
Atheroembolic Renal Disease

Cholesterol-crystal embolization of intrarenal arteries

Renal vascular pathology • mechanism, morphology and clinical interpretation

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Learning objectives

- Define the embolic lesion and identify its arterial source.
- Trace atheromatous debris from the aorta or renal artery into the kidney.
- Recognize cholesterol clefts and the relevant vascular compartments.
- Relate embolic burden and baseline renal function to clinical injury.
- Separate atheroembolism from stenosis, infarction and microangiopathy.

The defining lesion is intravascular

Atheromatous debris embolizes into intrarenal arteries.

- The source is an atherosclerotic plaque, not a primary glomerular process.
- An arterial lumen contains cholesterol-crystal-rich embolic material.
- Clinical significance depends on the embolic burden and renal reserve.

Embolization is not the same as stenosis

Atheroembolic disease	Atherosclerotic renal artery stenosis
Detached plaque fragments travel downstream	Plaque narrows a major renal artery in situ
Arcuate/interlobular arterial lumens may contain crystals	Persistent renal hypoperfusion is the main consequence
Acute injury possible after an arterial procedure	Chronic ischemia / renovascular hypertension can result

Place in renal vascular pathology

Renal vascular diseases



Large-artery narrowing / ischemia



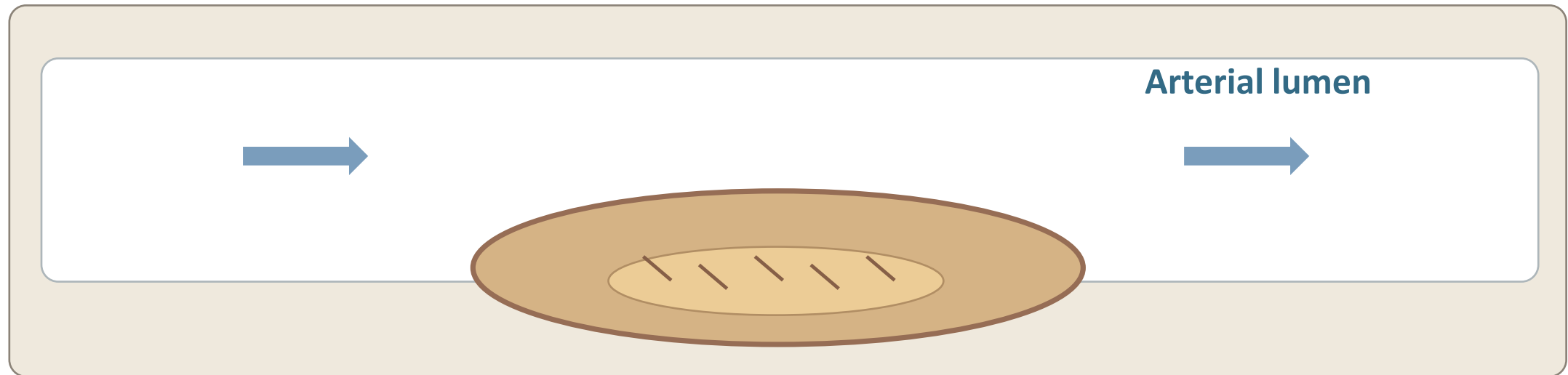
Cholesterol plaque-fragment embolization



Small-vessel thrombotic microangiopathy

The lesion begins in an atheroma

Original schematic: Longitudinal arterial section



Plaque core

Fibrous cap (lumen-facing surface)

Schematic, not a gross or microscopic specimen; Wall proportions are illustrative.

What is inside the plaque core?

- Extracellular cholesterol and cholesterol esters are major lipid constituents.
- Foam-cell debris, necrotic material and variable thrombotic material coexist.
- Crystalline cholesterol can be released if plaque integrity is disrupted.
- In routine sections, cholesterol crystals often leave empty clefts.

Atheroma develops in the arterial intima

Structural location

- An atherosclerotic plaque is an intimal lesion.
- A fibrous cap faces the arterial lumen.

Why it matters here

- Plaque disruption exposes core debris to flowing blood.
- The debris can reach renal arterial branches.

Plaque rupture releases solid debris

**Atheromatous
plaque**



**Surface
disruption**



**Debris enters
bloodstream**



**Distal
embolization**

Three major plaque complications differ

Complication	Immediate event	Renal implication
Stenosis	Progressive luminal narrowing	Reduced inflow / chronic ischemia
Thrombosis	Clot forms on disrupted plaque	Local arterial occlusion
Atheroembolism	Plaque material breaks free	Distal cholesterol-rich occlusion

Embolus material is diagnostically informative

Cholesterol-rich debris

- Derived from atheromatous plaque.
- Angular empty cholesterol clefts can remain after processing.

Thrombotic embolus

- Composed predominantly of intravascular clot.
- Do not identify atheroembolism solely from an occluded lumen.

The abdominal aorta is a major source

The aorta can shed plaque fragments into renal arterial flow.

- Severe atherosclerosis provides the source lesion.
- Proximity of the renal artery origins explains the renal pathway.
- Aortic procedures are emphasized as triggering contexts.

The renal artery itself can be the source

- Renal arterial atheroma can generate embolic plaque fragments.
- The debris moves from the source site to smaller intrarenal branches.
- Thus the source plaque and embolus can lie within one vascular territory.

Anatomic route of cholesterol emboli

Abdominal aorta



Renal artery



Intrarenal branches



Arcuate artery



Interlobular artery

Intrarenal arteries: Where to look

Renal artery

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graph TD; A[Renal artery] --> B[Segmental / interlobar branches]; B --> C[Arcuate arteries]; C --> D[Interlobular arteries];
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Segmental / interlobar branches

Arcuate arteries

Interlobular arteries

Emboli lodge distal to the parent plaque

**Source plaque is
disrupted**



**Debris follows
arterial flow**



**Intrarenal lumen
traps fragments**



**Downstream
perfusion may
suffer**

Focal lesions can be clinically silent

Atheroemboli are frequently of no significance.

- A histologic embolus does not automatically imply renal failure.
- A small embolic burden may produce little functional loss.
- Clinical interpretation must incorporate baseline renal function.

Severe atherosclerosis provides the substrate

- Atheroembolic events occur in people with substantial plaque burden.
- Atheromatous material must be available for detachment.
- Arterial manipulation may disturb plaque and promote embolization.

Older adults are the typical population

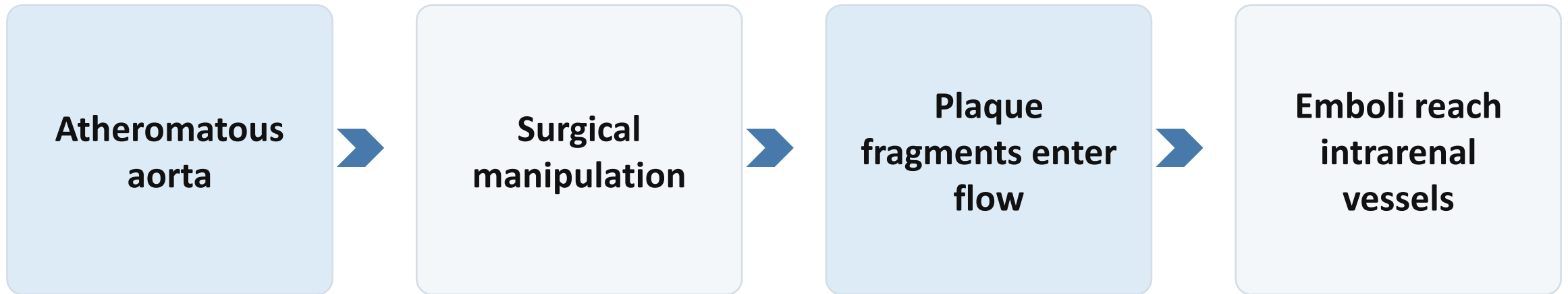
Clinical setting

- Older adults with severe atherosclerosis.
- Often vascular disease is already established.

Interpretive consequence

- Underlying renal impairment may coexist.
- A new embolic insult may unmask limited functional reserve.

Abdominal aortic surgery is a recognized trigger



Aortography is a recognized trigger

- Robbins specifically lists aortography among precipitating exposures.
- Catheter/instrument passage occurs in an atheromatous arterial environment.
- Temporal association with a procedure raises suspicion but is not proof.

Intra-aortic cannulation is a recognized trigger

- Intra-aortic cannulation is explicitly named in the Robbins description.
- Arterial instrumentation can be followed by atheromatous debris release.
- Clinical injury is not inevitable after manipulation.

Procedural exposure is a clue—not a criterion

What the exposure tells us	What it does not establish
A plausible moment of plaque disruption	That cholesterol emboli certainly formed
A mechanism connecting aorta and kidney	That renal dysfunction must occur
A reason to examine intrarenal vessels	That all postprocedural AKI is atheroembolic

The source text does not require a procedure

“Especially after” does not mean “only after.”

- Three interventional settings.
- Its definition rests on embolized plaque fragments and cholesterol crystals.
- No incidence or relative frequency of spontaneous events is provided.

From plaque disruption to renal injury

**Atheromatous
source**



**Cholesterol-rich
microemboli**



**Small intrarenal
arteries**



**Variable renal
consequence**

Embolic burden determines potential effect

Few emboli

- Limited number of affected vascular lumens.
- May have no clinically important consequence.

Numerous emboli

- More vascular territories may be compromised.
- An acute decrement in renal function becomes possible.

Baseline renal function is the second variable

Preserved reserve

- Some vessel loss may be tolerated.
- Do not automatically equate emboli with renal failure.

Already compromised

- Additional microvascular injury may precipitate AKI.
- The same insult may carry a greater functional cost.

An interaction—not a single threshold

Baseline × burden	Lower embolic burden	Higher embolic burden
Better baseline function	Often limited effect	Injury possible
Impaired baseline function	Injury possible	Greater concern for AKI

Conceptual interaction — no numerical risk thresholds implied.

The spectrum of clinical consequence

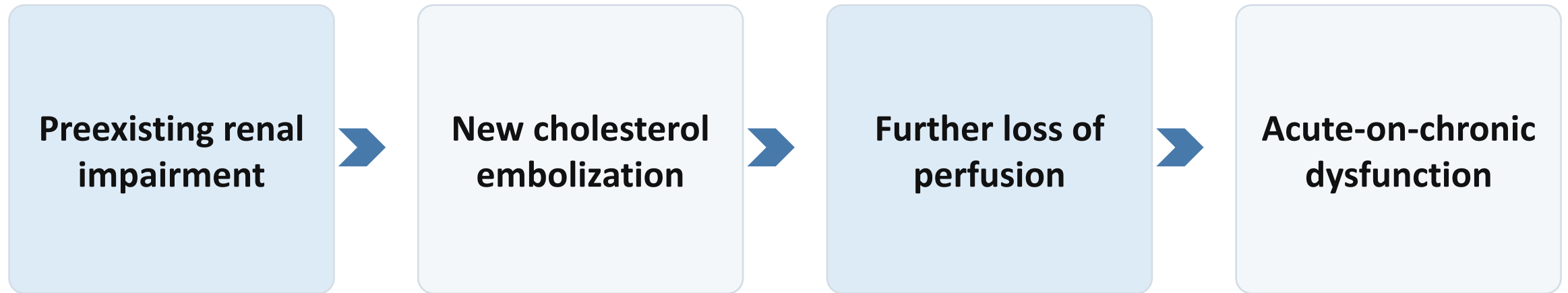


These are possible outcomes, not obligatory stages.

Why absence of AKI does not refute emboli

- Robbins explicitly says the emboli are frequently insignificant.
- Functional measurements reflect summed nephron activity.
- Sparse focal lesions may not produce a major global change.

Why vulnerable kidneys may deteriorate



The adverse event to recognize is AKI

Acute renal injury or failure can follow atheroembolization.

- Older adults with compromised renal function.
- The lesion is intrarenal arterial, not a primary tubular or immune deposit.
- Do not ascribe every postprocedure creatinine increase to this mechanism.

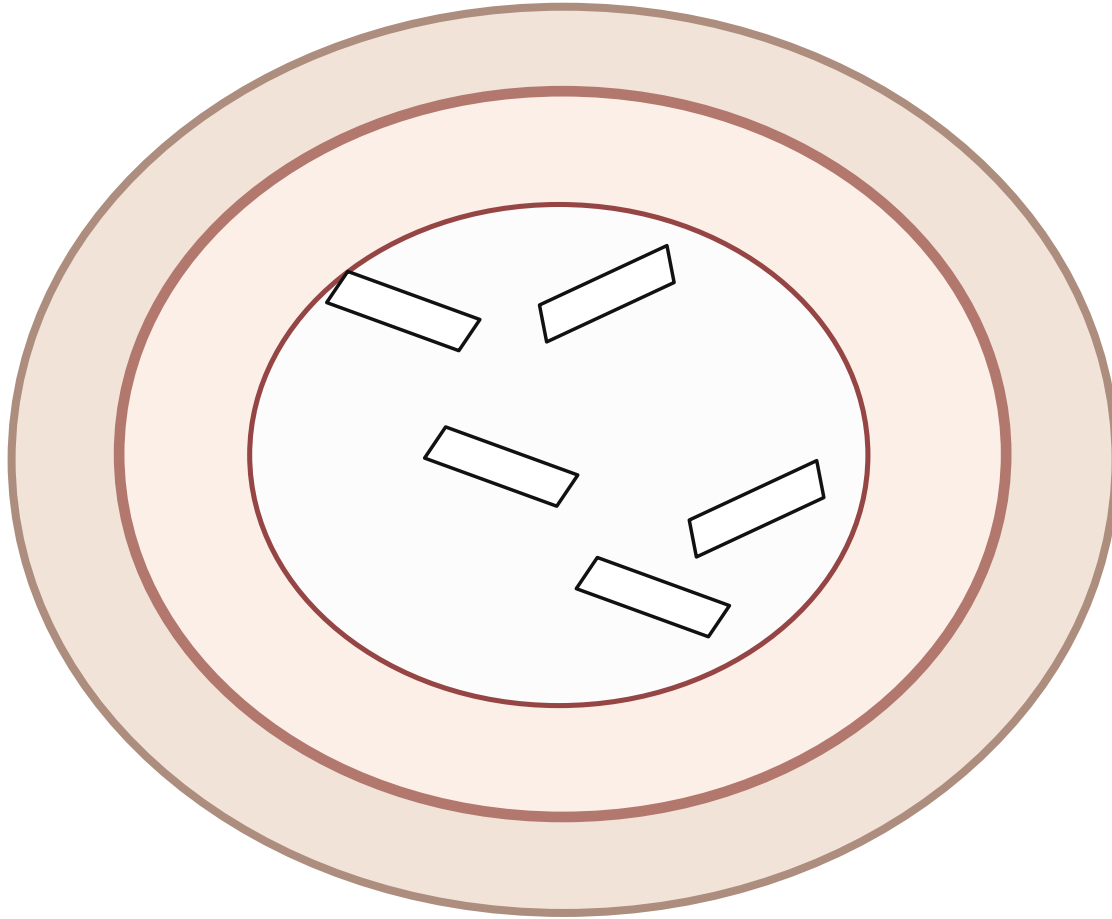
Histology anchors the diagnosis

Cholesterol crystals within an intrarenal arterial lumen are the signature.

- Look for angular or rhomboid-appearing empty clefts.
- Identify the vascular compartment surrounding the clefts.
- Correlate the finding with atherosclerotic disease and exposure history.

Vessel-wall versus luminal location matters

Original native PowerPoint schematic — NOT a photomicrograph



Arterial wall

Intrarenal vessel lumen

Empty angular cholesterol clefts

Cholesterol clefts are processing footprints

**Cholesterol
crystals in
plaque debris**



**Tissue fixation /
routine
processing**



**Lipid material
removed**



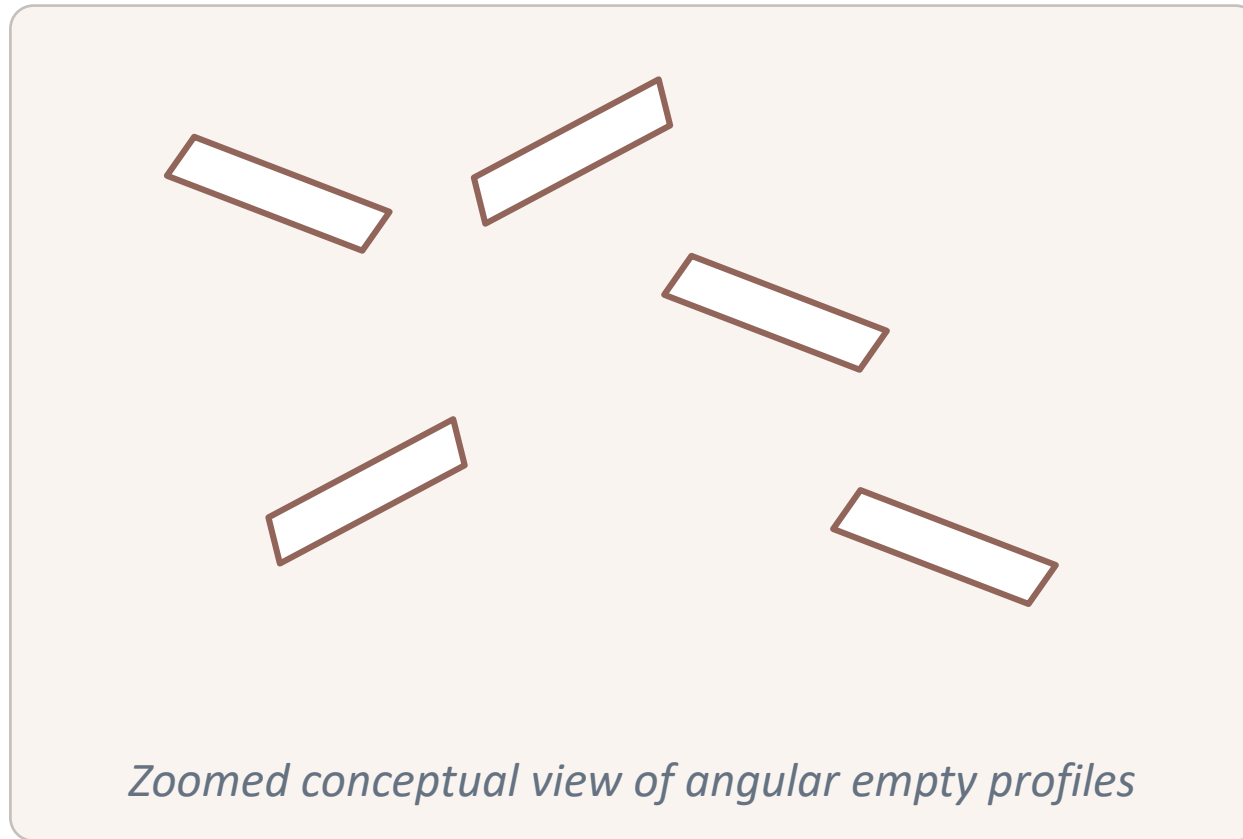
**Angular empty
clefts remain**

Why the clefts appear empty

- Routine paraffin processing extracts much of the lipid.
- The former crystal space persists as a sharply bounded empty profile.
- A pale cleft is therefore indirect morphologic evidence of cholesterol.

Rhomboid clefts are the textbook hallmark

Original native PowerPoint schematic — NOT a photomicrograph



**Rhomboid / angular
clefts**

Lipid has been removed
during ordinary histologic
processing.

Arcuate and interlobular arteries are involved

Site identified

- Emboli may occupy arcuate arteries.
- Interlobular arteries are also described.

What a pathologist should report

- The lesion is intraluminal.
- Describe cholesterol clefts and the artery involved when identifiable.

Do not confuse a cholesterol embolus with an atheroma

In situ atheroma	Downstream cholesterol embolus
Located in arterial intima	Located in an arterial lumen
Fibrous cap and lipid-rich core	Fragmented material with cholesterol clefts
Can generate stenosis or rupture	Represents material shed from a source plaque

The histologic label requires context

- First recognize the vessel, not merely an isolated clear space.
- Next identify a group of characteristic angular intraluminal clefts.
- Then assess whether their distribution fits downstream embolization.
- Finally integrate history without replacing tissue evidence by assumption.

Morphology cannot measure global renal function

What a section can show

- A discrete embolus in a sampled artery.
- Its luminal location and shape.

What a section cannot establish alone

- Exact total embolic burden across both kidneys.
- The clinical trajectory without laboratory context.

Gross morphology: Avoid false specificity

- The short Robbins atheroembolism section specifies the arterial microscopic lesion.
- It does not describe a unique gross renal appearance for this disorder.
- Gross wedge-shaped infarcts belong to the separate renal-infarct discussion.
- Do not treat a generic pale kidney as diagnostic of cholesterol emboli.

Three renal vascular mechanisms

Mechanism	Primary compartment	Signature result
Atheroembolism	Intrarenal arterial lumen	Cholesterol-crystal clefts
Renal artery stenosis	Large-artery intima/lumen	Chronic renal ischemia
TMA	Capillaries / arterioles	Microthrombi with endothelial injury

Compare with atherosclerotic ischemic renal disease

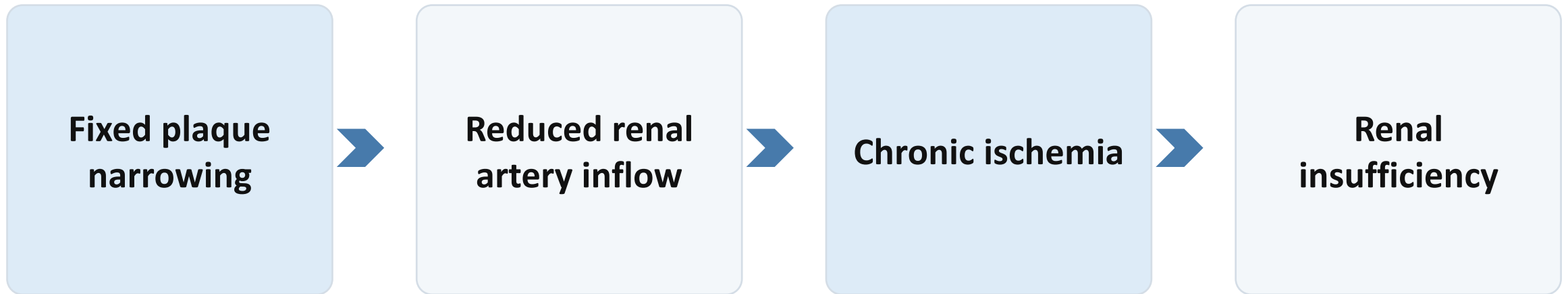
Ischemic renal disease

- Bilateral renal artery disease can cause chronic ischemia.
- Hypertension may or may not accompany insufficiency.

Atheroembolic renal disease

- Plaque fragments occlude downstream arteries.
- The source emphasizes a variable, sometimes acute injury.

Renal artery stenosis produces another pathway



Renovascular hypertension is a different clue

Renal artery stenosis

- An ischemic kidney can increase renin release.
- Renin–angiotensin activation can elevate blood pressure.

Atheroembolism

- A cholesterol cleft is the defining tissue finding.
- Hypertension is not specified as the hallmark in the core paragraph.

Renal infarction is not synonymous with microembolization

Renal infarct	Atheroembolic lesion
Commonly wedge-shaped ischemic necrosis	Arterial cholesterol-crystal microemboli
Often from thromboemboli of cardiac origin	From aorta or renal-artery atheroma
Can cause flank pain and hematuria	Can be silent or cause acute renal dysfunction

Thrombotic microangiopathy has a different core lesion

TMA	Atheroembolic disease
Platelet-rich microthrombi	Cholesterol-rich plaque fragments
Glomerular capillaries / arterioles	Arcuate / interlobular arteries described
Syndromic hemolysis and thrombocytopenia	Not diagnostic features in the Robbins core definition

Hypertensive nephrosclerosis: A different morphology

Nephrosclerosis

- Arteriosclerosis and arteriolosclerosis.
- Glomerulosclerosis with chronic tubulointerstitial scarring.

Atheroembolism

- Specific luminal cholesterol clefts.
- Potential acute worsening in compromised kidneys.

A structured comparison of the differentials

Disorder	Typical mechanism	Most discriminating clue
Atheroembolism	Embolized plaque debris	Cholesterol clefts
Renal artery stenosis	Flow-limiting large-artery lesion	Fixed stenosis / chronic ischemia
Renal infarct	Arterial occlusion with tissue necrosis	Wedge-shaped infarction
TMA	Endothelial / platelet thrombotic injury	Microvascular thrombi

Clinical suspicion is a chain of evidence

**Older adult with
atherosclerosis**



**Recent vascular
manipulation**



**New renal
dysfunction, if
present**



**Seek vascular
lesion evidence**

Three questions before attributing kidney injury

- Is there a plausible atheromatous source?
- Is the time course compatible with plaque disturbance?
- Is the renal lesion demonstrated or otherwise convincingly supported?
- Could fixed stenosis, infarction or TMA better explain the findings?

Case 1 — an incidental tissue finding

HYPOTHETICAL CLINICOPATHOLOGIC VIGNETTE

An older adult with advanced atherosclerosis has an intrarenal arterial cholesterol embolus, but renal function remains stable.

QUESTION

Does this contradict Robbins?

Interpretation: No. Atheroemboli are frequently clinically insignificant; Burden and baseline function affect outcome.

Case 2 — injury after aortic instrumentation

HYPOTHETICAL CLINICOPATHOLOGIC VIGNETTE

An older patient with reduced baseline renal function develops acute kidney injury after an aortic procedure.

QUESTION

What mechanism should enter the vascular differential?

Interpretation: Embolization of cholesterol-rich plaque material into intrarenal vessels. The time course alone is not proof.

Case 3 — chronic narrowing without emboli

HYPOTHETICAL CLINICOPATHOLOGIC VIGNETTE

An older individual has bilateral atherosclerotic renal artery disease with gradual renal insufficiency.

QUESTION

Which mechanism better fits this description?

Interpretation: Chronic ischemic renal disease caused by large-artery stenosis, not necessarily cholesterol embolization.

Case 4 — a wedge-shaped pale lesion

HYPOTHETICAL CLINICOPATHOLOGIC VIGNETTE

A renal specimen shows a sharply demarcated wedge-shaped zone of ischemic coagulative necrosis.

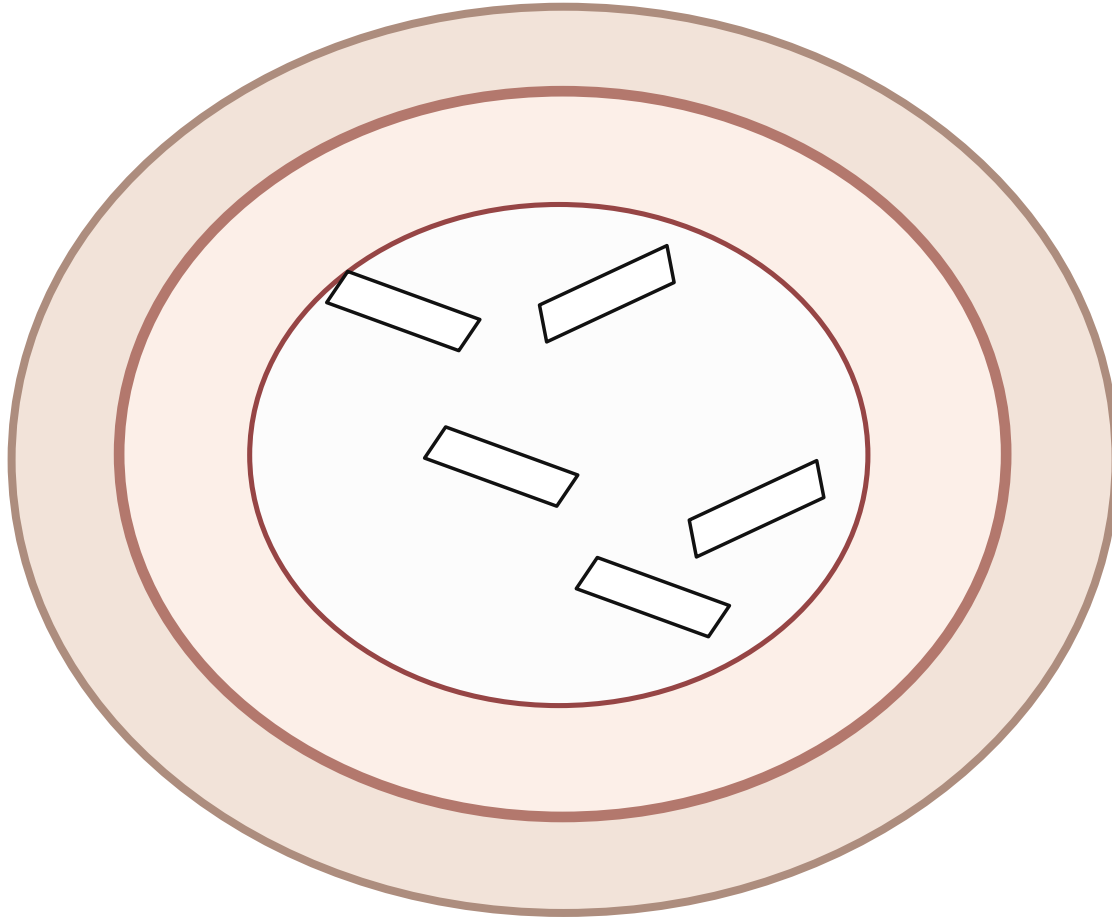
QUESTION

Which lesion must be distinguished from atheroembolism?

Interpretation: Renal infarction. A gross wedge is not the defining cholesterol-embolus lesion.

Microscopic spotter — reason from location

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Which compartment?

What are the angular empty profiles?

Which plaque source is plausible?

What establishes that this is an embolic lesion rather than an intimal atheroma?

Causal exercise — connect the modifiers

HYPOTHETICAL CLINICOPATHOLOGIC VIGNETTE

Two patients receive a similar plausible embolic insult. One has preserved renal function; The other has substantial preexisting impairment.

QUESTION

What does the source predict about variability?

Interpretation: Outcome depends on both embolic number and preexisting renal function. No numeric risk estimate is given.

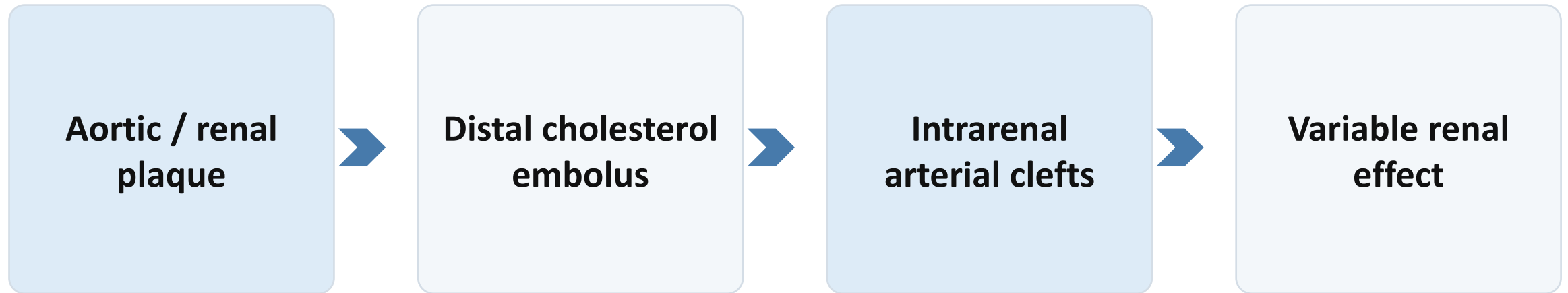
Diagnostic pitfall: Procedure does not equal disease

- Vascular instrumentation increases plausibility, not certainty.
- An isolated renal injury can have other mechanisms.
- Atheroembolic disease is defined by plaque-derived intravascular debris.
- Clinical correlation should not replace morphologic discrimination.

Diagnostic pitfall: All emboli are not identical

- An embolus may consist of thrombus, atheromatous debris or other material.
- The source and composition change the expected morphology.
- Cholesterol-crystal clefts specifically support plaque-fragment embolization.

Integrating source, lesion and consequence



What should be retained?

- Source: Severe aortic or renal arterial atherosclerosis.
- Triggers: Abdominal aortic surgery, aortography and intra-aortic cannulation.
- Microscopy: Cholesterol crystals leaving rhomboid clefts in intrarenal arteries.
- Outcome: Often inconsequential, but AKI can occur with impaired reserve.

Source, scope and image-access references

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. (2026). Chapter 20, p. 855: Atheroembolic Renal Disease.
- Robbins, Cotran & Kumar. Chapter 11: Atherosclerotic plaque composition, rupture and atheroembolism.
- Robbins, Cotran & Kumar. Chapter 20: Renal artery stenosis, thrombotic microangiopathy and renal infarcts.
- Open-licensed authentic renal biopsy micrographs: Wikimedia Commons, “Cholesterol embolus — intermed mag” (Nephron, CC BY-SA 3.0).
- The linked Commons specimens are not embedded in this deck. Morphologic illustrations here are original editable schematics.