

# **Benign Fibrous Histiocytoma** (Dermatofibroma)

Clinical presentation · Pathogenesis · Morphology · Differential diagnosis

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# Learning objectives: Recognize and distinguish dermatofibroma

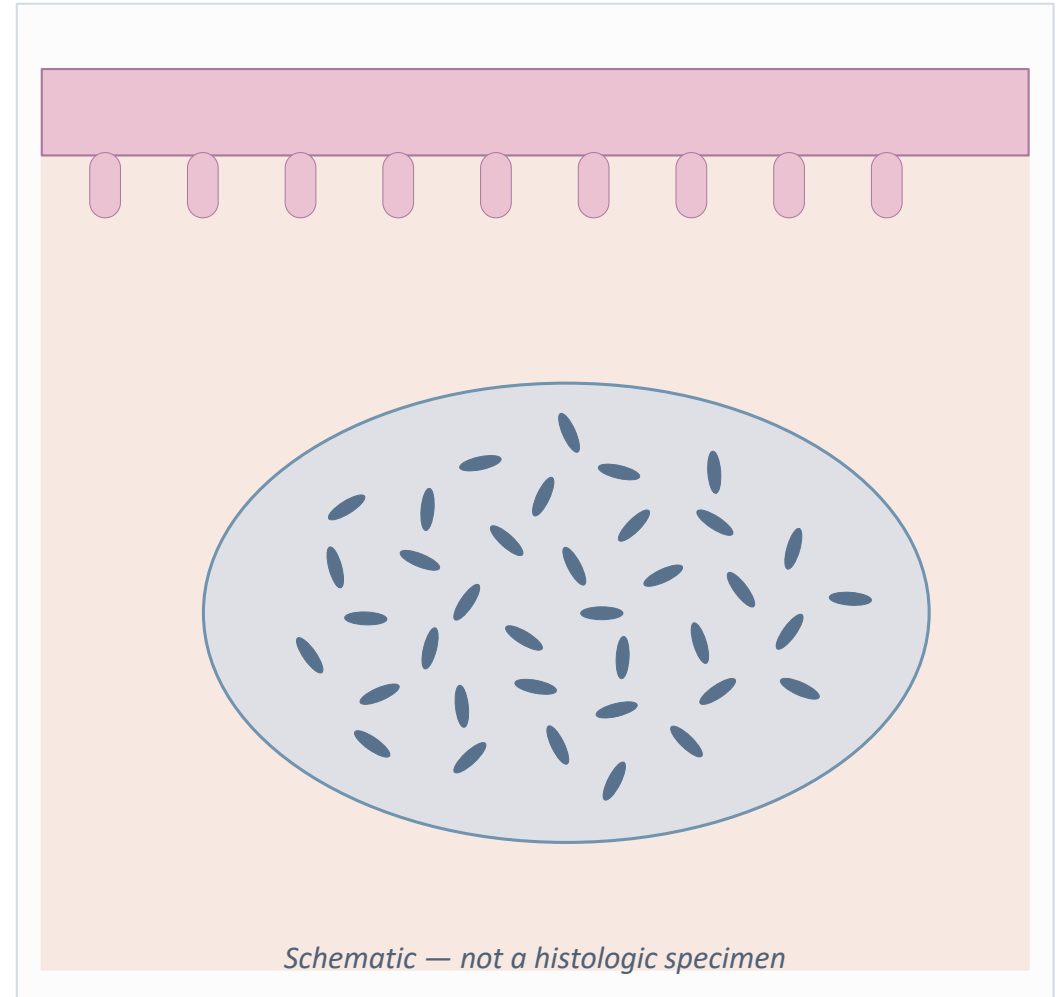
## AT THE END OF THIS LECTURE

- Recognize the usual clinical phenotype and indolent course.
- Explain the uncertain lineage and evidence of true neoplasia.
- Identify defining dermal, epidermal, and collagen-related microscopic findings.
- Distinguish common and selected atypical variants from dermatofibrosarcoma protuberans (DFSP).
- Choose a morphology-led diagnostic and management approach.

# The dermis is the anatomic compartment of origin

## WHY LOCATION MATTERS

- Dermis contains fibroblastic, vascular, neural, and other mesenchymal elements.
- Dermatofibroma is primarily a dermal spindle-cell proliferation.
- Epidermal alterations above the lesion are secondary diagnostic clues.
- Subcutaneous extension is possible and must be interpreted in context.



# Dermatofibroma is a benign fibrohistiocytic neoplasm

## CORE CONCEPT

- Represents the commonest form within the benign fibrous histiocytoma family.
- Morphologically related lesions have uncertain, probably heterogeneous lineage.
- Typical lesions are localized, slowly evolving, and biologically indolent.
- The term “benign” describes the usual course; It does not replace morphologic assessment of variants.

# Related names do not imply identical biology

## DIAGNOSTIC COMPARISON

### Dermatofibroma / BFH

- Common benign dermal fibrohistiocytic lesion.
- May show epidermal acanthosis and collagen entrapment.
- Typically small and confined largely to dermis.

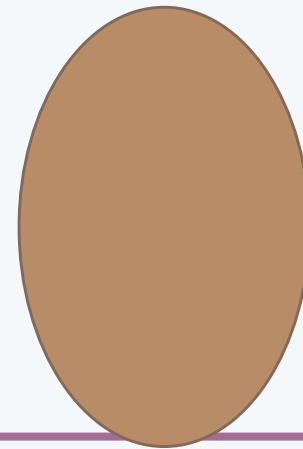
### Dermatofibrosarcoma protuberans

- Distinct infiltrative dermal neoplasm.
- Often grows into fat in a honeycomb pattern.
- Has characteristic COL1A1::PDGFB pathway in many cases.

# A firm tan-to-brown papule is the clinical prototype

## CLINICAL RECOGNITION

- Firm papule or small nodule with tan to brown coloration.
- Most lesions measure less than 1 cm.
- Common clinical sites include the lower extremities.
- Clinical appearance must be interpreted alongside evolution and symptoms.



Clinical schematic

# Patient distribution: A lesion encountered in adults

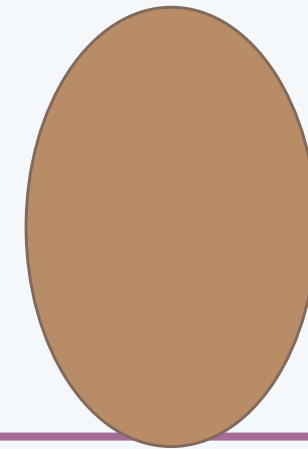
## TYPICAL PATIENT CONTEXT

- Usually recognized in adulthood.
- Particularly frequent on the legs of younger and middle-aged women.
- Occurs across ethnic groups and can arise elsewhere on the skin.
- Demography modifies clinical suspicion but is not itself diagnostic.

# The lower leg is a common but not exclusive site

## ANATOMIC DISTRIBUTION

- Lower extremity location supports—but does not establish—the clinical diagnosis.
- The lesion may occur on the trunk or upper extremities.
- A site unusual for the common phenotype should trigger closer assessment of morphology and history.



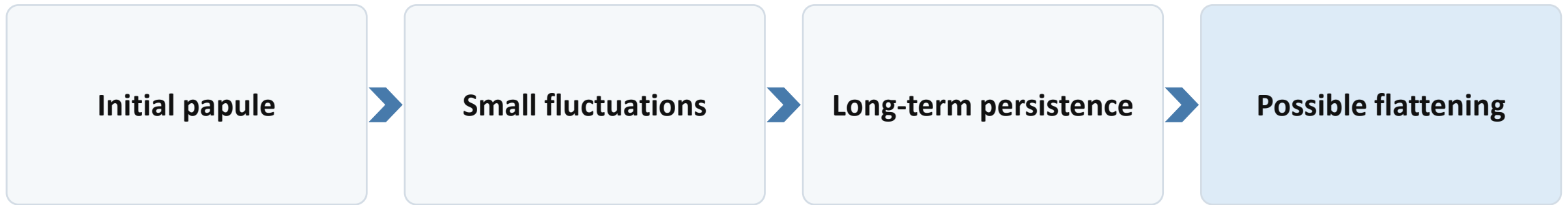
Clinical schematic

# Symptoms range from none to localized tenderness

## CORE CONCEPT

- Many dermatofibromas are asymptomatic.
- Some are tender or irritated by repetitive friction.
- Symptoms alone cannot separate benign dermatofibroma from its mimics.
- A new symptom in a changing lesion warrants clinical reassessment.

# Size evolution often reflects a slow, fluctuating course



## MECHANISTIC REASONING

- Slight increases or decreases in size over time.
- A growing lesion may occasionally reach several centimeters; Most remain small.
- Abrupt, disproportionate growth is a reason to reassess rather than assume a typical course.

# Most lesions are small; Size is context dependent

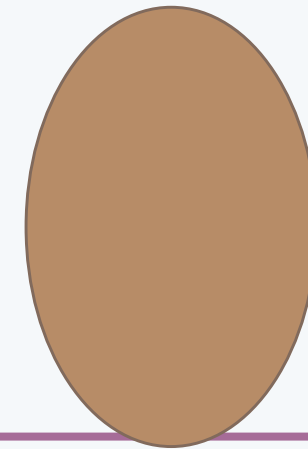
## CORE CONCEPT

- Most dermatofibromas are under 1 cm in diameter.
- Actively growing lesions occasionally become several centimeters across.
- Large size alone is not a histologic diagnosis.
- Size, depth, growth pattern, and cytology should be considered together.

# The dimple sign reflects tethering of the overlying skin

## CLINICAL EXAMINATION

- Lateral compression may cause central depression of a typical dermatofibroma.
- Dimpling is a supportive clinical observation, not a definitive diagnostic test.
- Absence of the sign does not exclude a variant dermatofibroma.



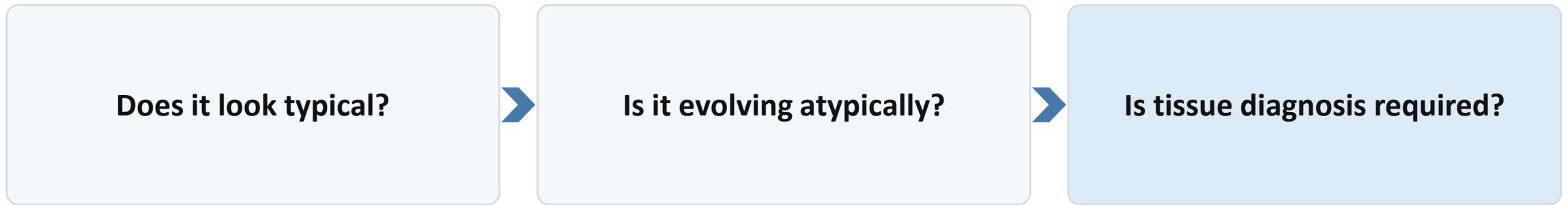
Clinical schematic

# Dermoscopic clues can support clinical recognition

## SUPPLEMENTARY CLINICAL CORRELATION

- A central white scar-like area with a peripheral pigment network is a common dermoscopic pattern.
- Dermoscopic appearances are variable across lesions and skin tones.
- Asymmetry, ulceration, or atypical evolution may justify biopsy.
- Histopathology resolves uncertainty when clinical findings are discordant.

# Clinical assessment asks three distinct questions



## MECHANISTIC REASONING

- Combine distribution, firmness, size, tenderness, and history.
- Consider dermoscopy as supportive rather than conclusive evidence.
- Proceed to tissue assessment when clinical uncertainty could change care.

# Clinical-to-pathologic correlation is multidimensional

## STRUCTURED APPRAISAL

### Surface

- Tan or brown color
- Possible dimple sign
- Epidermal thickening may be present

### Dermal mass

- Firm spindle-cell lesion
- Collagen trapping
- Mid-dermal centered

### Biologic behavior

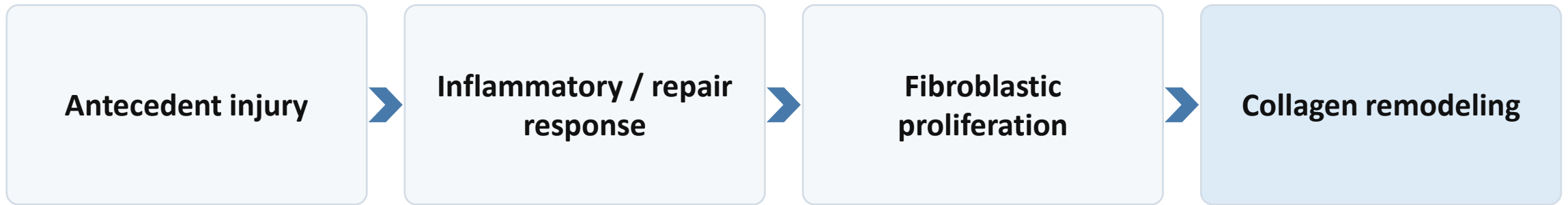
- Usually indolent
- Variable slight size changes
- Clinical red flags require review

# The precise initiating cause remains unresolved

## PATHOGENESIS

- Some lesions follow an antecedent minor injury.
- An abnormal repair or inflammatory response has been proposed.
- Other lesions carry kinase-related fusion genes, favoring a neoplastic process.
- Reactive and neoplastic observations should not be collapsed into a single proven pathway.

# The trauma-associated hypothesis is a model, not a proof



## MECHANISTIC REASONING

- Analogy with hypertrophic scar or keloid is proposed.
- Association with trauma cannot by itself establish causation.
- The molecular findings suggest that some lesions are true neoplasms.

# Kinase-fusion evidence supports true neoplasia in a subset



## MECHANISTIC REASONING

- Rearrangements involving kinases including ALK and protein kinase C are reported in subsets.
- No single driver is claimed to explain all conventional dermatofibromas.
- Fusion-associated subsets help reconcile lesion heterogeneity.

# Epithelioid fibrous histiocytoma highlights molecular diversity

## CORE CONCEPT

- Epithelioid fibrohistiocytic lesions may carry ALK rearrangements.
- Some authorities regard this phenotype as distinct from conventional dermatofibroma.
- An ALK result must be interpreted alongside cell morphology and lesion architecture.
- A molecular association is not evidence that every dermatofibroma is ALK driven.

# Dermal dendritic-cell differentiation is implicated

## CORE CONCEPT

- Dermal dendritic cells a likely partial component of lineage.
- The historical term fibrous histiocytoma reflects mixed fibroblastic/histiocytic appearances.
- Immunophenotype can support differentiation, but lineage is not uniquely established by a single stain.
- Avoid overspecifying the cell of origin.

# Reactive and neoplastic models are not interchangeable

## DIAGNOSTIC COMPARISON

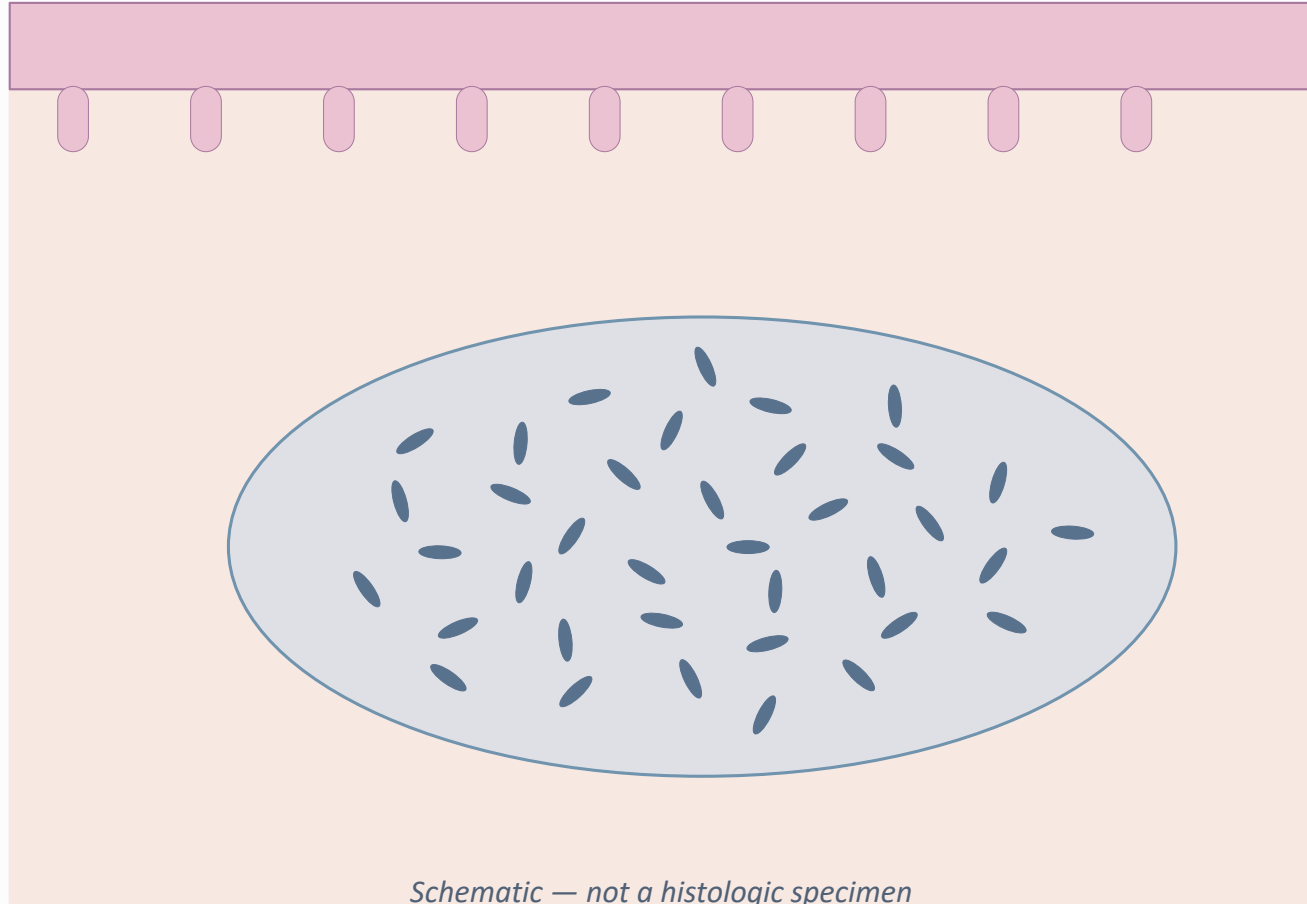
### Reactive hypothesis

- Reported history of local trauma in some cases.
- Morphology may resemble altered scar-like collagen deposition.
- Does not explain all lesions or prove initiating mechanism.

### Neoplastic evidence

- Somatic kinase-related fusions found in some cases.
- Supports autonomous proliferation in at least a subset.
- Does not define one universal pathway for the entire family.

# The classic lesion is a nonencapsulated dermal nodule



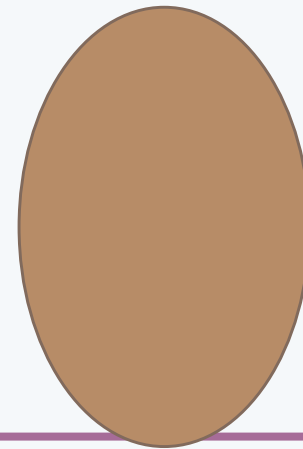
## LOW-POWER ARCHITECTURE

- Bland spindle-cell proliferation is centered in the mid-dermis.
- Typical mass is identifiable but lacks a true capsule.
- Overlying epidermis often shows reactive thickening.
- The relationship to collagen and fat aids recognition.

# Gross morphology: Firm, tan-to-brown, circumscribed papule

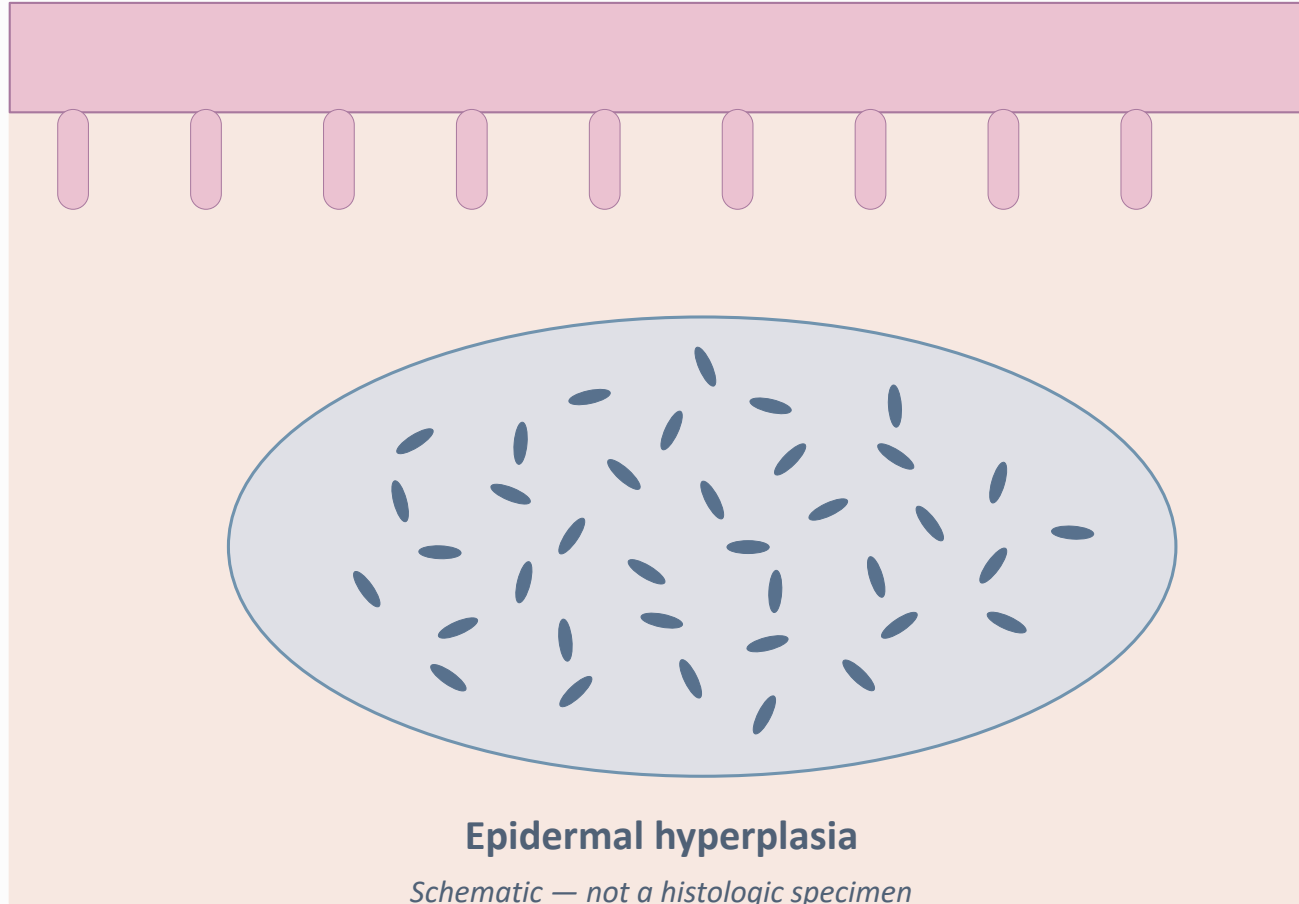
## MACROSCOPIC PATTERN

- Surface lesion commonly appears as a firm papule.
- Pigmentation ranges from tan to brown.
- The clinical border may be more apparent than the microscopic border.
- Morphologic examination determines whether an atypical lesion remains consistent with BFH.



Clinical schematic

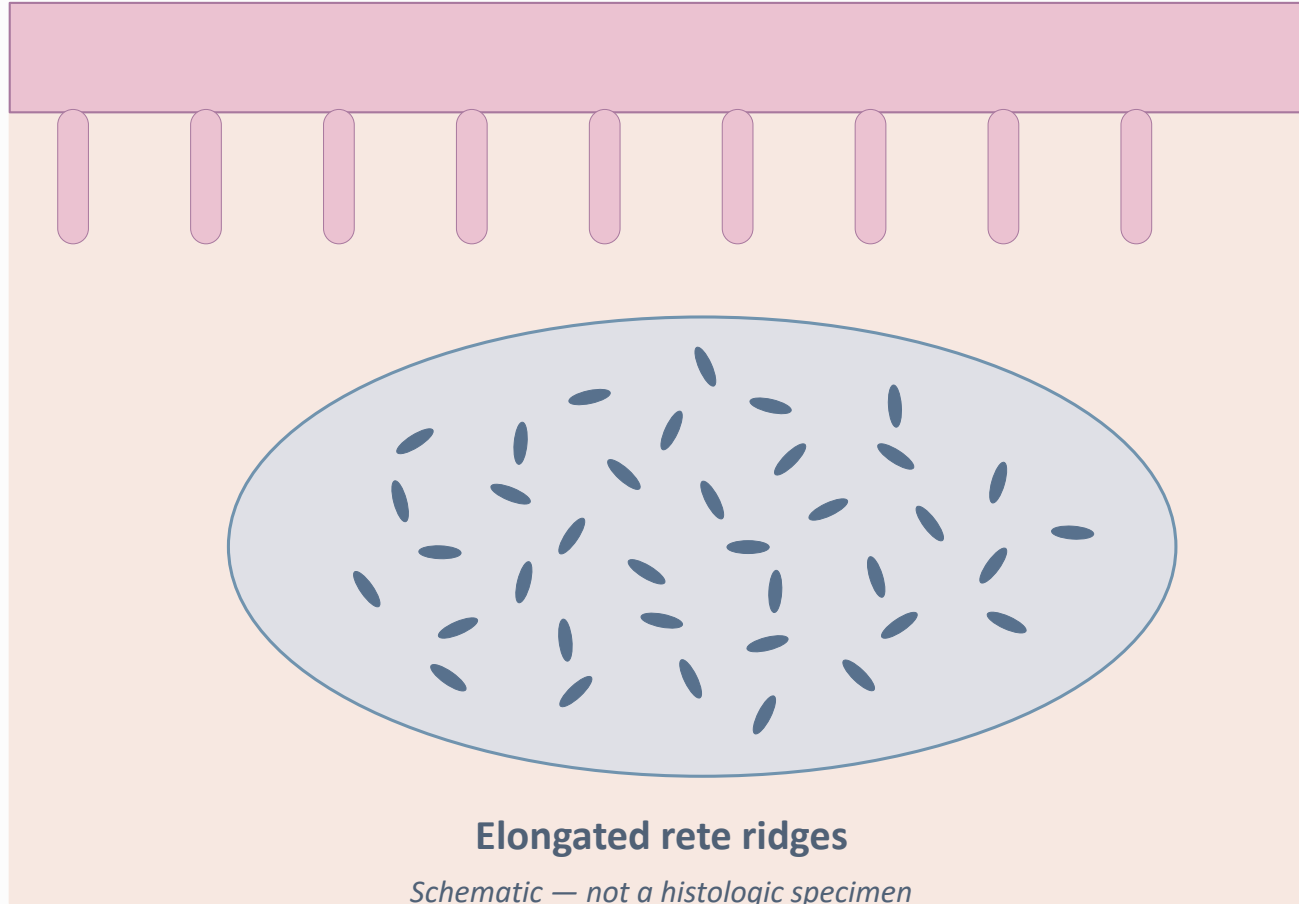
# Overlying epidermal hyperplasia is a recurring diagnostic clue



## READ THE MORPHOLOGY

- Many lesions exhibit thickened epidermis above the dermal mass.
- Rete ridges often extend downward.
- The surface reaction may be disproportionate to the cytologic blandness beneath.
- Not every dermatofibroma shows identical epidermal change.

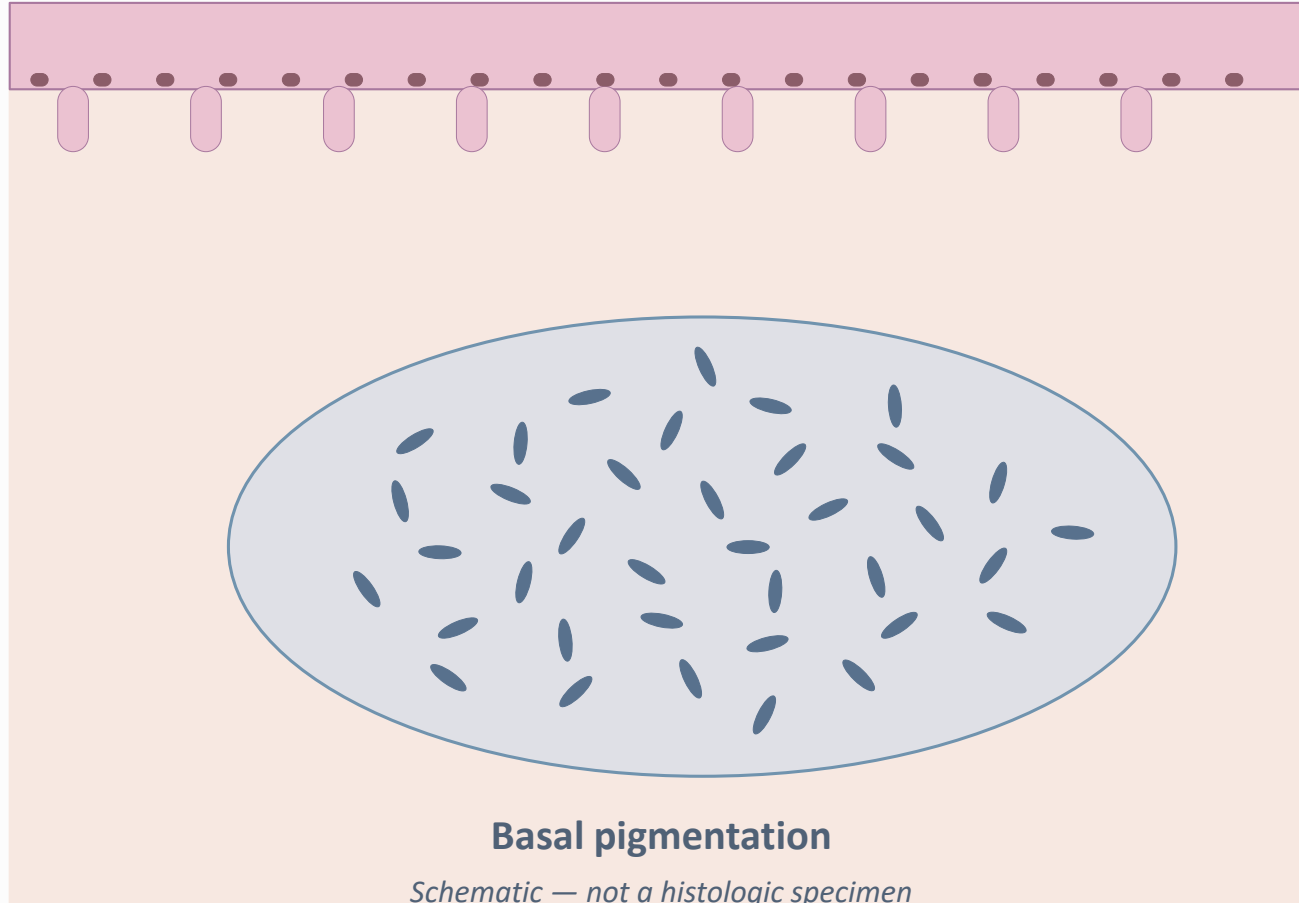
# Pseudoepitheliomatous hyperplasia has a precise meaning



## EPIDERMAL RESPONSE

- The rete architecture is elongated and can be pigmented.
- This reactive-appearing epithelial proliferation overlies a mesenchymal lesion.
- “Pseudoepitheliomatous” does not mean invasive squamous carcinoma.
- Always inspect the underlying dermis before labeling a reactive epithelial change neoplastic.

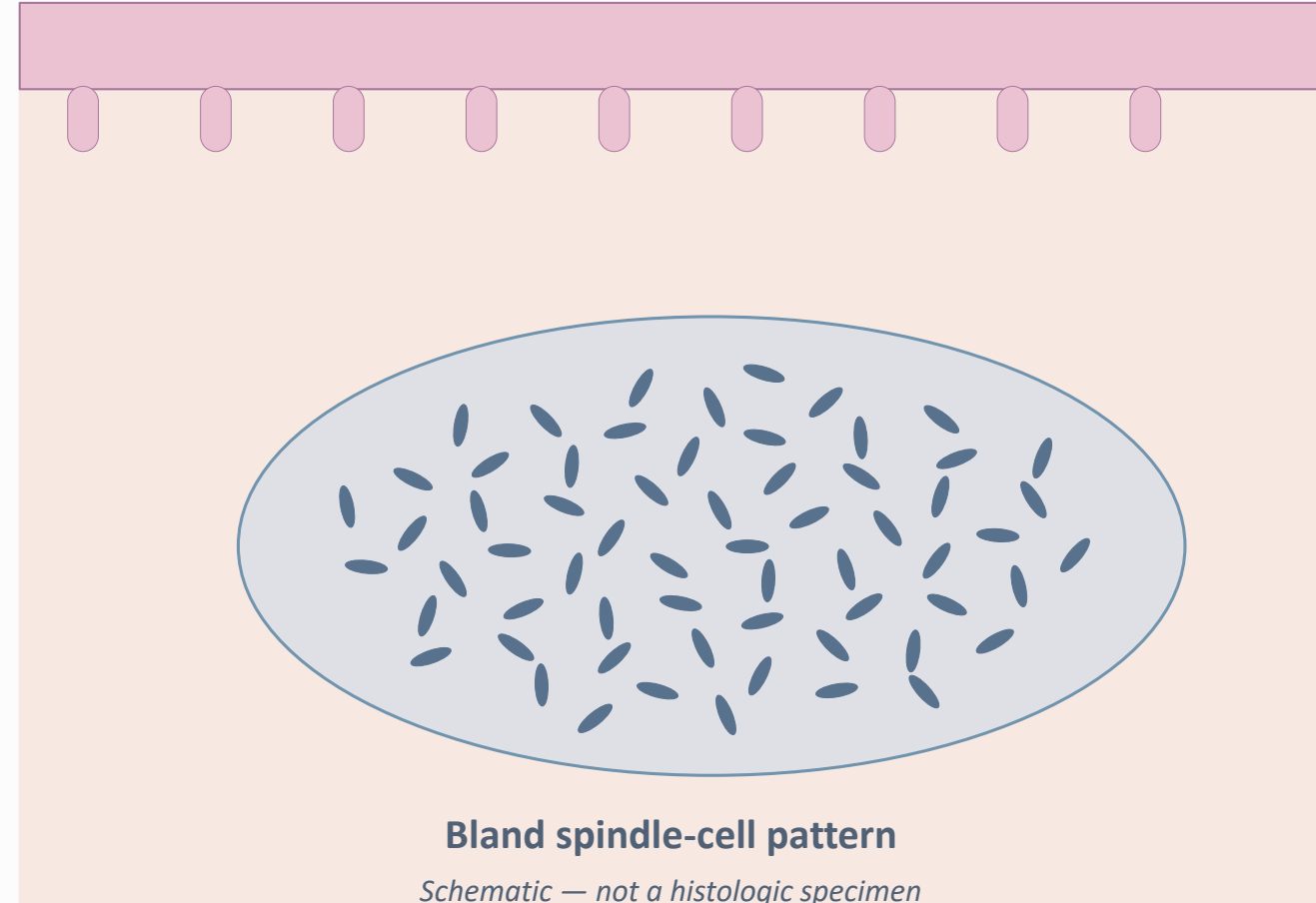
# Basal epidermal hyperpigmentation alters lesion color



## MICROSCOPIC–CLINICAL LINK

- Basal-layer pigmentation may be accentuated above the lesion.
- This contributes to a clinically brown appearance in some cases.
- Surface pigmentation alone cannot distinguish a dermal fibrohistiocytic lesion from a melanocytic mimic.

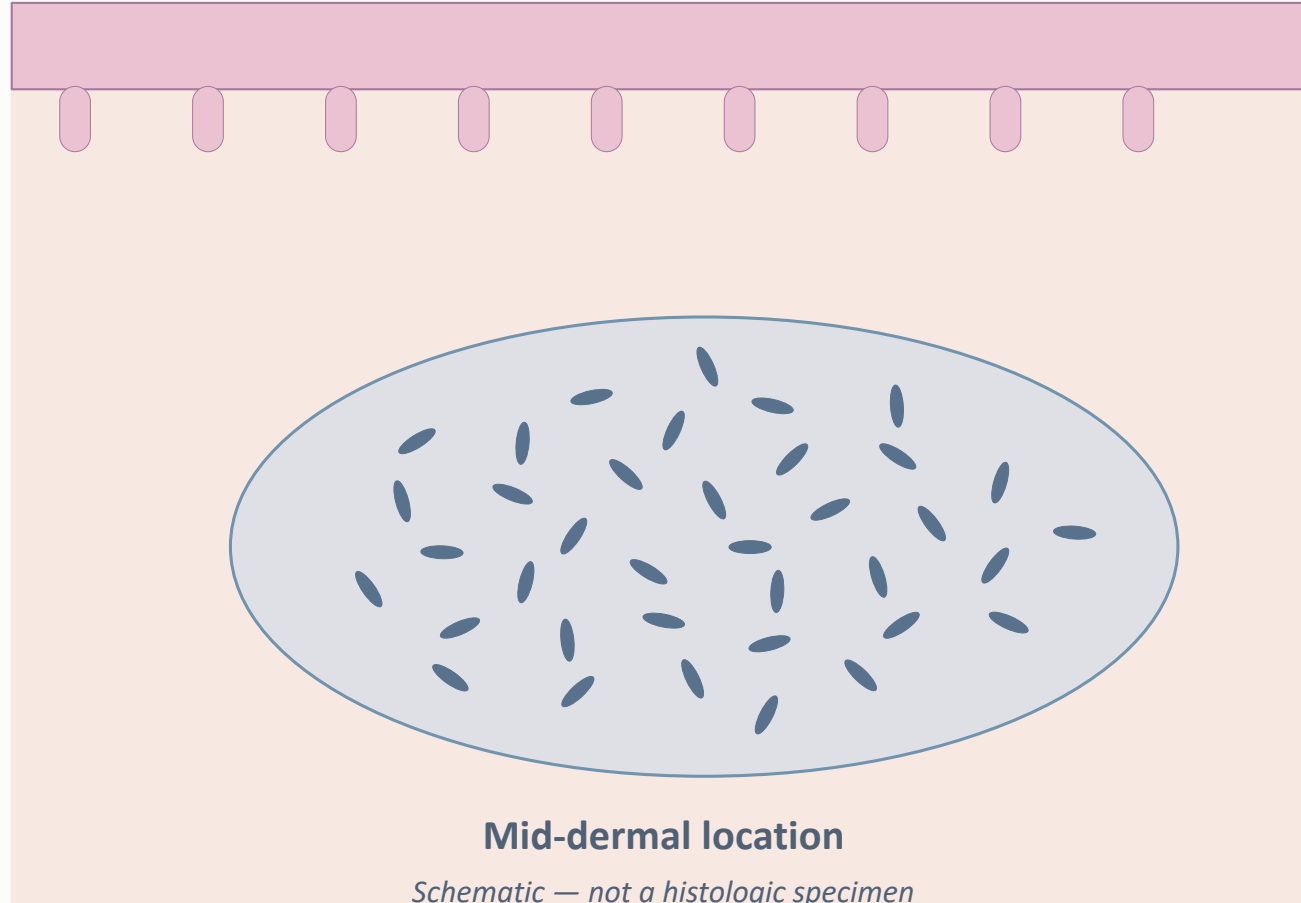
# Bland spindle cells are the principal lesional component



## READ THE MORPHOLOGY

- Cells are elongated with relatively benign-appearing nuclei.
- The proliferation usually occupies a collagen-rich dermal background.
- Cytologic evaluation should be integrated with growth pattern and lesion depth.
- Marked atypia demands variant assessment and exclusion of mimics.

# The lesion is centered in the mid-dermis



## MAPPING TUMOR DEPTH

- Mid-dermal localization is characteristic.
- Relatively superficial epidermal changes may accompany deeper spindle-cell proliferation.
- A biopsy limited to one component can obscure the full diagnostic pattern.

# Circumscribed does not mean encapsulated

## DIAGNOSTIC COMPARISON

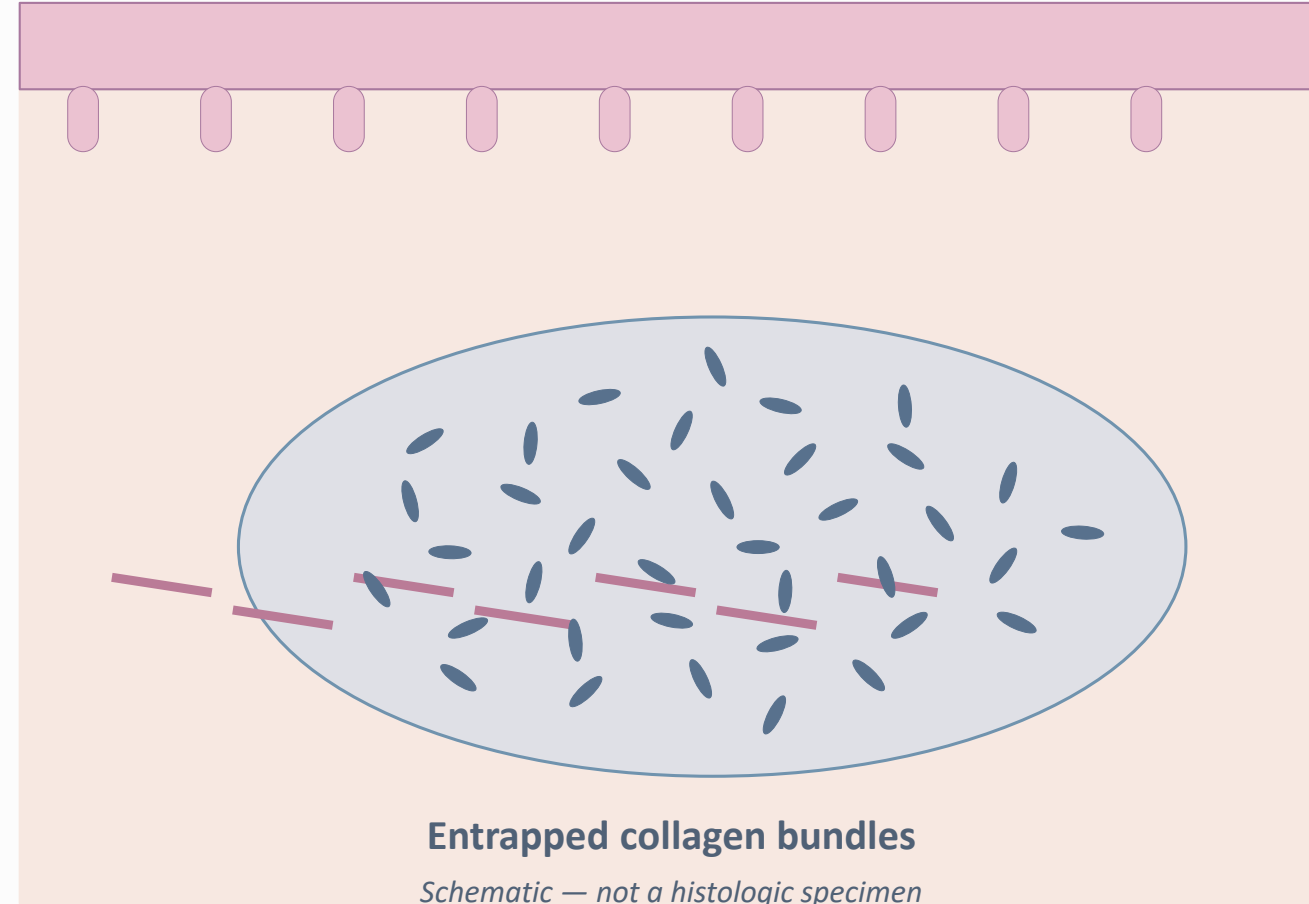
### Architectural circumscription

- A recognizable tumor is demarcated from nearby dermis.
- Margins can merge microscopically with native collagen.
- This is the usual meaning of well-defined BFH.

### True fibrous capsule

- An external encasing layer completely surrounds a mass.
- A typical dermatofibroma lacks this complete capsule.
- Capsule absence alone is not evidence of malignancy.

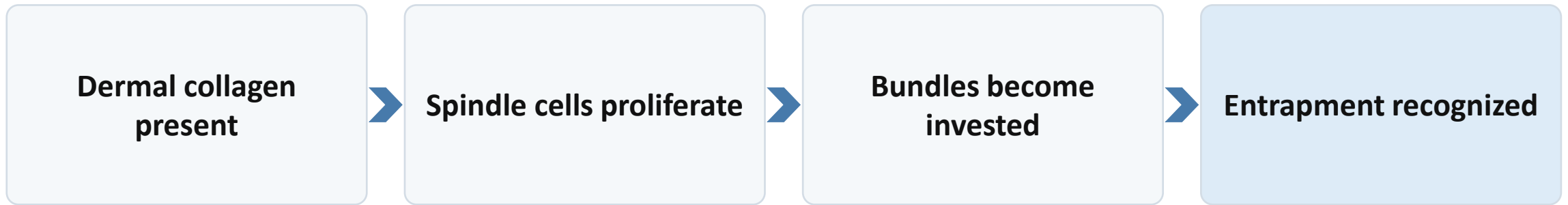
# Peripheral collagen entrapment is especially informative



## DERMAL INTERFACE

- Spindle cells invest and surround individual collagen bundles.
- Entrapped native collagen helps localize the advancing lesion boundary.
- A sampled region without the edge may omit this classic clue.
- Use the finding in combination with overall architecture.

# Why collagen trapping helps lesion recognition



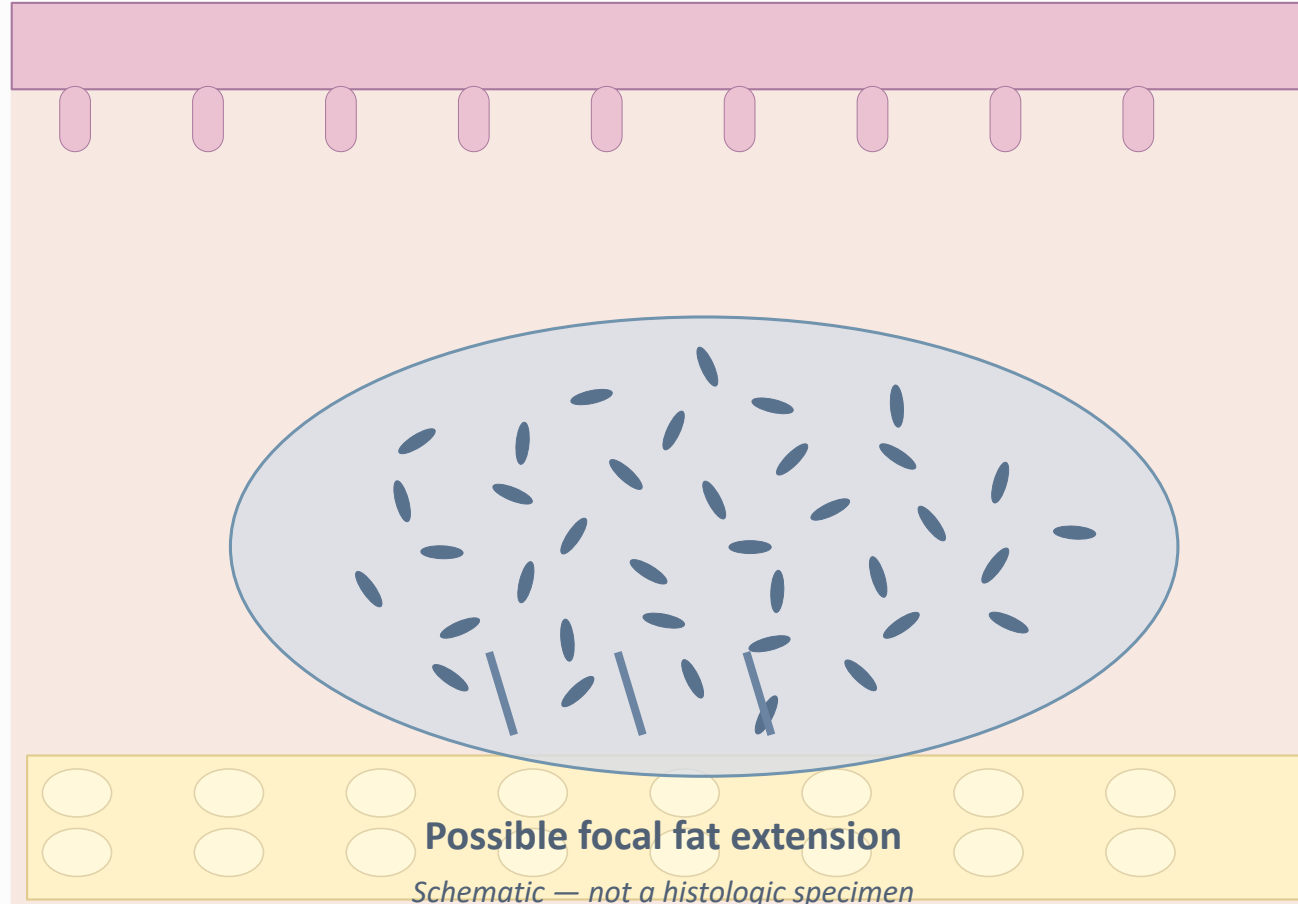
## MECHANISTIC REASONING

- The finding reflects interactions between lesional cells and pre-existing matrix.
- It supports a dermatofibroma pattern but is not a standalone molecular criterion.

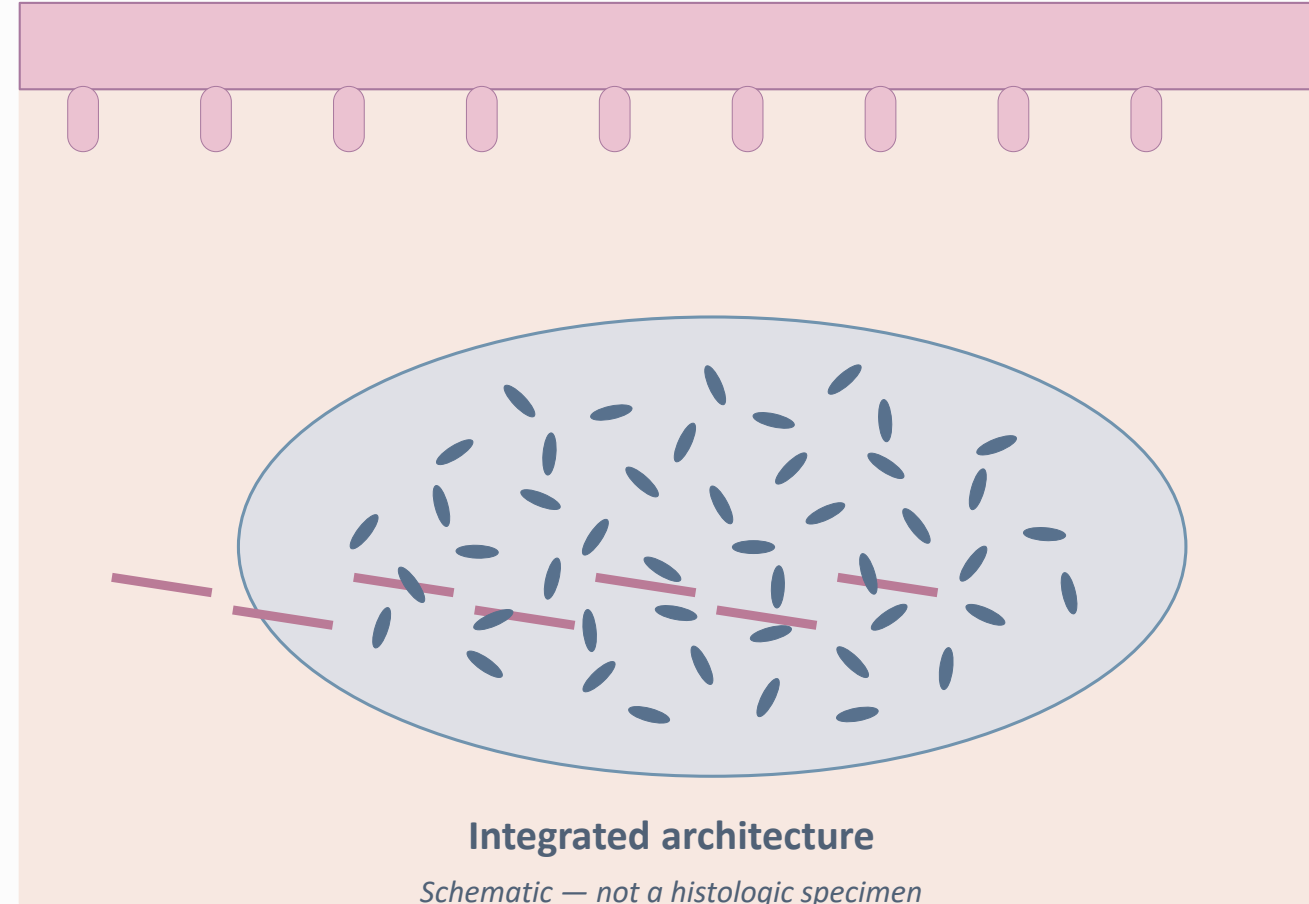
# Occasional subcutaneous extension requires contextual assessment

## READ THE MORPHOLOGY

- Lesional cells can extend into subcutaneous fat.
- This finding does not automatically convert a benign lesion to DFSP.
- Broad infiltration, architecture, and ancillary immunohistochemistry help resolve difficult cases.



# A combined architectural pattern is more diagnostic than a single sign



## READ THE MORPHOLOGY

- Mid-dermal spindle-cell proliferation with bland morphology.
- Reactive acanthosis with elongated or pigmented rete ridges.
- Collagen entrapment at the periphery.
- Nonencapsulation with an otherwise predominantly circumscribed outline.

# Cytologic blandness does not eliminate all diagnostic pitfalls

## CORE CONCEPT

- Typical cells show no conspicuous malignant pleomorphism.
- Cellularity, atypia, mitoses, depth, and vascular spaces vary among subtypes.
- Atypical histology requires more than the label “dermatofibroma.”
- Borderline cases should be evaluated for DFSP and other dermal spindle-cell entities.

# Conventional dermatofibroma: Integrated feature checklist

| Domain      | Expected finding                   | Diagnostic use                             |
|-------------|------------------------------------|--------------------------------------------|
| Clinical    | Small, firm, tan–brown papule      | Supports but does not prove diagnosis      |
| Compartment | Predominantly dermal proliferation | Distinguishes superficial and deep pattern |
| Cytology    | Bland spindle cells                | Favors common BFH phenotype                |
| Epidermis   | Hyperplasia / pigmented rete       | Associated surface response                |
| Matrix      | Collagen entrapment                | Helpful peripheral pattern                 |
| Margin      | Nonencapsulated but defined        | Avoids equating no capsule with malignancy |

# Histologic variants are a family of patterns

## STRUCTURED APPRAISAL

### Cell-rich

- Cellular variant
- More densely cellular spindle cells
- May be diagnostically challenging

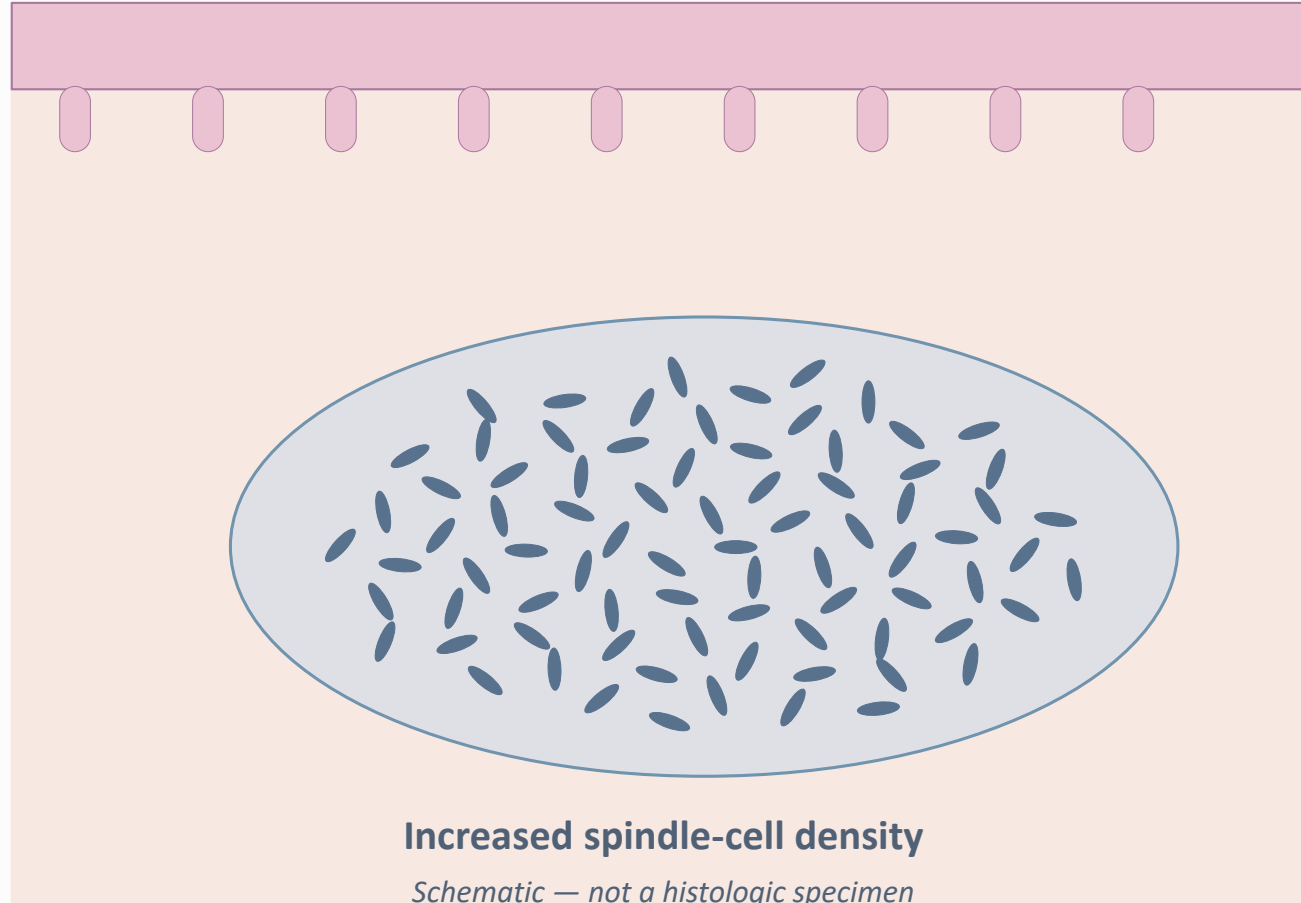
### Blood-related

- Aneurysmal variant
- Blood-filled spaces
- Hemosiderin and hemorrhagic change

### Unusual cytology

- Epithelioid or atypical patterns
- May have distinctive molecular associations
- Require appropriate differentials

# Cellular dermatofibroma shows increased tumor density



## VARIANT MORPHOLOGY

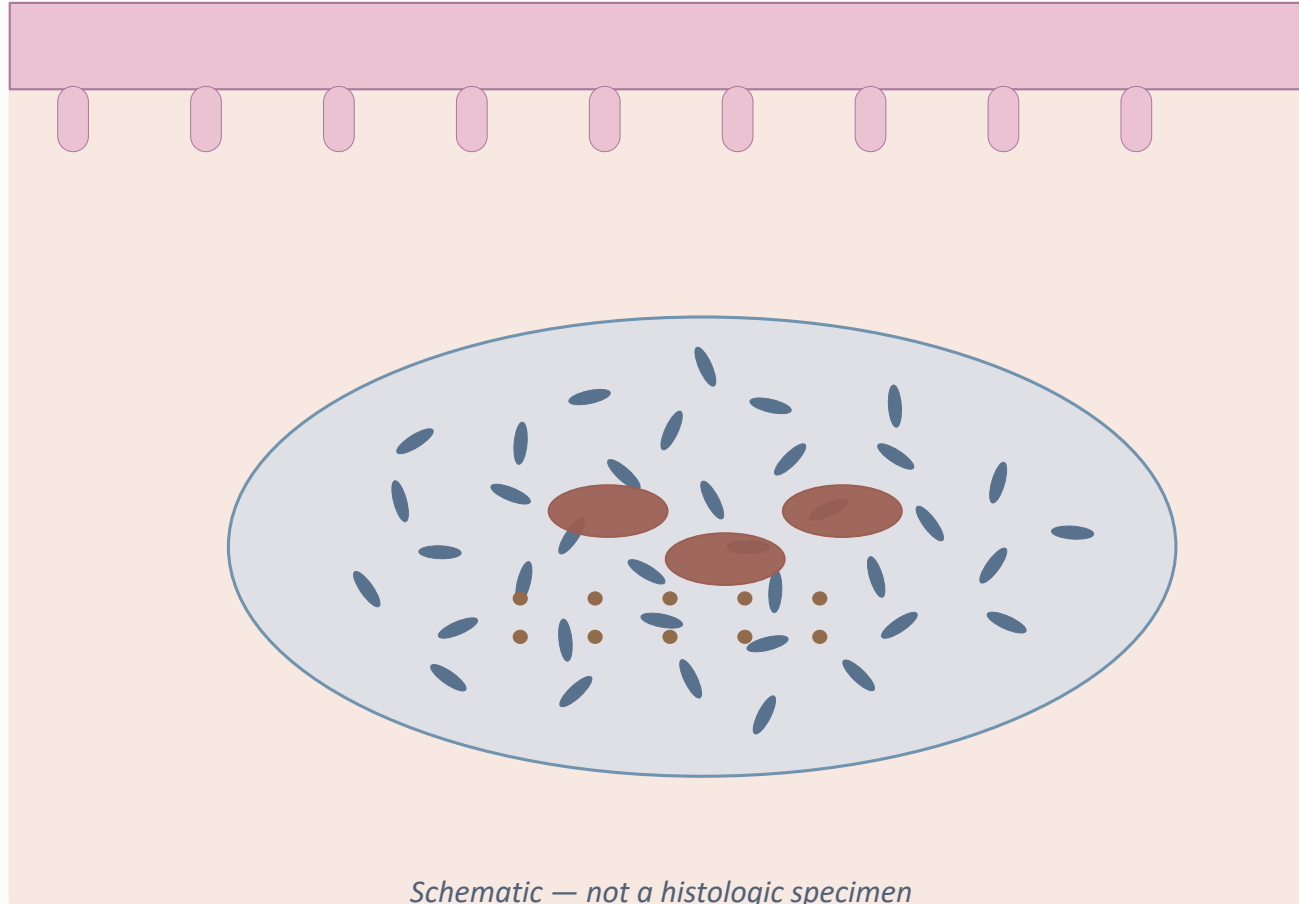
- More crowded spindle cells than in the common form.
- May show more extensive subcutaneous growth.
- Some lesions have increased mitotic activity and more conspicuous storiform architecture.
- Greater overlap with DFSP requires careful sampling.

# Cellular variants merit particular attention to margins

## CORE CONCEPT

- DermNet describes a greater chance of local recurrence than with conventional dermatofibroma.
- Apparent infiltration and high cellularity invite a DFSP differential.
- Immunostains may be useful but are not decisive in isolation.
- Extent of excision and follow-up depend on the specific pathology and clinical context.

# Aneurysmal dermatofibroma contains extravascular blood



*Schematic — not a histologic specimen*

## VARIANT MORPHOLOGY

- Blood pools and hemorrhagic spaces may develop within the tumor.
- Hemosiderin may accumulate around the spaces.
- The lesion can look blue-brown and may enlarge after bleeding.
- The apparent spaces are not equivalent to normal endothelial-lined vessels.

# Blood-filled spaces are not automatically a vascular neoplasm

## DIAGNOSTIC COMPARISON

### Aneurysmal dermatofibroma

- Blood occupies cleft-like spaces in a fibrohistiocytic tumor.
- Spaces lack a true endothelial lining.
- Additional dermatofibroma architecture may be present.

### True vascular structures

- Vessels have endothelial lining.
- Endothelial immunomarkers highlight actual vascular channels.
- The dominant growth pattern must be assessed.

# Hemosiderotic dermatofibroma may mimic a pigmented lesion

## CORE CONCEPT

- Hemosiderin deposition can make the lesion appear dark.
- Pigmentation should be localized morphologically: Epidermal melanin versus dermal iron.
- A suspected melanocytic tumor requires an appropriate clinical and histologic differential.
- Pigment alone cannot establish biologic lineage.

# Epithelioid fibrous histiocytoma is a distinct diagnostic challenge

## CORE CONCEPT

- Usually comprises epithelioid rather than ordinary slender spindle cells.
- ALK rearrangements and ALK protein expression are described in many examples.
- Some authorities separate this lesion from conventional BFH.
- Morphology and immunophenotype are interpreted together.

# Atypical dermatofibroma can display alarming large cells

## CORE CONCEPT

- “Monster cells” or focal pleomorphic cells are recognized in atypical variants.
- The presence of atypia mandates evaluation of the full lesion.
- Rare aggressive outcomes have been reported, but historical case classification can be uncertain.
- Do not extrapolate this behavior to all common dermatofibromas.

# Lipidized and atrophic variants broaden the morphology

## CORE CONCEPT

- Lipidized lesions can contain conspicuous foamy histiocytes and collagen sclerosis.
- Atrophic lesions have reduced thickness and prominent altered collagen.
- These patterns may be clinically or microscopically unfamiliar.
- An integrated pattern-based diagnosis remains preferable to a single isolated finding.

## Variants: Practical discriminating clues

| Variant            | Feature to seek                             | Potential pitfall            |
|--------------------|---------------------------------------------|------------------------------|
| Cellular           | Crowded spindle cells; deeper growth        | DFSP                         |
| Aneurysmal         | Extravascular blood spaces                  | Vascular lesion              |
| Hemosiderotic      | Iron deposition / brown color               | Melanocytic lesion           |
| Epithelioid        | Rounder eosinophilic cells; ALK association | Other epithelioid tumors     |
| Atypical           | Focal enlarged pleomorphic cells            | Sarcoma                      |
| Atrophic/lipidized | Depression or lipid-rich histiocytes        | Other fibroblastic processes |

# DFSP is the critical spindle-cell malignant comparator

## DIAGNOSTIC COMPARISON

### Dermatofibroma

- Usually a small firm papule.
- Predominantly dermal with collagen entrapment.
- Benign behavior in the conventional type.

### Dermatofibrosarcoma protuberans

- Slowly enlarging indurated plaque or protuberant nodule.
- May infiltrate subcutaneous fat extensively.
- Can recur locally; Metastasis is uncommon.

# Surface epidermis often points in opposite directions

## DIAGNOSTIC COMPARISON

### Dermatofibroma

- Overlying epidermis may be thickened.
- Downward elongated and sometimes hyperpigmented rete ridges.
- Pseudoepitheliomatous pattern supports DF.

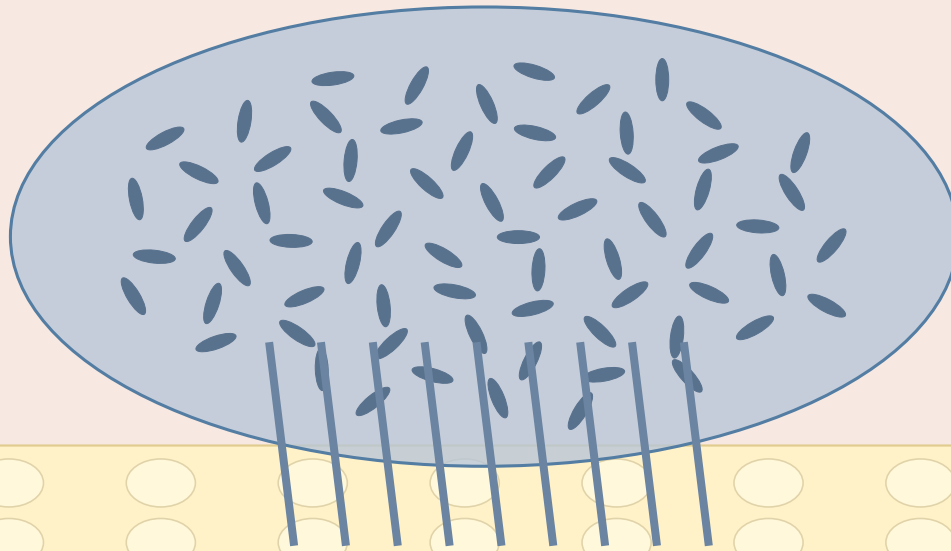
### DFSP

- Epidermis is often attenuated or thinned.
- Surface may ulcerate in larger lesions.
- The epidermal clue must be interpreted with depth and architecture.

# DFSP fat infiltration can produce a honeycomb pattern

## COMPARATOR SCHEMATIC

- DFSP commonly infiltrates adipose compartments from a dermal origin.
- Isolated fat cells become surrounded by spindle-cell proliferation.
- This broad infiltrative architecture differs from usual superficial BFH.
- Minor fat extension in BFH means the feature is not absolute.



*Schematic — not a histologic specimen*

# Storiform growth is not uniquely diagnostic of DFSP

## DIAGNOSTIC COMPARISON

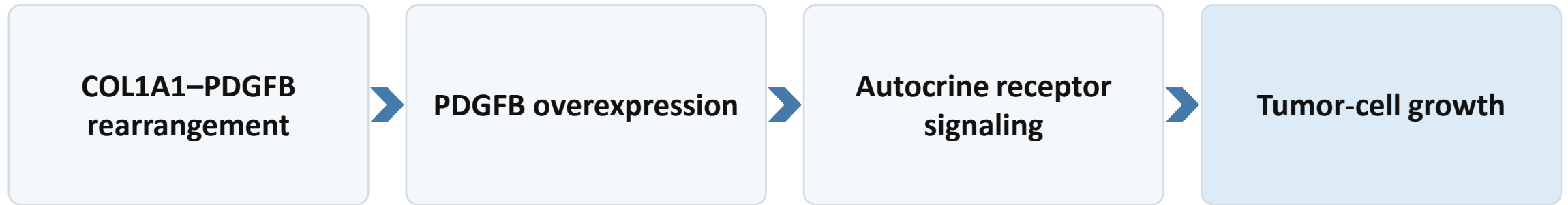
### In dermatofibroma

- Variable swirled or short fascicular spindle-cell proliferation.
- Collagen entrapment and reactive epidermis are useful companions.
- Cellular BFH can be particularly storiform.

### In DFSP

- Tightly packed spindle cells often form a prominent radial “cartwheel.”
- Usually accompanies an infiltrative, deeply extending growth pattern.
- Do not diagnose DFSP from a storiform pattern alone.

# DFSP has a distinct COL1A1::PDGFB oncogenic mechanism



## MECHANISTIC REASONING

- This pathway belongs to DFSP—not to conventional dermatofibroma.
- The signaling mechanism to receptor-targeted therapy in selected advanced DFSP.

# Factor XIIIa and CD34 can support the distinction

## DIAGNOSTIC COMPARISON

### Dermatofibroma

- Often factor XIIIa-positive in lesional cells.
- Typically CD34-negative or only focally positive.
- Staining patterns vary across BFH variants.

### DFSP

- Typically diffuse CD34 staining.
- Factor XIIIa highlights mainly entrapped or peripheral dendritic cells.
- A panel complements, rather than replaces, architecture.

# How to interpret a discordant immunostain panel

| Observation                            | Appropriate interpretation                                       |
|----------------------------------------|------------------------------------------------------------------|
| CD34 focal at periphery                | Can occur in dermatofibroma, especially cellular cases.          |
| CD34 diffuse in lesional spindle cells | Raises support for DFSP in the right architecture.               |
| Factor XIIIa lesional labeling         | Supports BFH, but not absolutely specific.                       |
| Overlapping or equivocal stains        | Review full lesion, depth, and alternate markers as appropriate. |
| Scant superficial biopsy               | May be insufficient to judge infiltrative architecture.          |

# Morphology-led diagnostic matrix: BFH versus DFSP

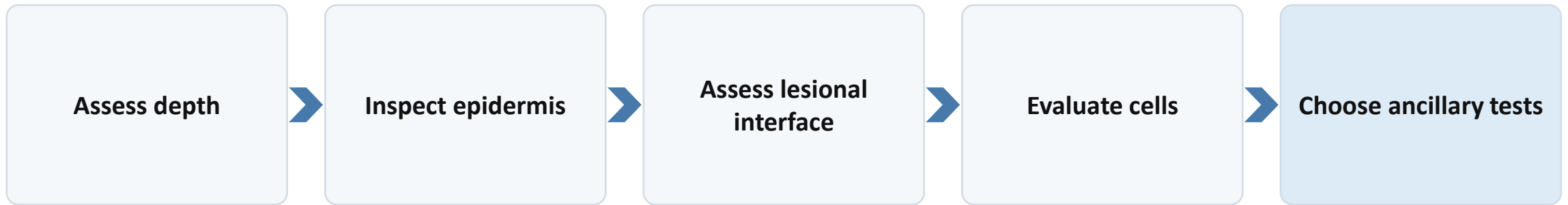
| Feature           | Dermatofibroma                          | DFSP                      |
|-------------------|-----------------------------------------|---------------------------|
| Typical skin site | Often leg                               | Often trunk               |
| Surface epidermis | Hyperplasia frequent                    | Often thinned             |
| Dermal interface  | Collagen trapping                       | More infiltrative         |
| Subcutis          | Occasional extension                    | Honeycomb invasion common |
| Immunophenotype   | FXIIIa+, often CD34–                    | Diffuse CD34 typical      |
| Molecular pathway | Heterogeneous; kinase fusions in subset | COL1A1::PDGFB hallmark    |

# Other lesions can enter the clinical or histologic differential

## CORE CONCEPT

- Pigmented melanocytic lesions: Resolve with epidermal/dermal pattern and lineage assessment.
- Hypertrophic scar or keloid: Trauma-associated collagen alteration, but no identical BFH architecture.
- Vascular lesions: Genuine endothelial-lined channels rather than aneurysmal blood pools.
- Other dermal spindle-cell neoplasms: Evaluated with depth, cytology, and targeted markers.

# Tissue diagnosis begins with architecture, not an antibody



## MECHANISTIC REASONING

- Look for collagen entrapment, subtype-specific features, and fat infiltration.
- Order ancillary tests when the morphology and differential warrant them.
- Reconcile discordant results rather than privileging one positive stain.

# Biopsy sampling determines which clues are accessible

## STRUCTURED APPRAISAL

| Surface                                                                                                                                           | Lesion body                                                                                                                                            | Deep interface                                                                                                                                                        |
|---------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"><li>• Can show acanthosis</li><li>• May show hyperpigmented rete</li><li>• Absent in deep-only sample</li></ul> | <ul style="list-style-type: none"><li>• Shows spindle-cell density</li><li>• Reveals hemorrhage or atypia</li><li>• Helps subtype assignment</li></ul> | <ul style="list-style-type: none"><li>• Can disclose fat invasion</li><li>• Distinguishes penetration patterns</li><li>• May be omitted by shallow sampling</li></ul> |

# Clinically atypical lesions deserve histologic assessment

## CORE CONCEPT

- Consider biopsy when there is recent conspicuous enlargement, ulceration, or asymmetric pigmentation.
- Histology should assess the whole growth pattern whenever feasible.
- Tenderness or clinical pigmentation alone does not identify a malignant lesion.
- Resolve the specific differential rather than using a nonspecific label.

# Usual dermatofibroma often needs no active treatment

## CORE CONCEPT

- A typical asymptomatic lesion can be clinically observed.
- Reassurance is usually sufficient once the diagnosis is adequately established.
- Removal may be appropriate for symptoms, cosmetic concerns, or unresolved diagnostic doubt.
- Management is not interchangeable with that for DFSP.

# Excision strategy depends on the actual diagnosis

## DIAGNOSTIC COMPARISON

### Common dermatofibroma

- Clinical observation can be reasonable when typical.
- Symptomatic or uncertain lesions may be removed.
- Routine oncologic margins are not a feature of usual DF.

### DFSP / concerning variant

- DFSP generally requires definitive complete surgical excision.
- Cellular/atypical BFH may need closer margin assessment.
- Specialist input is appropriate for disputed or higher-risk pathology.

# A useful pathology report communicates pattern and certainty

## CORE CONCEPT

- Identify the lesion and recognized variant when warranted.
- Describe relevant epidermal, dermal, collagen, and deep-margin findings.
- State atypia, cellularity, or hemorrhagic changes when diagnostically material.
- Document ancillary results together with interpretive limitations.
- State uncertainty and need for correlation when a differential remains unresolved.

## Case application: Stable firm nodule on the shin

### CLINICAL VIGNETTE

A 32-year-old woman has a small, firm tan-brown papule on the lower leg that has changed little over several years. Histology shows bland spindle cells in the mid-dermis, epidermal hyperplasia, and collagen entrapment.

### CLINICOPATHOLOGIC INTERPRETATION

- Pattern is most consistent with conventional dermatofibroma.
- The combination of location, evolution, and morphology supports a benign diagnosis.

# Case application: Hypercellular lesion extends into fat

## CLINICAL VIGNETTE

A densely cellular cutaneous spindle-cell lesion involves the dermis and focally reaches subcutaneous tissue. Overlying epidermis is reactive; CD34 is focal rather than diffusely strong.

## CLINICOPATHOLOGIC INTERPRETATION

- Consider cellular dermatofibroma and DFSP in the differential.
- Evaluate growth architecture, fat pattern, lesional factor XIIIa, and completeness of sampling.

# Case application: Suddenly darkening blue-brown nodule

## CLINICAL VIGNETTE

A previously small dermal nodule enlarges and becomes blue-brown. Sections reveal bland fibrohistiocytic areas and irregular spaces filled with blood but lacking a vascular lining.

## CLINICOPATHOLOGIC INTERPRETATION

- Aneurysmal dermatofibroma is a coherent histologic explanation.
- Exclude a true vascular tumor and consider appropriate excision assessment.

# Case application: Indurated plaque with fat honeycombing

## CLINICAL VIGNETTE

A slow-growing indurated plaque on the trunk forms a protuberant nodule. Histology shows tightly packed storiform cells, attenuated epidermis, and honeycomb invasion of adipose tissue.

## CLINICOPATHOLOGIC INTERPRETATION

- The constellation supports dermatofibrosarcoma protuberans rather than ordinary DF.
- Confirm the diagnosis with appropriate ancillary studies and plan definitive management.

## Knowledge check: Associate clue with interpretation

| Clue                                   | Most appropriate interpretation                           |
|----------------------------------------|-----------------------------------------------------------|
| Hyperpigmented elongated rete          | Supports reactive epidermal response over BFH             |
| Peripheral collagen entrapment         | Useful classic BFH interface feature                      |
| Blood lakes without endothelial lining | Think of aneurysmal BFH                                   |
| Diffuse lesional CD34 + honeycomb fat  | Strongly consider DFSP                                    |
| Kinase fusion involving ALK            | Possible fibrohistiocytic molecular subset; not universal |
| Growing lesion with marked atypia      | Reassess variant and malignant differential               |

# Integrated clinicopathologic synthesis

## CORE CONCEPT

- Dermatofibroma is usually a small, benign, firm cutaneous lesion.
- The diagnostic core is a dermal spindle-cell proliferation with epidermal reaction and collagen entrapment.
- Variant patterns explain deviations in cellularity, cytology, or hemorrhage.
- Distinction from DFSP relies on architecture, fat infiltration, and supportive immunophenotype.
- The reactive-versus-neoplastic debate is nuanced by kinase fusions in subsets.

# References and recommended diagnostic reading

## PRIMARY AND SUPPLEMENTARY READING

|                         |                                                                                      |
|-------------------------|--------------------------------------------------------------------------------------|
| <b>Primary textbook</b> | Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed (2026). Ch 25, p 1044. |
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| <b>Dermatopathology</b> | Cheng H, Emanuel P. Dermatofibroma pathology. DermNet NZ (2013). |
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| <b>Clinical correlation</b> | Oakley A. Dermatofibroma. DermNet NZ (revised 2020). |
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| <b>Comparator</b> | DermNet NZ. Dermatofibrosarcoma protuberans pathology. |
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| <b>Immunohistochemistry</b> | CD34 and factor XIIIa in DF versus DFSP. Am J Dermatopathol (1993); PMID 7694515. |
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