Vascular Dissection (Carotid, Vertebral)

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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 C_2 and skull base) - carotid artery is stretched over transverse process of C_2 by any injury involving hyperextension and rotation of head and neck.
- less frequently involved – vertebrobasilar system (most mobile V_1 and V_3 segments), intracranial ICA, MCA.

ETIOLOGY

– **trauma** (blunt*, penetrating, or even trivial**; see also p. TrS21 >>), 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, 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, α_1-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 ECA (esp. to lower face) see p. Eye19 >>
   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.

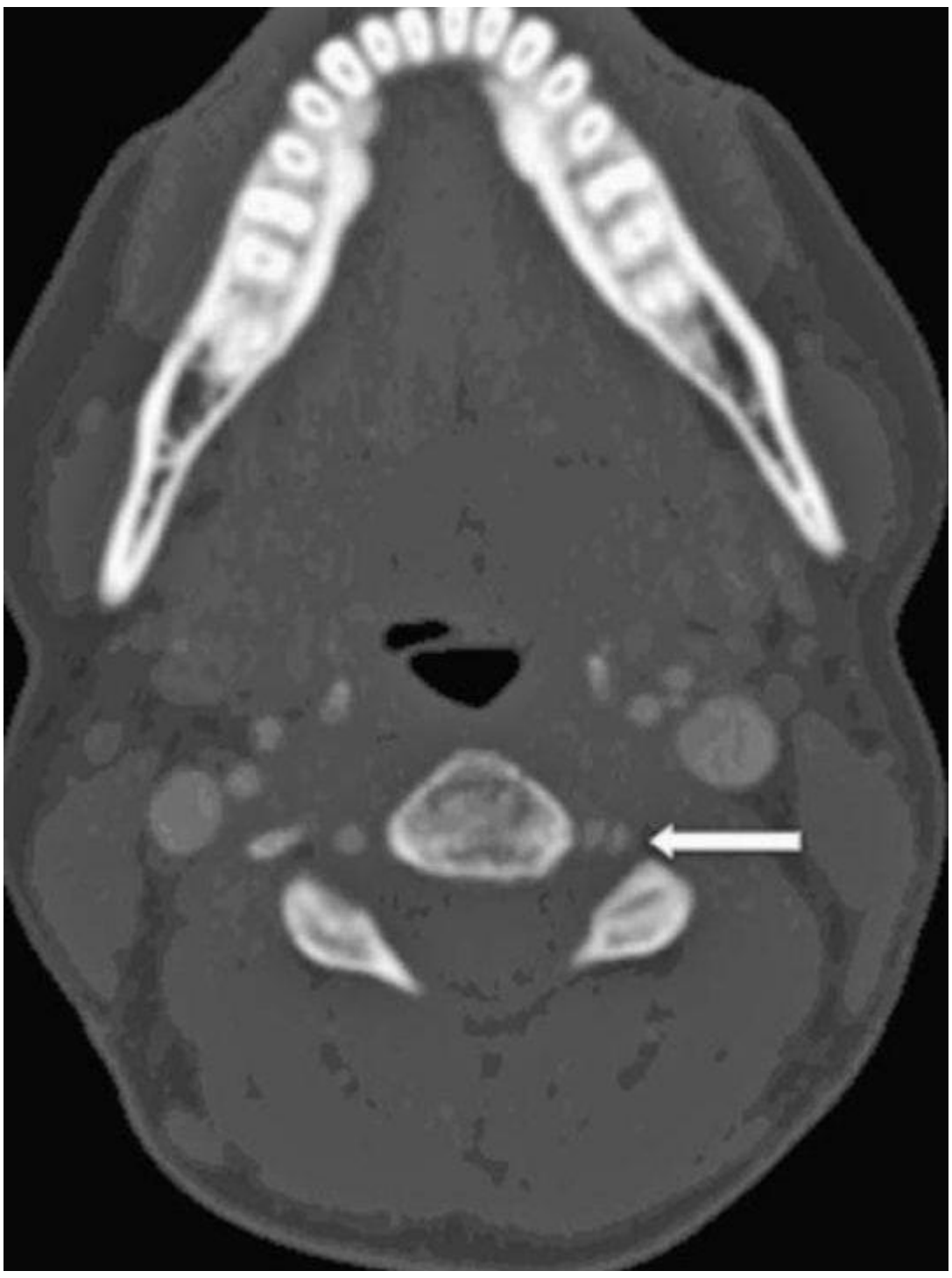
DIAGNOSIS

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.

CTA – most reliable noninvasive diagnosis!

Left vertebral artery intimal flap (*arrow*) secondary to vertebral artery dissection:



Ultrasound - reliable screening tool:

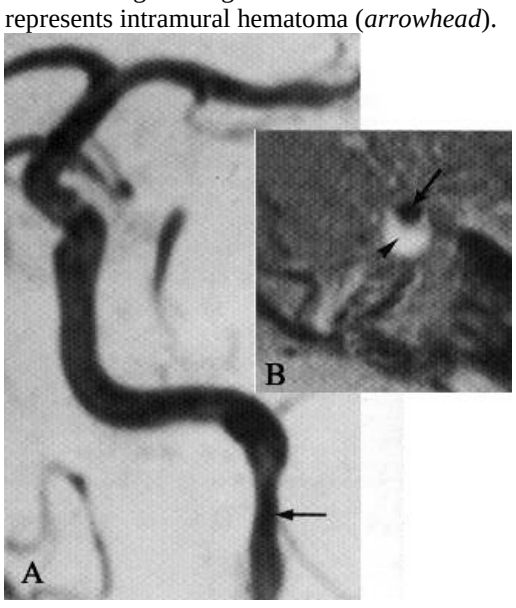
B-mode ultrasound - tapering of ICA lumen, irregular membrane crossing lumen, true and false lumens.

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 / **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.

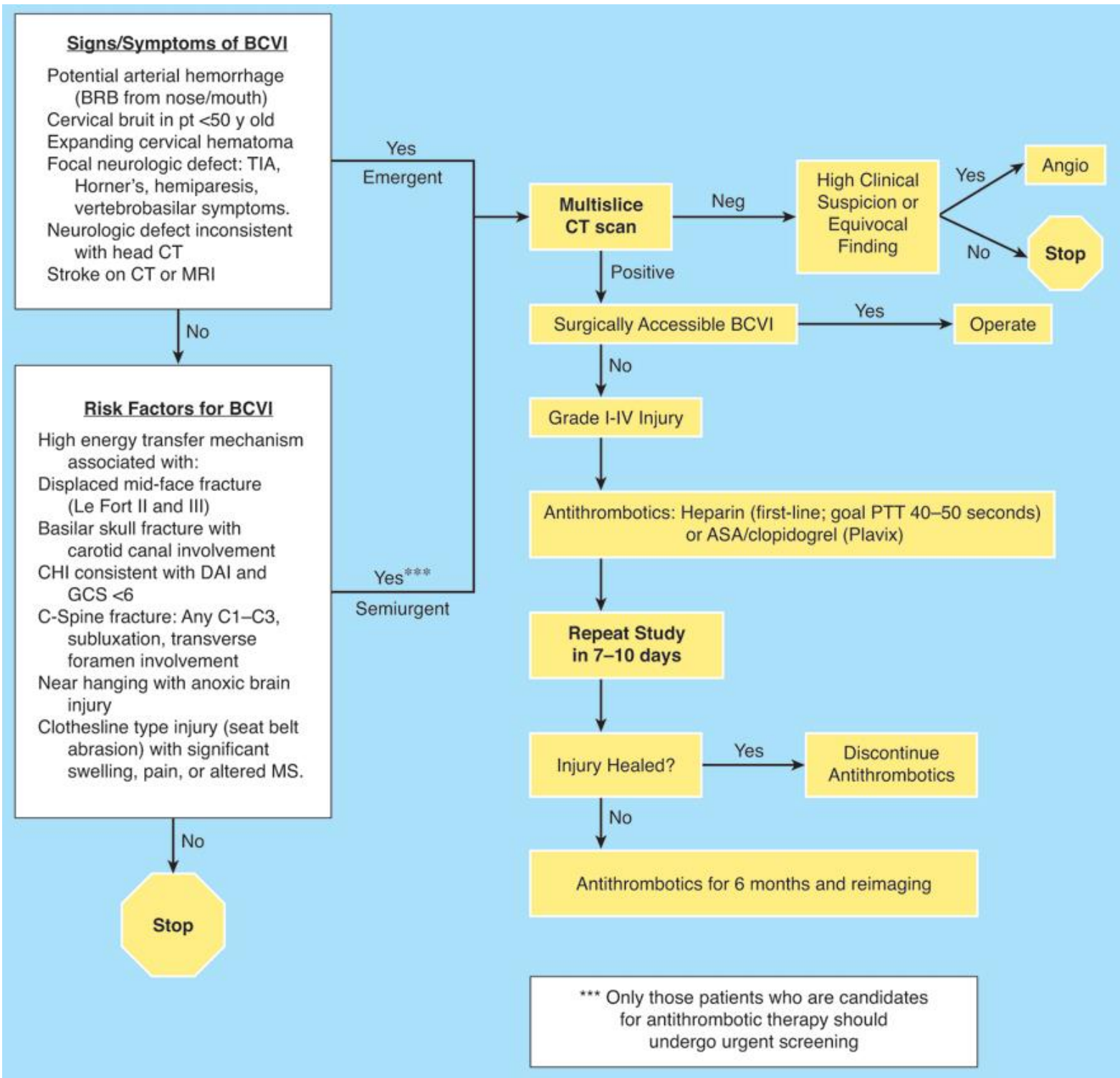
Endovascular stenting is a modern option.

- 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

b) low in neck (proximal) - OK to stent (e.g. pipeline)

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



BIBLIOGRAPHY for ch. "Neurovascular Disorders" → follow this LINK >>