Dissection of Arteries Supplying the Brain

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Overview and Epidemiology

Arterial dissection of arteries supplying the brain was first identified in the 1950s. However, more recently the number of patients identified with arterial dissection has increased principally due to improved imaging techniques. Arterial dissection represents an important potentially treatable injury to the extracranial carotid and vertebral arteries. Cervical and intracranial dissections of the carotid and vertebral arteries can be present as either traumatic, or commonly, spontaneous without an identifiable etiology. For this discussion, we group dissections into extracranial and intracranial, based on differences in the anatomy of arterial wall and their presenting clinical features. A thinner media and adventitia is a key difference with intracranial as compared with cervical extra-cranial arteries. This allows for dissections to occur beyond the media and into the outer adventitia layer; whereas, dissection of extracranial arteries occurs between the intima and media because of a comparatively thicker vessel wall.

The extracranial arteries most affected by dissection are the internal carotid and cervical vertebral arteries. The intracranial arteries most prone to dissection include the intradural vertebral arteries and less commonly the basilar, supraclinoid internal carotid, middle cerebral and anterior cerebral arteries. Traumatic dissections of the cervical carotid and vertebral arteries can occur with seemingly minor movements such as coughing or laughing and with more forceful movements such as cervical spine manipulation, whiplash injury or blunt trauma; whereas traumatic dissections can occur in patients of any age, spontaneous dissections mainly occur in younger patients between the ages of 25 – 45. Furthermore, up to 15% of patients with Fibromuscular Dysplasia and 1-5% of patients with Ehlers-Danlos also experience dissection. In patients over 50 years, stroke risk factors such as hypertension, smoking and diabetes mellitus are associated with, but not a directly a cause of dissection (6).

Extracranial Artery Dissection:
Pathophysiology

Dissection of extracranial carotid and vertebral arteries typically occurs at the subintima of the arterial wall. At the site of injury, blood entering the subintima can create a local thrombus and a subsequent false lumen for thrombus to propagate. As the dissection propagates longitudinally, the false lumen expands and the true (original) arterial lumen narrows. This can compromise forward blood flow leading to distal ischemia and can propagate thrombus causing emboli.
Cervical carotid artery dissection typically occurs just distal to the carotid bulb of the ICA and extends to the skull base. Rarely, ICA dissections can extend intracranially. The most common mechanism of injury leading to dissection is hyperextension and lateral rotation. Vertebral artery dissection often occurs at its entrance of the artery into the C6 transverse foramina or at its exit from the C1 foramina. Vertebral artery dissection often occurs spontaneously, but can also occur as a result of dislocation, distraction, fracture of the cervical vertebrae, or an intruding osteophyte.

**Clinical Features:**

Patients with internal carotid artery dissection often present with unilateral neck pain in the trigeminal and cervical dermatomes. Headaches also occur and can range from sharp to dull or throbbing in nature. Extracranial dissection of the internal carotid artery, can lead to damage of the sympathetic chain located in the carotid sheath. This injury to the ascending sympathetic nerves can lead to ptosis, miosis and anhydrosis of the ipsilateral eye (Horner’s Syndrome). If carotid artery flow is significantly interrupted, patients may exhibit hemispheric ischemic signs such as ipsilateral gaze deviation, contralateral weakness or a homonymous hemianopsia.

Patients with vertebral artery dissection often present with pain in the posterior neck. As flow diminishes or emboli are formed, patients can develop brainstem ischemia presenting as dizziness, ataxia, diplopia or dysarthria.

**Intracranial Artery Dissection**

**Pathophysiology:**

The overwhelming majority of dissections of the intracranial circulation occur within the intradural vertebral arteries. Often, intracranial dissection will be caused by arterial blood dissecting into the subadventitia, causing a pseudoaneurysm. If the pseudoaneurysm succumbs to the pressure created by the dissection, the artery artery can rupture and the patient will present with subarachnoid hemorrhage.

**Clinical Features:**

Intracranial artery dissection will present with either an infarction of the respective vascular territory or subarachnoid hemorrhage. Since the intradural vertebral arteries account for the majority of intracranial dissection, the classic presentation is non-aneurysmal subarachnoid hemorrhage.
Imaging:

*Magnetic resonance imaging (MRI)*
The key image sequences for diagnosing dissection is the T1 weighted “fat saturation.” Axial T1 “fat saturation” images demonstrate an intramural hematoma as a hyperintense curvilinear or crescent shaped focus within a vessel. This abnormal focus is usually eccentric and may widen the external diameter of the artery or eventually become circumferential. The hematoma can remain hyperintense for months, which is helpful in a patient with a suspected remote history of mechanical stress.

Since ischemia can be the presenting symptom of dissection, obtaining an MRI will help diagnose a dissection and precisely define any area of ischemia. Time of flight magnetic resonance angiography source images can complement MRI images by demonstrating “flow void” and a “double lumen.” Finally, once the diagnosis is made, MRI is an excellent technique to monitor response to therapy as an outpatient.

*Cerebral Angiography*
Cerebral angiography is a sensitive studies to diagnose dissection and prior to MRI and CTA, was the standard technique for making the diagnosis. Angiographic findings of dissection include segmental narrowing/stenosis due to intraluminal hematoma “string” sign, arterial occlusion, aneurysm formation, narrowing in sites unusual for atherosclerosis and segmental dilatation (pseudoaneurysm). Angiography cannot help distinguish between spontaneous and traumatic dissections.

*Computed Tomography Angiography (CTA)*
In addition to MRI, CTA imaging of the head and neck has become an excellent modality to diagnose dissection because it provides high quality images of the arterial lumen, vessel wall and is noninvasive. CTA has a high sensitivity and specificity for demonstrating a narrowed eccentric lumen, vessel contour changes or occlusion and aneurysm formation. Limitations of CTA include imaging vessels at the base of the skull (due to bony artifact) and the risk of renal failure due to intravenous contrast administration in patients with renal dysfunction.

*Ultrasound*
Ultrasound is a noninvasive, helpful bedside tool to follow extracranial carotid artery dissection. Flow direction, visualization of the true and false lumen (even if the lumen is thrombosed) and flow velocity can be easily obtained. These findings can be followed serially, especially in patients that are not suitable to travel outside of the ICU.

Treatment:

*Extracranial Artery Dissection*
To date there are no large randomized trials directly comparing anticoagulation to antiplatelet agents.
Furthermore, there are no randomized trials comparing either therapy to control. Current treatment is guided by observational studies and meta-analyses without a definitive treatment strategy (Cochrane 2010).

**Intracranial Artery Dissection**

Antiplatelet agents and anticoagulants should be avoided in patients with an intracranial dissection, presenting with a SAH. Endovascular therapy is an increasingly preferred treatment, which often requires coil embolization of the dissection (Flis 2007). A full review of this area is beyond the scope of this review. See references for additional information.

**REFERENCES**

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