

Endometrioid Ovarian Tumors

Epithelial neoplasia • pathogenesis • morphology • clinical interpretation

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Learning objectives

Locate endometrioid tumors within the ovarian epithelial tumor spectrum.

Connect endometriosis, shared molecular alterations, and tumor development.

Recognize the distinctive glandular and mixed gross–microscopic morphology.

Interpret simultaneous uterine disease, bilaterality, and stage-related outcome.

Where endometrioid tumors fit

Primary epithelial ovarian neoplasms (müllerian phenotype)



Serous

Tubal-like differentiation

Mucinous

Mucin-secreting differentiation

Endometrioid

Endometrial-type gland formation

Biologic categories across the epithelial family

Benign

Borderline

Carcinoma

Endometrioid identity rests on differentiation

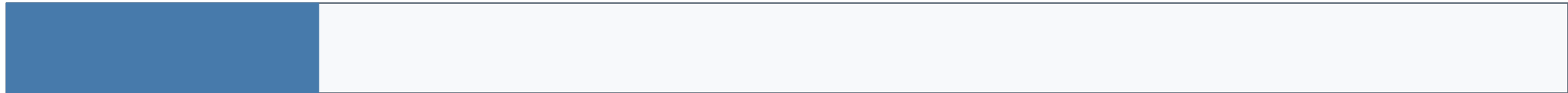
Neoplastic glands resemble the glands of the uterine endometrium.

“Endometrioid” is a morphologic designation—not proof of a uterine site of origin.

Ovarian tumors span benign, borderline, and frankly malignant forms.

Endometrioid carcinoma in the ovarian cancer spectrum

Share of ovarian cancers



20% | Reported proportion

0%

100%

Endometrioid carcinoma: Approximately 20% of all ovarian cancers in this Robbins table.

Denominator: Malignant ovarian tumors—not all ovarian masses.

The value is textbook-reported; It is not a claim about every population or current registry.

Three biologic categories

Benign

**Endometrioid
adenofibroma**

Uncommon

Borderline

**Endometrioid borderline
tumor**

Uncommon

Malignant

Endometrioid carcinoma

~20% of ovarian cancers

Benign endometrioid adenofibroma

A benign member of the endometrioid epithelial family.

The designation combines endometrioid epithelial differentiation with a fibrous component.

Distinguish benign lesions from borderline and invasive counterparts by epithelial proliferation and architecture.

Borderline endometrioid tumors

Recognized separately from benign adenofibroma and invasive carcinoma.

Uncommon according to the textbook.

Borderline status should not be equated automatically with invasive ovarian carcinoma.

Full histologic assessment is necessary before assigning a biologic category.

Benign, borderline, malignant: What the source establishes

Category	Explicit textbook point	Safe teaching interpretation
Benign	Endometrioid adenofibroma	Nonmalignant endometrioid entity
Borderline	Occurs; uncommon	Intermediate diagnostic category
Malignant	Endometrioid carcinoma	Invasive carcinoma with endometrial-like glands

Müllerian epithelial framework

Most primary ovarian epithelial neoplasms show müllerian differentiation.

Major textbook patterns include serous, mucinous, and endometrioid.

Classification combines epithelial phenotype and the extent of proliferation.

Phenotype must be integrated with invasive growth and disease distribution.

Endometrioid, serous, mucinous: Conceptual separation

Feature	Endometrioid	Serous	Mucinous
Epithelial resemblance	Endometrial glands	Tubal-like epithelium	Mucin-containing epithelium
Typical structure	Tubular glands	Cystic/papillary patterns	Multiloculated mucinous cysts
Teaching anchor	Endometrial-type morphology	Tubal/serous phenotype	Mucinous differentiation

Type I and type II ovarian carcinoma pathways

Type I: Described framework

Often follows benign / borderline disease or endometriosis

Includes endometrioid and certain other low-grade histotypes

Progression model varies by tumor type

Type II: Contrasting framework

High-grade serous tumors dominate the type II pathway

Often associated with fallopian-tube precursor lesions

Do not infer a tubal precursor for endometrioid carcinoma

Endometriosis-associated route: Supported association



Conceptual association; Not a proven obligatory sequence for every case

What is ovarian endometriosis?

Ectopic endometrial tissue is present outside the uterus.

The ovary is the most common site listed.

Ovarian endometriosis is one of three broad anatomic types of endometriosis.

Only a subset of ovarian endometrioid carcinomas is associated with it.

Endometriosis has several presentations

Superficial peritoneal endometriosis.

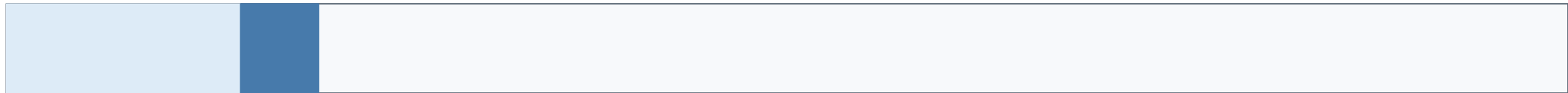
Ovarian endometriosis.

Deep infiltrating endometriosis.

Malignant transformation mainly with superficial and ovarian forms; Deep infiltrating lesions very rarely transform.

Endometriosis coexistence is not universal

Reported frequency range: 15%–20%



15–20% of the indicated population

0%

100%

Approximately 15%–20% of ovarian endometrioid carcinomas coexist with endometriosis.

Thus, many cases have no documented accompanying endometriosis.

Coexistence supports a pathogenetic link in some tumors, not a universal origin.

Age shift supports a biologic link

The peak incidence of endometriosis-associated ovarian endometrioid carcinoma is about one decade earlier.

Comparator: Ovarian endometrioid carcinomas without documented endometriosis.

Age-pattern evidence is supportive, not direct proof of lesion-by-lesion transformation.

Association versus causation

Observed

Coexistence of carcinoma and endometriosis

Earlier peak incidence of associated tumors

Overlapping genetic alterations

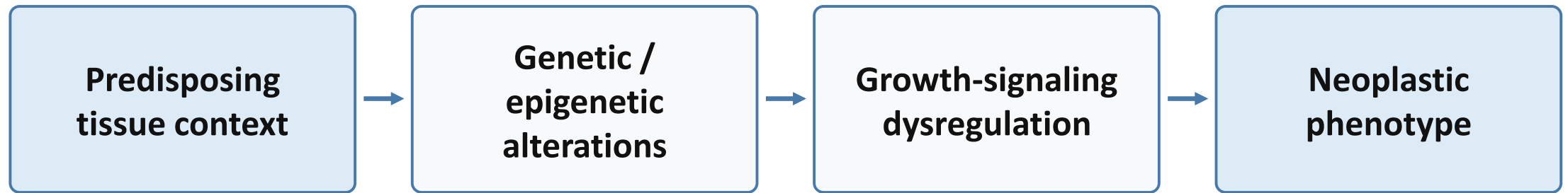
Not established by the excerpt

Every endometriotic focus becomes malignant

All ovarian endometrioid tumors arise from endometriosis

A mandatory linear precursor sequence

Pathogenesis as accumulation of changes



Teaching synthesis: Relationships are schematic and not quantified as a temporal series

A shared molecular signature with uterine carcinoma

Ovarian and endometrial endometrioid carcinomas share several commonly altered genes.

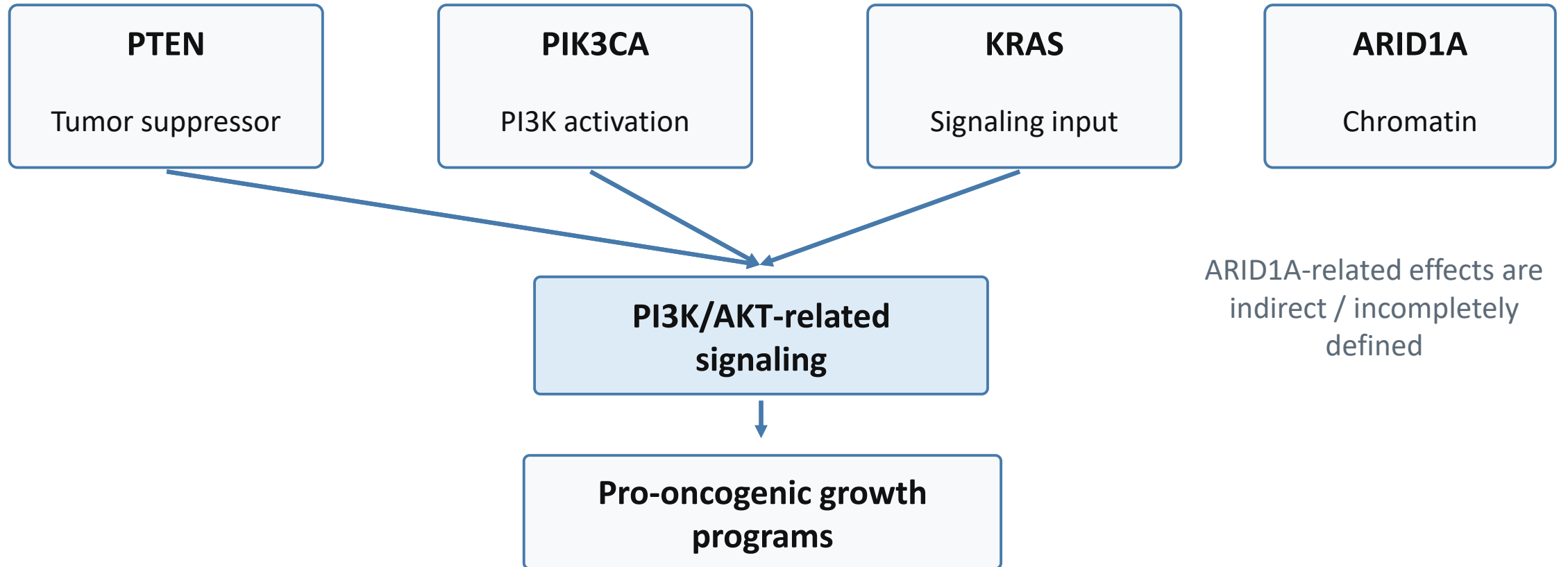
Changes that favor PI3K/AKT-related growth signaling.

Shared phenotype and pathways do not, by themselves, establish primary site.

Overlapping gene alterations provide a pathway framework, not a unique origin fingerprint.

PI3K/AKT: The organizing molecular theme

Alterations recurrently reported in ovarian endometrioid carcinoma



MMR abnormalities and TP53 (poorly differentiated cases) are related but distinct axes.

PTEN: Tumor-suppressor pathway context

PTEN appears among the genes altered in ovarian endometrioid carcinoma.

The companion endometrial chapter identifies PTEN as a tumor suppressor restraining PI3K/AKT signaling.

PTEN disruption is one mechanism consistent with increased growth signaling.

PTEN alteration alone does not determine tumor grade or organ of origin.

PIK3CA: Direct signaling-pathway component

PIK3CA is included in the shared ovarian/endometrial molecular alterations.

The companion uterine discussion identifies it as encoding a catalytic PI3K subunit.

Activating alteration can favor PI3K/AKT-pathway signaling.

Molecular identity and anatomic primary site are separate diagnostic questions.

KRAS: An oncogenic signaling input

KRAS mutations appear list of shared changes.

KRAS is an oncogene in growth-signal transduction networks.

The uterine parent section links KRAS activation with PI3K/AKT-related signaling.

A gene alteration alone is not sufficient to define ovarian tumor histotype.

ARID1A: Chromatin-regulatory biology

ARID1A alterations recur in ovarian endometrioid and clear-cell carcinomas.

ARID1A regulates chromatin structure.

The source associates its loss with pro-oncogenic transcriptional effects.

The precise causal connection to PI3K/AKT activation is described as unclear.

Mismatch-repair abnormalities

The ovarian endometrioid section mentions mutations in DNA mismatch-repair genes.

Mismatch repair preserves genomic stability by correcting replication errors.

Defects can contribute to accumulation of somatic genetic alterations.

Molecular defects complement—but cannot replace—morphologic classification.

TP53 and differentiation: A key distinction

Well-differentiated gland-forming tumors

Often display recognizable endometrial-type glands

TP53 mutations should not be assumed in every case

Architectural resemblance is central to recognition

Poorly differentiated tumors

TP53 mutations are common, per Robbins

Poor differentiation changes the molecular interpretation

Atypia and growth pattern require specific histologic assessment

Molecular map: What each alteration teaches

Gene/group	Core function or context	Textbook-supported inference
PTEN	Tumor suppressor	Loss may favor PI3K/AKT signaling
PIK3CA	PI3K catalytic component	Activation may enhance growth signaling
KRAS	Oncogenic signaling	Growth-signal activation
ARID1A	Chromatin remodeling	Indirect transcriptional effect; mechanism unclear
MMR genes	Replication-error repair	Genomic instability
TP53	Tumor suppression	Enriched in poorly differentiated tumors

Gross morphology: Mixed composition

Ovarian endometrioid carcinomas typically show solid and cystic areas.

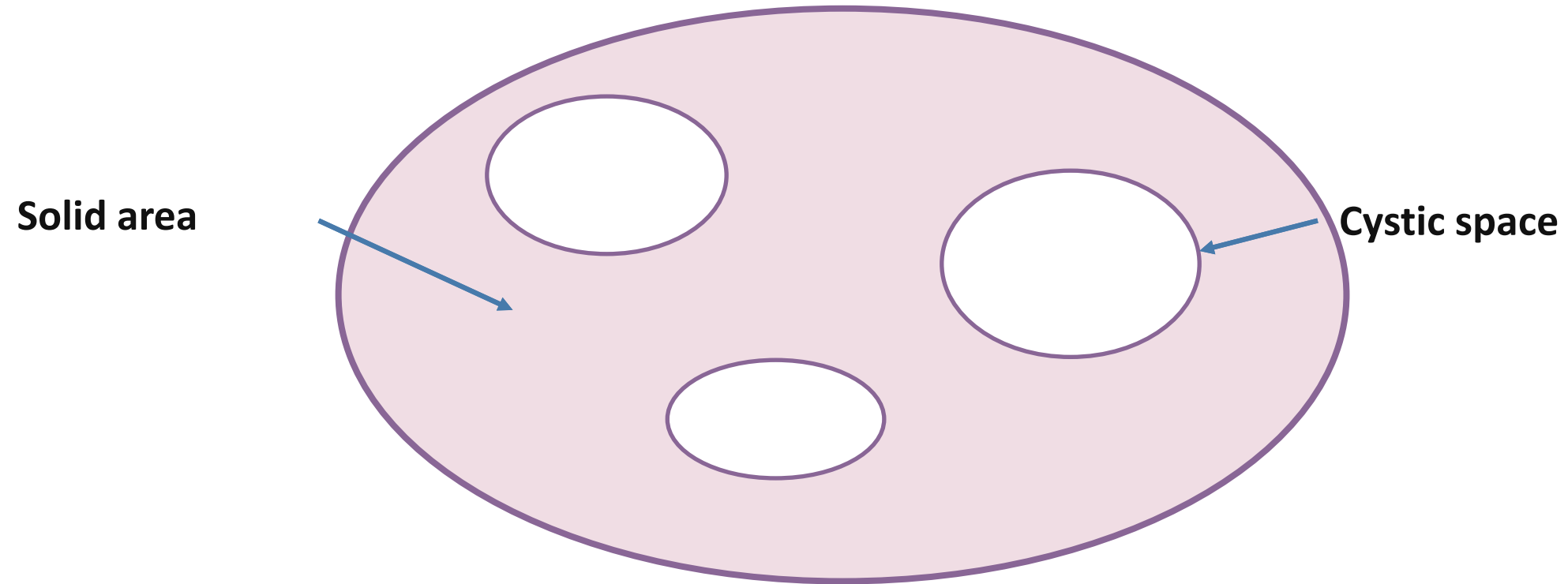
Solid and cystic appearance is a descriptive gross pattern, not a standalone diagnosis.

Histology is needed to establish endometrioid differentiation.

Specimen assessment also considers laterality and the presence of extraovarian disease.

Solid and cystic growth: Editable conceptual section

Simplified cross-section of a heterogeneous ovarian tumor



Schematic only — gross morphology is not pathognomonic without microscopy.

The histologic hallmark is tubular gland formation

Tubular glands resemble benign or malignant uterine endometrium.

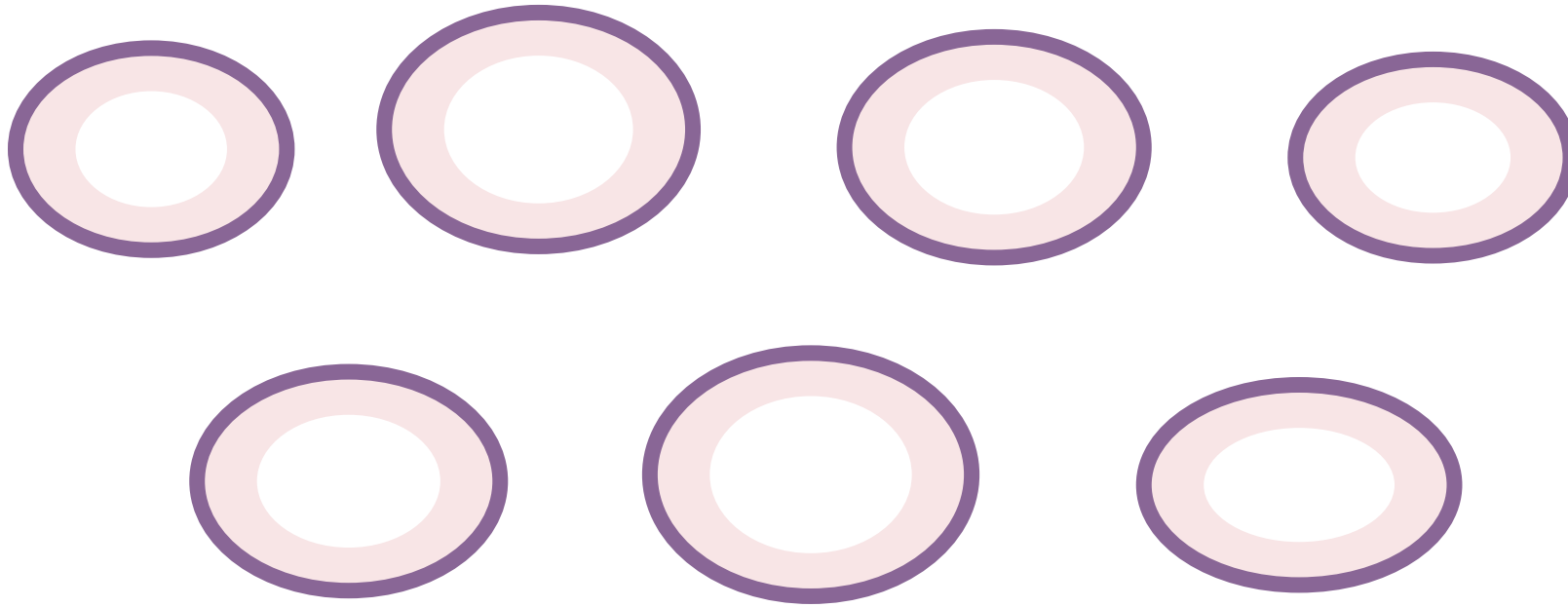
The glandular phenotype separates this type from many serous and mucinous patterns.

The architecture is assessed together with cytologic atypia and stromal invasion.

Do not confuse a glandular appearance with proof of a uterine primary.

Endometrial-type tubular glands

Tubular glands lined by endometrial-type epithelium



Editable conceptual schematic — not a histologic specimen; Assess actual H&E sections.

Glandular resemblance and tumor differentiation

Low-grade ovarian endometrioid carcinomas commonly retain a conspicuous glandular pattern.

The source also recognizes poorly differentiated tumors with frequent TP53 mutation.

Architectural resemblance becomes less informative as differentiation is lost.

Grade should not be inferred from gross solidity or an isolated genotype.

Glandular and nonglandular components

Glandular component

Tubular or closely packed endometrial-type glands

Phenotype anchors the endometrioid diagnosis

Compare pattern with uterine endometrioid neoplasia

Solid component

Solid growth may coexist with cysts

Gross solid areas do not equal microscopic grade

Histologic sampling resolves architecture and differentiation

Squamous differentiation can coexist

The textbook explicitly mentions squamous differentiation in ovarian endometrioid carcinoma.

Its presence does not automatically require a second, unrelated tumor category.

Interpret squamous areas in context of the accompanying glandular component.

Interpret squamous differentiation alongside the tumor's overall glandular phenotype.

Mucinous differentiation may also occur

Endometrioid ovarian carcinomas may contain mucinous foci.

A minor mucinous component need not reclassify the entire tumor as mucinous carcinoma.

A primary mucinous pattern is distinguished through the dominant histologic phenotype.

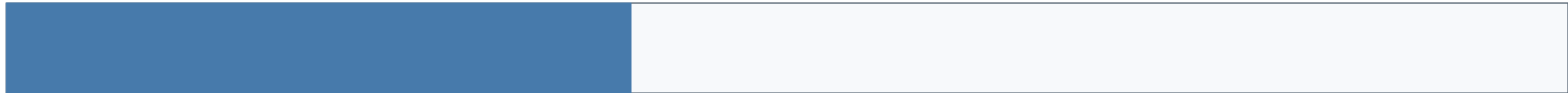
Assessment of dominant architecture and the distribution of components is essential.

Recognition checklist for an ovarian endometrioid lesion

Domain	Look for	Interpretive caution
Gross	Solid and cystic architecture	Nonspecific by itself
Microscopy	Endometrial-type tubular glands	Not proof of uterine origin
Differentiation	Squamous or mucinous foci	Can be within endometrioid carcinoma
Distribution	Bilateral disease / endometrium	Raise questions about spread or origin

Bilaterality is clinically meaningful

Both ovaries involved



40% | Reported proportion

0%

100%

That approximately 40% of ovarian endometrioid carcinomas involve both ovaries.

The text states this pattern usually implies extension beyond the genital tract.

Bilateral disease warrants careful assessment of distribution and possible secondary involvement.

Bilateral involvement differs across histotypes

Malignant tumor category	Bilateral involvement in Table 22.4	Interpretation
Endometrioid carcinoma	40%	Bilateral presentation is substantial
Serous carcinoma	65%	Commonly bilateral
Mucinous carcinoma	<5%	Usually unilateral; metastasis concern if bilateral
Clear-cell carcinoma	40%	Bilateral presentation occurs

Why bilaterality changes the question

Laterality is not simply an anatomic description; It informs the disease-distribution assessment.

Bilateral ovarian masses may reflect dissemination, an extraovarian primary, or multifocal disease.

For endometrioid morphology, inspection of the uterine endometrium is particularly relevant.

The textbook does not supply a definitive site-of-origin algorithm.

Differential diagnosis: Endometrioid versus serous

Feature	Endometrioid carcinoma	Serous carcinoma
Morphologic anchor	Endometrial-type glands	Tubal-like/serous epithelial differentiation
Textbook pathway	Type I often associated with endometriosis	High-grade serous often type II, tubal origin
Molecular emphasis	PTEN / PIK3CA / ARID1A / KRAS; MMR	High-grade serous: TP53, genomic instability
Main pitfall	Assuming uterine origin from appearance	Assuming identical precursor pathway

Differential diagnosis: Endometrioid versus mucinous

Feature	Endometrioid ovarian carcinoma	Primary mucinous ovarian tumor
Dominant differentiation	Endometrial-type tubular glands	Mucinous epithelial lining
Gross pattern	Frequently solid and cystic	Typically large multilocular mucinous cysts
Mucinous foci	May occur in endometrioid tumors	Dominant feature of mucinous tumors
Interpretation	Assess dominant architecture	Exclude metastatic disease when indicated

Differential diagnosis: Endometrioid versus clear-cell

Feature	Endometrioid carcinoma	Clear-cell carcinoma
Microscopic image	Endometrial-like glandular pattern	Large epithelial cells with clear cytoplasm
Association	Endometriosis in a subset	May also occur with endometriosis
Shared genetics	PTEN, PIK3CA, ARID1A, KRAS	Overlapping gene alterations
Caution	Preserve histotype distinction	Overlapping pathogenesis ≠ identical morphology

Brenner tumors are not endometrioid tumors

Brenner tumors show nests of urothelial-like epithelial cells in ovarian stroma.

Most are benign and their differentiation differs from endometrioid gland formation.

Overlapping benign ovarian mass presentations do not imply equivalent histopathology.

Use epithelial phenotype, not only gross appearance, for classification.

Evidence hierarchy: Histotype and primary site

Histotype: Morphologic evidence

Endometrial-type glandular phenotype

Coexisting squamous or mucinous foci

Solid and cystic growth as gross context

Primary site: Distribution evidence

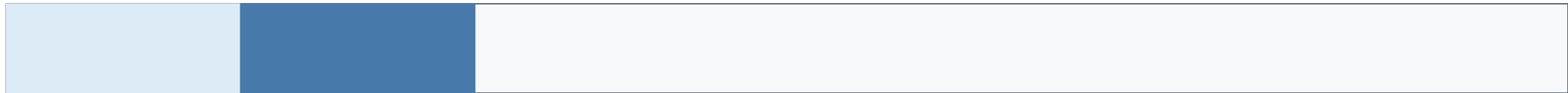
Concurrent uterine carcinoma

Bilateral ovarian involvement

Integrated anatomic and molecular
correlation

Concurrent uterine carcinoma requires evaluation

Concurrent endometrial carcinoma



15–30% of the indicated population

0%

100%

Approximately 15%–30% of ovarian endometrioid carcinomas coexist with endometrial carcinoma.

Coexistence can be clinically important even when the lesions appear histologically similar.

The source reports molecular evidence favoring endometrial-to-ovarian metastasis in most paired cases.

Paired endometrium and ovary: Two hypotheses

Independent primary tumors

Two separate primary sites might be considered

Morphologic resemblance alone cannot prove independence

Requires integrated disease mapping

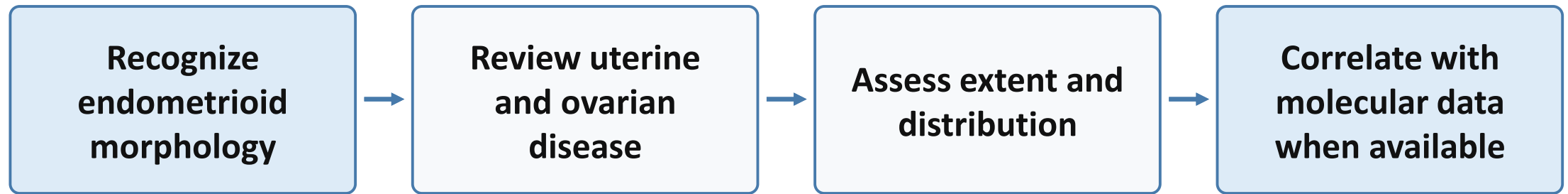
Endometrial primary with ovarian spread

Molecular evidence favors this in most paired cases

Endometrial tumor is the source of secondary ovarian disease

Recognition changes the interpretation of site and extent

Primary-site reasoning: A cautious workflow



Illustrative interpretive workflow; Not a formal diagnostic criterion set

Relatively favorable paired-case prognosis: Paradox

The text describes relatively good outcomes when ovarian and endometrial carcinomas coexist.

Favorable outcome does not, by itself, establish two independent primaries.

Molecular results may support a shared clonal origin despite the clinical course.

Do not equate outcome pattern with a definitive metastatic classification in an individual patient.

Stage I prognosis as reported

Approximately 75% 5-year survival for stage I ovarian endometrioid carcinoma.

That estimate pertains to the specified stage, not all cases.

Interpret the number as textbook-era teaching data; Not a personalized prognostic estimate.

No subtype-specific stage II–IV survival percentages are supplied here.

Why anatomic extent matters

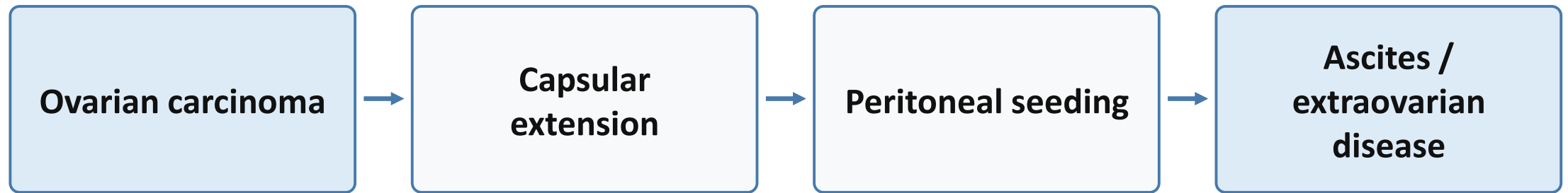
Ovarian carcinomas may extend through the capsule and seed peritoneal surfaces.

Peritoneal implantation can produce ascites and widespread abdominal disease.

Stage and extraovarian extent affect clinical behavior and treatment context.

These dissemination mechanisms are described for ovarian epithelial carcinoma generally.

A general epithelial ovarian cancer dissemination model



General ovarian epithelial carcinoma mechanism; Not unique to endometrioid histotype

Presentation: Often nonspecific

Lower abdominal pain or abdominal enlargement may prompt assessment.

Urinary frequency, pelvic pressure, and gastrointestinal symptoms can reflect mass effects.

Early benign lesions may be asymptomatic or incidentally detected.

These are general ovarian tumor features; None is histotype-specific.

CA-125: Defined role in the supplied source

Serum CA-125 is described for monitoring known ovarian carcinoma.

It may help follow recurrence or progression in a patient with established disease.

CA-125 lacks sufficient screening performance according to the textbook discussion.

Do not substitute a marker measurement for morphologic diagnosis.

Evidence boundaries in this lecture

Question	Supported by source	Not established here
Does endometriosis always precede tumor?	Association in a subset	Obligatory transformation
Do uterine + ovarian lesions equal two primaries?	Molecular evidence favors spread in most	Universal rule for individuals
Can stage I survival estimate be generalized?	~75% for reported stage I	All-stage personalized survival
Does Robbins give ovarian IHC cutoffs?	Not in the extracted unit	Any invented panel / threshold

Teaching case 1: An endometriosis-associated mass

Interpretation and teaching question

A hypothetical ovarian mass shows endometrial-type tubular glands.

Endometriosis is also found in the ovary.

Question: What pathogenetic association is supported? What claim would be too strong?

Answer and explanation on the next slide.

Case 1: Separate association from proof

Reasoned interpretation

Supported: Glandular morphology is endometrioid; Coexistence is biologically relevant.

Supported: Ovarian endometriosis may act as a precursor in some instances.

Not supported: Proving chronological transformation from the sampled focus.

Teaching case 2: Bilateral ovarian disease

Interpretation and teaching question

A hypothetical ovarian carcinoma is bilateral and endometrioid in appearance.

An endometrial mass is reported in the same patient.

Question: Which origin hypothesis must receive serious consideration?

Answer and explanation on the next slide.

Case 2: Correlate uterine and ovarian findings

Reasoned interpretation

Concurrent endometrial carcinoma occurs in approximately 15%–30%.

Molecular evidence favors an endometrial primary with ovarian spread in most paired cases in the text.

A diagnostic conclusion for an individual case needs integrated evaluation.

Teaching case 3: Mucin production

Interpretation and teaching question

A hypothetical ovarian epithelial carcinoma has endometrial-like glands.

Some regions show mucinous differentiation.

Question: Must any mucinous focus force classification as mucinous carcinoma?

Answer and explanation on the next slide.

Case 3: Use the dominant phenotype

Reasoned interpretation

Endometrioid tumors can contain mucinous areas.

The predominant architecture and epithelial phenotype remain central to interpretation.

The source provides no fixed numerical mucinous-component cutoff.

Teaching case 4: A poorly differentiated component

Interpretation and teaching question

A hypothetical ovarian endometrioid tumor contains poorly differentiated regions.

Question: Which additional molecular alteration becomes especially relevant?

Question: Does that alteration negate the possibility of endometrioid histotype?

Answer and explanation on the next slide.

Case 4: Connect grade and genotype cautiously

Reasoned interpretation

TP53 mutation enrichment in poorly differentiated endometrioid tumors.

This is a pattern, not a claim about every tumor.

Avoid extrapolating uterine-specific numerical gene frequencies to the ovary.

Teaching case 5: A molecular profile

Interpretation and teaching question

A hypothetical tumor harbors changes involving PTEN and ARID1A.

The ovarian mass also contains endometrial-type glands.

Question: What can the molecular findings support—and what can they not determine alone?

Answer and explanation on the next slide.

Case 5: Molecular phenotype is not organ assignment

Reasoned interpretation

PTEN and ARID1A are list of recurrent alterations.

Shared changes overlap with uterine endometrioid carcinoma.

Primary site still requires correlation with tumor distribution and pathology.

Synthetic educational vignette; Not patient data.

Teaching case 6: Early-stage counseling

Interpretation and teaching question

A hypothetical patient asks about prognosis after a tumor is described as stage I.

Question: Which number is available?

Question: Why should it not be framed as individual certainty?

Answer and explanation on the next slide.

Case 6: Report survival with its denominator

Reasoned interpretation

Use approximately 75% five-year survival only with the stage I qualifier.

Identify its textbook-source context; Do not convert it into a modern patient-specific estimate.

Stage, extent of disease, and differentiation inform clinical interpretation.

Synthetic educational vignette; Not patient data.

Final synthesis: Five dimensions of interpretation

Dimension	Core evidence	Common interpretive error
Type	Endometrial-like glands	Morphology proves uterine origin
Pathogenesis	Endometriosis association; PI3K/AKT alterations	All cases share one precursor
Distribution	40% bilateral; 15%–30% paired uterine lesion	Paired lesions must be independent
Differentiation	May show squamous/mucinous foci	Minor differentiation redefines whole tumor
Outcome	Stage I five-year estimate ~75%	One estimate predicts every case

Key take-home messages

Endometrioid ovarian tumors form a benign–borderline–malignant epithelial spectrum.

Carcinoma is recognized by endometrial-type tubular glands and often solid/cystic gross growth.

Endometriosis is associated with a subset, while PI3K/AKT-related genes and MMR pathways recur.

Concurrent uterine disease and bilaterality require careful assessment of origin and spread.