

Small Vessel Vasculitis

Pathogenesis • morphology • clinicopathologic recognition

Immune-complex and ANCA-associated disorders

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Learning objectives

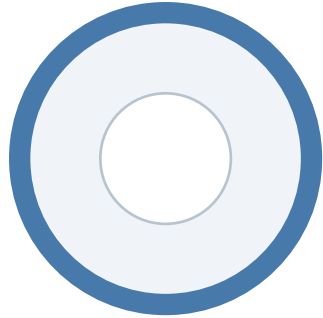
Classify small-vessel vasculitis by vascular immune deposits.

Explain immune-complex and ANCA-mediated injury to small vessels.

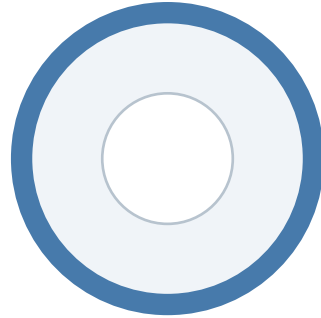
Recognize diagnostic morphology and characteristic organ involvement.

Differentiate microscopic polyangiitis, GPA, EGPA and major immune-deposit disorders.

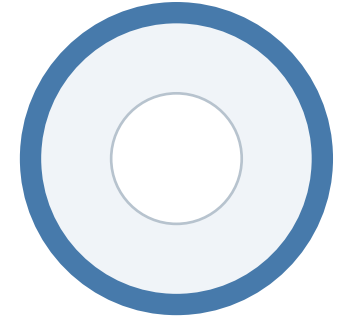
The vascular compartment defines the disease



Small artery / arteriole



Capillary bed



Postcapillary venule

SCHEMATIC — NOT HISTOLOGY

Most commonly affected: Small intraparenchymal arteries, arterioles, capillaries and venules.

Medium-sized arteries may also participate; Vessel caliber is a predominance, not an absolute boundary.

Organ manifestations reflect the vascular beds injured.

Two patterns of small-vessel immunopathology

Immune-deposit pattern

Ig and/or complement deposits
Often associated with immune-complex GN

Pauci-immune pattern

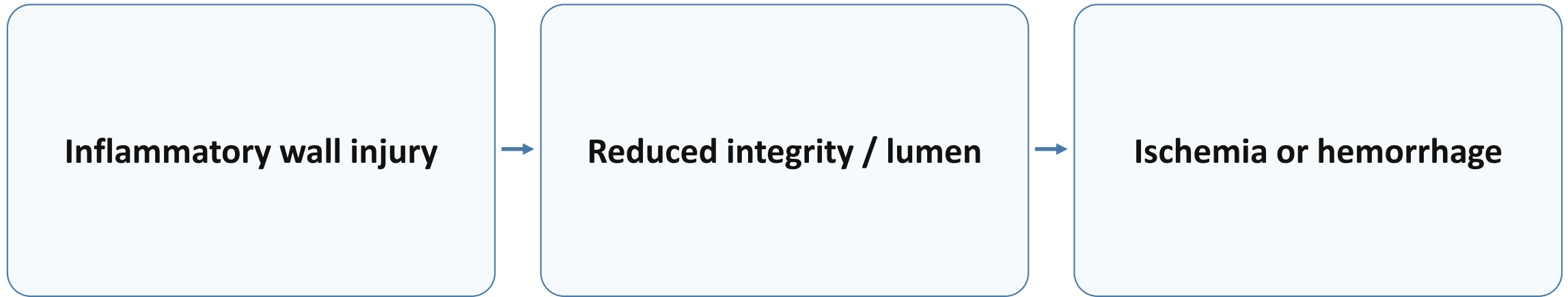
Few or no deposits in most lesions
Strong association with ANCA

Immune-complex disease: Vessel walls contain appreciable immunoglobulin and/or complement.

ANCA-associated disease: Necrotizing injury with few detectable immune deposits (pauci-immune).

Similar clinical and H&E appearances can arise by different mechanisms.

Vasculitis is vessel-wall inflammation



SCHEMATIC

Inflammatory destruction can narrow or occlude the lumen.

Vessel-wall injury can produce tissue hemorrhage, ischemia or thrombosis.

Constitutional symptoms may coexist with focal, organ-specific effects.

Mechanisms must be distinguished before treatment

Direct infectious injury

Vascular invasion by pathogen
Address underlying infection

Noninfectious immune injury

Autoantibodies, complexes or T cells
Mechanism guides therapy

Immune-mediated inflammation is a major cause of noninfectious vasculitis.

Microorganisms can also invade vessels directly or trigger indirect immune injury.

Immunosuppression can help immune vasculitis but may harm uncontrolled infection.

Why renal and pulmonary beds matter

Kidney

Glomerular capillary injury

Hematuria / proteinuria

Lung

Alveolar capillary injury

Hemorrhage / hemoptysis

Glomerular capillaries can undergo necrotizing glomerulonephritis.

Pulmonary capillaries can develop capillaritis and alveolar hemorrhage.

Concurrent hematuria and hemoptysis therefore suggest potentially severe small-vessel injury.

Immune deposits organize the differential



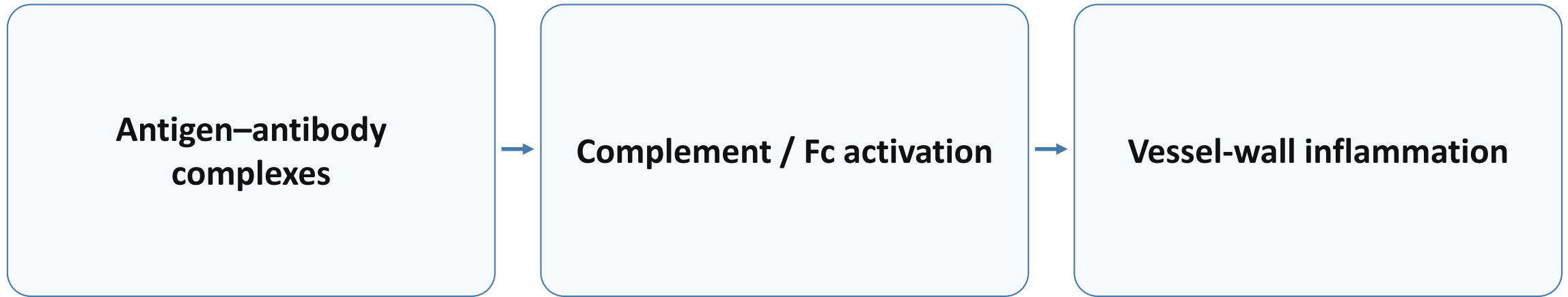
SCHEMATIC

Marked immune deposits favor immune-complex or antibody-mediated pathways.

Sparse deposits support a pauci-immune mechanism in an appropriate clinicopathologic context.

Leukocytoclastic change alone does not distinguish these categories.

Immune complexes initiate a vascular injury cascade



SCHEMATIC

Circulating complexes may deposit in vessel walls; Complexes may also arise locally.

The responsible antigen is often not identified.

Complement activation and Fc-receptor-bearing leukocytes propagate damage.

Immune-complex formation: Two possible locations

Preformed complexes

Circulate before vascular deposition

In-situ complexes

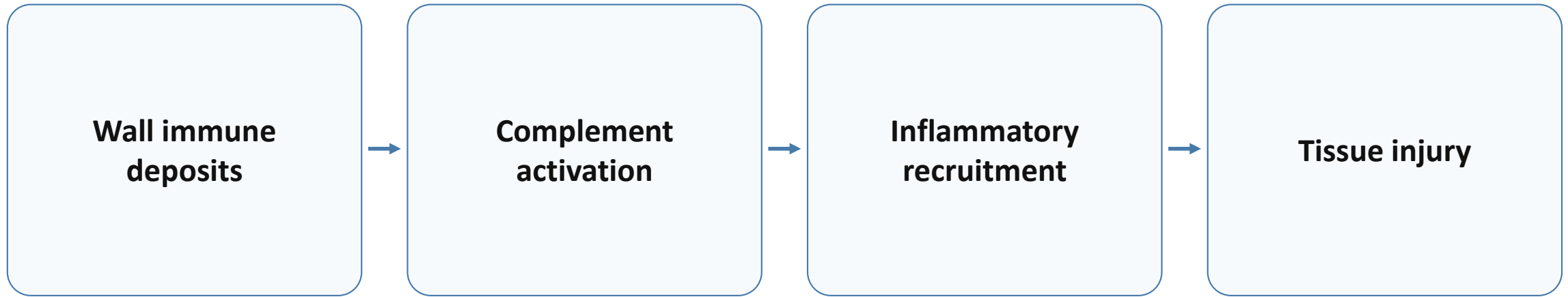
Antibody encounters antigen at tissue site

Circulating antigen–antibody complexes can become trapped in vessel walls.

Alternatively, complexes may form in situ after local antigen binding.

The relative importance of these routes often remains uncertain.

Complement amplifies immune-complex injury



SCHEMATIC

Vessel-wall antibody and/or complement deposition is detectable in lesional tissue.

Complement activation recruits and activates inflammatory effector cells.

The recruited inflammatory cells then damage the vascular wall.

Fc-receptor-bearing cells are central effectors



SCHEMATIC

Neutrophils bind antibody-containing complexes through Fc receptors.

Monocytes and macrophages can contribute to vascular inflammation.

Endothelial injury reflects activated inflammatory-cell interactions.

Immune-deposit vasculitis: Diagnostic tissue logic

Routine morphology

What kind of vessel injury exists?

Immunodeposit evaluation

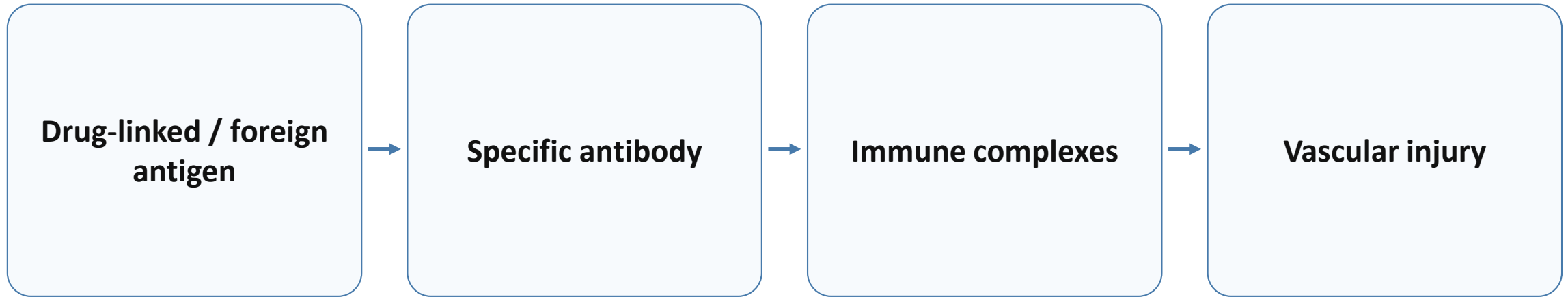
What immune material remains in the lesion?

H&E identifies inflammatory vascular injury; Immunostaining evaluates the deposited material.

Significant wall Ig and/or complement supports an immune-deposit pathway.

Immune-complex glomerulonephritis is a common associated renal pattern.

Drug hypersensitivity can form immune complexes



SCHEMATIC

Some drugs act as haptens after binding serum proteins (textbook example: Penicillin).

Some agents are foreign proteins themselves (textbook example: Streptokinase).

Antibodies then recognize these antigens and promote immune-complex formation.

Drug-associated vasculitis: Clinical priority

Exposure

Review drugs and timing

Seek a plausible trigger

Course

Disease can vary in severity

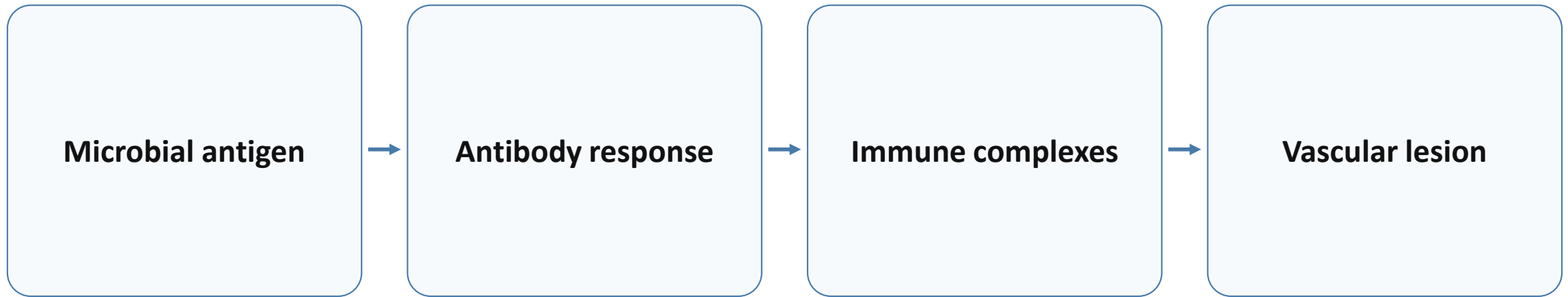
Removal may permit resolution

Skin lesions are the most frequent clinical manifestations.

The course ranges from mild self-limited disease to severe systemic illness.

Recognizing and withdrawing the offending agent is often decisive.

Infection may trigger indirect immune-complex injury



SCHEMATIC

Antibodies directed against microbial constituents can form circulating immune complexes.

Complexes may lodge in vascular walls and activate inflammatory pathways.

This indirect mechanism differs from direct microbial invasion of a vessel.

Hepatitis B illustrates the immune-complex bridge

Mechanism

HBV antigen–antibody complexes

Disease category

PAN: Predominantly medium-arterial vasculitis

A subset of polyarteritis nodosa (PAN) to HBsAg–anti-HBsAg complexes.

PAN remains classified under medium-vessel vasculitis, despite involving small arteries.

The example illustrates immune-complex pathogenesis, not reclassification of PAN as a small-vessel vasculitis.

Autoimmune disorders can produce vasculitis

SLE

Multiple autoantibodies

DNA–anti-DNA complexes highlighted

Rheumatoid arthritis

Long-standing severe disease

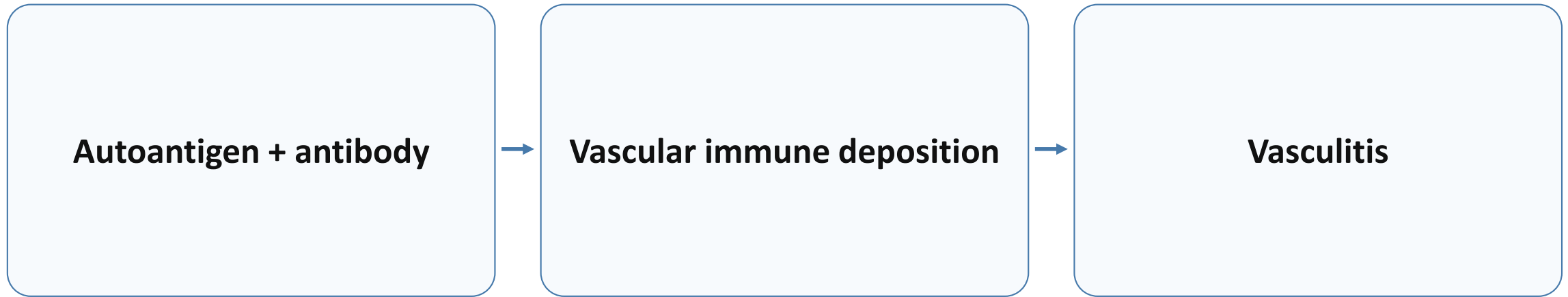
Small/medium-vessel vasculitis

Systemic lupus erythematosus, rheumatoid arthritis and antiphospholipid antibody syndrome are examples.

Autoantibodies may provide substrates for vascular immune-complex formation.

Some disorders have additional cell-mediated or antiendothelial contributions.

Systemic lupus erythematosus: Multiple complexes



SCHEMATIC

DNA–anti-DNA complexes are prominent candidates in SLE-associated vascular injury.

Other autoantibodies may also contribute to vasculitis.

SLE may affect small and medium-sized vessels.

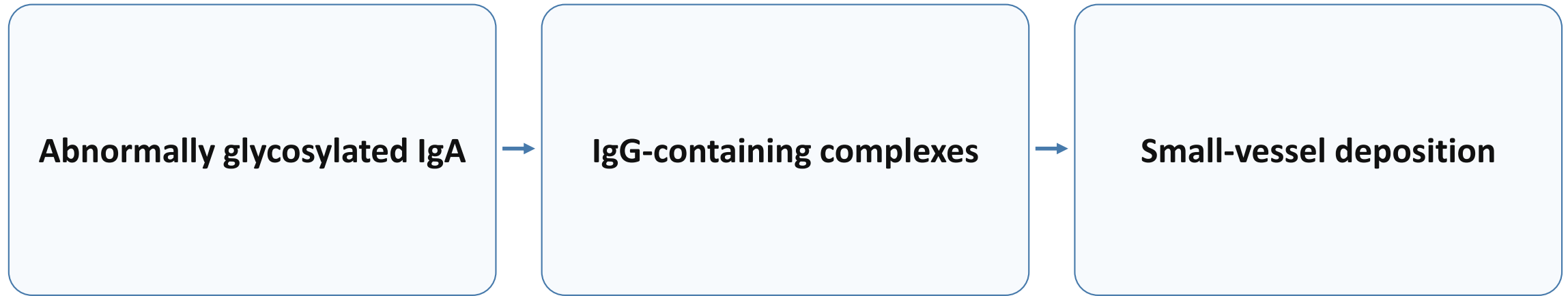
Rheumatoid vasculitis has a specific context

Typically occurs with long-standing, severe rheumatoid arthritis.

May involve both small and medium-sized vessels.

Antiendothelial antibodies and cell-mediated immunity may also participate.

IgA vasculitis: A distinctive immune-complex substrate



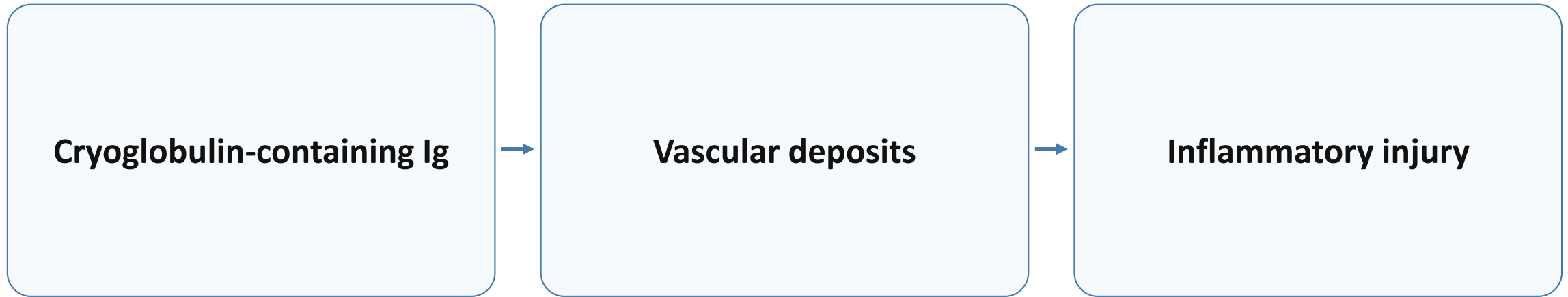
SCHEMATIC

IgA vasculitis was previously termed Henoch–Schönlein purpura.

Robbins implicates IgG complexes with abnormally glycosylated IgA.

It belongs to the immune-deposit subgroup of small-vessel vasculitis.

Cryoglobulinemic vasculitis: Deposit-related injury



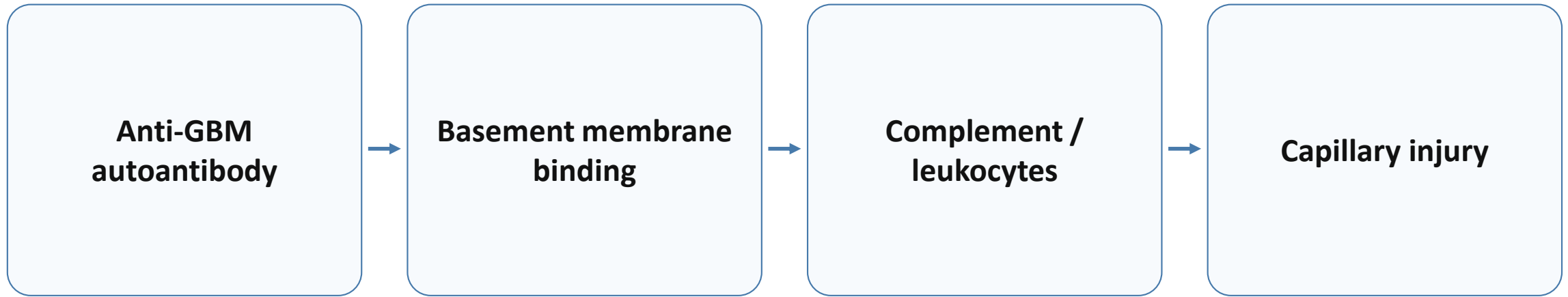
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Cryoglobulins are immunoglobulins that precipitate when cooled.

Cryoglobulin immune deposits are implicated in vascular inflammation.

Its placement within immune-deposit vasculitis reflects immunoglobulin deposition.

Anti-GBM disease: Direct antibody binding



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Autoantibodies directly recognize components of glomerular and pulmonary capillary basement membranes.

This is direct antibody binding rather than circulating immune-complex deposition.

Complement and leukocyte-mediated damage can be similar to immune-complex vasculitis.

Anti-GBM and immune complexes are not identical

Immune complexes

Antigen–antibody complexes
Deposited or formed in situ

Anti-GBM antibody

Direct tissue-antigen binding
Glomerular + pulmonary membranes

Both mechanisms may leave substantial immunoglobulin in involved tissue.

Immune complexes reflect antigen–antibody complexes; Anti-GBM antibody binds basement membrane directly.

Mechanistic interpretation requires immunostaining pattern plus clinical context.

ANCA targets are neutrophil-associated antigens

PR3-ANCA

Historically associated with c-ANCA labeling

MPO-ANCA

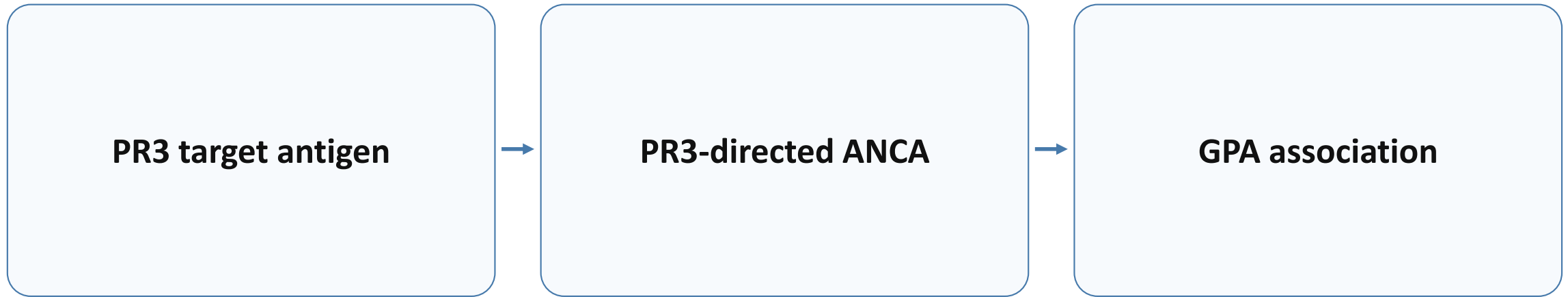
Historically associated with p-ANCA labeling

ANCAs react with constituents of neutrophil primary granules and related cell compartments.

The principal antigen-specific categories are proteinase 3 (PR3) and myeloperoxidase (MPO).

Antigen specificity is more useful than historical staining labels alone.

PR3-ANCA has a strong GPA association



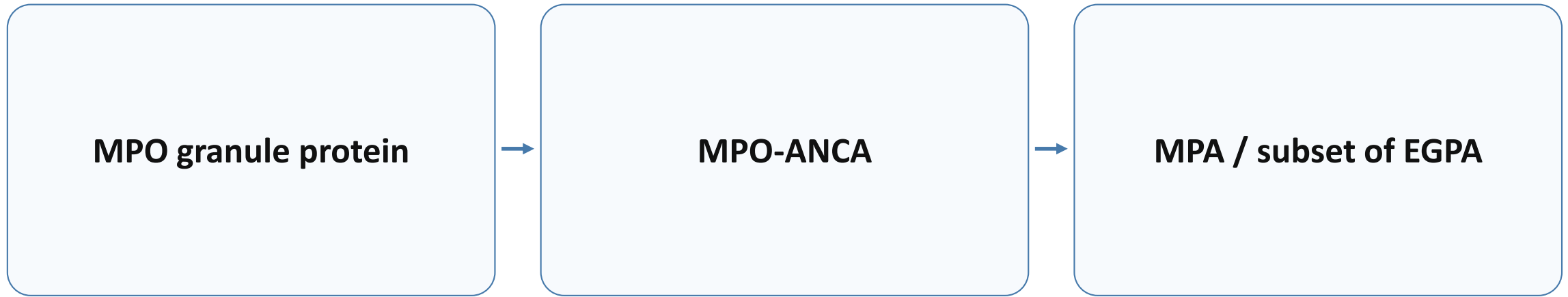
SCHEMATIC

Proteinase 3 is an enzyme in neutrophil azurophilic granules.

Microbial peptide homology as one possible explanation for PR3 autoimmunity.

PR3-ANCA is particularly associated with granulomatosis with polyangiitis.

MPO-ANCA links several phenotypes



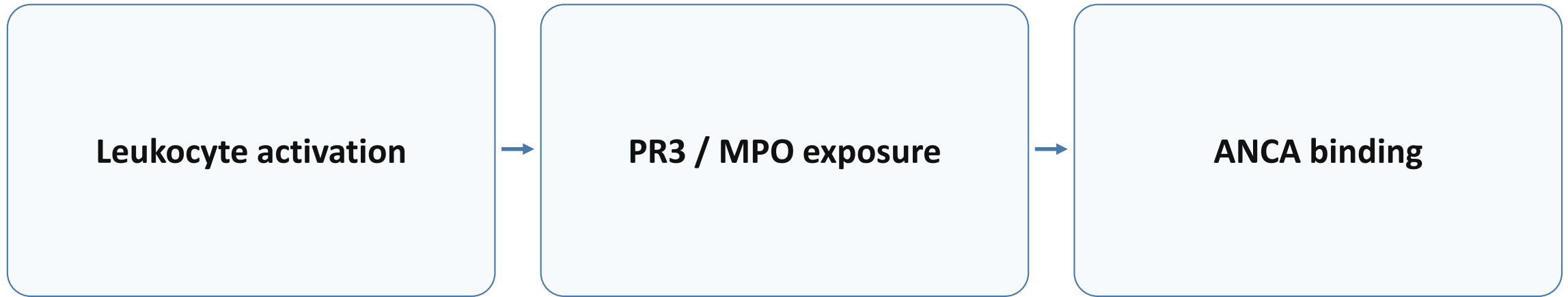
SCHEMATIC

Myeloperoxidase is a granule enzyme involved in reactive oxygen species production.

MPO-ANCA commonly accompanies microscopic polyangiitis.

It is also found in a subset of EGPA; Some therapeutic agents can induce it.

Intracellular antigens can become accessible



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ANCA antigens are principally intracellular in resting neutrophils.

Low-level surface expression or surface translocation occurs during activation or apoptosis.

Circulating ANCAs can then engage their targets on leukocytes.

How ANCAs promote pauci-immune vascular injury



SCHEMATIC

ANCA engagement can directly activate neutrophils.

Activated cells release reactive oxygen species and proteolytic enzymes.

Inflammatory-cell interactions with endothelium cause destructive vascular injury.

Pauci-immune does not mean nonimmune

Systemic immune driver

Circulating autoantibody activating cells

Local tissue appearance

Few detectable immune deposits despite injury

ANCA autoantibodies target cellular proteins rather than forming typical deposited immune complexes.

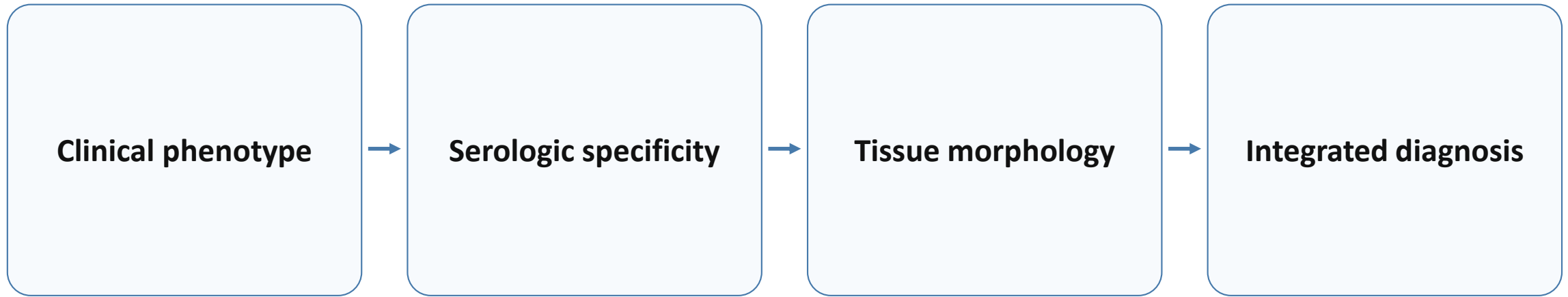
Lesions therefore usually contain few demonstrable antibodies or complement deposits.

Profound necrotizing inflammation can coexist with weak or absent tissue immunodeposits.

Three principal ANCA-associated syndromes

Entity	Defining tissue emphasis
Microscopic polyangiitis	Necrotizing small-vessel lesions; no granulomas
Granulomatosis with polyangiitis	Respiratory granulomas; necrotizing vasculitis
Eosinophilic GPA	Eosinophils and granulomas; asthma association

ANCA results require clinicopathologic integration



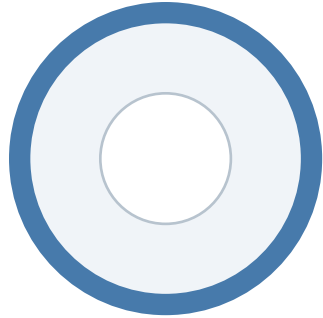
SCHEMATIC

ANCAs as useful diagnostic and disease-activity markers.

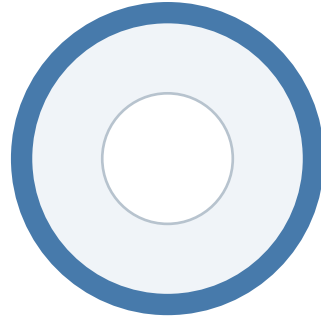
However, disease recognition also depends on affected organs and morphology.

The hallmark is a coherent constellation—not the antibody result alone.

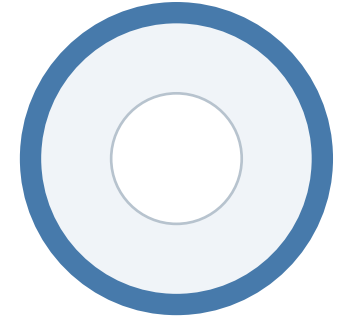
Microscopic polyangiitis: Disease definition



Capillary



Arteriole



Venule

SCHEMATIC — NOT HISTOLOGY

Necrotizing vasculitis of capillaries, arterioles and venules.

Medium-sized and large arterial involvement is not the dominant pattern.

MPO-ANCA is associated with most cases; Deposits are generally sparse.

Microscopic polyangiitis affects multiple beds

Kidney

Necrotizing glomerulonephritis

Hematuria / proteinuria

Lung

Pulmonary capillaritis

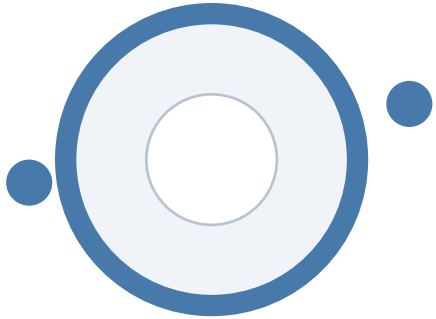
Hemoptysis

Skin, mucosa, lung, kidney, muscle, gastrointestinal tract and other organs may be involved.

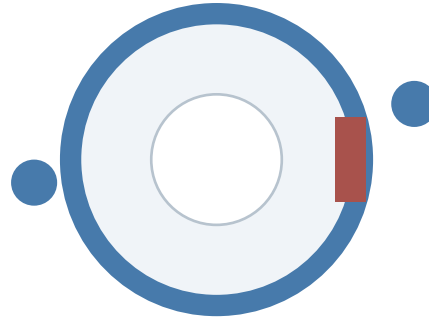
Necrotizing glomerulonephritis and pulmonary capillaritis are especially important.

Vascular distribution accounts for the diversity of presenting symptoms.

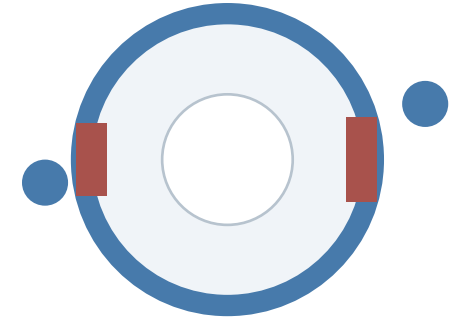
MPA morphology: Segmental necrotizing injury



Segmental media injury



Fibrinoid necrosis



Transmural extension

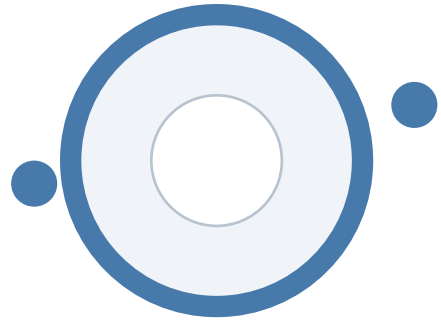
SCHEMATIC — NOT HISTOLOGY

A segment of the media develops fibrinoid necrosis.

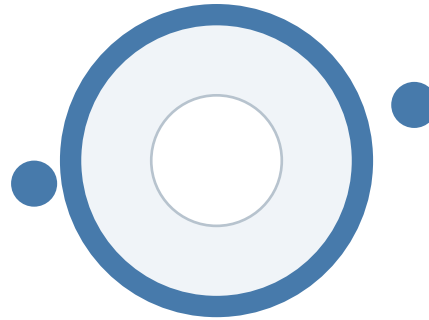
Transmural necrotizing foci may also occur.

Granulomatous inflammation is absent in classic MPA.

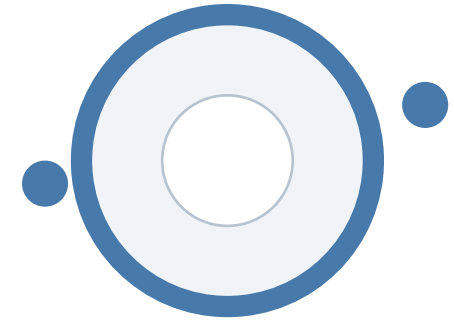
Leukocytoclastic vasculitis is a pattern



Neutrophil infiltration



Nuclear fragmentation



Wall damage

SCHEMATIC — NOT HISTOLOGY

Neutrophils infiltrate and fragment, especially in postcapillary venules.

Fragments of neutrophil nuclei produce leukocytoclasia.

The same pattern may accompany immune-complex vasculitis; It is not ANCA-specific.

MPA-associated renal injury



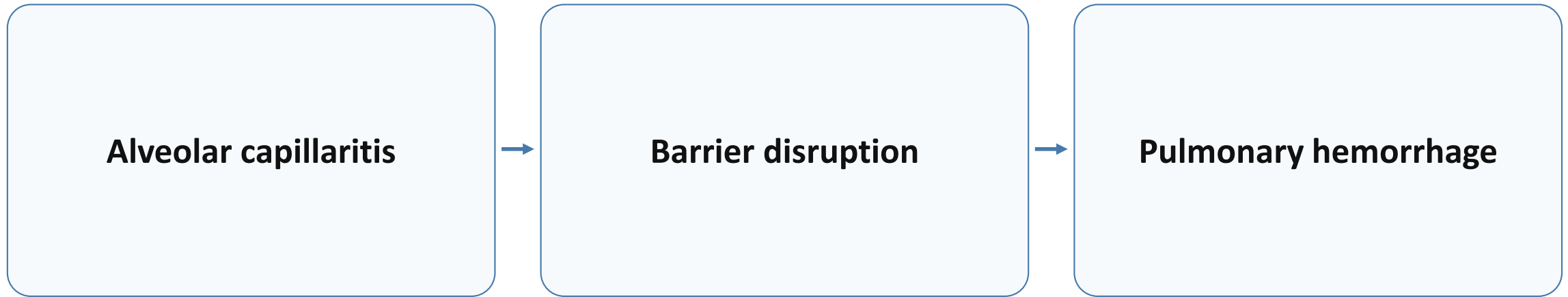
SCHEMATIC

Necrotizing glomerulonephritis is very common' description.

Capillary damage disrupts the glomerular filtration barrier.

Hematuria and proteinuria are resulting clinical manifestations.

MPA pulmonary capillaritis



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Alveolar capillaries are vulnerable to necrotizing inflammation.

Damage to capillary walls can produce alveolar hemorrhage.

Hemoptysis signals clinically significant pulmonary involvement.

The pulmonary–renal MPA pattern

Lung

Capillary inflammatory injury

Hemoptysis

Kidney

Necrotizing capillary injury

Hematuria

Simultaneous glomerular and pulmonary capillary injury provides a unifying mechanism.

Respiratory hemorrhage plus urinary blood/protein should raise concern for systemic small-vessel injury.

The pattern is not uniquely diagnostic of MPA; GPA and anti-GBM disease may also affect these organs.

MPA can follow environmental or drug triggers

The source cites hydralazine as a potential drug trigger.

Certain infections, including *Staphylococcus aureus*, are also discussed as possible triggers.

Trigger recognition must not replace tissue and clinical assessment.

MPA versus polyarteritis nodosa

Feature	MPA	PAN
Main vessels	Capillaries, venules, arterioles	Small/medium arteries
Glomerular inflammation	Characteristic	Absent by classification
Pulmonary capillaritis	May occur	Not typical
Lesion age	Often similar	May be mixed

MPA clinical findings follow organ injury

Skin

Postcapillary small vessels

Palpable purpura

Viscera

Renal / lung / bowel beds

Organ-specific symptoms

Palpable purpura reflects cutaneous small-vessel injury.

Hematuria/proteinuria, hemoptysis, abdominal pain/bleeding and myalgias may occur.

Widespread renal or CNS involvement can produce major morbidity.

MPA treatment response reflects immune activity

Durable remission is possible with immunosuppression in many patients.

Recognition and removal of a suspected inciting exposure is important.

Extent of renal or neurologic injury strongly influences clinical seriousness.

GPA is defined by a triad of lesions

Airways

Necrotizing granulomas

Upper or lower tract

Kidney

Necrotizing glomeruli

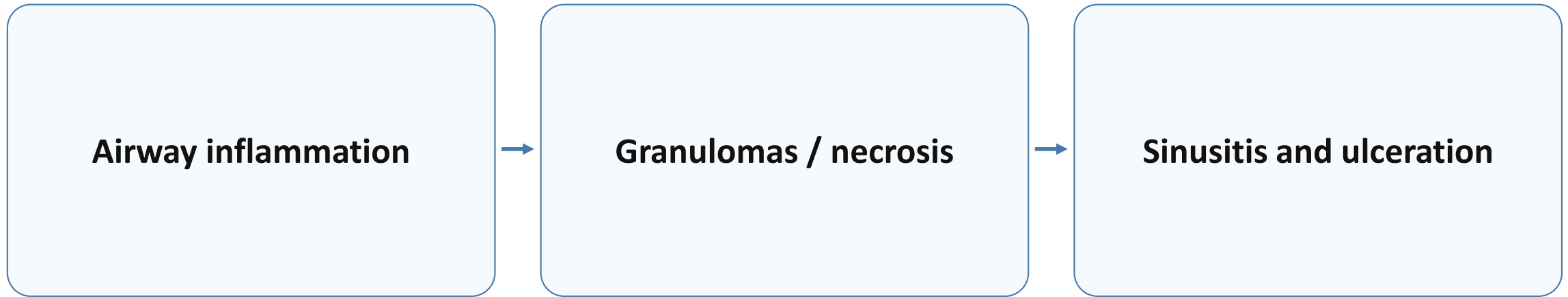
Crescents may form

Necrotizing granulomatous inflammation of upper and/or lower respiratory tract.

Necrotizing or granulomatous vasculitis of small to medium-sized vessels.

Focal necrotizing, often crescentic, glomerulonephritis.

Upper-airway GPA: Granulomatous inflammation



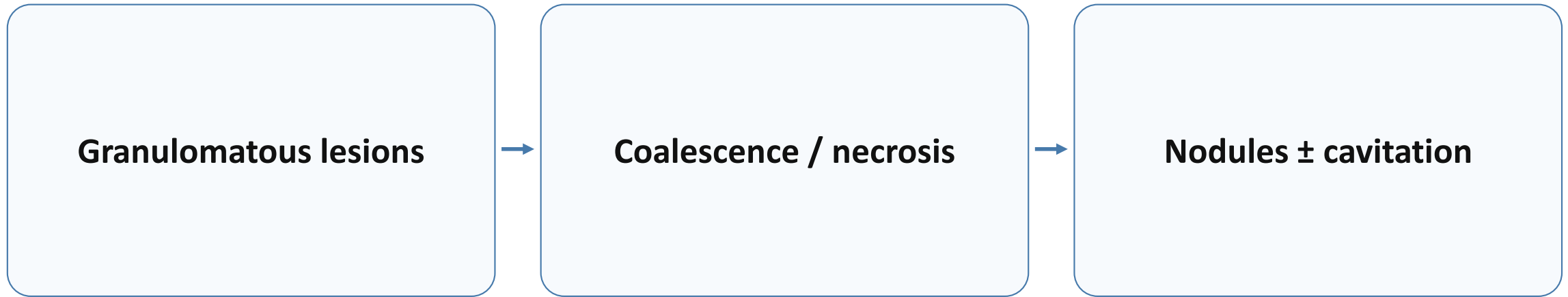
SCHEMATIC

Granulomatous sinusitis is a classic upper-tract lesion.

Nasal, palatal or pharyngeal ulceration may result from tissue destruction.

Respiratory tract disease can precede or occur without generalized organ injury.

Pulmonary GPA can form nodules and cavities



SCHEMATIC

Lung lesions range from diffuse infiltrates to granulomatous nodules.

Coalescing necrotizing granulomas may produce cavitating masses.

Alveolar hemorrhage may coexist with parenchymal destructive inflammation.

GPA granulomas are a major discriminator

GPA

Necrotizing granulomatous inflammation

MPA

Necrotizing vasculitis without granulomas

Epithelioid histiocytes and giant cells may occur adjacent to inflamed vessels.

Destructive necrotizing granulomatous parenchymal inflammation can be extensive.

Granulomatous lesions distinguish GPA from classic MPA.

GPA vessel lesions involve adjacent parenchyma



SCHEMATIC

Necrotizing inflammation can involve vessel walls and surrounding tissue.

A fibroblastic response may accompany granulomatous lesions.

Healing may progress toward organization and fibrosis.

GPA renal pathology can progress to crescents



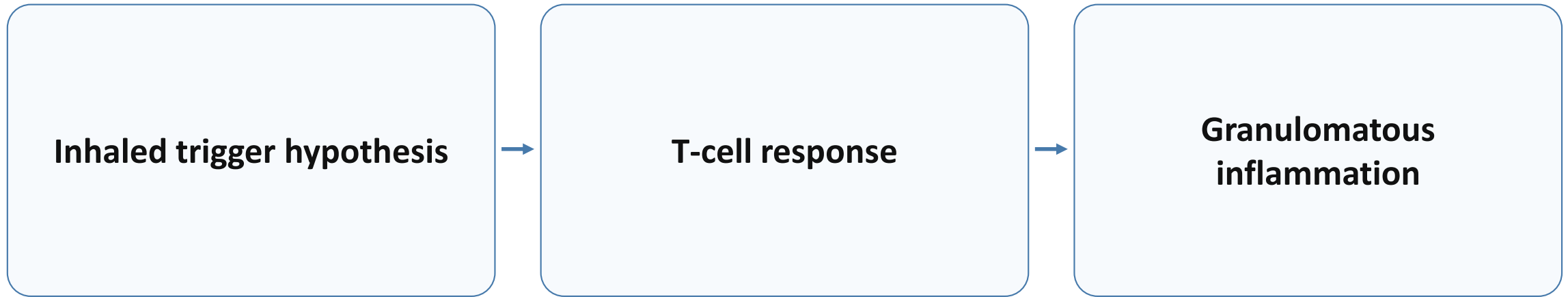
SCHEMATIC

Early renal lesions can show focal, segmental glomerular capillary necrosis.

More extensive injury may provoke parietal epithelial-cell proliferation.

The resulting crescentic glomerulonephritis marks severe glomerular injury.

GPA pathogenesis involves cellular immunity



SCHEMATIC

An abnormal T-cell-mediated response to inhaled environmental or microbial antigens.

Granuloma formation is consistent with a cellular inflammatory component.

PR3-ANCA-mediated neutrophil activation provides an additional pathogenic pathway.

PR3-ANCA is particularly informative in GPA

Strongly associated with GPA.

Antibody measurements may contribute to monitoring but must be interpreted alongside clinical findings.

The respiratory granuloma / renal lesion combination remains the defining clinicopathologic clue.

GPA ranges from limited to systemic disease

Localized phenotype

Upper/lower respiratory injury predominates

Systemic phenotype

Respiratory + kidney / other organs

Limited disease may remain restricted to the respiratory tract.

Disseminated disease may involve eyes, skin, cardiac tissue and other organs.

Renal disease is an important contributor to systemic severity.

GPA clinical recognition is multisystem

Upper airway

Chronic sinusitis / ulcers

Granulomatous injury

Lower airway

Nodules / cavitation

Necrotizing lesions

Chronic sinusitis or destructive upper-airway disease is a major clue.

Pulmonary infiltrates, nodules or cavities add a lower-airway clue.

Urinary abnormalities suggest associated necrotizing glomerulonephritis.

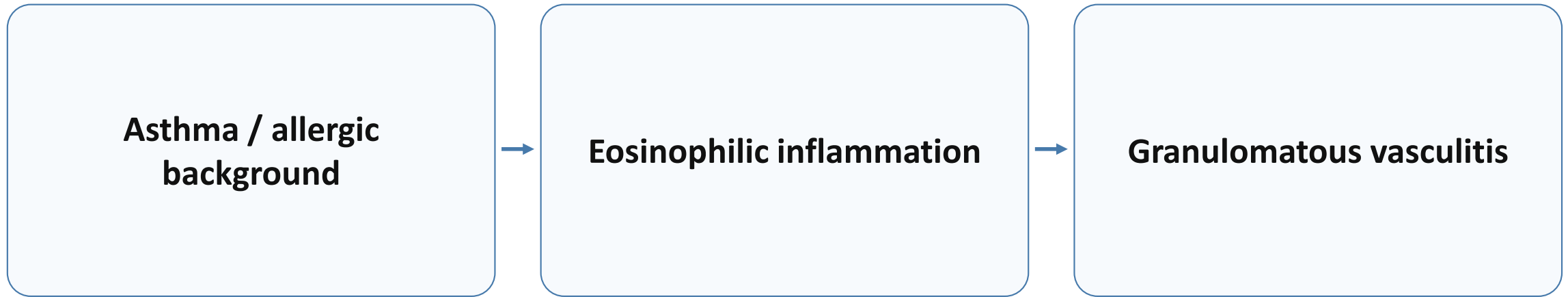
Untreated GPA is dangerous but treatable

High mortality for untreated classical systemic GPA.

Immunosuppression has transformed disease into a relapsing-remitting course for many patients.

Residual fibrosis and organ damage may persist despite control of activity.

EGPA links eosinophilia, allergy and vasculitis



SCHEMATIC

Classically associated with asthma and allergic rhinitis.

Peripheral eosinophilia and eosinophilic tissue infiltrates are characteristic.

Necrotizing granulomatous inflammation can affect small to medium-sized vessels.

Eosinophil-rich granulomas distinguish EGPA

EGPA

Eosinophil-rich lesions + granulomas

MPA

Necrotizing vessels; Granulomas absent

Extravascular necrotizing granulomas are described in EGPA.

Eosinophils infiltrate blood vessels and other tissues.

Both eosinophils and granulomas help distinguish EGPA from MPA.

EGPA respiratory phenotype

Upper respiratory

Allergic rhinitis

Allergic inflammatory setting

Lower respiratory

Asthma / infiltrates

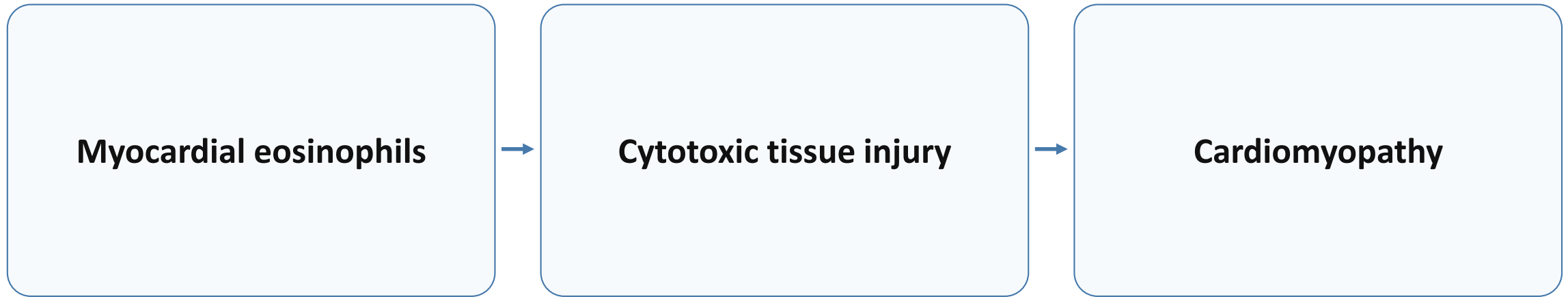
Eosinophilic tissue disease

Asthma is a central clinical association.

Allergic rhinitis and pulmonary infiltrates are common elements of the classical syndrome.

Extravascular granulomas can accompany respiratory inflammation.

EGPA cardiac involvement is consequential



SCHEMATIC

Eosinophilic myocardial infiltrates can damage cardiac myocytes.

Resultant cardiomyopathy is an important cause of morbidity and death.

Cardiac dysfunction in an eosinophilic vasculitic phenotype warrants particular attention.

EGPA can affect skin, gut and kidneys

Skin

Small-vessel involvement

Purpura

Other organs

Gastrointestinal / renal

Bleeding or renal signs

Palpable purpura reflects cutaneous vascular involvement.

Gastrointestinal bleeding is a recognized association.

Renal disease occurs, but the dominant clinical picture may be asthma/eosinophilic organ injury.

ANCA is not universal in EGPA

ANCA-positive subset

MPO reactivity often observed

ANCA-negative subset

Asthma + eosinophilic tissue disease remains relevant

Only a subset of EGPA patients has MPO-ANCA, unlike the stronger association of MPA with MPO-ANCA.

ANCA as more frequent with concurrent renal involvement.

ANCA negativity does not exclude the classical eosinophilic granulomatous phenotype.

MPA, GPA and EGPA: Morphologic contrasts

Finding	MPA	GPA	EGPA
Granulomas	Absent	Present	Present
Eosinophilic lesions	Not defining	Not defining	Characteristic
Respiratory pattern	Capillaritis	Necrotizing nodules / airways	Asthma / infiltrates
Typical ANCA	MPO	PR3	MPO in subset
Renal injury	Frequent GN	Necrotizing/crescentic GN	Variable

Behçet syndrome is a variable-vessel disorder

Small-vessel classification

Defined chiefly by vessel caliber and deposit pattern

Behçet syndrome

Variable-vessel inflammatory syndrome

Behçet syndrome adjacent to the small-vessel vasculitides.

It can involve both veins and arteries rather than a single vessel-caliber compartment.

The defining recognition pattern is multisystem inflammatory disease.

Behçet mucosal disease offers a clinical clue

Oral mucosa

Transient ulcers

Common recurrent feature

Genital / skin

Ulcers and variable skin lesions

Multisystem disease

Recurrent, self-healing oral ulcers occur in almost all cases.

Genital ulcers can be larger and persist longer.

Skin and articular involvement are also frequently seen.

Behçet vascular and neurologic complications

Eye / CNS

Uveitis; Brainstem inflammation

Vision / neurologic injury

Vascular system

Venous thrombosis; Aneurysms

Thrombotic / aneurysmal complications

Ocular involvement may include uveitis or hypopyon.

Venous thrombosis, arterial aneurysms and retinal vascular inflammation are described.

Central nervous system lesions, particularly in the brainstem, can cause persistent injury.

Behçet disease: Immune hypothesis and tissue



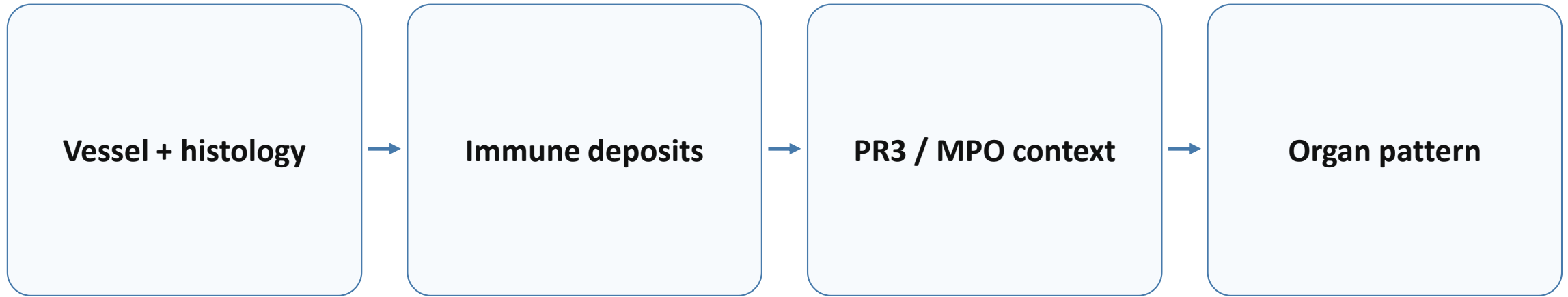
SCHEMATIC

The initiating cause is not established account.

Genetic susceptibility involving Th17 response regulation and environmental triggers are proposed.

Some skin biopsies show leukocytoclastic inflammation with mixed inflammatory cells.

Start diagnosis with the vessel injury pattern



SCHEMATIC

Identify the involved compartment: Venules/capillaries versus muscular arteries.

Characterize necrosis, neutrophil fragmentation, granulomas and eosinophils.

Then integrate tissue immune deposits, serum ANCA specificity and organ phenotype.

Immune deposits change the interpretation

Immune-positive lesion

Consider IgA, cryoglobulins, SLE,
drug/infection, anti-GBM

Pauci-immune lesion

Consider MPA, GPA or EGPA phenotype

Abundant immune material favors immune-complex or direct antibody-binding mechanisms.

Pauci-immune necrotizing injury is characteristic of ANCA-associated syndromes.

No single staining result replaces an integrated clinicopathologic diagnosis.

Clinical phenotype guides targeted tissue review

Presentation clue	Pathology question
Cavitating respiratory nodules	Is there necrotizing granulomatous vasculitis?
Asthma + eosinophilia	Are eosinophil-rich granulomas present?
Pulmonary–renal injury	Is necrotizing capillaritis / GN present?
Purpura with deposits	What immune material is deposited?

Case: Hemoptysis, hematuria and purpura

An adult develops hemoptysis and urinary blood with palpable purpura.

A vessel biopsy shows segmental necrosis and neutrophilic fragments.

No granulomas are seen; Tissue immune deposits are minimal.

Question: Which small-vessel syndrome best fits the combination?

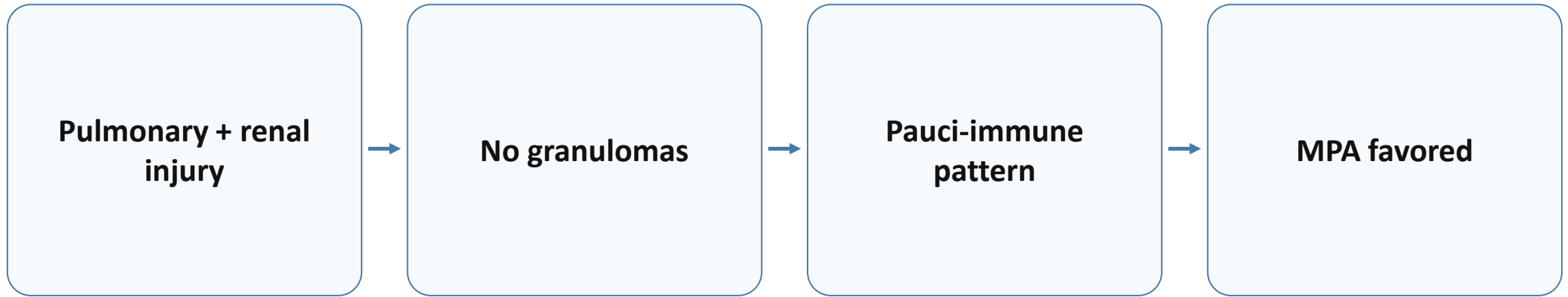
TRACE THE CLUES

Lung capillaritis

Renal capillary injury

Pauci-immune pattern

Case interpretation: Microscopic polyangiitis



SCHEMATIC

The pulmonary–renal syndrome is compatible with MPA.

Absence of granulomas separates this pattern from classical GPA.

MPO-ANCA supports the pattern, but renal/lung evaluation remains decisive.

Case: Chronic sinusitis and cavitating nodules

Persistent sinonasal disease is followed by cavitating pulmonary nodules.

Tissue shows necrotizing granulomatous inflammation involving small vessels.

Urinary blood raises concern for associated glomerular disease.

Question: Identify the unifying syndrome and its most associated ANCA.

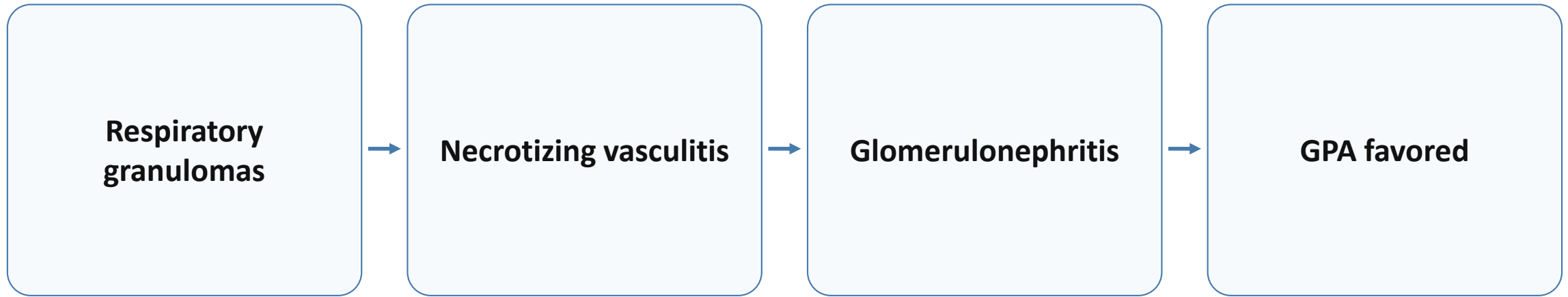
TRACE THE CLUES

Upper airway

Lower airway

Kidney

Case interpretation: GPA and PR3-ANCA



SCHEMATIC

Respiratory necrotizing granulomas and small-vessel vasculitis favor GPA.

Focal necrotizing or crescentic glomerulonephritis completes the classic triad.

PR3-ANCA is the characteristic antigen association.

Case: Asthma with eosinophilic organ injury

An individual with asthma and allergic rhinitis develops peripheral eosinophilia.

Tissue shows eosinophil-rich inflammation and necrotizing granulomas.

New cardiac dysfunction raises concern for eosinophilic myocardial injury.

Question: Which vasculitis unifies these observations?

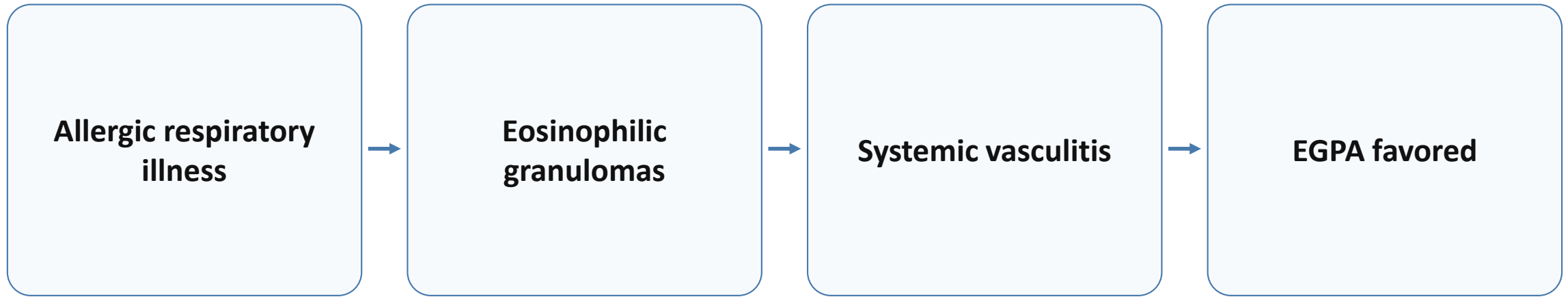
TRACE THE CLUES

Asthma

Eosinophilia / granulomas

Cardiac injury

Case interpretation: EGPA



SCHEMATIC

Asthma and eosinophil-rich granulomatous vasculitis strongly support EGPA.

Myocardial infiltration explains possible cardiomyopathy.

MPO-ANCA may be present but is not required for recognition of the phenotype.

Case: Palpable purpura after a new exposure

Palpable purpura appears after a newly introduced medication.

Small-vessel inflammation shows appreciable immunoglobulin/complement deposits.

Question: Is a deposited immune-complex pathway or pauci-immune ANCA mechanism more consistent?

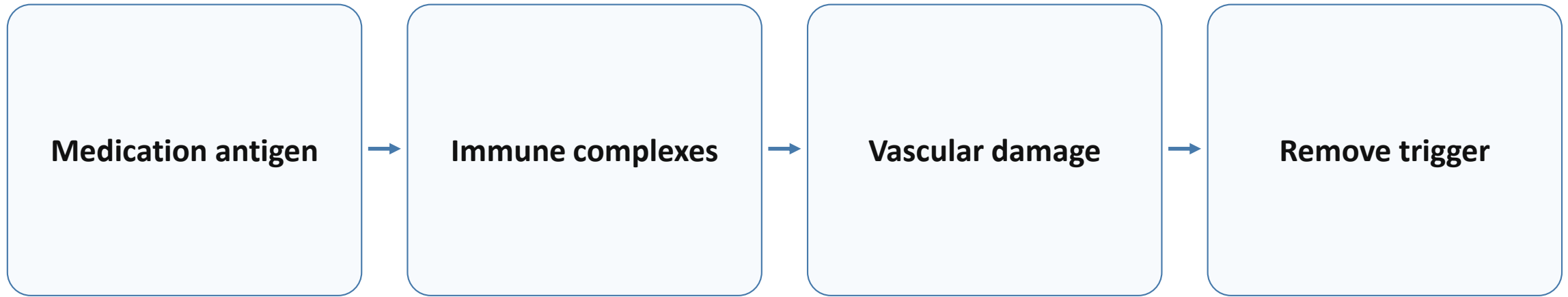
TRACE THE CLUES

Drug exposure

Vascular immune deposits

**Immune-deposit
mechanism**

Case interpretation: Drug-linked immune complexes



SCHEMATIC

A drug-related immune-complex process fits the deposited immune material.

Consider a drug hapten or other antigenic exposure as.

Identifying and discontinuing the culprit medication can permit resolution.

Mechanisms and morphology: Final synthesis

Immune-deposit disease involves vessel-wall antibody/complement accumulation and leukocyte activation.

ANCA disease causes necrotizing injury through leukocyte activation with few tissue deposits.

Granulomas, eosinophils, glomerular lesions and respiratory phenotype distinguish the ANCA syndromes.

Always distinguish immune injury from direct infection and interpret results in organ context.

Primary source and scope

Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).

Blood Vessels: Vasculitis and Small Vessel Vasculitis.

The discussion of immune complexes and ANCAs is developed within the same textbook section.

Related mechanisms: immune-mediated injury and glomerular basement membrane autoantibodies.