

Carotid And Vertebral Arterial Dissections In The Emergency Department

Abstract

Although carotid and vertebral (cervical) arterial dissections are not common presentations to the emergency department, timely and appropriate diagnostic strategies will allow early initiation of effective treatment therapies. Cervical arterial dissection occurs when the intimal wall of an artery is damaged as a result of trauma or defect. As blood fills the layers of the arterial wall, thrombi form, which can lead to stroke, pseudoaneurysm, vessel occlusion, and stroke. Intracranial dissections may result in subarachnoid hemorrhage. Because cervical arterial dissections may present with common signs and symptoms such as headache, neck pain, neurological deficits, and stroke, it is essential that dissection be considered early and ruled out quickly. Computed tomographic angiography, magnetic resonance angiography, and digital subtraction angiography may be used for diagnosis. Anticoagulation or antiplatelet therapy is the mainstay of treatment for spontaneous or traumatic dissections and will reduce the risk of stroke. Endovascular therapy or surgery may be indicated. Recurrence or rebleeding is a significant risk and must be managed.

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CME Objectives

Upon completion of this article, you should be able to:

1. Cite the presenting signs and symptoms of carotid, vertebral, and intracranial dissections.
2. Select appropriate imaging modalities for the evaluation of cervical artery dissection and when to pursue each.
3. Describe when to initiate aspirin, heparin anticoagulation, or endovascular therapy.
4. Summarize the indications and contraindications to initiation of IV tPA in the ED.
5. Demonstrate how to elicit key components of the patient's history that predispose to and alter the prognosis of cervical artery dissection.

Prior to beginning this activity, see "Physician CME Information" on the back page.

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Case Presentations

A 29-year-old woman is brought in by EMS after a motor vehicle accident in which she was an unrestrained driver. She was found by the medics lying across the steering wheel with a large gash on her forehead. At the scene, she was awake, but disoriented, and there was a strong odor of alcohol on her breath. Her ED assessment revealed a small right-sided subdural hematoma and a zygoma fracture. Her serum ETOH level was 260 dL/mL, and she was placed under observation status on the trauma service. Six hours later, she becomes agitated and then rapidly develops left-sided weakness and neglect. Your first thought is that she must have had a seizure and is left with a Todd paralysis . . . but it doesn't quite add up, and you wonder what else might have happened. Is there anything you should be doing?

A 41-year-old man presents to the ED after developing blurred vision 1 hour earlier while walking at the grocery store. He had the same blurry vision 3 years ago, at which time he was prescribed glasses. His vision improved within 3 weeks. Five years ago, he had a "couple of months" of mild neck tightness that caused him to seek out chiropractic treatment. He does not take any medications, works as a businessman, and drums in a band on the weekends. His drug screen and urine toxicology are negative. Examination shows a nystagmus on right-end gaze and mild incoordination with right finger-nose-finger testing. Neuro is paged, and you wonder if there is a tester intervention that you should be providing.

Introduction

Dissections of the carotid and vertebral arteries are uncommon conditions that are often the topic of morning emergency department (ED) conversations starting with, "You know that patient you saw last night?..." The diagnosis and intervention for cervical arterial dissection are often delayed until the patient develops irreversible neurologic sequelae. Awareness of this condition, both spontaneous and as a complication of traumatic injury, has increased with the parallel evolution of imaging technology. These have both conveniently intersected to a place where one can suspect the diagnosis and then obtain diagnostic imaging within a reasonable timeframe and without undue risk to the patient.

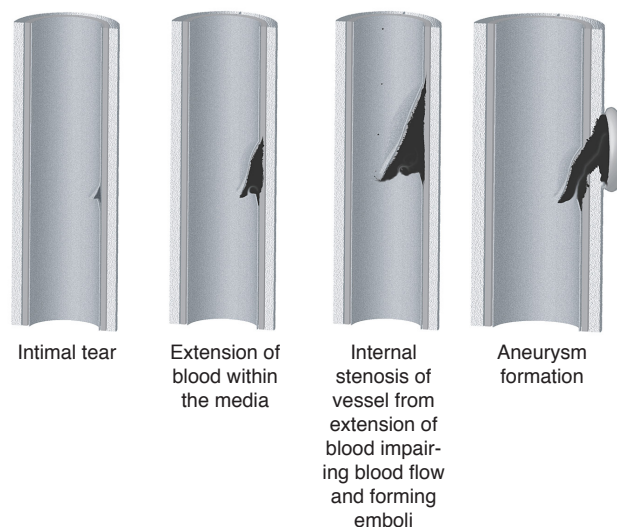
An arterial dissection occurs when the inner lining of the arterial wall (intima) is disrupted and blood extends between the layers of the blood vessel wall. (See Figure 1.) (Note: for color images of general anatomy of extracranial and intracranial arteries, please see Figure A at www.ebmedicine.net/CVADfigures.) This process can cause vessel stenosis, vessel occlusion, distal emboli, aneurysm formation, and subarachnoid hemorrhage (SAH).

Cervical artery dissections are dissections of the carotid or vertebral arteries. They occur in patients of all ages, and cervical artery dissections are the

cause of stroke in 8% to 10% of patients under age 40.^{1,2} Methods of detecting and treating the disease are complex and have been an active area of research for over 30 years. The disease was first recognized in 1947 when Dratz published dissection as a complication from direct injection of the carotid artery.^{3,4} The recognized incidence of spontaneous cervicocranial dissection in the United States and Europe is approximately 3 to 5 per 100,000. The rate of dissection in patients with significant traumatic injuries is much higher, approximately 1 in 1000.⁵⁻⁹

Spontaneous dissections present with a constellation of symptoms and signs that are common ED complaints – headache, dizziness, and weakness. While many patients with these presenting complaints improve with fluids, ibuprofen, reassurance, and counseling, the diagnosis of carotid or vertebral arterial dissection needs to be considered, as these patients require urgent intervention to prevent disastrous complications. The stakes are similarly high for the trauma patient. During a typical ED shift, emergency medical services (EMS) will bring in multiple patients with traumatic closed-head injuries; unless an aggressive screening protocol is in place, the only cervical artery dissections diagnosed will be those that are recognized too late.

Figure 1. Mechanisms Of Cervical Artery Dissection



Disruption of the intima leads to exposure of tissue factor to the blood, inducing thrombosis. Platelets adhere at the site of injury, leading to further thrombus formation. After the intima sustains damage, there may be extension, leading to the creation of an intimal flap. Blood may dissect anywhere along the media or adventitia. Intracranial vessels do not contain smooth muscle and have a weak adventitia, leading to a substantial risk of vessel rupture and subarachnoid hemorrhage.^{8,10,11}

Used courtesy of Kenneth Shea, MD.

Critical Appraisal Of The Literature

A literature search was performed using PubMed, Ovid MEDLINE®, and Google Scholar® with the search terms *carotid artery dissection, vertebral artery dissection, cervical artery dissection, dissecting intracranial aneurysm, intracranial dissection, and stroke dissection* from 1977 to present. Limitations were placed on the searches to yield only clinically relevant journal articles. More than 650 articles were reviewed. Only recent review articles from peer-reviewed journals published from 2001 to present were considered relevant, due to increasing use of computed tomographic angiography (CTA) and magnetic resonance angiography (MRA) after that time.

The Cochrane Database of Systematic Reviews was also queried, and the search yielded 1 relevant article.¹² No randomized controlled trials were found regarding medical, surgical, or endovascular treatment of cervical artery dissection, carotid artery dissection, or vertebral artery dissection. A search performed through the National Guidelines Clearinghouse (www.guidelines.gov) for guidelines regarding cervical artery dissection revealed approximately 20 guidelines relating to headache or stroke that mention consideration of arterial dissection. Diagnostic imaging consensus guideline recommendations from the American College of Radiology address imaging to screen for dissection in adults and children who present with general headache, unilateral headache, or ipsilateral Horner syndrome.¹³⁻¹⁶

Consensus guidelines addressing diagnostic imaging in the setting of trauma have been produced by the Eastern Association for the Surgery of Trauma and the American College of Radiology.¹⁷ MRA and CTA are listed as preferred imaging modalities in these guidelines. With regard to the treatment of stroke and transient ischemic attack (TIA), there are no guidelines regarding treatment with tissue plasminogen activator (tPA) in arterial dissection. The 2011 American Heart Association (AHA) Secondary Prevention of Stroke and Transient Ischemic Attack guidelines state that stroke prophylaxis with aspirin or warfarin is indicated for 3 to 6 months.¹⁸ For recommendations on dissection treatment from the 2008 AHA guideline, see **Table 1**.

Given that arterial dissection is a relatively uncommon condition, there are no randomized controlled trials regarding treatment or management. There are cohort studies reporting the use of tPA for dissection-associated stroke.²⁰⁻²⁴ While dissection patients do not show as much improvement as patients with nondissection acute stroke after receiving tPA, small studies have shown intravenous (IV) tPA to be beneficial. In 1 study comparing outcomes in stroke patients given tPA, 36% of cervical artery dissection patients showed stable neurologic improvements compared to 44% in the nondissection group.²⁵

There are nonrandomized treatment trials and pooled analyses of case reports comparing aspirin to warfarin for stroke prevention in dissection, but no statistically significant difference between treatments has been demonstrated.^{18,23,26,27}

In cohort and case-control studies, dissection patients have been compared to stroke patients, accident victims, or patients with symptoms such as headache, neck pain, or ipsilateral Horner syndrome. These studies have attempted to address the natural history of dissection,^{5,28-31} risk factors,^{29,32,33} causes,³⁴⁻³⁶ efficacy of treatment with tPA,^{22,25} or secondary stroke prevention.^{23,26,27} More-contemporary studies have attempted to closely match patients. The Cervical Artery Dissection and Ischemic Stroke Patients (CADISP) Group recently published a case-control analysis where the groups were matched by age, gender, and country of origin.³⁷

Etiology And Pathophysiology

Arterial dissection occurs when blood exits the intimal lining of the arterial wall through a tear or defect, creating an intramural thrombus. As blood fills within the layers of the arterial wall, the inner lumen of the artery may be compressed, creating a stenosis. Alternatively, the blood may track further away from the inner lining, leading to aneurysm formation. SAH may result from intracranial dissection because there is a lack of smooth muscle within the vasa vasorum of the adventitia.³⁸ Dissection may occur from trauma, mild mechanical stress to the artery, or as a spontaneous event.

Table 1. American Heart Association Treatment Recommendations For Stroke Patients With Arterial Dissections, 2008¹⁹

Recommendation	Class/Level of Evidence
For patients with ischemic stroke or TIA and arterial dissection, warfarin for 3 to 6 months or antiplatelet agents is reasonable.	Class IIa, Level B
Beyond 3 to 6 months, long-term antiplatelet therapy is reasonable for most ischemic stroke or TIA patients. Anticoagulant therapy beyond 3 to 6 months may be considered among patients with recurrent ischemic events.	Class IIb, Level C
For patients who have definite recurrent ischemic events despite antithrombotic therapy, endovascular therapy (stenting) may be considered.	Class IIb, Level C
Patients who fail or are not candidates for endovascular therapy may be considered for surgical treatment.	Class IIb, Level C

Abbreviation: TIA, transient ischemic attack.

Dissection typically presents as pain from disruption of the intimal lining of the artery. Stroke may occur immediately, or it can occur days or even years after the onset of pain.^{31,39-42} Ischemic stroke may occur from the occlusive low-flow state or from emboli. Compression of the vessel by the hematoma or occult thrombus at the site of the dissection may cause diminished flow. Emboli may form at the dissection from disruption of the lumen or from turbulence of blood flow.⁴³ Endothelial factors exposed by disruption of the intima could cause local thrombus formation with the potential to embolize.^{44,45} (See **Figure 1, page 2.**)

Spontaneous Cervical Artery Dissection

In patients without significant trauma, it is suspected that there is an underlying predisposition to spontaneous dissection. Over half of patients with a recurrence of spontaneous cervical artery dissection have been found to have a family history of cervical artery dissection, compared to only 5.8% of patients without recurrence.^{29,46} Patients who have been found to have a vertebral artery dissection have simultaneous dissection in another vessel more than patients with cervical artery dissection.^{39,47,48} Mechanical stressors that have been presumed to be causal include sneezing, chiropractic neck manipulation, sexual intercourse, paroxysmal hypertension, judo and yoga sessions, shaving, nose-blowing, coughing, ceiling painting, minor falls, rapid head turning or flexing, and swimming. Given the relatively minor nature of the trauma, they are typically referred to as provoked spontaneous dissection.^{8,49-67} No mechanical event is reported in 20% to 35% of patients, but truly spontaneous dissection is thought to be very uncommon.^{52,68-70}

Approximately 1% to 5% of dissection patients have an identifiable connective tissue disease. Features suggestive of a connective tissue abnormality are present in approximately 20% of dissections where criteria of known connective diseases are not met.^{71,72} Diseases that have been known to have increased risk for dissection include connective tissue diseases such as fibromuscular dysplasia, Ehlers-Danlos syndrome, osteogenesis imperfecta, and Marfan syndrome. Migraines have been shown to have an association, but no causal or pathological relationship has been determined.^{31,73-75} (See **Table 2.**) A very recent histopathological study implicates diffuse inflammation and microhemorrhages of the vessel wall in predisposing patients to dissection.³⁶ Some 22% to 64% of spontaneous vertebral artery dissection patients have multiple dissections on presentation, which suggests an unidentified underlying disease.^{39,47,48}

The location of nontraumatic dissection along an extracranial artery is most commonly where the artery transitions from mobile to immobile or is compressed externally by a bony structure. The

most vulnerable location to dissection along the extracranial internal carotid artery is from 2 cm above the bifurcation to the skull base.^{5,40,41,71,82} During hyperextension and torsion or abrupt flexion and torsion of the neck, the carotid artery may be injured by being stretched or compressed against the transverse processes of C2 or C3.^{47,83-86} (Note: color images of the anatomy of Horner syndrome from cervical arterial dissection [**Figure B**] and the anatomy of cranial neuropathies from cervical arterial dissection [**Figure C**] can be viewed at www.ebmedicine.net/CVADfigures.)

Vertebral artery dissection occurs far less commonly than carotid artery dissection.^{5,7} The annual incidence is approximately 1 per 100,000.⁷ There are several locations along the vertebral arteries that are thought to be vulnerable to injury. One location is from C1 to C2 where there are mechanical forces between the atlas and the C2 cervical foramina during head rotation.^{39,41,51,52,87-89} The other classical location is at C6, where the vessel becomes immobile at the cervical foramina.^{8,87-89} Head-turning may cause compression of the vertebral artery against skeletal muscle and fascial bands, resulting in a third possible site of dissection between the cervical foramina and the origin.^{39,41,69,90} (See **Figure 2.**) Multiple series have confirmed that the most common locations of vertebral artery dissection cluster around C1 to C2 at the atlantooccipital and atlantoaxial joint and from C5 to C6 where the vessel enters the cervical foramen.^{39,41,51,52,69,87-89} In children, there is a strong predisposition for vertebral artery dissection to occur at the C1-C2 level (53%).⁹¹

Table 2. Potential Causes Of Spontaneous Dissection

Association	Summary of Evidence
Hypertension	Multiple cohort studies including large international cohort (CADISP) ³⁷
Hyperhomocysteinemia	Multiple cohort studies
Collagen mutations	Collagen type III (COL3A1), collagen type IV (Ehlers-Danlos syndrome) in cohort studies ³³ and case reports ⁷⁶
Marfan syndrome	Case reports
Fibromuscular dysplasia	Case reports
Autoimmune thyroid disease	One small cohort study ⁷⁷
ICAM-1 gene polymorphism	One small cohort study ⁷⁸
MTHFR gene polymorphism	3 cohort studies supporting; 3 cohort studies with no significant difference
Alpha 1 antitrypsin deficiency	Single cohort study
Infections: syphilis, virus	Cohort study ⁷⁹
Migraine	Retrospective series ⁸⁰

A large multicenter international cohort of European patients with cervical artery dissection has been formed, known as CADISP.³⁷ Patients with cervical artery dissection complicated by stroke are more likely to be hypertensive but less likely to be obese, have hyperlipidemia, smoke, or have diabetes than young patients without dissection having stroke.³⁷ Similar risk factors have been shown in other, smaller cohorts.^{37,49,93-98} Because risk factors for cervical arterial dissection are similar to those for atherosclerosis, a relationship between the 2 conditions has been suggested, but there is insufficient evidence to determine whether or not there is a relationship.^{95,99}

Trauma is implicated as a cause of many arterial dissections that are subsequently classified as “spontaneous” due to the relatively minor nature of the associated trauma. The classification of a dissection as “traumatic” depends on the severity of the injury suffered by the patient. For the purposes of this discussion, traumatic dissections refer specifically to injuries due to blunt trauma forces to the head and/or neck. In the past 2 decades, injuries to the carotid and vertebral arteries as a result of blunt trauma have been recognized with increasing frequency. Early studies described the clinical presentation of traumatic dissections as unanticipated neurological deficits in the trauma victim, with often devastating consequences.^{34,100-106} More-recent studies have focused on early identification of carotid and vertebral artery dissections to allow for a larger window of opportunity in which to intervene before symptoms develop. Expert consensus is that early diagnosis and therapeutic intervention will improve clinical outcomes.¹⁰⁷⁻¹⁰⁹

Pathophysiology Of Traumatic Dissections

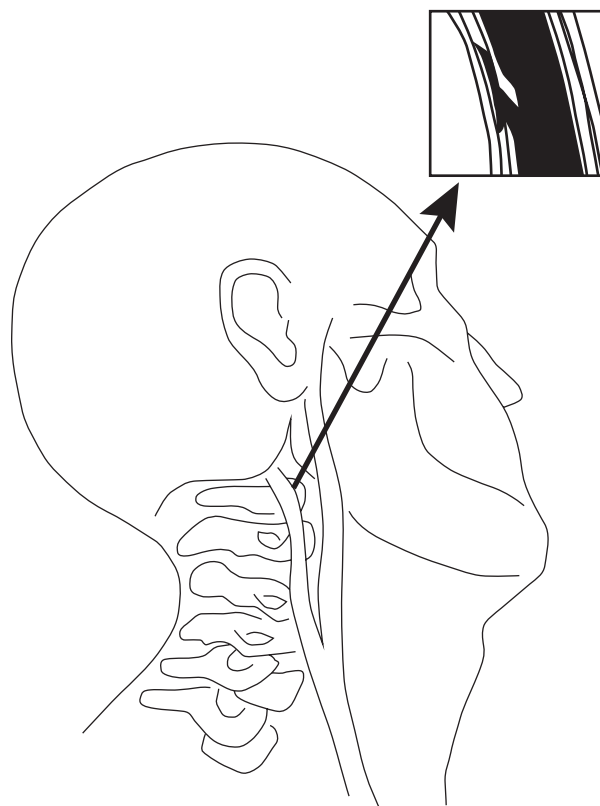
Traumatic cervical artery injuries result in either partial or complete disruption of the vessel wall. The injury and subsequent complications can be considered along a continuum, from its most minor form—a separation of the intima from the underlying media—to a frank dissection with an intimal flap, pseudoaneurysm formation, and then vessel occlusion due to mechanical obstruction or thrombus. The most severe result is arterial transection, which is usually recognized clinically or on subsequent surgical exploration of the neck.¹¹⁰ Simple dissections are often unrecognized at the time of initial evaluation due to a number of factors. First, the patient will often have coexisting injuries, particularly to the head and face, that may mask the clinical signs of dissection. Second, the onset of clinically significant symptoms is often delayed by hours to days following the injury. Finally, although the signs of cerebral dysfunction or ischemia may be apparent to the emergency clinician, the findings may be attributed to other causes, such as concussion or intoxication.

Mechanism Of Injury

Injuries to carotid and vertebral arteries are most often associated with motor vehicle accidents where the victims sustained significant injuries to the head and cervical spine. Nearly any form of trauma that results in either a direct blow or rotational injury to the head or neck has been reported, including fist fights, hangings, falls, local surgical procedures, and blunt intraoral trauma.¹⁰³⁻¹⁰⁵

Crissey and Bernstein identified 4 fundamental mechanisms of blunt carotid injury and classified them as Types I, II, III, or IV. Type I injuries result from a direct blow to the neck and account for a minority of injuries. Type II injuries are the most common and result from hyperextension and contralateral rotation of the head and neck. Type III injuries result from intraoral trauma. Type IV injuries are associated with skull base fractures that involve the sphenoid or petrous bones.¹¹¹ Regardless of the type of injury, the mechanism for most of these arterial lesions is intimal disruption that exposes the thrombogenic subendothelial collagen surface, promoting platelet aggregation with subsequent

Figure 2. Mechanical Causes Of Dissection⁹²



Manipulation or torsion of the neck has been implicated as a cause of cervicocranial dissection. This drawing depicts mechanical stretching by simultaneous extension and rotation of the neck. The vertebral arteries are most prone to dissect at the cervical foramina from mechanical stress (seen at the origin of the arrow).

embolization, partial thrombosis with low flow, or complete occlusion due to thrombus formation.

The vertebral arteries are particularly vulnerable to injury based on their anatomic relationship to the supporting musculature, tendons, and bony elements of the neck and cervical spine. Cervical fractures and dislocations can cause injury through direct impingement, resulting in intimal disruption, narrowing, or occlusion.^{103,112,113} These injuries most frequently involve the second segment that runs through the transverse foramina of C6 up to C1 due to the fixation of the vertebral artery within the transverse foramina. Ischemia and stroke result from luminal narrowing, complete occlusion, or embolization of thrombus arising from pseudoaneurysms or dissection. Vertebral artery injuries are often clinically asymptomatic when diagnosed through screening protocols.^{114,115} This is because there may be collateral flow from the ipsilateral vertebral artery, or the process of injury-induced thrombus formation and embolism is often delayed.

Presenting Signs And Symptoms Of Cervical Artery Dissection

Spontaneous Cervical Artery Dissection

Headache or migraine is a presenting symptom in 57% to 92% of carotid artery dissections and 69% to 72% of vertebral artery dissections.^{28,39,73,74} The location of the headache pain is neither sensitive nor specific for the artery of dissection; however, the pain from a carotid dissection tends to present as a frontal headache, whereas the pain in vertebral artery dissection is likely to be high posterior or occipital. Conversely, eye, ear, or face pain is very likely to indicate carotid involvement. In one series of 161 carotid and vertebral dissections, frontal headache location was present in 56% of carotid dissection headaches and 5.5% of vertebral dissection headaches, whereas occipital headache location was present in 67% of vertebral dissections and was not present in any carotid dissections. They also found anterior neck pain in 97% of carotid dissections and zero vertebral dissections.⁷³ Posteriorly located neck pain was present in all vertebral dissections with neck pain and 3% of carotid dissection patients. The neck pain of vertebral dissection frequently had a gradual onset, raising concern that it could be mistaken for musculoskeletal pain.^{28,39,73} It has been reported that only one-third of patients with carotid dissections have bruits; therefore, deciding to perform vascular imaging on a patient based on the presence of a bruit is not recommended.^{28,116}

Stroke or TIA is a complication of 73% to 85% of all cervical arterial dissections that present to a hospital, and it may be the presenting symptom in up to 72% of cases.^{30,31,47,73,74} There is an unknown number of patients with cervical arterial dissection who

never develop symptoms and therefore never seek medical attention. The average age of patients who present with stroke is relatively young, 45.9 years; thus, cervical arterial dissection is an important etiology to consider when managing a younger patient with stroke.³⁷ The symptoms created by stroke or TIA from the dissection depend upon which artery dissects. Presenting signs of stroke related to cervical arterial dissection include hemiparesis (87%-100%),^{5,28} hemisensory loss (39%-77%),^{5,28} slurred speech (45%),⁶⁸ aphasia (35%),²⁸ monocular vision loss (6%-25%),^{5,28,73} hemineglect, and gaze deviation. Cranial nerve palsies occur in 10% to 12% of cervical arterial dissections when disruption of the adventitia leads to aneurysm formation.^{46,116,117} Cervical arterial dissection that presents with cranial nerve deficits from aneurysmal dilatation of the internal carotid combined with hemiparesis from occlusion of the carotid at the dissection may be falsely localized to the brainstem. Dysgeusia (abnormal taste) is found in 7% of carotid dissection patients from a lesion of the chorda tympani, and it typically occurs without other CN VII involvement.⁷³ The presence of dysgeusia is highly suggestive of cervical arterial dissection.¹¹⁶ Horner syndrome is present in 20% to 48% of patients found to have a cervical arterial dissection,^{5,30,46,47} but the classic triad of Horner syndrome plus hemispheric stroke symptoms and headache is only found in 8% of cervical arterial dissections.³⁰

Spontaneous vertebral arterial dissection is one-half to one-third as common as cervical arterial dissection. This may be due to imaging technique rather than actual pathophysiologic differences between carotid and vertebral artery dissections. There is a normal asymmetry of the vertebral arteries that may be mistaken for normal when, in fact, there is arterial narrowing due to dissection.^{118,119} In addition, the site of vertebral artery dissection may be at the origin, which is frequently not well visualized or not included in the imaging study.¹¹⁹⁻¹²¹ Alternatively, it may be that fewer patients with vertebral arterial dissection survive to be included in observational studies or registries. Stroke symptoms from vertebral artery dissection have a wider variation compared to carotid artery dissection and have greater potential to be lethal because brainstem perfusion is at risk. Common stroke symptoms due to vertebral arterial dissection that have been reported include incoordination (67%),⁶⁸ lateral medullary syndrome (27%-65%),^{39,122} vertigo or dizziness (52%-57%),^{68,122} unilateral facial paresthesias (47%),¹²² disequilibrium or unsteadiness (42%),^{68,122} vertigo with nystagmus or "cerebellar signs" (25%-33%),^{39,122} isolated hemiparesis or quadraparesis (11%-25%),³⁹ hemisensory loss (11%-25%),^{39,122} visual field defects (15%),¹²² and dysarthria (15%).¹²² Other symptoms include cranial nerve deficits, loss of consciousness, perioral numbness, double vision, slurred speech,

and diplopia.^{39,68,122,123} (See Table 3.) Unlike the carotid artery, where external layer dissection and aneurysmal compression may cause cranial nerve palsies, vertebral artery dissection may compress the cervical nerve roots, resulting in cervical radiculopathy, and occurs in 1% of extracranial vertebral artery dissections.¹²³ This is most commonly seen at C5 and C6, which would result in rhomboid, supraspinatus, infraspinatus, deltoid, bicep, and tricep weakness with a decrease or loss of sensation along the lateral arm and hand.^{123,125-127} Physical examination findings include decreased or absent biceps or brachioradialis reflexes.¹²⁸

Spontaneous Dissection In Children

Strokes will occur in children, often with debilitating outcomes. Cervical arterial dissection is among the constellation of conditions that predispose children to stroke, which include arteriopathy, vascular malformations, congenital heart disease, sickle cell disease, and hematologic abnormalities. Arterial dissection accounts for 7.5% to 20% of childhood arterial ischemic strokes. In a 2008 systematic review of published studies and case reports of children with

cervical arterial dissection, 118 pediatric patients were identified. There was a significant male predominance for both anterior and posterior circulation dissection.¹³⁰ Dissections involving the carotid artery are more common in children, and nearly 14% had experienced multiple ischemic events prior to definitive diagnosis of dissection.^{87,129,130}

In children, cervical arterial dissection may present with slightly different features. In one cohort of 213 children with acute neck dissection, Rafay et al reported that 38% presented with seizures or altered consciousness.² There were also signs of increased intracranial pressure in 63%, speech deficits in 50%, cranial nerve deficits in 37.5%, and visual field deficits in 35%.² Cervical arterial dissection is likely to be detected earlier in children or young adults presenting with focal neurologic findings because a high rate of vascular abnormalities prompts early use of conventional angiography.^{130,131}

Traumatic Dissections

Significant vessel injury should be suspected in the patient with cervical spinal fractures, subluxations, or dislocation; lateral neck soft-tissue injury; Le Fort

Table 3. Presentation Of Spontaneous Cervical Artery Dissection

Symptoms	Location of Dissection	
	Carotid	Vertebral
Pain distribution	<ul style="list-style-type: none"> • Headache or migraine, 36%, 68%,⁶⁷ 65%^{5,68} • Occipital headache, 1%⁶⁷ • Frontal headache, 23%⁶⁸ • Facial or orbital pain, 52%⁶⁷ • Neck pain, 16%,⁵ 26%⁶⁷ • Chest pain* 	<ul style="list-style-type: none"> • Headache or migraine • Neck pain • Neck pain posteriorly • Chest pain*
Neurological deficits, by occlusion	<ul style="list-style-type: none"> • Hemiparesis and sensory loss • Monocular blindness • Hemineglect • Aphasia (if dominant hemisphere) 	<ul style="list-style-type: none"> • Locked-in syndrome • Respiratory failure • Hemianopsia or bilateral visual field loss
Neurological deficits, by emboli	<p>Anterior circulation deficits:</p> <ul style="list-style-type: none"> • Amaurosis fugax (monocular blindness), 17%⁵ • Hemineglect • Hemiparesis, 23%⁵ • Aphasia • Dysarthria • Hemisensory loss, 7%⁵ 	<p>Posterior circulation deficits:</p> <ul style="list-style-type: none"> • Hemianopsia or unilateral field deficit • Ataxia, 20%⁴⁰ • Diplopia • Dysarthria • Upside-down vision • Lateral medullary syndrome (Wallenberg syndrome), 32%⁴⁰: dysphagia, hemiparesis, diplopia, facial weakness, unilateral tinnitus
Neurological deficits by compressive aneurysmal dilatation and disruption of adventitia	<ul style="list-style-type: none"> • Horner syndrome, unilateral miosis, ptosis, anhidrosis, 35%, 20%-48%⁶⁸ • Dysgeusia, 0.5%⁶⁸ • Pulsatile tinnitus, 16%⁶⁸ • Ocular motor palsy: CN III, IV, VI, 2.6%¹¹⁷ • Palsy of CN V, 3.7%¹¹⁶ • Palsy of CN VII, IX-XII, 7.5%,⁷⁴ 12%¹¹⁶ 	<ul style="list-style-type: none"> • Pulsatile tinnitus, 5% • Unilateral radicular weakness (C5-C6 most common), 1%^{123,125,127,128}

*Should raise concern for aortic dissection, which is a contraindication to thrombolytic therapy and should be ruled out prior to tPA.¹²⁴

Note: percentages are from small case cohort series and should only be thought of as approximations.

Abbreviations: CN, cranial nerve; tPA, tissue plasminogen activator.

II or III facial fractures; skull-base fractures involving the foramen lacerum; or neurological abnormalities unexplained by intracranial injuries.^{34,114,115,132-134} The specific neurologic deficits reflect the vessel involved and do not differ significantly from those described for spontaneous dissections. Some notable differences are that headache, face and neck pain, and incomplete or partial Horner syndrome are less frequently noted on initial presentation.^{39,105,135-138} These findings, common to patients with spontaneous dissections, may have been present at the time of injury and either overlooked or attributed to clinically apparent injuries.

Clinical findings that raise suspicion for a vascular injury are an expanding neck hematoma, crepitus, hemodynamic instability without other obvious cause, hemiplegia in the alert patient, and the development of a depressed level of consciousness in the patient who was initially lucid, without obvious head injury, or who had a normal head CT. The significance of the lucid interval with deteriorating level of consciousness has been appreciated by many clinicians and points to the development of cerebral ischemia secondary to vascular injury. These patients require emergent evaluation of all 4 cervical vessels, either through CTA, 4-vessel angiography, or direct surgical exploration.

Differential Diagnosis

The differential diagnosis depends on the presenting complaints and circumstances. Stroke is the leading diagnosis for the patient presenting with focal neurologic deficits, and dissection must be considered when atypical features for classic stroke are present. These include: (1) the patient's age, (2) the lack of risk factors for thrombotic or embolic stroke, (3) neurologic deficits, and (4) the presence of pain. For the patient with subtle or no neurologic deficits, the presenting symptoms will determine the differential diagnosis. For the patient with headache alone, the diagnosis is likely to be missed unless surrounding circumstances (eg, rotational injury, chiropractic manipulation) or subtle neurologic deficits are recognized. In the trauma patient, the key discriminating feature is the presence or development of neurologic deficits in the awake patient. Traumatic injuries are likely to cause focal neurologic deficits, as with epidural and subdural hematomas, which are typically associated with a depression in level of consciousness.

Prehospital Care

The most difficult aspect of carotid or vertebral artery dissection in the prehospital setting is identification of arterial dissection as a possible diagnosis. EMS will be called for complaints of headache,

stroke-like symptoms, or trauma, and transport to a center with the resources to diagnose and intervene is a priority. Protocol-driven care for the patient with suspected stroke or head injury is likely appropriate for cervical arterial dissection as well. The critical action is to get the patient to a stroke or trauma center where the diagnoses can be suspected and treated if found in a timely and appropriate manner.

Emergency Department Evaluation

Making the diagnosis of carotid or vertebral artery dissection is difficult. The signs and symptoms vary and are similar to etiologies that are encountered far more frequently, so the diagnosis is dependent on a high level of suspicion.⁷⁴ Complications of arterial dissection occur mostly in the acute period, so expedient initiation of therapy in the ED could have a significant impact on outcomes. Most EDs have protocol-driven management plans that will likely lead to the correct diagnosis for the patient presenting with stroke. Arterial dissection is not a common cause of neck or head pain alone, but it should be suspected in patients that present with neck pain and stroke.^{5,68} In the absence of stroke, there are few defining features that distinguish dissection from migraine or musculoskeletal neck pain, which are much more common. Dissection should be considered in the patient with head or neck pain that occurred in the context of activities that cause torsion, distraction, or blunt injury to the neck – or even in the context of a cough or sneeze.⁶⁸

Awareness of cervical arterial dissection came initially through case reports, series, and reviews of trauma center experiences, which showed that the overall incidence of cervical arterial dissection was approximately 1 in 1000. Mortality rates from blunt injury to the cervical arteries were 23% to 28% in the 1980s and 1990s, with nearly one-half of all surviving patients having permanent neurologic disabilities.^{101-104,115,137,139} Literature improved the awareness of this injury and focused screening of blunt trauma patients. By the 2000s, the incidences were found to be much higher, 2.4 to 5 per 1000.^{101,114,115} These reports confirmed that the majority of these injuries were clinically occult, and liberal screening for asymptomatic patients with high-risk injuries was endorsed. There have been numerous reports of screening protocols that use high-risk criteria based on retrospective analysis of patients with cervical arterial dissection. The Western Trauma Association (WTA) has proposed a management algorithm based on extensive institutional experience and internal prospective validation over nearly 2 decades of experience.¹⁰⁷ High-risk groups are those who, on presentation, are found to have:

- Arterial hemorrhage from neck, nose, or mouth
- Expanding cervical hematoma

- Cervical bruit in patients < 50 years old
- Focal neurologic deficit
- Stroke on computed tomography (CT) or magnetic resonance imaging (MRI)
- Neurologic deficit inconsistent with head CT

Asymptomatic patients with any of the following mechanisms fall into WTA's high-risk group¹⁰⁷:

- An injury mechanism compatible with severe cervical hyperextension with rotation or hyperflexion
- Le Fort II or III fracture
- Basilar skull fracture with carotid canal involvement
- Diffuse axonal injury with Glasgow Coma Scale score less than 6
- Cervical vertebral body or transverse foramen fractures, subluxation, or ligamentous injury at any level
- Near-hanging with anoxic brain injury
- Seatbelt or clothesline injury with significant neck pain, swelling, or altered mental status

As experience with focused screening protocols builds, there is growing concern that the definition of "high-risk" needs to be expanded to include patients with a wider range of injuries to the head and neck. Raising the clinical suspicion for cervical arterial injury has identified greater numbers of traumatic dissections. It is unknown whether there has been improvement in clinical outcome. Studies that have adopted high-risk screening protocols have a nonrandomized design, so any reported outcomes can only be compared to historical controls. These studies have demonstrated the ability to identify significant injuries prior to the development of stroke. The screening protocols vary with respect to their inclusion criteria, but the largest studies have shown that approximately one-third of screened patients will have an injury to at least 1 vessel.^{34,105,106,115,134,140-143}

Diagnostic Imaging

The typical evaluation of worrisome head and neck pain in the ED is likely to include head imaging with CT and a lumbar puncture to rule out SAH. These tests do not screen for arterial dissections. Awareness and recognition of the disease are key, but so is obtaining the appropriate imaging modality. In recent years, improvement of imaging techniques has led to increased detection of the disease and earlier initiation of treatment.^{1,8} Diagnosis is typically reached in the acute setting when diagnostic studies support clinical suspicion. The gold standard is digital subtraction angiography, which is frequently required to make the diagnosis and exclude other intrinsic vessel abnormalities. (See Figure 3.) CTA or MRA of the head (circle of Willis) and neck

have been used for screening in recent years. CTA has a 90% sensitivity for detection of cervical artery dissection.^{121,144} MRA has a high sensitivity, between 75% to 100%, when compared to digital subtraction angiography.^{118,120,145} When compared directly, CTA has been determined to more accurately evaluate the vertebral arteries for dissection than MRA.¹⁴⁶

Dissection and its complications may show on CTA axial images as a characteristic crescent sign. The characteristic finding of arterial dissection on MRA is a flame-like tapering of the blood vessel. MRI T1 or T2 axial images may reveal an intramural thrombus.^{119,144,147} Conventional angiography may show an irregular tapering, intimal flap, or string sign from flow in a severe stenosis.¹⁴⁸ If MRI/MRA detects a double lumen, mural hematoma with stenosis, or aneurysmal dilatation, an arterial dissection can be assumed to be present, with relative confidence.¹²⁰ (See Figure 4, page 11.)

Ultrasonography is not as sensitive as CTA, MRA, or MRI for detection of extracranial lesions. A lesion that has been detected by a screening modality may be followed for resolution on ultrasonography; however, there is debate regarding the usefulness of ultrasonography to routinely follow extracranial carotid lesions.^{46,150}

Digital subtraction angiography is the gold standard imaging modality due to unmatched spatial resolution. In addition to clear images, angiography provides an assessment of blood flow that is not present on CTA or MRA imaging. Angiography should be used to provide information on collateral blood flow, as this may guide further management and stratify risk.¹⁵¹ Patients with an intact circle of Willis should tolerate an acute occlusion much better than those who do not.⁴⁴

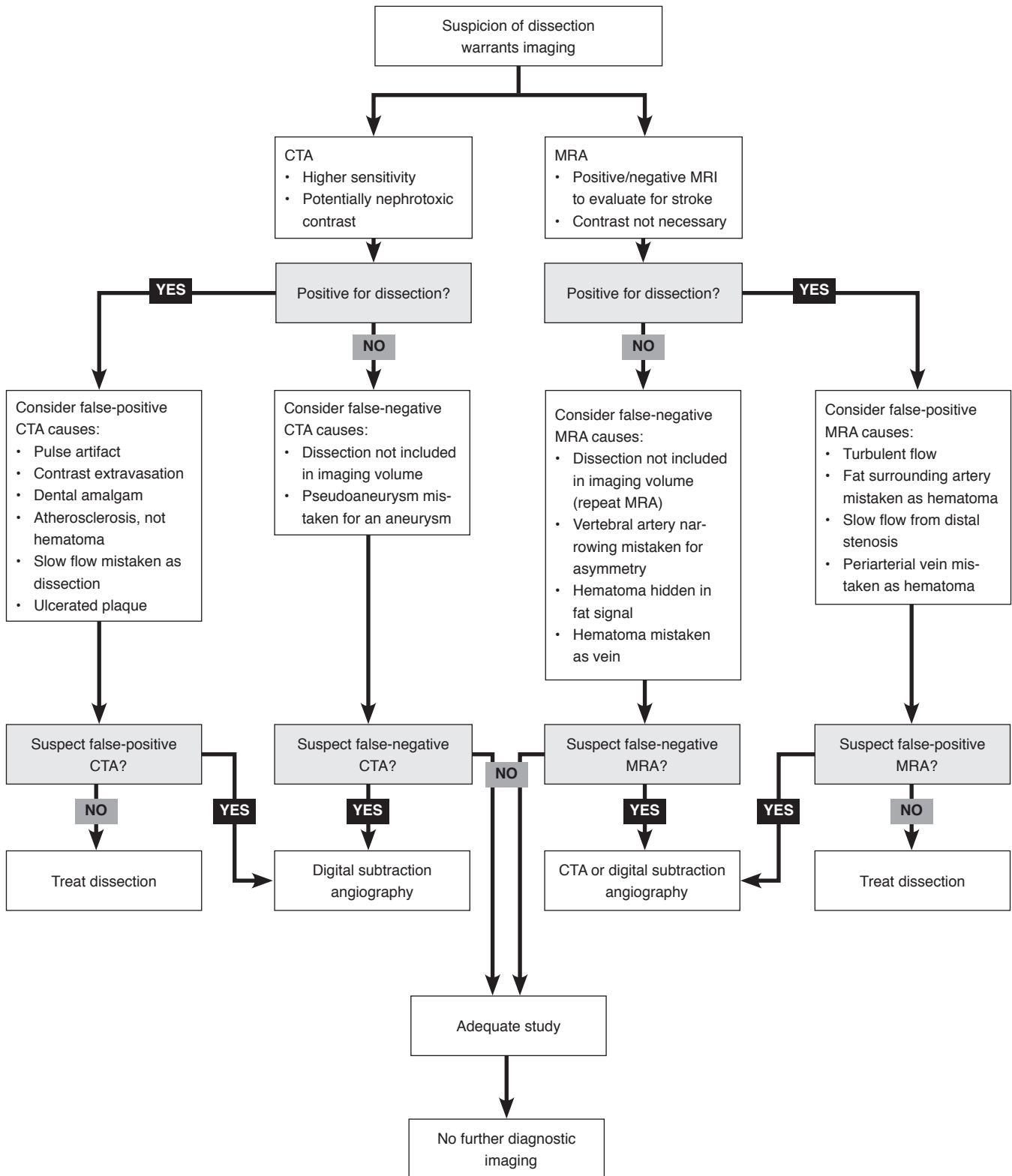
Figure 3. Digital Subtraction Angiography Of Spontaneous Dissection



- A. Significantly reduced flow with presence of a false lumen can be seen in the left vertebral artery, indicating the presence of a vertebral artery dissection.
- B. After a balloon occlusion test of the artery, the artery was coiled.

Used courtesy of Kenneth Shea, MD.

Clinical Pathway For Diagnosing Cervical Arterial Dissection



Abbreviations: CTA, computed tomographic angiography; MRA, magnetic resonance angiography.

Trauma Imaging

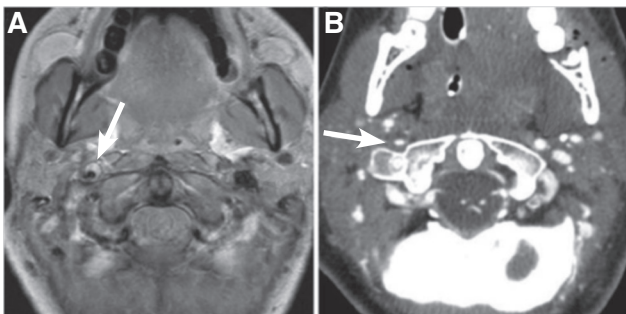
Diagnostic screening in the trauma patient must be performed in the context of the need to screen for coexisting injuries. Although diagnostic imaging of cervical arterial injury with 4-vessel angiography or digital subtraction angiography may be ideal, it is neither readily available nor without risk for many trauma patients at greatest risk for cervical arterial dissection. The single greatest radiological advance in the past quarter-century has been the refinement and increasing use of CT imaging for the diagnosis of surgical disease. CTA is increasingly replacing digital subtraction angiography for screening patients at risk for cervical arterial dissection. Pooled data from 6 studies (1368 patients) published between 2002 and 2006 show a sensitivity of 79% and a specificity of 97% for identification of cervical artery injuries in the trauma setting.^{115,140,152-157} The advantages of CTA are that it is noninvasive, can be performed quickly as part of the trauma screening protocol, and the imaging can be manipulated in 3 dimensions. (See Figure 5) Angiography still has a role in the management of patients with suspected cervical arterial dissection, but it is likely reserved for patients where the CTA is nondiagnostic and cervical arterial dissection is strongly suspected based on the development of neurologic findings and in patients where neuroradiological intervention is necessary.¹⁵⁸

Treatment

Initial Management

The risk of stroke is greatest in the first 24 hours after cervical artery dissection and decreases over the next 7 days. The risk of stroke after 2 weeks is

Figure 4. Imaging Cervicocranial Arterial Dissection¹⁴⁹



- A. Arrow points to intramural hematoma on MRI.
- B. Arrow points to intramural hematoma on CTA.

With kind permission from Springer Science+Business Media:
Emergency Radiology. MRI and MRA for evaluation of dissection of craniocerebral arteries: lessons from the medical literature. Volume 16, 2008, page 190, James M. Provenzale, Figure 4.

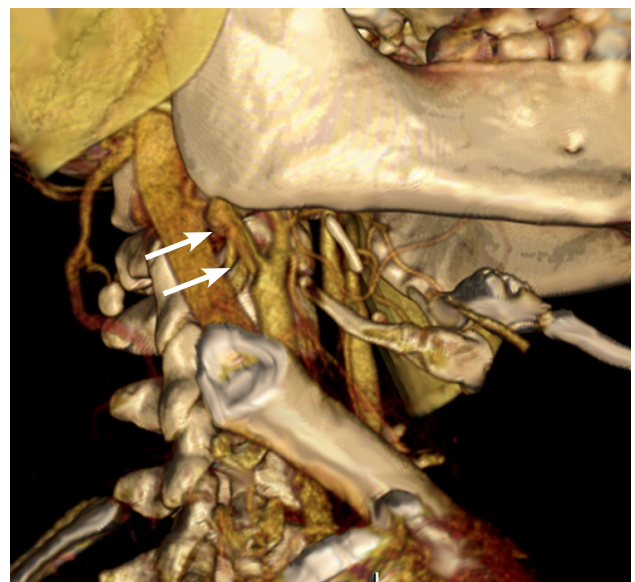
relatively low.³¹ Dissection patients who develop neurologic ischemic deficits are candidates for treatment with IV tPA or endovascular stroke therapy. Systolic blood pressure should not be above 185 mm Hg to minimize the risk of hemorrhagic complication. Hypotension should be avoided as well.

Medical Therapies

tPA is the first medication to be considered for the patient with spontaneous dissections and evidence of acute stroke. It is given in concordance with guidelines for typical ischemic stroke. Early reports suggest that cervical arterial dissection can be managed safely with IV tPA.^{23,24,26} Patients with stroke due to cervical arterial dissection were included in the National Institutes of Health (NIH) National Institute of Neurological Disorders and Stroke (NINDS) IV tPA trial without adverse events.^{21,23,159} Dissection patients who receive tPA do not have as much improvement as patients with nondissection acute stroke who receive tPA. In 1 study comparing outcomes in stroke patients given tPA, 36% of cervical arterial dissection patients showed stable neurologic improvements compared to 44% in the comparison group.²⁶ Lytic therapy should not be given if it is recognized that the dissection enters the skull (due to risk of intracranial hemorrhage) or if the aorta is involved (due to the risk of aortic rupture).^{124,160-164}

After the patient has been treated acutely, anticoagulation or antiplatelet therapy is the mainstay of therapy for all dissections. Antiplatelet therapy may

Figure 5. CTA Reconstruction Images With Focal Stenosis At The Origin Of The Carotid Artery (Arrows)



Used courtesy of Kenneth Shea, MD.

be started 24 hours after tPA is administered. While the true risk of stroke from dissection is unknown, it is clear that antiplatelet and anticoagulant therapy will reduce the risk of stroke in symptomatic patients without neurologic deficits. The benefits of aspirin or anticoagulation appear to be similar, and any differences in outcomes have been observed in the risk of recurrence. There is a risk of hemorrhagic complications in patients receiving anticoagulation that is clinically significant for dissections that extend intracranially, but is not thought to be a contraindication in patients with extracranial dissections.¹⁶⁵ A patient with severe neurological deficits or National Institutes of Health stroke scale (NIHSS) score ≥ 15 should not be placed on anticoagulation acutely due to a risk of hemorrhagic transformation.^{18,166} Dissections presenting with cranial neuropathies, aneurysmal dilatation, Horner syndrome, or cervical root compression may have a decreased risk of ischemic stroke, which makes antiplatelet therapy a favored choice.⁷⁴ Patients with evidence of emboli or free-floating thrombus at the site of dissection or multiple ischemic events may require full anticoagulation.^{167,168} (See Table 1, page 3.)

Endovascular Therapy

In the acute phase of stroke due to cervical arterial dissection, the goal is reconstitution of blood flow in the dissected blood vessel. There is evidence that endovascular therapy may be superior to IV tPA in patients with "tandem" lesions when there is a dissected vessel and embolic stroke downstream.²⁵ Endovascular therapy is an option for patients with contraindications to lytic therapy. Preliminary research indicates that improved outcomes are associated with reconstitution of flow within 6 hours, which expands the therapeutic window beyond that for lytic therapy.^{23,25,26} Endovascular therapy may also reduce the risk of stroke by stenting the dissecting vessel. Aneurysms complicating dissections can be filled with soft coils to induce a thrombosis. This method is a definitive treatment of aneurysms, and long-term rates of rupture are very low.¹⁶⁹⁻¹⁷²

Current AHA guidelines recommend proceeding to endovascular therapy for extracranial carotid or vertebral artery dissection when medical management fails.^{173,174} Initiation of medical therapy with tPA, antiplatelet therapy, or anticoagulation does not exclude patients for endovascular therapy; however, when the medical therapy fails it may be too late to prevent neurological deficits. There are no factors to predict the patients for whom medical therapy will fail and the patients for whom it will be successful. Endovascular stroke therapy may be pursued immediately following or during IV tPA infusion.^{25,175}

Treatment For Traumatic Dissections

Early anticoagulation will reduce the risk of stroke.^{104,115,156} While there are no randomized pro-

spective trials that demonstrate improved outcomes in those patients receiving therapies, it is clear that the rationale behind aggressive screening protocols is that early detection and therapy improves outcome. A 2002 study by Miller et al reported outcomes of 139 traumatic cervical dissections, of which 75 involved the carotid artery and 64 the vertebral artery.¹¹⁵ Forty-three of the 75 carotid artery injury patients and 39 of the 50 vertebral artery injury patients were treated before development of ischemia. The stroke rate for treated vessels (heparin, antiplatelet therapy) with carotid artery injuries was 6.8% compared with 64% in untreated vessels ($P < 0.001$). Treated patients with vertebral artery injuries had a stroke rate of 2.6%, whereas untreated patients developed stroke 54% of the time. In 2004, the Denver group reported the outcomes in 114 patients with carotid injuries. Seventy-three patients underwent anticoagulation after diagnosis (heparin in 54, low-molecular-weight heparin in 2, antiplatelet agents in 17); none had a stroke. Of the 41 patients who did not receive anticoagulation, usually due to contraindications to anticoagulation, 46% developed neurologic ischemia. Ischemic neurologic events occurred in 100% of patients who presented with symptoms before angiographic diagnosis and those receiving a carotid coil or stent without anticoagulation.¹⁷⁶

Endovascular stenting has been used in select patients who have contraindications to anticoagulation or are demonstrating neurologic deterioration on therapy. Early results are encouraging, but experience with this modality and data on late follow-up are still very limited.^{177,178} A 2011 survey of trauma surgeons, intensivists, and interventionalists indicated that the most commonly preferred treatment for traumatic cervical arterial injuries was anticoagulation (42.8%) and antiplatelet drugs (32.5%). Patients with intraluminal thrombus and no related symptoms were preferably treated with heparin and/or warfarin (65.7%), followed by antiplatelet drugs (22.9%) and thrombolytics (6.2%). Some 20.7% of the respondents recommend treatment of asymptomatic dissections and traumatic aneurysms with endovascular techniques, while 2.7% would not, and 51.6% would do so only if there was worsening of the lesion on follow-up imaging.¹⁰⁸

Treatment strategies clearly need to be tailored to the individual patient, injury, and associated injuries. A 2011 study reported the outcome of 222 patients with 263 blunt cerebrovascular injury (BCVI) — 115 carotid injuries and 148 vertebral injuries — and 22 patients had ischemic strokes before their angiographic diagnosis. Treatment options included endovascular treatment (41%), heparin drips (50%), aspirin (76%), and clopidogrel (52%), respectively. Seven patients developed infarcts after BCVI diagnosis, for a postdiagnosis rate of 4%.¹⁷⁹

Special Circumstances

Intracranial Dissections

In intracranial dissections, the intracranial portion of the vertebral artery wall becomes comparatively weak, which increases the risk of rupture and SAH.^{82,180} Intracranial dissection frequently results in aneurysmal dissection due to the differences in the intracranial vascular wall. Ninety percent of intracranial vertebral artery dissections began extracranially. Mechanical forces generated by the dural membrane can be a source of intracranial vertebral dissection. Patients with intracranial dissections have a younger average age, between 20 and 35 years old.¹³¹ Primary dissection of the anterior, middle, and posterior cerebral arteries are rare.^{148,181}

The most common presenting feature of intracranial dissection is infarct; however, the most significant contribution to morbidity and mortality is SAH.^{161-165,182} Symptoms include sudden onset of severe headache that may be associated with syncope, vomiting, and alteration in level of consciousness and abrupt neurologic deterioration.^{183,184} Terson syndrome (vitreous hemorrhage associated with SAH) may be present in 20% of vertebral dissection-associated SAHs.¹⁸⁵ Ischemic infarct is seen in 84% of intracranial dissections without hemorrhage.¹⁶⁵ Severe headache is frequently seen with ischemic stroke from intracranial dissection even in the absence of SAH.^{123,165}

No randomized controlled trials of anticoagulation or antiplatelet therapy have been performed for intracranial dissections. In intracranial dissection, anticoagulation has been shown to lead to increased death and disability compared with antiplatelet therapy.¹⁶⁵ Thrombolysis has been performed in a few patients but has never been effectively studied.

There is a high risk of rebleeding in the first 24 hours of intracranial aneurysmal dissection with SAH. Rebleeding rates have been reported between 57% and 58% in the first 24 hours and are associated with a very high mortality of between 50% and 79%.^{163,186} Endovascular or surgical treatment significantly decreases risk of rebleeding, and there are small series published that show no rebleeding events after endovascular treatment.¹⁸⁷ Endovascular treatment of dissecting aneurysms has been shown to decrease mortality to 20% to 27%.^{163,164,186} While the ability to treat intracranial dissection safely with endovascular techniques has been demonstrated, the impact of long-term outcome has not been conclusively studied.^{164,186}

Controversies And Cutting Edge

Endovascular Therapy

Medical and endovascular management of carotid and vertebral artery dissection is an area of current research. It is safe to give tPA to patients with stroke from dissection despite concern for enlarging the hematoma.^{21,23,25-27} Dissection will resolve in 80% of patients treated with antiplatelet or anticoagulation, without complication.²⁴ The low number of patients identified to have stroke from dissection limits the number of patients for enrollment into studies at any single institution. In the past few years, endovascular techniques have been employed in symptomatic dissections refractory to medical therapy; however, at this time there are no criteria to selectively predict high-risk patients that will have neurological deterioration.¹⁸⁸ Therefore, it is unknown which patients would benefit from endovascular therapy as a prophylactic measure against stroke.

Blood Pressure/Volume Enhancement

A randomized trial of albumin infusion to improve cerebral perfusion in acute ischemic stroke, known as Albumin In Acute Stroke (ALIAS), is currently underway.¹⁸⁹ Treatment of acute stroke with blood pressure augmentation with vasoactive or inotropic agents is being considered, but is not currently standard practice.¹⁹⁰⁻¹⁹⁴ Pilot studies demonstrating safety of increasing blood pressure by 10% to 20% (not exceeding 200 mm Hg) have been performed, and further investigations are underway.¹⁹⁰⁻¹⁹⁶

Stroke Risk Assessment Using Ultrasonography

Methods of identifying patients at increased risk of stroke from dissection have included transcranial Doppler ultrasound techniques. Transcranial Doppler ultrasound studies of patients presenting within 24 hours of dissection showed that microemboli were recordable downstream from the dissection in 46% of patients and were predictive of future ischemic events without medical therapy. After initiation of heparin, 70% of patients had complete resolution of microembolic signals.^{167,168}

Disposition

Spontaneous Dissection Prognosis

Extracranial cervical artery dissections that are detected in the hospital setting will be complicated by stroke within minutes to 24 hours in 36% to 56% of patients, and 78% to 82% will develop a stroke within the first 7 days of dissection warning symptoms.^{31,47} The risk substantially decreases over time, but stroke after 2 months of dissection is rare.⁴⁸ The outcomes are highly variable and are dependent on numerous factors such as stroke on presentation,

NIHSS score, age, the artery involved, and whether there is occlusion, presence of SAH, presence of dissecting aneurysm (intracranial), multiple dissections on presentation, or recruitment of collateral blood flow within 12 hours.^{47,48,151,197,198} Those who had occlusion of a dissected artery have worse outcomes of death and disability than those who presented with some flow or incomplete occlusion.^{47,199} Some studies have found that outcomes from an occluded internal carotid artery due to dissection have a worse outcome than from atherosclerotic disease.^{41,199} (See Table 4.)

Dissection recurrence occurs in 10% to 28% of cervical artery dissections, and the period of greatest risk is within the first 2 months after dissection. Patients with involvement of 2 cervical arteries are at a 4 times higher risk of recurrent dissection.^{48,197} Triple and quadruple dissections on presentation are rare, at 1.5% and 0.1% of spontaneous dissections, respectively. They are more likely to occur in women, and the risk of recurrence is the same as having 2 arteries involved.²⁰⁰ The risk of recurrent dissection is increased in patients who have a family history of arterial dissection and for those who present with dissection at a young age (< 45 years). For those with a known family history, recurrence rates as high as 50% to 100% have been reported in prospective studies and as low as 6% to 22% for those without.^{29,33,131}

For intracranial dissections, the overall prognosis is guarded compared to extracranial dissections. The presence of SAH with intracranial dissection portends the worst prognosis.^{82,183,202} There is some evidence that the size and location of the dissected vessel may impact the outcome of intracranial dissection. In a series of anterior cerebral artery dissections, a better prognosis was seen in patients that had distal dissection.¹⁸¹

Traumatic Dissection Prognosis

Aggressive screening protocols for dissection will identify increased numbers of cervical dissections before development of stroke symptoms. The ultimate rationale for these protocols is that early detection will reduce the incidence of stroke and death. Cervical injuries were classified in the 1990s with respect to treatment and prognosis, and this classification has remained viable for injuries to the carotid artery. Grade I injuries are mild intimal injuries that usually heal, regardless of therapy. Dissections or hematomas with luminal stenosis (grade II) are high-risk for progression and embolization, and anticoagulation remains the mainstay of therapy. Grade III injuries are the formation of pseudoaneurysms and usually require endovascular stent placement. Occlusions are grade IV injuries and usually do not recanalize. Grade V injuries are transections that are lethal and refractory to intervention.^{112,143} If untreated, carotid artery injuries are associated with a stroke rate of up to 50%, depending on injury grade, with increasing stroke rates correlating with increasing grades of injury. The bulk of the literature over the past decade has suggested that early anticoagulation in patients with BCVIs reduces stroke rates and resultant neurologic morbidity.^{143,156,176,179,203,204}

Complications from vertebral arterial injuries are also reduced with treatment, and their response to treatment appears to be independent of injury grade. The Denver Health Medical Center has a longstanding aggressive screening protocol for cervical arterial injuries, and they reported in 2000 that neurologic complications from vertebral arterial injuries were not related to injury grade. In their series, the incidence of stroke related to vertebral injury was 24% and the likelihood of stroke in spite of intervention was not related to injury grade.¹⁰⁰

Table 4. Prognostic Factors Of Head And Neck Arterial Dissections

Artery	Factors Improving Outcome	Factor Worsening Outcome	Factors Increasing Risk of Recurrence
Internal carotid	Spontaneous recruitment of collateral arteries within 12 hours ¹⁵¹	Occluded artery ⁴⁷	Bilateral involvement ^{10,199}
Vertebral	Extracranial	<ul style="list-style-type: none"> Intracranial SAH Dissecting aneurysm 	Bilateral involvement ¹⁹⁹
Intracranial	Distal vessel involvement ¹⁸⁰	<ul style="list-style-type: none"> Proximal vessel involvement¹⁸⁰ SAH 	
Any cervical artery	<ul style="list-style-type: none"> Collateralization of arteries within 12 hours¹⁵¹ Low NIHSS score¹⁹⁶ Young age¹⁸¹ 	<ul style="list-style-type: none"> Occluded artery⁴⁷ High NIHSS score¹⁶⁶ Older age²⁰⁰ 	<ul style="list-style-type: none"> Family history^{29,47} Multiple dissections on presentation¹⁹⁶

Abbreviations: NIHSS, National Institutes of Health Stroke Scale; SAH, subarachnoid hemorrhage.

It has been speculated that nonocclusive vertebral artery injuries are potentially more dangerous than those in the carotid artery for a number of reasons. First, they are likely to be clinically asymptomatic on initial presentation due to collateral flow from the contralateral artery, and recognition of an injury is delayed until neurologic sequelae develop. Second, intimal injuries, while nonocclusive, serve as substrate for platelet aggregation, clot formation, and embolic stroke. The stroke rate is higher in occlusive injuries of the carotid artery, presumably because the internal carotid circulation is less able to compensate for an interruption in flow.

Time- and Cost-Effective Strategies

- Elicit key features of the history:
 - Pain or neurologic deficit onset during activities such as sneezing, coughing, sex, rollercoaster riding, chiropractic manipulation, neck stretching, weight-lifting
 - New or change in headache quality in a chronic headache patient
 - Presence of dysgeusia, pulsatile tinnitus, or personal or family history of dissection
- Evaluate patients with headache or neck pain with a careful neurological examination, giving close attention to cranial nerve deficits, Horner syndrome, lateral arm numbness, and deltoid and/or biceps weakness.
- In headache- or neck-pain patients, improve your physical examination sensitivity for detecting Horner syndrome by turning down the lights in the room to elicit anisocoria.
- For patients being considered for IV tPA, expedite blood draw of complete blood count, electrolytes, international normalized ratio, and prothrombin time, and obtain a noncontrast CT prior to angiography.
- Obtain CTA or MRA with trauma imaging in patients where there are neck or skull-base injuries to prevent delay in making the diagnosis.
- Consider favoring CTA over MRI/MRA for screening for vertebral artery dissections.
- Obtain CTA or MRA for screening for carotid artery dissections.
- Quickly initiate antiplatelet or anticoagulation therapy to prevent stroke and/or recurrent stroke.
- Avoid blood pressure depression below the patient's baseline blood pressure to avoid risk of worsening of neurological deficits.

Summary

Carotid and vertebral artery dissections are a significant cause of stroke, and disproportionately more so in younger individuals. The mechanism of stroke is twofold, from occlusion at the dissection and distal emboli. Dissections are detected in patients with headache and neck pain alone. It is suspected that some underlying disease predisposes individuals to spontaneous dissections. Commonly, there is a mild mechanical event associated with a spontaneous dissection. Traumatic dissections should be suspected whenever there is evidence or suspicion of injury to the neck or head, and screening should not wait for development of neurologic signs or symptoms.

When a carotid or vertebral artery dissection is suspected, a CTA or MRA is an appropriate screening test; however, when clinical suspicion is high, digital subtraction angiography is necessary to rule out the diagnosis. In trauma, early consideration of dissection will help the emergency clinician obtain angiography to piggyback on other diagnostic imaging.

There are no randomized controlled data to guide management of cervical arterial dissection; however, acute stroke treatment secondary to dissection includes thrombolytic therapy with IV tPA despite concern for worsening the dissection. Endovascular therapy compares favorably with thrombolytics in patients with an embolic stroke in the vascular territory of the dissection (tandem lesions), and it may be used in patients with contraindications to thrombolytic therapy.

The majority of patients who develop an arterial dissection will not develop a stroke. Stroke prevention is with antiplatelet or anticoagulant therapy. Trials have not been performed to compare the efficacy or safety of either. An increased risk of hemorrhage in intracranial dissection and hemorrhagic conversion with large infarcts (NIHSS score > 15) favors treatment with antiplatelet therapy. Ultrasonic or clinical evidence of emboli while on antiplatelet therapy or presence of a pseudoocclusion favors placement on anticoagulation.

Case Conclusions

A subdural or epidural hematoma would have to be extensive to cause hemiparesis, hemisensory loss, and neglect. A Todd paralysis after seizure was possible, but was considered only after ischemic causes were ruled out. Since the initial noncontrast head CT showed a small subdural hematoma, ischemic stroke was the next most-worrisome possibility. Concern for dissection should be raised when ischemic stroke is considered in the setting of trauma. A CTA was obtained that showed near occlusion of the right

internal carotid artery. IV tPA was not administered for this traumatic dissection for concern of worsening or creating hemorrhagic complications. Interventional neuroradiology was consulted immediately, and the patient was placed on a heparin infusion as a bridge to the procedure. Stenting of the vessel was

performed, and though it was not successful in reversing her neurological deficits, it may have prevented further ischemic damage.

The CT brain was negative, and you obtained an MRA to rule out vertebrobasilar insufficiency. The imaged showed diffuse narrowing of the left vertebral

Risk Management Pitfalls For Cervical Arterial Dissection

(Continued on page 17)

1. **"We did a complete headache evaluation that included a noncontrast head CT and lumbar puncture that had no red blood cells. Her neuro exam is nonfocal. Let's send her out with tramadol and neurology follow-up."**

If the patient has not responded to headache cessation therapy in the ED, consider that a single-center study showed that 20 of 245 (8%) patients with headache or neck pain alone were found to have a cervical arterial dissection in the absence of neurological deficits. One-quarter of the patients with dissection actually had multiple dissections.⁷⁵

2. **"She recently started visiting a chiropractor for neck pain. This pain is different from her previous migraines. Her headache sounds like a migraine, with a visual aura, slow onset, and pulsation. She has light sensitivity and is starting to get nauseated. Let's rule out SAH and see if she gets better with some IV compazine."**

Not suspecting cervical arterial dissection in the beginning of the evaluation results in a delay to diagnosis. We have all seen 6 to 12 hours go by while trying to abort the migraine with medications and then an MRI or MRA shows intracranial pathology. At the onset of headache in those found to have carotid dissections, 92% reported an ipsilateral headache, 25% a pulsating headache, and 85% a gradual onset, all classic features of migraine.^{73,75} Migraineurs who develop dissection typically describe a pain that is different from usual, and dissections are found in those who have accompanying aura, nausea, vomiting, photophobia, and phonophobia.⁷⁵

3. **"The patient was rear-ended by someone in the grocery store parking lot. We cleared his c-spine, and his collar was removed. He developed a headache and feels "weird." There is no evidence of seat belt injury or external hematoma on his neck. He had a nonfocal neuro exam, and he has no bruits. The radiology resident is on the phone and wants to cancel the CTA. They are saying he does not meet criteria for requir-**

ing a CTA, and since there are no bruits, the study is going to be negative."

Tell radiology that you are not cancelling the CTA order. Although this patient does not have any strong symptomatic indications such as a Le Fort II or III fracture, basilar skull fracture, bruits, or external neck hematoma, and he is awake and alert, his neck was rotated and he may have hit his neck on the seatbelt. Presence of a bruit is not sensitive or specific for carotid stenosis or dissection. Only one-third of patients with carotid dissection have a bruit.^{28,116}

4. **"A 14-year-old boy fell off of his bike after hitting a mailbox and then had a seizure. The noncontrast CT head and c-spine were normal. I should do a neurological examination and clear his c-spine."**

In pediatric patients, consider a seizure as a manifestation of dissection. If there is clinical suspicion for neck injury followed by a seizure, vascular imaging is warranted.

5. **"He had acute onset of right-sided weakness and aphasia. His last known normal time was 2 hours ago. CT ruled out hemorrhage, and CTA showed an extracranial left carotid dissection. I wish we could give IV tPA, but we can't because that will just extend the hematoma and make things worse."**

The patient should be considered for IV tPA as long as he does not have other contraindications to receiving tPA. A few cohort studies have reported that patients with stroke secondary to dissection who received tPA improved compared to those who did not receive tPA. The rate of hemorrhagic complications is low or absent in these studies, and the authors felt the benefits outweigh the potential risks.^{21,23,25,26,175} Alternatively, endovascular therapy has been reported as superior to IV tPA for a proximal carotid dissection with distal MCA occlusion, "tandem lesions."²⁵

artery, with a left cerebellar infarct. The patient was started on heparin and continued to have intermittent symptoms. He was taken to interventional radiology, and his left vertebral artery was temporarily occluded without exacerbating symptoms, so the vessel was occluded to prevent embolic infarcts from the dissecting

vessel. He was asymptomatic from neurologic deficits at his 1-year follow-up appointment. He was advised to refrain from head-banging in the future.

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6. **“The CTA report for the 41-year-old female with Horner syndrome is normal except for a focal stenosis of the extracranial internal carotid artery that is not hemodynamically significant. There is no presence of mural hematoma to suggest carotid dissection, but that cannot be excluded. So her Horner syndrome is probably unrelated.”**

Not so fast. Sometimes detection of a mural hematoma is missed on either a CTA or MRI T1 fat suppression sequences for various reasons. Further, the resolution of CTA (or especially MRA) limits the ability to clearly see the intimal flap of a dissection. If dissection is suspected on the same side of a patient presenting with Horner syndrome and a focal stenosis is seen, follow-up studies should be obtained. The patient should probably have an MRI of her neck with “fat suppression sequences” or a digital subtraction angiogram.^{147,119,144}

7. **“We should wait to see what interventional radiology says before starting a heparin drip because they may want to place a stent.”**

Endovascular neurosurgical procedures can be performed if a patient is anticoagulated, on antiplatelet therapy, or both. During procedures, heparin boluses and heparinized saline are used to prevent thrombi from forming on the catheters or if the catheter disrupts the intima during a procedure. Since the risk of stroke or recurrent stroke is highest in the first 24 hours of dissection, treatment with anticoagulation or antiplatelet therapy should be initiated immediately.³¹

8. **“There was an intimal flap on the CTA, but the patient has no neurological deficits. The MRI was negative for stroke as well. He really wants to leave. I guess we don’t need to admit him; we could place him on aspirin and send him home.”**

There is a strong argument that the patient should be admitted. Some patients fail medical therapy in the first few days and require endovascular therapy, so a period of observation

or admission is warranted.^{19,27,123,176,177,187,196} It is also reasonable to obtain transcranial Doppler ultrasound with microemboli signal monitoring because patients who have emboli are at a higher risk of stroke.^{44,167,168} Furthermore, serious consideration should be given to whether the patient should be placed on anticoagulation instead of antiplatelet therapy because no randomized trials have ever been able to adequately compare the 2 treatments due to insufficient sample sizes.

9. **“We can’t start anticoagulation or antiplatelet therapy on the patient because she was in a motor vehicle collision and she could develop internal or intracranial bleeding.”**

Given the high risk of stroke after traumatic dissection (which is around 64% in carotid artery dissection and 54% in vertebral artery dissection), there should be a strong effort to ensure the patient is not bleeding by performing diagnostic imaging followed by initiation of antiplatelet or anticoagulant therapy. Treatment reduced stroke from carotid dissection to 6.8% and to 2.6% in vertebral dissection.¹¹⁵

10. **“Ms. Smith was sent in from clinic where a carotid ultrasound showed a right carotid dissection. She was given 325 mg of aspirin. The neurology consult resident thinks she can go home on aspirin and follow-up in clinic. That is OK because no benefit for anticoagulation over antiplatelet therapy has ever been demonstrated.”**

The patient should not be sent home without a full assessment of her vasculature. Patients who present with a dissection have multiple arteries with dissection approximately 6% to 28% of the time.^{7,48,74,87,131,196,199} If the patient were to have a vertebral artery dissection extending intracranially, she would be at significant risk of SAH.

This Month In *EM Practice Guidelines Update*

The April 2012 issue of *EM Practice Guidelines Update* reviews 2 guidelines on non-ST-elevation myocardial infarction (NSTEMI): (1) the 2011 American College of Cardiology Foundation/ American Heart Association guideline and (2) the 2010 National Institute for Health and Clinical Excellence (NICE) guideline from the United Kingdom. Subscribers to *Emergency Medicine Practice* have free access to this online publication at www.ebmedicine.net/NSTEMI.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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- An arterial dissection initially occurs when what is disrupted?**
 - Intima
 - Media
 - Adventitia
 - Thrombosis
- Cervical artery dissections are dissections of which arteries?**
 - Uterine
 - Carotid
 - Vertebral
 - Subclavian
 - B and C
- What is the current gold standard for screening and for diagnosing cervical dissections?**
 - MRA
 - CTA
 - Conventional angiography
 - Ultrasound
- Which of the following diseases is not known to have an increased risk for dissection?**
 - Fibromuscular dysplasia
 - Ehlers-Danlos syndrome
 - Osteogenesis imperfecta
 - Marfan syndrome
 - Insulin-dependent diabetes mellitus
- Vertebral artery dissection has a lower incidence than carotid artery dissection.**
 - True
 - False
- What is the most common mechanism of blunt carotid injury identified by Crissey and Bernstein?**
 - Type I - Direct blow to the neck
 - Type II - Hyperextension and contralateral rotation of the head and neck
 - Type III - Intraoral trauma
 - Type IV - Skull-base fractures

7. All of the following clinical findings raise suspicion for a vascular injury EXCEPT:
 - a. Expanding neck hematoma
 - b. Crepitus
 - c. Bilateral miosis
 - d. Hemodynamic instability without other obvious cause
 - e. Hemiplegia in the alert patient

8. After cervical artery dissection, the risk of stroke is greatest in the
 - a. First 24 hours
 - b. First 7 days
 - c. After 2 weeks
 - d. After 2 months

9. Tissue plasminogen activator (tPA) should not be given if the cervical dissection extends into the skull or if the aorta is involved.
 - a. True
 - b. False

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