Case 1: ICH

- 63y Male
- Clinical Symptoms

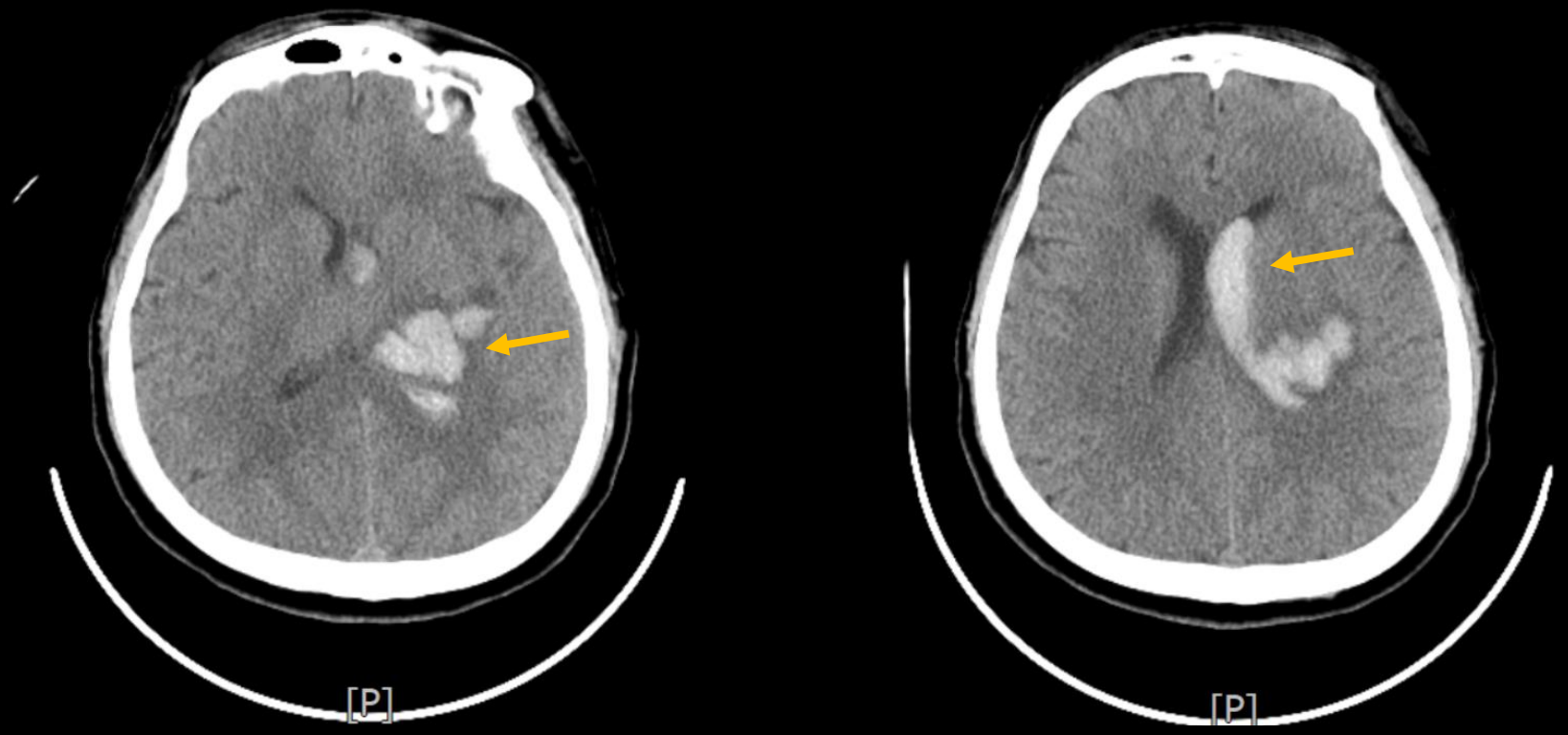
  Right side weakness, muscle power (RU/RL/LU/LL): 0/0/5/5
- Past History

  Hypertension

2022/05/07 CT

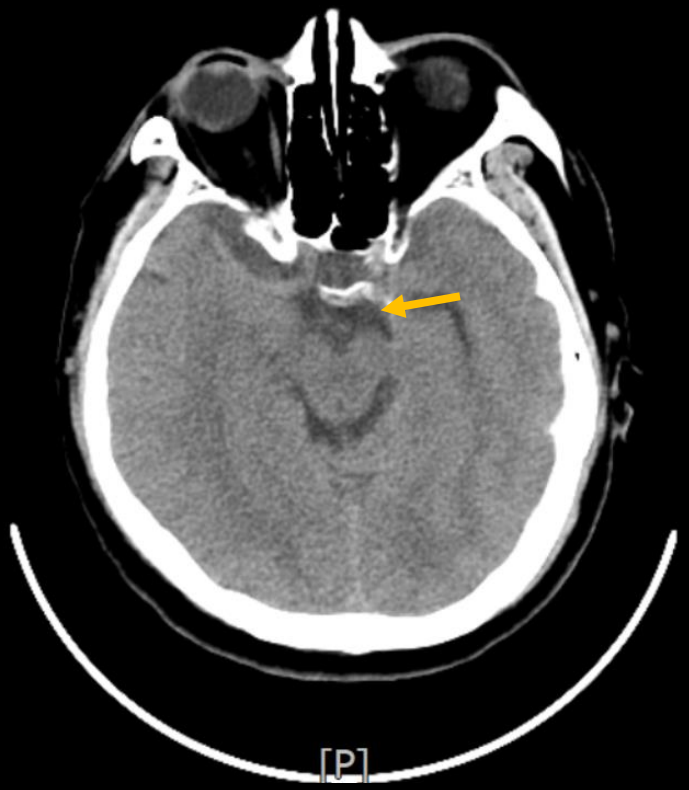
- Hyperdense mass in left thalamus, putamen and lateral ventricle
- Slightly dilatation of left lateral ventricle
- The midline is not shifted

-> deep ICH with IVH



2022/05/07 CT

- Clear suprasellar cistern and sulci -> no SAH
- Mild atrophy of brain tissue, correlated with aging (63y)
- Grey-white matter differentiation looks adequate

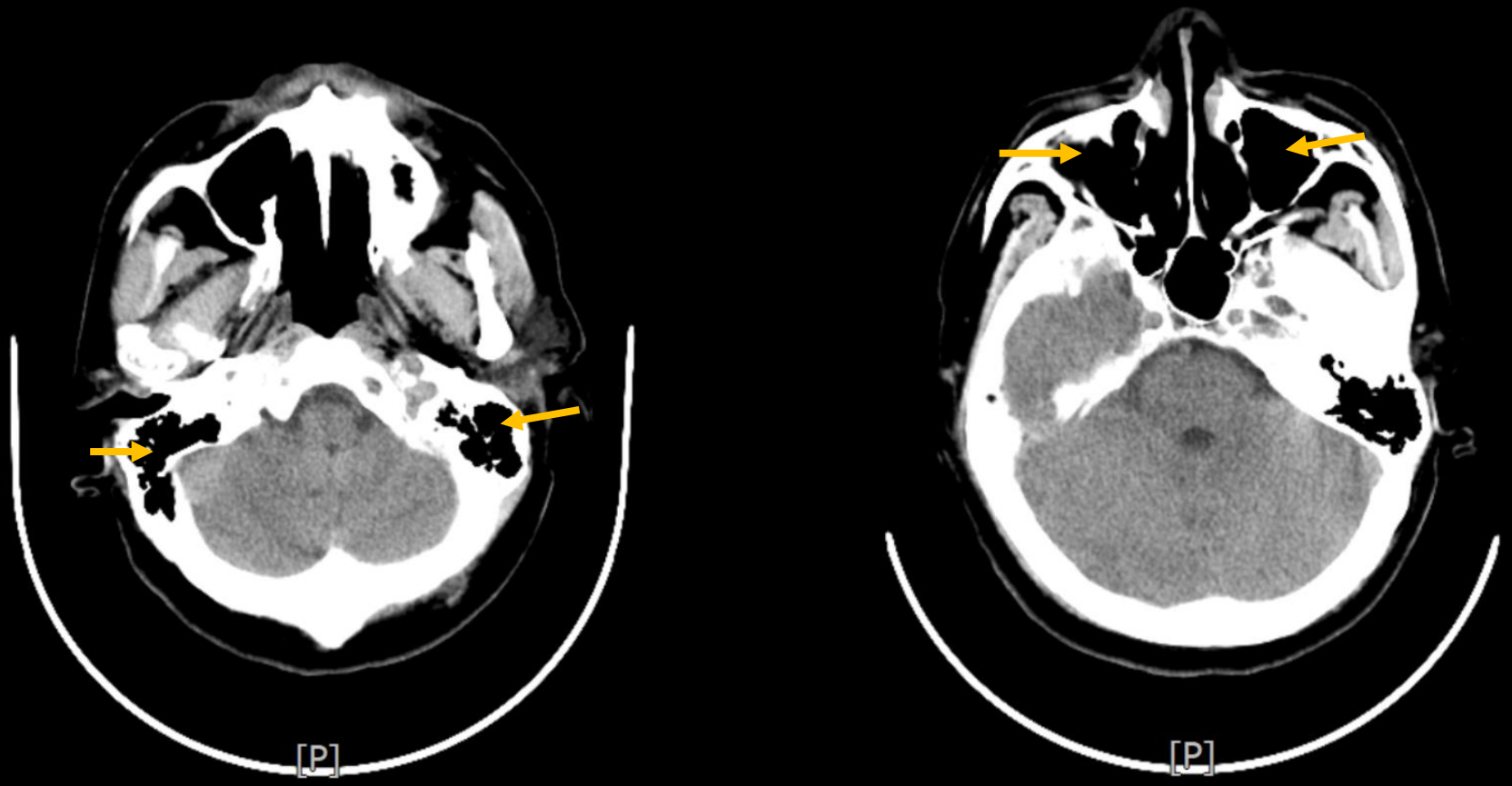




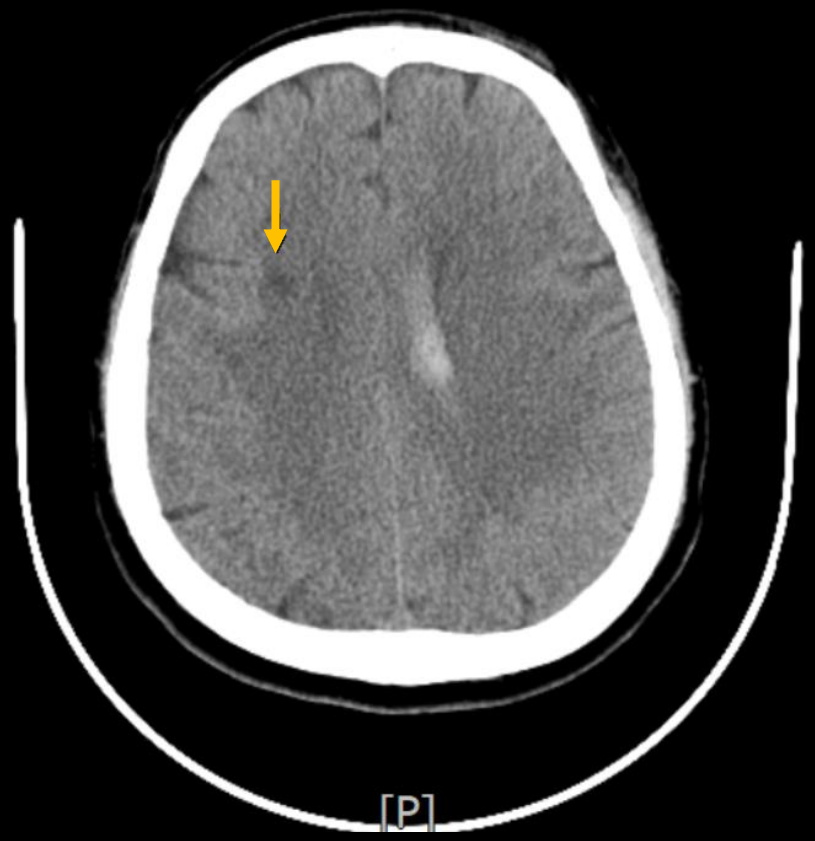
2022/05/07 CT

- Pneumatized mastoid sinus and paranasal sinus

-> no sinusitis or COM



2022/05/07 CT

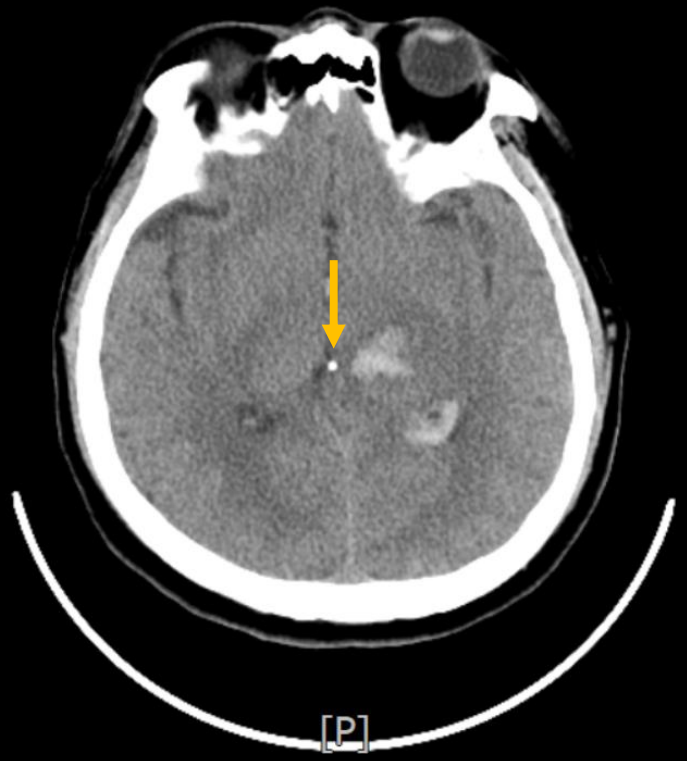


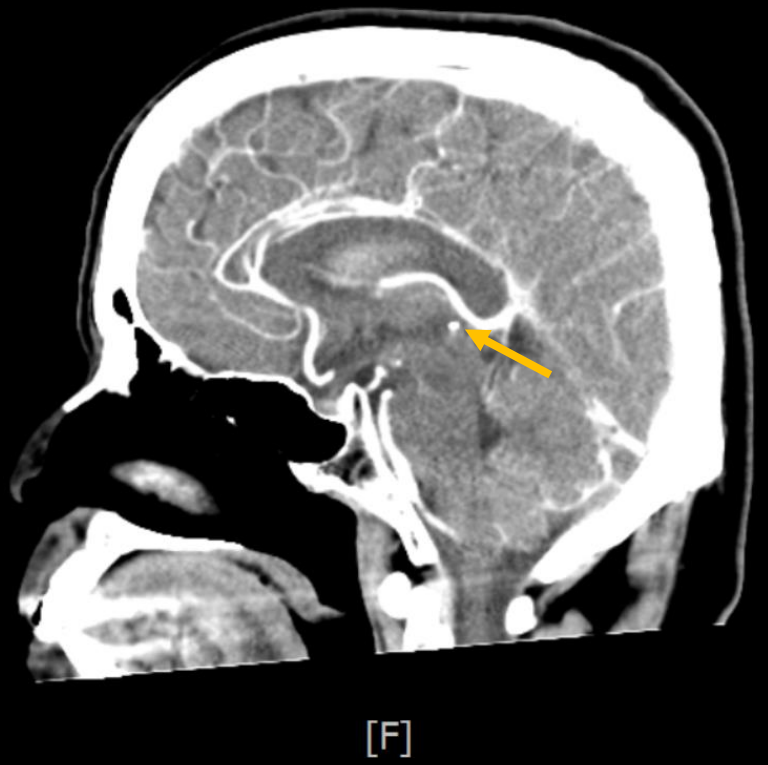
- A hypodense lesion at right corona radiata

-> May be old lacunar infarction? Look up for further information in MRI.

2022/05/07 CT

- Physiological calcification of pineal gland





2022/05/17 MRI (10 days after)

- Hematoma follow up: (orange arrows)

  Marginal T1 hyperintensity

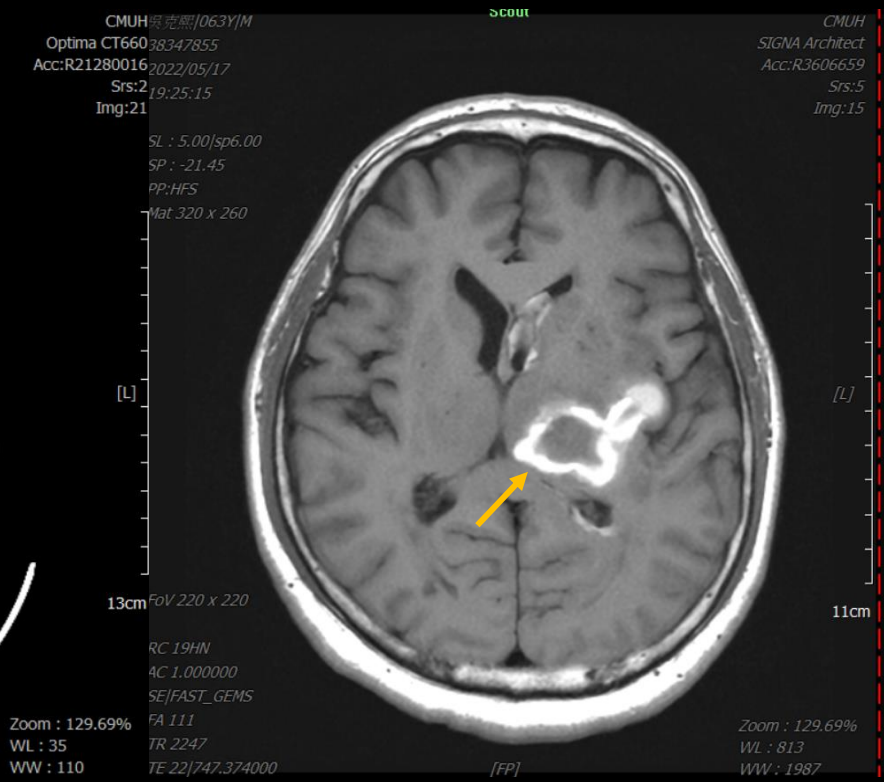
  Heterogeneous T2 intermediate to hypointensity

  -> early subacute stage of ICH

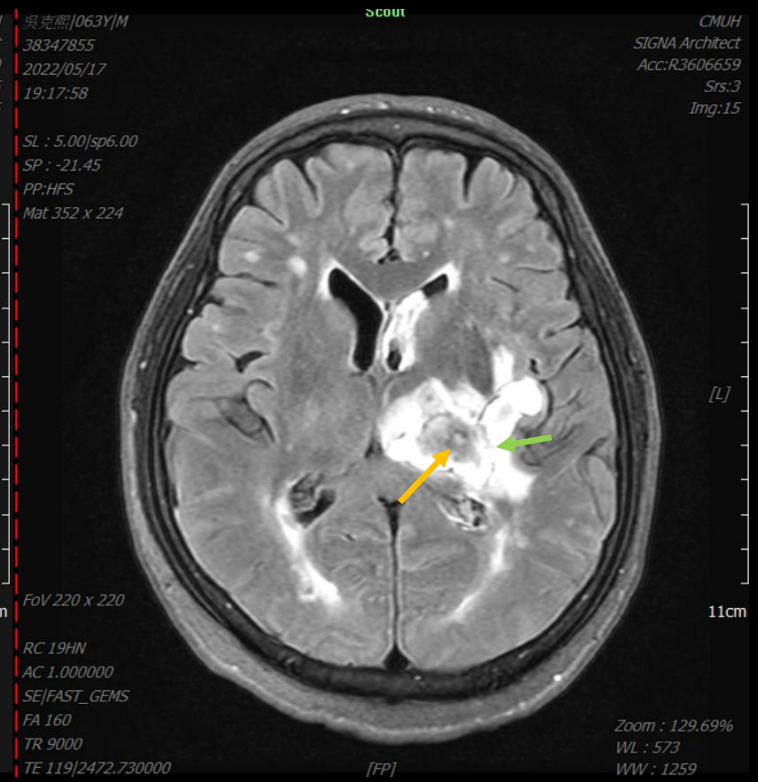
- T2 hyperintensity surrounding the hematoma -> **edema** (green arrows)



CT on 05/07



T1 FLAIR



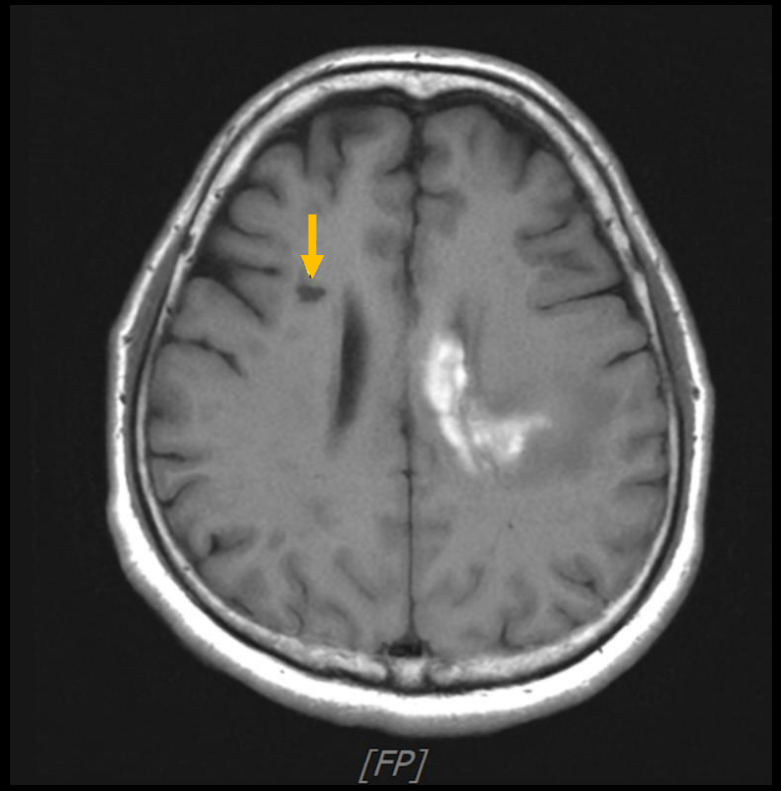
T2 FLAIR

2022/05/17 MRI

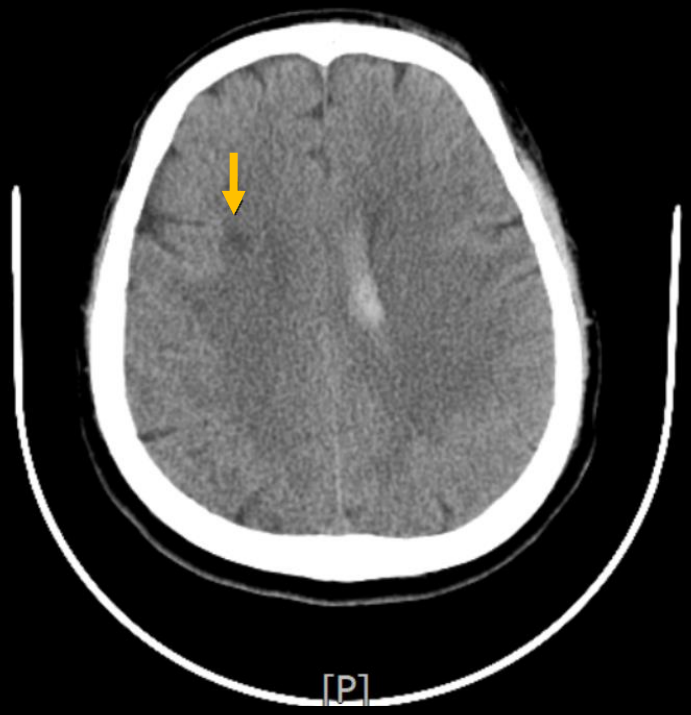
- Old lacuna at right corona radiata, consistent with CT image

-> lacunar infarction, a hint of small vessel disease (SVD)

- Atrophy of brain tissue more obvious in MRI than CT (dilatation of sulci)



T1 FLAIR

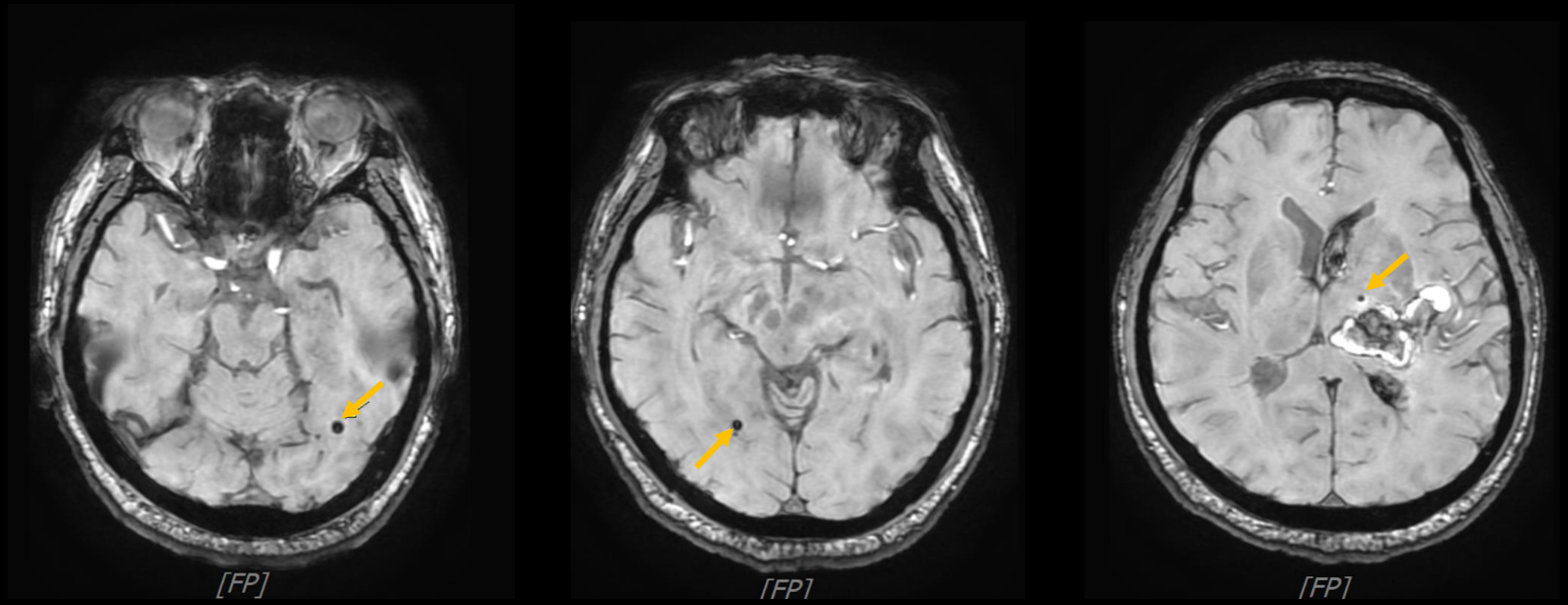


CT on 05/07

2022/05/17 MRI

- Dots of hypointensity in bilateral occipital lobes and left thalamus ventral to hematoma

-> microbleed, a hint of small vessel disease (SVD)

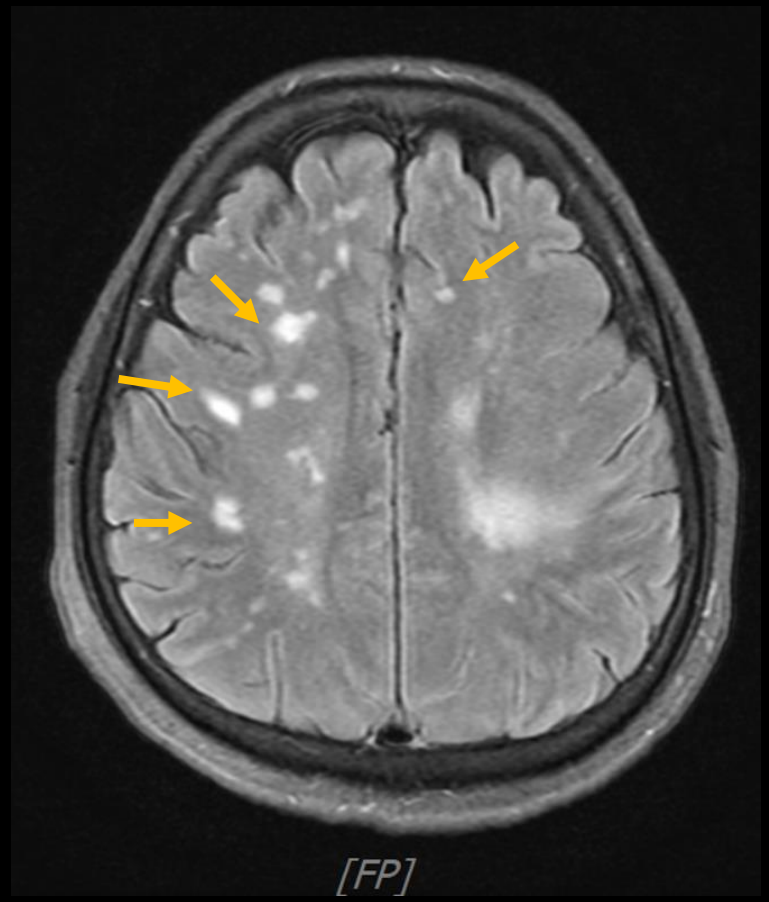


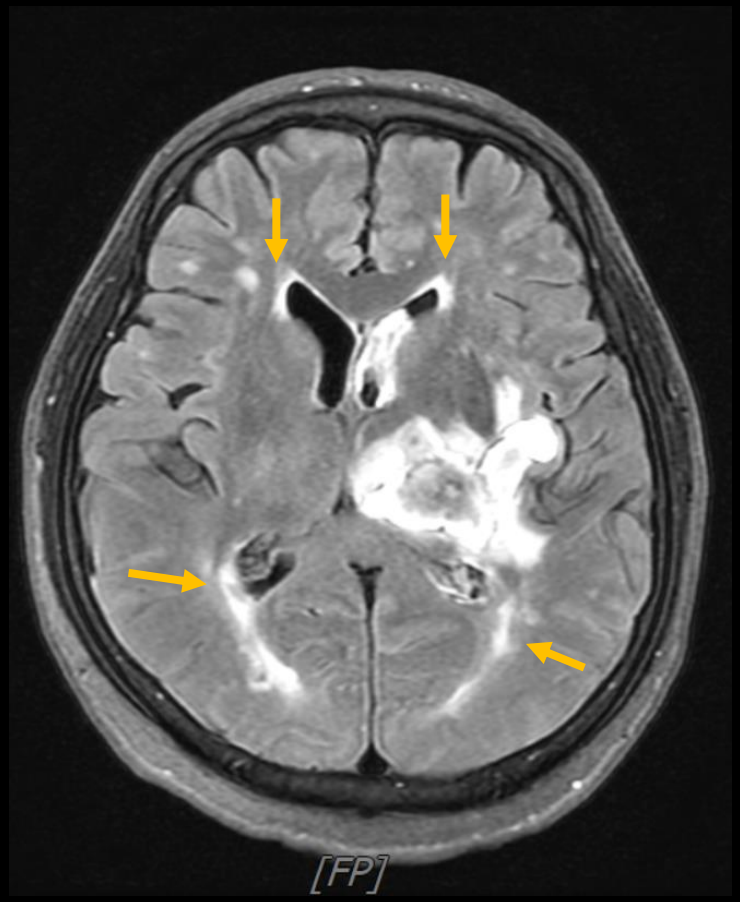
SWAN

2022/05/17 MRI

- Multiple small hyperintensities in periventricular and subcortical area of the bilateral cerebrum

-> leukoaraiosis, a hint of small vessel disease (SVD)

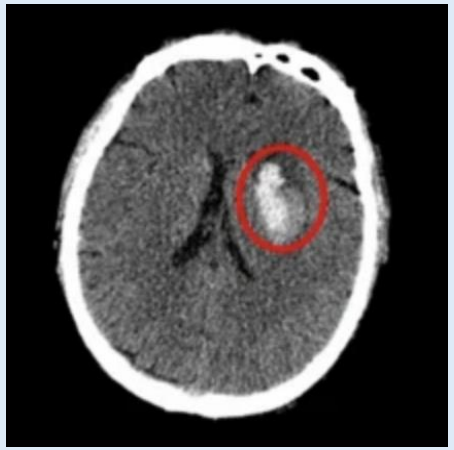




T2 FLAIR

Review of Hypertensive ICH

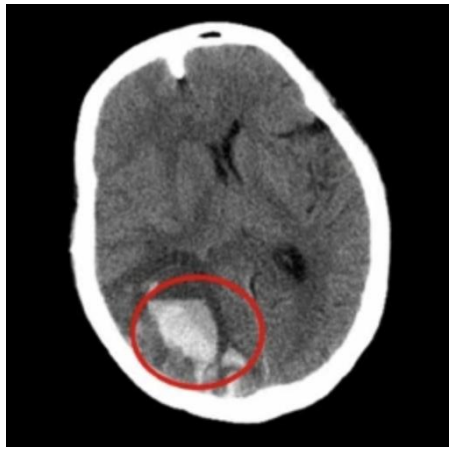
Types and Common Causes of ICH



Deep ICH

(putamen, thalamus, pons…)

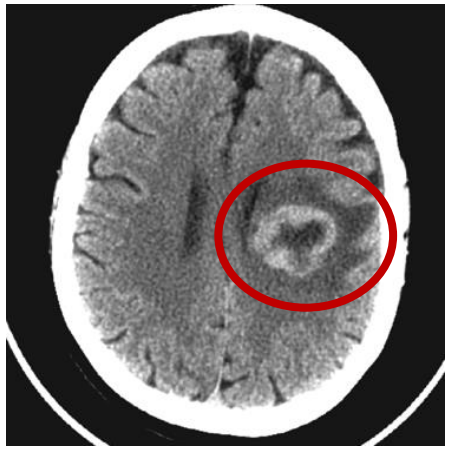
-> Hypertension



Lobar ICH

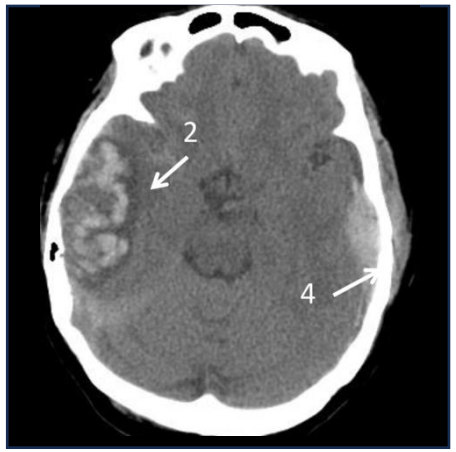
-> Old age: amyloid angiopathy

-> Young age: AVM



Acute ICH with edema

-> Tumor bleeding

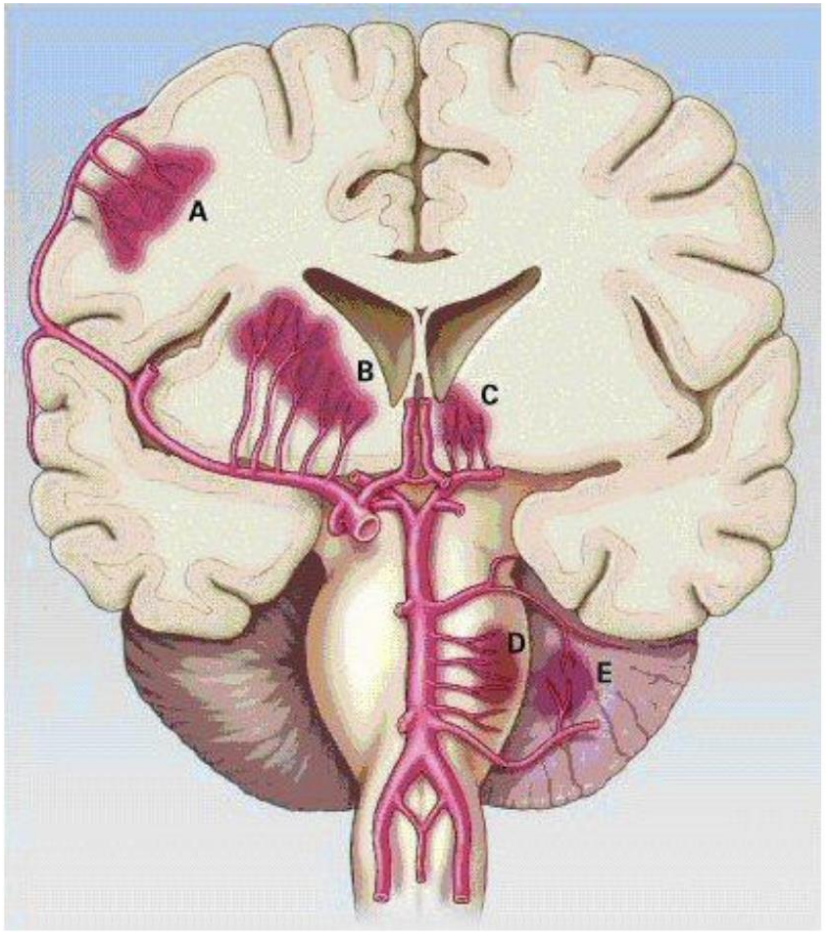


Contusion ICH with hematoma

-> Head injury

Location of Hypertensive ICH and related symptom

1. Putamen (B)
   -> Contralateral hemiparesis
2. Thalamus (C)
   -> Contralateral hemiparesis, sensory loss
3. Pons (D)
   -> Coma, pinpoint pupil, quadriplegia
4. Cerebellum (E)
   -> Ataxia, vertigo, vomiting
5. Subcortex of cerebrum (A)
   -> Contralateral motor/sensory deficits



https://dx.doi.org/10.26044/ecr2020/C-02134

Pathophysiology

Hypertension causes a number of abnormalities in vessel walls, the 2 most prominent :

- Large artery: atherosclerosis

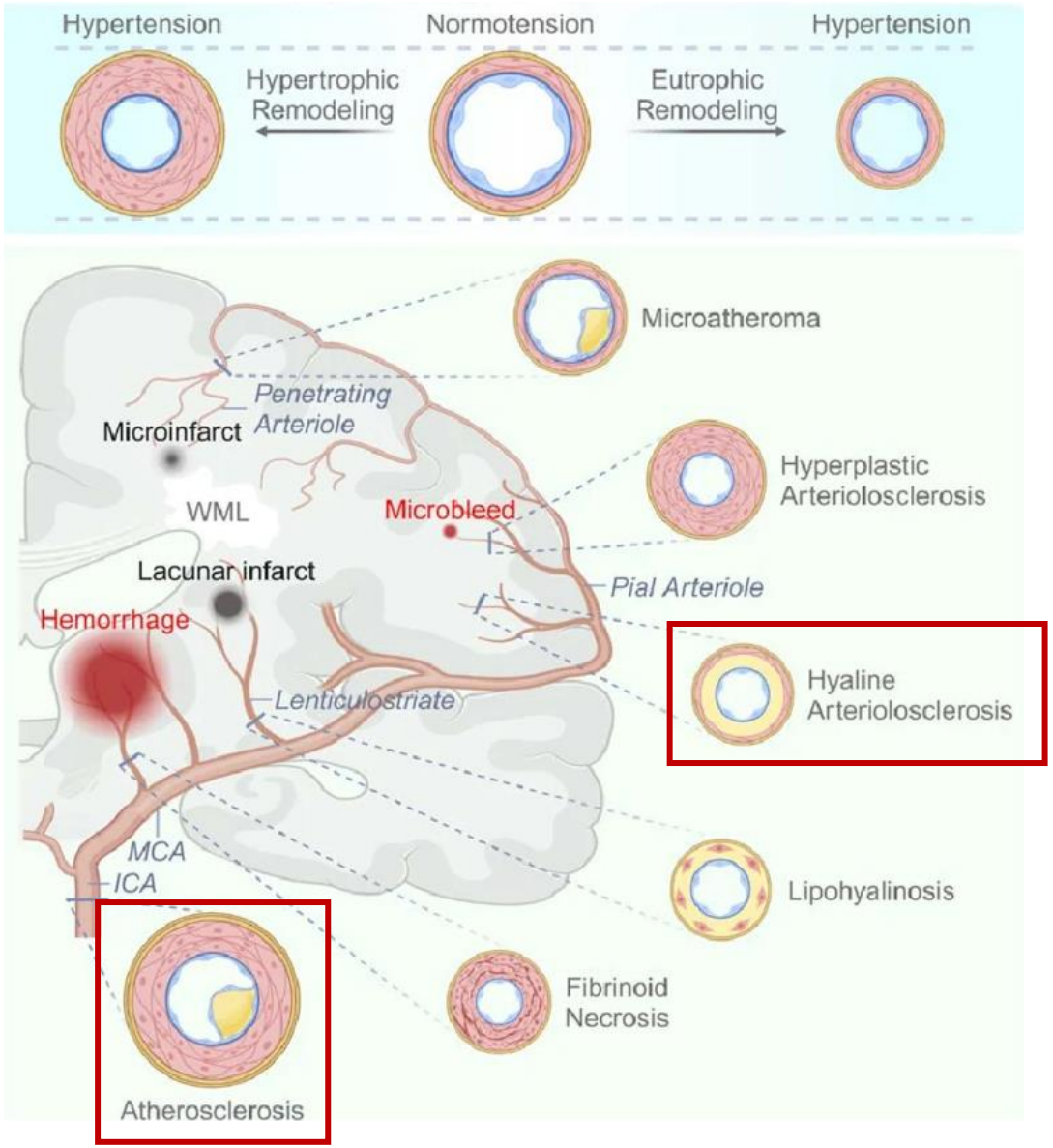
- Small artery: **hyaline arteriosclerosis**

-> Small vessel disease (SVD)

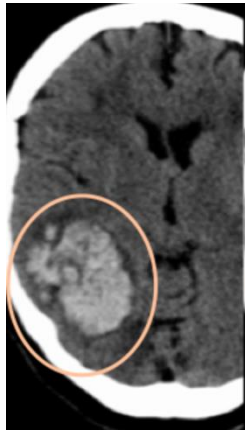
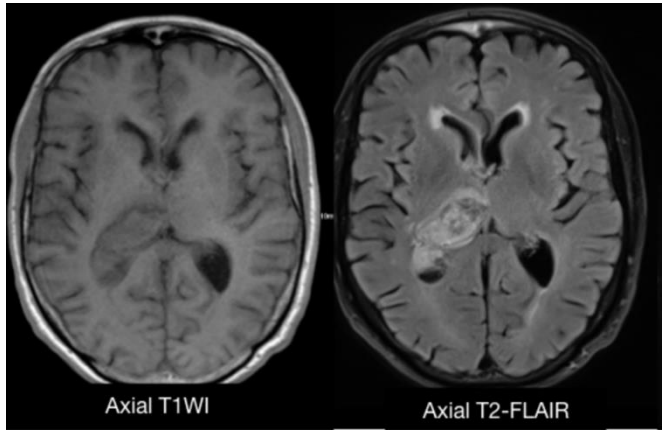
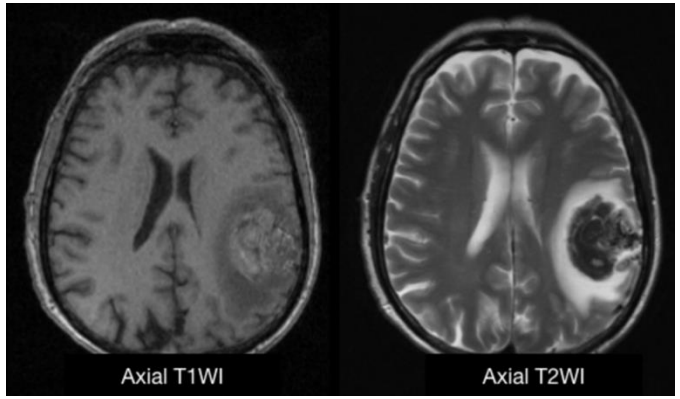
-> Small penetrating artery rupture

-> Deep ICH

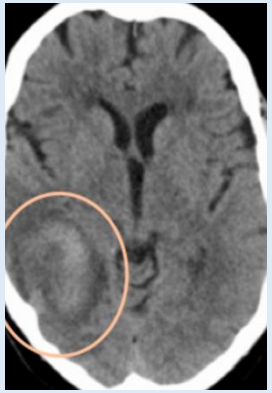
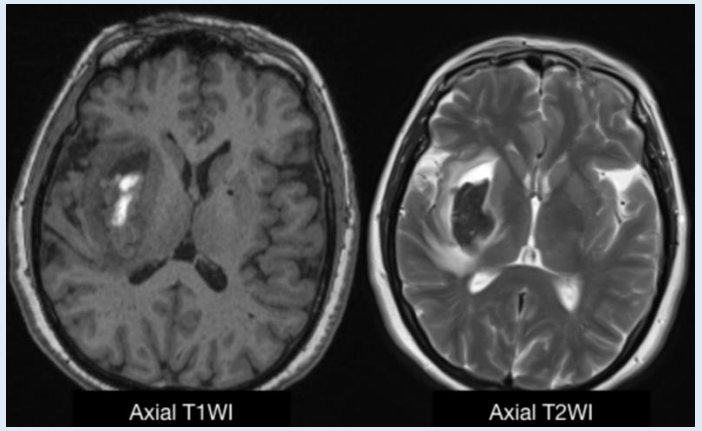
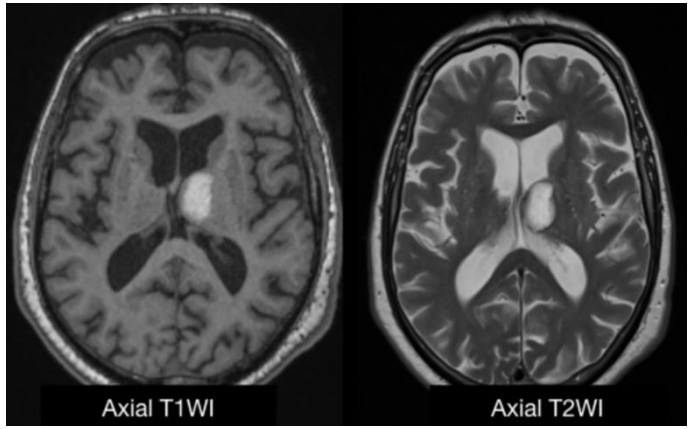
Other SVD symptoms:
lacunar infarction, microbleed, leukoaraiosis, amyloid angiopathy



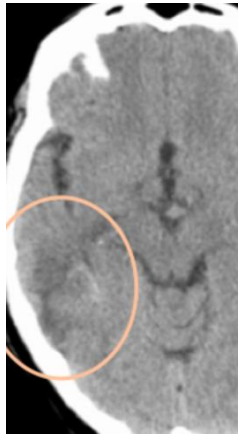
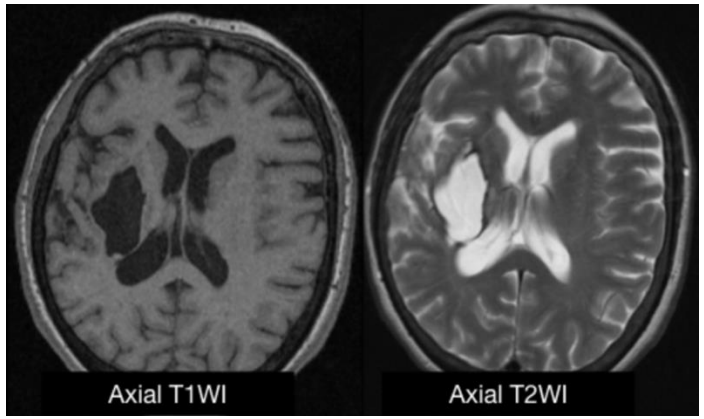
Evolution of ICH

Stage	CT	MRI
Hyperacute <12 hrs	 • Hyperdense hematoma • No edema	 Oxyhemoglobin • T1: Hypointensity • T2: Hyperintensity
Acute 12 hrs～ 2 days	 • Hyperdense hematoma • Perilesional edema	 Deoxyhemoglobin • T1: Hypointensity • T2: Hypointensity, with surrounding hyperintensity (edema)

Evolution of ICH

Stage	CT	MRI
Early subacute 2 days～ 2 weeks	 • Progressively deceased hyperdense hematoma • More edema	 Intracellular methemoglobin • T1: Hyperintensity • T2: Hypointensity
Late subacute 2 weeks～ 2 months	 • Hematoma becomes isodense or hypodense • Less edema	 Extracellular methemoglobin • T1: Hyperintensity • T2: Hyperintensity

Evolution of ICH

Stage	CT	MRI
Hyperacute >2 months	 • Hematoma becomes hypodense or linear cavity	 • T1: Cavitary change • T2: Hyperintensity (water in the cavity), and peripheral hypoitensity (hemosiderin)

https://dx.doi.org/10.26044/ecr2020/C-02134

Case 2: SAH

- 70y Female
- Clinical Symptoms

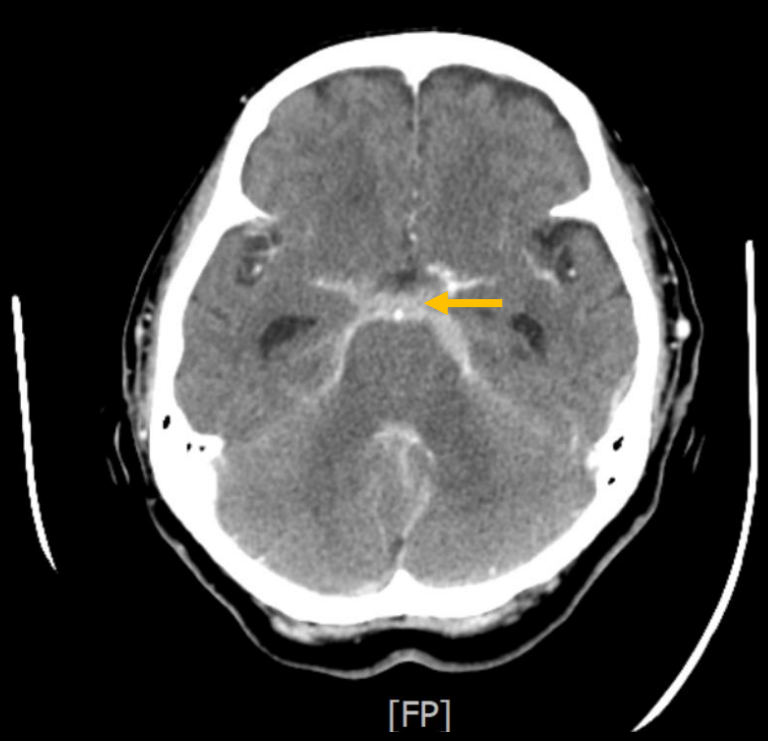
  Headache, vomiting, consciousness loss
- Past History

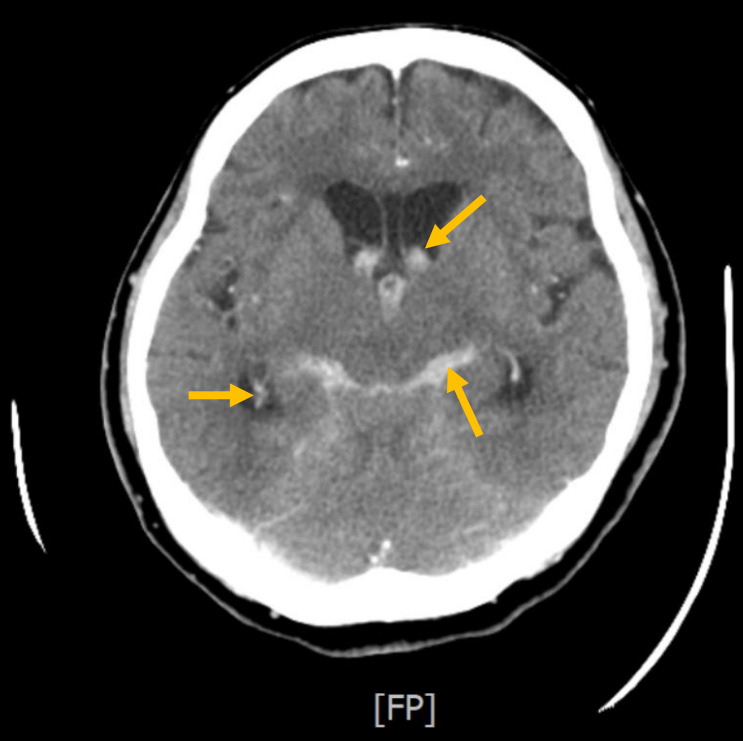
  Unknown

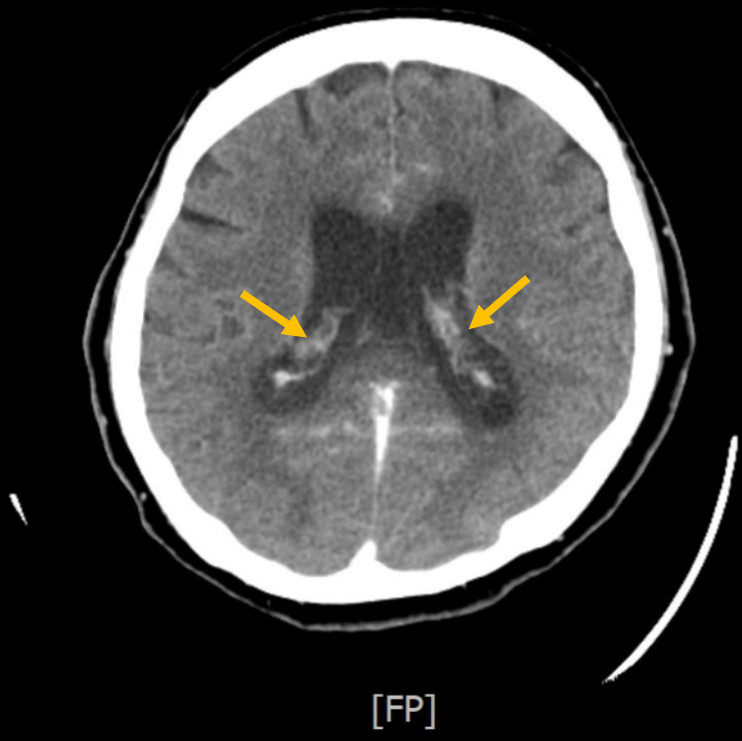
2026/10/15 CT

- Hyperdense mass in suprasellar cistern, ambient cistern and quadrigeminal cistern, lateral ventricles
- Dilatation of lateral ventricle and visible lateral horn
- No ICH was seen

-> Primary SAH with IVH, acute hydrocephalus

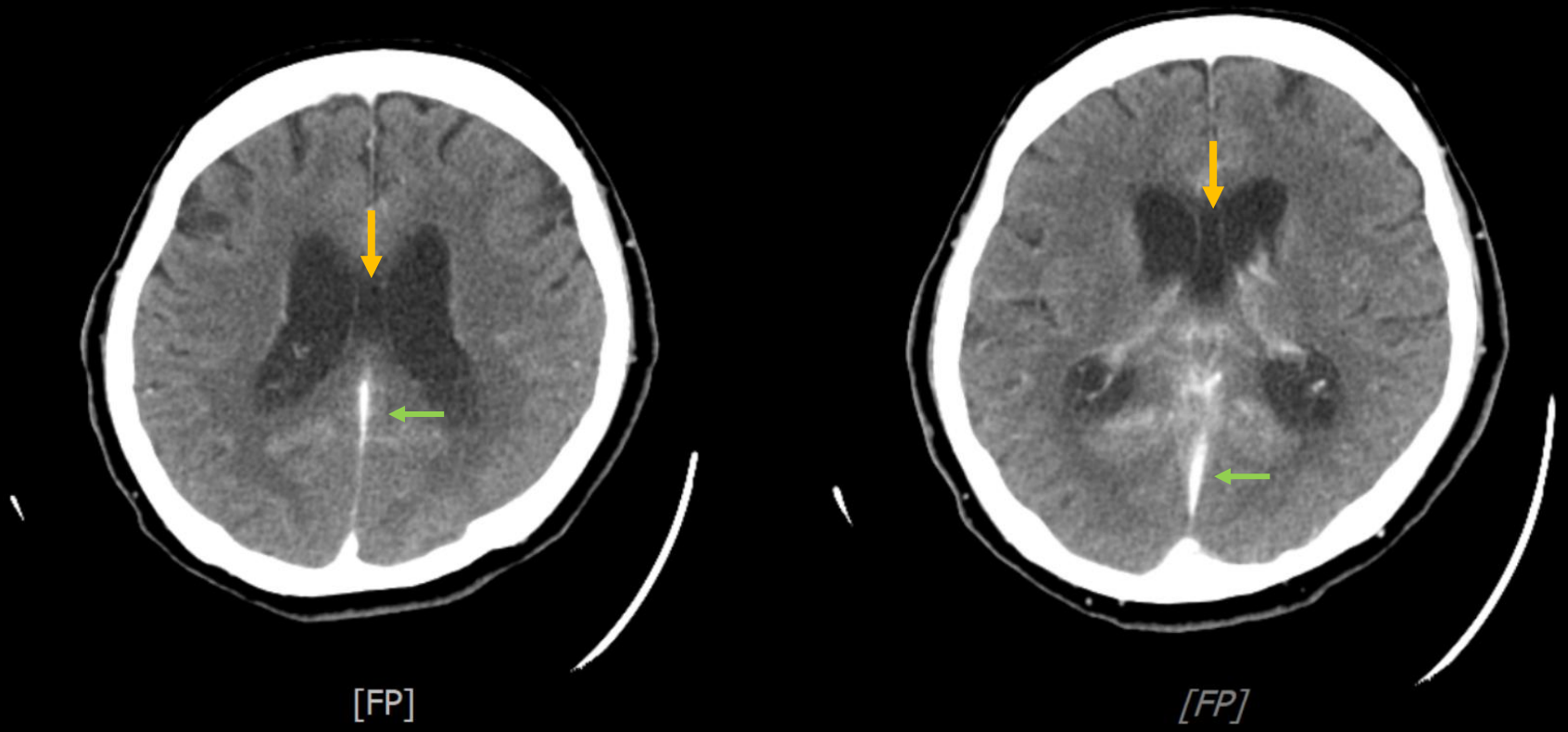






2026/10/15 CT

- 5th ventricle (orange arrow)
- Atrophy of the brain tissue, correlated to aging (70y)
- Physiological calcification of cerebral falx (green arrow)



2022/05/07 CT



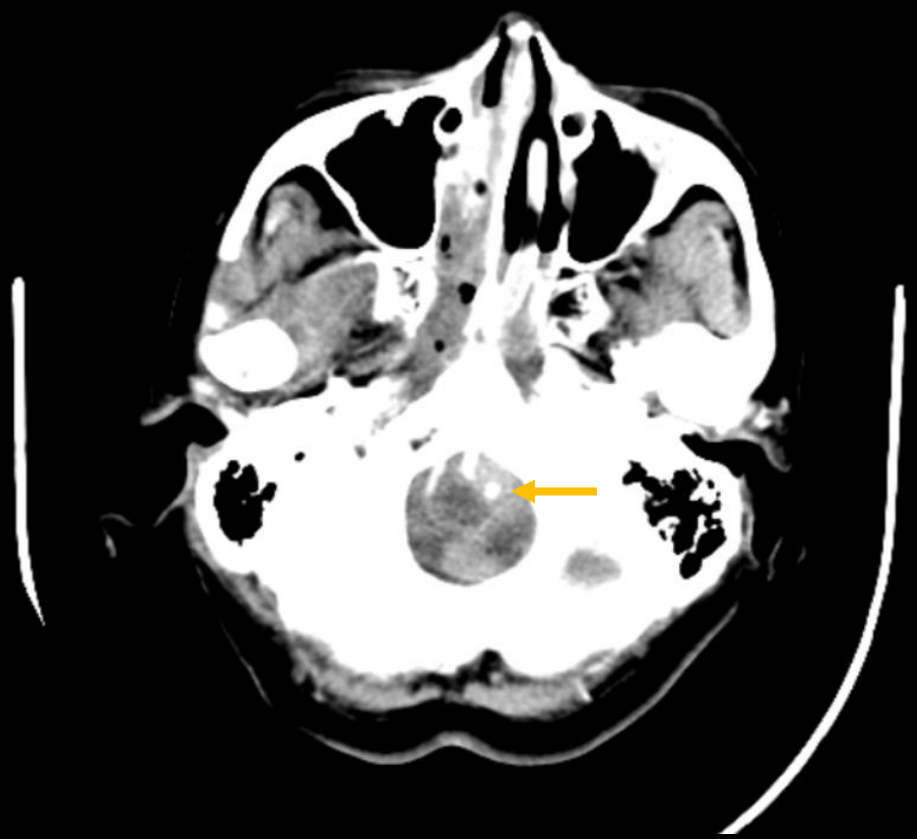
- Pneumatized mastoid sinus and paranasal sinus

-> No sinusitis or COM

2026/10/15 CTA

- Suspicious small aneurysm of left vertebral artery

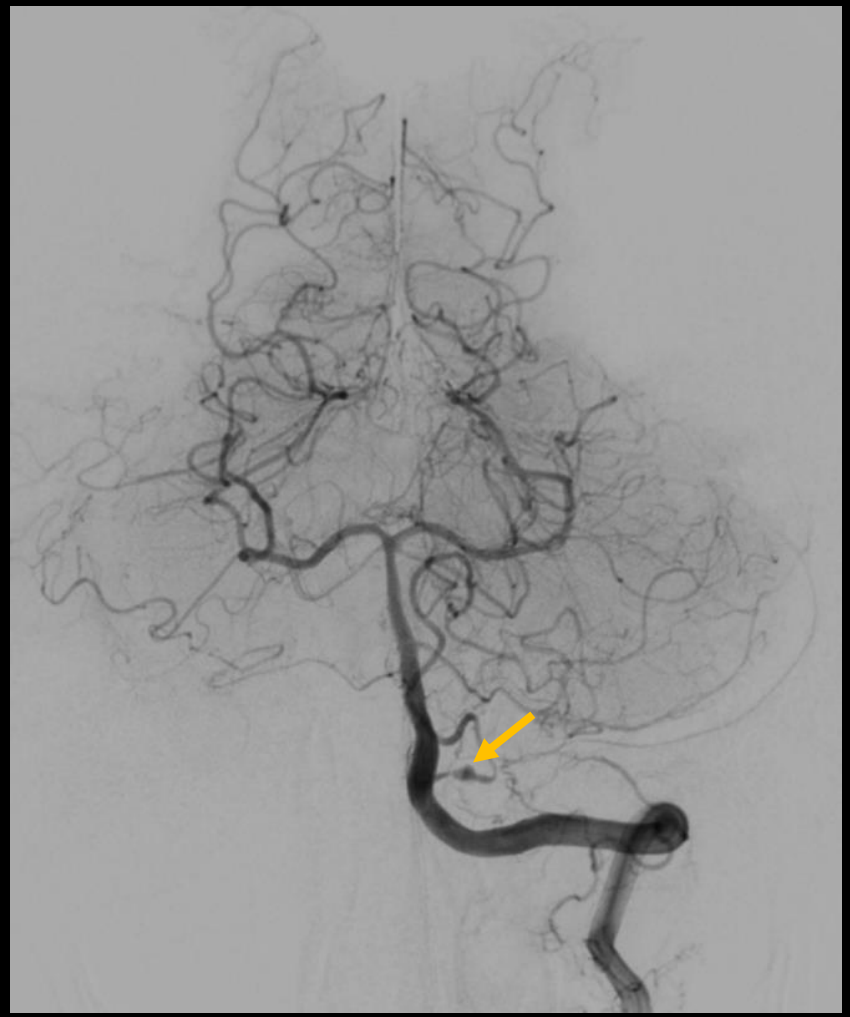
-> Arrange DSA for further evaluation





2026/10/15 DSA

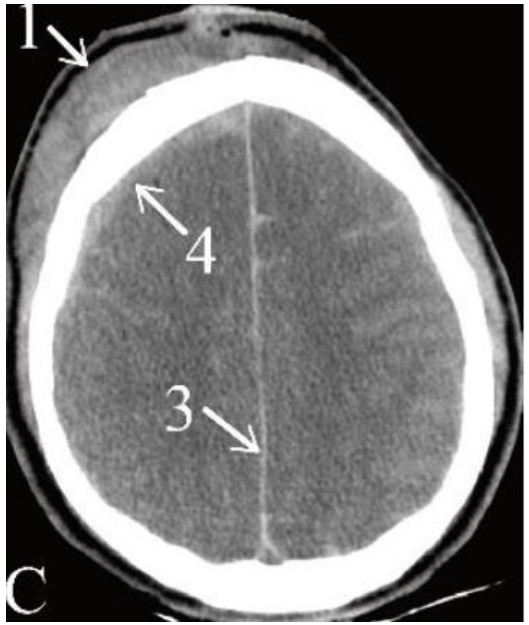
- Confirmed an aneurysm of left posterior inferior cerebellar artery (PICA), 2.12*1.76 mm





Review of Primary SAH

Types of SAH



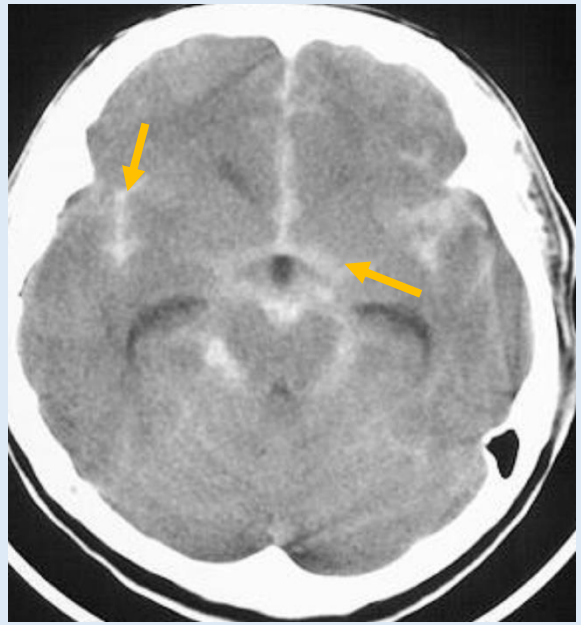
Traumatic SAH

- Most common in sulci and sylvian fissures, interhemispheric fissure
- Usually with sculp swelling, EDH, SDH…



Secondary SAH

- Developed from ICH (rupture into subarachnoid space or ventricle)



Primary SAH

- Extend from suprasellar cistern to other subarachnoid space
- Sometimes with IVH

Primary SAH

Symptoms

- Severe headache
- Vomiting
- Neck stiffness
- Conscious change

-> Similar with meningitis

Common Causes

1. Aneurysm: most common
2. AVM
3. Vasculitis: rare, hemorrhage in sulci
4. Arteriosclerosis and rupture

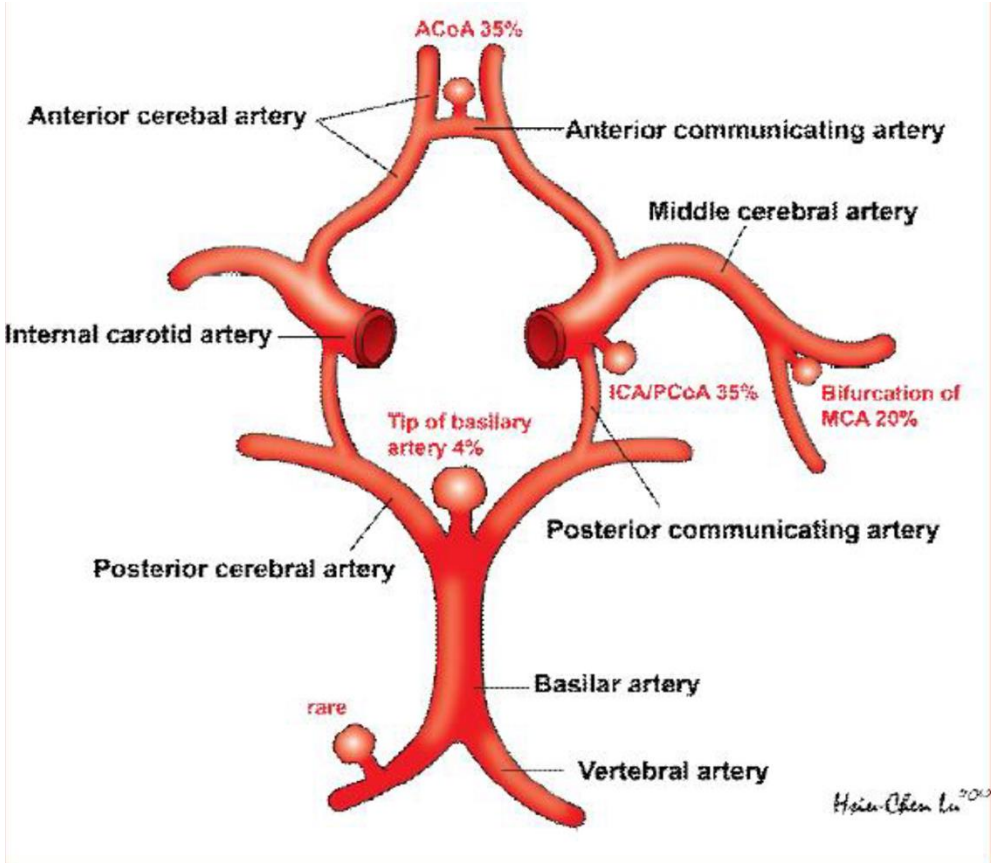
In this case, the patient developed typical symptoms of SAH, which was caused by aneurysm.

Common Location of Aneurysm

1. ACoA
2. ICA/PCoA
3. Tip of basilar artery

Diffuse from suprasellar cistern

4. Bifurcation of MCA
   -> Diffuse from sylvian fissure
5. Others: vertebral artery, basilar artery…
   -> Difuse from foramen magnum



⚠ An aneurysm located in a more distal segment of the artery
-> consider infected aneurysm

If SAH is accompanied by ICH, the location of ICH can help predict the location of the ruptured aneurysm.

e.g. ICH in frontal lobe -> aneurysm in ACoA; temporal lobe -> ICA/PCoA

Primary SAH Caused by Aneurysm

Sudden severe headache, vomiting, conscious change

Suspected subarachnoid hemorrhage (SAH)

↓

Non-contrast brain CT

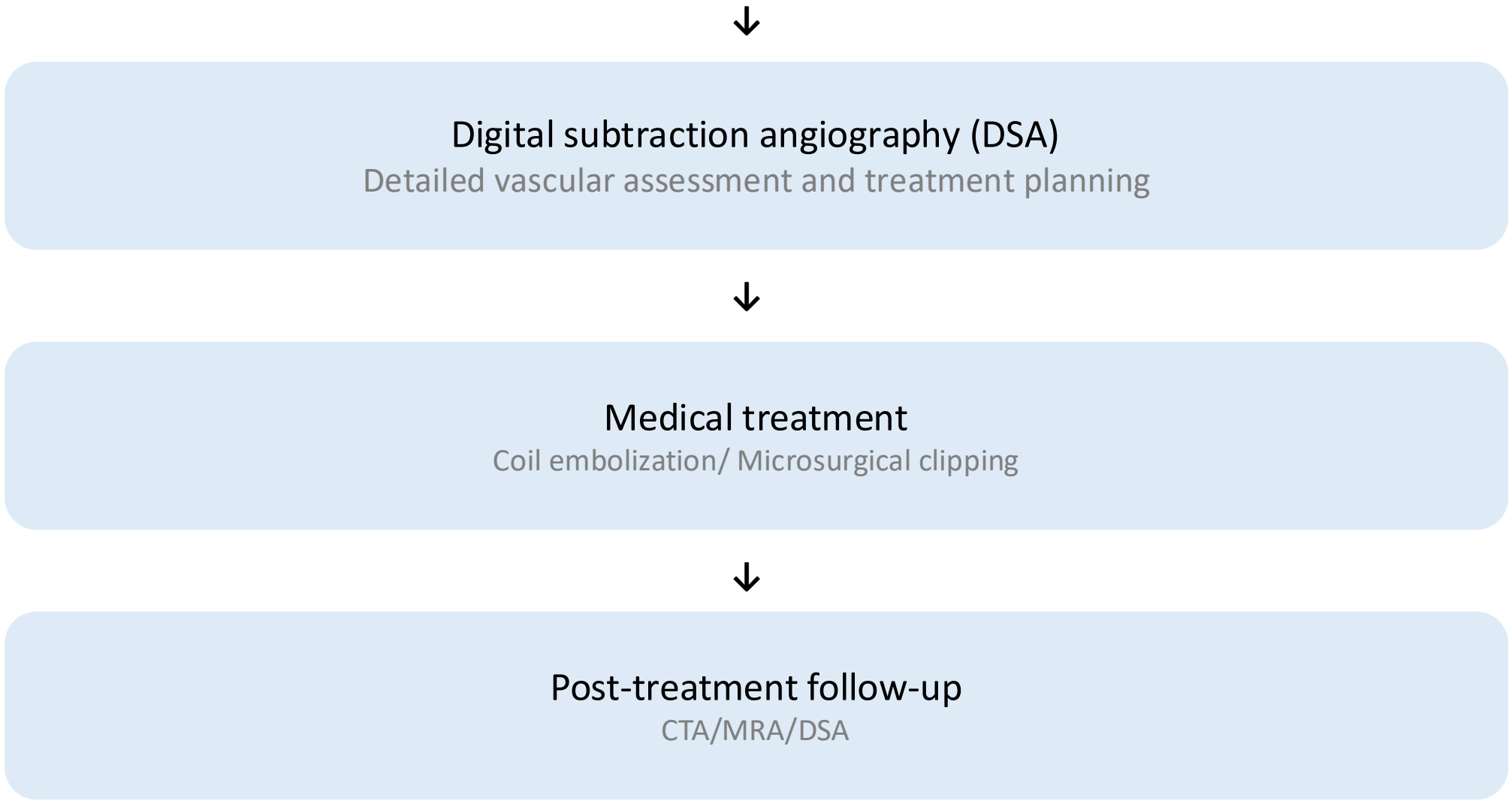
Confirm the presence of SAH

↓

CT angiography (CTA)

Identify the aneurysm and assess its location and morphology

↓


Digital subtraction angiography (DSA)
Detailed vascular assessment and treatment planning
Medical treatment
Coil embolization/ Microsurgical clipping
Post-treatment follow-up
CTA/MRA/DSA

References

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