

Colitis-Associated Neoplasia

Dysplasia, cancer risk, surveillance, and clinicopathologic decisions

Dr Abu Suraih Sakhri

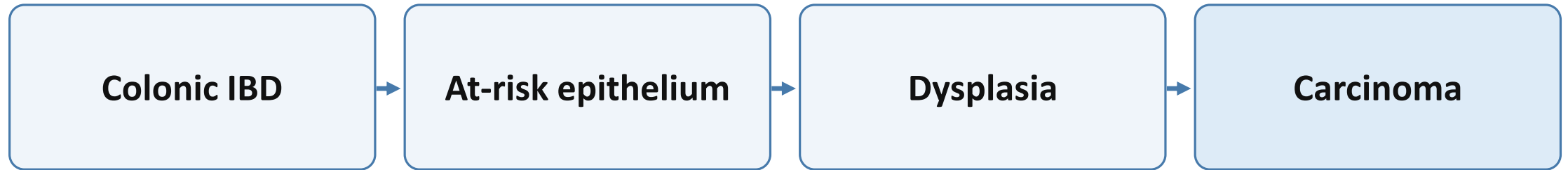
MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning objectives

- Identify which IBD populations have increased colorectal neoplasia risk.
- Explain how duration, extent, inflammation, and PSC alter surveillance.
- Recognize dysplasia categories and consequential diagnostic pitfalls.
- Relate lesion visibility and histologic grade to management.

Colitis-associated neoplasia: The core concept

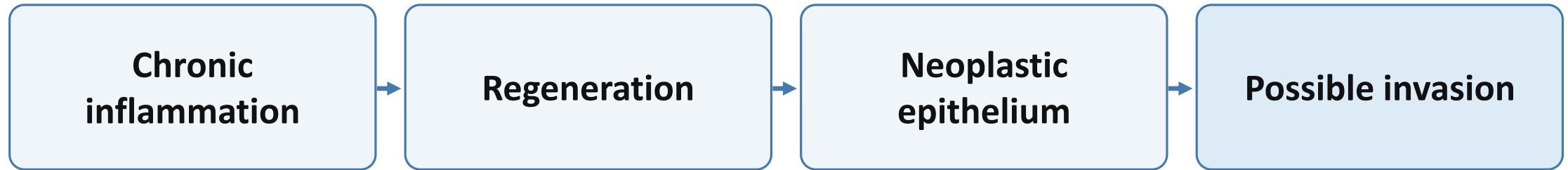


- Chronic colonic IBD increases the risk of dysplasia and colorectal carcinoma.
- Dysplasia is a neoplastic epithelial precursor without invasive growth.
- Risk is not uniform: Disease distribution and inflammatory history matter.

Which IBD patients are relevant?

Colonic disease	Relevance
Ulcerative colitis	At-risk group
Crohn colitis	At-risk group
Crohn without colitis	No increased risk via colitis pathway

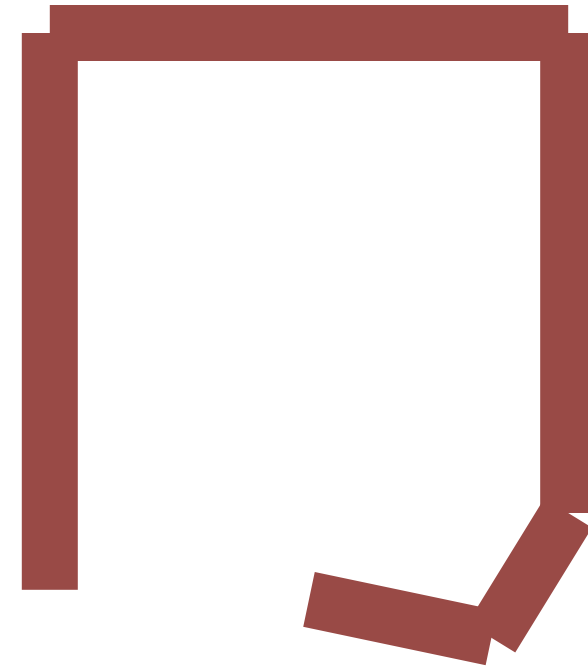
Normal colon, inflamed colon, dysplasia



- Colonic epithelial injury and regeneration provide the tissue context.
- Dysplasia describes true neoplasia—not merely active inflammation.
- Invasive carcinoma is a separate diagnostic state.

Ulcerative colitis: Extent is continuous

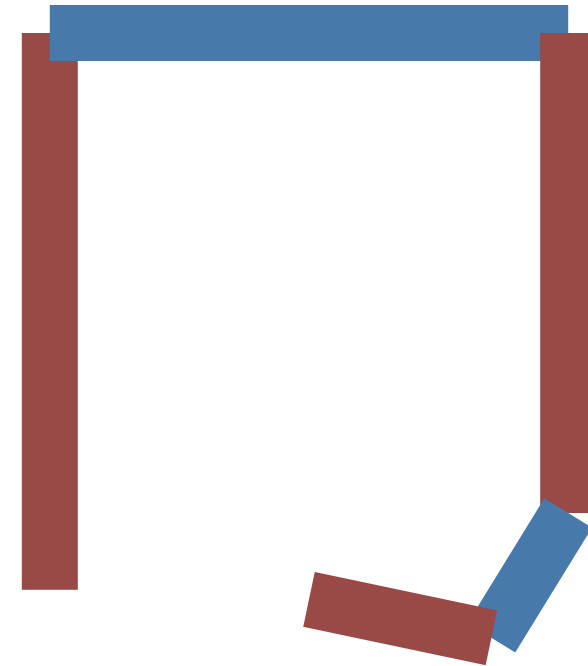
- UC generally starts in the rectum and extends proximally without skip lesions.
- Left-sided colitis and pancolitis differ in the amount of at-risk mucosa.
- Pancolitis carries higher neoplasia risk than left-sided disease.



Simplified distribution: Extensive colon involvement

Crohn colitis: Colonic involvement is decisive

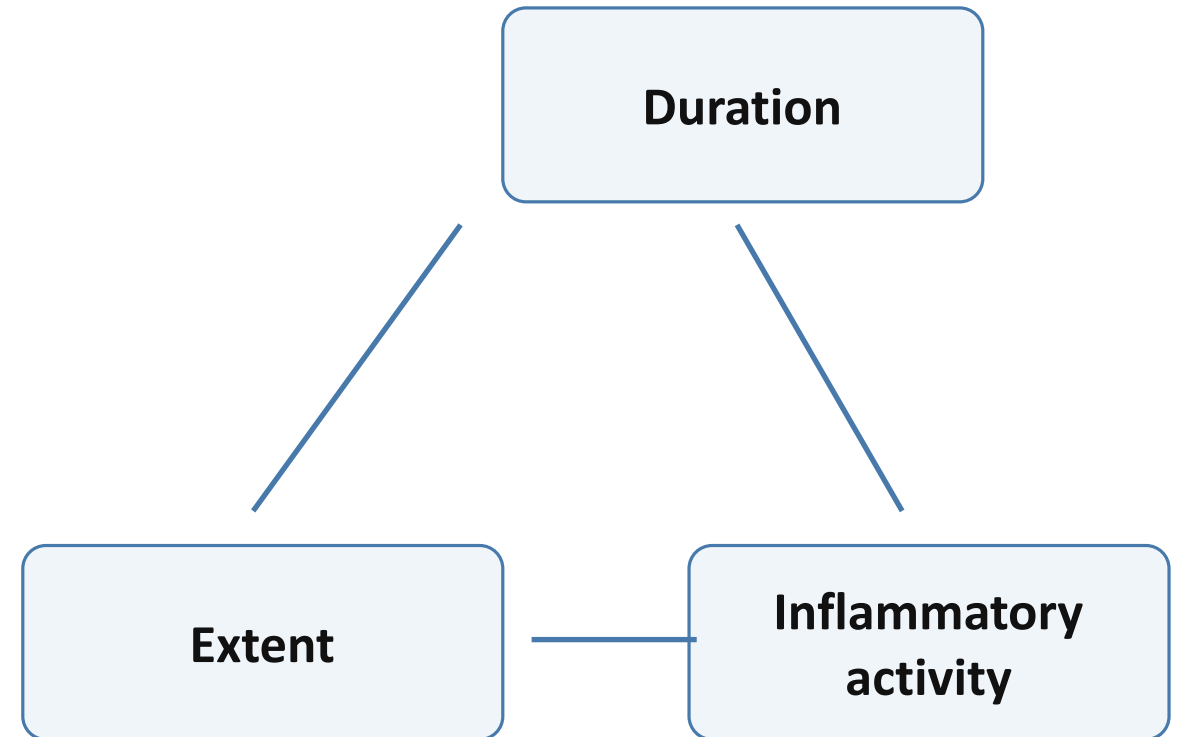
- Crohn disease can involve small and large bowel in discontinuous segments.
- The neoplasia risk statement concerns Crohn disease with colonic involvement.
- Do not extrapolate the same colon risk to isolated ileal disease.



Simplified distribution: Patchy Crohn colitis

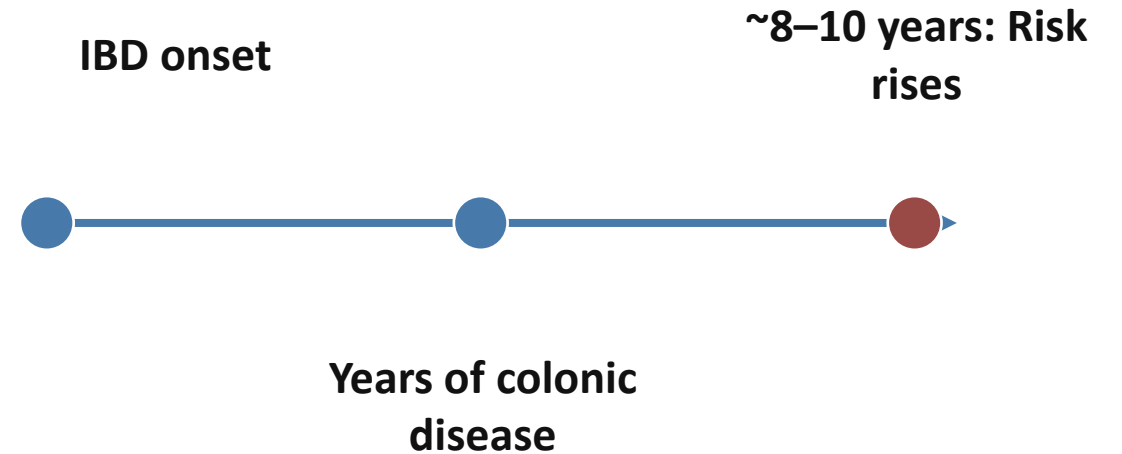
The three major risk dimensions

- Longer disease duration increases cumulative exposure.
- Greater colonic extent increases tissue at risk.
- More frequent and severe active inflammation increases risk.



Disease duration: The late complication

- A marked risk increase after approximately 8–10 years.
- Duration refers to the history of IBD, not merely current symptoms.
- A new episode of active symptoms does not reset the disease clock.



Extent: Pancolitis versus left-sided colitis

Disease distribution	Textbook risk relationship
Pancolitis	Greater relative risk
Left-sided colitis	Lower than pancolitis
Isolated proctitis	No routine IBD-specific program if truly confined to rectum (ECCO)

Active inflammation is a risk signal

- The presence of neutrophils characterizes histologically active inflammation.
- More frequent and more severe inflammatory episodes confer greater risk.
- Distinguish activity from chronic architectural injury on biopsy.

Neutrophilic activity



Repeated mucosal injury



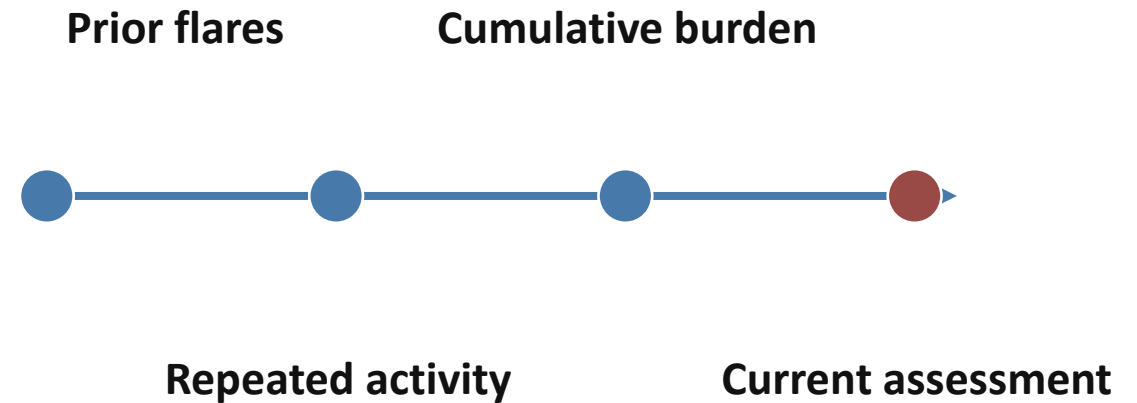
Higher neoplasia risk

Chronicity and activity are not synonyms

Feature	Histologic implication
Architectural distortion	Chronic mucosal injury
Cryptitis / crypt abscess	Current active inflammation
Dysplastic atypia	Neoplastic transformation

Inflammation burden is longitudinal

- Risk assessment considers how often inflammation has recurred.
- Histologic severity across examinations can be clinically informative.
- A single remission biopsy cannot summarize the entire disease course.



Primary sclerosing cholangitis changes risk

- Concurrent primary sclerosing cholangitis (PSC) identifies a higher-risk subgroup.
- Surveillance beginning at diagnosis for IBD with PSC.
- ECCO recommends annual surveillance following PSC diagnosis.

**Requires individualized
clinical action**

PSC: Do not apply the standard waiting period

Clinical context	Screening / surveillance start
Colonic IBD, no PSC	About 8 years after onset/diagnosis
Colonic IBD plus PSC	At PSC diagnosis

A patient-specific risk synthesis

- Record disease duration and historical extent.
- Review endoscopic and histologic inflammation burden.
- Identify PSC, prior dysplasia, stricture, and relevant family history for modern risk stratification.

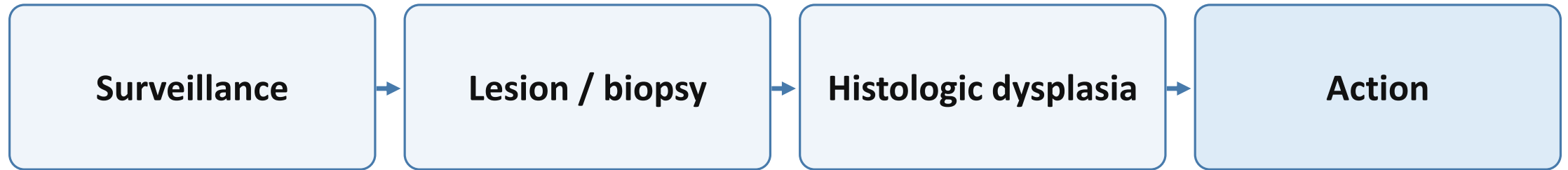
Duration

Extent

Inflammation

High-risk modifiers

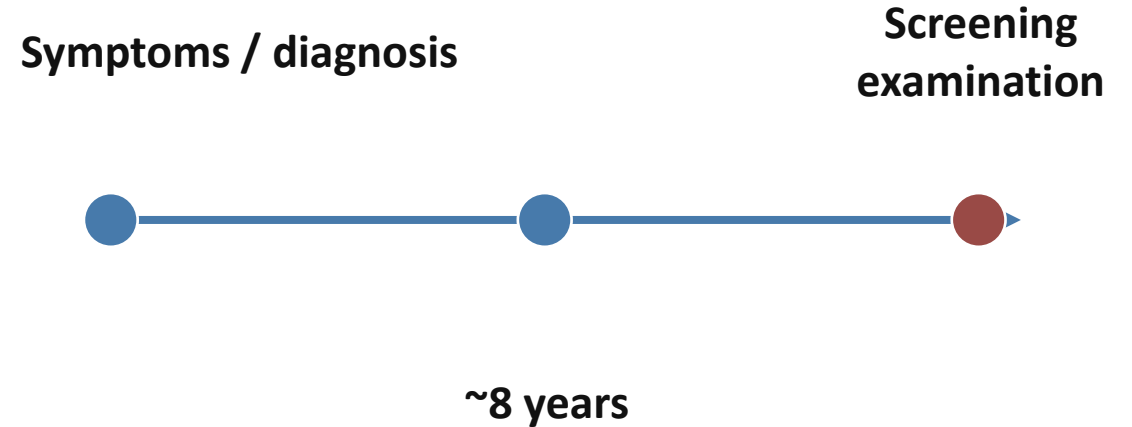
Why surveillance targets dysplasia



- Dysplasia precedes many colitis-associated carcinomas.
- Detecting a precursor may allow intervention before invasion.
- Biopsies must undergo histologic assessment to establish dysplasia.

The baseline surveillance timeline

- Robbins: Surveillance is generally initiated about 8 years after IBD diagnosis.
- ECCO: Screening colonoscopy 8 years after first symptoms to assess extent and dysplasia.
- The differing anchors should not be silently treated as identical.



Surveillance intervals depend on risk

- AGA advises repeat surveillance every 1–5 years after a negative examination.
- ECCO uses shorter intervals when high-risk features are present.
- The exact interval should be individualized using guideline-defined risk factors.

Lower risk

Longer reassessment interval

Intermediate risk

Intermediate interval

Higher risk

Shorter surveillance interval

Examine when inflammation is controlled

- Active inflammation obscures subtle lesions.
- Inflammation also complicates interpretation of reactive versus dysplastic atypia.
- ECCO recommends performing surveillance when IBD is in remission where feasible.

Better disease control



Clearer mucosal inspection



More reliable histology

High-definition colonoscopy changes detection

- Careful mucosal inspection is central to dysplasia detection.
- High-definition endoscopes reveal subtle surface and vascular differences.
- Histology remains necessary to classify suspected dysplasia.

Inspection → targeted tissue → histology

Three endoscopic visibility categories

Category	Clinical interpretation
Polypoid	Visible projecting lesion
Nonpolypoid	Visible subtle surface lesion
Invisible	Histologic dysplasia on nontargeted biopsy

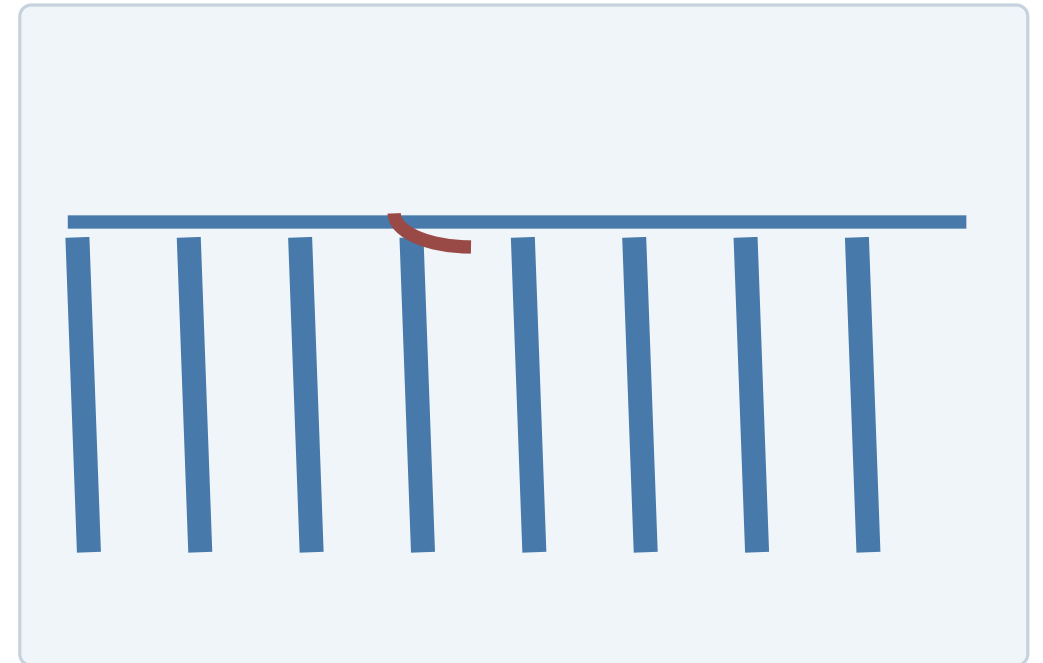
Polypoid does not automatically mean sporadic

- IBD patients can develop sporadic colorectal adenomas.
- Polypoid IBD-associated dysplasia may resemble sporadic adenoma.
- Distribution, surrounding colitis, and complete resectability matter.

**Morphology alone does not
establish lesion origin.**

Nonpolypoid dysplasia can be subtle

- A minimally raised or depressed lesion may escape casual inspection.
- High-quality bowel preparation and slow careful examination are important.
- Suspicious mucosal irregularities warrant targeted assessment.



Subtle visible surface change

Schematic (not endoscopic or histologic evidence)

Invisible dysplasia is a sampling diagnosis

- The diagnosis arises on nontargeted biopsy without a documented visible lesion.
- Invisible does not prove that no subtle lesion exists.
- Repeat high-definition chromoendoscopy may reveal a resectable focus.

Nontargeted biopsy + dysplasia



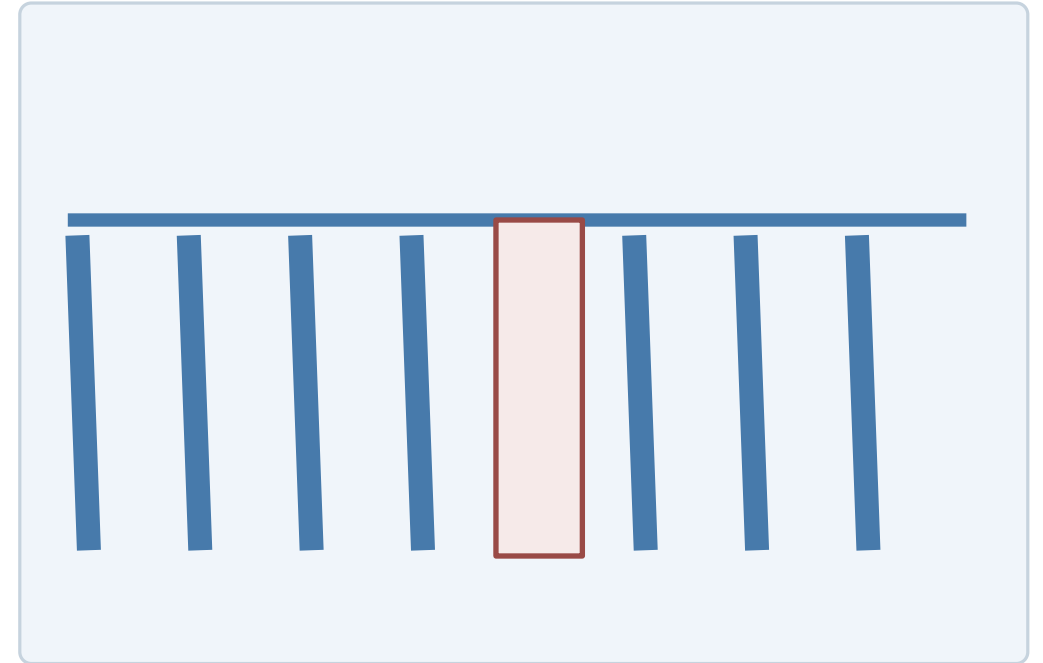
Expert confirmation



Repeat expert examination

Why apparently normal mucosa must be considered

- Dysplasia may develop in macroscopically flat mucosa.
- Endoscopic appearance and epithelial genotype are not equivalent.
- Surveillance strategies must address lesions that are not overt masses.



Mucosal surface may look unremarkable

Schematic (not endoscopic or histologic evidence)

Dye-spray chromoendoscopy

- Contrast dye accentuates mucosal surface architecture.
- AGA recommends considering dye-spray chromoendoscopy in colonic IBD surveillance.
- Dye highlights suspicious patterns; It does not substitute for tissue diagnosis.

Contrast-enhanced surface



Abnormality recognized



Targeted examination

Virtual chromoendoscopy

Method	Main principle
Dye-spray	Topical dye highlights surface relief
Virtual chromoendoscopy	Optical / digital image enhancement
Histology	Definitive dysplasia assessment

Targeted versus nontargeted biopsy

Sampling	When informative
Targeted	Suspected or altered mucosa
Nontargeted	White-light surveillance or selected high-risk patients

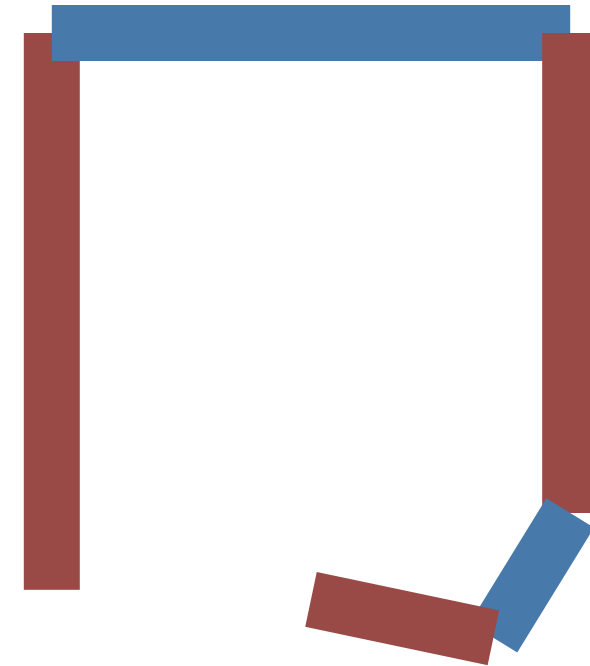
Nontargeted sampling remains situational

- AGA advises extensive nontargeted biopsies when surveillance relies on white-light endoscopy without chromoendoscopy.
- These are generally not routine with high-definition chromoendoscopy.
- History of dysplasia or PSC can justify added nontargeted sampling.

Sampling is tailored to endoscopic technique and patient risk.

Specimen mapping preserves clinical meaning

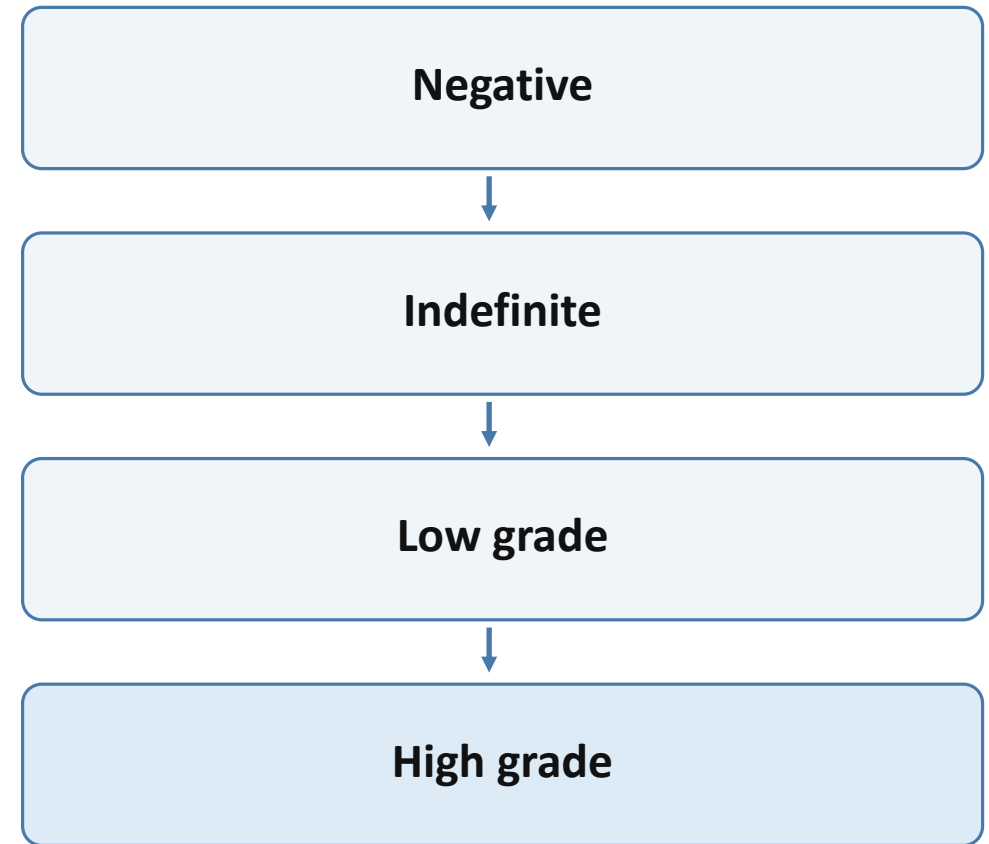
- Biopsies should be attributable to a colon region or targeted lesion.
- Report whether dysplasia was visible and resected versus biopsy-only.
- Anatomic distribution helps assess multifocality and future management.



Simplified distribution: Patchy Crohn colitis

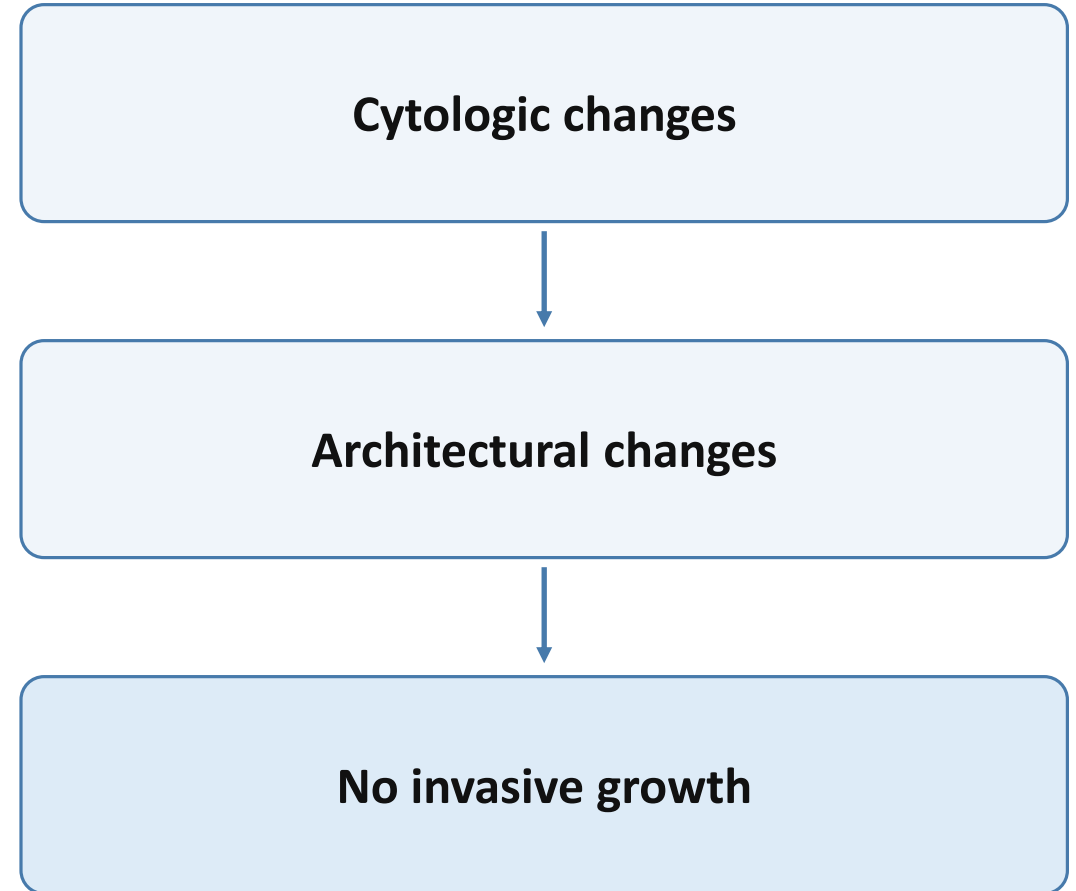
Morphologic classification begins with H&E

- Dysplasia is assessed using cytologic and architectural alterations.
- Conventional dysplasia is graded as low grade or high grade.
- The category “indefinite for dysplasia” addresses unresolved neoplasia versus reactive change.



Dysplasia is neoplasia without invasion

- ECCO defines dysplasia as unequivocal epithelial neoplasia without invasive growth.
- Atypia caused solely by inflammation or repair is not dysplasia.
- The designation requires integration of gland architecture and cytology.



Negative for dysplasia

- Epithelium may be normal, chronically inflamed, or regenerative.
- “Negative” refers to absence of established dysplasia, not absence of IBD.
- Activity and architectural injury can still be reported separately.

Negative for dysplasia does not mean absence of chronic colitis.

Reactive atypia can mimic neoplasia

Regeneration	Dysplasia
Inflammation / repair context	Persistent neoplastic cytology
Maturation may be preserved	Loss of normal epithelial maturation
May resolve with healing	Requires specific surveillance / therapy

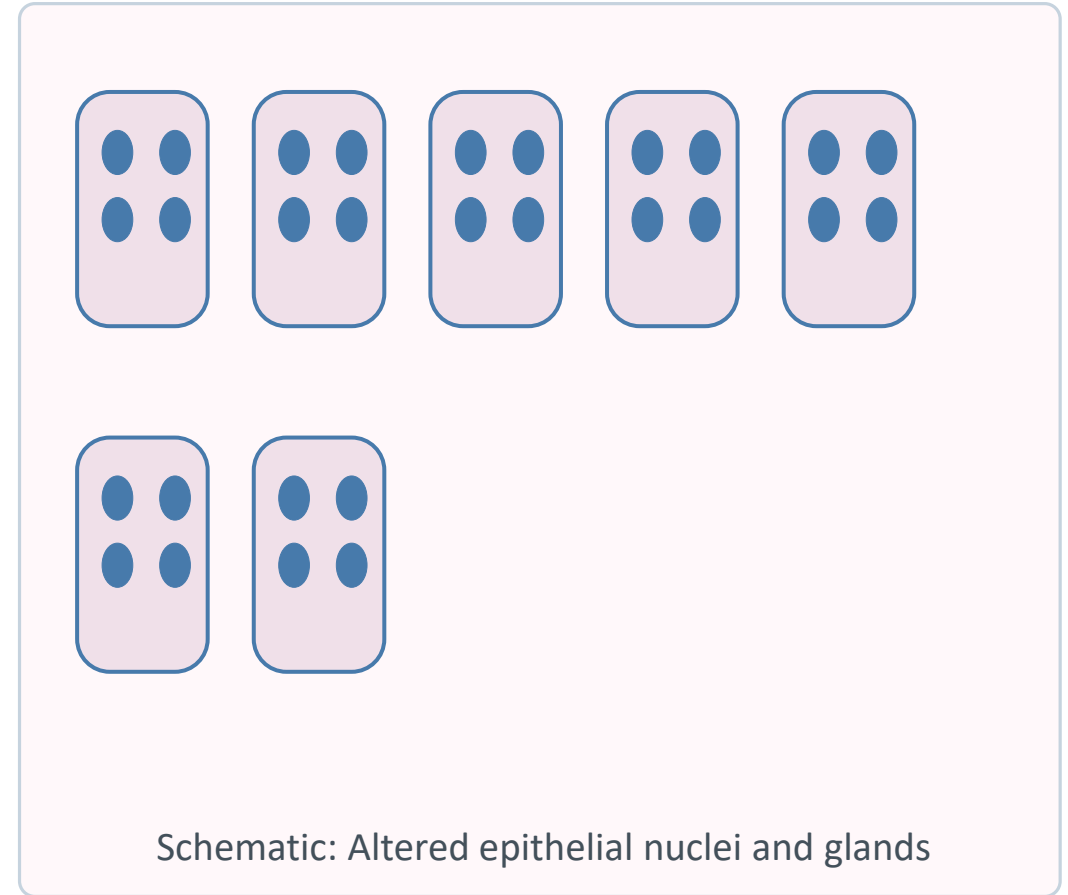
Indefinite for dysplasia

- Use this category when a definite distinction cannot be made.
- Severe active inflammation, tissue damage, and limited sampling may contribute.
- A definitive low-grade diagnosis should not be forced from equivocal material.

**Requires individualized
clinical action**

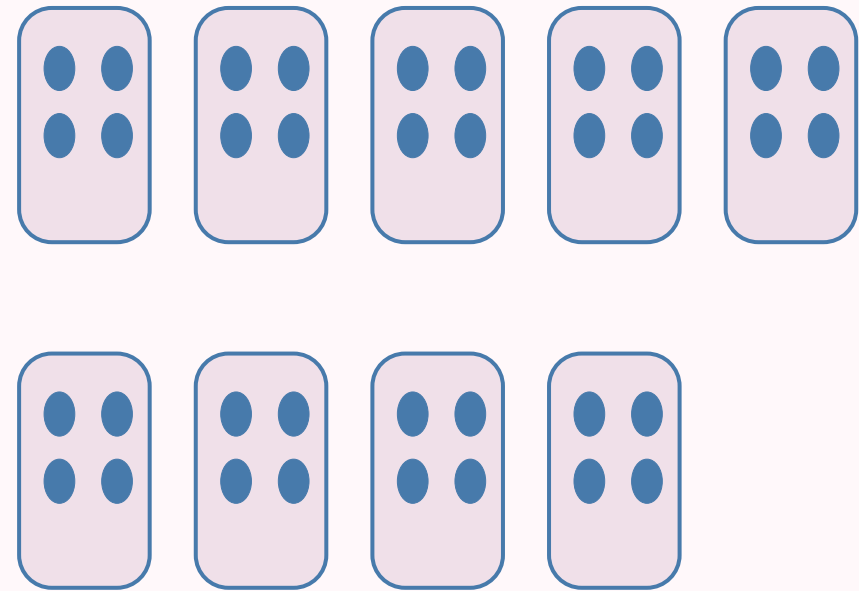
Low-grade dysplasia: Nuclear abnormalities

- Enlarged, elongated, hyperchromatic nuclei support dysplasia.
- Pseudostratification may be evident but polarity is relatively maintained.
- Interpret nuclei together with surrounding gland architecture.



Low-grade dysplasia: Architectural changes

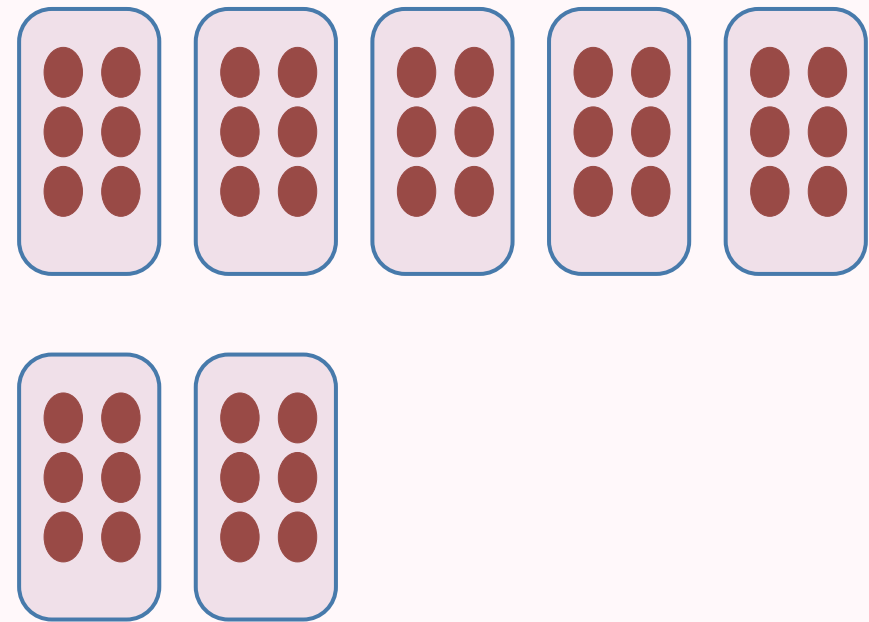
- Gland crowding or altered tubular organization may be present.
- Architectural abnormality is usually less marked than in high-grade lesions.
- Comparison with adjacent nondysplastic mucosa aids interpretation.



Schematic: Altered epithelial nuclei and glands

High-grade dysplasia: Cytologic escalation

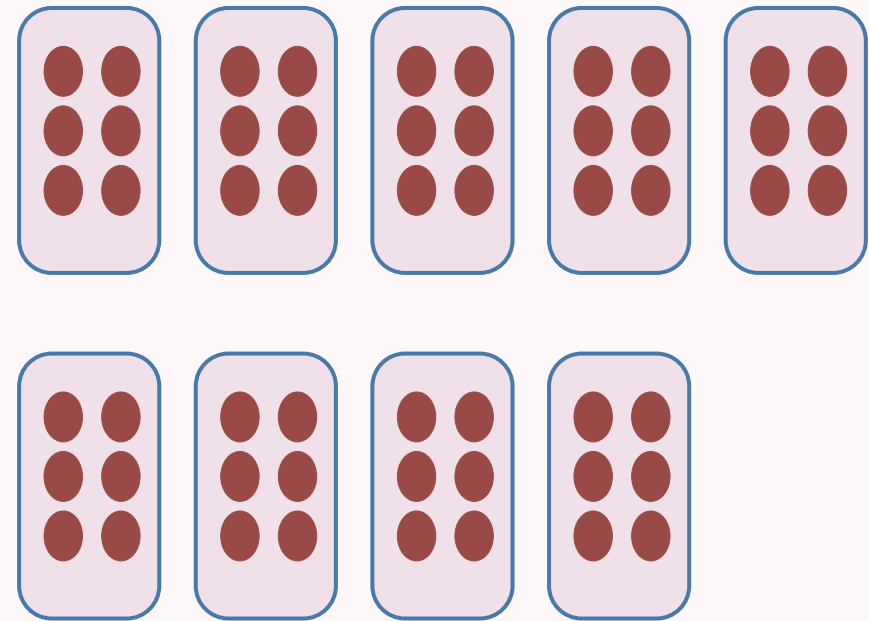
- Marked nuclear atypia and loss of polarity are concerning.
- Nuclei may occupy the upper half of epithelial cells.
- Prominent nucleoli and vesicular chromatin may be present.



Schematic: Marked nuclear crowding and gland complexity

High-grade dysplasia: Complex glands

- Back-to-back glands and cribriform configurations suggest advanced architecture.
- Loss of orderly epithelial maturation accompanies severe cytologic abnormalities.
- High-grade dysplasia remains noninvasive unless invasion is demonstrated.



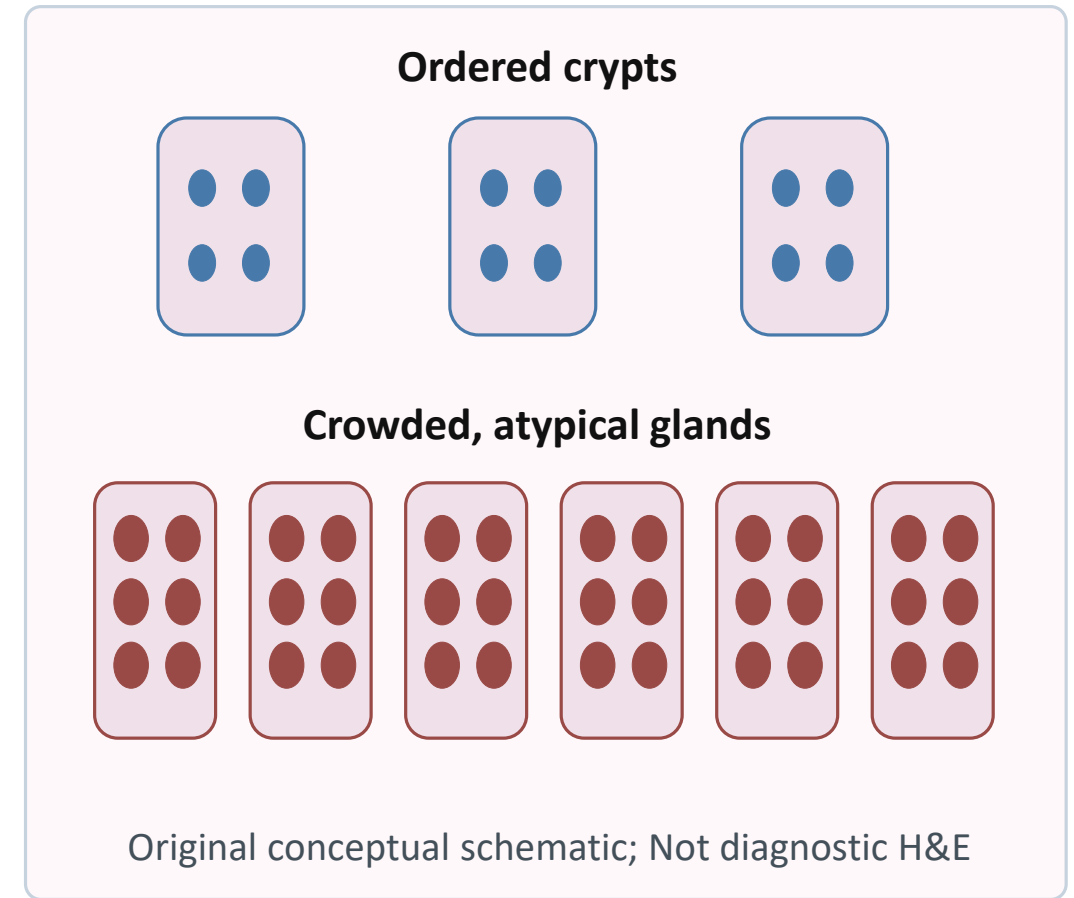
Schematic: Marked nuclear crowding and gland complexity

Low-grade versus high-grade dysplasia

Feature	Low grade	High grade
Nuclear atypia	Present, less severe	Marked / loss of polarity
Architecture	Variable crowding	Complex, cribriform possible
Invasion	Absent	Absent by definition

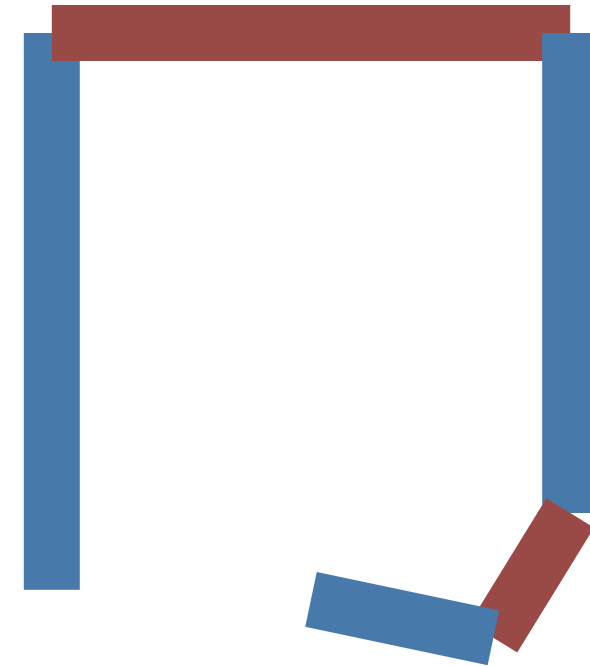
A schematic progression of gland complexity

- Normal crypts are ordered and separated by lamina propria.
- Dysplastic glands become progressively crowded and irregular.
- The schematic is conceptual; Real grading requires authentic H&E sections.



Multifocal dysplasia changes risk interpretation

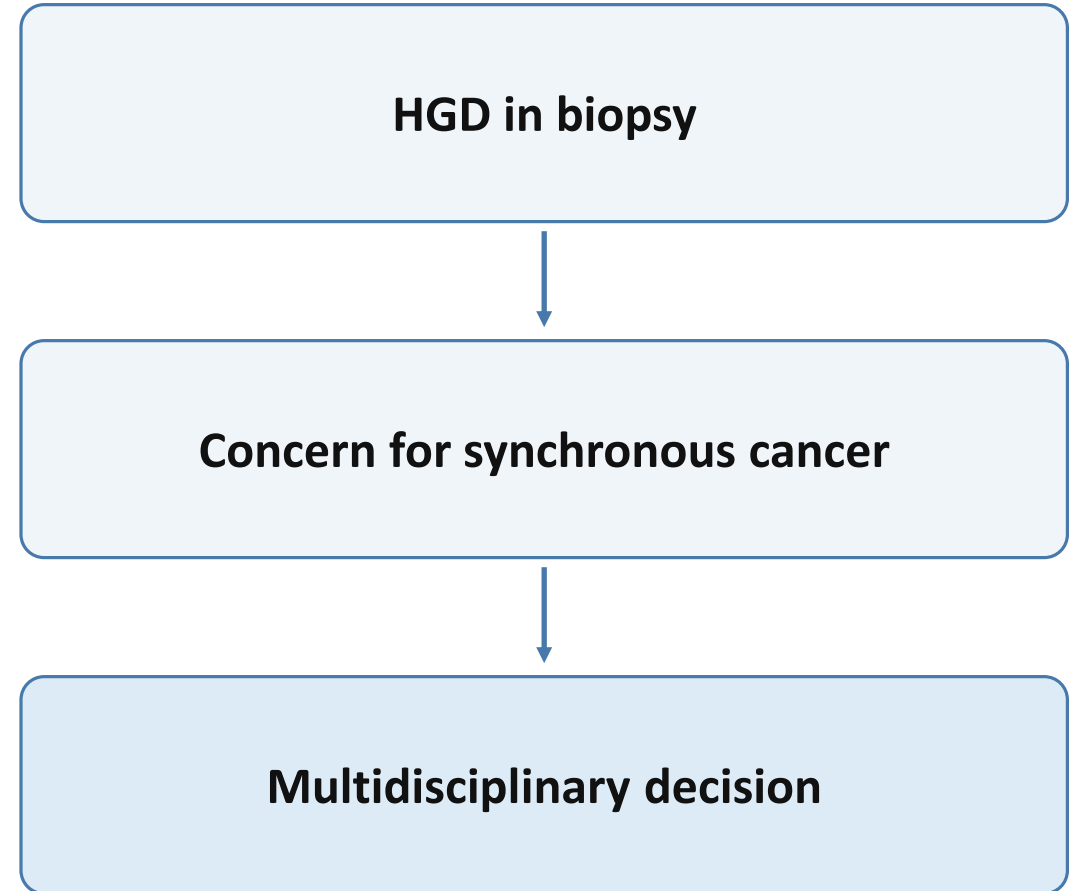
- IBD-associated dysplasia may arise at more than one colonic site.
- Multifocal nonpolypoid dysplasia increases concern for widespread at-risk mucosa.
- Management differs from an isolated completely resectable lesion.



Simplified distribution: Distinct sites

High-grade dysplasia may coexist with cancer

- Carcinoma at the same site or elsewhere in the colon.
- Sampling of one lesion does not exclude invasive neoplasia elsewhere.
- High-grade dysplasia commonly triggers colectomy consideration.



Superficial low grade may conceal deeper cancer

- Invasive adenocarcinoma beneath superficially low-grade dysplasia.
- Biopsy sampling may underrepresent the biologic extent of a lesion.
- Discordance between endoscopic appearance and biopsy demands caution.

Superficial biopsy: Low-grade area



**Deeper invasive focus may
be missed**

The pathologist needs endoscopic context

- Document lesion site, size and morphology.
- State whether it was visible, completely resected, or detected only on biopsy.
- Include surrounding inflammation and any previous dysplasia history.

Visibility, resection status, and prior dysplasia inform interpretation.

Expert review matters

- Active inflammation complicates grading and reproducibility.
- AGA advocates expert confirmation of dysplasia detected on nontargeted biopsies.
- Clinical and endoscopic information improves interpretive confidence.

Initial interpretation



GI pathology review



Endoscopic correlation

Sporadic adenoma versus IBD dysplasia

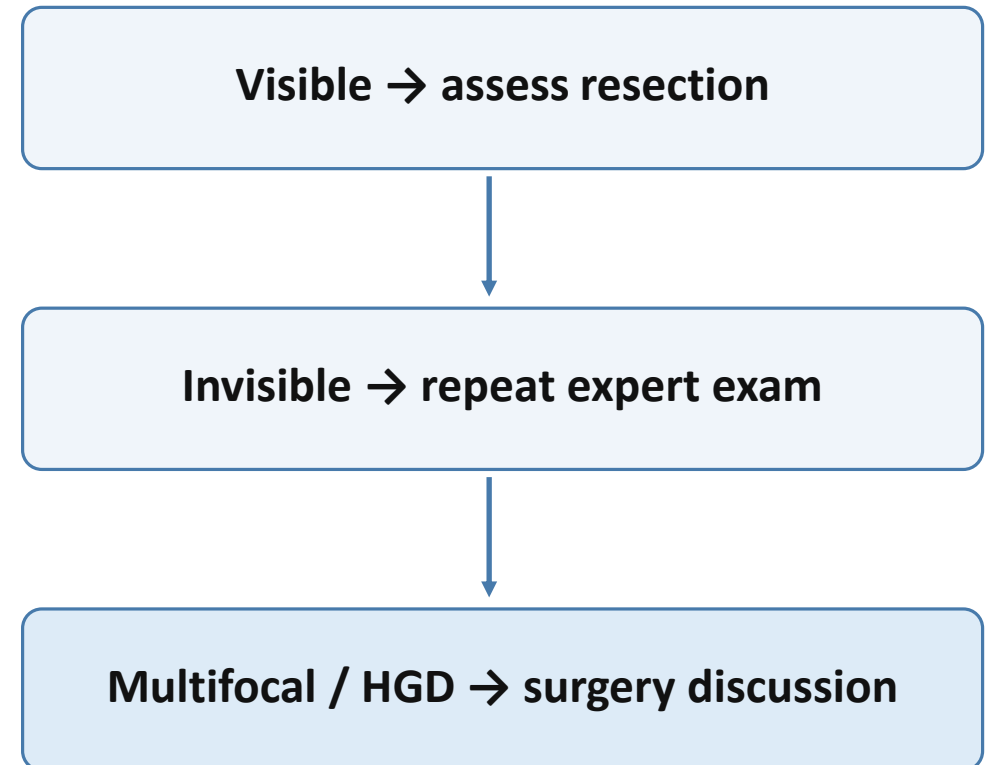
Question	Why it matters
Is the lesion completely removable?	Supports endoscopic strategy
Is dysplasia present elsewhere?	Raises field-risk concern
What is the colitis distribution?	Provides etiologic context

Use modern visibility terminology

Less useful legacy label	Preferred description
DALM / adenoma-like mass	Visible lesion: Morphology + resectability
Flat dysplasia (ambiguous)	Nonpolypoid visible or invisible

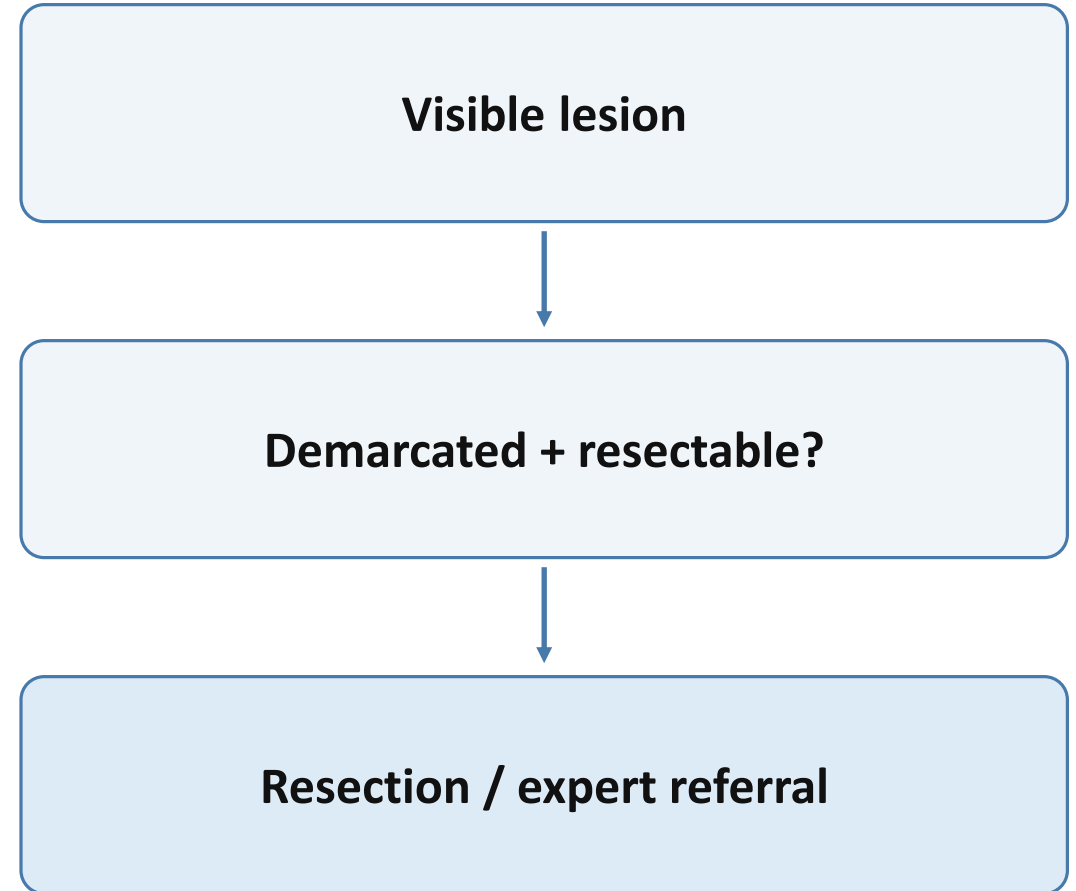
The first management fork: Visible or invisible

- Clearly delineated visible lesions should be assessed for resection.
- Invisible dysplasia requires expert confirmation and repeat optimized examination.
- Unresectability, multifocality, and grade influence surgery discussions.



Visible dysplasia: Assess resectability

- AGA favors endoscopic resection of well-demarcated lesions without invasive features or significant submucosal fibrosis.
- Questionable resectability warrants referral to an expert endoscopist or IBD center.
- Histology after complete resection guides follow-up.



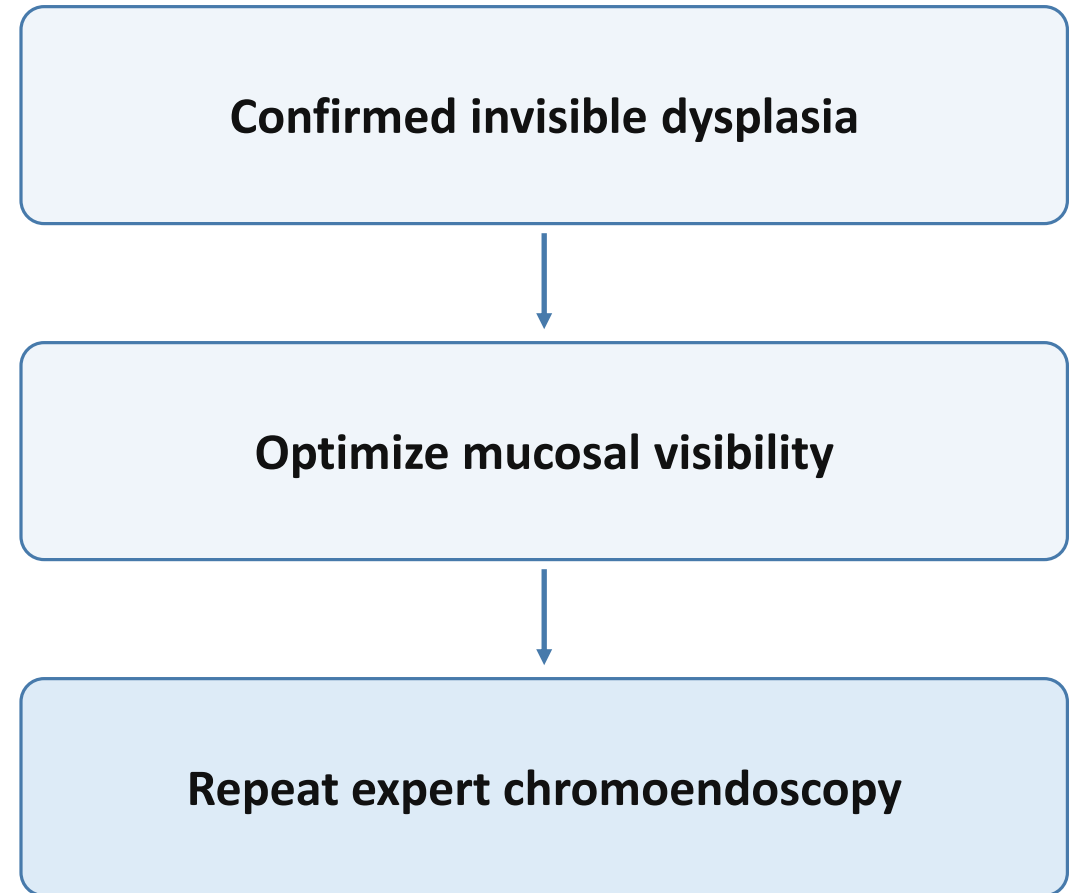
Resected solitary polypoid dysplasia

- Robbins permits endoscopic removal with careful follow-up when solitary and without flat dysplasia.
- Modern guidance extends assessment to any clearly demarcated resectable visible dysplasia.
- A complete resection does not eliminate the need for colitis surveillance.

Successful resection must be followed by colitis surveillance.

Invisible dysplasia: Repeat the examination

- Confirm the diagnosis, ideally through expert GI pathologist review.
- Perform high-definition dye-spray chromoendoscopy under optimal conditions.
- Sample the previously implicated region if no lesion becomes visible.



When colectomy is favored

- AGA recommends colectomy for unresectable visible dysplasia.
- Invisible multifocal dysplasia or high-grade dysplasia favors colectomy.
- Treatment decisions remain multidisciplinary and patient-specific.

**Requires individualized
clinical action**

High-grade dysplasia: The consequence

Context	Management implication
Invisible HGD	Colectomy recommended (AGA)
Unresectable visible lesion	Colectomy recommended (AGA)
Clearly resectable visible lesion	Expert endoscopic assessment

Low-grade dysplasia requires context

- Surveillance or colectomy depending on extent and lesion pattern.
- Multifocal flat dysplasia and long-standing extensive disease raise concern.
- Modern endoscopic visibility, resectability, and expert confirmation refine decisions.

Grade

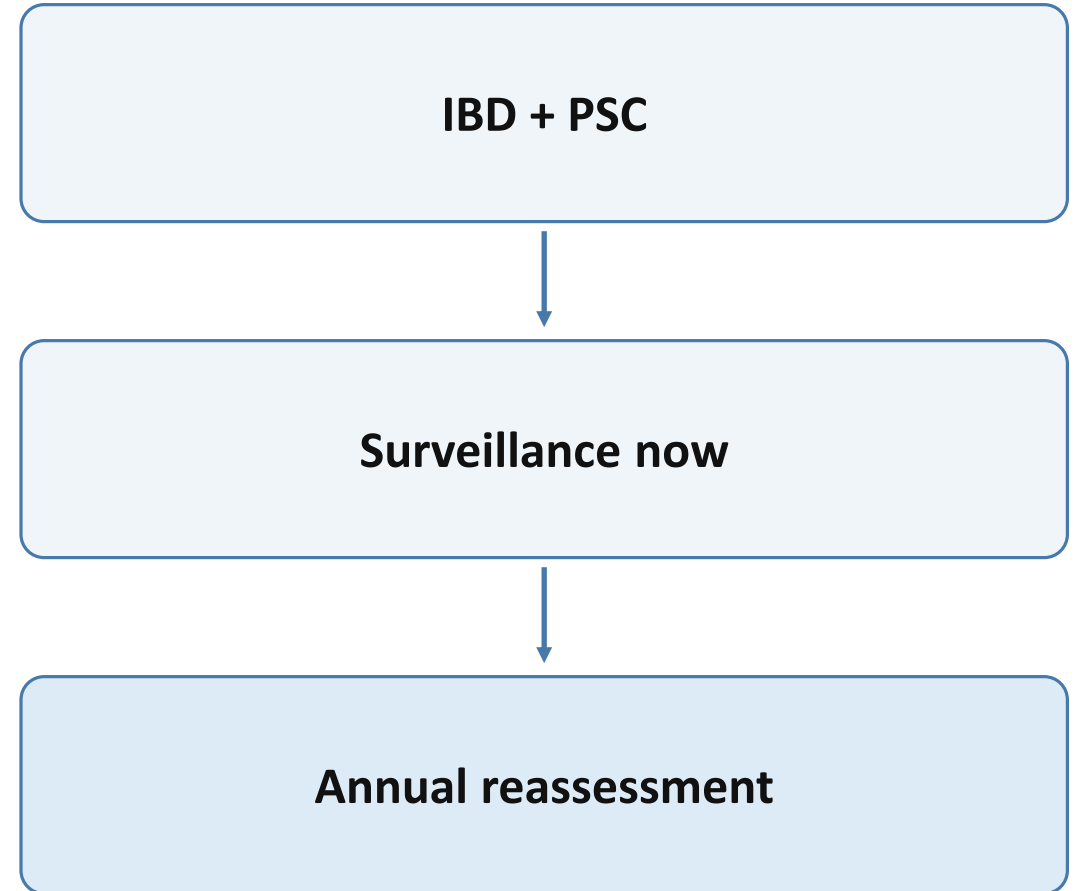
Number of foci

Visibility

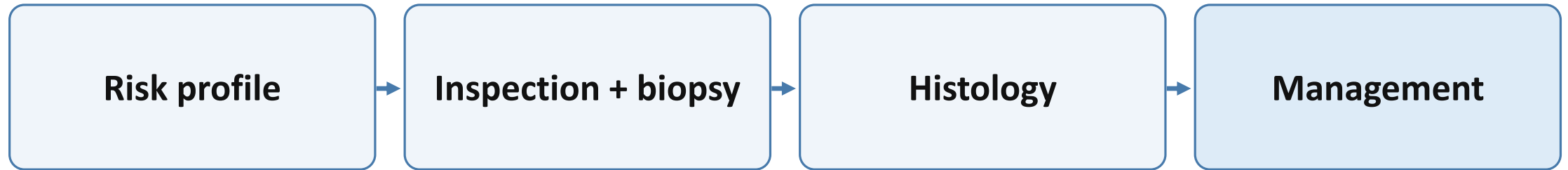
Resectability

PSC creates a different surveillance pathway

- Initiate surveillance at PSC diagnosis in patients with IBD.
- ECCO recommends annual colonoscopy in this subgroup.
- The intensity of current bowel symptoms does not remove this high-risk feature.



Risk and lesion management form one pathway



- First stratify colonic IBD risk and initiate surveillance.
- Then detect and histologically categorize abnormalities.
- Finally choose resection, intensified surveillance, or surgery according to lesion features.

Case 1: Long-standing extensive UC

- A patient with pancolitis of long duration enters surveillance.
- Recent examinations show recurrent histologic active inflammation.
- Question: Which risk dimensions are operating?

Clinical reasoning

Duration + pancolitis + inflammatory activity increase concern; Determine PSC status and past dysplasia.

Case 1: The surveillance strategy

- Do not assess risk from current symptoms alone.
- Review inflammation history, disease extent and prior biopsy maps.
- Use high-quality inspection with targeted biopsies of suspicious lesions.

Risk assessment



Optimized surveillance



Histologic classification

Case 2: An invisible dysplasia report

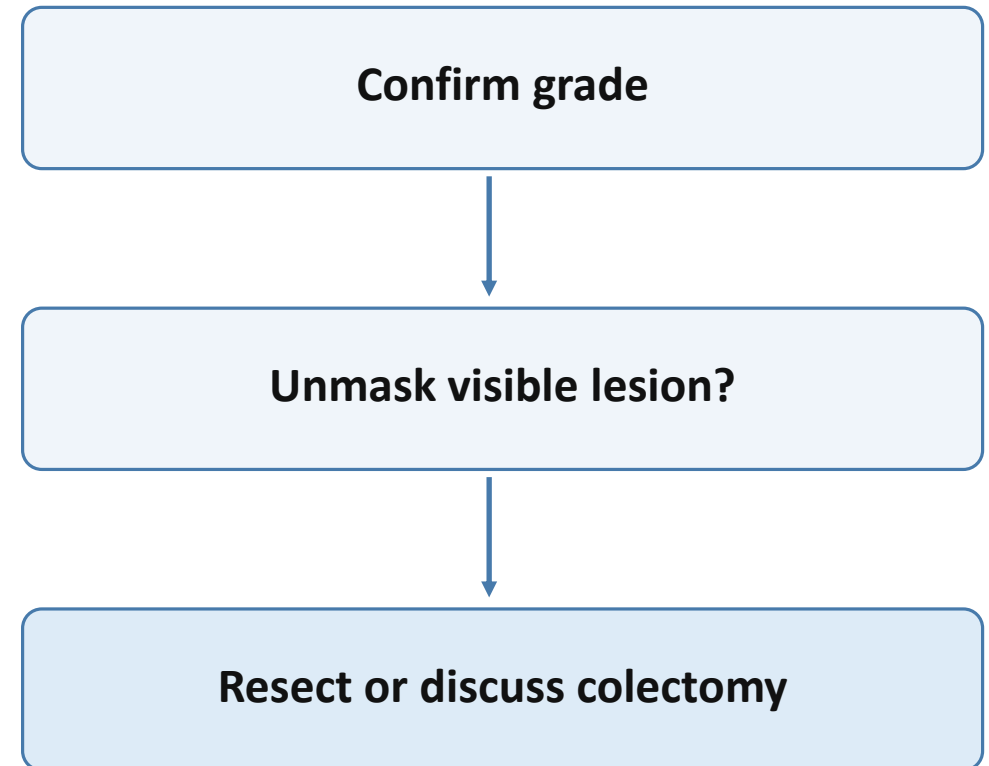
- A nontargeted biopsy is read as dysplasia, but no lesion was described.
- The original examination occurred during active inflammation.
- Question: What verification is needed before definitive treatment?

Clinical reasoning

Expert pathology review,
inflammation control when possible,
and repeat high-definition
chromoendoscopy.

Case 2: Grade and distribution direct action

- Ask whether dysplasia is confirmed, low grade or high grade.
- Determine if repeat inspection identifies a resectable lesion.
- Persistent invisible multifocal or high-grade dysplasia warrants colectomy discussion.



What is source-based, and what is updated?

Information layer	Use in this lecture
Robbins 11e (2026)	Core textbook disease concepts
AGA (2021)	Endoscopic dysplasia management
ECCO (2023–24)	Risk surveillance and histologic criteria

Integrated take-home messages

- Cancer risk depends on colitis duration, extent and cumulative activity.
- PSC requires earlier and more intensive surveillance.
- Dysplasia is diagnosed histologically and may be nonpolypoid or invisible.
- Visibility, histologic grade, multifocality and resectability guide decisions.

Selected references

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. 2026. Ch 17, p 733.

Murthy SK et al. AGA Clinical Practice Update. Gastroenterology. 2021.

Gordon H et al. ECCO Guidelines: IBD and Malignancies. J Crohns Colitis. 2023.

ECCO Position Paper: Definitions of Histological Abnormalities in IBD. J Crohns Colitis. 2024.

Bressenot A et al. World J Gastroenterol. 2014;20:3164–3172.