HIGH YIELD

HEAD & NECK

EMERGENCIES

CNS infection: Meningitis & Encephalitis

Status Epilepticus

Giant cell arteritis

Raised ICP

Intracranial haemorrhage & basal skull #s

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Intended Learning Outcomes:
(for your own reference only after the session)

CNS Infection:

- Identify the 2 most common organisms implicated in bacterial meningitis from their gram stains
- Interpret LP results, to discriminate between likely viral or bacterial cause of meningitis
- Presentation and management of meningitis
- Clinical features and management of encephalitis

Giant Cell Arteritis:

- Risk factors for GCA
- Presenting features
- Ix and empirical managment

Status Epilepticus:

- Define/recognise and management status

Raised ICP

- Define and recognise the clinical signs of raised ICP
- Outline the emergency management of raised ICP

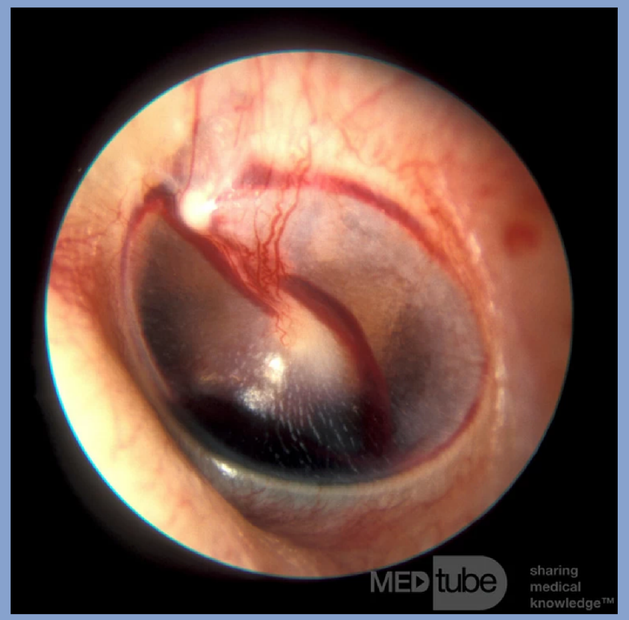
Cranial # and intracranial haemorrhage

- Outline a classical illness script for extradural haematoma
- Outline a classical illness script for subdural haematoma
- Identify and describe CT head suggestive of ICH
- Explain the CT appearance with reference to anatomy of the osteology of the skull and meninges
- Identify the clinical signs suggestive of basal skull #

Brain warm up!

Battle's sign (mastoid ecchymosis)

Haemotympanum

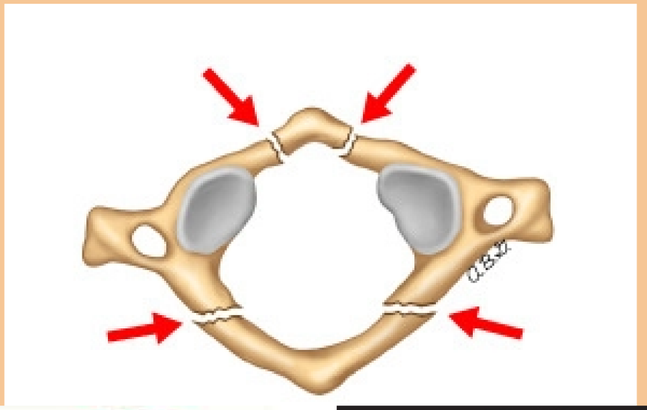


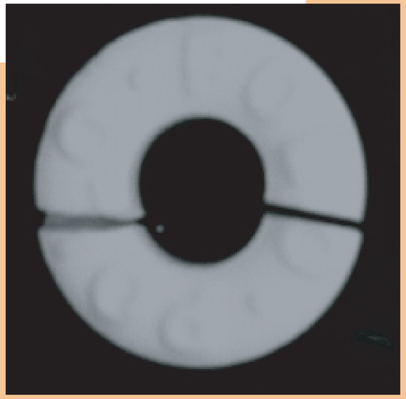
Bilateral periorbital ecchymosis, 'Racoon Eyes'

Cervical #'s

C1/Atlas

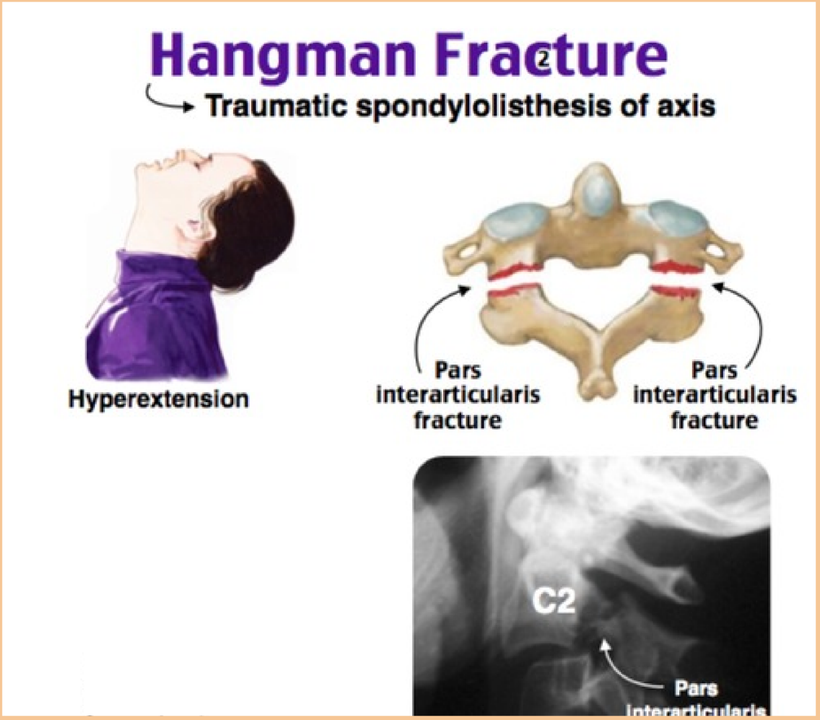
Jefferson





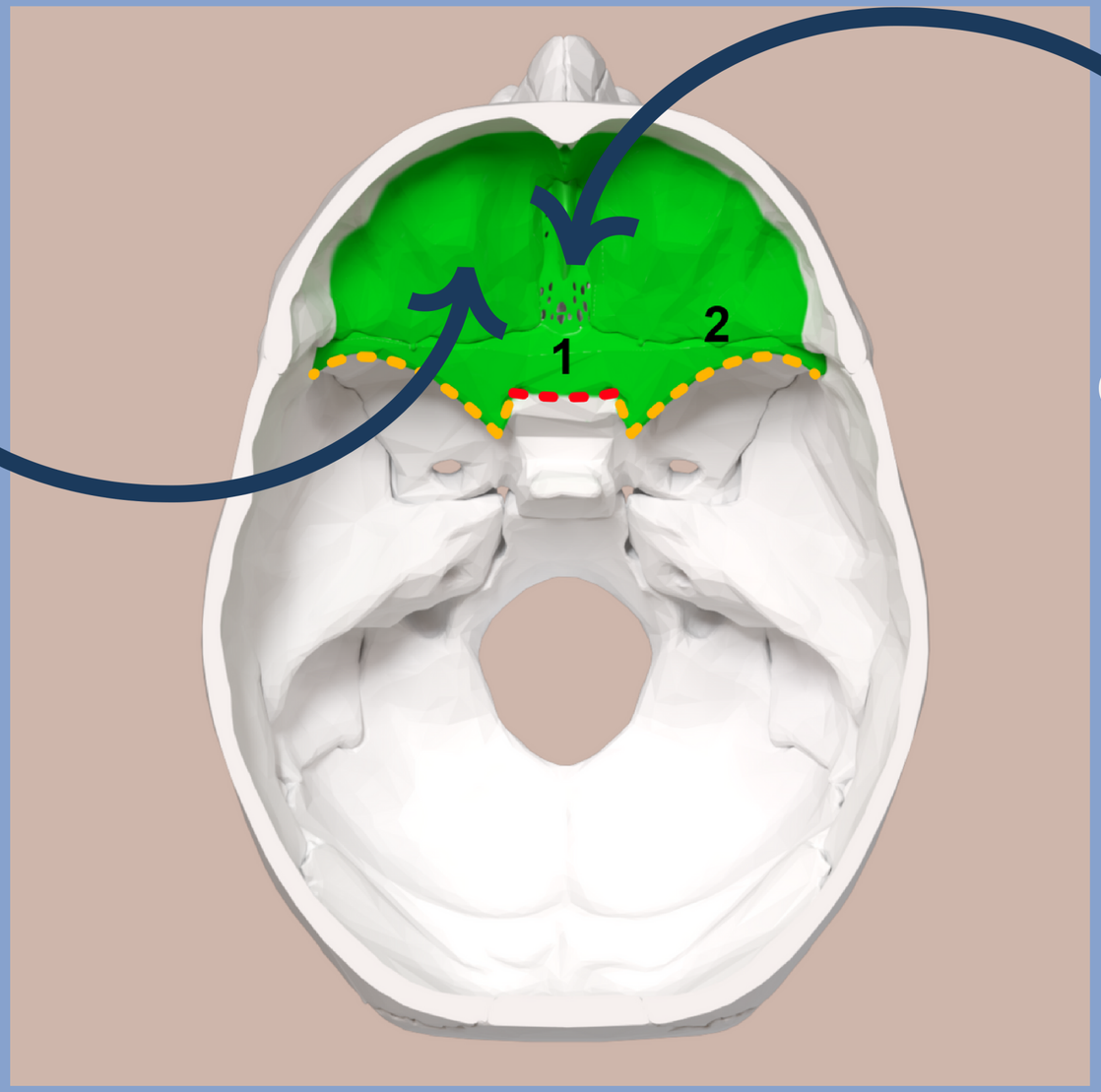
C2/Axis

Hangman's



Anatomical basis of basal skull # linked to clinical signs

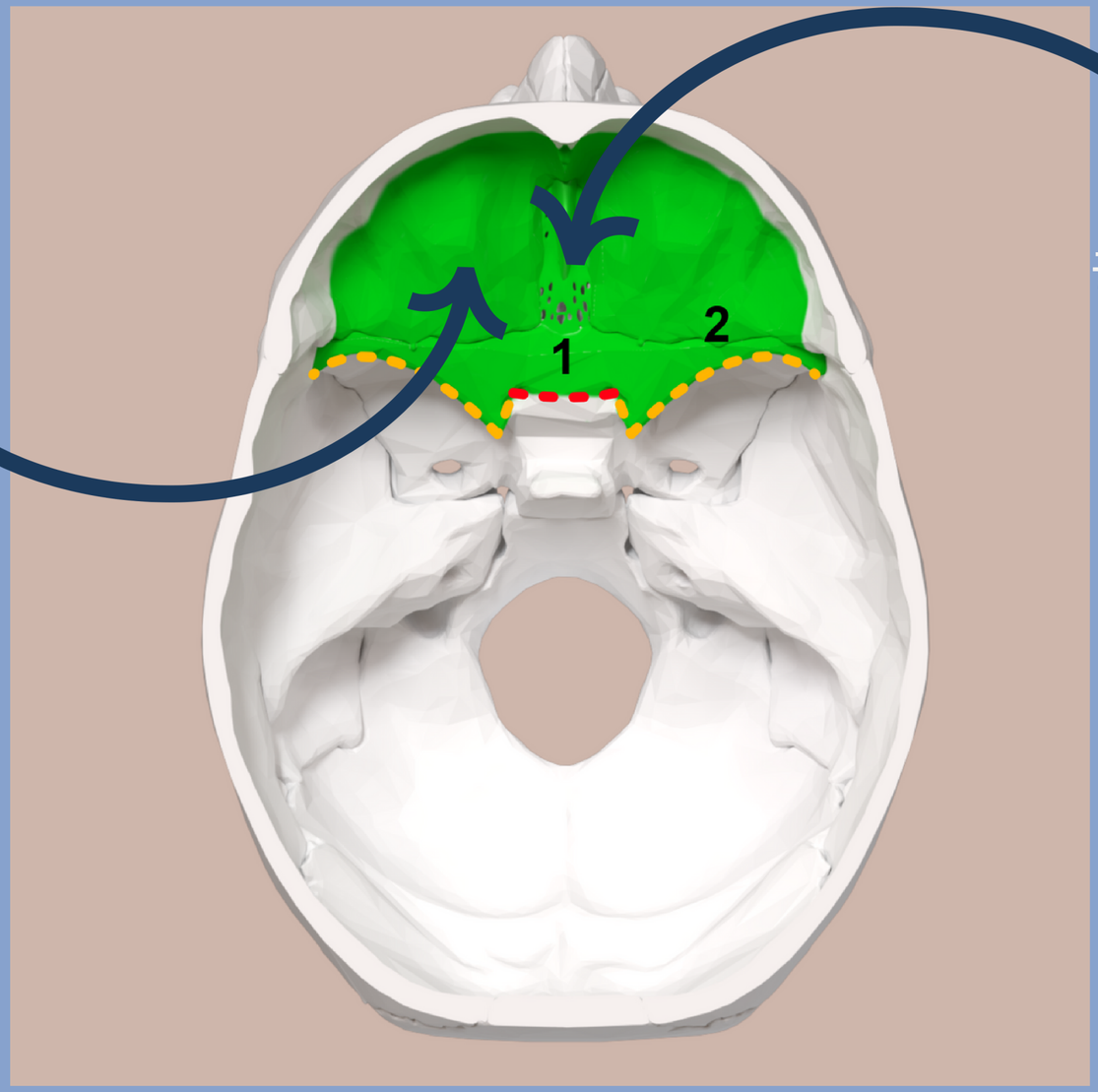
orbital plate



cribiform plate of ethmoid bone

Anatomical basis of basal skull # linked to clinical signs

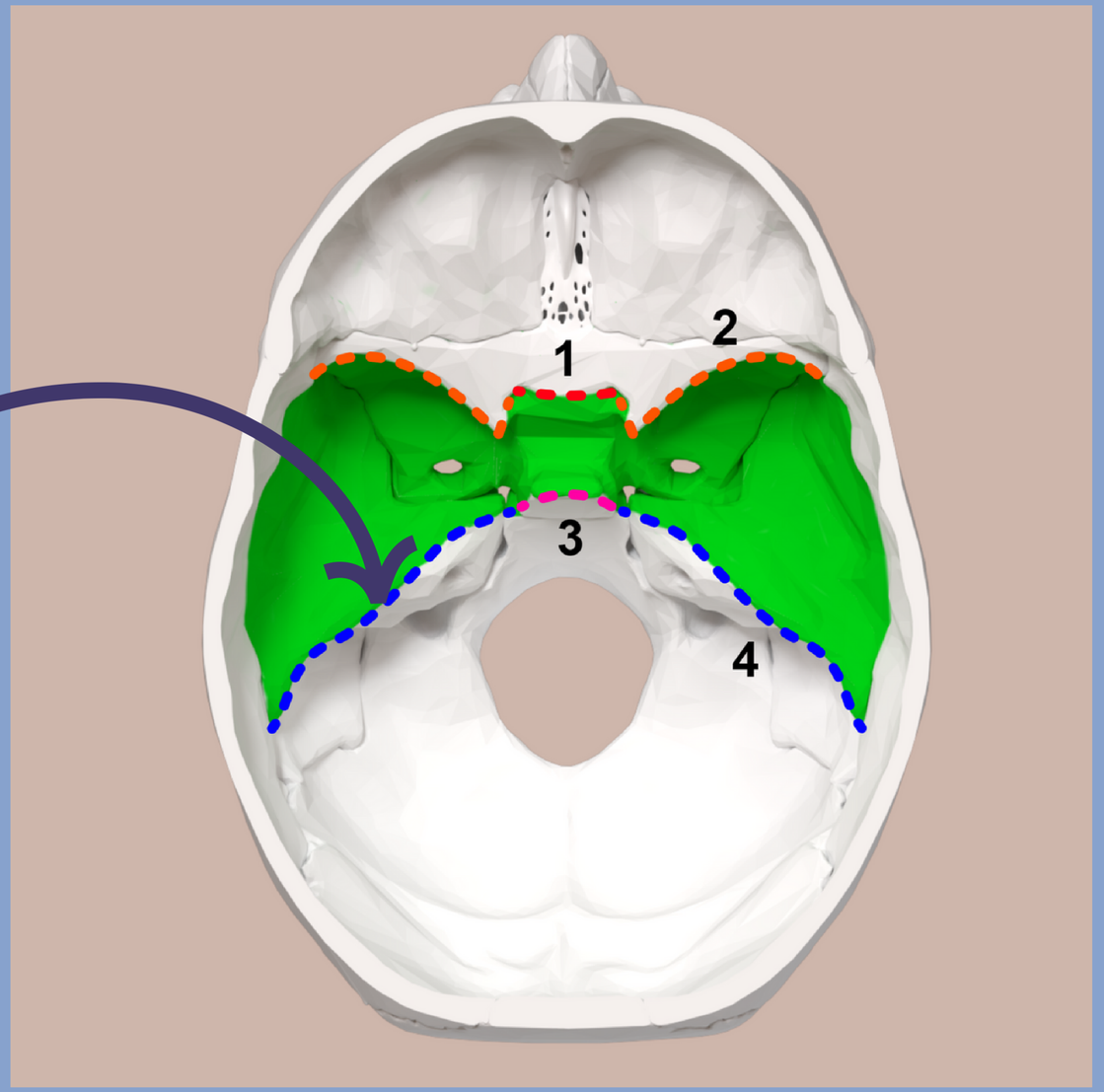
orbital plate
= bilateral peri-orbital ecchymosis



cribiform plate
= CSF rhinorrhoea

MCF: # of Petrous part of temporal bone (3)

- Battle's sign - mastoid ecchymosis
- Haemotympanum
- CSF/blood otorrhea



S

A 25 year old man who was involved in a fight at outside a bar, He was knocked to the floor, were he remained unconscious for a few minutes.

B

He subsequently walked off from the scene of the accident. His partner rang hours later as he has started to become confused followed by drowsiness

A

- GCS: 9
- BP: 140/95
- HR: 102
- RR: 16, 02 Sats 99%

R

ETA: 5 minutes

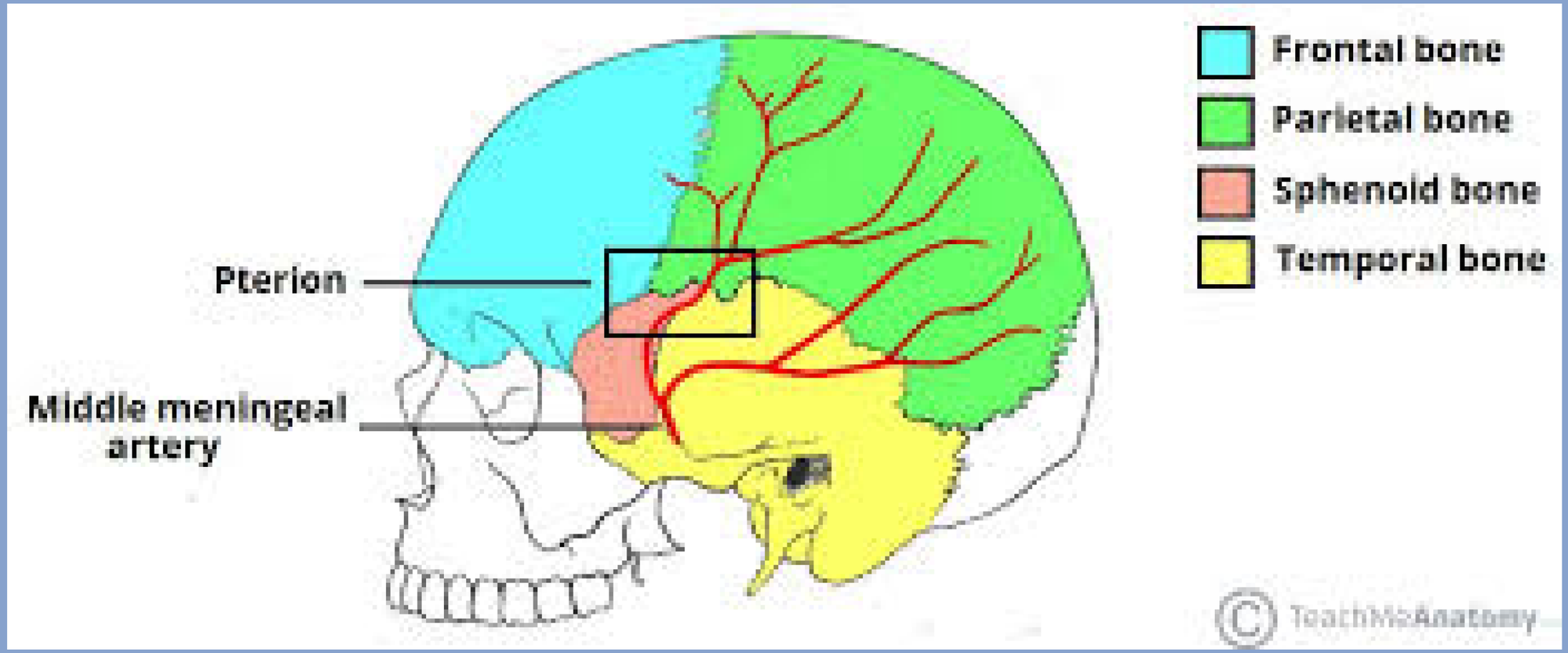
Initial Management of Patient 1?

A-E assessment & Initial stabilisation

Ix: FBC, U&Es, LFTs, clotting, group & save, cross match
VBG: glucose, lactate

CT

Bright white/hyperdense lentiform/ biconvex appearance suggestive of extradural


Frontal bone
Parietal bone
Sphenoid bone
Temporal bone
Pterion
Middle meningeal artery
© TeachMeAnatomy

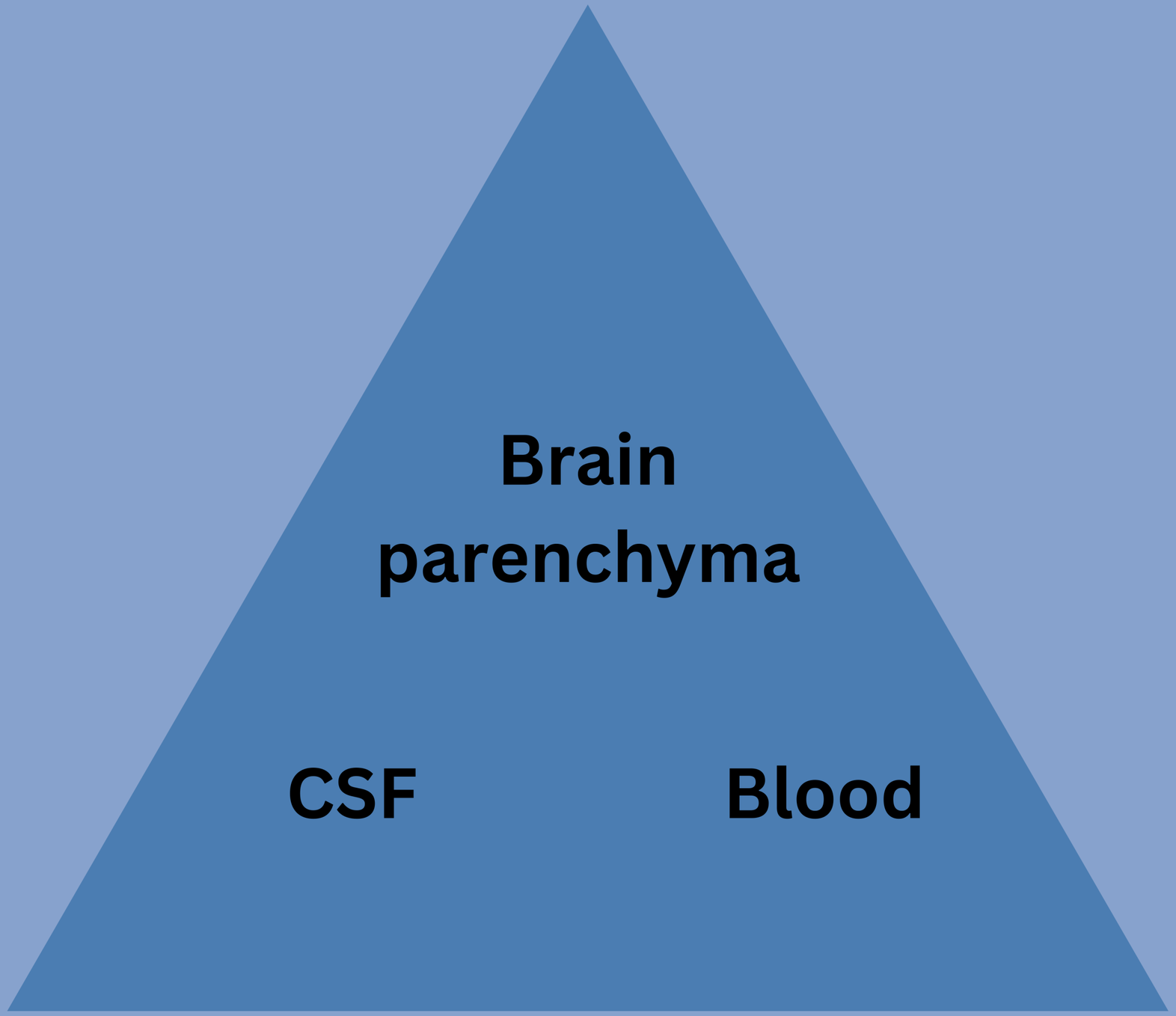
Patient 2:

Headache with gradual onset of neurological symptoms

Normal range of ICP:

Adults: 5-l5mmHg

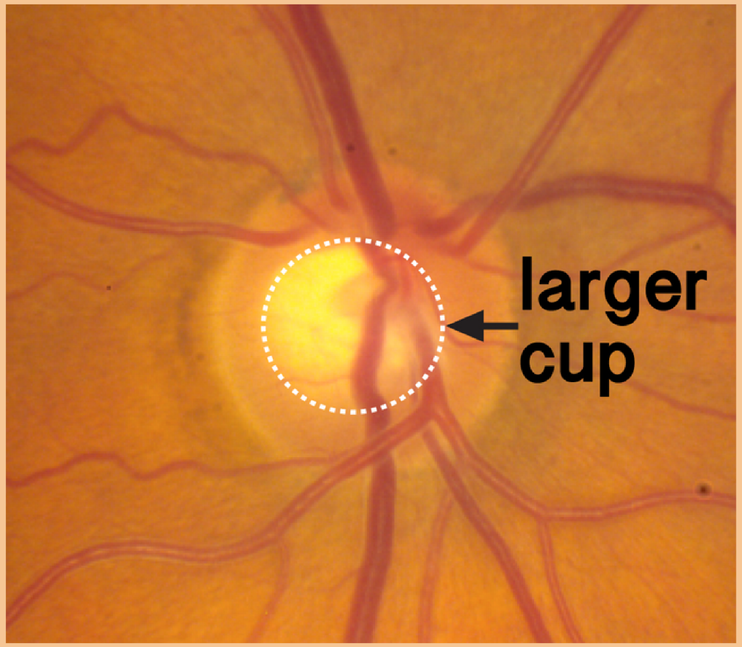
Pathological raised ICP in adults is sustained (minutes) rise in ICP >20mmHg


Brain
parenchyma
CSF
Blood

Early signs

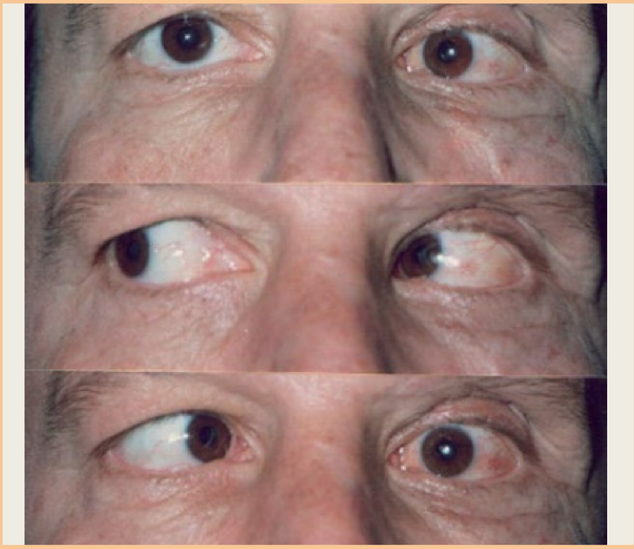
- Decreased visual acuity
- Visual field defect
- Papilledema

Late signs

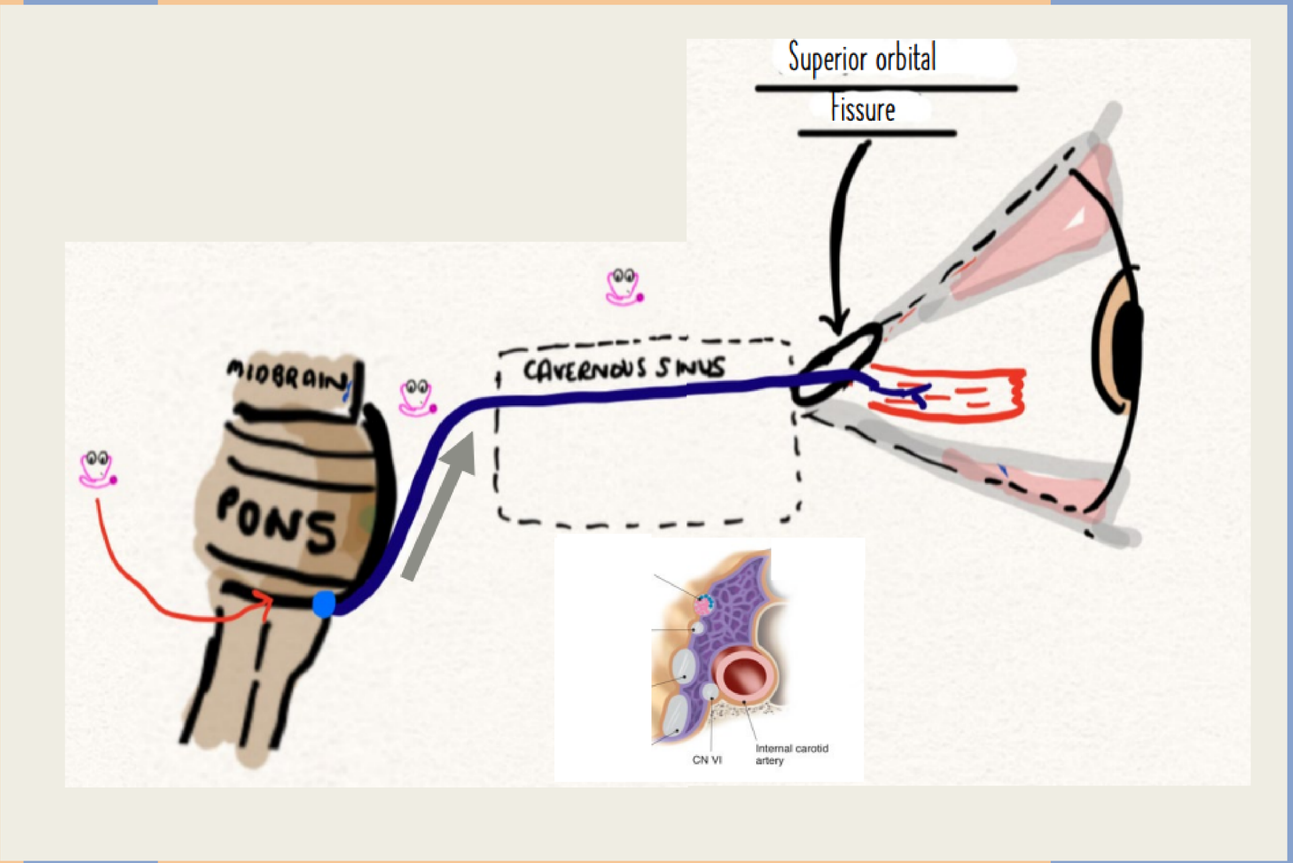


Early signs

- Decreased visual acuity
- Visual field defect
- Papilledema
- Strabismus & diplopia (abducens)



Late signs

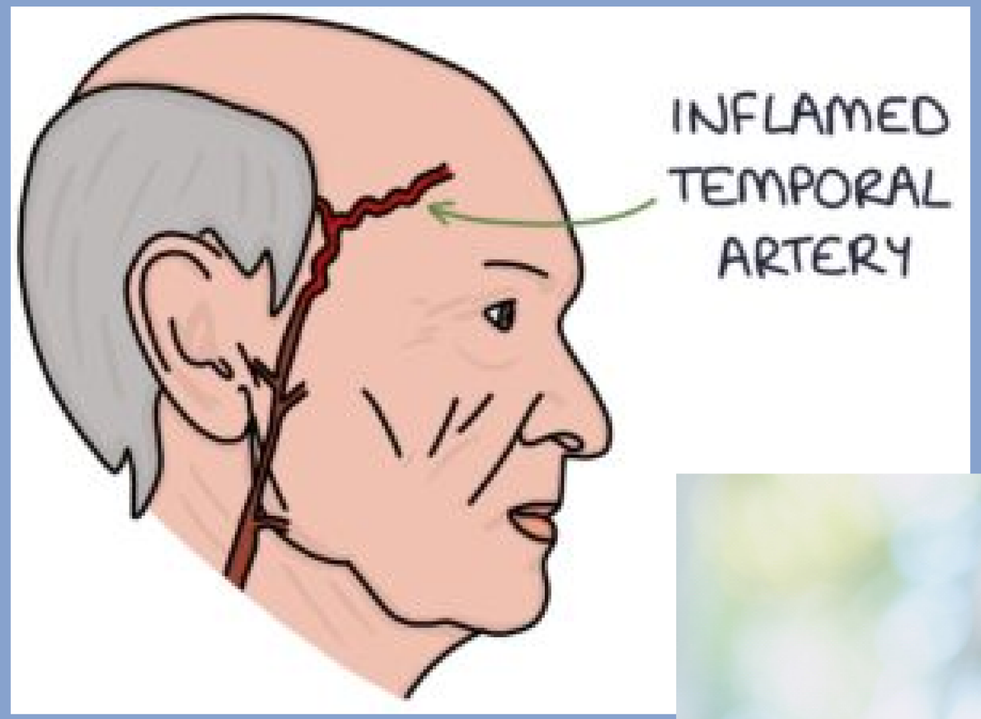


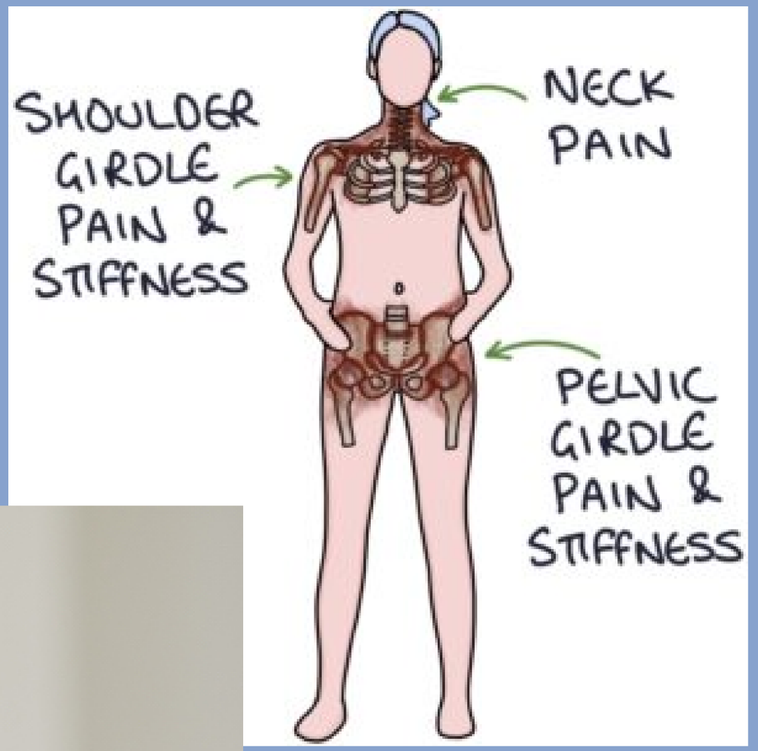
Early signs

- Decreased visual acuity
- Visual field defect
- Papilledema
- Strabismus & diplopia (abducens)
- Headache: +/- N&V
- Provoked by straining/coughing or position ie. worse on standing, lying or bending over/leaning forwards

Late signs

- Reduced GCS
- Focal neurology
- Seizures
- Pupils: constriction at first, later dilation
- Cushing's triad: falling pulse, rising BP, bradypnea (v late sign)


INFLAMED
TEMPORAL
ARTERY


SHOULDER
GIRDLE
PAIN &
STIFFNESS
NECK
PAIN
PELVIC
GIRDLE
PAIN &
STIFFNESS

G

I

A

N

T

Nodular, pulseless
THIS SIGN HAS V HIGH PPV!

Temporal artery abnormalities

G

I

A

N

T

New temporal headache (typically unilateral)

Temporal artery abnormalities

G

I

Increased inflammatory markers

A

Age (decades over) 50

N

New temporal headache (typically unilateral)

T

Temporal artery abnormalities

G

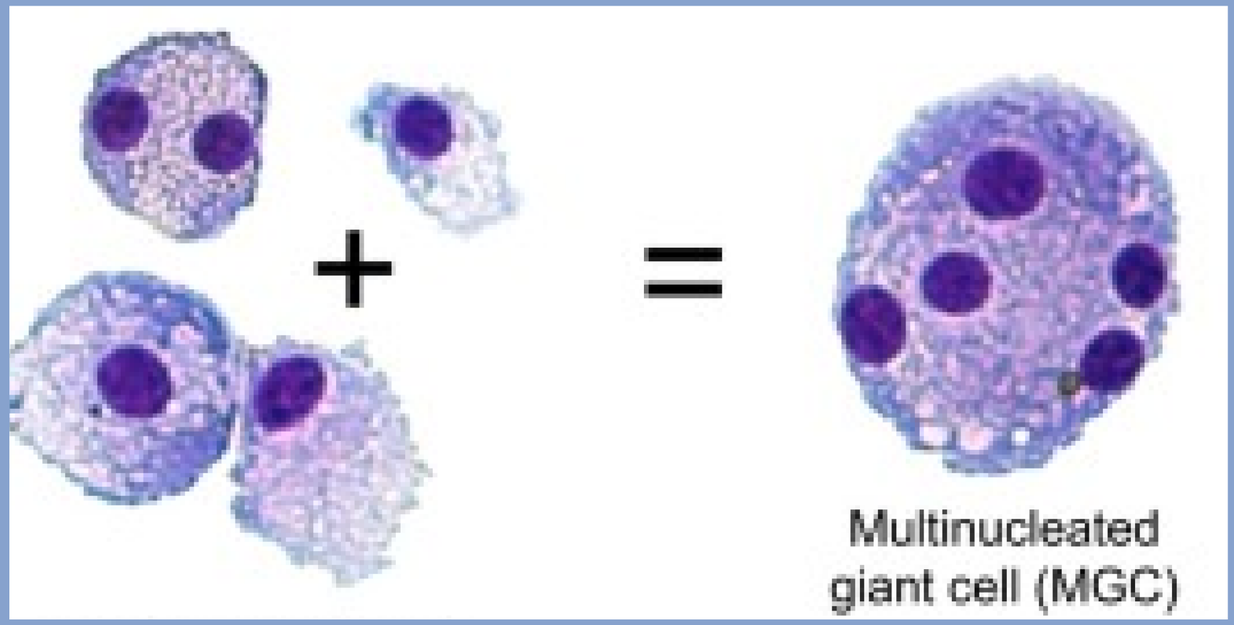
I

A

N

T

Granulomatous inflammation ft giant cells



G Granulomatous inflammation ft giant cells

I Increased inflammatory markers

A Age (decades over) 50

N New temporal headache (typically unilateral)

T Temporal artery abnormalities

Empirical pred

Do not delay treatment waiting for Ix results to diagnose!
HAVE HIGH CLINICAL SUSPICION & If suspected need to treat immediately to prevent loss of sight!

Empirical pred

Visual symptoms

Blurred or double vision (visual phenomena may occur weeks/months after onset of other symptoms)

Empirical pred

Visual symptoms

Yes - high dose 60mg

No - low dose 40mg

75mg aspirin OD decreases risk of visual loss & stroke

DON’T – Don’t stop taking steroids abruptly. Risk of adrenal crisis.

S Sick day rules

T Treatment card

O Osteoporosis prevention with bisphosphonates and supplemental calcium and vitamin D

P PPI for gastric protection

What is Status Epilepticus?

Seizure lasting > 5 minutes

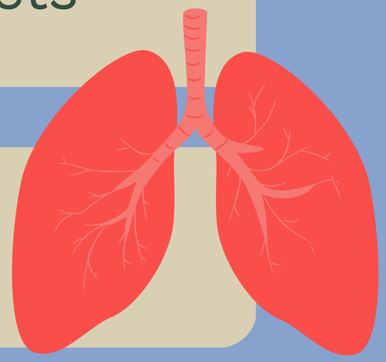
or

more than 3 seizures within l hour without complete recovery between

Management?

TIME

A Secure airway! Advanced or adjuncts



B 02, 15L/min

C Continuous BP monitoring: seizures & AEDs can cause hypotension, & obtain IV access, IV pabrinex if alcoholism or malnourished

D BM (50ml 50% glucose if <4)

E Exporsure, recovery position, 1st dose of full dose Benzo: 4mg Lorazepam. Neuro consult

Management?

Repeat your A-E!

After 5 mins
2nd Full dose benzo: REPEAT 4mg IV lorazepam

5-10 mins: LD Phenytoin, Levetiracetam (Kepra), Sodium valproate (caution in females of repro age)

ITU consult
> 30 mins: ITU involvement: IV thiopentone & mech ventilation

Assess precipitating cause...

Evidence of:

Meningism

Raised ICP/ focal neurology

Drug/alcohol abuse

Recent head injury

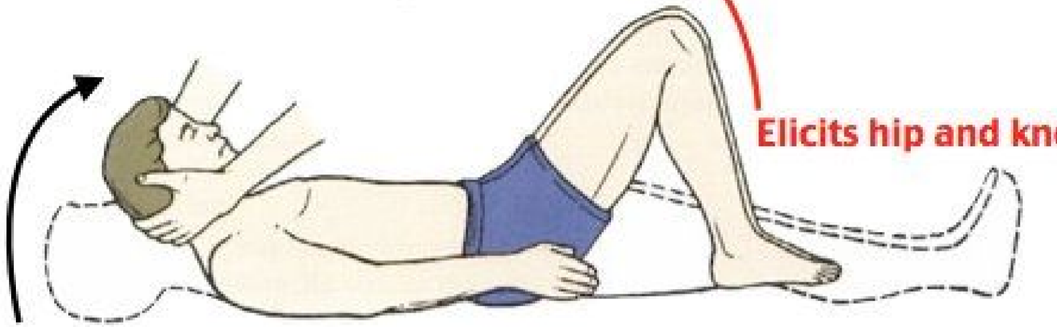
Stigmata of hepatic failure

Accurate recent drug history

A 24-year-old university student presents to the MAU with a 2-day Hx of feeling generally unwell. They have been in bed all day today with the blinds drawn. She complains of headache that came on gradually but has persisted and is now severe. Feeling nauseated but no vomiting as of yet. Not noticed any rash. As she's very uncomforable and not giving much Hx you proceed to examination...

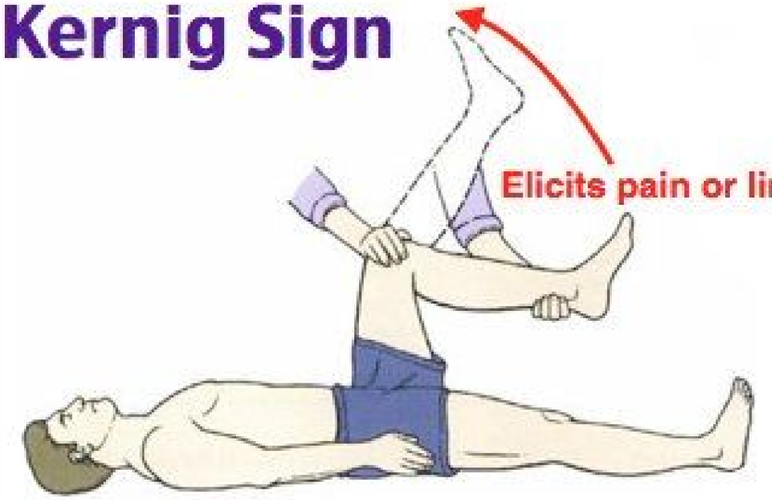
Alert, but appears unwell
RR 22, Sats >94%
HR 105
BP 115/85
Temp: 38.1
Unable to touch chin to chest
Brudinski sign +ve, Kernig sign -ve

Brudzinski Sign



1. Passive flexion of neck

Kernig Sign



1. Knee is flexed to 90 degrees
2. Hip is flexed to 90 degrees
3. Extension of the knee is painful or limited in extension

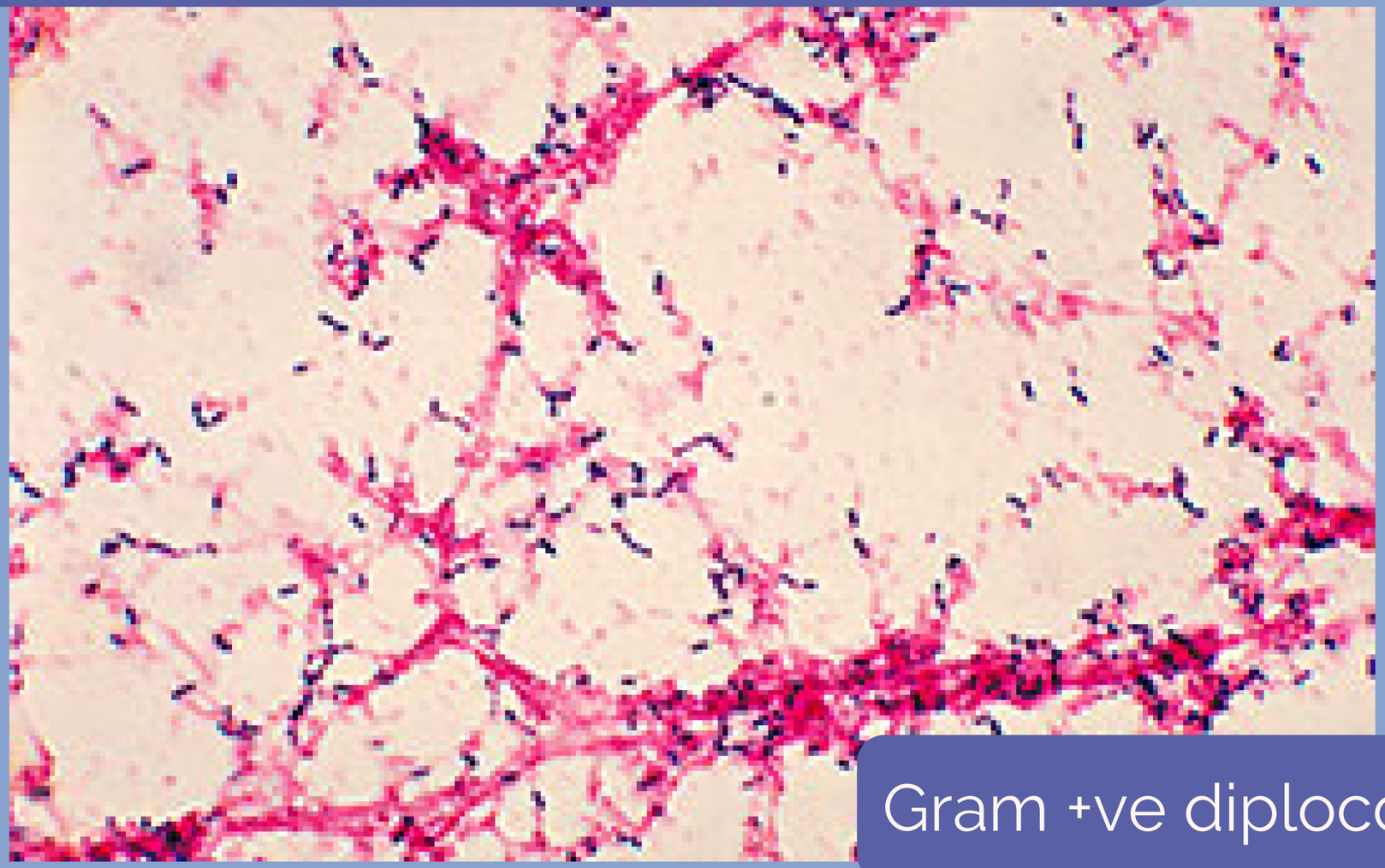
Bacterial

- Cloudy
- High WCC (neutrophils)
- Low glucose
- High protein

Viral

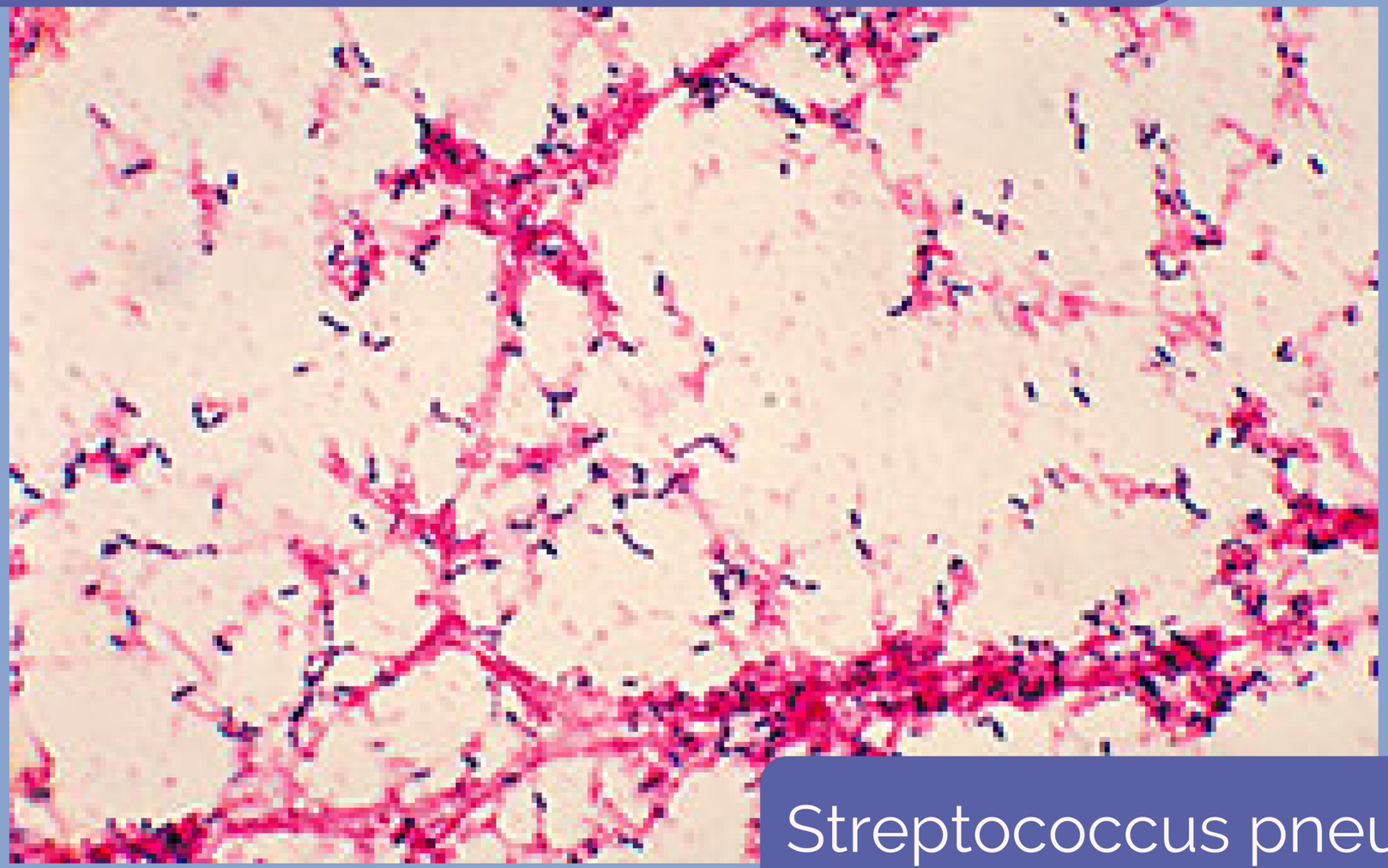
- Clear
- Mild rise in WCC (lymphocytes)
- Normal glucose
- Normal or mild rise in protein

Describe the gram stain. State the most common causative organism:



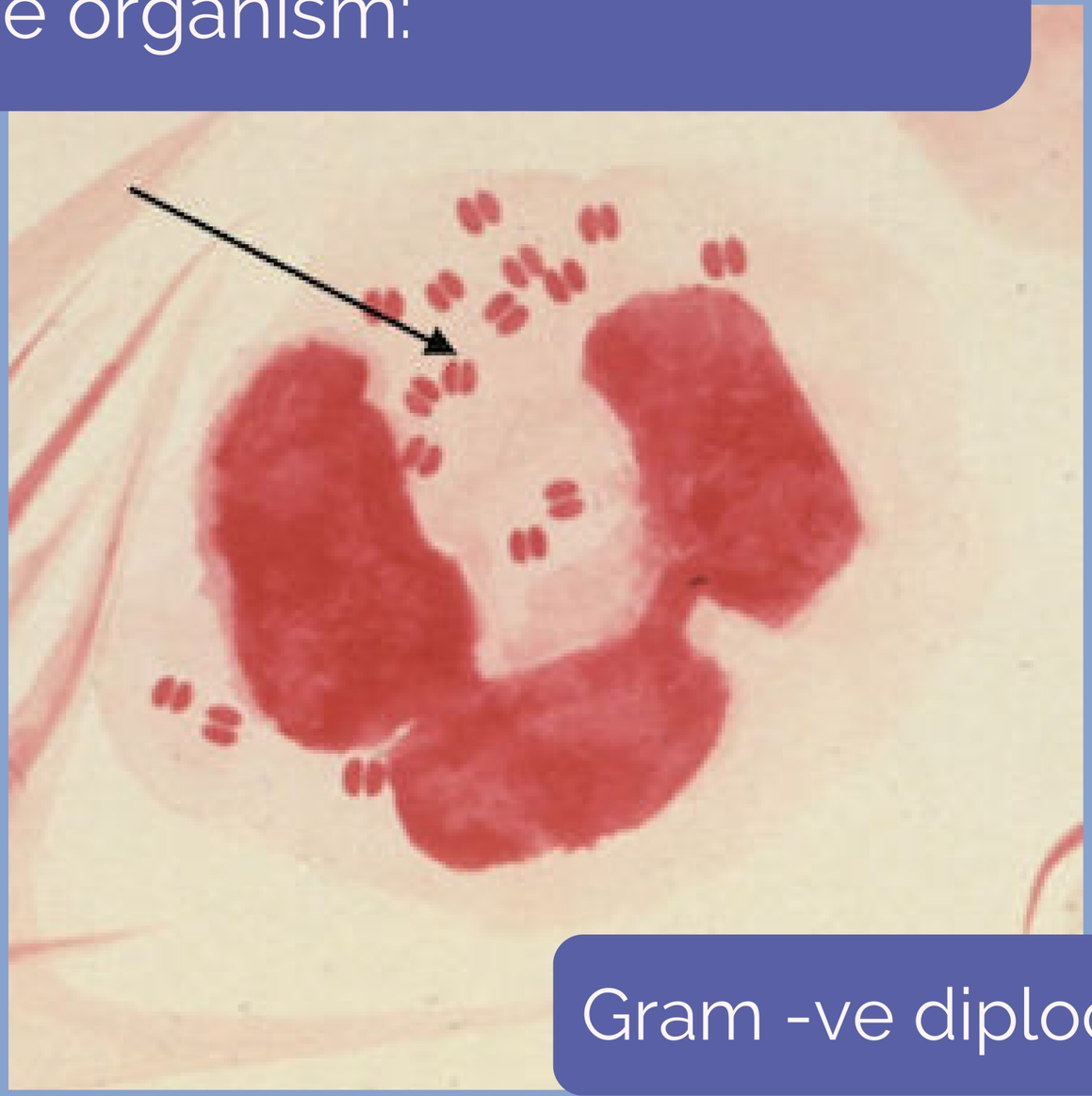
Gram +ve diplococci

Describe the gram stain. State the most common causative organism:



Streptococcus pneumoniae

Describe the gram stain. State the most common causative organism:



Gram -ve diplococci

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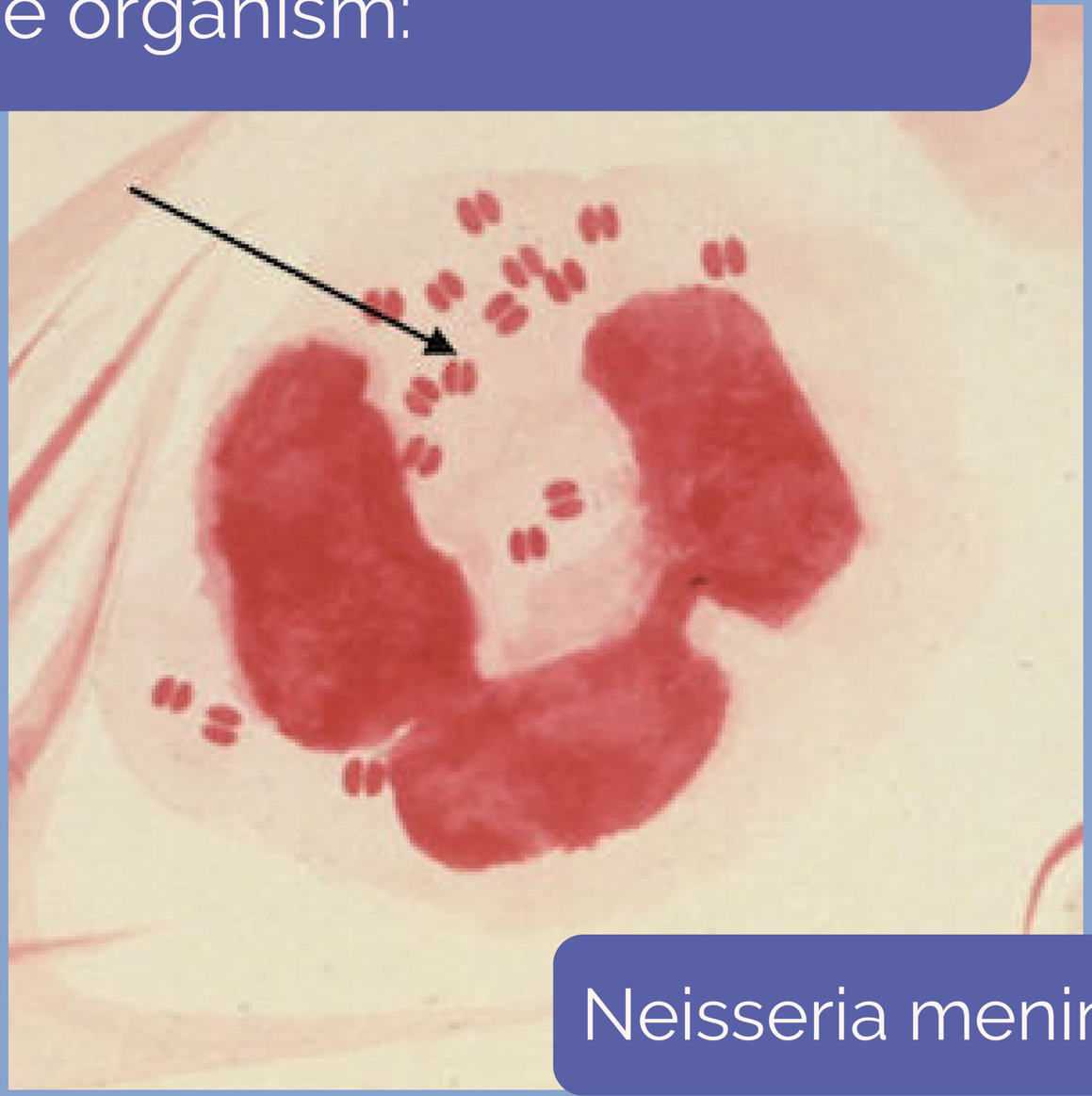


Neisseria meningitidis

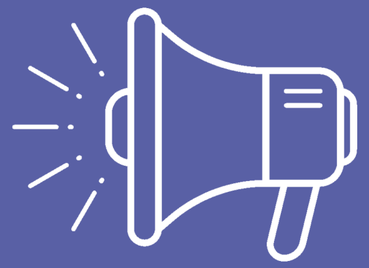
Empirical abx therapy: 1st line: IV Ceftriaxone

Empirical abx therapy: 1st line in penicillin allergy: IV Meropenem

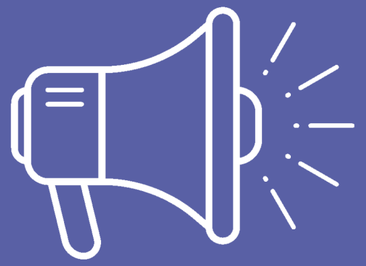
Additional pharm management if suspect pneumococcal meningitis

10mg IV dexamethasone
(first dose within 12hrs of abx)

Reduces intracranial inflammation: reduces post-meningitis complications of SNHL and neurological impairment



Notify PHE



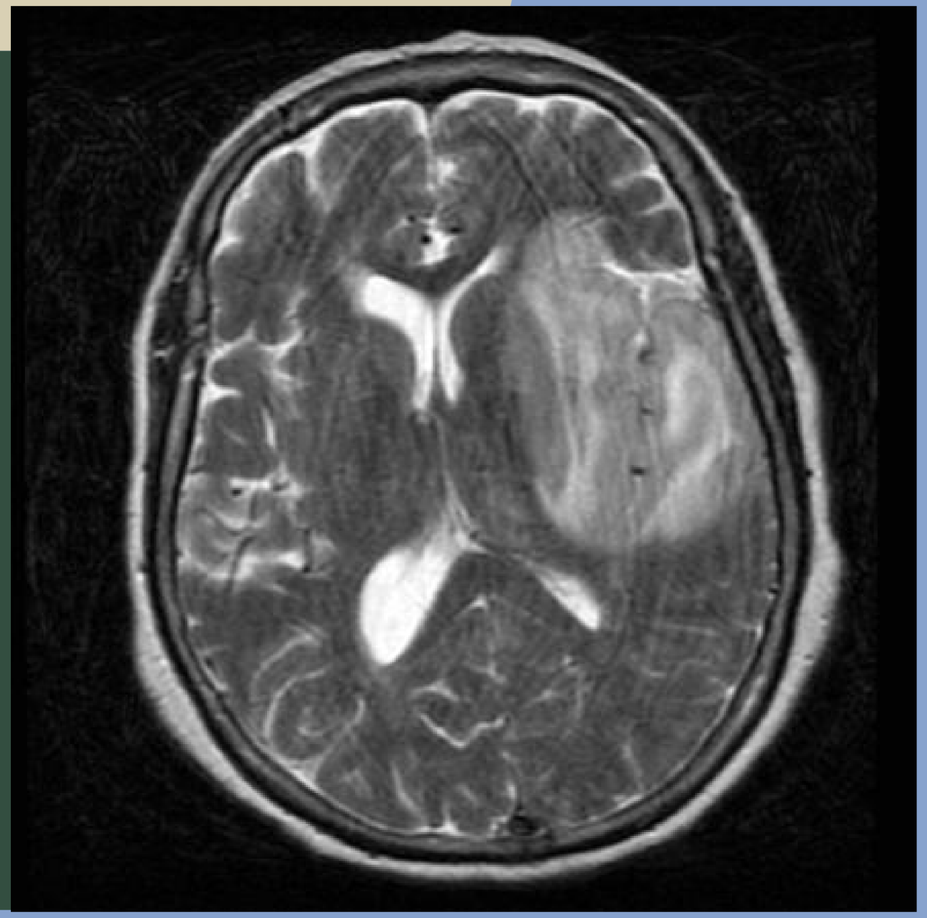
What additional symptoms/clinical features would make you concerned about encephalitis?

Confusion
Encephalopathic/psychiatric symptoms
Focal neurology e.g. aphasia, focal/partial seizures
↓ GCS

With reference to the lobes most commonly effected in encephalitis, why do you get focal seizures?

Temporal lobes most commonly effected

MR Appearance: Hyperdensity of the white matter and cortex in the medial temporal lobes and insular cortex



What's the most common cause of encephalitis?

HSV 1 and 2
Herpes simplex virus

HSV-1 in children & adults (cold sores)
HSV-2 in neonates (genital herpes, contracted during birth)


thANK
you