

Clinical Course, Detection, and Prevention of Ovarian Epithelial Tumors

Ovarian epithelial neoplasms • Clinicopathologic interpretation

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraisakhri.com

Learning objectives

- Distinguish benign, borderline, and malignant ovarian epithelial tumor courses.
- Connect local mass effects, peritoneal dissemination, and constitutional symptoms with underlying disease.
- Explain why early detection remains difficult and how CA-125 should be interpreted.
- Relate hereditary predisposition and tubal origins to risk-reducing interventions.

The central clinical problem

- Ovarian carcinomas commonly produce overlapping, nonspecific complaints.
- Carcinomas often present after extension beyond the ovary or tube.
- The burden of extraovarian disease strongly influences clinical outcome.

Symptoms are shared

**Spread changes
prognosis**

Detection is difficult

Biologic categories predict different courses

Benign

Well-differentiated epithelial proliferation;
Usually cured by resection.

Borderline

Increased epithelial proliferation without destructive stromal invasion.

Malignant

Stromal invasion; May disseminate along peritoneal surfaces and beyond.

Histologic type is distinct from biologic behavior

Differentiation

Serous, mucinous, and endometrioid are the major epithelial phenotypes.

Behavior

Benign, borderline, and malignant describe invasive potential and clinical course.

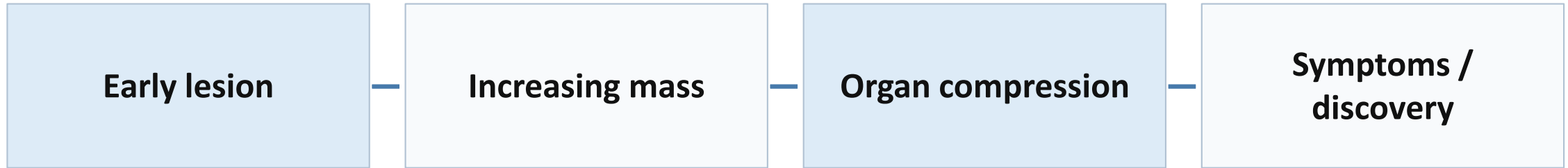
Most ovarian tumors are benign

- Approximately 80% of all ovarian tumors are benign overview.
- Benign neoplasms predominate in younger patients.
- Malignant epithelial tumors occur more often in older patients; High-grade serous tumors account for many carcinomas.

**Tumor frequency ≠
cancer frequency**

**Age influences case
mix**

Some tumors remain undetected until large



- Benign lesions can be incidental and asymptomatic.
- Nonfunctional tumors frequently cause symptoms only after reaching substantial size.

Type I and type II: Distinct trajectories

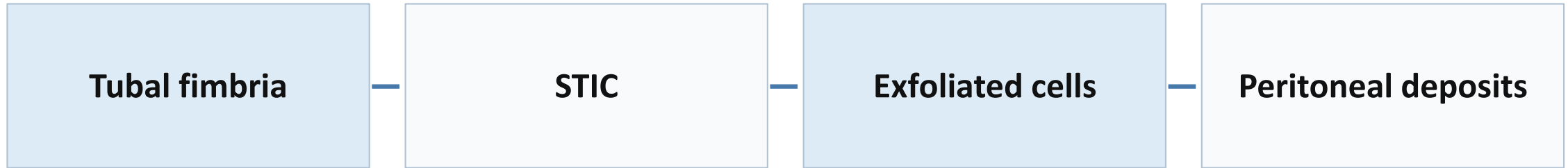
Type I group

Often associated with borderline tumors or endometriosis; Typically lower-grade progression.

Type II group

High-grade serous carcinoma commonly arises from the distal fallopian tube and presents aggressively.

A tubal precursor explains apparently ovarian disease



- Serous tubal intraepithelial carcinoma (STIC) is a recognized precursor associated with many high-grade serous carcinomas.
- Tubal carcinoma cells may exfoliate and implant on ovarian or peritoneal surfaces.

High-grade serous carcinoma: Anatomic origin matters

- The source considers most high-grade serous carcinomas likely to begin in fallopian tube epithelium.
- A primary ovarian mass does not necessarily indicate that transformation began in ovarian surface cells.
- The tubal-origin model informs the rationale for risk-reducing salpingectomy.

Tumor site ≠ origin site

**Prevention target:
Tube**

Serous tumors: Grade shapes prognosis

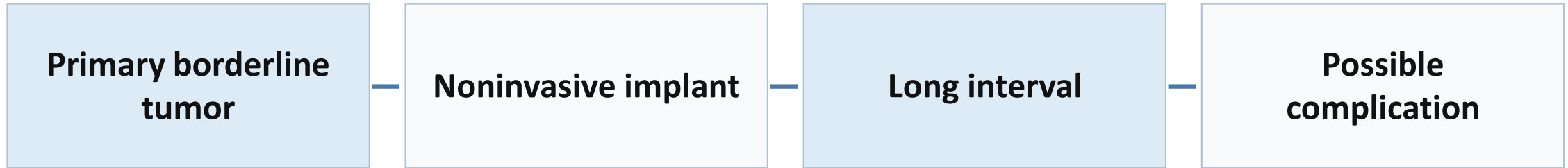
Low-grade serous carcinoma

May arise with serous borderline lesions; Relatively indolent even after extraovarian extension.

High-grade serous carcinoma

Often widely metastatic within the abdomen at diagnosis; Typically rapid clinical deterioration.

Borderline serous disease can involve the peritoneum



- Serous borderline tumors may have noninvasive peritoneal implants.
- Indolent implants can remain asymptomatic for extended periods; Progressive disease can eventually cause obstruction.

Peritoneal spread need not imply rapid progression

Borderline / low grade

May persist or recur for many years after dissemination.

High grade

May involve extensive abdominal surfaces at first presentation and worsen quickly.

Mucinous tumors: Primary cancer is uncommon

- The majority of ovarian mucinous tumors are benign or borderline.
- Primary ovarian mucinous carcinoma is an uncommon ovarian cancer subtype.
- Mucinous epithelial tumors may form very large multiloculated cystic masses.

**Mass effect can
dominate**

**Malignancy must be
assessed separately**

Bilateral ovarian mucinous masses require caution

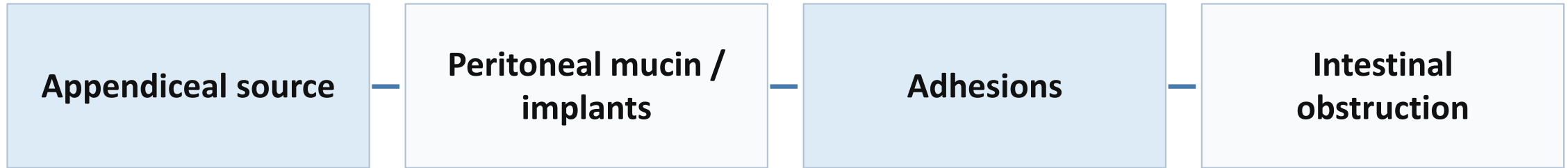
Primary ovarian mucinous tumor

Typically unilateral description.

Bilateral mucinous involvement

Requires exclusion of metastatic disease from a nonovarian primary.

Pseudomyxoma peritonei: Avoid mistaken attribution



- Mucinous ascites and peritoneal epithelial implants can cause adhesions and bowel obstruction.
- The appendix, rather than the ovary, as the usual primary site.

Endometrioid ovarian carcinoma: Linked clinical context

- Endometriosis coexists with a subset of ovarian endometrioid carcinomas.
- Concomitant endometrial and ovarian carcinoma demands evaluation of a possible uterine primary with ovarian metastasis.
- Ovarian endometrioid tumors can be bilateral when disease extends beyond the genital tract.

**Endometriosis
association**

**Primary-site
distinction**

Ovarian clear cell carcinoma: Stage is critical

Disease confined to ovary

The source reports a favorable outcome relative to advanced disease.

Advanced-stage disease

Clear-cell histology is associated with a poor outcome once disease is advanced.

Brenner tumors: An important clinical contrast

Benign Brenner tumor

Usually an incidental lesion with benign behavior, even when large.

Malignant transitional phenotype

Uncommon lesions have a course that differs by grade and may resemble other ovarian carcinomas.

Lower abdominal pain is a frequent presentation

- Lower abdominal pain is a common manifestation across ovarian carcinoma subtypes.
- Pain alone does not identify serous, mucinous, or endometrioid histology.
- Interpret the symptom in the context of pelvic mass, distribution of disease, and associated complaints.

Frequent

Nonspecific

Anatomy-dependent

Abdominal enlargement has more than one cause

Growing adnexal mass

Large ovarian tumors can increase abdominal girth by their own bulk.

Peritoneal disease with ascites

Capsular extension and peritoneal implants may generate substantial ascitic fluid.

Gastrointestinal complaints are part of the presentation

- Ovarian tumors may produce gastrointestinal complaints before their origin is clinically obvious.
- Local mass effect and peritoneal disease can contribute to bowel symptoms.
- Advanced or indolent peritoneal disease can eventually lead to intestinal obstruction.

Mass effect

Serosal involvement

**Obstructive
complications**

Urinary symptoms may arise from pelvic involvement

- Urinary frequency and dysuria occur among the reported symptoms.
- Pelvic pressure can accompany urinary or gastrointestinal complaints.
- Symptom location reflects anatomy but does not establish a specific tumor subtype.

Frequency

Dysuria

Pelvic pressure

Clinical manifestations overlap across subtypes

Local

Lower abdominal pain;
Pelvic pressure.

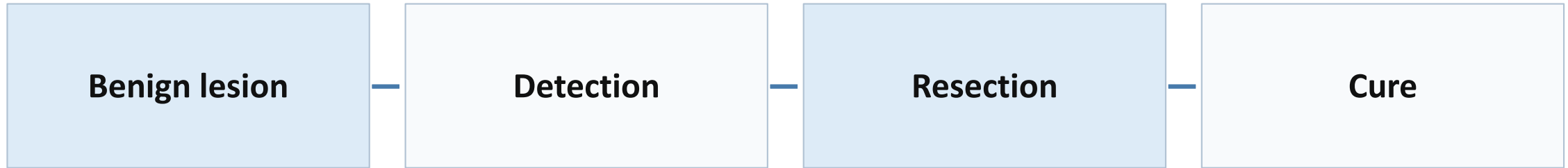
Compression

Urinary frequency,
dysuria, and
gastrointestinal
complaints.

Systemic / advanced

Weakness, weight loss,
cachexia; Ascites after
peritoneal spread.

Benign lesions usually follow a favorable course



- Benign ovarian epithelial lesions can be found incidentally or because they become large.
- Surgical resection is generally curative according to the source.

Malignancy may produce constitutional decline

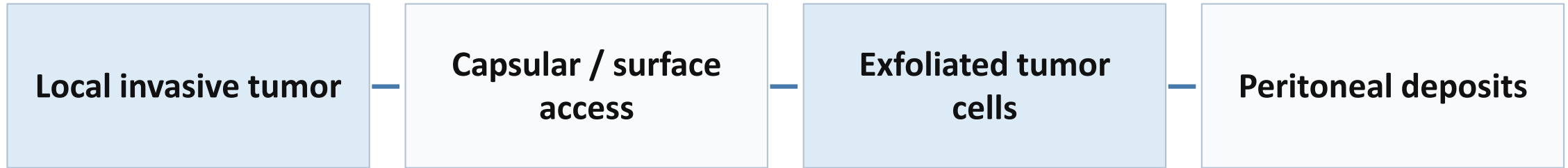
- Progressive weakness, weight loss, and cachexia are described in ovarian malignancy.
- These systemic features contrast with the predominantly local or incidental course of many benign lesions.
- Constitutional deterioration often accompanies established tumor burden.

Weakness

Weight loss

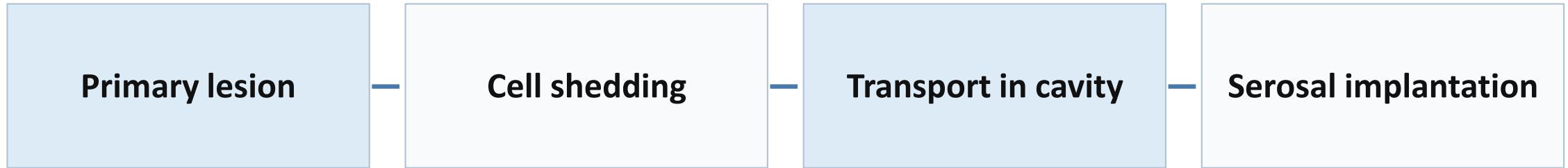
Cachexia

Capsular extension precedes peritoneal seeding



- Carcinoma extends through the tumor capsule or reaches the peritoneal surface.
- Exfoliated malignant cells can seed the peritoneal cavity.

Exfoliation provides a route of abdominal dissemination



- Tumor cells may detach into the peritoneal cavity rather than relying solely on direct deep tissue invasion.
- This dissemination pattern helps explain widespread serosal disease.

Ovarian carcinoma has a characteristic serosal pattern

- Diffuse seeding of peritoneal serosal surfaces is described.
- Implants are often small, about 0.1–0.5 cm description.
- These nodules may be extensive despite limited invasion of underlying organ parenchyma.

Widespread

Often small nodules

**Predominantly
surface-based**

Peritoneal implants are not equivalent to deep organ invasion

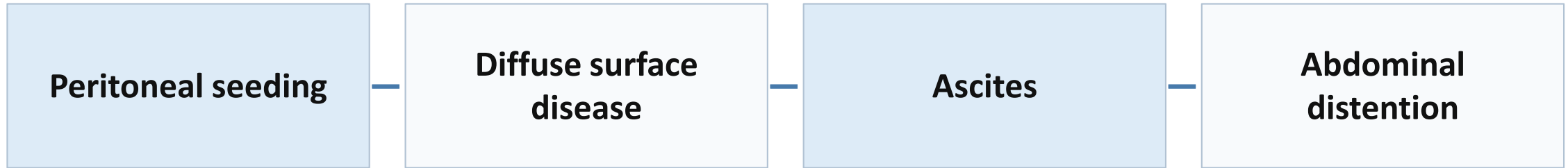
Serosal seeding

Numerous tumor nodules coat peritoneal surfaces.

Parenchymal invasion

The text notes that these serosal implants only rarely penetrate deeply into underlying organ tissue.

Ascites marks a major complication of dissemination



- When ovarian carcinoma spreads throughout the peritoneal cavity, ascites often develops.
- Fluid accumulation can be massive and contribute substantially to abdominal enlargement.

Ascitic fluid can contain malignant exfoliated cells

- Ascitic fluid containing exfoliated tumor cells.
- This finding connects tumor biology with the possibility of cytologic evaluation.
- No sensitivity, specificity, or negative-test exclusion rule for ascites cytology is given in the source.

Tumor exfoliation

Fluid-based cells

**Interpretation requires
context**

Regional lymph nodes may become involved

- Ovarian carcinoma can spread to regional lymph nodes.
- Nodal involvement represents a separate route from free intraperitoneal exfoliation.
- The textbook does not provide a nodal distribution map or incidence percentage in this section.

Peritoneal route

**Lymphatic
involvement**

Distant metastatic sites extend beyond the pelvis

Liver

Metastatic involvement is described.

Lungs

An extraabdominal metastatic site reported in the source.

Gastrointestinal tract / others

Visceral metastases may occur in addition to peritoneal surface implants.

Three dissemination patterns have different meanings

Peritoneal

Cells exfoliate and implant on serosal surfaces.

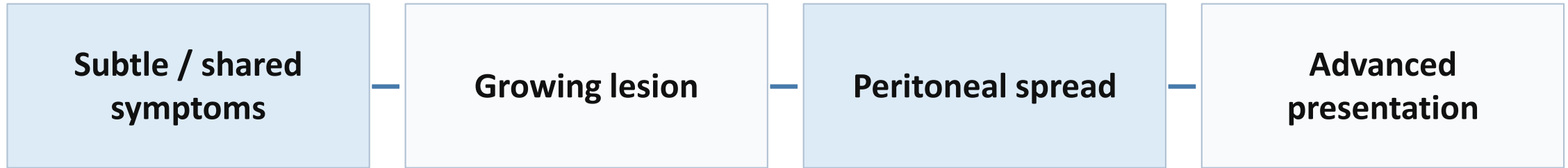
Nodal

Tumor reaches regional lymph nodes.

Distant

Metastases appear in the liver, lung, gastrointestinal tract, or elsewhere.

Why is ovarian cancer frequently advanced at diagnosis?



- Initial manifestations can be nonspecific.
- The tumor may enlarge or seed peritoneal surfaces before detection.
- Many patients therefore first present with advanced disease.

Extent of peritoneal disease is prognostically important

- Serous tumor prognosis is related to both microscopic grade and distribution of peritoneal disease.
- Spread outside the ovary changes the stage of disease.
- Extent and grade should be interpreted independently rather than treated as interchangeable.

Histologic grade

Anatomic extent

Biologic tempo

Grade influences clinical tempo after extraovarian spread

Lower-grade disease

Can progress slowly despite extension beyond the ovary; Survival may be prolonged.

Higher-grade disease

Frequently extensive at presentation and associated with rapid deterioration.

Textbook-reported five-year outcomes differ by extent

Disease category	Confined to ovary	Peritoneal involvement
Borderline serous tumors	100%	Approximately 90%
Malignant serous tumors	70%	Approximately 25%

Rates reported for the specified serous tumor categories; Not patient-specific predictions.

Five-year survival is not equivalent to cure

- Borderline tumors can recur many years after original presentation.
- A favorable five-year survival percentage therefore does not exclude delayed progression or recurrence.
- Time horizon matters when interpreting outcomes in indolent ovarian tumors.

Delayed relapse

Long follow-up

Survival $\neq$ cure

The diagnostic challenge is early-stage recognition

- Most patients with ovarian carcinoma are diagnosed with advanced disease.
- The source identifies late stage at presentation as the principal reason for poorer long-term survival relative to cervical or endometrial carcinoma.
- Improved early-detection assays remain an important research goal.

Late presentation

**Worse long-term
outcomes**

**Need for early
detection**

Screening tests require both sensitivity and specificity

Sensitivity

An early-detection test should identify a sufficiently high proportion of patients with disease.

Specificity

A screening test must also avoid excessive false positives among people without disease.

No proposed blood antigen assay is adequate for screening

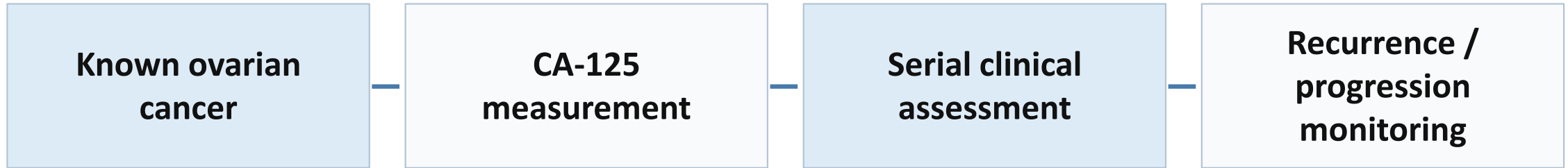
- Tumor antigens and secreted tumor products have been investigated for early detection.
- The proposed assays do not achieve the sensitivity and specificity needed for clinical utility as general early-detection tests.
- This conclusion should not be confused with their separate roles in managing known disease.

Research biomarkers

**Unmet performance
standard**

**Distinct surveillance
indication**

CA-125 has a defined role in known disease



- Serum CA-125 is used to follow patients with diagnosed ovarian carcinoma.
- Its stated role is monitoring recurrence or progression, not establishing population-wide screening efficacy.

Marker monitoring is not general-population screening

Known disease

A longitudinal marker can assist follow-up of a recognized malignancy.

No diagnosed disease

Robbins does not support CA-125 as a screening assay for asymptomatic populations.

High-risk identification differs from cancer detection

Risk identification

Family history and hereditary variants identify individuals with increased susceptibility.

Tumor detection

A separate question: Whether current disease can be identified accurately before symptoms.

Familial susceptibility is clinically consequential

- Inherited BRCA1 or BRCA2 variants increase susceptibility to ovarian and breast cancer.
- Strong family history is another signal for elevated inherited risk.
- The source places risk identification before discussion of preventive surgery.

BRCA1 / BRCA2

Strong family history

Risk management

BRCA-associated risk is substantial but heterogeneous

- The textbook estimates lifetime ovarian cancer risk by age 70 of approximately 20%–60% for BRCA1/2 mutation carriers.
- The broad estimate spans heterogeneous hereditary groups and is not an individual prediction.
- Genetic susceptibility changes prevention discussions but does not establish that a tumor is present.

By age 70

20%–60% in source

Risk ≠ diagnosis

Reproductive history modifies risk associations

Nulliparity / low parity

The source associates lower parity with increased frequency of serous ovarian carcinoma.

Oral contraceptives / tubal ligation

In women aged 40–59 years, prior oral contraceptive use or tubal ligation is associated with reduced ovarian cancer risk.

The fallopian tube is a prevention-relevant site



- High-grade serous carcinoma frequently originates in the distal fallopian tube.
- Removing the tube may interrupt a potential anatomic source before invasive cancer emerges.
- This rationale is not proof that every ovarian carcinoma is preventable by salpingectomy.

Risk-reducing salpingectomy targets tubal epithelium

- The source describes salpingectomy as a developing approach for reducing serous ovarian carcinoma risk.
- The mechanism-based rationale is removal of a frequently implicated site of origin.
- Long-term preventive impact was not established discussion.

Anatomic prevention

Potential benefit

Outcome uncertainty

Salpingo-oophorectomy is an established risk-reducing option

- For individuals at hereditary or strong familial risk, risk-reducing salpingo-oophorectomy.
- The strategy includes the fallopian tube as well as ovary, reflecting the tubal-origin evidence.
- The textbook excerpt does not specify timing, patient selection thresholds, or surgical adverse effects.

High inherited risk

Tube + ovary

**Individual decision
context**

Prevention effectiveness still requires long follow-up

Rationale

Inherited risk and a tubal precursor pathway support risk-reducing interventions.

Evidence horizon

The textbook explicitly notes uncertainty about the long-term effect of preventive strategies in high-risk groups.

Case: Pelvic symptoms without a specific histology

A patient has lower abdominal pain, gradual abdominal enlargement, urinary frequency, and pelvic pressure.

Discuss:

- Which features suggest an adnexal process?
- Why can symptoms alone not identify epithelial tumor subtype?

Case: Ascites and diffuse serosal nodules

An ovarian carcinoma is accompanied by massive ascites and numerous small nodules coating peritoneal surfaces.

Discuss:

- What route of spread explains the pattern?
- Does diffuse serosal involvement require deep organ parenchymal invasion?

Case: How should a CA-125 result be used?

A patient with an established ovarian carcinoma is being assessed for possible progression.

Discuss:

- Which use of CA-125 is supported by the source?
- Why does this not validate CA-125 screening in asymptomatic individuals?

Case: Inherited risk and prevention

A person with a pathogenic BRCA variant asks why discussion of ovarian cancer prevention includes the fallopian tubes.

Discuss:

- What precursor concept underlies the explanation?
- Which two risk-reducing operations are described, and what uncertainty remains?

Three clinical decisions should be kept separate

Course

Determine histology, grade, and distribution of disease.

Detection

Distinguish evaluation of symptoms from screening and monitoring known cancer.

Prevention

Identify hereditary risk and discuss risk reduction without overclaiming efficacy.

References and source boundaries

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. (2026), Chapter 22, pp. 923–929.
- Primary topic: Clinical Course, Detection, and Prevention of Ovarian Epithelial Tumors, pp. 928–929.
- Parent context: ovarian epithelial tumor classification, serous and other subtypes, pp. 923–928.
- No original chapter PDF or licensed textbook pathology plates were available for figure-level cross-checking.