Subarachnoid haemorrhage

Epidemiology of subarachnoid haemorrhage (SAH) in the UK

- Definition and scope:
  - Epidemiological data typically refer to spontaneous SAH, most commonly due to ruptured intracranial aneurysm; traumatic SAH is more common on CT in head-injury populations but is considered separately.
  - Non-aneurysmal perimesencephalic SAH accounts for roughly 10–15% of spontaneous SAH and has a distinct, more benign course.
- Incidence and burden:
  - Annual incidence in the UK is approximately 6–9 per 100,000 person-years, equating to around 6,000–8,000 new cases per year.
  - SAH accounts for about 5% of all strokes but a disproportionate share of stroke-related years of life lost due to younger age at onset.
- Demographics:
  - Age: peak incidence in middle age (approximately 45–60 years); median age at presentation is in the early-to-mid 50s.
  - Sex: higher in women (female-to-male ratio roughly 1.3–1.6:1), with the sex gap widening after menopause.
- Outcomes:
  - Case fatality remains high despite improvements in care: about one-third die within 30 days; 10–15% die before reaching hospital.
  - Among survivors, long-term neurocognitive and functional impairment is common, contributing substantially to societal and caregiver burden.
- Risk factors and population patterns:
  - Strong modifiable risks: cigarette smoking, hypertension, and heavy alcohol intake; additional risks include cocaine/amphetamine use.
  - Non-modifiable risks: family history (first-degree relatives have ~3–5× higher risk), certain heritable arteriopathies (e.g., ADPKD, connective tissue disorders), and female sex.
  - Socioeconomic gradients are observed in the UK, with higher incidence in more deprived areas—partly reflecting the distribution of vascular risk factors.
  - Incidental unruptured intracranial aneurysms are found in roughly 2–3% of the general population; most never rupture, but risk rises with aneurysm size, posterior circulation location, smoking, and hypertension.
- Temporal trends:
  - In high-income settings including the UK, aneurysmal SAH incidence has shown a gradual decline over the past two decades, likely linked to reductions in smoking and improved blood pressure control.
  - Improved pre-hospital pathways, rapid CT availability, and special-

ist neurovascular care have reduced early rebleeding and improved survival, though overall mortality and morbidity remain substantial.

- Radiology-specific implications:
  - High clinical index of suspicion and early non-contrast CT (ideally within 6 hours of ictus) underpin case ascertainment and affect measured incidence.
  - Negative CT with persistent clinical concern drives lumbar puncture and/or CTA/MRA pathways; perimesencephalic SAH and angiography-negative SAH comprise a meaningful minority of cases.
  - The background prevalence of incidental aneurysms means radiologists frequently adjudicate rupture likelihood and surveillance versus treatment decisions in a screening or work-up context. ## Pathophysiology of Subarachnoid Haemorrhage (UK context)

Subarachnoid haemorrhage (SAH) refers to bleeding into the cerebrospinal fluid (CSF) within the subarachnoid space. In the UK, most non-traumatic SAH is aneurysmal (~80–85%), with non-aneurysmal perimesencephalic SAH (PMSAH) accounting for ~10% and other vascular lesions (e.g., arteriovenous malformations, dissections) comprising the remainder. Traumatic SAH is common in emergency presentations but has distinct mechanisms and clinical trajectories.

Aneurysm formation and rupture - Haemodynamic stress: Saccular aneurysms arise at arterial branch points (e.g., anterior communicating, posterior communicating, MCA bifurcation, basilar tip) where pulsatile flow and wall shear stress induce endothelial dysfunction and remodelling. - Wall degeneration: Chronic hypertension and smoking (prevalent UK risk factors) drive inflammatory infiltration, extracellular matrix degradation via matrix metalloproteinases, and elastin/collagen loss, weakening the aneurysm dome. - Rupture mechanics: Sudden blood pressure surges and cyclical stress lead to focal wall failure. Intraluminal thrombus and uneven wall thickness exacerbate susceptibility.

Immediate intracranial events after rupture (early brain injury, minutes to 72 hours) - ICP surge and global ischaemia: Rapid cisternal blood release acutely elevates intracranial pressure (ICP), decreasing cerebral perfusion pressure and cerebral blood flow. Brief loss of consciousness is common. - Subarachnoid blood distribution: High-attenuation blood tracks within basal cisterns and fissures. Pattern often reflects the source: - ACom/anterior circulation: suprasellar cistern, interhemispheric fissure. - MCA: Sylvian fissure dominance. - PCom/ICA: suprasellar and ambient cisterns. - Basilar apex/vertebrobasilar: interpeduncular, prepontine cisterns. - Blood–brain barrier (BBB) disruption: Mechanical and biochemical injury increases permeability, causing vasogenic oedema; concurrent ischaemia produces cytotoxic oedema. - Catecholamine surge: Sympathetic storm can cause myocardial stunning and neurogenic pulmonary oedema, relevant to peri-imaging haemodynamics.

Biochemical toxicity of blood in CSF - Oxyhaemoglobin-mediated effects: Scavenges nitric oxide (NO), reducing vasodilation; generates reactive oxygen species, driving lipid peroxidation and endothelial injury. - Endothelin-1 upregulation

and calcium influx: Potent, sustained vasoconstriction of large and small cerebral arteries. - Inflammation: Microglial activation, cytokines (e.g., IL-1, TNF-), complement activation, and TLR4 signalling amplify endothelial dysfunction and BBB breakdown. - Coagulation and microthrombosis: Platelet activation and microthrombi impair microcirculatory flow independent of large-vessel changes. - Erythrocyte lysis and bilirubin formation: Over hours to days, haemoglobin breakdown produces xanthochromia in CSF; radiologically, evolving density of cisternal blood and dependent layering may be seen.

Delayed cerebral ischaemia (DCI) and vasospasm (classically days 3–14) - Large-vessel vasospasm: Smooth muscle contraction in proximal arteries (detectable by CTA/DSA; TCD velocities may be used in UK units). Risk correlates with subarachnoid clot burden and intraventricular haemorrhage. - Microvascular dysfunction: Endothelial swelling, perivascular inflammation, and microthrombi reduce capillary perfusion even without angiographic spasm. - Cortical spreading depolarisations: Energy-demanding waves in vulnerable cortex exacerbate mismatch between supply and demand, contributing to DCI. - Clinical–radiological correlation: The modified Fisher scale (widely used in UK practice) grades cisternal and intraventricular blood to estimate DCI risk; larger, thicker cisternal clots and IVH confer higher risk.

Hydrocephalus - Acute communicating hydrocephalus: Fibrin and blood obstruct arachnoid villi and basal cistern CSF pathways, elevating ventricular pressure and size; periventricular interstitial oedema may be seen. Early external ventricular drainage is often required. - Chronic communicating hydrocephalus: Impaired CSF resorption leads to later ventricular enlargement with gait/cognitive changes; more frequent with heavy cisternal blood.

Non-aneurysmal perimesencephalic SAH (PMSAH) - Likely venous source within the interpeduncular/prepontine cisterns; limited blood load, minimal IVH. - Distinct pathobiology: Low inflammatory burden and minimal NO scavenging lessen vasospasm and DCI risk; hydrocephalus is uncommon and usually transient. - Imaging hallmark: Prepontine/interpeduncular clot centred around the midbrain without extensive sylvian or interhemispheric spread.

Systemic and metabolic sequelae relevant to imaging and care - Hyponatraemia: Cerebral salt wasting and natriuretic peptides increase renal sodium loss; can reduce intravascular volume, worsening cerebral perfusion. - Impaired autoregulation: Pressor-dependent cerebral blood flow may necessitate careful haemodynamic control during imaging and interventions.

Radiological implications for trainees - Early non-contrast CT best detects cisternal blood and guides aneurysm targeting by pattern recognition. - Clot burden predicts vasospasm/DCI risk; monitor with CTA/DSA and, where available, CT perfusion for at-risk territories. - Vigilance for hydrocephalus (ventricular enlargement, transependymal oedema) and evolving infarction is key to timely intervention.

Although the biological mechanisms are universal, the UK context emphasises

high prevalence of aneurysmal SAH among non-traumatic cases, widespread use of the modified Fisher scale for DCI risk stratification, and protocolised CTA/DSA pathways to confirm the bleeding source and monitor vasospasm. ## Imaging features of spontaneous subarachnoid haemorrhage

- First-line: non-contrast CT head (NCCT)
  - Hallmark: acute hyperdensity in subarachnoid spaces (basal cisterns, Sylvian fissures, interhemispheric fissure, cortical sulci). Typical attenuation 50–70 HU.
  - Sensitivity is time-dependent: highest within 6 hours of ictus and falls over subsequent days as blood becomes isodense.
  - Distribution patterns that suggest aetiology:
    - Aneurysmal SAH: diffuse basal cisternal blood, often prominent in suprasellar, interpeduncular, perimesencephalic and bilateral Sylvian cisterns; frequent intraventricular extension and hydrocephalus. Focal parenchymal haematoma may localize the ruptured aneurysm (e.g., MCA territory).
    - Perimesencephalic non-aneurysmal SAH (PNSAH): blood centered anterior to the brainstem (prepontine/perimesencephalic cisterns) with little or no extension into Sylvian/interhemispheric fissures, minimal IVH, and rare hydrocephalus.
    - Convexity SAH (cSAH): sulcal blood over cerebral convexities with little basal cisternal involvement; consider reversible cerebral vasoconstriction syndrome, cerebral amyloid angiopathy, coagulopathy, venous sinus thrombosis.
  - Complications on CT: acute hydrocephalus (ventricular dilatation, transependymal oedema), intraventricular haemorrhage, intraparenchymal haematoma, early infarction (hypodensity) and mass effect/herniation.
  - Grading on CT:
    - Fisher and modified Fisher scales estimate vasospasm risk based on cisternal clot thickness and presence of intraventricular blood.
    - Hijdra score (optional) semiquantitatively scores blood in cisterns and ventricles to monitor clearance.
  - Pitfalls and mimics:
    - Pseudo-SAH appearance in severe diffuse cerebral oedema.
    - Intrathecal contrast (post–angiography/myelography) and meningitis can produce high-attenuation sulci.
    - Severe anaemia may decrease conspicuity; motion and beam hardening can obscure thin SAH.
- CT angiography (CTA)
  - Rapid identification of ruptured aneurysm(s): size, neck, morphology (lobulations/blebs), relation to branches.
  - Can show vasospasm (arterial narrowing) and vessel anomalies (dissections, fenestrations).
  - Helps infer rupture site by clot burden adjacency when multiple

aneurysms are present.
  - Limitations: very small aneurysms, thrombosed or blister aneurysms may be missed; motion/beam-hardening artifacts; if CTA is negative and the CT pattern is not perimesencephalic, proceed to DSA and consider repeat angiography.
- Digital subtraction angiography (DSA)
  - Reference standard for detecting aneurysms, AVMs, dural AV fistulas, vasculopathies and for endovascular therapy.
  - Assesses vasospasm dynamically and guides intra-arterial treatment.
  - "Angiogram-negative SAH": if initial DSA is negative and the bleed pattern is non-perimesencephalic or diffuse, repeat DSA (typically 7–10 days) is recommended.
- MRI and MRA
  - Useful when CT is negative but clinical suspicion remains high, or in the subacute/chronic phases.
  - Key sequences:
    * FLAIR: sulcal/cisternal hyperintensity from proteinaceous blood; very sensitive after the first 6–24 hours. Beware flow/oxygen therapy artifacts and meningitis.
    * T2*/GRE and SWI: detect paramagnetic blood products; excellent for subacute/chronic SAH and cortical superficial siderosis.
    * DWI: detects delayed cerebral ischaemia/infarcts from vasospasm.
    * T1-weighted: subacute methemoglobin may appear hyperintense.
  - MRA (TOF or contrast-enhanced): can detect many aneurysms but is generally less sensitive than CTA/DSA for small or blister lesions.
  - High-resolution vessel wall MRI (where available): enhancement of the culprit aneurysm wall may suggest the ruptured lesion among multiple aneurysms; can help differentiate vasculitis from reversible vasoconstriction.
- Follow-up and complications imaging
  - Hydrocephalus: serial CT for ventricular size; assess EVD position if inserted.
  - Vasospasm and delayed cerebral ischaemia (DCI): CTA for arterial narrowing; CT perfusion for perfusion delay (e.g., prolonged Tmax, reduced CBF); MRI DWI for infarcts.
  - Rebleeding: new or increased hyperdensity on CT; renewed sulcal FLAIR/SWI signal on MRI.
  - Cerebral oedema and herniation: mass effect, basal cistern effacement, midline shift.
- Practical reporting checklist
  - Confirm presence and distribution of SAH; estimate clot burden.
  - Note intraventricular extension, hydrocephalus and any parenchymal haematoma.
  - Comment on side/pattern most suggestive of bleeding source.

- Provide a Fisher/modified Fisher grade and any Hijdra scoring if used locally.
- On CTA/MRA/DSA: describe aneurysm presence, size, neck, morphology, and relationship to the clot; mention vasospasm or other vascular lesions.
- Recommend next steps (e.g., DSA, repeat angiography) based on pattern and findings. ## Imaging management of spontaneous subarachnoid haemorrhage

Goals: confirm SAH, identify/secure the bleeding source, stratify vasospasm/DCI risk, and monitor complications. Imaging choices should be tailored to haemorrhage pattern, clinical stability, and initial angiographic findings.

Initial imaging - Non-contrast CT (NCCT) head - First-line test; near-maximal sensitivity within 6 hours of ictus (98–100%), declining thereafter (90% at 24 h; lower beyond 72 h). - Report distribution and burden: basal cisterns, fissures, sulci, intraventricular/intraparenchymal extension; hydrocephalus; herniation. - Grade haemorrhage burden with the modified Fisher scale to estimate vasospasm risk. - If NCCT negative but clinical suspicion high - Options include lumbar puncture (for xanthochromia 6–12 h post ictus) or MRI. - MRI: FLAIR is sensitive to SAH; SWI/GRE detect blood products; be aware of FLAIR false positives (oxygen therapy, slow flow, motion).

Source detection and vascular imaging - CT angiography (CTA) head/neck - Immediate CTA is standard to detect aneurysm(s) and plan treatment. - High sensitivity for aneurysms 3 mm; assess dome-to-neck, branching, calcification/thrombus, vasospasm, and anatomical variants. - If CTA shows a ruptured aneurysm: proceed to securing (endovascular or surgical) without delay; additional DSA may be performed for treatment planning. - Digital subtraction angiography (DSA) - Gold standard for aneurysm detection and treatment; use 3D rotational angiography. - Indicated when: - CTA is negative or equivocal in a diffuse/aneurysmal SAH pattern. - Small/blister/dissecting aneurysm is suspected. - Non-aneurysmal vascular causes are possible (AVM, dural AVF, vasculitis, RCVS). - If initial DSA is negative but the pattern is non-perimesencephalic or diffuse, repeat DSA at 7–14 days (yield 5–15%). - Pattern-specific considerations - Perimesencephalic SAH (PM-SAH): haemorrhage centred around the prepontine/interpeduncular cisterns, minimal lateral extension. - If high-quality CTA is negative, many centres forgo immediate DSA; obtain delayed vascular imaging (CTA or DSA) in 1–2 weeks to be certain. - Convexity (cortical) SAH: - Consider cerebral amyloid angiopathy (older patients), reversible cerebral vasoconstriction syndrome (RCVS), cerebral venous sinus thrombosis (CVST), vasculitis, and dissection. - Perform CTA/MRA of head/neck and venous imaging (CTV or MRV). Vessel wall MRI can help (vasculitis shows concentric enhancement; RCVS typically shows minimal or no wall enhancement). - Non-localising/diffuse aneurysmal pattern: pursue DSA even if CTA is negative.

Imaging surveillance for complications - Hydrocephalus - Serial NCCT to assess ventricular size, transependymal oedema; confirm/monitor EVD position and catheter-related haemorrhage. - Vasospasm and delayed cerebral ischaemia (DCI) [typically days 3–14] - Clinical monitoring ± transcranial Doppler (TCD) daily where available. - CTA to depict luminal narrowing if neurological decline or high TCD velocities. - CT perfusion (CTP) to detect tissue-level hypoperfusion (prolonged Tmax/MTT, reduced CBF); guides haemodynamic and endovascular therapy. - MRI with diffusion/perfusion when stable enough to transport, to confirm infarction or reversible hypoperfusion. - Post-treatment assessment - After coiling: catheter angiography confirms occlusion class; NCCT for complications. - After clipping: CTA or DSA if concern for residual neck; CT to assess for infarct/haematoma. - Surveillance for coiled aneurysms: centre-dependent; commonly MRA or DSA at 6–12 months, then tailored by occlusion class and risk factors.

Angionegative SAH work-up (no aneurysm on initial CTA/DSA) - Reassess haemorrhage pattern (PM-SAH vs non-PM vs convexity); this drives further imaging. - If non-PM/diffuse: repeat DSA at 7–14 days. - If convexity SAH: - CTV/MRV for CVST; MRA/CTA for vasculopathy; vessel wall MRI if vasculitis suspected; screen for RCVS with repeat CTA/MRA in 1–2 weeks to document reversibility. - If persistent uncertainty and focal spinal symptoms: MRI of the spine to exclude spinal vascular malformations or tumours.

Reporting checklist for trainees - NCCT: distribution and burden of SAH; modified Fisher grade; intraventricular/intraparenchymal components; hydrocephalus; herniation; infarcts. - CTA/DSA: - Aneurysm: location, size (maximal dome, neck, dome–neck ratio), morphology (blister, dissecting, fusiform), branch involvement, thrombus/calcification, multiplicity. - Alternative causes: AVM/dAVF, vasculitis/RCVS features, dissection, mycotic aneurysm. - Vasospasm: arterial segments involved; severity (mild/moderate/severe); distal vs proximal predominance. - Devices/therapy: EVD position; stents/coils/clips; complications. - Follow-up: recommend appropriate interval imaging based on pattern and findings.

Common pitfalls - Missing small blister aneurysms (supraclinoid ICA, ACom, PCom, PICA) on CTA; use high-quality CTA, thin slices, and DSA with 3D runs if suspicion persists. - Assuming PM-SAH without carefully excluding lateral/sylvian or diffuse extension. - Over-calling SAH on FLAIR due to artefact; corroborate with SWI/NCCT and clinical context. - Neglecting venous imaging in convexity SAH. - Underestimating vasospasm when relying on lumenography alone; add CTP when clinical-radiological mismatch exists.

Suggested workflow (summary) - Suspected SAH → NCCT. - Positive → CTA immediately. - Aneurysm found → secure aneurysm; baseline NCCT; plan vasospasm surveillance (TCD ± CTA/CTP as indicated). - CTA negative: - PM-SAH pattern → consider no immediate DSA; delayed CTA/DSA at 1–2 weeks. - Non-PM/diffuse pattern → proceed to DSA; if negative, repeat DSA at 7–14 days. - Convexity SAH → CTV/MRV + vasculopathy work-up; con-

sider vessel wall MRI; repeat vascular imaging to assess reversibility (RCVS). - Negative NCCT but high suspicion → LP for xanthochromia and/or MRI; if SAH confirmed, proceed as above.

Notes - Early aneurysm securing (ideally within 24 hours) reduces rebleeding; coordinate imaging to avoid delaying therapy. - Minimise cumulative contrast dose across serial CTA/DSA, especially in renal impairment. - Align with local protocols and current guidelines (e.g., AHA/ASA 2023) and discuss equivocal cases in multidisciplinary neurovascular conference. # Suggested figures and captions for "Subarachnoid haemorrhage" (trainee radiologist textbook)

Epidemiology and burden

1) Figure 1. Epidemiology snapshot of SAH in the UK

- Panel A: UK map heat-map of annual incidence (6–9 per 100,000 person-years).
- Panel B: Age–sex incidence curves showing peak in early-to-mid 50s and higher female rates post-menopause.
- Panel C: Pie chart of spontaneous SAH aetiologies: aneurysmal (~80–85%), perimesencephalic (~10–15%), other (~5%). Caption: SAH accounts for ~5% of strokes but a disproportionate burden of years of life lost. Incidence has declined modestly with reduced smoking and better BP control.

2) Figure 2. Population risk and aneurysm prevalence

- Panel A: Bar chart of key modifiable risks (smoking, hypertension, heavy alcohol) and their relative contributions.
- Panel B: Schematic showing incidental aneurysm prevalence (2–3%) and factors increasing rupture risk (size, posterior circulation, smoking, hypertension). Caption: Modifiable risk factors dominate at a population level; incidental aneurysms are common but most never rupture.

Pathophysiology and timelines

3) Figure 3. Aneurysm formation and rupture mechanics

- Annotated diagram of a saccular aneurysm at a branch point with flow streamlines, wall shear stress, inflammatory infiltration, elastin loss, and focal dome thinning. Caption: Endothelial injury, matrix degradation, and haemodynamic stress interact to produce wall failure at aneurysm domes.

4) Figure 4. Early brain injury and delayed complications timeline

- Horizontal timeline (minutes → days → weeks) mapping: ICP surge/global ischaemia (minutes–hours), BBB disruption/oedema (hours–days), vasospasm/DCI window (days 3–14), hydrocephalus (early

and chronic). Caption: The biological cascade after rupture underpins imaging targets: detect blood early, anticipate vasospasm, and monitor hydrocephalus and infarction.

Core CT and MRI appearances

5) Figure 5. NCCT essentials: recognising SAH

- Panel A: Aneurysmal SAH with dense basal cisternal blood (suprasellar, interpeduncular, bilateral Sylvian).
- Panel B: Intraventricular extension with early hydrocephalus (ventricular enlargement, transependymal oedema).
- Panel C: Density measurement (50–70 HU) within cisterns; tip on narrow windowing to accentuate subtle sulcal blood. Caption: Non-contrast CT is most sensitive within 6 hours; report distribution, density, IVH, and hydrocephalus.

6) Figure 6. CT distribution patterns that suggest source

- Panel A: Anterior communicating aneurysm pattern—blood in suprasellar cistern and interhemispheric fissure.
- Panel B: MCA aneurysm—dominant Sylvian fissure blood ± adjacent parenchymal haematoma.
- Panel C: PCom/ICA—suprasellar/ambient cisterns.
- Panel D: Basilar apex—interpeduncular/prepontine cisterns. Caption: Pattern recognition can localise the likely ruptured aneurysm and guide targeted angiography.

7) Figure 7. Perimesencephalic SAH (PMSAH) on NCCT

- Axial images centred on prepontine/interpeduncular cisterns with minimal extension to Sylvian/interhemispheric fissures and little/no IVH. Caption: PMSAH has a benign course with low DCI risk; a negative high-quality CTA may obviate immediate DSA.

8) Figure 8. Convexity SAH (cSAH) patterns and differentials

- Panel A: cSAH from reversible cerebral vasoconstriction syndrome—sulcal blood over high convexity; inset CTA with multifocal "beading."
- Panel B: cSAH in probable cerebral amyloid angiopathy—superficial siderosis on SWI with recent sulcal FLAIR hyperintensity.
- Panel C: cSAH from cerebral venous sinus thrombosis—FLAIR sulcal hyperintensity plus MRV showing superior sagittal sinus thrombosis. Caption: Convexity-predominant SAH prompts evaluation for RCVS, CAA, and venous thrombosis.

9) Figure 9. MRI in SAH

- Panel A: FLAIR hyperintensity in basal cisterns (subacute phase).
- Panel B: SWI demonstrating cortical superficial siderosis.

- Panel C: DWI showing territorial infarcts from DCI. Caption: MRI complements CT when presentation is delayed or CT is negative; SWI is sensitive to chronic blood products.

10) Figure 10. CT sensitivity vs time from ictus

- Line graph of NCCT sensitivity: ~98–100% at 6 h, ~90% at 24 h, falling thereafter; overlay of LP xanthochromia window (6–12 h). Caption: Time from ictus informs test choice—early CT is best; beyond the first day, LP/MRI can aid diagnosis.

Grading and risk stratification

11) Figure 11. Modified Fisher scale (worked examples)

- Four axial NCCT panels illustrating grades 0–4 with callouts: cisternal clot thickness and IVH presence. Caption: Modified Fisher grading predicts DCI risk more accurately than the original Fisher scale by incorporating IVH.

12) Figure 12. Hijdra scoring demonstration

- Template showing cisternal and ventricular regions with example scores overlayed on NCCT. Caption: Hijdra score provides a semiquantitative measure for clot burden and clearance over time.

Vascular imaging and source identification

13) Figure 13. CTA of ruptured aneurysm

- Panel A: ACom aneurysm with lobulation/bleb (MIP/VR reconstructions).
- Panel B: Clot adjacency map on NCCT correlating with aneurysm location. Caption: CTA rapidly identifies aneurysm morphology and supports rupture adjudication when multiple aneurysms are present.

14) Figure 14. DSA for difficult lesions

- Panel A: Blister aneurysm of the supraclinoid ICA missed on CTA, seen on 3D rotational DSA.
- Panel B: Dissecting PICA aneurysm with irregular tapering. Caption: DSA remains the reference standard for small, blister, or dissecting aneurysms and for endovascular therapy.

15) Figure 15. Vessel wall MRI to identify the culprit

- High-resolution pre/post-contrast T1 images showing focal wall enhancement and thickening in the ruptured aneurysm among multiple candidates. Caption: Vessel wall enhancement may indicate instability/rupture, aiding selection of the target lesion.

Complications, surveillance, and treatment

16) Figure 16. Vasospasm and DCI: multimodal imaging

- Panel A: CTA day 7 with proximal MCA and A1 narrowing.
- Panel B: CT perfusion map with prolonged Tmax/reduced CBF in corresponding territory.
- Panel C: Follow-up MRI DWI with infarcts if therapy delayed/insufficient. Caption: Combine lumenography (CTA/DSA) with tissue perfusion (CTP) to detect treatable ischaemia in the DCI window.

17) Figure 17. Transcranial Doppler (TCD) in practice

- Graph of daily MCA mean velocities with Lindegaard ratio trend; threshold lines for mild/moderate/severe spasm. Caption: TCD offers bedside surveillance; rising velocities prompt confirmatory CTA/DSA and pre-emptive management.

18) Figure 18. Hydrocephalus on CT and EVD assessment

- Panel A: Acute communicating hydrocephalus with transependymal oedema.
- Panel B: CT showing EVD tip position in the frontal horn; small catheter-tract haemorrhage. Caption: Recognise hydrocephalus early and report device position/complications after CSF diversion.

19) Figure 19. Post-treatment imaging

- Panel A: Coiled aneurysm—early DSA occlusion class; inset NCCT excluding procedure-related haemorrhage.
- Panel B: Clipped aneurysm—CTA demonstrating clip artifact and screening for residual neck.
- Panel C: Follow-up MRA at 12 months showing stable occlusion vs small neck remnant. Caption: Tailor post-treatment surveillance to occlusion class, device type, and risk factors.

Special situations and mimics

20) Figure 20. Pseudo-SAH and other pitfalls

- Panel A: Diffuse cerebral oedema with pseudo-SAH appearance; measured low HU in "hyperdense" sulci.
- Panel B: Intrathecal contrast after angiography mimicking SAH; dual-energy CT or time course clarifies.
- Panel C: Severe anaemia reducing hyperdensity of true SAH. Caption: Avoid over- or under-calling SAH by checking HU, clinical context, and recent contrast exposure.

21) Figure 21. Angionegative SAH workflow

- Flowchart: PM-SAH pattern with negative CTA → delayed CTA/DSA at 1–2 weeks; diffuse/non-PM pattern → immediate DSA and repeat at 7–14 days if negative; convexity SAH → CTV/MRV and vasculopathy work-up. Caption: The haemorrhage pattern dictates the vascular work-up strategy and timing of repeat angiography.

22) Figure 22. RCVS reversibility

- Panel A: CTA at presentation with multifocal distal arterial narrowing.
- Panel B: Repeat CTA/MRA at 2 weeks showing resolution of narrowing. Caption: Reversibility distinguishes RCVS from vasculitis; vessel wall MRI typically shows minimal/no wall enhancement in RCVS.

Practical "how to look" and reporting

23) Figure 23. "Where to look for blood" on NCCT

- Annotated axial slices highlighting basal cisterns (suprasellar, interpeduncular, ambient), Sylvian fissures, interhemispheric fissure, posterior fossa cisterns, and dependent ventricular recesses. Caption: Systematic search improves detection of thin SAH and small IVH; adjust window width/level to highlight subtle sulcal blood.

24) Figure 24. Reporting checklist overlay

- Example NCCT/CTA with callouts aligned to a reporting template: SAH distribution, IVH, hydrocephalus, suspected source side, modified Fisher grade, aneurysm metrics (size, neck, morphology), vasospasm, and recommended next steps. Caption: Structured reporting ensures key management-critical details for the neurovascular team.

Acquisition and display tips (call-outs to accompany figures)

- NCCT: Thin slices (1 mm) reconstructed to 3–5 mm; review at standard brain windows and a narrower window (e.g., W/L ~100/40) for subtle sulcal blood.
- CTA: High-quality arterial phase with thin slices and 3D reconstructions; scrutinise common blind spots (supraclinoid ICA, ACom complex, PICA origin).
- MRI: Include FLAIR, SWI/GRE, DWI; consider high-resolution black-blood T1 post-contrast where available.
- Perfusion: Standardise maps (Tmax, MTT, CBF, CBV) and interpret alongside CTA lumenography and clinical data.

These figures collectively illustrate epidemiology, mechanistic underpinnings, characteristic imaging appearances, differential patterns, grading, vascular work-up, complications, treatment follow-up, and common pitfalls in the UK

context. Each caption is designed to reinforce actionable imaging points for trainee radiologists.