

# 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<sub>2</sub> and skull base) - carotid artery is stretched over transverse process of C<sub>2</sub> by any injury involving hyperextension and rotation of head and neck.
- **less frequently involved** – vertebrobasilar system (most mobile V<sub>1</sub> and V<sub>3</sub> 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<sub>1</sub>-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.

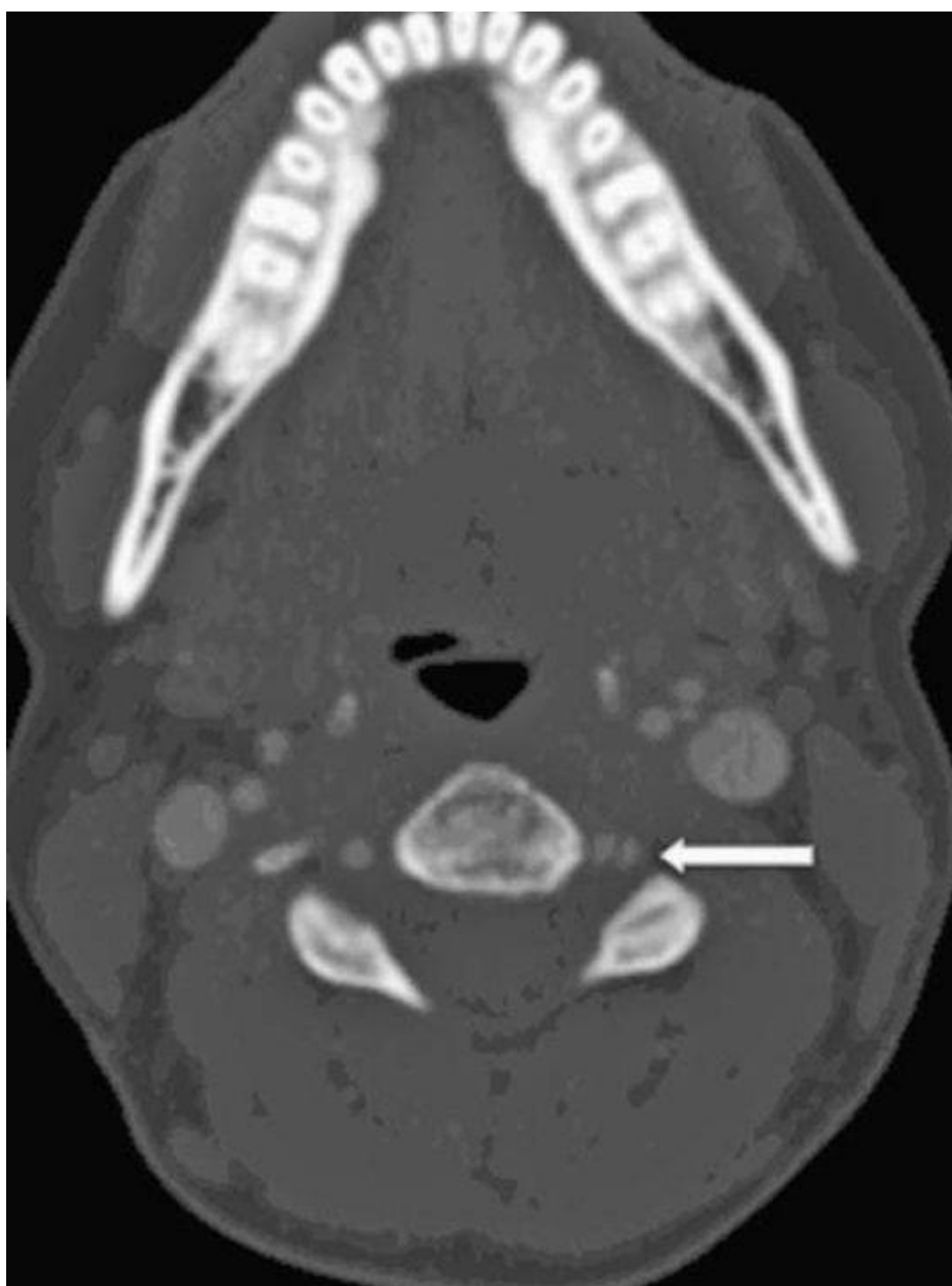
## DIAGNOSIS

**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:

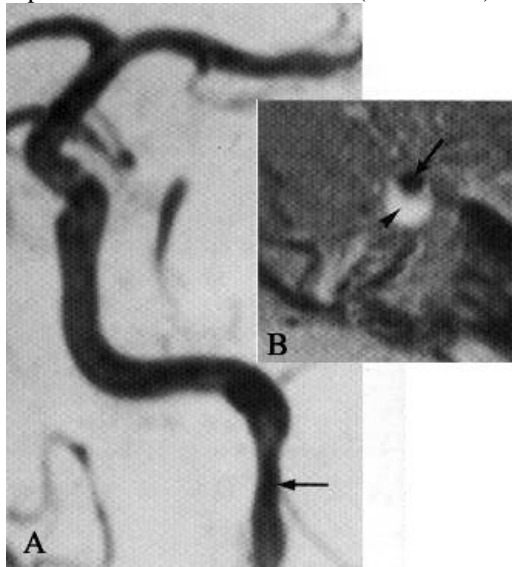
Diminished intracranial velocities in young patients who have normal ICA bifurcations  
→ diagnosis of dissection is quite likely.

**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 viable option.

Indications for carotid dissection stenting:

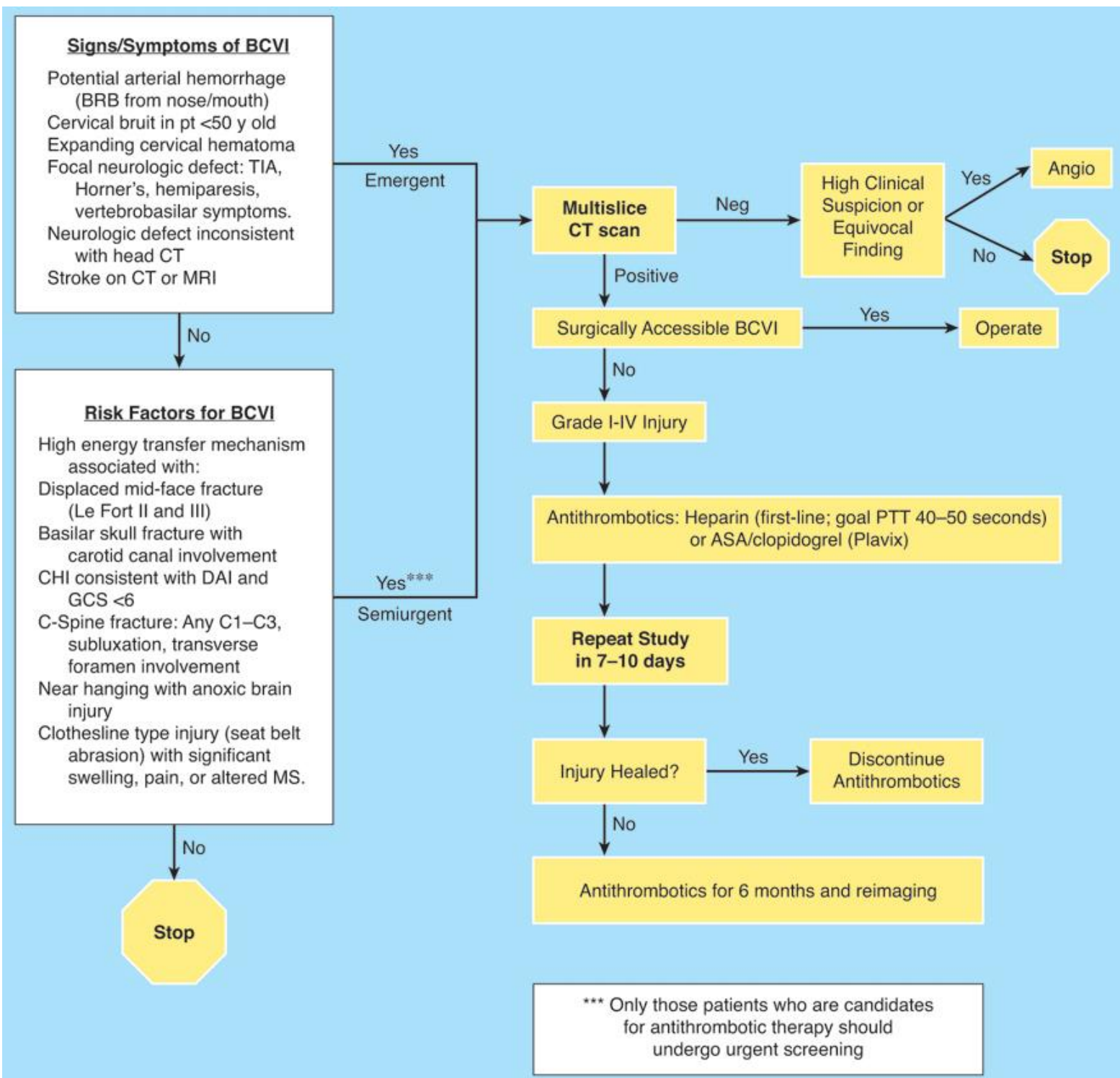
1. Patient cannot take antiplatelets
2. Worsening exam on antiplatelets
3. Worsening pseudoaneurysm on repeat angio
4. Brain perfusion asymmetry on angio (i.e. flow limiting dissection)

Carotid dissections:

with complete occlusion - NTD (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)



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