fact that there is no level 1 evidence for the natural history of cerebral dissections or for the best treatment. We present 26 case studies derived from 70 pediatric patients affected by dissections, occlusions, and pseudoaneurysms.

Keywords Cerebral traumatic dissection · Carotid dissection · Vertebral dissection · Stroke · Pseudoaneurysm

Introduction

Carotid artery dissection (CAD) and vertebral artery dissection (VAD) are uncommon and traumatic dissections that appear to be more common than non-traumatic dissections [20, 23, 39, 44, 46, 50, 55]. The annual incidence of spontaneous CAD ranges from 2.5 to 3.0/100,000 patients and for spontaneous VAD, it ranges from 1 to 1.5/100,000. The incidence of traumatic dissection for carotid and vertebral artery is 0.08–0.4% of the entire trauma population [23, 46]. A review of the US National Pediatric Trauma Registry 1987–1997 discovered an incidence of 0.03% (15 of 57,659 blunt trauma patients) [29]. Through autopsy, Verneuil was the first to describe CAD in 1872 [59]. In 1955, Suechting et al. reported the first VAD after severe neck trauma [57]. These pathological entities are underdiagnosed because of the lack of early warning symptoms, and traumatic pseudoaneurysms may be misdiagnosed as saccular aneurysms, atherosclerotic lesions, or vasospasm following subarachnoid hemorrhage [9, 44]. However, recent greater physician awareness regarding dissections has led to an increase in the number of reported cases when compared with the cases reported a few decades ago.

CAD can lead to thrombosis and occlusion of the vessel [4, 9, 11, 58], and is one of the major causes of stroke in children [62]. The most common mechanism in traumatic CADs is direct blows to the neck/head or hyperextension [44]. Sporting events, fights, and falls can all cause arterial dissections, but motor vehicle accidents play the most significant role [23, 46]. Physical examination provides little evidence of carotid or vertebral artery injury as the condition may be asymptomatic or result in minor symptoms; however, lack of an early diagnosis may lead to stroke or death [23, 47].

The gold standard for diagnosing CAD is cerebral angiography [16], but MR angiography (MRA) [27] and CT angiography (CTA) have both been successful at detecting dissections and are gaining popularity as the resolution is improving. Ultrasonography [50] has also shown to be helpful in some cases for detecting dissections. Watridge et al. concluded that angiography should be performed on patients with focal neurological deficits when the CT scan is normal [60] as often times, a normal CT scan of the brain masks the possibility of a persistent dissection prior to, or early after an ischemic event. The reasons why angiography is not performed as primary diagnostic practice is because it is time-consuming, expensive and invasive, and carries a slight risk of causing neurological injury. As the quality of CTA continues to improve, CTA

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has replaced angiography as the first study of choice at some institutions [52]. Currently, there are ongoing randomized controlled trials comparing CTA with angiography. Because of its invasive nature [18, 48] and the radiation, angiography and CTA are often avoided in children, and instead, MRA is performed.

Children usually present with symptoms of cerebral ischemia [61], most commonly hemiparesis, whereas only 49–79% of adults present with ischemia. Pain is not a common symptom for children but it is for adults [27].

Treatment is assumed to target stopping the propagation of thrombus formation by the dissection and reducing embolization and occlusion, while the dissection is being endothelialized over time [60]. There is debate on which treatment option should be employed. Some authors have argued for surgical therapy [21] and endovascular treatment [1, 31, 45], whereas others have recommended conservative options such as close observation in asymptomatic patients. Others have proposed the administration of anticoagulation, antiplatelet agents, and thrombolytics [16, 49].

In summary, conventional cerebral angiogram is the golden standard of work-up; meanwhile, a quickly improving CTA [52] is gaining popularity and several randomized controlled trials are underway comparing CTA to angiogram. There is a common consensus on treating all the dissection with anticoagulants and considering endovascular therapy for the pseudoaneurysms. There is also a debate on the optimal non-invasive method. Some authors recommend antiplatelet therapy, whereas others advocate using anticoagulation therapy [10, 12, 14, 34, 35, 51, 56]. It is important to note that complete anticoagulation is difficult to apply and regulate in children; on the other hand, antiplatelet therapy is much easier to administer [9]. However, there is little known on the natural history of the non-treated traumatic dissections. So far, published studies are primarily case reports and retrospective. Also, the reviews on this subject so far, have discussed the previous works done without commenting on the fact that the different treatment options started after the first ischemic episode. To the best of authors' knowledge, there are no randomized control studies comparing Coumadin/Heparin with anti-platelet therapy prior to the first ischemic event. Therefore, the current study aimed to elucidate this pathology through a comprehensive review of the literature.

Materials and methods

Literature search

The search was limited to studies in the English language, and PubMed was used as the main search engine. The search terms included "traumatic cerebral dissection," "traumatic carotid dissection," "traumatic carotid artery dissection," "traumatic vertebral dissection," and "traumatic vertebral artery dissection." The search was limited to children (0–18 years of age). The abstracts and full-texts of these articles were then reviewed and some publications included from the reference lists of the individual papers.

Study selection criteria

The initial PubMed search provided a total of 1,033 articles dealing with traumatic cerebral/carotid/vertebral artery dissections. Figure 1 summarizes the results. Six hundred thirty-five papers were excluded because of duplicity. The inclusion criteria included prospective randomized controlled studies, prospective case control studies, and retrospective studies dealing with traumatic artery dissections. One hundred forty-one case reports dealing with

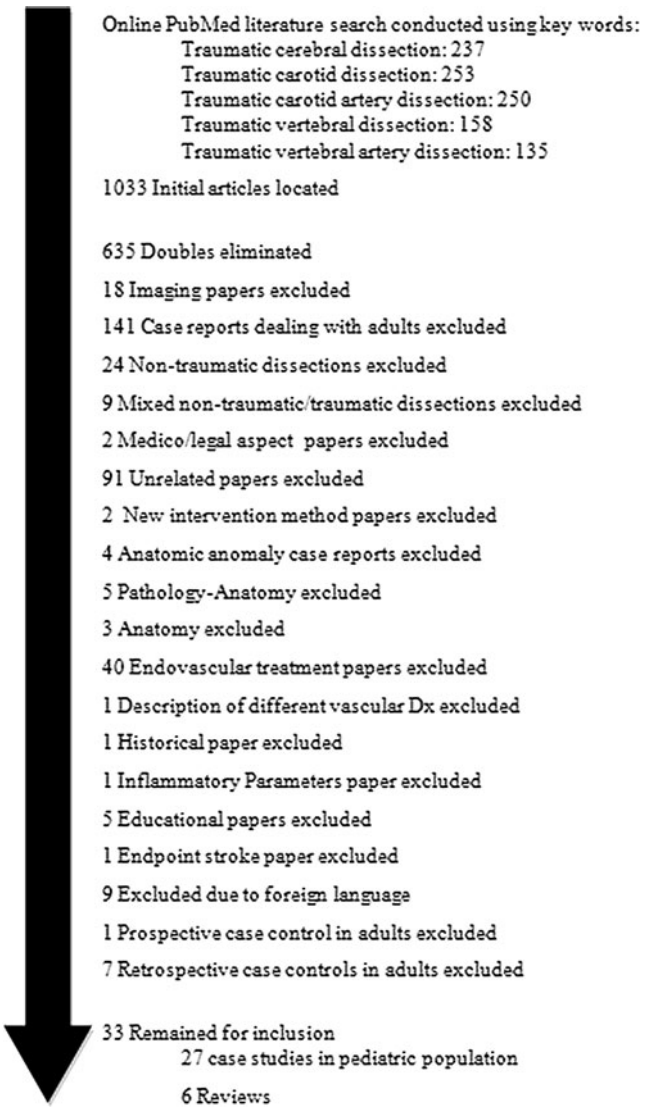

Fig. 1 Literature search

adult patients, 24 non-traumatic dissections, and 40 endovascular treatment papers were excluded. Also, 91 papers were found to be unrelated to the purpose of this review, and therefore, were excluded. The following papers were also excluded: imaging papers, new intervention methods, anatomical anomalies, pathology and anatomy, historical papers, educational reviews, and papers that involved medico/legal aspects. One retrospective study was excluded because its endpoint was stroke. Seven retrospective case control studies and one prospective case control study were also excluded because the studies did not involve pediatric populations. There were no prospective randomized studies, prospective case control studies, and retrospective studies dealing with traumatic artery dissections found in pediatric patients. Therefore, the search was extended to case control studies in pediatric populations. Two papers dealt with both pediatric and adult case studies and were included because of the relevant pediatric cases. Twenty-seven such case studies were found dealing with young patients.

Data extraction

The data extracted were: study design, patient demographics and number of patients, method of diagnosis, treatment employed, number of ischemic events prior to surgery, number of ischemic events post surgery, the length of follow-up, and finally, patient outcomes. The overall outcome criteria included neurological outcome as well as anatomical extent of the dissection.

Results

Risk factors

Risk factors seen in pediatric cases that can potentially lead to carotid, vertebral, or cerebral dissections are mostly similar to risk factors seen in adults. In the 26 case studies reviewed, 16 patients had encountered injury from a motor vehicle accident [2, 7, 13, 15, 25, 26, 29, 33, 40, 43, 53, 54], 1 from shoulder seatbelt [28], 4 patients were struck by a motor vehicle [29], 1 was run over by a truck [17], 2 patients underwent an auto-pedestrian accident [15], and 1 bicycle accident [43]. Fifteen patients encountered injury from falling [15, 17, 19, 22, 29, 32, 37, 42], 1 from a water slide [30], and 4 patients experienced intraoral trauma [33, 35, 36]. Three others were struck, one of whom was struck during a boxing game [24, 29]. One patient underwent a "laser tag" accident and either tripped or was pushed into one of the padded walls leading to neck and head injury [41], and another underwent sports trauma [43]. Two cases encountered injury due to child abuse [1]. For four patients, the authors mentioned that the patient had experienced traumatic injury; however, the mechanism of injury was not mentioned [8, 15, 24, 29].

Symptoms

Pediatric patients like adult patients, experienced similar symptoms due to cerebrovascular dissection (see Table 1). Five patients experienced facial palsy [13, 17, 30, 42, 54] and 18 patients suffered from decreased consciousness [6, 8, 29, 40, 53, 54]. Frantzen et al. reported that all six of the cases presented suffered from unconsciousness or somnolence in the initial phase of the disease [17].

The patient in Brunworth et al.'s study and one of the patients in Pierrot et al.'s study were asymptomatic [7, 42]. The patient in Levack et al.'s study suffered from right hemiplegia and aphasia due to his shoulder seatbelt when involved in a motor vehicle accident [28].

The patient in de Brost et al.'s study had a left-sided hematoma and experienced paralysis of the left arm and leg [13]. The patient in the study of Horowitz et al. presented with mild hemiparesis and ataxia [22]. Most other patients also suffered from hemiparesis [2, 3, 6, 15, 17, 19, 24, 26, 32, 33, 36, 37, 43].

Headache, vomiting, neck pain, numbness of the left limbs, hemiplegia, and slurred speech were all symptoms experienced by the patient in the study of Lin et al. [30]. The patient in Jariwala et al.'s study experienced increased confusion and lack of sensation and in the entire left upper extremity after the automobile accident [25]. One of the cases explained in Pierrot et al.'s study experienced decreased consciousness, left hemiplegia, and central facial palsy after falling with her mouth open against a door handle [42].

The patient in the study of Park et al. had incurred a brachial plexus injury after a motorcar accident and suffered from left arm monoplegia. The patient also displayed signs of cerebral concussion and loss of consciousness [40]. The patient who was injured in a "laser tag" accident experienced slurred speech and complained of a headache [41]. On examination, the patient's tongue was deviated to the right side and the eyes were deviated to the left. In addition, the patient's speech was dysarthric and he was confused. The patient of Singh et al. was admitted post-motor vehicle accident with symptoms of headache and loss of consciousness; however, 11 days after admission, the patient developed a right Horner's syndrome [53].

Out of the 12 patients in Chabrier et al.'s study, 8 suffered from decreased consciousness and all 12 encountered motor deficit. Three of the 12 cases also experienced seizures [8]. Thirteen of the 15 cases reported in the Lew et al.'s study suffered from altered consciousness [29]. Four out of five patients in Duke and Parrington study suffered from an

Table 1 Presenting symptoms and cause of injury in pediatric case control studies

Study/year	No. of patients	Cause of injury	Symptoms possibly linked with dissection	Ischemic symptom
Brunworth/2009	1	MVA	None	–
Levack/2009	1	Shoulder seatbelt	Right hemiplegia & aphasia	Y
Lin/2007	1	Water slide injury	Headache, vomiting, neck pain, numbness over left limbs, facial palsy, hemiplegia, & slurred speech	Y
de Burst	1	MVA	Facial palsy, paralysis of left arm & leg, left-side hematoma	Y
Jariwala/2006	1	MVA	Increased confusion & lack of strength & sensation of entire left upper extremity; GCS 9	Y
Pierrot/2006	2	Fall	Decreased consciousness, left hemiplegia, central facial palsy	Y
		Fall	Asymptomatic	–
Agner/2005	2	Child abuse	Seizures, left gaze unsteady gait, tremors right arm	Y
Park/2005	1	MVA	Left arm monoplegia & loss of consciousness	Y
Payton/2004	1	"Laser tag" accident—bumped head	Slurred speech & headache; tongue deviated to right side and eyes deviated to left side, speech was dyarthric and pt was confused	Y
Singh/2004	1	MVA	Headache & loss of consciousness. Developed Homer's syndrome	N
Chabrier/2003	12	Not stated	Motor deficit: 12; decreased consciousness: 8; seizures: 3	Y
Bar/2002		Intraoral trauma	Right hemiparesis, aphasia	Y
Borges/2000	2	Intraoral trauma	Left hemiparesis, somnolence, confusion	Y
		Bicycle accident—intraoral trauma	Left hemiparesis, aphasia	Y
Lew/1999	15	MVA: 4; struck by MV: 4; fall: 4; struck: 2; NS: 1	Altered consciousness: 13; mean GCS: 10±4.4	–
Ganesan/1997	1	Fall	Left hemiparesis, blurred vision, disoriented	Y
Nance/1997	1	Fall	Right-sided weakness, slurred speech	Y
Moriarty/1997	1	Intraoral trauma	Right hemiparesis, aphasia	Y
Duke/1996	5	Auto-ped	Right hemiparesis, aphasia; GCS 4	Y
		Non-accidental trauma	Infarct; GCS 13	Y
		Auto-ped	Infarct; GCS 15	Y
		MVA	Infarct; GCS 4	Y
		Fall	Infarct; GCS 15	Y
Horowirz/1994	1	Fall	Mild hemiparesis & ataxia	Y
Mann/1993	2	Fall	Right hemiparesis, aphasia	Y
		Birth delivery	Seizure activity, right hemiparesis	Y
Pozzati/1989	4	MVA	Right hemiparesis, dysarthia	Y
		Bicycle accident	Right hemiparesis, dysarthia	Y
		Sports trauma	Left-sided weakness	Y
		MVA	Right hemianopia, loss of vision in left eye	Y
An/1992	1	MVA	Right-sided hemiparesis, aphasia, seizures	Y
Martin/1991	2	Intraoral trauma	Left hemiplegia	Y
		MVA	Left hemiparesis	Y
Stringer/1980	1	MVA	Unconsciousness, 3rd and 6th cranial nerve palsy	Y
Kak/1972	1	MVA	Right hemiparesis, mute	Y
Jacob/1970	2	Boxing	Transient numbness of right-side diplopia	Y
		Thought traumatic—but unknown	Right hemiplegia	Y
Frantzen/1961	6	Fall	Right hemiplegia	Y
		Fall	Somnolent, left hemiparesis	Y
		Fall	Somnolent, diplopia, left hemiparesis, face weakness	Y
		Fall	Left hemiparalysis	Y
		Run over by truck	Loss of consciousness, right hemiparalysis, seizures	Y
		Shot	Left hemiparesis	Y

MVA motor vehicle accident

infarct [15]. Finally, the patient in Stringer & Kelly study suffered from possible third and sixth nerve palsy [54].

As can be seen in Table 1, the presence of some of the symptoms was taken as an indication for an ischemic event. These symptoms included hemiparesis, aphasia, facial palsy, forced eye deviation, dilated pupil, slurred speech, and monoplegia. Even though lack of consciousness, confusion, and headaches could be associated with ischemia, these symptoms alone were not linked with the presence of an ischemic event (see Table 1).

Diagnosis

Angiography and MRA were popular methods of diagnosis among the pediatric population with 26 out of the 70 patients (37%) undergoing only angiography and 9 out of the seventy patients undergoing just MRA (13%). Two case studies reported cases where patients were diagnosed with CTA [7, 15], three with Doppler [2, 3, 29], and one with cervical duplex [6]. Both patients in Pierrot et al.'s study were diagnosed through MRI [42].

Some patients underwent more than one angiographic exam. In two cases, the patients underwent both MRA and angiography [22, 30], and in de Borst et al.'s study, the patient underwent both MRA and CTA [13]. The 12 patients in Chabrier et al.'s study underwent MRA and/or angiography; however, the specifics were not provided [8]. Lew et al. did not state the method of diagnosis for 9 out of their 15 patients [29].

Overall, 32 patients (46%) were diagnosed with a dissection, 20 patients (28.6%) with an occlusion, and 3 patients (4%) had pseudoaneurysms. Lew et al. did not specify whether their patients had a dissection or an occlusion for their 15 cases that they reported. ICA was affected in 37 patients (53%), VA in 6 patients (8.6%), SCA in 7 patients (10%), and MCA in 4 patients (5.7%). Again in Lew et al.'s study, all 15 patients either had the ICA or common carotid artery (CCA) affected. One of the patients in Frantzen et al.'s study had both PCA and MCA occlusion (see Table 2).

Treatment and outcome

Two patients were treated with only heparin [42, 54]. However, three were treated with both heparin and ASA [3, 22, 42], another was treated with heparin and ASA followed by warfarin [41], and one was treated with heparin and then warfarin, wherein after 3 months, warfarin was substituted with ASA [53]. Another three patients were treated with both heparin and warfarin [36, 37, 40]. One patient was treated with heparin followed by enoxaporin [7], and another was treated by heparin followed by carbasalte calcium [13]. One patient was given antiplatelets and then heparin and warfarin therapy [25].

Ten patients were treated with only ASA [6, 8] and three were treated by only warfarin [1, 30, 32]. One patient was treated with corticosteroids and low-molecular-weight dextran [24]. Four case studies encompassing of ten patients mentioned that the patients were treated with anticoagulantion [8, 17, 19, 28]; however, the specific kind was not mentioned. Out of these ten patients, six belonged to Frantzen et al.'s study, in which each patient was treated with anticoagulation and repeated stellate blocks, with the exception of one who was also treated with hypothermia [17].

In Pozzati et al.'s study, three patients underwent surgery. One underwent aneurysmorrhapy and patch venous graft, another underwent thrombectomy, and the third underwent ICA ligation [43]. Nine patients were treated conservatively and were put under observation. These patients were not given anticoagulation, and no surgery was performed [2, 6, 8, 15, 24, 26, 33, 43]. For 16 patients, the treatment information was not available [29, 32] (see Table 2).

The patient in Brunworth et al.'s study was treated with a heparin drip and therapeutic enoxaporin (Lovenox) 1 mg/kg subcutaneously twice a day. The patient underwent a complete recovery, and a 39-day post-injury CTA showed complete resolution of the right VA thrombosis [7]. The Levack et al.'s study mentioned that the patient underwent anticoagulation; however, no specifics were given. On a 2-year follow-up, the patient had completely recovered. The imaging of this patient was never followed [28].

The patient of de Borst et al. was treated with low-molecular-weight heparin for 1 week and the regimen was switched to antiplatelet therapy, carbasalate calcium. The patient's neurological condition improved significantly. A 6-month follow-up angiogram showed normal carotid arteries [13]. In Horowitz et al.'s study, the patient underwent heparin therapy, and his hemiparesis, ataxia, and speech improved. The patient was released from the hospital with ASA. A 5-month follow-up shows almost complete recovery with slight foot drop still present. The patient did not undergo imaging follow-up for his dissection [22].

In Lin et al.'s study, the patient received warfarin therapy for 3 months, and after 3 months follow-up, the patient has partially recovered and walked with a walking stick [30]. In Jariwala et al.'s study, the patient was treated initially by antiplatelet therapy and then by anticoagulation that involved heparin and then warfarin. The patient's condition had somewhat improved by day 23 and she was transferred to a rehabilitation facility. The case study mentions that the patient continues to improve; however, the follow-up interval is not mentioned [25]. The Pierrot et al.'s study consisted of two patients. The first, a 4.5-year-old girl was treated with heparin for 4 months, followed by oral treatment with ASA (100 mg/day). Three-month follow-up color Doppler imaging showed good permeability of the

Table 2 Compilation of data dealing with case control studies of 70 patients (27 case studies) with traumatic cerebrovascular dissection

Case reports	No. of patients	Age	Imaging	Artery affected	Diagnosis	Treatment	No. of ischemic events		Fu Period	Outcome	
							Prior to Dx	After Dx		Stroke symptoms	Dissection
Brunworth/2009	1	8	CTA	VA	Occlusion	Heparin followed by enoxaporin	1	0	2 months	Complete recovery	39 days complete resolution of VA thrombosis
Levack/2009	1	14	MRA	MCA	Occlusion	Anticoagulation[a]	1	0	2 years	Complete recovery	Not followed
Lin/2007	1	7	MRA+Angio	ICA	Dissection	Warfarin	1	0	3 months	Patient requires walking stick	Not followed
de Borst/2006	1	13	MRA+CTA	ICA	Dissection	Heparin followed by carbasalate calcium	1	0	6 months	Hemiparesis improved but slight facial asymmetry	6-month follow-up angio: normal
Jariwala/2006	1	17	Angiography	ICA	Dissection	Antiplatelets then heparin & warfarin	1	0	Not stated	Partial recovery	Not followed
Pierrot/2006	2	4.5	MRI	ICA	Dissection	Heparin 4 months & then ASA	1	0	10 months	Complete recovery	3-month-Color Doppler showed good permeability
		3.5	MRI	ICA	Dissection	Heparin	0	0	21 months	Remained asymptomatic	21-month-Color Doppler showed normal permeability
Agner/2005	2	4 months	MRA	ICA	Dissection	No anticoagulation	1	0	1 year	Complete recovery	Not followed
		2	MRA	VA	Dissaayection	Warfarin for 3 month	1	0	18 months	Complete recovery	Not followed
Park/2005	1	4	Angiography	VA	Occlusion	Heparin 5 days & Coumadin 6 months	1	0	9 months	Monoplegia improved	Not followed
Payton	1	11	MRA	ICA	Dissection	Heparin & ASA followed by warfarin	1	0	Not stated	Neurological improvement	Not followed
Singh/2004	1	11	MRA	ICA	Pseudo-aneurysm	Heparin then warfarin (3 months) substituted with ASA	0	0	6 months	Complete recovery	Persistent dissection on 6-month follow-up
Chabrier/2003	12	2–12	ICA and MRA	Supraclinoid CA: 7; ICA: 3; VA: 2	Dissection: 10; occlusion: 2	ASA: 9; anticoagulant: 2[a]; observation: 1	12	0	41 months (8 month–8.6 year)	Complete recovery: 8; Recurrent stroke: 1; neurological disability: 4	6 patients followed: 5 recovered, 1 unchanged image; not followed 6
Bar/2002	1	9	Doppler	ICA	Occlusion	Thrombectomy, Post-op: heparin & ASA	1	0	1 year	Minor hemiparesis	Not followed
Borges/2000	2	16	Cervical duplex	ICA	Occlusion	Supportive care	1	0	8 days	Left hemiparesis	Not followed
		4	Cervical duplex+MRA	ICA	Occlusion	ASA	1	0	2 months	Minor mouth deviation and right Babinski sign	Not followed
Lew/1999	15	5.5±5.8	Angiography: 3; ultrasound: 2; MRA: 1; not stated: 9	ICA or CCA[b]	Not stated	Not stated	Not stated	Not stated	Not stated	Neuro compl: 6; dead: 2; 12 patients discharged home, 1 to rehab	Not followed
Genesan/1997	1	15	MRA	ICA	Dissection	Anticoagulant treatment 6 months[a]	1	0	Not stated	Residual dense hemiparesis	Not followed
Moriarty/1997	1	2.5	MRA	ICA	Occlusion	Heparin & then Coumadin	1	0	15 weeks	Complete recovery	Not followed

Table 2 (continued)

Case reports	No. of patients	Age	Imaging	Artery affected	Diagnosis	Treatment	No. of ischemic events		Fu Period	Outcome	
							Prior to Dx	After Dx		Stroke symptoms	Dissection
Nance/1997	1	11	Angiography	ICA	Dissection	Heparin for 1 week then warfarin for 9 months	1	0	1 year	Complete recovery	9 month-MRA and Doppler showed persistent reduced flow in left ICA
Duke/1996	5	1.2–8	Angiography: 4; CTA: 1	ICA: 5	Dissection: 3; occlusion: 2	Observation. No surgery of anticoagulation	5	0	24 h: 1; Not stated: 4	Left hemiparesis: 4; death: 1	Not followed
Horowitz/1994	1	5	MRA+ angiography	VA	Dissection	Heparin followed by ASA	1	0	5 months	Partial recovery	Never followed
Mann/1993	2	10	MRA	ICA	Dissection	Warfarin	1	0	Not stated	Partial recovery	Never followed
		9 days	MRA	ICA	Dissection	Not stated	1	0	10 months	Partial recovery	10-month MRA: encephalomal acia in region of left MCA infarction & recanalization of left ICA
An/1992	1	16	Doppler autopsy	ICA	Pseudo-aneurysm	Observation	1	0	76 h	Death	–
Martin/1991	2	12 & 15	Angiography: 2	ICA	Dissection: 2	Observation	1	0	Not stated: 2	Partial recovery: 1; no deficit: 1	Never followed
Pozzati/1989	4	15	Angiography	ICA	Dissection	Aneurysmorrha py and patch venous graft	1	1	Not stated	Partial recovery	Never followed
		5	Angiography	ICA	Dissection	No anticoaguation, observation, supportive care	1	1	12 years	Complete recovery	12-year Doppler ultrasonography did not reveal any abnormality
		18	Angiography	ICA	Occlusion	Thrombectomy	0	0	5 days	Death	–
		16	Angiography	ICA	Pseudo-aneurysm	ICA ligation	1	1	Not stated	Partial recovery	Never followed
Stringer/1980	1	15	Angiography	ICA	Dissection	Heparin	1	0	3 years	Complete recovery	Never followed
Kak/1972	1	1.4	Angiography	ICA	Occlusion	None	1	0	1 year	Partial recovery	Never followed
Jacob/1970	2	14	Angiography	ICA	Dissection	None	1	0	3 weeks	Complete recovery	Never followed
		7	Angiography	MCA	Occlusion	Corticosteriods & LMW dextran	1	0	3 days	Death	Autopsy showed lt middle cerebral artery occluded by a thrombus-hematoma found
Frantzen/1961	6	14	Angiography	MCA	Occlusion	Anticoagulant therapy[a] & stellate blocks	1	0	9 months	Partial recovery	9-month Angio showed normal results
		6	Angiography	PCA & MCA	Occlusion	Anticoagulant therapy[a] & stellate blocks	1	0	8 months	Partial recovery	1 month better flow in MCA, but not normal
		11	Angiography	MCA	Occlusion	Anticoagulant therapy[a] & stellate blocks					
		5	Angiography	ICA	Occlusion	Anticoagulant therapy[a] & stellate blocks	1	0	7 months Partial recovery		6-weekocclusion still present
		1.8	Angiography	ICA	Occlusion	Anticoagulant therapy[a]	1	0	1.5 years	Minimal recovery	Electroencep halogram

Table 2 (continued)

Case reports	No. of patients	Age	Imaging	Artery affected	Diagnosis	Treatment	No. of ischemic events		Fu Period	Outcome	
							Prior to Dx	After Dx		Stroke symptoms	Dissection
						& stellate blocks					severely abnormal
		14	Angiography	ICA	Occlusion	Anticoagulant therapy[a], stellate blocks & hypothermia	1	0	1.5 years	Partial recovery	Never followed

cCT contrast enhanced computed tomography; *MRA* magnetic resonance angiography; *CTA* computed tomography angiography; *VA* vertebral artery; *ICA* internal carotid artery; *ECA* external carotid artery; *ASA* acetylsalicylic Acid; *MCA* middle cerebral artery; *LMW* low-molecular weight

[a] Anticoagulant therapy given but the kind not specified

[b] not stated how many cases with ICA or CCA

right CCA. Ten-month follow-up showed complete recovery in the patient. The second patient, a 3.5-year-old girl remained asymptomatic after being treated with low-molecular-weight heparin. Twenty-one-month follow-up showed normal permeability of the CCA on the cervical color Doppler imaging, and the patient remained asymptomatic [42].

Agner and Weig presented two cases of child abuse where the first patient was treated for her ICA dissection through observation. Because of the patient's subdural and subhyaloid bleeding, she was not anticoagulated. She was treated for her seizures through seizure medication, and at 1-year follow-up, the patient's only symptom was decreased vision in left eye. Her imaging was not followed. The second patient, a 2-year old boy was treated with warfarin for 3 days for his VA dissection and showed complete recovery on 18-month follow-up [1].

In Park et al.'s study, the patient received intravenous heparin for 5 days and was discharged from the hospital on Coumadin therapy for 6 months. Nine months follow-up showed improved monoplegia. There was no imaging follow-up performed on this patient [40].

Payton et al. provided their patient with heparin and ASA, which were followed by warfarin therapy. The patient experienced gradual neurological improvements; however, no follow-up imaging data is available. The patient was transferred to a rehabilitation hospital for further care [41].

In Singh et al.'s study, the patient was managed with heparin therapy and was discharged on warfarin therapy for 3 months. The patient's 6-month follow-up MRA showed persistent pseudoaneurysm and warfarin was substituted with ASA. The patient has been asymptomatic since, but no data on imaging follow-up after the 6-month period is available [53].

Chabrier et al. divided their results into two groups: cervical arterial dissection that dealt with ICA and VA and intracranial arterial dissection that dealt with supraclinoid carotid artery dissection. Out of the five patients that had cervical arterial dissection, two were treated with anticoagulation (kind not specified) and three were treated with ASA. Both patients treated with anticoagulation had a favorable outcome; however, the specific outcome of the three patients treated with ASA was not clearly stated. Whereas, six out of the seven patients in the intracranial dissection group were treated with ASA, and none of them were treated with anticoagulation. No recurrences were observed in these six patients and angiographic follow-up showed the lesions disappeared or regressed in all but one patient whose image was unchanged [8]. In Bar et al.'s study, an immediate ICA thrombectomy was performed and much improvement was seen after surgery; however, hemiparesis persisted. After surgery, the patient underwent treatment with amoxicillin clavulanate potassium and

clindamycin and with heparin and ASA. On 1-year follow-up, the patient is doing well, but slight hemiparesis persisted. No data on imaging follow-up are available [3]. Borges et al.'s study consisted of two patients. The first patient, a 16-year-old boy, was admitted for supportive care, and the study did not mention that he was treated with anticoagulation. After 8 days of hospitalization, the patient was discharged with stabilized symptoms. There was no mention of imaging follow-up on this patient. On the second patient, a 4-year-old boy, was discharged after 9 days from admittance with persistent aphasia and somewhat improvement of symptoms. The patient was treated with 100 mg of ASA and 2-month follow-up showed partial recovery showing normal muscle strength, but minor mouth deviation, homonymous hemianopia, brisk reflexes, and right Babinski sign. Once again, no image follow-up is available on this patient [6].

In Ganesan et al.'s study, the patient was treated with anticoagulant treatment for 6 months; however, the specific type of anticoagulation method used was not mentioned. The patient improved slowly. The case study reports that the patient could now walk, but had not gained useful function in his arm. There was no imaging follow-up provided and the follow-up duration was not mentioned [19]. In Nance et al.'s study, the patient was started with intravenous heparin and 7 days after trauma, the patient started on oral warfarin. On 1-year follow-up, the patient had no neurological sequelae, but MRA after 9 months still showed abnormalities of ICA, and reduced flow on Doppler persisted [37]. In Moriarty et al.'s study, the patient was given intravenous heparin, and Coumadin was started when the patient's neurologic signs stabilized. The patient also underwent rehabilitation. Fifteen weeks follow-up showed complete recovery, but there was no imaging follow-up data available [36].

In the study of Duke and Parrington, none of the five patients underwent surgery or anticoagulation. One patient with bilateral carotid thromboses died on the first hospital day. Since all patients had developed large infarcts prior to the diagnosis of their dissection, the decision to not anticoagulate was made. The rest of the four patients all had partial recovery. They all had hemiparesis but ambulated independently and none of them suffered any further ischemic events [15]. Mann et al. consisted of two patients. The first, a 10-year-old boy, was treated with warfarin sodium and had left hemiparesis at the time of discharge. The duration for further follow-up is not stated and no further imaging data is available on this patient. The second patient in this study, a newborn child, was attempted to be vaginally delivered, but after that failed vacuum delivery was then attempted. Finally, an urgent c-section was performed. The newborn was treated with seizure medication, but no mention of anticoagulants was made. A 10-month MRA follow-up showed encephalomalacia in region of left MCA and infarction and recanalization of left ICA [32].

Pozzati et al.'s study consisted of four patients, three of whom were treated with surgery. The first patient, a 15-year-old boy, had aneurysmorraphy, and patch venous graft was performed. The patient suffered from palsies of the seventh, ninth, and twelfth cranial nerves; however, follow-up showed partial recovery of these symptoms. The follow-up duration is not mentioned and no follow-up imaging data is available for this patient. The second patient, a 5-year-old girl, was not treated with any anticoagulation. Her symptoms cleared rapidly, and 12-year follow-up Doppler ultrasonography did not reveal any abnormality. The third patient, an 18-year-old boy, underwent a thrombectomy; however, his left hemiplegia was unchanged. The patient died 5 days later. The fourth patient, a 16-year-old boy, underwent ICA ligation, and follow-up showed marked improvement of the visual field in the right eye; however, his left eye remains blind. The follow-up duration or imaging follow-up is not present in the case description of this patient [43].

In An et al.'s study, the patient died on the fourth hospital day. He was not treated with anticoagulation or surgery for his pseudoaneurysm of the ICA [2]. Out of the ten patients in Martin et al.'s study, two were relevant for this study because of the traumatic injury to the ICA leading to dissection. Both were treated on supportive care, and no anticoagulation or surgery was performed. One patient had a complete recovery with no neurological deficit, whereas the other underwent partial recovery. Follow-up duration or imaging follow-up was not present for either of the patients [33]. The patient in Stringer and Kelly's study was treated with heparin for 5 days, after which he began to follow commands appropriately and equally on both sides. Heparin was discontinued in this patient because of hypotension, anemia, and massive melena. Three-year follow-up imaging showed no change in the pseudoaneurysm, and the boy seems to have no neurological deficits [54]. The patient in Kak et al.'s study was not given any anticoagulation, and no surgery was performed. The patient underwent a partial recovery shown on 1-year follow-up. No imaging follow-up is available [26]. The first patient in Jacob et al.'s study, a 14-year-old boy, was also treated conservatively and no anticoagulation was given. Three-week follow-up showed complete recovery, and subsequent follow-ups have shown no relevant abnormalities. No angiographic imaging follow-up is present for this patient. The second patient in this study, a 7-year-old girl, was treated with corticosteroids and low-molecular-weight dextran for her MCA occlusion. The patient remained drowsy and died on the third hospital day. Her autopsy showed a turgid left MCA and seemed occluded by a thrombus [24].

Frantzen et al.'s study consisted of six patients, and they were all treated with anticoagulation and repeated stellate blocks. The specific type of anticoagulation was not mentioned. On 9-month follow-up, the first patient, a 14-year-old boy was somewhat demented with a slight right-sided spastic hemiparesis. The angiography showed normal imaging, but an electroencephalogram was slightly abnormal. The second patient, a 6-year-old boy, suffered from left hemiparesis on the eighth month of follow-up. The 1-month follow-up angiographic imaging had shown better flow in the MCA, but not normal. The third patient, an 11-year-old boy, had an eventful history. He was diagnosed with an occlusion of the MCA, but 15 months later, his symptoms reappeared, and this time, the angiography showed a complete occlusion of the right anterior cerebral artery. After treatment, 1.5-year follow-up from first diagnosis, he was demented. His left hemianopia persisted, and there was a diffuse paresis of his left arm, and also, his gait was spastic. No further imaging follow-up is available. The fourth patient, a 5-year-old girl, had good recovery after her treatment. Her paralysis of the arm persisted on the seventh month of follow-up. The fifth patient, a 21-month-old boy, was diagnosed to be slightly retarded on 1.5-year follow-up. The patient still suffered from slight facial paralysis, spastic paralysis of the right arm, and slight spastic paralysis of the leg. The sixth patient, a 14-year-old boy, was treated with hypothermia in addition to anti-coagulation and stellate blocks. One-and-a-half-year follow-up showed partial recovery. The patient still suffers from slight dementia and exhibits behavior disorders. No imaging follow-up is available for this patient [17].

Lew et al.'s study of 15 patients did not mention the treatment method or the follow-up duration; however, they did mention the outcome for few of their patients. Six patients had neurological complications and two patients died. Out of 13 left, 12 patients were discharged home, but one was discharged to rehabilitation. There was no imaging follow-up data available [29]. None of the patients encountered new ischemic symptoms after treatment of dissection. Follow-up ranged from 3 to about 12 years.

Discussion

Traumatic dissections are being given increasing attention as an important treatable diagnosis group, but with serious outcomes if untreated [5, 9, 16, 18, 38, 46]. This is following almost two decades of increasing awareness, secondary to improving imaging techniques. Although angiogram continues to be the gold standard for diagnosis [16, 38], modern CTA are continuing to gain popularity as their specificity and sensitivity are approaching that of cerebral angiograms. With increasing awareness of this diagnosis, the focus would naturally be on the natural history of untreated dissections. Our review of the current literature shows that there is no level 1 evidence on natural history of dissections. Most of the literature consists of case reports, and there are a few studies consisting of less than ten patients per study, where some were treated and some were not, that do not show any difference in outcome. There is a propensity to treat all the dissections so most of the studies are biased towards treatment. Additionally, most of the outcomes are measured by neurologic outcome and many of them lack angiographic follow-up.

When looking at different treatment options, we see again a bias towards treatment. Heparin, coumadin, dipyridamol, ASA, and clopidrogel, have all been used alone or in conjunction, and although some studies have shown benefit towards one or the other in different trials, there is not even a level 2 or level 3 reproducible positive outcome for any of them.

A very noteworthy distinction we have observed in the literature is that most of the treatment options are started after patient's ischemic event and then have been compared to each other. This might naturally be due to the fact that the advent of this new diagnosis group was boosted by search for the etiology of unexplainable delayed post-traumatic strokes. Therefore, the radiological work-up for dissection was initiated first after the ischemic event. Hence, we do not know the difference between treatment and observation from diagnosis of the dissection until the first ischemic event.

As more attention is being given to this diagnosis, and less invasive, more sensitive, and more reachable imaging are being utilized in the modern time, more dissection prior to causing ischemic events will be diagnosed, and therefore, we would expect the literature to include prospective randomized trials comparing anticoagulation agents to anti-thrombotic agent and observation from the diagnosis of the dissection rather than from the diagnosis of the dissection-induced stroke. We would also expect radiological follow-ups to be made looking into the epithelialization of the dissection over time.

The only works in the pediatric population were 27 case reports. Risk factors, symptoms, and treatment regimens seemed to be similar to adults. Most of the patients were started on anticoagulant or antiplatelet therapy with good prognosis. A few patients were observed conservatively. The follow-up imaging was more frequent compared to the adult population and showed resolution of the dissection. However, once again, all the treatments were given after the first ischemic event.

Conclusions

Traumatic carotid, cerebral, and vertebral artery dissections are rare conditions that appear to have a relative good

prognosis if treated but have a grave prognosis causing ischemic stroke if untreated. Our knowledge is mostly limited to a few retrospective studies. The natural history is unknown. Furthermore, the studies neither indicate if treatment leads to better outcome, nor point towards one treatment being better than the other. The different treatments have also begun after the dissection-induced ischemic event, rather than before. Standardized imaging follow-up is lacking. The pediatric population follows similar patterns, although the literature is even scarcer. Currently, the consensus is that dissection should be treated with anticoagulant or anti-thrombotic agents. Prospective randomized trials starting at the diagnosis of the dissection and prior to the ischemic event, and follow-ups including imaging outcome, are warranted.

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