

Gastric Adenoma

Morphology • dysplasia • malignant potential

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning outcomes

Place gastric adenoma within the spectrum of gastric polyps.

Explain its links to atrophic gastritis, intestinal metaplasia, and inherited susceptibility.

Recognize dysplastic cytology, high-grade architecture, and features that influence cancer risk.

Distinguish adenoma from common reactive and fundic gland polyps.

Adenoma is not merely a raised mucosal lesion

Gastric polyp

A protruding lesion seen at endoscopy.
May reflect inflammation, hyperplasia, ectopia, or neoplasia.

Gastric adenoma

Epithelial neoplastic polyp with dysplasia.
Histologic examination establishes its character.

How often are gastric polyps encountered?

$\leq 5\%$

Of upper GI endoscopies

The endoscopic designation “polyp” describes gross configuration.

Histology separates common reactive proliferations from adenomas.

Three relevant epithelial polyp categories

Hyperplastic / inflammatory

Often linked to chronic gastritis.

Foveolar gland elongation,
dilation, inflammation.

Fundic gland

Usually body or fundus.

Cystic glands with oxyntic
elements.

Adenoma

Typically antral.

Dysplastic epithelial proliferation.

Most gastric polyps are not adenomas

≤75%

Inflammatory / hyperplastic

Together these account for up to three quarters of gastric polyps.

Their shared associations do not make them identical to an adenoma.

Gastric adenomas: A minority of polyps

$\leq 10\%$

Of gastric polyps

A frequency of up to 10%.

This denominator is gastric polyps—not endoscopies or all gastric cancers.

Endoscopic appearance versus histologic identity

Endoscopy clarifies

Whether a raised lesion is present.

Site, size, multiplicity, and surface contour.

Histology clarifies

Whether epithelial dysplasia is present.

Dysplasia grade and glandular architecture.

Typical age at presentation

50–60

Years of age

Frequency increases progressively with age.

The quoted decade describes usual patients; It is not an exclusion criterion.

Male predominance

3 : 1

Male-to-female ratio

Approximately three times as many males as females.

The ratio is epidemiologic; Sex does not determine the diagnosis in an individual.

Geographic variation parallels gastric cancer

Gastric adenoma prevalence differs among populations.

This population pattern parallels the incidence of gastric adenocarcinoma.

A parallel distribution does not mean every adenoma advances to carcinoma.

Interpretation

Shared risk context is plausible.

The source does not quantify progression for every lesion.

Gastric adenomas usually arise in the antrum



Regional schematic; Not an anatomic drawing or a lesion distribution map

Gastric adenomas are usually solitary lesions in the antrum.

Fundic gland polyps are typically found in the body and fundus.

Multiplicity provides diagnostic context

Typical gastric adenoma

Usually a solitary lesion.

Commonly located in the gastric antrum.

Other gastric polyp patterns

Hyperplastic polyps may be multiple.

Fundic gland polyps may be single or multiple.

Sporadic adenomas usually have an injured mucosal field

Most arise against chronic gastritis.

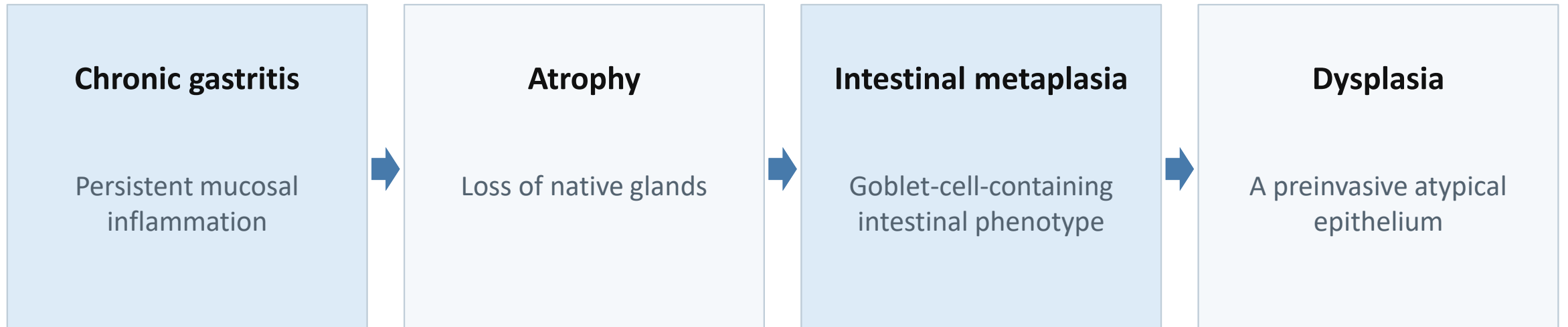
Associated changes include glandular atrophy and intestinal metaplasia.

These changes provide histologic context for interpreting the lesion.

Important exception

Inherited APC- or MUTYH-associated adenomas need not follow this usual background.

Atypical epithelium may emerge after chronic injury



Schematic of associated tissue states; This is not an inevitable linear fate for every patient.

Atrophy changes the glandular compartment

Chronic gastritis can cause substantial loss of acid-producing parietal cells.

Oxyntic gland atrophy frequently accompanies intestinal metaplasia.

The atrophic background signals a mucosa at elevated risk of gastric neoplasia.

Intestinal metaplasia has a visible cellular clue

Goblet cells identify intestinal-type metaplasia in gastric mucosa.

Metaplasia is associated with increased risk of gastric adenocarcinoma.

Metaplasia alone is not equivalent to an adenoma: Dysplasia must be established.

Metaplasia and adenoma answer different questions

Intestinal metaplasia

A change in epithelial differentiation.
Recognizable by intestinal-type goblet cells.

Adenoma

A localized dysplastic epithelial neoplasm.
Characterized by nuclear and glandular atypia.

Inflammatory injury contributes to genetic stress

Chronic gastritis exposes epithelium to inflammation-related free radicals.

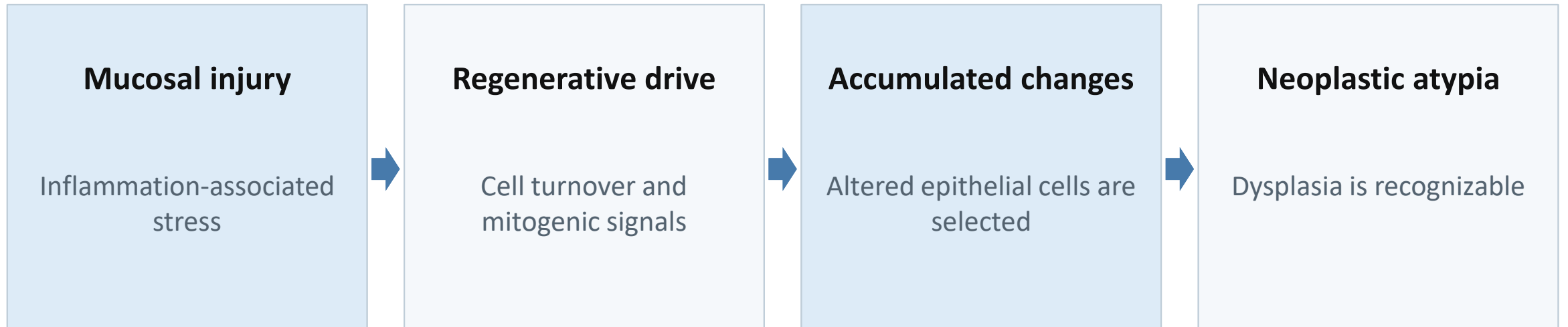
Regeneration and increased gastrin add proliferative stimuli.

Over time, genetic alterations can accumulate and support neoplasia.

Scope

This explanation describes the chronic-gastritis field, not a molecular signature unique to gastric adenoma.

Pathogenesis: Injury, proliferation, genetic change



Mechanistic teaching sequence; Specific molecular drivers for gastric adenoma are not assigned in this source.

APC and MUTYH define important exceptions

Some gastric adenomas occur with germline APC mutations.

Others occur with germline MUTYH mutations.

These from the typical atrophy–metaplasia-associated setting.

Sporadic context versus inherited context

Usual setting

Chronic gastritis in the surrounding mucosa.
Atrophy and intestinal metaplasia may coexist.

Germline APC / MUTYH setting

An inherited genetic predisposition is present.
The usual atrophic/metaplastic backdrop is not required.

Do not equate a fundic gland polyp with an adenoma

APC/MUTYH disorders are also discussed with fundic gland polyps.

Fundic gland polyps have cystically dilated oxyntic-type glands.

A shared inherited setting does not erase distinct polyp histology.

Diagnostic principle

Classify the tissue actually sampled; Do not diagnose by syndrome association alone.

Dysplasia is obligatory in gastrointestinal adenomas

All gastrointestinal adenomas demonstrate dysplasia.

Dysplasia is graded as low-grade or high-grade.

Grading integrates cytologic atypia with glandular architecture.

The nuclear abnormalities of gastric adenoma

Size and shape

Enlarged nuclei.
Elongated nuclear contours.

Arrangement

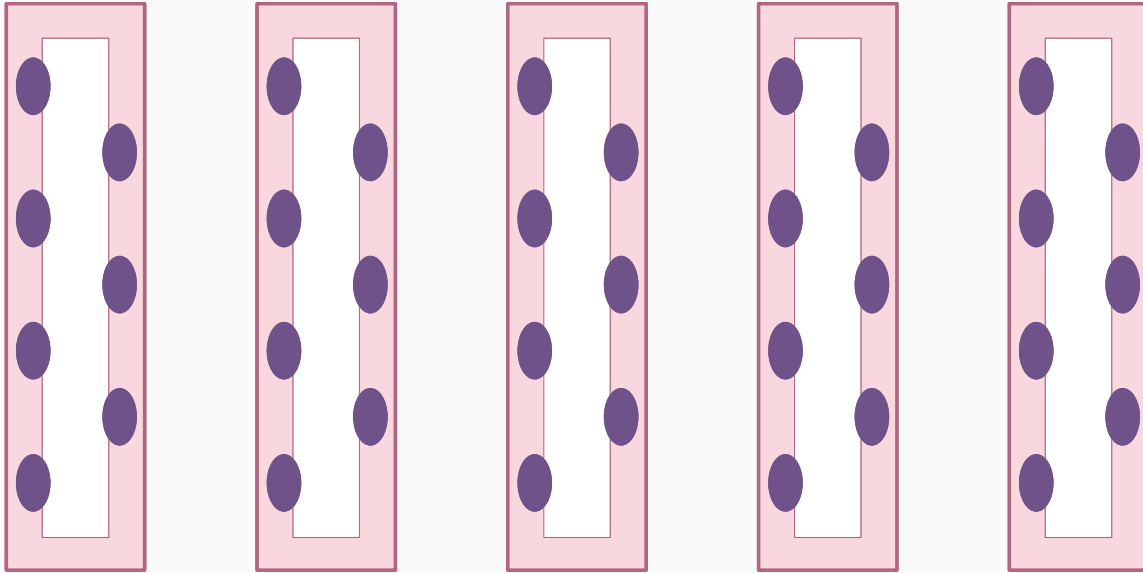
Nuclei occupy multiple apparent levels.
Pseudostratification is visible.

Chromatin

Hyperchromasia reflects darkly staining nuclei.
Atypia persists in dysplastic epithelium.

Schematic: Columnar cells with dysplastic nuclei

SCHEMATIC — not a histology specimen



Read the architecture

Nuclear crowding and elongation.

Multiple nuclear levels.

Darker nuclear staining than bland mucosa.

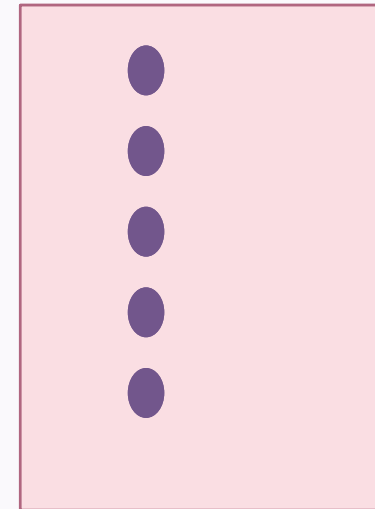
Nuclear enlargement: A cellular warning sign

Dysplastic nuclei are enlarged relative to ordinary epithelial nuclei.

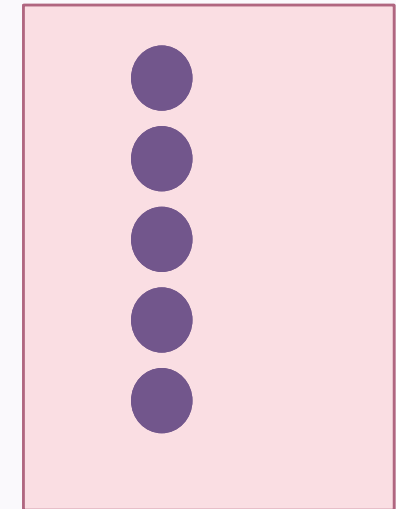
Interpret enlargement together with elongation, hyperchromasia, and organization.

Enlargement alone is not sufficient to separate dysplasia from injury-related regeneration.

Epithelial nuclei — conceptual comparison



Compared with



Feature highlighted

SCHEMATIC · not an H&E specimen

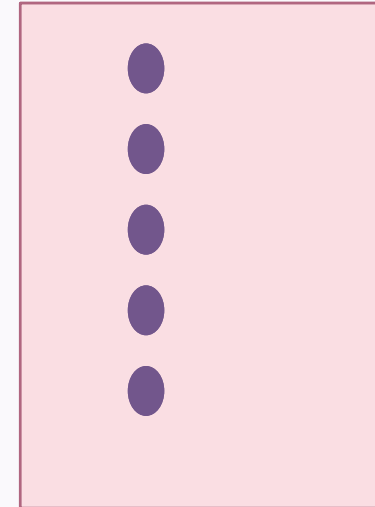
Nuclear elongation adds a morphologic clue

The adenomatous nuclei often appear elongated rather than rounded.

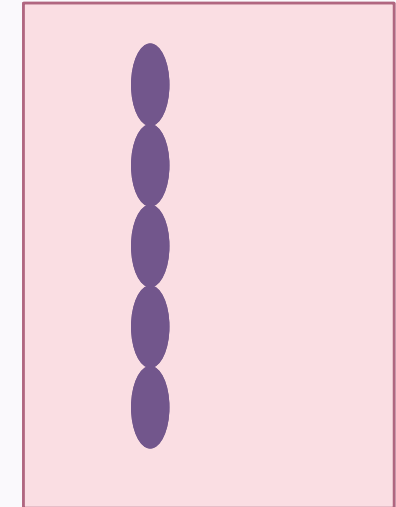
Elongation accompanies pseudostratification and hyperchromasia.

Assess the composite epithelial pattern rather than one isolated nucleus.

Epithelial nuclei — conceptual comparison



Compared with



Feature highlighted

SCHEMATIC · not an H&E specimen

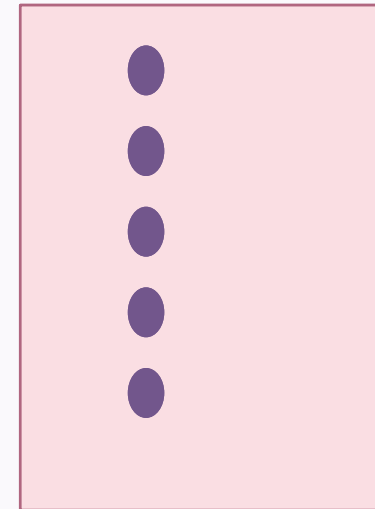
Pseudostratification alters the nuclear plane

Nuclei appear at several heights within a single lining epithelium.

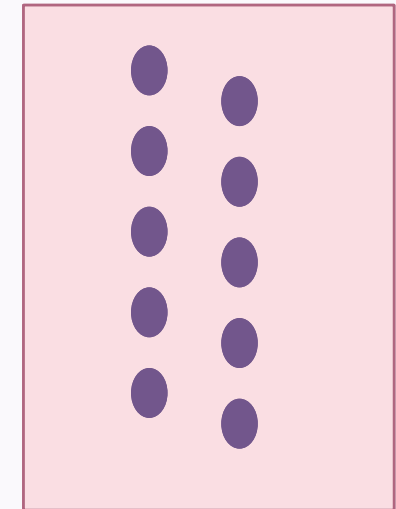
This produces an impression of multiple nuclear layers.

Pseudostratification is a cytologic clue; It is not evidence of invasion.

Epithelial nuclei — conceptual comparison



Compared with



Feature highlighted

SCHEMATIC · not an H&E specimen

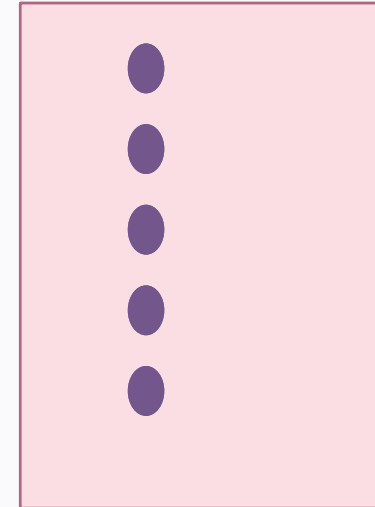
Hyperchromasia makes nuclear atypia conspicuous

Hyperchromatic nuclei stain darker on hematoxylin-based sections.

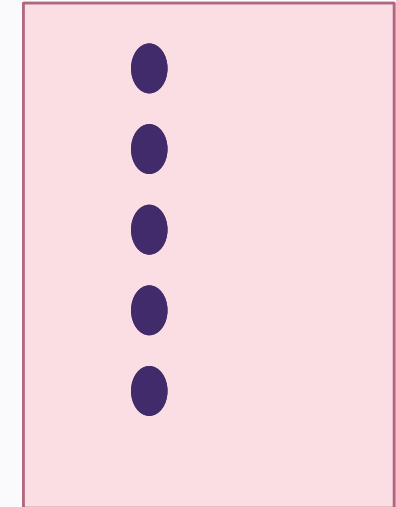
This feature in both low- and high-grade dysplasia.

Dark staining must be integrated with architectural and maturation changes.

Epithelial nuclei — conceptual comparison



Compared with



Feature highlighted

SCHEMATIC · not an H&E specimen

Low-grade and high-grade share core cytology

Features in both grades

Nuclear enlargement and elongation.

Pseudostratification.

Hyperchromasia.

What identifies high grade?

Greater severity of cytologic atypia.

More complex and abnormal glandular architecture.

Low-grade dysplasia: Coherent but abnormal epithelium

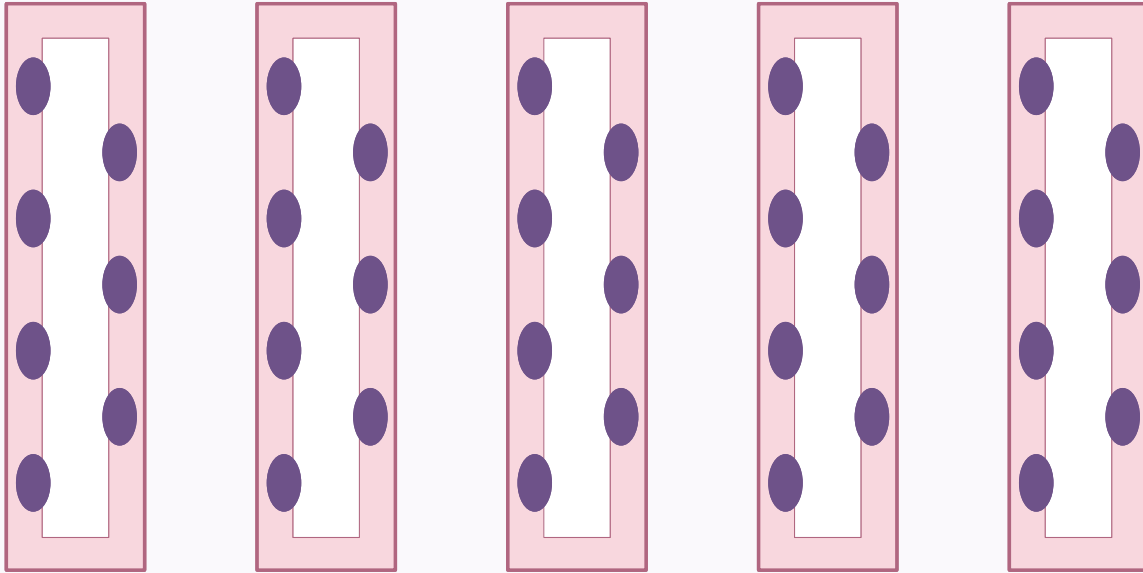
Dysplasia is present, although cytologic disturbance is less severe than high grade.

Nuclear changes still require a neoplastic interpretation.

The distinction from high grade depends on the overall cytologic and architectural pattern.

Schematic: Lower-grade gland organization

SCHEMATIC — not a histology specimen



Read the architecture

Elongated hyperchromatic nuclei.

Dysplastic epithelial crowding.

Less severe architectural disturbance
than in high grade.

High-grade dysplasia: Cytologic severity increases

Nuclear atypia is more severe than in low-grade lesions.

Architectural abnormalities become important diagnostic evidence.

Budding and cribriform gland-within-gland structures.

Assessment

Review both nuclei and gland architecture; Grade is not assigned by size alone.

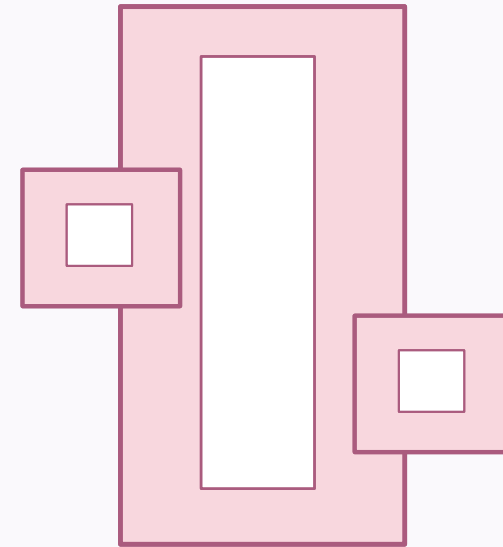
Glandular budding signals architectural complexity

Buds arise from an existing gland profile.

Glandular budding is named by Robbins as a high-grade architectural abnormality.

Do not equate budding by itself with proven stromal invasion.

Schematic gland architecture



Side buds project from the parent gland

SCHEMATIC · not an H&E specimen

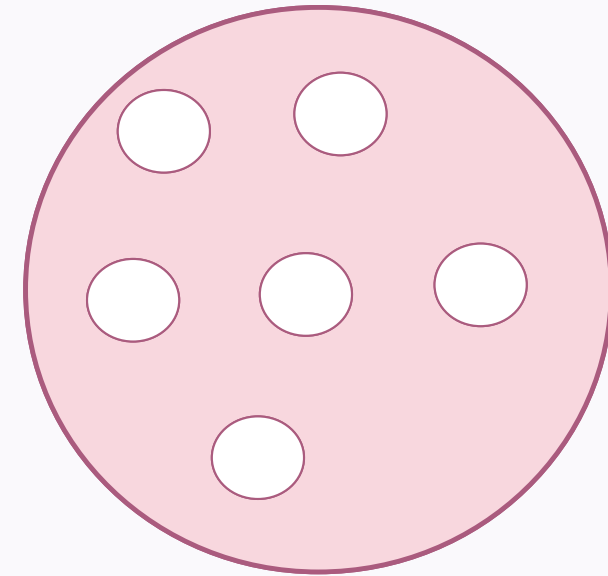
Cribriform glands have gland-within-gland complexity

Cribriform architecture is a gland-within-gland configuration.

It is one of the architectural changes listed for high-grade dysplasia.

Its presence raises concern about the grade of epithelial atypia.

Schematic gland architecture



Several lumina within a complex gland

SCHEMATIC · not an H&E specimen

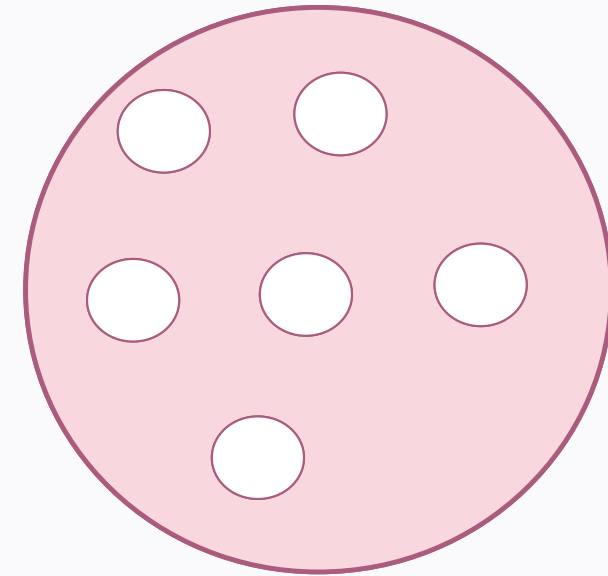
Schematic: Complex high-grade gland pattern

High-grade dysplasia requires more severe cytologic atypia.

Complex architecture, including cribriform change, is characteristic.

Actual diagnosis requires examination of histologic tissue.

Schematic gland architecture



Several lumina within a complex gland

SCHEMATIC · not an H&E specimen

Histologic features used to grade dysplasia

Feature	Low-grade dysplasia	High-grade dysplasia
Nuclear atypia	Enlarged, elongated, hyperchromatic	More severe abnormalities
Nuclear distribution	Pseudostratification possible	Pseudostratification with greater atypia
Architecture	Dysplastic glands	Budding; gland-within-gland / cribriform
Key distinction	Cytologic dysplasia present	Greater cytologic + structural complexity

Dysplasia versus repair is an important pitfall

Active inflammation induces regeneration and mitotic activity.

These changes can resemble neoplastic atypia.

Surface maturation as a useful distinction.

Surface maturation helps distinguish reactive atypia

Reactive epithelium

Proliferation occurs near injury.

Cells mature as they approach the surface.

Dysplastic epithelium

Cytologic immaturity persists.

Atypia is not confined to a basal regenerative zone.

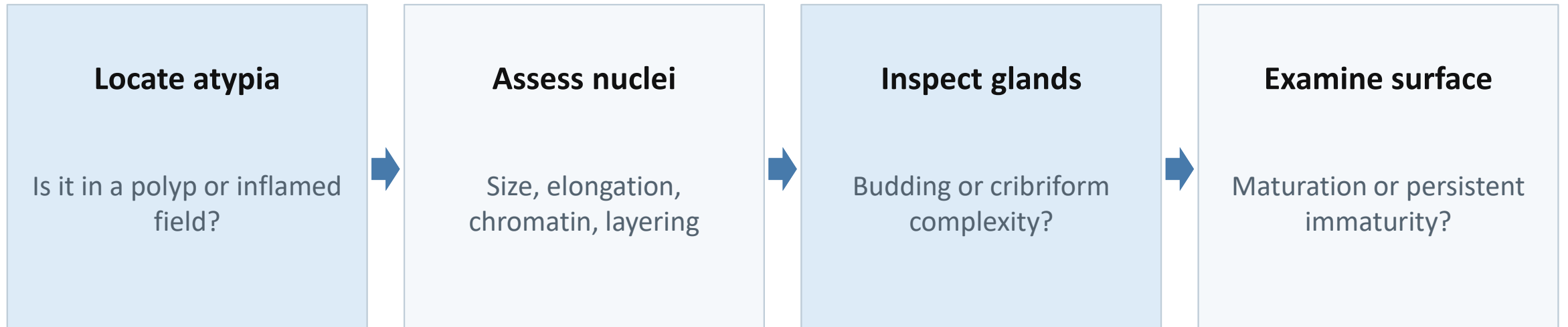
Atypia should be assessed in its inflammatory context

A background of active gastritis may exaggerate nuclear irregularity.

Increased mitoses can occur in reactive and dysplastic epithelia.

Use surface maturation and the complete cytologic pattern to avoid overcalling dysplasia.

Reading a suspicious gastric biopsy



Teaching logic, not an official reporting algorithm or a substitute for full specimen assessment.

Location helps organize the gastric polyp differential



Regional schematic; Not an anatomic drawing or a lesion distribution map

Gastric adenomas are usually solitary lesions in the antrum.

Fundic gland polyps are typically found in the body and fundus.

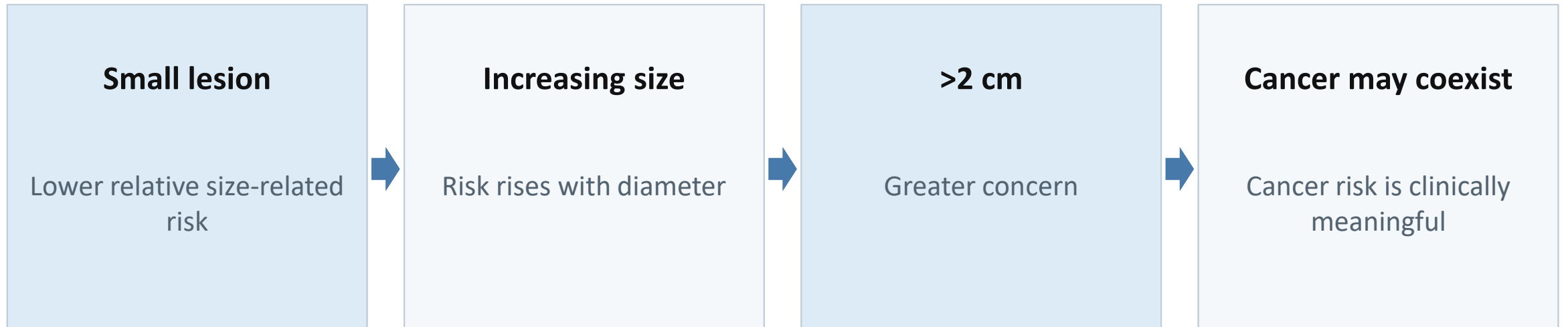
Macroscopic context: A usually solitary antral lesion

Gastric adenomas are usually solitary rather than numerous.

The antrum is a typical location.

Neither site nor multiplicity can replace microscopic demonstration of dysplasia.

Malignancy risk increases with adenoma size



Qualitative risk schematic: Box spacing and height do not indicate measured probabilities.

Size threshold highlighted by Robbins

>2 cm

Higher adenocarcinoma risk

Risk of adenocarcinoma within an adenoma correlates with lesion size.

Lesions greater than 2 cm are particularly concerning.

Large adenomas carry substantial cancer risk

Up to 30%

When large

The statement applies to large gastric adenomas.

“Up to” is a reported upper estimate, not a universal probability for an individual.

Clinical significance extends beyond histologic grade

A lesion with adenomatous dysplasia is a clinically important neoplasm.

Larger diameter increases concern for associated adenocarcinoma.

Gastric adenomas warrant more aggressive management than colonic adenomas.

Boundary of source

No specific endoscopic technique, margin threshold, or surveillance interval is prescribed by this excerpt.

Do not confuse risk with certainty

Supported by Robbins

Cancer risk rises with size.

Large lesions may have cancer, up to 30%.

Not supported by that statistic

Every >2 cm adenoma contains cancer.

Size alone demonstrates carcinoma.

Hyperplastic polyps: The principal common comparison

Inflammatory/hyperplastic polyps arise with chronic gastritis.

Their glands are elongated, irregular, and often cystically dilated.

Edematous lamina propria and inflammatory cells support this category.

Adenomatous dysplasia versus hyperplastic architecture

Adenoma

Dysplastic epithelial nuclear changes.
Pseudostratification and hyperchromasia.
High grade may form budding or cribriform glands.

Hyperplastic polyp

Elongated and cystically dilated foveolar glands.
Inflamed or edematous lamina propria.
May arise as a regenerative response to gastritis.

Inflammatory and hyperplastic polyps differ grossly

Hyperplastic polyps may have a smooth surface and ovoid shape.

Superficial erosions are common in inflammatory polyps.

Both share a chronic-gastritis relationship; They are differentiated histologically.

Fundic gland polyps occupy a different mucosal zone

Fundic gland polyps most often arise in the body and fundus.

They are circumscribed lesions with a smooth surface.

Their typical glandular composition distinguishes them from adenomas.

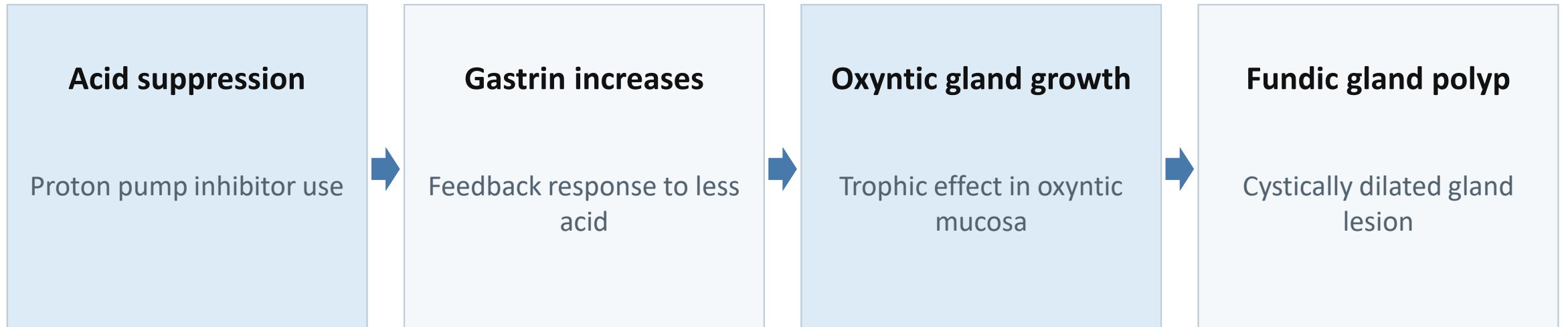
Fundic gland polyps: Recognize oxyntic-type glands

Characteristic glands are irregular and cystically dilated.

They contain parietal, chief, and foveolar mucous cells.

Inflammation is usually absent or minimal.

Why proton pump inhibitors relate to fundic gland polyps



This mechanism belongs to fundic gland polyps, not the ordinary pathogenesis of gastric adenoma.

Fundic gland neoplasia risk depends on setting

Sporadic / PPI-associated

No significant neoplastic risk reported.
Often related to acid suppression.

FAP-associated

Dysplasia can occur.
Rare cancers have been reported.

Gastric epithelial polyps: High-yield comparison

Dimension	Adenoma	Hyperplastic / inflammatory	Fundic gland
Typical location	Antrum	Variable; gastritis-associated	Body / fundus
Core histology	Dysplasia	Long, cystic foveolar glands	Cystic oxyntic glands
Background	Atrophy, metaplasia often	Chronic gastritis	PPI exposure / APC-MUTYH
Cancer implication	Size-dependent, substantial	Dysplasia may occur	Sporadic: not significant

A practical lesion description separates observations

Endoscopy: Location, approximate diameter, and multiplicity.

Microscopy: Epithelial dysplasia, its grade, and architectural pattern.

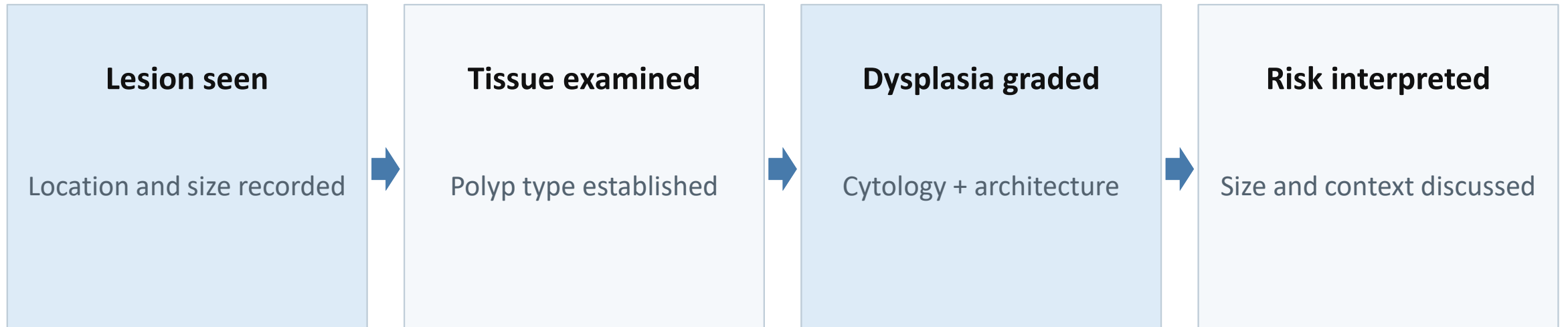
Background mucosa: Gastritis, atrophy, and intestinal metaplasia if observed.

Do not infer carcinoma from lesion diameter alone.

Teaching application

These are source-derived descriptive variables, not a formal synoptic reporting standard.

From lesion observation to clinicopathologic interpretation



An educational workflow inferred descriptions; No treatment protocol is asserted.

Case 1: Antral polyp in atrophic gastritis

A 58-year-old man has a solitary antral polyp. Biopsy shows elongated hyperchromatic nuclei with pseudostratification.

Which category is most strongly supported?

Which feature makes a purely reactive polyp less likely?

What background change would you specifically inspect?

Case 1: Interpret the dysplastic epithelium

Gastric adenoma is favored

Dysplastic nuclear pattern supports neoplastic epithelium.

Solitary antral location is concordant but not diagnostic.

Inspect nearby mucosa for atrophy and intestinal metaplasia.

Case 2: Gland-within-gland architecture

An antral polyp displays markedly atypical nuclei and complex budding and cribriform glands.

What dysplasia grade is indicated?

Which findings justify that grade?

Does a cribriform gland, alone, prove invasion?

Case 2: Architecture points to high grade

High-grade dysplasia

Severe nuclear atypia supports high grade.

Glandular budding and cribriform structures are named high-grade features.

The described architecture alone is not proof of stromal invasion.

Case 3: A body/fundus polyp with cystic glands

A smooth polyp in the gastric body has cystically dilated glands lined by parietal, chief, and foveolar cells.

Which polyp is most likely?

Which medication association is relevant?

How does sporadic neoplastic risk compare with gastric adenoma?

Case 3: Recognize fundic gland morphology

Fundic gland polyp

Body/fundus location and oxyntic-type cystic glands are characteristic.

Proton pump inhibitor use is an established association.

Sporadic fundic gland polyps have no significant neoplastic risk.

Case 4: A 2.6 cm gastric adenoma

A hypothetical 2.6 cm gastric adenoma has dysplasia on limited sampling.

What feature raises concern about associated adenocarcinoma?

Can size alone establish malignant transformation?

What limitation applies when quoting a 30% estimate?

Case 4: Risk is elevated but not certain

Size >2 cm increases concern

Larger adenoma diameter with risk of adenocarcinoma.

A lesion larger than 2 cm falls into a particularly concerning group.

“Up to 30% when large” is not an individual predicted probability.

Five distinctions worth retaining

What it is	How to recognize it	Why it matters
An epithelial adenoma with dysplasia. Usually solitary and antral.	Enlarged, elongated, hyperchromatic nuclei. High-grade: Severe atypia, budding/cribriform.	Often linked to atrophy and metaplasia. Cancer risk increases with size, especially >2 cm.

Source and visual provenance

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. Chapter 17: The Gastrointestinal Tract; pp. 702–705.

Gastric adenoma: printed p. 705; adjacent material: chronic gastritis/dysplasia, gastric polyps.

Histologic figures in this deck are editable simplified teaching schematics—not actual photomicrographs.

The original textbook histology image (Fig. 17.19D) should be consulted for diagnostic morphology.