

Oral Candidiasis (Thrush)

Host susceptibility • mucosal morphology • clinicopathologic diagnosis

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Learning objectives

- Distinguish oral colonization from candidal mucosal infection.
- Recognize pseudomembranous, erythematous, and hyperplastic patterns.
- Connect host risk, clinical morphology, and diagnostic decisions.
- Differentiate thrush from non-scrapable lesions and assess possible extension.

A commensal organism can become a pathogen

- Candida albicans is frequently present without symptomatic infection.
- The same organism may produce superficial mucosal disease.
- Clinical disease reflects interaction between fungus and oral host milieu.
- Positive microbiology alone is not synonymous with candidiasis.

Normal oral carriage is common

- C. Albicans as oral flora in approximately 50% of people.
- Carriage describes organism presence, not invasive or symptomatic disease.
- Interpret a positive swab in the context of visible lesions and symptoms.

Colonization-to-infection continuum



The clinical threshold is defined by compatible mucosal disease, not by recovering *Candida* alone.

Three determinants of infection

Fungal factor

C. Albicans strain

Virulence varies among strains

Ecologic factor

Oral microbiome composition

Antibiotics change competition

Host factor

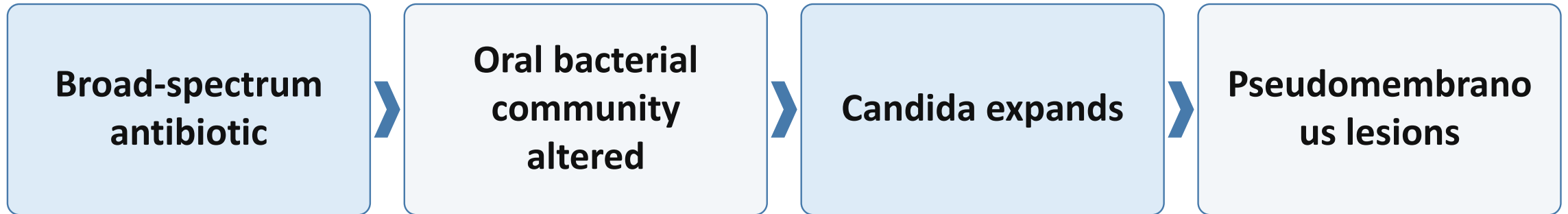
Immune status

Deficient protection increases susceptibility

Why oral bacterial flora matters

- A mixed bacterial community normally shares the oral mucosal niche.
- Broad-spectrum antibiotics can eliminate or alter bacterial flora.
- The altered ecosystem can permit a candidal bloom and thrush.

Antibiotic-associated thrush: Causal sequence



The causal sequence explains why fungal disease may follow antibacterial therapy.

Strain-dependent susceptibility

- The C. Albicans strain as a disease determinant.
- Organism identity alone does not determine clinical severity.
- Host and community factors modify whether infection manifests.

Immunosuppression removes a key restraint

- Immunosuppression as the principal context for deeper risk.
- Oral mucosal candidiasis ordinarily remains superficial.
- Immune compromise can increase persistence and disease burden.

Important high-risk clinical settings

Transplantation

Solid-organ transplant

Hematopoietic stem-cell
transplant

Hematologic / oncologic

Neutropenia

Chemotherapy-related
immunosuppression

Systemic disease

AIDS / advanced HIV

Diabetes mellitus

Transplant-associated risk

- Both organ and hematopoietic stem-cell transplantation are named.
- Immune suppression after transplantation creates a susceptible host.
- Examine new white mucosal lesions rather than assuming they are nonspecific.

Neutropenia and candidiasis

- Neutropenia is explicitly identified as a predisposing clinical condition.
- Reduced innate defenses permit opportunistic fungal infection.
- In neutropenic patients, oral lesions require attention to overall clinical context.

Chemotherapy-associated susceptibility

- Chemotherapy can produce immunosuppression and mucosal vulnerability.
- A new coating or erythematous lesion should trigger clinical assessment.
- Severe host immunosuppression alters the concern for extension beyond the mouth.

AIDS and oral candidiasis

- AIDS among the causes of increased susceptibility.
- NIH recognizes oral/esophageal candidiasis as manifestations of immune suppression in HIV.
- The lesion warrants attention to immune status and associated symptoms.

Diabetes as a host factor

- Diabetes is explicitly included among Robbins risk conditions.
- In a patient with recurrent or atypical thrush, systemic context matters.
- Candida presence without disease still requires careful interpretation.

Local predisposition: Beyond the Robbins core

- DermNet notes dentures and inhaled corticosteroids as promoting conditions.
- Other local modifiers include smoking and oral dryness.
- Risk assessment should combine systemic immunity and local mucosal ecology.

Systemic and local susceptibility

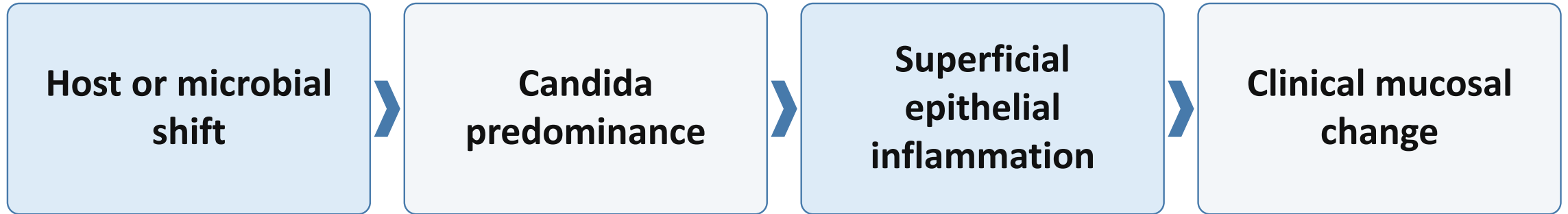
Systemic susceptibility

- HIV-associated immune suppression
- Neutropenia or chemotherapy
- Organ or stem-cell transplantation
- Diabetes mellitus

Local and ecological susceptibility

- Recent broad-spectrum antibiotics
- Dentures or altered local environment
- Inhaled corticosteroids
- Dryness and smoking

From altered defenses to a visible lesion



A white membrane is one possible clinical expression; Erythema or hyperplasia can predominate.

Three major mucosal forms

Pseudomembranous

Gray-white superficial membrane

Typically removable

Erythematous

Predominantly red mucosal change

Plaque may be absent

Hyperplastic

Persistent white plaque

Non-scrapable pattern

Thrush means pseudomembranous disease

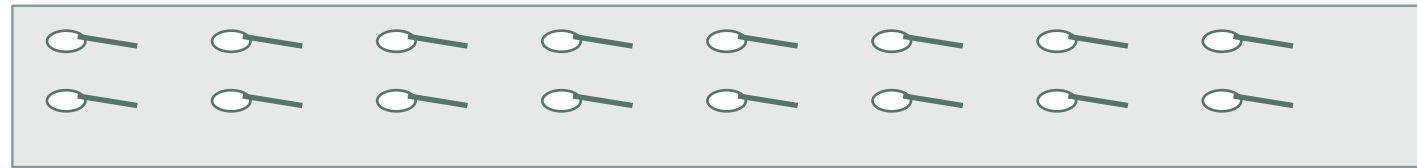
- A superficial gray-to-white inflammatory membrane is characteristic.
- Lesions represent fungal organisms admixed with inflammatory exudate.
- The membrane is typically easy to scrape from the underlying mucosa.

The pseudomembrane is a composite

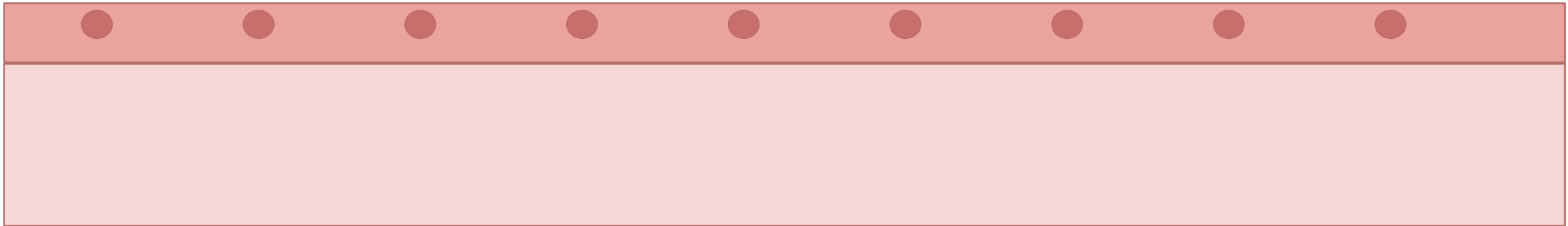
- Matted fungal organisms.
- They are enmeshed in fibrinosuppurative exudate.
- The gray-white coating is inflammatory material—not simply keratin.

Pseudomembrane architecture: Schematic

Fungi + fibrinopurulent exudate



Detachable surface layer



Erythematous inflamed mucosal surface

Schematic cross-section — not a histologic specimen

Why removal reveals an erythematous base

- The superficial layer can be detached from the inflamed mucosal surface.
- After removal, an erythematous inflammatory base is exposed.
- This surface response helps distinguish thrush from adherent keratotic plaques.

Where pseudomembranes may occur

- NIH describes lesions on the tongue and buccal mucosa.
- The hard palate, soft palate, gums, and oropharynx may also be involved.
- Anatomic distribution alone is less discriminating than the characteristic removable coating.

Removable vs persistent white plaques

Pseudomembranous Candida

- Creamy or gray-white surface material
- Scrapes off in many cases
- Inflamed mucosa may be exposed
- Confirm risk factors and symptoms

Non-removable plaque

- Persistent white lesion
- Consider hyperplastic Candida or other causes
- Hairy leukoplakia is an important alternative
- Persistent unexplained lesions need evaluation

Erythematous oral candidiasis

- This is a recognized form, even without a white membrane.
- Clinical appearance may be dominated by smooth red mucosal areas.
- Tenderness or burning may be prominent in symptomatic patients.

An atrophic red pattern may follow antibiotics

- DermNet describes acute atrophic candidiasis with red shiny patches.
- A sore tongue after antibiotic use is a meaningful clinical clue.
- Do not require a visible white pseudomembrane for every diagnosis.

Denture-associated mucosal erythema

- Chronic atrophic candidiasis can occur under dentures.
- The denture-bearing mucosa may be erythematous and inflamed.
- Assess the prosthesis, hygiene, and other coexisting local factors.

Median rhomboid glossitis

- DermNet includes a central posterior-tongue erythematous pattern.
- It appears as a circumscribed, often diamond-shaped area.
- Interpret the finding in clinical context; Not all tongue erythema proves Candida.

Angular cheilitis: An associated pattern

- Inflammation and fissuring affect one or both oral commissures.
- Candida can contribute, sometimes with bacterial coinfection.
- Local saliva-related irritation can contribute independently.

Hyperplastic candidiasis

- A hyperplastic form of oral candidiasis.
- The lesion may produce a chronic, non-scrapable white plaque.
- Persistent lesions require evaluation for another keratotic disorder or dysplasia.

Three forms: Morphology and bedside clue

Form	Dominant appearance	Practical discriminator
Pseudomembranous	White / gray coating	Often wipes away
Erythematous	Red smooth area	May lack white plaque
Hyperplastic	Persistent white plaque	Does not scrape away

Symptoms can be variable

- NIH describes many white oral lesions as painless.
- Erythematous disease may cause soreness or a burning sensation.
- Symptoms alone cannot establish the subtype or exclude another diagnosis.

Why immunologic context changes interpretation

- Superficial Candida in a healthy host usually has limited anatomic extent.
- In marked immunosuppression, infection can be more persistent.
- Screen for swallowing symptoms that suggest esophageal involvement.

Oropharyngeal vs esophageal candidiasis

- Oropharyngeal disease causes oral or pharyngeal mucosal lesions.
- Esophageal involvement may cause odynophagia and dysphagia.
- Esophageal disease can occur even when oral thrush is absent.

Oropharyngeal and esophageal patterns

Oropharyngeal candidiasis

- Typical removable surface plaques
- Tongue, buccal mucosa, palate
- Clinical diagnosis often sufficient
- Topical or systemic therapy by severity

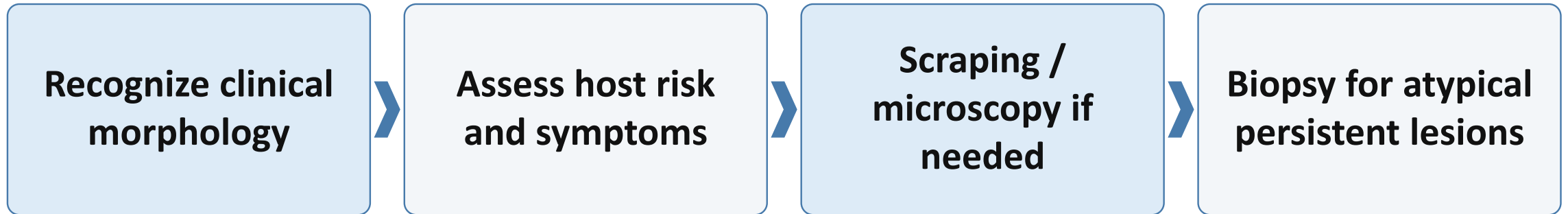
Esophageal candidiasis

- Retrosternal pain or burning
- Odynophagia / dysphagia
- May occur without oral lesions
- Requires systemic antifungal therapy

Oral findings can signal immune dysfunction

- In people with HIV, thrush often occurs with significant immune suppression.
- NIH reports that oral or esophageal candidiasis is most often seen with CD4 counts below 200 cells/mm³.
- A single oral lesion does not independently quantify immune function.

A lesion-to-laboratory diagnostic sequence



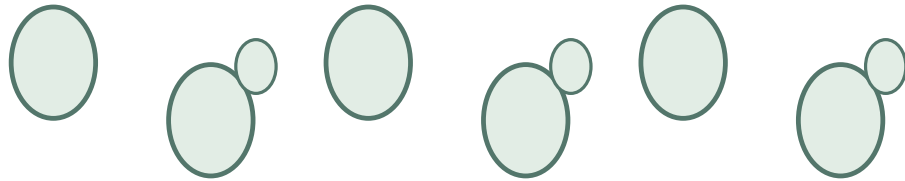
The decision to test depends on uncertainty, severity, treatment response, and alternate diagnoses.

Clinical diagnosis is usually the starting point

- Typical removable plaques support a clinical diagnosis of thrush.
- Look for an erythematous base and compatible susceptibility factors.
- Consider alternatives if the lesion is atypical or does not respond as expected.

Yeast and filamentous forms: Schematic

Conceptual fungal forms examined in an oral scraping



Budding yeast forms



Elongated filamentous forms

Educational schematic: Demonstration of fungal forms must be interpreted with the clinical lesion.

Direct microscopy and potassium hydroxide

- An oral scraping can be examined by potassium hydroxide preparation.
- Characteristic budding yeasts and filamentous forms support the diagnosis.
- Correlate with visible disease, because colonization can also occur.

Culture: Useful but not diagnostic in isolation

- Culture can identify the Candida species recovered.
- Because Candida may colonize normal mucosa, a positive culture is not enough.
- Culture or further testing is useful when the presentation or response is atypical.

Tissue biopsy: When the pattern is discordant

- Persistent non-scrapable plaques merit evaluation for other pathology.
- Biopsy may be needed when leukoplakia or dysplasia is a concern.
- Tissue interpretation must distinguish surface colonization from lesion causation.

Histologic principle: Organisms plus tissue response

- Fungal forms and surface inflammatory change may coexist.
- Inflammatory exudate explains the pseudomembranous coating.
- Pathology is most persuasive when organisms and lesional inflammation fit the clinical presentation.

White mucosal lesions: Diagnostic contrast

Feature	Thrush	Oral hairy leukoplakia
Surface	Creamy removable material	Hairy / corrugated keratosis
Scraping	Often removable	Not scrapable
Etiology	Candida-associated	Epstein–Barr virus
Clinical context	Various susceptible hosts	Often immunocompromised

Oral hairy leukoplakia is not thrush

- Hairy leukoplakia to Epstein–Barr virus.
- It typically involves the lateral border of the tongue.
- The lesion does not scrape off, unlike classic pseudomembranous Candida.
- Candida may secondarily coexist, complicating surface appearance.

Persistent leukoplakia is a different problem

- Leukoplakia is a clinical designation for a persistent white plaque requiring exclusion of other causes.
- One cannot label all non-scrapable plaques as chronic Candida.
- Assess for dysplasia or neoplasia when clinically warranted.

Candida infection vs herpes infection

Oral candidiasis

- White pseudomembrane or erythema
- Many plaques wipe off
- Fungal forms may be found
- Strongly shaped by host factors

Oral herpes simplex

- Vesicles and subsequent ulcers
- Ulcerative rather than fungal plaques
- Often painful primary stomatitis
- Viral process; Different workup

Broad oral differential: Pattern first

- Scrapable white coating: Consider pseudomembranous Candida.
- Persistent white plaques: Assess hairy leukoplakia and leukoplakia.
- Predominantly red lesions: Consider erythematous Candida and other mucosal disorders.
- Discrete vesicles or ulcers: Investigate nonfungal etiologies.

When oral white plaques are found

1. Inspect distribution, texture, and adjacent mucosa

2. Determine whether the surface plaque is removable

3. Review antibiotics, immune suppression, diabetes, and local modifiers

4. If atypical, persistent, or treatment-refractory, obtain further evaluation

5. Ask about swallowing pain, dysphagia, and substernal discomfort

Mild oropharyngeal disease: Local therapy

- IDSA recommends topical approaches for mild disease.
- Examples include clotrimazole troches and miconazole buccal tablets.
- Nystatin suspension or pastilles are alternative topical options.
- Treatment selection depends on patient factors and local availability.

Moderate-to-severe disease: Systemic therapy

- IDSA recommends oral fluconazole for moderate-to-severe oropharyngeal candidiasis.
- The guideline distinguishes this from topical management of mild disease.
- Drug interactions, tolerability, and underlying conditions require clinical review.

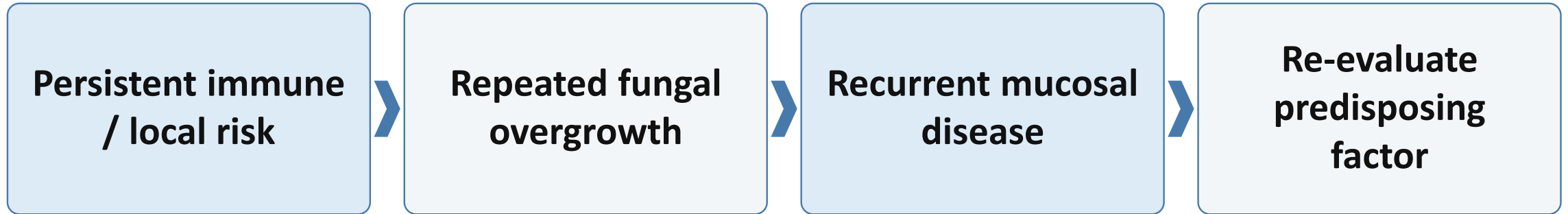
Esophageal symptoms alter treatment requirements

- Esophageal candidiasis requires systemic antifungal therapy.
- Oral-only topical regimens are not appropriate for esophageal infection.
- Assess dysphagia or odynophagia before assuming disease is restricted to the mouth.

Refractory disease requires reassessment

- Re-evaluate diagnosis, adherence, and immune or local predisposing factors.
- Non-albicans species and reduced azole susceptibility may contribute.
- IDSA provides alternative systemic agents for fluconazole-refractory disease.

Why candidiasis recurs



Symptom treatment without addressing major susceptibility factors can be followed by recurrence.

Local risk modification

- Review denture hygiene and fit when a prosthesis is involved.
- Assess inhaled corticosteroid technique and local oral care.
- Use antibacterial agents appropriately; Unnecessary spectrum may perturb flora.
- Evaluate relevant systemic conditions when infection is recurrent.

Deep fungal infections: Adjacent Robbins scope

- Robbins groups several deeper fungal infections under head-and-neck disease.
- Named agents include Histoplasma, Blastomyces, Coccidioides, Cryptococcus, mucormycosis, and aspergillosis.
- These infections are distinct from routine superficial oral thrush.

When the oral fungal differential broadens

- Marked immunocompromise increases concern for unusual fungal disease.
- Atypical ulcerating or destructive lesions should not be assumed to be Candida.
- Escalate diagnostic investigation when morphology or course is discordant.

Case 1: White coating after antibiotics

A patient develops removable creamy plaques on the tongue shortly after a course of broad-spectrum antibiotics. Red mucosa is visible after gentle removal.

Discuss

- Identify the most likely candidal form.
- Explain why antibiotics could be a trigger.
- Explain why a positive swab alone is insufficient.

Case 2: Transplantation and painful swallowing

A transplant recipient has oral plaques and new odynophagia, together with a markedly immunosuppressed clinical state.

Discuss

- Which symptom raises concern for esophageal disease?
- Why is management different from mild, isolated oral thrush?
- Which host factor is central to the presentation?

Case 3: A white plaque that cannot be removed

An immunocompromised patient has a persistent corrugated white plaque along the lateral tongue; Gentle scraping does not remove it.

Discuss

- What competing diagnosis deserves attention?
- Which bedside feature opposes classic thrush?
- Why is the label “Candida” premature?

Clinicopathologic synthesis

- Candida can be commensal: Distinguish carriage from disease.
- The pseudomembrane contains fungi and inflammatory exudate.
- Scrapability, mucosal response, and host risk guide recognition.
- Systemic symptoms and atypical persistence demand further evaluation.

Selected references

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DermNet. Oral candidiasis; Candidiasis pathology; Angular cheilitis.