

Infectious Vasculitis

Direct vessel-wall infection, vascular injury and clinical consequences

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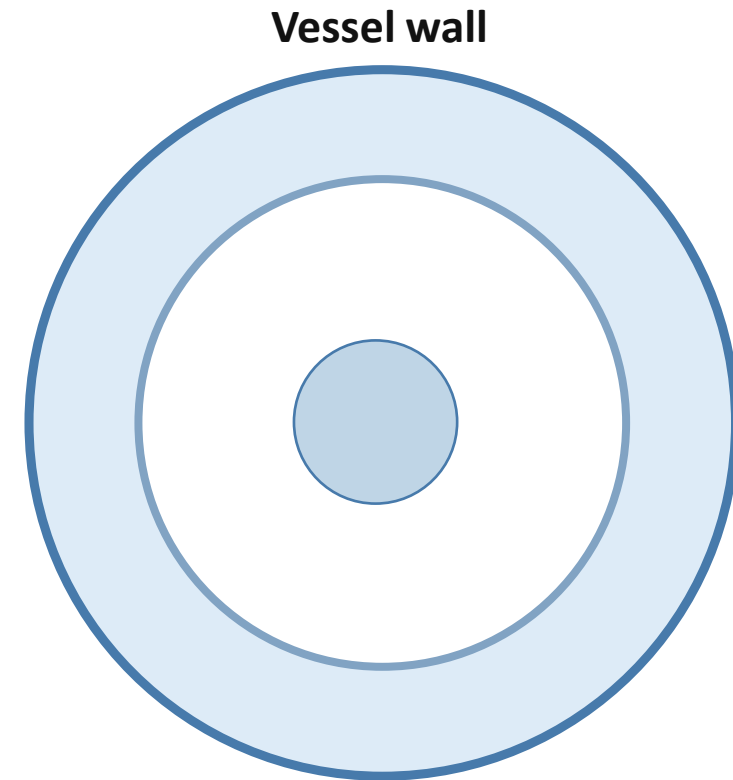
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Learning objectives

- Distinguish direct infectious vasculitis from immune-mediated vasculitis
- Trace infection to the arterial wall through local and blood-borne routes
- Link wall injury to aneurysm, thrombosis and infarction
- Interpret angioinvasive fungal and infective endocarditis examples

Vasculitis means inflammation of the vessel wall

- The defining process is inflammation centered on a vascular wall.
- Manifestations depend on the affected vascular bed.
- Disease can be organ-limited or affect multiple vascular beds.



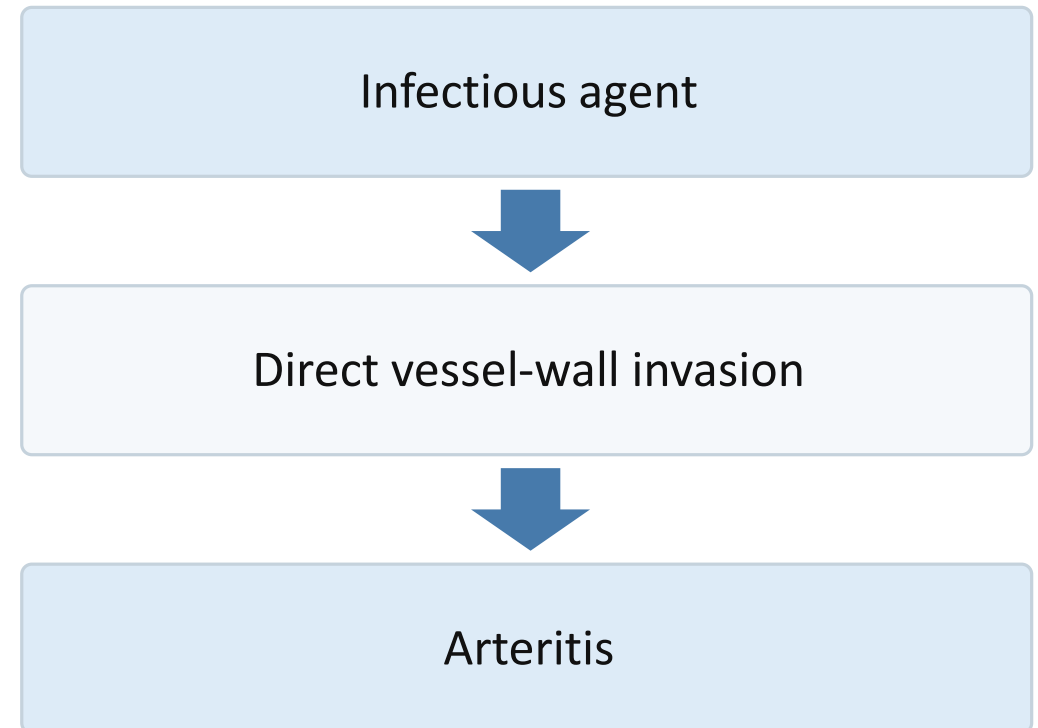
Vascular lumen

Wall inflammation

Schematic vessel cross-section — not a histologic specimen

Infectious vasculitis requires direct vascular invasion

- Infectious agents directly invade and inflame vascular walls.
- The mechanism differs from an infection that only triggers a remote immune reaction.
- Arteritis is the arterial-wall form emphasized in this Robbins subsection.



Schematic

Two major routes to vasculitic injury

- Direct microbial wall invasion produces infectious arteritis.
- Immune-mediated inflammation produces noninfectious vasculitis.
- The distinction changes the implications of immunosuppression.

Direct microbial invasion

Infectious vasculitis

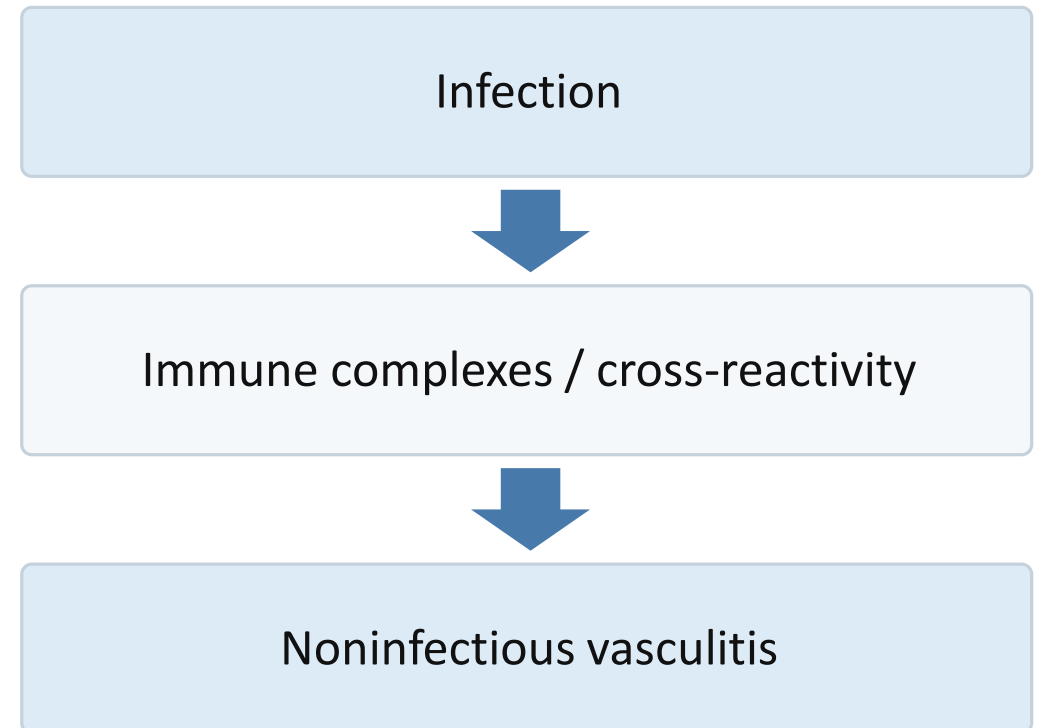
Immune-mediated inflammation

Noninfectious vasculitis

Schematic

Infection may also drive indirect immune vasculitis

- An infectious trigger can generate immune complexes.
- An infection can also initiate vascular cross-reactivity.
- These mechanisms do not establish direct organisms within a vascular wall.



Schematic

Why the infectious–immune distinction matters

Question	Direct infectious	Immune-mediated
Immediate driver	Organisms invade wall	Inflammatory immune response
Robbins warning	Immunosuppression may harm	May require immunosuppression

Distribution: Local disease versus systemic involvement

- Vasculitis may initially be restricted to a single organ.
- Systemic disease may involve several vascular beds.
- Organ-specific injury and constitutional inflammation may coexist.

Single affected territory

Organ-limited disease

Several vascular beds

Systemic disease

Schematic

Symptoms derive from two simultaneous processes

- Vascular-bed injury produces local functional effects.
- Systemic inflammation may cause fever, myalgias, arthralgias and malaise.
- The clinical phenotype depends on where vessel damage occurs.

Affected vascular bed

Organ findings

Systemic inflammatory response

Constitutional findings

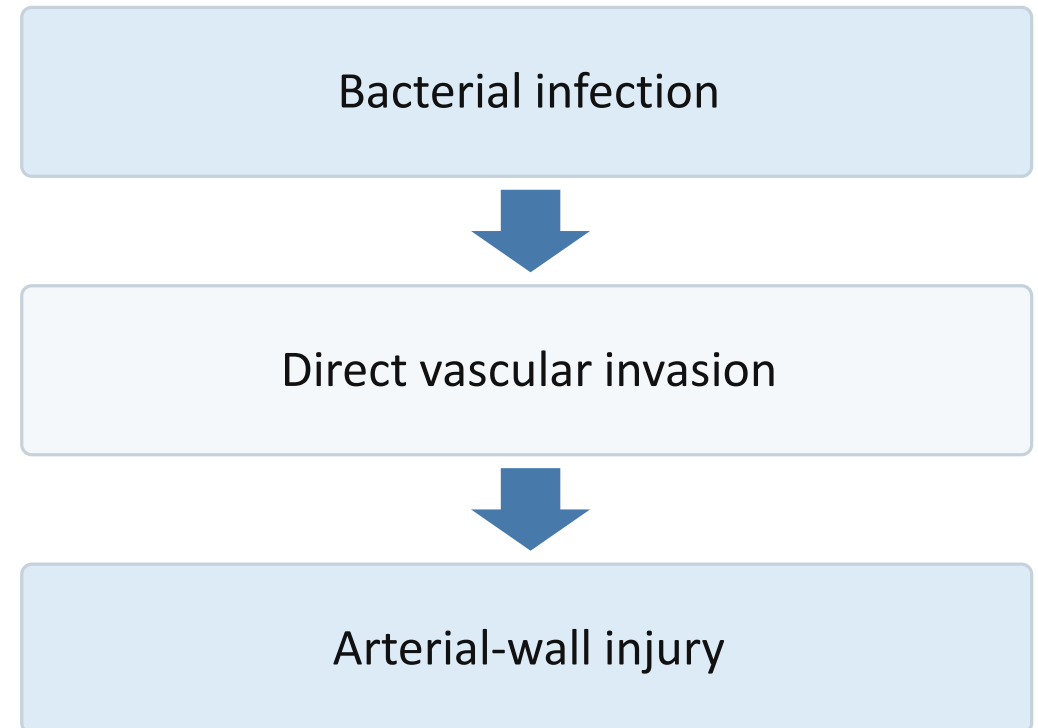
Schematic

The principal infectious agents named

Category	Named example	Teaching relevance
Bacteria	Pseudomonas	Direct infectious arteritis
Fungi	Aspergillus	Angioinvasion; infarction
Fungi	Mucor	Arterial invasion; thrombi

Pseudomonas: Bacterial arteritis as a mechanism

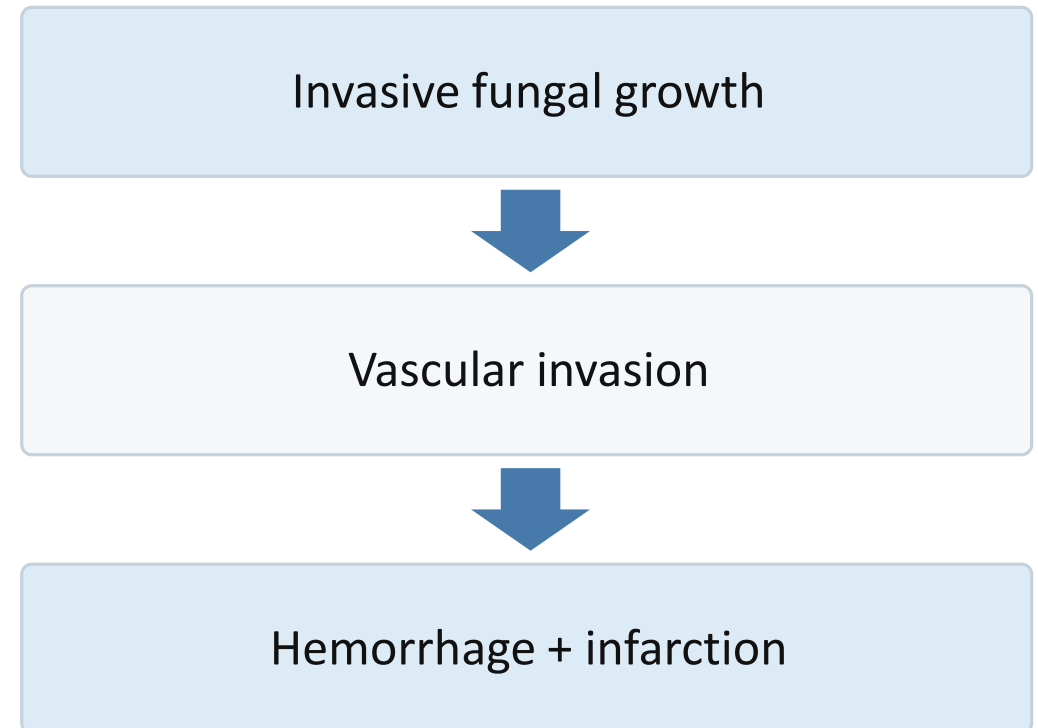
- Pseudomonas as a bacterial example of direct arterial-wall infection.
- Vessel injury can accompany an existing tissue infection or occur through hematogenous seeding.
- The organisms and source of infection require patient-specific evaluation.



Schematic

Aspergillus: Fungal infection with vascular tropism

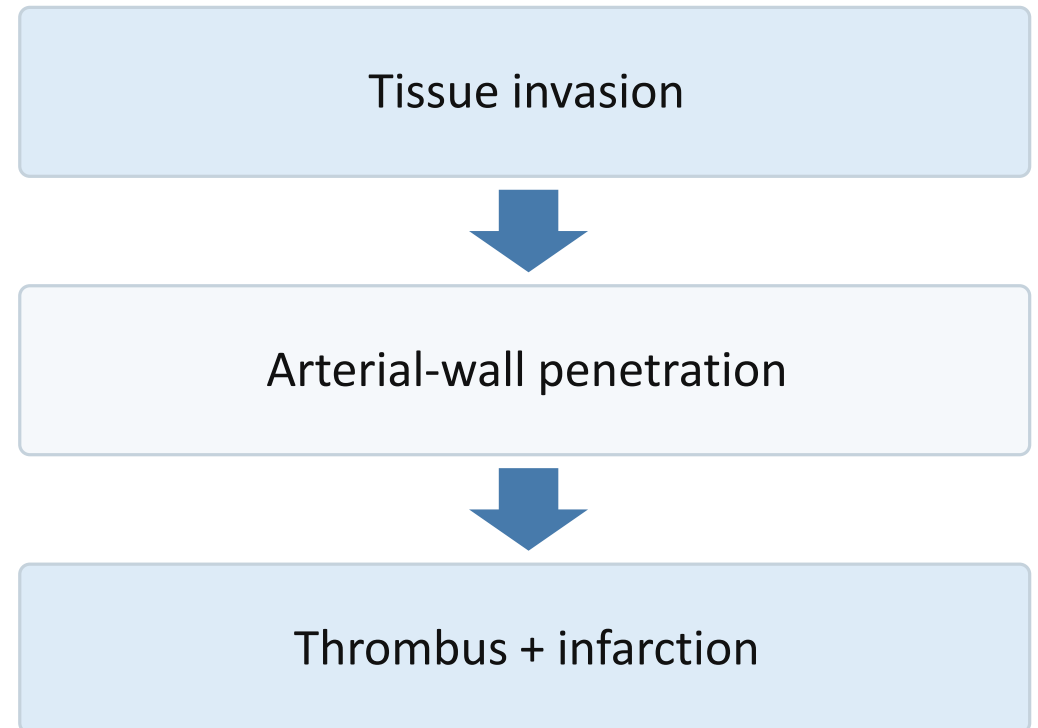
- Invasive aspergillosis occurs in immunosuppressed hosts.
- Aspergillus has a propensity for vascular invasion.
- Hemorrhage and infarction often accompany necrotizing infection.



Schematic

Mucor species: Vascular injury in invasive infection

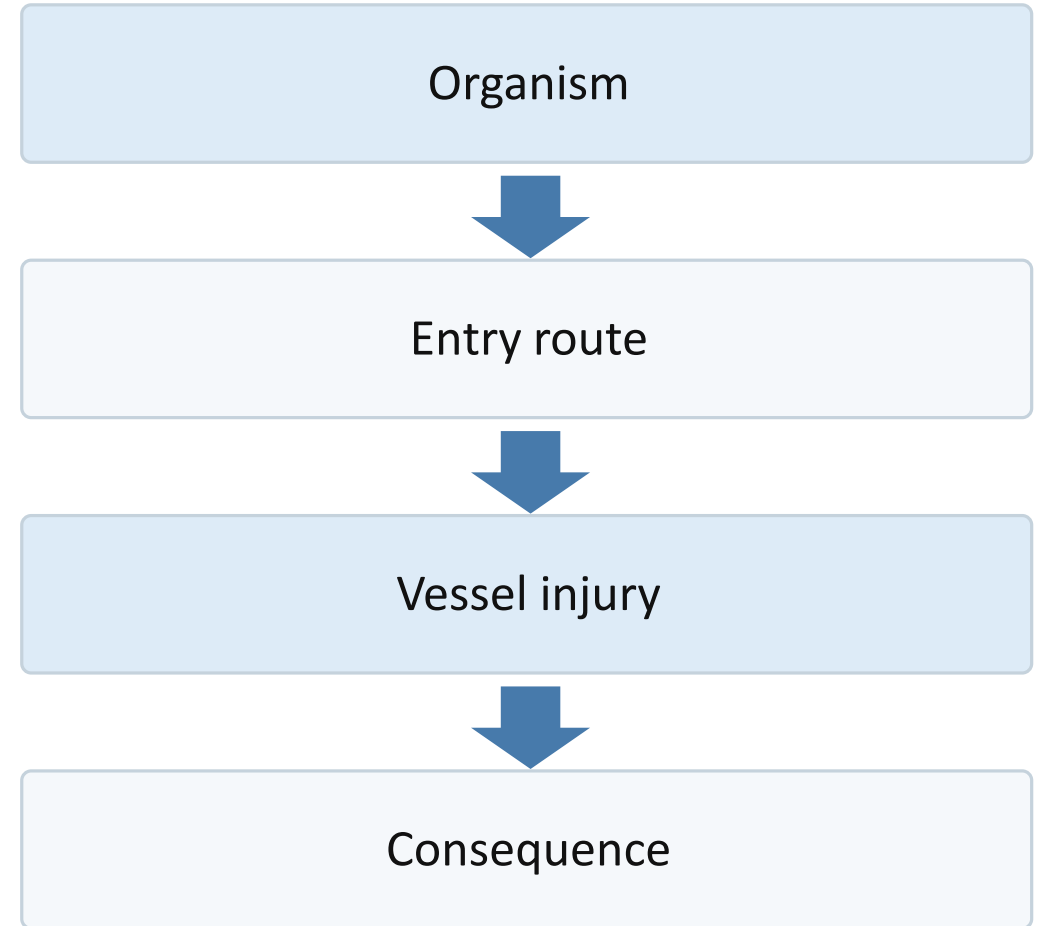
- Invasion of arterial walls by mucormycetes.
- Thrombosis can produce cerebral or pulmonary infarction.
- Anatomic spread from the nasal sinuses may affect orbit and brain.



Schematic

A microbial label is not a complete mechanism

- “Infectious vasculitis” specifies direct wall invasion, not merely a positive blood culture.
- Different pathogens may converge on thrombosis or aneurysmal wall damage.
- Always describe organism, entry route, involved vessel and downstream lesion separately.



Schematic

Two anatomic routes deliver microbes to vessels

- Contiguous spread begins in an infected neighboring tissue.
- Hematogenous spread brings organisms through the bloodstream.
- Local spread as a familiar setting and hematogenous seeding as less common.

Local pneumonia / abscess

Contiguous invasion

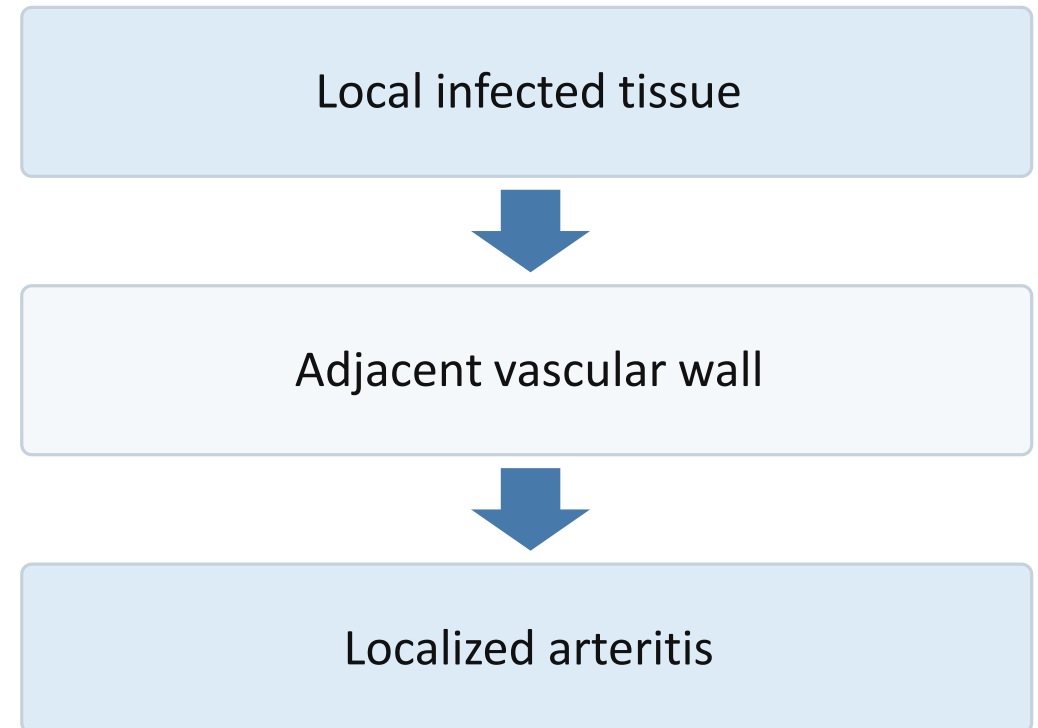
Septicemia / septic embolus

Blood-borne seeding

Schematic

Local spread: Infection abutting a vascular wall

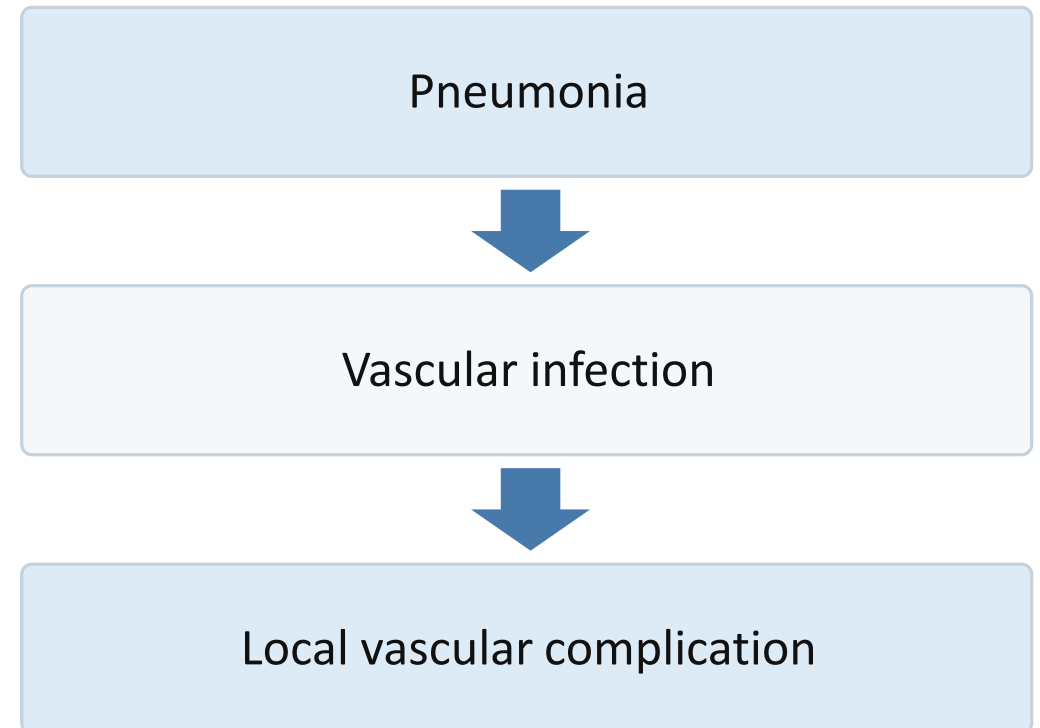
- Bacterial pneumonia can extend into nearby vascular structures.
- An abscess is another source of adjacent microbial invasion.
- The spatial relationship between infected tissue and vessel matters.



Schematic

Pneumonia-associated vascular invasion

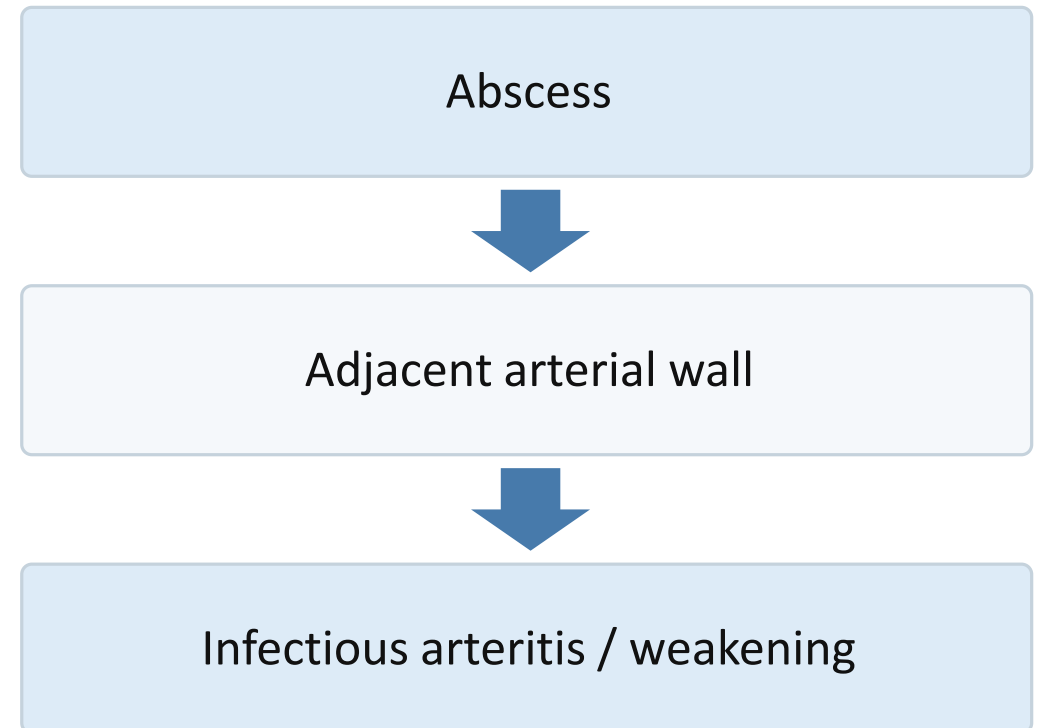
- Pneumonic inflammation is an example of a local infectious source.
- Microbes can invade vessels within the infected tissue field.
- Vascular injury may then compound parenchymal tissue damage.



Schematic

Abscess-associated vascular invasion

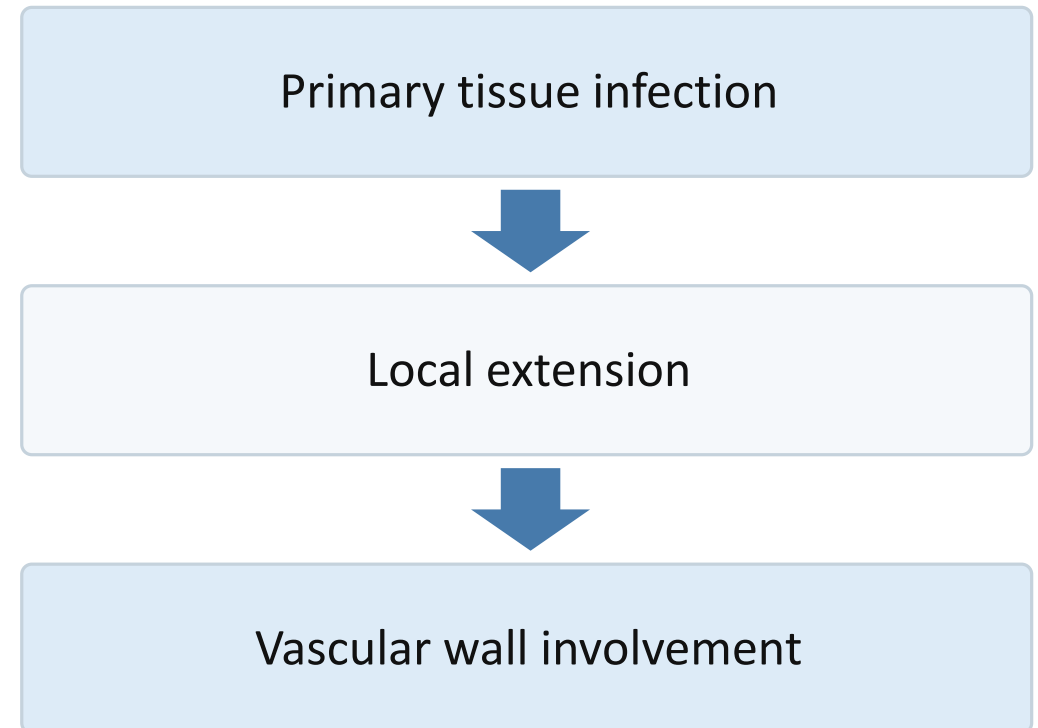
- An abscess contains an infectious inflammatory focus.
- A nearby artery may be reached by extension of the suppurative process.
- The same extension pathway can contribute to an infected aneurysm.



Schematic

Contiguous spread is an anatomical sequence

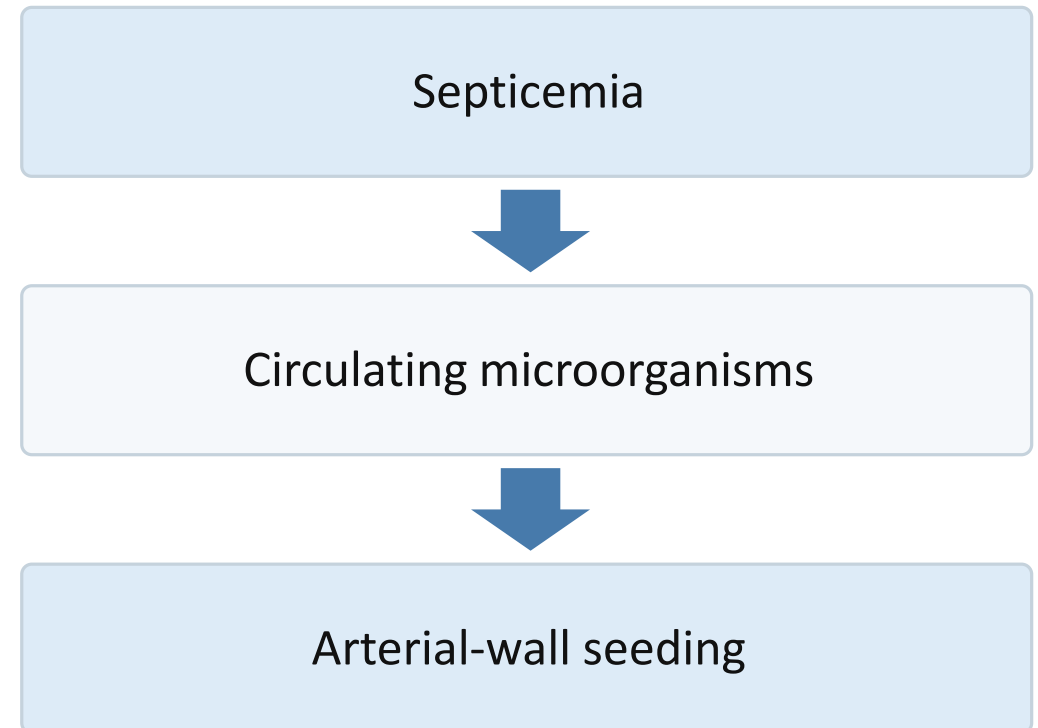
- The original infection need not begin in the vessel.
- Direct invasion occurs when the infected focus reaches its wall.
- This is distinct from circulating microorganisms seeding a remote vessel.



Schematic

Hematogenous route: Organisms reach vessels in blood

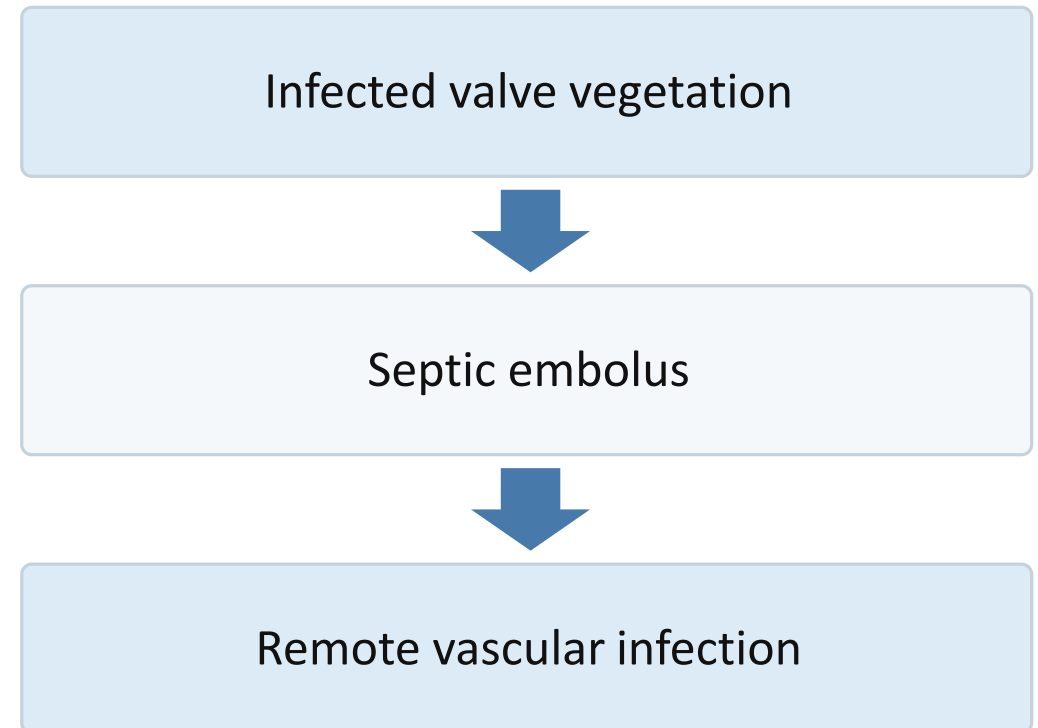
- Septicemia can distribute organisms through the circulation.
- Circulating organisms may directly infect an arterial wall.
- Blood-borne infection is a separate pathway from contiguous spread.



Schematic

Infective endocarditis supplies infected emboli

- Infective endocarditis forms organism-containing valvular vegetations.
- Friable vegetations can embolize.
- The embolic fragments may infect arterial sites where they lodge.



Schematic

An infected embolus can injure two compartments

- The embolus may occlude an arterial lumen.
- Its organisms can also establish infection at the lodging site.
- Distinguish mechanical obstruction from subsequent destructive infection.

Luminal obstruction

Ischemic consequence

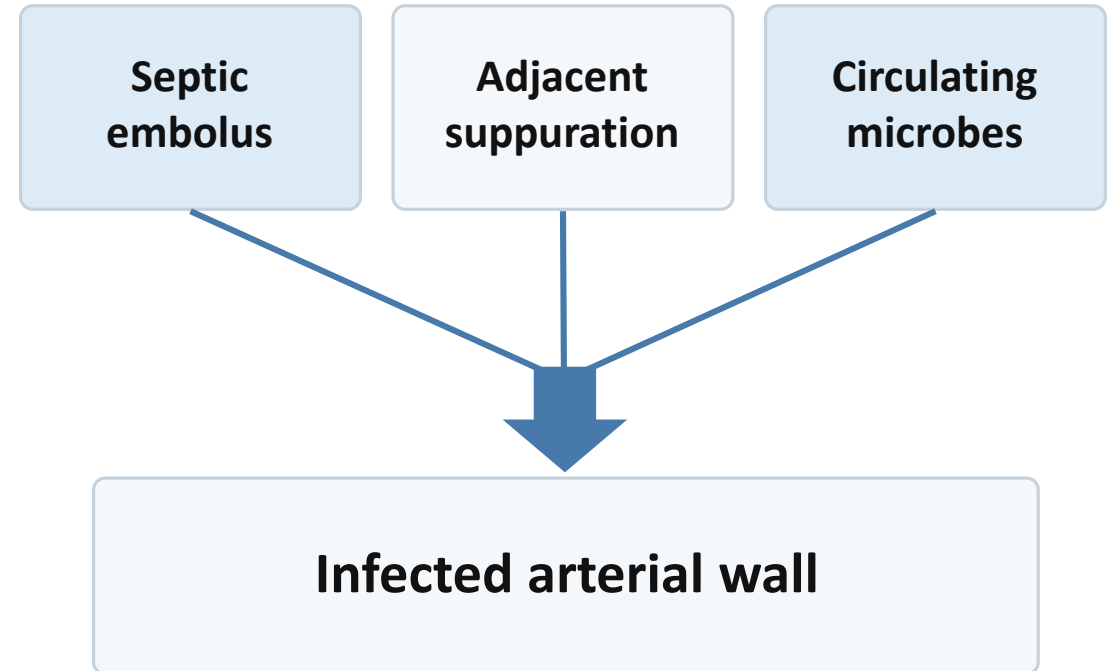
Microbial seeding

Infected vessel wall

Schematic

Three origins of a mycotic aneurysm

- Septic embolus, commonly from infective endocarditis.
- Direct extension of an adjacent suppurative focus.
- Circulating microorganisms infecting the arterial wall.



Schematic — three alternative routes converge on the same lesion

The word “mycotic” does not identify a fungus

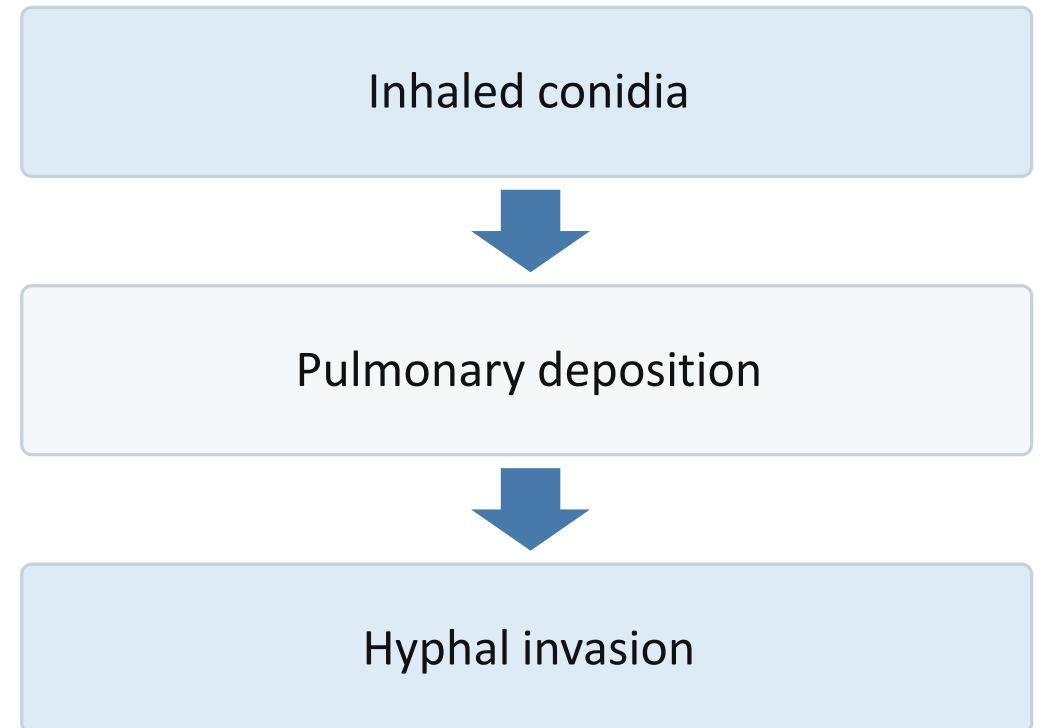
Term	What it identifies	What it does not prove
Infectious arteritis	Infected arterial wall	A single causative organism
Mycotic aneurysm	Infection-associated aneurysm	Fungal infection specifically

Fungal angioinvasion adds organ-specific mechanisms

Fungus	Important portal / territory	Vascular consequence
Aspergillus	Lung; later dissemination	Hemorrhage or infarction
Mucormycetes	Sinus–orbit–brain; lung	Thrombi and infarction

Aspergillus enters the host as inhaled conidia

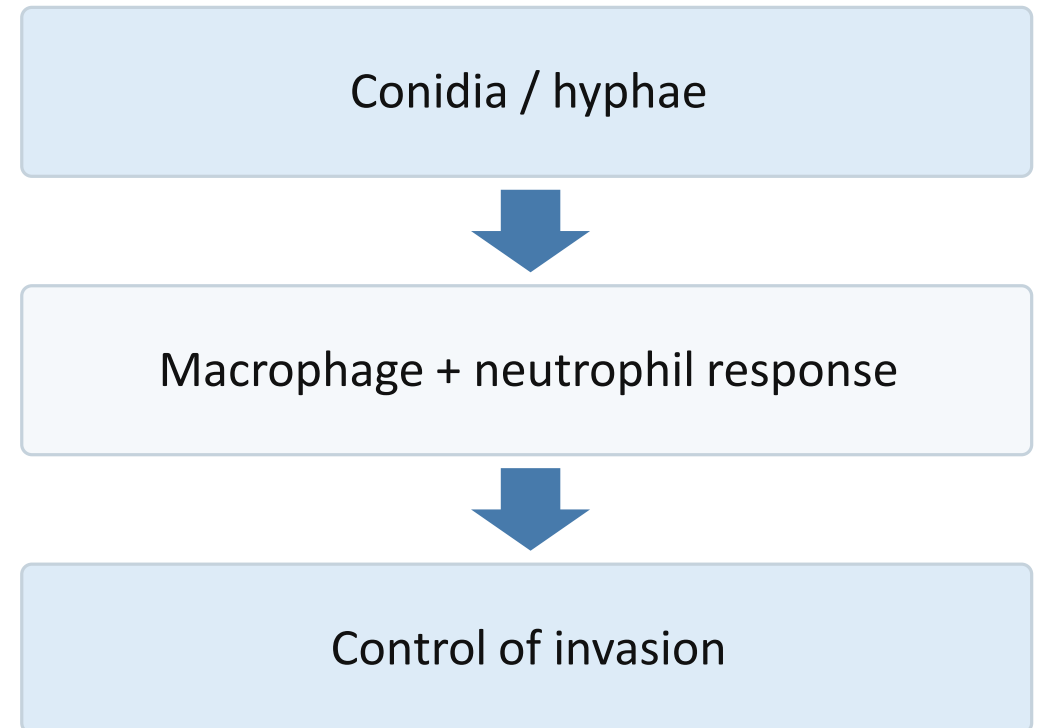
- The pulmonary tract is the major portal of entry.
- Inhaled conidia can reach distal airspaces.
- Invasive disease follows germination and tissue invasion in susceptible hosts.



Schematic

Neutrophil defense normally restricts *Aspergillus*

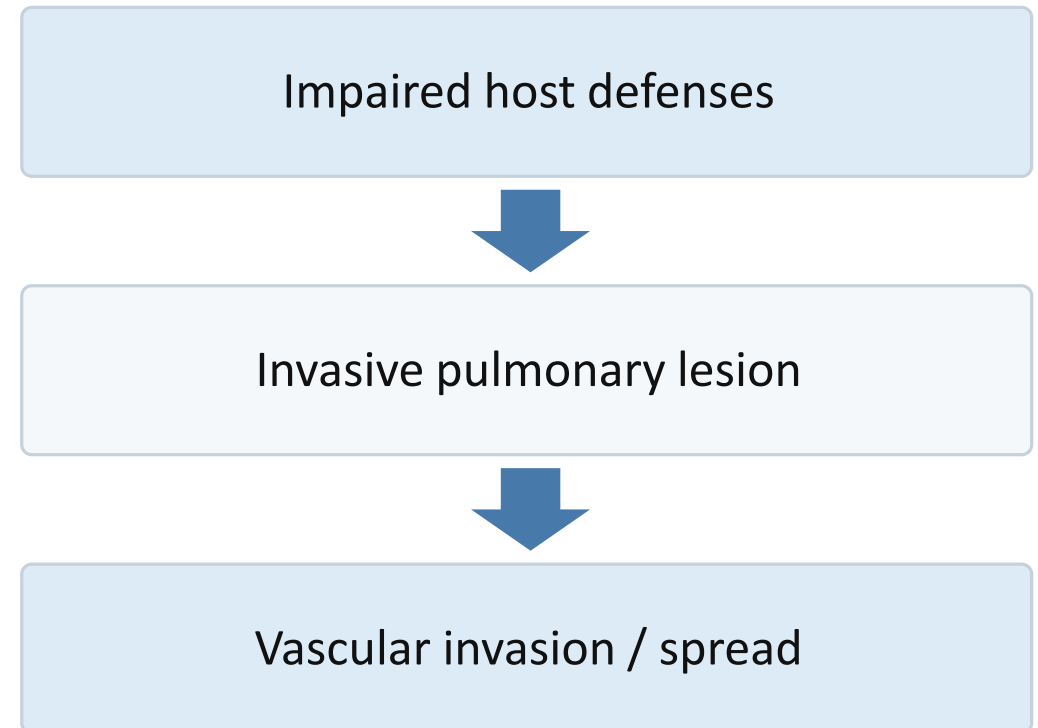
- Alveolar macrophages recognize and remove conidia.
- Neutrophils help kill fungal hyphae.
- Neutropenia and impaired neutrophil function favor invasive infection.



Schematic

Invasive aspergillosis centers on tissue penetration

- Invasive aspergillosis is an opportunistic infection in immunosuppressed hosts.
- Lung lesions commonly show necrotizing pneumonia.
- Its vascular tropism permits infarction and hematogenous dissemination.



Schematic

Pulmonary aspergillosis: The necrotizing target lesion

- Sharply demarcated rounded gray foci.
- The lesions have hemorrhagic borders.
- Vascular invasion explains associated hemorrhage and infarction.

Necrotizing focus

Rounded gray pulmonary lesion

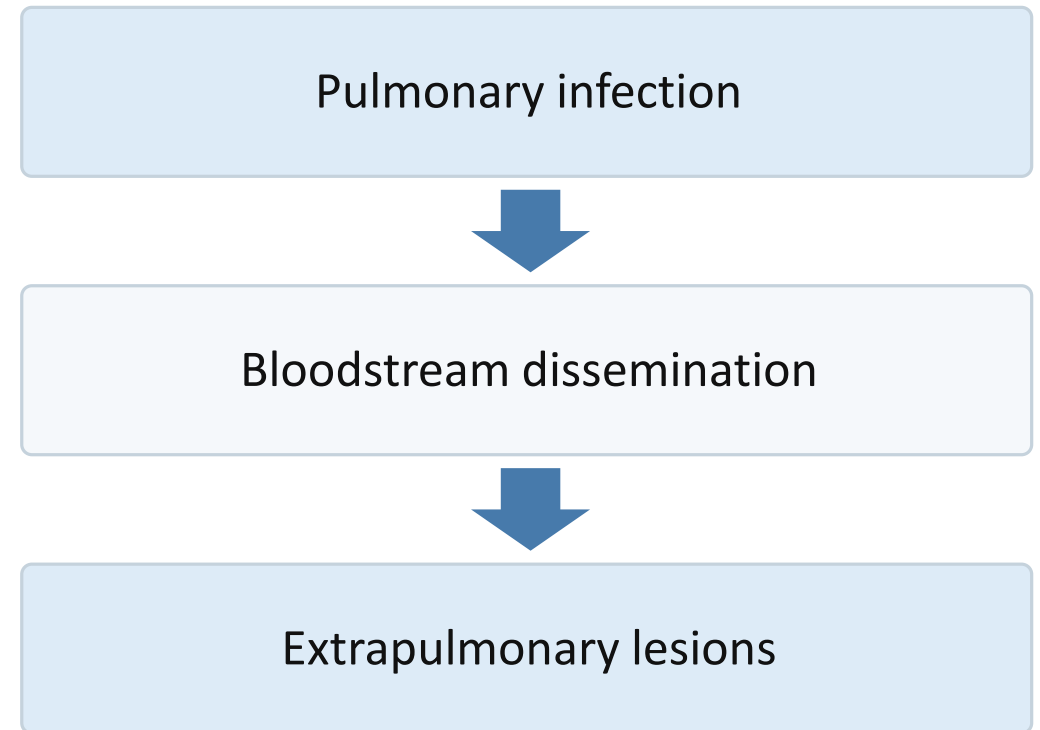
Hemorrhagic peripheral border

Associated vascular injury

Schematic

Aspergillus vascular invasion enables dissemination

- Fungal hyphae invade vascular structures in the primary lesion.
- Hematogenous spread can extend infection beyond the lung.
- Skin, brain, eyes, liver and kidneys among affected sites.

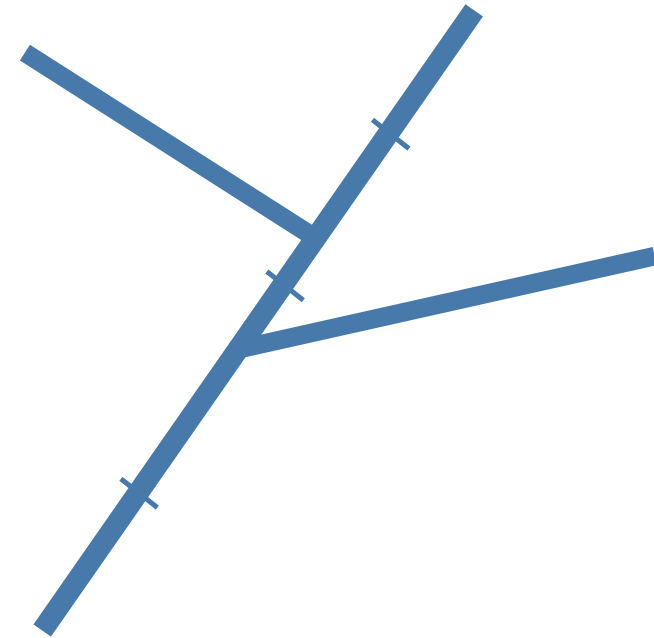


Schematic

Aspergillus histomorphology: Septate hyphae

- Septate filaments with acute-angle branching.
- The pattern is useful for recognizing an angioinvasive mold in tissue.
- Observe whether fungi are within vessel walls or lumina, not only nearby parenchyma.

Aspergillus hyphae



Septate / acute-angle

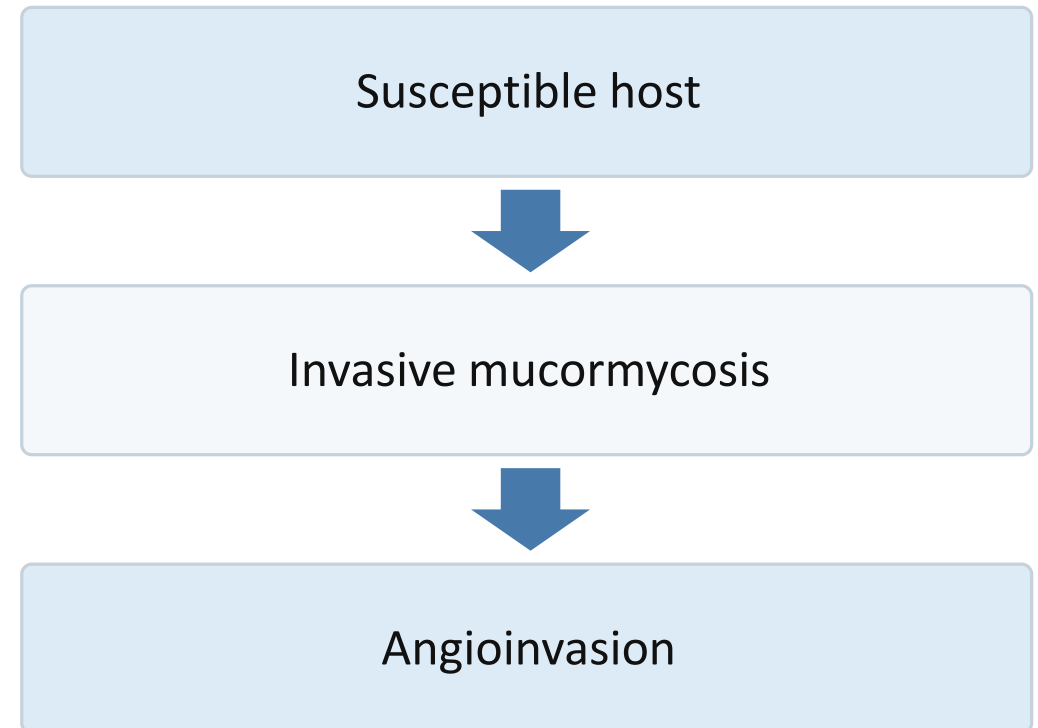
Simplified original schematic; Not a tissue photomicrograph

Fungal morphology alone has diagnostic limits

Observation	Supports	Does not establish
Septate acute-angle hyphae	Compatible Aspergillus pattern	Definitive organism species
Hyphae inside a vessel	True angioinvasion	Every underlying predisposing factor

Mucormycosis particularly affects susceptible hosts

- Mucormycetes cause opportunistic invasive disease.
- Immunosuppression and diabetes with ketoacidosis in relevant presentations.
- Their ability to invade arteries drives major tissue injury.



Schematic

Mucormycete entry sites determine the disease field

- Primary invasion may involve nasal sinuses, lungs or gastrointestinal tract.
- The route depends on inhaled or ingested spores.
- The involved site determines the adjacent vascular territories at risk.

Nasal sinuses

Rhinocerebral spread

Lungs

Pulmonary lesions

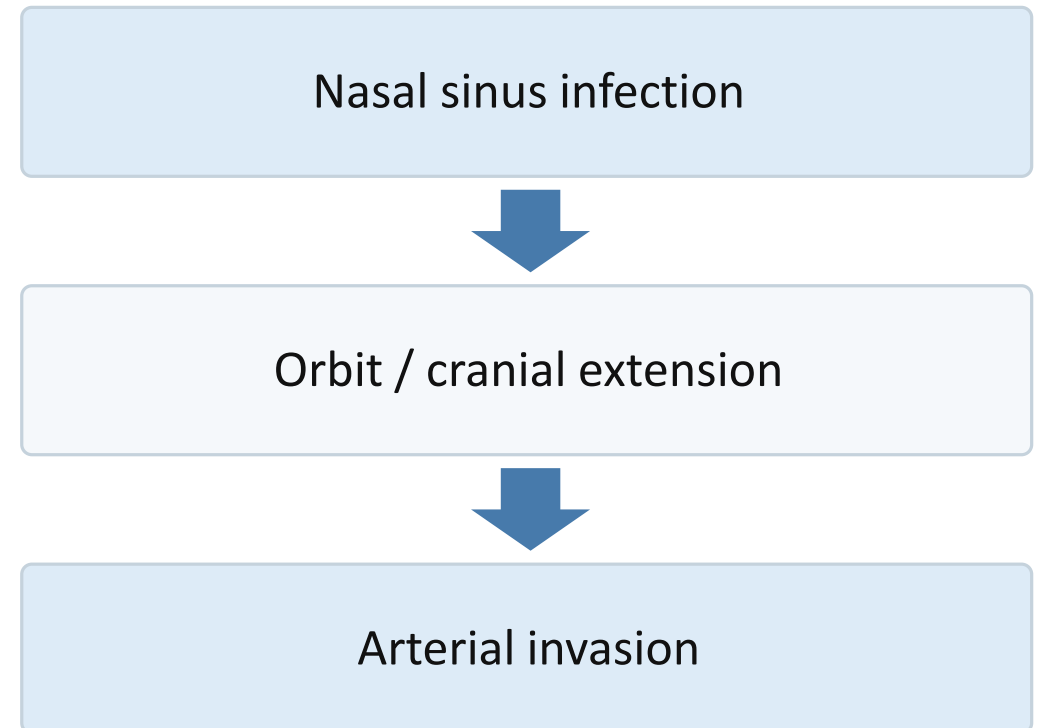
Gastrointestinal tract

Local infection

Schematic

Rhinocerebral mucormycosis follows local extension

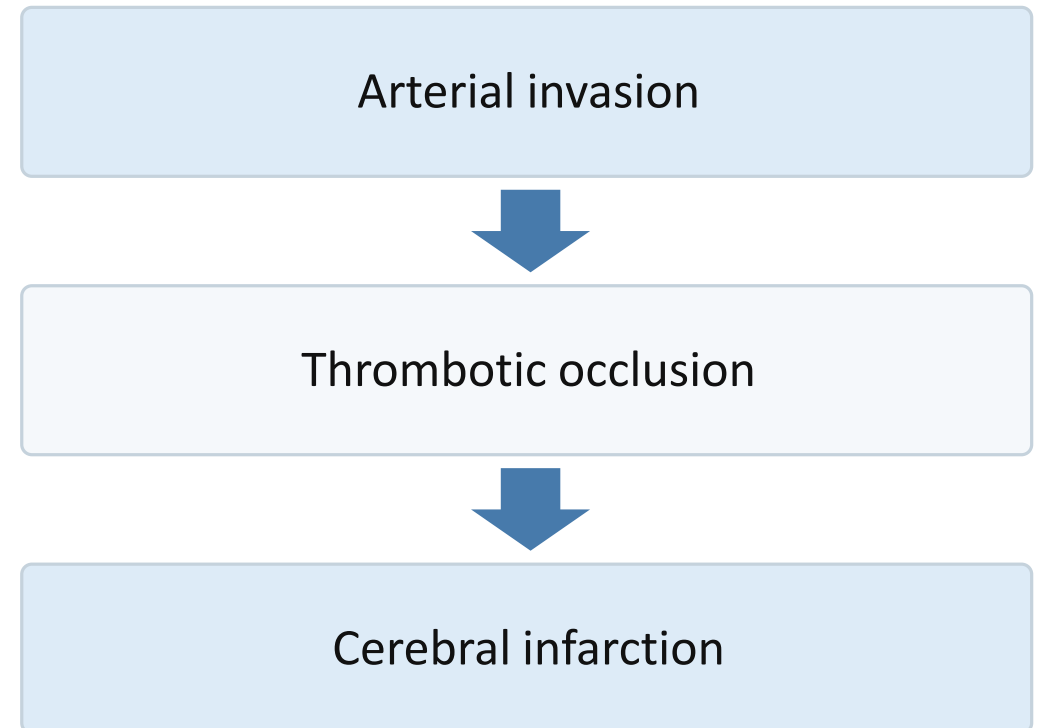
- Invasion may advance from the nasal sinuses toward the orbit and brain.
- The organism can cross into periorbital tissues and cranial structures.
- Infected cerebral arteries may become thrombosed.



Schematic

Cerebral infarction in mucormycosis

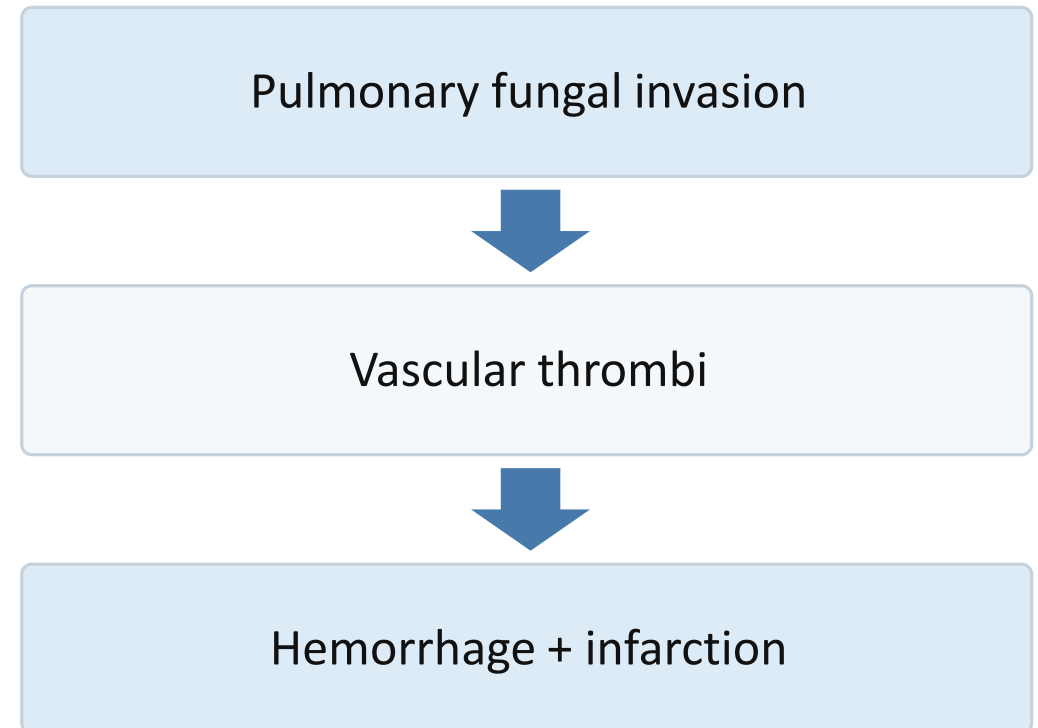
- Mucormycetes penetrate arterial walls.
- Intravascular thrombosis may obstruct cerebral blood supply.
- Brain infarction can accompany meningoencephalitis.



Schematic

Pulmonary mucormycosis: Vascular thrombi

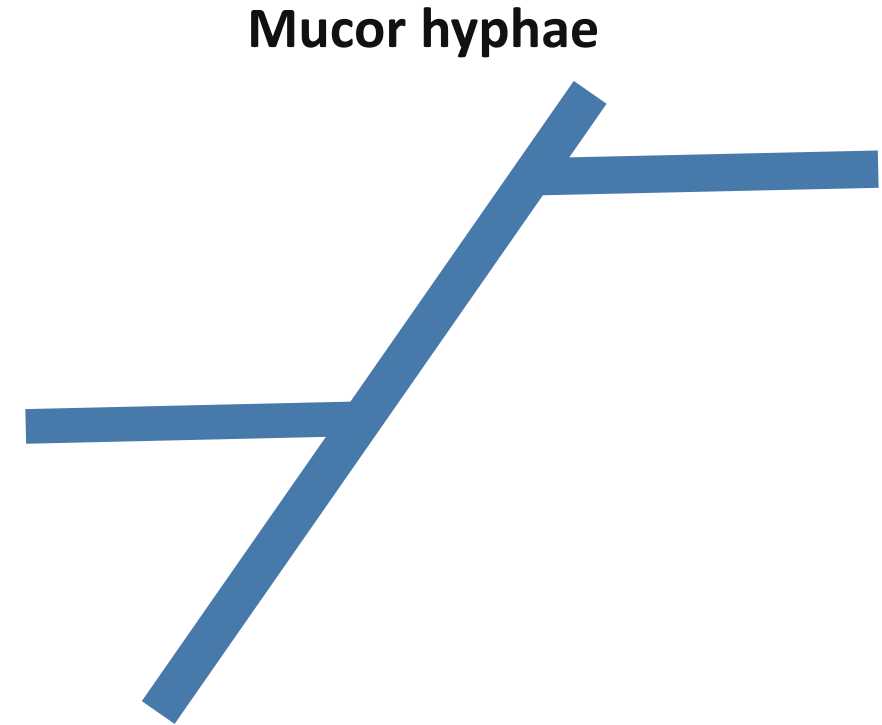
- Lung disease may be primary in severe immunodeficiency.
- Hemorrhagic pneumonia can coexist with vascular thrombosis.
- Distal infarction follows compromised arterial perfusion.



Schematic

Mucormycete morphology: Broad irregular hyphae

- Nonseptate hyphae with irregular width.
- Near right-angle branching contrasts with the Aspergillus pattern.
- Angioinvasive fungi in meningeal vessels.



Nonseptate / near right-angle

Simplified original schematic; Not a tissue photomicrograph

Aspergillus versus Mucor: Tissue comparison

Feature	Aspergillus	Mucormycetes
Hyphal septation	Septate	Nonseptate
Branching	Acute angles	Often near 90°
Vascular effect	Hemorrhage, infarction	Thrombosis, infarction

Vascular injury produces two major outcomes

- Wall destruction and weakening predispose to aneurysm formation.
- Inflammation-associated thrombosis compromises downstream blood flow.
- These are distinct, potentially coexisting complications.

Wall weakening

Infected aneurysm

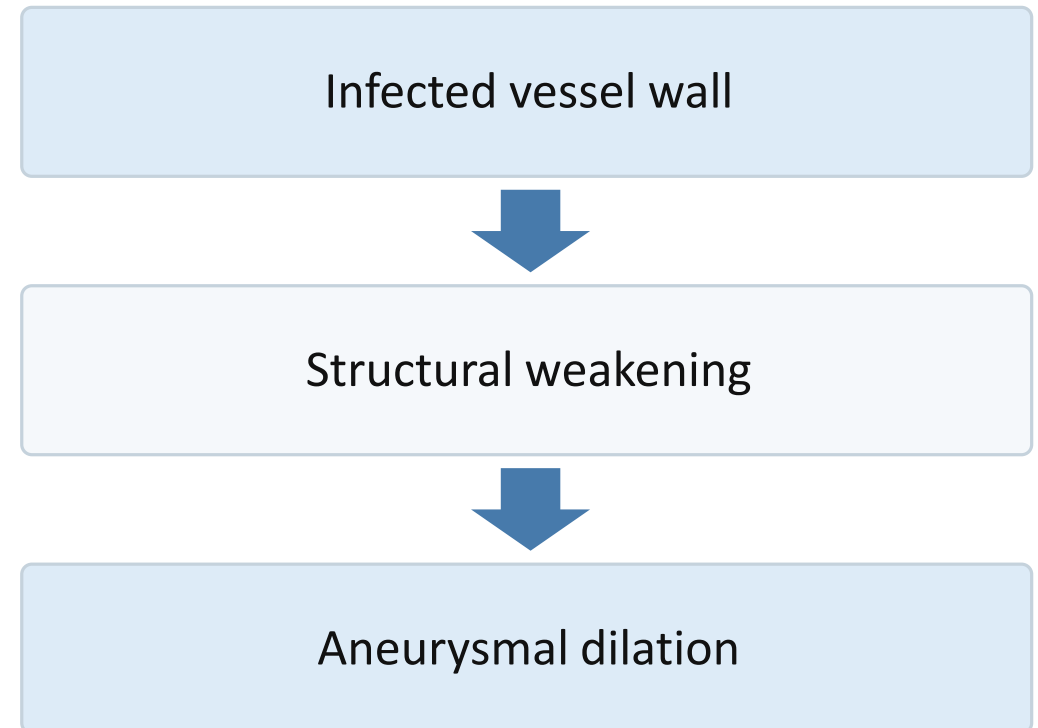
Luminal thrombosis

Downstream infarction

Schematic

Infected aneurysm: Failure of arterial wall integrity

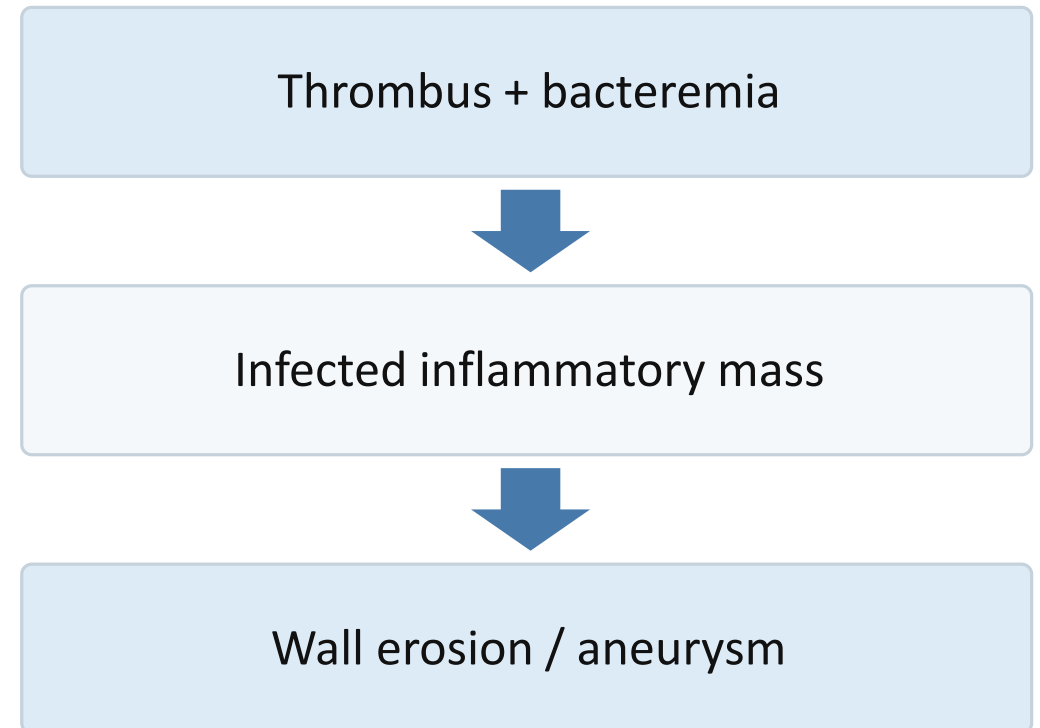
- Direct infection weakens the normal vessel-wall structure.
- Loss of mechanical integrity can permit abnormal dilation.
- An infection-associated aneurysm is termed mycotic.



Schematic

An infected thrombus can erode the artery

- Thrombi becoming infected during bacteremia.
- An infected thrombus can form an inflammatory mass.
- Erosion and weakening of the wall may produce a mycotic aneurysm.



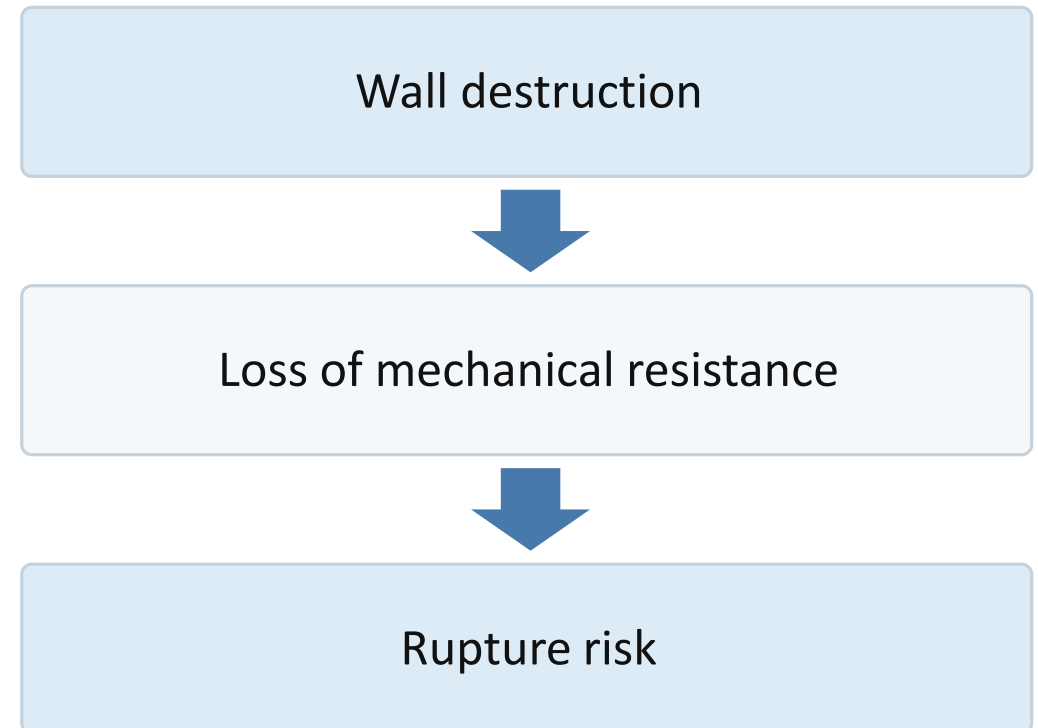
Schematic

Aneurysm and pseudoaneurysm are not synonymous

Lesion	Defining structural relationship
True aneurysm	Dilated vessel wall remains part of sac
Pseudoaneurysm	Wall breach; blood outside wall communicates with lumen

Why an infected aneurysm is dangerous

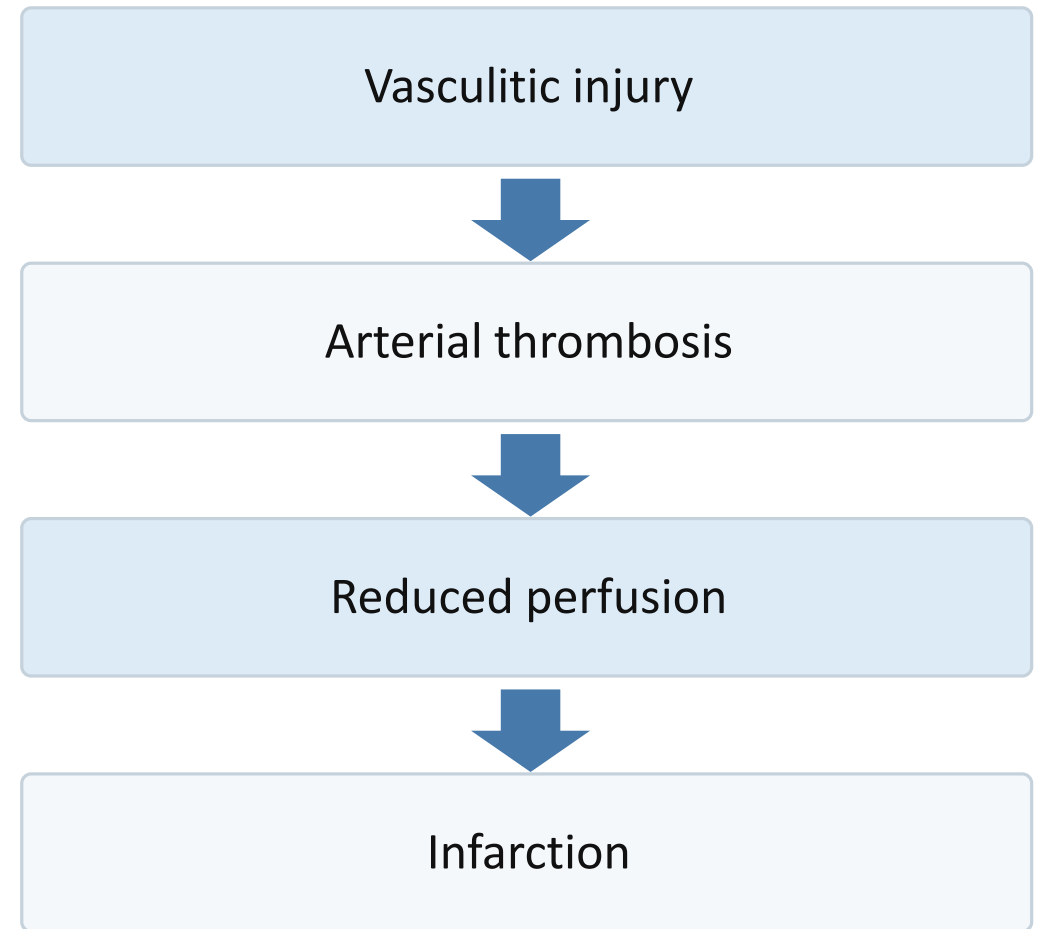
- Arterial wall damage reduces resistance to hemodynamic stress.
- Progressive disruption may predispose to rupture.
- Location and the involved vessel determine clinical severity.



Schematic

Thrombosis: The alternative vascular pathway

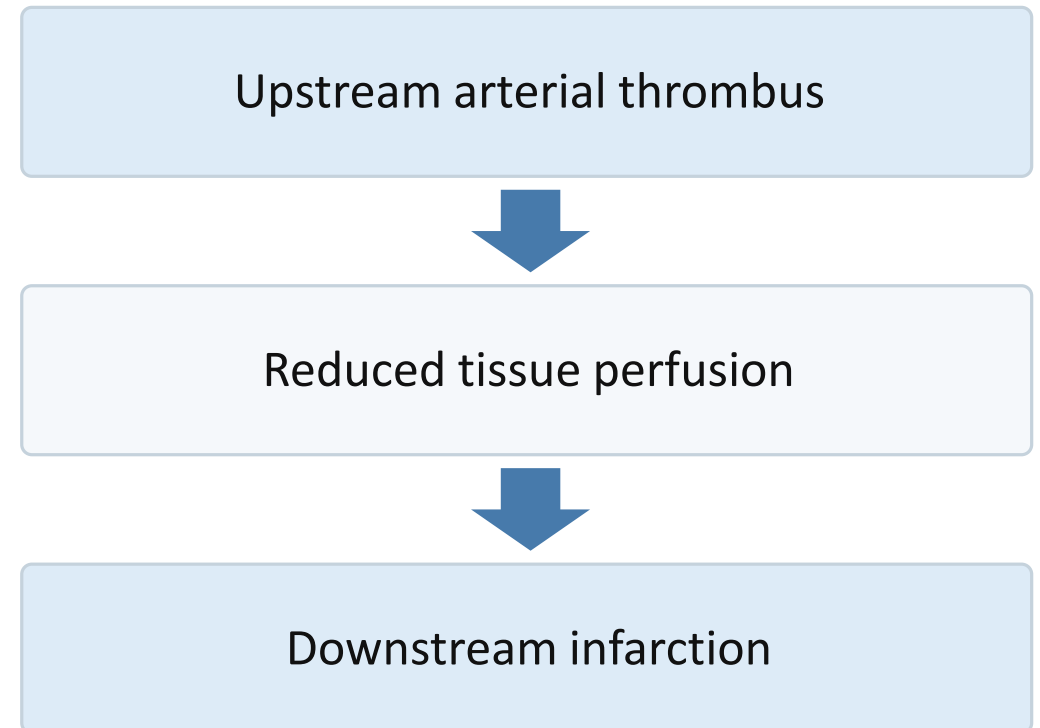
- Infectious inflammation can promote formation of a luminal thrombus.
- The thrombus may restrict or obstruct perfusion.
- Vulnerable downstream tissue can become infarcted.



Schematic

A thrombus is a local lesion; Infarction is downstream

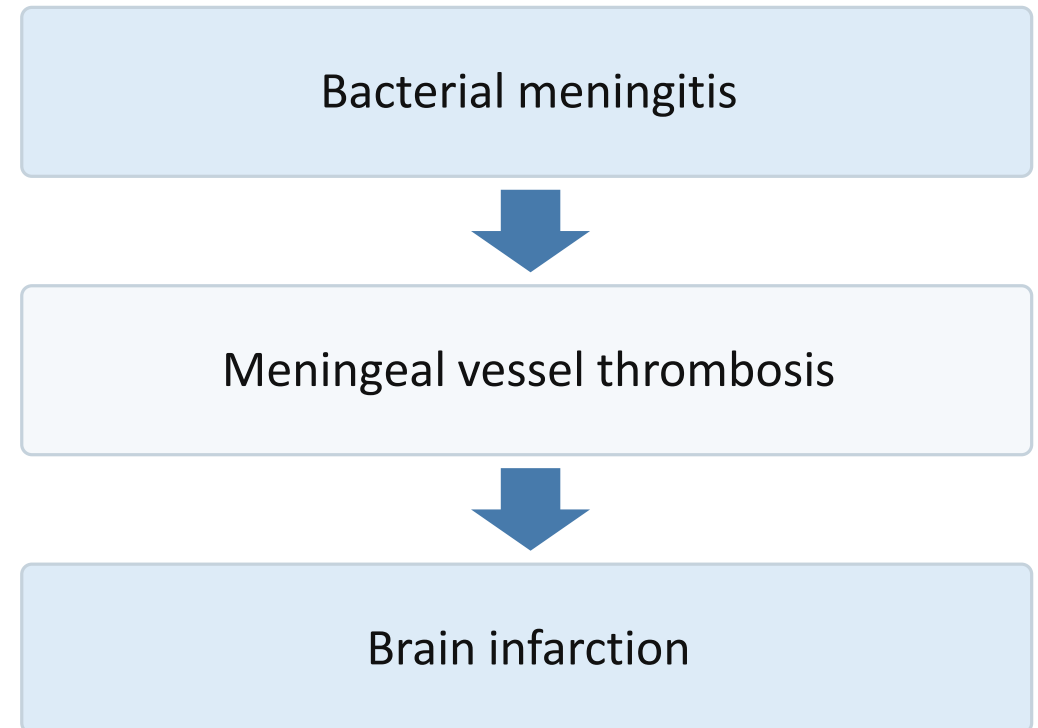
- The obstructing event is in the vessel lumen.
- The tissue injury develops in the supplied vascular territory.
- Identifying both sites clarifies the causal interpretation.



Schematic

Bacterial meningitis can cause meningeal vasculitis

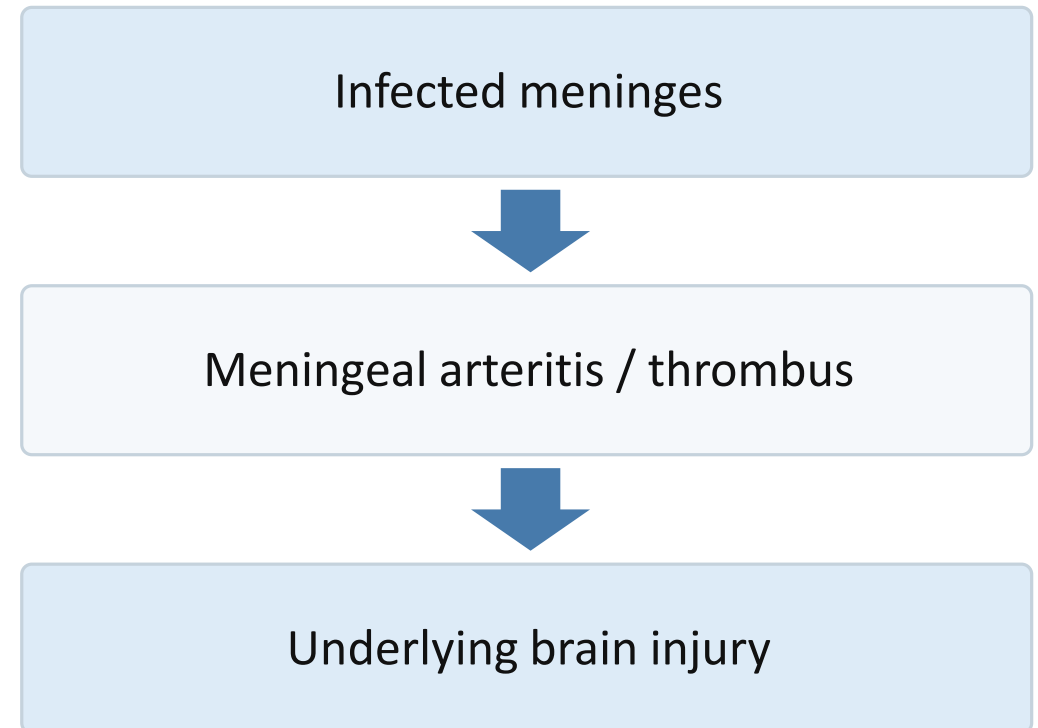
- Inflammation can involve meningeal vessels during bacterial meningitis.
- Associated thrombosis may reduce perfusion of the underlying brain.
- Cerebral infarction is a recognized complication.



Schematic

The meningitis example links adjacent compartments

- The infection is centered in meningeal structures.
- Vessel-wall inflammation extends the injury to the vascular compartment.
- Infarction involves underlying brain supplied by affected vessels.



Schematic

Hemorrhage and infarction can occur together

- Angioinvasive fungal lesions can produce hemorrhagic tissue damage.
- Vascular occlusion simultaneously favors ischemic necrosis.
- This combination in invasive pulmonary mold disease.

Vessel-wall damage

Hemorrhage

Vascular occlusion

Ischemic infarction

Schematic

Compare four source-supported clinical settings

Setting	Route of vessel injury	Consequence
Abscess / pneumonia	Local extension	Infected arteritis
Endocarditis	Septic embolization	Infected aneurysm
Bacterial meningitis	Meningeal inflammation	Brain infarction
Angioinvasive molds	Tissue / wall penetration	Thrombi, hemorrhage, infarction

Interpretation requires locating the infected wall

- Infected tissue adjacent to a vessel is not automatically proof of vessel-wall invasion.
- A finding of intravascular or wall-associated organisms is stronger evidence of angioinvasion.
- Never label a synthetic diagram as a histological finding.

Adjacent infection

Does not alone prove angioinvasion

Organisms invading vessel wall

Supports direct infectious vasculitis

Schematic

Distinguishing mechanisms at the microscope

Key question	Direct infectious mechanism	Indirect immune mechanism
What drives injury?	Microbial invasion	Immune effector response
Required causal link	Organisms reach wall	Infection triggers immunity
Pitfall	Mistaking adjacency for invasion	Assuming every postinfection vasculitis is microbial

Clinical exercise: Infarction after bacterial meningitis

HYPOTHETICAL TEACHING CASE

Bacterial meningitis → new focal neurological deficit

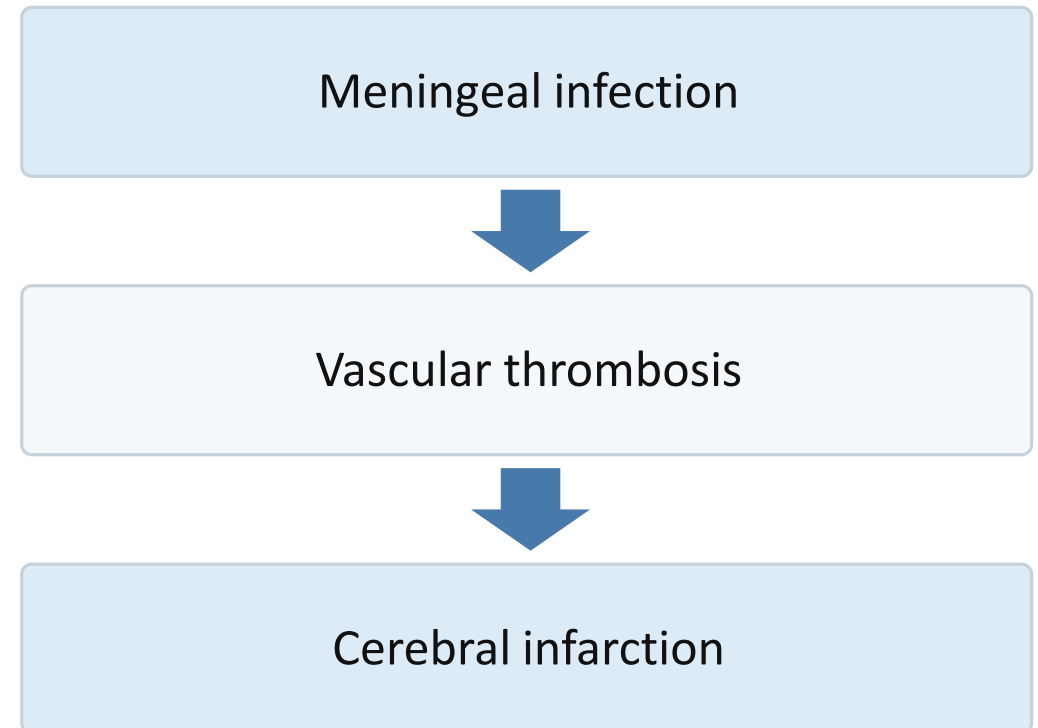
PATHOLOGY QUESTION

Which vascular event could connect these findings?

Apply the source-based pathogenesis to this illustrative scenario.

Case interpretation: Meningeal thrombosis

- Meningeal infection can cause inflammation involving meningeal vessels.
- Thrombotic occlusion may interrupt supply to the underlying brain.
- This mechanism can produce infarction rather than only diffuse inflammation.



Schematic

Clinical exercise: An embolizing valve vegetation

HYPOTHETICAL TEACHING CASE

Infected valve vegetation → septic
arterial embolus

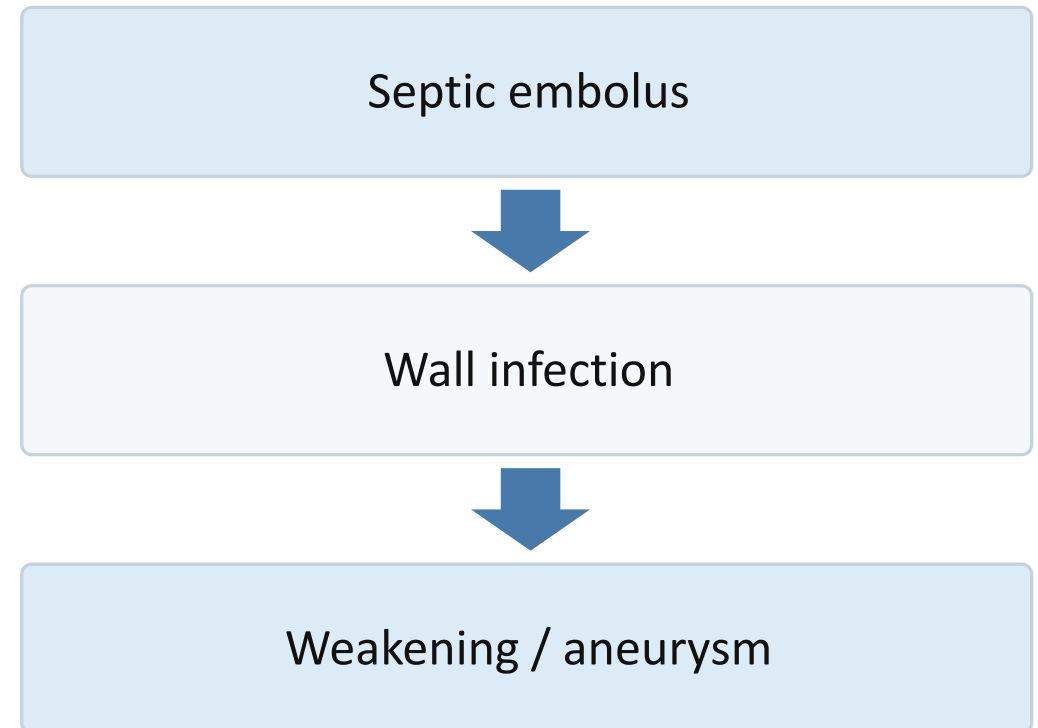
PATHOLOGY QUESTION

How could this cause aneurysmal
enlargement?

Apply the source-based pathogenesis to this illustrative scenario.

Case interpretation: Infected aneurysm

- An embolus containing organisms lodges in an artery.
- Local infection and inflammation can damage the vessel wall.
- Loss of structural integrity favors a mycotic aneurysm.



Schematic

Clinical exercise: Necrotizing pulmonary aspergillosis

HYPOTHETICAL TEACHING CASE

Immunosuppressed host →
pulmonary necrotizing focus

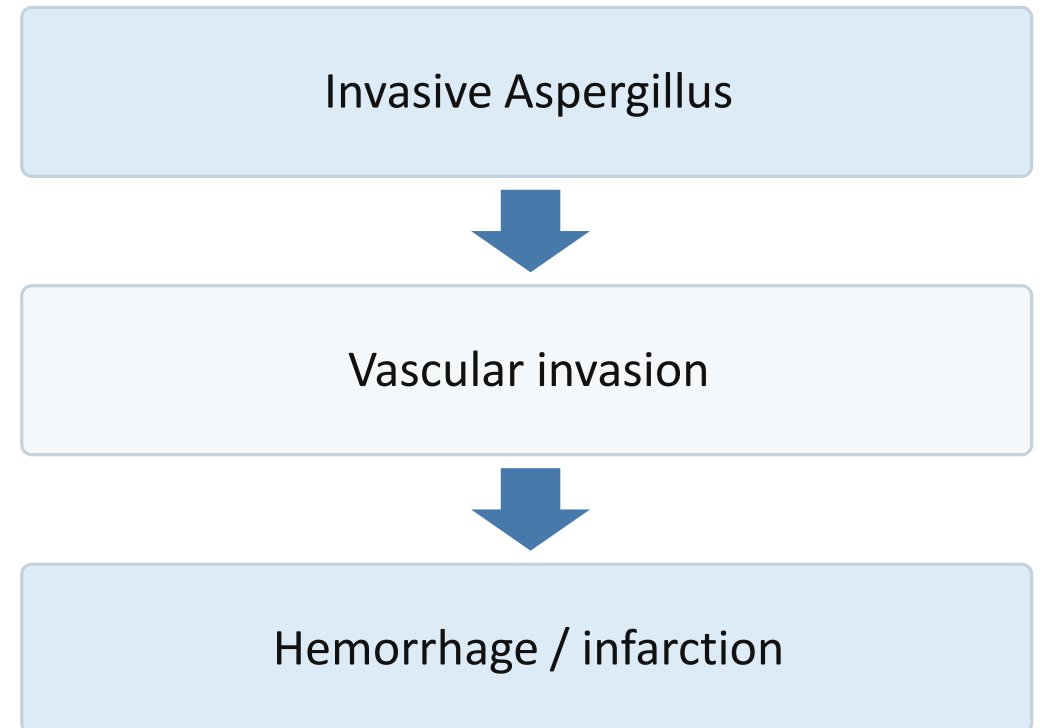
PATHOLOGY QUESTION

What mechanism can explain
hemorrhage plus necrosis?

Apply the source-based pathogenesis to this illustrative scenario.

Case interpretation: Fungal vascular invasion

- Hyphal tissue invasion reaches vessel walls.
- Vascular compromise produces hemorrhagic and ischemic injury.
- The pathology links the necrotizing lung lesion to angioinvasion.



Schematic

Clinical exercise: Sinus-to-brain fungal infection

HYPOTHETICAL TEACHING CASE

Sinus infection → orbit / brain extension

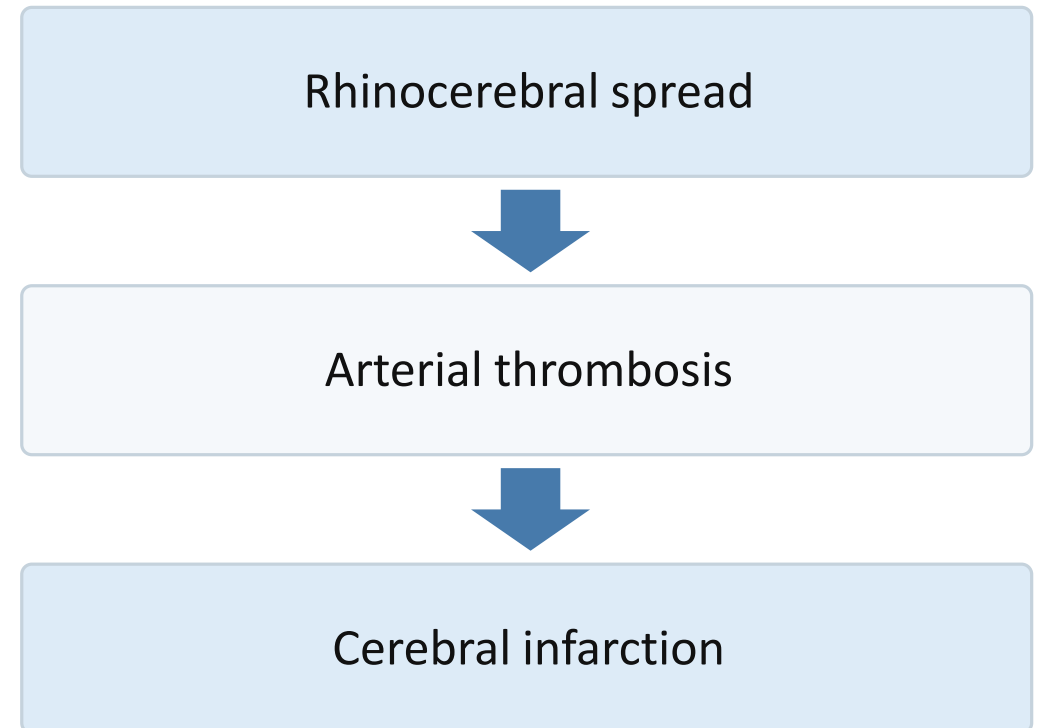
PATHOLOGY QUESTION

Why might cerebral infarction develop?

Apply the source-based pathogenesis to this illustrative scenario.

Case interpretation: Thrombosed cerebral arteries

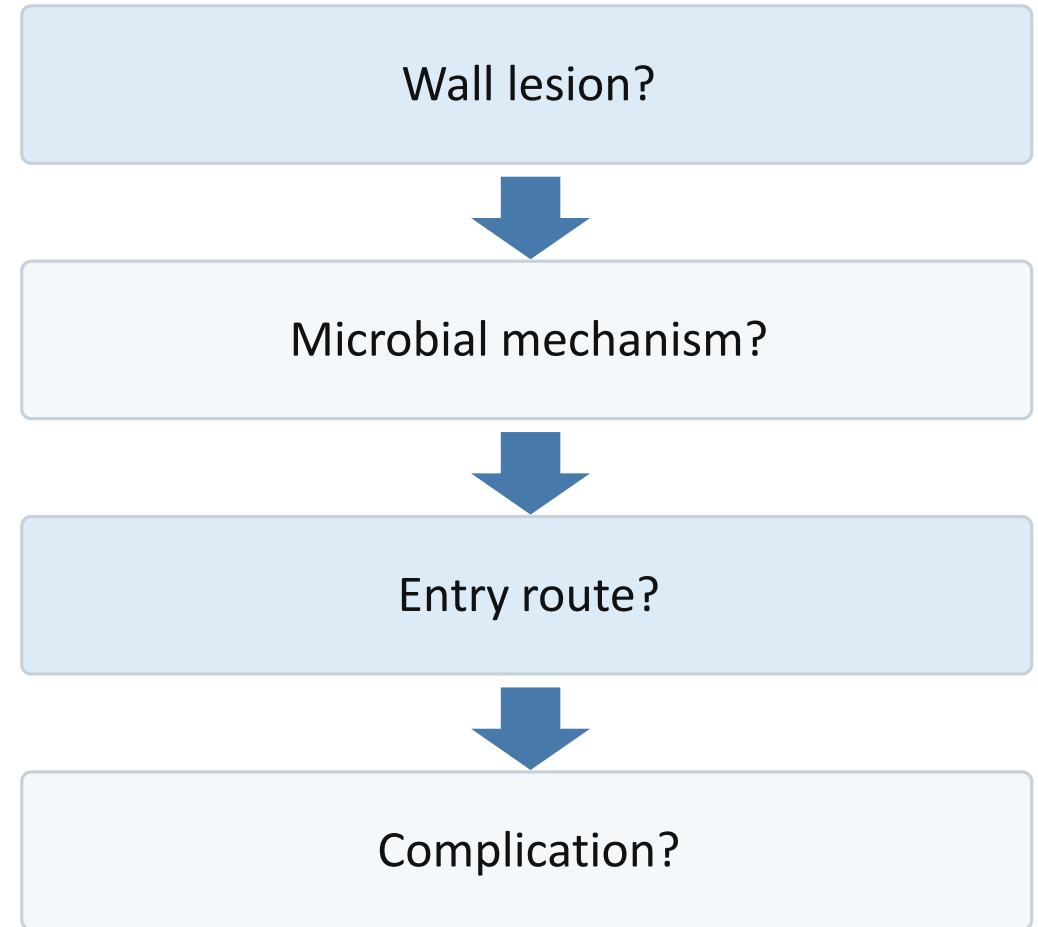
- Mucormycetes can invade cranial and meningeal vessels.
- Arterial-wall invasion promotes thrombotic obstruction.
- Downstream cerebral infarction may complicate meningoencephalitis.



Schematic

Diagnostic reasoning: Separate four questions

- Is vessel-wall inflammation actually present?
- Is direct microbial invasion documented or suspected?
- Did infection spread locally or through blood?
- Is the dominant complication aneurysm, thrombosis, or infarction?



Schematic

The most consequential diagnostic pitfall

- Systemic inflammatory symptoms alone cannot assign an infectious or immune cause.
- An infection may directly invade vessels or indirectly trigger immune vasculitis.
- The two mechanisms have substantially different therapeutic implications.

Direct infection

Treat infection as the driver

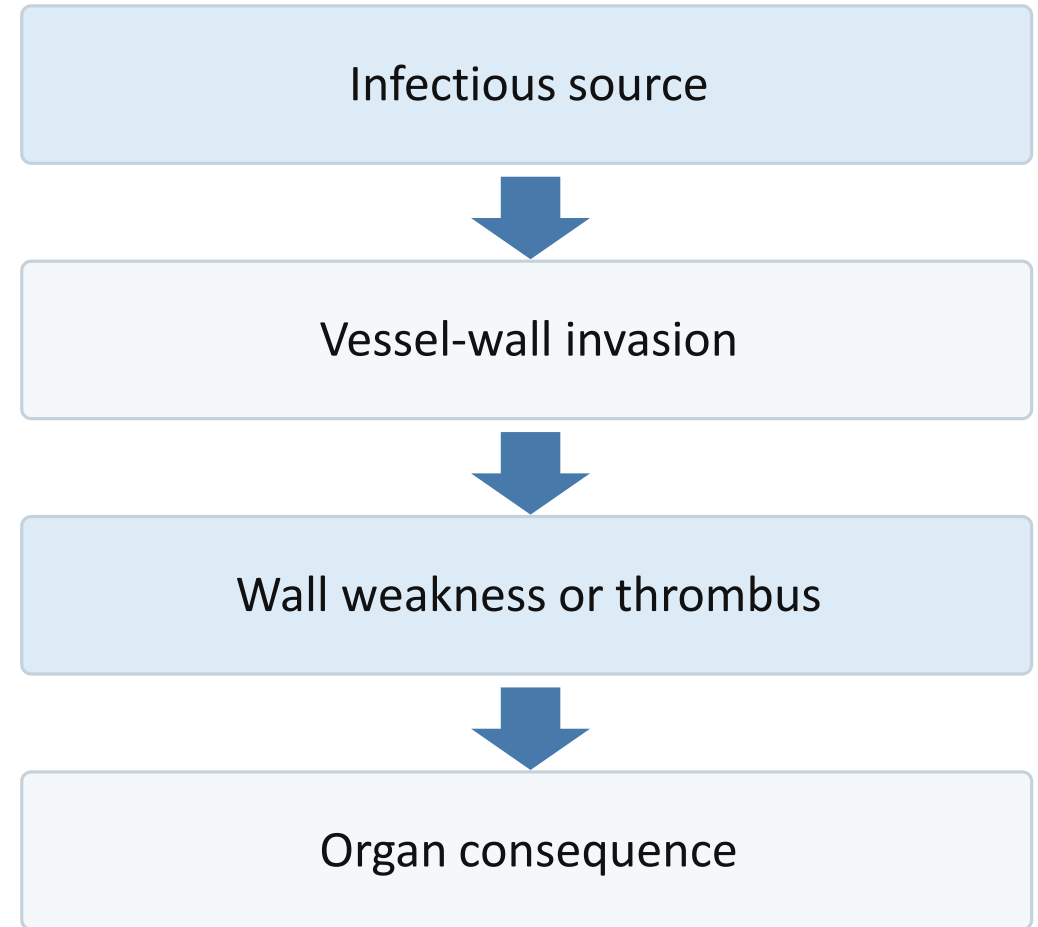
Immune process

Different management logic

Schematic

Mechanism summary: Trace the complete causal chain

- Identify the tissue source or circulating route.
- Establish direct involvement of the vessel wall.
- Recognize either loss of wall integrity or luminal occlusion.
- Predict the relevant organ consequence.



Schematic

Five facts to retain

- Direct microbial invasion distinguishes infectious arteritis from indirect immune vasculitis.
- Local extension and hematogenous seeding are principal access routes.
- Pseudomonas, Aspergillus and Mucor are Robbins examples.
- Mycotic aneurysm denotes an infection-associated lesion, not necessarily a fungus.
- Vascular thrombosis can produce downstream infarction.

Source references

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026), Ch 11 — Vasculitis and Aneurysms.
- Related Robbins source sections: Ch 8 — Aspergillosis and Mucormycosis; Ch 12 — Infective Endocarditis.
- Related Robbins source sections: Ch 4 — Thrombosis; Ch 15 — Lung Abscess.
- Exact markdown source locators and visual provenance appear in speaker notes and accompanying manifest.