

Onchocerciasis

Pathogenesis, morphology, and clinicopathologic consequences

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Onchocerciasis: A tissue-centered infection

- *Onchocerca volvulus* is a filarial nematode.
- Infection has a characteristic subcutaneous nodule and cutaneous inflammatory disease.
- Microfilariae can injure the cornea and intraocular tissues, causing loss of vision.

Core distinction

Adult worms and microfilariae occupy different pathological roles.

Learning outcomes

- Identify the parasite, vector, and river-associated exposure setting.
- Explain why microfilarial inflammation dominates tissue damage.
- Recognize onchocercomas, chronic skin changes, and progressive eye lesions.
- Relate ivermectin activity and treatment-associated inflammation to parasite stage.

Emphasis: Mechanism → morphology → organ damage.

The etiologic organism

- The disease is attributed to *Onchocerca volvulus*.
- *O. Volvulus* belongs to the filarial nematodes.
- Adult parasites and their microfilariae produce distinct patterns of tissue involvement.

Core distinction

Name the species, then
locate its adult and
immature forms.

Filarial context: Two sites of disease

Lymphatic filariasis

Adult parasites in lymphatic channels

Lymphedema / elephantiasis

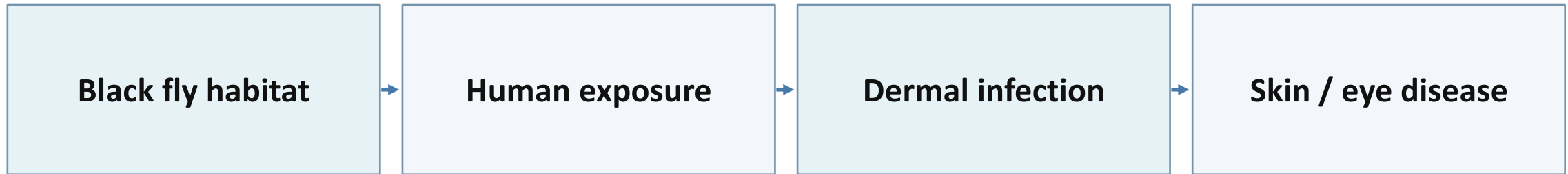
Onchocerciasis

Adult parasites in dermis

Dermatitis / ocular injury

Avoid transferring elephantiasis mechanisms directly to onchocerciasis.

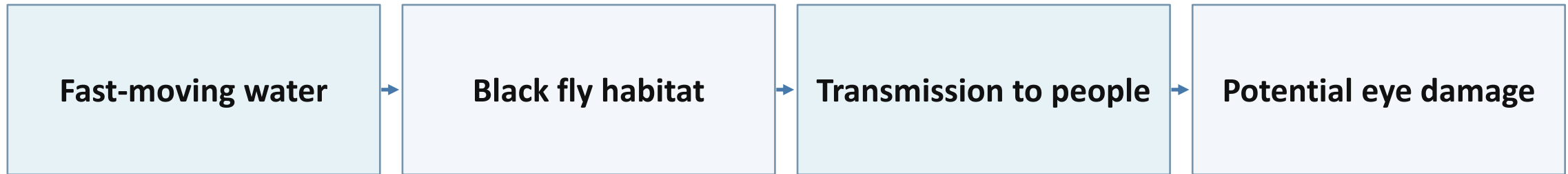
Black flies transmit the parasite



- The source identifies black flies as the vectors.
- Reported transmission locations include Africa, South America, and Yemen.
- The local vector habitat is central to geographic risk.

Vector ecology helps explain the disease distribution.

Why “river blindness”?



- Black flies have habitats near fast-moving water.
- Human incidence is therefore higher near rivers.
- The name links transmission ecology to a severe ocular outcome.

River proximity predicts exposure context—not the anatomical site of injury.

The distribution

- Africa, South America, and Yemen as transmission settings.
- The description emphasizes blindness in sub-Saharan Africa.
- These are textbook statements, not a current surveillance map.

Core distinction

Distribution statements should be dated to the edition being taught.

Elimination is location-specific

- The text reports WHO-declared elimination in Colombia, Ecuador, Mexico, and Guatemala.
- Elimination in named countries does not imply elimination everywhere.
- Historical program achievements must be separated from present-day surveillance.

Core distinction

Textbook claim; No independent current-status verification performed.

Mass ivermectin campaigns

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graph LR; A[Community treatment] --> B[Immature-stage killing]; B --> C[Lower disease burden]; C --> D[Need for repeat dosing];
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Community treatment → **Immature-stage killing** → **Lower disease burden** → **Need for repeat dosing**

- A large decline in incidence to mass ivermectin administration.
- Campaign impact is described for Africa and South America.
- Continued treatment is conceptually linked to persistence of adult parasites.

Public-health effect and individual parasitologic clearance are different endpoints.

A compartmental view of infection

Dermis

Adult worms mate

Subcutaneous onchocercoma

Skin

Microfilariae accumulate

Chronic dermatitis

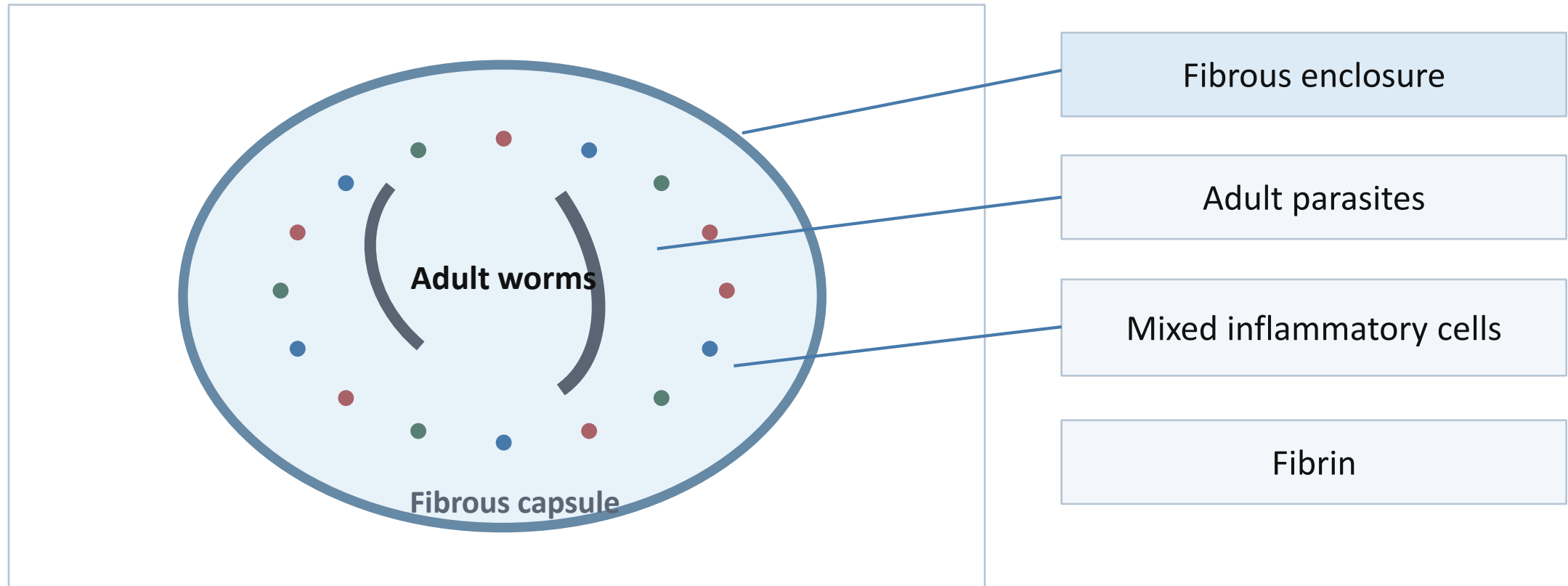
Eye

Microfilariae disseminate

Keratitis / inflammation

- Dermis: Adult worms mate and persist.
- Skin: Offspring microfilariae accumulate.

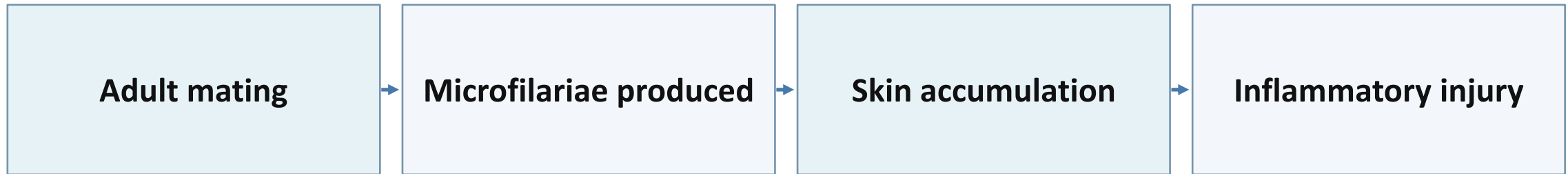
Adult worms establish the dermal focus



Simplified nodule schematic — not a histology image

- Adult male and female parasites mate in the dermis.
- Host cells accumulate around these adult worms.

Reproduction creates a mobile disease stage



- Inseminated adult females produce microfilariae.
- Microfilariae collect in skin rather than remaining confined to the adult nodule.
- Their distribution explains lesions beyond the original nodule.

Adult persistence can seed ongoing microfilarial exposure.

The eye is an additional target

Cornea

Punctate → sclerosing keratitis

Anterior chamber / ciliary body

Iridocyclitis ± glaucoma

Choroid and retina

Atrophy → loss of vision

Linking factor

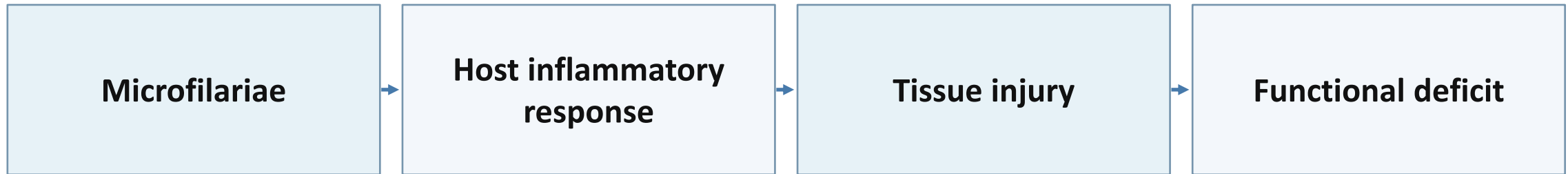
Microfilarial inflammation



Anatomical site map; Not a scaled anatomical drawing

- Microfilariae disseminating into the eye chambers.
- Ocular inflammation can affect the cornea, anterior chamber, and posterior structures.

The core pathogenic principle



- Clinical disease is primarily attributed to inflammation induced by microfilariae.
- Immature parasite degeneration is prominent in corneal inflammatory injury.
- Host inflammatory responses are central to the destructive phenotype.

Organ damage is not simply the physical presence of worms.

Adults versus microfilariae

Adult worms

Predominantly dermal nodules

Fibrous capsule + mixed cells

Source of progeny

Microfilariae

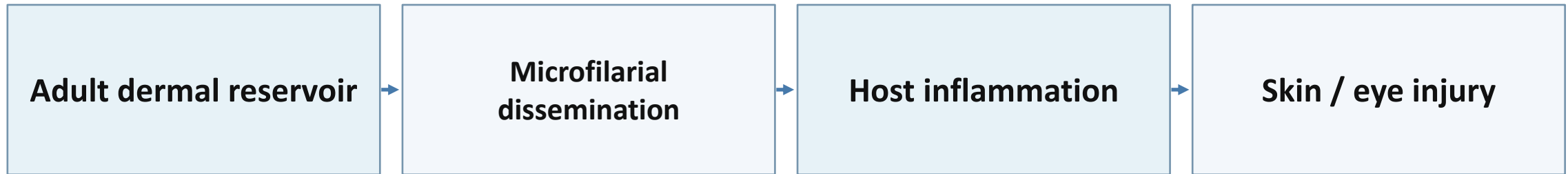
Distributed through skin

Diffuse dermatitis and eye inflammation

Key inflammatory effector stage

Different parasite stages explain distinct pathology patterns.

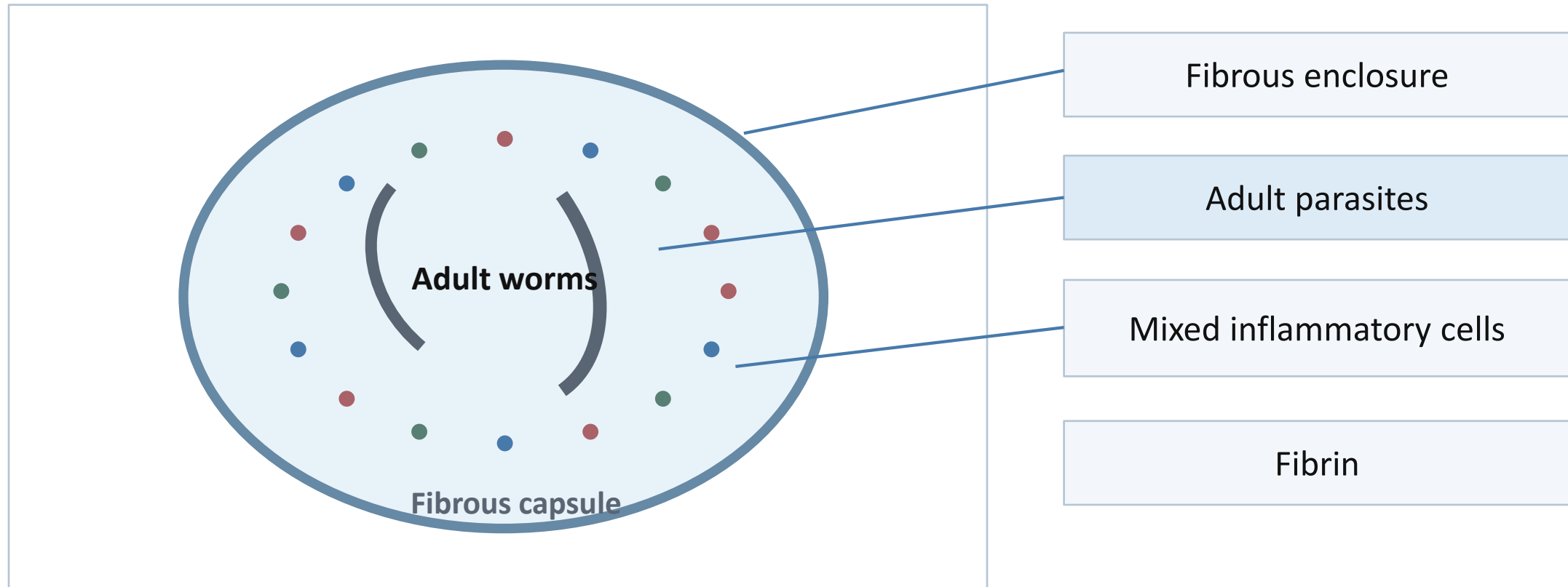
A three-part mechanistic model



- Parasite reservoir: Reproductive adults in a dermal nodule.
- Dissemination: Microfilariae reach skin and eye.
- Host response: Inflammation changes skin architecture and ocular transparency.

Reservoir → dissemination → inflammatory damage.

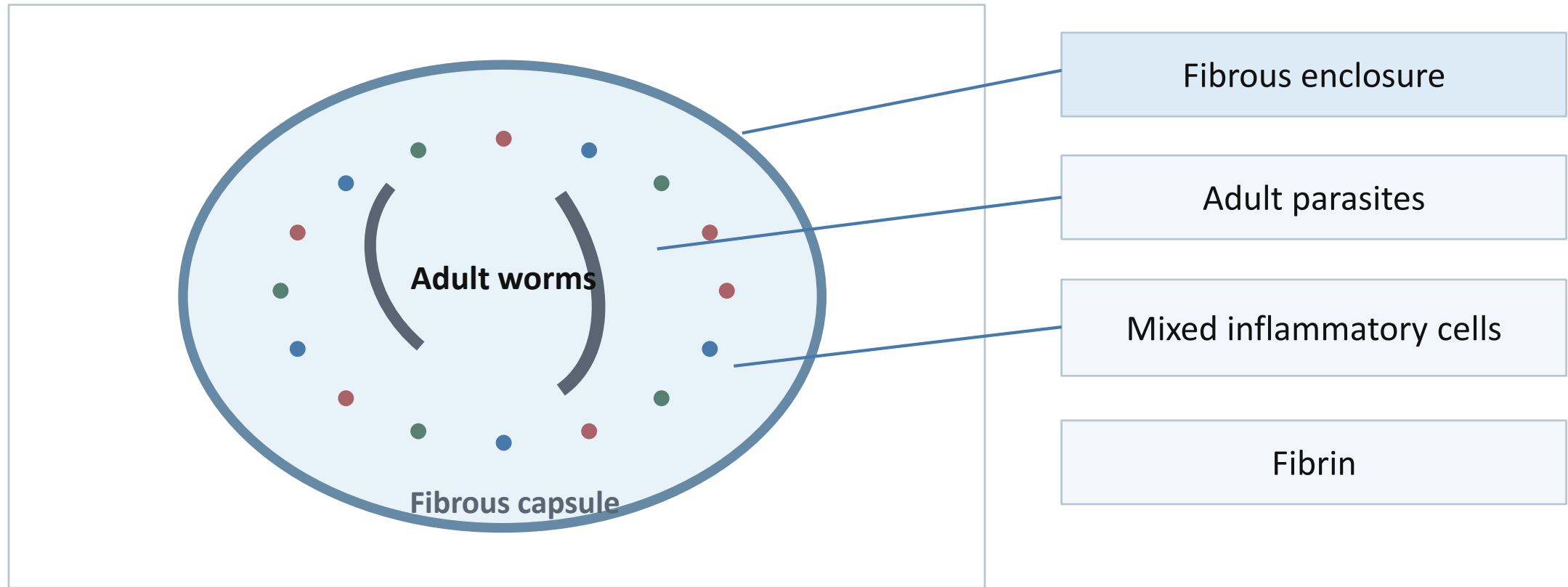
Onchocercoma: The signature nodule



Simplified nodule schematic — not a histology image

- An onchocercoma is a subcutaneous nodule surrounding adult parasites.
- It contains a fibrous capsule and inflammatory cells.

Onchocerca: Capsule architecture



Simplified nodule schematic — not a histology image

- A fibrous capsule encircling the adult worms.
- A capsule separates the worm-containing focus from adjacent tissue.

The inflammatory-cell mixture

Fibrin

Neutrophils

Eosinophils

Lymphocytes

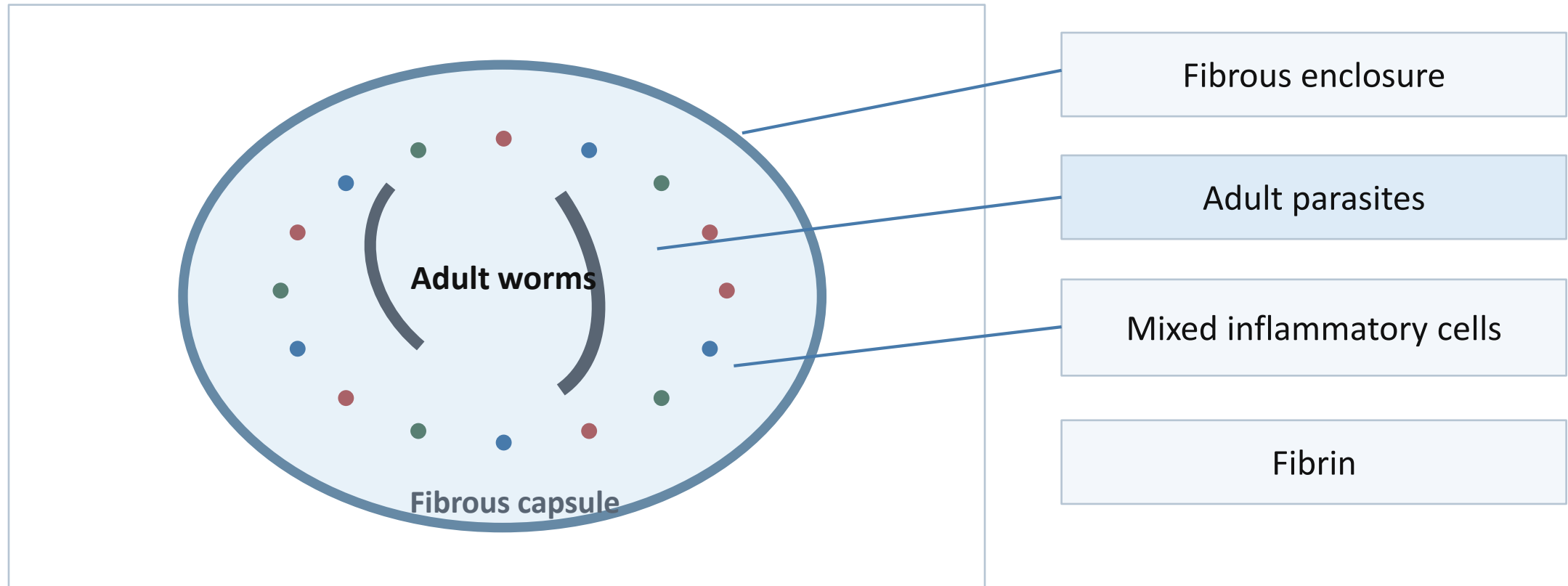
Giant cells

Adult worms

Elements listed for the onchocercoma

- The nodule contains neutrophils, eosinophils, and lymphocytes.
- Giant cells are part of the host response.

Histologic identification of an onchocercoma



Simplified nodule schematic — not a histology image

- Find the encapsulated adult-worm compartment.
- Recognize its mixed inflammatory background.

Why nodules do not explain all injury

Focal process

Adult worm in capsule

Localized subcutaneous nodule

Distributed process

Microfilariae beyond nodule

Dermatitis and ocular lesions

Distinguish focal structural lesions from distributed inflammatory lesions.

Cutaneous disease: The clinical pattern

Epidermal surface

Focus: Morphologic pattern

Epidermis: Focal atrophy ↔ hyperkeratosis

Epidermis + dermis

Dermis: Elastic-fiber injury, pigment incontinence, fibrosis

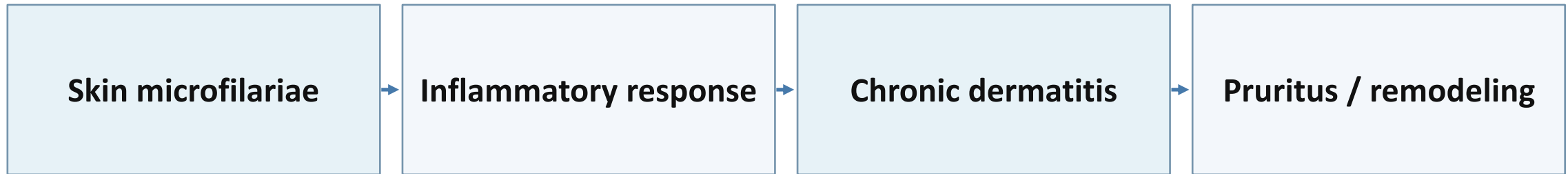
Schematic tissue layers



Schematic: Relative layer positions, not diagnostic microscopy

- A chronic, itchy dermatitis.
- Pigment may become focally darker or reduced.

Pruritus reflects an inflammatory skin disease



- Itch is included in the primary cutaneous morphology.
- The source places dermatitis downstream of microfilarial inflammation.
- Persistent irritation can coexist with longer-term tissue remodeling.

Pruritus and architectural changes belong to one pathological process.

Cutaneous pigment may increase or decrease

Epidermal surface

Focus: Pigment change

Epidermis: Focal atrophy ↔ hyperkeratosis

Epidermis + dermis

Dermis: Elastic-fiber injury, pigment incontinence, fibrosis

Schematic tissue layers



Schematic: Relative layer positions, not diagnostic microscopy

- Both focal darkening and loss of pigmentation are reported.
- The patterns may coexist with scale.

“Leopard,” “lizard,” and “elephant” skin

- These terms for chronic onchocercal skin patterns.
- They refer to visible alterations in pigmentation, scale, and surface architecture.
- The labels describe appearance; They are not separate etiologic entities.

Core distinction

Use descriptive morphology rather than assuming distinct pathogens.

Epidermal atrophy: Tissue loss

Epidermal surface

Focus: Tissue atrophy

Epidermis: Focal atrophy ↔ hyperkeratosis

Epidermis + dermis

Dermis: Elastic-fiber injury, pigment incontinence, fibrosis

Schematic tissue layers



Schematic: Relative layer positions, not diagnostic microscopy

- Areas of epidermal atrophy are described.
- The source also reports breakdown of elastic fibers.

Elastic-fiber breakdown

Epidermal surface

Epidermis: Focal atrophy ↔ hyperkeratosis

Dermis: Elastic-fiber injury, pigment incontinence, fibrosis



Focus: Elastic fiber injury

Dermal matrix

Schematic tissue layers

Schematic: Relative layer positions, not diagnostic microscopy

- Degeneration of elastic fibers is specifically included in the skin lesion.
- The dermal matrix is therefore involved, not just the epidermal surface.

Hyperkeratosis in chronic dermatitis

Epidermal surface

Focus: Hyperkeratosis

Epidermis: Focal atrophy ↔ hyperkeratosis

Epidermal keratin

Dermis: Elastic-fiber injury, pigment incontinence, fibrosis

Schematic tissue layers



Schematic: Relative layer positions, not diagnostic microscopy

- Some skin regions become hyperkeratotic.
- Hyperkeratosis may coexist with hyperpigmentation.

Pigment incontinence

Epidermal surface

Focus: Pigment change

Epidermis: Focal atrophy ↔ hyperkeratosis

Epidermis + dermis

Dermis: Elastic-fiber injury, pigment incontinence, fibrosis

Schematic tissue layers



Schematic: Relative layer positions, not diagnostic microscopy

- Dermal pigment incontinence.
- This is distinct from a simple visual description of darker skin.

Dermal atrophy and fibrosis

Epidermal surface

Focus: Tissue atrophy

Epidermis: Focal atrophy ↔ hyperkeratosis

Epidermis + dermis

Dermis: Elastic-fiber injury, pigment incontinence, fibrosis

Schematic tissue layers



Schematic: Relative layer positions, not diagnostic microscopy

- The dermis can become atrophic and fibrotic.
- Loss of normal tissue and deposition of fibrous tissue may coexist.

The patchwork morphology of skin disease

Relatively atrophic foci

Epidermal atrophy

Elastic-fiber breakdown

Dermal atrophy

Thickened / pigmented foci

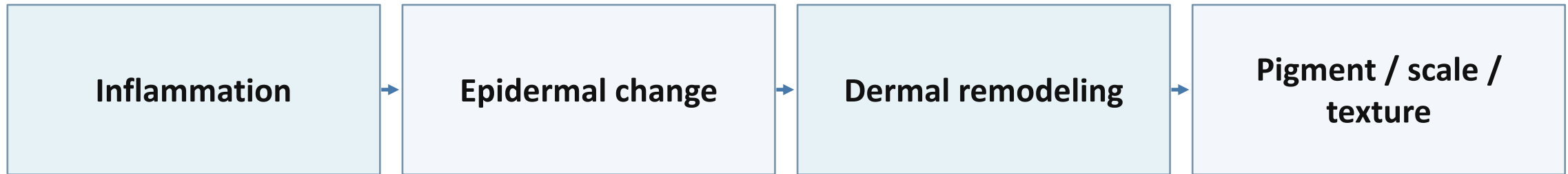
Hyperkeratosis

Hyperpigmentation

Pigment incontinence / fibrosis

Alternating patterns are an explicit finding.

Cutaneous structure–function reasoning



- Microfilarial inflammation produces chronic dermatitis.
- Ongoing injury is associated with epidermal and connective-tissue alterations.
- The clinical surface changes are the visible readout of underlying morphology.

Histology explains the visible skin phenotype.

Cutaneous recognition: What to describe

- Record pruritus and scaling as inflammatory symptoms/signs.
- Describe whether pigmentation is increased, decreased, or mixed.
- Distinguish atrophic from hyperkeratotic regions and note dermal fibrosis.

Core distinction

These are morphology descriptors, not a validated diagnostic algorithm.

Why the skin pattern is not one lesion

Subcutaneous lesion

Encapsulated adult worms

Mixed infiltrate

Focal nodule

Cutaneous diffuse lesion

Microfilaria-related inflammation

Pigment and keratin changes

Patchy tissue remodeling

Separate lesion type, anatomical site, and parasite stage.

Ocular injury: Multiple anatomical targets

Cornea

Punctate → sclerosing keratitis

Anterior chamber / ciliary body

Iridocyclitis ± glaucoma

Choroid and retina

Atrophy → loss of vision

Linking factor

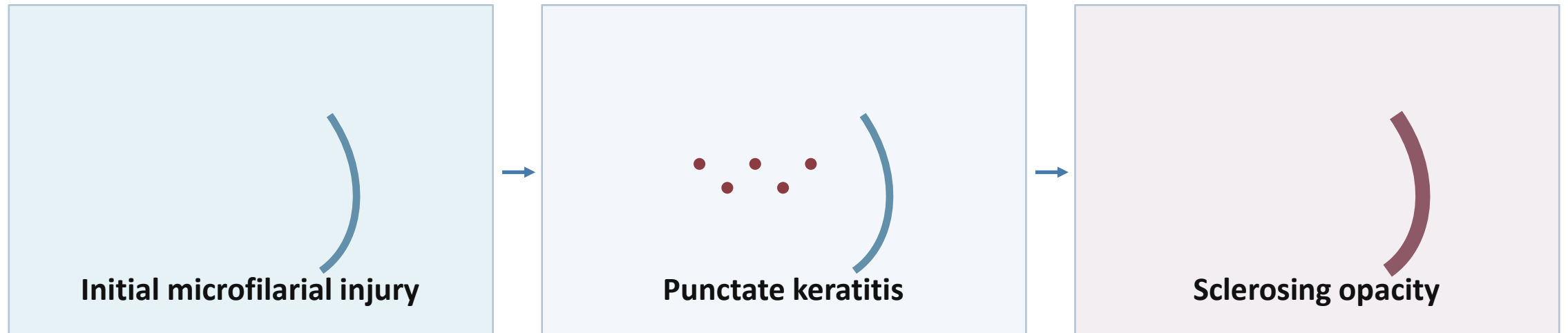
Microfilarial inflammation



Anatomical site map; Not a scaled anatomical drawing

- The cornea is affected by punctate and later sclerosing keratitis.
- Anterior chamber and ciliary body may develop iridocyclitis.

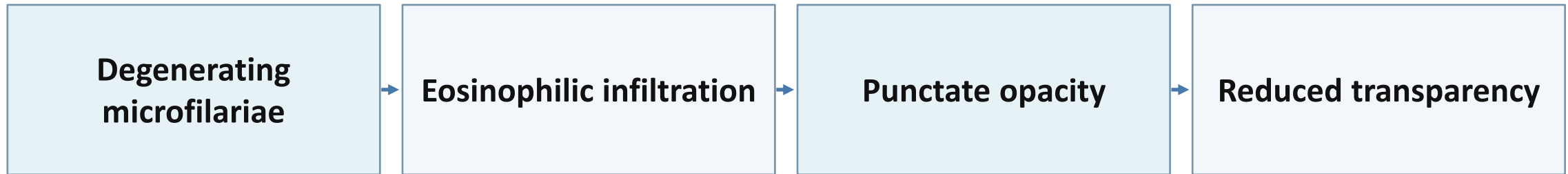
Early corneal change: Punctate keratitis



Punctate stage · Conceptual corneal schematic, not a clinical image

- The initial described corneal lesion is punctate keratitis.
- It appears as small, fluffy corneal opacities.

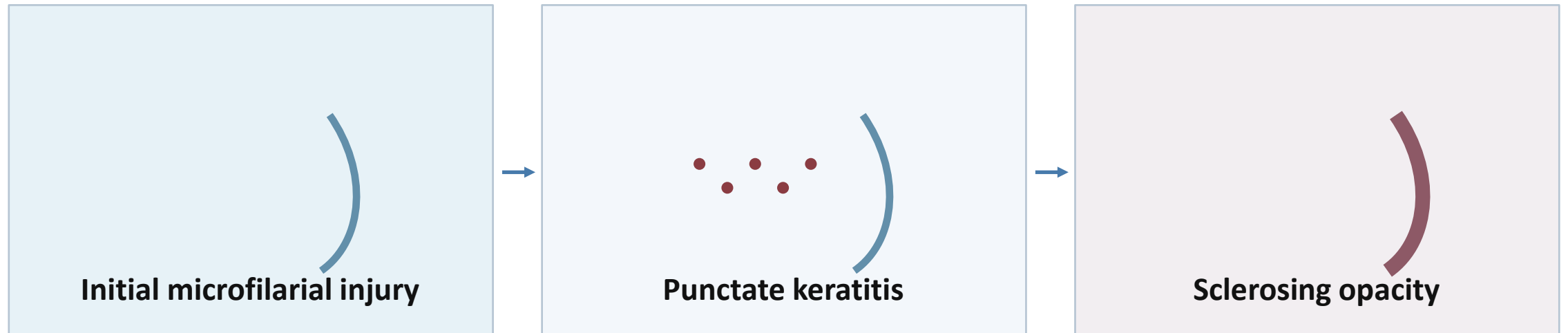
Why microfilariae cloud the cornea



- Degeneration of microfilariae occurs in the cornea.
- The degenerating parasites elicit an eosinophilic infiltrate.
- Inflammation accompanies the punctate, fluffy opacities.

Parasite degeneration → inflammation → loss of corneal clarity.

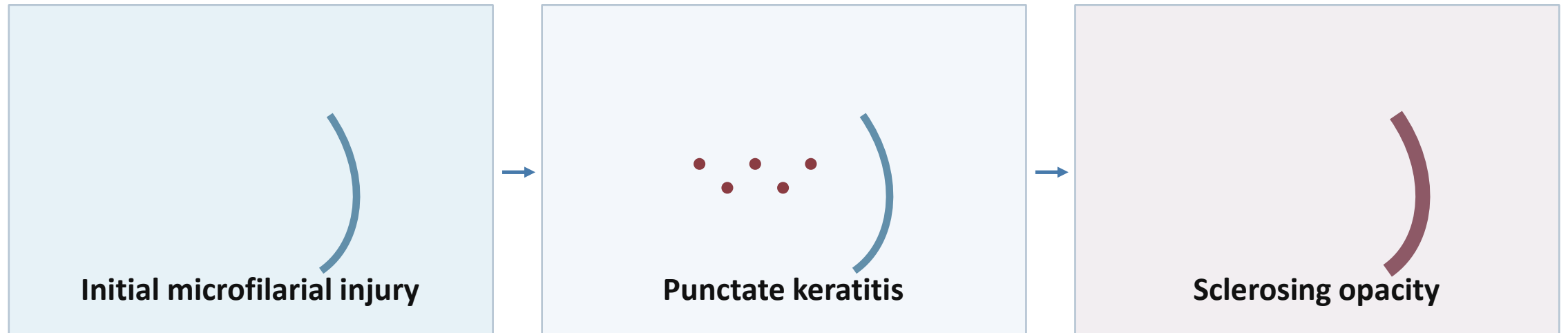
Eosinophils in early corneal disease



Eosinophilic inflammation · Conceptual corneal schematic, not a clinical image

- The infiltrate accompanying degenerating microfilariae includes eosinophils.
- This connects ocular pathology to host inflammatory response.

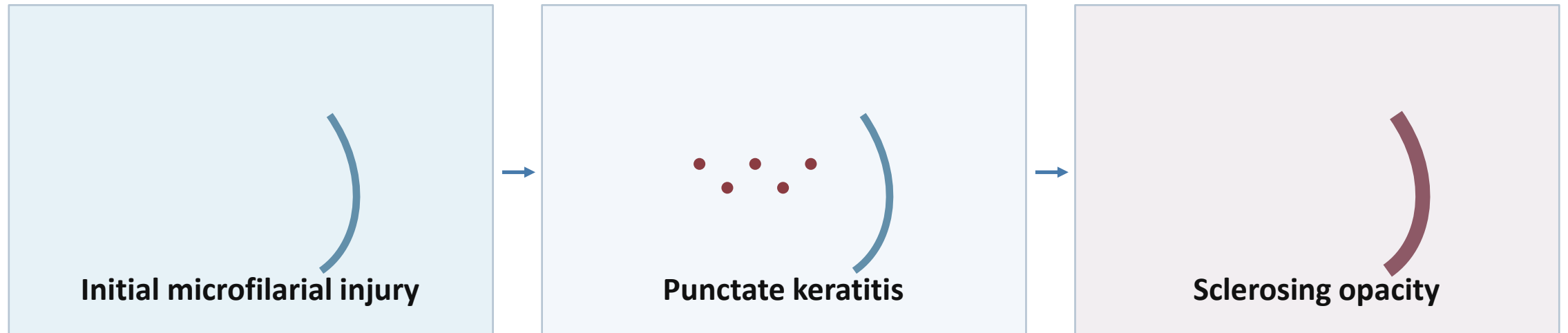
Corneal disease can become sclerosing



Sclerosing opacity · Conceptual corneal schematic, not a clinical image

- Progression from punctate to sclerosing keratitis.
- The later lesion opacifies the cornea.

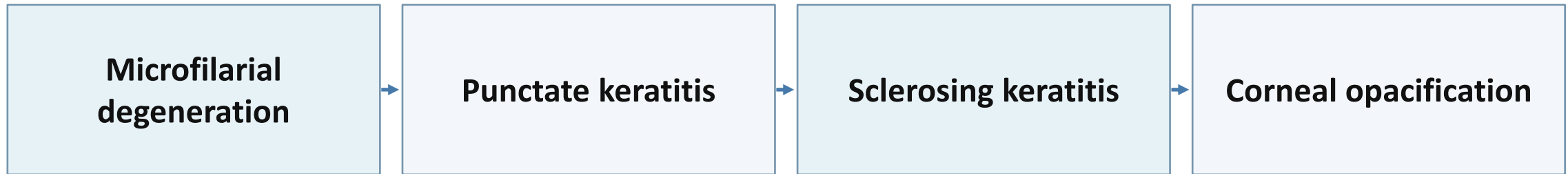
The limbal starting point



Limbal onset of sclerosis · Conceptual corneal schematic, not a clinical image

- Sclerosing corneal opacity begins at the scleral limbus.
- Its peripheral onset is an anatomical clue in the described sequence.

Corneal progression at a glance



- Degenerating microfilariae are associated with early punctate keratitis.
- Fluffy corneal opacities and eosinophils mark the inflammatory stage.
- Sclerosing keratitis creates progressively more opaque corneal tissue.

A morphologic continuum links early inflammation to advanced damage.

Anterior chamber involvement

Cornea

Punctate → sclerosing keratitis

Anterior chamber / ciliary body

Iridocyclitis ± glaucoma

Choroid and retina

Atrophy → loss of vision

Linking factor

Microfilarial inflammation



Anatomical site map; Not a scaled anatomical drawing

- Microfilariae may occupy the anterior chamber.
- Their presence is associated with anterior chamber inflammation.

Iridocyclitis: Anterior ocular inflammation

Cornea

Punctate → sclerosing keratitis

Anterior chamber / ciliary body

Iridocyclitis ± glaucoma

Choroid and retina

Atrophy → loss of vision

Linking factor

Microfilarial inflammation



Anatomical site map; Not a scaled anatomical drawing

- The source describes inflammation of the anterior chamber and ciliary body.
- It names this lesion iridocyclitis.

Glaucoma as a complication

Cornea

Punctate → sclerosing keratitis

Anterior chamber / ciliary body

Iridocyclitis ± glaucoma

Choroid and retina

Atrophy → loss of vision

Linking factor

Microfilarial inflammation



Anatomical site map; Not a scaled anatomical drawing

- Glaucoma is listed with anterior-chamber / iridocyclitic involvement.
- It is an ocular complication, not a synonym for keratitis.

Posterior eye involvement

Cornea

Punctate → sclerosing keratitis

Anterior chamber / ciliary body

Iridocyclitis ± glaucoma

Choroid and retina

Atrophy → loss of vision

Linking factor

Microfilarial inflammation



Anatomical site map; Not a scaled anatomical drawing

- Choroidal involvement is described.
- Retinal involvement is also described.

Atrophy in choroid and retina

Corneal disease

Punctate → sclerosing keratitis

Opacified optical tissue

Transparency compromised

Chorioretinal disease

Choroid / retina involvement

Posterior tissue atrophy

Loss of retinal function associated with tissue loss

Two major patterns: Loss of clarity versus tissue atrophy.

A site-based ocular damage map

Cornea

Punctate → sclerosing keratitis

Anterior chamber / ciliary body

Iridocyclitis ± glaucoma

Choroid and retina

Atrophy → loss of vision

Linking factor

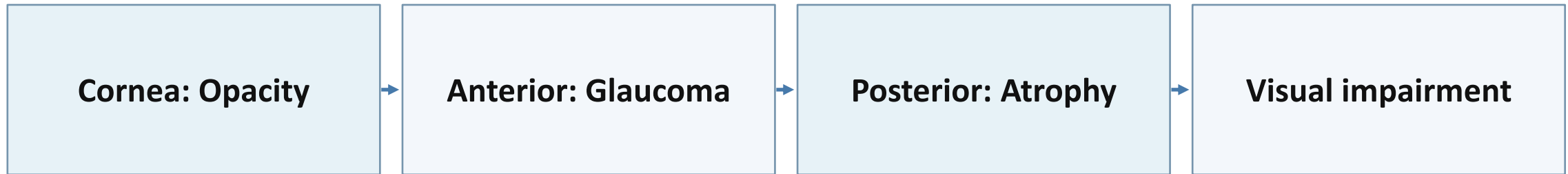
Microfilarial inflammation



Anatomical site map; Not a scaled anatomical drawing

- Cornea: Punctate keratitis and sclerosing opacity.
- Anterior segment: Iridocyclitis with glaucoma.

Vision loss has more than one mechanism



- Corneal damage changes optical transparency.
- Anterior intraocular inflammation is associated with glaucoma.
- Posterior chorioretinal atrophy is another route to loss of vision.

Separate the three lesion pathways when teaching blindness.

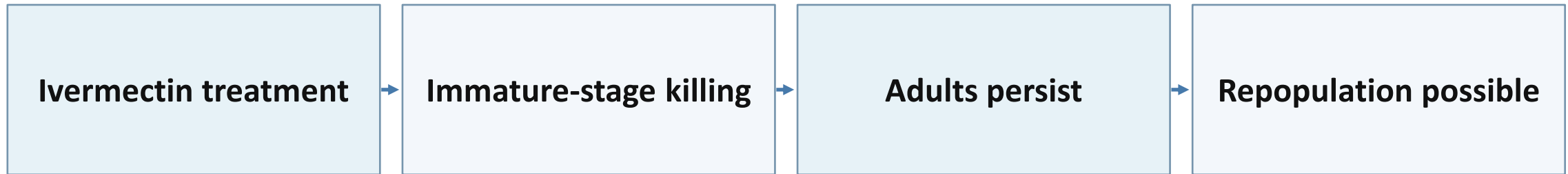
Mazzotti reaction: A treatment-linked change

- Keratitis may be accentuated by antifilarial treatment.
- This treatment-associated exacerbation is called the Mazzotti reaction.
- It underscores inflammation during parasite-directed therapy.

Core distinction

Anti-parasitic efficacy
does not mean
inflammation always
subsides immediately.

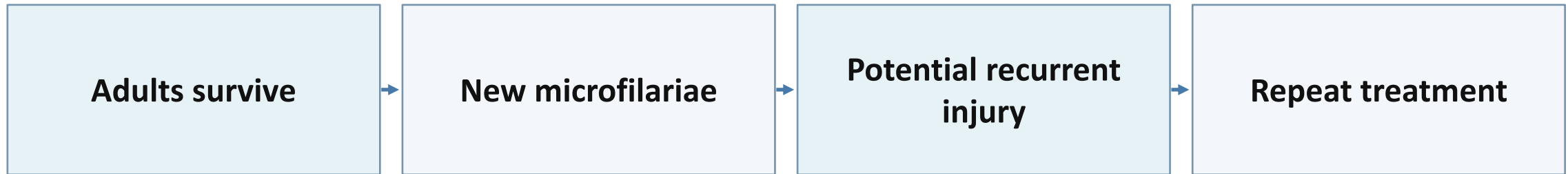
Ivermectin and developmental stage



- Ivermectin kills immature worms, not adults.
- It does not eradicate the adult reproductive reservoir described in the dermis.
- Surviving adult parasites can produce additional microfilariae.

This slide follows the textbook's stage-based wording.

Why repeat treatment is needed



- Adult parasites are not killed by ivermectin as.
- Microfilariae can recur while adults remain reproductively active.
- The text therefore describes retreatment every 3–6 months until symptoms resolve.

Regimen quoted; Not a current prescribing guideline.

Treatment: Parasite and host response

Parasitologic effect

Reduction in immature stages

Adults can persist

Retreatment may be needed

Inflammatory consequence

Keratitis may intensify

Mazzotti reaction is named

Ocular injury still requires recognition

Keep pathogen control separate from transient tissue inflammation.

Skin and eye: A unifying mechanism

Skin	Eye
Pruritus + scaling	Punctate keratitis
Pigment / keratin change	Sclerosing corneal opacity
Dermal atrophy / fibrosis	Iridocyclitis / chorioretinal atrophy

One etiologic process produces contrasting organ morphologies.

Onchocerciasis versus lymphatic filariasis

Lymphatic filariasis (parent text)	Onchocerciasis (topic text)
Adults occupy lymphatic channels	Adults mate in dermis
Chronic lymphedema / elephantiasis	Subcutaneous onchocercoma
Lymphatic remodeling	Dermatitis and eye damage

Do not interpret “elephant skin” as equivalent to lymphatic elephantiasis.

Morphology-based differential reasoning

- A subcutaneous fibrous nodule with adult worms favors onchocercoma.
- Chronic pruritic pigmentary dermatitis points to a broader cutaneous process.
- Corneal eosinophilic inflammation and chorioretinal atrophy indicate separate ocular compartments.

Core distinction

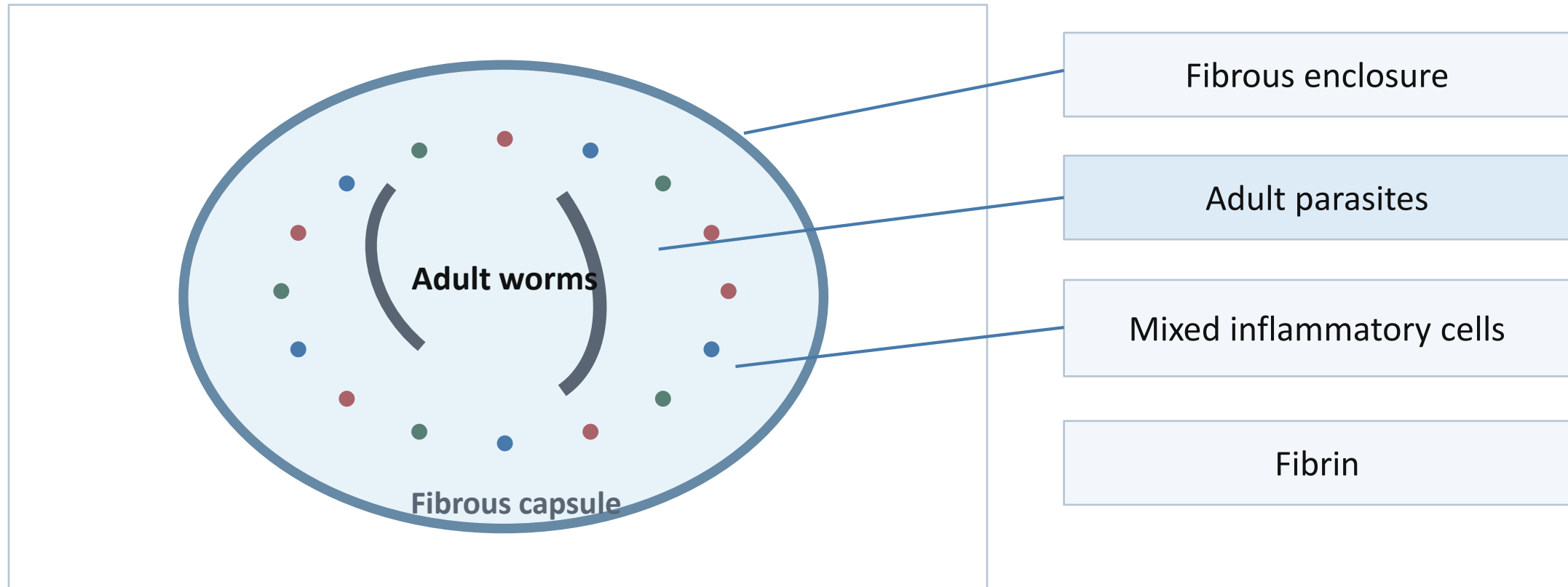
These are source-based lesion associations—not formal diagnostic test criteria.

Case 1: Chronic skin disease near a river

- An individual from a river-associated exposure area has intense itch and patchy light / dark scale.
- A subcutaneous nodule is present alongside diffuse dermatitis.
- Question: Which parasite stage explains each lesion?

Interpret the adult nodule and microfilarial inflammation separately.

Case 1: Compartment-specific explanation



Simplified nodule schematic — not a histology image

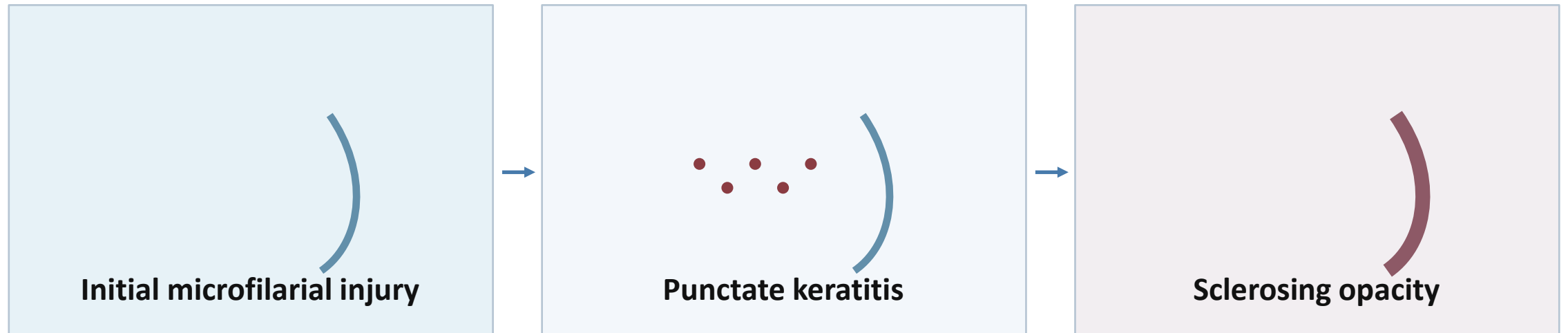
- Adult worms surrounded by a fibrous capsule and mixed cells produce an onchocercoma.
- Skin microfilariae account for the inflammatory, pigmentary dermatitis.

Case 2: Progressive visual impairment

- A patient with relevant exposure develops punctate, fluffy corneal opacity.
- Later, corneal opacity extends from the limbal region.
- Question: Identify the morphological stages and the inflammatory trigger.

Differentiate early punctate from later sclerosing keratitis.

Case 2: Clinicopathologic explanation



Inflammation precedes sclerosis · Conceptual corneal schematic, not a clinical image

- Degenerating microfilariae evoke an eosinophilic infiltrate.
- Punctate keratitis precedes sclerosing, opacifying keratitis.

Five interpretation pitfalls

- Mistaking adult worm nodules for the primary cause of every manifestation.
- Treating “leopard / lizard / elephant skin” as separate infections.
- Assuming keratitis is the only ocular lesion.
- Assuming ivermectin eliminates dermal adult worms.
- Confusing lymphatic elephantiasis with cutaneous onchocerciasis.

Each mistake arises from ignoring site, stage, or morphology.

Integrated pathology: What to retain

- Agent and ecology: *O. Volvulus* transmitted by black flies near running water.
- Parasite stages: Adults form nodules; Microfilariae drive inflammation.
- Skin: Pruritus, pigment / scale changes, atrophy, hyperkeratosis, and fibrosis.
- Eye: Punctate → sclerosing keratitis; Anterior / posterior inflammatory damage.

This slide synthesizes facts established across the lecture.

Source and boundaries

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026).
- Chapter 8, “Onchocerciasis,” printed pp. 365–366.
- Related comparison: preceding “Lymphatic Filariasis” section, pp. 364–365.
- Epidemiologic statements and treatment intervals reproduce the supplied textbook, not a contemporary guideline.