

Infarction

A clinicopathologic approach to ischemic tissue necrosis

Vascular causes, red versus white infarcts, evolution and modifying factors

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Learning outcomes

- Define infarction and discriminate it from vascular obstruction without tissue death.
- Predict red or white morphology from blood supply and tissue properties.
- Describe necrosis, inflammation, repair and the four key outcome determinants.

Definition: An area of ischemic necrosis

- An infarct is a region of tissue death caused by loss of perfusion.
- The obstructed pathway can be arterial supply or venous drainage.
- Necrosis, not simply impaired blood flow, defines an infarct.

Vascular obstruction



Insufficient perfusion



Irreversible necrosis

Occlusion does not invariably cause infarction

- Some vascular obstructions produce little detectable tissue injury.
- Other occlusions cause organ dysfunction or lethal necrosis.
- The tissue outcome depends on perfusion alternatives, time and oxygen needs.

Residual perfusion



Speed of obstruction



Tissue vulnerability

Arterial supply versus venous drainage

Arterial obstruction	Venous outflow obstruction
Reduces oxygenated inflow	Causes impaired drainage / congestion
Most common infarct cause	More often causes congestion alone
Often pale in solid organs	When infarction occurs, often hemorrhagic

Why infarction is clinically important

- Myocardial and cerebral infarction cause major morbidity and death.
- Pulmonary infarction complicates some thromboembolic events.
- Bowel infarction and extremity gangrene are further tissue-specific outcomes.

Heart

Brain

Lung / bowel

Extremity

Most common proximate causes

- Arterial thrombosis and arterial embolism underlie most infarctions.
- The occlusion deprives a vascular territory of its usual supply.
- Recognition of the vascular cause explains the territorial distribution of necrosis.

Arterial thrombus / embolus



Downstream obstruction



Tissue infarction

Thrombosis: Local obstruction

- Thrombosis forms within the vasculature at or near the obstructed site.
- Local loss of arterial flow may result in downstream necrosis.
- The effect depends on how much collateral perfusion is available.

Local thrombus



Reduced downstream flow



Variable ischemic injury

Embolism: Distant obstruction

- An embolus moves from a remote source and lodges in a smaller vessel.
- Its arrival can abruptly interrupt regional arterial perfusion.
- Sudden occlusion may leave little time for compensatory collateral flow.

Embolic source



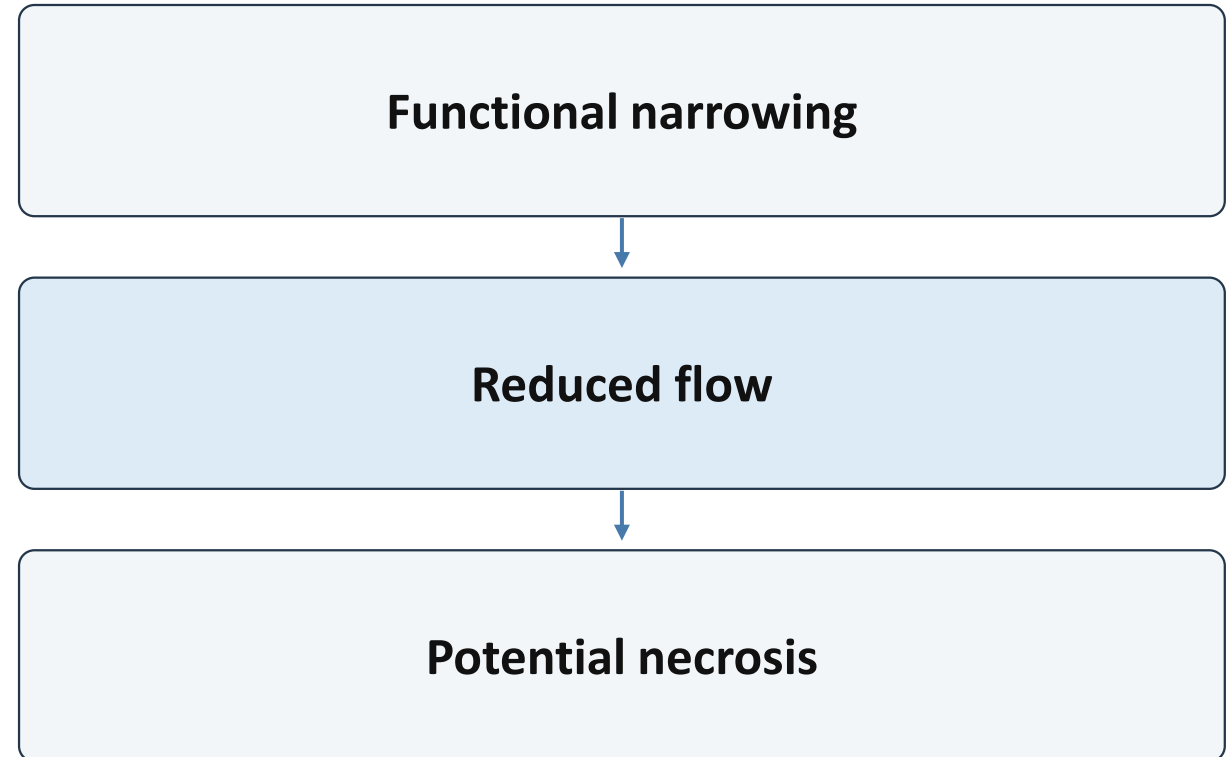
Transport in blood



Distal vascular blockage

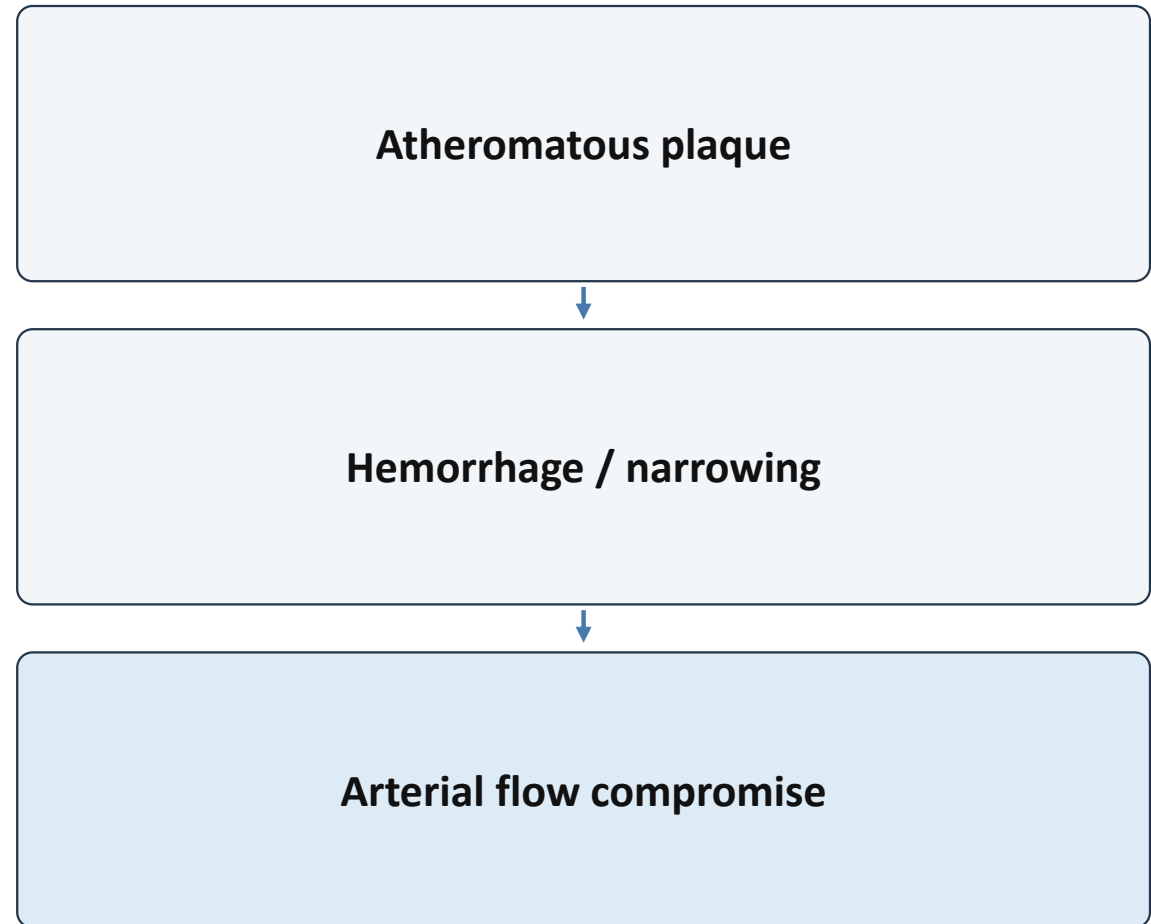
Less-common cause: Vasospasm

- Local vascular spasm can markedly reduce arterial inflow.
- Severe or prolonged reduction can contribute to ischemic necrosis.
- This mechanism differs from an obstructing thrombus or embolus.



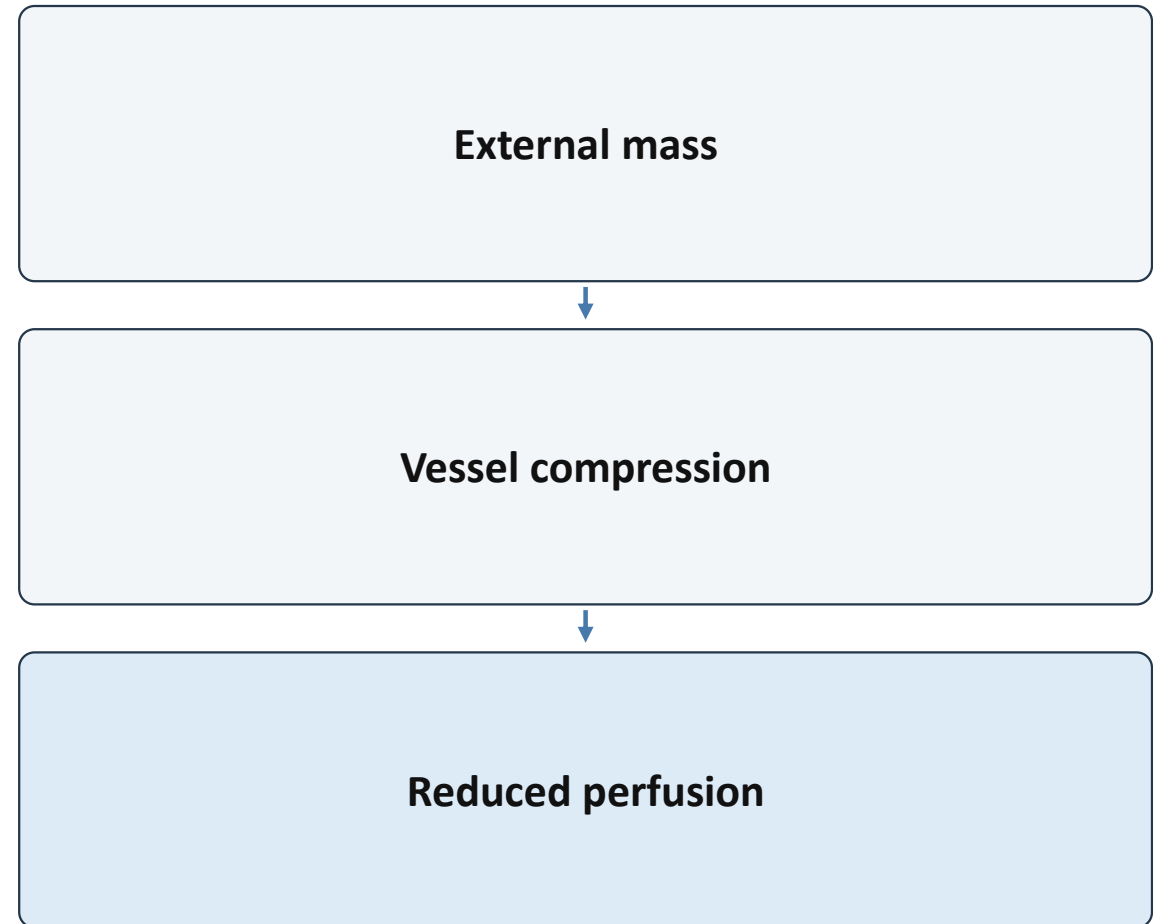
Hemorrhage into an atheromatous plaque

- Bleeding within a plaque may abruptly worsen arterial narrowing.
- The resulting reduction in blood flow can produce an infarct.
- The occlusion mechanism is local plaque change rather than a traveling embolus.



Extrinsic vascular compression

- A mass such as a tumor may compress a supplying vessel.
- Loss of perfusion arises from pressure exerted outside the lumen.
- Both intraluminal and extraluminal causes must be considered.



Vascular torsion

- Torsion twists vascular channels and impairs tissue perfusion.
- Examples include testicular torsion and bowel volvulus.
- Venous drainage may be particularly vulnerable in twisted pedicles.

Torsion / volvulus



Vascular compromise



Ischemic tissue injury

Traumatic vascular disruption

- Traumatic rupture may abolish blood flow through an arterial segment.
- Loss of downstream supply can lead to tissue ischemia.
- Here tissue damage is caused by vascular disruption, not necessarily thrombosis.

Vessel injury



Perfusion failure



Possible infarction

Edema and compartment pressure

- Increased local tissue pressure can compromise vessel perfusion.
- Anterior compartment syndrome of the leg as an example.
- The surrounding tissue compartment becomes the obstructive factor.

Swelling / pressure



Vascular compromise



Ischemic necrosis risk

Entrapment in a hernia sac

- Entrapped bowel may suffer vascular compromise.
- Both inflow and outflow can be affected in a constricted segment.
- The pathologic endpoint can be bowel infarction.

Entrapment



Vascular obstruction



Bowel ischemic necrosis

Venous thrombosis: Congestion is common

- Venous thrombi more often produce congestion rather than infarction.
- Alternative venous channels may open rapidly and decompress the tissue.
- Improved drainage can restore effective arterial inflow.

Venous blockage



Alternative drainage



Preserved tissue viability

The single-efferent-vein exception

- Organs with a single dominant venous exit have fewer outflow alternatives.
- The testis and ovary are examples identified.
- Venous blockage here is more likely to progress from congestion to infarction.

Limited outflow routes



Severe congestion



Hemorrhagic infarction

Compare arterial and venous pathways

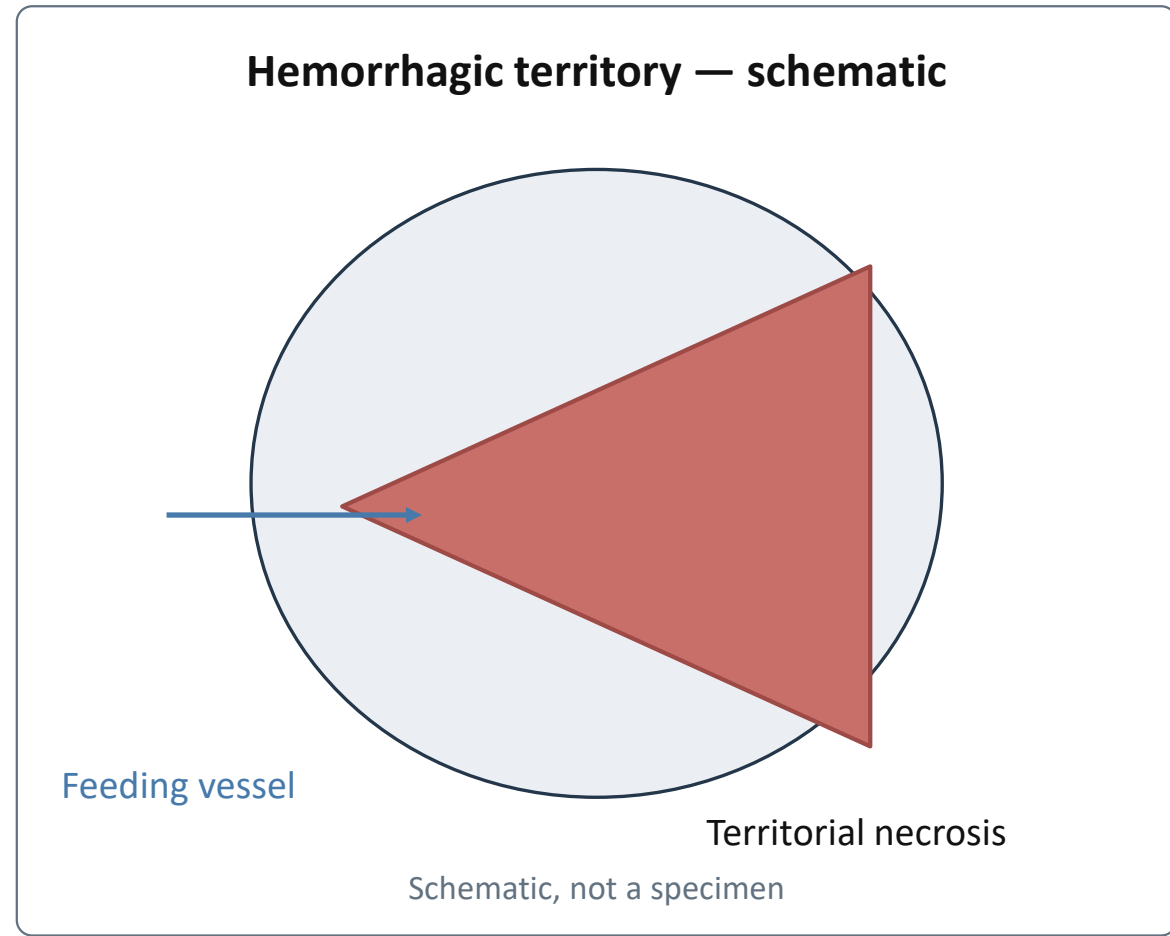
Arterial lesion	Venous lesion
Common infarct mechanism	Often congestion without infarction
May be pale in compact organs	Typically red when infarction occurs
Collateral arteries modify risk	Alternative draining veins modify risk

Two axes of infarct classification

Color axis	Infection axis
Red / hemorrhagic	Septic / infected
White / anemic	Bland / noninfected

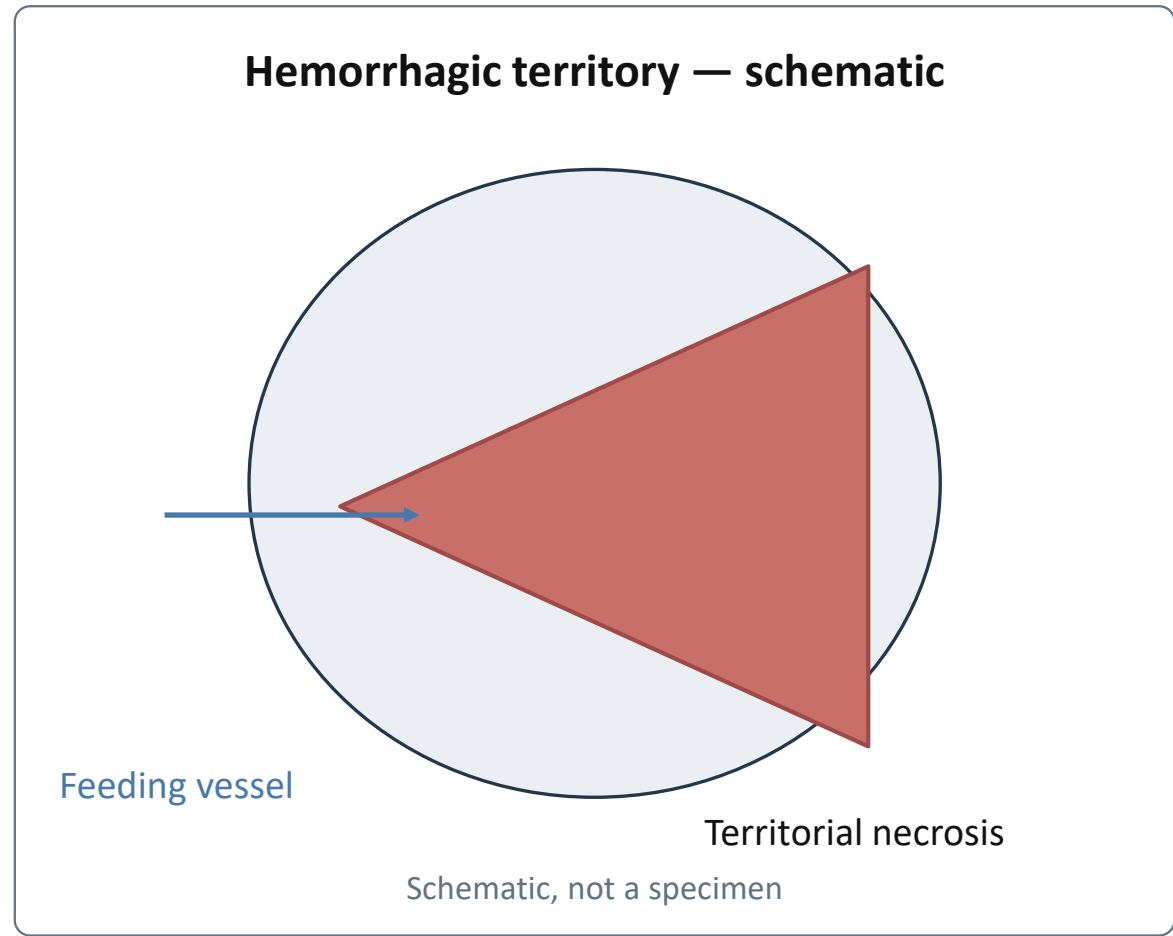
Red infarction: The central principle

- A red infarct contains extravasated blood within the necrotic territory.
- Vascular anatomy and tissue looseness determine whether blood can accumulate.
- Venous obstruction, dual supply, congestion and reperfusion are typical settings.



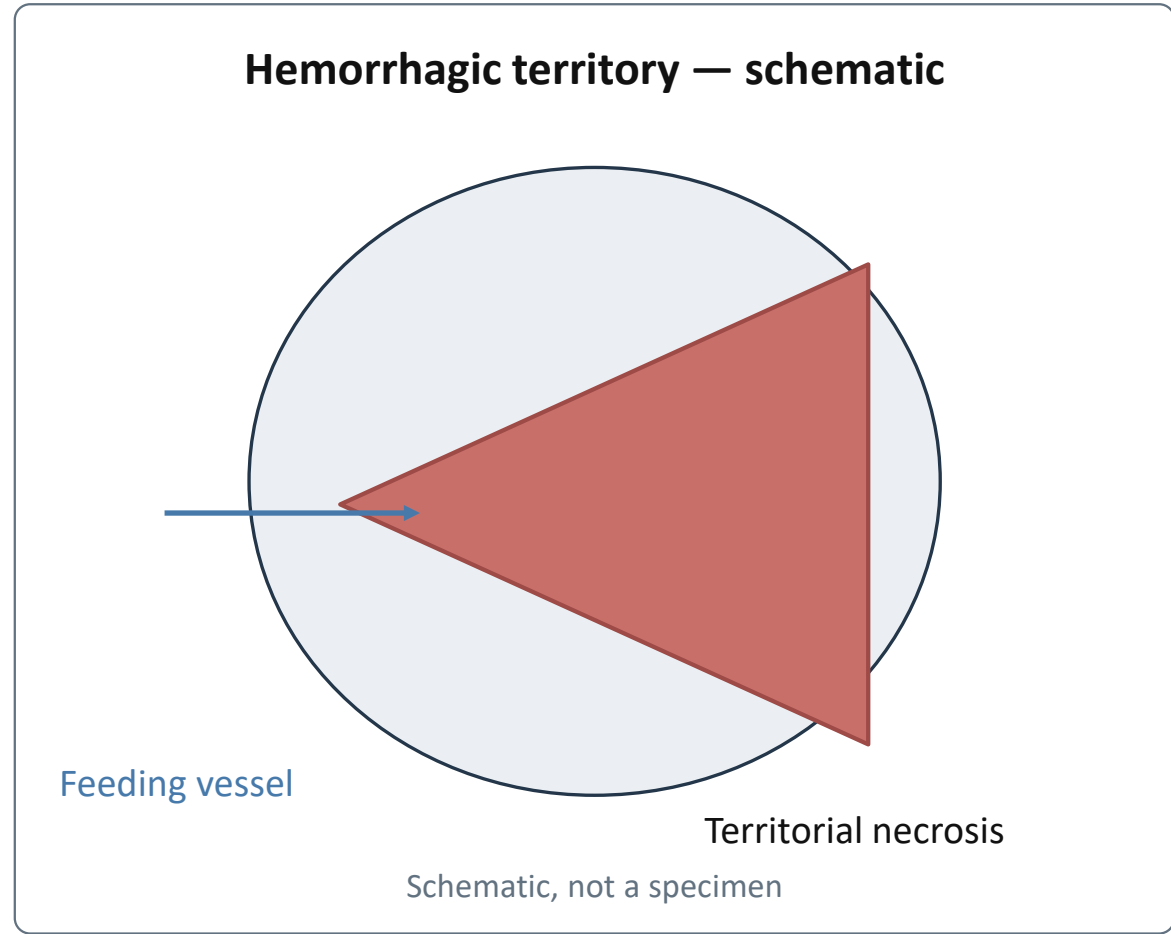
Red infarcts with venous occlusion

- Venous outflow obstruction can produce blood-filled necrotic tissue.
- Testicular torsion exemplifies a setting predisposed to hemorrhagic infarction.
- The lesion is red because blood cannot drain normally.



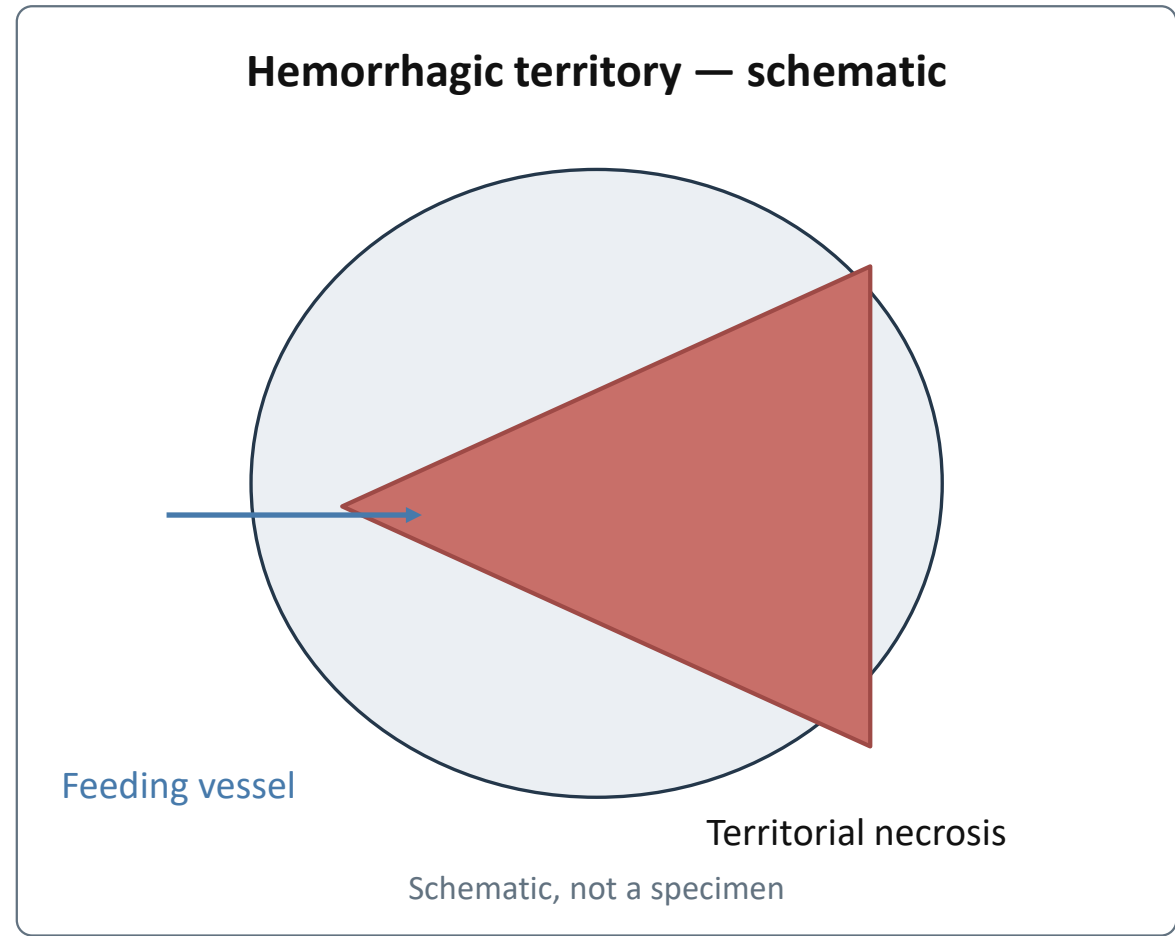
Red infarcts in loose or spongy tissue

- Lung parenchyma provides space in which blood can accumulate.
- Consequently, pulmonary infarcts are typically hemorrhagic.
- Tissue structure contributes to color, beyond the identity of the occluded vessel.



Red infarcts in dual circulation

- Lung and small intestine are examples of tissues with parallel inflow routes.
- An unobstructed supply may introduce blood into the necrotic region.
- Thus residual inflow does not necessarily prevent a hemorrhagic appearance.



Red infarcts after prior congestion

- Sluggish venous drainage can leave a tissue vascular bed congested.
- If infarction supervenes, the lesion is predisposed to hemorrhage.
- The pre-existing hemodynamic state modifies the gross appearance.

Venous congestion



Tissue ischemia



Hemorrhagic infarct

Red infarcts after reperfusion

- Flow can return to tissue already injured by arterial occlusion.
- Reperfusion may permit blood entry into a necrotic zone.
- Robbins gives angioplasty after arterial blockage as an example.

Arterial occlusion



Tissue necrosis



Reflow → hemorrhagic infarct

Five conditions favoring red infarcts

- Venous occlusion or preceding congestion.
- Loose pulmonary-type architecture or dual vascular supply.
- Reperfusion of a previously necrotic vascular territory.

Venous / congested



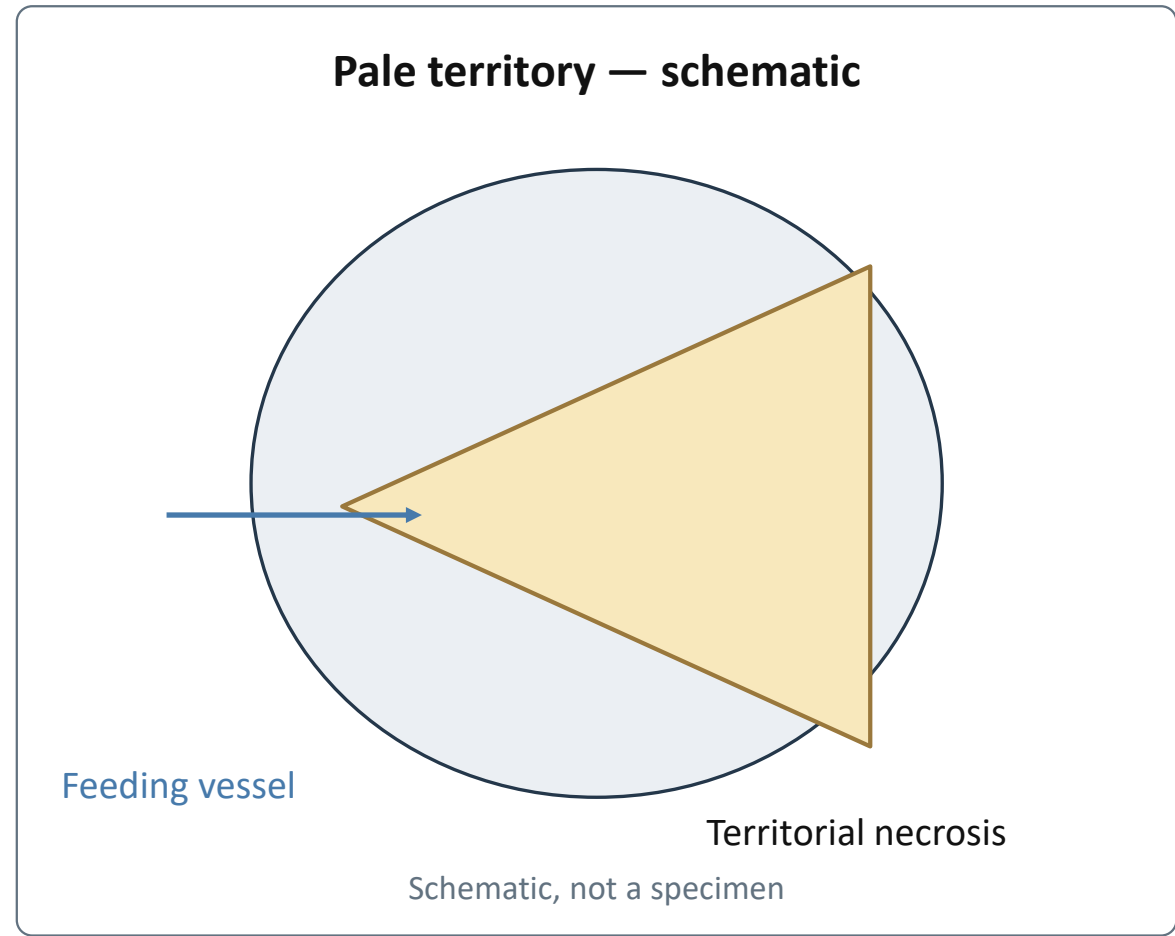
Spongy / dual supply



Reperfused

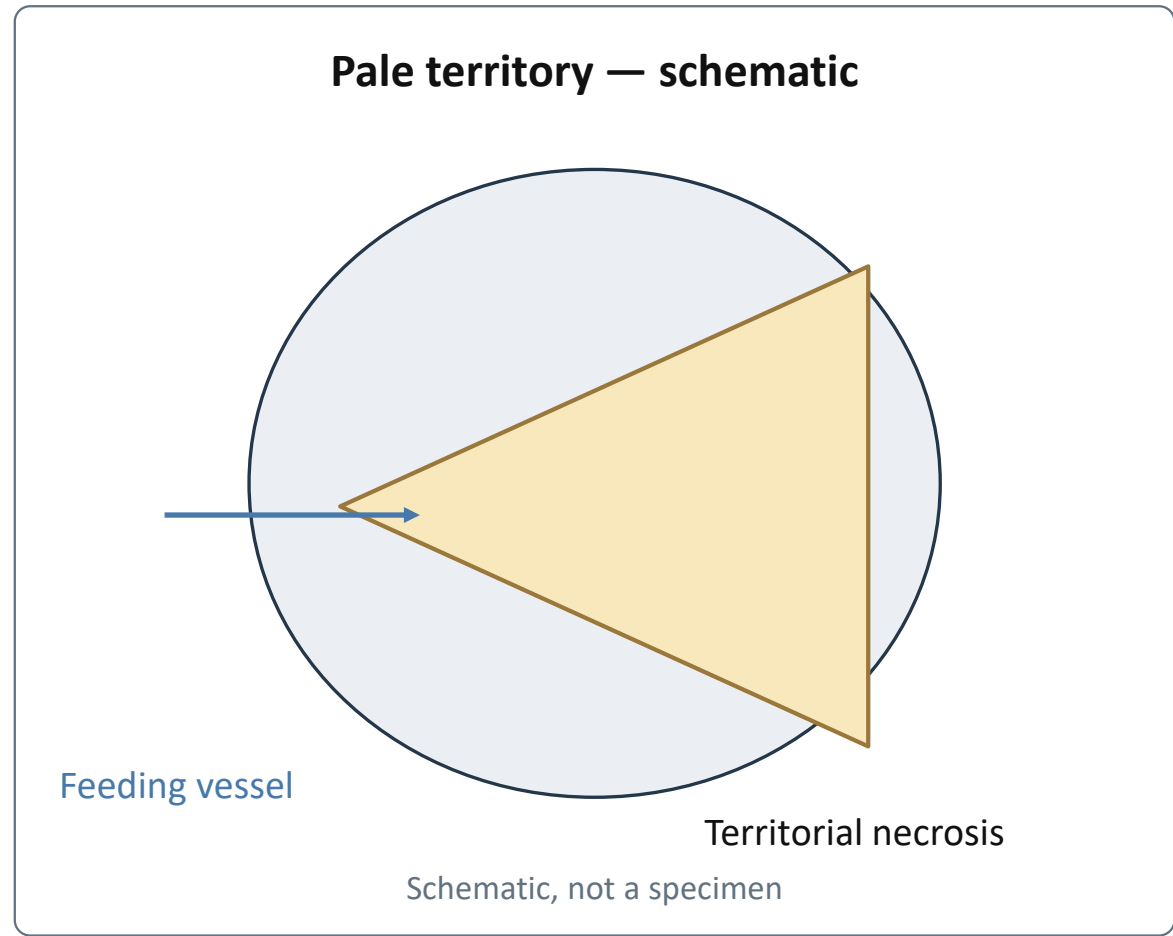
White infarction: The central principle

- White infarcts occur after arterial occlusion in compact solid organs.
- End-arterial supply limits alternative perfusion.
- Dense parenchyma restricts seepage of blood from adjacent capillaries.



End-arterial circulation

- Kidney and spleen are examples with limited collateral alternatives.
- Blocking the relevant artery typically kills its downstream territory.
- The infarct is commonly pale because there is little hemorrhagic re-entry.



Tissue density limits hemorrhage

Loose lung parenchyma	Compact renal or splenic parenchyma
Blood can accumulate in necrotic spaces	Blood seepage is constrained
Often hemorrhagic infarct	Often pale infarct

White infarction: Common sites

- Heart, spleen and kidney as common solid-organ settings.
- Arterial occlusion and limited collateral supply explain the tissue loss.
- Color and shape are consequences of vascular arrangement and tissue density.

Myocardium



Spleen



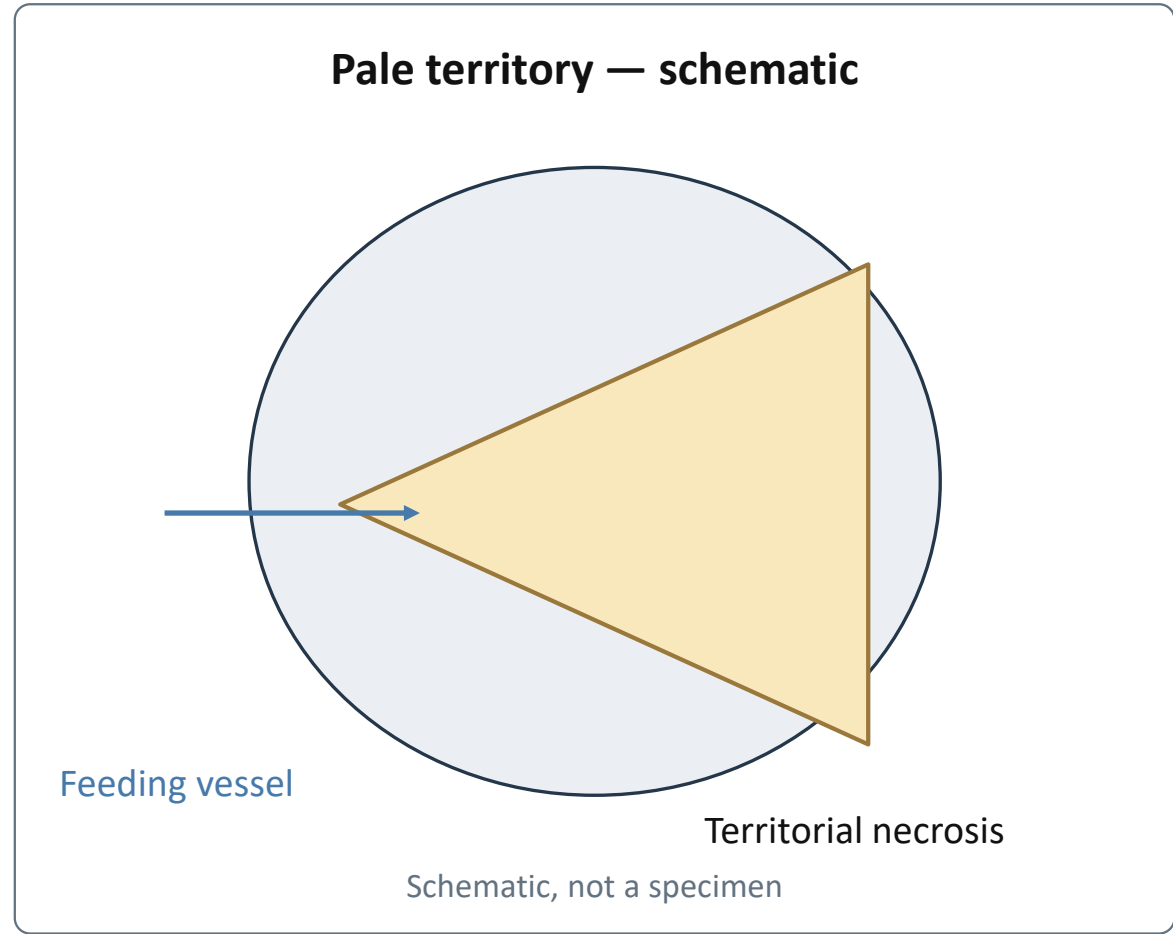
Kidney

Red versus white: Diagnostic comparison

Red / hemorrhagic	White / anemic
Often venous obstruction	Often arterial obstruction
Spongy or dual-supply tissue	Compact end-arterial tissue
Lung and intestine examples	Heart, kidney, spleen examples

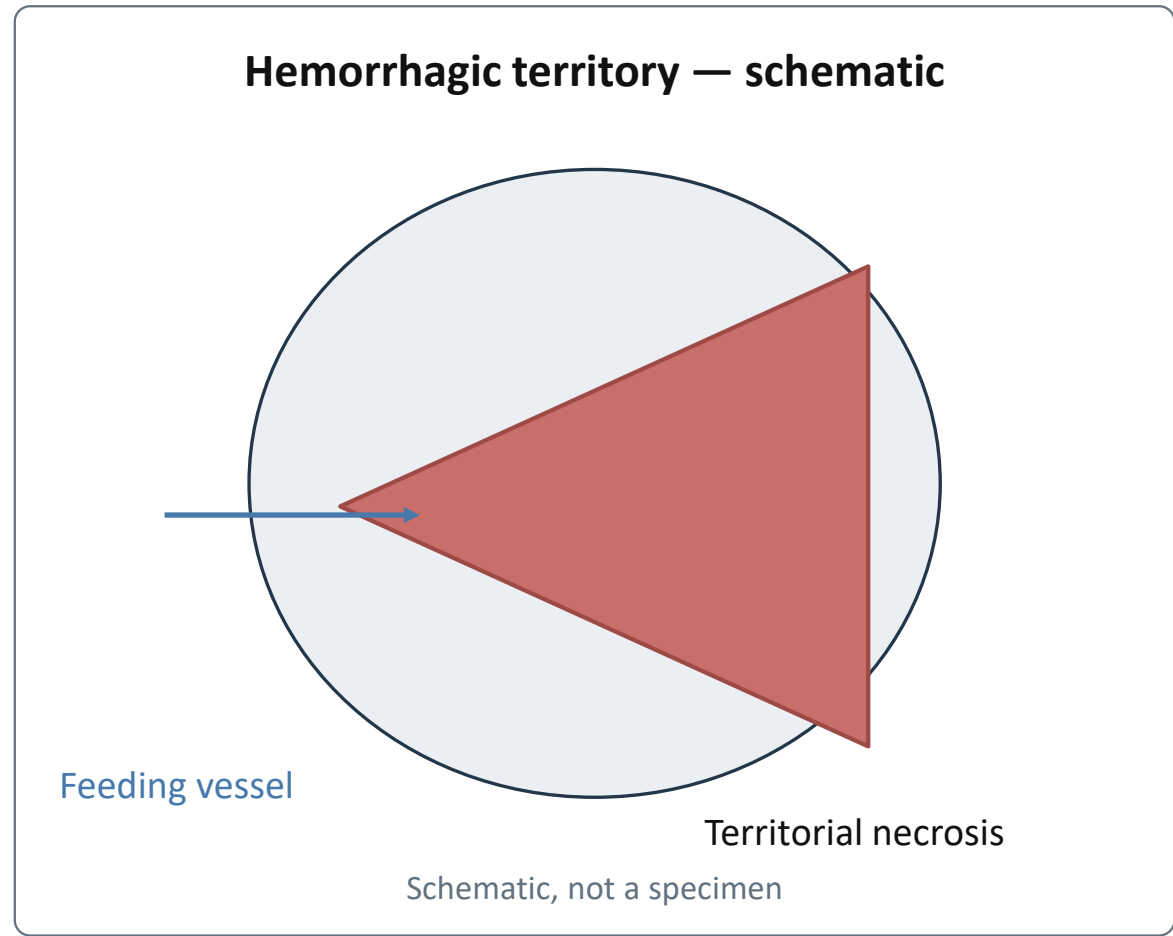
Wedge shape reflects vascular geography

- Many infarcts are roughly wedge-shaped.
- The occluded vessel corresponds to the narrow apex.
- The organ periphery often forms the broad base.



Apex points toward the blocked vessel

- A branching vascular territory expands from a proximal feeding vessel.
- When that vessel is occluded, downstream necrosis follows its distribution.
- The apex-to-base pattern therefore records the anatomy of blood supply.



Serosal surface may develop fibrin

- If an infarct reaches a serosal surface, fibrinous exudate can overlie it.
- Necrotic cells generate mediators that recruit acute inflammation.
- Surface fibrin is a local reaction to the underlying tissue injury.

Necrosis near serosa



Inflammatory response



Fibrinous surface exudate

Fresh infarcts are poorly demarcated

- Early infarcts may be subtle and ill-defined grossly.
- Slight hemorrhage may be present even in lesions that ultimately become pale.
- Appearance evolves with necrosis and inflammation.

Early: Indistinct



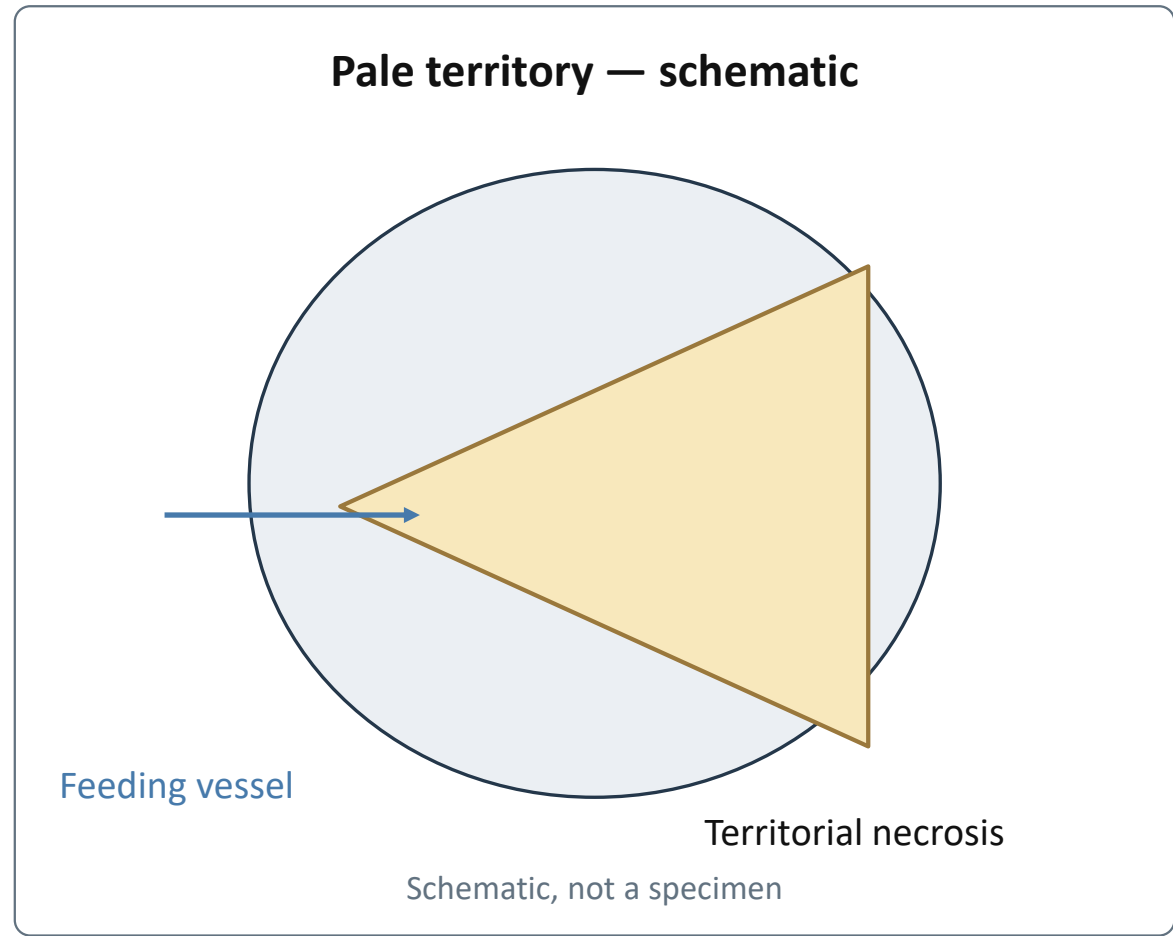
Days: Clearer margins



Later: Repair / scar

Inflammatory congestion defines the edge

- Over several days the infarct boundary becomes more visible.
- A narrow hyperemic rim corresponds to the inflammatory reaction.
- The edge demarcates necrotic tissue from preserved parenchyma.



White infarcts become paler with time

- In organs without dual supply, arterial infarcts tend to become progressively pale.
- Their borders become more sharply demarcated.
- The gross appearance therefore depends partly on lesion age.

Fresh lesion



Paler and distinct



Scar formation

Red cell removal in hemorrhagic infarcts

- Extravasated erythrocytes are removed by macrophages.
- Heme iron is converted to hemosiderin.
- Extensive old hemorrhage can leave a firm brown residuum.

Extravasated RBCs



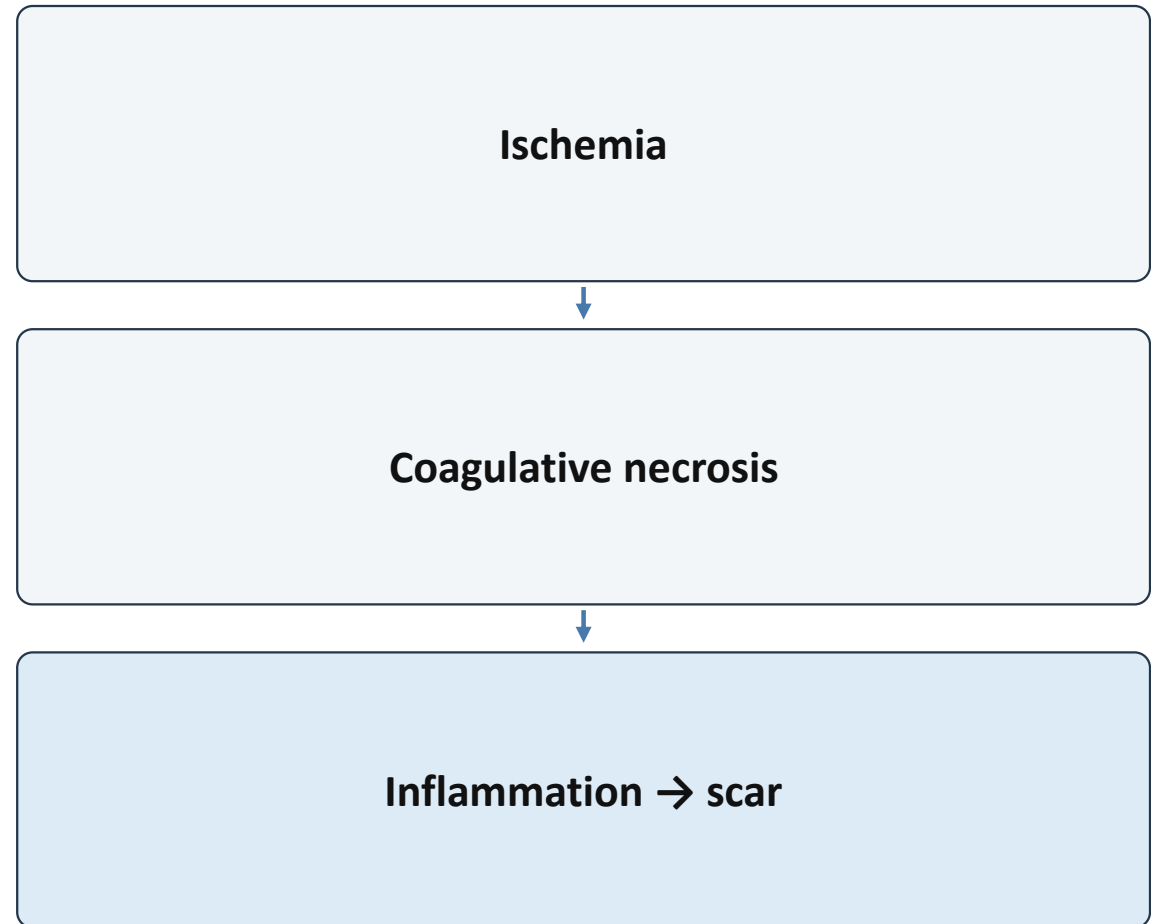
Macrophage phagocytosis



Hemosiderin deposition

Coagulative necrosis is the usual pattern

- Most non-CNS infarcts show coagulative necrosis.
- The dominant morphologic finding is ischemic death of cells.
- The tissue reaction progresses through inflammation toward fibrosis.



Microscopic injury can lag occlusion

- Immediately after a lethal occlusion, histologic necrosis may not yet be visible.
- Characteristic microscopic changes require about 4–12 hours.
- An apparently bland section cannot exclude very recent ischemic injury.

Minutes–hours: Injury



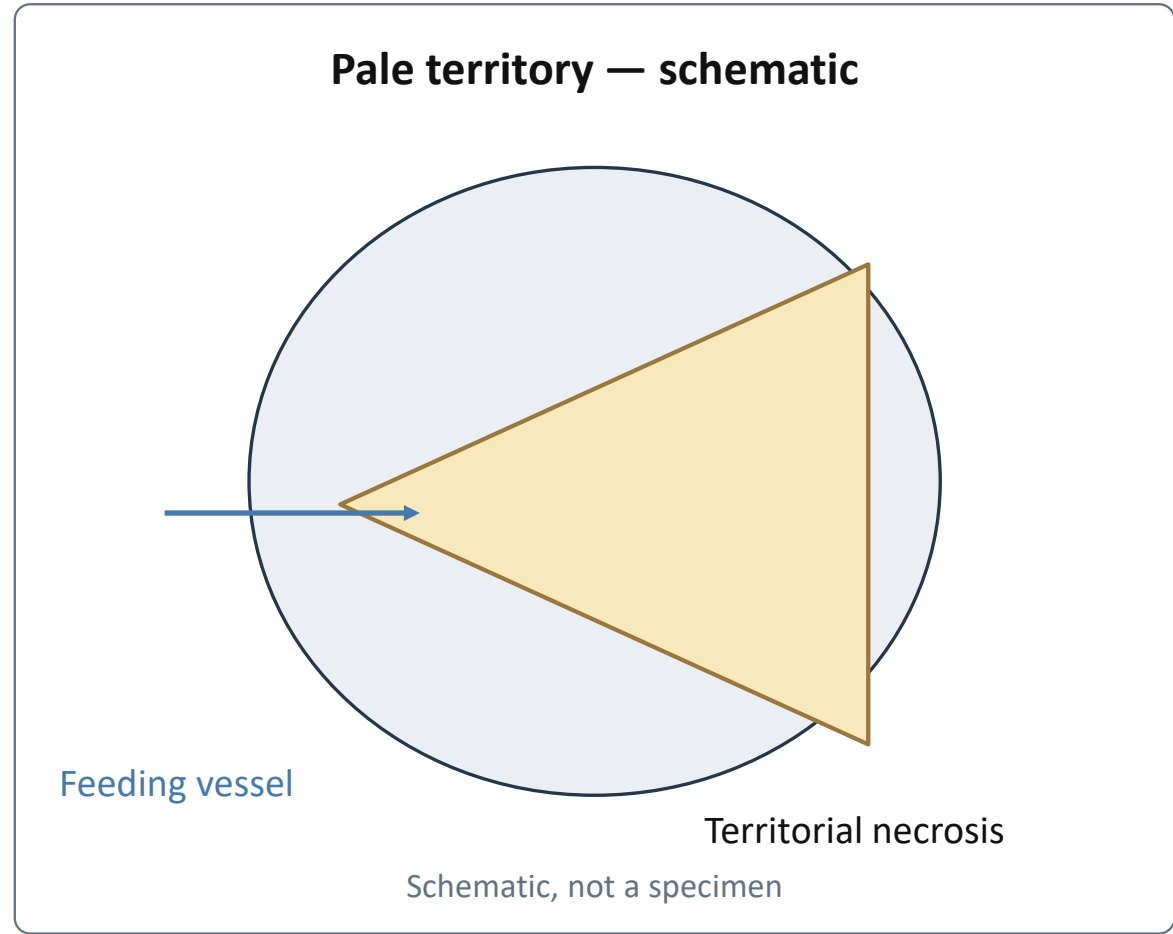
4–12 h: Visible necrosis



Later: Inflammation / repair

Early inflammation at the infarct edge

- Inflammatory cells begin accumulating near the margins within hours.
- The preserved margins supply the substrate for a reparative response.
- Spatial separation of dead center and inflamed border is important morphologically.



The 1–2-day inflammatory stage

- The inflammatory margin as well defined within 1–2 days.
- Inflammation helps demarcate the zone of necrosis.
- This timing must be interpreted in relation to tissue and clinical context.

Occlusion



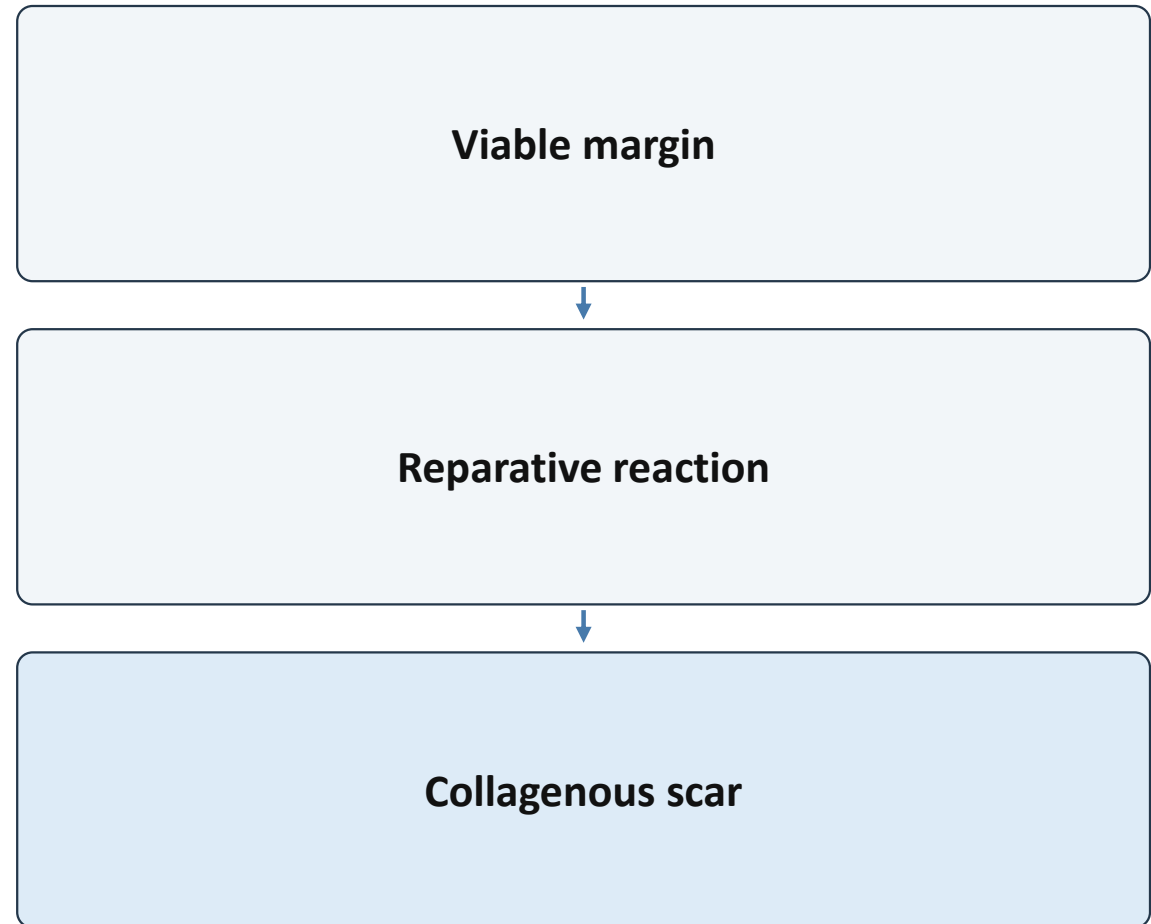
Acute inflammatory margin



Organization / repair

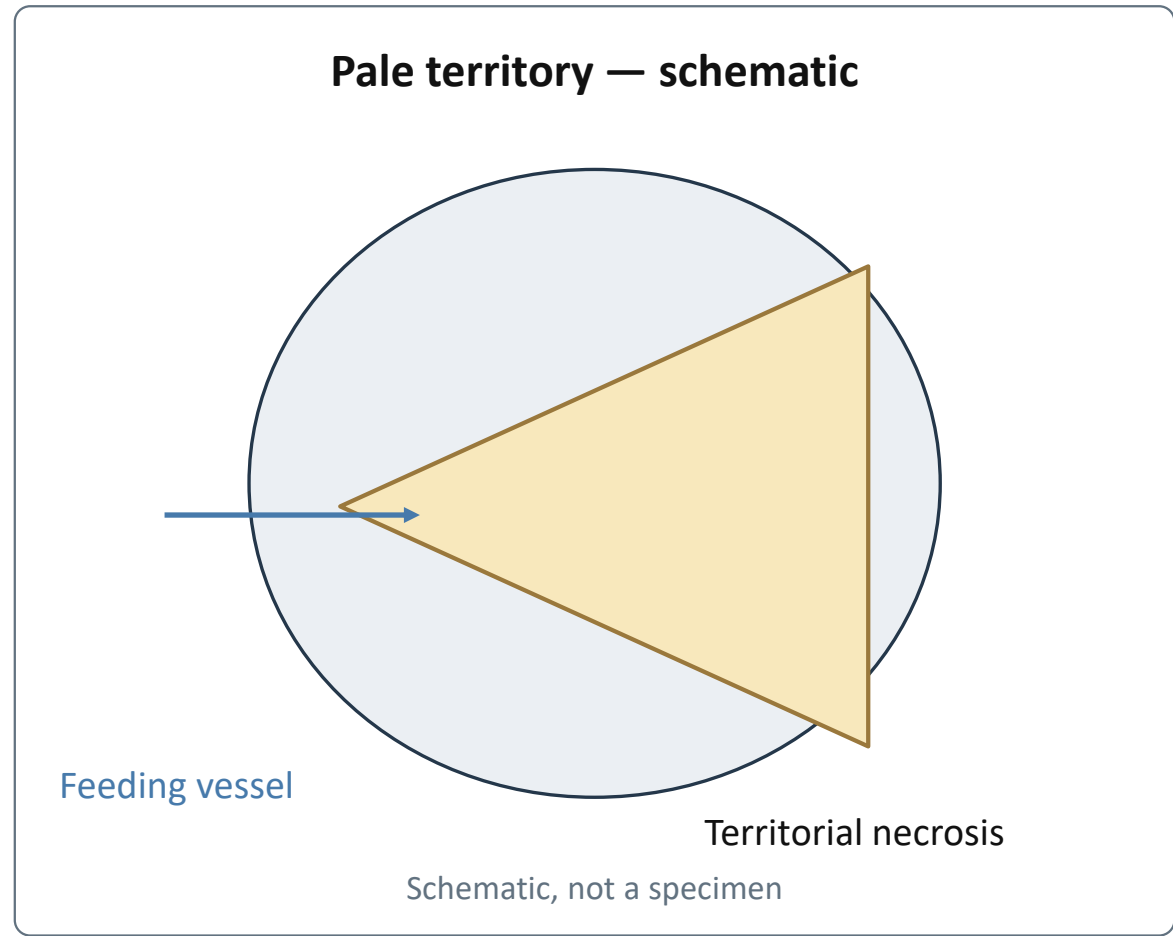
Reparative response begins at viable margins

- Repair develops from tissue surviving at the edge of an infarct.
- The central necrotic zone cannot itself regenerate simply by restored blood flow.
- Organization proceeds toward replacement by fibrous tissue.



Most infarcts heal by fibrosis

- Scar replacement is the usual end stage for many infarcts.
- A remote renal infarct replaced by fibrotic scar.
- The healed shape may retain evidence of the original vascular territory.



Brain infarction is the major exception

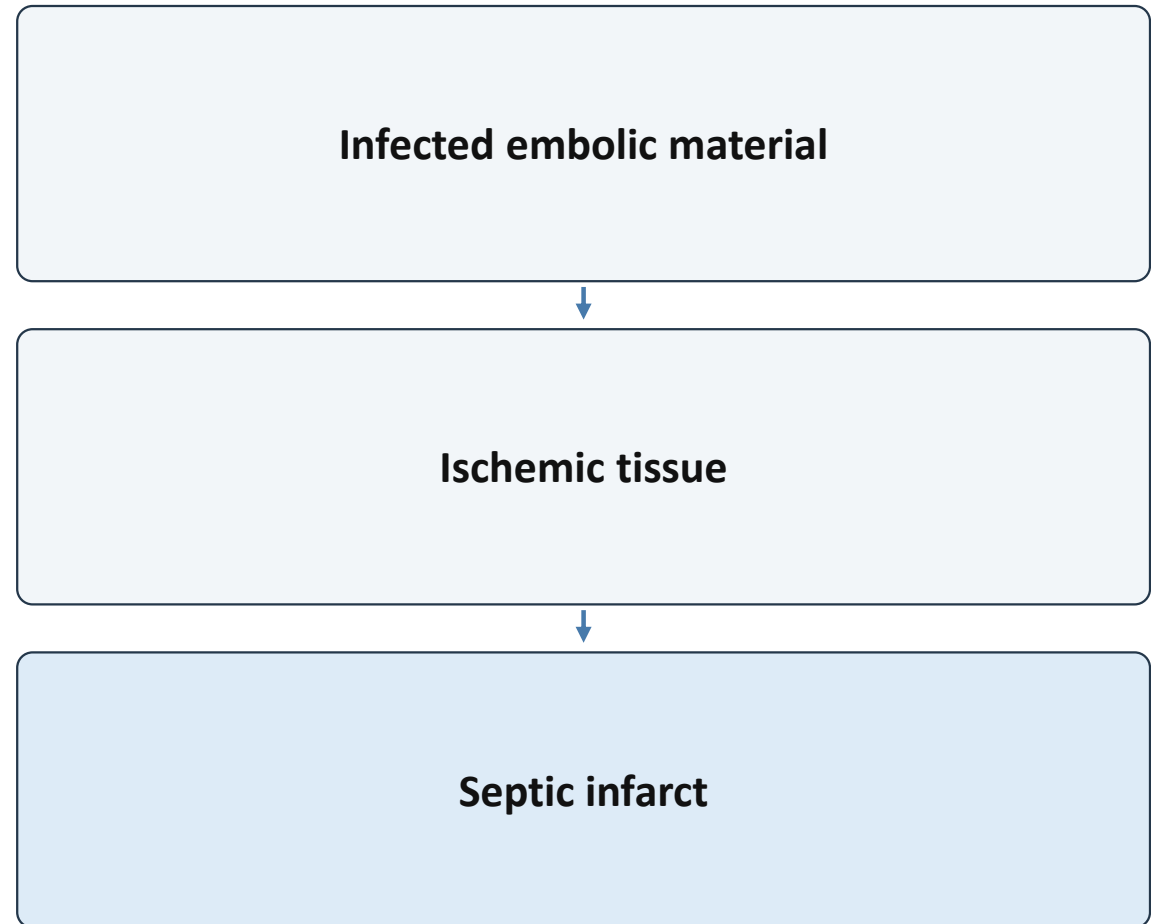
Most solid organs	Central nervous system
Coagulative necrosis	Liquefactive necrosis
Fibrous scar often remains	Different tissue removal / repair pattern

Bland versus septic infarction

Bland infarct	Septic infarct
Ischemic necrosis without infection	Necrosis involving microbes
Sterile inflammatory repair	Often greater inflammatory response
Progression to scar	May progress through abscess formation

Septic emboli may seed infarcts

- Infected cardiac valve vegetations can embolize into arteries.
- Microbes carried with embolic material can infect the ischemic territory.
- Necrotic tissue can also become secondarily seeded by microorganisms.



Septic infarction may become an abscess

- Microbial infection changes the evolution of the necrotic focus.
- Conversion of a septic infarct into an abscess.
- The inflammatory response is greater than in a bland lesion.

Ischemic necrosis + microbes



Abscess formation



Organization / repair

Four determinants of infarction risk

- Collateral vascular anatomy is the most important determinant.
- Rate of occlusion affects the opportunity for bypass perfusion.
- Tissue sensitivity to hypoxia and the oxygen content of blood also matter.

Vascular anatomy

Rate of occlusion

Tissue vulnerability

Blood oxygenation

Anatomy: Alternative perfusion protects

Protected by alternative flow	More end-arterial
Lung: pulmonary + bronchial arteries	Kidney: regional arterial supply
Liver: hepatic artery + portal vein	Spleen: limited collateral flow
Hand/forearm: radial + ulnar	Greater risk with occlusion

Pulmonary dual supply

- Pulmonary arterial flow and bronchial arterial flow provide alternative sources.
- This helps explain why not every pulmonary embolus causes infarction.
- If compensatory perfusion is inadequate, pulmonary infarction can occur.

Pulmonary artery blocked



Bronchial flow persists



Potentially spared parenchyma

Liver and limb arterial alternatives

- The liver receives hepatic arterial and portal venous inflow.
- The hand and forearm receive blood via radial and ulnar arteries.
- These parallel circulations provide resilience against isolated obstruction.

Hepatic artery



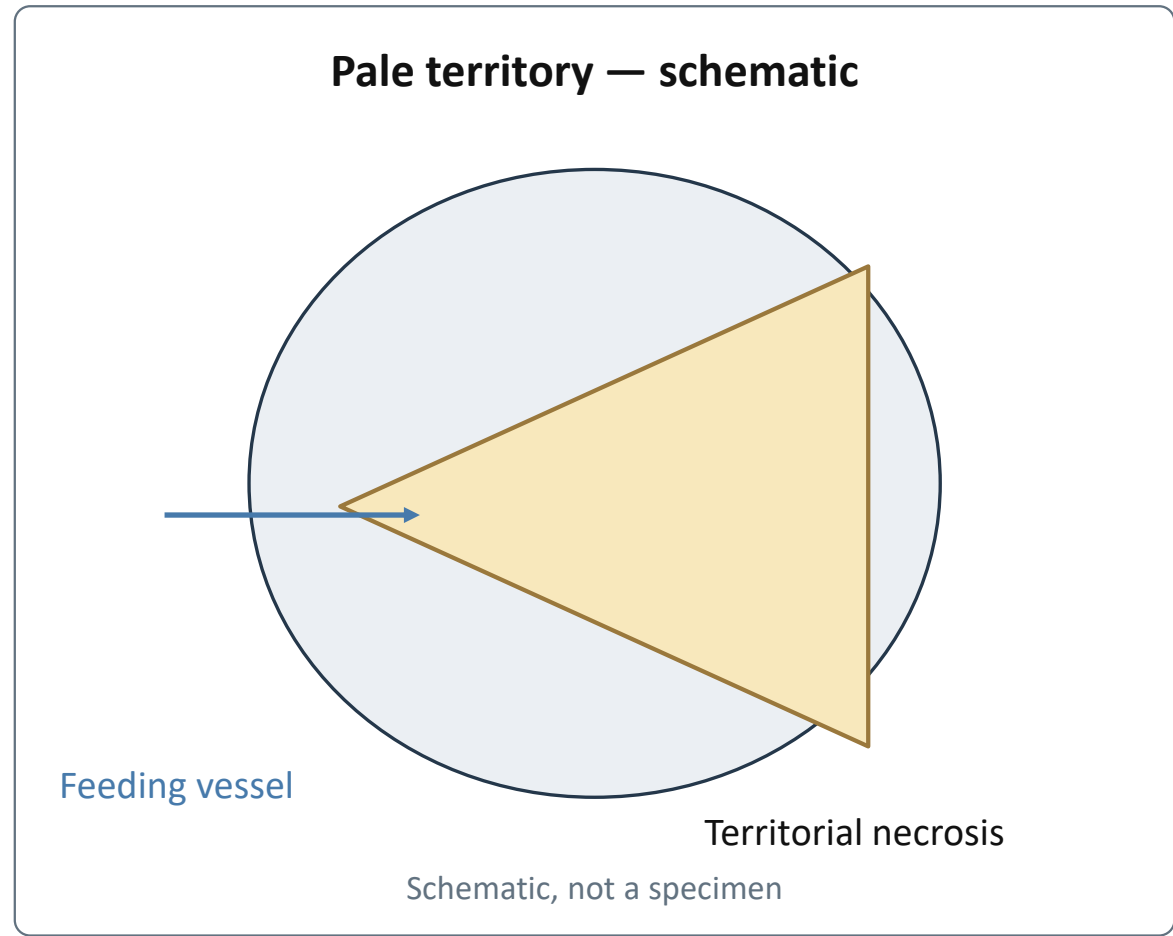
Portal vein



Radial / ulnar arteries

End-arterial vulnerability of kidney and spleen

- Regional occlusion in kidney or spleen usually leaves little functional bypass.
- These organs are prone to sharply demarcated pale infarction.
- The morphology can therefore reveal the vascular architecture.



Speed of occlusion modifies tissue outcome

Slow occlusion	Abrupt occlusion
Collateral adaptation possible	Limited adaptation time
Infarction may be avoided	Higher ischemic risk
Example: gradual coronary plaque growth	Example: abrupt arterial embolism

Coronary collateral example

- Small anastomoses connect major coronary arterial distributions.
- If obstruction progresses slowly, collateral flow can increase.
- This can prevent infarction even when a major artery eventually closes.

Gradual coronary stenosis



Collateral recruitment



Myocardial perfusion maintained

Neurons: Extremely ischemia-sensitive

- Neurons may be irreversibly damaged after 3–4 minutes without blood supply.
- This emphasizes the clinical impact of rapid loss of cerebral perfusion.
- The number describes cellular vulnerability, not the visibility of later histology.

Flow stops



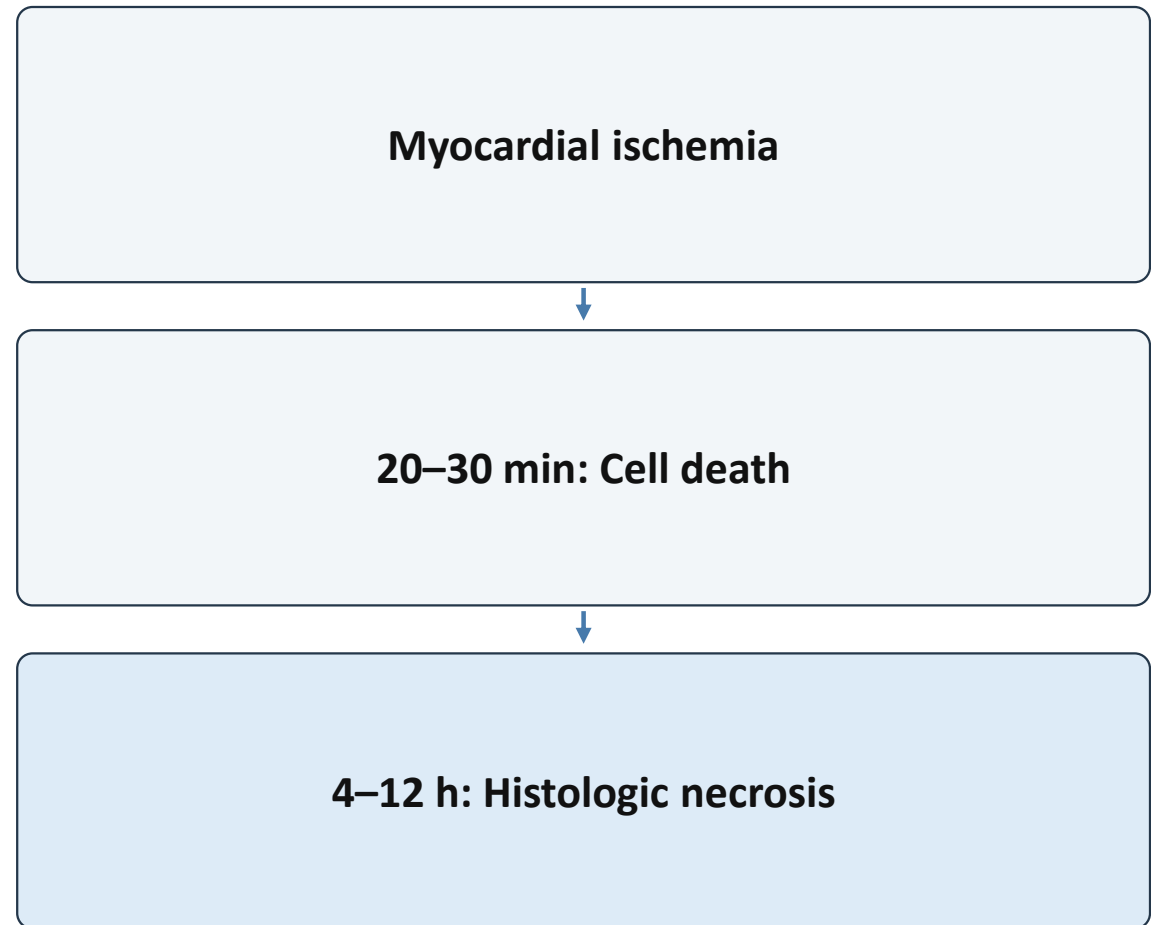
3–4 min: Neuronal injury



Later: Morphologic change

Cardiomyocytes: Vulnerable but less rapid

- Myocardial cells may die after 20–30 minutes of ischemia.
- The cells are more resilient than neurons but still highly oxygen-dependent.
- Microscopic visibility of necrosis still lags for approximately 4–12 hours.



Fibroblasts: Relatively ischemia-tolerant

- Myocardial fibroblasts remain viable for many hours of ischemia.
- Different cell populations in the same organ can therefore survive differently.
- This helps explain how repair may occur at margins of injured tissue.

Neurons: Minutes



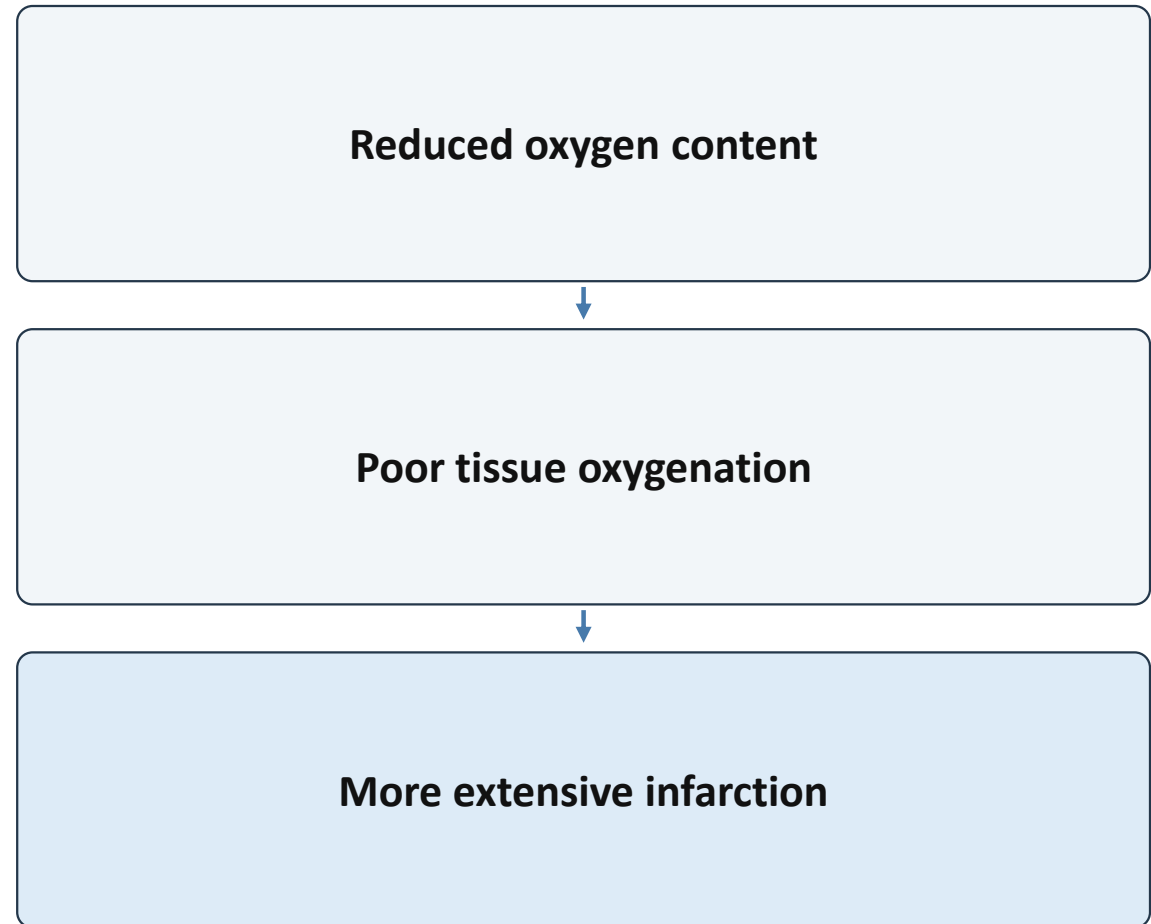
Myocytes: Tens of minutes



Fibroblasts: Many hours

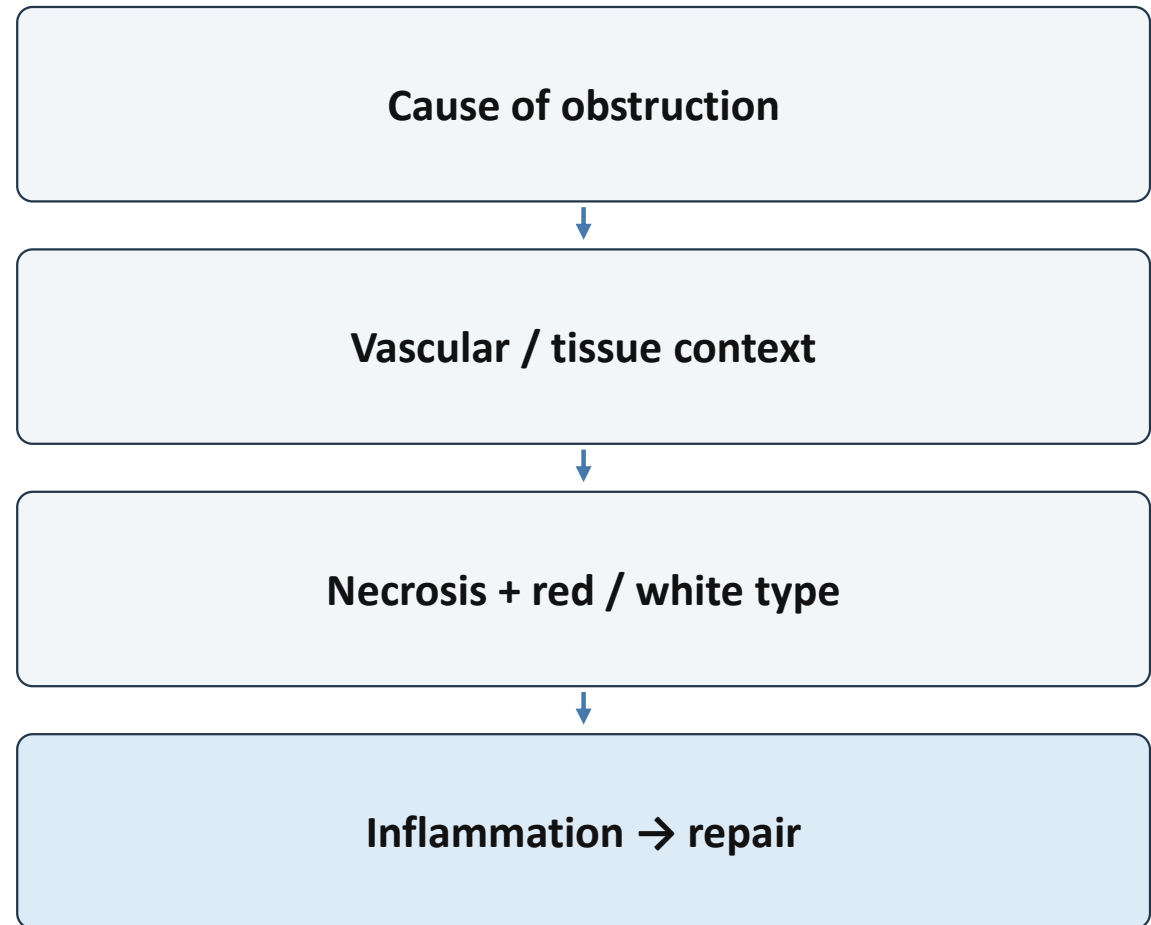
Hypoxemia lowers the ischemic threshold

- Low blood oxygen content worsens oxygen delivery through remaining vessels.
- It increases both the likelihood and the extent of infarction.
- Vascular patency and oxygen content must be considered together.



Integrate cause, morphology and susceptibility

- Identify the blocked inflow or outflow route.
- Predict hemorrhagic versus pale morphology using tissue and collateral anatomy.
- Estimate injury likelihood from rate of occlusion, tissue type and oxygenation.



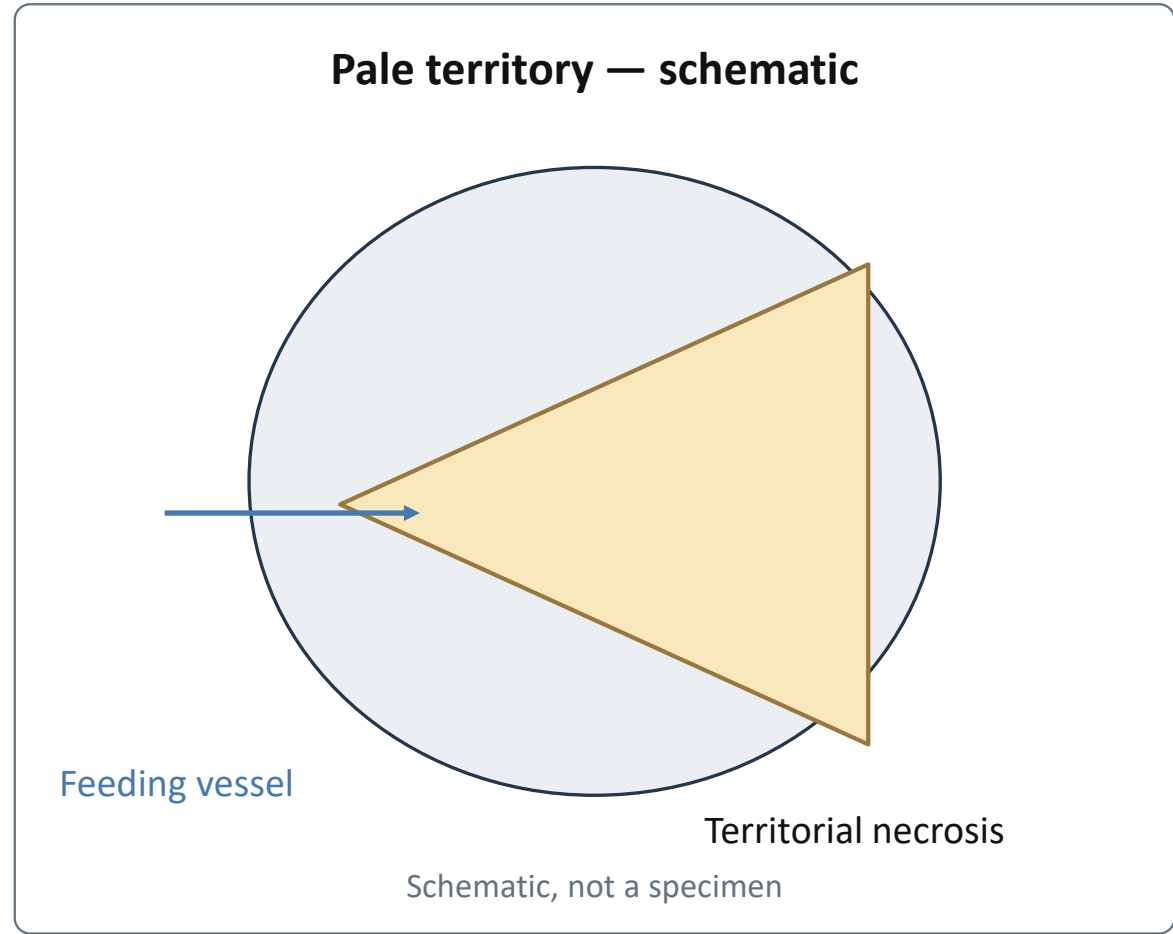
Case: A wedge-shaped pale splenic lesion

Clinical reasoning

- A splenic arterial branch is abruptly obstructed.
- A peripheral wedge is pale and relatively sharply demarcated.
- Which anatomic and histologic principles predict this appearance?

Case reasoning: The splenic infarct

- The spleen has a largely end-arterial circulation.
- Dense splenic parenchyma limits blood seepage into the dead area.
- The lesion is a white infarct whose apex points toward the obstructed vessel.



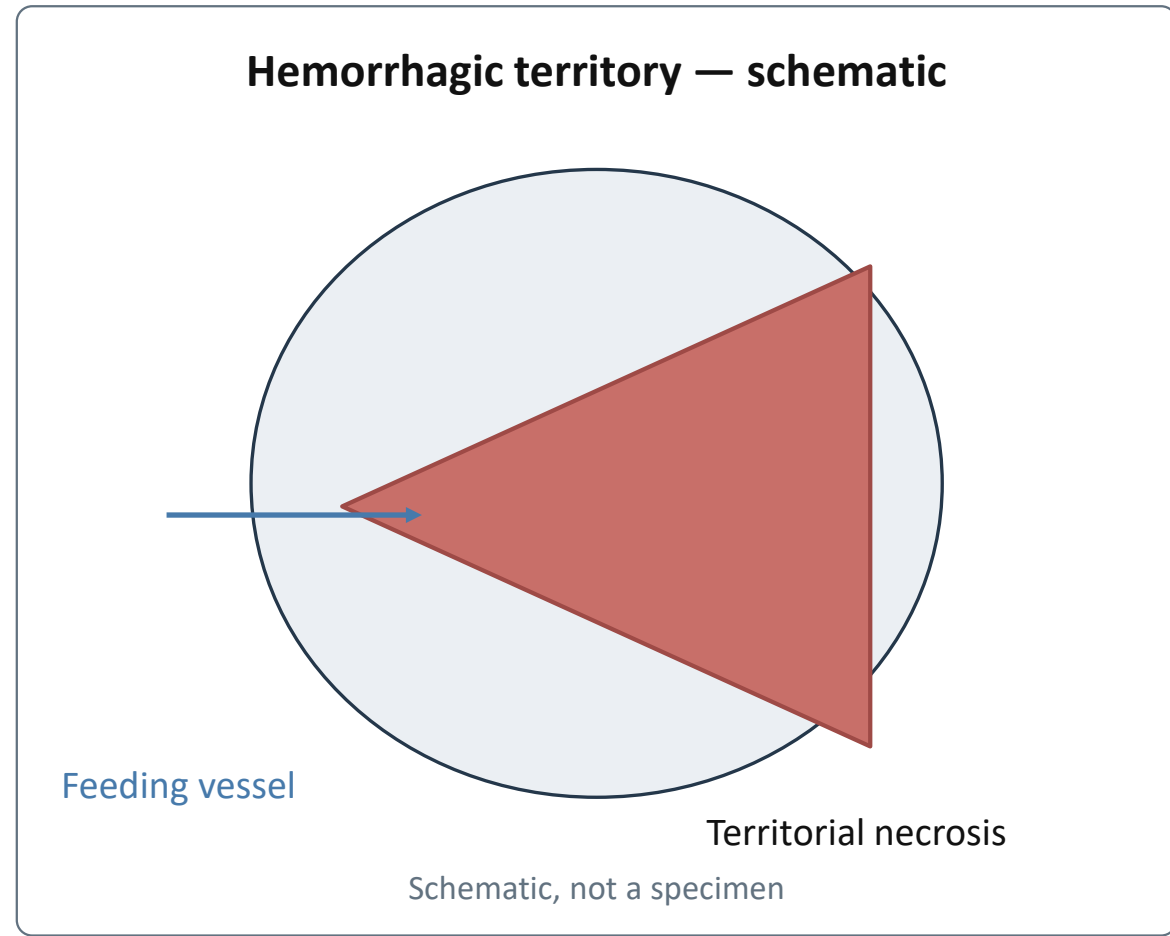
Case: Torsion with a hemorrhagic lesion

Clinical reasoning

- An organ with limited venous outflow develops sudden torsion.
- Marked congestion is followed by tissue necrosis with extensive hemorrhage.
- Which hemodynamic mechanism produces a red infarct?

Case reasoning: Venous infarction

- Torsion blocks venous drainage and can compromise further inflow.
- Congested vessels allow blood to accumulate in necrotic tissue.
- The result is a hemorrhagic infarct, as classically occurs in testicular torsion.



Synthesis: Four essential questions

- Did arterial inflow or venous outflow fail?
- Is the tissue solid/end-arterial or loose/dual-supply?
- How long and how rapidly was perfusion impaired?
- What is the tissue's intrinsic susceptibility and oxygen delivery?

Cause

Vascular anatomy

Time course

Oxygen needs

Source and interpretation

- Primary teaching source: Robbins, Cotran & Kumar, Pathologic Basis of Disease, 11th ed., chapter on hemodynamic disorders.
- The infarction topic extract had a misplaced thrombosis passage; this deck follows the verified full-text infarction section.
- Diagrams are editable schematic representations, not photographs of clinical specimens.