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CONCISE REVIEW

Craniocervical Artery Dissections: A Concise Review for Clinicians

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CME Activity

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Learning Objectives: On completion of this article, you should be able to (1) recognize symptoms of acute craniocervical artery dissection (CAD) and initiate the work-up with an appropriate level of urgency, (2) provide basic management for stroke prevention and pain control, and (3) identify long-term complications of CAD and counsel patients with CAD on activity restrictions.

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Abstract

Craniocervical artery dissection (CAD), although uncommon, can affect the young and lead to devastating complications, including stroke and subarachnoid hemorrhage. It starts with a tear in the intima of a vessel with subsequent formation of an intramural hematoma. Most CAD occurs spontaneously or after minor trauma. Patients with CAD may exhibit isolated symptoms of an underlying subclinical connective tissue disorder or have a clinically diagnosed connective tissue disorder. Emergent evaluation and computed tomography angiography or magnetic resonance imaging/angiography of the head and neck are required to screen for and to diagnose CAD. Carotid ultrasound is not recommended as an initial test because of limited anatomic windows; diagnostic catheter-based angiography is reserved for atypical cases or acutely if severe neurologic deficits are present. Patients with CAD can present with focal neurologic deficits due to ischemia (thromboembolism or arterial occlusion) or subarachnoid hemorrhage (pseudoaneurysm formation and rupture). Also common are local symptoms, such as head and neck pain, pulsatile tinnitus, Horner syndrome, and cranial neuropathy, or cervical radiculopathy from mass effect. Acute management of transient ischemic attack/stroke in CAD is not different from the management of ischemic stroke of other

causes. Patients with CAD need long-term antithrombotic therapy for secondary stroke prevention. Anticoagulation or dual antiplatelet therapy followed by single antiplatelet therapy is recommended for extracranial CAD and antiplatelet therapy for intracranial CAD. Recurrent ischemic events and dissections are rare and typically occur early. Patients with CAD should avoid deep neck massage or chiropractic neck manipulation involving sudden excessive, forced neck movements.

 ■ Mayo Clin Proc. 2022;97(4):777-783

The vessel wall of craniocervical arteries consists of 3 layers: the tunica intima, the inner layer; the tunica media, the middle and muscular layer; and the tunica adventitia, the outer layer. Craniocervical artery dissection (CAD) is a sudden tear in the intimal layer with subsequent bleeding into the subintimal space. This causes progressive vessel wall incursion into the lumen and narrowing, which at times proceeds to occlusion.[1] The site of dissection becomes thrombogenic because of turbulent blood flow and exposure of thrombogenic factors (Figure 1A). Enlargement of the vessel wall can also lead to compression on surrounding structures like cranial nerves.[2] If the intramural hematoma grows into the adventitia, it can lead to pseudoaneurysm formation (Figure 1B). Rupture of a pseudoaneurysm can cause subarachnoid hemorrhage (SAH) if the site of dissection extends to the intracranial vasculature.[1]

EPIDEMIOLOGY AND RISK FACTORS

The annual incidence of CAD is about 3 per 100,000, but the actual incidence is likely to be higher because of asymptomatic cases.[3] Although CAD is uncommon, up to one-fourth of cases of ischemic stroke in young patients are a result of dissection.[4]

More than half of CAD cases occur spontaneously, and close to 90% of traumatic dissections are due to minor trauma.[5] Chiropractic neck manipulations, heavy lifting, sports-associated injuries, and whiplash are the most common minor traumas identified in CAD.[5] Other minor traumas associated with CAD include childbirth, yoga, vigorous exercise, vomiting, coughing, and sneezing.

Craniocervical artery dissection can be associated with connective tissue disorders, such as fibromuscular dysplasia and Ehlers-Danlos, Marfan, and Loeys-Dietz syndromes.[4,6-8] Interestingly, many patients with CAD have isolated mild connective tissue abnormalities, such as joint hypermobility, easy bruising, and poor wound healing, suggesting that CAD may be another expression of a yet undefined connective tissue disorder.[9] Recent infections, hypertension, oral contraceptive use, smoking, pregnancy, migraine, and elongated styloid process are other comorbidities associated with CAD,[4,10] but their role in the pathogenesis of CAD is debated.

CLINICAL PRESENTATION

Patients with CAD can present with a transient ischemic attack (TIA) or acute ischemic stroke due to thromboembolism or arterial occlusion or with SAH due to rupture of a dissecting aneurysm (intracranial vasculature). Headache or neck pain, pulsatile tinnitus, Horner syndrome, compressive cranial neuropathy, and cervical radiculopathy are other CAD symptoms due to the rapid expansion of the vessel diameter.

Headache or Neck Pain

Head or neck pain, even in the absence of SAH, is the most common symptom after CAD; 80% of patients with CAD will present with pain in the head or neck.[3] The headache can be nonspecific and poorly localized, but a recent or sudden onset of intractable, persistent head or neck pain in a young adult should suggest acute dissection. The location of the pain may suggest which vessel is involved. In the carotid artery, CAD can cause ipsilateral cervical, retro-orbital, or temporal pain. In the vertebral artery, CAD can cause ipsilateral posterior cervical or occipital pain (Figure 2).[11] Based on case reports and small series, CAD should also be in the differential diagnosis of a thunderclap, hemicrania continua, or trigeminal

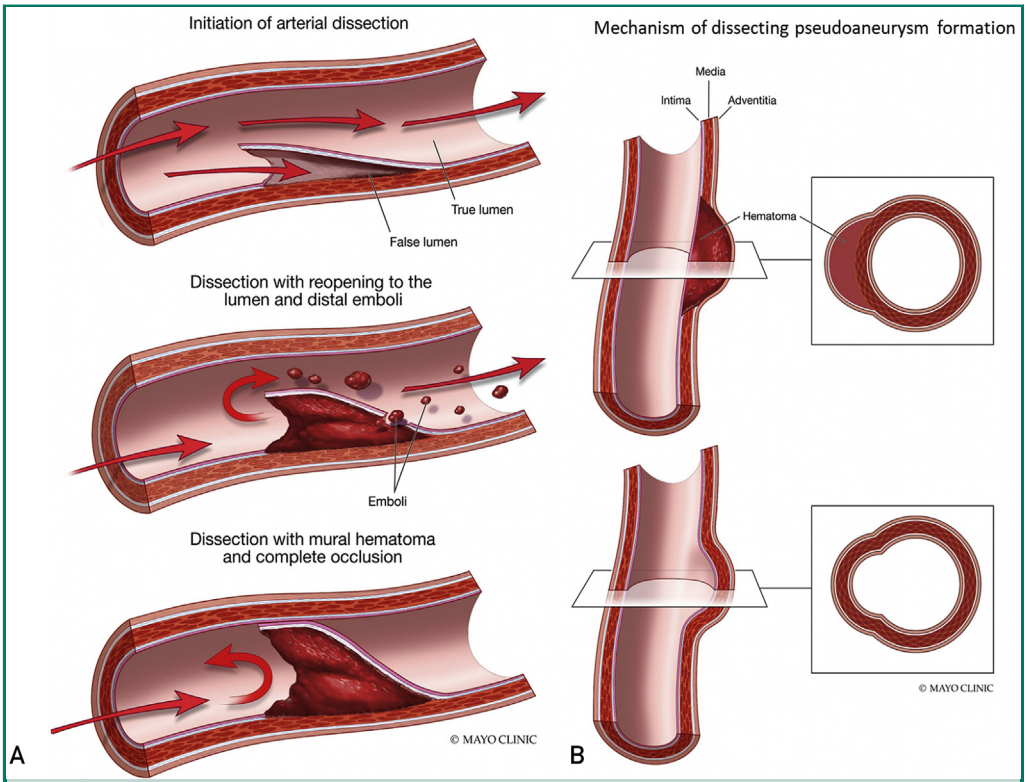

FIGURE 1. Mechanism and evolution of a dissection formation (A) and dissecting pseudoaneurysm (B). (Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.)

neuralgia–like headache.[11] Patients with preexisting migraine can experience worsening of their baseline headaches with new CAD.

TIA/Ischemic Stroke

More than half of the patients with symptomatic CAD experience TIA/acute ischemic stroke.[3] Depending on the site of CAD and location of the affected vessel, patients with CAD can present with sudden focal neurologic deficits, such as unilateral weakness, speech impairment, facial droop, vision loss or double vision, balance issues, or a combination of these symptoms. Craniocervical artery dissection should always be considered in a young patient with a stroke.

Subarachnoid Hemorrhage

Sudden thunderclap headache (headache reaching maximal intensity within seconds) with or without alteration in mental status and focal neurologic deficits should trigger an emergent evaluation for SAH due to rupture of a dissecting aneurysm.[1] This complication is typically seen in intracranial CAD, especially that involving the distal vertebral artery.

Other Compressive Effects of CAD

Ptosis and miosis without anhidrosis, partial Horner syndrome, can be seen in up to one-fourth of patients with CAD due to compression of sympathetic fibers in the carotid sheath (Figure 2).[3] Ipsilateral new-onset pulsatile tinnitus is reported by 8% of patients with CAD.[12] Patients with CAD can present with ipsilateral cranial neuropathies (any cranial nerve from 3 to 12), and interestingly, ipsilateral hypoglossal nerve palsy is the most common cranial neuropathy in CAD.[2] Thus, the strength and range of tongue movements should be tested in suspected cases of CAD. Although rare, patients with CAD can experience ipsilateral

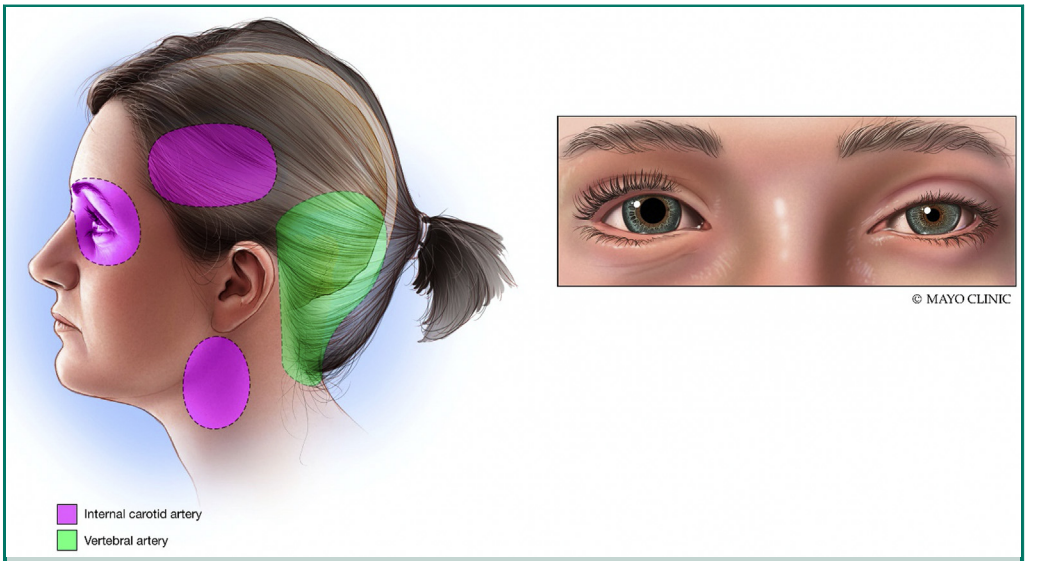

FIGURE 2. Dissection-related headache and neck pain (left). Miosis and ptosis of the left eye without anhidrosis, partial Horner syndrome. It is commonly seen in ipsilateral carotid dissections (right). (Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.)

compressive radiculopathy, most commonly at the C5-C6 level.[13]

WORK-UP

Depending on the level of suspicion, an emergent work-up is typically required whenever CAD is suspected. Head and neck computed tomography (CT) angiography, magnetic resonance imaging (MRI), or magnetic resonance angiography (MRA) is the initial recommended diagnostic test.[14] Many tertiary hospitals have an MRI-based dissection protocol that includes a T1 sequence with fat saturation to better visualize intramural hematoma. Especially in the presence of focal neurologic deficits, a non–contrast-enhanced head CT or, ideally, MRI scan should be obtained to rule out ischemic stroke or SAH. In patients with allergy to contrast material or advanced chronic kidney disease, MRA can be performed without intravenous administration of contrast material. Carotid ultrasound is not recommended for initial testing as it can only partially visualize craniocervical vasculature. Diagnostic angiography is the "gold standard" to detect CAD when the diagnosis is unclear or an intervention, such as endovascular stenting or acute mechanical clot retrieval, is needed.[15] Genetic testing is typically not recommended in CAD unless another established underlying connective tissue disorder, such as Ehlers-Danlos syndrome, is strongly suspected.

Because of the increased association of CAD with other connective tissue disorders, a workup for syndromes like Marfan, Ehlers-Danlos type IV, and Loeys-Dietz should be considered with features such as hyperflexible joints, disproportionately long arms and fingers, pectus carinatum or excavatum, hyperlucent skin, scoliosis, clubfoot, bifid uvula, or cleft palate.

MANAGEMENT

Acute CAD

Most patients with acute CAD should be treated emergently, especially when focal neurologic deficits are present. Recombinant tissue plasminogen activator should be administered intravenously under typical acute stroke guidelines.[14] Other hyperacute treatments, such as tenecteplase and mechanical thrombectomy, are also options for appropriate patients with extracranial CAD and acute ischemic stroke. Based on a small case series, thrombolysis and mechanical thrombectomy in intracranial CAD seem

relatively safe.[16,17] Patients with CAD experiencing SAH might require surgical intervention, such as aneurysm repair, external ventricular drain for hydrocephalus, or decompression for herniation.[1]

Long-term Management

Headache, Neck Pain, and Other Local Compressive Symptoms. There are no specific treatment recommendations for headache or neck pain for CAD. Beta blockers (such as propranolol), tricyclic antidepressants (such as amitriptyline), and antiepileptics (such as gabapentin, valproic acid, and topiramate) can be used for headache maintenance therapy. For acute pain attacks, over-the-counter agents such as acetaminophen can be used to treat pain. Although no data exist to advise against vasoactive agents such as triptans, it is reasonable to prioritize other agents. The safety and efficacy of the calcitonin gene—related peptides in patients with recent cerebrovascular disorders have not yet been tested. Other compressive symptoms, such as Horner syndrome, cranial neuropathies, and cervical radiculopathy, are managed conservatively. As the dissection heals, pain and compressive symptoms are expected to lessen and resolve, usually within the first 6 to 12 weeks after diagnosis.

Stroke Prevention. Antithrombotic therapy is recommended for at least 3 months for patients with TIA/ischemic stroke and extracranial CAD.[14] Despite large-scale trials,[18,19] the antithrombotic regimen (single antiplatelet, dual antiplatelet, or anticoagulation) is controversial. For extracranial CAD, expert opinion, including ours, favors anticoagulation or dual antiplatelet therapy for the first 3 months, followed by single antiplatelet therapy (typically low-dose aspirin) lifelong.[20] However, a short course of dual antiplatelet therapy in the setting of a cervical artery dissection has never been formally investigated, but data from dual antiplatelet therapy in nondisabling stroke can be extrapolated in the setting of cervical dissection. Only heparin products and warfarin have been tested so far in terms of anticoagulation,[18,19] but limited retrospective data suggest equal safety and efficacy for direct oral anticoagulants.[21] Other factors, such as intraluminal thrombus formation and significant luminal stenosis (hypothetically increased incidence of turbulent blood flow), can favor anticoagulation over antiplatelet therapy for the first few months. For the management of intracranial CAD, despite the knowledge gap in the guidelines,[14] single antiplatelet therapy is reasonable for ischemic stroke prevention. Expert opinion, such as from vascular medicine and neurology (ideally vascular neurology) specialists, should be sought to determine the type and duration of the antithrombotic management in CAD. Optimizing other vascular risk factors, such as hypertension and hyperlipidemia, is equally essential.

Up to 13% of patients can experience progressive luminal narrowing or recurrent TIA/ischemic strokes.[4] This might require consideration of endovascular intervention.[14]

Iatrogenic dissections are documented complications of endovascular diagnostic and therapeutic procedures. They typically have a benign course and do not require endovascular intervention.[22]

Unilateral or bilateral extracranial CAD as an extension of aortic dissection is not uncommon and increases the risk of stroke, especially in the acute stage.[23] As for spontaneous CAD, medical therapy with an antithrombotic regimen is recommended for stroke prevention.

Other Complications. Patients with known CAD may experience new-onset or worsening symptoms, such as pulsatile tinnitus, severe head or neck pain, and cranial or cervical radiculopathy, and require repeated imaging as new or expanding dissecting aneurysms develop in the first year in 10% to 20% of patients with CAD.[24] In addition, up to 5% of patients with CAD can have recurrent dissections,[4] the risk of which is higher during the first year after the original presentation. Patients with known connective tissue disorders can experience long-term complications, such as recurrent TIA/stroke,

recurrent CAD formation, and pseudoaneurysm formation. Antithrombotic therapy and surgical or endovascular intervention for the dissecting pseudoaneurysm formation can be pursued, depending on the location (extracranial vs intracranial vasculature, anterior vs posterior circulation), size, evolution, and associated symptoms.[1,24,25]

Counseling. It is recommended that patients with CAD avoid activities such as deep neck massage or chiropractic neck manipulation that involves sudden excessive and forced neck movements.[26] It is also wise to avoid heavy lifting or collision contact sports that include excessive neck straining. Otherwise, patients can resume their regular activities of daily living, including mild to moderate physical exercise as tolerated.

PROGNOSIS

The overall prognosis of CAD, including with mild ischemic symptoms, is generally benign, with most patients recovering fully. On radiologic evaluation, about one-third of patients with CAD show resolution of the arterial stenosis or occlusion, and most healing occurs within the first year after the initial tear.[3] Nearly 20% of patients with CAD develop pseudoaneurysm. The natural history of small (<10 mm in diameter) pseudoaneurysms in the neck is benign. Patients can be reassured that the risk of rupture with bleeding is virtually nil and the risk of recurrent thromboembolism very low when antithrombotic medication is being taken. Intracranial pseudoaneurysms are different and may require intervention in selected cases.[1,24] Good clinical recovery is expected in 75% to 92% of patients with CAD, and mortality remains below 5%.[3,8] Stroke severity at presentation, complete arterial occlusion, and older age are associated with worse clinical outcomes.[4]

SUMMARY AND FUTURE DIRECTIONS

Craniocervical artery dissection represents a common cause of stroke in young adults. Dissection starts with a tear in the intima of the vessel, followed by the formation of an intramural hematoma. Most cases occur spontaneously or after minor trauma. Emergent evaluation is required in most cases, and recommended initial diagnostic tests are head and neck CT angiography with brain CT and head and neck MRI or MRA with brain MRI. In the acute setting, CAD may lead to TIA/stroke, SAH, local compressive symptoms such as head or neck pain, cranial neuropathies or cervical radiculopathies, or a combination of these complications. Acute treatment of ischemic stroke in CAD may include intravenous thrombolytic therapy and, in select cases, mechanical thrombectomy. Patients with CAD, especially with TIA/stroke, need antiplatelet or anticoagulation therapy. Although there is no clear consensus on the optimum strategy to prevent stroke in extracranial CAD, expert opinion favors anticoagulation or dual antiplatelet therapy for at least 3 months. Anticoagulation should typically be avoided in intracranial CAD. There are no specific treatment recommendations for pain syndromes and other compressive causes. One-third of patients with CAD show spontaneous healing within the first year. About 20% to 50% of patients have dissecting pseudoaneurysms, and surgical or endovascular intervention is recommended in selected cases. Recurrent ischemic events and dissections are rare and typically occur in the first few months after diagnosis.

In addition, there is a knowledge gap in terms of type of antithrombotic regimen, stenting, and duration of antithrombotic therapy for stroke prevention in CAD. A well-designed trial including advanced imaging and genetic biomarkers is required to compare various antithrombotic approaches, and stenting in CAD is required.

ACKNOWLEDGMENTS

We thank Stephen Graepel, a senior medical illustrator at Mayo Clinic, Rochester, for creating illustrative figures in this article.

SUPPLEMENTAL ONLINE MATERIAL

Supplemental material can be found online at http://www.mayoclinicproceedings.org. Supplemental material attached to journal articles

has not been edited, and the authors take responsibility for the accuracy of all data.

Abbreviations and Acronyms: **CAD,** craniocervical artery dissection; **CT,** computed tomography; **MRA,** magnetic resonance angiography; **MRI,** magnetic resonance imaging; **SAH,** subarachnoid hemorrhage; **TIA,** transient ischemic attack

Potential Competing Interests: The authors report no competing interests.

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