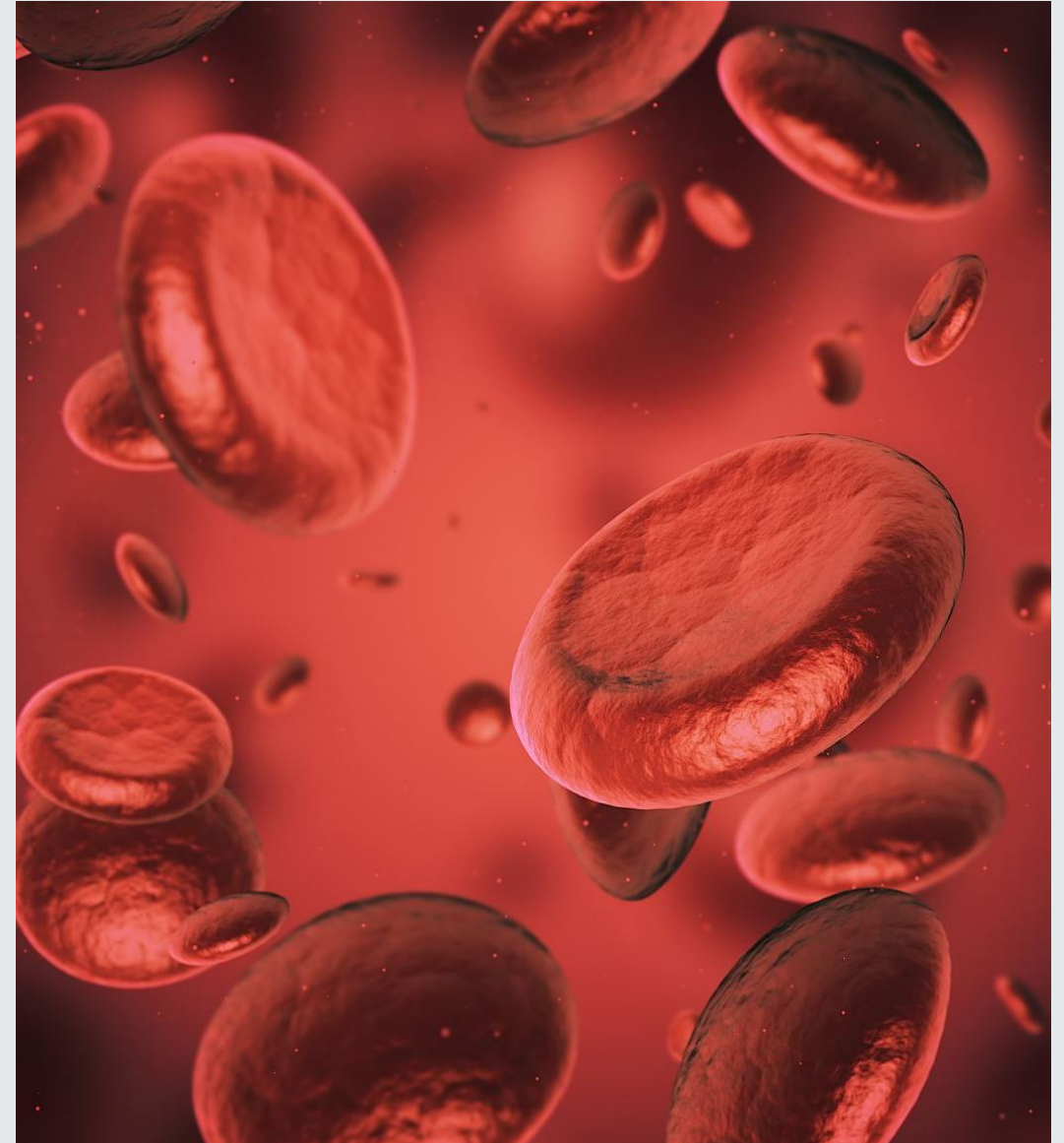

Non-traumatic intracranial bleeds

Dr Maureen Dumba
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National Hospital for Neurology and
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London, UK



OBJECTIVES

01

Distinguish types of intracranial haemorrhage

02

Formulate a differential diagnosis using a location-based approach

03

Review underlying pathology of bleeds

Intracranial Haemorrhage

Intra-axial -
within the brain
parenchyma

- **parenchymal haematoma – deep versus lobar**
- *(traumatic - cerebral contusions, diffuse axonal injury (DAI))*

Extra-axial -
outside the brain
parenchyma

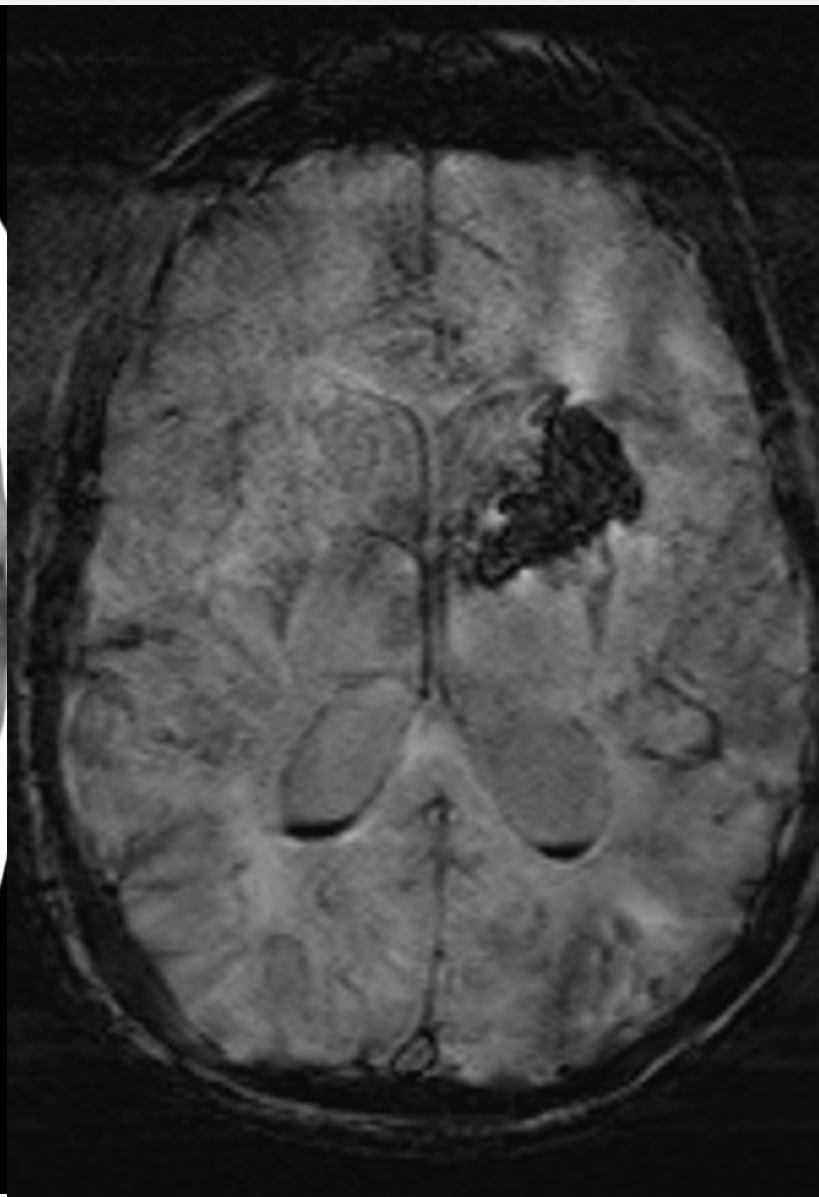
- **subarachnoid haemorrhage**
- *(traumatic - epidural (extradural) haematoma, subdural haematoma)*

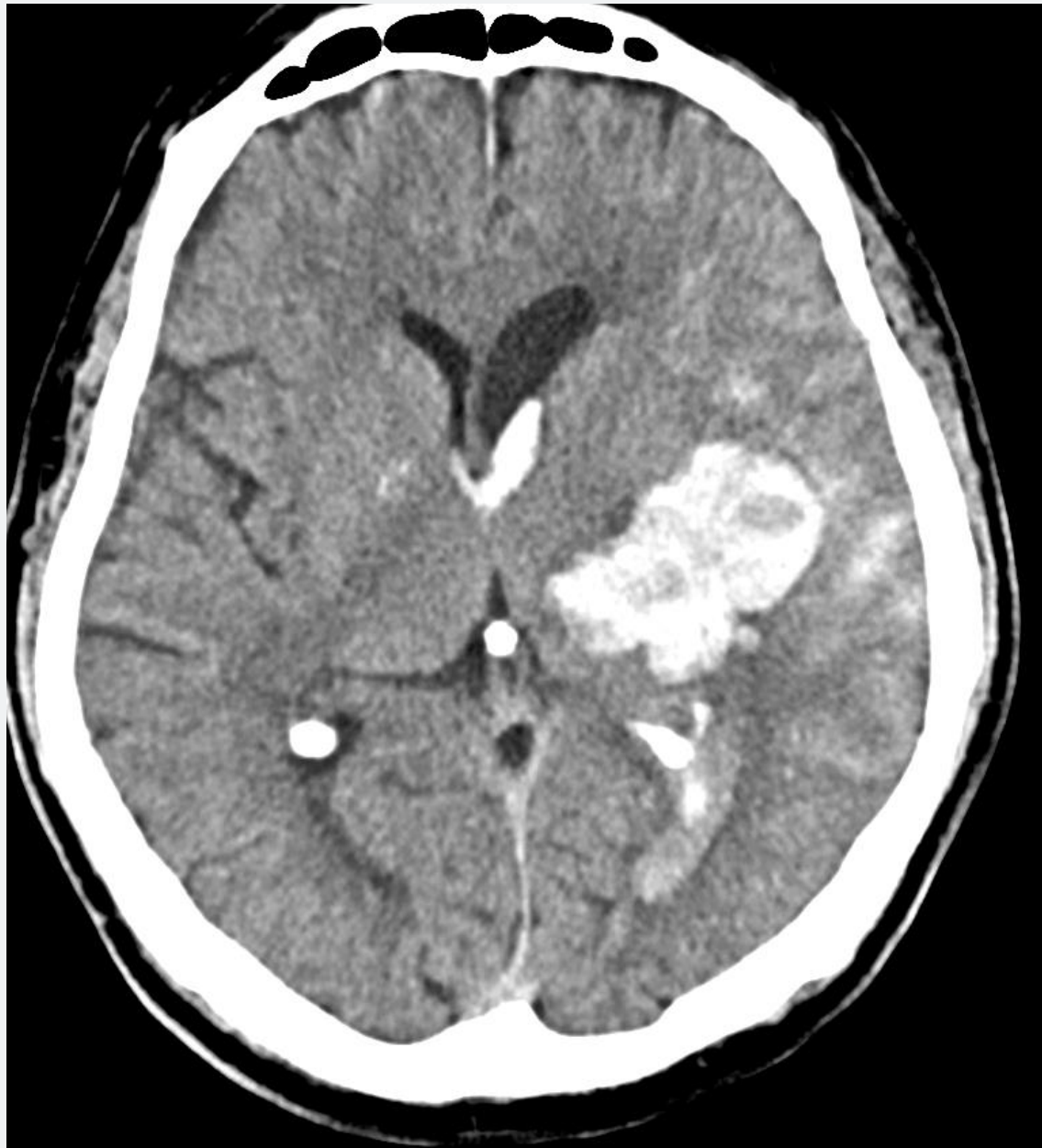
Density of blood
on CT: black ≠
chronic

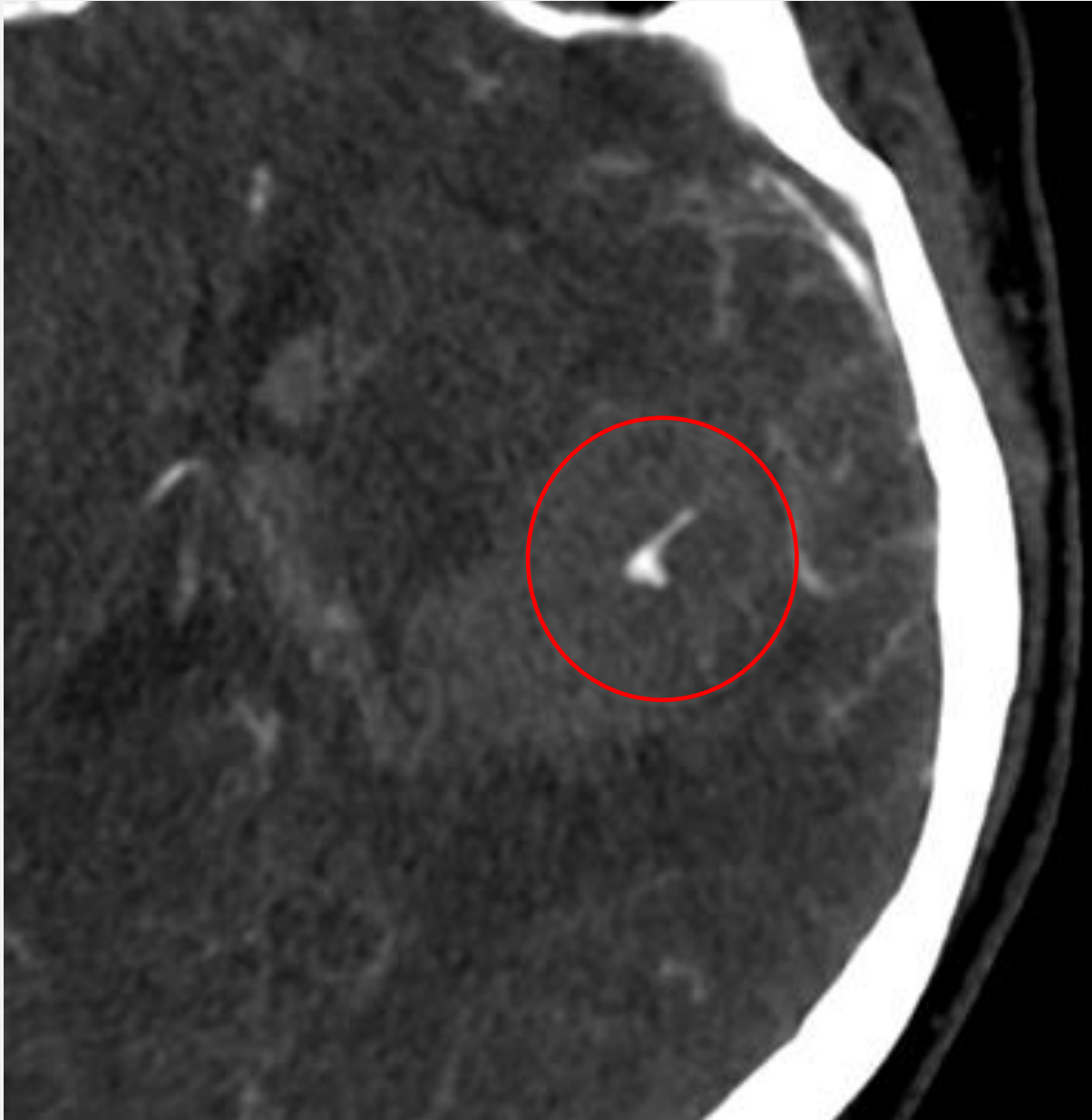
- black (hyperacute) → white (acute) → grey (subacute) → black (chronic)
- Consider anaemic patients or associated arachnoid tears that can “dilute” appearance of acute bleed, and **ACTIVE** bleeding

Parenchymal haematoma

Deep – hypertension

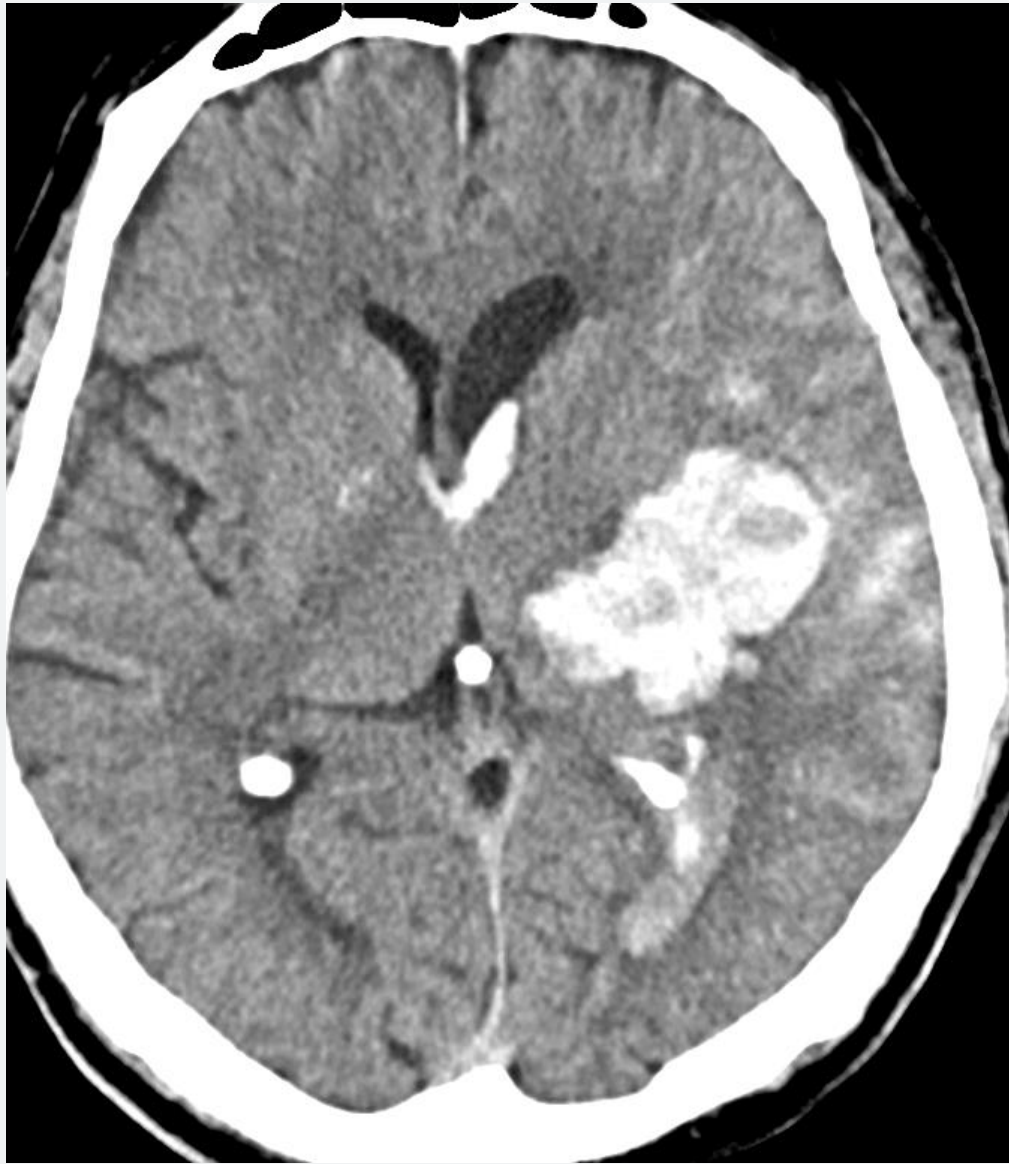




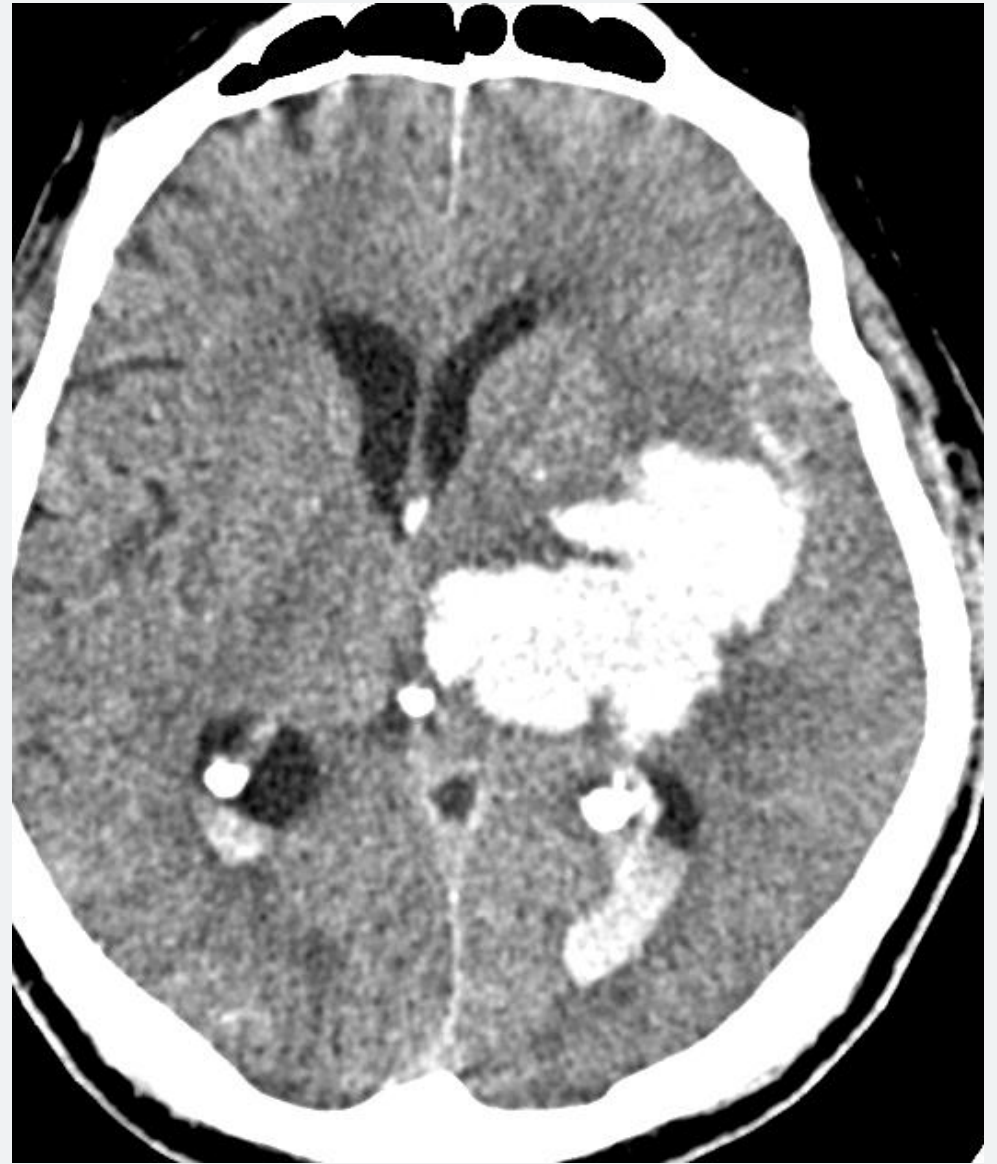


“Spot sign” - CT angiogram

- Focal pooling of contrast
- Not related to a vessel or hyperdense focus on the unenhanced study
- risk of haematoma expansion!



12 hours
later

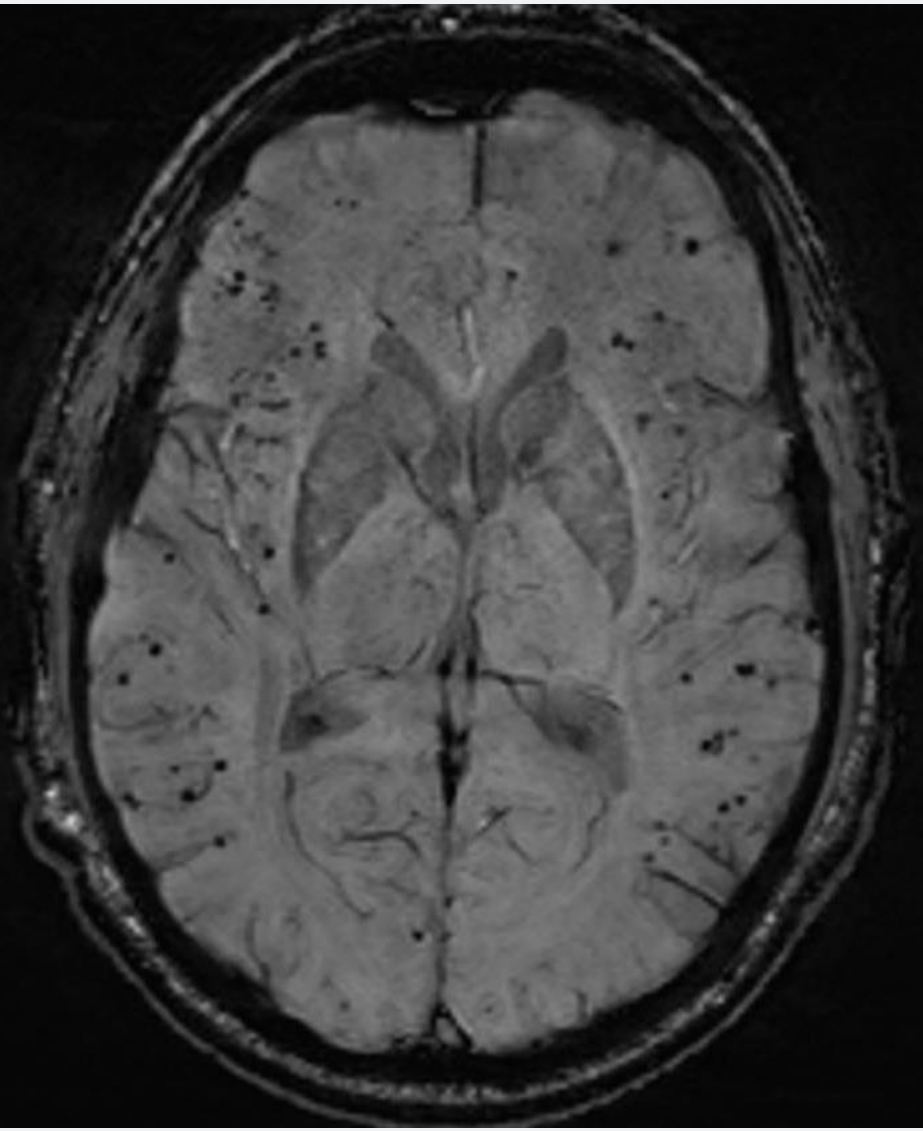
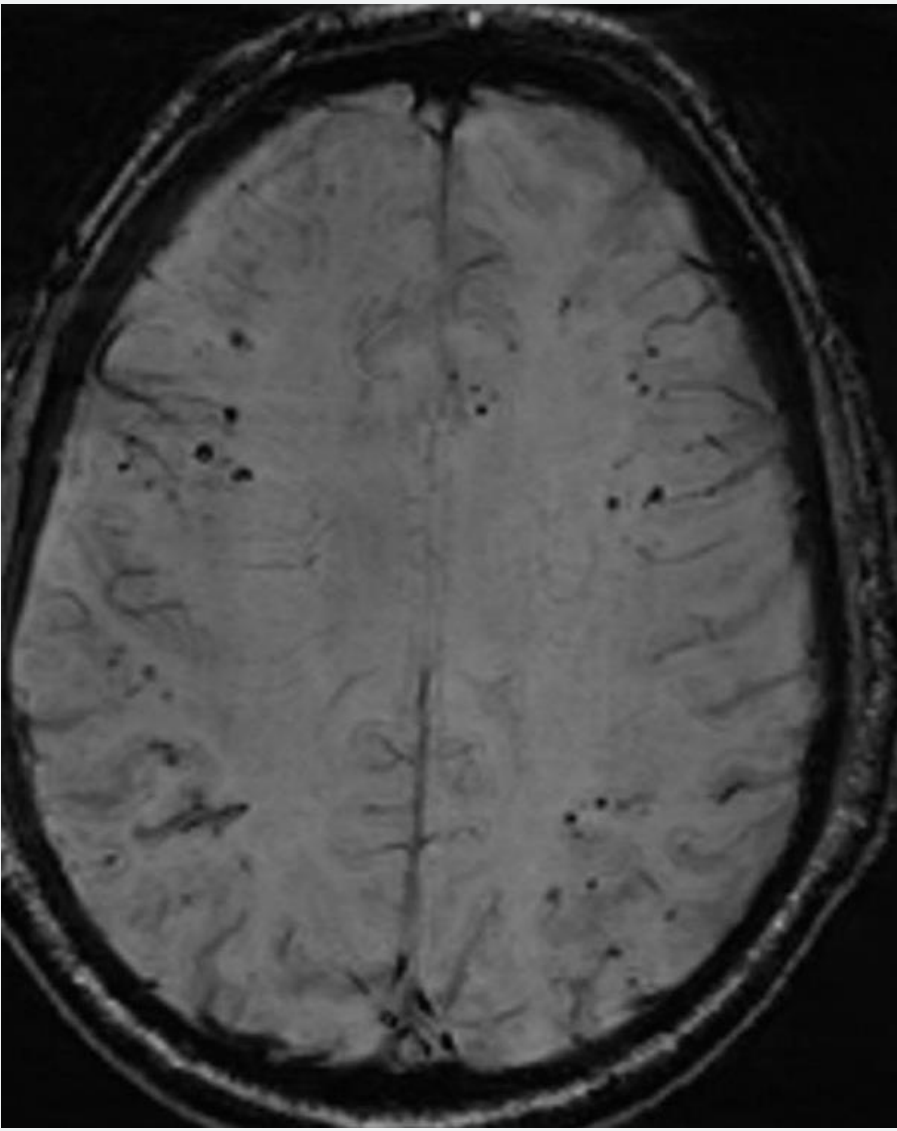
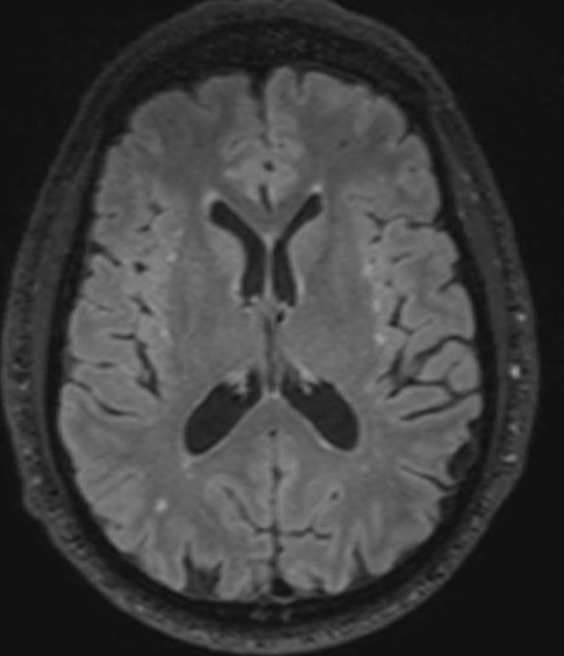


Hypertensive bleeds

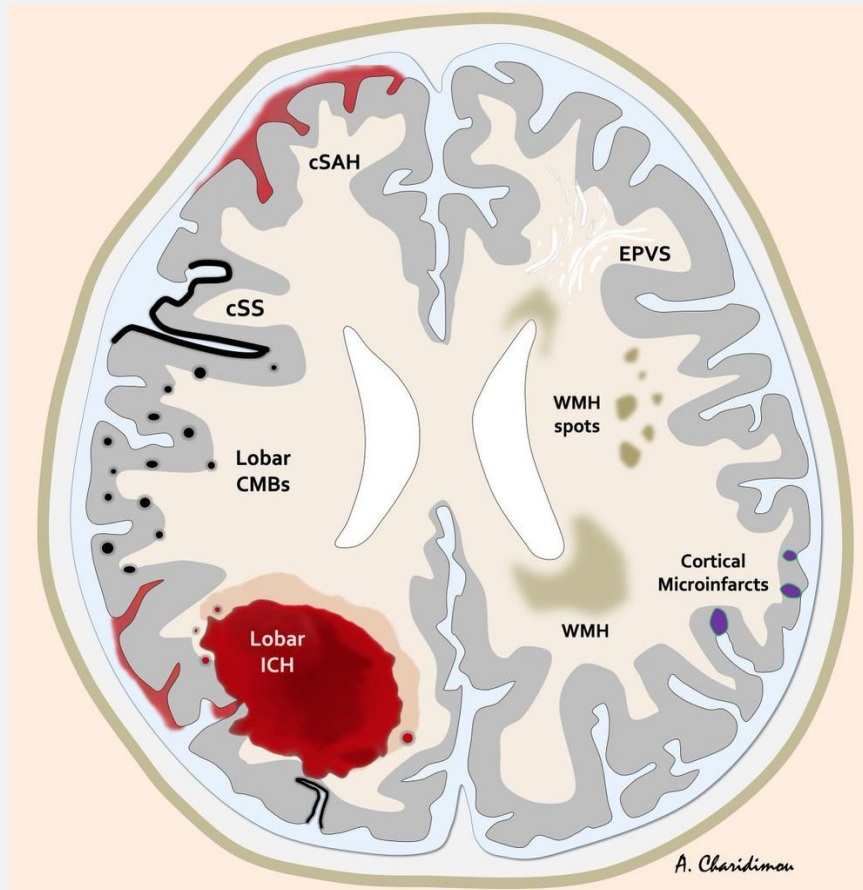
- Damage to small and large vessels
- Typically deep in location – basal ganglia, thalami, pons and cerebellum
 - but not always - atypical
- In younger patients, ask about illicit drugs (cocaine)
- Negative predictors – posterior fossa, intraventricular extension, significant mass effect.

Parenchymal haematoma

Lobar – CAA, venous,
(atypical hypertension)



Cerebral amyloid angiopathy





Sharma R, Cerebral amyloid angiopathy (illustration). Case study, Radiopaedia.org (Accessed on 20 Apr 2023) <https://doi.org/10.5334/7/rID-97818>

- Accumulation of beta-amyloid in walls of small/medium leptomeningeal and cortical vessels
- Lobar – macro- and micro-bleeds
 - Finger-like projections
- Convexity SAH, superficial siderosis
 - SS typically cortical but can be cerebellar
- Periventricular and deep white matter T2/FLAIR hyperintensities
 - Posterior predilection
- Enlarged perivascular spaces in deep white matter

Modified Boston Criteria 2.0

ARTICLES | VOLUME 21, ISSUE 8, P714-725, AUGUST 2022

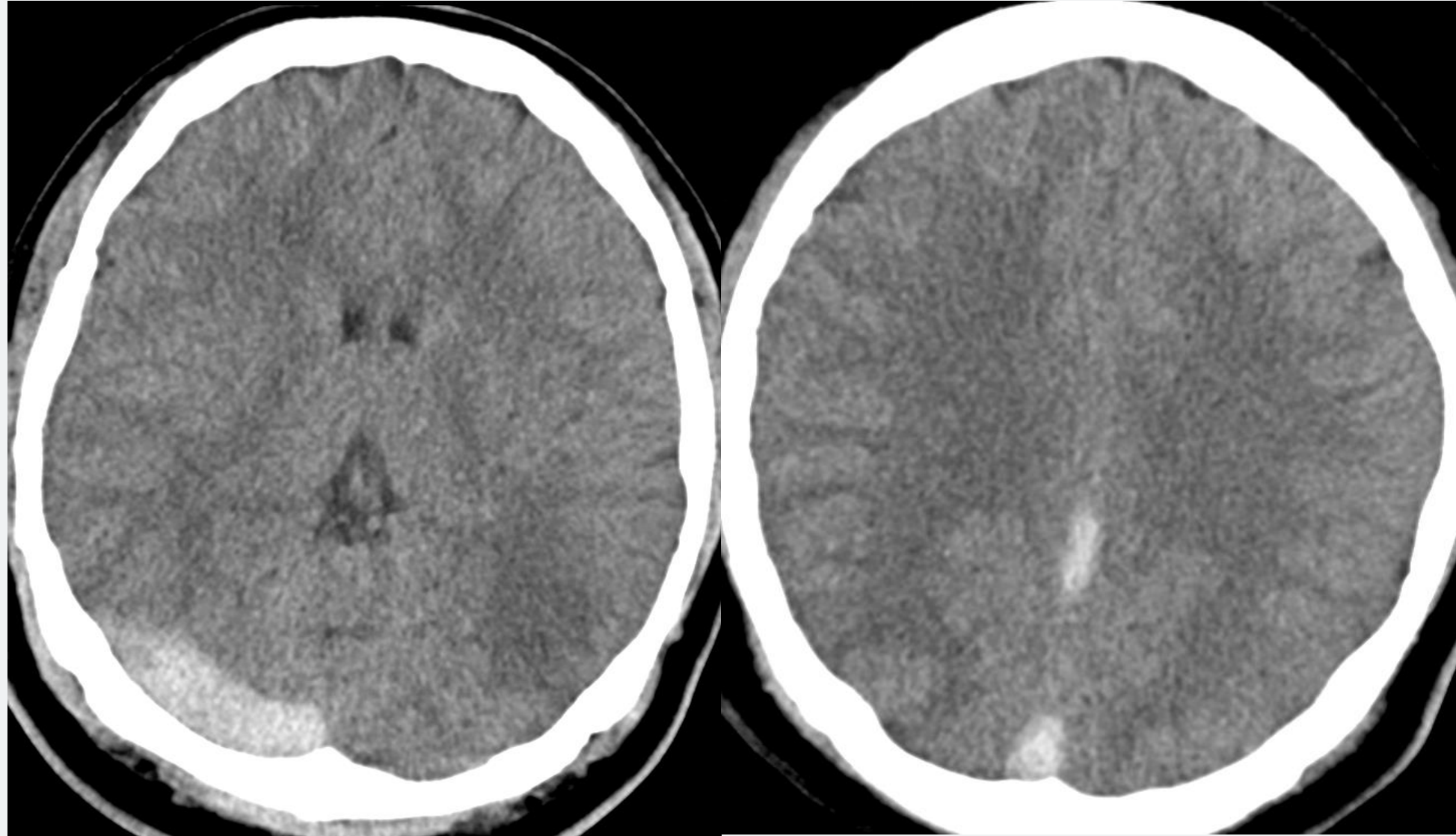
The Boston criteria version 2.0 for cerebral amyloid angiopathy: a multicentre, retrospective, MRI–neuropathology diagnostic accuracy study

Andreas Charidimou, MD   • Gregoire Boulouis, MD • Matthew P Frosch, MD • Prof Jean-Claude Baron, ScD • Marco Pasi, MD • Jean Francois Albucher, MD • et al. [Show all authors](#)

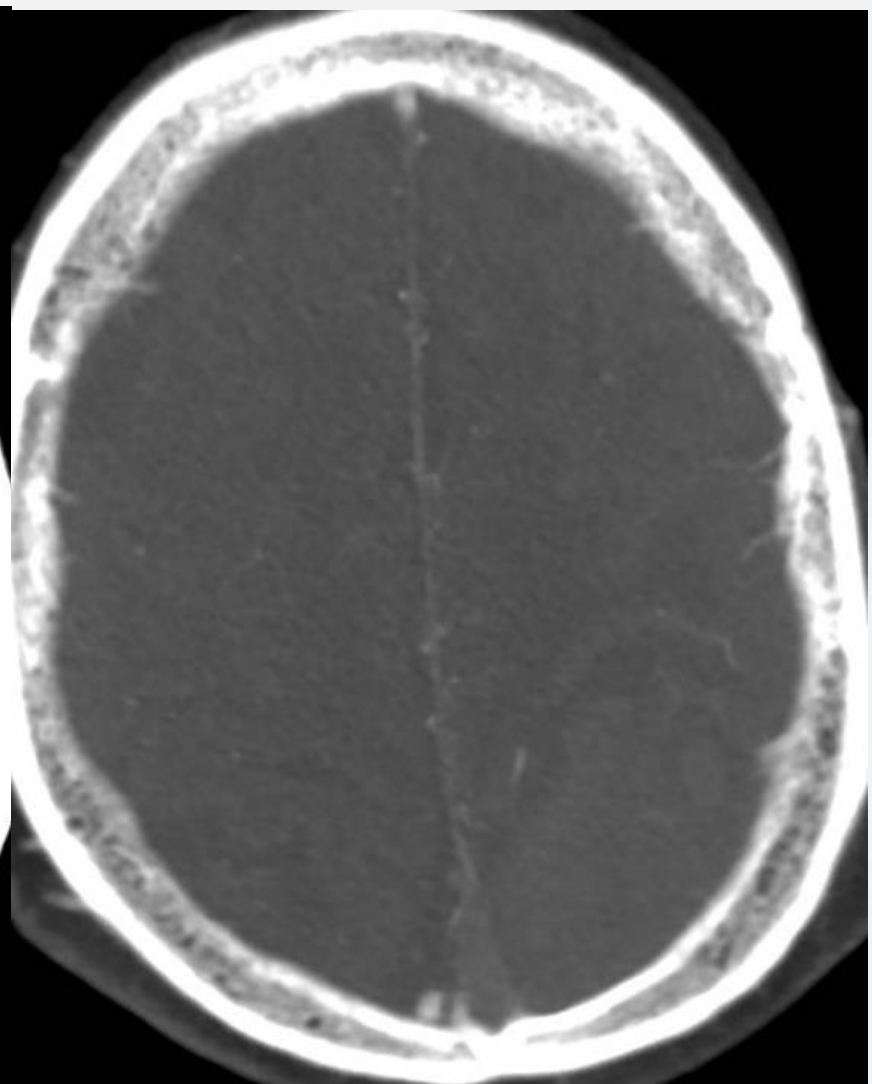
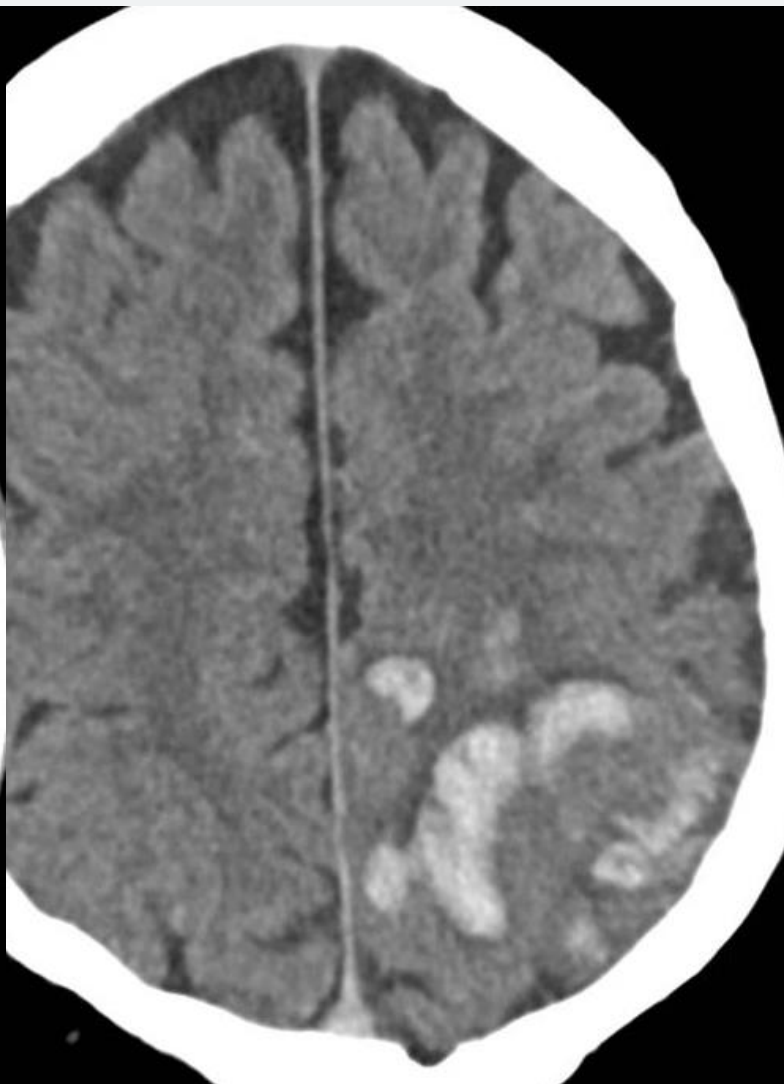
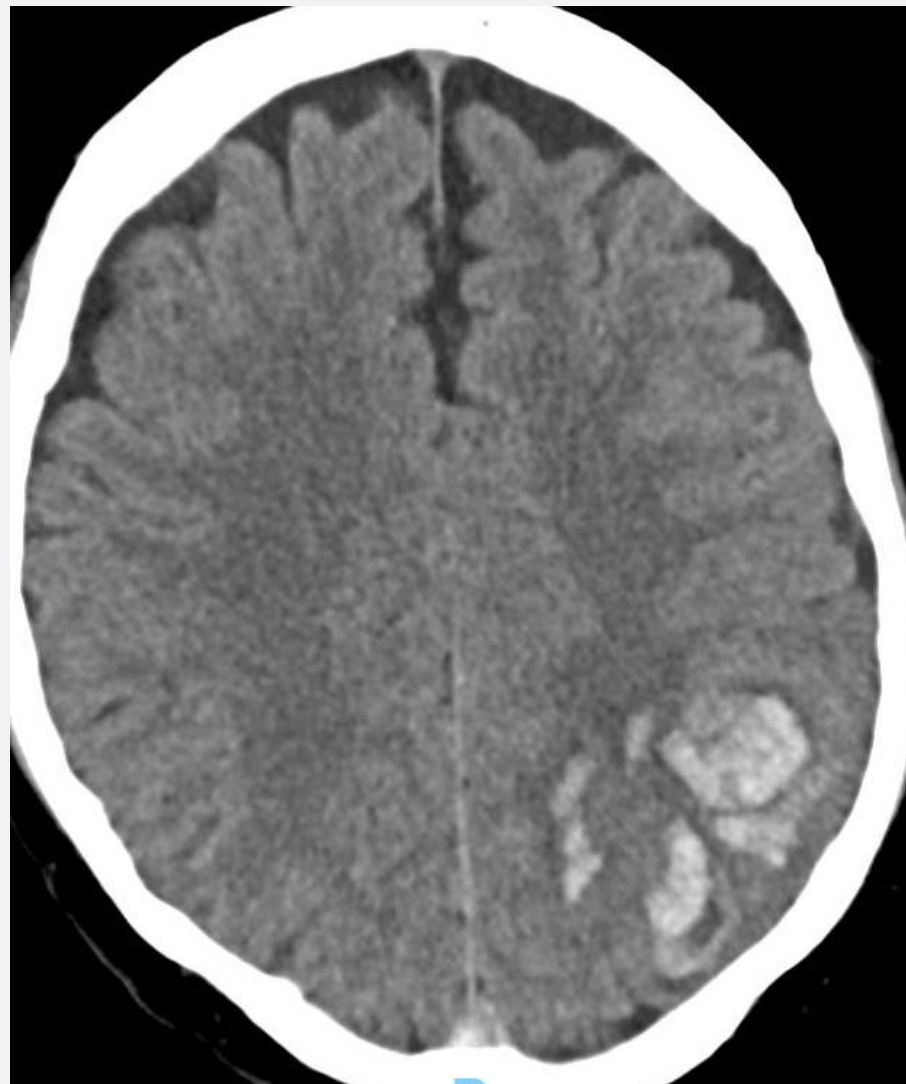
- **probable CAA**
 - pathological confirmation not required
 - for patients aged 50 years and older
 - presentation with spontaneous **intracerebral haemorrhage**, transient focal neurological episodes, or cognitive impairment or dementia
 - MRI criteria:
 - demonstrates either:
 - at least two of the following strictly lobar haemorrhagic lesions on T2*-weighted MRI, in any combination: **intracerebral haemorrhage**, **cerebral microbleeds**, or foci of cortical **superficial siderosis** (multiple distinct foci are counted as independent haemorrhagic lesions) or **convexity subarachnoid haemorrhage** (multiple distinct foci are counted as independent haemorrhagic lesions); **or**
 - one lobar haemorrhagic lesion plus one white matter feature (severe **perivascular spaces** in the centrum semiovale or **white matter hyperintensities** in a multispot pattern)
 - absence of:
 - any deep haemorrhagic lesions on T2*-weighted MRI; **and**
 - haemorrhagic lesion in cerebellum not counted as either lobar or deep haemorrhagic lesion
 - other cause of haemorrhagic lesions

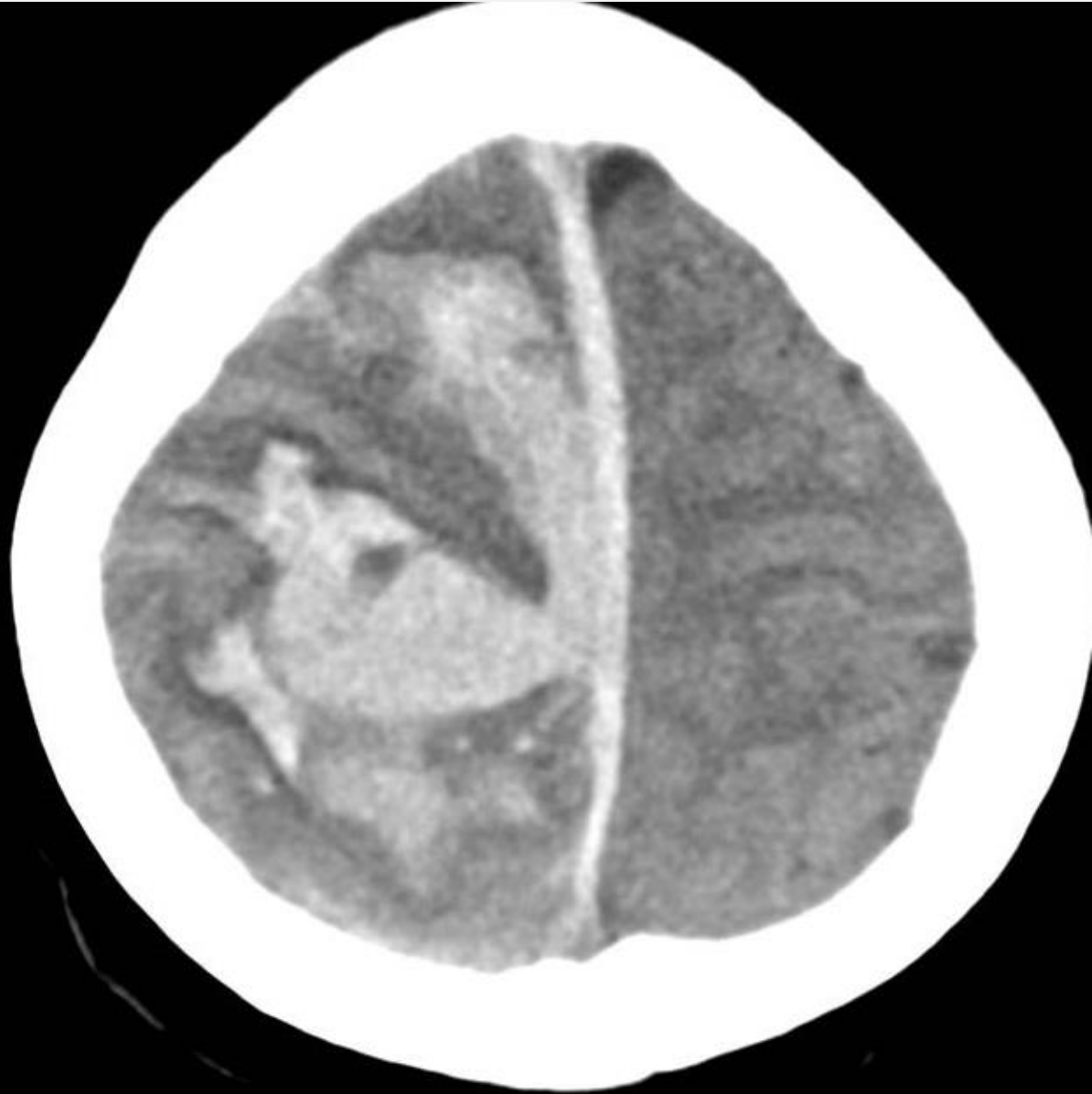
Sharma R, Kearns C, Rodrigues M, et al. Boston criteria 2.0 for cerebral amyloid angiopathy. Reference article, Radiopaedia.org (Accessed on 20 Apr 2023) <https://doi.org/10.53347/rID-149257>

**VENOUS
SINUS
THROMBOSIS**



VENOUS SINUS THROMBOSIS







Further investigations

Negative:

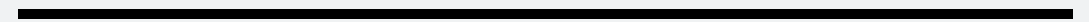
- CT angiogram/venogram
- Delayed MRI+c (12w)
- Delayed intracranial MRA (12w)

Both known poorly controlled hypertensives with multiple other risk factors - ?atypical hypertensive bleed



**Both
(deep/lobar)**

Tumours, vascular
malformations

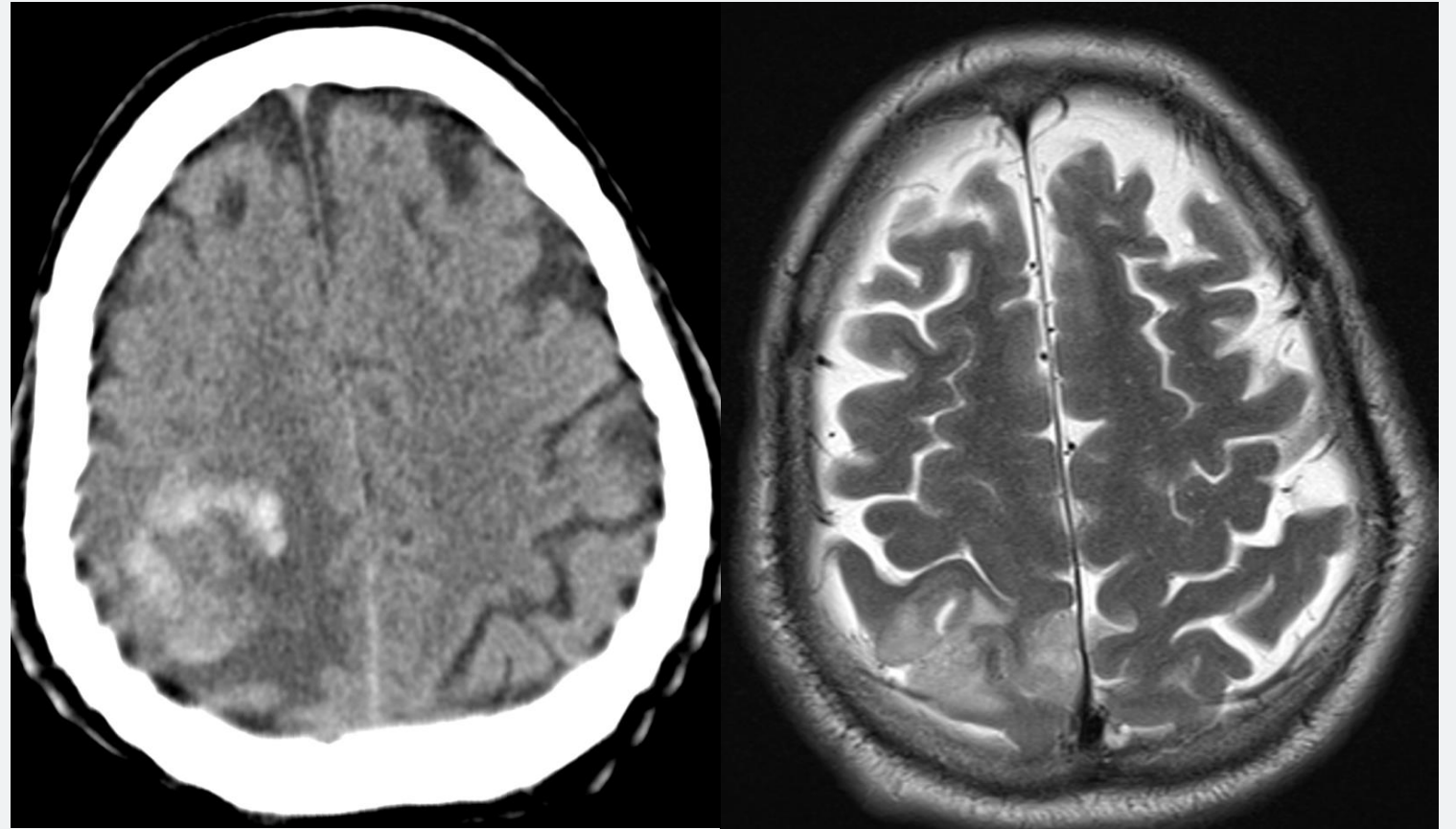


Tumours

- Tumours
 - Primary: glioblastoma, haemangioblastoma
 - Secondary: renal, thyroid (papillary), choriocarcinoma, melanoma
 - Breast, lung: uncommon but such high number of cases -> common

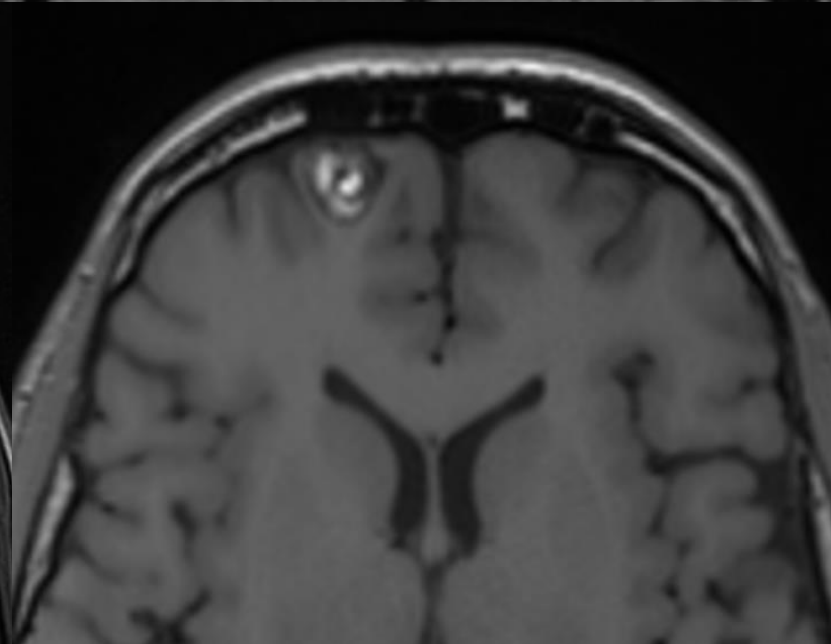
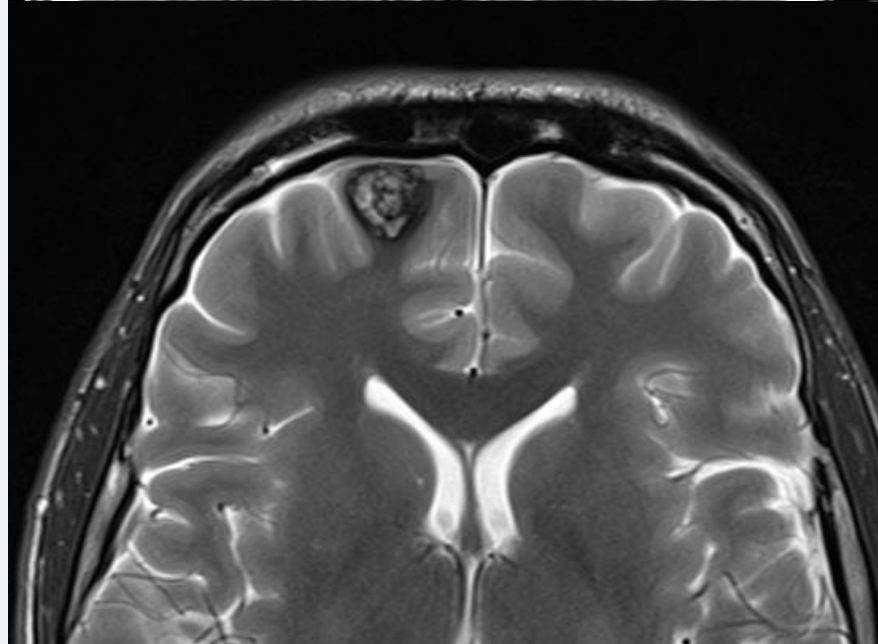
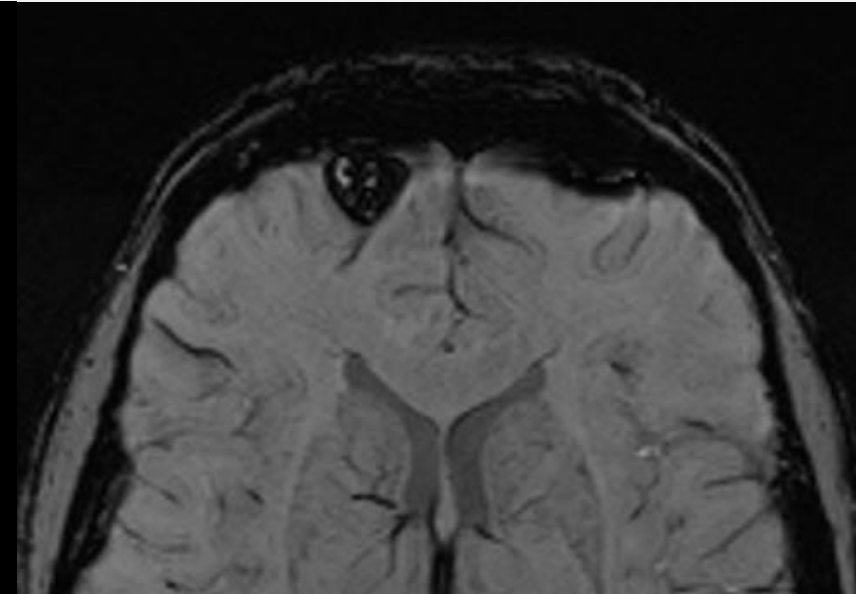
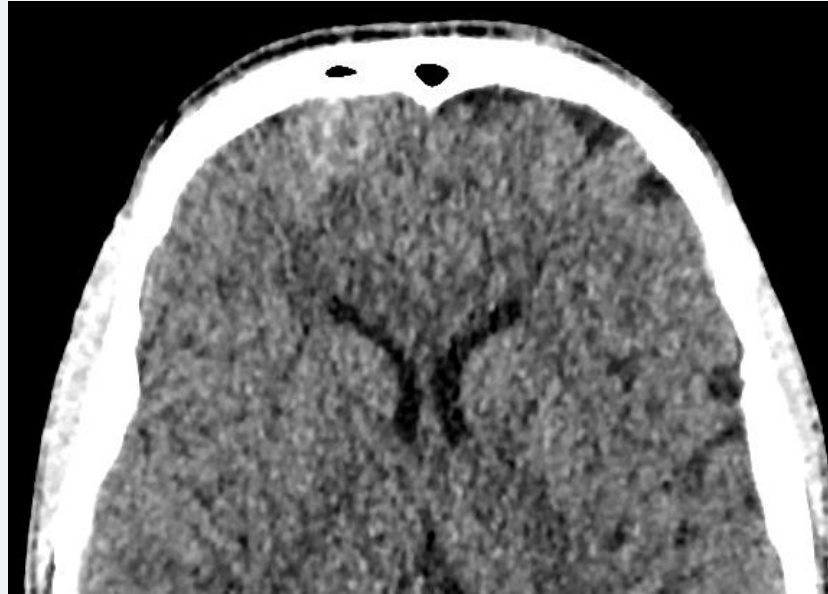
Tumours

Glioblastoma

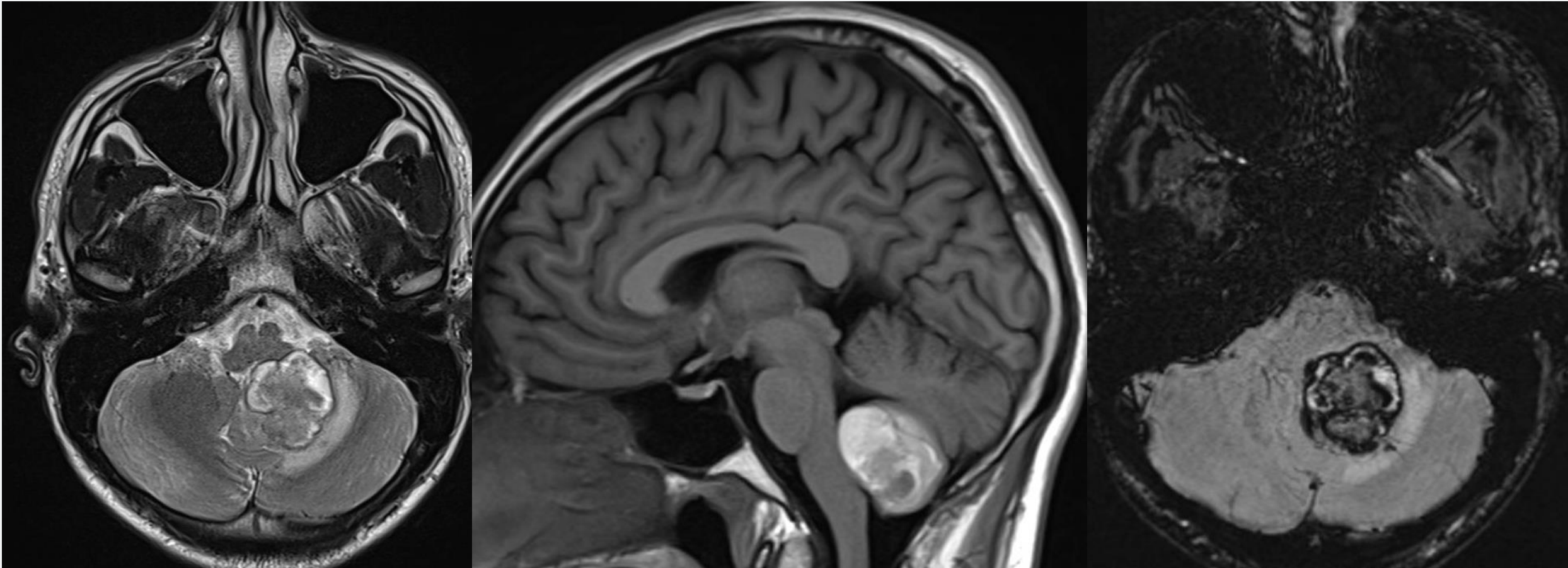


CAVERNOMA

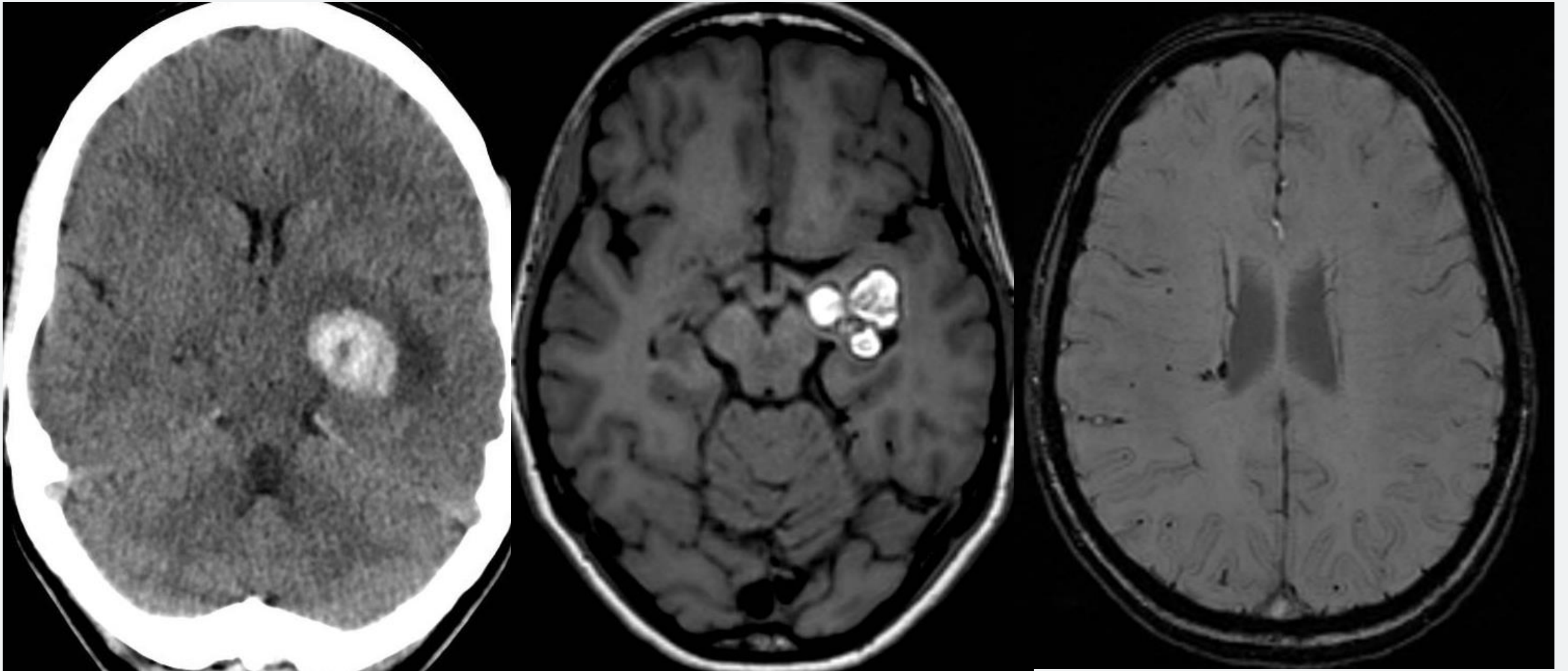
- Slow-flow venous malformation
 - no normal intervening brain
- Popcorn appearance on T2
- Intrinsic variable foci of T1-shortening
- Surrounding oedema: recent bleed - oedema

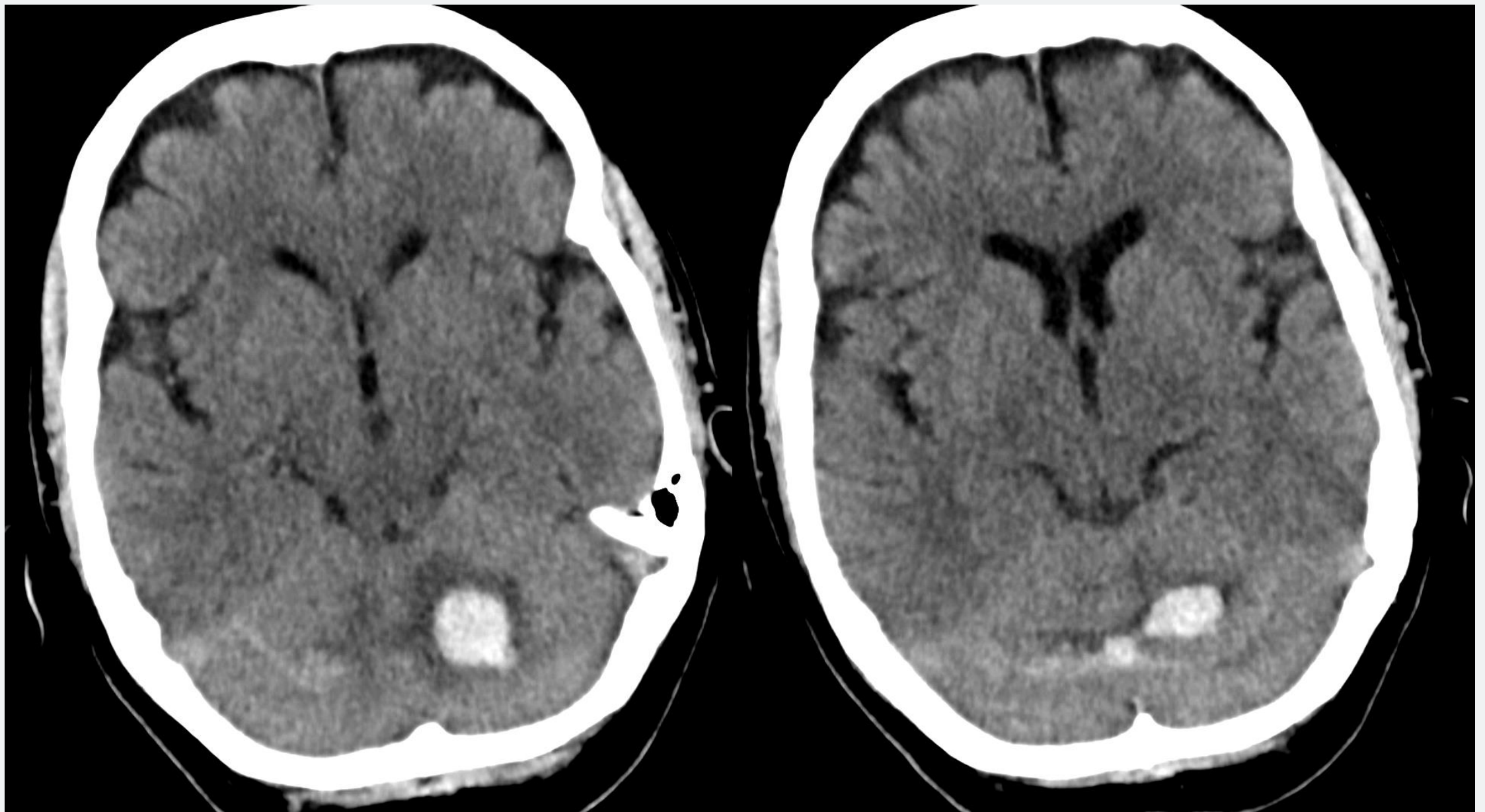


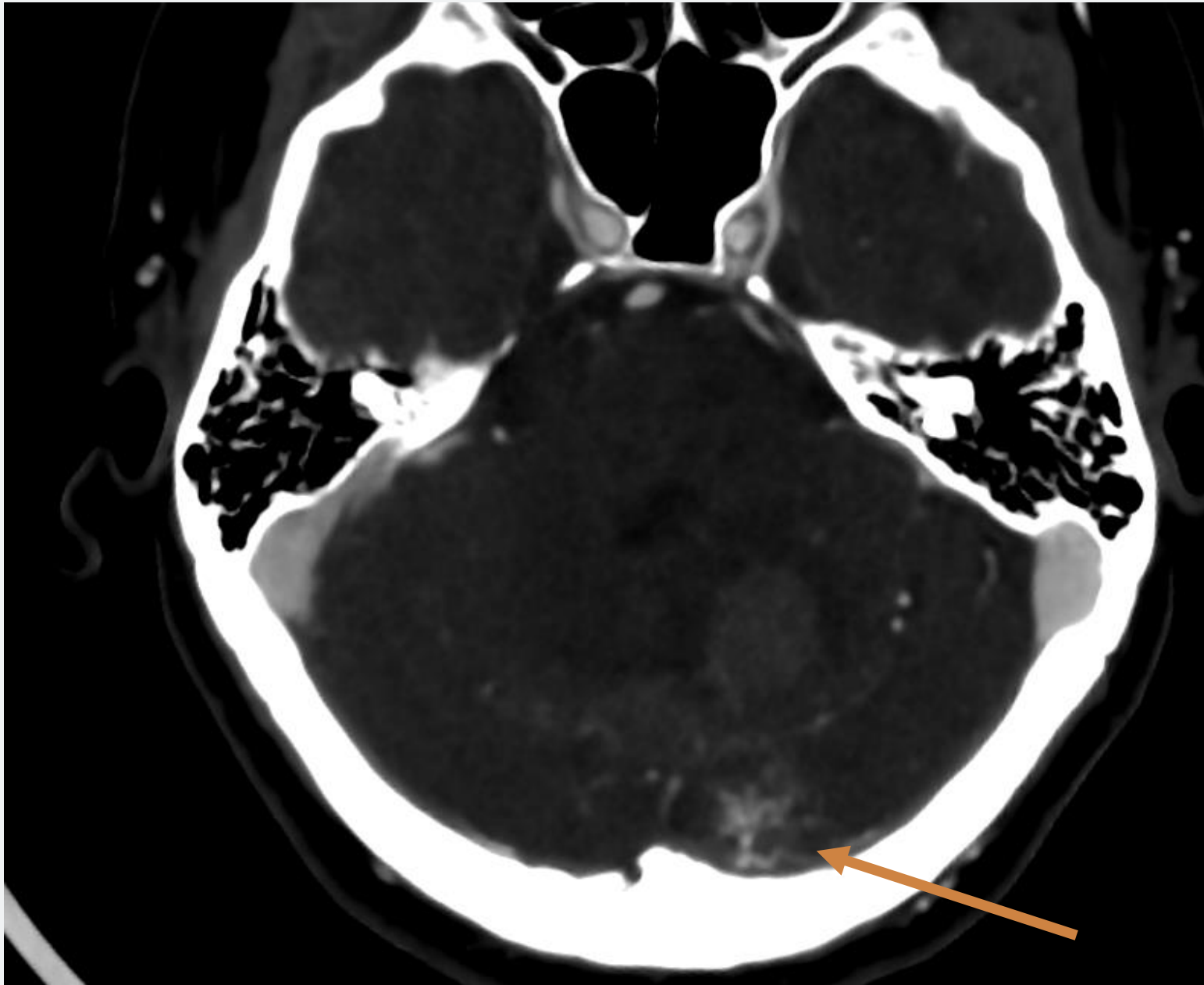
CAVERNOMA



CAVERNOMA

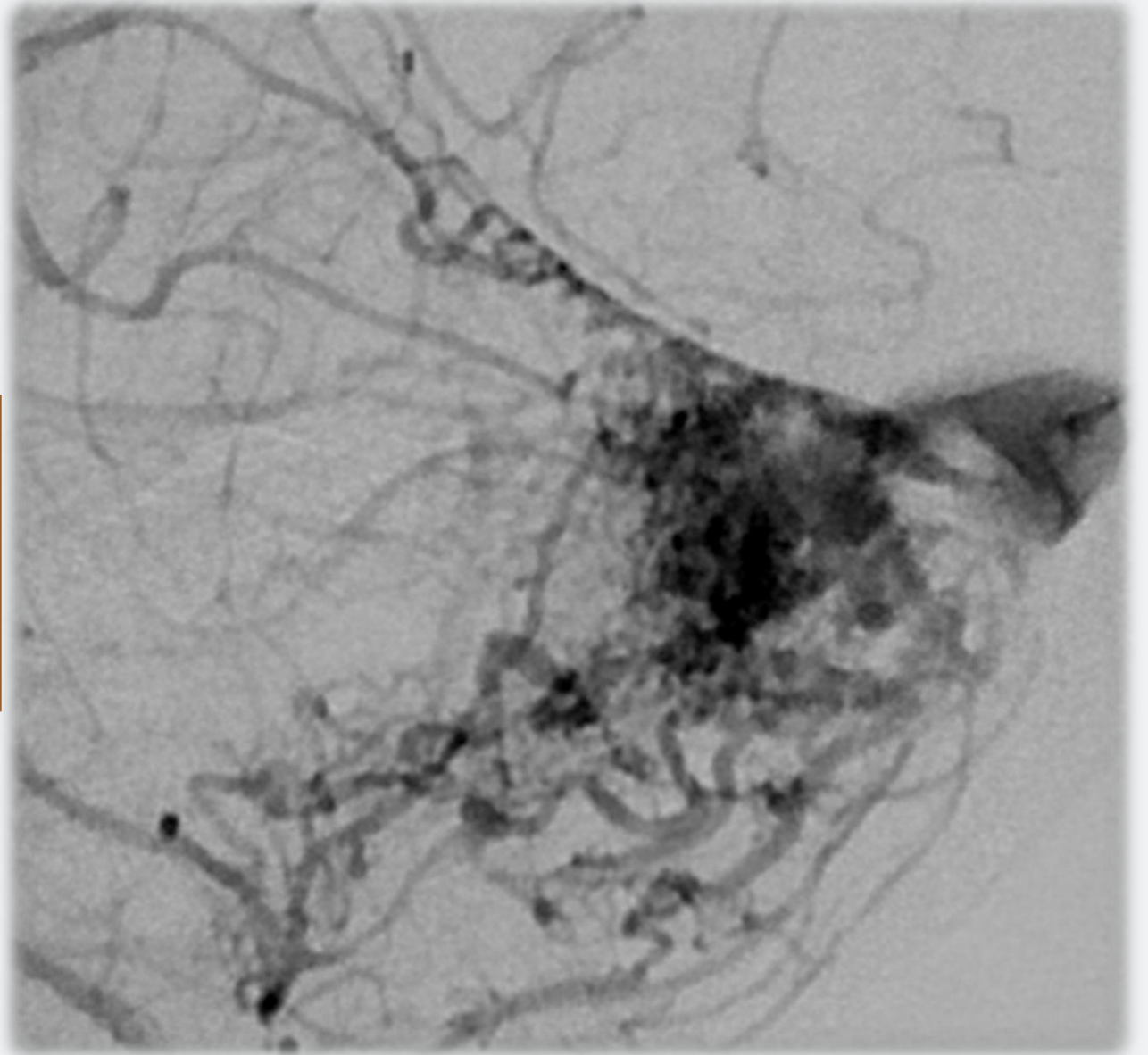
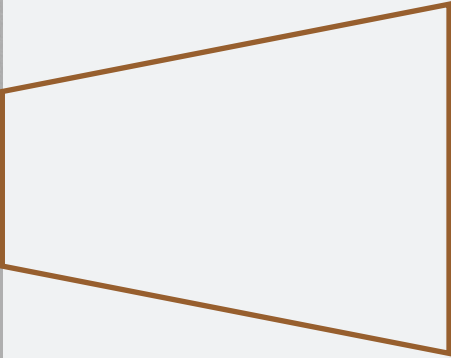






ARTERIOVENOUS MALFORMATION

Cerebellar AVM with multiple arterial PICA and SCA feeders and short pedicle superficial venous drainage.



Spetzler- Martin AVM grading scale

Size

- Small <3cm = 1
- Medium 3-6cm = 2
- Large >6cm = 3

Eloquence

- Noneloquent = 0
- Eloquent = 1

Venous drainage

- Superficial only = 0
- Deep component = 1

Subarachnoid haemorrhage

Aneurysms, RCVS, (CAA)

SAH – overlap with trauma

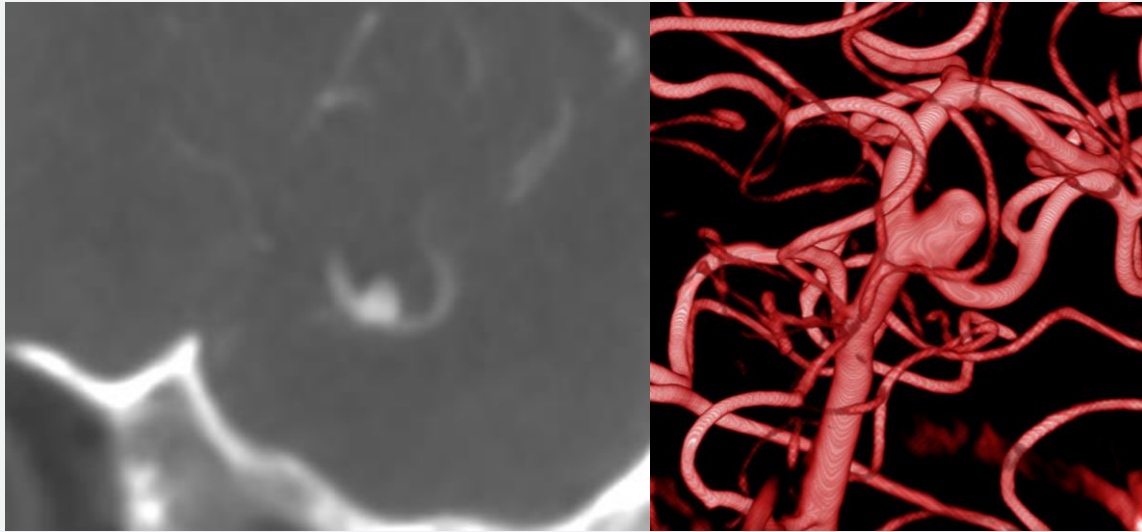
What came first...?

- fall, hit their head and bled
- bleed then fall and hit their head

It can be difficult to differentiate in the acute setting:

- significant volume of blood +/- location may imply an underlying ruptured aneurysm
- Smaller volumes in traumatic SAH – look for coup/contrecoup patterns
- Convexity SAH tends to be traumatic

Aneurysms



Anterior circulation (90%) far more common than posterior circulation (10%)

ACA/ACOM 1/3

- septum pellucidum, interhemispheric fissure & intra-ventricular

Terminal ICA/PCOM 1/3

- Sylvian fissure

MCA 1/3

- temporal lobe, Sylvian fissure & intra-ventricular

Basilar/SCA/PICA ~5%

- prepontine cistern
- foramen magnum

Consider family history and risk factors

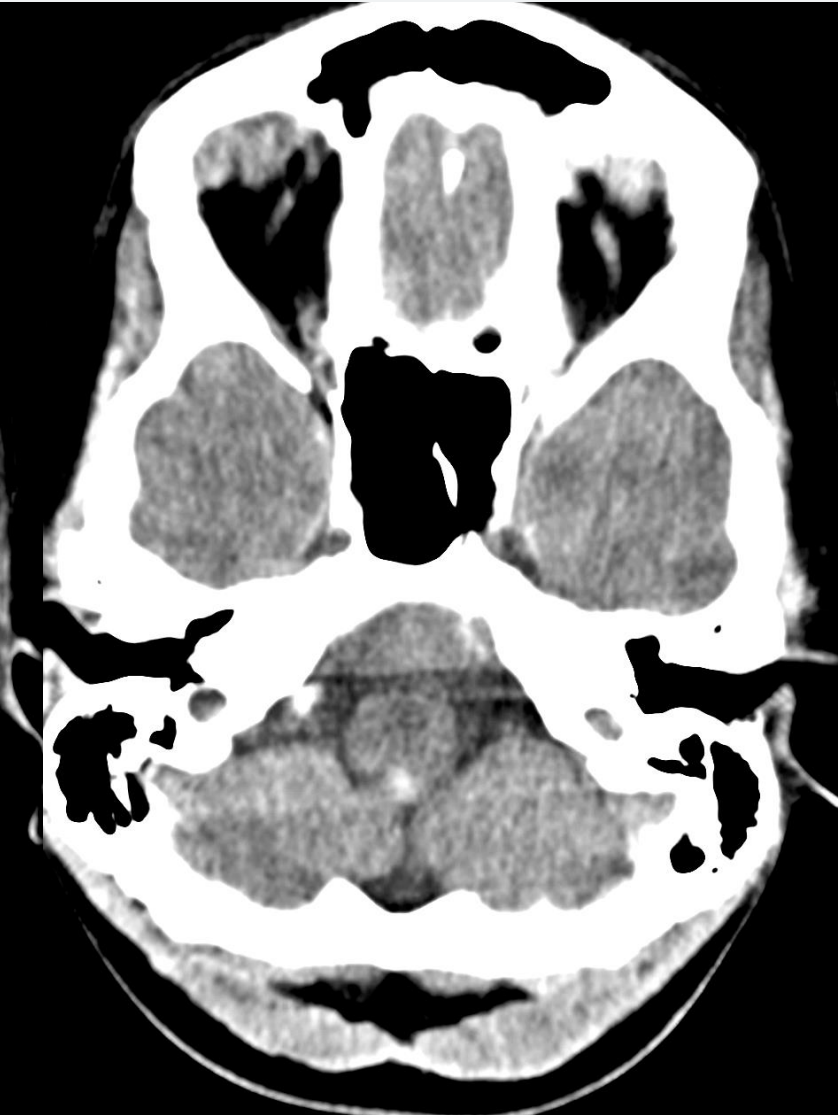
Can be multiple – don't stop looking once found one!

2016 Modified WFNS grading system

- Grade I:** GCS 15
- Grade II:** GCS 14
- Grade III:** GCS 13
- Grade IV:** GCS 7-12
- Grade V:** GCS 3-6

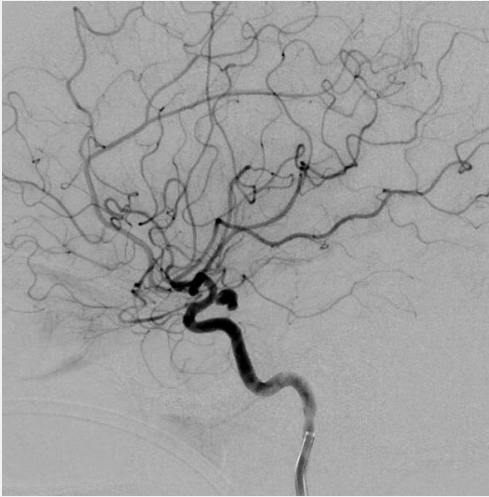


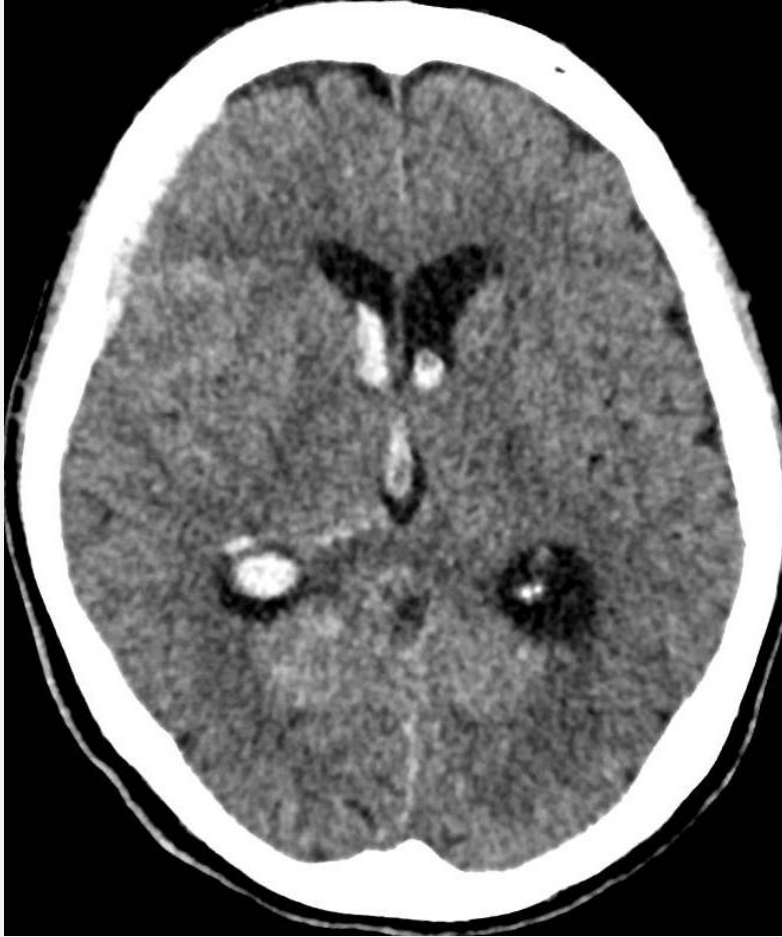
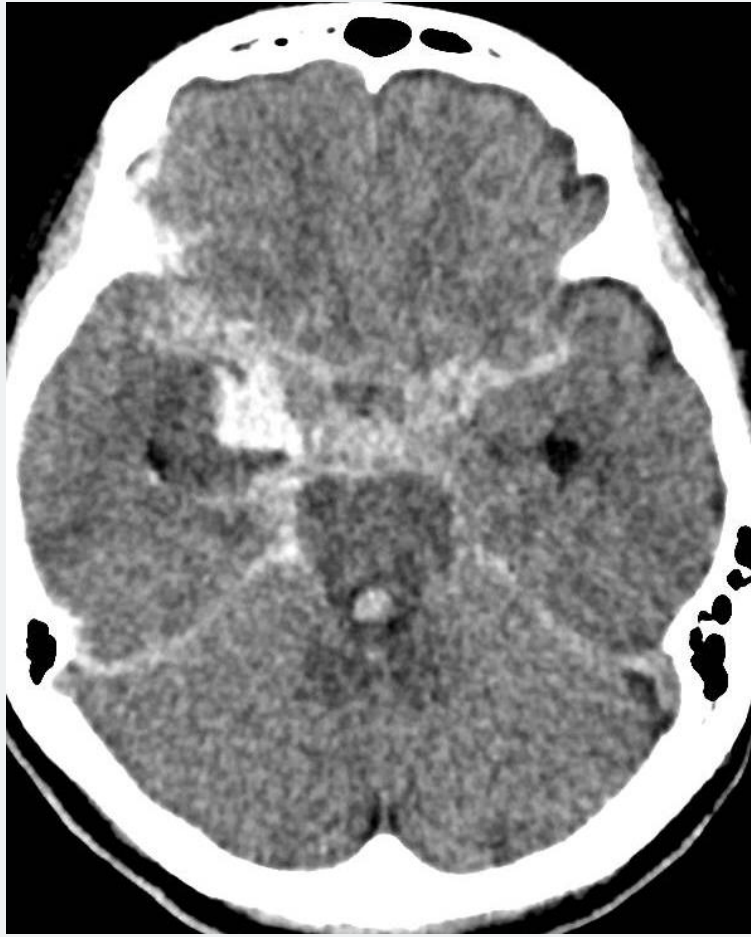


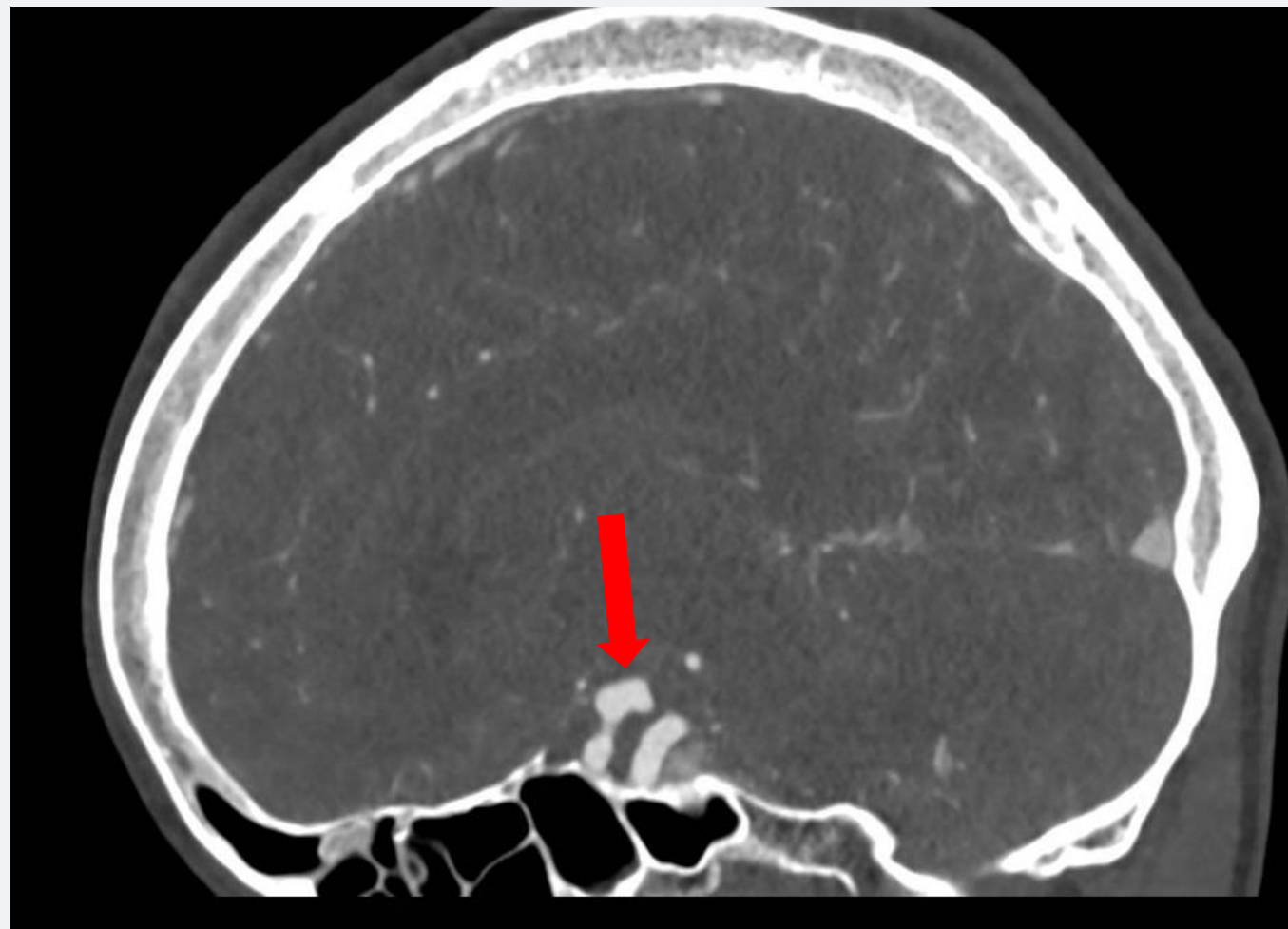
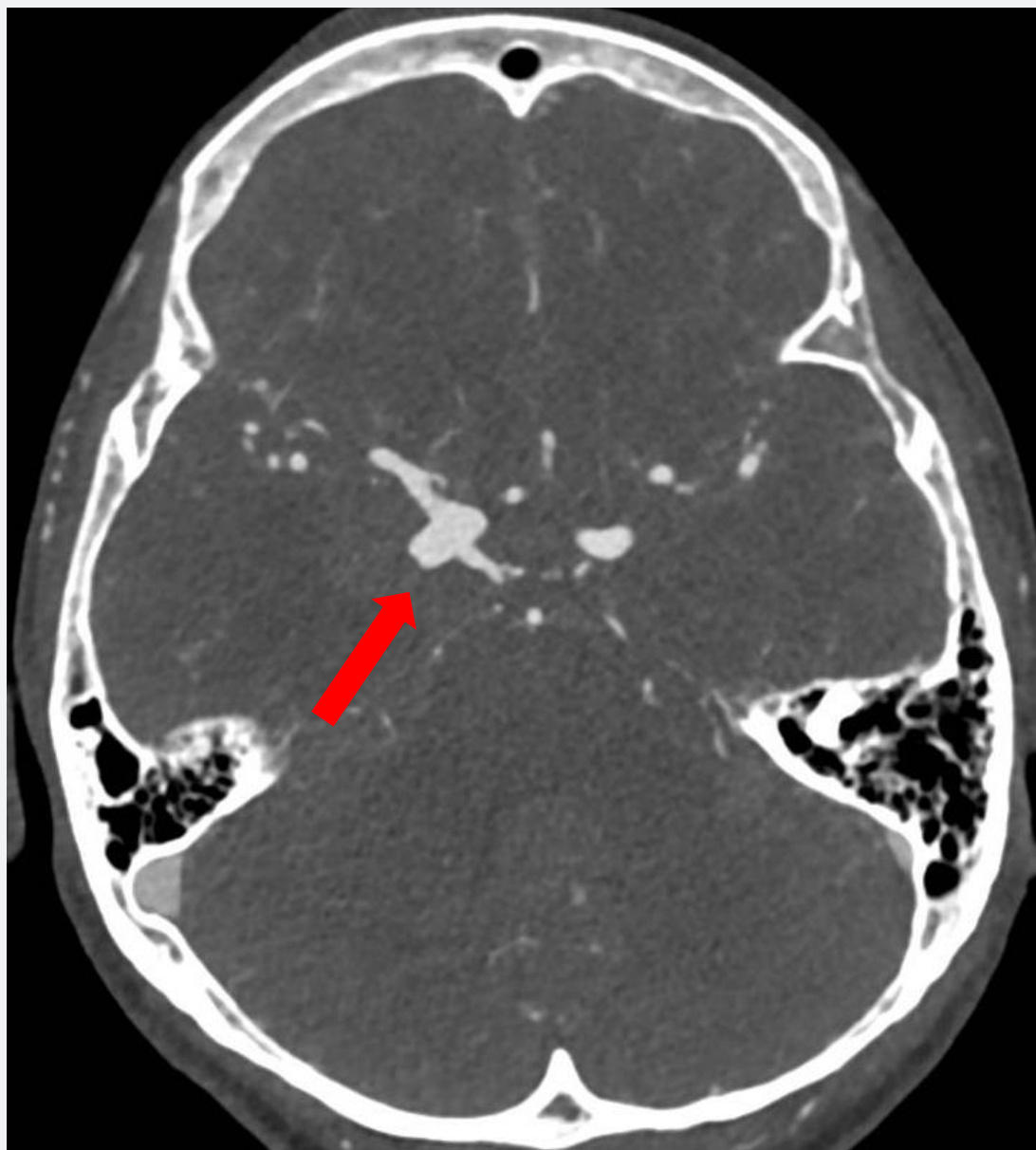




Posterior communicating artery
aneurysm







Perimesencephalic SAH



Involves the perimesencephalic cisterns

May extend to medial Sylvian fissure or interhemispheric fissure

No *significant* intraventricular component (caution – dependent occipital blood may be seen)

Typically not aneurysmal

- >90% cases no aetiology found
- ?venous aetiology
- poorer prognosis in the <10% with an aneurysm

RCVS

Reversible cerebral
vasoconstriction syndrome

Typically affects young women

Thunderclap headache – can
be recurrent

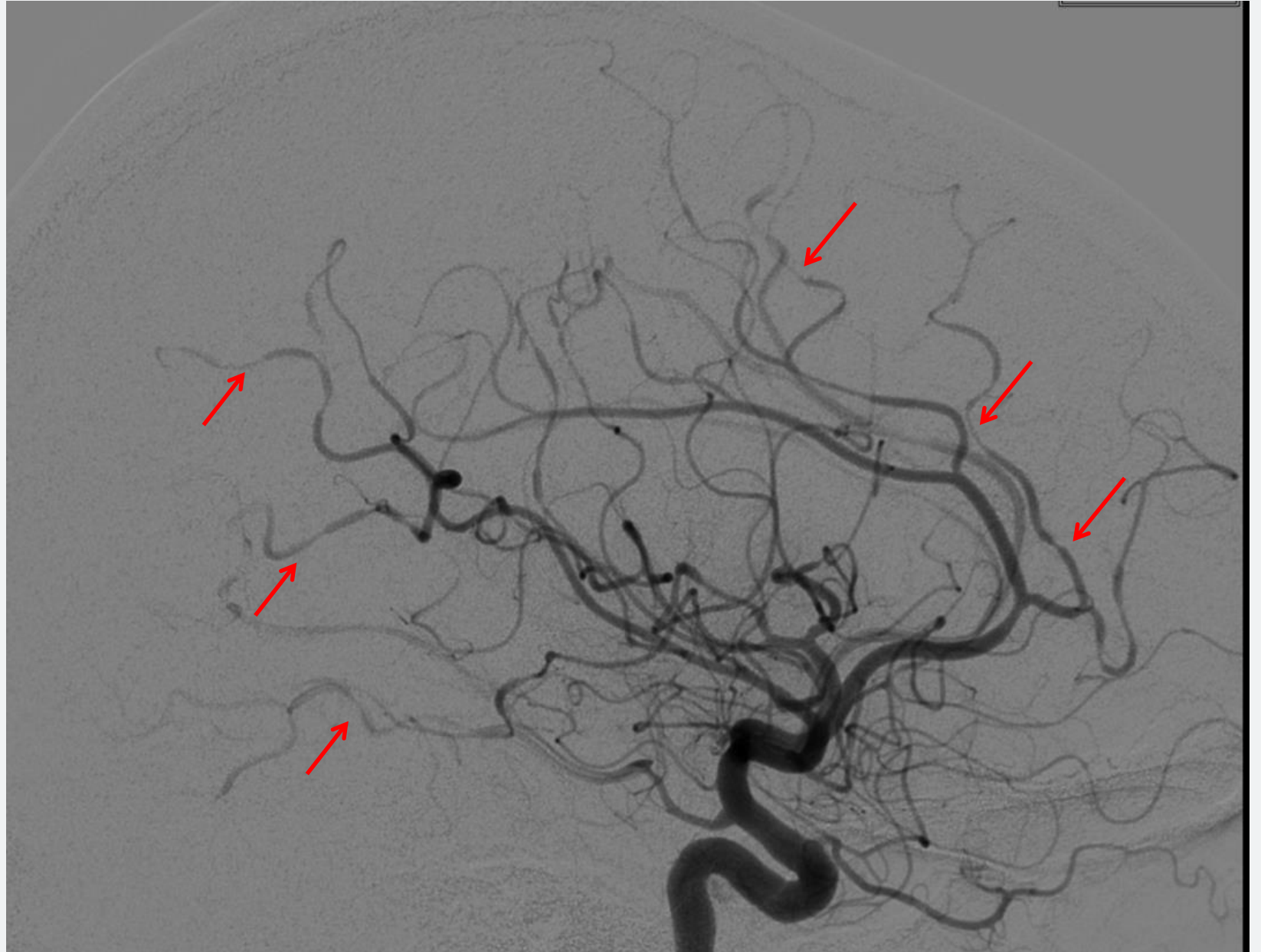
Irregular narrowing of vessels
leads to:

- Convexity SAH
- Infarcts
- Oedema

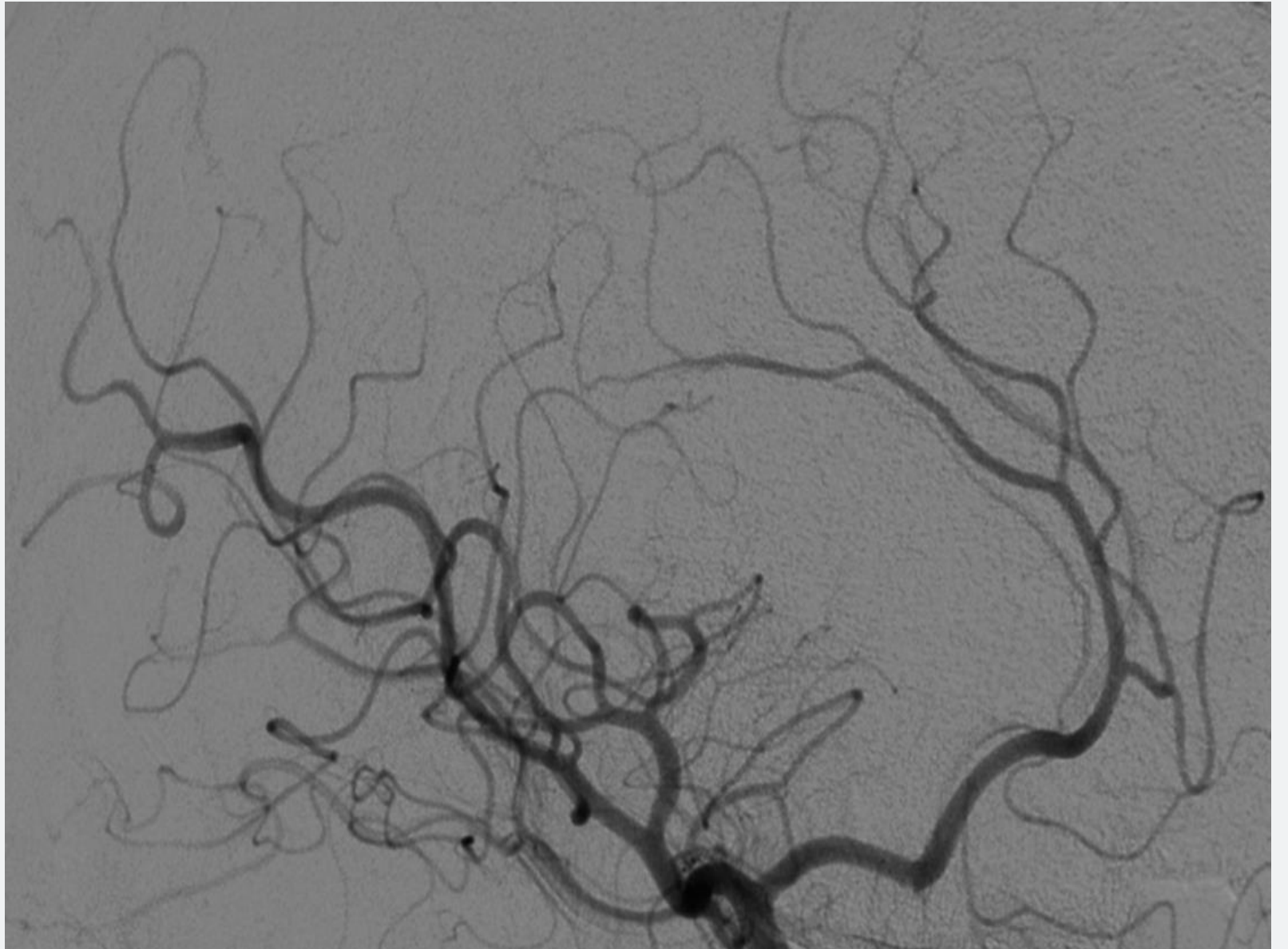
Mitchell P, Convexity subarachnoid haemorrhage:
reversible cerebral vasoconstriction syndrome. Case
study, Radiopaedia.org (Accessed on 20 Apr 2023)
<https://doi.org/10.53347/rID-34462>



March 2017



**3 months
later...**

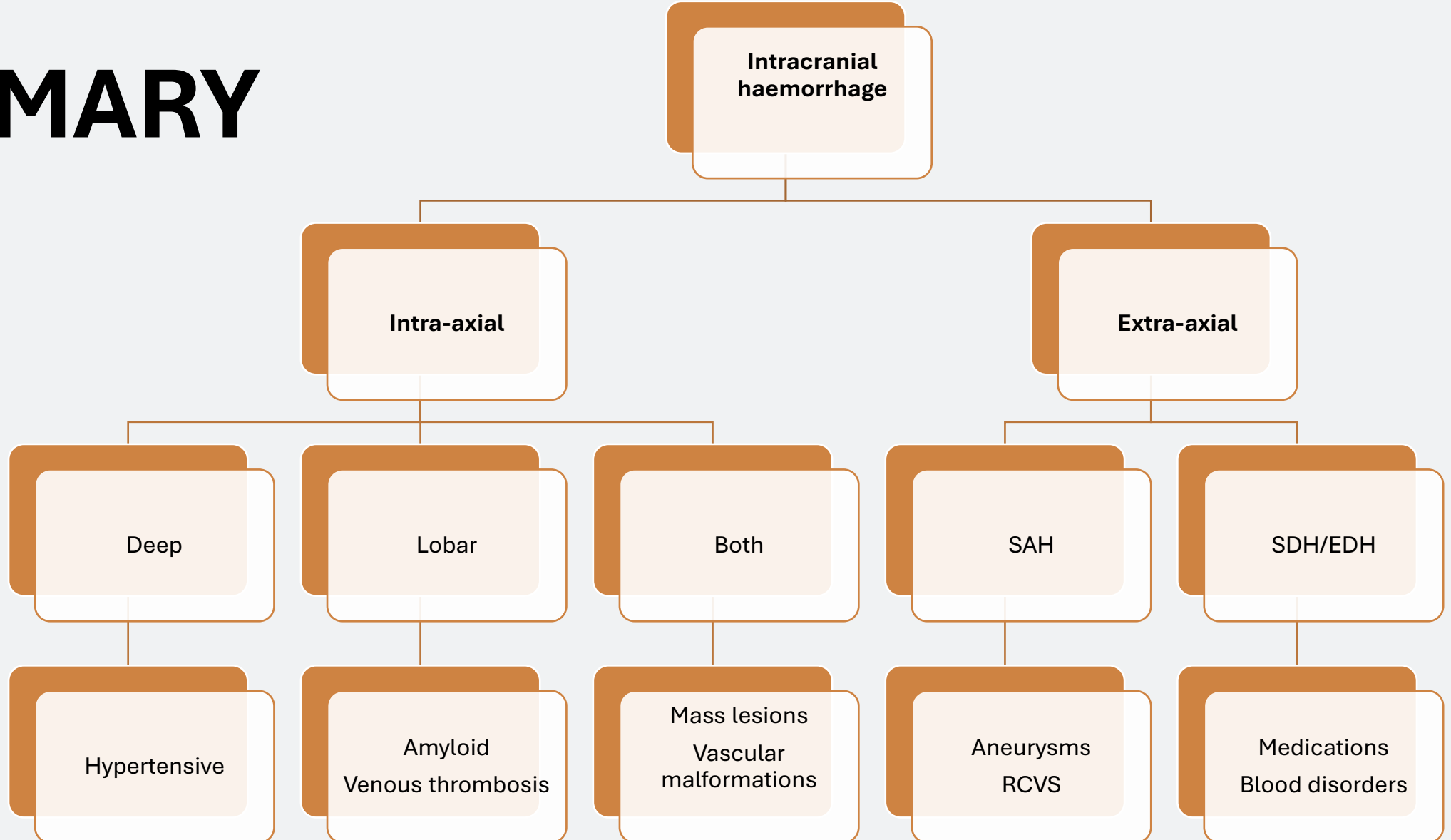


Extra-axial

- subdural
- extra-dural
- typically traumatic
- consider “occult” trauma and medication (DOAC, warfarin etc).



SUMMARY



Thank you



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