



Placental pathology

A review of Gross and Microscopic features

Dr. Bimal Patel, GAIMS, Bhuj

The 2014 Amsterdam International Consensus Group, as published in *Archives of Pathology & Laboratory Medicine* (2016), provides standardized guidelines for placental examination. Key requirements include noting whether the specimen is fresh or fixed—as fixation increases weight by 3% to 6%; documenting any basal plate sampling, and calculating the fetoplacental weight ratio to evaluate fetal growth restriction. (Key point: **Placental weight**)

Placental disk assessment involves measuring three dimensions—maximum length, perpendicular width, and the range of mural thickness—which can identify potential associations with pregnancy complications such as fetal growth restriction. Furthermore, documenting these measurements is critical for determining functional reserve by correlating the size of lesions to the total placental volume.

The **umbilical cord description** must document the diameter, length, and insertion site distance from the nearest placental margin, along with strictures and segmental coiling patterns (hypo- or hypercoiled). A thin cord may indicate FGR, while a thick cord is associated with maternal diabetes or fetal hydrops, and both excessively long/short cords and marginal (<1cm) or velamentous insertions are linked to adverse outcomes. Furthermore, hypocoiling (<1 coil/10cm) and hypercoiling (>3 coils/10cm) present risks, with deep grooves in hypercoiled cords specifically associated with stillbirth.

Membranes should be assessed for completeness, color, and opacity. It is essential to record the presence of circumvallate or circummarginate configurations, including the percentage of the placental circumference they affect. Because circumvallation is often linked to early pregnancy bleeding, the membranes may exhibit iron deposition, which typically manifests as a noticeable brown or yellow-brown discoloration.

Placental lesion descriptions should document the number (single or multiple) and location (central, paracentral, peripheral) of findings, alongside size measurements indicating maximum dimensions or the percentage of parenchyma affected. To ensure thorough pathological assessment, a minimum of one tissue block must be sampled from each identified lesion apart from the sampling as described below.

Sampling of placenta:

Submit 4 blocks as a minimum:

1 block: a roll of the extraplacental membranes from the rupture edge to the placental margin, including part of the marginal parenchyma;

1 block: 2 cross sections of the umbilical cord, one from the fetal end and another approximately 5 cm from the placental insertion end;

3 blocks: each containing a full-thickness section of normal-appearing placenta parenchyma. Full-thickness samples should be taken from within the central two-thirds of the disc and include one adjacent to the insertion site itself.

* A full-thickness sample should be taken from close to the umbilical cord insertion site to document fetal vascular ectasia and fetal and/or maternal inflammatory response.

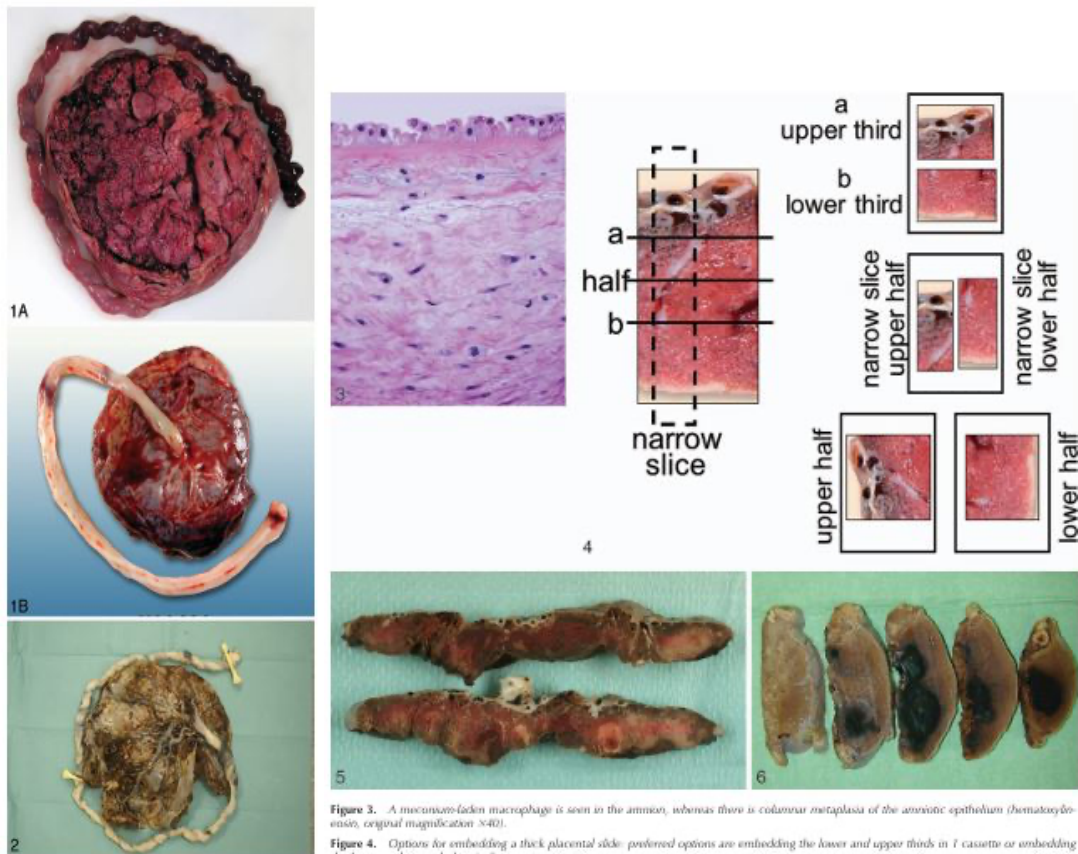


Figure 1. A, A hypercoiled umbilical cord. B, A hypercoiled cord.
Figure 2. A long and hypercoiled umbilical cord with areas of relatively less coiling (eg, lower right).

Figure 3. A meconium-laden macrophage is seen in the amnion, whereas there is columnar metaplasia of the amniotic epithelium (hematoxylin-eosin, original magnification $\times 40$).

Figure 4. Options for embedding a thick placental slide: preferred options are embedding the lower and upper thirds in 1 cassette or embedding the lower and upper halves in 2 separate cassettes.

Figure 5. Chronologic dating of infarcts is easier in the fixed placenta because the lesions are better demarcated: the older infarcts appear tan colored, whereas the fresher ones appear red and congested.

Figure 6. Fresh placental abruption resulting in marked congestion of the overlying placental parenchyma.

All images are taken from the following article and can be referred for better understanding- (Arch Pathol Lab Med. 2016;140:698–713; doi: 10.5858/arpa.2015-0225-CC)

Placental classification (incorporating the 2014 Amsterdam Placental Workshop Group criteria)

1. Placental vascular processes

a. Maternal stromal-vascular lesions

Developmental

- Superficial implantation/decidual arteriopathy
- Increased immature extravillous trophoblast

Malperfusion

Global/partial

- Early: distal villous hypoplasia
- Late: accelerated villous maturation

Segmental/complete

- Villous infarct(s)

Loss of integrity

- Abruption/placenta (arterial)
- Marginal abruption (venous)
 - Acute
 - Chronic

b. Fetal stromal-vascular lesions

Developmental

- Villous capillary lesions
- Delayed villous maturation (maturation defect)
- Dysmorphic villi

Malperfusion

Global/partial

- Obstructive lesions of umbilical cord
- Recent intramural fibrin in large fetoplacental vessels
- Small foci of avascular or karyorhectic villi

Segmental/complete

- Chorionic plate or stem villous thrombi
- Large foci of avascular or karyorhectic villi

Loss of integrity

- Large vessel rupture (fetal hemorrhage)
- Small vessel rupture (feto-maternal hemorrhage)
- Villous edema

2. Placental inflammatory- immune processes

a. Infectious inflammatory lesions

Acute

- Maternal inflammatory response: chorioamnionitis, subchorionitis
- Fetal inflammatory response: chorionic/umbilical vasculitis

Chronic

- Villitis (CMV, others)
- Intervillositis (malaria, others)

b. Immune/ idiopathic inflammatory lesions

Villitis of unknown etiology and related/ associated lesions

Chronic villitis

- Chronic chorioamnionitis
- Lymphoplasmacytic deciduitis
- Eosinophil T-cell fetalvasculitis

Chronic histiocytic intervillositis

3. Other placental processes

- Massive perivillous fibrin (old) deposition (maternal floor infarction)
- Abnormal placental shape or umbilical insertion site
- Morbidly adherent placentas (accreta)
- Meconium-associated changes
- Increased circulating nucleated red blood cells

Placental lesions

Maternal vascular malperfusion (MVM) is identified through a combination of gross findings, including placental hypoplasia—defined as a placental weight or umbilical cord diameter below the 10th percentile and/or a thin cord (<10th centile or <8mm dia at term), retroplacental hemorrhage, and infarction (in preterm or term placenta more than 5% of non-peripheral infarction should be described). Microscopic features of MVM, which are best evaluated in fixed specimens for chronological dating, include villous abnormalities and pyramidal-shaped infarcts (central > peripheral), specifically those involving the basal parenchyma or maternal floor.

Retroplacental hemorrhage is identified grossly as maternal surface blood accumulation causing parenchyma compression. Indentation of the placental parenchyma and two dimensions (length and width) or the percentage of maternal surface area involved should be recorded. Any separate clot should be measured and weighed. At least 1 block should be submitted. Microscopically blood dissecting the decidua with associated villous compression, crowding, and early necrotic changes is noted. Also, a smudged appearance maybe seen as evidence of early coagulation necrosis of the syncytiotrophoblast nuclei and pale appearance of syncytiotrophoblast nuclei. While *placental abruption* represents a clinical diagnosis, the accurate pathological descriptor is retroplacental hemorrhage or hematoma.

Chronicity of infarction:

Placental infarction progresses from early congestion, hemorrhage, and neutrophil infiltration to late-stage necrosis characterized by pyknosis, karyorrhexis, and the development of "ghost villi." This transition involves the loss of nuclear staining in both the stroma and trophoblast over time.

Distal villous hypoplasia (DVH) is defined by a lack of villi relative to the surrounding stem villi, where the existing villi appear thin, elongated, and show an increase in syncytial knots. This condition is best observed under low power by comparing the lower two-thirds of the placental parenchyma to the subchorionic third. A diagnosis is made when these features involve at least 30% of one full-thickness slide. DVH is graded as "focal" if features present in only one slide and "diffuse" if seen in two or more. This condition is significantly associated with maternal vascular malperfusion (MVM) and early-onset fetal growth restriction (FGR).

Accelerated villous maturation (AVM) is defined as the presence of small or short hypermature villi for gestational period, usually accompanied by an increase in syncytial knots. The diagnostic criterion involves identifying a diffuse pattern of term-appearing villi with increased syncytial knots and intervillous fibrin, usually alternating with areas of villous paucity. Note areas adjacent to infarcts should not be relied on for the diagnosis of accelerated villous maturation.

AVM may be found in mild, moderate, or severe forms of placental insufficiency, which includes FGR, preeclampsia, and preterm labor.

Delayed villous maturation (DVM)

(Deferred terms: villous maturation defect, variable villous maturation, or villous dysmaturity)

DVM is defined as a monotonous villous population (defined as at least 10 such villi) with centrally placed capillaries and decreased vasculosyncytial membranes, recapitulating the histology in early pregnancy, with at least presence of 30% of 1 full thickness parenchymal slide as a diagnostic criterion. Proposed Grading schema: focal- the lesion in 1 full-thickness parenchymal slide only; diffuse- lesion is present in 2 or more full-thickness slides sampled.

Syncytial Knots: Syncytiotrophoblast is the multinucleated epithelium of the placenta, in which many nuclei are dispersed within the syncytioplasm, while others are aggregated into specializations referred to as **true and false syncytial knots**, and **syncytial sprouts**. Nuclei within true knots display highly condensed chromatin and are thought to be aged. True knots increase in frequency with gestational age. Excessive formation of knots (Tenney-Parker change) is associated with placental pathology. A knotting index is used to assess severity. However, this index is potentially confounded by the creation of artifactual appearances (false knots) through tangential sectioning. Syncytial sprouts, markers of trophoblast proliferation, are defined as collections of nuclei attached to the villous surface by a stalk of cytoplasm.

Decidual arteriopathy refers to a group of structural changes in the blood vessels located within the **basal plate**, the **membrane roll**, or both areas of the placenta. This condition is marked by **acute atherosclerosis** and **fibrinoid necrosis**, which sometimes involves the presence of **foam cells**. It also includes features such as **mural hypertrophy**, **chronic perivasculitis**, and the formation of **arterial thrombi**. Pathologically, it is defined by a lack of proper **spiral artery remodeling**—or even the **total absence of spiral arteries**—along with the abnormal **persistence of intramural endovascular trophoblasts** into the third trimester.

Fetal Vascular Malperfusion

Fetal Vascular Malperfusion (FVM) is a condition where fetal blood flow is obstructed. While it was previously called **fetal thrombotic vasculopathy**, that term is now discouraged. This condition is identified by several microscopic findings, including blood clots (thrombosis), patches of villi without blood vessels (avascular villi), and damaged cells within the villous tissue. Other signs include fibrin build-up in vessel walls, hardened or narrowed vessels, and stretched, dilated veins.

FVM is divided into two categories: **low grade** and **high grade**. Low grade FVM is segmental, meaning it is limited to specific areas where blood vessels are blocked or narrowed.

High grade FVM is more widespread (global) and is often caused by partial or repeated blockage of the umbilical cord. Diagnostic criteria of FVM is finding of more than one focus of avascular villi (a cumulative assessment of ≥ 45 avascular villi over 3 sections examined or an average of >15 villi per section) with or without thrombus, or 2 or more occlusive or nonocclusive thrombi in chorionic plate or major stem villi, or multiple nonocclusive thrombi.

Thrombosis

It is essential to determine its origin as arterial or venous and to document the exact location, including the umbilical cord, chorionic plate, or stem vessels. Cases often involve combinations of these sites, providing critical insights into the severity and pattern of blood flow obstruction.

Avascular villi

A note of how they are spread out and how many are affected is essential. These villi have lost all their blood vessels and show a type of scarring called hyaline fibrosis. They are categorized by the size of the groups, or "foci," that they form. **Small foci** are defined as having three or more groups of 2 to 4 damaged villi. **Intermediate foci** consist of groups of 5 to 10 villi, while **large foci** contain more than 10 damaged villi in a single group.

Intramural fibrin deposition

(formerly called an "intimal fibrin cushion") is a lesion found within the walls of large fetal vessels. These can be either recent or remote; recent deposits consist of fresh fibrin or fibrinoid material, while older, remote deposits often show signs of calcification. These can be located at subendothelial or intramuscular, and thus intramural. By definition, these deposits are non-occlusive. To identify this, look for fibrin located just beneath the vessel lining or within the muscle of the vessel wall. When multiple deposits are found, they typically indicate global Fetal Vascular Malperfusion (FVM). However, if only a single deposit is seen on a slide, its clinical significance is less certain.

Villous Stromal-Vascular Karyorrhexis

It is a condition where fetal cells within the villi, such as red blood cells, white blood cells, and structural cells, begin to break apart or fragment. This process was previously known as **hemorrhagic endovasculitis**, but that term is now deferred. To diagnose this, three or more groups (foci) containing 2 to 4 terminal villi that show this cell fragmentation should be noted. A key feature of this lesion is that while the internal fetal cells are damaged, the surrounding outer layer, known as the **trophoblast**, remains intact.

Stem Vessel Obliteration

It is also known as **fibromuscular sclerosis**, occurs when the walls of the stem vessels become significantly thickened. This thickening eventually causes the lumen to close up or be "obliterated," stopping blood flow. This finding was previously called **stem vessel endovasculopathy**, but that term is now deferred.

Vascular Ectasia

Vascular ectasia is characterized by the finding of vessels that are four times the luminal diameter of the surrounding corresponding vessel.

Placental inflammatory-immune processes

Three important aspects to under this process are:

- **The Anatomical Interfaces:** The **cervicovaginal interface**, where placenta faces the outside world (bacteria, viruses). At the **maternal-fetal interface**, it faces the mother's immune system, which should technically see the fetus as a "foreign invader" (antigenically different).
- **The Immune Balancing Act:** To keep the fetus safe, the mother's **cell-mediated immunity** (the part that attacks foreign tissue) is naturally dampened. While this prevents "fetal rejection," it leaves the door slightly ajar for certain pathogens, which is why pregnant individuals are more vulnerable to specific infections like **Listeria** or **Malaria**.
- **Cellular Inflammation:** Because this balance is so delicate, "accidental" inflammation is common. When the body fails to suppress the immune response perfectly, you see **villitis** (inflammation of the placental villi), which is essentially the mother's immune cells starting to treat the placenta like a mismatched organ transplant.

Ascending intrauterine infection

Acute cellular inflammatory responses by infectious agents involve 2 separate immune systems: (1) maternal, with neutrophils entering chorioamnion via decidual venules in the membranes and the chorionic plate from the intervillous space (acute chorioamnionitis) and (2) fetal, with neutrophils entering the chorionic plate and Wharton's jelly by migrating through the walls of large chorionic and umbilical vessels (fetal and/or umbilical vasculitis). Poor fetal outcome is more often associated with fetal inflammatory response.

Grading and staging of inflammation:

Maternal inflammatory response		Fetal inflammatory response
Staging		
Stage 1	acute subchorionitis or chorionitis	chorionic vasculitis or umbilical phlebitis
Stage 2	acute chorioamnionitis: polymorphonuclear leukocytes extend into fibrous chorion and/or amnion	involvement of the umbilical vein and one or more umbilical arteries
Stage 3	necrotizing chorioamnionitis: karyorrhexis of polymorphonuclear leukocytes, amniocyte necrosis, and/or amnion basement membrane hypereosinophilia	necrotizing funisitis
Grading		
Grade 1	not severe as defined	not severe as defined
Grade 2	severe: confluent polymorphonuclear leukocytes or with subchorionic microabscesses	severe: near-confluent intramural polymorphonuclear leukocytes with attenuation of vascular smooth muscle

It is recommended that histologic acute inflammation in the placenta, extraplacental membranes, and umbilical cord be reported as: acute chorioamnionitis (or acute chorionitis) with/without fetal inflammatory response in (specify) chorionic vessels, umbilical vein, and/or umbilical artery (or arteries).

Some pathologists may prefer to use the terms maternal inflammatory response and fetal inflammatory response.

Villitis of Unknown Etiology (VUE)

VUE is a histological diagnosis used when inflammation is found in the placental villi without a clear cause, such as a viral or bacterial infection. VUE can be categorized into two main categories based on the extent of the inflammation: **Low Grade** and **High Grade**. **Low-grade VUE** involves small clusters of fewer than 10 inflamed villi; it is labeled "focal" if these clusters appear on only one slide and "multifocal" if they appear across several. In contrast, **high-grade VUE** is more extensive, requiring multiple inflamed areas across more than one slide, with at least one cluster containing 10 or more villi. This grade is further divided into "patchy" or "diffuse," with the latter meaning more than 30% of the distal villi are affected. In rare cases where only a single cluster is found, it is reported as "ungradable," though it may be noted as "possible low" or "high" grade based on its size. Accurately distinguishing these grades is clinically vital, as **high-grade VUE** is significantly linked to **fetal growth restriction (FGR)**, long-term neurodevelopmental issues, and a higher risk of the condition returning in future pregnancies.

Clinical Correlation Table

Placental Feature	Clinical Implication / Association
Low Placental Weight (<10th centile)	Marker for Placental Hypoplasia and FGR
Thin Umbilical Cord (<8mm at term)	Associated with Fetal Growth Restriction (FGR)
Thick Umbilical Cord	Associated with Maternal Diabetes and fetal hydrops
Distal Villous Hypoplasia (DVH)	Associated with MVM and early-onset FGR
Accelerated Villous Maturation	Found in Preeclampsia , Preterm Labor, and FGR
Fetal Inflammatory Response	Higher association with poor fetal outcome than MIR
High Grade Villitis (VUE)	Associated with FGR and neurodevelopmental impairment

Summary of Placental lesions:

S.no	Lesion	Findings
1.	Maternal vascular malperfusion	villus abnormality, infarctions, blood accumulation beneath and dissecting the decidua
2.	Retroplacental hemorrhage	blood accumulation beneath and dissecting the decidua and compression of the overlying intervillous space, with villous crowding, congestion, and/or intravillous hemorrhage with touching villi
3.	Distal villous hypoplasia (DVH)	paucity of villi in relation to surrounding stem villi. Villi are thin and relatively elongated, syncytial knots increased. Best observed under low power.
4.	Accelerated villous maturation	diffuse pattern of term-appearing villi (small or short hypermature villi for gestational period) with increased syncytial knots and intervillous fibrin, usually alternating with areas of villous paucity
5.	Decidual Arteriopathy	acute atherosclerosis, fibrinoid necrosis with or without foam cells, mural hypertrophy, chronic perivasculitis, absence of spiral artery +/- remodeling, arterial thrombosis, persistence of intramural endovascular trophoblast in the third trimester
6.	Fetal Vascular Malperfusion	thrombosis, segmental avascular villi, villous stromal-vascular karyorrhexis vascular intramural fibrin deposition, stem vessel obliteration/fibromuscular sclerosis, vascular ectasia
7.	Avascular Villi	total loss of villous capillaries and bland hyaline fibrosis of the villous stroma
8.	Intramural Fibrin Deposition	fibrin or fibrinoid deposition (subendothelial or intramuscular) within the wall of large fetal vessels (indicates recent), with calcification (indicates remote)
9.	Villous Stromal-Vascular Karyorrhexis	2 to 4 terminal villi showing karyorrhexis of fetal cells (nucleated erythrocytes, leukocytes, endothelial cells, and/or stromal cells) with preservation of surrounding trophoblast
10.	Stem Vessel Obliteration	Marked thickening of the vessel wall and resultant obliteration of the vascular lumen
11.	Vascular Ectasia	vessels that are four times the luminal diameter of the surrounding corresponding vessel
12.	Delayed villous maturation	a monotonous villous population with centrally placed capillaries and decreased vasculosyncytial membranes
13.	Ascending intrauterine infection	Maternal inflammatory response —acute subchorionitis or chorionitis —acute chorioamnionitis —necrotizing chorioamnionitis Fetal inflammatory response —chorionic vasculitis or umbilical phlebitis —involvement of the umbilical vein and one or more umbilical arteries —necrotizing funisitis

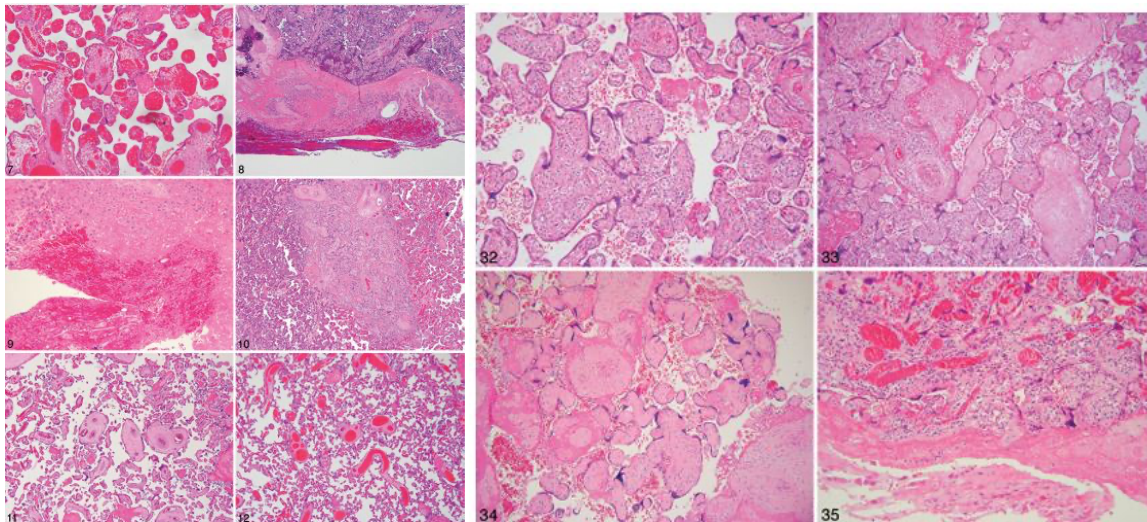


Figure 7. Intervillous hemorrhage accompanying retroplacental hemorrhage (hematoxylin-eosin, original magnification $\times 10$).
Figure 8. Dissection of the basal plate in retroplacental hemorrhage. There is congestion of the intervillous space immediately above the hemorrhage (hematoxylin-eosin, original magnification $\times 2$).
Figure 9. Neutrophils as a vital reaction to the retroplacental hemorrhage (hematoxylin-eosin, original magnification $\times 40$).
Figure 10. Early infarction showing crowding and compression of the intervillous space in the center of the infarct and congestion of villi around its periphery (hematoxylin-eosin, original magnification $\times 2$).
Figure 11. Distal villous hypoplasia: there is a paucity of villi, many of which are thin and elongated (hematoxylin-eosin, original magnification $\times 4$).
Figure 12. Avascular villous maturation: there is a combination of areas with increased cytotrophoblastic and intervillous fibrin deposition (hematoxylin-eosin, original magnification $\times 4$).
Figure 32. High-grade villitis of unknown etiology: this focus affects more than 10 contiguous villi (hematoxylin-eosin, original magnification $\times 10$).
Figure 33. Occasional avascular villi with scattered inflammatory cells suggesting "burnt-out villitis" (hematoxylin-eosin, original magnification $\times 10$).
Figure 34. Contiguous, uniformly hyalinized, avascular villi suggesting upstream vascular occlusion (hematoxylin-eosin, original magnification $\times 4$).
Figure 35. Bandlike parasitiform villitis (hematoxylin-eosin, original magnification $\times 10$).

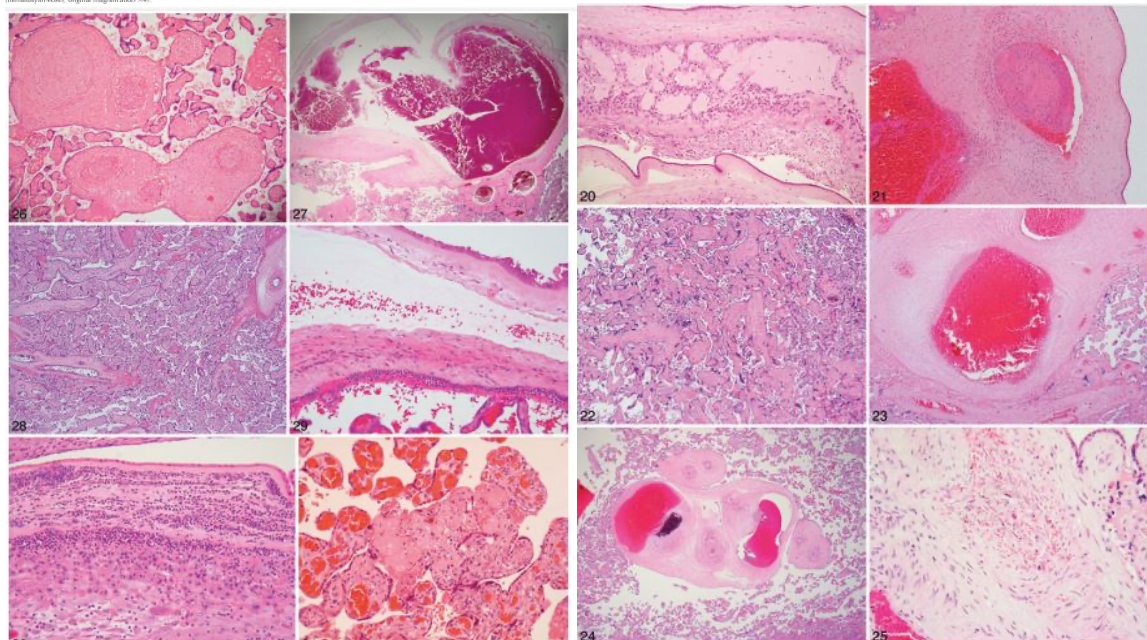


Figure 20. Contiguous pseudo-cysts in the chorionic laeve (hematoxylin-eosin, original magnification $\times 10$).
Figure 21. Thrombosis of a fetal artery (overlying a vein) in the chorionic plate (hematoxylin-eosin, original magnification $\times 10$).
Figure 22. A large focus of avascular villi, to the right of which vascularized villi are seen (hematoxylin-eosin, original magnification $\times 4$).
Figure 23. Intramural fibrin deposition in a large main stem vessel (hematoxylin-eosin, original magnification $\times 4$).
Figure 24. Intramural fibrin deposition and calcification, indicating remoteness of lesion, within a wall of the large fetal vessel (hematoxylin-eosin, original magnification $\times 2$).
Figure 25. Villous stromal vascular karyorrhexis: karyorrhexis of fetal cells with preservation of surrounding trophoblast (hematoxylin-eosin, original magnification $\times 40$).

Figure 26. Stem vessel obliteration: there is marked thickening of the vessel wall with resultant obliteration of the vascular lumen (hematoxylin-eosin, original magnification $\times 10$).
Figure 27. Vascular ectasia: the luminal diameter of the chorionic vessel is more than four times that of the adjacent vessels (hematoxylin-eosin, original magnification $\times 2$).
Figure 28. Term placenta showing delayed villous maturation: there is a monotonous villous population of villi with centrally placed capillaries and decreased vasculosyncytial membranes (hematoxylin-eosin, original magnification $\times 4$).
Figure 29. Neutrophils in the subchorionic intervillous space, in situ indicating subchorionitis (hematoxylin-eosin, original magnification $\times 10$).

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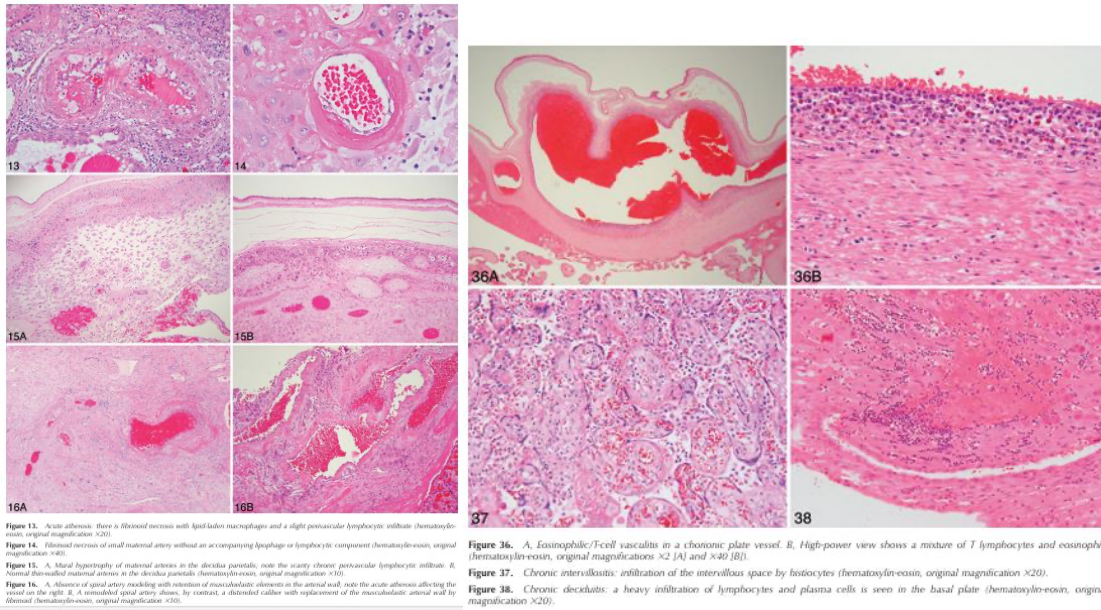


Figure 13. Acute atherosclerosis: there is fibrotic necrosis with lipid-laden macrophages and a slight postvascular lymphocytic infiltrate (hematoxylin-eosin, original magnification $\times 200$).
Figure 14. Infarcted necrosis of small maternal artery without an accompanying lymphocytic component (hematoxylin-eosin, original magnification $\times 40$).
Figure 15. A. Mild hypertrophy of maternal arteries in the decidua parietalis, note the scanty chronic postvascular lymphocytic infiltrate. B. Normal decidualized maternal arteries in the decidua parietalis (hematoxylin-eosin, original magnification $\times 100$).
Figure 16. A. Absence of spiral artery remodeling with retention of muscularis elements in the arterial wall, note the acute atherosclerosis affecting the vessel on the right. B. A intussuscepted spiral artery shows, by contrast, a distended lumen with replacement of the muscularis arterial wall by fibrotic (hematoxylin-eosin, original magnification $\times 300$).

Figure 36. A. Eosinophilic/T-cell vasculitis in a chronic plate vessel. B. High-power view shows a mixture of T lymphocytes and eosinophils (hematoxylin-eosin, original magnifications $\times 2$ [A] and $\times 40$ [B]).
Figure 37. Chronic intervillitis: infiltration of the intervillous space by histiocytes (hematoxylin-eosin, original magnification $\times 200$).
Figure 38. Chronic decidualitis: a heavy infiltration of lymphocytes and plasma cells is seen in the basal plate (hematoxylin-eosin, original magnification $\times 200$).

References and suggested further reading:

1. Arch Pathol Lab Med. 2016;140:698–713; doi: 10.5858/ arpa.2015-0225-CC
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