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Cerebral Artery Dissection

History of Presenting Illness:

RIGHT AFTER THE INJURY:

- young person
- severe occipital headache
- posterior neck pain
- maybe reduced ROM
- recent minor head or neck injury.
  - trauma is generally from a trivial mechanism but is associated with some degree of cervical distortion.

~ 3 DAYS AFTER THE INJURY:

maybe much longer, ? weeks….

- Focal Neuro Signs in 85%
- Usually looks like a LATERAL MEDULLARY SYNDROME (!)
- Facial pain and numbness are commonest presenting symptoms
- Plus, you can get vertigo, disequilibrium, nystagmus, Horner's; nausea, dysphagia, dysarthria, etc etc...

PHYSICAL EXAMINATION

Be warned, that it may be tempting to attribute the neck pain and headache to a musculoskeletal problem.

FULL NEURO EXAM:

- Looking for CN palsies
- Looking for cerebellar dysfunction
- Looking for lateral medullary syndrome signs

IS THERE POINT TENDRENESS ON THEIR NECK?

- It may be a C-spine fracture

Are they FEBRILE?

- It may be a brain abscess or meningitis

HAVE THEY GOT PAPILLOEDEMA?

- Better not miss an intracranial hematoma, subdural or epidural

INVESTIGATIONS:

Need to rule out the distressing possibilities: eg.

- Subarachnoid haemorrhage → CT of head; LP after 12 hrs… xanthochromia?
- Ischaemic Stroke → CT of head; { But if they are having an event in the posterior fossa, CT is next to useless, so just go with the MRI
- Vasculitis → ESR, CRP;
- C-spine fracture → Neck X-ray, with odontoid views

OTHERWISE: you are mainly interested in…

- Establishing that there is a vertebral artery dissection
  - Establishing a baseline for anticoagulation.

MRI — MRI angiography will show your dissection, the intramural hematoma, mural thrombi, etc

PTT, aPTT, INR — They are going on anticoags soon, so you need this screen

A YOUNG PERSON WITH WHAT SEEMS LIKE A BUNCH OF STROKE SYMPTOMS!
Does not compute…

WHATS ACTUALLY GOING ON?

There is a hematoma in the middle of the arterial wall, and its expanding- thus effectively occluding the lumen, and maybe even causing a mass effect.

THIS CAN EVOLVE IN SEVERAL WAYS:

- **If its small,** its asymptomatic. No cause for dismay.
- **If its Extensive:** (those that extend intracranially and involve the basilar artery) result in infarctions of the brain stem, cerebellum or, rarely, the spinal cord.
- **After a while,** the weakened dilated arterial wall can form a **PSEUDOANEURYSM** which is just itching to rupture and become one of **those 5% of subarachnoid haemorrhages** which get caused by dissected arteries.
- The whole business of arterial dissection causes a **LOW FLOW STATE** which leads to stasis and will eventually begin to form CLOTS which can sail downstream and cause a more **conventional stroke syndrome**.

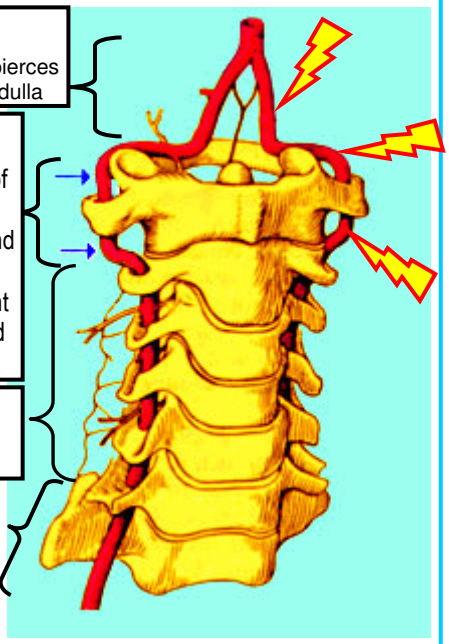
BARELY RELEVANT ANATOMY:

SEGMENT IV:
intracranial segment: begins as it pierces the dura; continues up to pons / medulla

SEGMENT III
begins at the transverse foramen of C2, runs posterolaterally to loop around the posterior arch of C1, and passes subsequently between the atlas and the occiput. This segment is encased in muscles, nerves, and the atlanto-occipital membrane

SEGMENT II
runs entirely within the transverse foramina of C5/C6 to C2.

SEGMENT I
Runs from subclavian artery to the transverse foramina of C5 or C6



Seg. III is the one where it usually dissects; may continue into Seg. IV

MANAGEMENT:

Major goal of treatment is to prevent THROMBOEMBOLIC COMPLICATIONS.

- **SO: if CT shows that there is NO BLEEDING- START ANTICOAGULANTS.**
  - **Heparinise until therapeutic range INR (2.0-3.0)**
  - **Taper heparin off** while preparing for warfarin
  - **Warfarin for 3-6months thereafter**
  - **Regular monitoring of INR**
  - **WATCH THEM CAREFULLY for new neuro signs and symptoms, as there may be subarachnoid haemorrhage from the burst artery**

? is here any need to get neurosurgeons involved?

- There may be, if there is a pseudoaneurysm and it looks like its about to burst.

SUPPORTIVE MANAGEMENT:

THEY ARE IN PAIN: attend to analgesia: paracetamol + opiates

- **They are dysarthric:** arrange speech therapy consult
- **They have hemiparesis:** arrange physio consult, walking aids etc.
- **They are nauseous: METOCLOPRAMIDE Intravenously**
- **They are not able to swallow:** Nil by mouth initially
  - Arrange formal swallowing assessment
  - Nasogastric feeding until swallowing cleared
  - **ASPIRATION PNEUMONIA** is the only real danger if there is no likelihood of subarachnoid haemorrhage or ischaemic stroke.

METOCLOPRAMIDE (MAXOLLON):

Antiemetic of choice for this sort of nausea.

- antagonises the action of dopamine in the chemoreceptor trigger zone, which is located in the area postrema in the floor of the fourth ventricle. **This disables the vomit / nausea centre.**
- works peripherally by stimulating 5-HT4 receptors, **increasing gastric peristalsis and removing the stimulus to vomit.**
- In high doses, metoclopramide antagonises 5-HT3 receptors, thereby having a similar action to drugs such as ondansetron.

ONDANSETRON (ZOFRAN): the OTHER antiemetic

- **According to MIMS:** Ondansetron is a potent, highly selective $5HT_3$-receptor antagonist. Its precise mode of action in the control of nausea and vomiting is not known. Chemotherapeutic agents and radiotherapy may cause release of 5HT in the small intestine, initiating a vomiting reflex by activating vagal afferents via $5HT_3$-receptors. Ondansetron blocks the initiation of this reflex. Activation of vagal afferents may also cause a release of 5HT in the area postrema, located on the floor of the fourth ventricle, and this may also promote emesis through a central mechanism…
- **DOES NOT SEDATE**