Extracranial Cervical Artery Dissections

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KEYWORDS

• Dissection • Stroke • Carotid • Vertebral • Cervical

KEY POINTS

- Cervical artery dissections (CeAD) are rare but important causes of stroke, especially in the younger population.
- Consider CeAD in patients with new-onset headache and neck pain with or without strokelike symptoms.
- Imaging is key to diagnosis, with several options available.
- Management involves treating acute stroke with thrombolysis or surgical therapy for eligible candidates. All others may be candidates for anticoagulation or antiplatelet therapy to reduce the risk of potential or worsening stroke symptoms. Either agent may be used.
- Prognosis remains good with low morbidity and mortality rates.

INTRODUCTION

The cervical arteries comprise bilateral internal carotid and vertebral arteries. These arteries are important structures of the neck, as they carry the main blood flow to the brain. Any thrombosis or damage to these vessels, including dissection, can lead to complications, such as cerebral ischemia, stroke, blindness, or death.[1,2] Although cervical artery dissections (CeADs) are rare causes of stroke overall, they are important causes of stroke in the younger population.[3–5] Unfortunately, given its rarity and nonspecific symptoms, CeAD is a difficult diagnosis to make. Affected patients can remain completely asymptomatic or asymptomatic for long periods of time. Patients may sustain delayed-onset stroke, which can contribute to difficulty in making the diagnosis.[3,6,7] Long-term, severe morbidity, such as stroke with loss of independence, can occur, and, thus, it is important to avoid missing this diagnosis.[8]

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Although patients with CeAD may deny history of trauma, the reality is that most patients tend to have a history of trauma, albeit very mild.[9,10] In addition, spontaneous extracranial CeAD and extracranial CeADs that develop due to minor trauma are more commonly discussed in the literature.[11–14] This is opposed to pure intracranial CeADs and those due to major trauma and blunt aortic dissection.[3] This article focuses on the epidemiology, pathophysiology, diagnosis, and management of spontaneous extracranial CeADs and extracranial CeADs due to negligible trauma.

EPIDEMIOLOGY

The estimated incidence of CeAD is 2.6 to 5.0 per 100,000 per year, but recent epidemiologic studies are lacking.[5,14–16] An epidemiologic study in 2014 demonstrated that of nearly 1400 patients with stroke, CeAD accounted for only 2% of cases.[16] Most recently, a study in Vancouver found that of 438 patients with transient ischemic attack (TIA) or ischemic stroke, approximately 5.9% were due to CeAD. This percentage included both internal carotid artery dissection (ICAD) and vertebral artery dissection (VAD), but 1 patient did have an intracranial dissection.[17]

Compared with thrombosis, ischemic strokes due to CeAD are rare, approximating a total of only 1% to 2% of all ischemic stroke cases.[15] However, CeAD accounts for a much larger percentage of ischemic strokes in the younger population.[1,3,15] CeAD is most common in the fifth decade of life, and is rare in patients older than 65.[14,18] In the Vancouver study, the mean age of patients with stroke/TIA due to CeAD was 49.1 years.[17] In the 2014 study by Bejot and colleagues,[16] a mean age of 49.1 years also was observed. Overall, the incidence of ICAD is approximately twice that of VAD.[14] VAD tends to occur more commonly in younger women, whereas ICAD is more prevalent in older men.[19,20] Importantly, most epidemiologic studies on CeAD are observational and based on European and American populations, and, thus, the incidence of CeAD is estimated and patterns may differ in other populations.[3,14,16]

PATHOPHYSIOLOGY

The pathogenesis of CeAD is not well delineated. However, as will be mentioned in a subsequent section, CeAD is thought to be caused by minor trauma. It also may occur spontaneously in patients with predisposing arterial defects.[3] CeAD is characterized as a hematoma within the wall of the internal carotid artery or vertebral artery.[3,21] Initially, a tear occurs in the artery where blood enters the wall under pressure and causes separation of the layers. A false lumen develops, resulting in a hematoma. This narrows the arterial lumen, increasing risk of occlusion by the hematoma.[22] The hematoma or thrombus can lead to cerebral thromboembolism, decreased blood flow, and subsequent ischemic stroke.[21,23] The hematoma may also cause a mass effect on surrounding structures, leading to Horner syndrome (ptosis, anhidrosis, and miosis, though anhidrosis may not be present).

Thromboembolism, rather than hypoperfusion, has been found to cause most ischemic strokes in CeAD.[24]

CeAD is likely a multifactorial process that is not well understood but may be related to underlying risk factors, such as genetics, connective tissue diseases, and prior trauma.[3,9,25] It is estimated minor trauma plays a role in approximately 40% of cases of spontaneous extracranial CeAD.[9] In 2013, Engelter and colleagues[9] compared patients with CeAD who had sustained known neck trauma, patients with ischemic stroke with other etiologies, and healthy subjects. Overall, the investigators found that prior mechanical trauma was more common in patients with CeAD than in patients