

Gastric Polyps and Tumors

An integrated clinicopathologic approach

Reactive polyps · epithelial tumors · lymphoma · NET · GIST

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Learning objectives

- Classify common gastric mucosal polyps by pathogenesis and histology.
- Differentiate reactive polyps and adenomas from malignant neoplasms.
- Relate adenocarcinoma morphology to molecular mechanisms.
- Recognize key features of gastric MALT lymphoma, NET, and GIST.

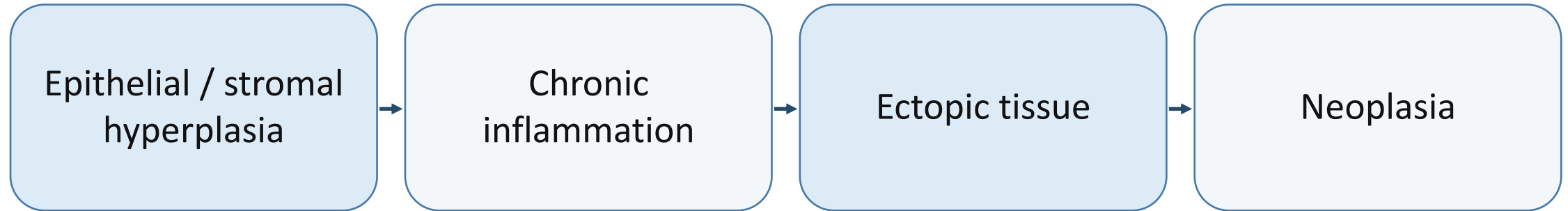
What is a gastric polyp?

A mucosal projection that rises above the surrounding gastric surface.

Detected in up to 5% of upper gastrointestinal endoscopies.

The term describes a shape—not a single biologic diagnosis.

Four mechanisms generate polypoid lesions



Polypoid protrusion seen at endoscopy

Lineage—not shape—organizes gastric masses

Gastric polyp or tumor

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graph TD; A[Gastric polyp or tumor] --> B[Epithelial]; A --> C[Lymphoid]; A --> D[Neuroendocrine]; A --> E[Mesenchymal]; B --> B1[Hyperplastic polyp · fundic gland polyp · adenoma · adenocarcinoma]; C --> C1[Gastric MALT lymphoma]; D --> D1[Well-differentiated gastric neuroendocrine tumor]; E --> E1[Gastrointestinal stromal tumor (GIST)];
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Epithelial

Hyperplastic polyp ·
fundic gland polyp ·
adenoma ·
adenocarcinoma

Lymphoid

Gastric MALT
lymphoma

Neuroendocrine

Well-differentiated
gastric
neuroendocrine
tumor

Mesenchymal

Gastrointestinal
stromal tumor
(GIST)

A mucosal polyp and a mural mass are different



- Hyperplastic polyps and adenomas arise in the mucosa.
- Neuroendocrine tumors often form intramural/submucosal nodules.
- GIST typically forms a mass in the gastric wall and may ulcerate mucosa.

Original conceptual cross-section (schematic)

Endoscopy provides shape; Histology provides identity

Endoscopic description

Polypoid / nodular / ulcerated / infiltrative
Solitary or multiple
Antrum versus oxyntic mucosa

Tissue assessment

Gland architecture and cytology
Lymphoid versus neuroendocrine cells
Spindle / epithelioid mesenchymal cells

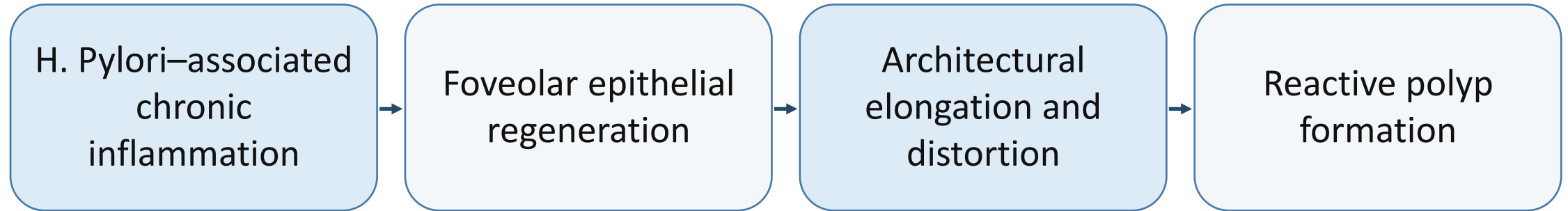
Inflammatory and hyperplastic polyps dominate

Together account for up to 75% of gastric polyps.

Share inflammatory associations but differ by the histologic emphasis.

Other common polyp diagnoses include fundic gland polyps and adenomas.

Helicobacter-associated mucosal injury matters



Association, not a universal obligatory pathway

Inflammatory and hyperplastic are related—not identical

Feature	Hyperplastic polyp	Inflammatory polyp
Surface	Usually smooth	Superficial erosion common
Predominant emphasis	Foveolar gland change	Inflammatory / erosive change
Shared background	Often chronic gastritis	Often chronic gastritis

Age and multiplicity provide context

- Most common in the fifth to sixth decades of life.
- Most lesions measure less than 1 cm.
- They may be multiple, particularly with atrophic gastritis.
- Regional prevalence tracks the prevalence of H. Pylori infection.

Gross morphology: Smooth versus eroded

Hyperplastic

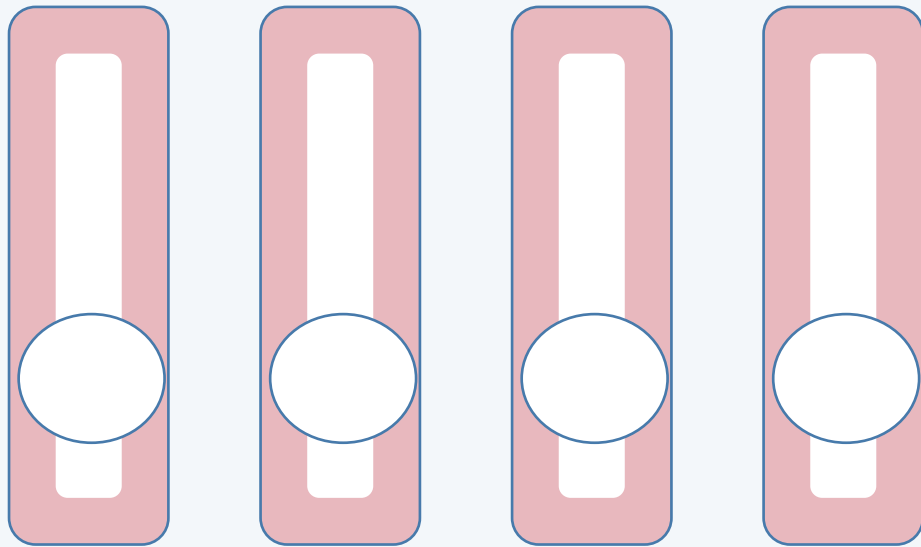
Commonly ovoid
Smooth mucosal surface
Frequently less than 1 cm

Inflammatory

Often superficial erosions
Similar reactive gland changes
Inflammation more conspicuous

The foveolar apparatus is the site of hyperplasia

SCHEMATIC — not a histologic specimen



Tortuous / dilated foveolae

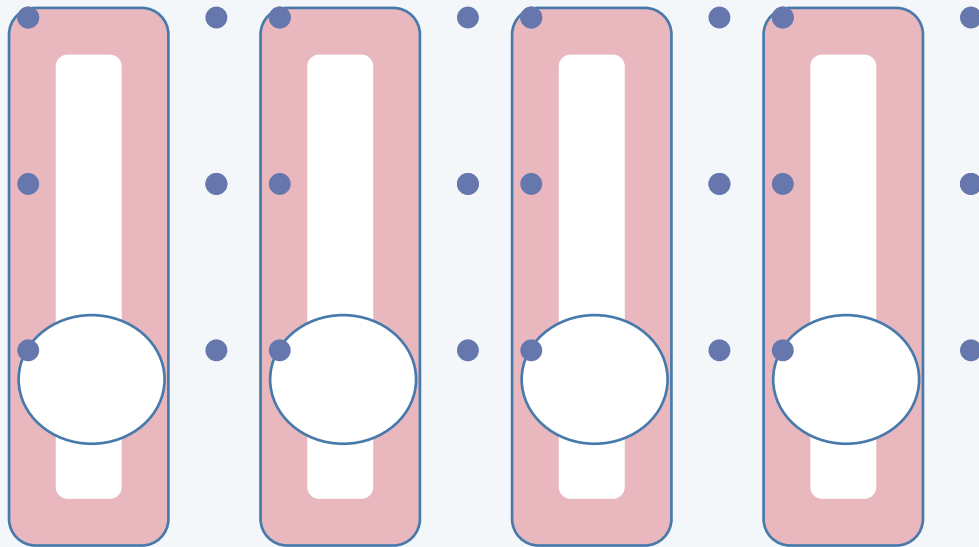
- Surface and pit epithelium expands into irregular elongated glands.
- Marked tortuosity produces a “corkscrew” architectural pattern.
- Variable cystic dilation adds luminal size variation.

Histology: Distorted and dilated foveolar glands

- Irregular gland profiles replace uniform foveolar architecture.
- Some glands are long and corkscrew-shaped; Others are cystically dilated.
- This is a predominantly regenerative epithelial response.
- Architectural distortion alone is not evidence of adenomatous dysplasia.

The lamina propria is edematous and inflamed

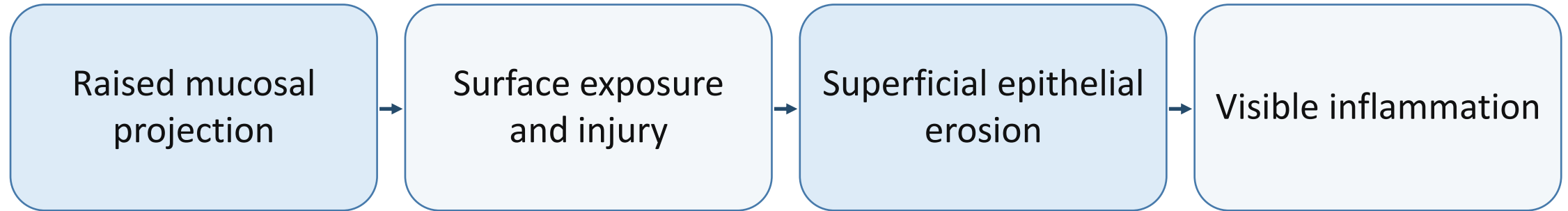
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Foveolar change + stromal inflammation

- Edema expands the interglandular stroma.
- The inflammatory infiltrate varies in acuity and intensity.
- Foveolar growth and stromal inflammation coexist.

Why erosions occur in an inflammatory polyp



Mechanistic interpretation of described morphology

Multiplicity suggests a mucosal field process

Single protrusion

- Characterize its surface
- Assess gland architecture
- Check background mucosa

Multiple protrusions

- Consider accompanying atrophy
- Look for diffuse chronic gastritis
- Do not equate multiplicity with malignancy

Hyperplastic polyp versus adenoma

Axis	Hyperplastic polyp	Gastric adenoma
Core biology	Reactive hyperplasia	Dysplastic epithelial neoplasm
Histology	Tortuous dilated foveolae	Nuclear atypia / stratification
Background	Chronic gastritis	Atrophy / intestinal metaplasia frequent

Applied morphology: A small eroded polyp

Teaching vignette — constructed from textbook descriptions

Presentation A patient in the sixth decade has multiple small gastric protrusions in a stomach with chronic gastritis.

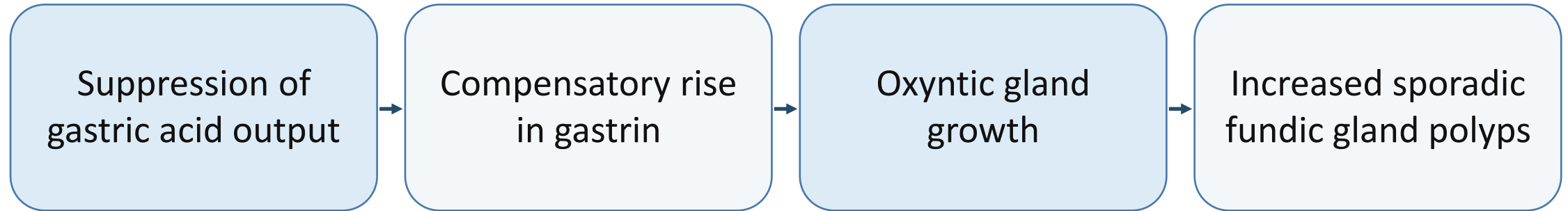
Finding The biopsied nodule shows elongated foveolar glands, edematous lamina propria, and a superficial erosion.

Interpretation Most consistent with an inflammatory/hyperplastic polyp; Assess histology rather than shape alone.

Fundic gland polyps arise in oxyntic mucosa

- Typical distribution: Gastric body and fundus.
- Well-circumscribed lesions with a smooth surface.
- May occur singly or in multiples.
- Can be sporadic or associated with inherited polyposis syndromes.

Proton-pump inhibition alters the oxyntic trophic axis



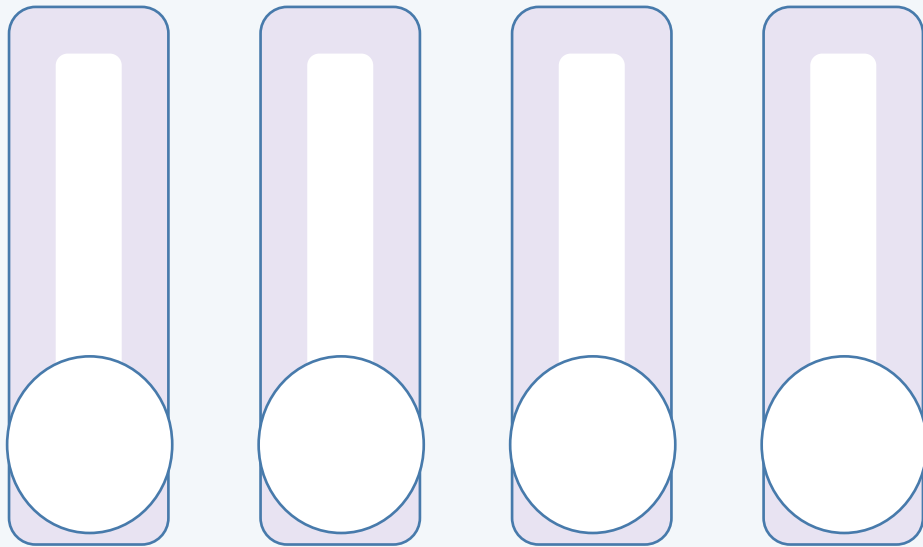
Association

Sporadic and syndromic polyps have different contexts

Feature	Sporadic / PPI-associated	Hereditary context
Setting	Often prolonged PPI exposure	Germline APC or MUTYH
Dysplasia	Not a significant neoplasia risk	Dysplasia may occur in FAP
Risk signal	Clinical medication context	Polyposis syndrome context

Microscopy: Cystic dilation of oxyntic glands

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Cystic oxyntic glands

- Cystically dilated, irregular glandular spaces.
- Lining includes parietal, chief, and foveolar mucous cells.
- The background inflammatory infiltrate is absent or minimal.

Histologic pitfall: Cysts do not imply adenoma

Fundic gland polyp

Cystically dilated glands
Recognizable oxyntic cell types
Little inflammation

Gastric adenoma

True epithelial dysplasia
Nuclear elongation / hyperchromasia
Architectural atypia at higher grade

Dysplasia risk depends on clinical setting

- FAP-associated fundic gland polyps can harbor dysplasia.
- Rare cancers have been described in that inherited setting.
- Sporadic lesions, including PPI-associated polyps, do not carry a significant neoplastic risk.

Applied morphology: Body/fundus cystic polyps

Teaching vignette — constructed from textbook descriptions

Presentation A patient taking a proton-pump inhibitor has several smooth gastric body polyps.

Finding Histology shows dilated oxyntic glands with parietal and chief cells and scant inflammation.

Interpretation Interpretation: Fundic gland polyp; Distinguish sporadic from inherited-polyposis context.

Adenoma is epithelial neoplasia with dysplasia

Unlike a reactive polyp, a gastric adenoma is defined by dysplastic epithelium.

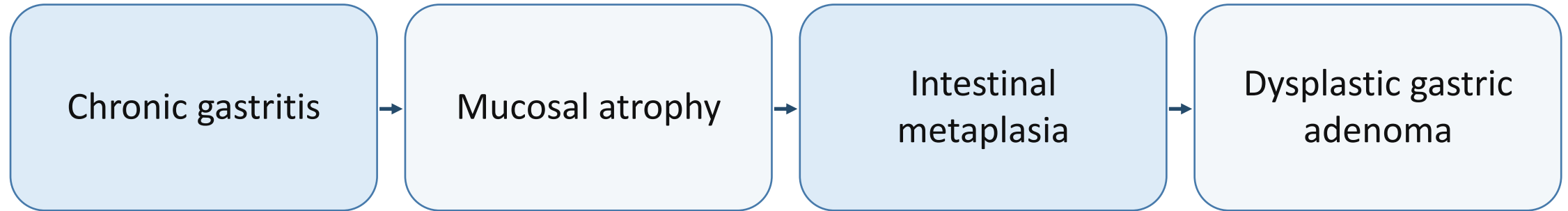
Usually develops in chronic atrophic gastritis with intestinal metaplasia.

APC- or MUTYH-associated syndromic adenomas are exceptions to that common background.

Adenomas are antral lesions in older adults

- Usually solitary, commonly located in the antrum.
- Frequency rises with age; Typical patient age is 50–60 years.
- Male predominance is described.
- Population prevalence parallels gastric carcinoma risk.

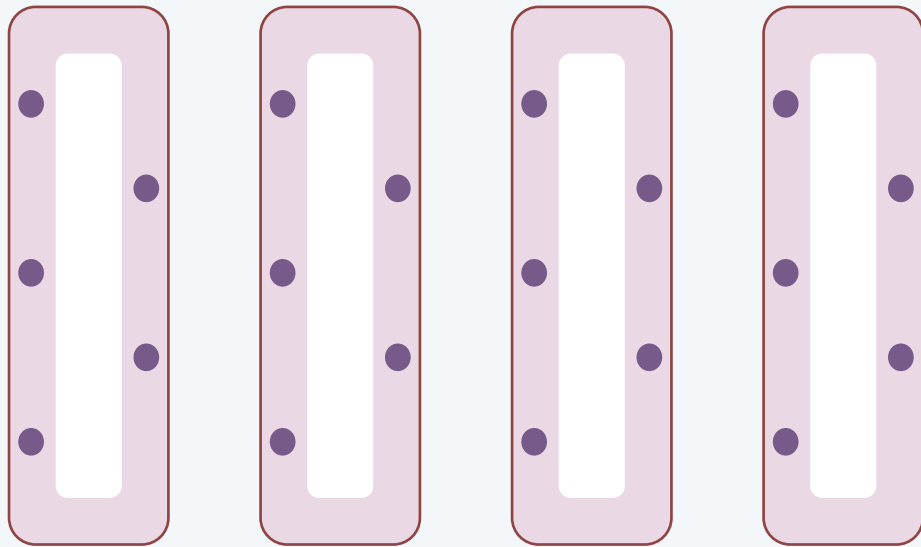
Chronic injury creates a permissive precursor mucosa



Typical background; Not a mandatory sequence for every lesion

Low-grade dysplasia has recognizable nuclear changes

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Dysplastic nuclei

- Nuclear enlargement, elongation, and hyperchromasia.
- Nuclear pseudostratification within glandular epithelium.
- Architectural disorder less severe than in high-grade dysplasia.

High-grade dysplasia adds complex architecture

Cytology

- More pronounced nuclear atypia
- Greater epithelial disorder
- Altered epithelial maturation

Gland architecture

- Budding of dysplastic glands
- Gland-within-gland profiles
- Cribriform configurations

Size is a meaningful neoplastic risk variable

Carcinoma risk within an adenoma increases with polyp size.

Lesions exceeding 2 cm deserve particular concern.

Cancer in up to 30% of large gastric adenomas.

This risk justifies a more aggressive approach than for typical colonic adenomas.

Distinguish a dysplastic gland from a reactive gland

Observation	Reactive foveolar proliferation	Dysplastic adenoma
Architecture	Elongated, tortuous or dilated	Crowded, atypical glands
Nuclei	No defining neoplastic atypia	Hyperchromasia + pseudostratification
Grade	Not assigned as adenoma grade	Low-grade or high-grade dysplasia

Applied morphology: Antral polyp with pseudostratification

Teaching vignette — constructed from textbook descriptions

Presentation A solitary antral polyp is found in a patient with atrophic gastritis.

Finding The epithelial cells show enlarged hyperchromatic, elongated and pseudostratified nuclei.

Interpretation A diagnosis of gastric adenoma requires histologic grading and assessment for carcinoma.

Adenocarcinoma dominates gastric malignancy

Accounts for more than 90% of gastric cancers.

The major morphologic patterns are intestinal-type and diffuse-type.

This division predicts differences in gland formation, growth pattern, and pathogenesis.

Symptoms are subtle until disease is advanced

Early / nonspecific

Dyspepsia
Nausea
Dysphagia

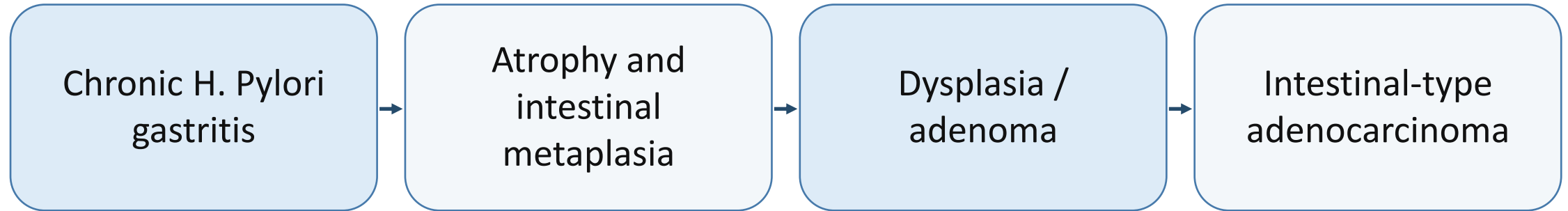
Later / advanced

Weight loss and anorexia
Anemia and hemorrhage
Early satiety (especially diffuse type)

Gastric cancer burden varies by geography

- Marked regional differences are documented.
- High-incidence areas have used population endoscopic screening.
- Reduced H. Pylori burden is associated with declining intestinal-type cancer.
- Risk also correlates with multifocal atrophy and intestinal metaplasia.

A mucosal field can progress toward intestinal-type cancer



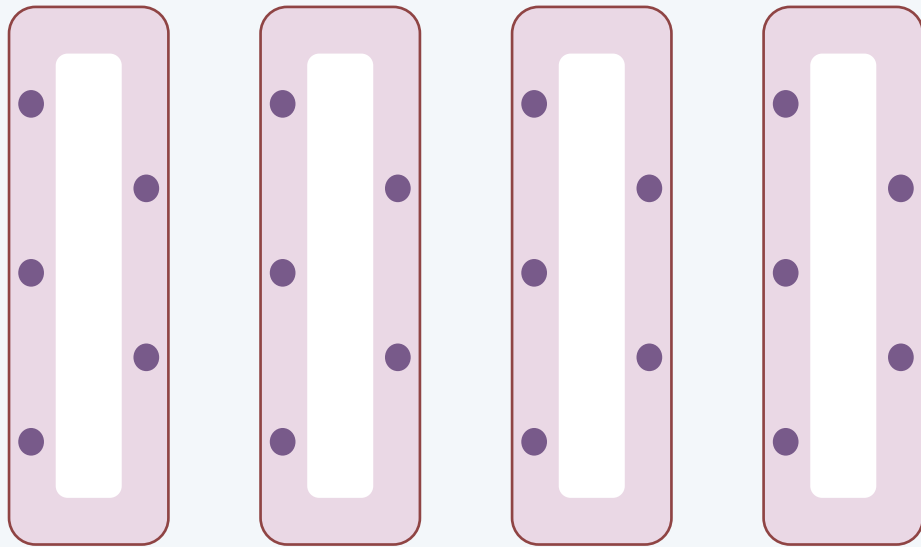
Conceptual precursor pathway from textbook associations

Intestinal and diffuse cancers build different lesions

Feature	Intestinal type	Diffuse type
Gross form	Bulky exophytic / ulcerated mass	Diffuse infiltration / wall rigidity
Cell organization	Cohesive malignant glands	Discohesive cells; no glands
Typical pattern	Mucin within glands	Mucin-filled signet-ring cells

Intestinal-type cancer forms cohesive glandular fronts

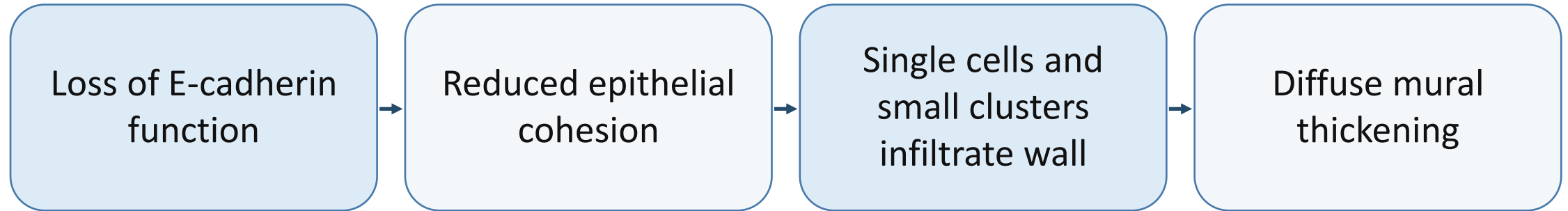
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Irregular malignant glands

- Invasive irregular gland structures within desmoplastic stroma.
- Cohesive fronts may produce an exophytic or ulcerated mass.
- Tumor cells can have apical mucin vacuoles and luminal mucin.

Diffuse-type cancer is a cell-adhesion disorder



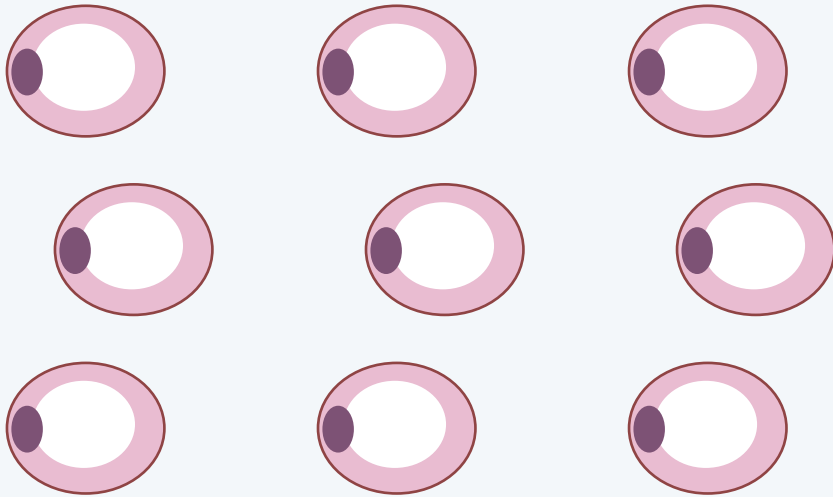
Central biology of diffuse gastric carcinoma

CDH1 connects hereditary and sporadic diffuse cancer

- CDH1 encodes the adhesion protein E-cadherin.
- Germline loss-of-function mutations predispose to hereditary diffuse gastric cancer.
- Sporadic tumors frequently lose E-cadherin through mutation or promoter methylation.
- CTNNA1 is another hereditary diffuse gastric cancer-associated gene.

Signet-ring morphology reflects intracellular mucin

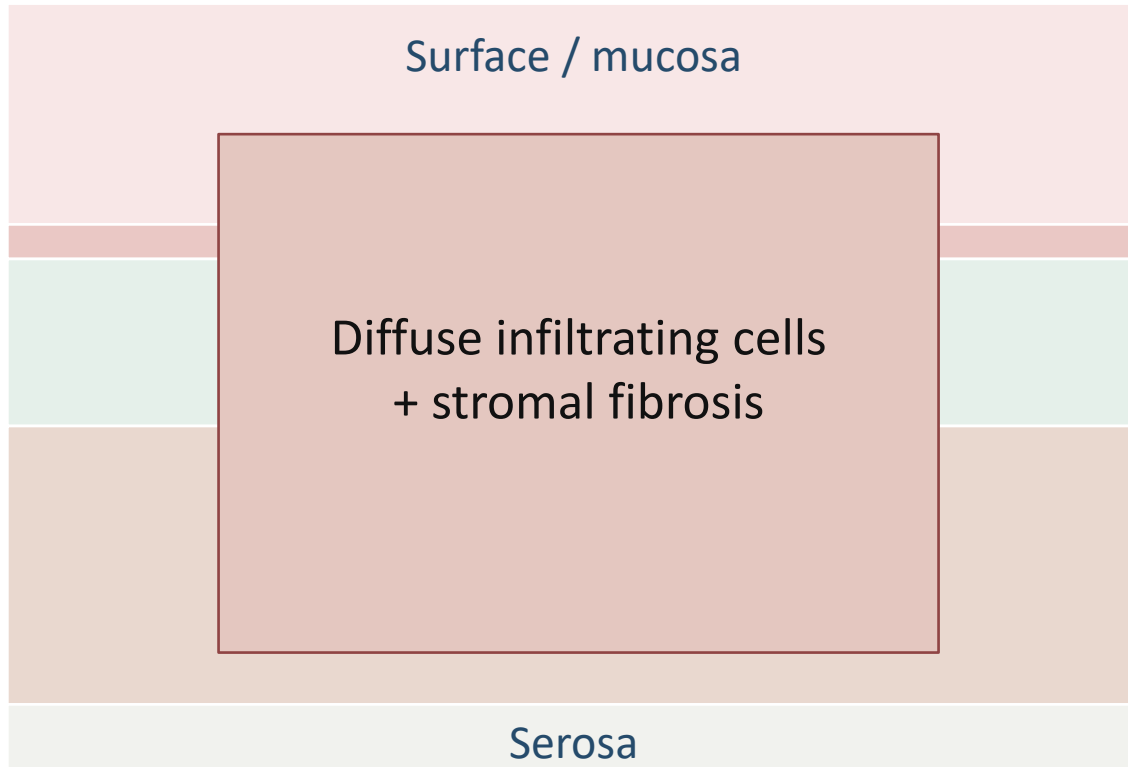
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Discohesive mucin-filled cells

- An expanded mucin vacuole displaces the nucleus to the cell periphery.
- Diffuse tumors infiltrate individually or in small cell clusters.
- At low power malignant cells can resemble inflammatory cells.

Linitis plastica results from infiltrative desmoplasia



- Diffuse infiltration occurs without a conspicuous dominant mass.
- A desmoplastic response stiffens and thickens the wall.
- Loss of rugal folds produces the characteristic “leather bottle” stomach.

Original conceptual cross-section (schematic)

Molecular diversity in intestinal-type tumors

CIN

Chromosomal instability
RTK–RAS, TP53, Wnt / TGF- β
alterations

MSI

Mismatch-repair defect
pattern
Often MLH1 promoter
silencing

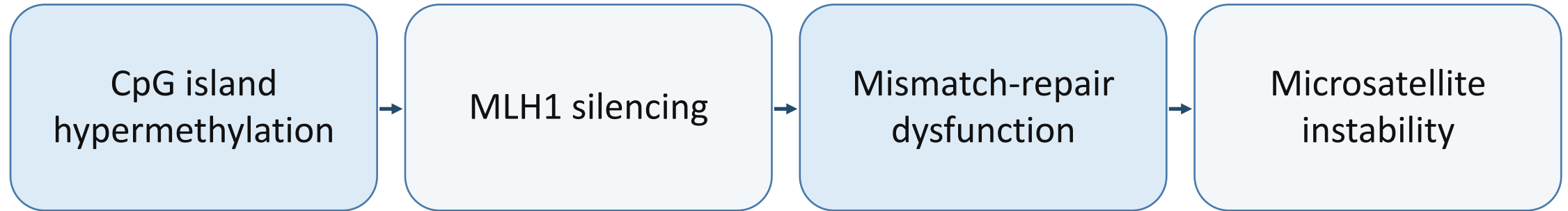
EBV-related

Extensive methylation
PIK3CA / PD-L1/2 associated
features

CIN tumors often harbor receptor-signaling alterations

- Chromosomal instability is associated with aneuploidy.
- Commonly altered signaling genes include ERBB2, EGFR, MET, and KRAS.
- APC, CTNNB1, SMAD2, SMAD4, and TP53 can also be involved.
- An antral / pyloric predilection.

MSI gastric tumors have a distinct methylation axis



Book-described molecular subtype

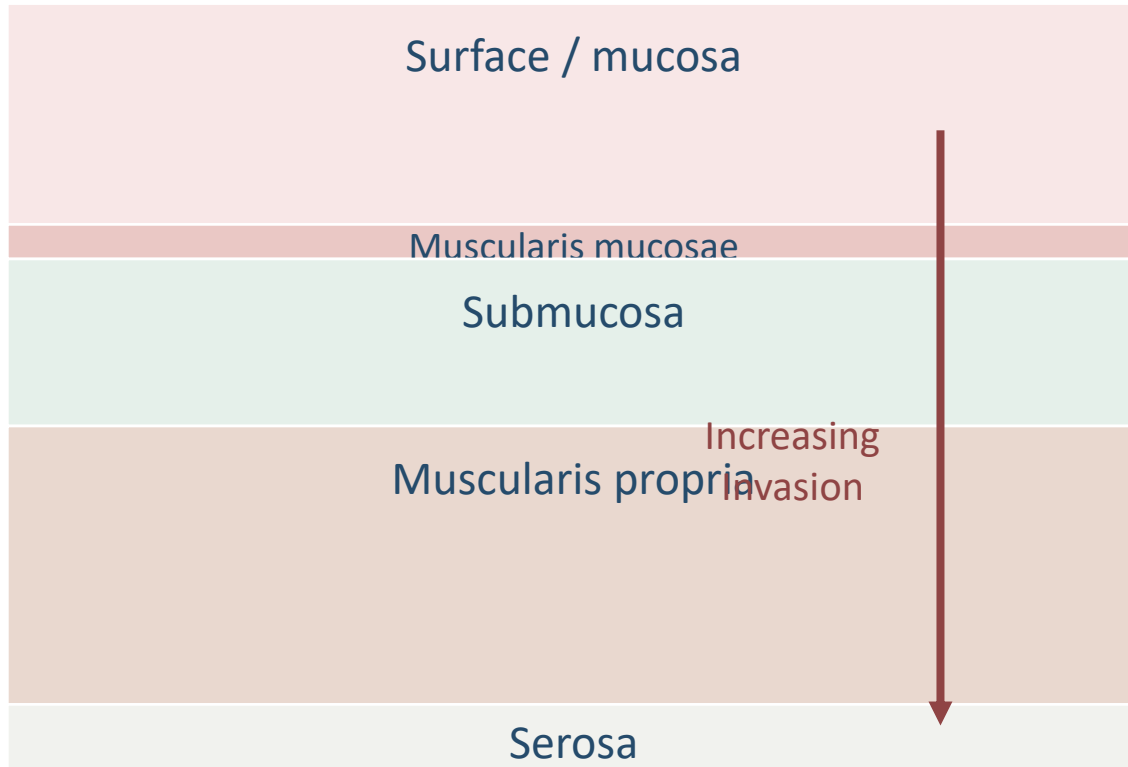
EBV-associated tumors form another molecular group

- A minority of gastric adenocarcinomas with EBV.
- Extensive promoter methylation may suppress CDKN2A, without MLH1 silencing.
- PIK3CA mutations and increased PD-L1/2 expression are frequent.
- Lymphocytic infiltrates commonly accompany these tumors.

Diffuse-type pathogenesis differs from CIN tumors

Axis	Diffuse pathway	CIN-rich intestinal pathway
Biologic defect	Cell–cell adhesion	Receptor signaling + genomic instability
Examples	CDH1; RHOA; CLDN18::ARHGAP26	ERBB2; EGFR; KRAS; TP53
Growth	Discohesive single cells	Cohesive malignant glands

Local growth and dissemination determine clinical stage



- Invasion advances through gastric wall layers and adjacent structures.
- Regional nodal spread and distant metastases markedly influence prognosis.
- Depth, nodal disease, and metastases outweigh gross appearance as prognostic factors.

Original conceptual cross-section (schematic)

Recognizable metastatic presentations

Lymphatic / cutaneous

Left supraclavicular (Virchow) node
Periumbilical Sister Mary Joseph nodule
Left axillary nodes

Other sites

Ovaries: Krukenberg tumor
Pouch of Douglas
Direct extension to neighboring organs

Early detection sharply changes prognosis

“Early gastric cancer” is confined to mucosa or submucosa.

Extent of invasion and metastasis are the strongest prognostic determinants.

Markedly better survival following resection of early disease than of advanced disease.

Applied morphology: Infiltrative thick-walled stomach

Teaching vignette — constructed from textbook descriptions

Presentation Endoscopy shows rigid thickened wall and flattened rugae without a prominent mass.

Finding Biopsy contains isolated mucin-filled cells with eccentric crescent-shaped nuclei.

Interpretation Diagnosis: Diffuse-type gastric adenocarcinoma; Consider altered E-cadherin pathway.

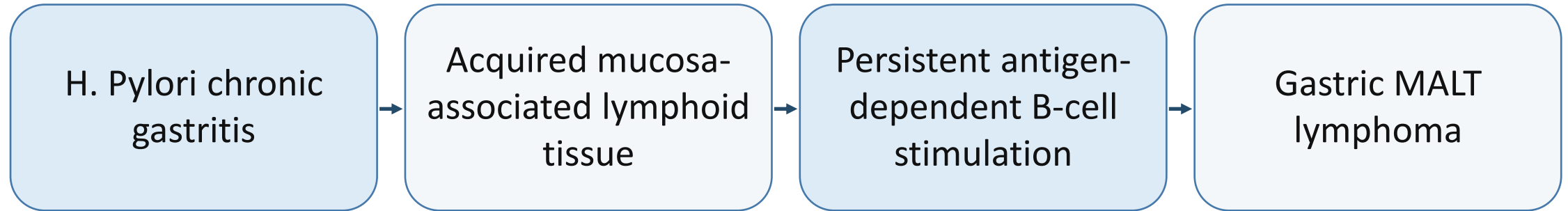
The stomach is a favored extranodal lymphoma site

The GI tract—particularly the stomach—is the most common extranodal lymphoma site.

Approximately 5% of gastric malignancies are primary gastric lymphomas.

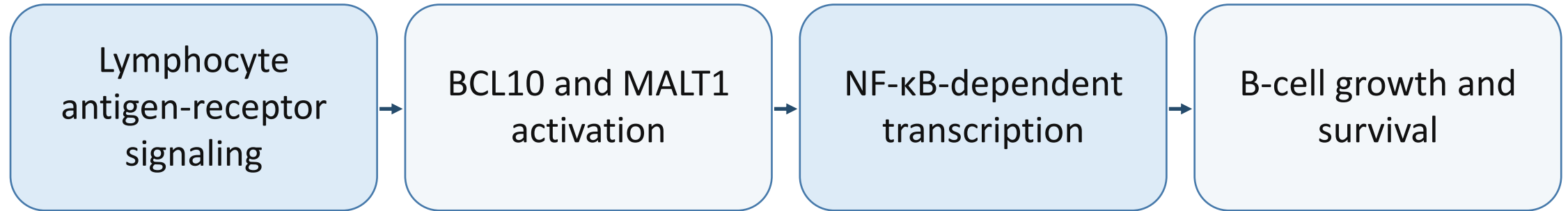
Extranodal marginal zone B-cell lymphoma of MALT type is a major subtype.

MALT is acquired in a stomach with chronic gastritis



Normal gastric mucosa lacks organized MALT

NF- κ B links antigen dependence to cell survival



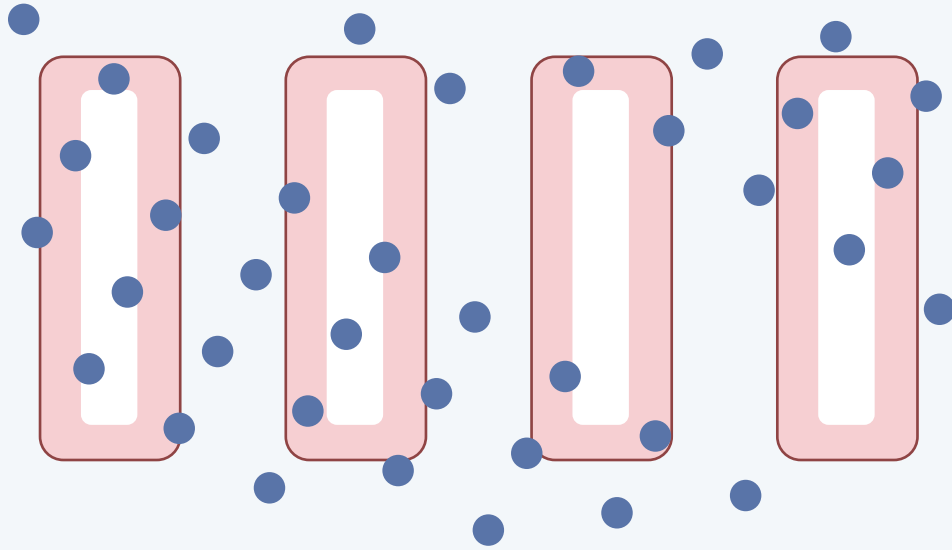
Translocations can bypass the antigen stimulus

Translocations create alternative routes to activation

Rearrangement	Target / consequence
t(11;18)	API2::MALT1 fusion
t(1;14)	BCL10 upregulation
t(14;18)	MALT1 upregulation
t(3;14)	FOXP1 upregulation

MALT lymphoma infiltrates and damages gastric glands

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Lymphocytes breach gastric glands

- Dense neoplastic lymphoid cells expand the lamina propria.
- Infiltration of gland epithelium creates lymphoepithelial lesions.
- Reactive-appearing follicles and plasmacytic differentiation may occur.

Immunophenotype supports marginal-zone B-cell lineage

Typical positive

CD19

CD20

Sometimes CD43

Usually negative / clonality

CD5 and CD10 negative

Light-chain restriction may demonstrate clonality

Clonal IGH rearrangements may be informative

Dependence on H. Pylori predicts eradication response

Feature	More likely H. pylori-responsive	More likely resistant
Activation	Antigen-dependent signals	MALT1/BCL10-related translocations
Depth	More superficial	Muscularis propria invasion
Spread	Limited disease	Nodal involvement

Applied morphology: Lymphocytes invade gastric glands

Teaching vignette — constructed from textbook descriptions

Presentation A patient with chronic H. Pylori gastritis presents with dyspepsia and epigastric pain.

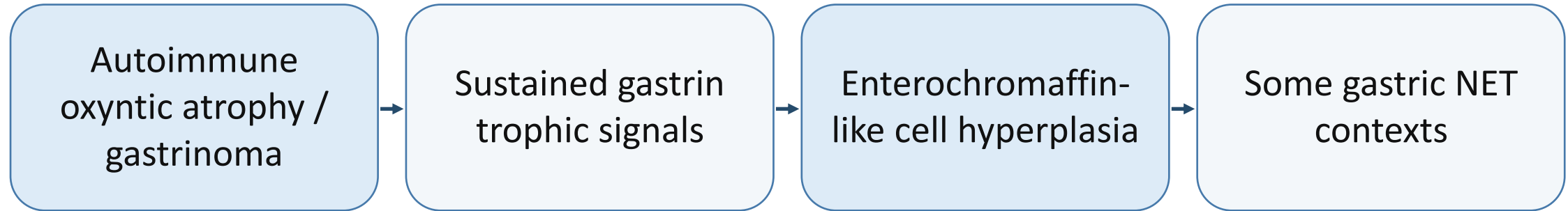
Finding A gastric biopsy shows a dense B-cell infiltrate forming lymphoepithelial lesions.

Interpretation Favor MALT lymphoma; Integrate morphology, immunophenotype, and clonality if required.

Gastric NETs arise from endocrine-lineage cells

- Well-differentiated NETs were historically termed carcinoids.
- Gastric lesions often arise in oxyntic mucosa and form submucosal nodules.
- Associations include autoimmune atrophic gastritis, MEN1, and Zollinger–Ellison syndrome.
- PPI-associated endocrine-cell hyperplasia rarely progresses to neoplasia.

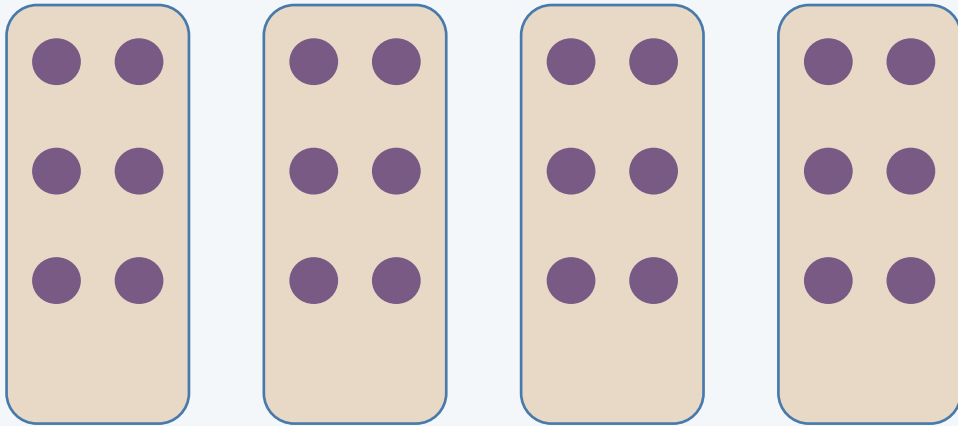
Hypergastrinemic settings can produce endocrine hyperplasia



Schematic association; Not every NET has this pathway

NET morphology: Uniform cells and stippled chromatin

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Organoid cell nests / trabeculae

- Nests, trabeculae, strands, glands, or sheets of uniform cells.
- Scant pink granular cytoplasm with round-to-oval nuclei.
- Coarse and fine chromatin creates a “salt-and-pepper” pattern.

Differentiate well-differentiated NET from NEC

Axis	Well-differentiated NET	Poorly differentiated NEC
Morphology	Uniform cells; organoid pattern	Small-cell or large-cell carcinoma
Proliferation	Mitotic / Ki-67 assessment	High proliferative activity
Molecular note	Not specified here	TP53 and RB alterations common

Neuroendocrine markers and prognostic variables

Lineage evidence

Synaptophysin
Chromogranin A
Compatible neuroendocrine morphology

Risk stratification

Degree of differentiation
Mitotic rate and Ki-67 index
Tumor size and location

Applied morphology: A small oxyntic NET

Teaching vignette — constructed from textbook descriptions

Presentation An intramural gastric nodule arises in the setting of autoimmune atrophic gastritis.

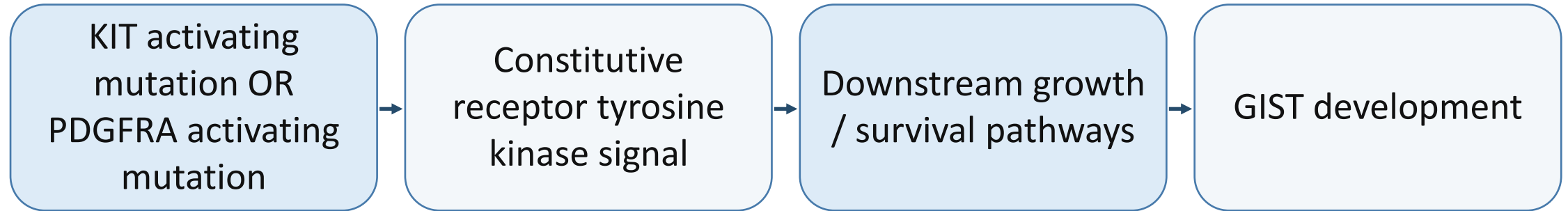
Finding Biopsy shows monotonous cells with salt-and-pepper chromatin and synaptophysin expression.

Interpretation Diagnosis: Gastric NET; Assess differentiation, proliferation, size and clinical background.

GIST is a mesenchymal tumor of Cajal-cell lineage

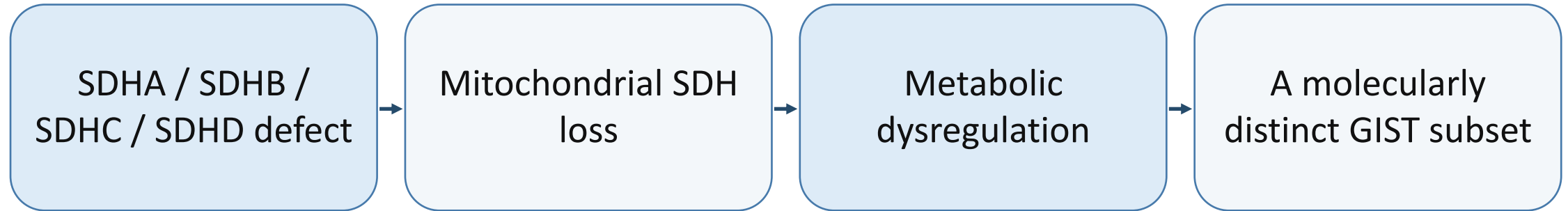
- Most common mesenchymal tumor of the abdomen.
- More than half of GISTs arise in the stomach.
- Interpreted as arising from interstitial cells of Cajal or related precursors.
- Distinguish from rare leiomyoma, schwannoma, and glomus tumors.

Constitutively active kinases drive most GISTs



KIT and PDGFRA mutations are generally mutually exclusive

A subset is linked to succinate dehydrogenase loss



An incompletely understood oncogenic mechanism

Inherited contexts can signal GIST predisposition

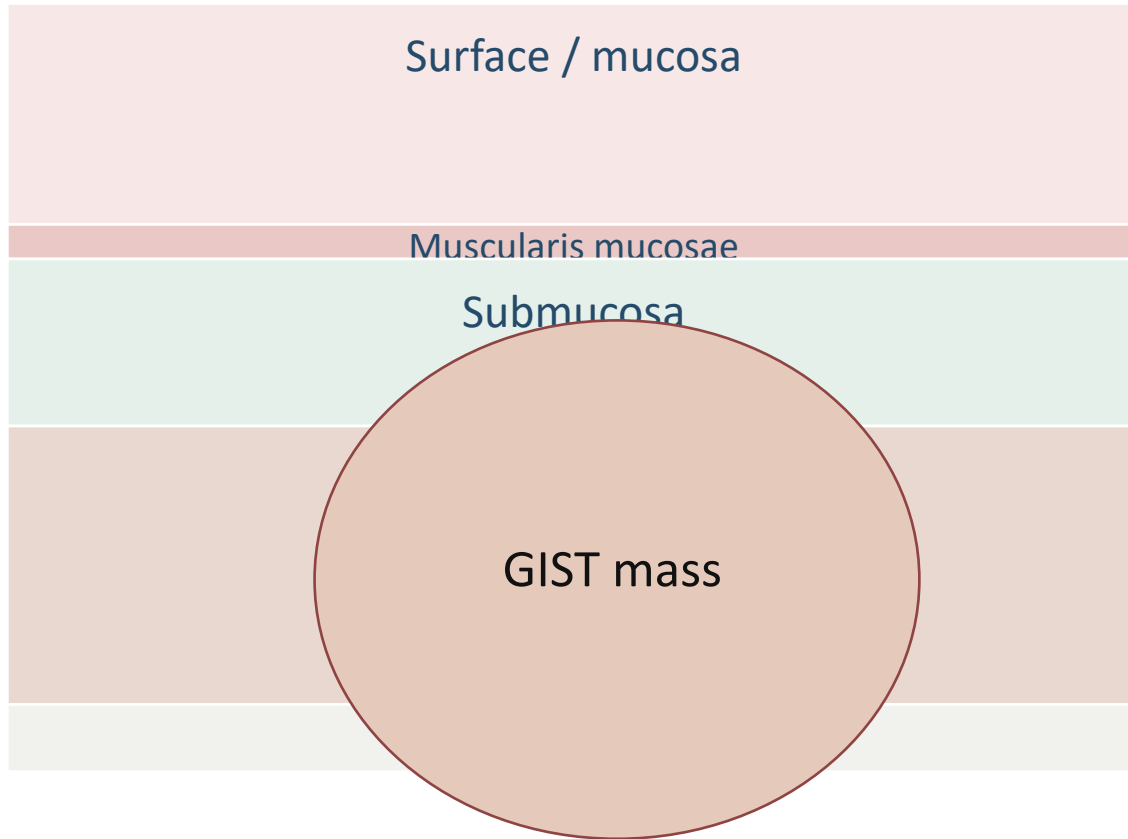
Familial / syndromic

KIT or PDGFRA germline variants
SDH-related Carney–Stratakis syndrome
Neurofibromatosis type 1

Carney triad

GIST
Paraganglioma
Pulmonary chondroma

Gross GIST: A fleshy wall-based mass



- Often a solitary, well-circumscribed mural mass.
- May show a whorled cut surface and overlying mucosal ulceration.
- Bleeding and anemia may result from mucosal breakdown.

Original conceptual cross-section (schematic)

Spindle and epithelioid patterns are morphologic variants

Pattern	Spindle-cell GIST	Epithelioid GIST
Cytology	Thin elongated cells	Plump epithelial-like cells
Architecture	Fascicles / bundles	Sheets or less-defined fascicles
Pitfall	Other spindle-cell tumors	May mimic carcinoma

KIT and DOG1 provide a useful diagnostic pair

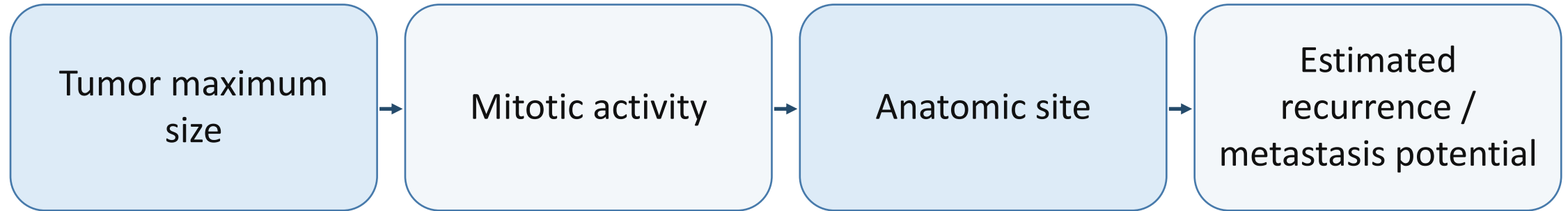
KIT (CD117)

Expressed by normal Cajal cells
Detected in most gastric GISTs
Can be absent in epithelioid tumors

DOG1

Strongly useful GIST marker
Also detects many KIT-negative tumors
Interpret with morphology and site

Risk assessment combines size, mitoses, and location



Gastric tumors generally behave less aggressively than small-bowel GISTs

Kinase genotype informs systemic drug sensitivity

Mutation-driven tumors

KIT / PDGFRA alterations
May respond to kinase inhibition
Secondary variants can confer resistance

Mutation-negative tumors

Many lack the target alteration
Often less sensitive to imatinib
Interpret genotype in clinical context

Applied morphology: An ulcerated spindle-cell mass

Teaching vignette — constructed from textbook descriptions

Presentation A gastric wall mass causes gastrointestinal blood loss and anemia.

Finding Histology demonstrates bundles of spindle cells; KIT and DOG1 are positive.

Interpretation Diagnosis: GIST. Record size, mitotic activity, location, and relevant mutation testing.

Gastric lesions are separable by cellular lineage

Lesion	Dominant cells	Useful clue
Hyperplastic polyp	Foveolar epithelium	Dilated corkscrew glands
Adenoma	Dysplastic glandular epithelium	Nuclear pseudostratification
MALT lymphoma	B lymphocytes	Lymphoepithelial lesions
NET	Neuroendocrine cells	Salt-and-pepper chromatin
GIST	Cajal-lineage mesenchyme	Spindle / epithelioid; DOG1

A site-based orientation narrows the possibilities

Antrum

Gastric adenoma common
Intestinal-type carcinoma
often distal

Body and fundus

Fundic gland polyp
Gastric NET within oxyntic
mucosa

Deep gastric wall

GIST, submucosal NET
Diffuse cancer may rigidly
thicken wall

Four actionable clues in a pathology report

- State the histologic lineage and whether dysplasia or invasion is present.
- Link the lesion to its background mucosa, especially atrophy and H. Pylori-associated injury.
- For malignant neoplasms, report extent of involvement and salient classification/risk features.
- Apply markers and molecular tests only when they answer a diagnostic or treatment question.

Integrated case: Three lesions, three questions

Teaching vignette — constructed from textbook descriptions

Presentation Smooth body polyps with cystic parietal/chief-cell glands → fundic gland polyps; Identify syndromic setting.

Finding Solitary antral polyp with pseudostratified hyperchromatic epithelium → dysplastic adenoma; Assess grade.

Interpretation Wall mass with spindle cells and DOG1 expression → GIST; Estimate risk using size and mitoses.

Take-home distinctions

- Polypoid gross appearance is not a diagnosis: The cellular lineage determines behavior.
- Reactive hyperplastic polyps, fundic gland polyps, and adenomas have different mechanisms and risks.
- Intestinal-type gastric cancer forms glands; Diffuse type loses cohesion and infiltrates as individual cells.
- MALT lymphoma, NET, and GIST have distinct morphology, immunophenotype, and therapeutic implications.

Primary reference

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th edition. 2026.
- Chapter 17 — The Gastrointestinal Tract.
- Gastric Polyps and Tumors and related source sections, printed pages 704–709.
- Histologic diagrams in this lecture are original editable schematics, not actual specimens.