Internal carotid artery dissection

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Internal carotid artery dissection is a separation of the layers of the artery wall supplying oxygen-bearing blood to the head and brain and is the most common cause of stroke in young adults.

Epidemiology

Cervical artery dissection accounts for only 1–2% of all ischemic strokes, but in young and middle-aged people it accounts for 10–25% of strokes [1)].

Classification

Internal carotid artery dissection (ICAD) can be classified based on various criteria including **etiology**, **location**, **extent**, and **imaging characteristics**.

1. Etiological Classification

- **Spontaneous Internal Carotid Dissection (sICAD):**
  - Occurs without a known traumatic cause.
  - Associated with connective tissue disorders (e.g., Ehlers-Danlos, Marfan) or fibromuscular dysplasia.
- **Traumatic Internal Carotid Dissection (tICAD):**
  - Caused by direct or indirect trauma (e.g., cervical hyperextension, blunt trauma, penetrating injury).

2. Anatomic Location Classification

- **Extracranial ICAD (EC-ICAD):**
  - Involves the cervical portion of the **internal carotid artery (ICA)**.

    - Most common (~80% of cases).
- **Intracranial ICAD (IC-ICAD):**
    - Affects the petrous, cavernous, or supraclinoid segments of the ICA.
    - Higher risk of subarachnoid hemorrhage (SAH).

3. Extent and Severity Classification

- **Localized Dissection:** Confined to a short segment.
- **Extensive Dissection:** Involves a long segment, possibly extending extracranially to intracranially.
- **Stenotic vs. Aneurysmal Dissection:**
    - **Stenotic Type:** False lumen compresses the true lumen, causing vessel narrowing and ischemia.
    - **Aneurysmal Type:** Pseudoaneurysm formation due to vessel wall weakening.

4. Imaging-Based Classification (Modified Schievink)

Type	Description
I	Narrowing of the lumen with an intimal flap or double lumen.
II	Irregular lumen narrowing without an apparent intimal flap.
III	Pseudoaneurysm formation due to vessel wall weakening.
IV	Complete occlusion due to thrombosis in the false lumen.
V	Dissection extending into distal branches.

5. Clinical Severity and Prognosis

- **Mild:** No neurological deficits, incidental finding.
- **Moderate:** Transient ischemic attack (TIA) or minor stroke.
- **Severe:** Major ischemic stroke, SAH (if intracranial dissection), or significant mass effect.

Causes

The causes of internal carotid artery dissection can be broadly categorised into two classes: spontaneous or traumatic.

Once considered uncommon, spontaneous carotid artery dissection is an increasingly recognised cause of stroke that preferentially affects the middle-aged.

The incidence of spontaneous carotid artery dissection is low, and incidence rates for internal carotid artery dissection have been reported to be 2.6 to 2.9 per 100,000.

Observational studies and case reports published since the early 1980s show that patients with spontaneous internal carotid artery dissection may also have a history of stroke in their family and/or hereditary connective tissue disorders, such as Marfan syndrome, Ehlers-Danlos syndrome, autosomal dominant polycystic kidney disease, pseudoxanthoma elasticum, fibromuscular dysplasia, and osteogenesis imperfecta type I.

However, although an association with connective tissue disorders does exist, most people with

spontaneous arterial dissections do not have associated connective tissue disorders. Also, the reports on the prevalence of hereditary connective tissue diseases in people with spontaneous dissections are highly variable, ranging from 0% to 0.6% in one study to 5% to 18% in another study.

Carotid artery dissection is thought to be more commonly caused by severe violent trauma to the head and/or neck. An estimated 0.67% of patients admitted to the hospital after major motor vehicle accidents were found to have blunt carotid injury, including intimal dissections, pseudoaneurysms, thromboses, or fistulas.

Of these, 76% had intimal dissections, pseudoaneurysms, or a combination of the two.

The probable mechanism of injury for most internal carotid injuries is rapid deceleration, with resultant hyperextension and rotation of the neck, which stretches the internal carotid artery over the upper cervical vertebrae , producing an intimal tear.

After such an injury, the patient may remain asymptomatic, have a hemispheric transient ischemic event, or suffer a stroke.

Pathophysiology

Arterial dissection of the carotid arteries occurs when a small tear forms in the innermost lining of the arterial wall (known as the tunica intima). Blood is then able to enter the space between the inner and outer layers of the vessel, causing narrowing (stenosis) or complete occlusion. The stenosis that occurs in the early stages of arterial dissection is a dynamic process and some occlusions can return to stenosis very quickly.

When complete occlusion occurs, it may lead to ischaemia. Often, even a complete occlusion is totally asymptomatic because bilateral circulation keeps the brain well perfused. However, when blood clots form and break off from the site of the tear, they form emboli, which can travel through the arteries to the brain and block the blood supply to the brain, resulting in an ischaemic stroke, otherwise known as a cerebral infarction. Blood clots, or emboli, originating from the dissection are thought to be the cause of infarction in the majority of cases of stroke in the presence of carotid artery dissection.

Cerebral infarction causes irreversible damage to the brain. In one study of patients with carotid artery dissection, 60% had infarcts documented on neuroimaging.

Pathogenesis

Embolism from thrombus forming at the dissection site is thought to play the major part in stroke pathogenesis.

This suggestion is supported by transcranial Doppler studies showing cerebral micro- emboli soon after dissection, and by the distribution of infarcts after dissection, which suggests an embolic pattern.

Clinical features

The signs and symptoms of carotid artery dissection may be divided into ischemic and non-ischemic

categories:

Non-ischemic signs and symptoms

Headache or neck pain

Decreased pupil size with drooping of the upper eyelid (Horner syndrome)

Ischemic signs and symptoms

Temporary vision loss

Ischemic stroke

see Hypoglossal nerve palsy due to internal carotid artery dissection.

Diagnosis

If a diagnosis of spontaneous internal carotid artery dissection is under consideration, laboratory studies are largely irrelevant for diagnostic purposes. However, if contrast-enhanced computed tomography (CT) or arteriography is planned, it is appropriate to obtain a baseline creatinine concentration.

With the use of noninvasive imaging, including magnetic resonance and computed tomography angiography, the diagnosis of carotid dissection has increased in frequency

Treatment

Treatment options include thrombolysis, antiplatelet or anticoagulation therapy, endovascular or surgical interventions. The choice of appropriate therapy remains controversial as most carotid dissections heal on their own.

The goal of treatment is to prevent the development or continuation of neurologic deficits.

The risk of early recurrence of stroke has led many clinicians to advocate the use of anticoagulation from presentation until 3 or 6 months after dissection. However others believe that antiplatelet drugs might be sufficient.

Anticoagulants might prevent embolism from a fresh thrombus but they are also more hazardous than antiplatelet drugs and can result in extension of the intramural haemorrhage, which occurs in a third of patients according to MRI [2].

see Cervical Artery Dissection in Stroke Study (CADISS)

Endovascular management of acute ischemic stroke (AIS) in the setting of carotid artery dissection (CAD) is a feasible, safe, and promising strategy [3].

Outcome

Some studies suggest a significantly increased risk of stroke in patients presenting with dissection either with local symptoms, such as headache and Horner's syndrome, or with stroke or transient ischaemic attack, with estimates of the risk of secondary stroke after presentation of 15–20% [4) 5) 6)] although other studies have reported a much lower proportion [7)].

Case reports

A patient with transient left hypoglossal nerve palsy caused by mechanic compression from intramural hematoma in higher extracranial portion of dissected carotid artery confirmed in MRI and CT scans [8)].

1)
Debette S, Leys D. Cervical artery dissections: predisposing factors, diagnosis, and outcome. Lancet Neurol 2009; 8: 668–78.
2)
Machet A, Fonseca AC, Oppenheim C, et al. Does anticoagulation promote mural hematoma growth or delayed occlusion in spontaneous cervical artery dissections? Cerebrovasc Dis 2013; 35: 175–78.
3)
Haussen DC, Jadhav A, Jovin T, Grossberg J, Grigoryan M, Nahab F, Obideen M, Lima A, Aghaebrahim A, Gulati D, Nogueira RG. Endovascular Management vs Intravenous Thrombolysis for Acute Stroke Secondary to Carotid Artery Dissection: Local Experience and Systematic Review. Neurosurgery. 2015 Oct 21. [Epub ahead of print] PubMed PMID: 26492430.
4)
Biousse V, D'Anglejan-Chatillon J, Toouboul PJ, Amarenco P, Bousser MG. Time course of symptoms in extracranial carotid artery dissections: a series of 80 patients. Stroke 1995; 26: 235–39.
5)
Beletsky V, Nadareishvili Z, Lynch J, Shuaib A, Woolfenden A, Norris JW. Cervical artery dissection: time for a therapeutic trial? Stroke 2003; 34: 2856–60.
6)
Weimar C, Kraywinkel K, Hagemeister C, et al. German Stroke Study Collaboration. Recurrent stroke after cervical artery dissection. J Neurol Neurosurg Psychiatry 2010; 81: 869–73.
7)
Touzé E, Gauvrit JY, Moulin T, Meder JF, Bracard S, Mas JL. Multicenter Survey on Natural History of Cervical Artery Dissection. Risk of stroke and recurrent dissection after a cervical artery dissection: a multicenter study. Neurology 2003; 61: 1347–51.
8)
Mes M, Palczewski P, Szczudlik P, Łusakowska A, Maj E, Gawel M. Hypoglossal nerve palsy as an isolated syndrome of internal carotid artery dissection: A review of the literature and a case report. Neurol Neurochir Pol. 2018 Jul 6. pii: S0028-3843(17)30477-2. doi: 10.1016/j.pjnns.2018.06.006. [Epub ahead of print] PubMed PMID: 30082078.