

Intracranial hemorrhage

Anatomical compartments, vascular mechanisms, morphology and clinical consequences

Epidural

Subdural

Subarachnoid

Intraparenchymal

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Learning outcomes

- Localize intracranial bleeding to the correct anatomical compartment.
- Connect hemorrhage patterns to their principal vascular lesions.
- Explain acute tissue injury and the evolution of hematomas.
- Differentiate common traumatic and spontaneous hemorrhages.

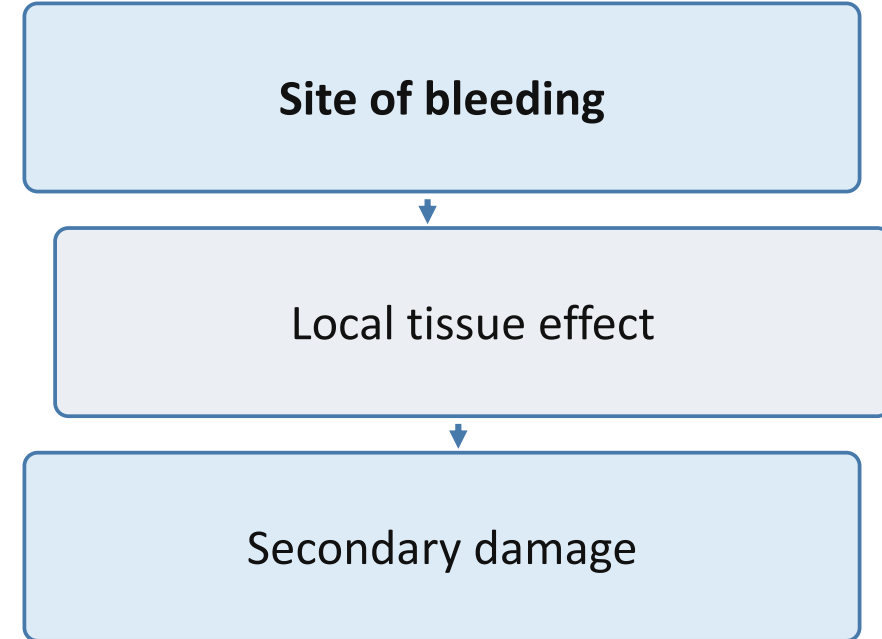
Hemorrhage can occur in four compartments

- Epidural: Between inner skull and dura.
- Subdural: Within the dural border region.
- Subarachnoid: Within CSF-filled meningeal space.
- Intraparenchymal: Within neural tissue.

Skull
Epidural interface
Dura
Subdural border
Arachnoid
Subarachnoid space (CSF)
Pia + cerebral cortex

Anatomical location determines the pathology

- Extra-axial collections primarily compress underlying brain.
- Parenchymal hematomas destroy, displace and infarct adjacent tissue.
- Subarachnoid blood contacts cerebral vessels and CSF pathways.

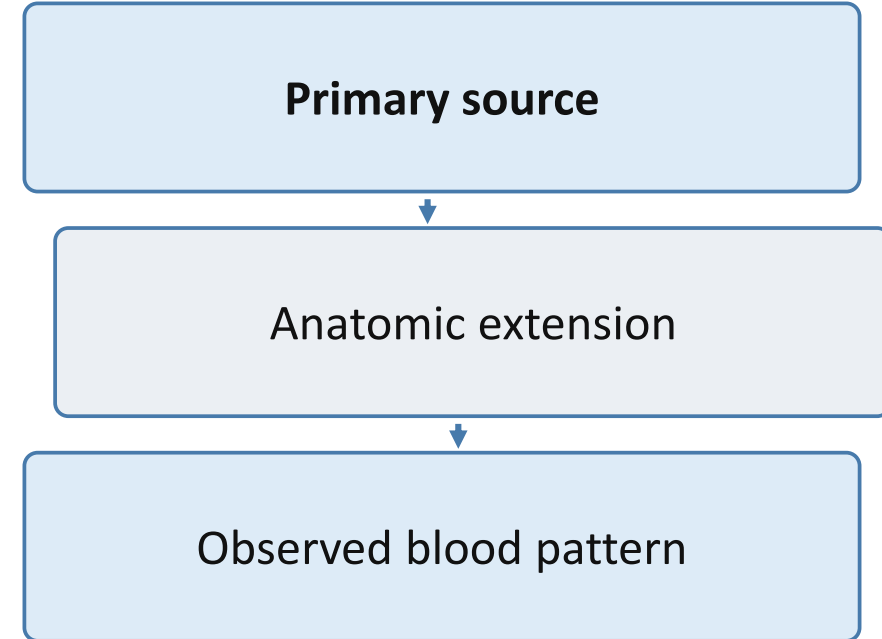


Trauma and vascular disease produce different patterns

Pattern	Typical setting	Important mechanism
Epidural / subdural	Trauma	Meningeal artery / bridging vein
Deep parenchymal	Chronic hypertension	Fragile penetrating arterioles
Lobar parenchymal	Cerebral amyloid angiopathy	Amyloid-bearing cortical vessels
Subarachnoid	Ruptured aneurysm	Failure at arterial branch point

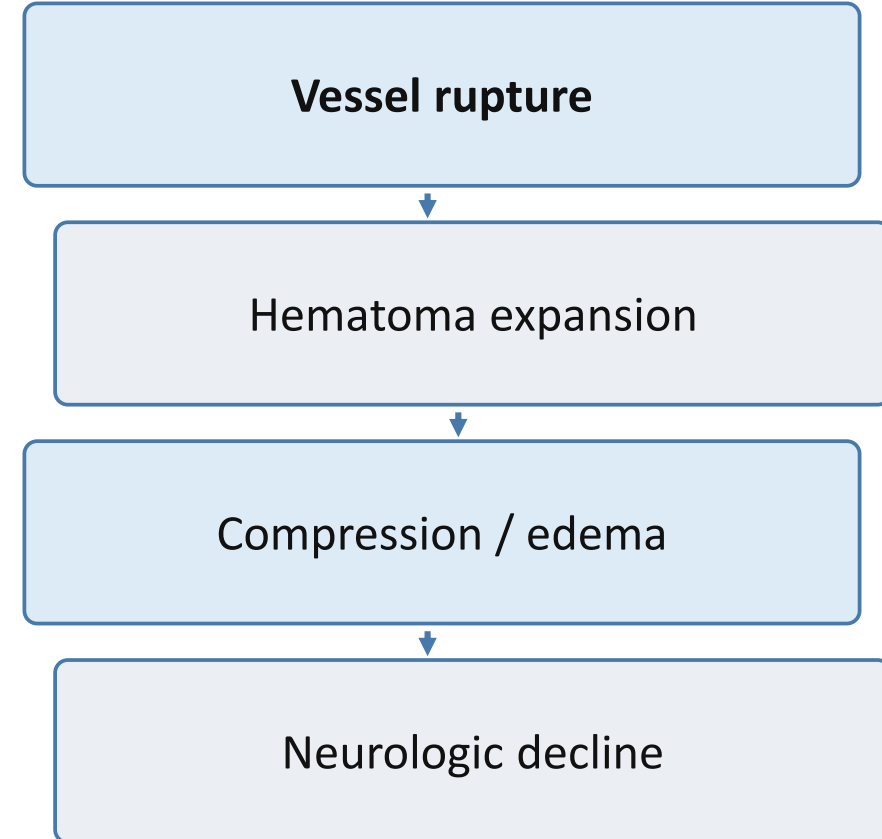
Blood distribution is not the same as bleeding origin

- A primary brain hemorrhage may enter ventricles or the subarachnoid space.
- A traumatic contusion may extend into the subarachnoid space.
- The secondary site of blood does not necessarily identify its source.



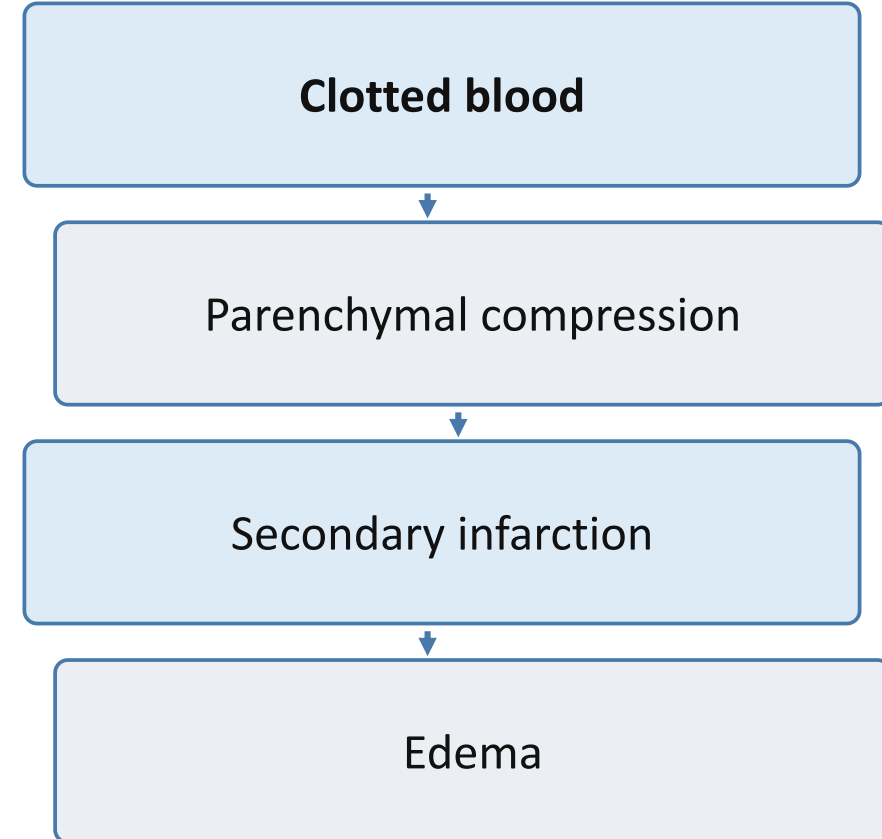
The rigid cranial cavity magnifies hemorrhagic injury

- Expanding blood collections compress adjacent tissue.
- Compression reduces local perfusion and aggravates ischemic damage.
- Large lesions can rapidly deteriorate despite an initially limited deficit.



Clot expansion and edema cause secondary tissue injury

- Accumulating blood increases pressure on nearby parenchyma.
- Associated edema intensifies tissue compression.
- Local tissue ischemia contributes to neurological dysfunction.



A location-based approach starts with three questions

- Is blood extra-axial, subarachnoid, or intraparenchymal?
- Is the likely initiating event traumatic or spontaneous?
- Does the anatomic distribution suggest a particular vessel lesion?

Where?

Compartment

Why?

Vascular or traumatic cause

**What
next?**

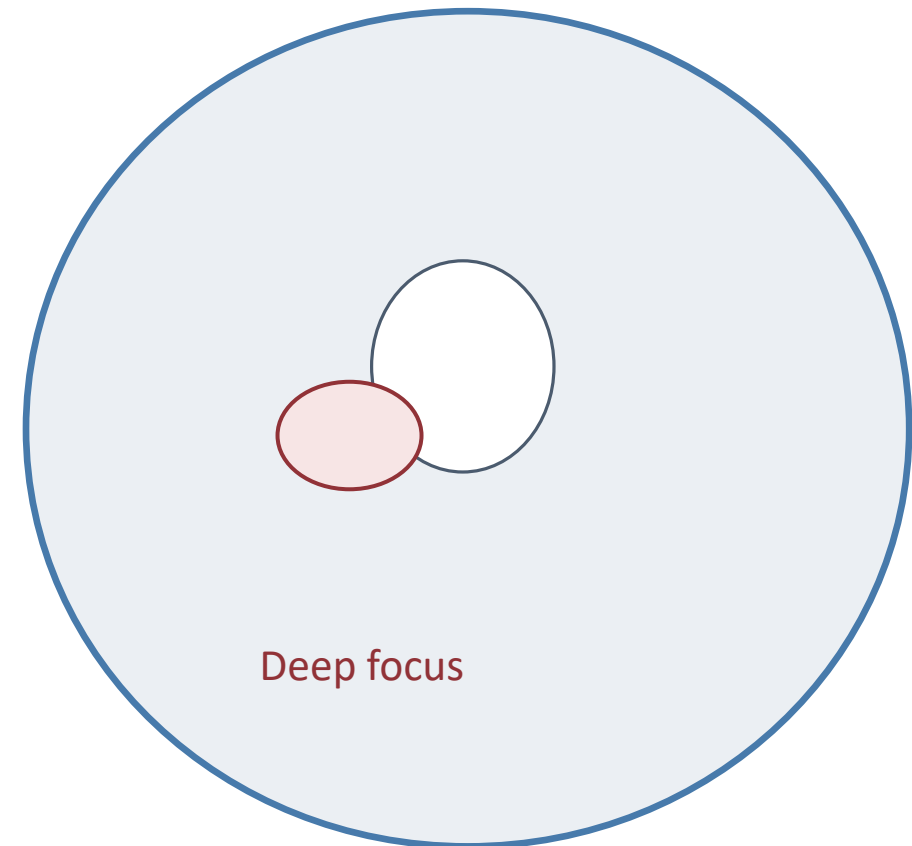
Tissue effects

Acute hemorrhage is not identical to hemorrhagic infarction

Feature	Primary hemorrhage	Hemorrhagic infarction
Initiating event	Vessel rupture	Occlusion / ischemia
Typical blood pattern	Hematoma / clot	Punctate, potentially confluent
Core tissue process	Compression + secondary infarct	Ischemic necrosis preceding bleed

Intraparenchymal hemorrhage begins inside brain tissue

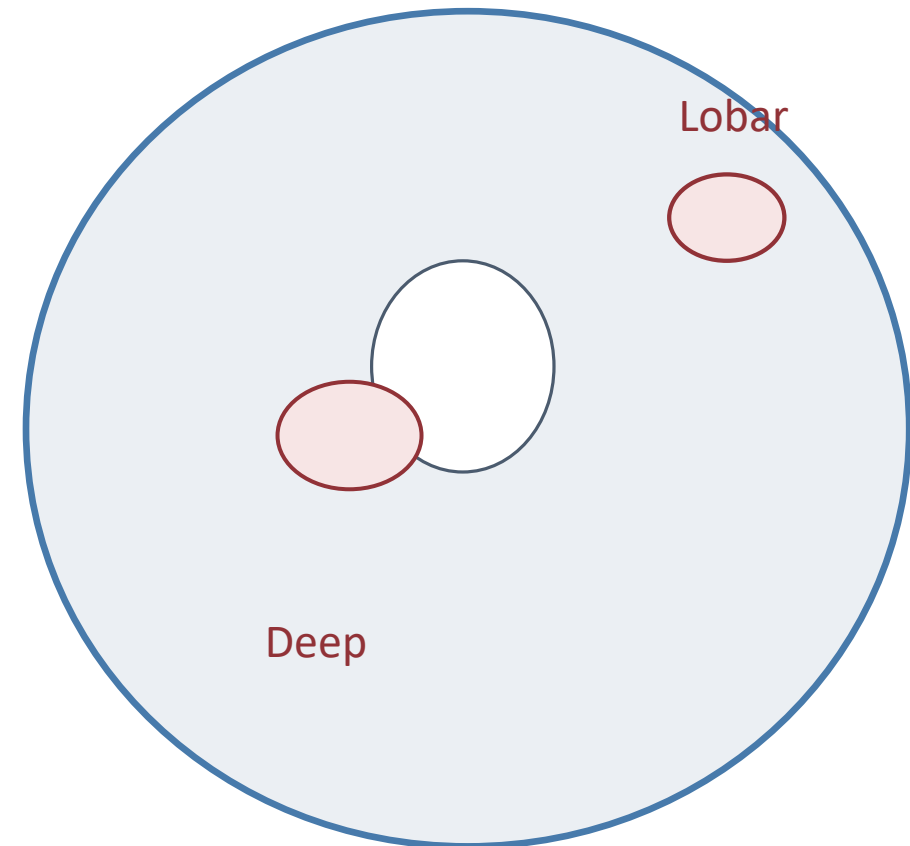
- Rupture of an intraparenchymal vessel creates a primary hematoma.
- Sudden neurological deficits often resemble other stroke syndromes.
- Anatomical site determines the observed deficits.



Schematic anatomical localization — not a specimen

Deep and lobar hemorrhages are distinct patterns

- Deep or ganglionic hemorrhage involves basal ganglia or thalamus.
- Lobar hemorrhage lies more superficially within cerebral hemispheres.
- These patterns suggest different underlying vasculopathies.



Schematic anatomical localization — not a specimen

Deep hemorrhage points toward chronic hypertension

- Hypertension is the dominant association for deep parenchymal hemorrhage.
- Typical locations include putamen, thalamus, pons and cerebellum.
- Long-standing small-artery damage makes rupture more likely.

Putamen

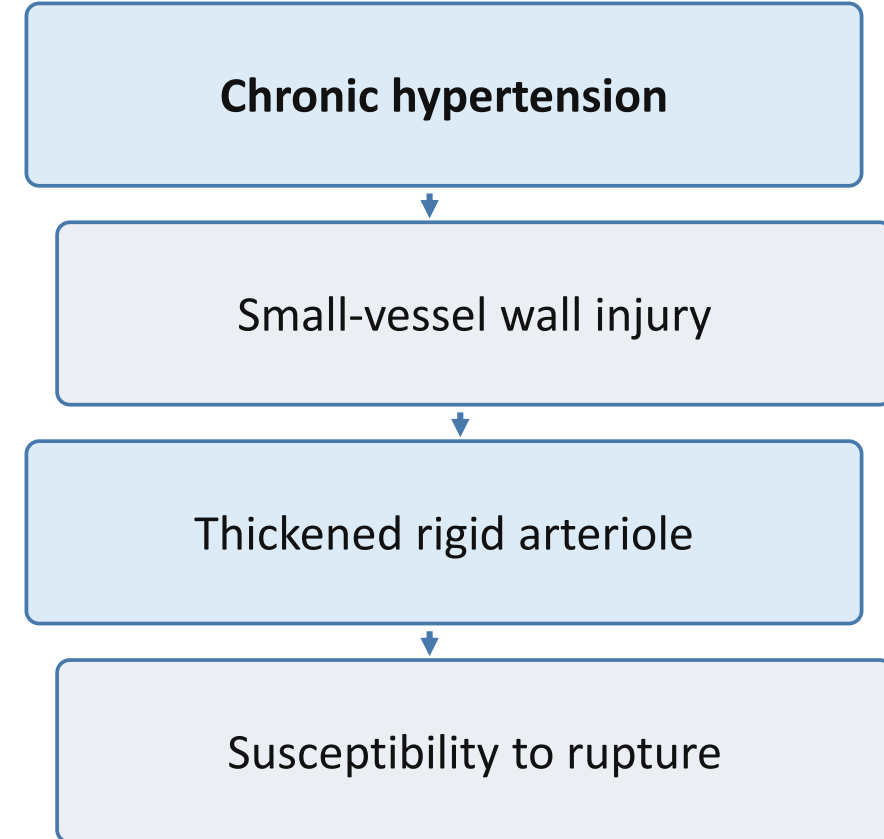
Thalamus

Pons

Cerebellum

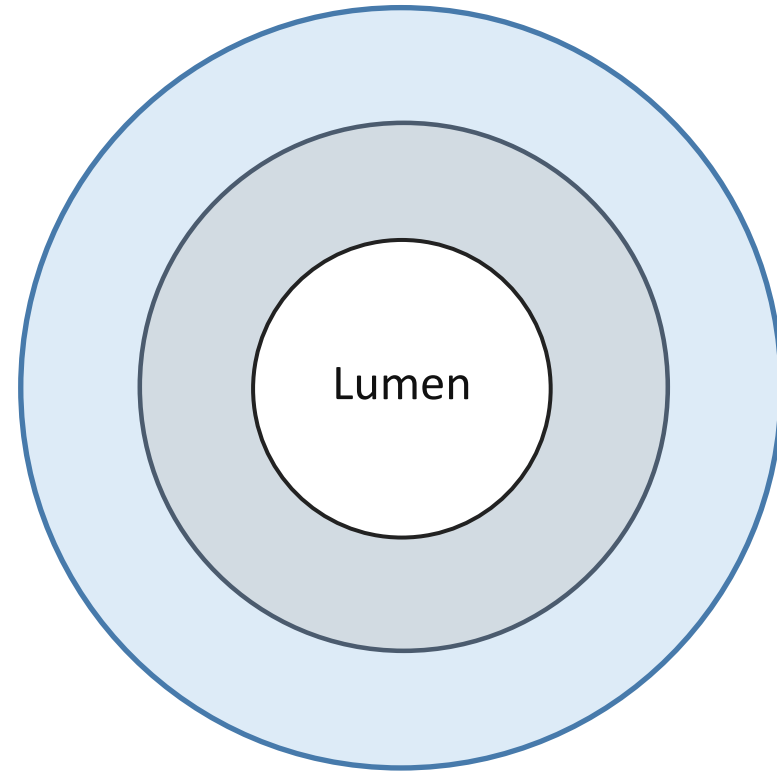
Hypertension remodels arteries at several calibers

- Large arteries can develop accelerated atherosclerosis.
- Small arteries develop hyaline arteriolosclerosis.
- Severe hypertension may produce proliferative change and arteriolar necrosis.



Hyaline arteriolosclerosis creates a rigid vessel wall

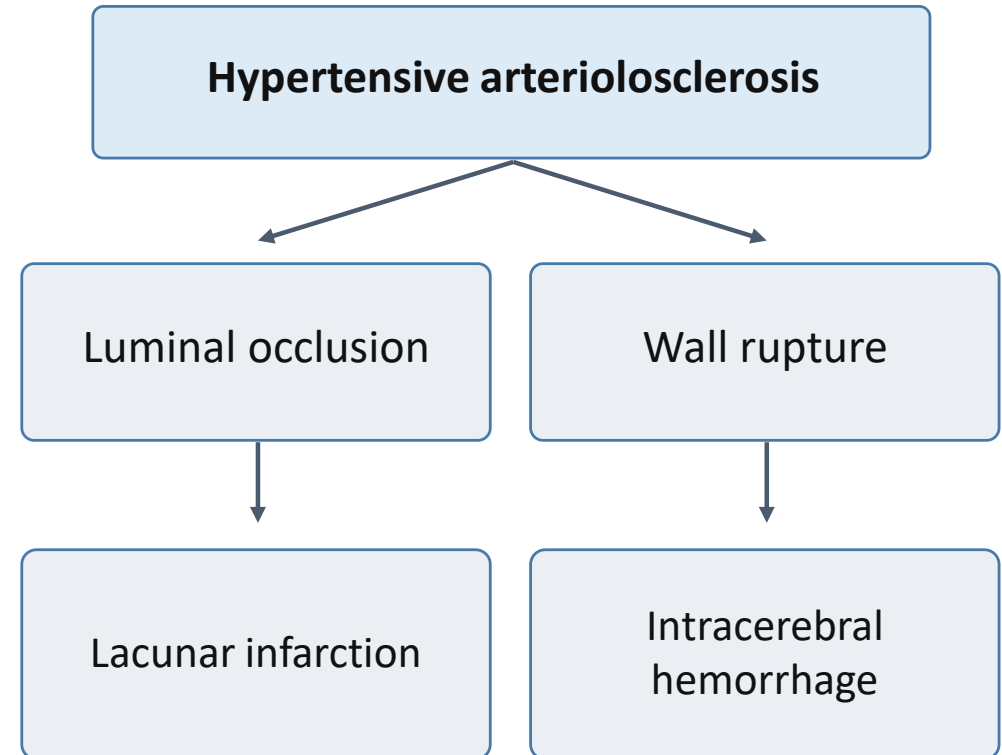
- Hyaline thickening reduces normal vessel-wall compliance.
- Affected small arteries are especially prominent in basal ganglia and white matter.
- The resulting wall is vulnerable to rupture under pressure.



Thickened vessel wall
Hyaline arteriolosclerosis

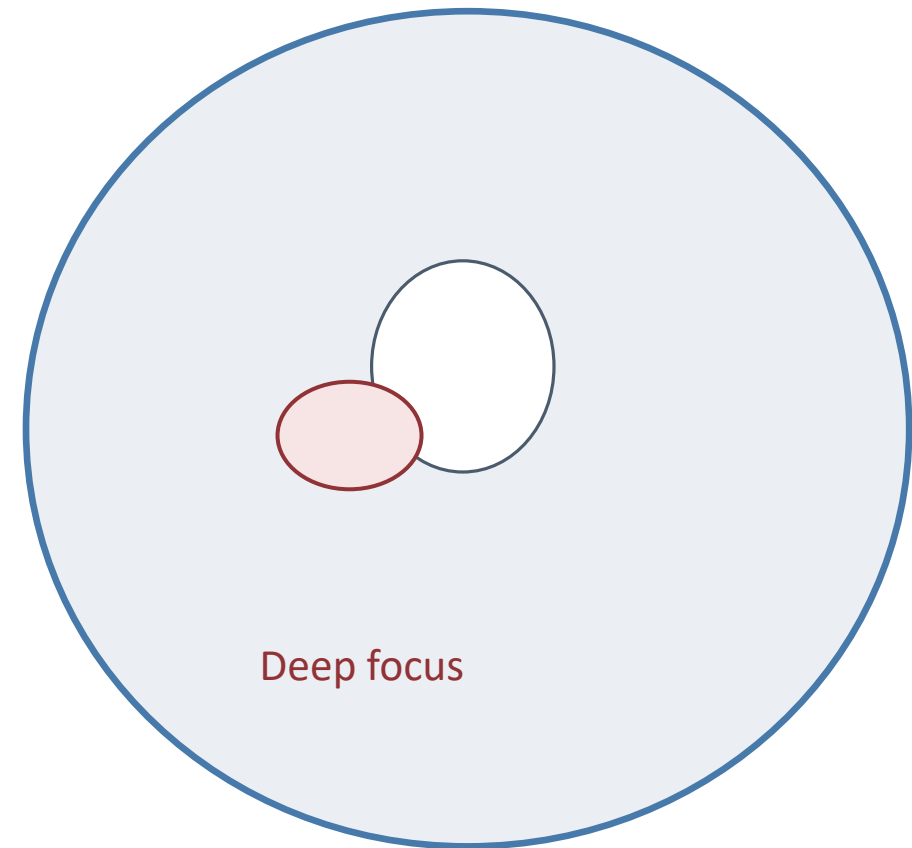
One small-vessel lesion causes two vascular outcomes

- Luminal narrowing or occlusion can produce lacunar infarction.
- Failure of the damaged vessel wall can cause parenchymal hemorrhage.
- Both outcomes can arise from hypertensive arteriolosclerosis.



The putamen is a characteristic hemorrhage site

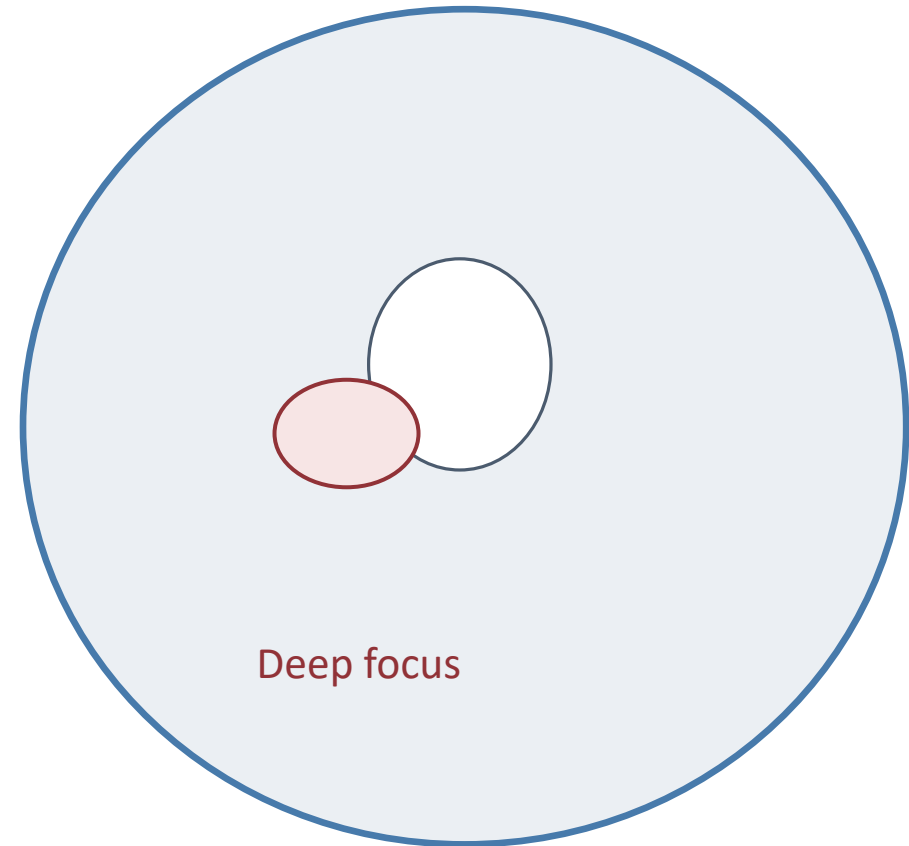
- Hypertensive hematomas frequently originate in the putamen.
- Blood may involve neighboring deep structures and white matter.
- Extension into the ventricular system can worsen the injury.



Schematic anatomical localization — not a specimen

Thalamic and brainstem lesions affect compact pathways

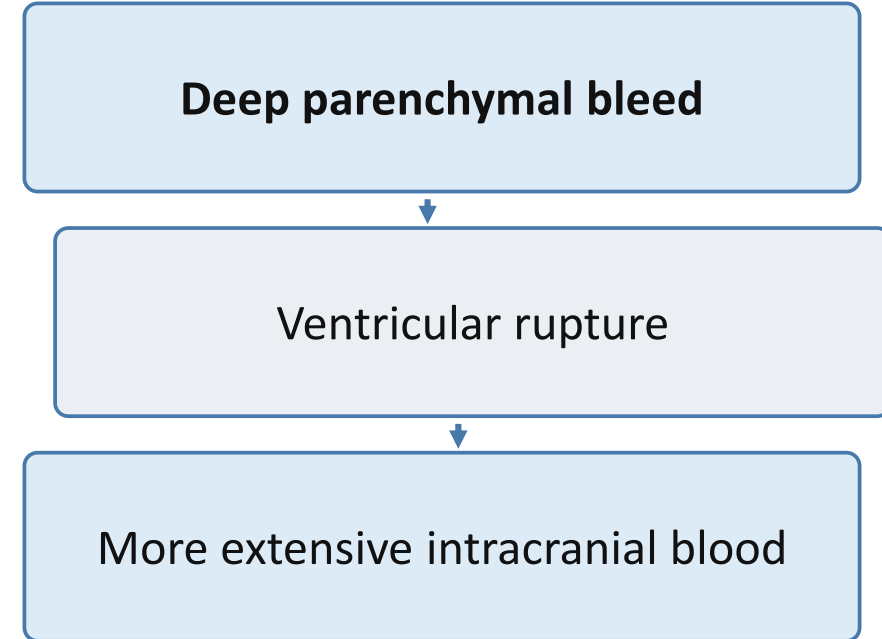
- A small deep hematoma can interrupt tightly packed circuits.
- The clinical deficit follows the neuroanatomic location, not simply clot size.
- Hypertensive hemorrhage may also involve the cerebellum and pons.



Schematic anatomical localization — not a specimen

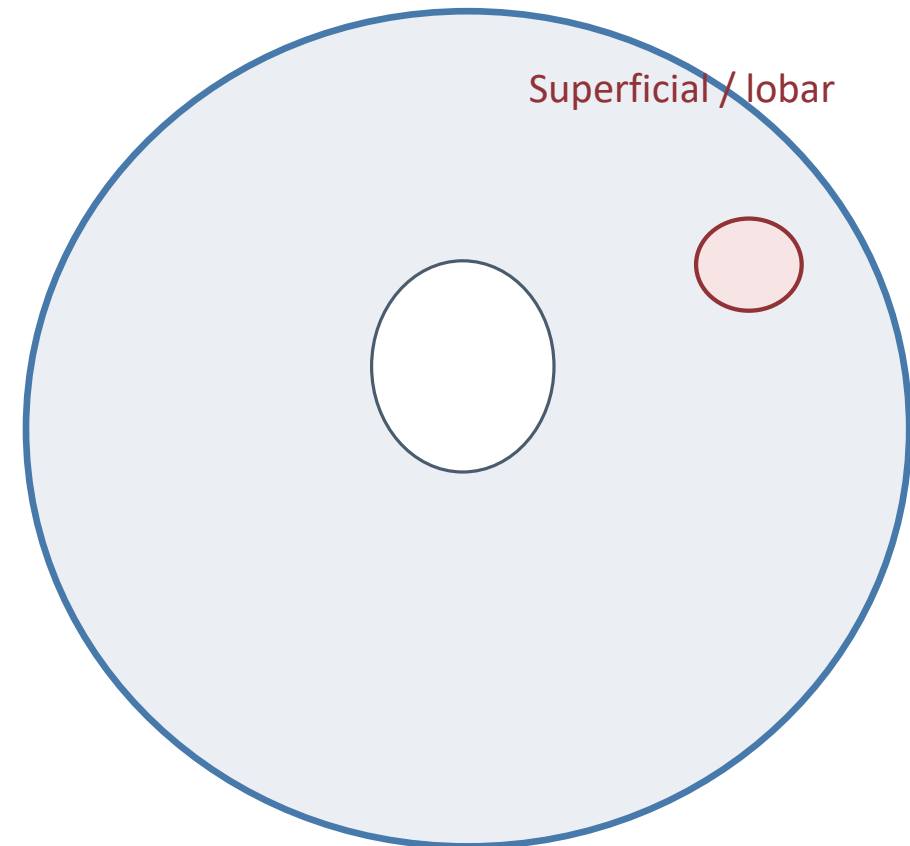
Intraventricular extension increases the affected territory

- Deep hematomas may rupture into the ventricular system.
- Ventricular blood signifies spread beyond the original parenchymal focus.
- Large or extensive hemorrhage may cause severe neurological deterioration.



Amyloid angiopathy preferentially causes lobar bleeding

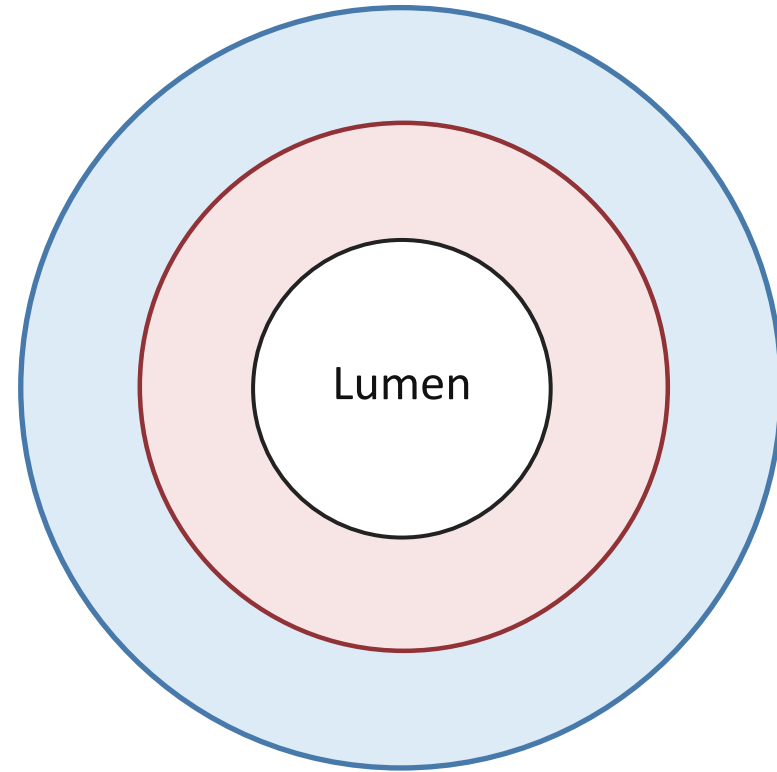
- Cerebral amyloid angiopathy deposits A β in vessel walls.
- Affected vessels are predominantly cortical and leptomeningeal.
- Vessel wall fragility favors superficial or lobar hemorrhage.



Schematic anatomical localization — not a specimen

CAA damages vessels by extracellular A β deposition

- Amyloid accumulates in small and medium meningeal and cortical vessels.
- The vessel wall becomes rigid and prone to rupture.
- H&E may resemble hyaline change, but the deposited material is A β .



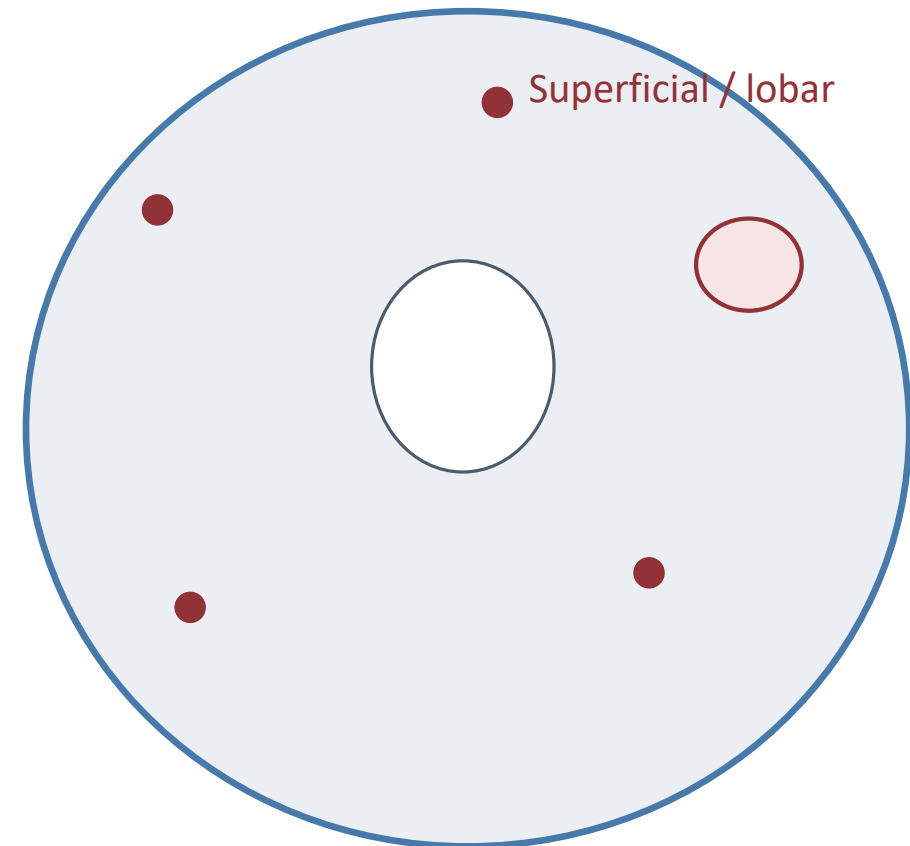
Thickened vessel wall
A β deposition

CAA and hypertension have different tissue distributions

Feature	Hypertensive arteriopathy	Cerebral amyloid angiopathy
Typical site	Deep / ganglionic	Lobar / superficial
Affected vessels	Deep small arteries	Cortical / leptomeningeal
Wall deposit	Hyaline / collagenous	A β amyloid
Other manifestation	Lacunar infarcts	Multiple small bleeds

CAA may produce multiple small hemorrhages

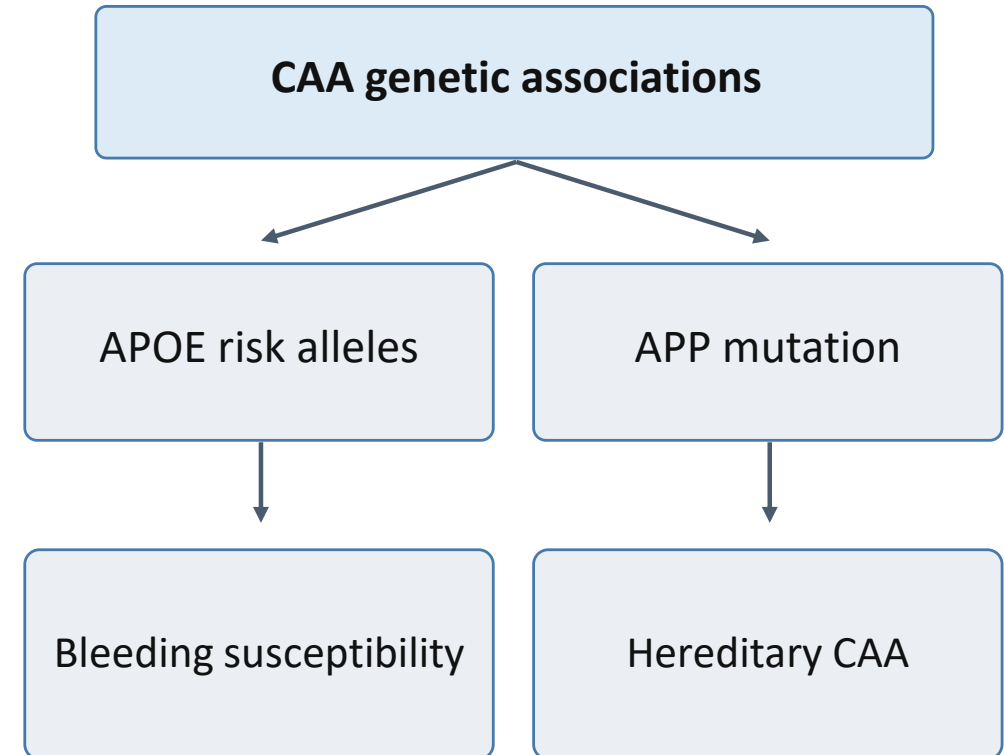
- Amyloid-containing vessel walls can fail repeatedly.
- Small cerebral microbleeds may accompany a major lobar hematoma.
- Imaging studies can reveal these small hemorrhagic foci.



Schematic anatomical localization — not a specimen

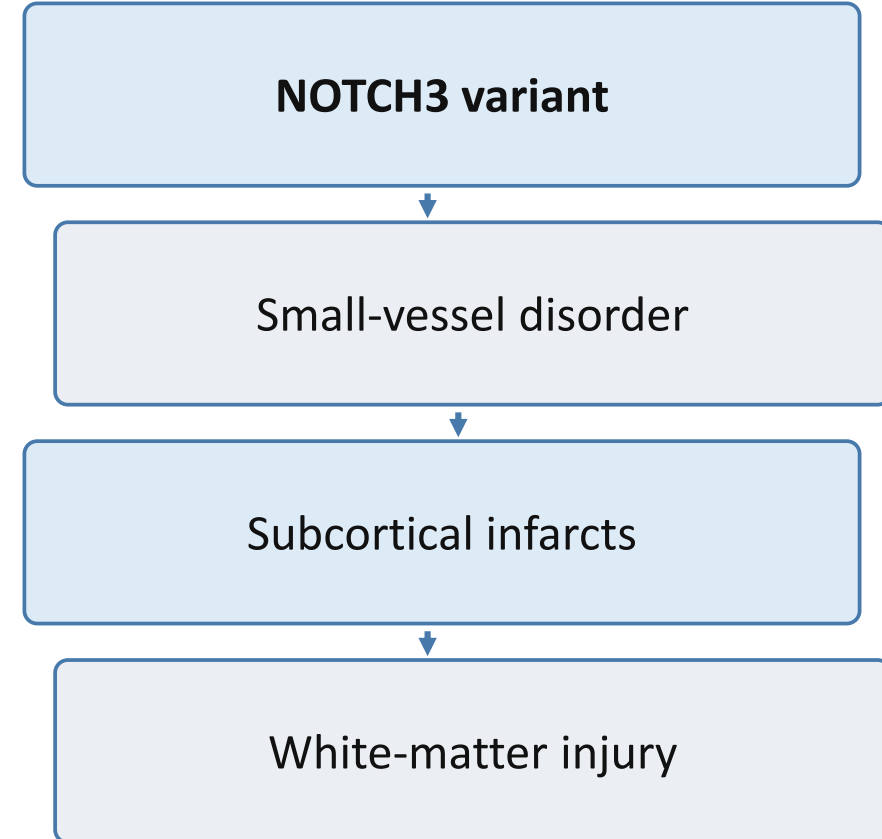
CAA-associated genetic factors modify bleeding susceptibility

- APOE $\epsilon 2$ or $\epsilon 4$ alleles are associated with increased hemorrhage risk in CAA.
- Some autosomal dominant CAA forms involve APP mutations.
- Genetic susceptibility is not equivalent to a deterministic diagnosis.



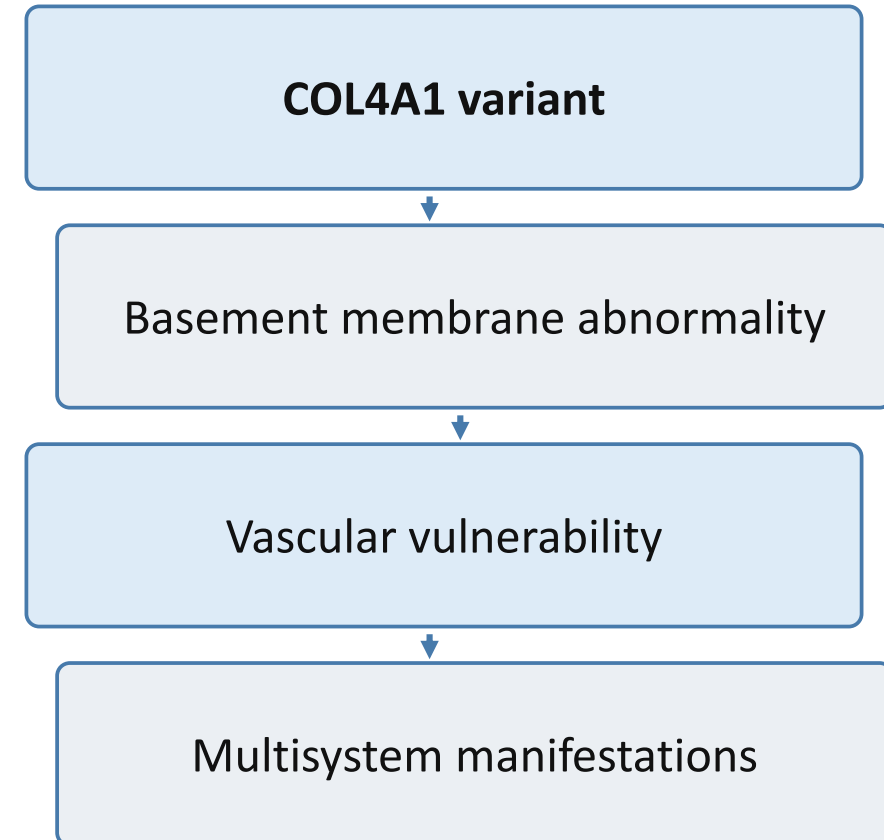
CADASIL is a contrasting hereditary small-vessel disorder

- Pathogenic NOTCH3 variants lead to abnormal extracellular receptor protein.
- Recurrent small-vessel infarcts are typical; Hemorrhage is less common.
- White-matter changes and cognitive impairment are characteristic features.



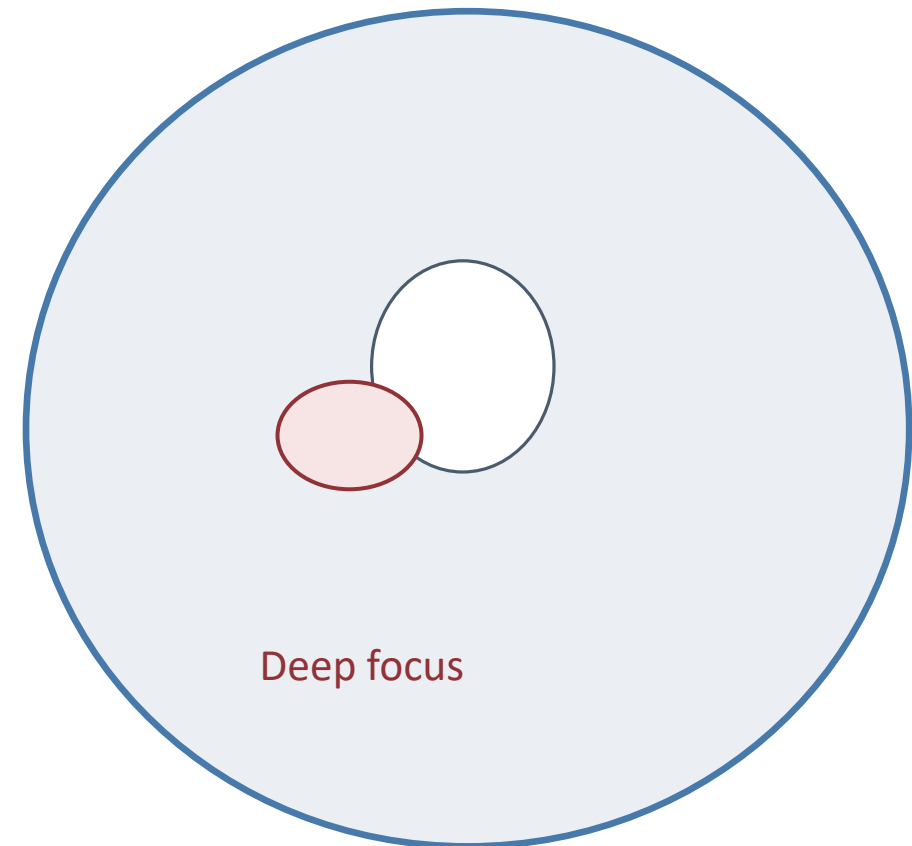
COL4A1-associated disease can include intracranial bleeding

- COL4A1 encodes a component of the vascular basement membrane.
- Inherited variants may be associated with brain hemorrhage.
- Other manifestations described include cataract, hematuria and epilepsy.



A primary hematoma has a central blood clot

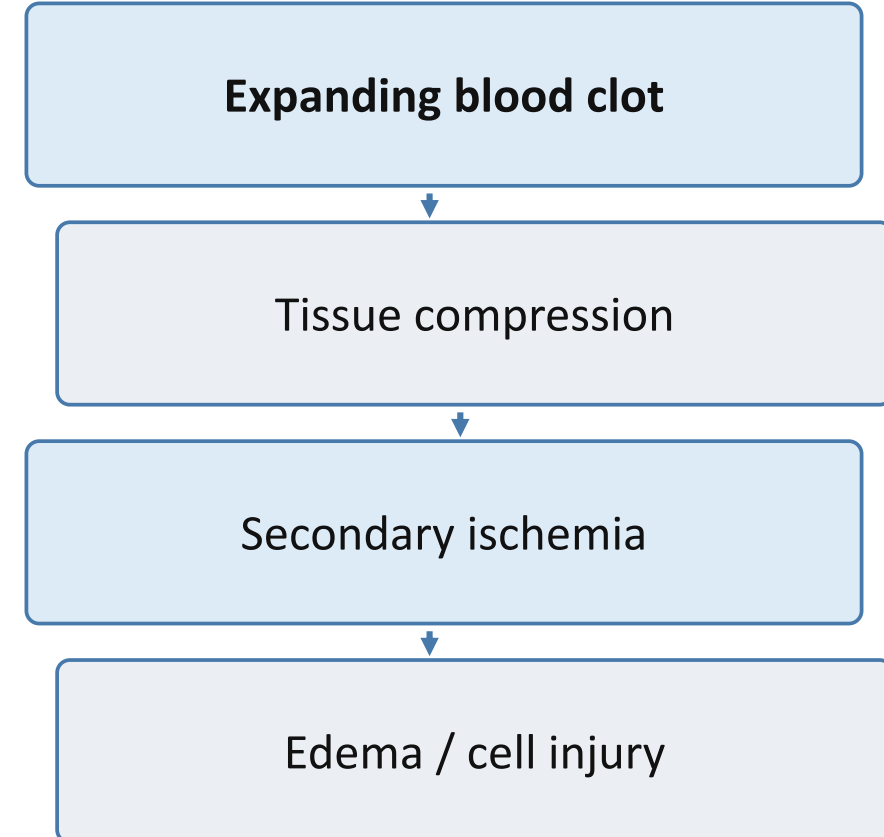
- Acute bleeding creates a central mass of coagulated blood.
- The hematoma displaces and compresses surrounding tissue.
- The surrounding parenchyma develops ischemic injury and edema.



Schematic anatomical localization — not a specimen

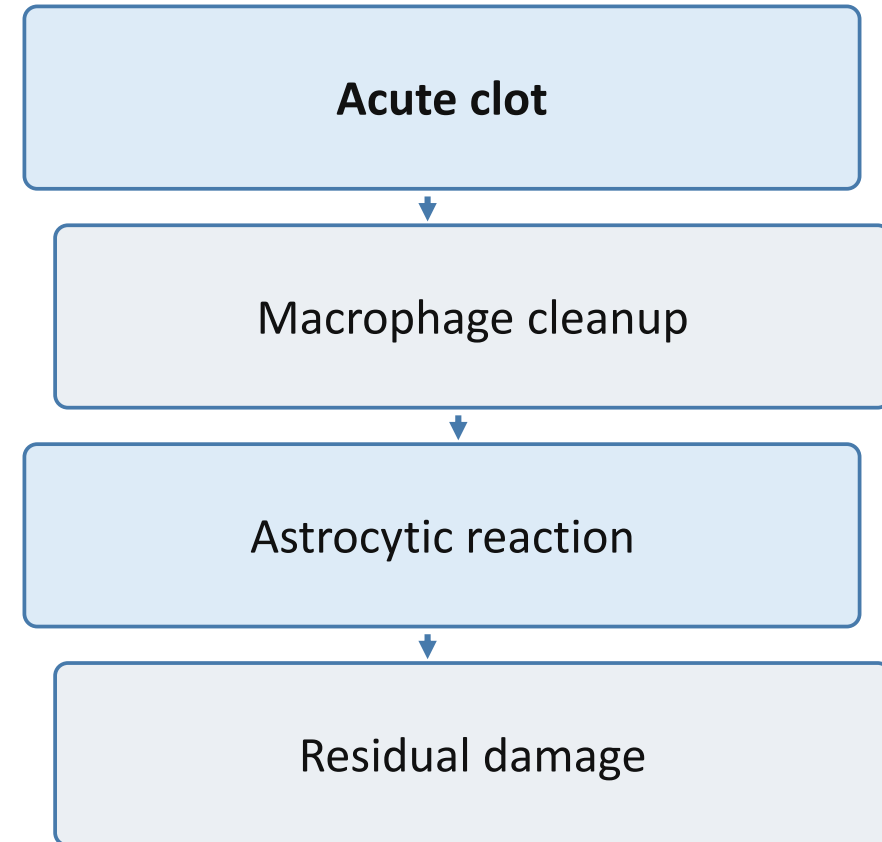
Adjacent ischemic injury is secondary to compression

- Clot expansion mechanically compresses neural tissue.
- Damaged surrounding cells show anoxic neuronal and glial changes.
- Edema adds further tissue injury around the hematoma.



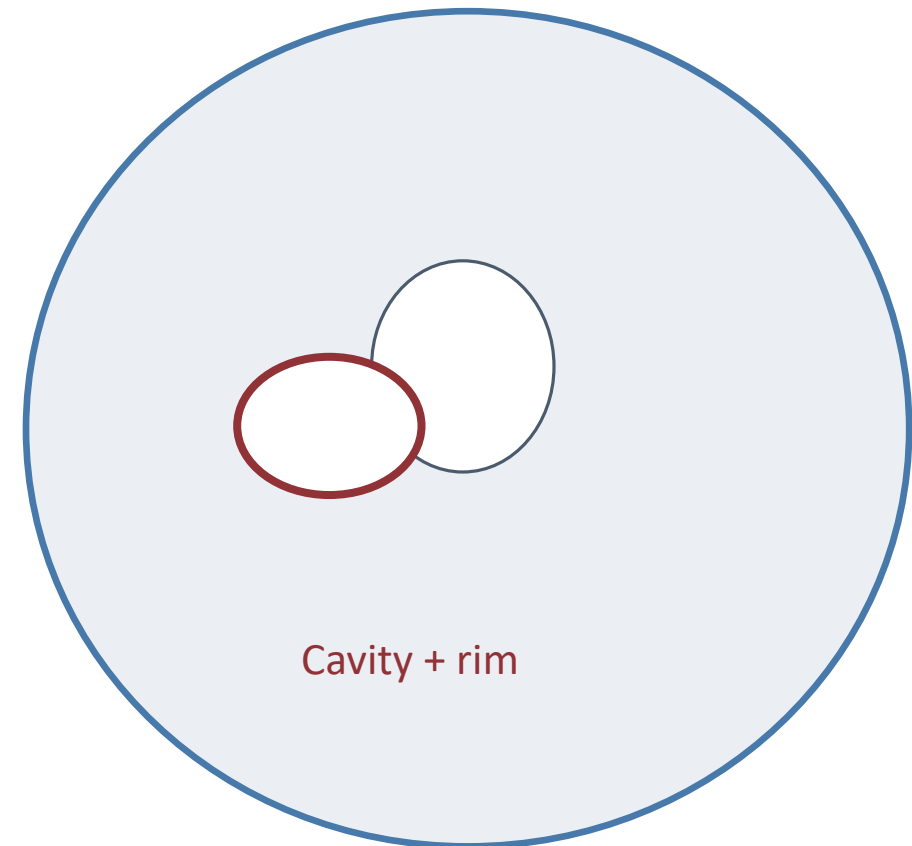
Healing begins with hematoma clearance

- Extravasated blood is gradually removed over weeks to months.
- Macrophages accumulate lipid and hemosiderin from injured tissue.
- Reactive astrocytes proliferate at the lesion margin.



Old hemorrhages leave characteristic cavities

- Severely injured parenchyma is permanently lost.
- Chronic lesions may form cavities with brown hemosiderin staining at the edge.
- Residual structural injury does not disappear when blood is cleared.



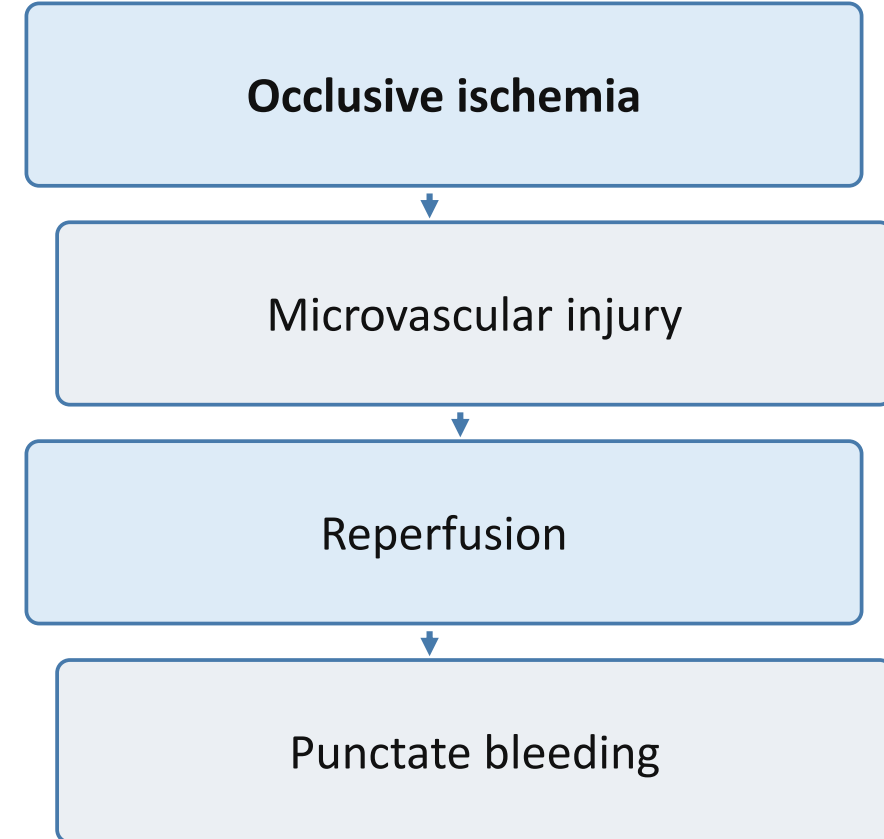
Schematic anatomical localization — not a specimen

A large hematoma or ventricular extension predicts severe injury

Distribution	Tissue impact	Clinical course
Small focal hematoma	Limited adjacent damage	May improve during resorption
Large parenchymal hematoma	Extensive destruction / edema	Often severe neurological deficit
Ventricular extension	More extensive blood distribution	Potentially devastating

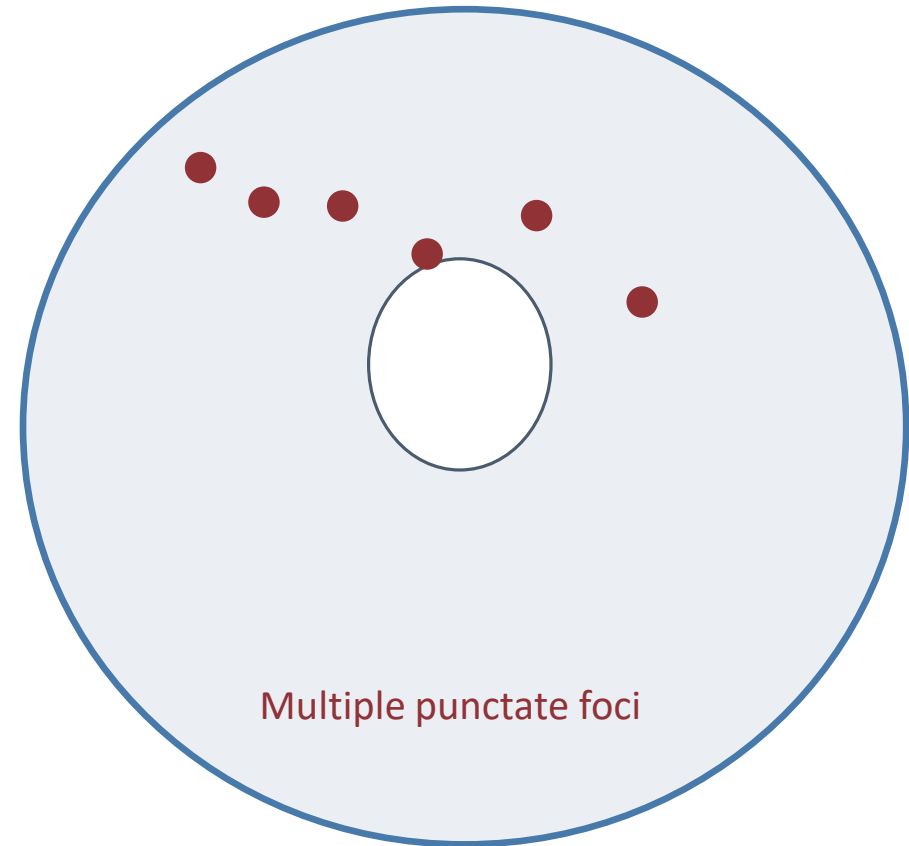
Hemorrhagic transformation follows an ischemic event

- An occlusive infarct initially may be nonhemorrhagic.
- Recanalization or fragmentation of the obstruction restores flow.
- Damaged microvessels then permit secondary hemorrhage.



Secondary hemorrhage commonly appears punctate

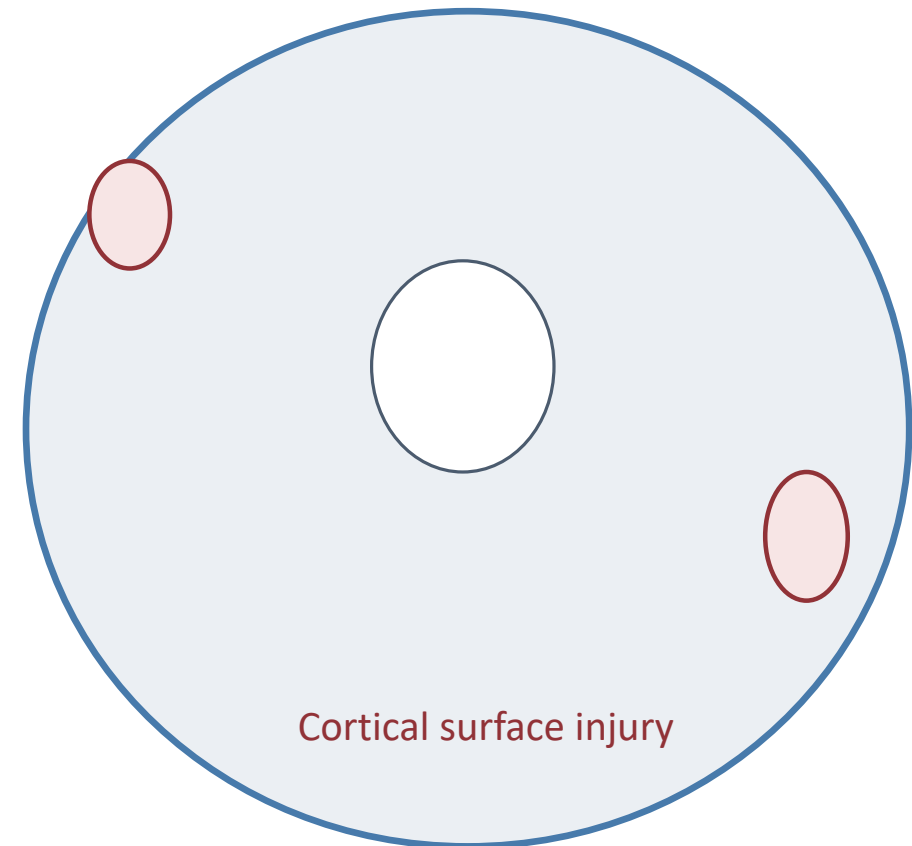
- Hemorrhagic transformation often produces multiple small hemorrhages.
- Blood may follow the previously ischemic cortical ribbon.
- More confluent secondary bleeding can also occur.



Schematic anatomical localization — not a specimen

Contusions are traumatic intraparenchymal hemorrhages

- Blunt kinetic force disrupts superficial tissue and blood vessels.
- Hemorrhage develops with parenchymal damage and edema.
- The crests of gyri are particularly susceptible to impact.



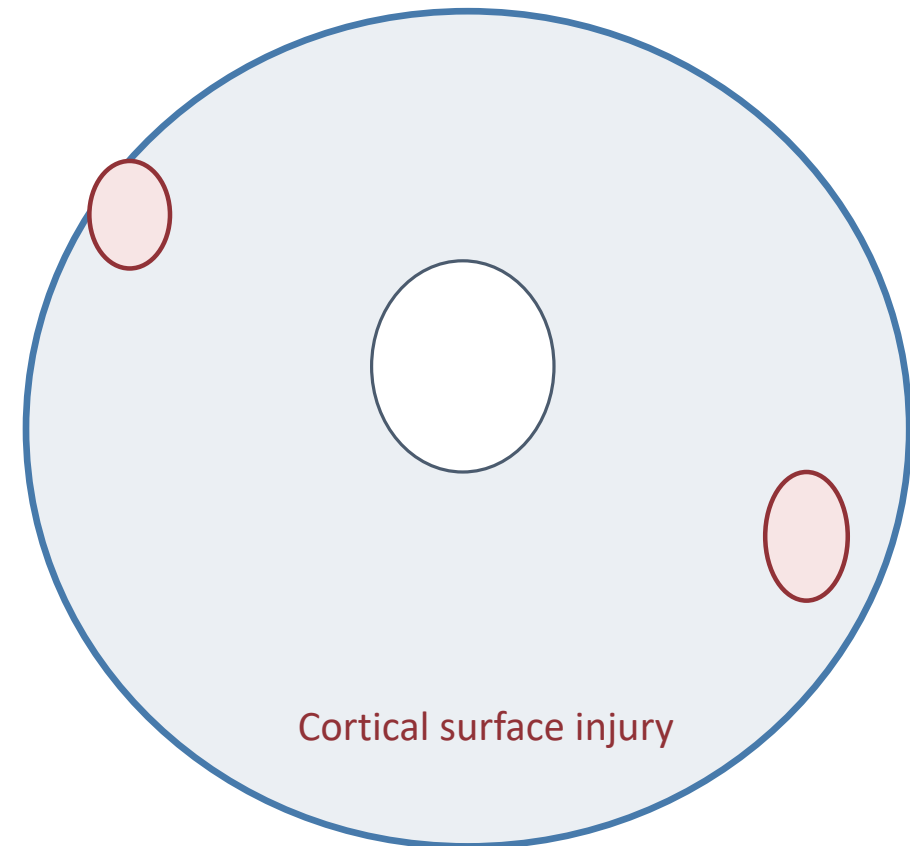
Schematic anatomical localization — not a specimen

Coup and contrecoup describe impact geometry

Feature	Coup	Contrecoup
Location	At point of impact	Opposite point of impact
Mechanism	Direct contact force	Brain strikes opposite skull
Morphology	Hemorrhagic contusion	Hemorrhagic contusion

Frontal and temporal surfaces are prone to contusions

- Orbital frontal and temporal cortex overlie irregular skull surfaces.
- The superficial gyri may develop wedge-shaped hemorrhagic lesions.
- Old contusions may leave yellow-brown, depressed plaques (plaque jaune).



Schematic anatomical localization — not a specimen

Additional causes complicate hemorrhage localization

- Coagulopathies may aggravate bleeding in several compartments.
- Neoplasms can bleed within parenchyma.
- Vasculitis, aneurysms and vascular malformations can also cause hemorrhage.

Some tumors are predisposed to intratumoral hemorrhage

- High-grade gliomas among relevant primary tumors.
- Examples of hemorrhage-prone metastases include melanoma.
- Choriocarcinoma and renal cell carcinoma metastases are also cited.

Subarachnoid blood occupies the CSF compartment

- Blood fills spaces around the brain normally containing CSF.
- Bleeding can arise from trauma or a ruptured vascular lesion.
- Rupture of a saccular aneurysm is the leading spontaneous cause.

Skull
Epidural interface
Dura
Subdural border
Arachnoid
Subarachnoid space (CSF)
Pia + cerebral cortex

Spontaneous subarachnoid hemorrhage has several causes

- Ruptured saccular aneurysm is the principal cause.
- Other causes include vascular malformations and blood disorders.
- A primary cerebral hemorrhage or tumor may also contribute.

Subarachnoid blood

Saccular aneurysm

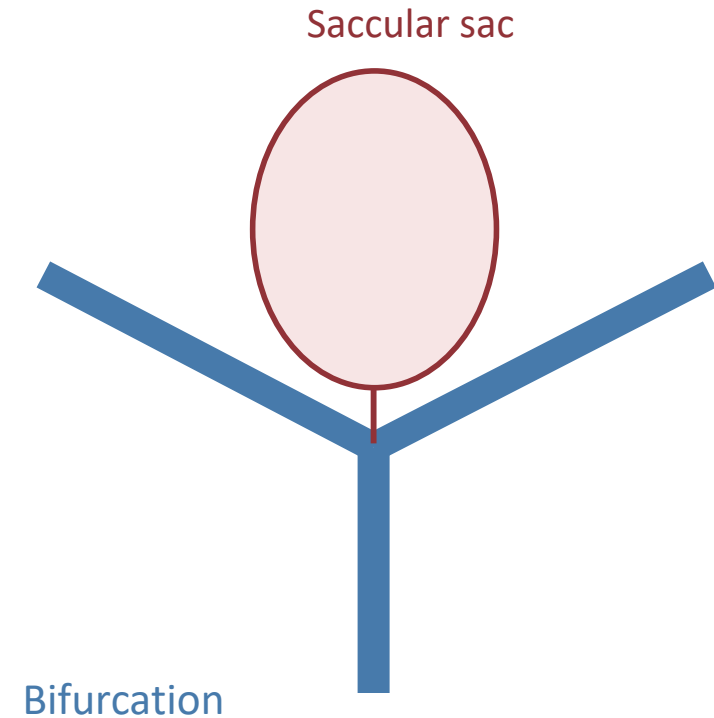
Vascular
malformation

Parenchymal
extension

Other disorders

Saccular aneurysms arise near major branch points

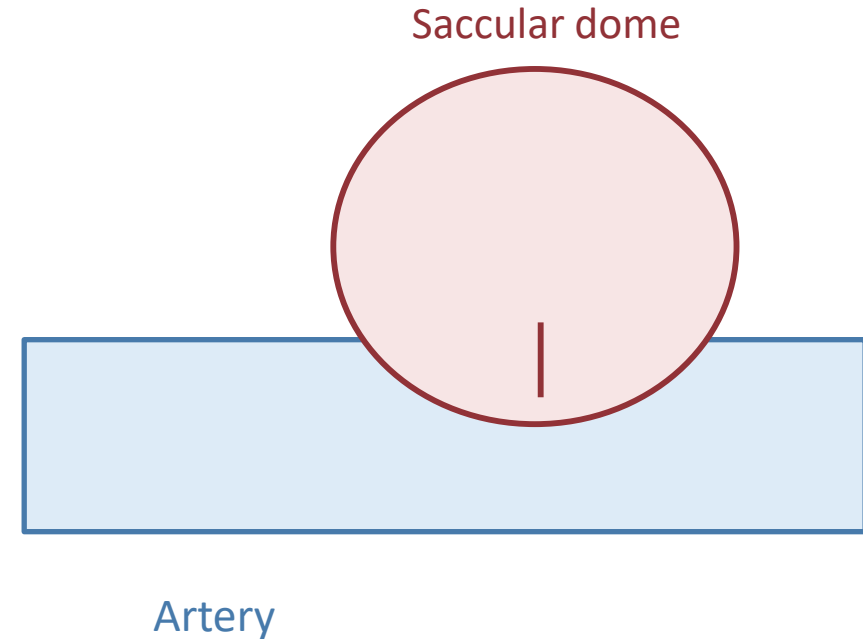
- Saccular outpouchings commonly occur along the circle of Willis.
- Most are located in the anterior cerebral circulation.
- Branch-point flow stress is implicated in their development.



Branch-point wall vulnerability

The aneurysm sac lacks normal arterial wall support

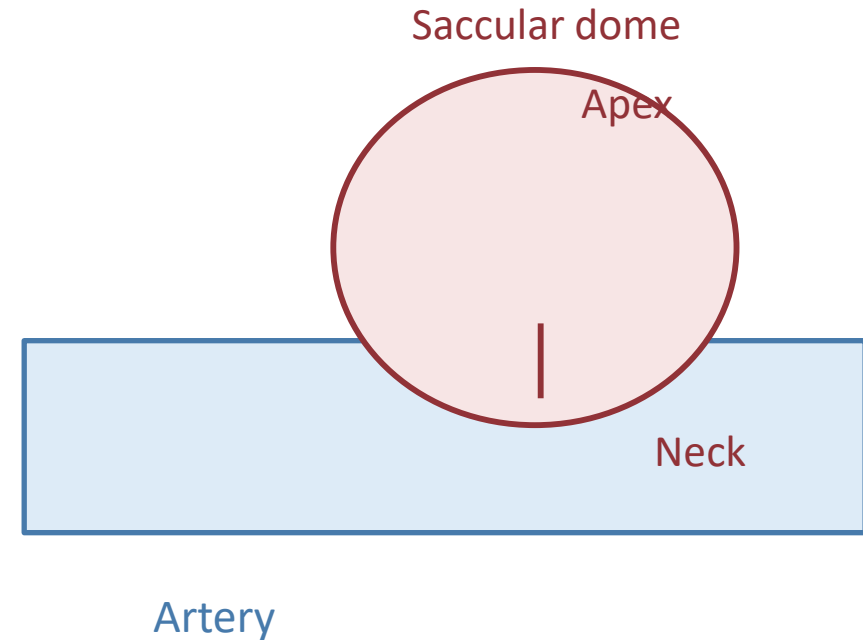
- Smooth muscle and internal elastic lamina are absent from the sac.
- The wall consists mainly of hyalinized intima and adventitia.
- The weakened sac is susceptible to rupture.



Sac lacks media and internal elastic lamina

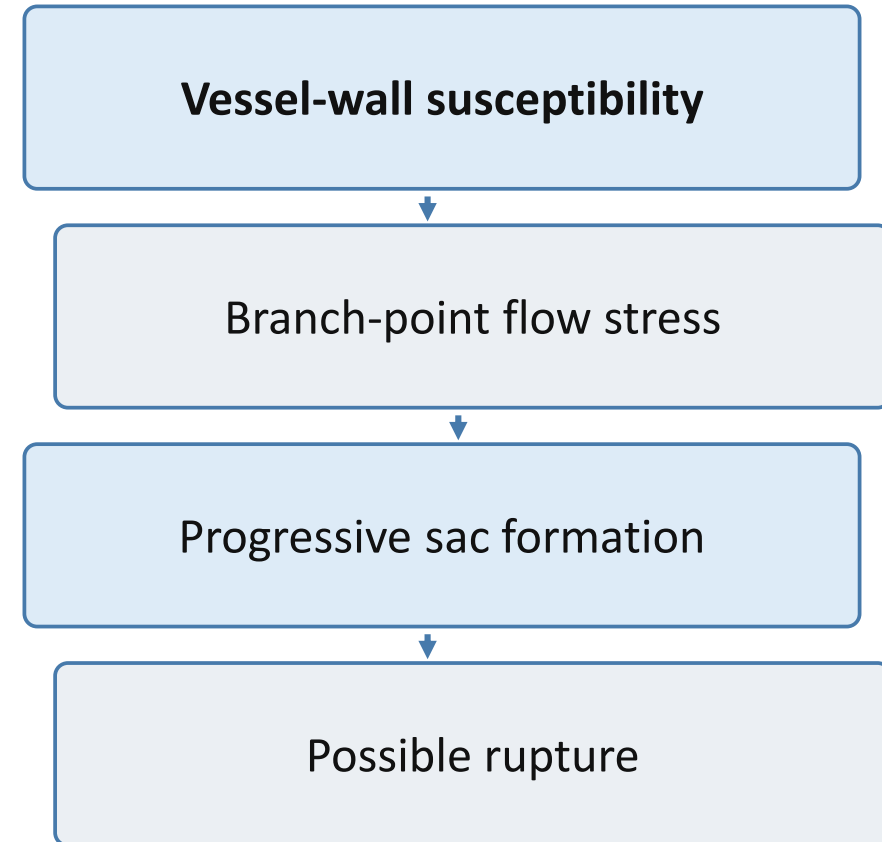
A saccular aneurysm has a neck, dome and apex

- A narrow or broad neck connects the sac to the parent artery.
- The dome forms a thin-walled rounded outpouching.
- Rupture generally occurs near the apex.



Aneurysms evolve rather than existing fully formed at birth

- Underlying structural susceptibility predisposes to sac formation.
- Hemodynamic stress at an arterial branch point contributes.
- The term “congenital aneurysm” is therefore potentially misleading.

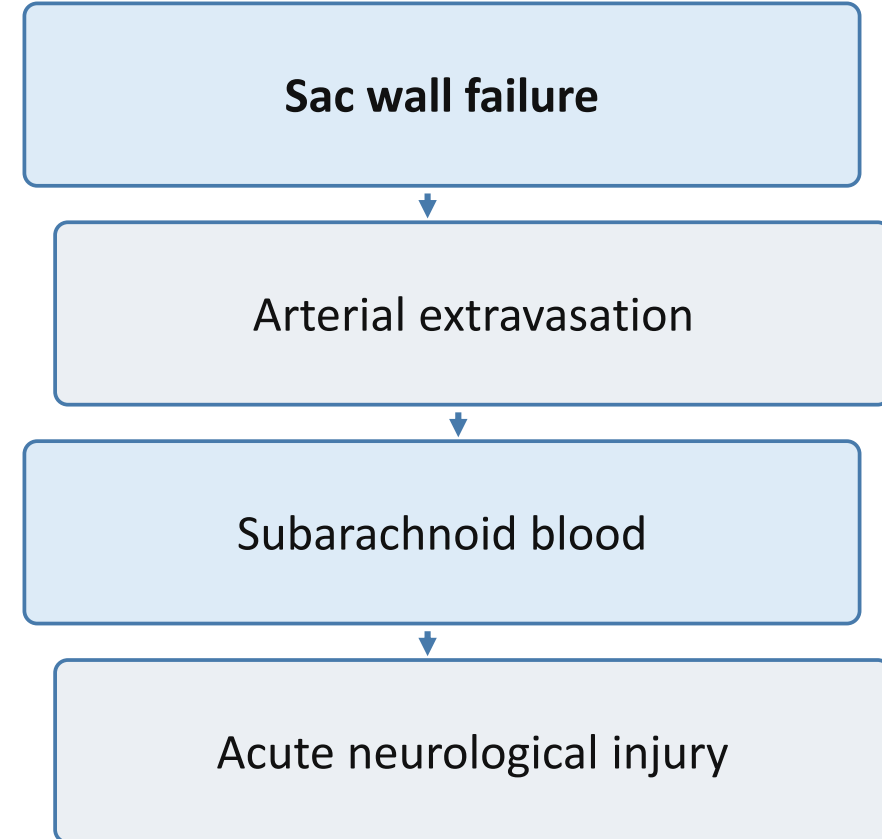


Aneurysm predisposition includes inherited and acquired factors

- First-degree family history suggests a genetic contribution.
- Associated disorders include autosomal dominant polycystic kidney disease.
- Smoking and chronic hypertension are recognized risk factors.

Rupture introduces arterial blood into the subarachnoid space

- A sac may break at its dome/apex.
- High-pressure arterial blood spreads around the brain.
- Some ruptures extend into the brain parenchyma as well.

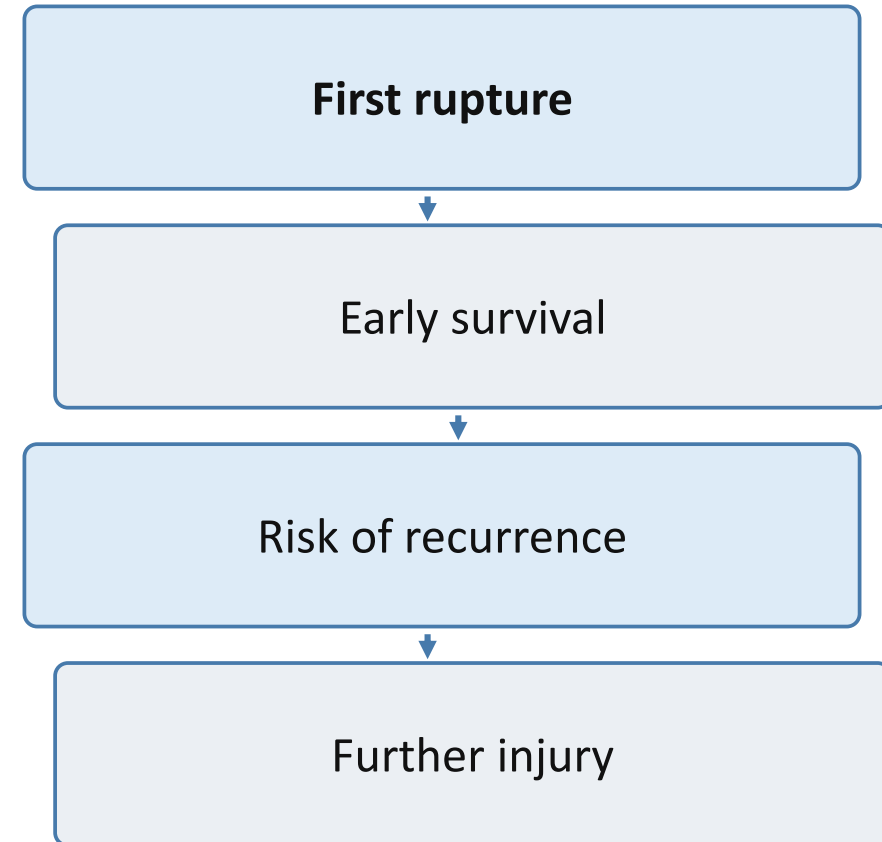


Aneurysmal SAH often causes abrupt severe headache

- Classically described as sudden, excruciating headache.
- Rapid loss of consciousness or deterioration may accompany rupture.
- The presentation reflects acute bleeding into a sensitive intracranial space.

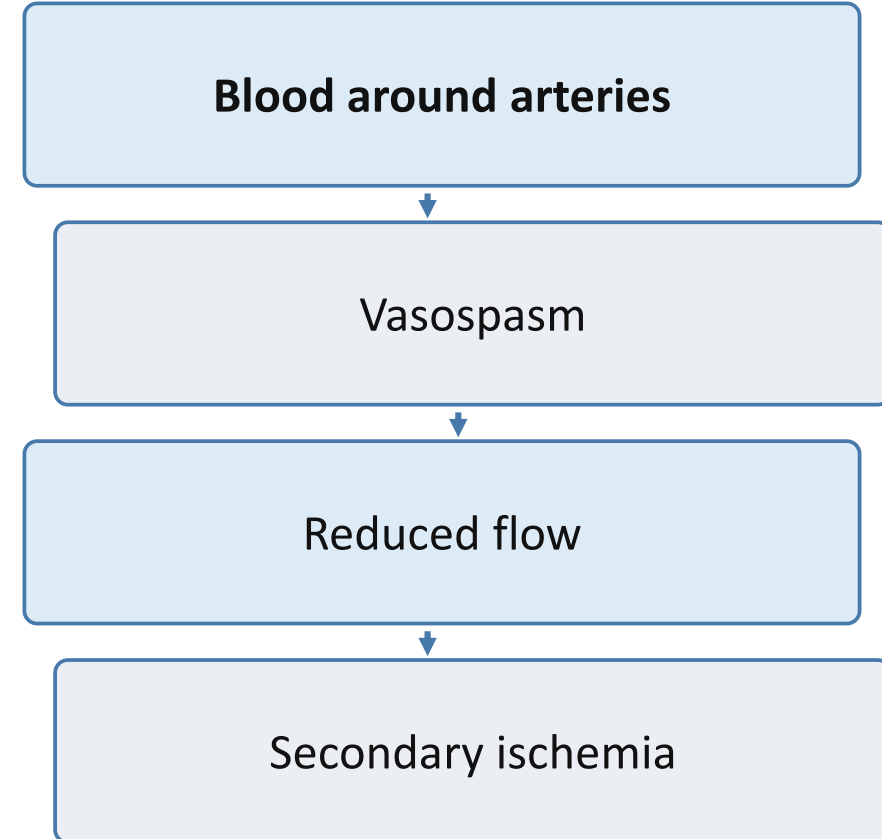
Rebleeding worsens the prognosis after aneurysm rupture

- Survivors of initial rupture may recover consciousness.
- Additional episodes of hemorrhage can occur unpredictably.
- Each further bleeding episode can worsen the outlook.



Subarachnoid blood can provoke arterial vasospasm

- Extravasated blood bathes basal cerebral vessels.
- Arterial narrowing may cause secondary cerebral ischemia.
- This risk is especially important in basal subarachnoid hemorrhage.



Several candidate mediators influence vasospasm

- Endothelins have been implicated in vascular contraction.
- Nitric oxide signaling and arachidonic acid metabolites may contribute.
- These as proposed mechanisms, not a single proven pathway.

Subarachnoid blood

Endothelin pathways

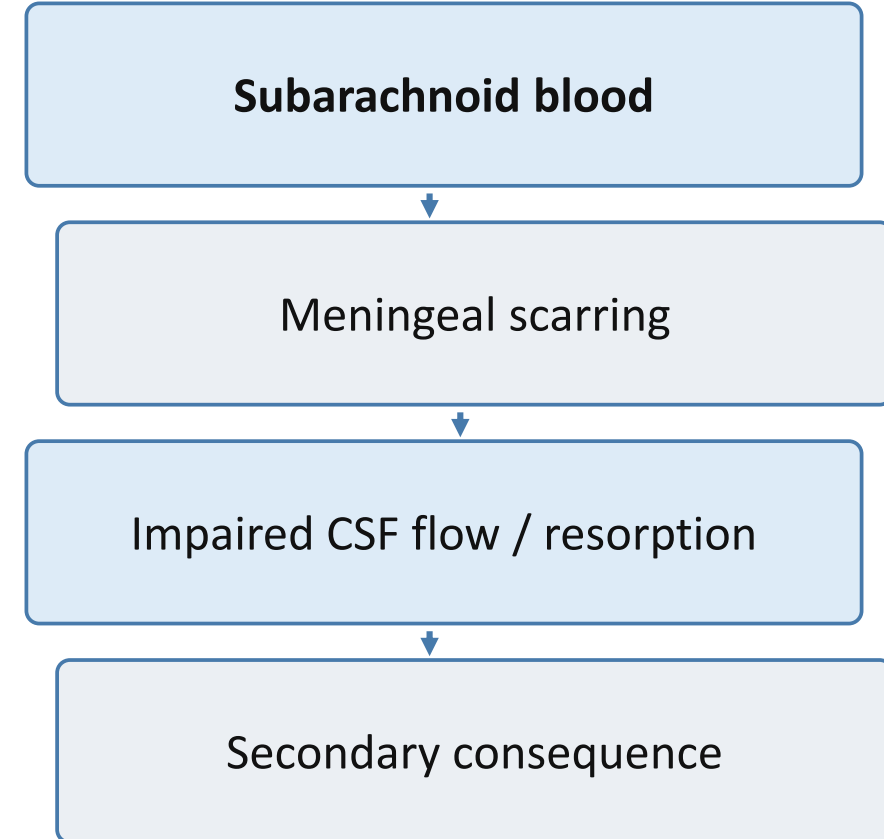
Nitric oxide signaling

Arachidonic products

Vessel caliber change

Healing of SAH may interfere with CSF circulation

- Meningeal fibrosis and scarring develop during repair.
- Scar formation can obstruct CSF flow.
- Disruption of CSF resorption provides an additional mechanism of injury.



Saccular aneurysms are not the only vascular abnormalities

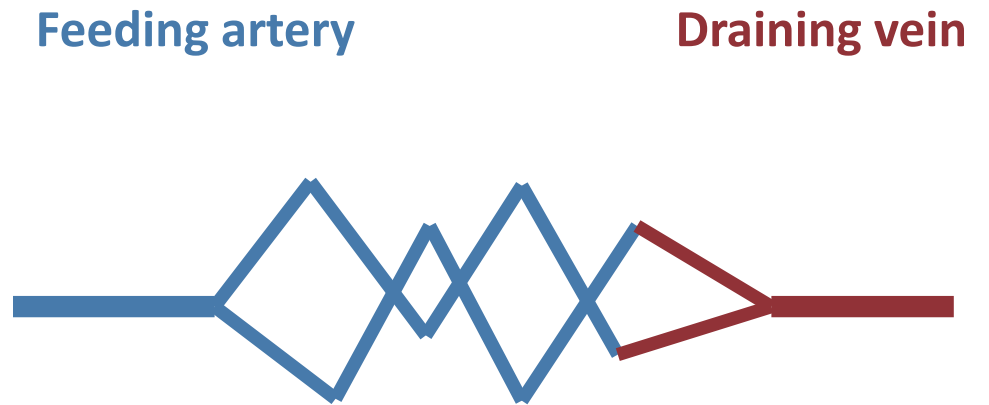
- Other intracranial aneurysm types include fusiform and dissecting lesions.
- Mycotic and traumatic aneurysms are also described.
- Different vascular lesions can produce overlapping bleeding distributions.

Four principal cerebral vascular malformations are recognized

Malformation class	Potential clinical importance
Arteriovenous malformation	Seizures; cerebral or subarachnoid bleed
Cavernous malformation	Bleeding and neurologic symptoms
Capillary telangiectasia	Recognized vascular malformation group
Venous angioma	Recognized vascular malformation group

AVMs connect arteries to veins abnormally

- Abnormal vessels form a high-flow arteriovenous shunt.
- Normal intervening capillary resistance is bypassed.
- Rupture can cause parenchymal or subarachnoid hemorrhage.



Direct arteriovenous connection

Intervening capillary bed bypassed

AVMs often present with seizures or bleeding

- Presentations commonly occur in children and young adults.
- Seizures and intracerebral or subarachnoid hemorrhage are important presentations.
- High-flow neonatal shunts may cause cardiac failure.

Arteriovenous malformation

Seizures

Intracerebral
bleeding

Subarachnoid
bleeding

Neonatal cardiac
failure

Somatic signaling alterations occur in vascular malformations

Entity	Reported molecular alterations	Interpretation
AVM	Endothelial KRAS activation	RAS pathway dysregulation
Cavernous malformation	Loss of CCM-complex function	Defective vascular-growth restraint
Aggressive cavernous lesion	Additional PIK3CA activation	Growth-promoting signaling

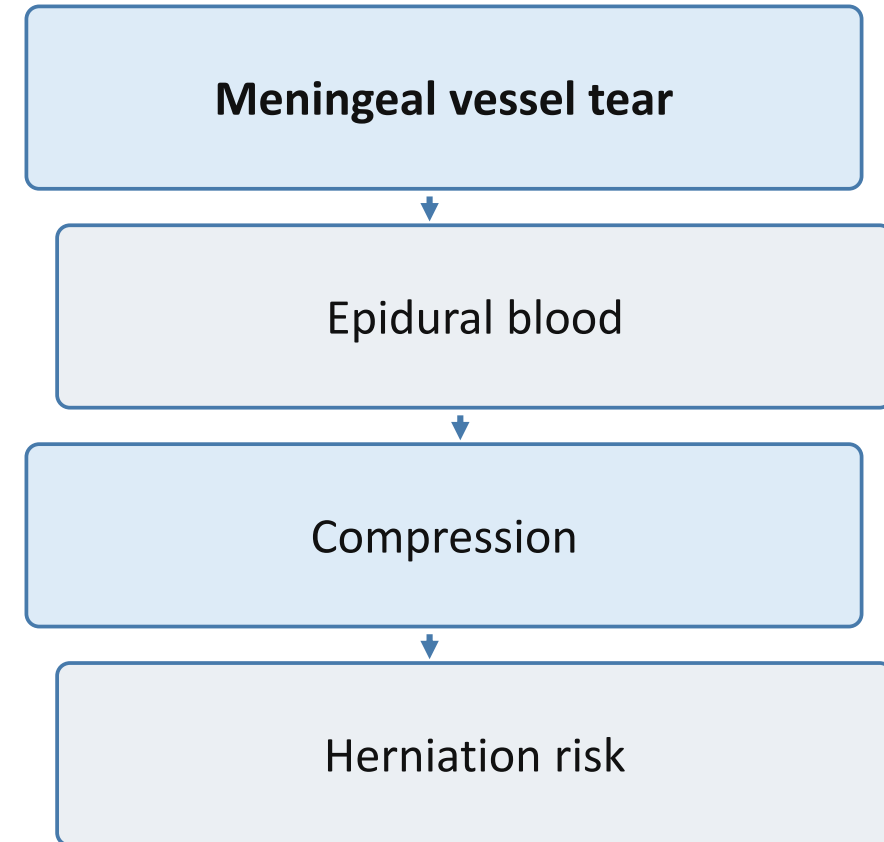
Epidural hematoma is extra-axial arterial bleeding

- The middle meningeal artery is a characteristic injured vessel.
- A fracture crossing its course is common in adults.
- Arterial pressure strips dura from the overlying skull.

Skull
Epidural interface
Dura
Subdural border
Arachnoid
Subarachnoid space (CSF)
Pia + cerebral cortex

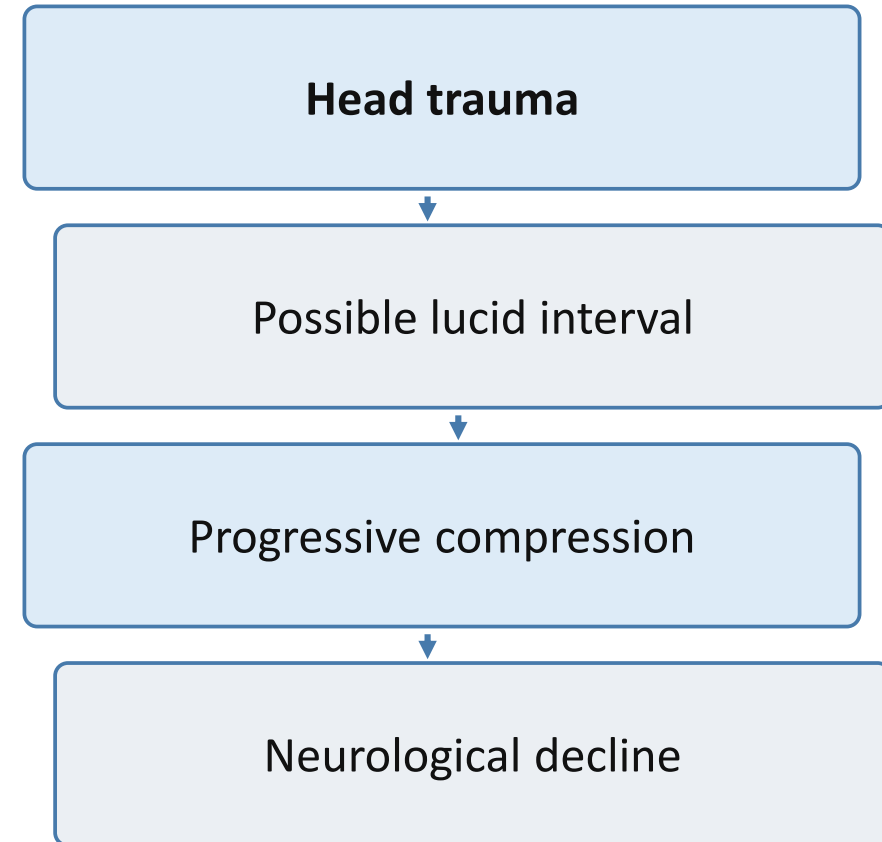
Epidural bleeding produces rapidly expanding mass effect

- Arterial blood creates a potential space against the skull.
- The growing collection compresses the adjacent brain.
- Without prompt treatment, dangerous herniation can develop.



A lucid interval is possible but not obligatory

- An initially conscious patient may deteriorate as the hematoma grows.
- The symptom-free period depends partly on the rate of bleeding.
- A symptomatic epidural hematoma is a neurosurgical emergency.

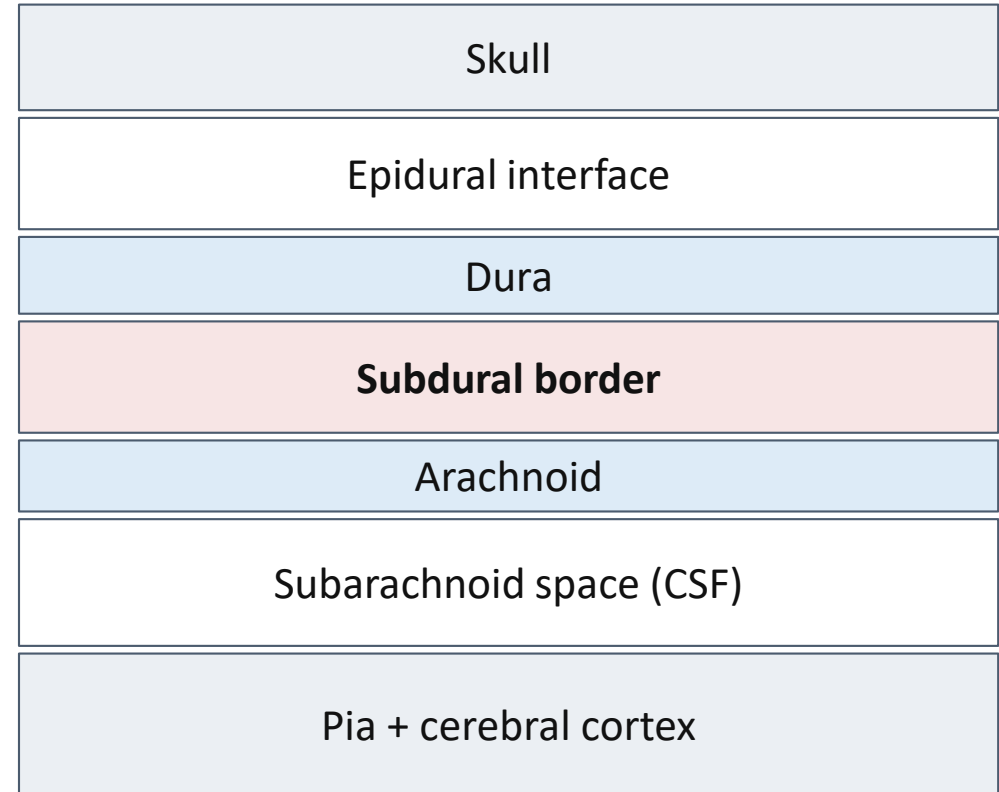


Epidural hemorrhage can occur without an obvious fracture

- In children, skull deformation may transiently tear a meningeal artery.
- A permanent fracture need not accompany vascular injury.
- The essential lesion is the arterial breach and expanding extradural blood.

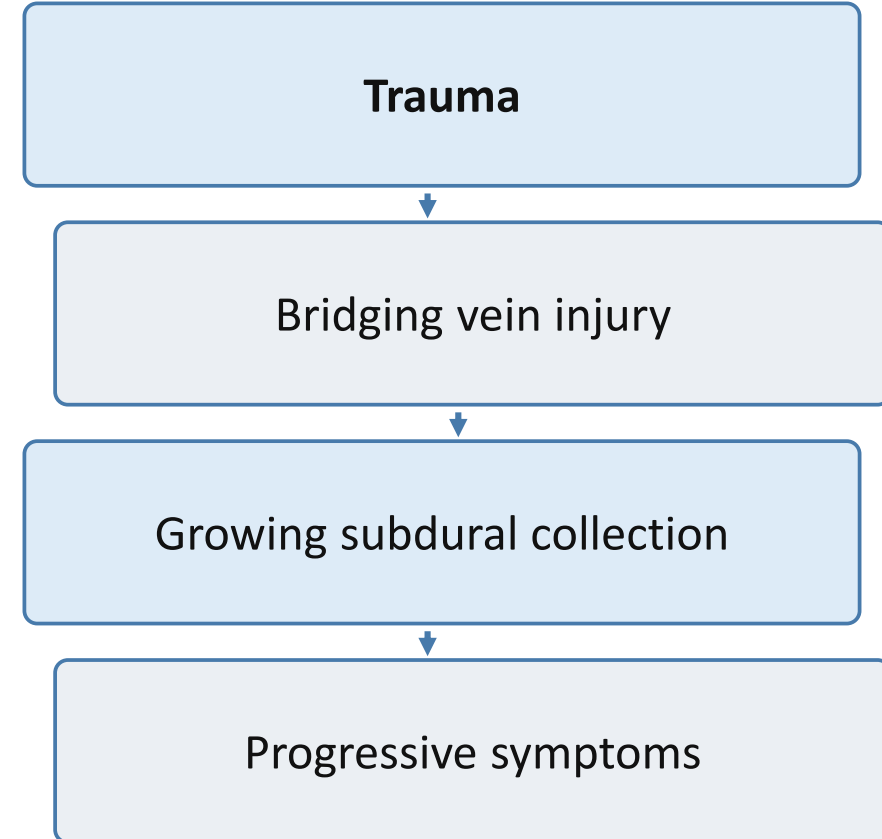
Subdural hematoma usually begins with venous tearing

- Bridging veins cross from the cerebral convexity toward dural sinuses.
- Brain displacement can tear veins near their dural entry.
- Blood dissects within the dural border region.



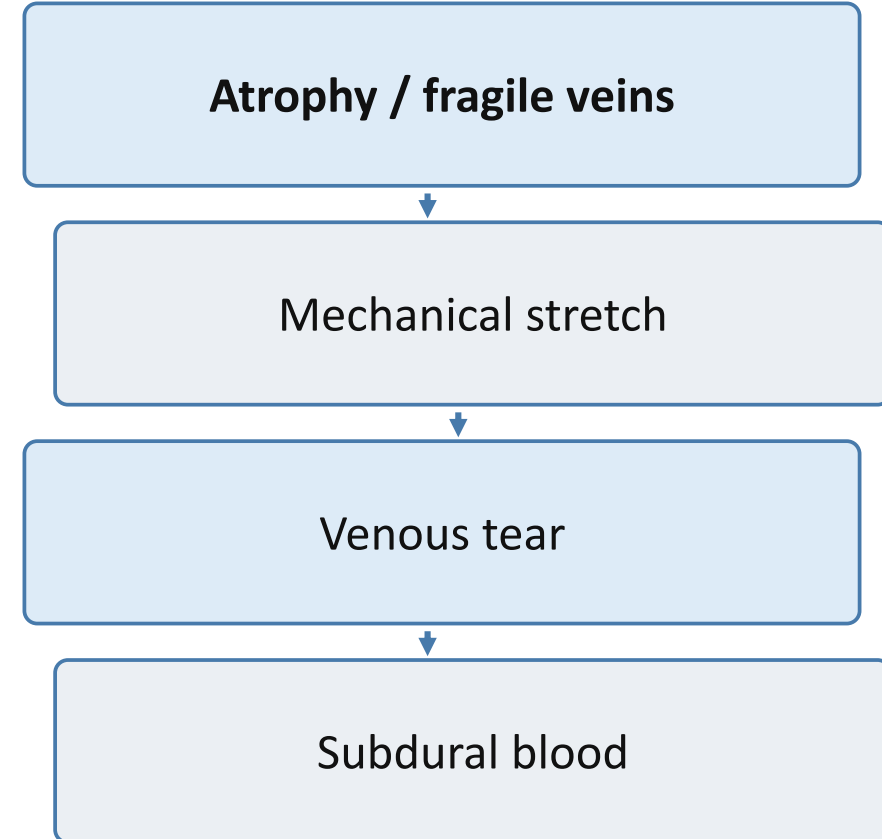
Subdural bleeding may evolve relatively slowly

- Headache or confusion may appear after a delay.
- Nonlocalizing symptoms are common, although focal signs can occur.
- Deterioration may be progressive or occasionally acute.



Brain atrophy increases bridging-vein vulnerability

- Atrophy places bridging veins under greater stretch.
- Consequently older adults may bleed after minor head trauma.
- Infants are susceptible because the bridging veins are thin-walled.

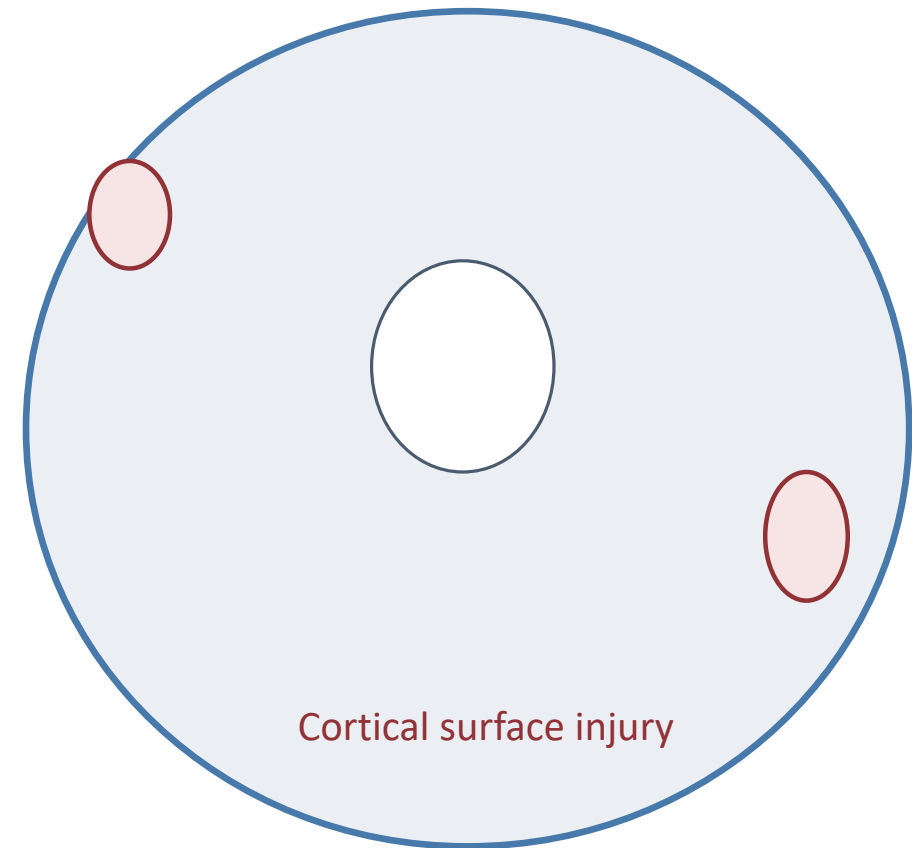


Epidural and subdural hematomas differ mechanistically

Feature	Epidural hematoma	Subdural hematoma
Source	Meningeal artery	Bridging veins
Typical setting	Skull fracture / trauma	Trauma; minor trauma possible
Bleeding compartment	Skull–dura interface	Dural border region
Course	May deteriorate rapidly	Often delayed / progressive

Trauma may involve multiple compartments at once

- An impact may injure skull, superficial brain and blood vessels together.
- Parenchymal contusions often coexist with subarachnoid blood.
- An extra-axial collection does not exclude additional brain injury.



Schematic anatomical localization — not a specimen

The complete compartment matrix localizes the source

Compartment	Typical lesion	Distinguishing context
Epidural	Meningeal arterial tear	Head trauma / fracture
Subdural	Bridging vein tear	Atrophy / minor trauma
Subarachnoid	Aneurysm or AVM rupture	Sudden severe headache
Parenchymal	Hypertensive / amyloid vessel rupture	Deep vs lobar location

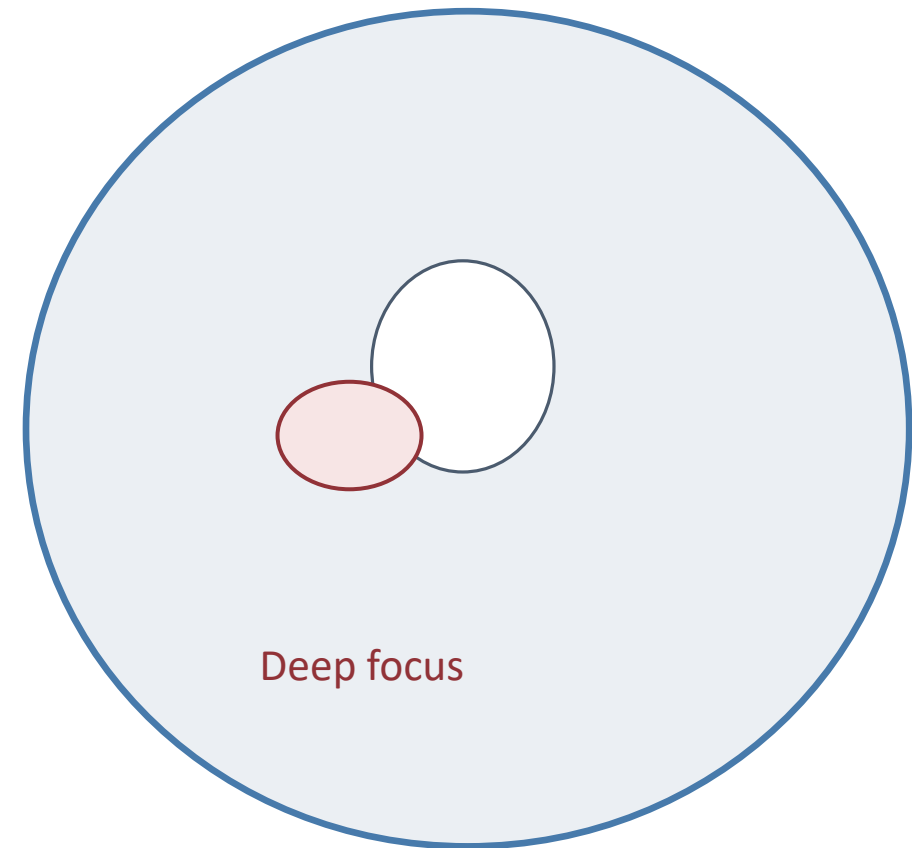
Case: Sudden deficit with a deep putaminal hematoma

Clinical vignette — interpret the pathology

- An adult develops abrupt focal neurological deficit.
- Bleeding is centered deep within the basal ganglia.
- Which underlying arterial disease best explains this distribution?

Deep putaminal hemorrhage suggests hypertensive arteriopathy

- Hypertension injures deep penetrating small arteries.
- Hyaline thickening makes the vessel wall prone to rupture.
- Ventricular extension may complicate a large deep hematoma.



Schematic anatomical localization — not a specimen

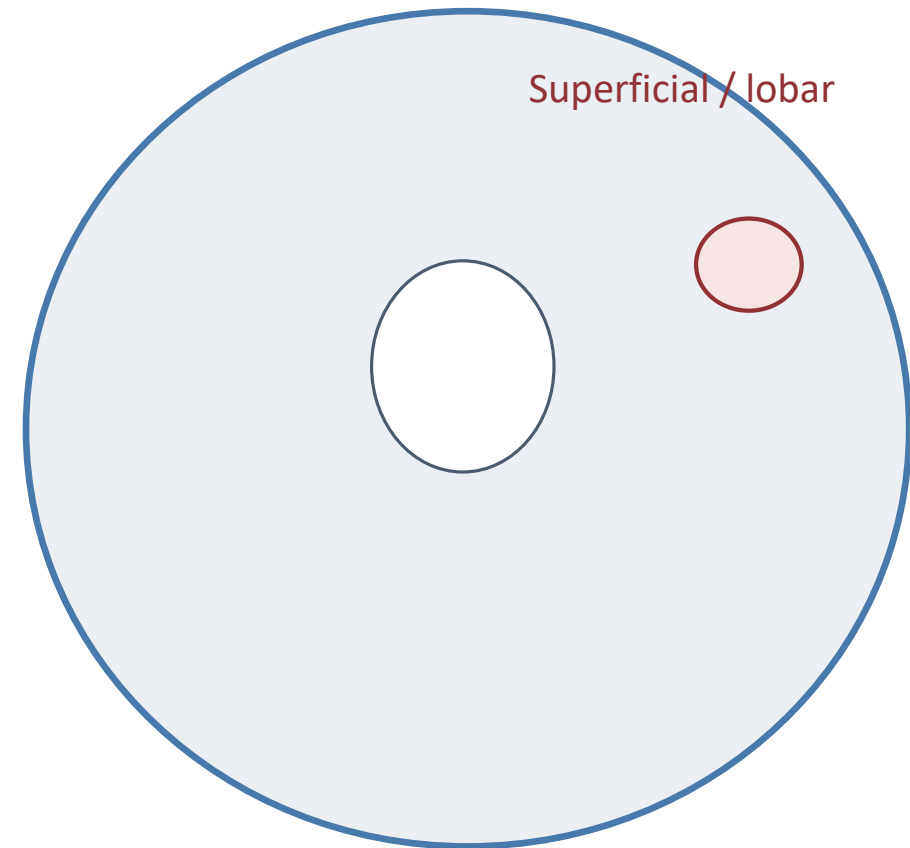
Case: Superficial lobar hemorrhage with microbleeds

Clinical vignette — interpret the pathology

- An older adult has a superficial cerebral hematoma.
- Several small prior hemorrhagic foci are also present.
- Which vessel-wall deposit should be considered?

Lobar hemorrhage with microbleeds raises CAA

- Cortical and leptomeningeal vessels accumulate A β deposits.
- A weakened vessel wall predisposes to superficial hemorrhage.
- The distribution differs from classic deep hypertensive bleeding.



Schematic anatomical localization — not a specimen

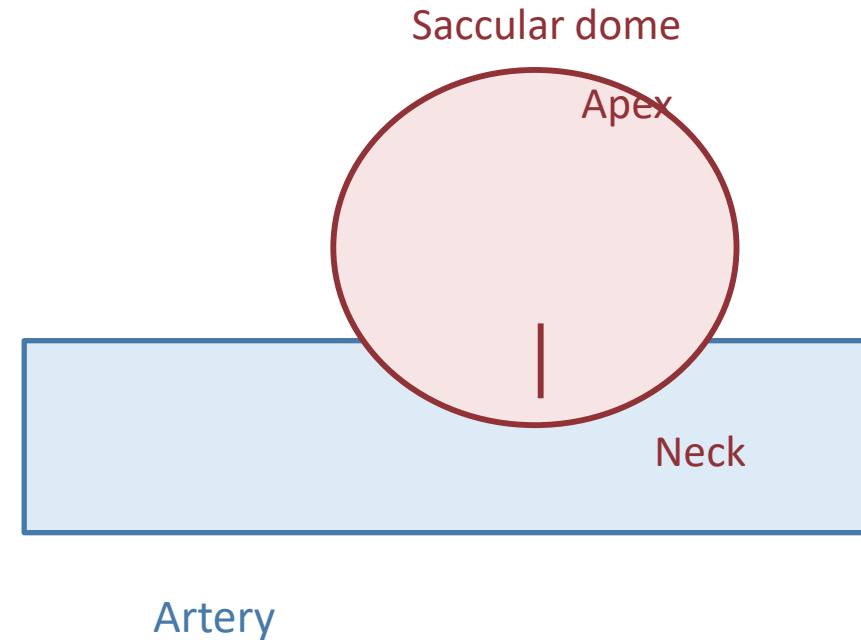
Case: Sudden excruciating headache and collapse

Clinical vignette — interpret the pathology

- A patient develops a sudden maximal-intensity headache.
- Consciousness is briefly lost; Subarachnoid blood is identified.
- Which ruptured arterial lesion is a leading possibility?

Aneurysm rupture explains spontaneous basal SAH

- The saccular arterial wall lacks normal media and elastic lamina.
- Rupture releases arterial blood into the subarachnoid compartment.
- Vasospasm and later CSF scarring are important secondary hazards.



Case: Deterioration after temporal head trauma

Clinical vignette — interpret the pathology

- Following a head injury, an adult is initially responsive.
- Neurological function deteriorates as an extra-axial collection grows.
- What arterial injury should be considered urgently?

Rapid extra-axial deterioration can indicate epidural blood

- Meningeal arterial rupture may follow temporal skull fracture.
- The expanding epidural collection compresses the brain.
- The possibility of a lucid interval must not delay recognition.

Skull
Epidural interface
Dura
Subdural border
Arachnoid
Subarachnoid space (CSF)
Pia + cerebral cortex

Synthesis: Infer mechanism from location and tissue response

- Deep clot-centered hemorrhage: Think hypertensive small-vessel injury.
- Lobar bleeding with microbleeds: Consider cerebral amyloid angiopathy.
- Subarachnoid blood: Search conceptually for trauma or vascular lesion.
- Extra-axial blood: Distinguish meningeal arterial from bridging-vein injury.

References and source scope

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. (2026).
- Primary parent heading: Intracranial Hemorrhage; expanded using adjacent CNS sections.
- All illustrations in this deck are original editable schematics, not clinical images.
- Clinical vignettes are constructed teaching examples.