
Gestational and Placental Disorders

Placental structure • pregnancy loss • vascular pathology • inflammation • trophoblastic disease

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Learning outcomes

- Map the maternal and fetal circulations and the trophoblast compartments.
- Link early gestational disorders to their characteristic mechanisms and morphology.
- Differentiate maternal and fetal vascular malperfusion and inflammatory lesions.
- Explain the placental basis of preeclampsia and identify the major trophoblastic lesions.

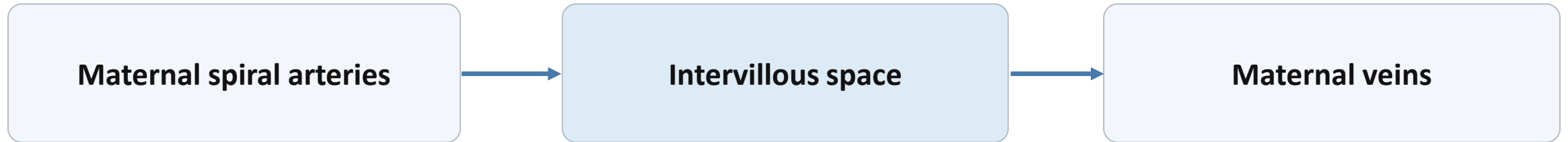
Why placental pathology matters

- Can explain fetal intrauterine or perinatal death and fetal growth restriction.
- Provides information about congenital abnormalities and maternal morbidity.
- Identifies morphologic processes relevant to immediate neonatal and maternal care.
- May inform counseling about recurrence risk in a later pregnancy.

Placental findings should be interpreted with gestational age and clinical context.

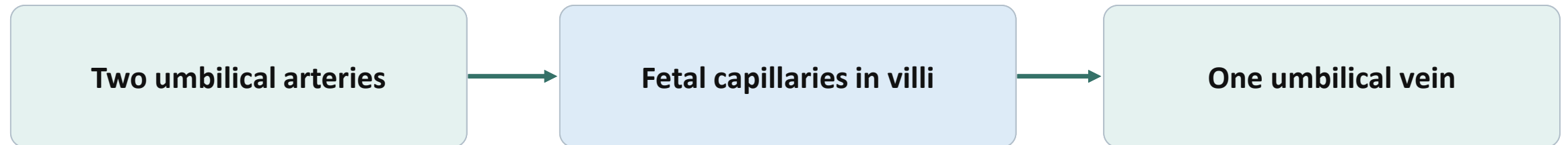
The placenta is a dual-circulation exchange organ

Maternal blood

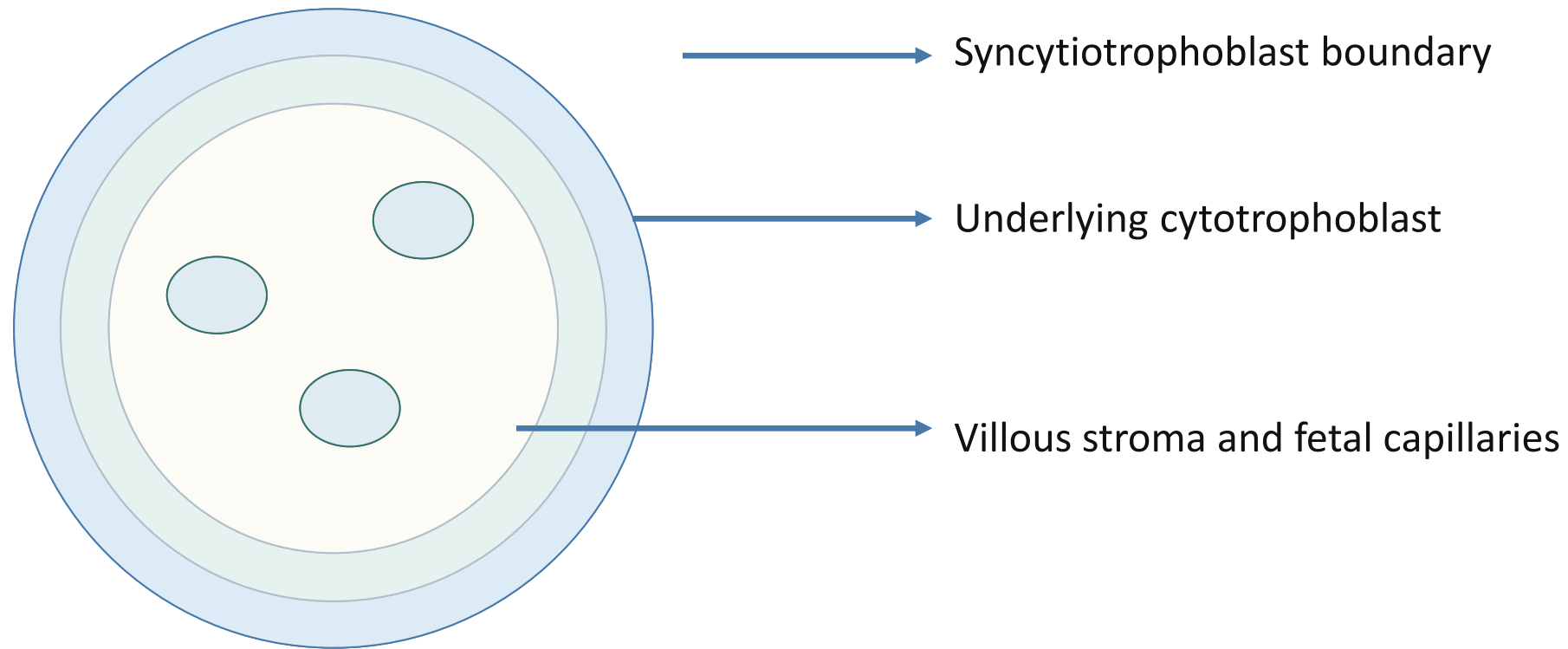


Molecule exchange across villous barrier; Blood remains in separate circulations

Fetal blood



Chorionic villi are exchange units



Schematic villus cross-section—not histology

Three functional trophoblast compartments

Syncytiotrophoblast

Cytotrophoblast

Extravillous trophoblast

**Maternal interface • exchange •
endocrine function**

**Villous trophoblast epithelial
compartment**

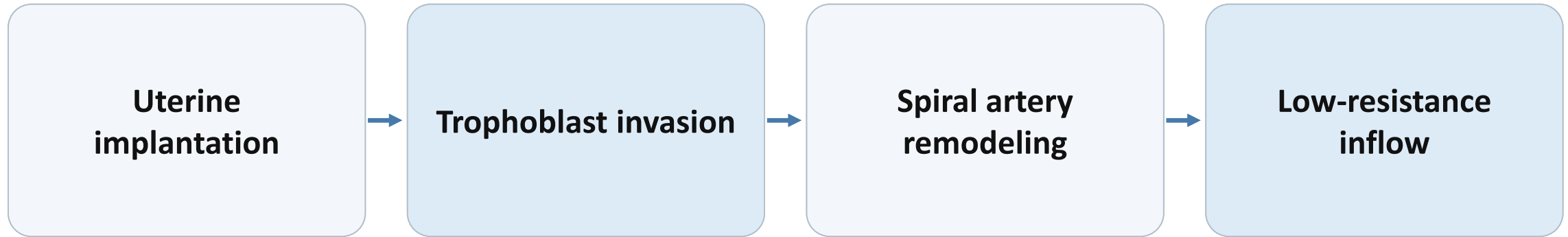
**Anchorage • uterine invasion •
vessel remodeling**

The major trophoblast groups serve different, complementary placental functions.

Villous trophoblast: Interface with maternal blood

- The syncytiotrophoblast is the outer villous epithelial surface.
- Cytotrophoblasts form an underlying trophoblast compartment.
- Together they participate in hormone secretion, transport, and barrier function.
- Fetal capillary endothelium completes the thin exchange boundary.

Extravillous trophoblast establishes uterine perfusion



Normal implantation converts maternal resistance vessels into high-capacitance placental inflow.

Normal spiral artery transformation

Before remodeling

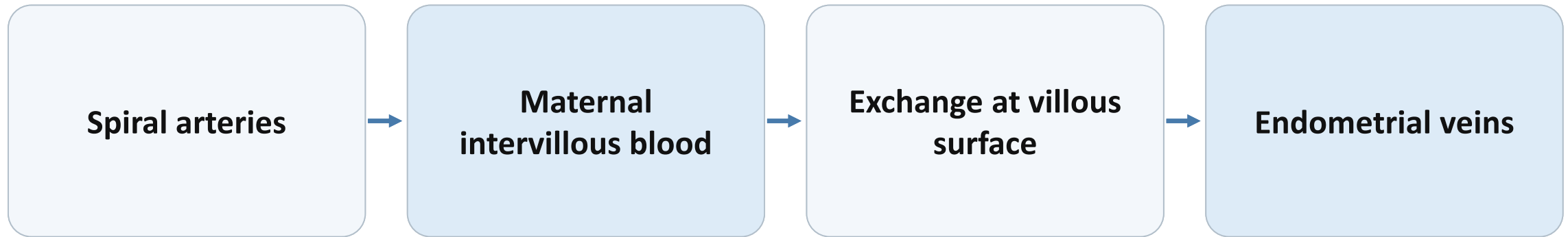
- Narrow-caliber resistance vessel
- Muscular vascular wall
- Limited flow capacity

Physiologic remodeled artery

- Extravillous trophoblast remodels wall
- Smooth muscle diminished or removed
- High-capacitance, low-resistance inflow

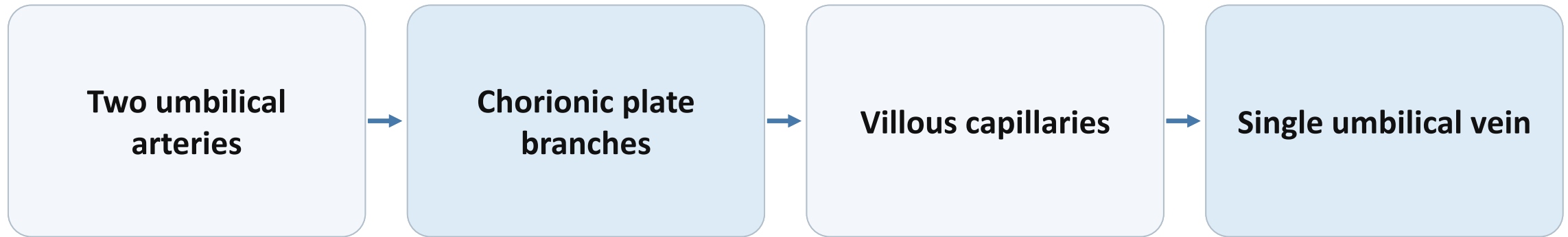
A failure of remodeling predisposes to placental ischemia.

Maternal blood enters the intervillous space



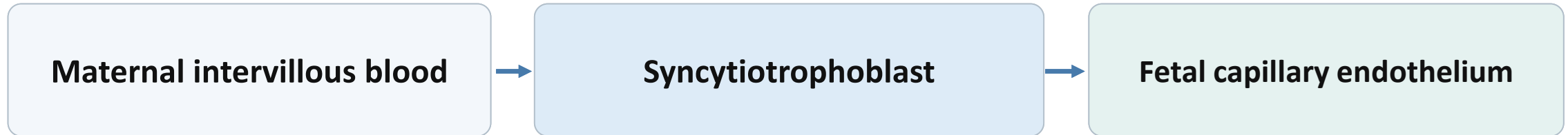
Perfusion of the intervillous space begins during early gestation; Exchange occurs without routine blood mixing.

Fetal blood traverses the villous capillary tree



Deoxygenated fetal blood is delivered to villi; Oxygenated blood returns to the fetus.

Exchange barrier becomes thin in mature villi



- Fetal capillary network lies close to the trophoblast surface.
- A thin epithelial–endothelial interface favors gas and nutrient exchange.
- Maternal and fetal blood ordinarily remain in distinct compartments.

Villous maturation changes histologic architecture

First trimester

- Prominent inner cytotrophoblast layer
- Outer syncytiotrophoblast well defined
- Delicate central stroma

Third trimester

- Dense fetal capillary network
- Thinned vasculosyncytial membrane
- Cytotrophoblast less conspicuous

Conceptual developmental comparison; Not a diagnostic micrograph.

Normal exchange does not require blood mixing

- Maternal blood bathes villi in the intervillous space.
- Fetal blood remains within capillaries inside chorionic villi.
- Small amounts of cell-free fetal DNA enter maternal blood, likely through syncytiotrophoblast shedding.
- The latter principle supports prenatal genetic testing, without implying wholesale blood mixing.

Approach placental disease by mechanism

Pregnancy establishment

Abortion • ectopic implantation

Perfusion abnormalities

Maternal and fetal malperfusion

Inflammation

Ascending or hematogenous infection

Trophoblast disorders

Hydatidiform mole • neoplasia

Timing helps organize the differential

First / early second

**Spontaneous abortion
Ectopic pregnancy**

Mid second

**Fetal vascular malperfusion
Infection • placental hypoplasia**

Third / peripartum

**Cord lesions • malperfusion
Implantation disorders •
preeclampsia**

Timing is a diagnostic framework, not an exclusive or rigid boundary.

Spontaneous abortion: Definition and distribution

- The textbook defines miscarriage as pregnancy loss before 20 weeks of gestation.
- Most reported miscarriages occur before 12 weeks.
- An important proportion of clinically recognized gestations end in miscarriage.
- Sensitive HCG testing reveals additional very early pregnancy losses.

Definition and frequency follow Robbins; Local clinical terminology and legal cutoffs may differ.

Etiologic framework for pregnancy loss

Fetal genetics

**Aneuploidy
Polyploidy
Rearrangements**

Maternal endocrine

**Luteal-phase defect
Uncontrolled diabetes**

Uterine structure

**Submucosal leiomyoma
Polyp • malformation**

Vascular / immune / infectious

**Antiphospholipid syndrome
Infection • immune dysregulation**

Chromosomal abnormalities drive many early losses

- Aneuploidy, polyploidy, and chromosomal translocations are major fetal factors.
- Chromosomal anomalies in approximately half of early abortuses.
- Additional subtle genetic defects may evade routine testing.
- A detected fetal chromosome anomaly identifies a mechanism, not necessarily a maternal disorder.

Maternal factors may disrupt implantation

Endocrine

Structural

Vascular

Hormonal / metabolic support

Uterine implantation site

Maternal placental perfusion

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graph TD; Endocrine[Endocrine] --> Hormonal[Hormonal / metabolic support]; Structural[Structural] --> Uterine[Uterine implantation site]; Vascular[Vascular] --> Placental[Maternal placental perfusion]; Hormonal --> Impaired[Impaired establishment or maintenance of pregnancy]; Uterine --> Impaired; Placental --> Impaired;
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Impaired establishment or maintenance of pregnancy

Pregnancy loss: Vascular, infectious, immune factors

- Antiphospholipid syndrome, coagulopathy, and hypertension may compromise maternal vessels.
- Pathogens can ascend from the cervicovaginal tract or spread hematogenously.
- Ascending infection is particularly relevant to second-trimester losses.
- Disturbed maternal tolerance of fetal trophoblast may contribute to recurrent loss.

Maternal immune tolerance protects the hemi-allograft

- The fetoplacental unit carries paternal genetic material.
- The maternal immune system must support extravillous trophoblast invasion without destructive rejection.
- Robbins implicates disturbances in innate and adaptive immunity in some recurrent miscarriages.
- Defective regulatory T-cell activity is discussed as a possible mechanism, not a universal explanation.

Ectopic pregnancy: Implantation outside the uterine cavity

Normal site
Endometrial uterine cavity

Most frequent ectopic site
Fallopian tube

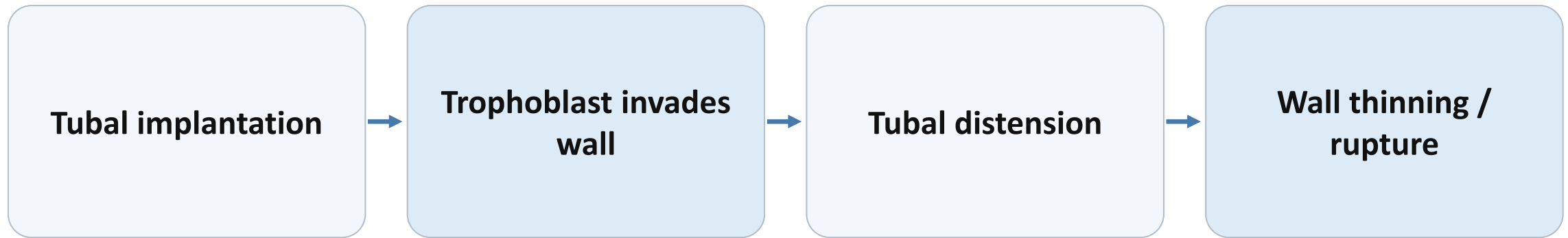
Other sites
Ovary • abdomen • scar •
interstitial region

- An extrauterine location cannot accommodate normal placental invasion and growth.
- Fallopian tube as the dominant site (~90%).

Tubal scarring is a major ectopic risk factor

- Chronic salpingitis from pelvic inflammatory disease can scar the tubal lumen.
- Peritubal adhesions can follow endometriosis, appendicitis, or abdominal/pelvic surgery.
- Prior surgery and abnormal tubal transport can impair passage toward the uterine cavity.
- Some ectopic pregnancies occur in tubes without recognizable lesions.

Tubal pregnancy progresses from implantation to rupture



Rupture can cause massive intraperitoneal hemorrhage and shock.

Morphology of tubal ectopic gestation

- Immature villi and trophoblast implant within the tubal lumen.
- Trophoblast and villi invade the tubal wall as gestation develops.
- The tube becomes distended; Hematosalpinx is an important morphologic clue.
- Rupture produces hemorrhage into the peritoneal cavity.

Ruptured ectopic pregnancy is an emergency

- Abdominal pain and vaginal bleeding may occur after several weeks of amenorrhea.
- Rapid intraperitoneal blood loss can cause an acute abdomen and hemorrhagic shock.
- Investigation may use serial HCG assessment and pelvic ultrasonography.
- Endometrial decidual change without villi may support—but alone does not prove—extrauterine implantation.

Clinical pathway is source-derived; It does not replace urgent patient-specific care.

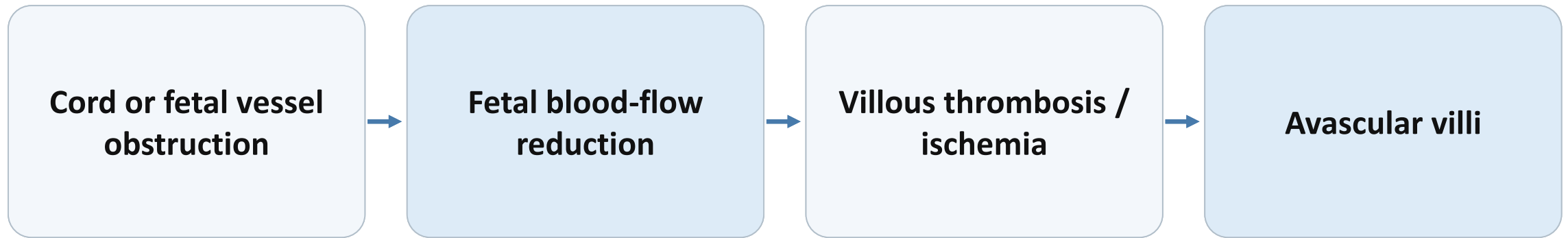
Miscarriage versus ectopic pregnancy

Feature	Spontaneous abortion	Tubal ectopic pregnancy
Primary process	Loss of an intrauterine gestation	Implantation within fallopian tube
Key mechanisms	Fetal genetic and maternal factors	Scarring / altered tubal passage
Morphologic clue	Products of conception may be present	Hematosalpinx; Villi invade tube
Immediate hazard	Bleeding; Retained gestational tissue	Tubal rupture; Intraperitoneal hemorrhage

Second trimester: Demand exceeds placental reserve

- Fetal metabolic and nutritional needs rise rapidly during the second trimester.
- An immature placenta has relatively limited reserve for exchange.
- Infection, placental hypoplasia, and diminished fetal vascular perfusion may precipitate loss.
- The same lesions may cause fetal demise earlier than they would at term.

Fetal vascular malperfusion: Mechanism



Cord compression or thrombosis may produce a pattern of fetal-side villous injury.

Umbilical cord pathology compromises fetal blood flow

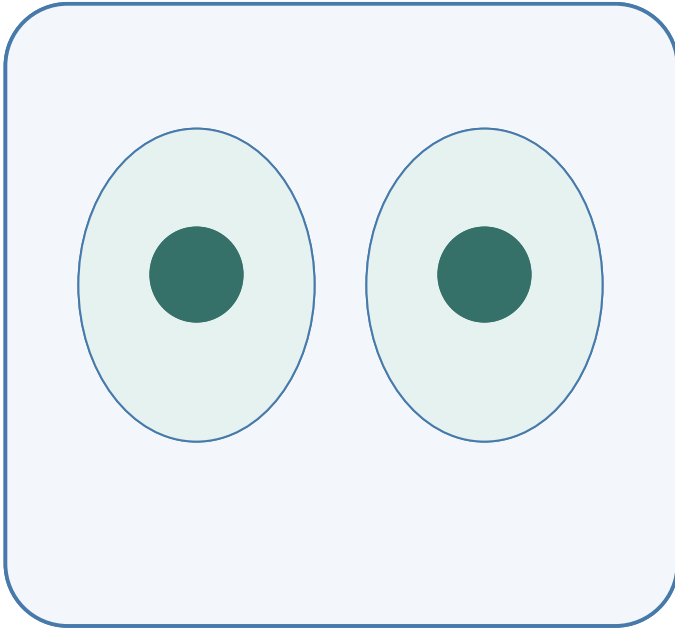
- True knots, kinking, prolapse, and compression can obstruct umbilical vessels.
- Acute obstruction may cause fetal death while leaving few specific placental changes.
- Chronic or intermittent compromise can produce fetal vascular thrombi and avascular villi.
- Fetal vascular lesions should be interpreted with cord and clinical findings.

Maternal versus fetal vascular malperfusion

	Maternal vascular malperfusion	Fetal vascular malperfusion
Primary circulation	Uteroplacental / spiral arteries	Umbilical or fetal placental vessels
Proximal defect	Poor maternal supply to intervillous space	Impaired flow within fetal circulation
Typical findings	Infarcts; Distal villous hypoplasia	Thrombi; Avascular villi
Clinical associations	Growth restriction; Preeclampsia	Cord lesions; Fetal compromise

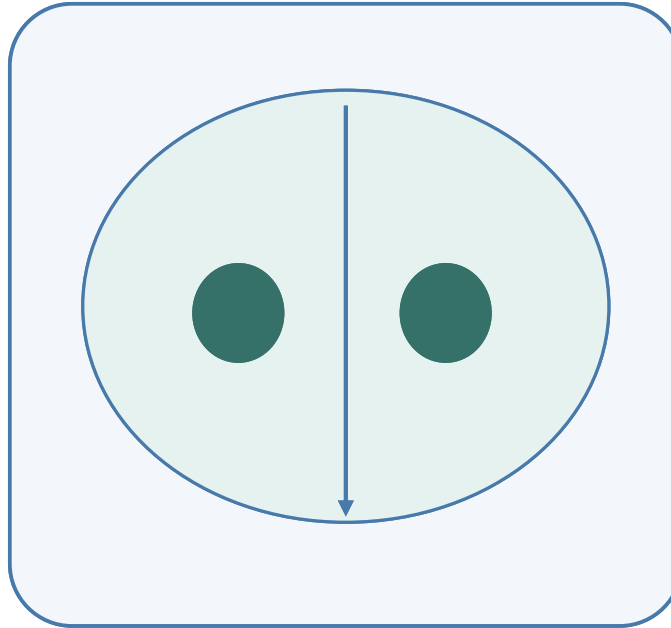
Twin placentation determines shared vascular risk

Dichorionic diamniotic



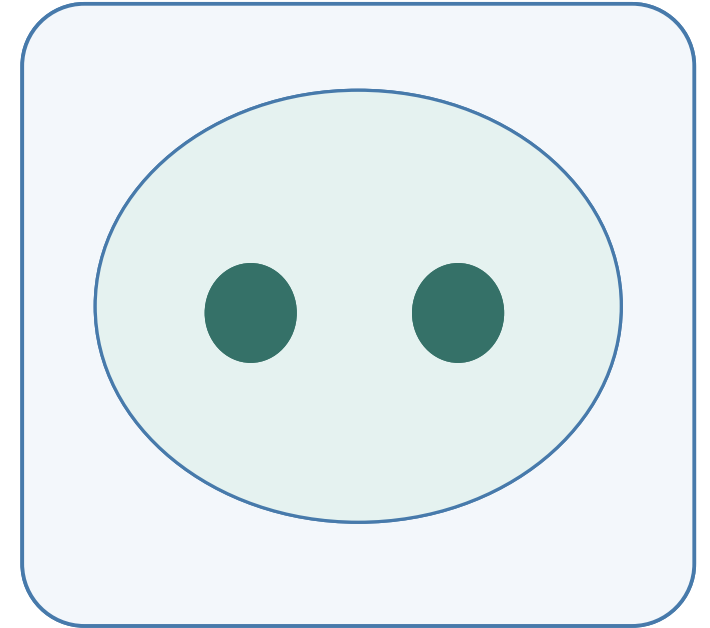
Chorionic compartments: 2
Amniotic sacs: 2

Monochorionic diamniotic



Chorionic compartments: 1
Amniotic sacs: 2

Monochorionic monoamniotic



Chorionic compartments: 1
Amniotic sacs: 1

Twin-to-twin transfusion follows unequal shunting

Shared monochorionic placenta



Placental vascular anastomoses

Donor twin
Relative underperfusion

Recipient twin
Fluid overload

Third-trimester placental pathology: A clinical map

Fetal-side flow

Umbilical cord lesions • thrombi

Implantation

Placenta previa • accreta spectrum

Maternal-side flow

Placental infarction • malperfusion

Inflammation

Chorioamnionitis • Membrane or villous infection

Maternal vascular malperfusion: Core mechanism

- Inadequate maternal delivery of blood to the intervillous space produces placental ischemia.
- Causes include abnormal implantation/remodeling and maternal vascular disease.
- Consequences range from fetal growth restriction to severe uteroplacental injury.
- The pathologic diagnosis is based on a constellation of findings rather than one isolated lesion.

Morphologic pattern of maternal vascular malperfusion

Placental hypoplasia

Weight below gestational-age expectation

Decidual vasculopathy

Abnormal spiral vessel remodeling

Infarcts

Ischemic parenchymal injury

Villous ischemia

Distal villous hypoplasia / syncytial knots

Placental infarcts reflect reduced maternal supply

- Infarction is one recognized component of maternal vascular malperfusion.
- An ischemic placenta may show numerous or extensive infarcts.
- Placental weight and infarct burden require interpretation relative to gestational age.
- Infarction and impaired flow help explain uteroplacental insufficiency and restricted fetal growth.

Abnormal implantation: Two distinct clinical hazards

Placenta previa

- Implantation over / near internal cervical opening
- May cause severe bleeding in late pregnancy
- Placental location is the principal abnormality

Placenta accreta spectrum

- Decidua deficient at implantation site
- Villi appose or extend into myometrium
- Failure of separation may cause postpartum hemorrhage

These disorders may coexist but are not synonyms.

Placenta previa: Malpositioned implantation

- Placental tissue lies in the lower uterine segment and covers the cervical region.
- May be associated with significant third-trimester hemorrhage.
- Bleeding risk arises from placental attachment at the site of cervical change.
- Placental site and obstetric context determine management.

Placenta accreta spectrum: Loss of the decidual plane

Normal separation plane

Placental villi

Decidua

Myometrium

Decidual interface between villi and myometrium

Accreta spectrum

Placental villi

Myometrium

Focally absent decidua: Direct villous–myometrial contact

Placental infection: Two routes, different tissues

Ascending route

- Microbes ascend from the cervicovaginal canal
- Usually involve membranes and amniotic fluid
- Acute chorioamnionitis with neutrophils

Hematogenous route

- Bloodstream transmission from maternal infection
- Microbes reach placental villi
- Often associated with chronic villitis

The route of infection predicts the predominant inflammatory compartment.

Acute chorioamnionitis: Maternal response

- Ascending bacterial infection can inflame amnion and chorion.
- Amniotic fluid may appear cloudy or purulent.
- Maternal neutrophils infiltrate placental membranes.
- Local inflammation may accompany premature rupture of membranes or preterm delivery.

Maternal and fetal inflammatory responses differ

Response	Principal site	Interpretation
Maternal acute chorioamnionitis	Chorion-amnion membranes	Maternal neutrophilic response
Fetal inflammatory response	Umbilical vessels / chorionic plate vessels	Fetal vasculitic response
Hematogenous villitis	Chorionic villi	Inflammation in exchange parenchyma

Hematogenous infection predominantly affects villi

- Maternal sepsis, viremia, or parasitemia can seed the placenta.
- Transplacental infections may cause chronic villitis.
- Infectious villitis is associated with impaired fetal growth and infection risk.
- Pathogen identification requires clinicopathologic correlation; A pattern alone is not species-specific.

Placental infectious patterns: Diagnostic comparison

Process	Main compartment	Usual pattern
Ascending infection	Membranes / amniotic fluid	Neutrophilic chorioamnionitis
Fetal response	Umbilical / chorionic vessels	Inflammation of fetal vessels
Transplacental infection	Villi	Villitis; Often chronic
Uncommon bloodborne bacteria	Villi / intervillous region	May elicit acute inflammation

Preeclampsia: Placental disease with systemic effects

- A maternal syndrome with new-onset hypertension after 20 weeks and evidence of organ involvement.
- Maternal endothelial dysfunction is central to its manifestations.
- Renal, hepatic, hematologic, pulmonary, and neurologic complications may occur.
- Eclampsia denotes seizures complicating the syndrome.

Textbook clinical description is retained; No separate external guideline criteria are asserted.

Preeclampsia complications follow endothelial injury

Placenta-derived endothelial disturbances

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graph TD; A[Placenta-derived endothelial disturbances] --> B[Vasoconstriction]; A --> C[Leaky endothelium]; A --> D[Prothrombotic state]; B --> E[Hypertension]; C --> F[Edema / proteinuria]; D --> G[Organ microthrombi]
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The diagram is a flowchart illustrating the path from placenta-derived endothelial disturbances to various complications of preeclampsia. At the top, a light blue box labeled 'Placenta-derived endothelial disturbances' has three arrows pointing down to three separate light blue boxes: 'Vasoconstriction', 'Leaky endothelium', and 'Prothrombotic state'. Below each of these boxes is a corresponding light green box representing a complication: 'Hypertension' under 'Vasoconstriction', 'Edema / proteinuria' under 'Leaky endothelium', and 'Organ microthrombi' under 'Prothrombotic state'.

Vasoconstriction

Hypertension

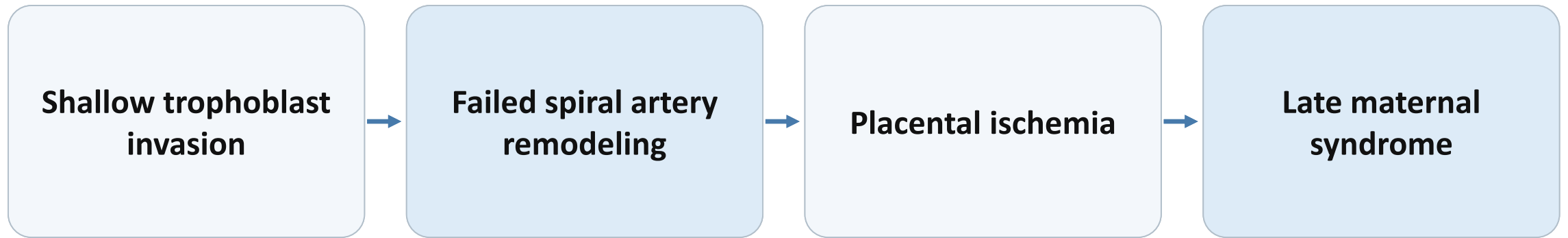
Leaky endothelium

Edema / proteinuria

Prothrombotic state

Organ microthrombi

Preeclampsia begins with defective placentation



The vascular defect begins early; Clinical manifestations commonly appear later.

Normal remodeling versus preeclamptic placentation

Normal

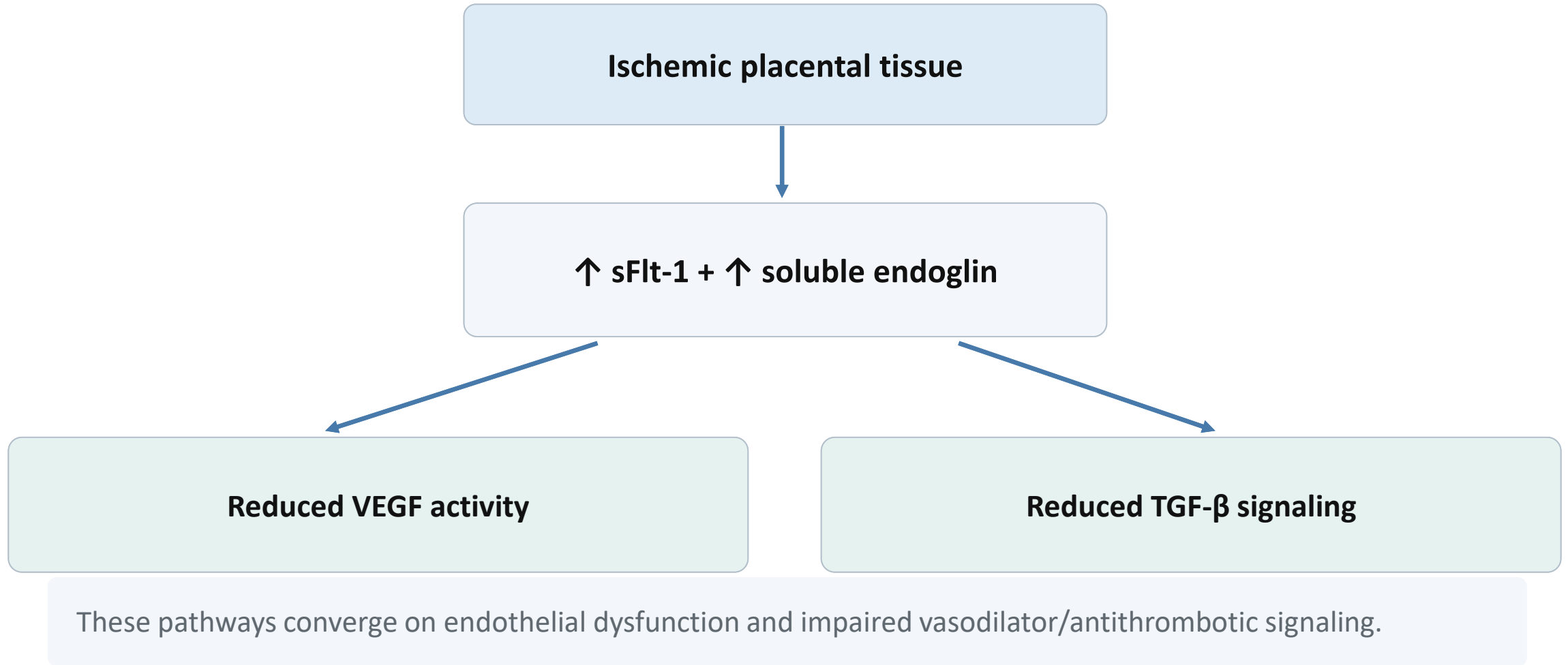
- Extravillous trophoblast enters vessel walls
- Smooth-muscle media reduced
- Large-caliber, low-resistance uteroplacental vessel

Preeclampsia

- Invasion and remodeling are incomplete
- Spiral arteries retain resistance-vessel character
- Placental supply may become inadequate as fetal demands grow

Shallow implantation is an initiating lesion, not merely a consequence of late hypertension.

Antiangiogenic mediators link placenta to maternal vessels



Endothelial dysfunction explains systemic injury

Maternal endothelial dysfunction

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graph TD; A[Maternal endothelial dysfunction] --> B[Vascular tone]; A --> C[Permeability]; A --> D[Hemostasis]; B --> E[Vasoconstriction • hypertension]; C --> F[Proteinuria • edema]; D --> G[Microthrombi • ischemia];
```

Vascular tone

Vasoconstriction • hypertension

Permeability

Proteinuria • edema

Hemostasis

Microthrombi • ischemia

Preeclampsia generates a prothrombotic state

- Widespread endothelial injury favors microvascular thrombus formation.
- Placenta, kidneys, liver, brain, and pituitary may be affected.
- Reduced VEGF signaling may lower endothelial prostacyclin (PGI₂) activity.
- The resulting imbalance promotes coagulation and tissue hypoperfusion.

Placental lesions associated with preeclampsia

Placental hypoplasia

Low placental mass for gestation

Distal villous hypoplasia

Small, shrunken villi; Syncytial knots

Infarction

Extensive ischemic foci

Decidual arteriopathy

Acute atherosclerosis / fibrinoid necrosis

Renal and hepatic pathology in severe preeclampsia

Kidney

- Glomerular endothelial swelling
- Endothelial-side dense material / fibrin
- In severe cases, glomerular and cortical thrombosis

Liver

- Subcapsular or intraparenchymal hemorrhage
- Microvascular fibrin thrombi
- Focal hemorrhagic necrosis

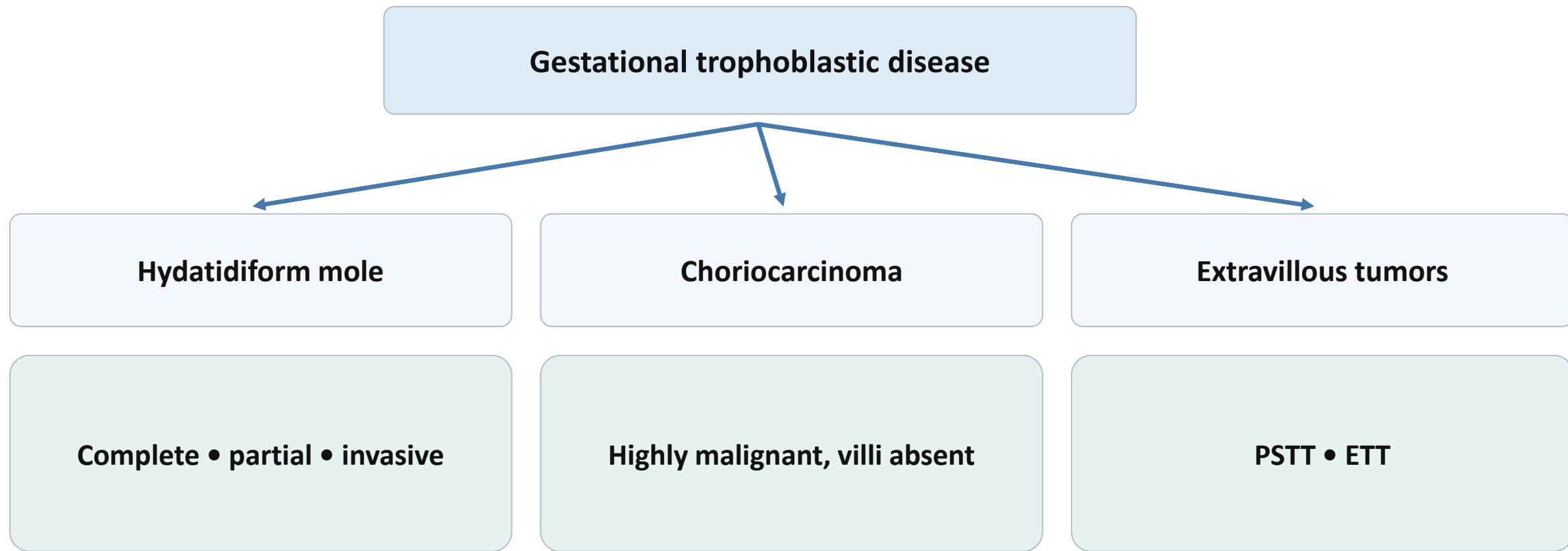
Clinical pathology includes proteinuria, renal failure, and hepatic complications.

Gestational hypertension, preeclampsia, eclampsia, HELLP

Term	Defining feature context
Gestational hypertension	New pregnancy-associated hypertension without preeclampsia features
Preeclampsia	New hypertension plus evidence of maternal systemic involvement
Eclampsia	Seizures complicating preeclampsia
HELLP syndrome	Hemolysis • elevated liver enzymes • low platelets

Gestational trophoblastic disease: A spectrum

Gestational trophoblastic disease



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graph TD; A[Gestational trophoblastic disease] --> B[Hydatidiform mole]; A --> C[Choriocarcinoma]; A --> D[Extravillous tumors]; B --> E[Complete • partial • invasive]; C --> F[Highly malignant, villi absent]; D --> G[PSTT • ETT];
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The diagram illustrates the spectrum of gestational trophoblastic disease. It starts with a central box labeled 'Gestational trophoblastic disease'. Three arrows point from this box to three sub-categories: 'Hydatidiform mole', 'Choriocarcinoma', and 'Extravillous tumors'. Below each of these sub-categories is a box listing specific types or characteristics: 'Complete • partial • invasive' for Hydatidiform mole, 'Highly malignant, villi absent' for Choriocarcinoma, and 'PSTT • ETT' for Extravillous tumors.

Hydatidiform mole

Complete • partial • invasive

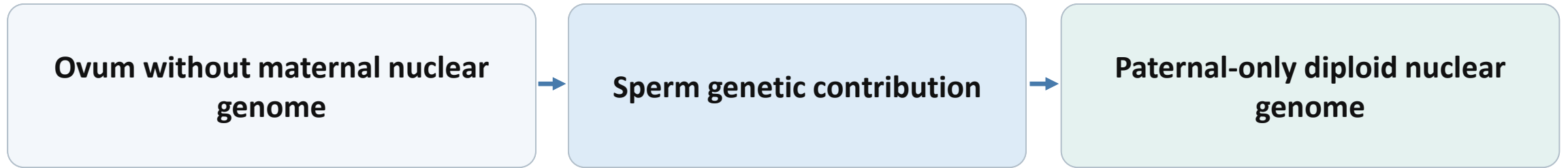
Choriocarcinoma

Highly malignant, villi absent

Extravillous tumors

PSTT • ETT

Complete mole: Androgenetic diploid concept



- Commonly: One sperm genome duplicates → 46,XX.
- Less commonly: Dispermy of an empty ovum → 46,XX or 46,XY.
- Embryonic development is absent in typical complete mole.

Partial mole: Usually diandric triploidy



- Typical triploid constitutions include 69,XXY and 69,XXX.
- Some fetal development may occur but is abnormal.
- Persistent molar disease is possible; Postmolar choriocarcinoma is uncommon.

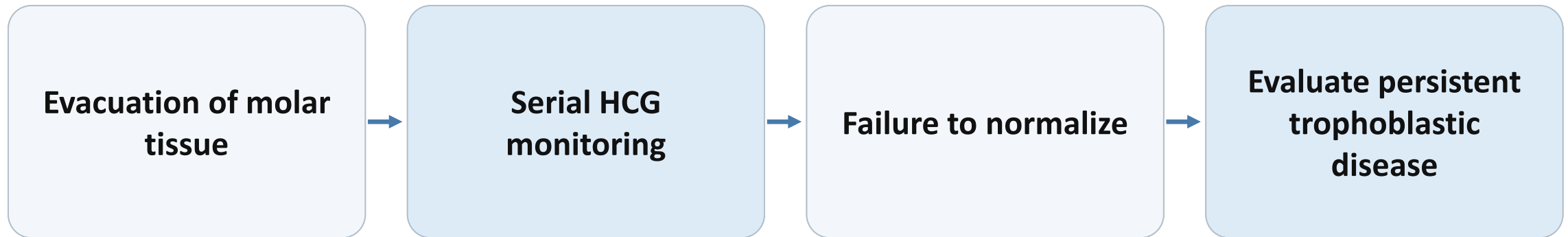
Complete versus partial mole: Diagnostic morphology

Feature	Complete mole	Partial mole
Villi	Diffuse enlarged, hydropic villi	Subset enlarged, often scalloped
Trophoblast	Marked circumferential proliferation	More focal / mild proliferation
Villous outline	Typically smooth, rounded	Irregular; Trophoblastic inclusions
Embryonic tissue	Absent	May be present
Typical genetics	Paternal-only diploid	Usually triploid

Hydatidiform mole: Clinical recognition and follow-up

- Abnormal ultrasound appearance or early miscarriage may prompt evacuation.
- HCG levels in complete mole can substantially exceed those in normal pregnancy.
- Molar villi are hydropic; Histologic assessment distinguishes complete and partial patterns.
- Serial HCG follow-up after evacuation detects persistent trophoblastic disease.

Persistent HCG after molar evacuation is a warning



Persistent HCG warrants assessment for invasive mole or choriocarcinoma; No patient-specific protocol is depicted.

Invasive mole: Villi extend into myometrium

- Hydropic villi with trophoblastic proliferation penetrate the uterine wall.
- Myometrial infiltration may cause persistent bleeding and uterine enlargement.
- HCG may remain elevated following evacuation.
- Villi may extend into vessels and extrauterine sites; Unlike choriocarcinoma, villi are present.

Choriocarcinoma: Malignant trophoblast without villi

- The lesion comprises proliferating malignant cytotrophoblast and syncytiotrophoblast.
- Chorionic villi are absent, a decisive morphologic distinction from invasive mole.
- Gross tumors show extensive hemorrhage and necrosis.
- Destructive vascular invasion permits early hematogenous metastasis.

Three major malignant trophoblastic patterns

Entity	Distinguishing morphology	Clinical tendency
Invasive mole	Hydropic chorionic villi invading myometrium	Persistent HCG; Occasional emboli
Choriocarcinoma	Malignant biphasic trophoblast; Villi absent	Hemorrhagic invasion and metastases
Placental site trophoblastic tumor	Mononuclear extravillous trophoblast; Vessel tropism	Uterine mass; Variable HCG
Epithelioid trophoblastic tumor	Mononuclear epithelioid cells; Hyaline material	Mass lesion; Metastatic potential

Gestational choriocarcinoma: Clinical behavior

- May arise after molar pregnancy, miscarriage, or apparently normal gestation.
- Bleeding can present during or after pregnancy; Diagnosis may be delayed.
- Common metastatic destinations include lung and vagina.
- Gestational choriocarcinoma is highly chemosensitive, unlike its nongestational counterpart.

Extravillous trophoblast tumors: PSTT and ETT

- Placental site trophoblastic tumor (PSTT) is composed of invasive mononuclear extravillous trophoblast.
- PSTT may infiltrate myometrium and show affinity for maternal vessels.
- Epithelioid trophoblastic tumor (ETT) comprises epithelioid mononuclear trophoblast and hyaline material.
- Both lesions can be malignant; Their morphology differs from biphasic choriocarcinoma.

Pathologic interpretation: Integrate location and lesion

Site of gestation

Intrauterine versus ectopic

Compartment

Villi • membranes • cord • decidua

Pattern

Ischemia • inflammation • trophoblast

Clinical context

Gestational age • fetal growth • maternal disease

Applied case: Early pregnancy with shock

A patient with amenorrhea, pelvic pain and vaginal bleeding develops hypotension. At surgery, a distended fallopian tube and hemoperitoneum are found.

- What is the most likely lesion?
- What histologic structures establish gestational implantation?
- Why can the lesion become immediately life-threatening?

Case interpretation: Ruptured tubal pregnancy

- The combination of a tubal gestation and intraperitoneal bleeding supports ruptured ectopic pregnancy.
- Chorionic villi and trophoblast within tubal tissue demonstrate implantation.
- Invasion and distension may thin and rupture the tubal wall.
- Massive blood loss is the immediate mechanism of hemodynamic collapse.

Applied case: Hypertension with fetal growth restriction

After midgestation, new maternal hypertension and proteinuria accompany fetal growth restriction. Placental examination shows infarcts, small distal villi and abnormal decidual arteries.

- Which placental vascular process explains the findings?
- How does abnormal early implantation produce a late maternal syndrome?
- What maternal organs may be damaged by endothelial dysfunction?

Case interpretation: Preeclampsia with malperfusion

- The placental pattern supports maternal vascular malperfusion.
- Defective spiral artery remodeling can limit placental perfusion.
- Placenta-derived antiangiogenic mediators contribute to widespread endothelial dysfunction.
- Maternal renal, hepatic, cerebral and hematologic complications may follow.

Applied case: Enlarged hydropic chorionic villi

After evacuation of an abnormal early gestation, pathology shows enlarged hydropic villi and trophoblast proliferation. The clinician asks whether this is complete or partial mole and whether surveillance is needed.

- What microscopic patterns distinguish the mole types?
- What genetic origin supports complete mole?
- Why does persistently elevated HCG matter after evacuation?

Case interpretation: Hydatidiform mole

- Complete mole has diffuse villous hydropic enlargement with circumferential trophoblast hyperplasia.
- Partial mole shows heterogeneous scalloped villi, inclusions, and more focal proliferation.
- Complete mole usually has a paternal-only diploid nuclear genome.
- Persistent postevacuation HCG can indicate invasive/persistent trophoblastic disease.

Synthesis: Lesion → compartment → clinical consequence

Pathology	Diagnostic compartment	Principal consequence
Tubal ectopic pregnancy	Fallopian tube villi / trophoblast	Rupture, hemorrhage
Fetal malperfusion	Cord / fetal villous vessels	Fetal ischemia; Avascular villi
Maternal malperfusion	Decidua / intervillous supply	Growth restriction; Infarcts
Ascending infection	Membranes / fetal vessels	Preterm delivery / perinatal injury
Preeclampsia	Placental bed plus maternal endothelium	Hypertension and organ dysfunction
Gestational trophoblastic disease	Villous / extravillous trophoblast	Persistent disease or malignancy

Selected reference

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

Foundational pathology reference