

Vascular Dissection (Carotid, Vertebral)

Updated: April 17, 2010

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PATHOPHYSIOLOGY

- tear within arterial wall → **blood extravasation (longitudinal dissection) into medial or subintimal layers** → expanded arterial wall → **lumen compromise**.
- dissection can produce second intimal tear, allowing blood clot to reenter lumen → embolization.
- clot is absorbed within several weeks, and lumen usually returns to its normal size.
- **most commonly involved** – **ICA high in neck** (between C₂ and skull base) - carotid artery is stretched over transverse process of C₂ by any injury involving hyperextension and rotation of head and neck.
- **less frequently involved** – verteobasilar system (most mobile V₁ and V₃ segments), intracranial ICA, MCA.

ETIOLOGY

- **trauma** (blunt*, penetrating, or even trivial**), but may occur **spontaneously**.
 - *e.g. fall on popsicle in mouth, abuse with whiplash-shake injuries
 - **e.g. prolonged neck holding in eccentric position, chiropractic manipulation
- usually occur in young people.
- **associated conditions** (congenital / degenerative changes in vessel wall) - **fibromuscular dysplasia** (!), Marfan's syndrome, Ehlers-Danlos type IV syndrome, pseudoxanthoma elasticum, atherosclerosis, migraine, pronounced vessel tortuosity, moyamoya, cystic medial degeneration, pharyngeal infections, α₁-antitrypsin deficiency, luetic arteritis.

CLINICAL FEATURES

1. **Pain** (important symptom that helps to diagnose this cause of brain ischemia!!!):
 - carotid dissections** → ipsilateral throbbing headache (forehead, eye, face), intense local sharp pain in neck.
 - vertebral dissections** → pain in occiput, posterior neck.
2. **Ischemia** – TIAs (due to luminal compromise), stroke (due to embolization within first few days). Arterial dissection is important cause of ischemic strokes in young people!
3. Other associated symptoms:
 - 1) **Horner syndrome** (in carotid dissection)
 - 2) self-audible **bruits** (but auscultation is poor screening tool)
 - 3) **tenderness** over neck
 - 4) pulsatile **tinnitus**.

COMPLICATIONS

- if dissection extends between media and adventitia:
 - 1) **dissecting aneurysms** → space-occupying lesions (compress adjacent cranial nerves, brain parenchyma), SAH. see p. Vas25 >>
 - 2) **tears through adventitia** → SAH.

DIAGNOSIS

CT / MRI can directly visualize intramural bleeding and expansion.

- MRI *after few days* - rim of high signal (subacute intramural hematoma) expanding outer diameter of artery and narrowing its lumen.
- MRI *in acute stage* - intramural hematoma is isointense to muscle - difficult to detect.

Ultrasound - reliable screening tool:

B-mode ultrasound - tapering of ICA lumen, irregular membrane crossing lumen, true and false lumens.

Duplex scans - decreased pulsatility, intravascular abnormal echoes, decreased flow.

TCD - effect of neck pathology on poststenotic intracranial circulation:

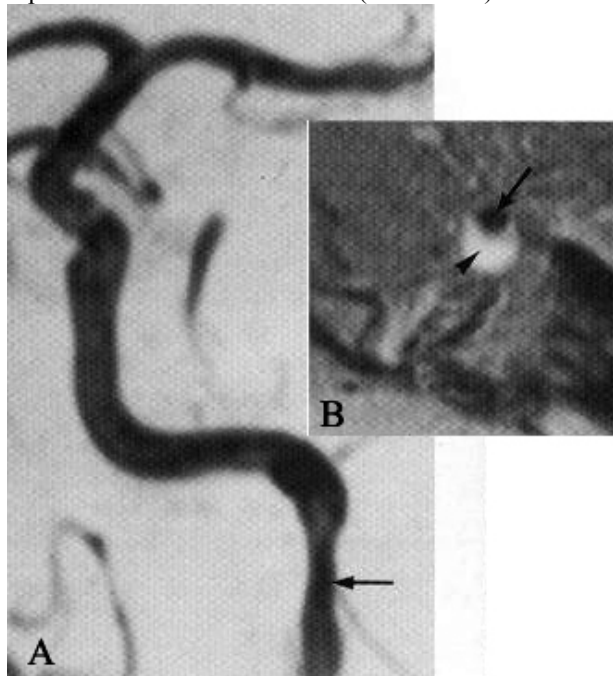
Diminished intracranial velocities in young patients who have normal ICA bifurcations → diagnosis of dissection is quite likely.

MRA - reliable noninvasive diagnosis for extracranial ICA.

ICA dissection (3D TOF MRA):

A. Focal narrowing as ICA enters skull base (*arrow*).

B. Axial image through that level - flow void in residual vessel lumen (*arrow*) and high signal crescent, which represents intramural hematoma (*arrowhead*).



Conventional **angiography** (more useful for VA);

- regions of **severe narrowing** ("string sign") or **total occlusion** beginning > 2 cm distal to ICA origin, sparing siphon, and having gradually tapering segment.
- aneurysmal sacs or outpouchings.

Any **trauma patient** having focal neurological deficits (esp. with Horner's syndrome) that cannot be explained from imaging studies → early angiography to diagnose carotid artery dissection!

TREATMENT

Most extracranial dissections *heal spontaneously!*

- if complete occlusion has occurred, arteries often do not recanalize.
- arteries that retain some residual lumen invariably heal and become normal.

ANTICOAGULANTS shortly after dissection should prevent stroke;

N.B. risk of embolization exists *only during acute period!* TIAs often precede infarction, leaving time for therapeutic intervention!

- do not seem to increase extent of dissection.
- **HEPARIN** → **WARFARIN** is continued until lumen is not severely compromised (e.g. for 3-24 months; target INR 2-3) → **ANTIPLATELET AGENTS** for at least 2 years.
- anticoagulation is contraindicated in intracranial dissections complicated by SAH.

SURGICAL REPAIR indications:

- 1) SAH
 - 2) persistent high-grade stenosis
- location high in neck makes surgical carotid repair difficult.
 - *endovascular stenting* is viable option.

BIBLIOGRAPHY for ch. "Neurovascular Disorders" → follow this [LINK >>](#)