

Vascular Pathology in Hypertension

Mechanisms • Small-vessel morphology • Organ injury

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Learning objectives

- Recognize hyaline, hyperplastic, and necrotizing arteriolar lesions
- Explain how chronic pressure injury produces luminal narrowing and ischemia
- Trace hypertensive renal vascular injury to nephrosclerosis
- Distinguish arteriolosclerosis from other arteriosclerotic patterns

Why the vascular wall matters

- Persistently elevated arterial pressure damages target-organ vessels
- Structural adaptation may preserve integrity but reduce luminal caliber
- Small-vessel ischemia and large-artery complications arise by different routes
- Histologic patterns provide clues about injury severity and duration

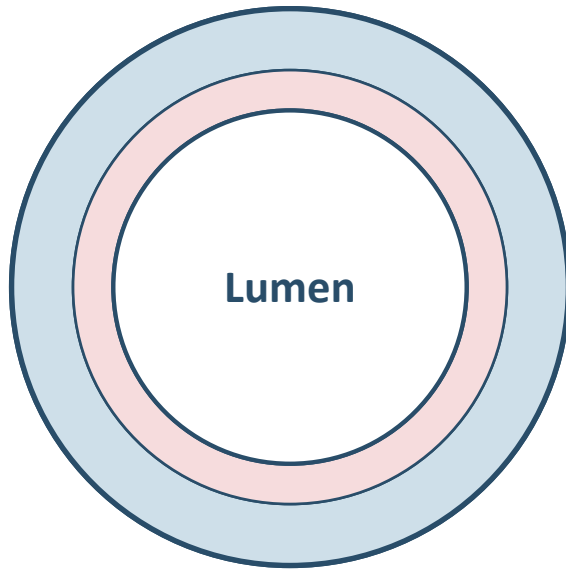
Scale of hypertensive vascular injury

Vascular territory	Dominant consequence	Illustrative pathology
Arterioles / small arteries	Wall thickening; perfusion loss	Hyaline or hyperplastic lesions
Large / medium arteries	Degeneration and superimposed atherosclerotic injury	Dissection; cerebrovascular hemorrhage
Pulmonary arterioles	Wall remodeling with raised pulmonary pressure	Intimal fibrosis; medial hyperplasia

Arteriolosclerosis is not atherosclerosis

- Arteriolosclerosis affects arterioles and small arteries
- Atherosclerosis is an intimal lipid-rich plaque process of larger arteries
- Hypertension can cause arteriolosclerosis and accelerate atherogenesis
- The lesions have different microscopic architecture

Normal arteriole: Baseline for comparison

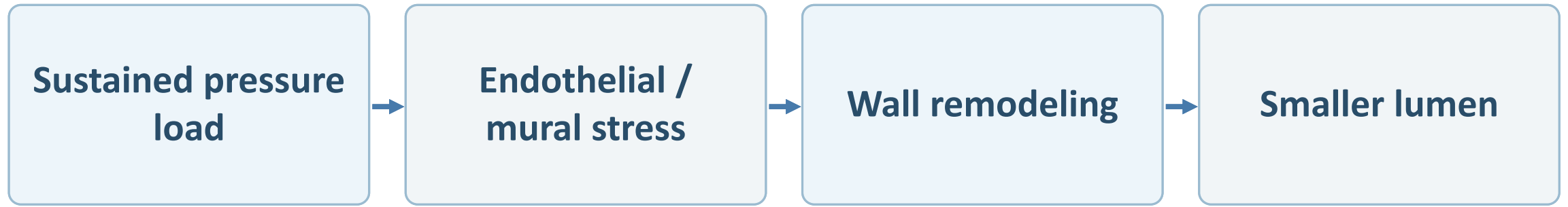


Schematic vessel cross-section — not a histologic specimen

Morphologic interpretation

- Thin vessel wall relative to an open central lumen
- Endothelium forms the luminal barrier
- Smooth muscle regulates vessel caliber and resistance

From hemodynamic load to structural remodeling



The morphologic endpoint depends on intensity and pattern of vascular injury.

Structural adaptation versus tissue injury

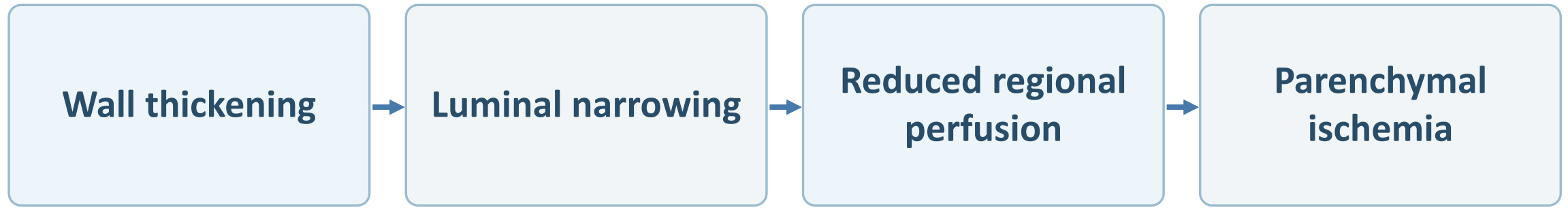
Remodeling

- Wall thickening can represent a response to prolonged hemodynamic stress
- Matrix synthesis and mural proliferation alter vessel architecture
- Remodeling may coexist with impaired perfusion

Injury

- Luminal loss restricts downstream blood supply
- Severe endothelial injury permits plasma-protein leakage
- Necrosis signals destructive vascular damage

Why a narrow arteriole damages its organ

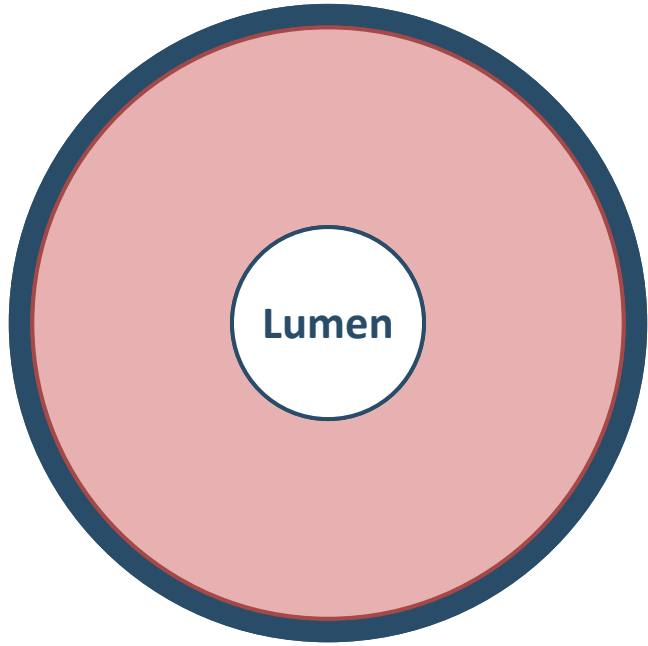


In the kidney this sequence contributes to glomerular and tubulointerstitial scarring.

Three hypertension-related small-vessel lesions

Pattern	Hallmark	Typical context
Hyaline arteriolosclerosis	Homogeneous eosinophilic wall thickening	Chronic hypertension; diabetes; aging
Hyperplastic arteriolosclerosis	Concentric laminated 'onion-skin' wall	Severe hypertension
Necrotizing arteriolitis	Fibrinoid injury with wall necrosis	Severe/malignant hypertension, particularly kidney

Hyaline arteriolosclerosis: Wall morphology

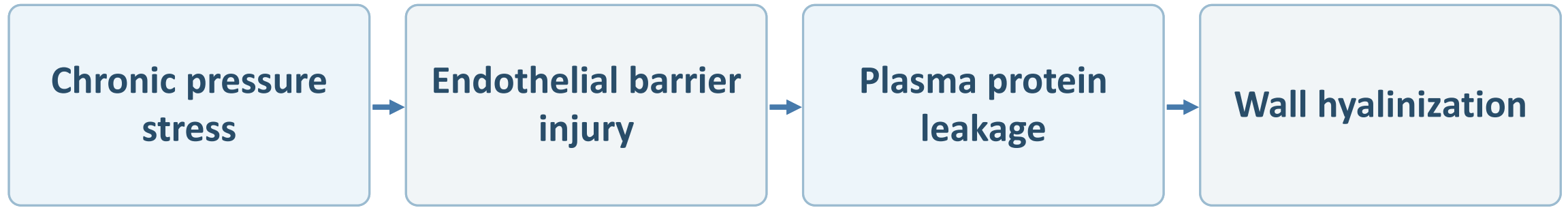


Schematic vessel cross-section — not a histologic specimen

Morphologic interpretation

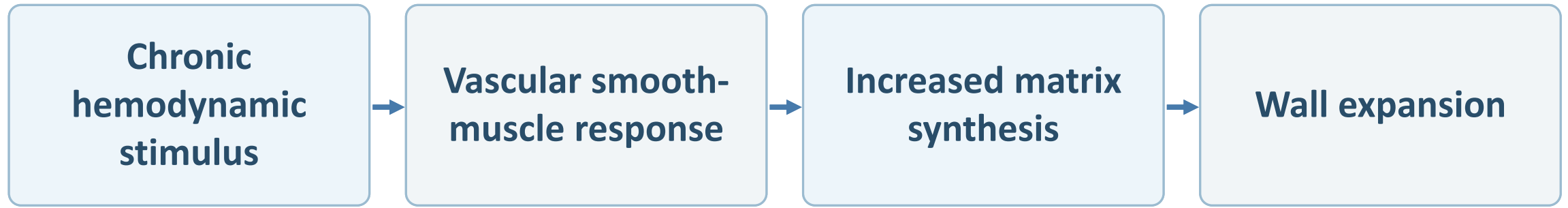
- Homogeneous pink, glassy material expands the arteriolar wall
- The residual lumen is smaller than that of an unaffected vessel
- Microscopic descriptor: Hyaline thickening

Hyalinization begins with endothelial injury



Protein deposition is one source of the characteristic eosinophilic change.

Smooth-muscle matrix synthesis adds to hyaline wall thickening



Hyaline arteriosclerosis is not explained by protein leakage alone.

Two contributors to hyaline arteriolosclerosis

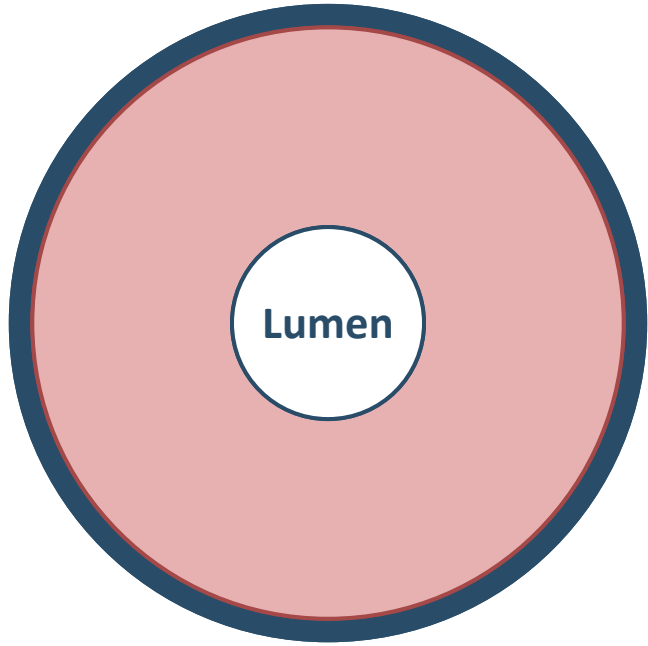
Plasma-protein leakage

- Barrier dysfunction permits plasma constituents to accumulate in the wall
- Protein leakage reflects endothelial injury
- Adds to homogeneous hyaline appearance

Matrix production

- Smooth-muscle cells synthesize increased extracellular matrix
- Pressure-related remodeling enlarges the wall
- Contributes to luminal narrowing

Hyaline lesion: Wall–lumen relationship



Schematic vessel cross-section — not a histologic specimen

Morphologic interpretation

- An expanded wall occupies more of the cross-sectional area
- A diminished lumen may limit downstream perfusion
- The most important physiologic effect is ischemia

Hyaline arteriolosclerosis and aging

- Hyaline changes are also seen in older normotensive patients
- Their presence alone does not establish hypertension
- Hypertension makes the process more generalized and severe
- Interpret the lesion with age and clinical context

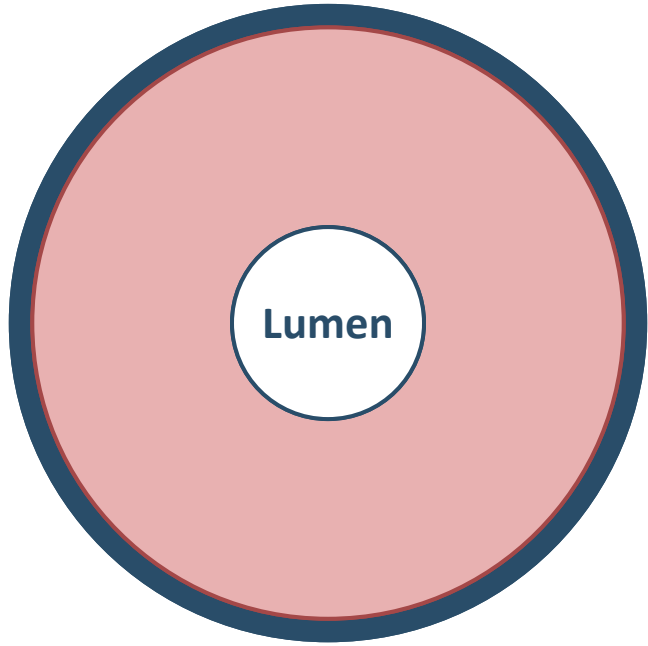
Diabetes increases the burden of hyaline injury

- Hypertension and diabetes both associate with hyaline arteriolosclerosis
- The combination may produce widespread small-vessel disease
- A glomerular lesion in a diabetic kidney may reflect additional pathology
- Do not attribute every hyalinized glomerulus to hypertension alone

Hyaline arteriolosclerosis in the kidney

- Renal arteriolar wall thickening reduces blood delivery to parenchyma
- Ischemic glomerular scarring can develop downstream
- Tubules and interstitium may also undergo chronic ischemic injury
- This is a central vascular component of nephrosclerosis

Recognizing hyaline arteriolosclerosis



Schematic vessel cross-section — not a histologic specimen

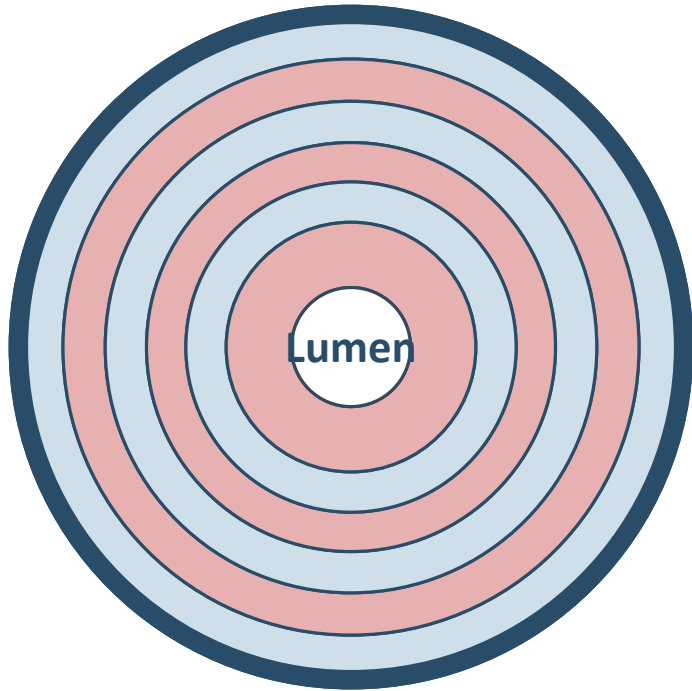
Morphologic interpretation

- Identify an arteriole rather than a glomerular capillary loop
- Assess circumferential hyaline wall thickening
- Confirm that the residual lumen is narrowed
- Evaluate accompanying chronic parenchymal scarring

What hyaline change cannot establish

- It is not specific for hypertension
- Histology alone does not determine an individual's blood pressure
- The lesion may coexist with diabetes-related and age-related changes
- Vascular findings should be integrated with the whole renal biopsy

Hyperplastic arteriolosclerosis: Defining architecture

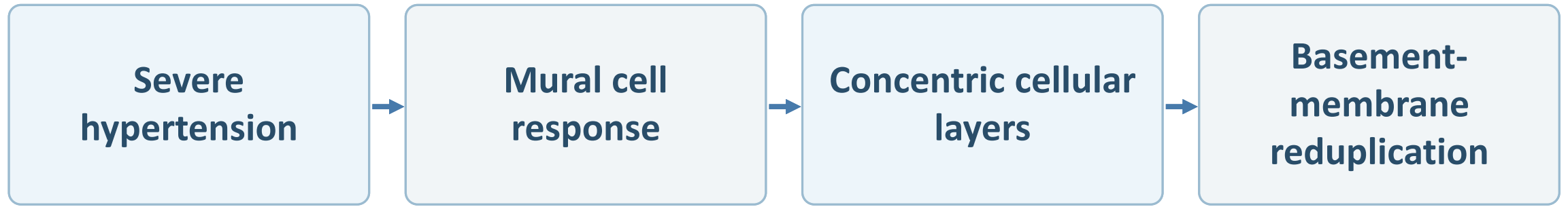


Schematic vessel cross-section — not a histologic specimen

Morphologic interpretation

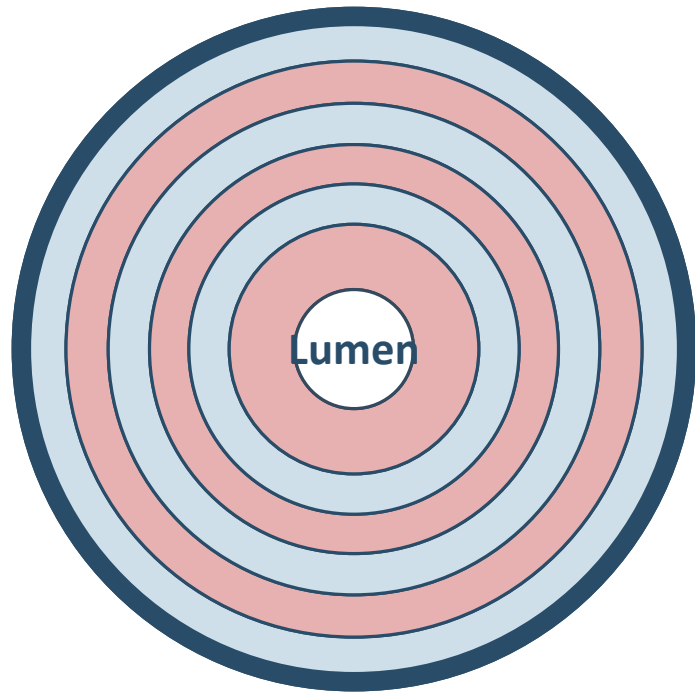
- Marked concentric, laminated thickening of the vessel wall
- Classic descriptor: “onion-skin” appearance
- The lumen may be extremely narrow or obliterated

Cellular and basement-membrane contributors



The laminations contain smooth-muscle cells and thickened, reduplicated basement membranes.

Concentric laminae produce 'onion-skinning'

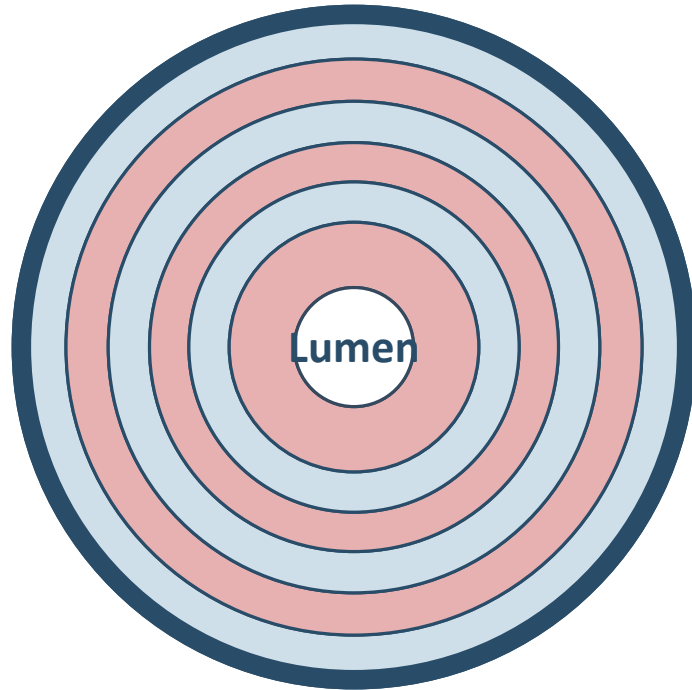


Schematic vessel cross-section — not a histologic specimen

Morphologic interpretation

- Multiple circumferential layers surround the lumen
- The repeated rings distinguish this pattern from homogeneous hyaline deposition
- This is an architectural rather than merely a color-based diagnosis

Hyperplastic arteriolosclerosis narrows the lumen



Schematic vessel cross-section — not a histologic specimen

Morphologic interpretation

- Each lamination adds to wall thickness
- Severe lesions can nearly obliterate arteriolar channels
- Downstream flow restriction can rapidly compromise renal function

Hyaline versus hyperplastic arteriolosclerosis

Hyaline

- Homogeneous eosinophilic mural material
- Protein leakage and increased matrix synthesis
- Seen in aging, hypertension and diabetes

Hyperplastic

- Concentric, laminated mural proliferation
- Smooth-muscle cells + reduplicated basement membranes
- Especially associated with severe hypertension

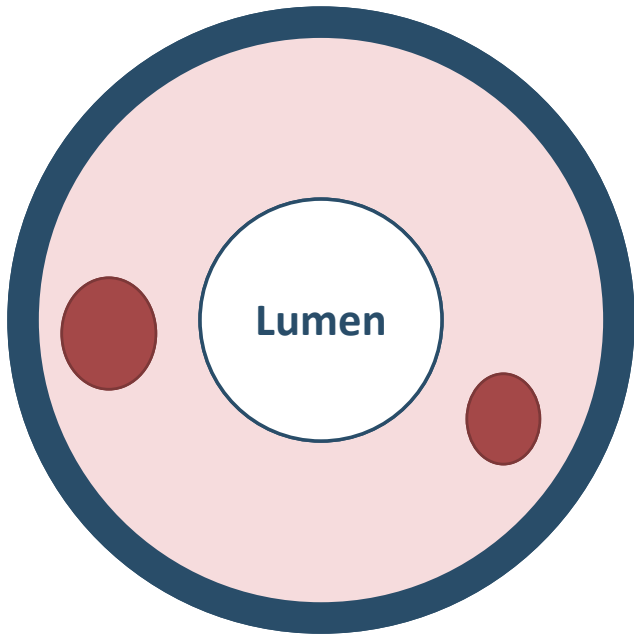
Diagnostic comparison of the arteriolar wall

Feature	Hyaline	Hyperplastic
Microscopic pattern	Uniform glassy pink expansion	Concentric cellular / membranous layers
Main structural process	Protein leakage + matrix deposition	Cellular proliferation + membrane duplication
Lumen	Narrowed	Often markedly narrowed
Clinical setting	Chronic hypertension; age; diabetes	Severe hypertension

Severe hypertension changes the lesion spectrum

- Hyperplastic arteriolosclerosis characterizes severe pressure injury
- Wall necrosis and fibrinoid deposits may accompany the lamination
- These changes are not obligatory features of every hyperplastic arteriole
- Examine multiple vessels because patterns can coexist

Necrotizing arteriolitis: Destructive mural injury



Schematic vessel cross-section — not a histologic specimen

Morphologic interpretation

- Fibrinoid material is deposited in injured walls
- Vascular wall necrosis differentiates it from simple chronic thickening
- The kidney is a particularly important site

Fibrinoid material versus simple hyalinization

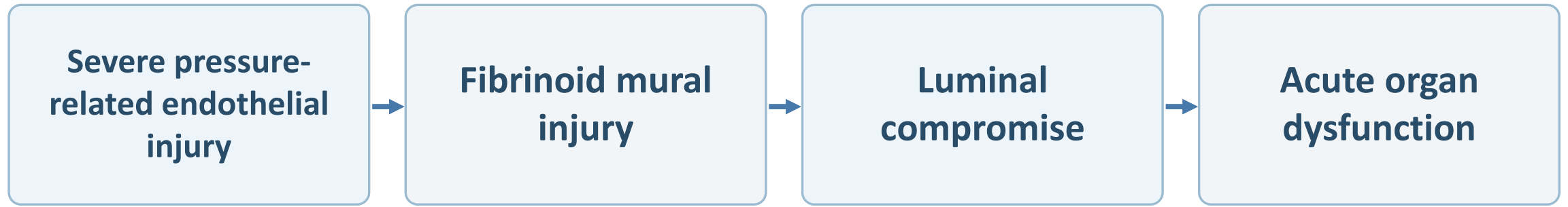
Hyaline arteriolosclerosis

- Chronic homogeneous wall thickening
- Strong association with vascular protein leakage and matrix synthesis
- Typically accompanies a longer-standing remodeling pattern

Necrotizing injury

- Destructive injury with fibrinoid deposits
- Can occur in severe/malignant hypertension
- May be accompanied by glomerular microthrombi

Destructive renal microvascular injury



Necrotizing arteriolitis can coexist with hyperplastic lesions in severe hypertension.

Necrotizing arteriolitis: Renal distribution

- Arterioles are the characteristic vessels involved
- The process may also involve glomerular capillary structures
- Microthrombi can occur within glomeruli
- The biopsy may show both acute injury and chronic vascular remodeling

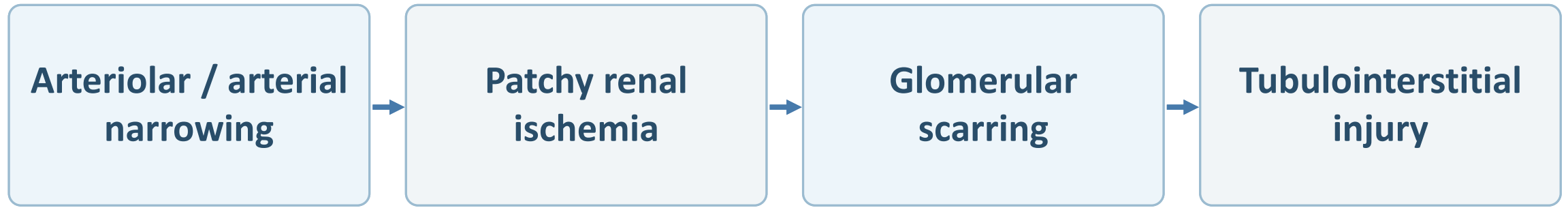
Three morphologic patterns — why they matter

Lesion	Dominant morphology	Primary implication
Hyaline	Glassy eosinophilic thickening	Chronic narrowing / ischemia
Hyperplastic	Concentric laminated thickening	Severe stenosis or obliteration
Necrotizing	Fibrinoid injury and necrosis	Destructive acute vascular injury

Nephrosclerosis is a constellation, not one lesion

- Arteriosclerosis and arteriolosclerosis coexist with parenchymal scarring
- Glomerulosclerosis is a frequent downstream consequence
- Tubular atrophy and interstitial fibrosis complete the morphologic pattern
- The diagnosis is associated with hypertension but is not absolutely specific

Nephrosclerosis: Vascular cause to parenchymal effect

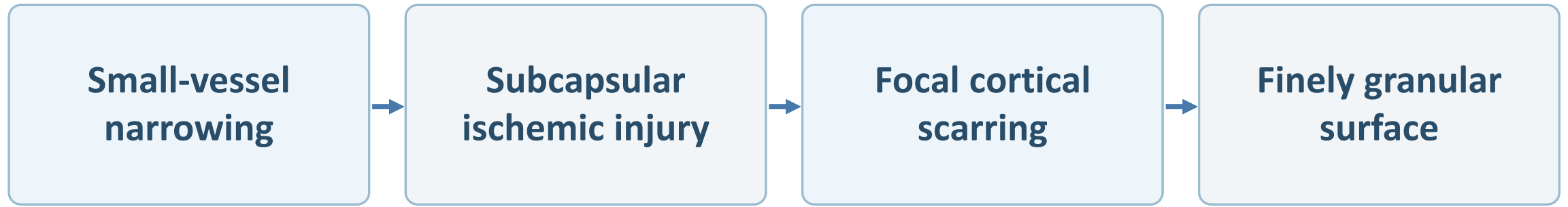


Loss of functioning renal parenchyma links the vascular lesion to organ dysfunction.

Renal surface in chronic nephrosclerosis

- Kidneys may be normal sized or moderately reduced in size
- The cortical surface can become finely and evenly granular
- Surface granularity reflects microscopic subcapsular scars
- Loss of mass is largely due to cortical shrinking and scarring

Why the cortex becomes finely granular



The gross finding reflects multiple small zones of parenchymal loss.

Glomerular consequences of chronic hypoperfusion

- Some glomeruli develop basement-membrane collapse
- Periglomerular fibrosis and collagen in Bowman space may occur
- End-stage affected glomeruli can become globally sclerosed
- Uninjured and scarred units may be intermixed

Tubular and interstitial consequences

- Ischemia may cause focal tubular atrophy
- Chronic injury leads to interstitial fibrosis
- Tubular dropout can align with subcapsular glomerular scars
- These changes contribute to reduction of functional renal mass

Larger renal vessels remodel as well

- Interlobular and arcuate arteries may show medial hypertrophy
- The internal elastic lamina can be replicated
- Intimal myofibroblastic tissue increases
- This fibroelastic hyperplasia augments vascular narrowing

Patchy injury despite systemic hypertension

Relatively preserved regions

- Some vascular territories retain relatively preserved perfusion
- Glomeruli and tubules can appear comparatively intact
- Findings depend on the sampled region

Ischemic regions

- Other vascular territories have marked ischemia
- Sclerosed glomeruli and tubular dropout accumulate
- Focal areas may merge into broader scars

The renal ischemic lesion is heterogeneous

- Microscopic scars may alternate with better-preserved cortex
- Pronounced lesions can involve large parenchymal regions
- Wedge-shaped infarcts or regional scars may develop
- A biopsy represents only a fraction of the kidney

Clinical course of uncomplicated nephrosclerosis

- Progression to uremia is unusual in uncomplicated nephrosclerosis
- Renal functional consequences depend on severity of vascular narrowing
- Severe blood pressure elevations increase the risk of renal failure
- Clinical course cannot be inferred from one arteriole alone

Severe hypertension: A combined clinicopathologic syndrome

- Severe hypertension can present with rapid renal function loss
- The supplied Robbins section describes retinal hemorrhages and exudates
- Hyperplastic vascular remodeling may coexist with fibrinoid necrosis
- Acute end-organ injury, not a single morphologic label, is clinically consequential

Chronic nephrosclerosis versus severe-pressure renal injury

Chronic vascular pattern

- Hyaline arteriolosclerosis and fibroelastic hyperplasia
- Patchy chronic cortical scarring
- Glomerulosclerosis with tubular atrophy

Severe injury pattern

- Concentric hyperplastic lesions
- Possible fibrinoid arteriolar / glomerular injury
- Potentially rapid loss of renal function

Severe hypertension and TMA overlap morphologically

- Endothelial injury and microvascular damage occur in both settings
- Microangiopathic hemolytic anemia may accompany microvascular injury
- Glomerular microthrombi can be present with severe hypertension
- Morphology alone may not determine the initiating process

TMA is not synonymous with hypertension

- The pathogenesis of hypertension-related lesions from primary TMA
- Some molecular defects characteristic of TMA are absent in hypertension
- Clinical data and targeted evaluation are needed for causal attribution
- Avoid equating every thrombus-containing renal biopsy with primary TMA

Renal patterns at a glance

Compartment	Chronic narrowing	Severe injury
Arteriole	Hyaline wall expansion	Onion-skin lamination; possible necrosis
Small artery	Medial / intimal thickening	Concentric intimal proliferation
Glomerulus	Collapse, sclerosis, periglomerular fibrosis	Possible fibrinoid necrosis, microthrombi
Tubulointerstitium	Atrophy and fibrosis	Ischemic damage accompanying acute injury

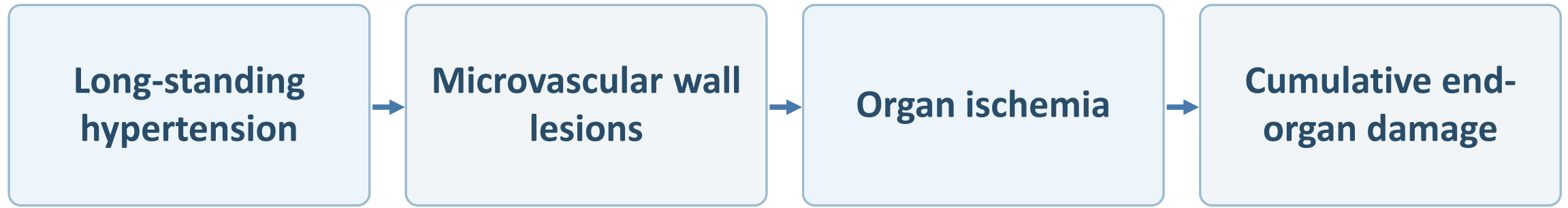
Hypertension accelerates atherogenesis

- Atherosclerosis and arteriolosclerosis are distinct but can coexist
- Hypertension is a major atherosclerotic risk factor
- Large-artery plaques add another mechanism of ischemia
- Do not misclassify lipid-rich intimal plaque as hyaline arteriolosclerosis

Large-artery complications are not arteriolosclerosis

- Elevated pressure can contribute to degenerative changes in arterial walls
- Aortic dissection represents a large-artery complication
- Cerebrovascular hemorrhage is another important consequence
- Vessel caliber determines the dominant morphology and clinical hazard

Pressure injury across vessel calibers



Large and medium arteries may develop separate degenerative or atheromatous complications.

Pulmonary hypertension produces a different remodeling pattern

- Pulmonary vascular pressure can rise for several distinct reasons
- Affected lung arterioles undergo intimal and medial alterations
- Fibrotic intimal thickening and medial hyperplasia are prominent
- Pulmonary remodeling should not be equated with systemic hyaline arteriosclerosis

Causes of pulmonary hypertension cited

Pathophysiologic setting	Example
Left-sided pressure transmission	Left heart failure; valvular disorders
Abnormal pulmonary blood flow	Congenital heart disease
Pulmonary parenchymal / airway disease	Obstructive or interstitial lung disease
Vascular obstruction	Recurrent thromboemboli

Pulmonary arteriolar remodeling

Intimal fibrosis

- Fibrotic thickening affects the intimal layer
- The channel available for blood flow may decrease
- Interpreted in pulmonary hemodynamic context

Medial hyperplasia

- Smooth-muscle compartment becomes more prominent
- Wall thickening reflects medial hyperplasia
- May coexist with intimal fibrosis

Arteriosclerosis: An umbrella term

- Arteriosclerosis means arterial wall thickening and loss of elasticity
- It is a morphologic family, not one histologic lesion
- Different patterns affect different vessel compartments
- Hypertensive arteriolosclerosis belongs to this broader family

Four major patterns of arteriosclerosis

Pattern	Primary wall change	Typical vessel
Arteriolosclerosis	Hyaline or hyperplastic wall thickening	Small arteries / arterioles
Mönckeberg medial sclerosis	Medial calcification	Muscular arteries
Fibromuscular intimal hyperplasia	Cellular / matrix expansion of intima	Larger muscular arteries
Atherosclerosis	Lipid-rich intimal atheroma	Large / medium arteries

Mönckeberg medial sclerosis is not an intimal plaque

Mönckeberg sclerosis

- Medial calcification, often near the internal elastic membrane
- Typically no meaningful luminal encroachment
- Commoner in older adults and renal dysfunction

Hypertensive arteriolosclerosis

- Hyaline deposition or laminated mural thickening
- Directly narrows small-vessel lumens
- Associated with chronic or severe hypertension

Fibromuscular intimal hyperplasia: Repair-related stenosis

- Occurs in muscular arteries larger than arterioles
- Can follow mechanical injury, inflammation or transplant arteriopathy
- Intimal expansion may produce substantial stenosis
- This lesion is a distinct response from hypertensive arteriolar hyaline sclerosis

Differential by arterial compartment

Compartment affected	Morphologic pattern	Interpretive clue
Arteriolar wall	Hyalinization or lamination	Hypertension-associated small-vessel disease
Medial muscular artery	Calcification	Mönckeberg sclerosis
Muscular arterial intima	Fibromuscular expansion	Repair-related stenosis
Larger-artery intima	Atheromatous plaque	Atherosclerosis

Case 1 — a narrowed renal arteriole

Clinical and histologic vignette

A patient with long-standing hypertension has cortical scarring and arterioles with homogeneous eosinophilic wall thickening and narrowed lumens.

Which vascular lesion best explains the renal ischemia?

Case 1 — morphologic interpretation

- The described lesion is hyaline arteriosclerosis
- Endothelial protein leakage and smooth-muscle matrix synthesis account for thickening
- Luminal loss reduces renal blood supply
- Glomerulosclerosis and tubulointerstitial fibrosis can develop downstream

Case 2 — concentric arteriolar layers

Clinical and histologic vignette

A patient with markedly severe hypertension and deteriorating renal function has arterioles displaying concentric laminated wall thickening with virtual luminal obliteration.

What vascular pattern is present and what is it composed of?

Case 2 — interpreting the onion-skin lesion

- The vascular lesion is hyperplastic arteriosclerosis
- Concentric smooth-muscle cellular layers and reduplicated basement membranes form the lamination
- Associated fibrinoid necrosis suggests more destructive injury
- The lesion can markedly impair renal perfusion

Case 3 — calcified arterial media

Clinical and histologic vignette

Histology shows calcification in the media of a muscular artery without significant encroachment on the lumen.

Is this the typical small-vessel lesion of systemic hypertension?

Case 3 — distinguish wall compartment

- The finding is characteristic of Mönckeberg medial sclerosis
- The dominant process is medial calcification rather than arteriolar wall hyalinization
- The lumen is commonly preserved
- It must be distinguished from both atherosclerosis and arteriosclerosis

Integrated lesion-to-consequence synthesis

Pressure-related setting	Morphology	Functional implication
Chronic systemic pressure	Hyaline arteriolosclerosis	Slow renal ischemic scarring
Severe systemic pressure	Hyperplastic lesions ± necrotizing injury	Marked perfusion loss; acute renal injury
Pulmonary vascular hypertension	Intimal fibrosis / medial hyperplasia	Pulmonary arteriolar remodeling
Large-artery pressure injury	Degeneration; accelerated atherogenesis	Dissection, hemorrhage, ischemic disease

Five high-yield recognition rules

- Glassy pink arteriolar wall → think hyaline arteriosclerosis
- Concentric onion-skin lamination → think hyperplastic arteriosclerosis
- Fibrinoid wall necrosis → recognize destructive vascular injury
- Renal scarring reflects downstream consequences of arterial narrowing
- Medial calcification without stenosis → consider Mönckeberg sclerosis

Selected sources and scope

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. 2026. Chapter 11, pp. 445–449.
- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. 2026. Chapter 20, pp. 851–852.
- Original editable vascular schematics created for conceptual explanation; no microscopy reproduced.