

Premalignant and Malignant Lesions

Colorectal adenomas · serrated precursors · early invasion

Pathology of the gastrointestinal tract

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Learning objectives

Apply the morphology to the clinical decision.

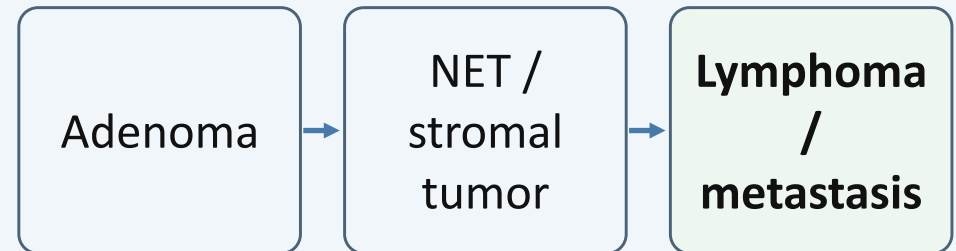
- Distinguish polyp shape from histologic lineage and malignant potential.
- Recognize tubular, villous, and serrated epithelial lesions.
- Identify the muscularis mucosae as the key boundary for invasion.
- Explain how size, dysplasia, depth, and resection findings affect risk.

A polyp is a gross configuration

The word “polyp” does not specify a histologic diagnosis.

- An elevated mucosal lesion may be neoplastic or nonneoplastic.
- Multiple unrelated tumor classes can create a polypoid mass.
- Histology determines precursor status and subsequent decisions.

Polyp = growth configuration

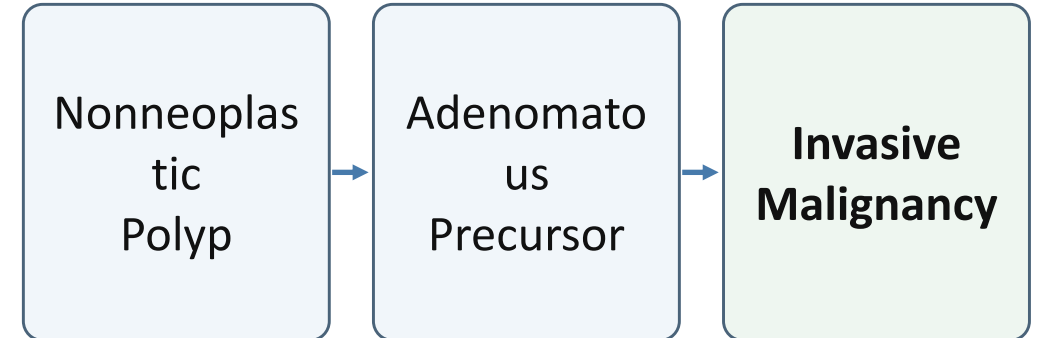


Morphology alone does not establish lineage

Intestinal polyps: The first classification

Epithelial neoplasia is one branch of the polyp differential.

- Nonneoplastic: Hyperplastic, inflammatory, hamartomatous.
- Neoplastic: Adenomatous and selected serrated precursor lesions.
- Invasive malignancy may itself appear polypoid.



Where intestinal neoplasia concentrates

Colorectal adenomas are the most common neoplastic intestinal lesions.

- They are precursors to most colorectal adenocarcinomas.
- Polyp detection therefore has a cancer-prevention role.
- Colonic adenocarcinoma is the most common GI malignancy.

Colorectum

Adenomas common
Adenocarcinoma is the
predominant GI malignancy

The small-intestine paradox

Tumor frequency is not proportional to intestinal length.

- The small intestine contributes roughly 75% of GI tract length.
- Nevertheless, small-intestinal neoplasms are comparatively uncommon.
- The contrast emphasizes organ-specific carcinogenesis.

Small intestine

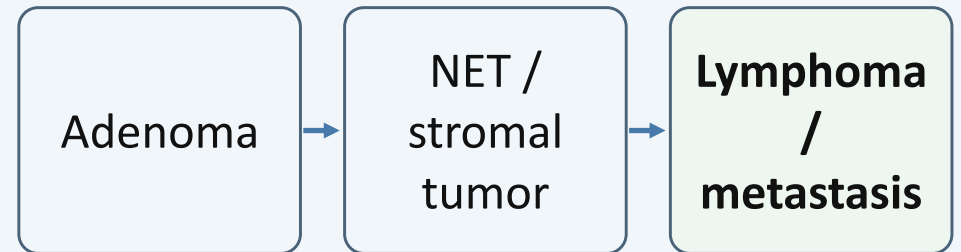
~75% of GI tract length
Tumors comparatively uncommon
Adenocarcinoma ≈ well-differentiated
NET among malignancies

Spectrum of small-intestinal malignancies

Adenocarcinoma is not the only epithelial or mural tumor to consider.

- Adenocarcinomas and well-differentiated neuroendocrine tumors have roughly equal incidence.
- Lymphomas and sarcomas are less frequent malignant categories.
- Biopsy diagnosis must consider the full tumor spectrum.

Polyp = growth configuration



Morphology alone does not establish lineage

Adenomas: A defining concept

An adenoma is an epithelial neoplasm with dysplasia.

- It may be a small pedunculated or a large sessile polyp.
- Adenomas can be precursors without already being invasive cancers.
- Epithelial dysplasia supplies the morphologic basis for diagnosis.

Cytology

Nuclear elongation · hyperchromasia · stratification

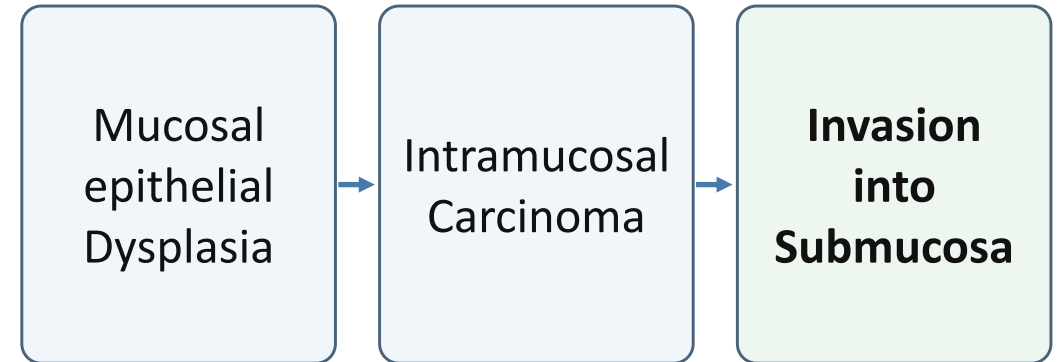
Architecture

Tubular / villous growth patterns

Precursor status does not mean inevitable cancer

Most colorectal adenomas do not progress to carcinoma.

- The majority are benign at diagnosis.
- A subset acquires changes that permit invasive growth.
- An individual adenoma cannot be reliably predicted to progress from morphology alone.



Population prevalence and geographic trend

Adenomas are common in adulthood.

- The text reports adenomas in approximately 30% of Western adults by age 60.
- Historically lower prevalence in Asia has risen alongside westernized diet and lifestyle.
- Population adenoma rates correlate with colorectal carcinoma rates.

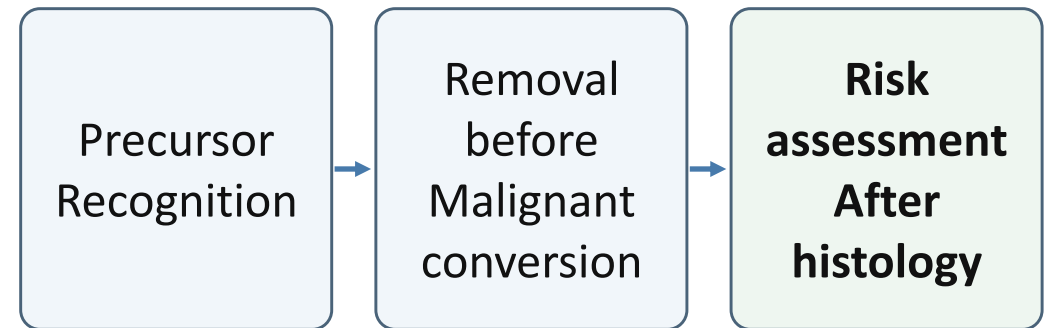
Colorectum

Adenomas common
Adenocarcinoma is the
predominant GI malignancy

Anatomic correlation of precursor and carcinoma

Adenoma and adenocarcinoma patterns broadly track one another.

- Their distributions within the colon are similar.
- This coherence supports the adenoma–carcinoma precursor relationship.
- Population correlations do not imply that every adenoma becomes cancer.



Most adenomas are clinically silent

Absence of symptoms does not exclude significant neoplasia.

- Small adenomas usually cause no clinical manifestations.
- Large lesions can cause occult bleeding.
- Chronic blood loss may lead to anemia.

Most adenomas

Clinically silent

Large adenomas

Occult bleeding and iron-deficiency anemia can occur

Why an adenoma may bleed

Lesion size can turn an incidental precursor into a symptomatic polyp.

- Large mucosal protrusions are exposed to luminal trauma.
- Occult bleeding may precede overt symptoms.
- Anemia can be a clue prompting colorectal evaluation.

Most adenomas

Clinically silent

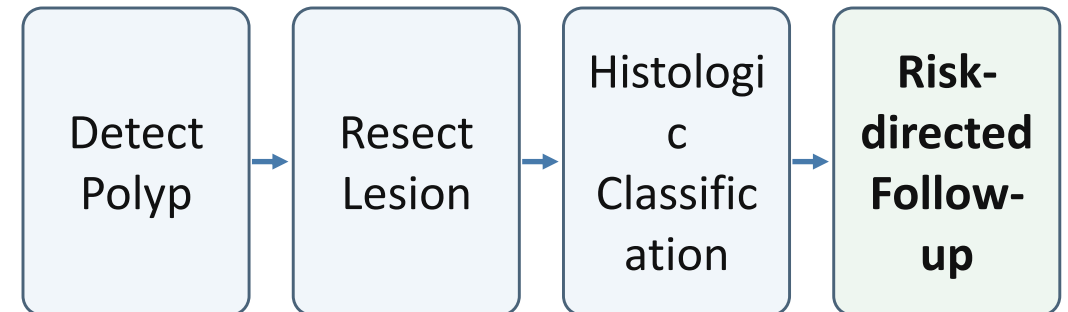
Large adenomas

Occult bleeding and iron-deficiency anemia can occur

Colonoscopy links diagnosis and prevention

Endoscopic removal interrupts the progression of an excised precursor.

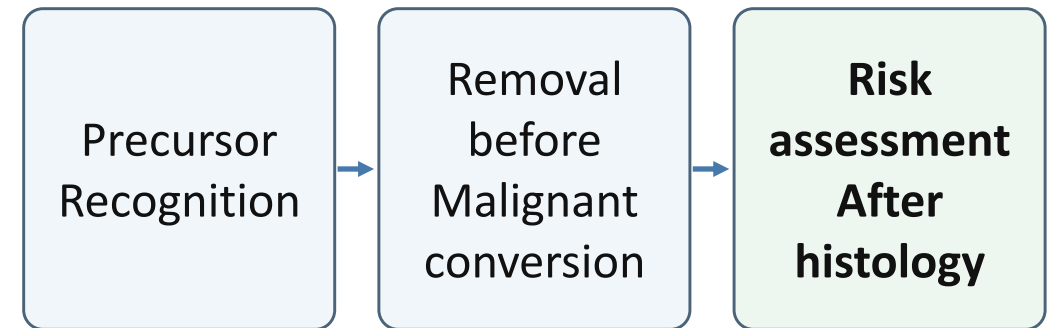
- Colonoscopy detects lesions and allows removal of many polyps.
- Histology classifies the excised tissue and identifies invasion.
- A removed adenoma cannot subsequently become a carcinoma at that site.



Screening as stated in the source

Risk-based detection precedes symptoms.

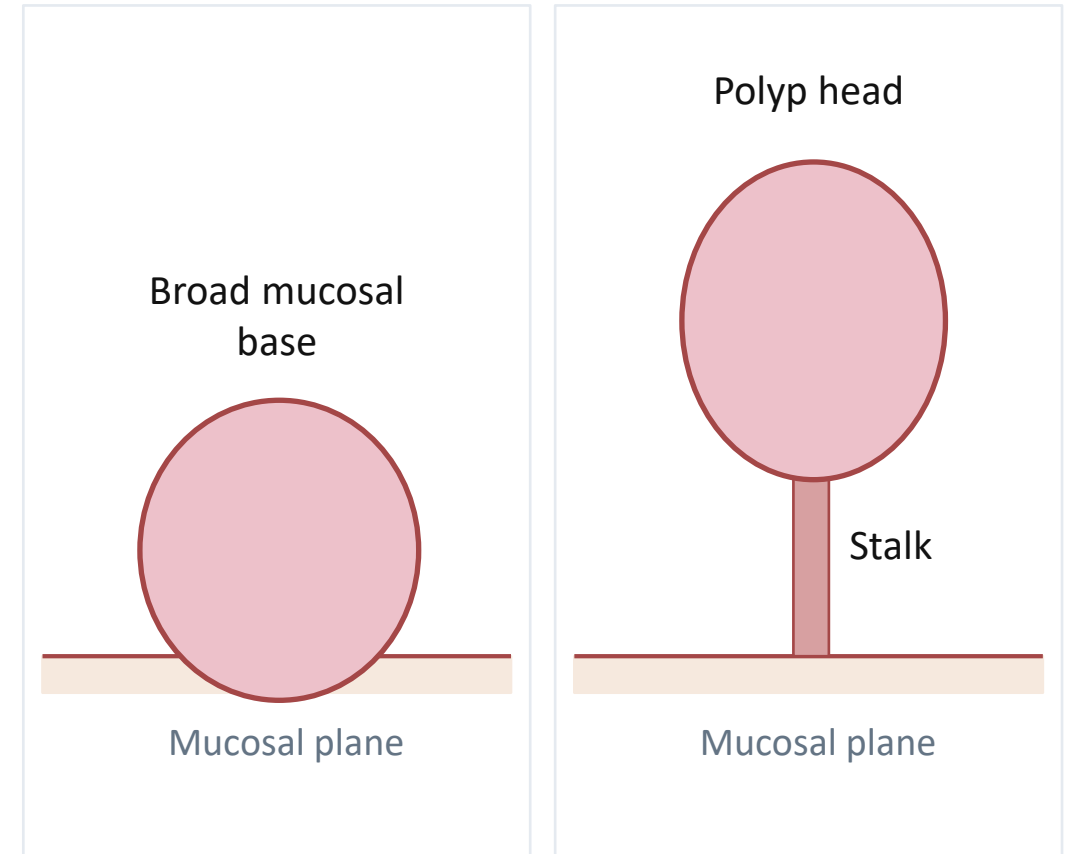
- Surveillance colonoscopy beginning at age 45.
- Earlier screening is considered when family history increases risk.
- These are textbook statements; Clinical schedules require contemporary guideline review.



Gross forms: Sessile and pedunculated

These terms describe attachment, not microscopic grade.

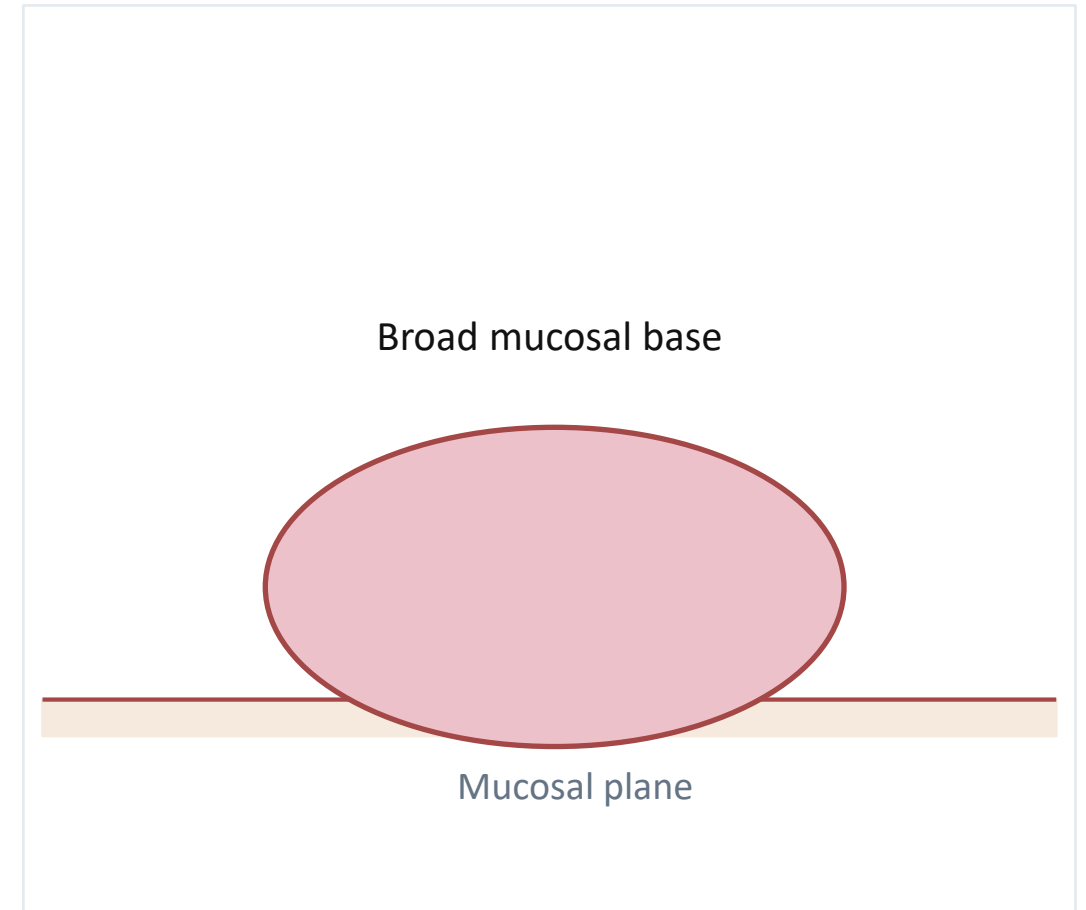
- Sessile lesions have a broad mucosal base.
- Pedunculated lesions project on a stalk.
- Both forms may contain dysplastic epithelium.



Adenomas usually begin as sessile elevations

Stalk formation can accompany enlargement.

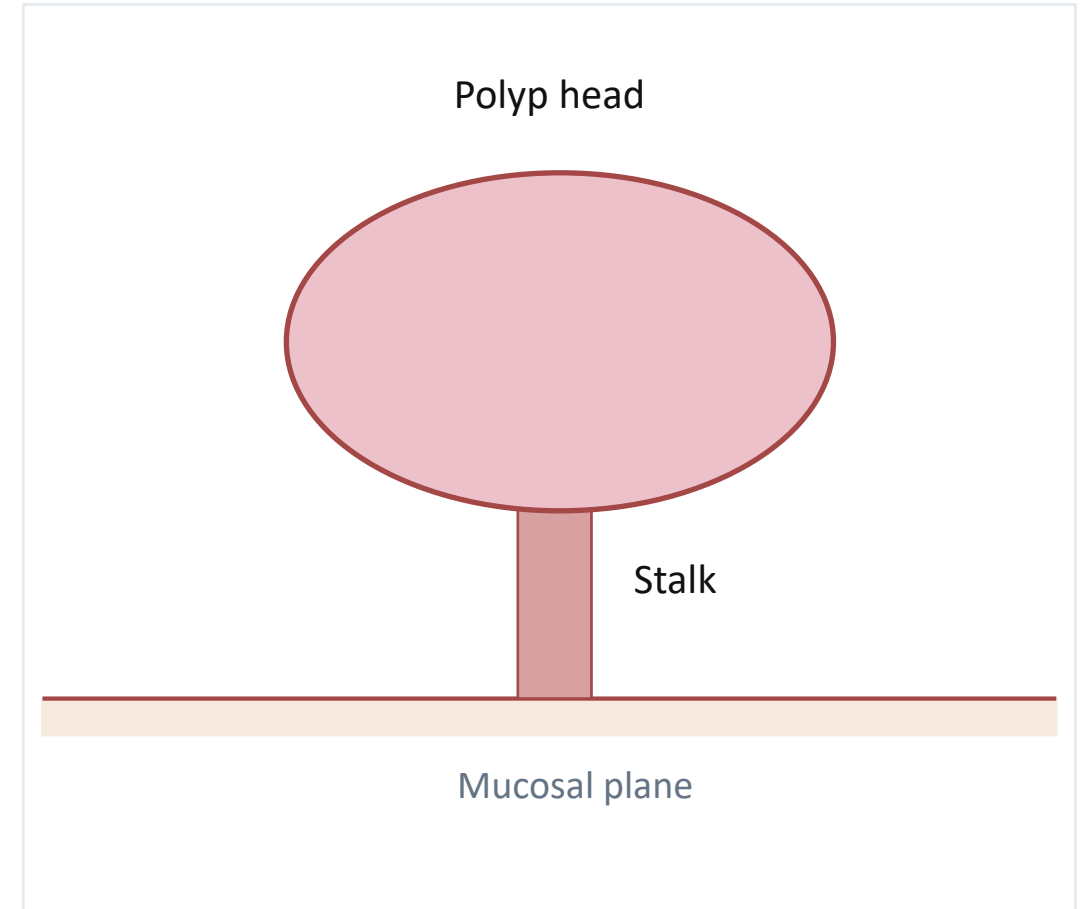
- The early lesion begins as a mucosal elevation.
- Proliferation around the lesion and traction may contribute to a stalk.
- The final contour does not identify histologic subtype.



Pedunculated adenoma: Anatomical components

The polyp head and stalk must be assessed separately.

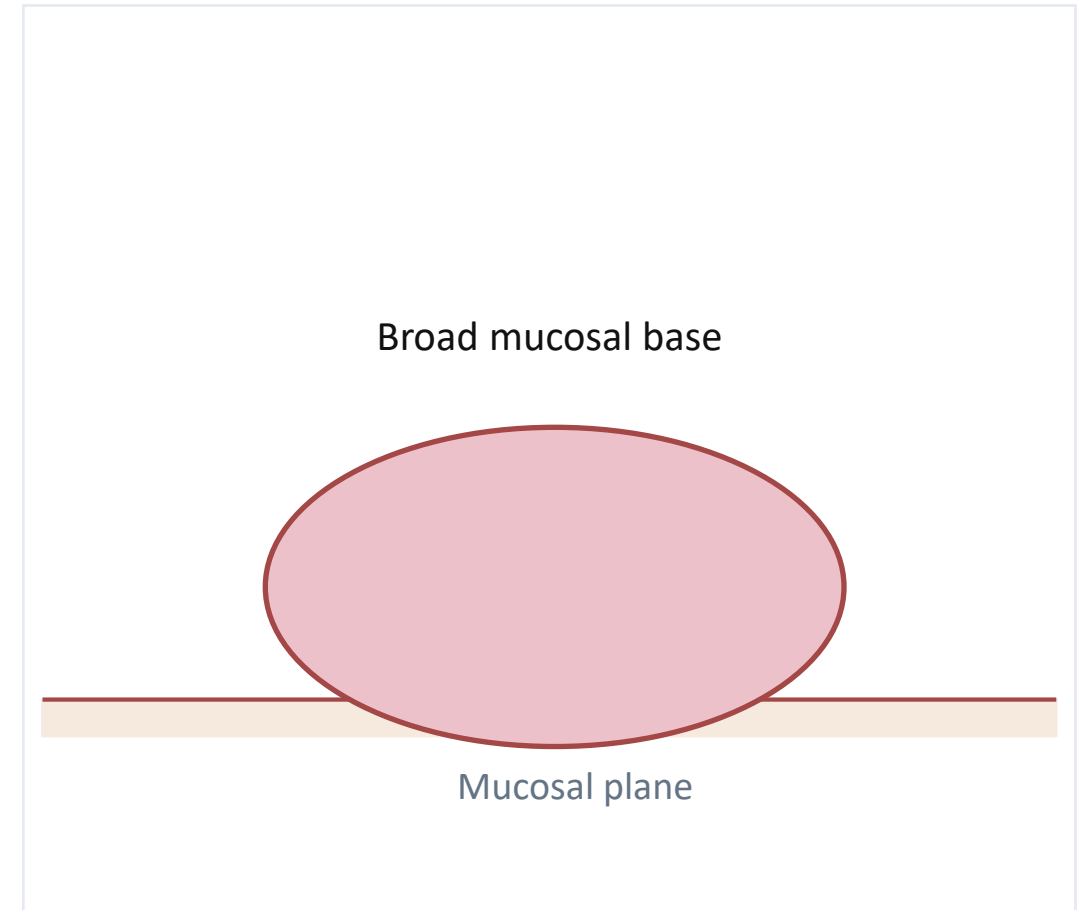
- The stalk contains a slender fibromuscular core.
- Prominent stalk vessels originate in the submucosa.
- Stalk covering is often nondysplastic but may show dysplasia.



Sessile lesions: Broad attachment

The entire mucosal interface deserves careful sampling.

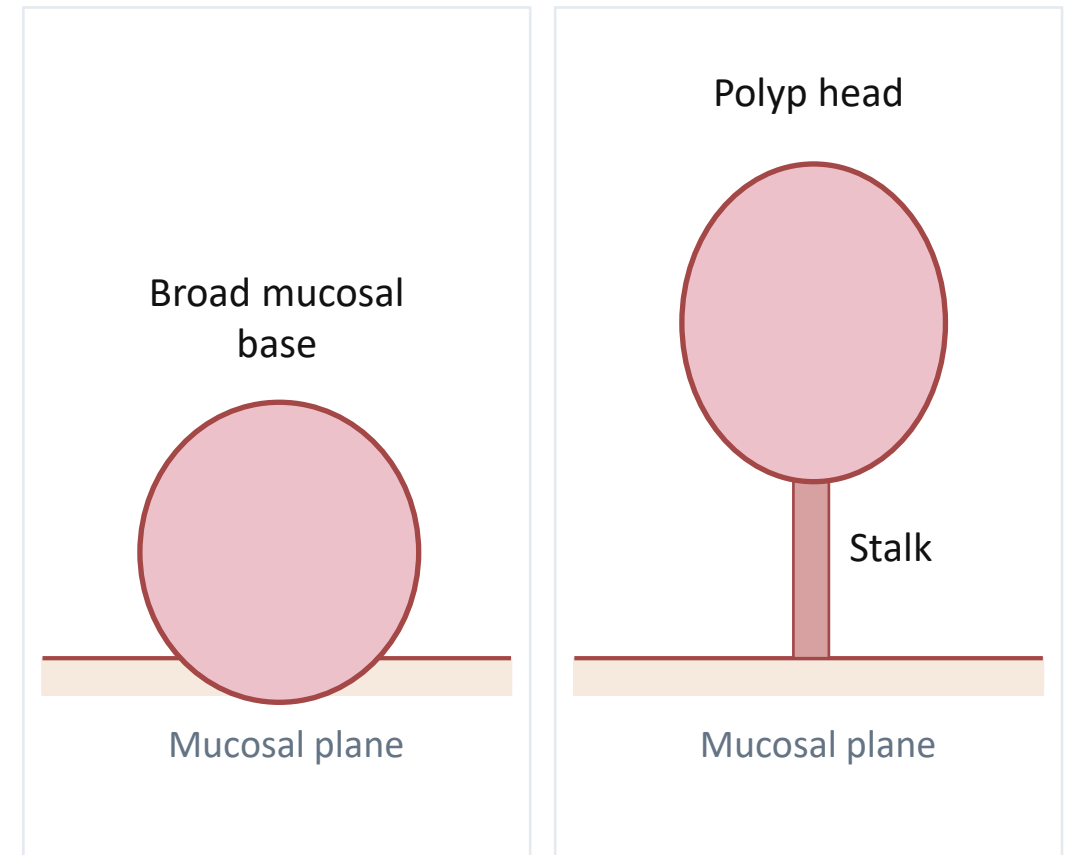
- Broad-based growth offers no discrete stalk for orientation.
- Large sessile lesions may show adenomatous or serrated architecture.
- Gross contour cannot establish the depth of invasion.



Gross appearance of colorectal adenomas

Surface texture often reflects the glandular proliferation beneath.

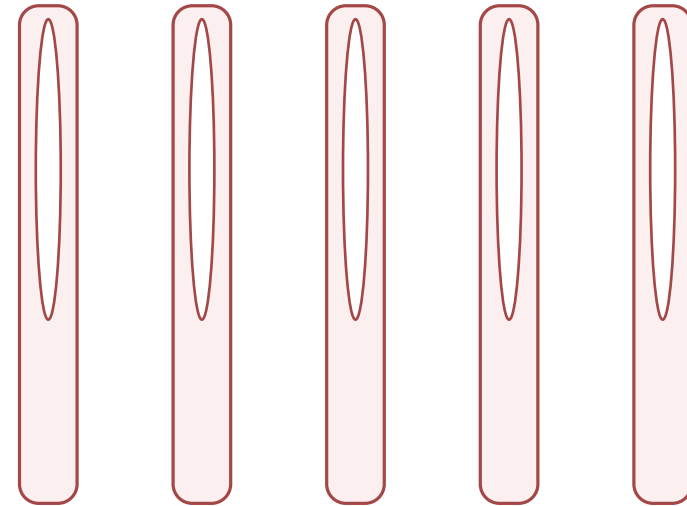
- Typical adenomas measure approximately 0.3–10 cm.
- Surfaces may look velvety or raspberry-like.
- Endoscopic appearance is informative but not a substitute for pathology.



Tubular architecture

Rounded glandular profiles dominate a tubular adenoma.

- Often presents as a relatively small pedunculated lesion.
- Crowded rounded/tubular glands build the adenomatous mass.
- Dysplastic epithelium, not the gland shape alone, defines an adenoma.

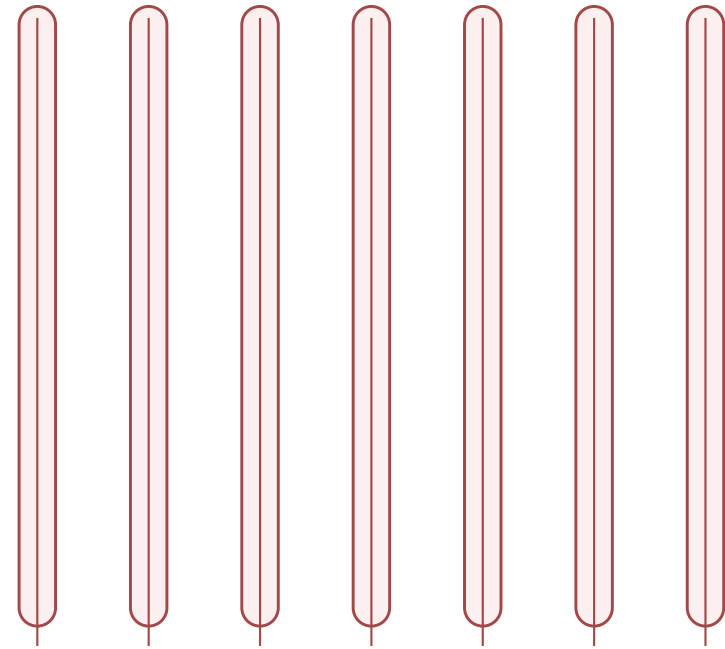


Original simplified architecture schematic — not a photomicrograph

Villous architecture

Long slender mucosal projections create a villous pattern.

- Architecture consists of villus-like epithelial fronds.
- Villous adenomas are commonly larger than tubular adenomas.
- Higher cancer frequency in this group is associated partly with lesion size.

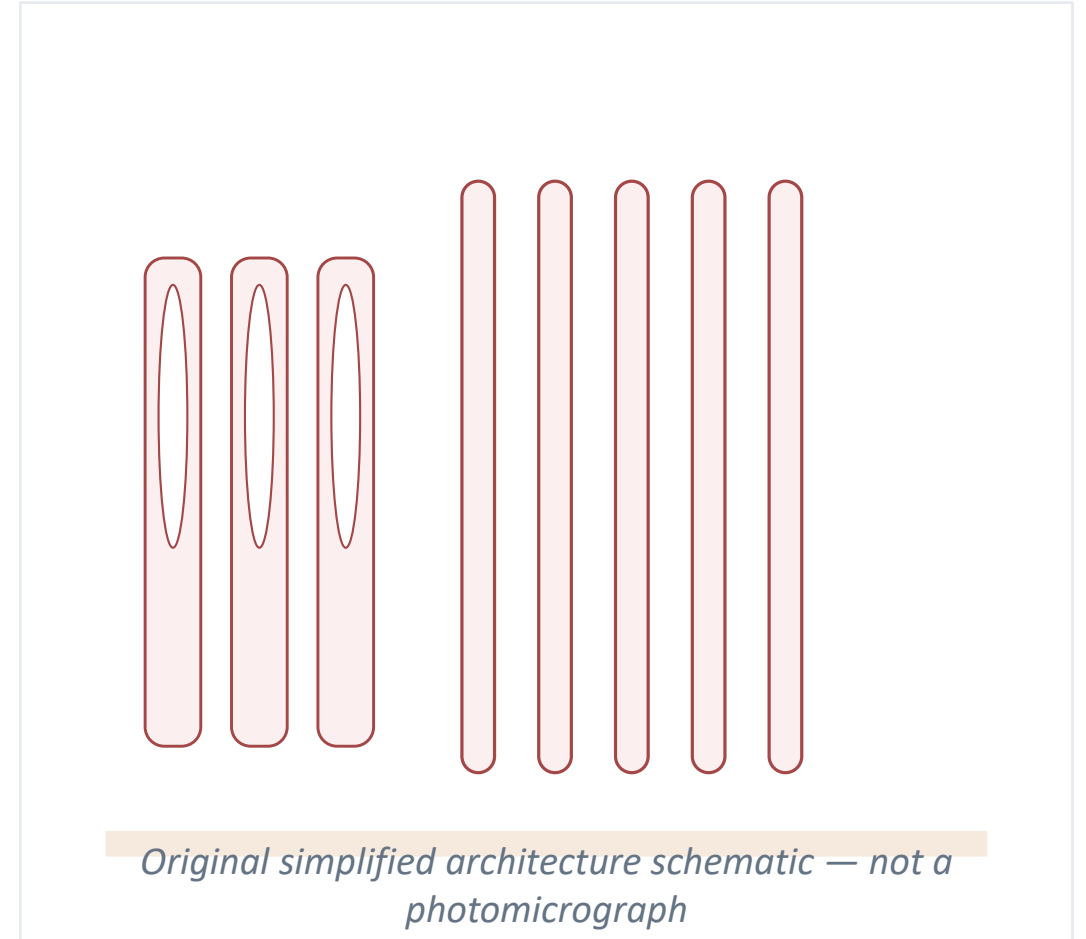


Original simplified architecture schematic — not a photomicrograph

Tubulovillous architecture

Mixed glandular and villous features coexist.

- Tubular glands and villous projections occupy the same adenoma.
- Classification describes architecture rather than invasive depth.
- Size and dysplasia remain critical separate observations.



Architecture and dysplasia are different axes

Do not treat a villous label as synonymous with carcinoma.

- Tubular / tubulovillous / villous = architectural pattern.
- Dysplasia describes epithelial cytologic and architectural atypia.
- Invasion requires assessment of the lesion–stroma interface.

Architectural type

Tubular / tubulovillous / villous

Epithelial atypia

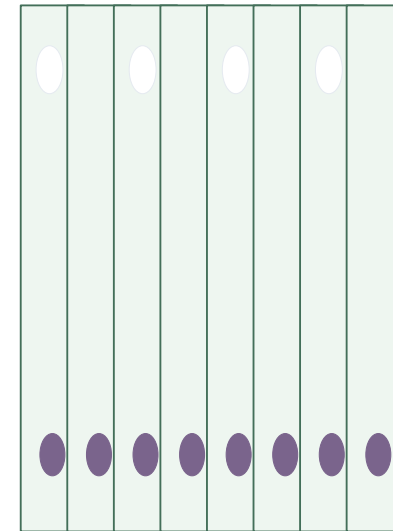
Low or high grade dysplasia

Adenomatous dysplasia: Defining nuclear pattern

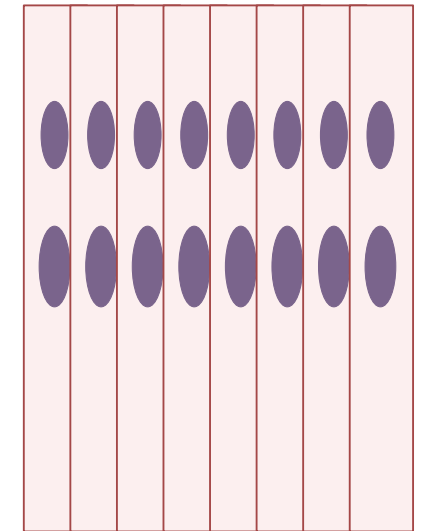
Nuclear elongation, hyperchromasia, and stratification are central.

- Dysplastic nuclei appear crowded and darker than normal.
- Nuclear layering disrupts the normal basal nuclear alignment.
- These findings can be easiest to see near the adenoma surface.

Normal epithelium



Adenomatous dysplasia



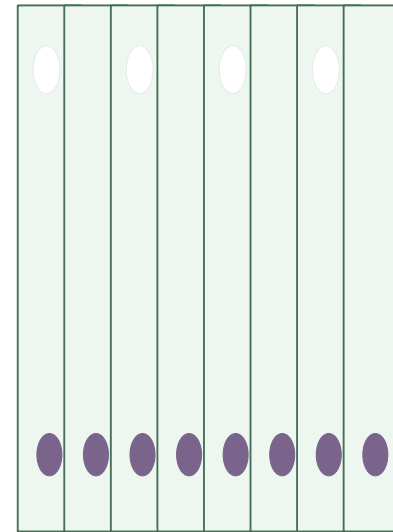
Original epithelial schematic — not tissue microscopy

Nuclear hyperchromasia

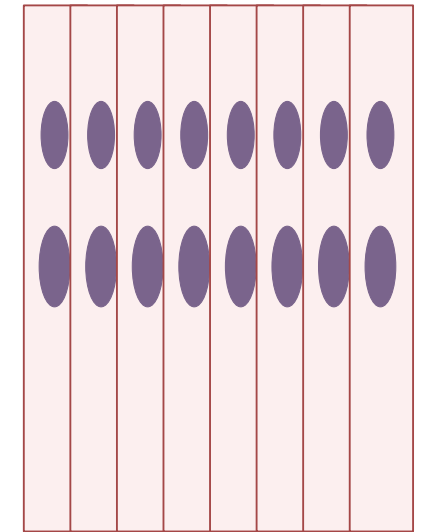
Increased nuclear staining is part of dysplastic cytology.

- Nuclei may be relatively dark and crowded.
- The change is interpreted together with shape and stratification.
- Inflammation or repair should not be equated automatically with neoplasia.

Normal epithelium



Adenomatous dysplasia



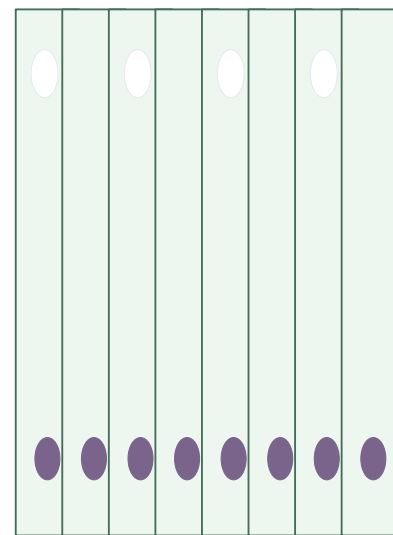
Original epithelial schematic — not tissue microscopy

Elongation and stratification of nuclei

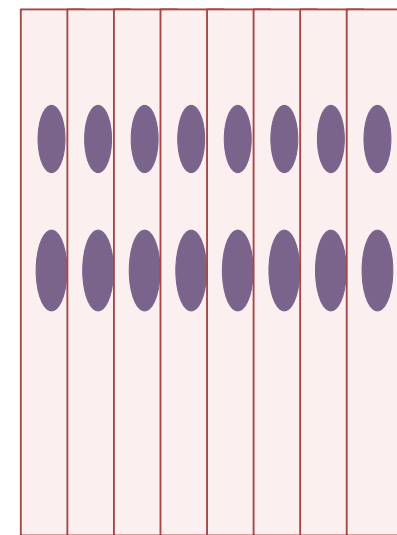
Nuclear polarity and spacing are diagnostically informative.

- Normal colonic epithelium has relatively basal nuclear positioning.
- Dysplastic epithelium shows elongated, layered nuclei.
- Compare atypical glands with nearby nondysplastic crypts when available.

Normal epithelium



Adenomatous dysplasia



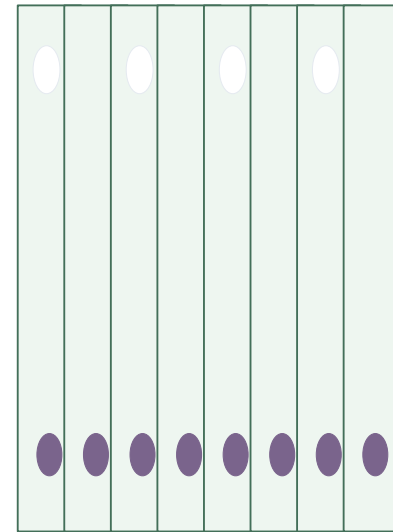
Original epithelial schematic — not tissue microscopy

Other epithelial changes in adenomas

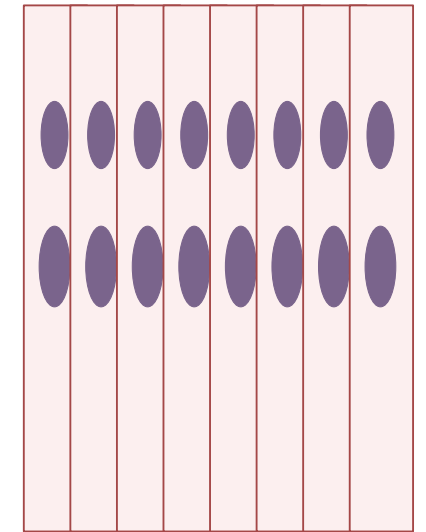
The dysplastic phenotype extends beyond the nucleus.

- Prominent nucleoli may accompany nuclear atypia.
- Cytoplasm may be more eosinophilic.
- Goblet-cell numbers are often reduced.

Normal epithelium



Adenomatous dysplasia



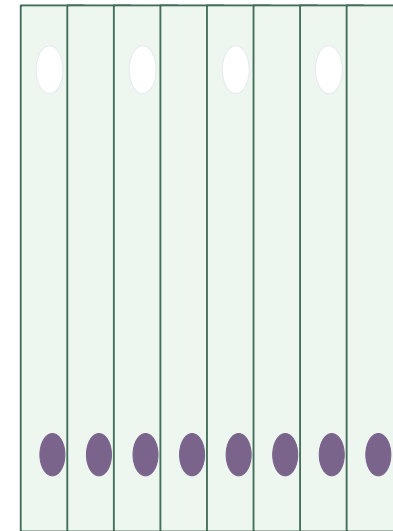
Original epithelial schematic — not tissue microscopy

Surface epithelium can reveal dysplasia

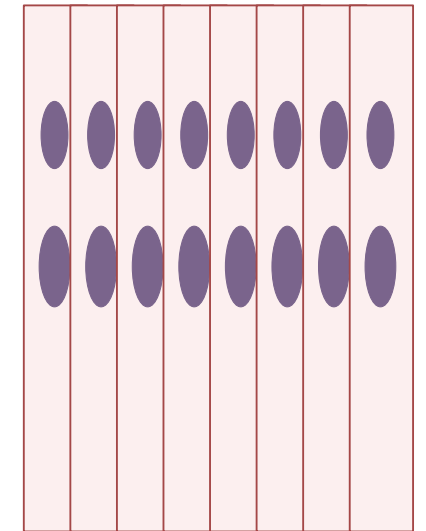
Assess the surface as well as the gland bases.

- Dysplastic atypia is often prominent at the adenoma surface.
- The surface may show hyperchromatic, stratified nuclei.
- The normal maturation pattern of colonic crypts is disrupted.

Normal epithelium



Adenomatous dysplasia



Original epithelial schematic — not tissue microscopy

Microscopic assessment: Two simultaneous tasks

Classify the epithelium and interrogate the interface.

- First identify glandular/villous architecture and cytologic atypia.
- Then look for invasion of the surrounding lamina propria or deeper tissue.
- Describe grade and invasion independently.

Specimen architecture

Look for tubular, villous, or serrated crypt organization

Cellular details

Identify cytologic dysplasia and stromal invasion

Size is the leading correlate of invasive cancer

Larger adenomas are more likely to contain invasive foci.

- Invasive carcinoma is rare in adenomas smaller than 1 cm.
- Nearly 40% of lesions larger than 4 cm contain invasive cancer.
- The comparison describes cancer found within a polyp, not future annual risk.

Adenoma <1 cm

Invasive carcinoma is rare

Adenoma >4 cm

Nearly 40% contain foci of invasive cancer

Textbook comparison; Not a continuous risk curve

Interpreting the >4 cm observation

An association in detected lesions is not a per-patient forecast.

- The ~40% figure applies to large polyps containing invasive foci at detection.
- It does not imply that 40% of all adenomas will progress.
- Size directs attention to adequate sampling and histology.

Adenoma <1 cm

Invasive carcinoma is rare

Adenoma >4 cm

Nearly 40% contain foci of invasive cancer

Textbook comparison; Not a continuous risk curve

Villous type and cancer probability

Histologic architecture and lesion size are correlated.

- Villous adenomas harbor cancers more often than tubular adenomas.
- Villous lesions also tend to be larger.
- Architecture should not be interpreted in isolation as an independent risk estimate.

Architectural type

Tubular / tubulovillous / villous

Epithelial atypia

Low or high grade dysplasia

High-grade dysplasia: Local significance

High-grade dysplasia increases concern for cancer in that polyp.

- It is a marker of severe epithelial atypia.
- It increases concern for carcinoma within the index lesion.
- Diagnostic evaluation still requires assessment for stromal invasion.

Architectural type

Tubular / tubulovillous / villous

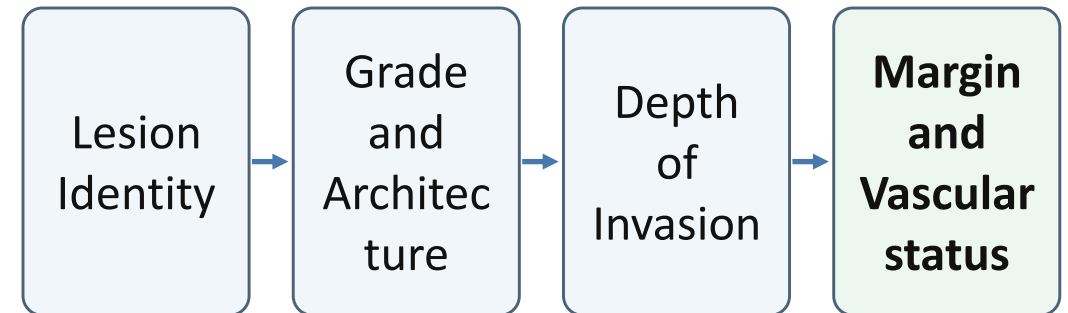
Epithelial atypia

Low or high grade dysplasia

High-grade dysplasia does not predict another polyp

Separate the index-lesion finding from patient-wide assumptions.

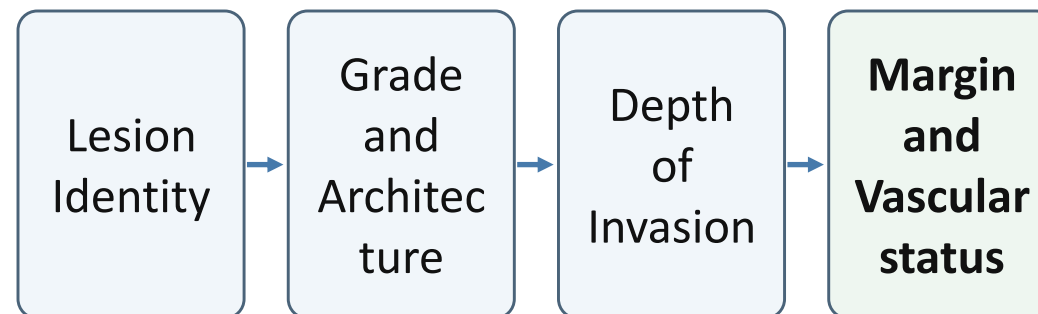
- The source states that high-grade dysplasia does not independently increase cancer risk in other polyps.
- This distinction prevents extrapolation from one adenoma to all others.
- Each detected lesion needs its own classification and assessment.



Adenoma risk synthesis

Combine separate observations rather than rely on one label.

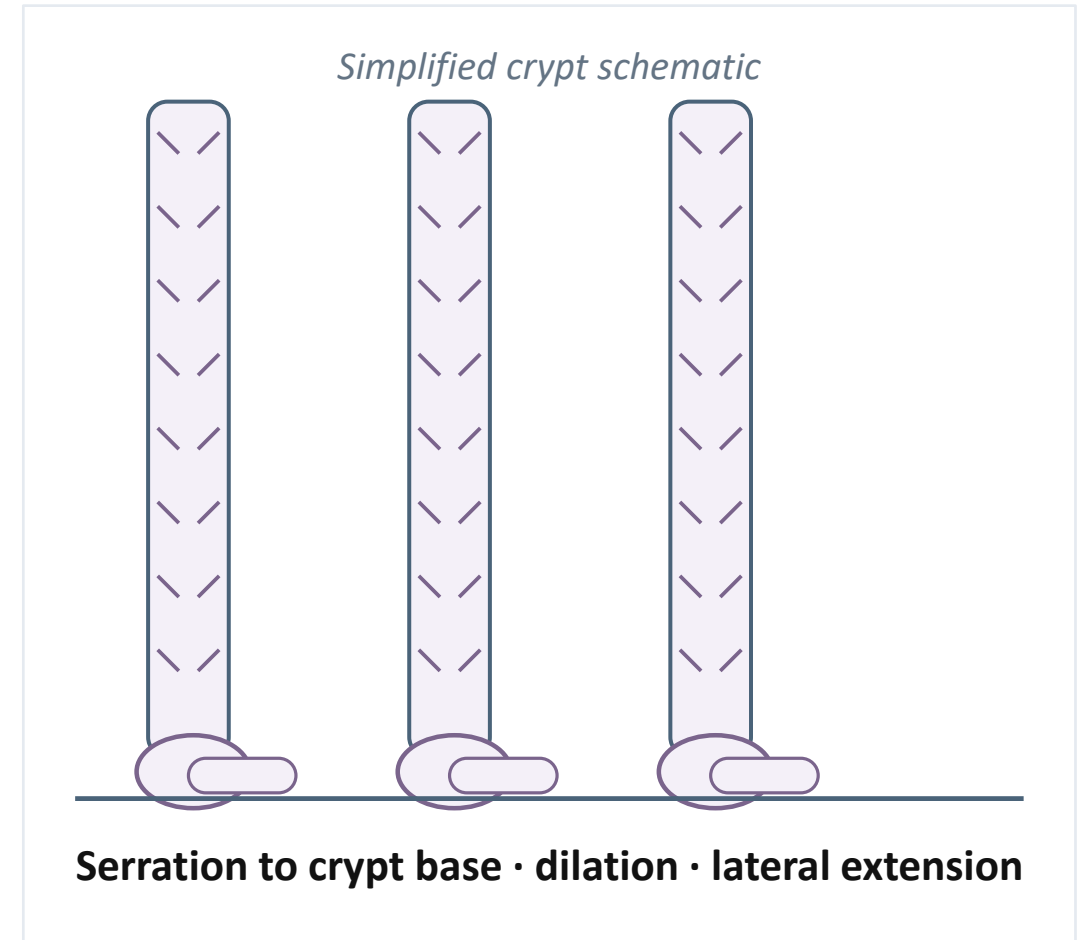
- Size is a strong correlate of invasive foci.
- High-grade dysplasia raises concern in the sampled lesion.
- Architecture describes growth pattern and covaries with size.
- Actual invasion determines whether adenocarcinoma is present.



The serrated precursor category

Not every premalignant colonic polyp looks like a conventional adenoma.

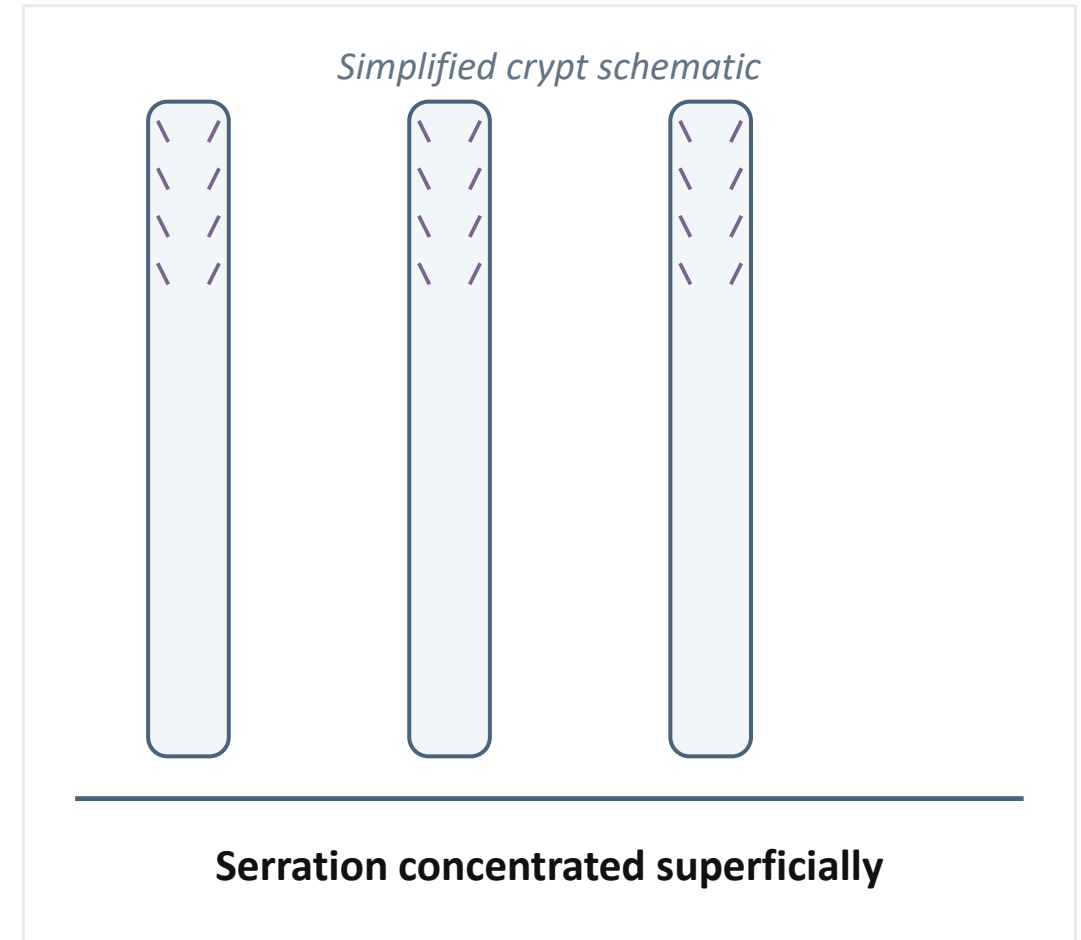
- Sessile serrated polyps are premalignant epithelial lesions.
- They overlap in appearance with hyperplastic polyps.
- They often lack classic adenomatous cytologic dysplasia.



Hyperplastic polyp: Benign serration

A hyperplastic polyp is not equivalent to a sessile serrated polyp.

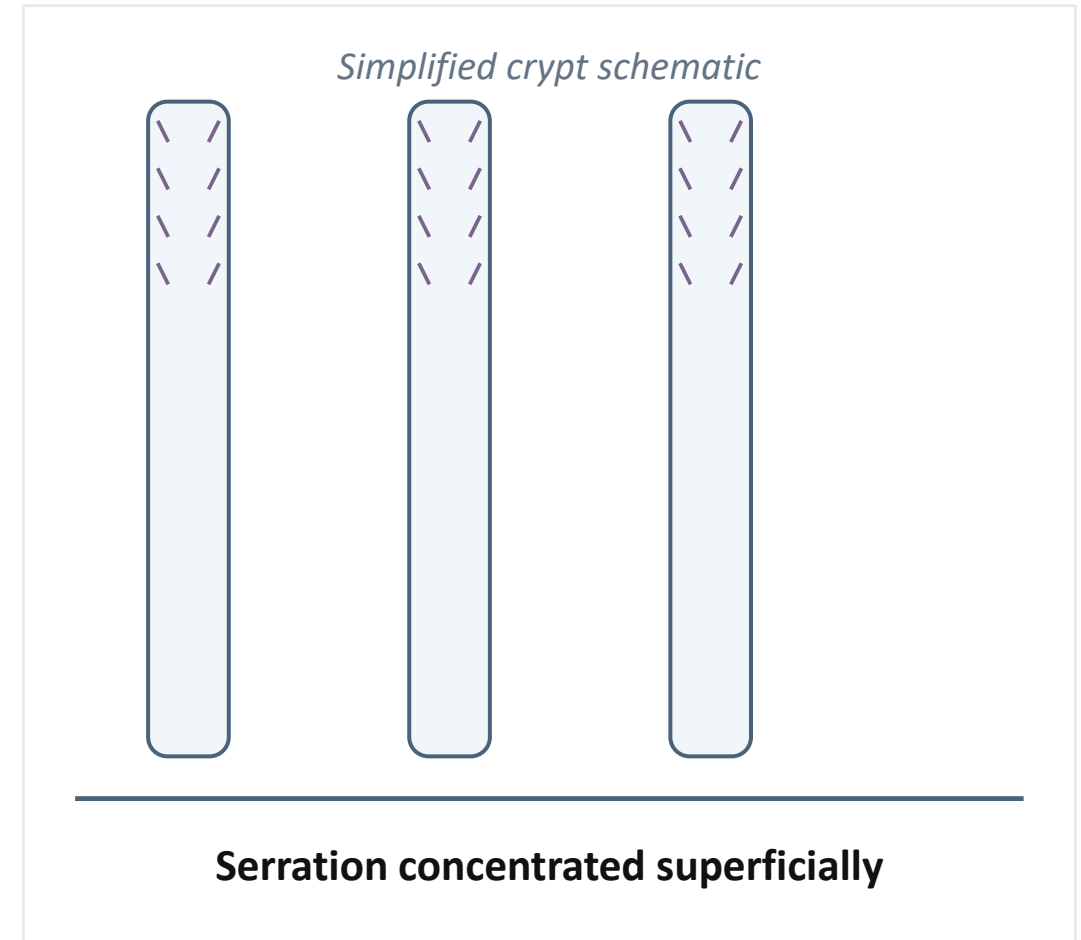
- Usually a small lesion, commonly in the left colon.
- Contains mature goblet and absorptive cells.
- No malignant potential for these lesions.



Hyperplastic architecture is superficial

Upper-crypt serration is the defining distribution.

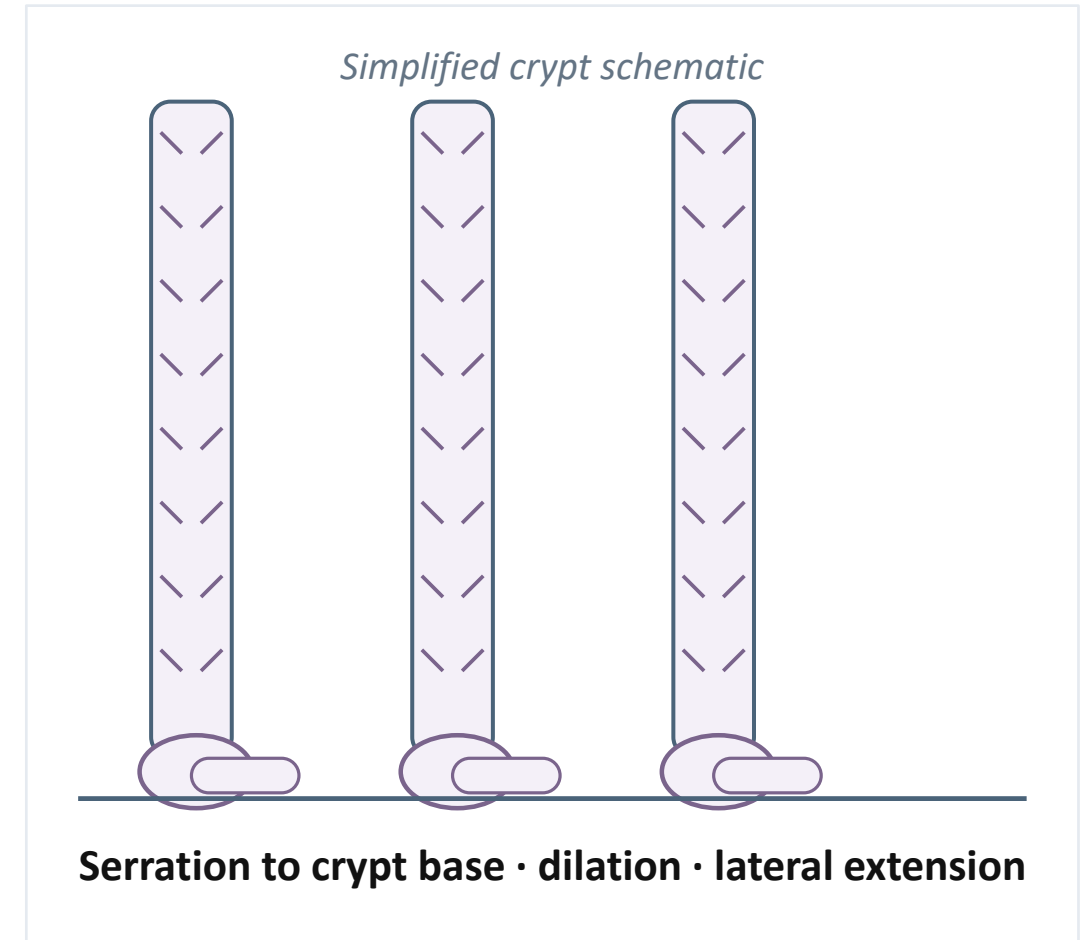
- Sawtooth epithelial folds are mainly in the upper crypt.
- Lower crypts preserve their more regular configuration.
- The crypt base helps distinguish this from a sessile serrated lesion.



Sessile serrated polyp: Basal crypt involvement

Abnormal architecture reaches the crypt bases.

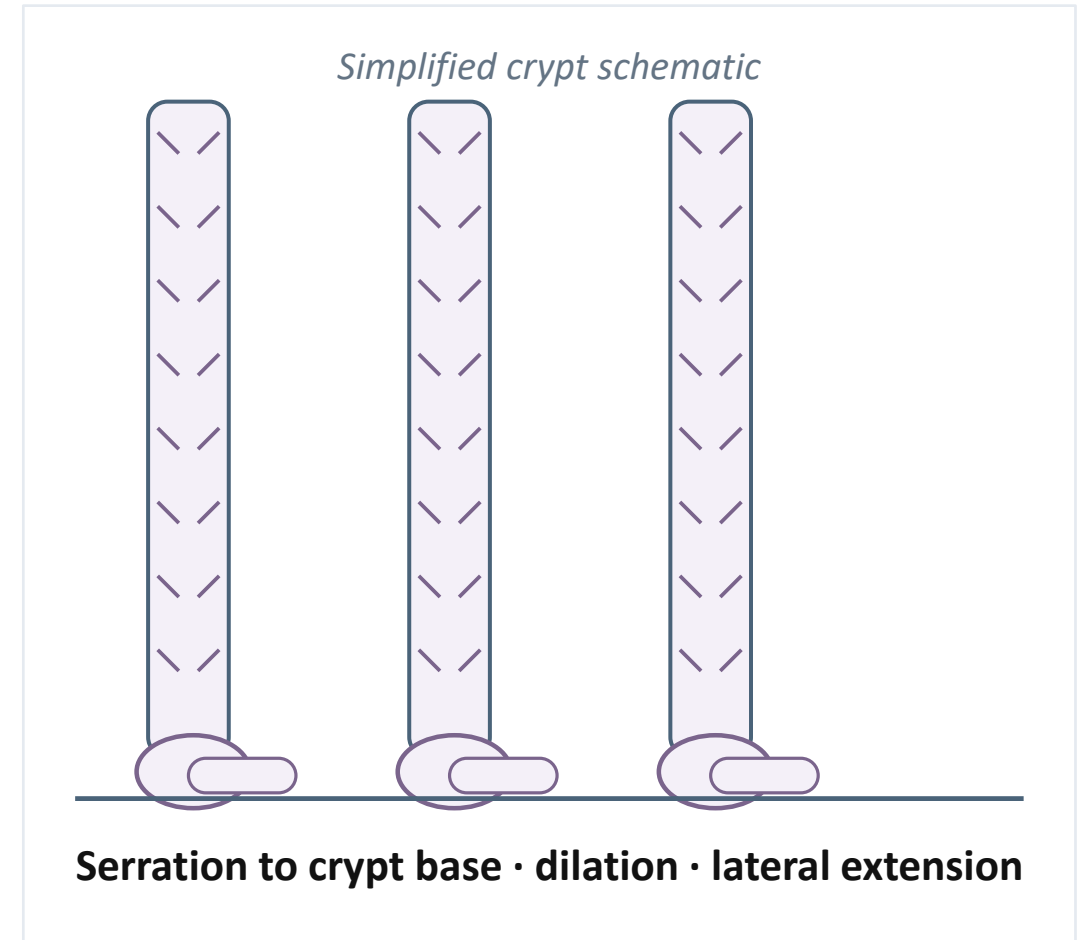
- Serration extends along the full gland length.
- Basal crypt dilation is characteristic.
- The gland bases may extend laterally.



Lateral crypt growth is diagnostically useful

Basal horizontal extension changes crypt topology.

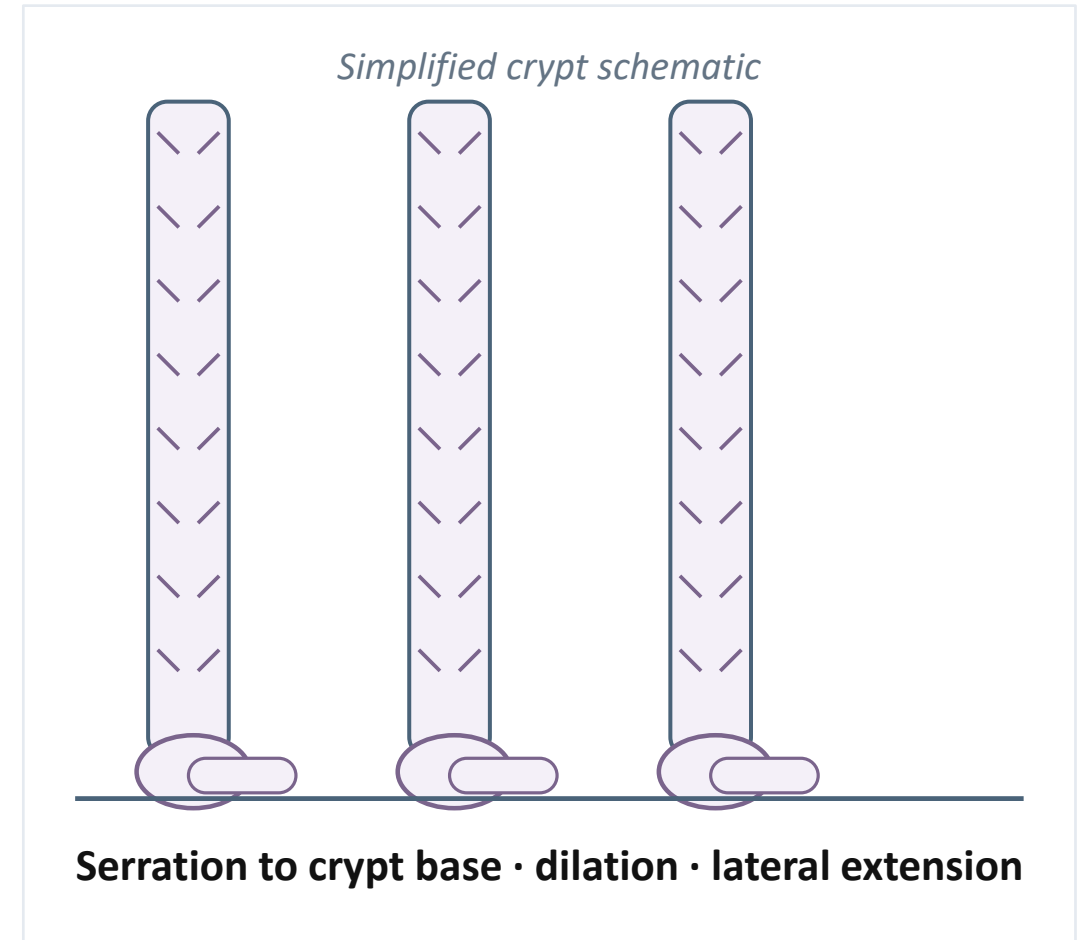
- Crypts can run along the muscularis mucosae instead of vertically.
- Basal dilation and full-length serration strengthen recognition.
- The distinction is architectural even without overt cytologic dysplasia.



Right-sided tendency of sessile serrated polyps

Anatomic site adds context, not a standalone diagnosis.

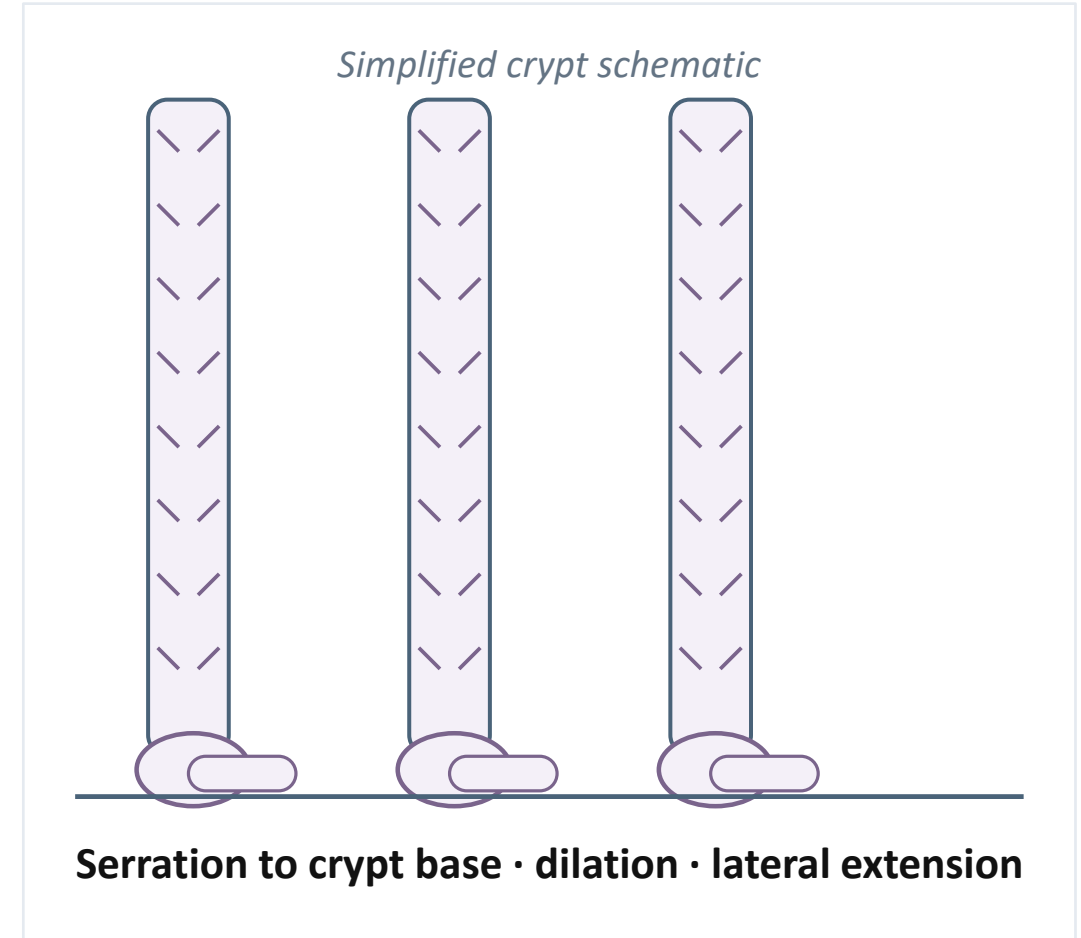
- Sessile serrated polyps are more often in the right colon.
- Hyperplastic polyps more often occur in the left colon.
- Location cannot replace full crypt-base assessment.



Why cytology alone can miss a serrated precursor

A lack of conventional dysplasia does not prove benignity.

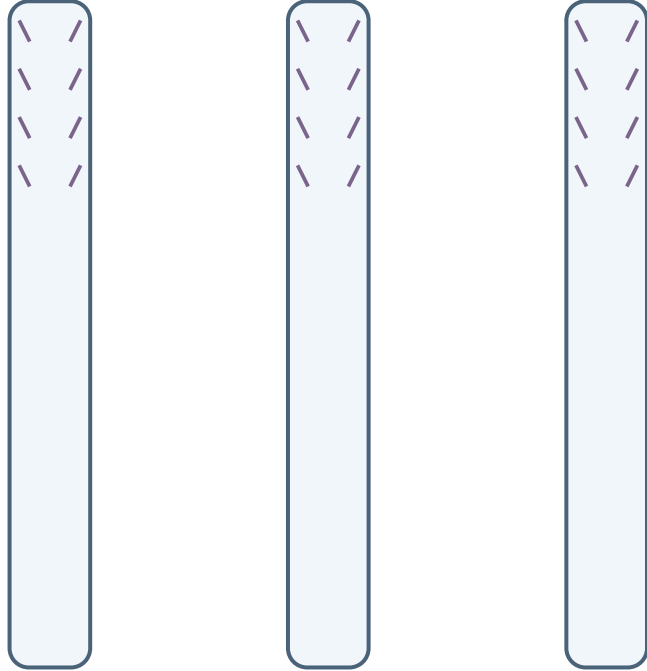
- Sessile serrated polyps may retain bland-appearing epithelial nuclei.
- Malignant potential is indicated by abnormal crypt architecture.
- This is a key exception to the usual adenoma diagnostic pattern.



Hyperplastic versus sessile serrated polyp

The critical distinction is the entire crypt, especially its base

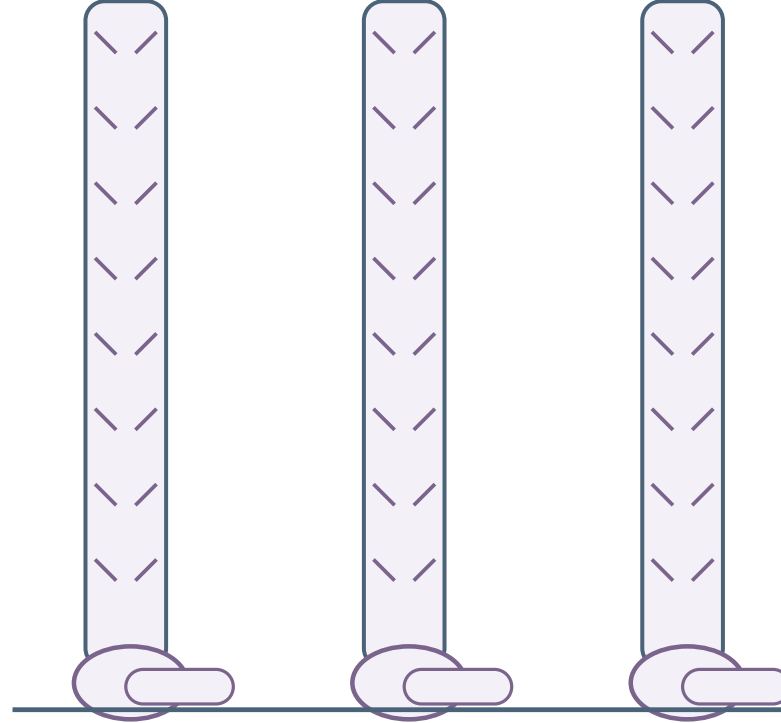
Simplified crypt schematic



Serration concentrated superficially

HYPERPLASTIC

Simplified crypt schematic



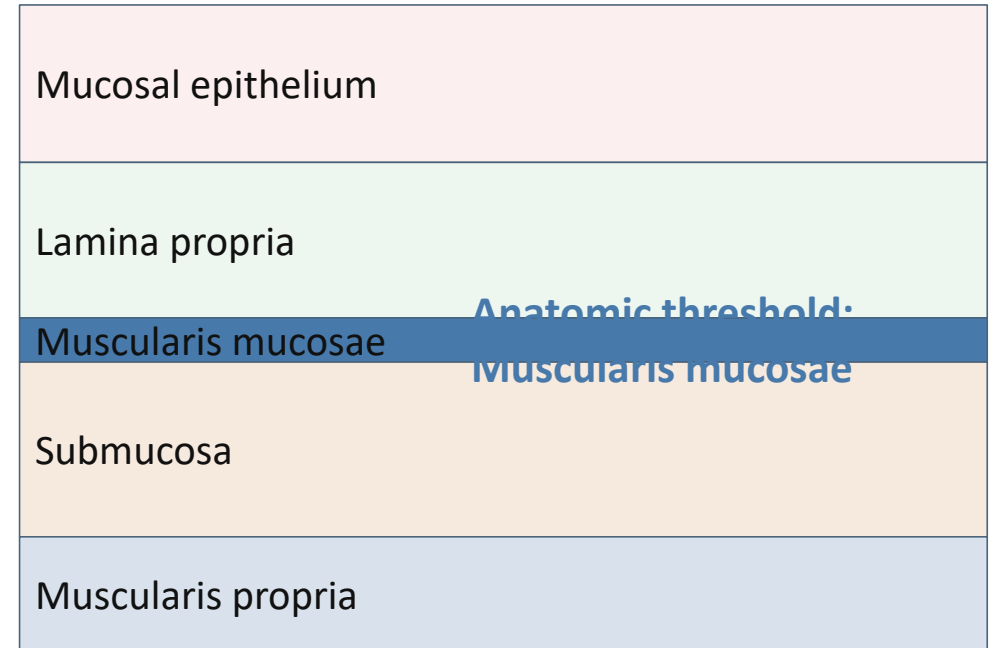
Serration to crypt base • dilation • lateral extension

SESSILE SERRATED

Normal colonic wall: Map the boundaries

Depth changes biological behavior.

- Epithelium rests on basement membrane over lamina propria.
- Muscularis mucosae separates mucosa from submucosa.
- Invasion beyond this boundary carries different metastatic implications.

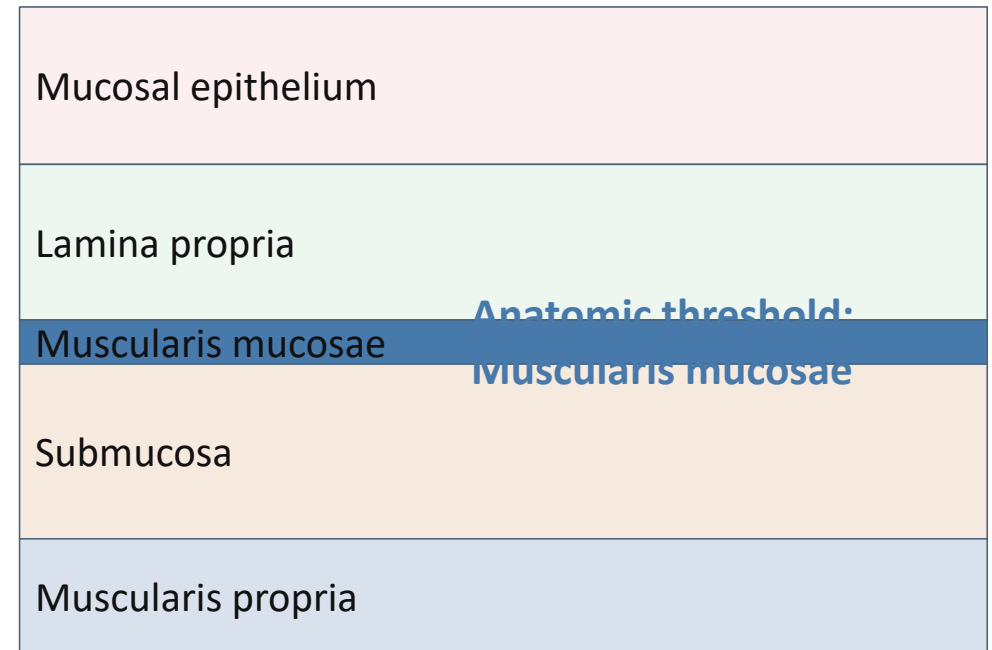


Schematic, not a histologic section

Dysplasia remains intraepithelial

Nuclear atypia alone does not establish stromal invasion.

- Dysplastic cells remain in the epithelial compartment.
- Preserved epithelial boundaries distinguish dysplasia from intramucosal carcinoma.
- Examine the gland–stroma relationship, not just cytologic severity.

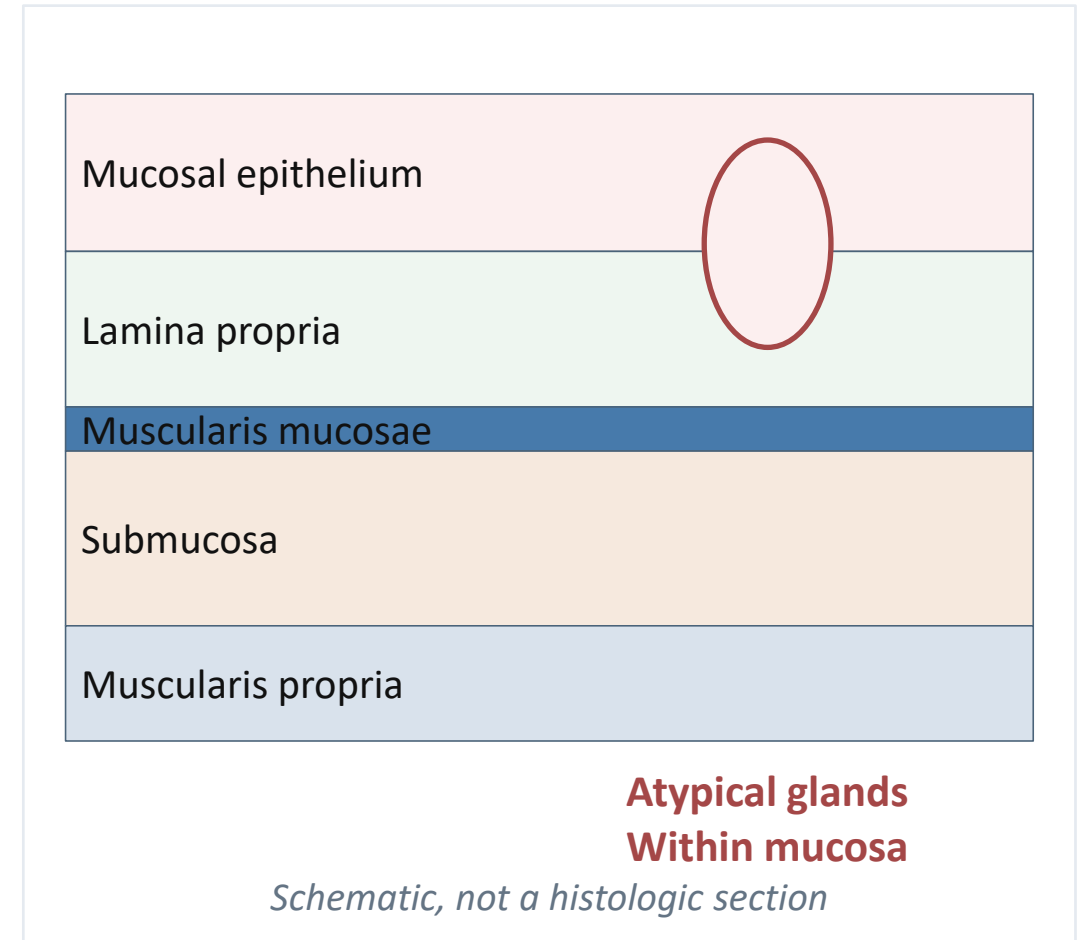


Schematic, not a histologic section

Intramucosal carcinoma: Breached membrane

Dysplastic epithelium invades lamina propria.

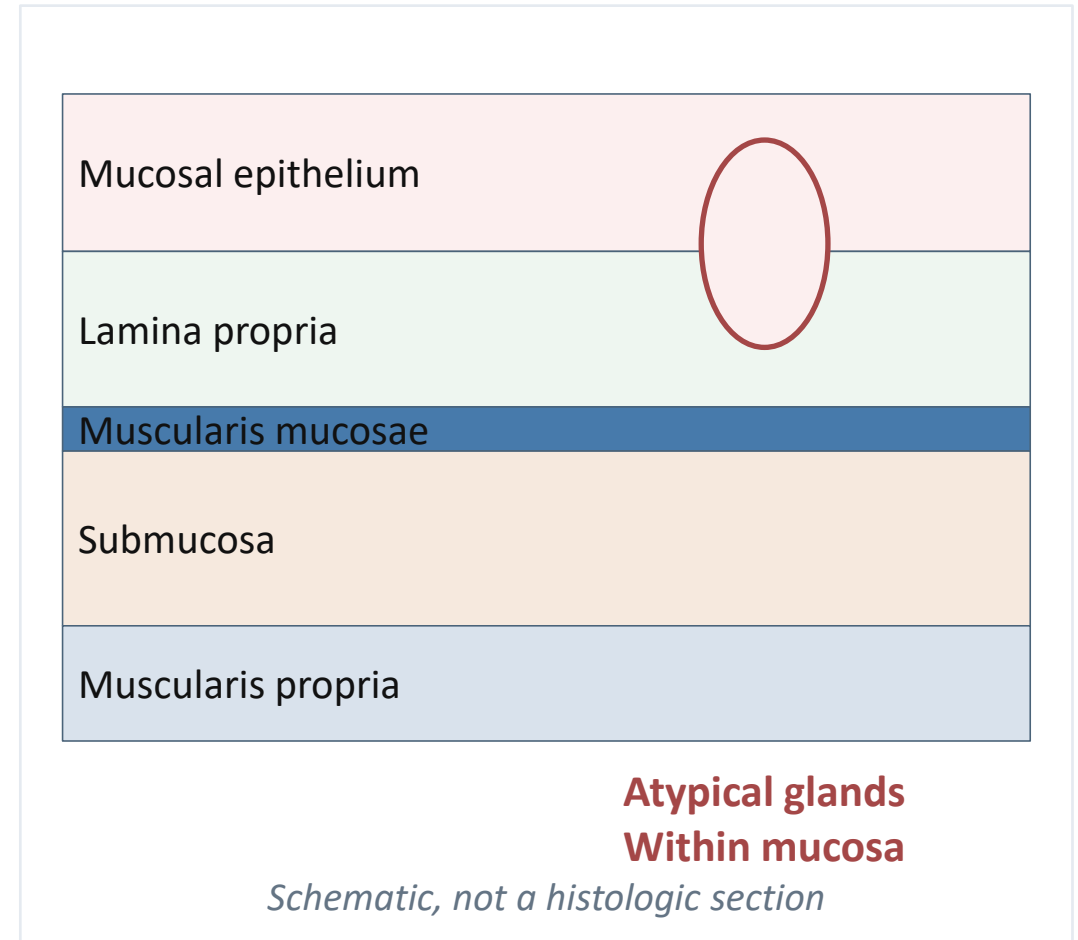
- Cells cross the epithelial basement membrane.
- Invasion remains confined to the colonic mucosa.
- It may extend into, but not through, the muscularis mucosae.



Intramucosal carcinoma and dissemination

Anatomic lymphatic distribution explains the clinical distinction.

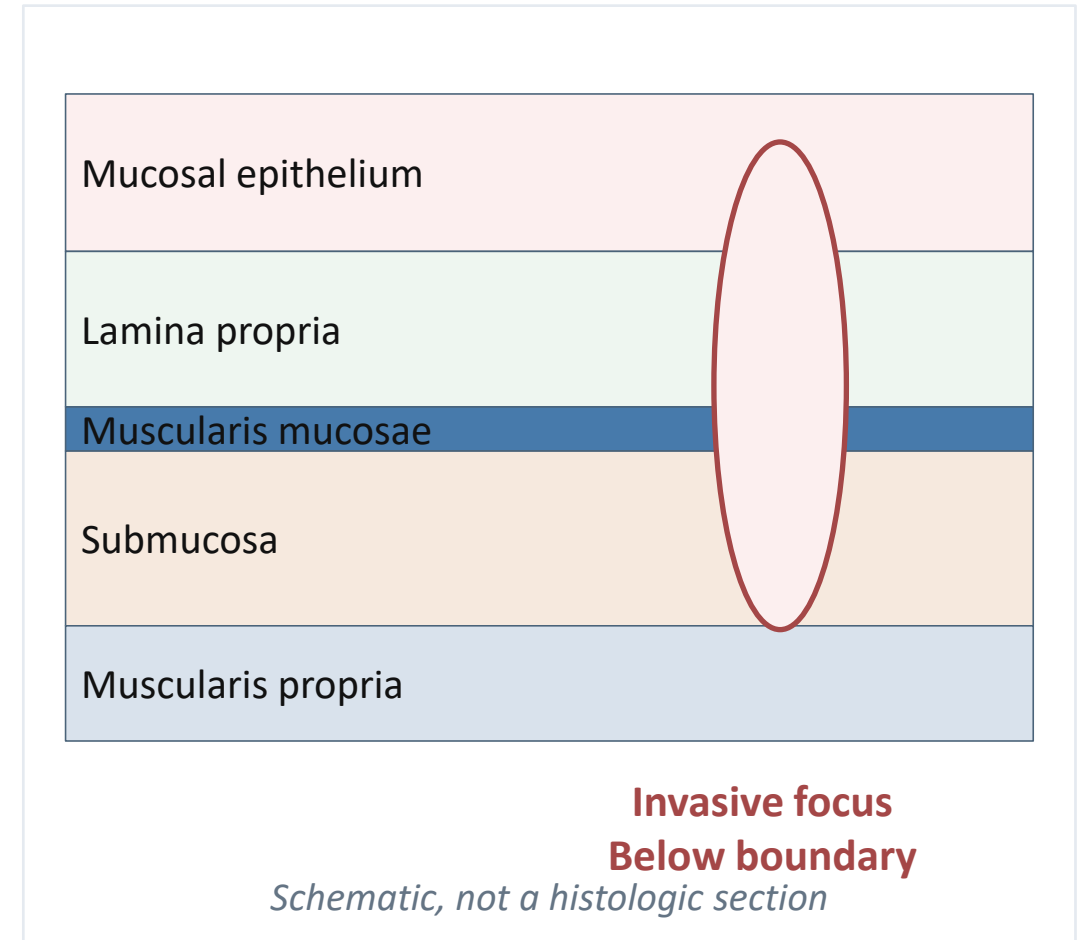
- Functional lymphatic channels are absent from colonic mucosa account.
- Intramucosal carcinoma consequently has little or no metastatic potential.
- Complete polypectomy is generally curative in this situation.



Submucosal invasion marks invasive adenocarcinoma

Crossing the muscularis mucosae introduces metastatic risk.

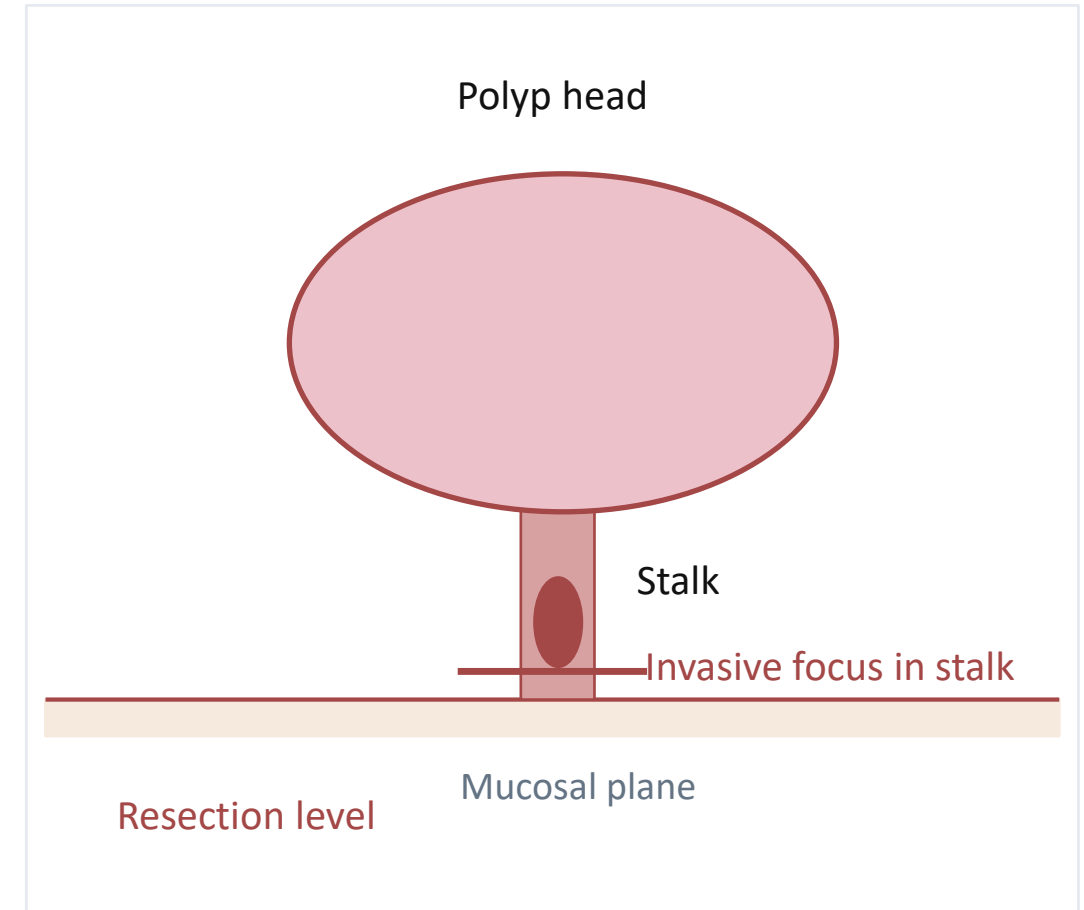
- Neoplastic glands extend into submucosal tissue.
- The lesion is invasive adenocarcinoma, including in a stalk.
- Additional pathologic factors are needed to plan management.



Pedunculated polyp: Invasion may enter the stalk

A polyp head may conceal deeper invasion.

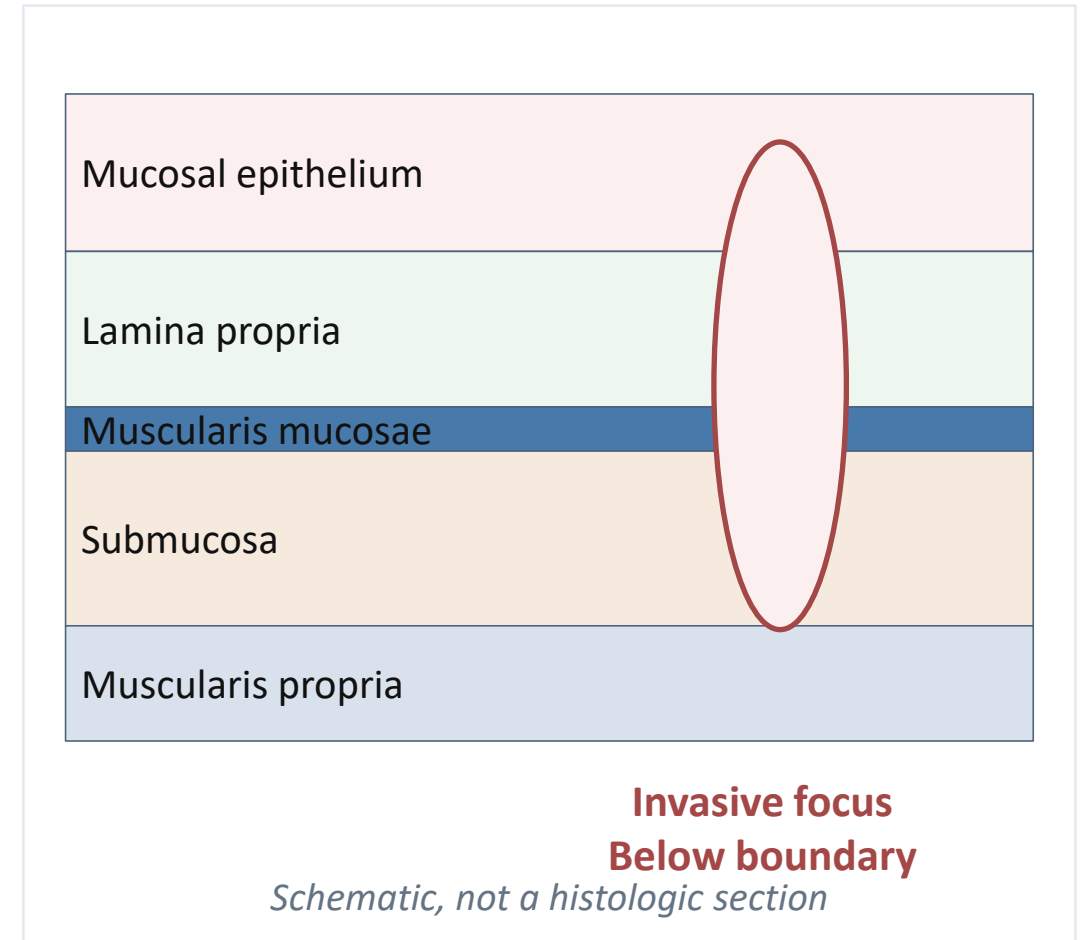
- The stalk contains submucosal tissue and vascular structures.
- Invasive glands in the submucosal stalk constitute adenocarcinoma.
- The relationship to the resection margin must be determined.



Desmoplasia supports invasive growth

Stromal response may accompany the invasive component.

- The source illustrates a desmoplastic reaction around invading carcinoma.
- This interface contrasts with noninvasive adenomatous epithelium.
- Morphologic interpretation should consider native anatomic layers.



Cancer within a polyp: Clinical categorization

“Malignant polyp” implies invasive adenocarcinoma.

- Noninvasive dysplasia is managed differently from true submucosal invasion.
- Describe the depth and accompanying adverse histologic features.
- Further management is individualized rather than inferred from the word “polyp.”

Mucosal invasion

Minimal metastatic potential in colon

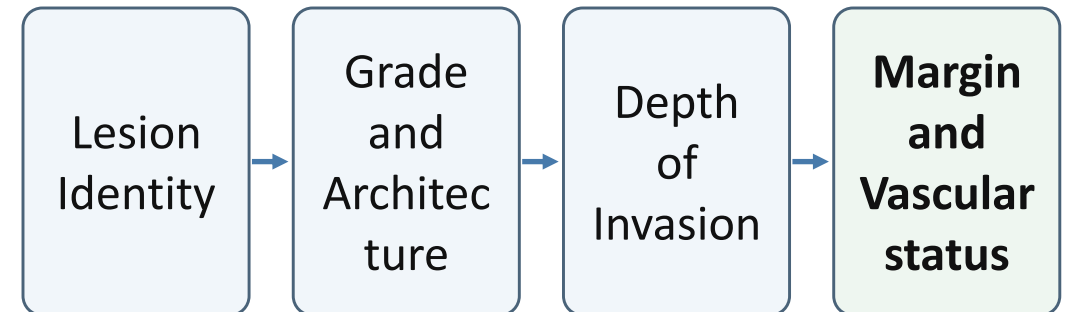
Submucosal invasion

Potential lymphovascular access and spread

Invasive component: Histologic grade matters

Grade is one input to post-polypectomy assessment.

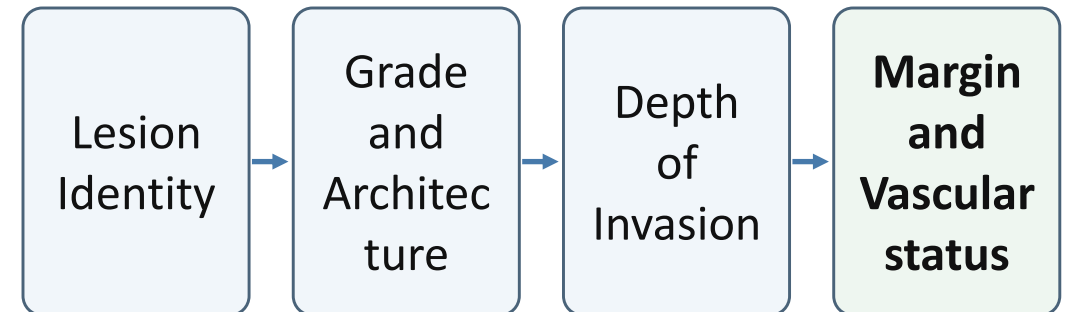
- Assess differentiation within the invasive carcinoma component.
- Grade is not the same concept as adenoma architecture.
- Combine grade with invasion, vascular status, and clearance.



Vascular or lymphatic invasion

Detection of tumor in channels changes risk assessment.

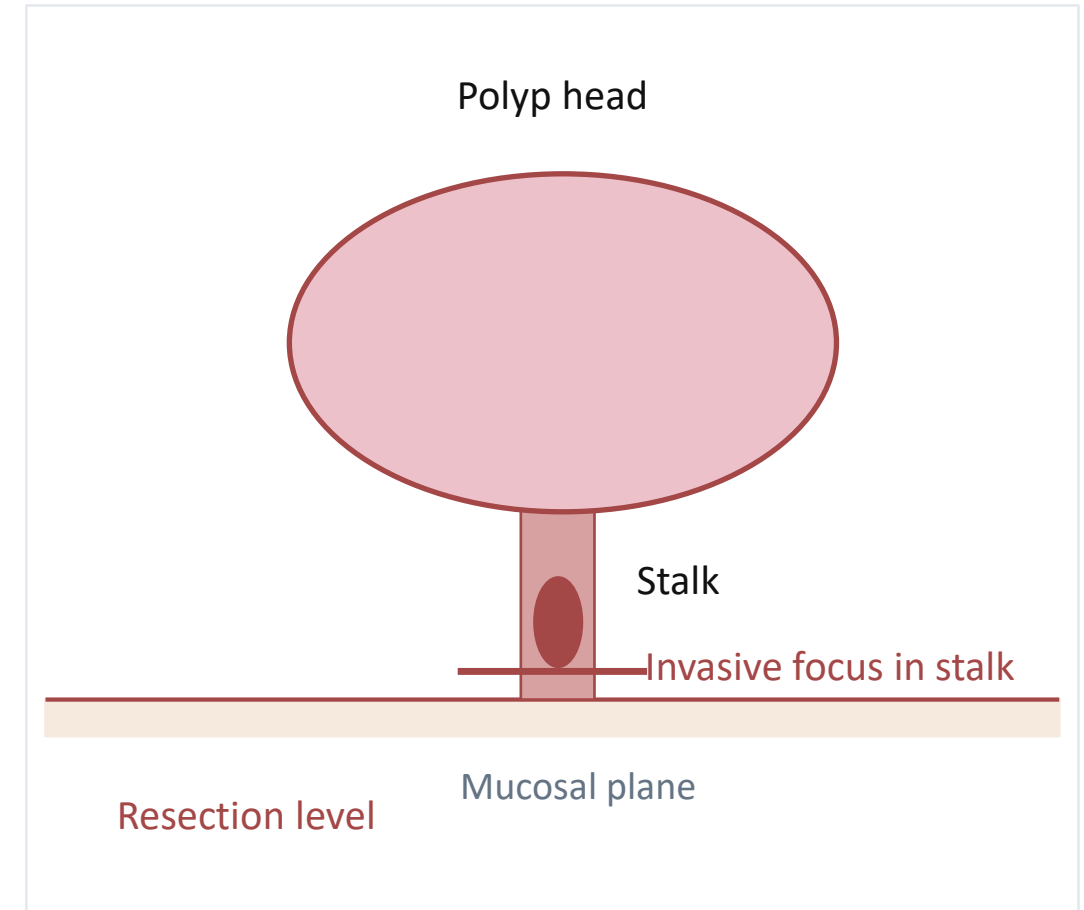
- Identify vascular or lymphatic invasion within malignant-polyp tissue.
- Its presence is considered during further therapy planning.
- A polyp cannot be classified as low risk from size alone.



Resection margin and tumor clearance

Distance to the invasive focus is clinically consequential.

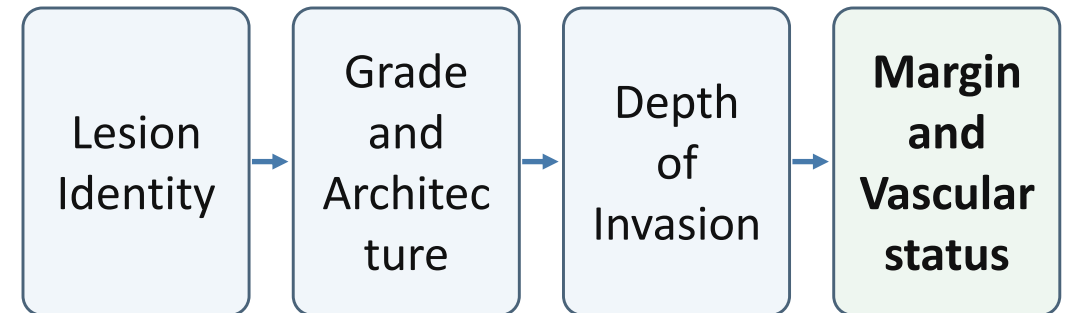
- Examine how close invasive carcinoma approaches the excision margin.
- Interpret this alongside invasive grade and lymphovascular involvement.
- A technically removed polyp is not automatically an adequately treated invasive carcinoma.



Pathology handoff after polypectomy

A useful report separates descriptive and decision-relevant findings.

- Polyp type and architectural pattern.
- Dysplasia grade and presence or absence of invasion.
- If invasive: Grade, lymphovascular invasion and resection margin relationship.

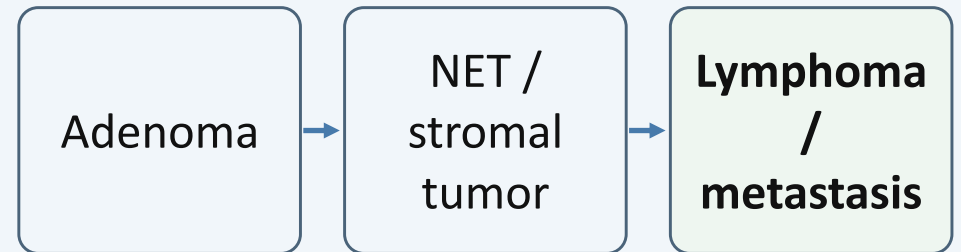


A polypoid malignant mass has many lineages

Avoid labeling every neoplastic protrusion an adenoma.

- Polypoid adenocarcinoma can mimic a precursor lesion grossly.
- Neuroendocrine, stromal and lymphoid neoplasms can be polypoid.
- Metastases may also present as gastrointestinal mass lesions.

Polyp = growth configuration

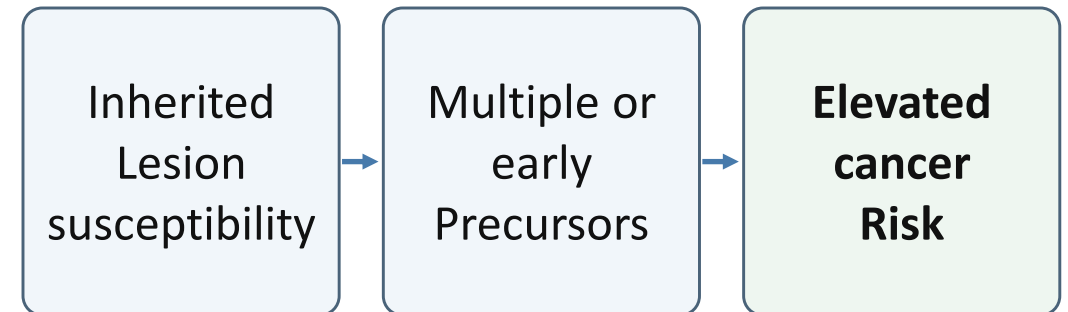


Morphology alone does not establish lineage

Hereditary syndromes reveal precursor biology

Familial lesions provide mechanistic context, not a replacement for morphologic diagnosis.

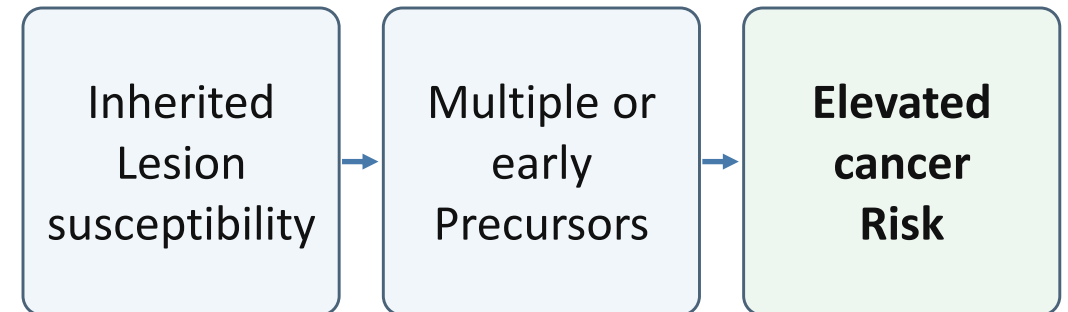
- APC pathway disruption underlies familial adenomatous polyposis.
- Defective DNA mismatch repair underlies Lynch syndrome.
- Different inherited pathways converge on colorectal malignancy risk.



Familial adenomatous polyposis: Brief context

Polyp burden makes the precursor mechanism visible.

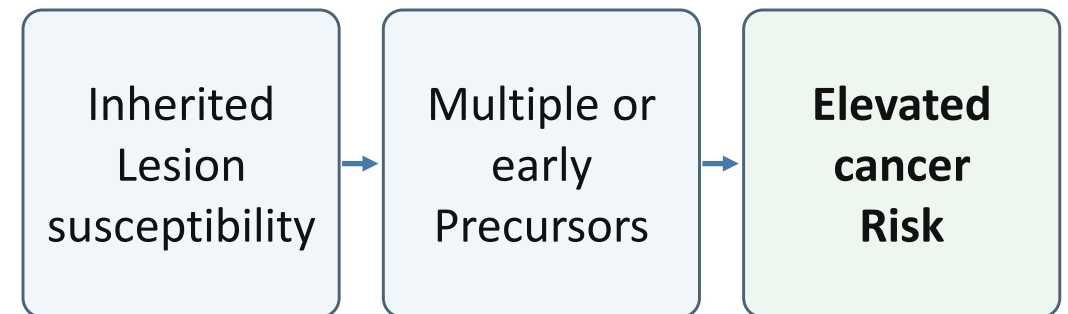
- Autosomal dominant APC-associated syndrome produces numerous adenomas.
- Classic FAP requires at least 100 polyps in the cited textbook.
- Sporadic and FAP-associated adenomas are morphologically alike.



Lynch syndrome: Mismatch-repair context

Carcinogenesis may occur without an extensive polyposis phenotype.

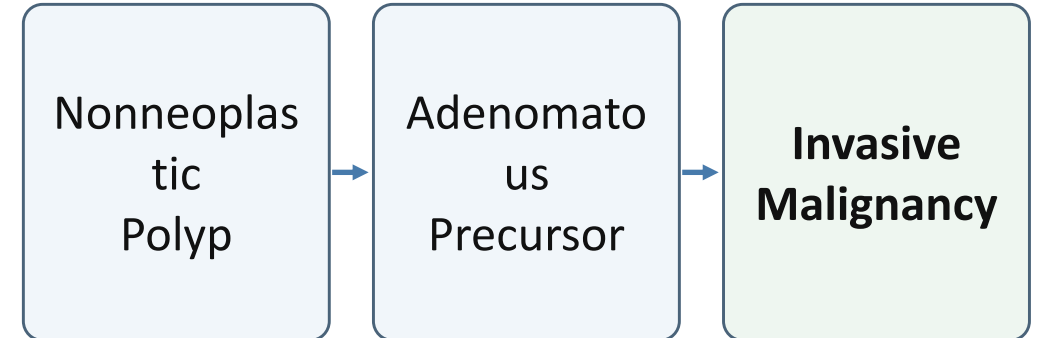
- Inherited defects in mismatch-repair genes produce microsatellite instability.
- Colon cancers frequently arise at relatively young ages and on the right.
- The hereditary syndrome is explored in its separate Robbins unit.



Hamartomatous polyps: Important exceptions

A histologically benign-looking polyp may signal syndromic cancer risk.

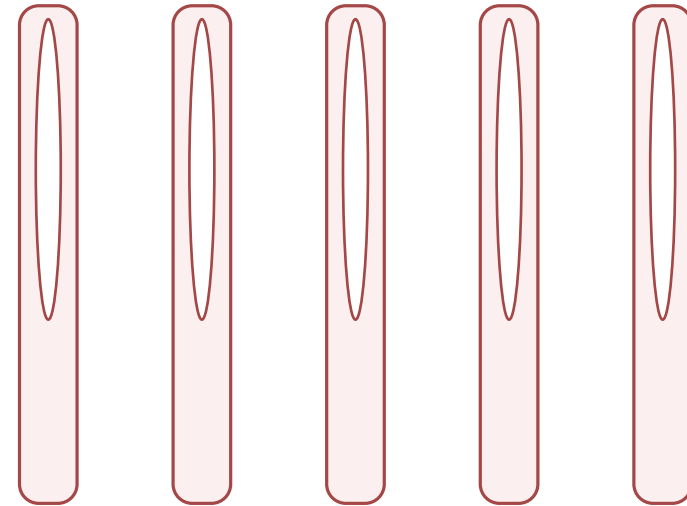
- Sporadic juvenile polyps and juvenile polyposis syndrome differ in risk.
- Peutz–Jeghers syndrome includes characteristic polyps and increased malignancy risk.
- Clinical phenotype and family history help identify these conditions.



Case: A small pedunculated adenoma

Clinical interpretation: A polyp can be neoplastic without being invasive.

- A short stalk supports pedunculated gross configuration.
- Crowded rounded glands with dysplastic nuclei support tubular adenoma.
- No invasion is established by architectural subtype alone.

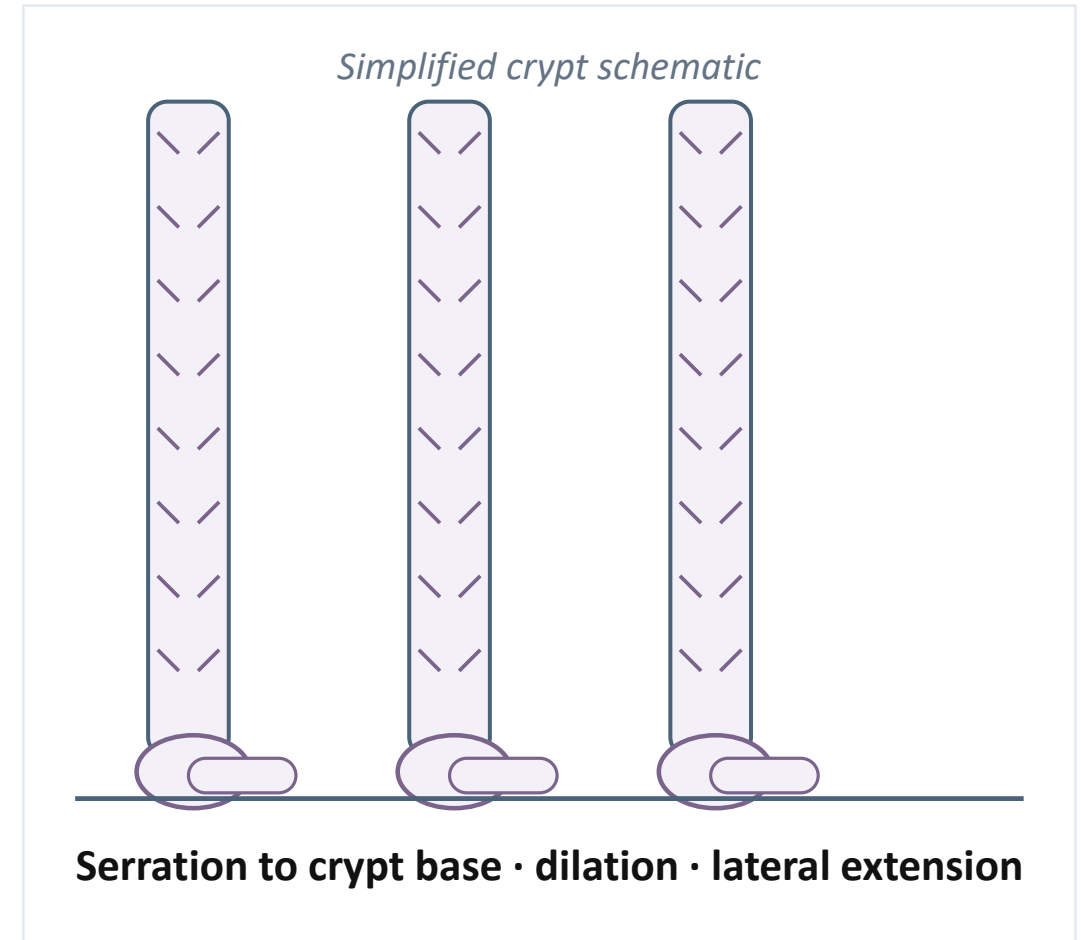


Original simplified architecture schematic — not a photomicrograph

Case: A right-sided flat serrated lesion

What must be seen at the crypt base?

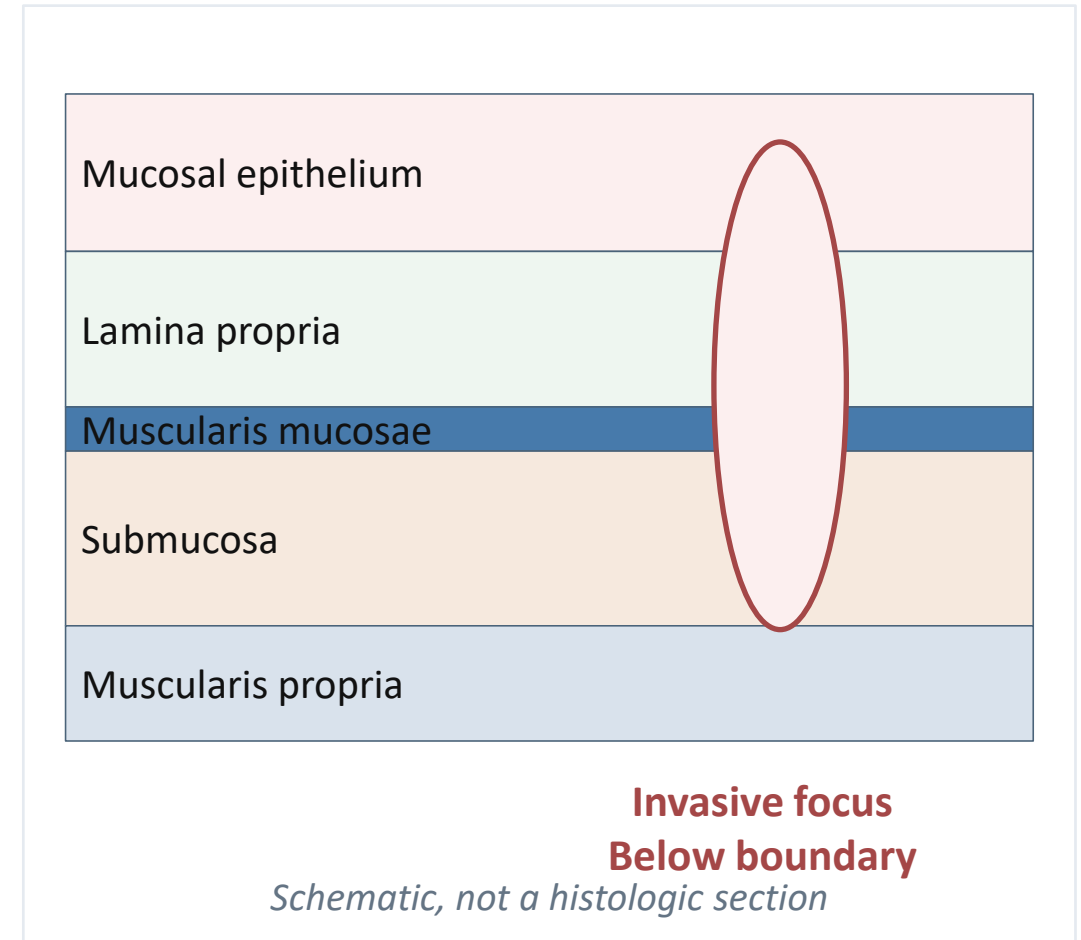
- Bland cytology does not exclude a premalignant serrated lesion.
- Full-length serration, dilation and lateral base extension favor sessile serrated polyp.
- Upper-crypt-only serration supports a hyperplastic lesion.



Case: Intramucosal versus submucosal carcinoma

Locate the deepest tumor glands relative to the muscularis mucosae.

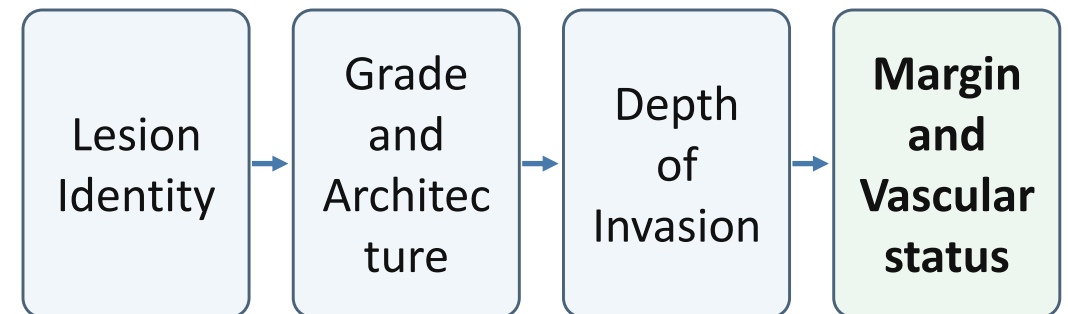
- Lamina-propria infiltration is intramucosal carcinoma.
- Invasion beyond muscularis mucosae constitutes invasive adenocarcinoma.
- The distinction changes the significance of polypectomy and potential spread.



Integrated diagnostic decision sequence

Use a stable order for endoscopic and pathology information.

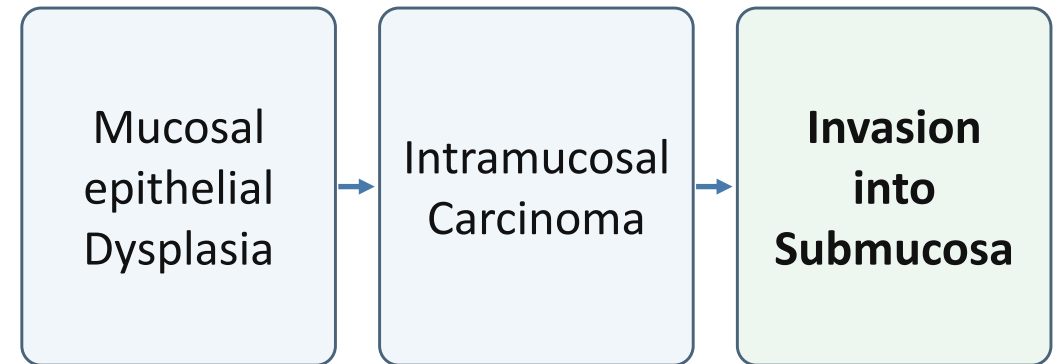
- Identify histologic lineage, growth pattern and dysplasia.
- Search for carcinoma and document exact anatomic depth.
- For submucosal invasion, evaluate grade, channels and margin.
- Relate findings to appropriate clinical follow-up.



Synthesis: What determines clinical meaning?

Biology is established by histology and anatomic relationships.

- An adenoma is a precursor; Most never become carcinoma.
- Invasive risk increases with lesion size and some advanced features.
- Serrated precursors may lack classic cytologic dysplasia.
- Submucosal invasion creates metastatic potential.



Primary source and figure provenance

Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).

- Chapter 17, The Gastrointestinal Tract: intestinal polyps, adenomas and the premalignant/malignant lesions section (pp. 735–741).
- Adjacent FAP and Lynch syndrome sections were used only for brief context.
- Illustrations are original, editable educational schematics; no diagnostic image is presented as a real specimen.