Vascular Dissection (Carotid, Vertebral)

PATHOPHYSIOLOGY

- tear within arterial wall → blood extravasation (longitudinal dissection) into medial or subintimal layer → 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 – vertebrobasilar system (most mobile V and V segments), intracranial ICA, MCA.

ETIOLOGY

- trauma (blunt*, penetrating, or even trivial**; see also p. TSS21 >>), but may occur spontaneously.
- e.g. fall on popsicle in mouth, abuse with whiplash-shakes injuries
- *e.g. prolonged neck holding in eccentric position, chiropractic manipulation, coughing
- 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, alpha-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): N.B. in ICA dissection Horner syndrome is incomplete – sympathetic fibers to face sweat glands and blood vessels travel along ICA (esp. to lower face); see p. Eye19 >>
   2) self-anultrate bruise (see auscultation is poor screening tool)
   3) tenderness over neck
   4) pulsatile tinnum.

COMPLICATIONS

- if dissection extends between media and adventitia:
  1) dissecting aneurysm → space-occupying lesions (compress adjacent cranial nerves, brain parenchyma), SAH. see p. Vasc25 >>
  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.

CTA – most reliable noninvasive diagnosis!

Left vertebral artery intimal flap (arrow) secondary to vertebral artery dissection.
**Vascular Dissection (Carotid, Vertebral)**

**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):**
  1. Focal narrowing as ICA enters skull base (arrow).
  2. 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 / Antiplatelets** 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 (s. flow-limiting) stenosis
- location high in neck makes surgical carotid repair difficult.

**Endovascular stenting** is a modern option.
- stenting in mobile neck carries the risk of stent fracture (cf. intracranial stenting or stenting VA ostia – much less vessel movement); solution might be a softer stent (e.g. pipeline).
- **carotid dissections:**
  - with complete occlusion - observation (continue ASA for life) with slight contrast wisp (high grade stenosis):
    a) high intracranial - do not touch it if brain is well perfused (risk of even slightest dissection extension and may occlude PComA ostia → massive
stroke), if brain hypoperfused - document it with pCT and proceed with stenting.

- indications for carotid dissection stenting:
  - worsening exam on antiplatelets
  - worsening pseudoaneurysm on repeat angio
  - brain perfusion asymmetry on angio (i.e. flow limiting dissection)

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<tr>
<th>Vasculardissection (carotid, vertebral)</th>
<th>indications for carotid dissection stenting</th>
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<tbody>
<tr>
<td>High energy transfer mechanism associated with:</td>
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<tr>
<td>Displaced mid-facet fracture (e.g. for I and II)</td>
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<td>Basilar skull fracture with carotid canal involvement</td>
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<td>ChI consistent with DAI and GCCS 6</td>
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<tr>
<td>C-spine fracture: Any C1-C3, subluxation, transverse foramen involvement, near hanging with acute brain injury</td>
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<td>Clohtesline type injury (seat belt abrasion) with significant swelling, pain, or altered M5.</td>
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### Bibliography for ch. “Neurovascular Disorders”
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