# Carotid and Vertebral Artery Dissections

Lauren E. Mamer, MD, PhD[a,*], Robert Stephen, MD[b]

**KEYWORDS**

• Carotid • Vertebral • Artery • Dissection • Stroke

**KEY POINTS**

- Carotid and vertebral artery dissections are a disproportionate cause of stroke in patients 50 years of age and younger.
- These dissections are often associated with a traumatic mechanism, although it may be minor or unnoticed at the time.
- Treatment consists of a risk/benefit approach with antiplatelet or anticoagulant medications and typically is continued for 3 to 6 months.

## INTRODUCTION

Carotid and vertebral artery dissections, referred to collectively as cervical artery dissection (CeAD), are an infrequent cause of ischemic stroke (2%) that disproportionately affects patients younger than age 50 (up to 25% of strokes in this group).[1,2] Presenting symptoms can range from subtle headache and neck pain to cerebral or retinal ischemia. Diagnosis is made more challenging still by the fact that some patients do not have or do not report a clear traumatic mechanism for dissection.[3] In a younger patient population with a low baseline incidence of stroke and a variably present preceding mechanism for dissection, the diagnosis of CeAD can be easily delayed or missed.[4] This review focuses on the epidemiology, pathophysiology, diagnosis, and treatment in these patients.

## EPIDEMIOLOGY

CeAD is a relatively rare event and was not truly recognized until the mid-twentieth century. The first clear case of a spontaneous dissecting aneurysm of the internal carotid artery was described by Anderson and Schechter in 1959,[5] whereas the first

[a] Michigan Medicine, Department of Emergency Medicine, 1500 E Medical Center Drive, TC B1 220, Ann Arbor, MI 48109-5305, USA; [b] Department of Emergency Medicine, University of Utah School of Medicine, 30 N Mario Capecchi, HELIX Building, Level 2 South, Salt Lake City, UT 84112, USA
* Corresponding author.
*E-mail address:* lmamer@umich.edu

Emerg Med Clin N Am 44 (2026) 239–248
https://doi.org/10.1016/j.emc.2025.08.015 **emed.theclinics.com**

| Abbreviations | |
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| AHA | American Heart Association |
| CeAD | cervical artery dissection |
| CTA | computed tomography angiography |
| DOAC | direct oral anticoagulant |
| DSA | digital subtraction angiography |
| MRA | magnetic resonance angiography |
| TIA | transient ischemic attack |
| VKA | vitamin K antagonist |

report of spontaneous vertebral dissection is credited to a Canadian neurologist, C. Miller Fisher in their case series in the 1970s.[6]

CeAD is an uncommon cause of ischemic stroke with an incidence that has been reported to vary widely across different studies and populations. The American Heart Association (AHA) notes an incidence of 2.6 to 3.0 per 100,000 people, but this is based on older studies.[7] A study from Pisa, Italy reported an incidence rate of 1.88 per 100,000 person-years for cerebrovascular events owing to CeAD.[8] However, a more recent population-based study in Olmsted County, Minnesota found an overall incidence rate of 4.69 per 100,000 person-years, with a notable increase from 2.30 per 100,000 person-years in 2002 to 2006 to 8.93 per 100,000 person-years in 2017 to 2020.[9] It is thought the true incidence is likely even higher, possibly because some patients with dissection may not seek medical attention owing to self-limited or minor symptoms. The increasing use of noninvasive vascular imaging has likely contributed to this rising detection rate of CeAD, with one study finding an increase in Emergency Department use of computed tomography angiography (CTA) of the neck of 1300% and CTA of the head of 1100% in the period from 2007 to 2017.[10]

Although uncommon, CeAD accounts for approximately 2% of all ischemic strokes.[11] However, it is a significant cause of ischemic stroke in patients less than 50 years of age, responsible for up to 25% of events in this cohort.[12] The mean age at diagnosis is approximately 44 to 50 years.[7,13] The literature is inconsistent with regard to a sex preference for CeAD, with some studies showing a slightly higher risk in men, and others showing a slightly higher risk in women.[7,14] The most common comorbidities noted in the Olmstead County study were a history of smoking (39.8%), migraine headache (36.6%), hyperlipidemia (36.6%), and hypertension (35.8%).[9] Although most of these comorbidities are not unusual in patients with vascular pathologic condition, the presence of a history of migraine headache is important to emphasize because there is a well-known link between migraine headaches, mostly without aura, and CeADs.[15] Although most patients with a diagnosis of CeAD will not have a connective tissue disorder, people with known connective tissue disorders, such as fibromuscular dysplasia, Ehlers-Danlos, or Marfan, do have an increased risk of CeAD.[16,17] Interestingly, although the number of patients studied was small, even when an established diagnosis of a connective tissue disorder is not present, clinical signs of connective tissue abnormalities, including joint hypermobility, skin hyperelasticity, and craniofacial dysmorphisms, are more frequent in patients with CeAD.[18] Indeed, a skin biopsy study showed that 68% of CeAD cases have connective tissue ultrastructural abnormalities.[19]

## PATHOPHYSIOLOGY AND PROGNOSIS

In CeAD as in other types of dissection, damage or weakness in the artery wall allows blood to flow between the layers of the arterial wall, resulting in hematoma formation[20]

and causing ischemic injury distal to the dissection. This ischemic injury occurs through one of two mechanisms. In the first, the hematoma expands to such a degree that the true lumen of the artery becomes stenosed or occluded (estimated to be <10% of patients) causing distal hypoperfusion. In the second and more common scenario, involvement of the intima can cause the development of an intravascular clot with thrombosis that can break off and obstruct blood flow to the downstream tissue in an embolic stroke mechanism.[21]

More relevant for clinical care is the origin of the vessel injury, because certain historical features can suggest CeAD as an important diagnosis to consider. CeAD can be spontaneous, associated with genetic or environmental factors, or associated with a traumatic mechanism that may seem trivial at the time. Minor traumatic mechanisms include sports (such as jogging, scuba-diving, combat sports, and weight lifting),[2] rapid head movements, chiropractic manipulation, and whiplash injuries.[22] Nontraumatic risk factors have also been studied, and although most studies are complicated by small sample size, selection bias, and lack of blinding, there is evidence to support genetic and connective tissue disorders, migraine, and vessel abnormalities as risk factors for CeAD. Although some studies have cited recent or acute infection as a risk factor, this risk may be attributed to the mechanical stress of minor traumatic mechanisms associated with illness, such as coughing or sneezing.[23]

In the context of major trauma, CeAD is often referred to as blunt cerebrovascular injury, and this injury may be clinically silent in the immediate trauma evaluation period, presenting a diagnostic challenge. Over the last 25 years, multiple screening protocols have been developed to set guidelines for imaging to screen for CeAD based on a patient's injuries and mechanism.[24–27] These criteria generally include a "high-risk mechanism" of trauma, such as hyperextension, hyperflexion, or a direct trauma to the neck, along with patterns of other associated injuries, such as severe facial fractures, "seatbelt-sign,"[3] expanding neck hematomas, audible bruits, or neurologic deficits.[24]

The understanding of the prognosis of CeAD has been informed by large multicenter clinical trials in recent years.[1] Early studies specifically in CeAD resulting from known major trauma suggested a subsequent stroke rate in untreated CeAD as high as 25%.[28] Subsequent studies evaluating the question of antiplatelet versus anticoagulation therapy have found a much lower stroke rate of 1% to 2% in patients with CeAD treated with either therapy.[1,29] Studies in which follow-up imaging was performed around 3 to 4 months after diagnosis show evidence of healing vasculature and good outcomes with respect to disability as measured by modified Rankin score of 0 to 2.[1,28,30]

## CLINICAL FEATURES

The challenge in the timely diagnosis of a CeAD in any patient is due to the relative rarity of occurrence and the common, nonspecific presenting complaints. The most common complaints are ipsilateral headache and neck pain.[9] Other symptoms variably present can be dizziness, facial pain, and tinnitus (often pulsatile).[31] The symptoms can precede diagnosis and any neurologic deficits by a disconcertingly variable period of time, up to 2 weeks.[4] A careful history may reveal a recent trauma preceding the symptoms, but this event can be so mild and quotidian as to escape notice. Such events can include strenuous physical activity or exercise,[32] coughing,[33] Valsalva during childbirth,[34] neck cracking,[35] and certain sudden movements, including dancing[36] and even riding roller coasters.[37]

Internal carotid dissections can be associated with a partial Horner syndrome (meiosis and ptosis without anhidrosis), facial pain (to include teeth, ear, and eye),

ipsilateral anterior neck pain, and headache limited to the frontal and temporal regions.[31,38] Vertebral artery dissections often have posterolateral ipsilateral neck pain, although it can be bilateral, and posterior headaches.[38]

Focal neurologic deficits may also be present because of compression of adjacent structures by the expanding vessel or ischemia owing to embolization of thrombus or watershed ischemia owing to stenotic vascular occlusion. Compression of adjacent structures can cause radiculopathies (most commonly C5 and C6)[39] in vertebral dissections and cranial nerve deficits (CN XII most commonly and then IX and X) in carotid dissections.[31]

Ischemic signs and symptoms indicating the presence of a transient ischemic attack (TIA) or stroke occur in up to 67% of patients and can occur at any time, with or without preceding compressive symptoms.[30] Carotid dissection affects the anterior and middle cerebral arteries, resulting in typical signs and symptoms of an anterior circulation stroke (unilateral sensory changes, hemiparesis, gaze deviation, neglect, aphasia, and dysarthria), whereas vertebral dissections affect the posterior circulation and can present with symptoms in that distribution (vertigo, dysmetria, ataxia, nausea/vomiting, and nystagmus). One factor that points toward a dissection as the cause of a stroke or TIA is the presence of headache and/or neck pain, which is more common in these cases than in those strokes caused by other causes.

In general, a dissection should be suspected in a patient with any of the following.

1. Headache
2. Neck pain
3. Neurologic symptoms/findings
4. A history of risk factors (recent mild trauma, Valsalva events, a personal or family history of connective tissue disorder, or migraine headaches)

## DIAGNOSTIC TESTS

The gold standard in radiographic evaluation for CeAD is digital subtraction angiography (DSA), although this is not a practical screening test in the acute setting and is reserved for cases in which there is a diagnostic dilemma or uncertainty from other imaging.[40] Practically, diagnosis is most commonly achieved by CTA or magnetic resonance angiography (MRA).[20,21,41] Systematic review of available studies with a standard (DSA) comparator has shown comparable performance of MRA and CTA for detection of CeAD.[42] CTA may provide greater detail about vessel pathologic condition, such as hematoma formation or intimal flap, whereas MRA provides more detail about subsequent ischemic injury.[43] Ultimately, the decision regarding imaging modality is likely to be institution-dependent.

Frequently used in the acute setting for other pathologic conditions, and for evaluation of carotid atherosclerosis in stroke, carotid duplex ultrasound has also been evaluated as a possible diagnostic modality for CeAD. The challenge of this technique is that, although ultrasound may be able to visualize arterial narrowing, it is not sufficient to rule out dissection in asymptomatic patients, or patients with only local symptoms, likely because there is not sufficient luminal narrowing to be visualized on ultrasound in these cases.[44,45]

## TREATMENT

Because of the very low incidence of CeAD, it is quite difficult to conduct large, randomized studies with robust numbers of patients that allow for definitive investigation and determination of the optimal treatment strategy. Although antithrombotic therapy

is generally indicated and accepted as the preferred treatment, the evidence supporting whether antiplatelet therapy or anticoagulation should be the preferred option is inconclusive. The following sections review the available evidence and present the AHA recommendations for CeAD management (https://www.ahajournals.org/doi/epub/10.1161/STR.0000000000000457, refer to the Figure 3 in the link).[7]

## ANTITHROMBOTICS

The TREAT-CAD Trial was a relatively recent multicenter, open-label, randomized, controlled, noninferiority trial comparing aspirin with vitamin K antagonists (VKA) in the treatment of CeAD. Of the 194 patients with CeAD, 71% presented with clinical signs of cerebral ischemia and 29% had local symptoms only. The primary end point in TREAT-CAD, a composite of clinical outcomes (stroke, major hemorrhage, death) and MRI outcomes (new ischemic or hemorrhagic brain lesions) assessed at 14 days (clinical and MRI outcomes) and 90 days (clinical outcomes only) after treatment onset, failed to meet its threshold for the noninferiority of aspirin as monotherapy compared with VKA.[46]

The CADISS Trial was a multicenter, randomized, controlled, open-label trial that had 250 participants, 118 with carotid dissection and 132 with vertebral artery dissection. The presenting symptoms were cerebral ischemia in 224 and local symptoms in 26 participants. The participants were randomly allocated to either anticoagulation in 124 patients with a VKA (most with heparin bridging) or antiplatelet therapy in 126 patients for 3 months. The latter treatment was variable and included aspirin, clopidogrel, and dipyridamole either as a single agent or in some form of combination therapy. The primary study endpoint was ipsilateral stroke or death, and no statistical significance was found between the treatment groups at the end of the 3-month study period nor at the 3- to 12-month observational follow-up time frame.[47]

## VITAMIN K ANTAGONISTS VERSUS DIRECT ORAL ANTICOAGULANTS

Both the CADISS and the TREAT-CAD trials used VKA for anticoagulation treatment and not the newer direct oral anticoagulants (DOACs). These two classes of anticoagulant medications were compared in a systematic review and meta-analysis of observational data with 699 patients in the VKA group and 53 patients in the DOAC group. The study showed decreased rates of stroke/TIA, 5.7% versus 12.3%, respectively, for DOACs compared with VKAs. It also showed less intracranial and major bleeding episodes, 0% versus 1.2%, respectively, for both types, again favoring DOACs over VKAs. However, there are major limitations to the study, including the observational nature of the data and the very limited number of patients in the DOAC group.[48]

## TREATMENT GUIDELINES

Because there is no clear evidence supporting one treatment over another, current guidelines from the AHA do not recommend a specific regimen of either antiplatelet or anticoagulation, but rather stipulate a tailored approach that balances individual patient bleeding risk with the presence or absence of high-risk features.[7] Duration of treatment is usually 3 to 6 months.

## THROMBOLYTIC THERAPY

Although it seems counterintuitive, thrombolytics are not contraindicated in patients presenting with stroke owing to a CeAD. In studies looking at patients with acute ischemic stroke from CeAD who received thrombolytics, there was a similar rate of

intracerebral hemorrhage when compared with those without CeAD as the cause of the stroke.[49,50] Therefore, in the absence of data suggesting an increased risk of harm or evident safety concerns, it is reasonable to consider thrombolytic therapy for patients with acute ischemic stroke with CeAD as the cause if they meet all other standard inclusion criteria as recommended by current guidelines and local stroke protocols.

Given the complexity of these patients, the questions surrounding optimal treatment, the duration of treatment, and the need for ongoing close follow-up, it is prudent to consult a specialist with expertise in this area (often neurology or neurosurgery, but may be institution dependent) to discuss management, the need for admission to the hospital, and continuing care.

## SURGICAL TREATMENTS

For patients who meet the inclusion criteria for mechanical thrombectomy for acute large-vessel occlusion stroke, stenting and thrombectomy are potentially helpful treatment modalities.[7] The optimal sequence of treatment for patients with large vessel occlusion stroke and dissection is an active area of study.[7] Interpretation of the data on tandem occlusions is made more difficult by the fact that occlusions from atherosclerotic disease and dissection occur by fundamentally different mechanisms and on different time scale, but are analyzed together in many major studies.[4,51] A multicenter retrospective cohort study of 526 patients with tandem lesions showed more frequent distal embolism and lower rates of successful recanalization in those with a dissection etiology compared with atherosclerosis, but further studies are required.[52] Subacute treatment of CeAD with stenting is controversial, but may be considered in the setting of recurrent cerebral ischemia while on optimal antithrombotic treatment.[7]

## DISPOSITION

Patients with ongoing, persistent neurologic symptoms or progressive neurologic deterioration, or who have either received, or are in the time window to receive, thrombolytic therapy, should be admitted to the hospital. In addition, those patients deemed to be candidates for possible stenting or thrombectomy should also be considered for admission. In the few patients who have no ongoing, persistent neurologic symptoms, who have a good social support system, who can return to the hospital expeditiously if needed, who are low risk for complications from antithrombotic therapy, and for whom definite follow-up can be arranged, discharge from the Emergency Department with antithrombotic therapy may be an option after discussion with a specialist.

## SUMMARY

Carotid and vertebral artery dissections are an important and frequently missed cause of ischemic stroke in young patients. Identification of this important cause of neurologic symptoms requires the emergency clinician to be alert to risk factors and symptoms that may seem atypical in this age group. Vessel injury can occur spontaneously, or be caused by major or seemingly trivial trauma. The goal of therapy is to prevent future stroke, and long-term prognosis is typically good.

## CLINICS CARE POINTS

- Carotid and vertebral artery dissection should be suspected in younger patients with stroke syndromes.

- Cervical artery dissection also be a cause of stroke syndromes in the setting of trauma (both major and minor).
- Treatment is with antiplatelet agents or anticoagulants depending on bleeding risk.
- Stenting or thrombectomy can be considered for acute tandem lesions in combination with a large-vessel occlusion stroke, or for subacute lesions in the setting of medical treatment failure.

## DISCLOSURES

L.E. Mamer and R. Stephen have no pertinent disclosures or conflicts of interest.

## FUNDING

L.E. Mamer would like to acknowledge support of her K12 Award: K12TR004374.

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