

Nonneoplastic Epithelial Disorders

Vulvar leukoplakia, lichenoid conditions, and benign exophytic lesions

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Learning objectives

- Interpret leukoplakia as a clinical descriptor rather than a specific histologic diagnosis.
- Distinguish lichen sclerosus from lichen simplex chronicus using morphology and mechanism.
- Recognize benign exophytic vulvar lesions and the hallmark morphology of condyloma acuminatum.
- Explain the nuanced association of chronic vulvar disorders with HPV-independent carcinoma.

The vulva: Several lesion patterns share the same site

White plaques

Leukoplakia encompasses inflammatory, reactive, premalignant, and malignant causes.

Raised lesions

Benign exophytic proliferations may be infectious or noninfectious.

Neoplastic mimics

VIN, Paget disease, and invasive carcinoma can enter the clinical differential.

Why cutaneous disorders matter on the vulva

- Inflammatory skin diseases such as psoriasis, eczema, and allergic dermatitis can also involve vulvar skin.
- Exposure to moisture and secretions favors superficial infections at this anatomic site.
- The clinical appearance of a plaque does not identify its underlying histologic process.
- This lecture focuses on common nonneoplastic lesions and their neoplastic mimics.

Leukoplakia: A descriptive, not etiologic, term

- Literally “white plaques”; Denotes opaque, white, plaquelike epithelial thickening.
- Pruritus and scaling may accompany the white thickening.
- The same descriptive appearance can arise from biologically distinct conditions.
- Clinical leukoplakia alone therefore does not establish benignity.

**White plaque ≠
diagnosis**

Three broad causes of vulvar leukoplakia

Group	Examples	Interpretive implication
Inflammatory	Psoriasis; Chronic dermatitis	Dermatosis rather than tumor
Nonneoplastic epithelial	Lichen sclerosus; Squamous cell hyperplasia	Look for characteristic architectural change
Neoplastic	VIN; Paget disease; Invasive carcinoma	White color cannot exclude neoplasia

Pruritus and scaling are not specific diagnoses

- Leukoplakic plaques may produce itch and superficial scaling.
- Lichen simplex chronicus specifically develops in the setting of rubbing and scratching to relieve itch.
- Lichen sclerosus also causes white plaques but has a distinct atrophic and sclerotic phenotype.
- Interpret symptoms together with lesion architecture and microscopy.

White vulvar lesions: Distinguish observation from diagnosis

Observation

A smooth or thickened white surface is a clinical finding.

Hypothesis

Lichen sclerosus, lichen simplex chronicus, and dermatoses are possibilities.

Tissue category

Histomorphology separates atrophy/sclerosis, hyperplasia, and neoplasia.

Why neoplastic lesions remain in the differential

- Vulvar intraepithelial neoplasia, Paget disease, and invasive carcinoma among causes of leukoplakia.
- A white surface is compatible with both nonneoplastic and neoplastic tissue processes.
- The later vulvar neoplasia section discusses HPV-associated and HPV-independent carcinogenic pathways.
- Nonneoplastic plaque morphology should not be equated with absence of a distinct adjacent neoplastic lesion.

The two principal nonneoplastic white-plaque disorders

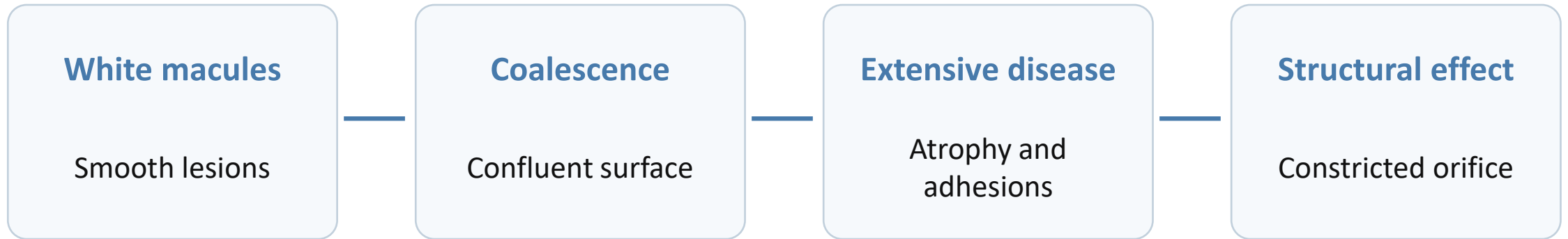
Feature	Lichen sclerosis	Lichen simplex chronicus
Core process	Atrophy and dermal sclerosis	Reactive epidermal hyperplasia
Clinical context	Smooth, porcelain/parchment plaques	Pruritic, scratched leukoplakic skin
Microscopy	Thin epidermis, basal injury	Acanthosis, hyperkeratosis

Lichen sclerosis: Characteristic clinical surface

- Begins as smooth, white plaques or macules.
- Plaques may enlarge and coalesce rather than remaining isolated.
- The resulting surface is likened to porcelain or parchment.
- This clinical phenotype differs from a purely hyperplastic, thick epidermal response.

**Smooth · white ·
atrophic**

How lichen sclerosis alters vulvar architecture



- The sequence summarizes the changes described with extensive vulvar involvement.

Lichen sclerosis: Consequences of widespread involvement

- Atrophic labia and adhesions may develop when the entire vulva is affected.
- Changes in tissue architecture may narrow the vaginal orifice.
- Visible surface color alone understates the potential structural consequences.
- Gross appearance and functional anatomy should be considered alongside microscopy.

Lichen sclerosis: Age and distribution

- The disease can be encountered in every age group.
- It is most common in postmenopausal females.
- Lichen sclerosis is not confined to the vulva; It also occurs elsewhere on the skin.
- An older age distribution is an epidemiologic tendency, not a defining criterion.

Lichen sclerosis: Evidence suggesting immune involvement

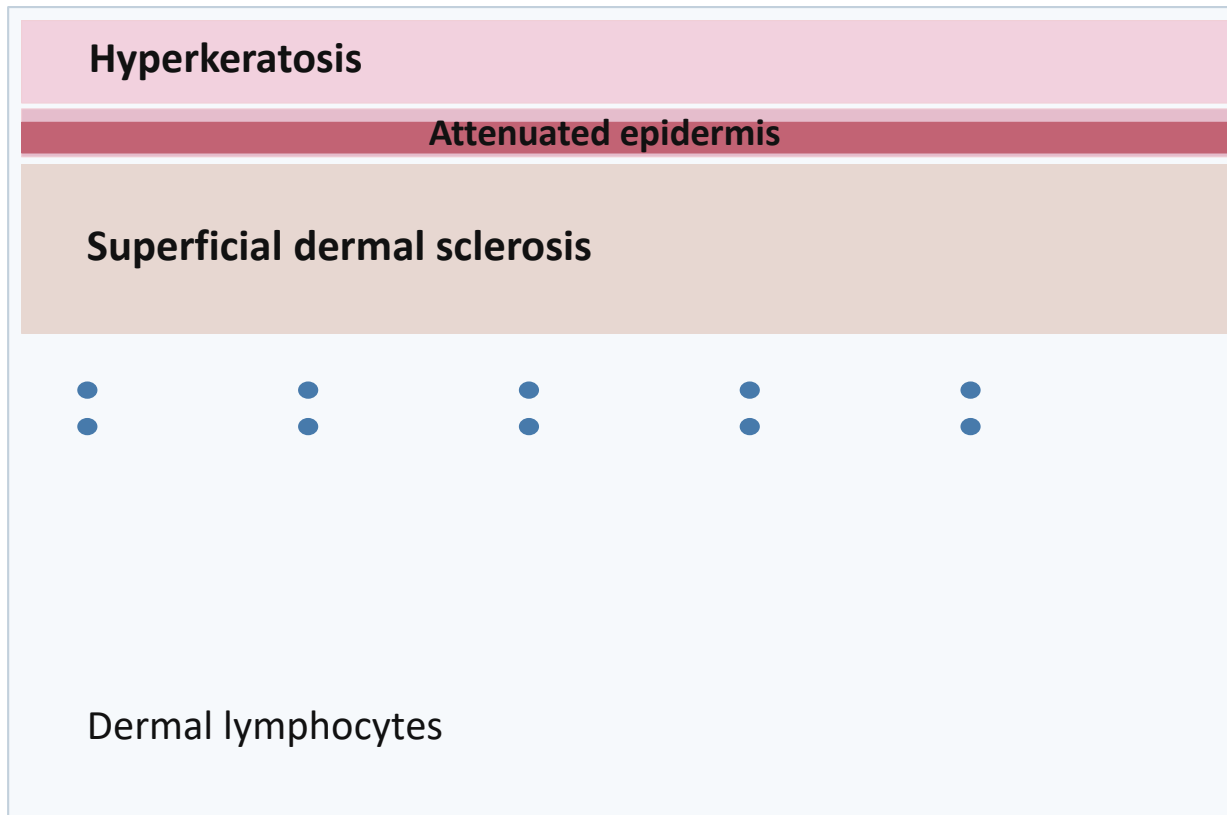
- Activated T cells are present in the subepithelial inflammatory infiltrate.
- Associated autoimmune disorders occur more frequently in affected females.
- Together these findings suggest an autoimmune reaction may contribute.
- The source does not establish a single definitive initiating mechanism.

**Evidence supports
an immune
hypothesis — not
proof of etiology**

Lichen sclerosis: Observation versus interpretation

Observed	Mechanistic interpretation
Activated T cells in infiltrate	Consistent with a cellular immune response
More autoimmune comorbidity	Supports an autoimmune association
Uncertain pathogenesis	Do not present one immune mechanism as proven

Lichen sclerosis: The histologic pattern



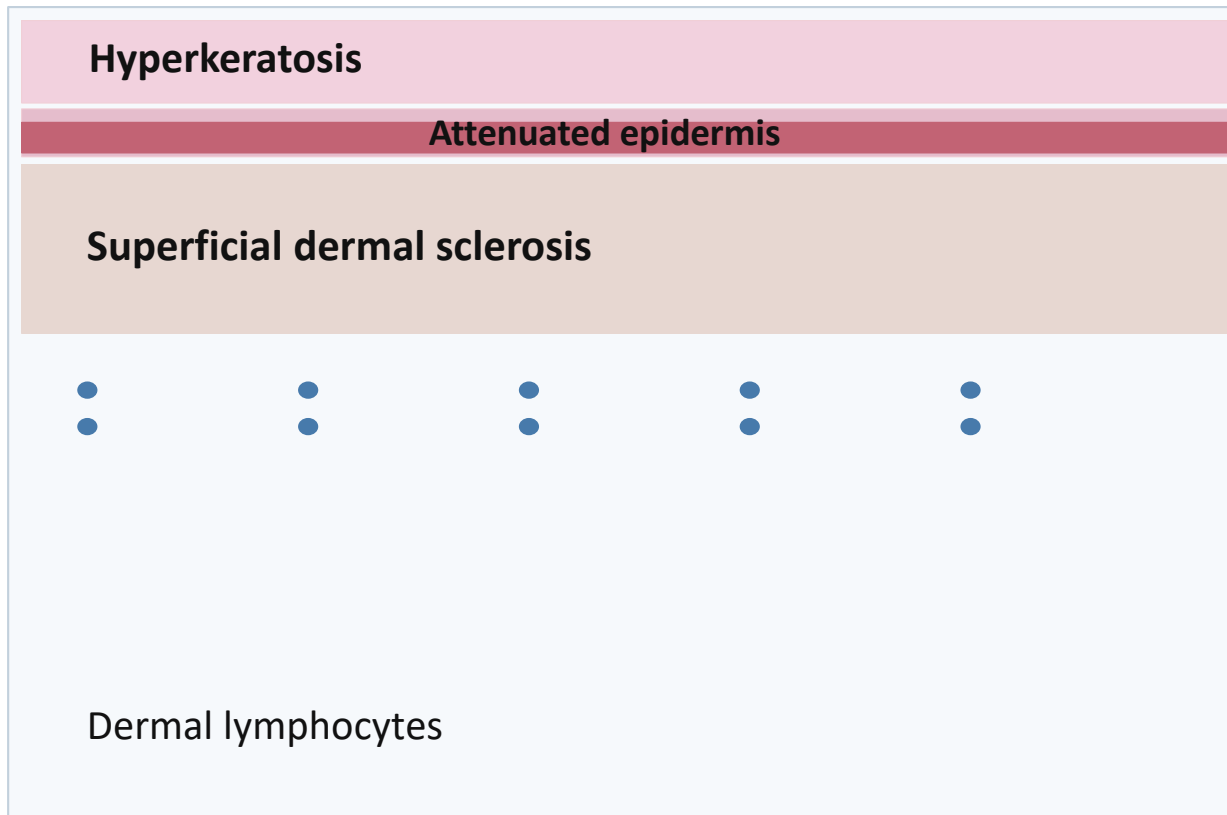
- Marked attenuation of the epidermis is a central feature.
- The basal epithelial layer shows degenerative injury.
- Hyperkeratosis can coexist with epidermal thinning.
- Superficial dermal sclerosis and deeper inflammation complete the pattern.

Schematic cross-section — not a photomicrograph

Lichen sclerosis: Epidermal attenuation

- Thinning affects the stratified squamous epidermis beneath the keratin surface.
- The source identifies marked epidermal thinning, not a thick acanthotic epidermis.
- Recognize atrophy in the context of subjacent superficial dermal sclerosis.
- This architectural contrast is especially valuable in the LS versus LSC differential.

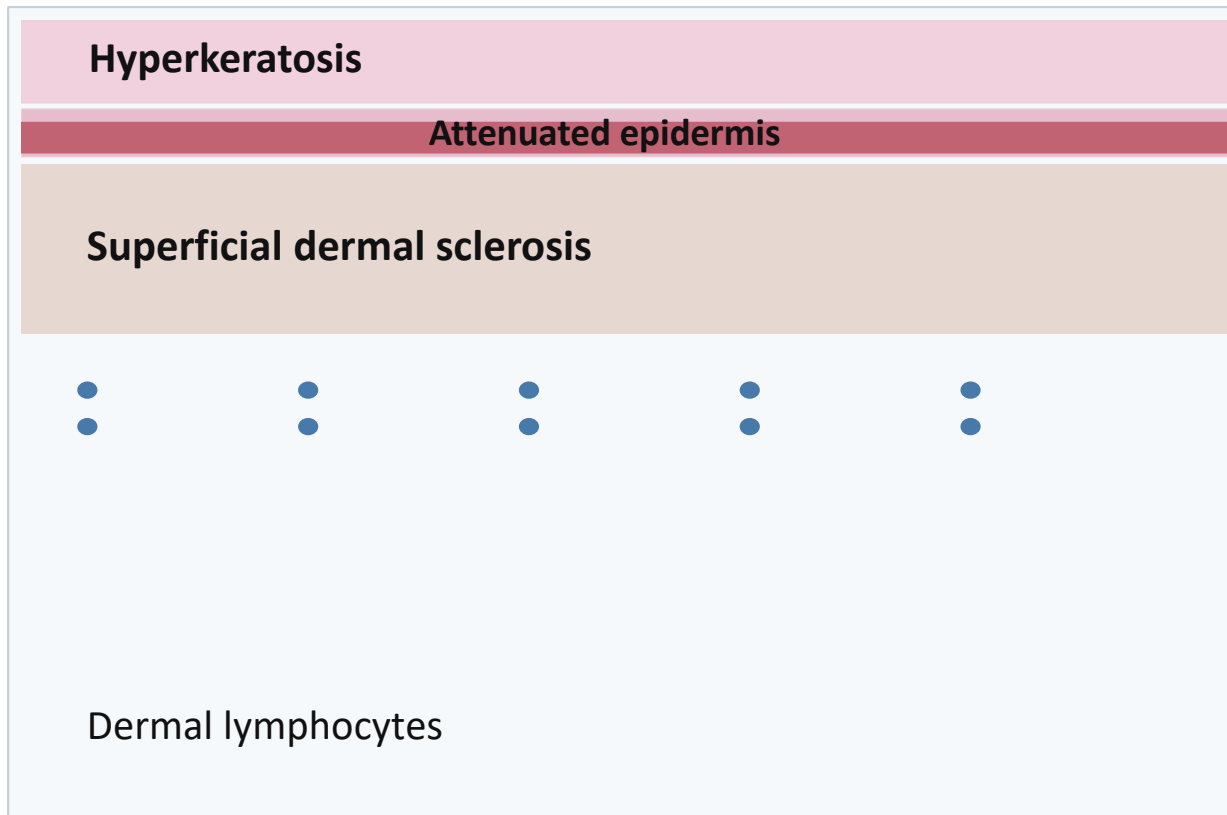
Lichen sclerosis: Basal injury and keratinization



- Basal epithelial cells show degenerative change.
- Hyperkeratosis is excessive keratin production at the surface.
- Thin viable epidermis and increased surface keratin are not contradictory.
- Interpret surface change and living epidermal thickness separately.

Schematic cross-section — not a photomicrograph

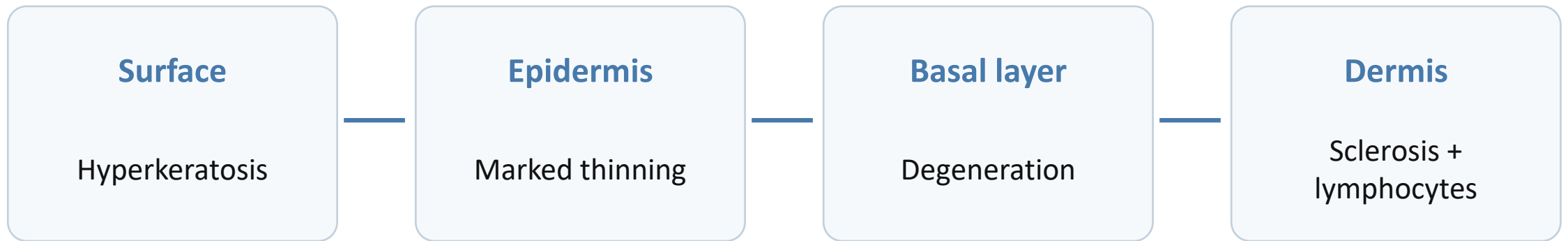
Lichen sclerosis: The superficial dermis



- Superficial dermis shows sclerosis beneath the epithelial layer.
- A lymphocytic inflammatory band is described in the underlying dermis.
- Location of sclerosis and the inflammatory infiltrate is part of the pattern.
- Both epithelial and dermal changes are needed for an integrated reading.

Schematic cross-section — not a photomicrograph

Read the lichen sclerosis biopsy by compartment



- This is a morphologic reading sequence, not a validated scoring system.

Lichen sclerosis: Clinicopathologic concordance

Clinical observation	Pathologic substrate
Smooth porcelain-like white plaques	Surface keratinization with epidermal attenuation
Atrophic vulvar changes	Marked epithelial thinning and dermal sclerosis
Chronic plaque disorder	Bandlike lymphocytic infiltrate in dermis

Cancer risk: Separate the lesion from the patient

- Lichen sclerosus is not itself a premalignant lesion.
- Nevertheless, affected females have a slightly increased chance of HPV-independent vulvar squamous cell carcinoma.
- This association is not equivalent to inevitable progression of an LS plaque.
- Distinguish a nonneoplastic inflammatory dermatosis from a separate epithelial precursor lesion.

**Nonpremalignant
lesion ≠ zero
associated cancer
risk**

The HPV-independent neoplasia context

Associated background

Long-standing lichen sclerosus or lichen simplex chronicus may be present.

Distinct neoplastic precursor

Differentiated VIN is an epithelial precursor, not another name for lichen sclerosus.

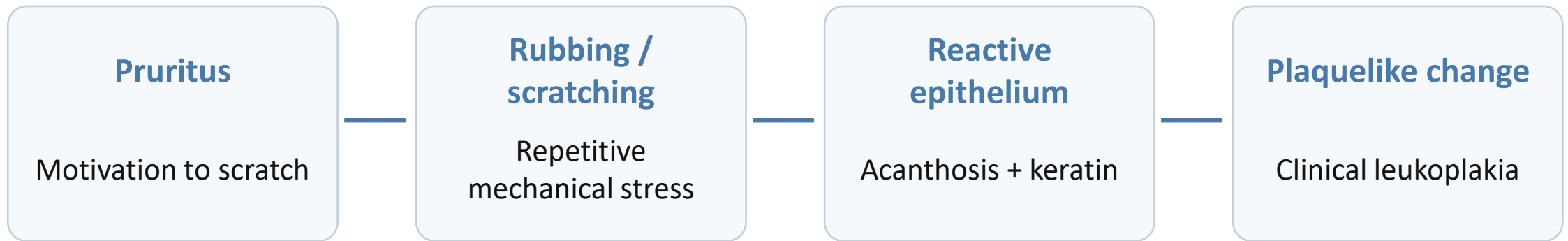
Malignancy

HPV-independent vulvar squamous cell carcinoma may arise; Chronic irritation is a proposed contributor.

A diagnostic pitfall: Assuming “leukoplakia” is benign

- A white plaque may reflect lichen sclerosus, lichen simplex chronicus, VIN, Paget disease, or carcinoma.
- A history compatible with chronic dermatosis cannot on its own rule out another process.
- The source describes some HPV-independent cancers as insidious and mistaken for dermatitis or leukoplakia.
- Preserve the distinction between presenting phenotype and histologic diagnosis.

Lichen simplex chronicus: The itch–scratch cycle



- The source characterizes the disorder as a nonspecific response to rubbing or scratching.

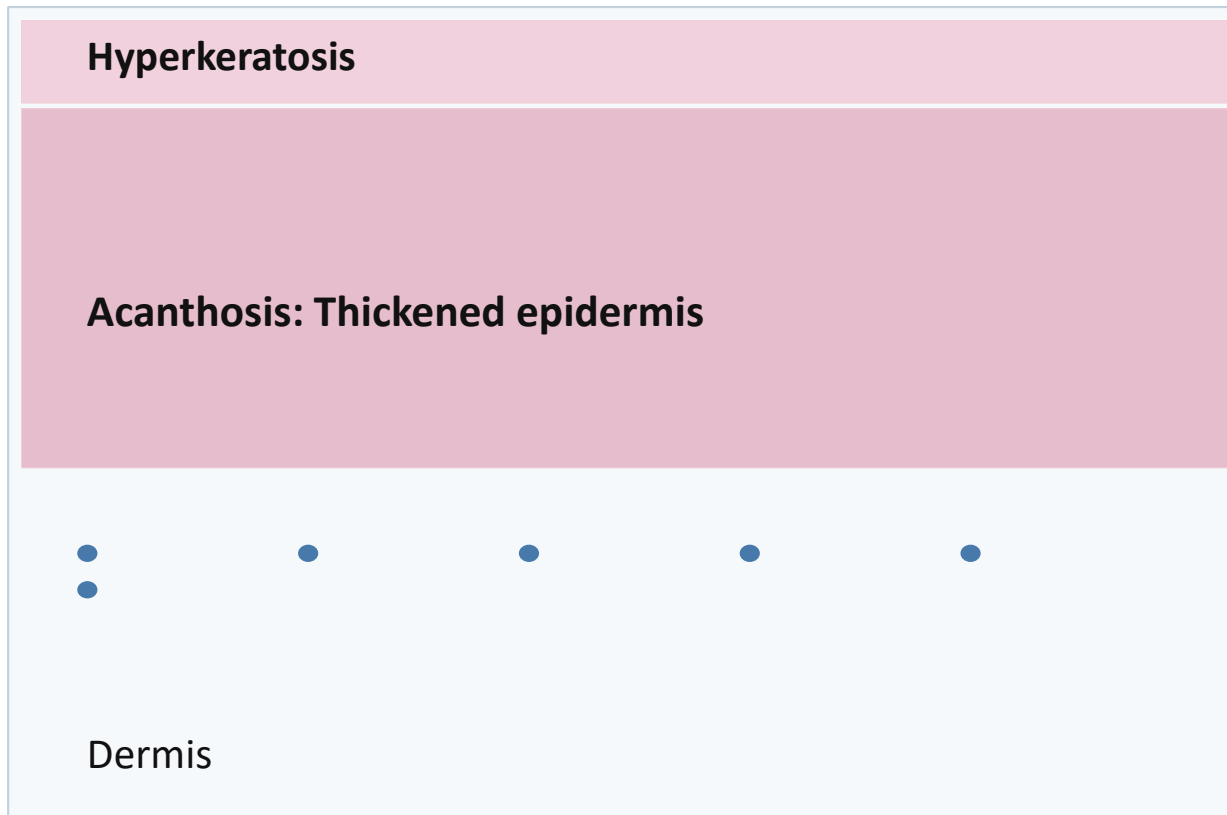
Lichen simplex chronicus: Historical terminology

- This disorder was formerly termed “hyperplastic dystrophy.”
- The term “squamous cell hyperplasia” is also used.
- These labels emphasize epidermal proliferation rather than atrophy.
- Use the current name while recognizing older terminology in reports and teaching material.

Lichen simplex chronicus: Clinical presentation

- The clinical appearance can be leukoplakic, much like lichen sclerosis.
- Its pathogenic setting is repeated scratching or rubbing to relieve itch.
- Epidermal thickening is a reactive consequence of ongoing mechanical stress.
- Color alone does not distinguish it from other leukoplakic processes.

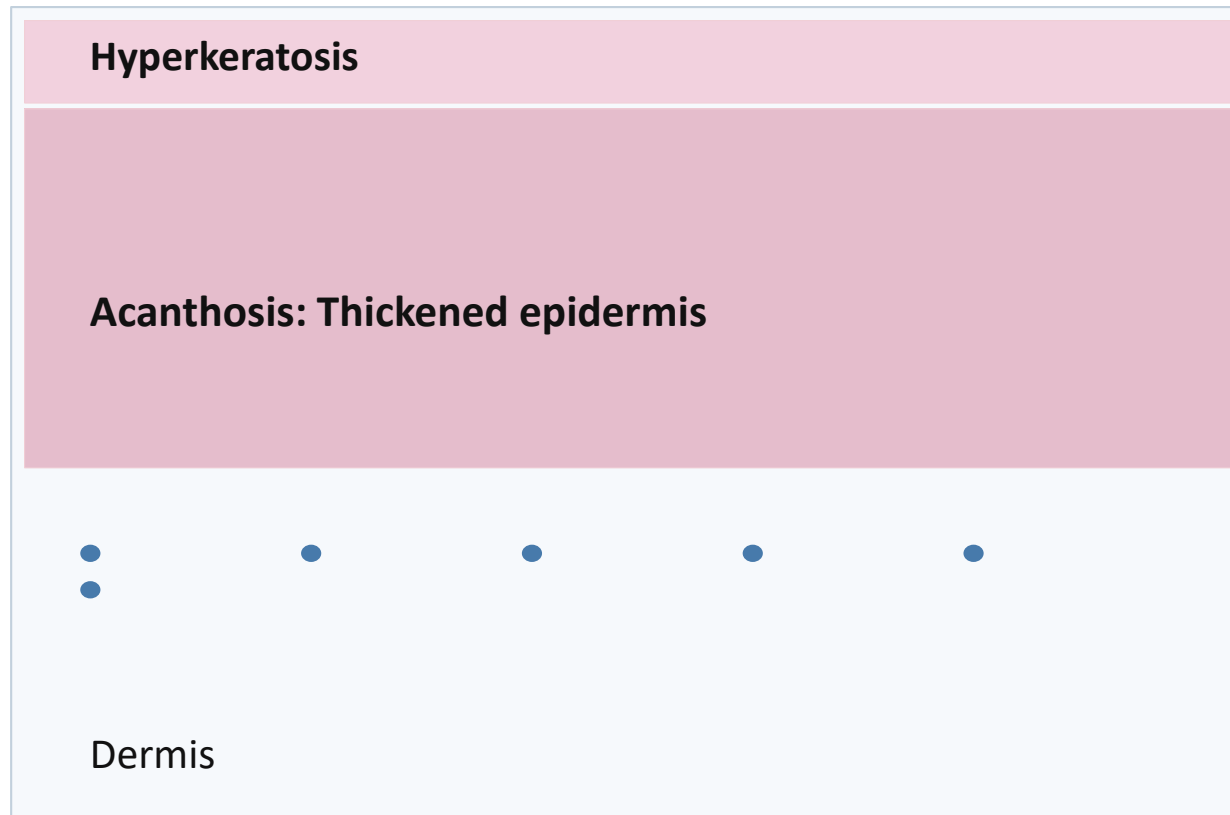
Lichen simplex chronicus: Epidermal acanthosis



- Acanthosis means expansion of the viable squamous epidermis.
- This epidermal thickening contrasts with the thinning of lichen sclerosus.
- Hyperkeratosis overlies the acanthotic epidermis.
- The combination accounts for a thickened plaquelike appearance.

Schematic cross-section — not a photomicrograph

Lichen simplex chronicus: Hyperkeratosis



- Surface keratin is increased in addition to thickening of the epidermis.
- Hyperkeratosis alone does not differentiate the two lichenoid disorders.
- The underlying viable epithelial compartment provides the distinction.
- Look for hyperplasia rather than LS-type atrophy.

Schematic cross-section — not a photomicrograph

Reactive proliferation is not the same as dysplasia

- Lichen simplex chronicus may show mitotic activity in hyperplastic epithelium.
- That this epithelium lacks cellular atypia.
- Proliferation, by itself, is not evidence of intraepithelial neoplasia.
- The presence or absence of atypia alters the pathologic interpretation.

**Mitoses may
occur; Atypia is
absent**

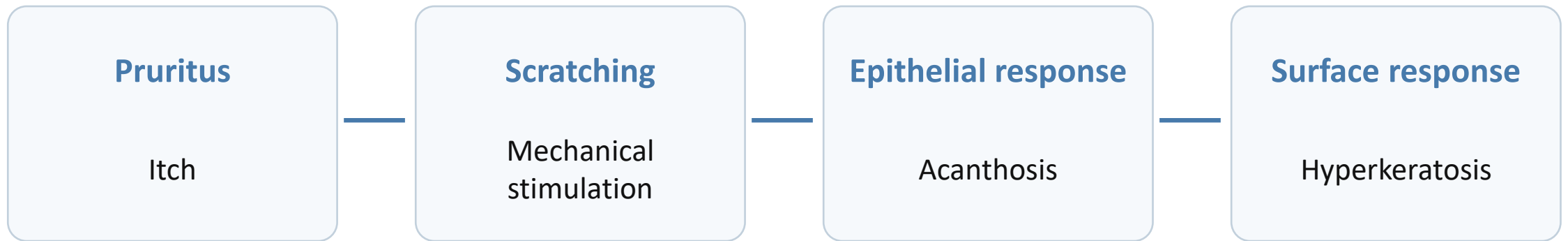
Dermal inflammation in lichen simplex chronicus

- Lymphocytic infiltration of the dermis may be seen.
- It is described as occasional rather than a defining uniform requirement.
- Primary diagnostic emphasis in this source is on acanthosis and hyperkeratosis.
- Do not mistake any dermal lymphocytes for the distinctive LS sclerosis pattern.

Lichen simplex chronicus: Relationship to vulvar cancer

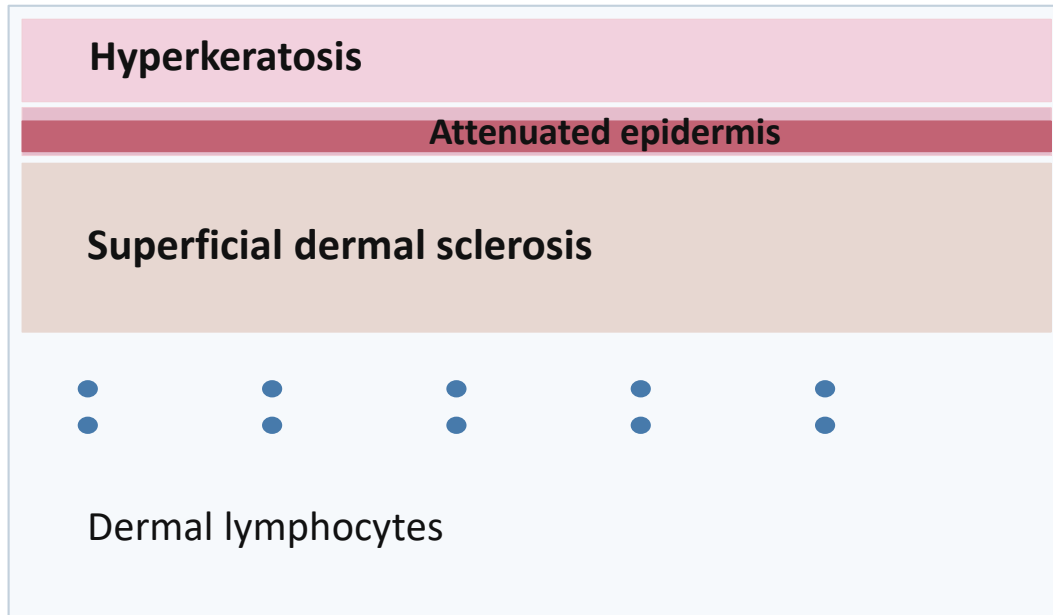
- Lichen simplex chronicus is not itself regarded as premalignant.
- It may be present at the margins of HPV-independent vulvar cancers.
- The adjacent chapter reports an association between long-standing chronic irritation and HPV-independent carcinoma.
- Spatial association is not proof that the sampled hyperplasia is neoplastic.

Translate the LSC mechanism into microscopy



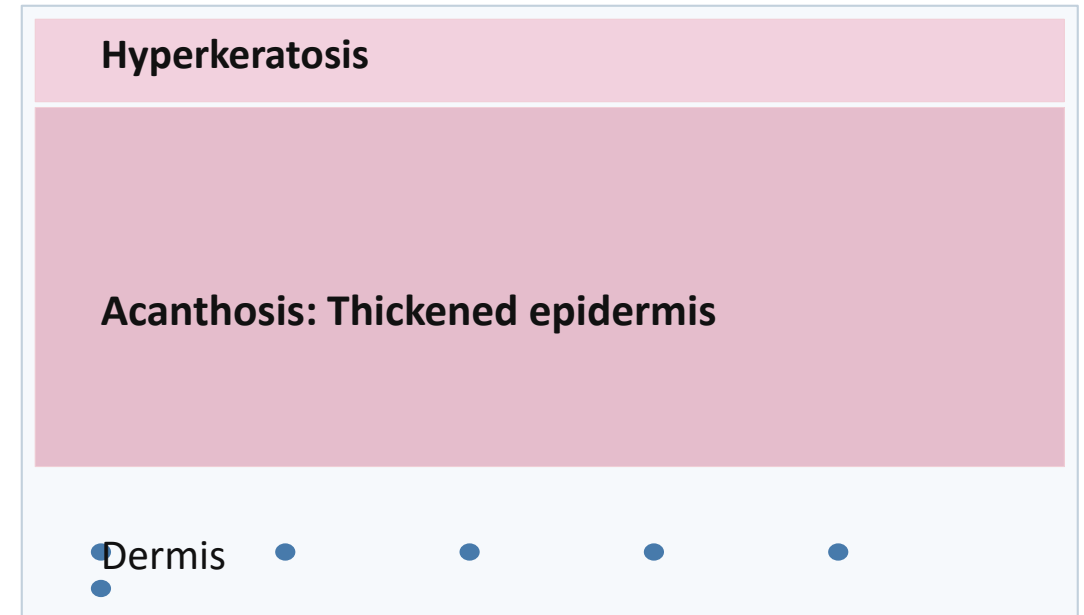
- The epithelial response is reactive and lacks cellular atypia in the described disorder.

Atrophy and hyperplasia: Opposite epidermal patterns



Lichen sclerosis

Thinned epidermis · sclerotic upper dermis



Lichen simplex chronicus

Thickened epidermis · prominent hyperkeratosis

Lichen sclerosis versus lichen simplex chronicus

Compartment	Lichen sclerosis	Lichen simplex chronicus
Surface	Hyperkeratosis	Hyperkeratosis
Epidermis	Marked thinning	Acanthosis
Basal layer	Degenerative change	No cellular atypia stated
Dermis	Sclerosis + bandlike lymphocytes	Sometimes lymphocytes

Clinical discriminators for the white plaque

Question	Favor lichen sclerosis	Favor lichen simplex chronicus
Surface	Smooth / porcelain-like	Plaque resulting from itch–scratch response
Extent	May coalesce and distort anatomy	Rubbing or scratching is defining history
Histology	Thinning plus sclerosis	Thickening plus hyperkeratosis

What the two disorders share — and do not share

Shared

Both may present as white vulvar plaques and show hyperkeratosis.

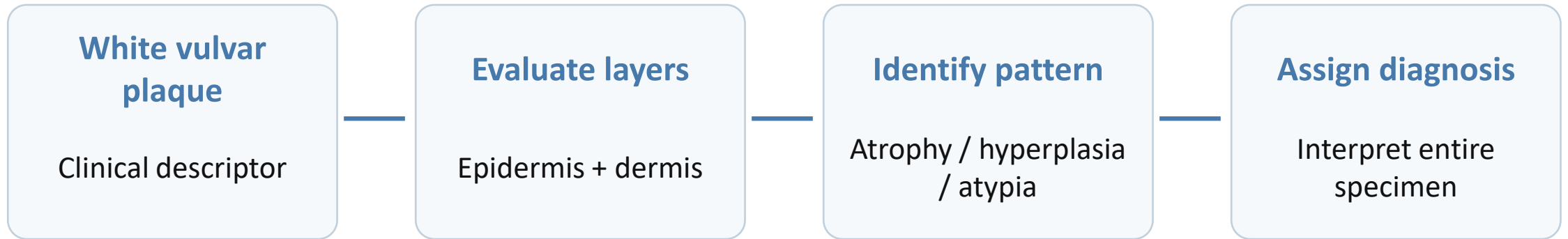
Different

LS attenuates the epidermis;
LSC expands it through
acanthosis.

Risk nuance

Neither is intrinsically premalignant; Both may coexist with HPV-independent carcinoma.

Pattern-based assessment of leukoplakia



- This teaching algorithm summarizes source morphologic contrasts; It is not a clinical practice guideline.

Benign exophytic vulvar lesions: Defining appearance

- Exophytic lesions project outward from the epithelial surface.
- Robbins groups raised or wartlike benign lesions by infectious and reactive/noninfectious origin.
- Clinical “wartlike” appearance alone does not establish HPV causation.
- The grouped examples include skin tags, squamous papillomas, and condylomata.

Benign exophytic lesions: Etiologic categories

Lesion	Described context	What is established
Condyloma acuminatum	Low-risk HPV infection	HPV types 6 and 11 mainly
Condyloma latum	Syphilitic infection	Sexually transmitted lesion
Fibroepithelial polyp	Noninfectious skin-tag analogue	Etiology unknown
Squamous papilloma	Benign squamous proliferation	Etiology unknown

Fibroepithelial polyps: Vulvar skin tags

- Vulvar fibroepithelial polyps are compared with skin tags elsewhere on the body.
- They fall within the noninfectious benign exophytic group.
- The etiology is unknown in the source text.
- Clinical recognition should not conflate a skin tag with an HPV-induced wart.

Squamous papillomas: A distinct benign proliferation

- Squamous papillomas arise as exophytic proliferations on vulvar surfaces.
- They are covered by nonkeratinized squamous epithelium.
- Lesions may occur singly or in multiples.
- Robbins does not assign a specific established cause.

Vulvar papillomatosis does not mean condylomatosis

Squamous papilloma

Benign exophytic epithelium;
May be single or multiple.

Vulvar papillomatosis

Term used when numerous
vulvar squamous papillomas
occur.

Condyloma acuminatum

Papillomavirus-induced
genital warts; Distinct viral
cytopathic features.

Condyloma acuminatum: Low-risk HPV disease

- Condylomata acuminata are benign genital warts caused by HPV infection.
- The major causative types are low-risk HPV 6 and HPV 11.
- The word “low-risk” refers to oncogenic potential, not the degree of visible surface proliferation.
- These lesions are not classified as precancerous.

HPV 6 / HPV 11

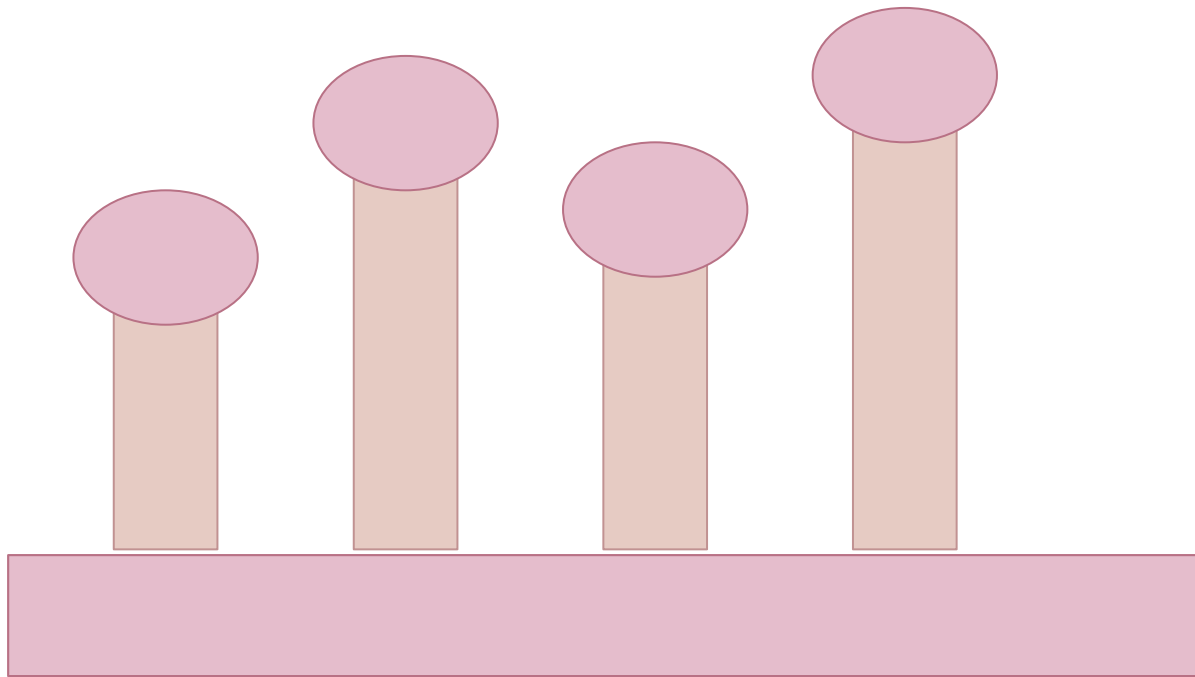
Condyloma: Solitary or multifocal distribution

- A single condyloma may occur, but multifocal disease is more frequent.
- Vulvar, perineal, and perianal regions can be involved.
- Lesions may extend to the vagina and, less commonly, the cervix.
- The same lesion type occurs at male genital and perianal sites.

Condyloma: Low-power histologic architecture

- The lesion is exophytic and papillary rather than a flat leukoplakic plaque.
- Papillary projections contain branching, treelike stromal cores.
- The cores are invested by thickened squamous epithelium.
- Low-power architecture is complemented by high-power viral cytopathic findings.

Condyloma: Papillary projections with stromal cores



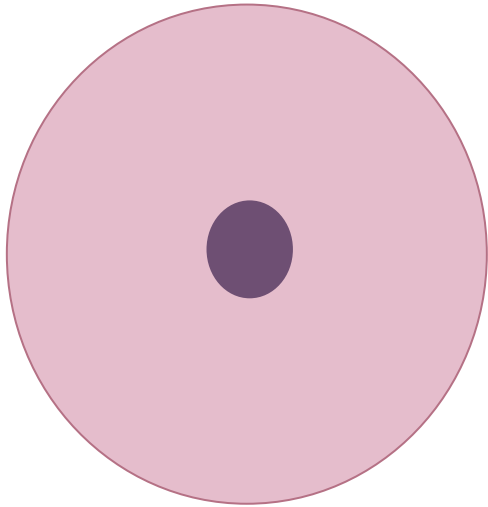
Schematic: Papillary projections with stromal cores and squamous covering

- Treelike stroma forms the central framework of papillary outgrowths.
- Proliferating squamous epithelium covers each projection.
- The schematic shows architecture, not a patient specimen.
- Koilocytic changes are evaluated in the epithelial covering.

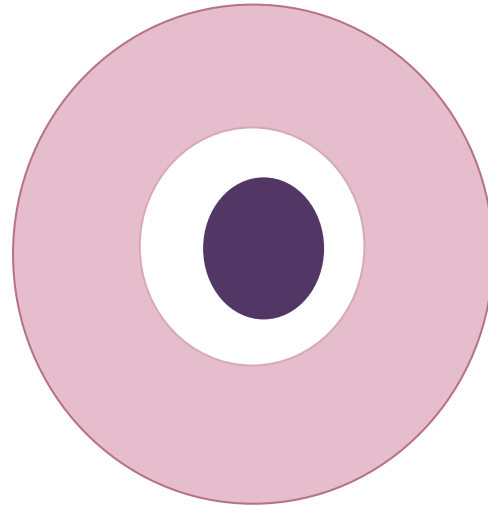
Koilocytic atypia: HPV-related cytopathic effect

- Koilocytic atypia is the characteristic cytopathic change named.
- The nucleus is enlarged and hyperchromatic.
- A perinuclear halo is seen within the cytoplasm.
- This viral effect explains the term “koilocytic,” but is not synonymous with invasive carcinoma.

Koilocyte morphology: Nucleus and perinuclear halo



Conventional nucleus



Koilocytic viral effect

Illustrative cytologic features; Not diagnostic microscopy

- Enlargement is a change in nuclear size.
- Hyperchromasia refers to increased nuclear staining intensity.
- Perinuclear clearing produces a pale cytoplasmic halo.
- Assess these features together with papillary lesion architecture.

Condyloma does not equal a precancerous lesion

- Condylomata acuminata are not precancerous.
- The associated HPV types are low-risk, principally 6 and 11.
- This is conceptually separate from high-risk HPV-associated vulvar intraepithelial neoplasia.
- Do not infer invasive potential from the mere presence of the term “atypia” in koilocytic atypia.

HPV vaccination and genital warts

- HPV vaccines provide excellent protection against infection by low-risk HPV and genital warts.
- The statement concerns prevention of HPV infection and its associated benign exophytic disease.
- No vaccine schedule or efficacy percentage is supplied in the source excerpt.
- Keep preventive implications distinct from histologic diagnosis of an existing wart.

Do not collapse all genital condylomata into HPV warts

Term	Etiology	Instructional distinction
Condyloma acuminatum	Papillomavirus (mainly HPV 6, 11)	Benign genital wart; Koilocytic effect
Condyloma latum	Syphilitic infection	Separate sexually transmitted etiology
Squamous papilloma	Unknown	Benign nonkeratinized squamous covering

Exophytic lesion comparison: Three discriminators

Finding	Condyloma acuminatum	Squamous papilloma / skin tag
Underlying category	Infectious HPV-associated	Noninfectious group; Cause unknown
Described epithelium	Thick squamous covering with koilocytosis	Squamous papilloma: Nonkeratinized covering
Configuration	Papillary, often multifocal	Papilloma may be solitary or numerous

A patterned differential is stronger than a color label

- White plaques: Begin with lichen sclerosus, lichen simplex chronicus, other dermatoses, and neoplasia.
- Raised papillary lesions: Consider condyloma acuminatum, squamous papillomas, and skin tags.
- Epidermal thinning, epidermal thickening, and viral koilocytosis represent different morphologic clues.
- Interpret neoplastic risk with the particular histologic entity rather than the word “leukoplakia.”

Clinical case A: White plaques and architectural change

An older patient has smooth white vulvar plaques that have gradually merged. Examination notes atrophic changes and adhesions.

- Which nonneoplastic vulvar disorder best fits this clinical pattern?
- Which two microscopic tissue compartments would you inspect first?
- What is the correct way to describe its association with vulvar carcinoma?

Case A: Explanation

Most consistent with lichen sclerosis, based on the smooth coalescent white plaques and architectural distortion.

- Expected tissue findings: Epidermal attenuation/basal injury; Superficial dermal sclerosis with lymphocytic inflammation.
- Lichen sclerosis is not itself premalignant.
- An association with a slightly increased chance of HPV-independent vulvar carcinoma is nevertheless described.

Clinical case B: Pruritus and thickened epithelium

A patient with persistent vulvar itching frequently rubs and scratches the area. A white plaque is sampled. The squamous epithelium is thickened and hyperkeratotic.

- What process explains the increased epithelial thickness?
- Which microscopic finding would support a reactive, rather than atypical, squamous proliferation?
- How does this pattern contrast with lichen sclerosis?

Case B: Explanation

This is a typical instructional description of lichen simplex chronicus: Reactive hyperplasia arising in the itch–scratch setting.

- Acanthosis and hyperkeratosis account for epithelial thickening.
- Mitoses may be present, but cellular atypia is absent in the source description.
- Lichen sclerosus instead features a thinned epidermis and superficial dermal sclerosis.

Clinical case C: Multifocal papillary vulvar lesions

Multiple outward-projecting vulvar lesions are sampled. Each has branching stromal cores covered by thickened squamous epithelium. Nuclei are enlarged with cytoplasmic halos.

- Which named vulvar lesion has this architecture?
- What viral types principally cause it?
- Is this particular lesion described as precancerous?

Case C: Explanation

The architecture and cytopathic changes are characteristic of condyloma acuminatum.

- Low-risk HPV 6 and 11 account for most such lesions in the source.
- Koilocytic atypia includes enlarged hyperchromatic nuclei and perinuclear clearing.
- Robbins explicitly distinguishes these benign genital warts from precancerous lesions.

Frequent interpretive errors

- Error: Leukoplakia is a histologic diagnosis. Correction: It is only a white-plaque description.
- Error: Hyperkeratosis proves lichen simplex chronicus. Correction: Lichen sclerosus may also be hyperkeratotic.
- Error: LS or LSC is itself carcinoma in situ. Correction: Neither condition is intrinsically premalignant.
- Error: Koilocytic “atypia” means condyloma is precancerous. Correction: HPV 6/11 warts are benign.

Summary: Diagnose by pattern, not label

Entity	Most discriminating features	Oncologic interpretation
Lichen sclerosus	Thin epidermis; Basal injury; Superficial dermal sclerosis	Not premalignant; Slight HPV-independent SCC association
Lichen simplex chronicus	Itch–scratch; Acanthosis + hyperkeratosis; No atypia	Not premalignant; May border SCC
Condyloma acuminatum	Exophytic papillae; Koilocytosis; Low-risk HPV 6/11	Not precancerous

Integrated clinicopathologic synthesis

Describe

Separate white flat plaques from papillary exophytic lesions.

Explain

Relate atrophy/sclerosis, reactive acanthosis, and viral cytopathic change to mechanism.

Classify

Preserve the distinction between nonneoplastic disease, tumor precursors, and invasive tumors.

Primary reference and source boundaries

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th edition (2026), Chapter 22: The Female Genital Tract, pages 899–901.
- Main passage: nonneoplastic epithelial disorders and adjacent lichen sclerosus, lichen simplex chronicus, benign exophytic lesions, and condyloma acuminatum.
- Contextual comparison: vulvar squamous neoplastic lesions in the following section.
- All tissue drawings are editable schematics, not Robbins figures or authentic photomicrographs.