FINDINGS THAT MATTER IN PLACENTAL PATHOLOGY

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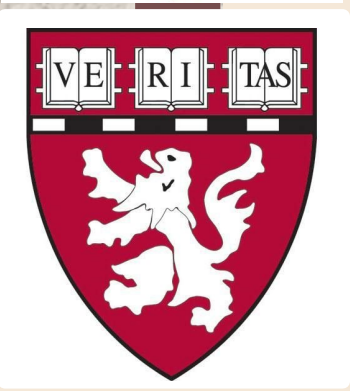


CONFLICT OF INTEREST STATEMENT

- I have no conflicts of interests or financial relations to disclose

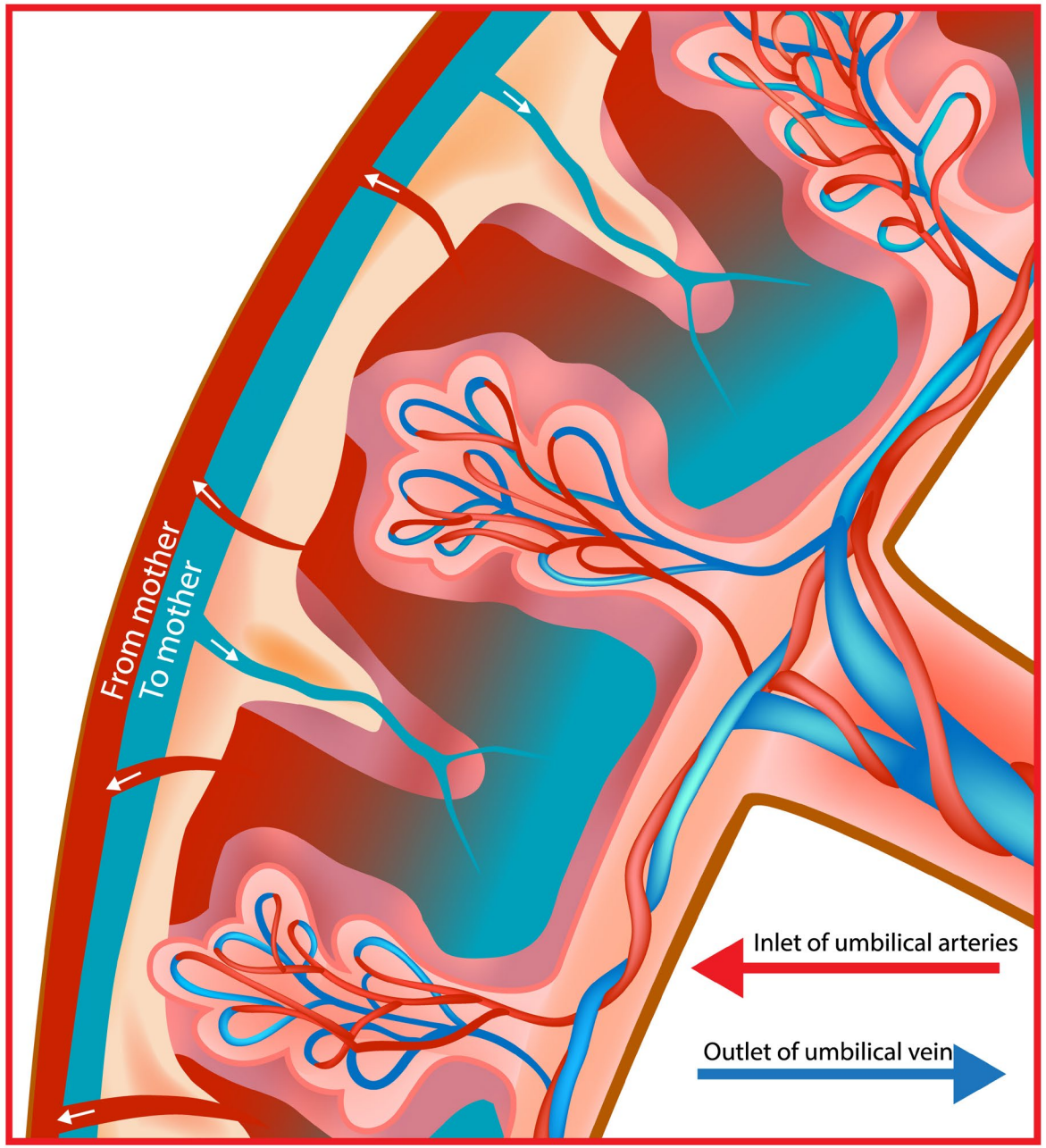
PLACENTAL LESIONS – OUTLINE

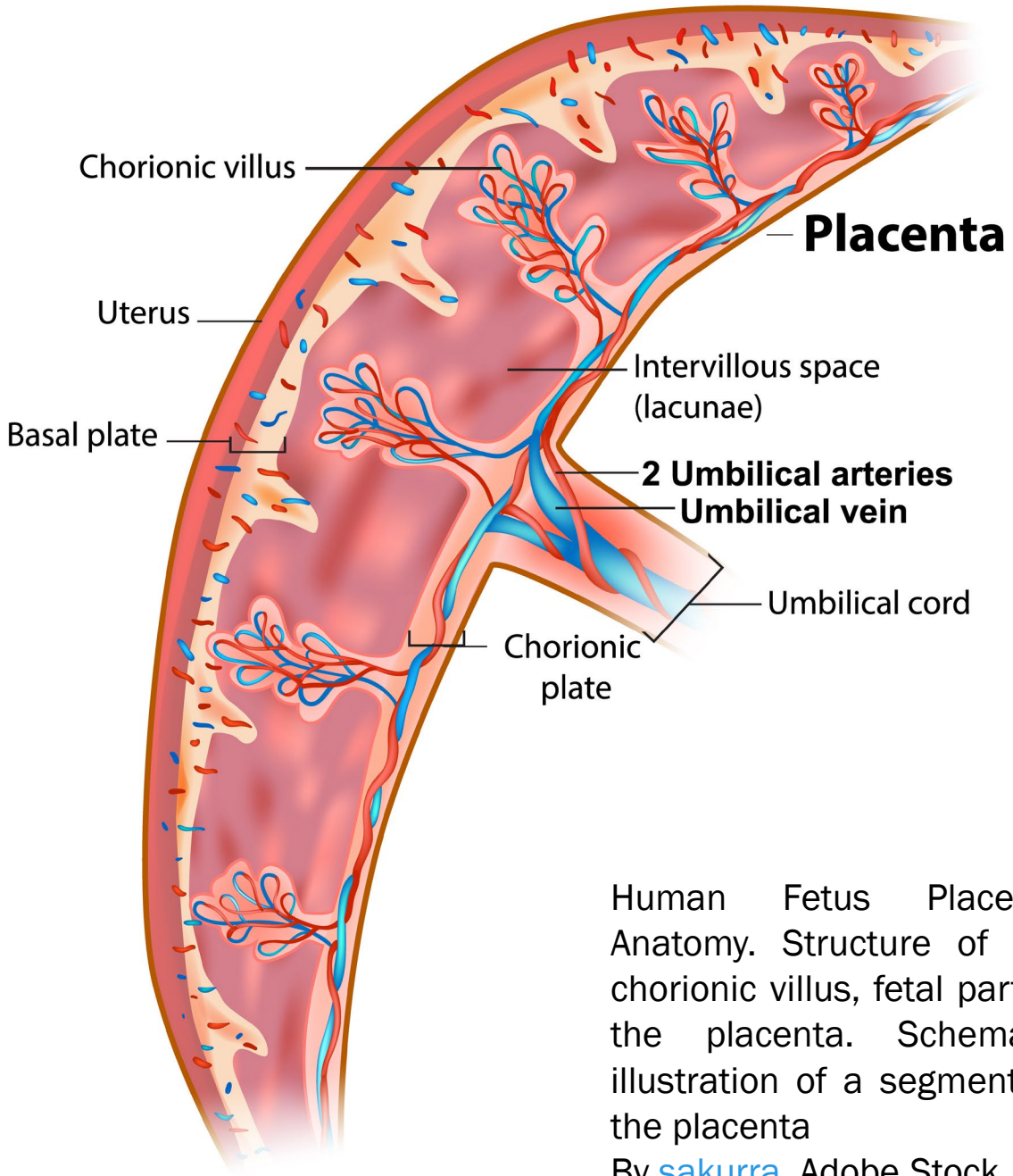
- NON-INFLAMMATORY
  1. *Maternal vascular malperfusion*
  2. *Fetal vascular malperfusion*
  3. *Placenta accreta spectrum*
  4. *Basal plate myometrial fibers*
  5. *Massive fibrin deposition*

- INFLAMMATORY
  6. *Histiocytic intervillositis*
  7. *Villitis of unknown etiology*
  8. *Ascending infection*

WHY DO THEY MATTER?

A. Explain adverse maternal and/or fetal outcomes
B. Imply recurrent materno-fetal morbidity
C. Require immediate intervention





Human Fetus Placenta Anatomy. Structure of the chorionic villus, fetal part of the placenta. Schematic illustration of a segment of the placenta

By sakurra Adobe Stock

PLACENTAL LESIONS A

- Placental lesions that explain an adverse maternal and / or fetal outcome
  1. *Maternal vascular malperfusion*
  2. *Fetal vascular malperfusion*
  3. *Placenta accreta spectrum*

1. MATERNAL VASCULAR MALPERFUSION (MVM)

- MVM replaces former terms
  - *Uteroplacental under-perfusion*
  - *Placental insufficiency*
- Constellation of findings seen in the setting of abnormal blood supply to the feto-placental unit
- Impaired spiral artery remodeling
  - *Insufficient circulation with acute /subacute / chronic ischemia*
  - *Turbulent, high-momentum blood ejection with villous damage*

Arch Pathol Lab Med 2016;140(7):698-713
Surg Pathol Clin 2022;15(2):175-196

1. MATERNAL VASCULAR MALPERFUSION (MVM)

- Associated with hypertensive disorders
  - *Chronic hypertension*
  - *Pregnancy-induced hypertension (PIH)*
    - Eclampsia, preeclampsia, HELLP syndrome
- Marker of cardiovascular and metabolic risk
  - *Subsequent high-risk cardiometabolic profile*
  - *High cholesterol, high blood pressure*

Placenta 2017;52:106–13
Am J Obstet Gynecol 2021;225:660.e1–12
Virchows Arch 2018;472(3):415–23
BJOG 2018;125(8):1009–17

1. MATERNAL VASCULAR MALPERFUSION (MVM)

- Increased risk of fetal and neonatal death
- More frequent in the late 2nd and 3rd trimesters
  - *14% at 37-42 weeks* *17% at 32-37 weeks*
  - *22% at 23-32 weeks* *4% below 23 weeks*

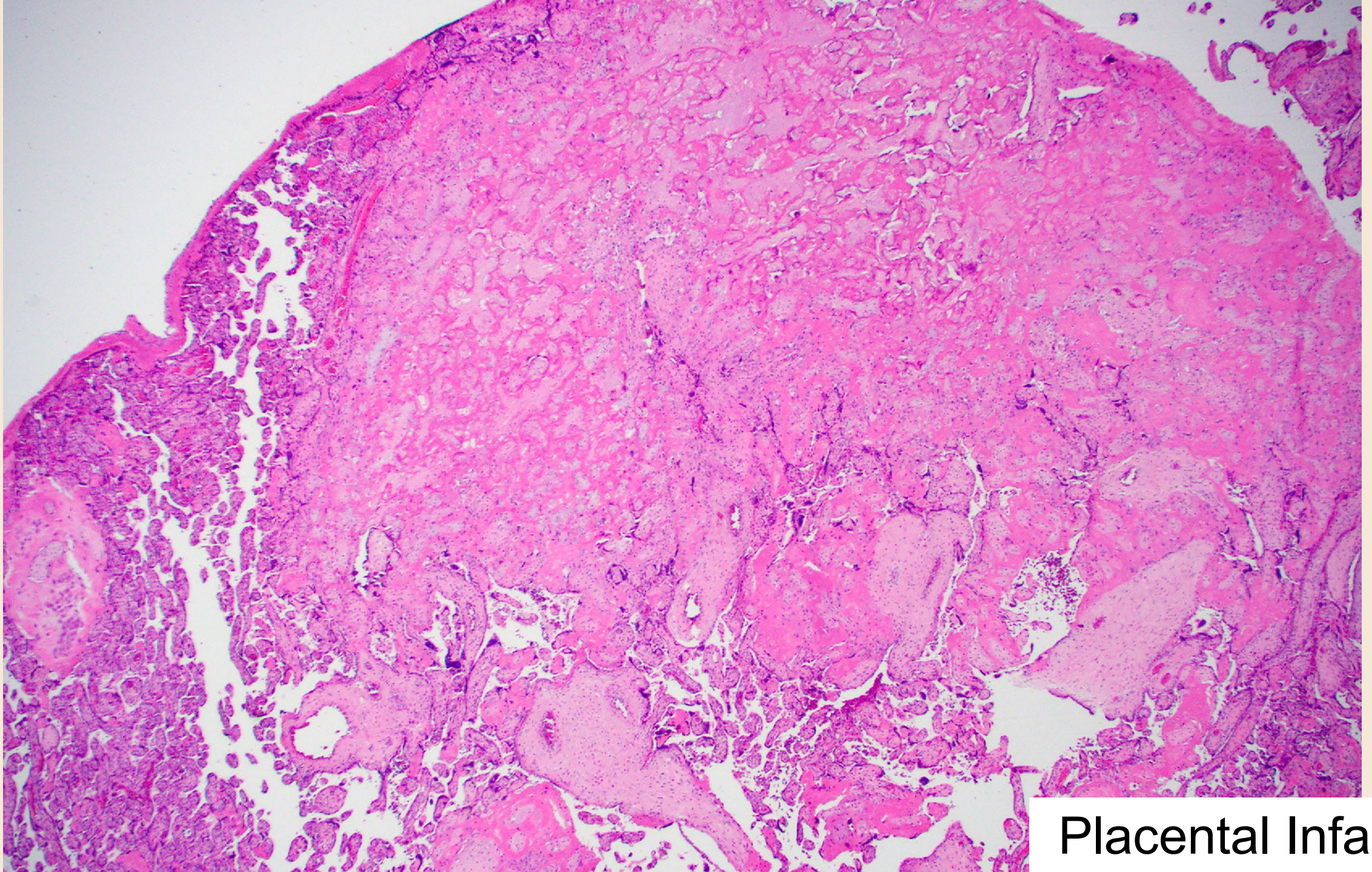
PREMATURITY

- Early onset (<34 weeks) >>>> Late onset (≥34 weeks) PIH
  - *Acute arteriolar atherosis*
  - *Distal villous hypoplasia*
  - *Accelerated villous maturation*

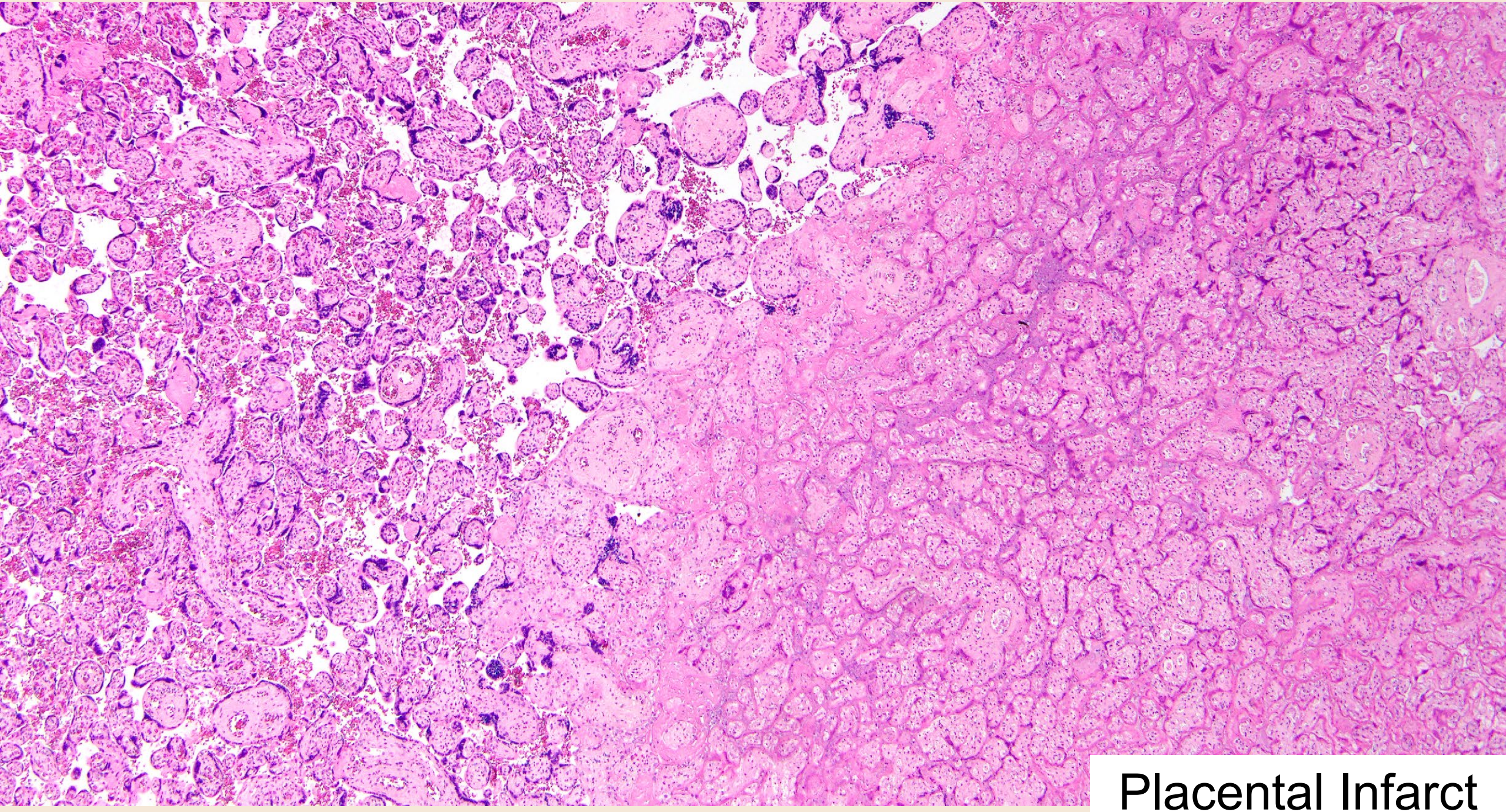
Am J Obstet Gynecol 2021;225:660.e1–12
J Perinat Med 2021;49(4):412–30
Mod Pathol 2021;34(6):1074–92
Virchows Arch 2018;472(4):635–42

1. MATERNAL VASCULAR MALPERFUSION (MVM)

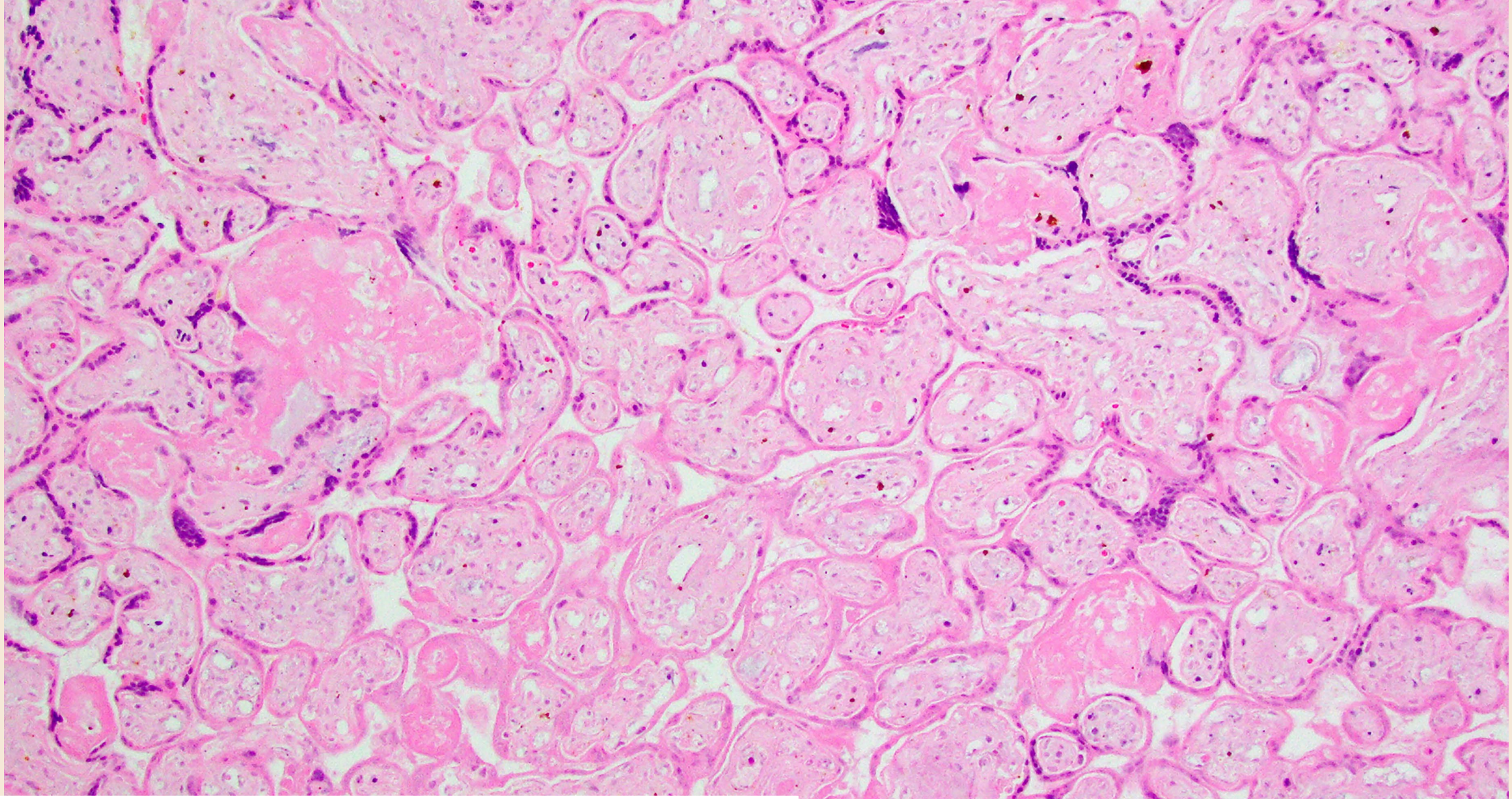
- Macroscopic features
  - *Low placental weight (<10th percentile for gestational age)*
  - *Thin umbilical cord (<10th percentile or <8 mm after term)*
  - *Placental infarcts*
  - *Retroplacental hemorrhage*
- Microscopic features
  - *Accelerated villous maturation*
  - *Distal villous hypoplasia*
  - *Decidual arteriopathy*
  - *Excess extravillous trophoblast*



Placental Infarct



Placental Infarct

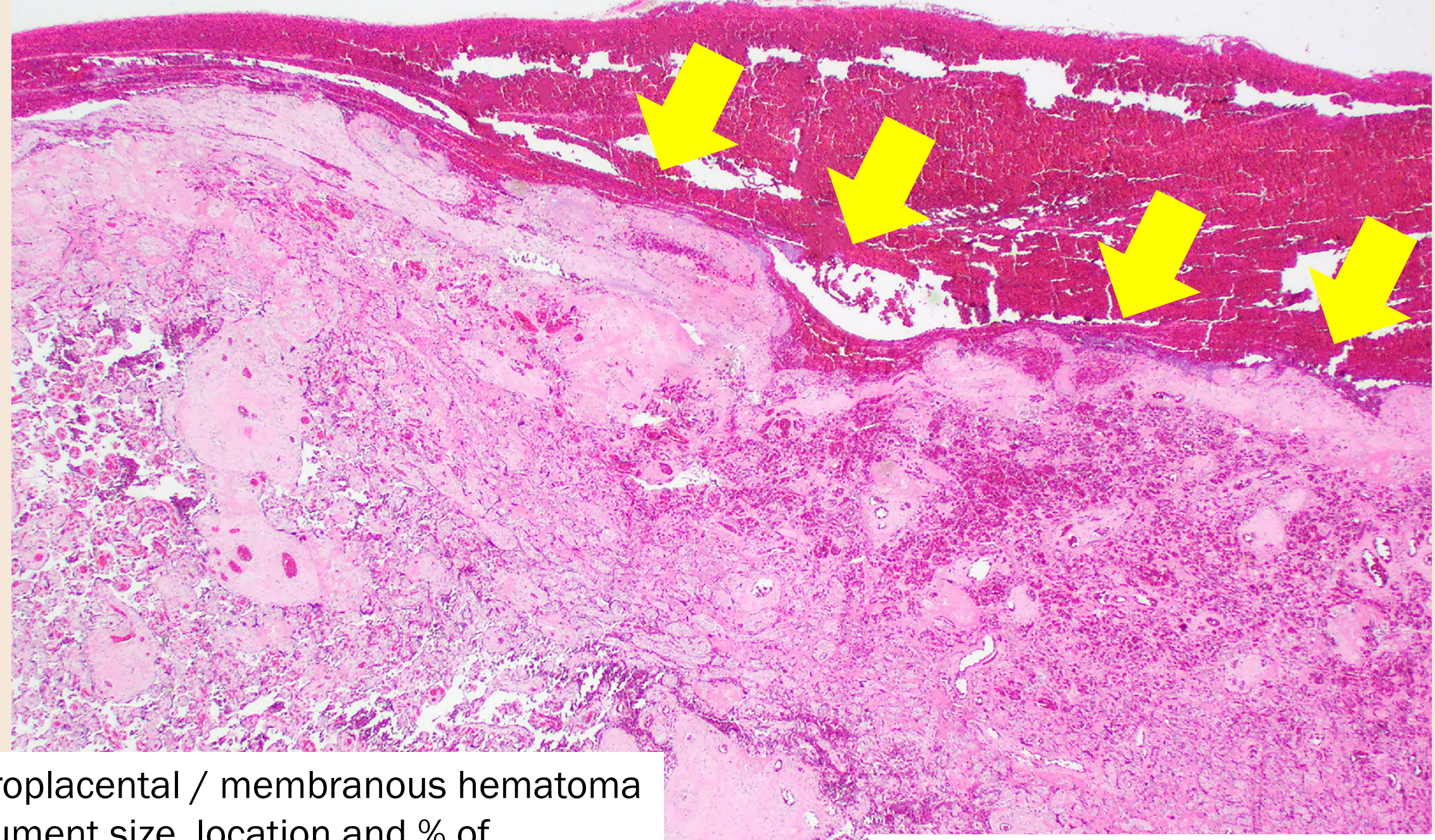


Significant infarcts

Before term: Any infarct

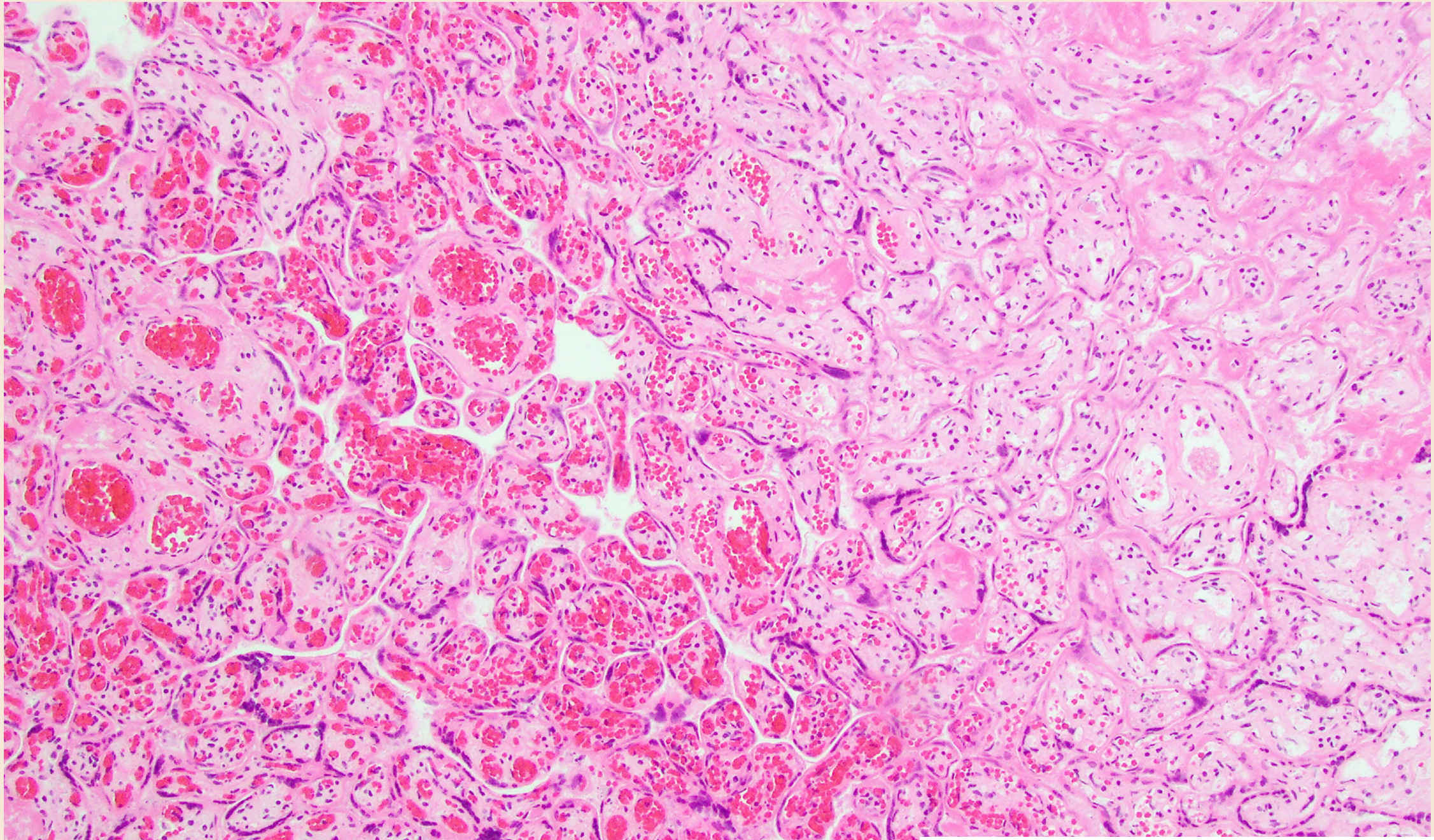
After term: Non-marginal, >5% of parenchyma

Placental Infarct

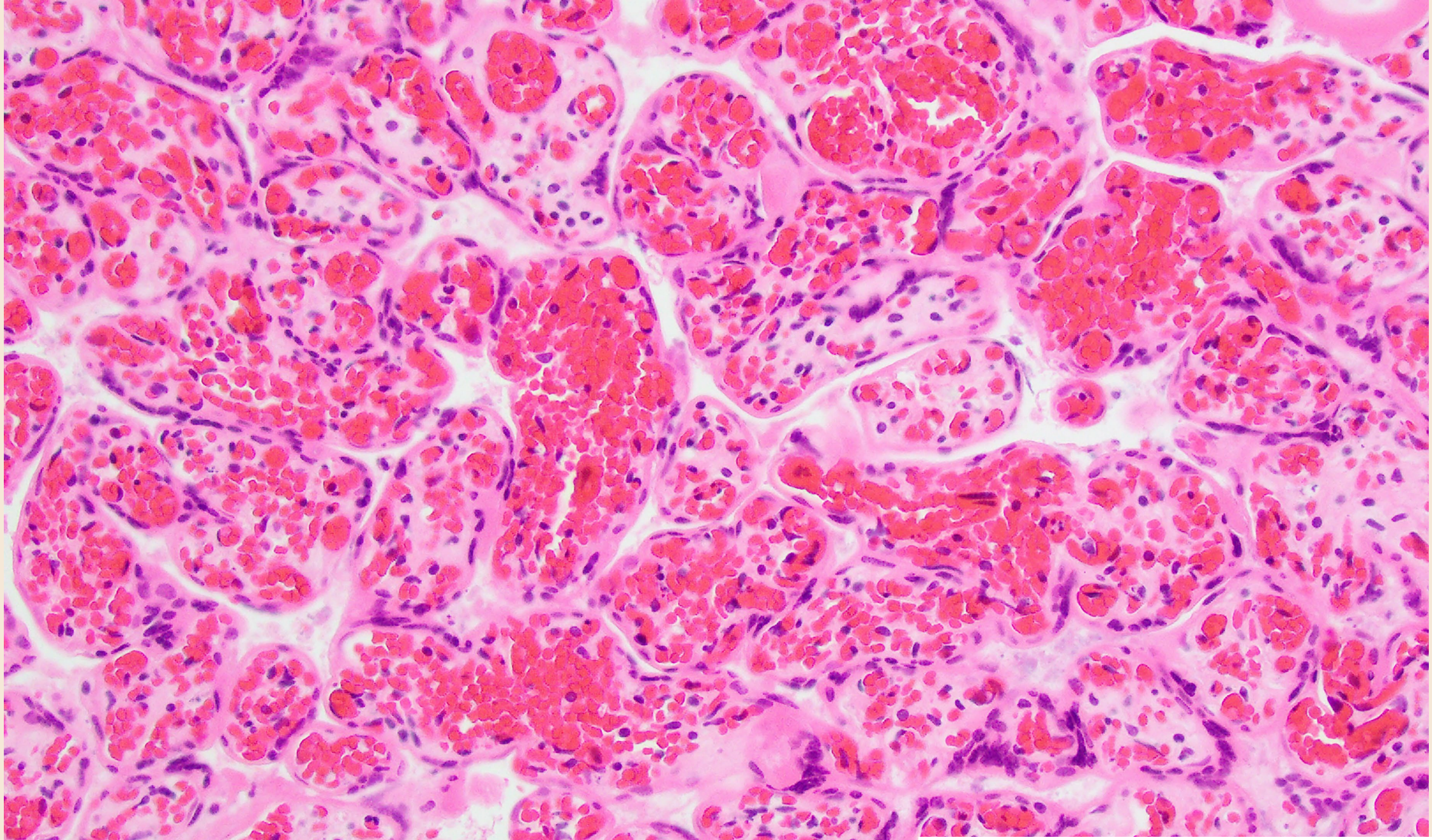


Retroplacental / membranous hematoma
Document size, location and % of parenchyma covered by it

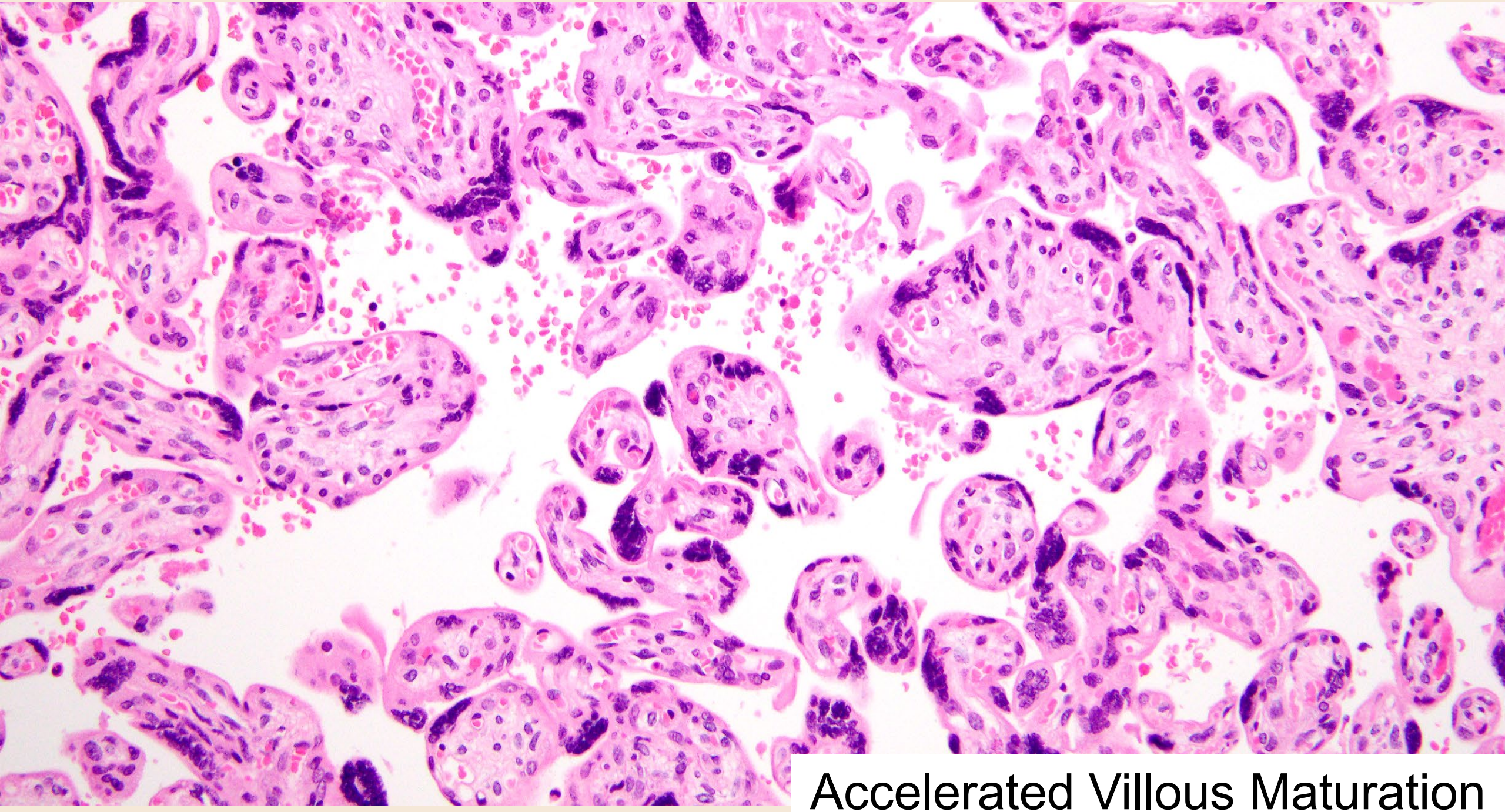
Retroplacental hematoma



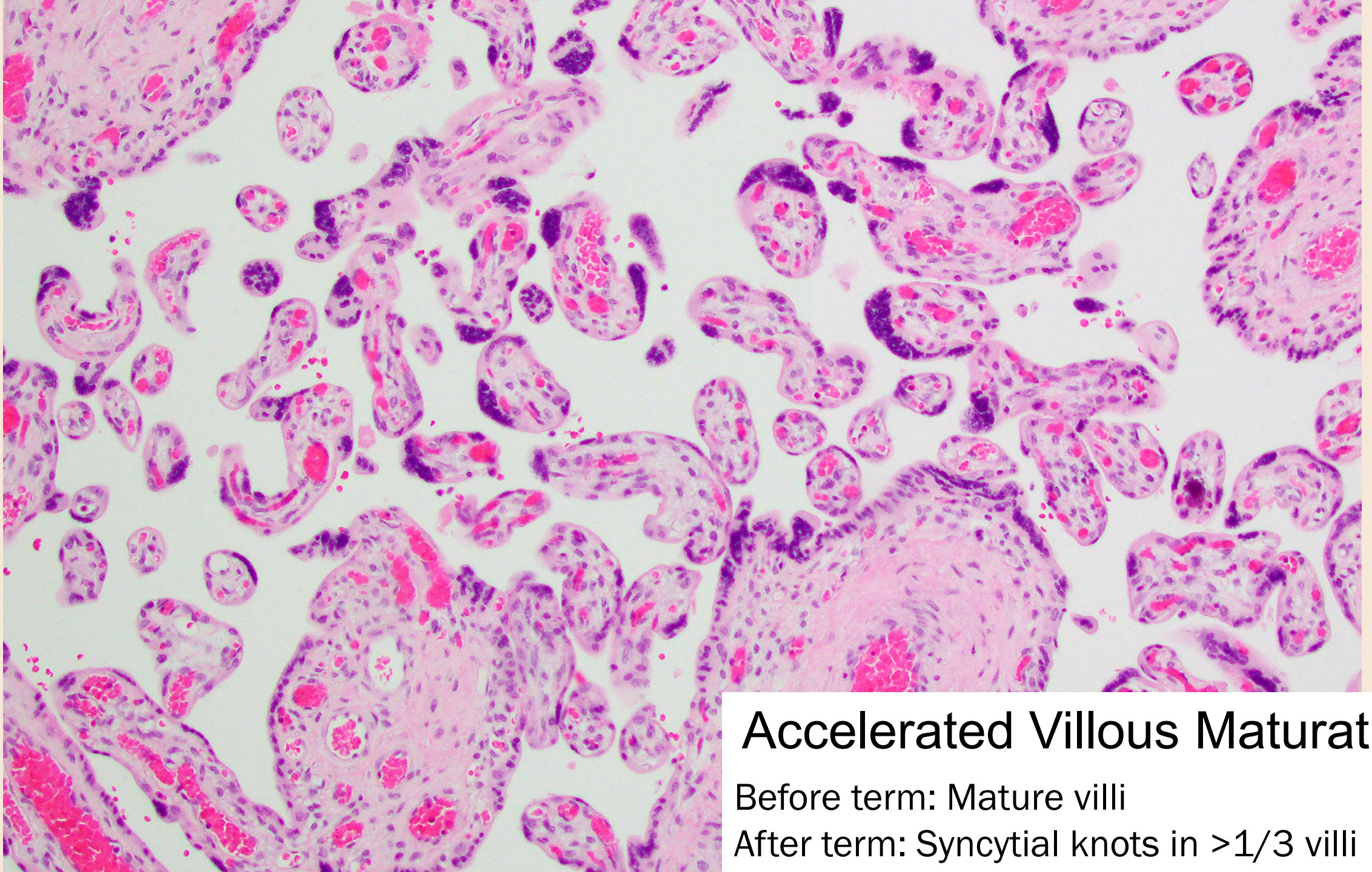
Intravillous hemorrhage



Intravillous hemorrhage



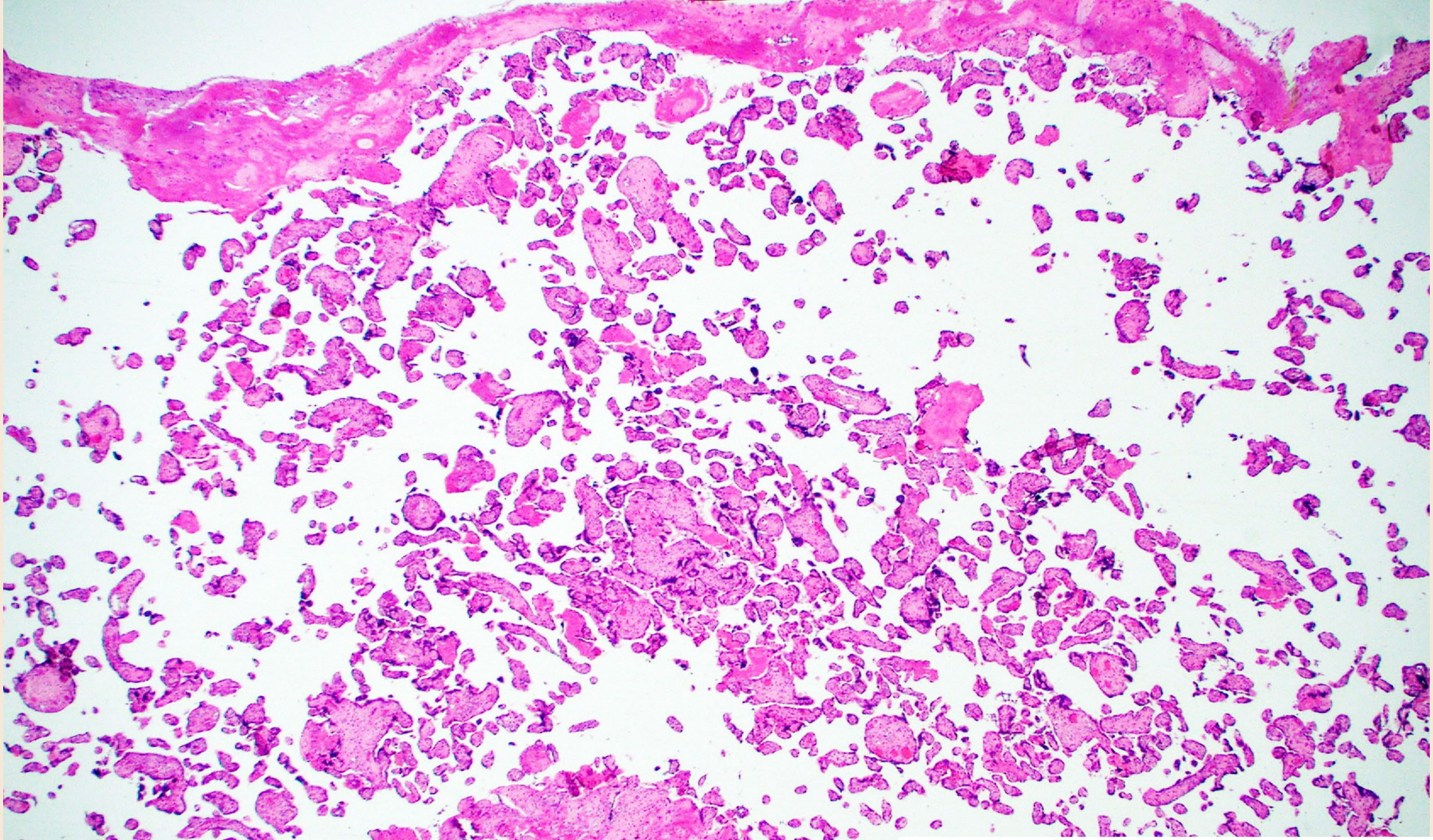
Accelerated Villous Maturation



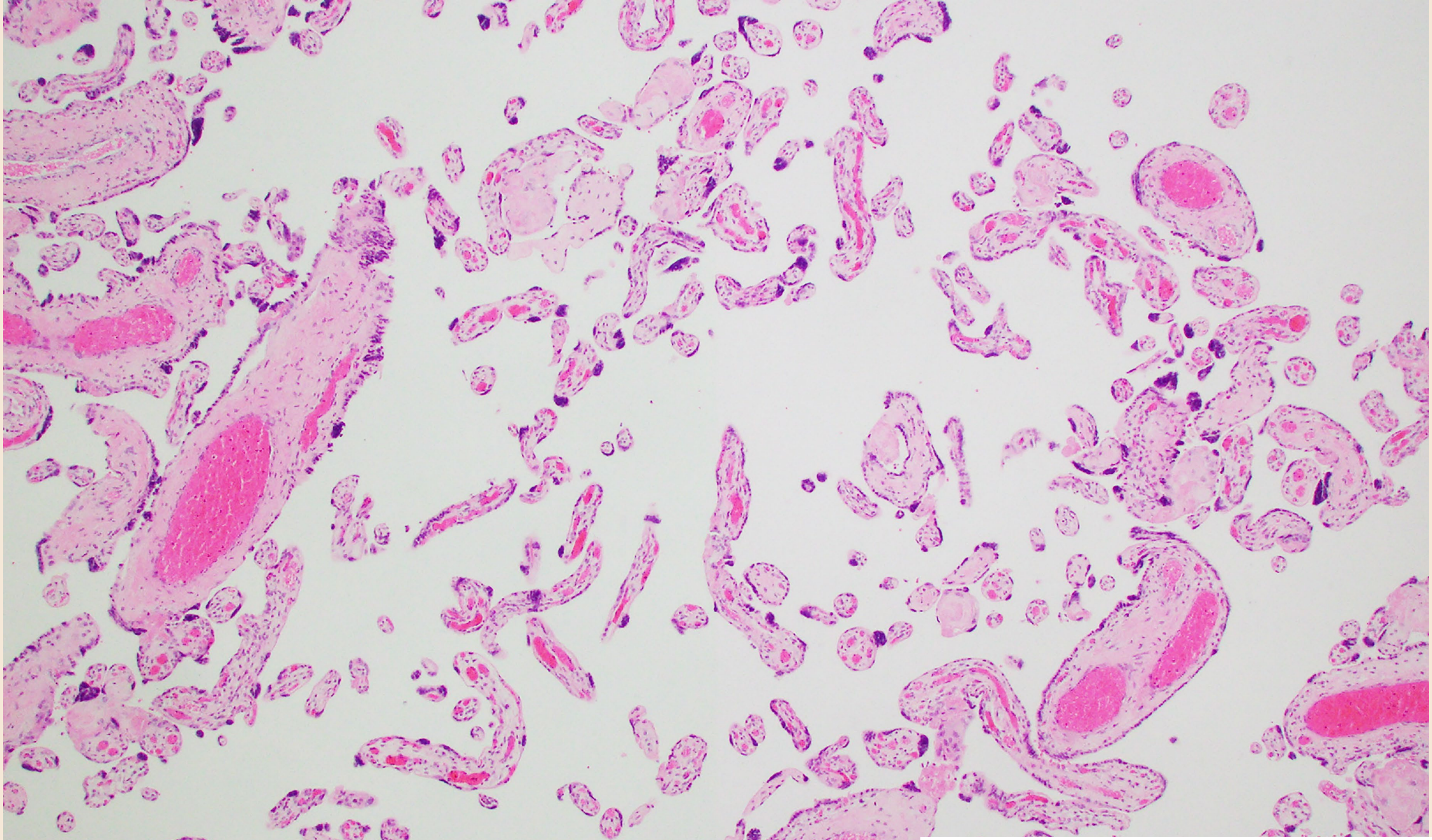
Accelerated Villous Maturation

Before term: Mature villi

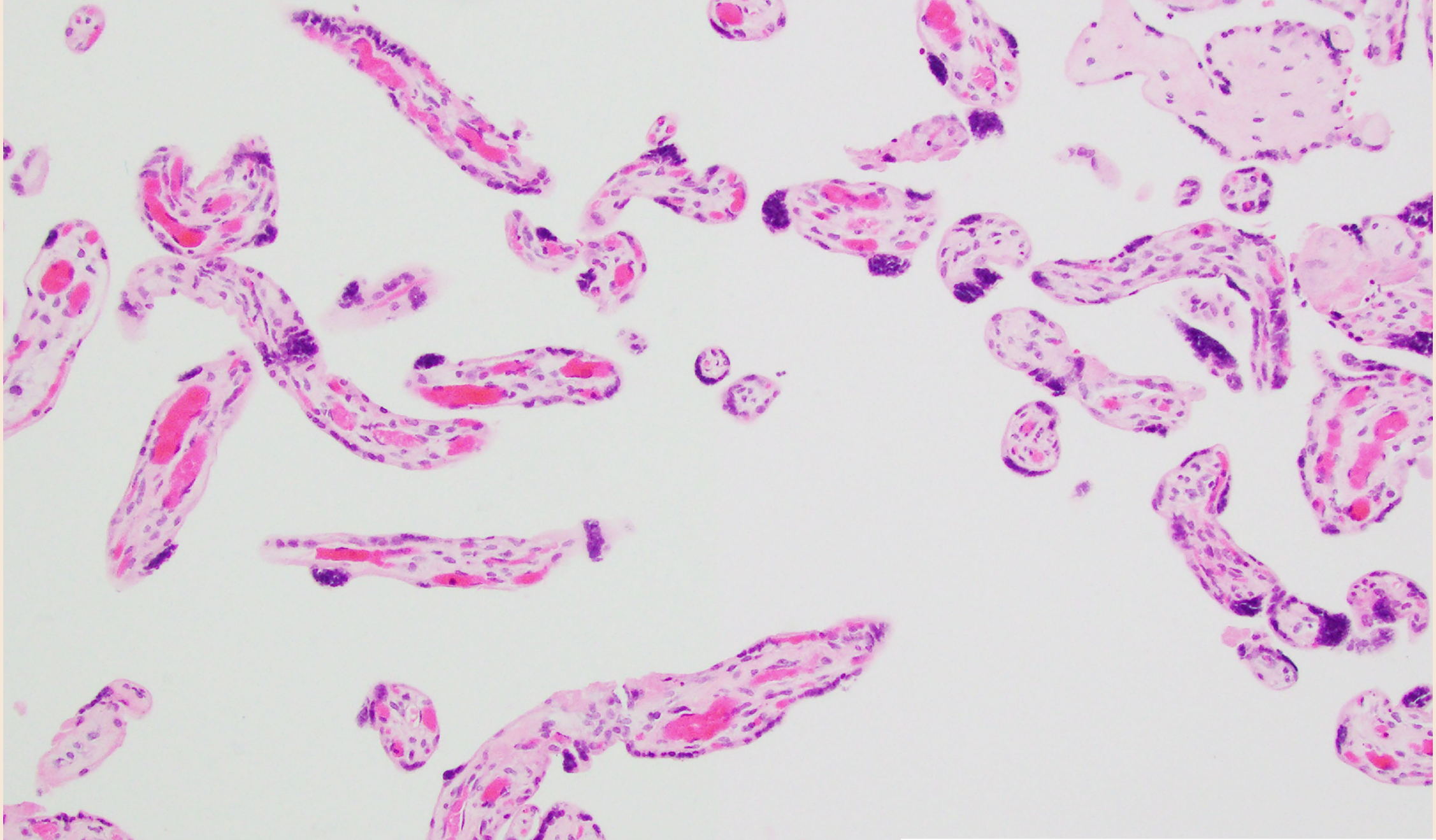
After term: Syncytial knots in >1/3 villi



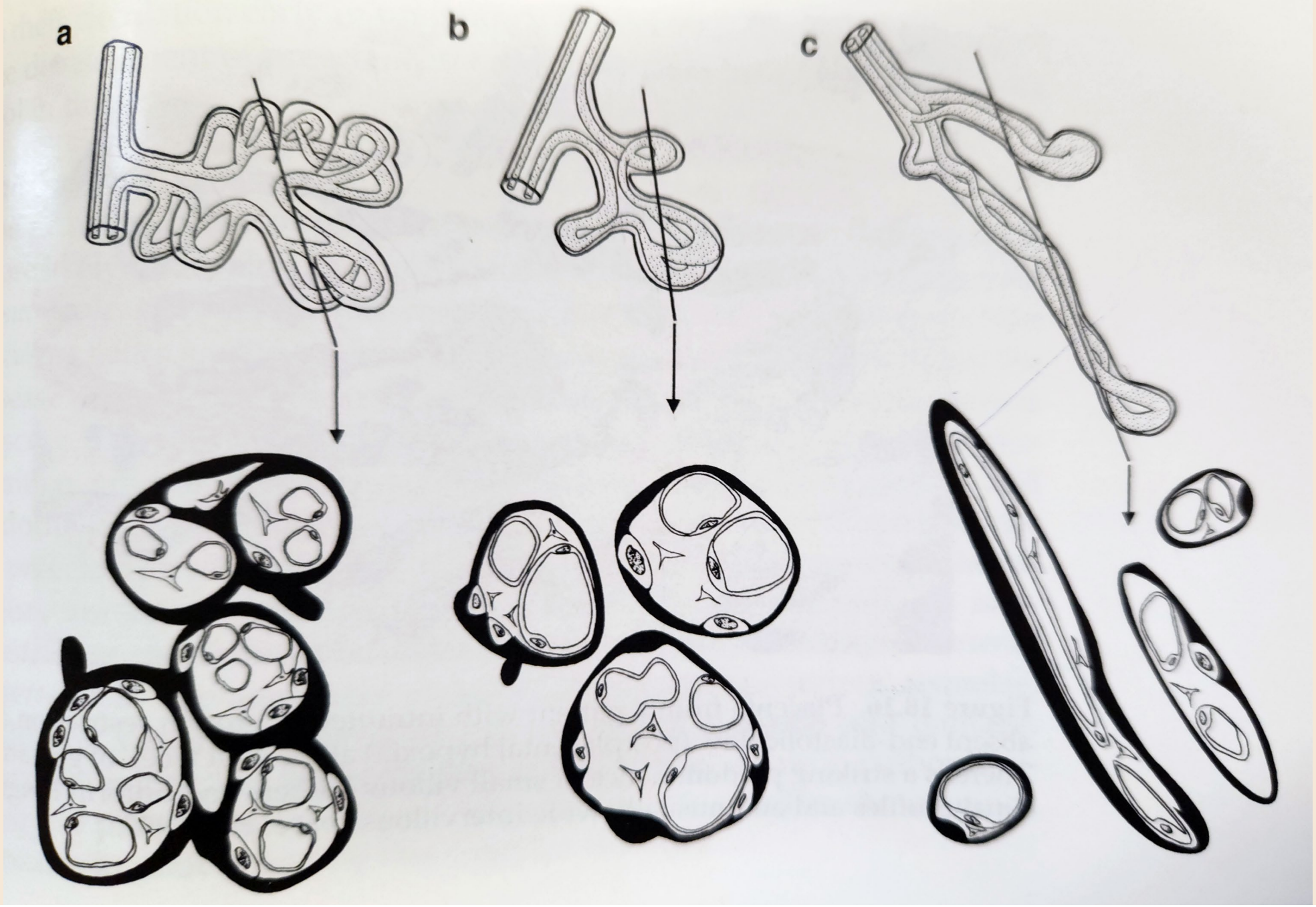
Distal Villous Hypoplasia



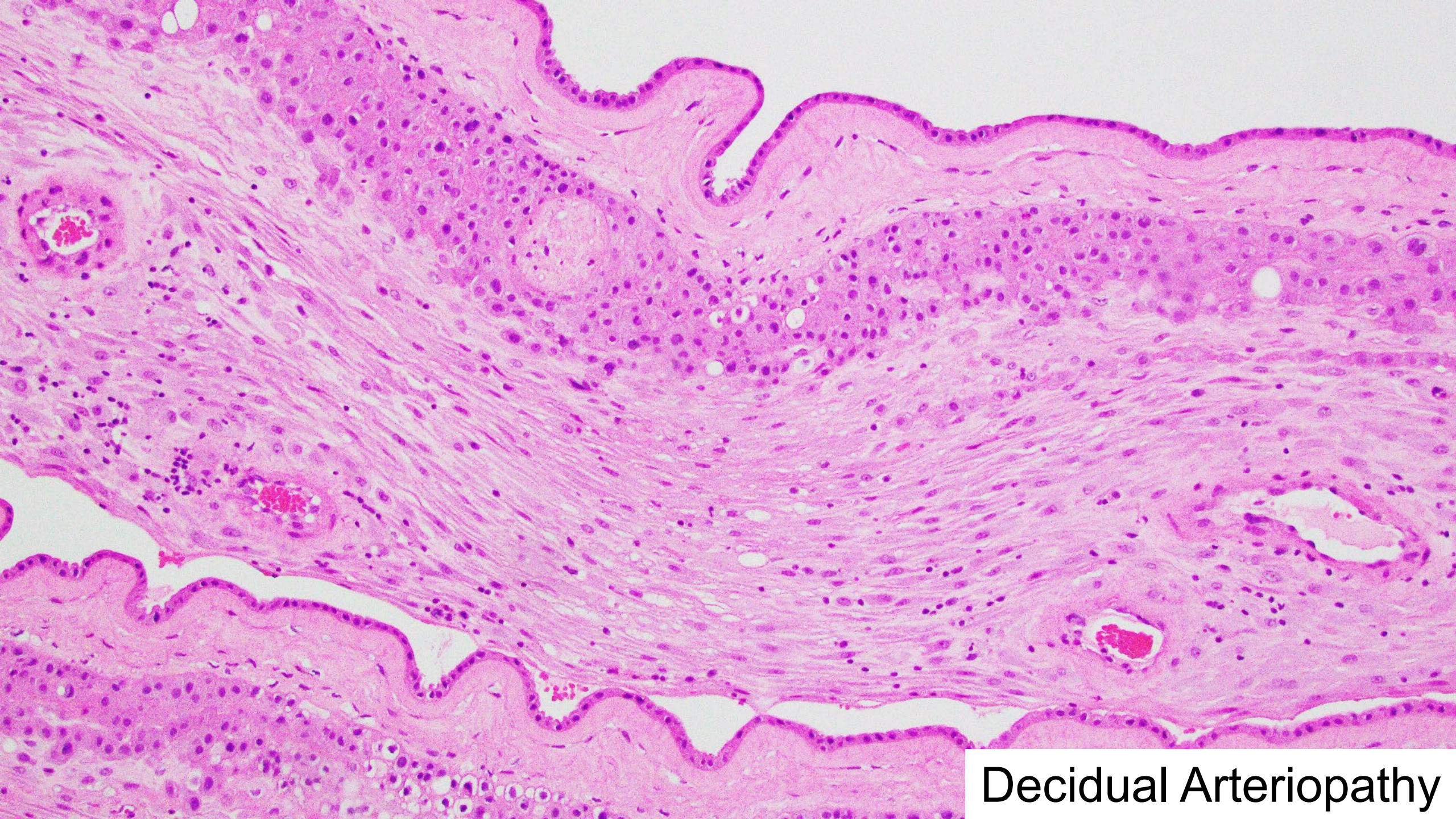
Distal Villous Hypoplasia

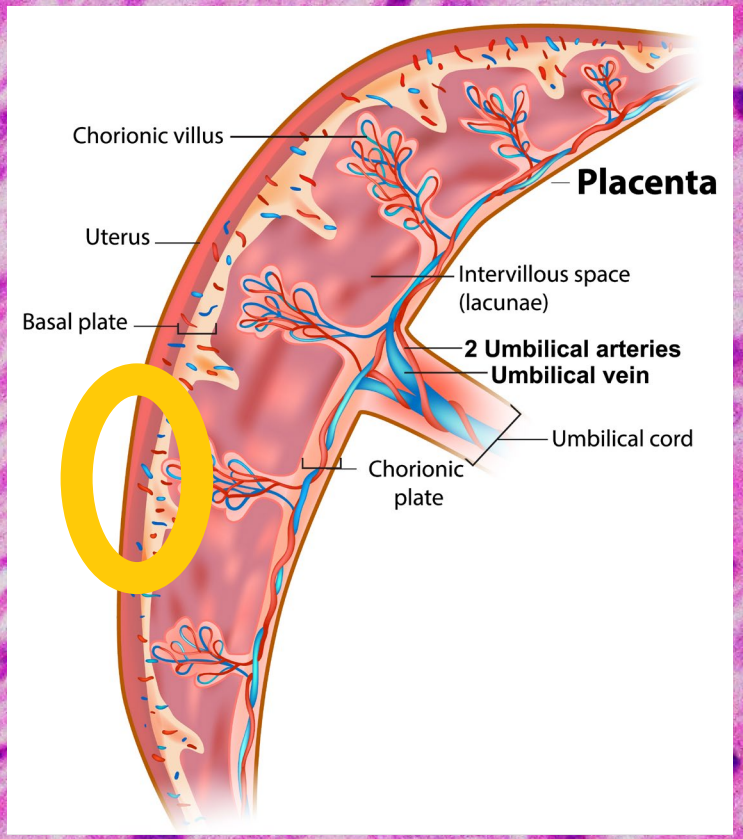


Distal Villous Hypoplasia

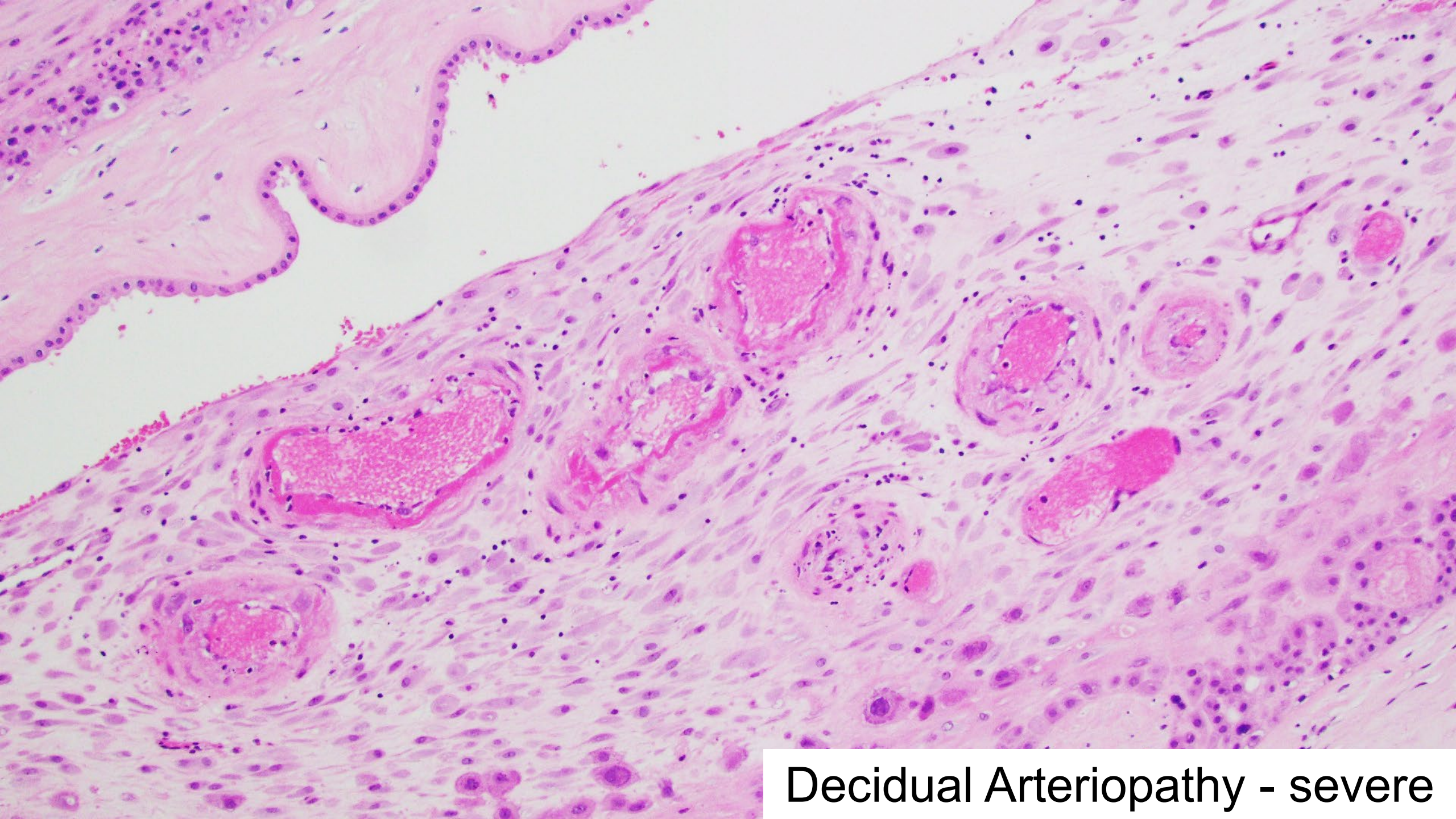


From Baergen's Manual of Pathology of the Human Placenta. Springer, 2nd ed. 2011. p.347

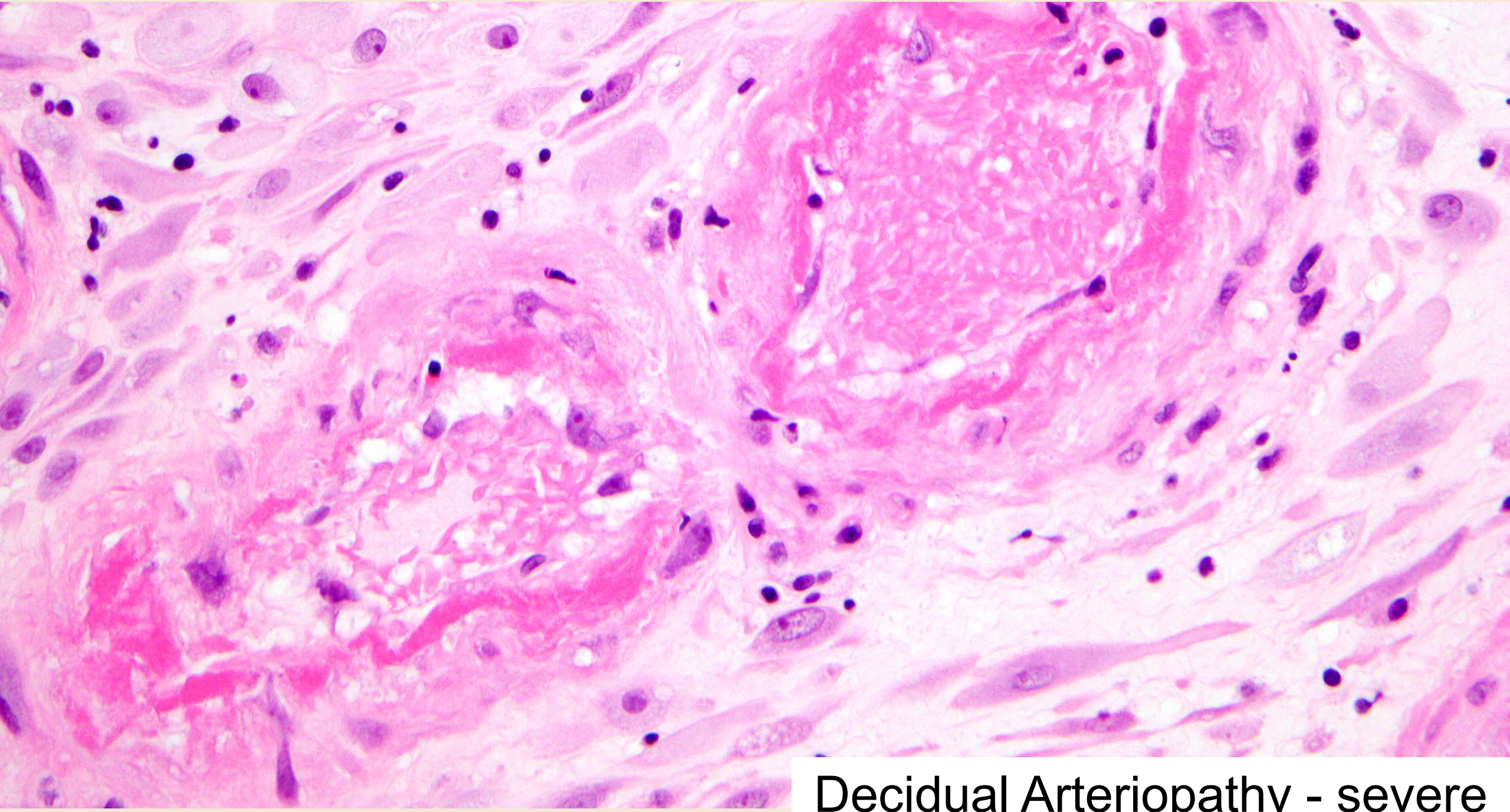

Decidual Arteriopathy



Decidual Arteriopathy



Decidual Arteriopathy - severe



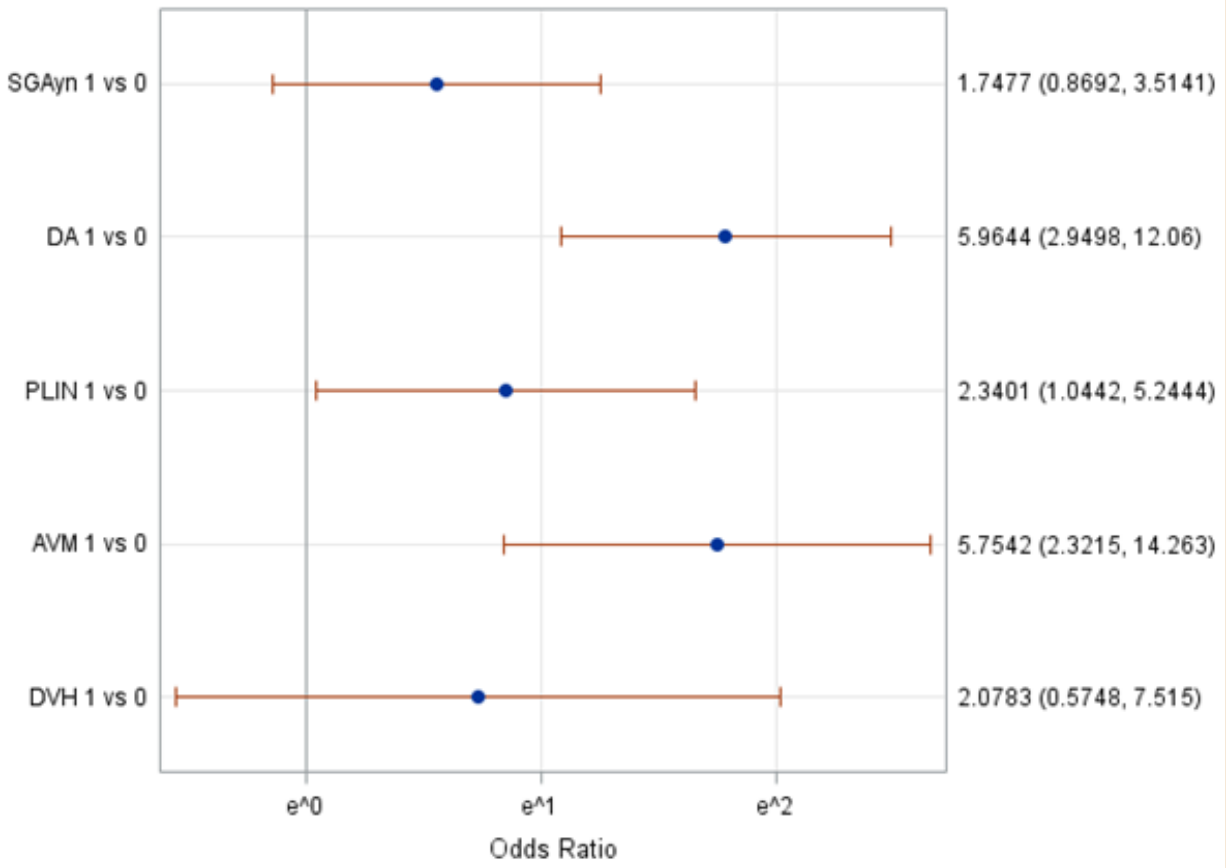
Decidual Arteriopathy - severe

MVM - ISSUES

- Poor interobserver reproducibility
  - *Accelerated villous maturation (Kappa 0.16, among experts 0.18)*
  - *Distal villous hypoplasia (Kappa 0.59)*
- When do I make the diagnosis of MVM?
  - *With any feature?*
  - *Or a combination of features?*

Arch Pathol Lab Med 2022;146(3):372-378
Pediatr Dev Pathol 2016;19(1):31–6

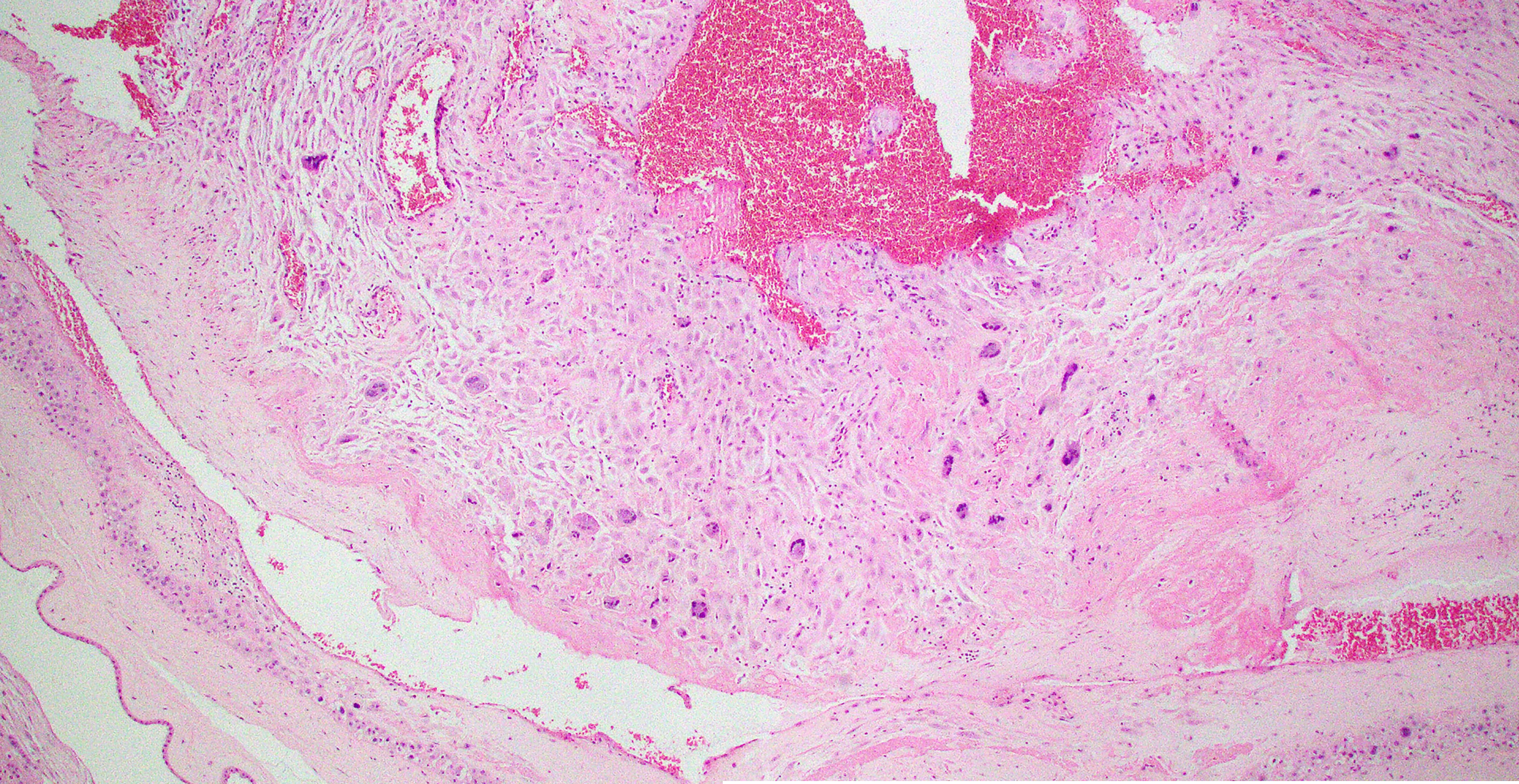
SGA = Placenta small (<10th percentile) for gestational age
PLIN = Placental infarct DA = Decidual arteriopathy
AVM = Accelerated villous maturation
DVH = Distal villous hypoplasia

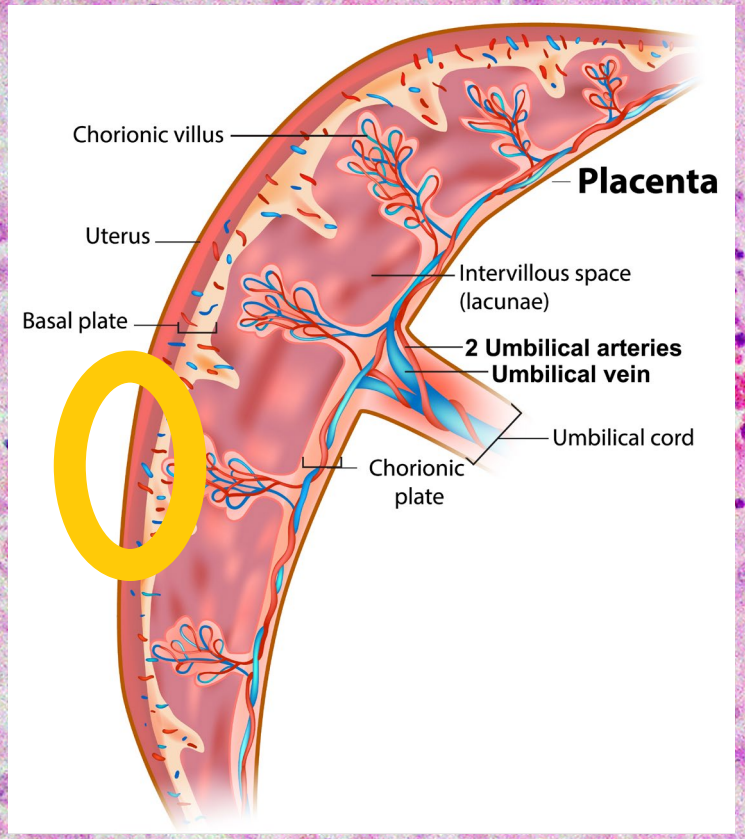


Pathologic feature(s)	*Log(odds)**
SGA only	0.23
PLIN only	0.30
PLIN + SGA	0.42
DA only	0.51
AVM only	0.52
DA + SGA	**0.65**
AVM + SGA	**0.65**
DA + PLIN	**0.72**
AVM + PLIN	**0.73**
DA + SGA + PLIN	**0.82**
AVM + SGA + PLIN	**0.82**
DA + AVM	**0.87**
DA + AVM + SGA	**0.92**
DA + AVM + PLIN	**0.94**
DA + AVM + SGA + PLIN	**0.97**

Lab Invest 2023;103(suppl 1):880-881 abstract #883

* For adverse maternal (HTN, PET, DM) or fetal (IUGR, IUFD) outcome(s)

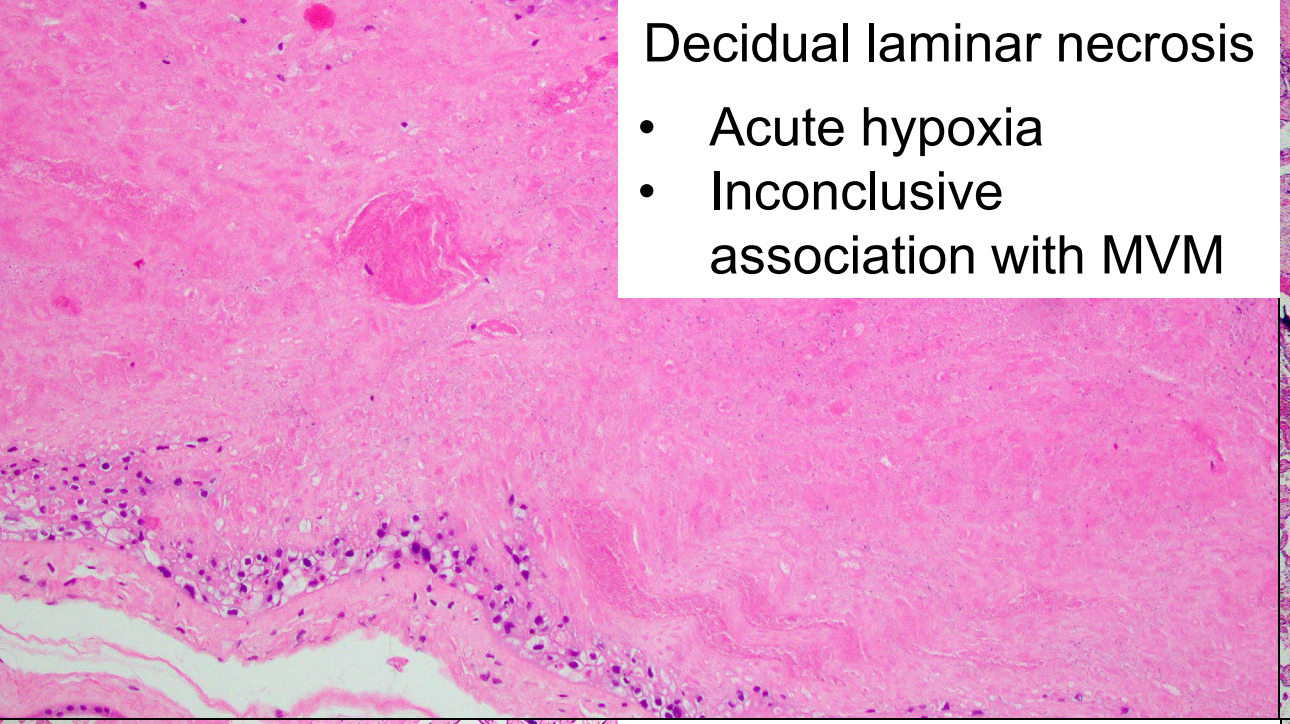

Excess multinucleated trophoblast



Excess multinucleated trophoblast

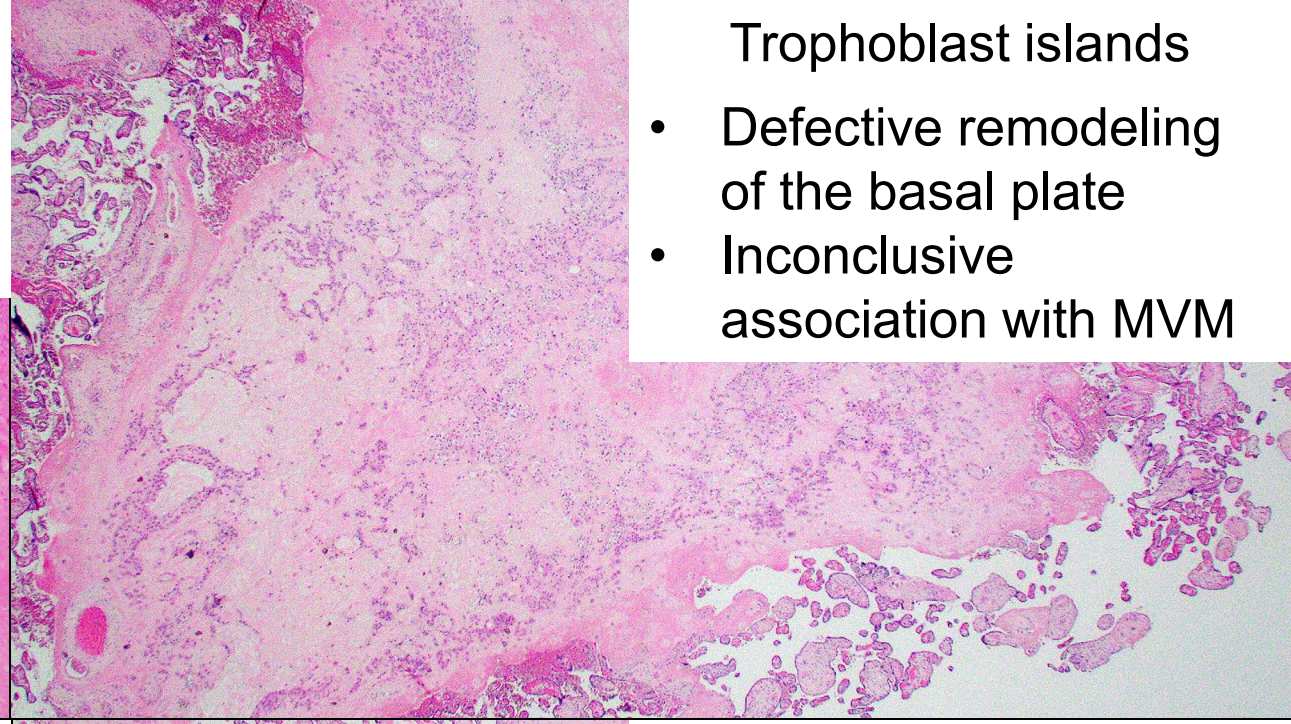
Decidual laminar necrosis

- Acute hypoxia
- Inconclusive association with MVM



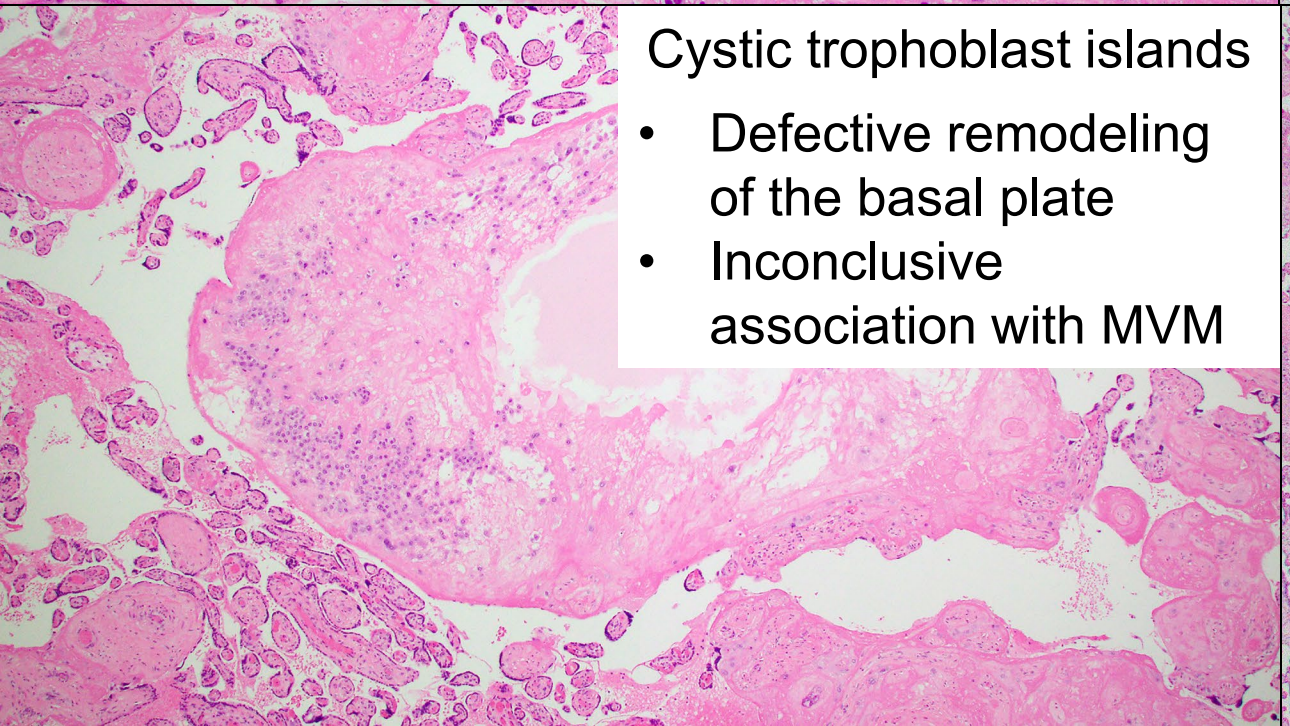
Trophoblast islands

- Defective remodeling of the basal plate
- Inconclusive association with MVM



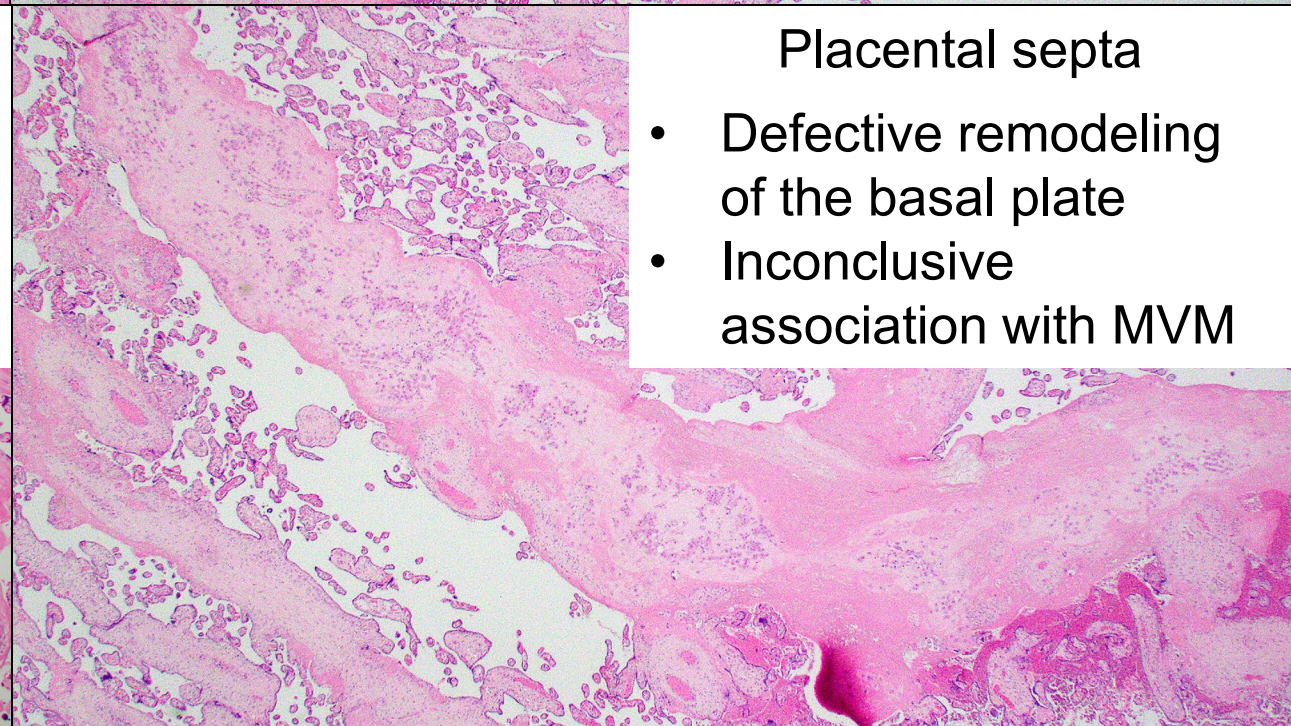
Cystic trophoblast islands

- Defective remodeling of the basal plate
- Inconclusive association with MVM



Placental septa

- Defective remodeling of the basal plate
- Inconclusive association with MVM



2. FETAL VASCULAR MALPERFUSION (FVM)

- Constellation of lesions resulting from impaired / obstructed fetal blood flow
- Replaces “fetal thrombotic vasculopathy”

Arch Pathol Lab Med 2016;140(7):698-713
Surg Pathol Clin 2022;15(2):175-196

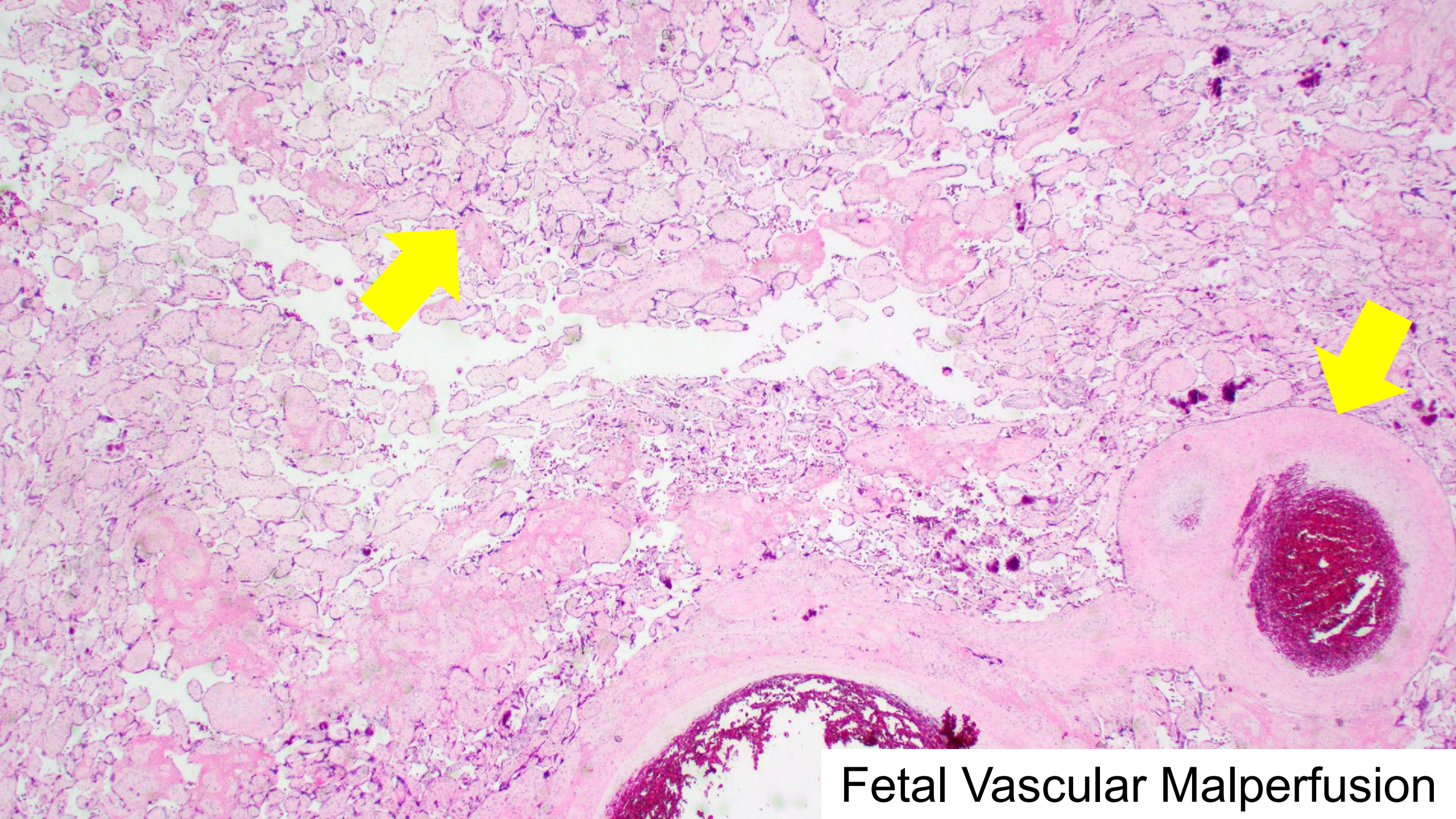
2. FETAL VASCULAR MALPERFUSION (FVM)

- Umbilical cord abnormalities / accidents
  - *Cord compression (oligohydramnios, multiple gestation)*
  - *Nuchal cord* *True knots*
  - *Long cord (>70 cm)* *Hypercoiled cord (>5 coils / 10 cm)*
  - *Velamentous cord insertion (compression of free vessels)*
- Other causes: maternal diabetes, fetal cardiac insufficiency, hyperviscosity syndromes, inherited thrombophilias

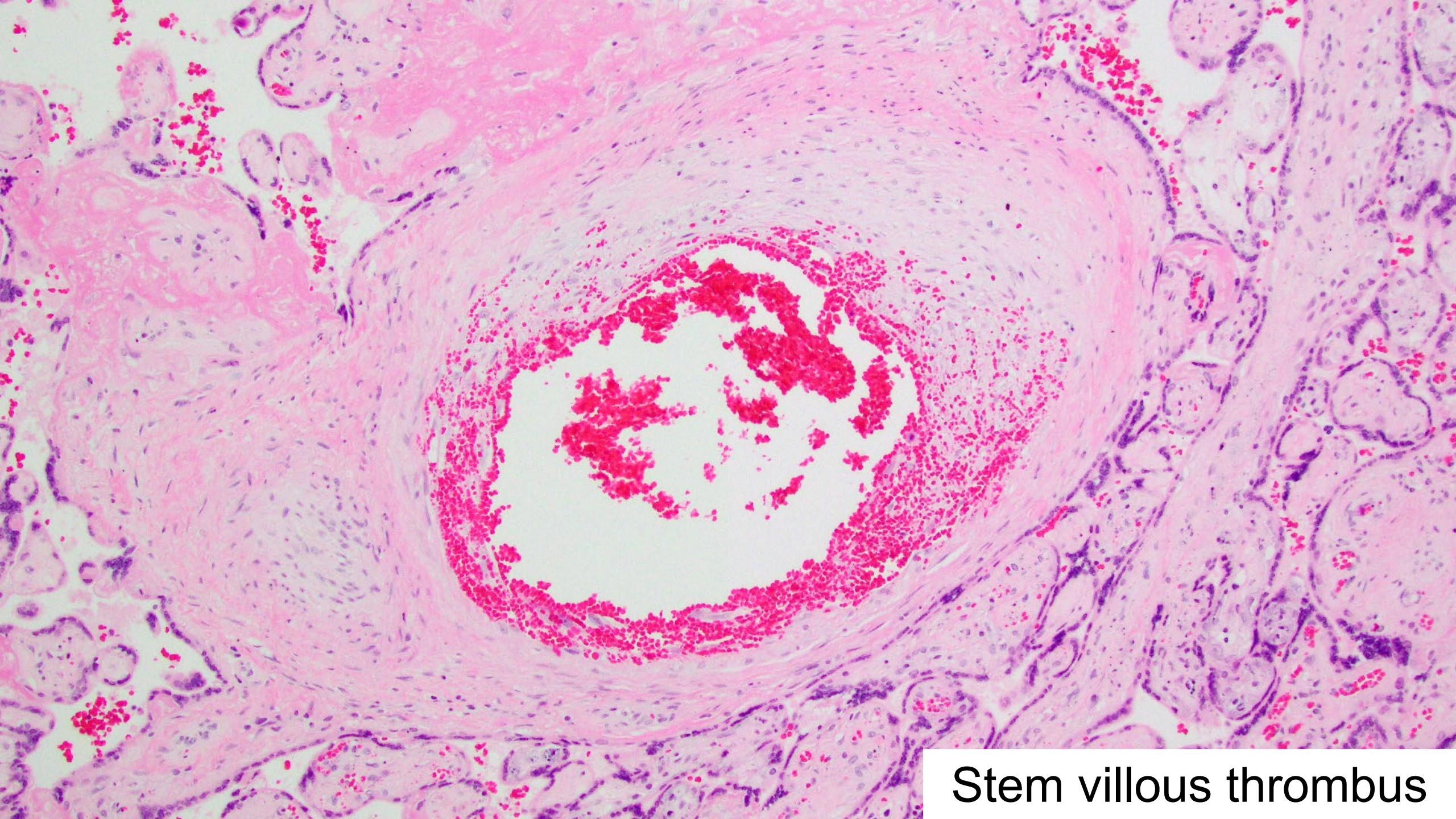
APMIS 2018;126(7):561–9

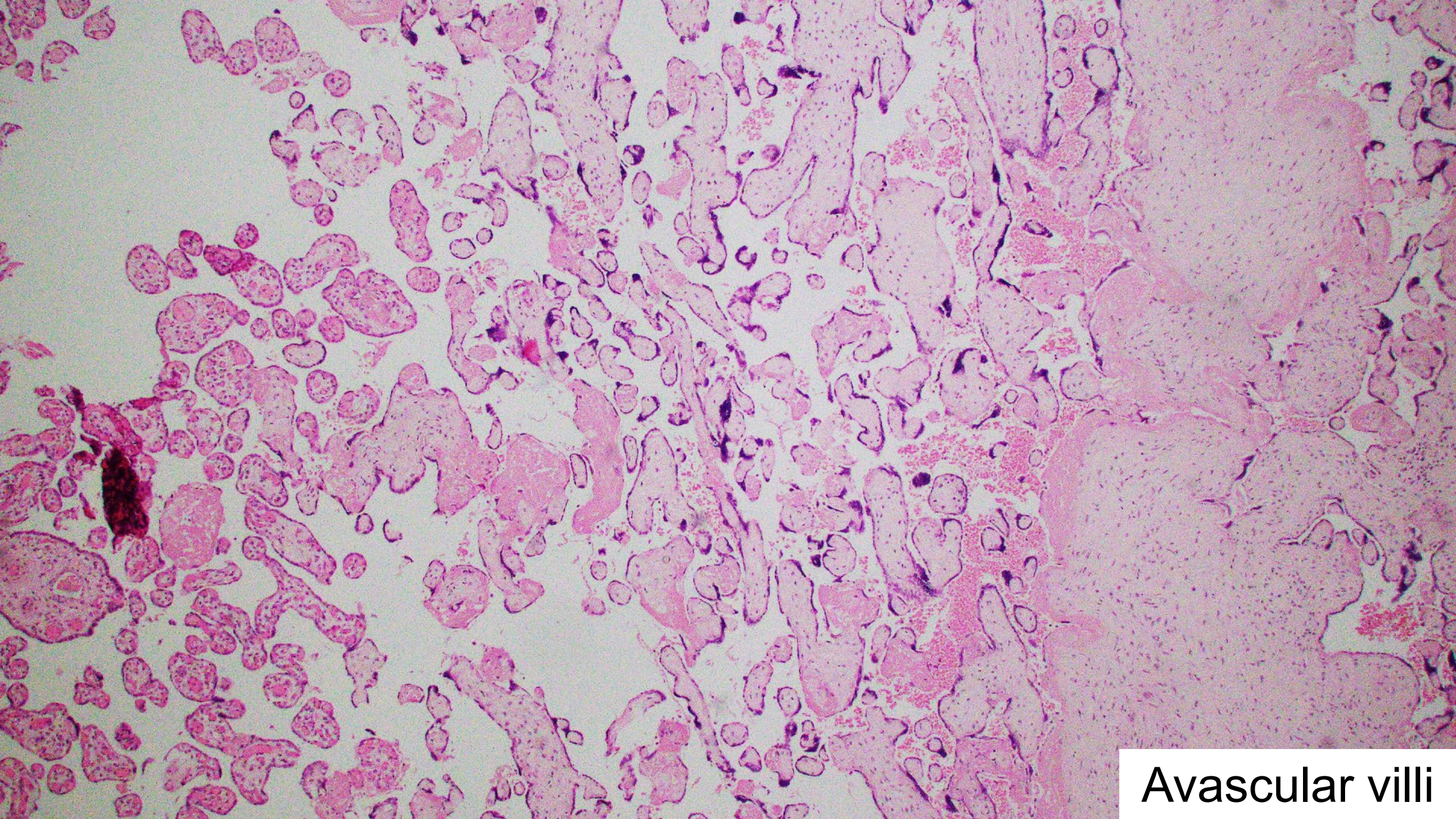
2. FETAL VASCULAR MALPERFUSION (FVM)

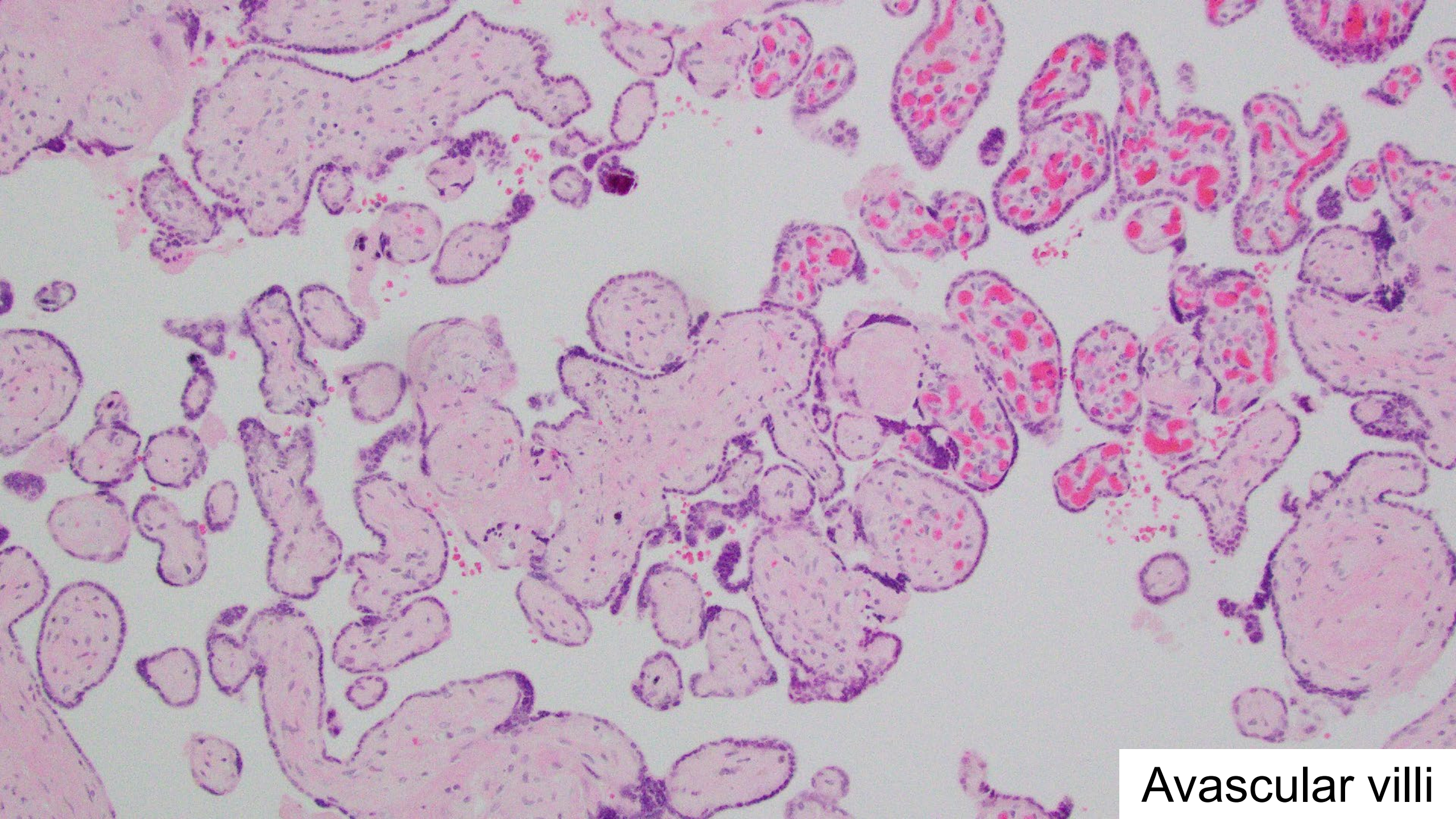
- Major vasculature changes
  - *Fetal vessel thrombosis*
  - *Stem vessel obliteration*
  - *Vascular ectasia*
  - *Vascular intramural fibrin deposition*
- Downstream villous effects
  - *Avascular villi*
  - *Villous stromal karyorrhexis*

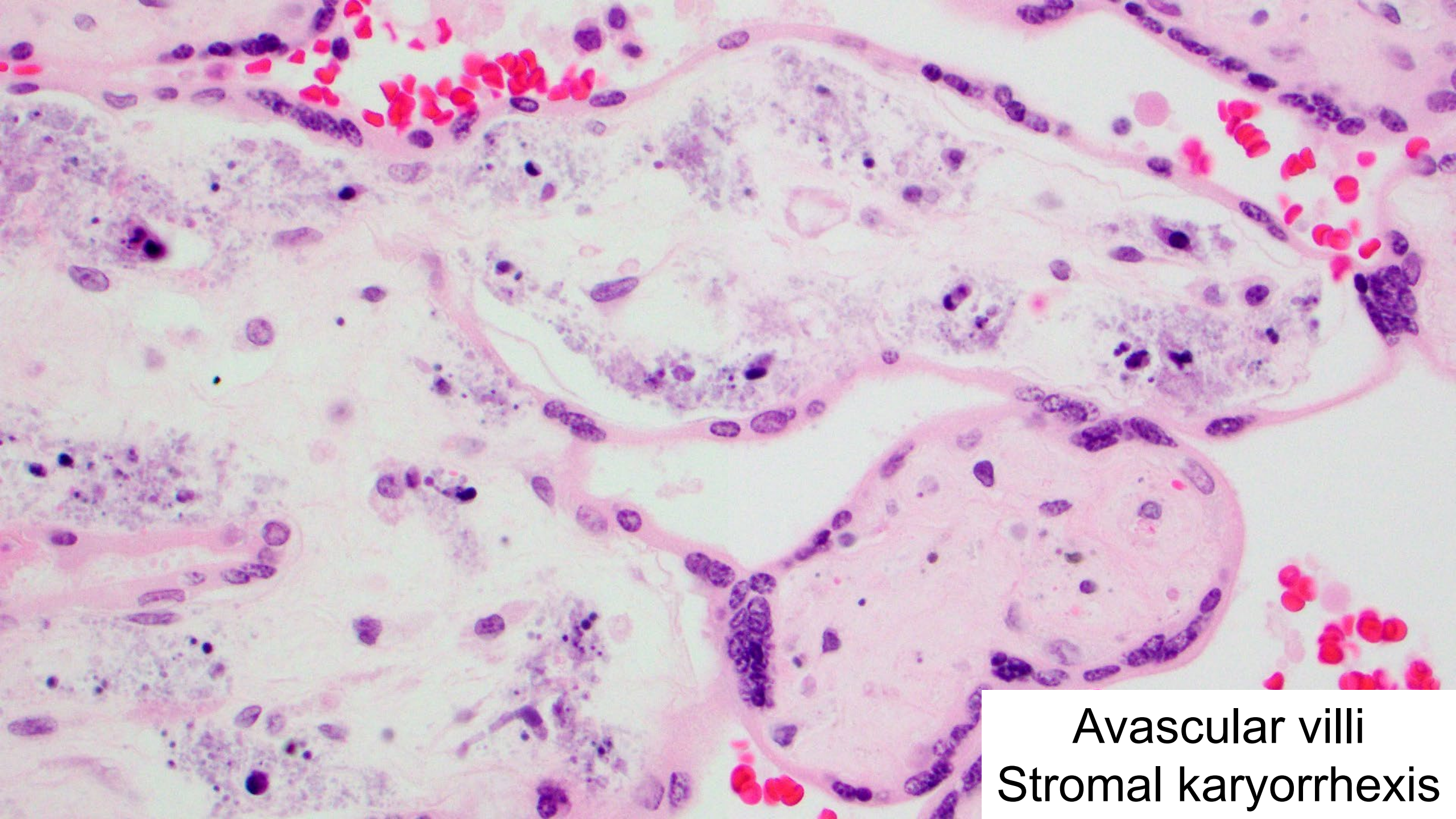


Fetal Vascular Malperfusion


Stem villous thrombus


Avascular villi


Avascular villi


Avascular villi
Stromal karyorrhexis

2. FETAL VASCULAR MALPERFUSION (FVM)

- Segmental
  - *Occlusion of one stem villous vessel with downstream effect*
  - *Focal, with villous thrombi and discrete large AVV areas (>10 villi)*
- Global
  - *Partial / intermittent umbilical cord occlusion*
  - *Diffuse, with vascular ectasia, fibrin deposition and multiple small AVV areas (<10 villi)*

Arch Pathol Lab Med 2016;140(7):698-713
Surg Pathol Clin 2022;15(2):175-196

2. FETAL VASCULAR MALPERFUSION (FVM)

- High grade = one of the following
  - *≥ 45 avascular villi over 3 sections examined (≥ >15 villi per section)*
  - *≥ 2 occlusive or non-occlusive thrombi in chorionic or stem villi*
  - *Multiple non-occlusive thrombi*
- Low grade = not high-grade as defined above

Arch Pathol Lab Med 2016;140(7):698-713

FVM - ISSUES

- Poor interobserver reproducibility
- Low-grade FVM / isolated lesions are common in normal pregnancies
  - *A single lesion of FVM seen in ~20% (small AVV foci, non-occlusive vessel intimal fibrin deposition)*
- High-grade FVM / ≥ 2 lesions are more specific
  - *<1% of normal pregnancies*
  - *Intrauterine fetal demise and neurodisability*

Arch Pathol Lab Med 2022;146(3):372-378
Pediatr Dev Pathol 2010;13(6):459–64
Pediatr Dev Pathol 2016;19:237-43
J Perinat Med 2018;46(6):613–30
Placenta 2009;30(12):1083–8
Pediatr Dev Pathol 2011;14:345-52

3. PLACENTA ACCRETA SPECTRUM

- Incidence has increased 10-fold in the past 50 years
- Now the leading cause of peripartum hysterectomy (50–65%)
- Risk factors:
  - *Placenta previa & low implantation*
  - *Previous Caesarian sections, uterine curettage or myomectomy*

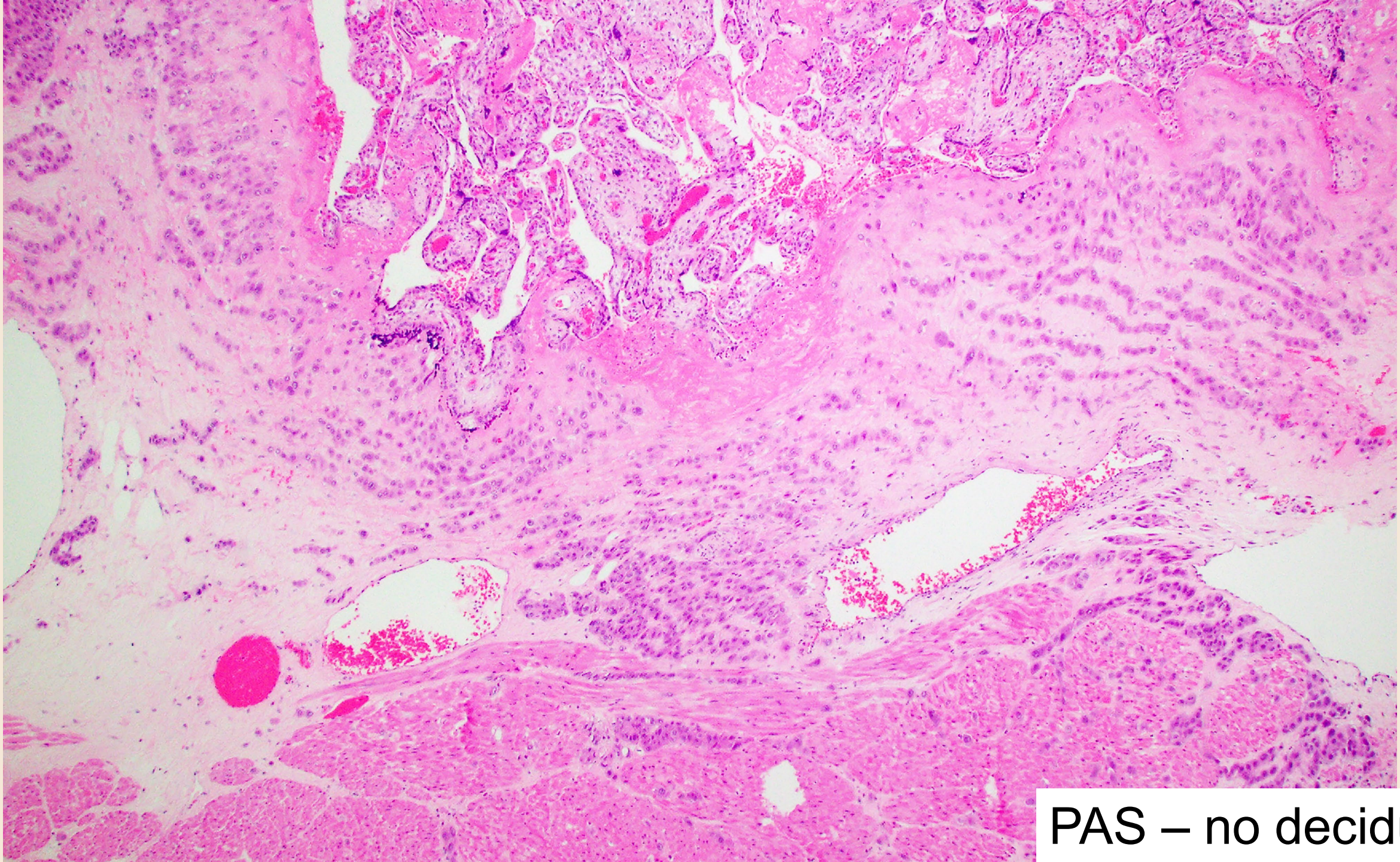
Uterine (endometrial) scarring
DEFECTIVE DECIDUA

J Clin Pathol 2008;61(12):1243–6
Am J Obstet Gynecol 2010;203(5):430–9

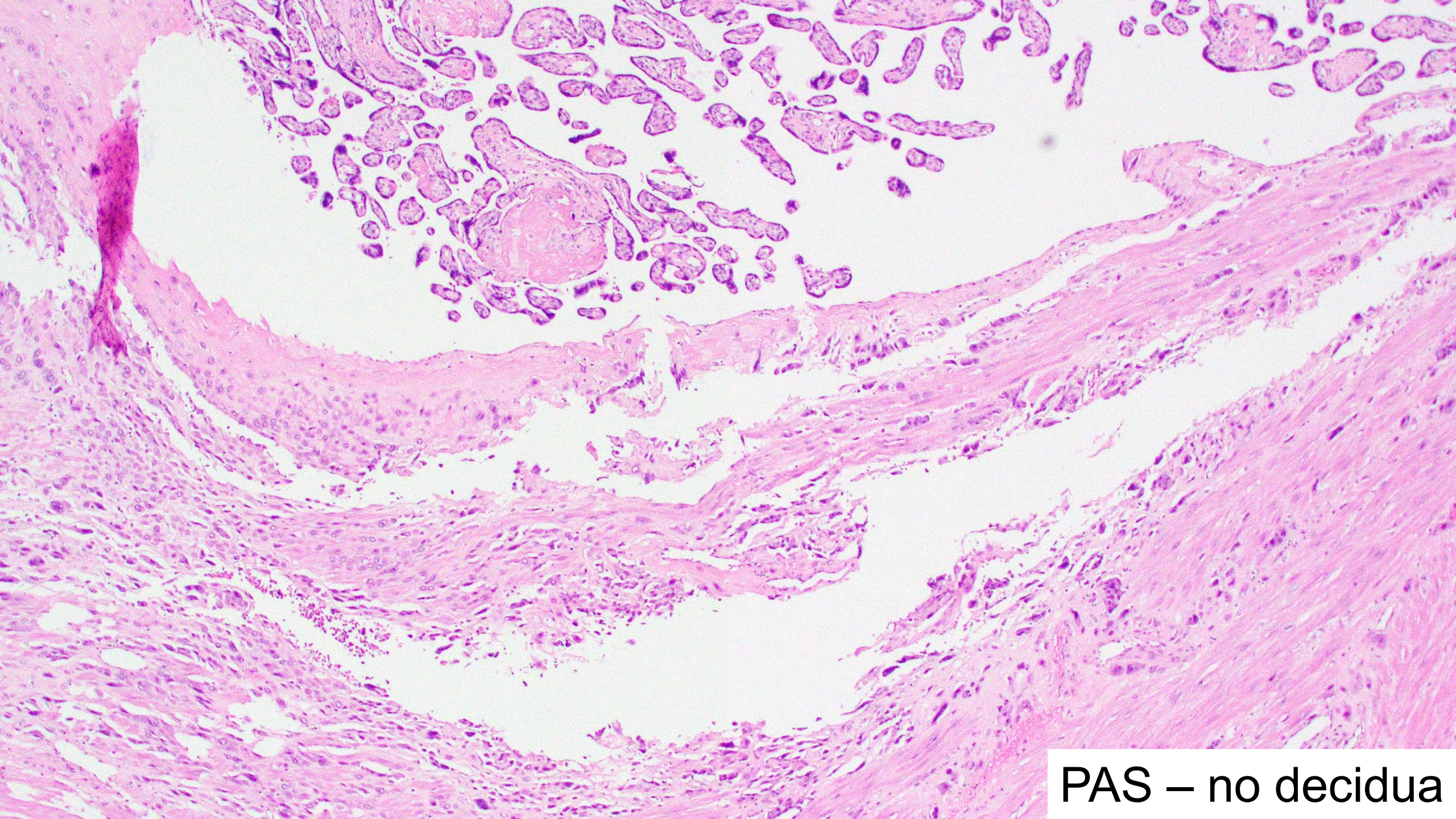
3. PLACENTA ACCRETA SPECTRUM

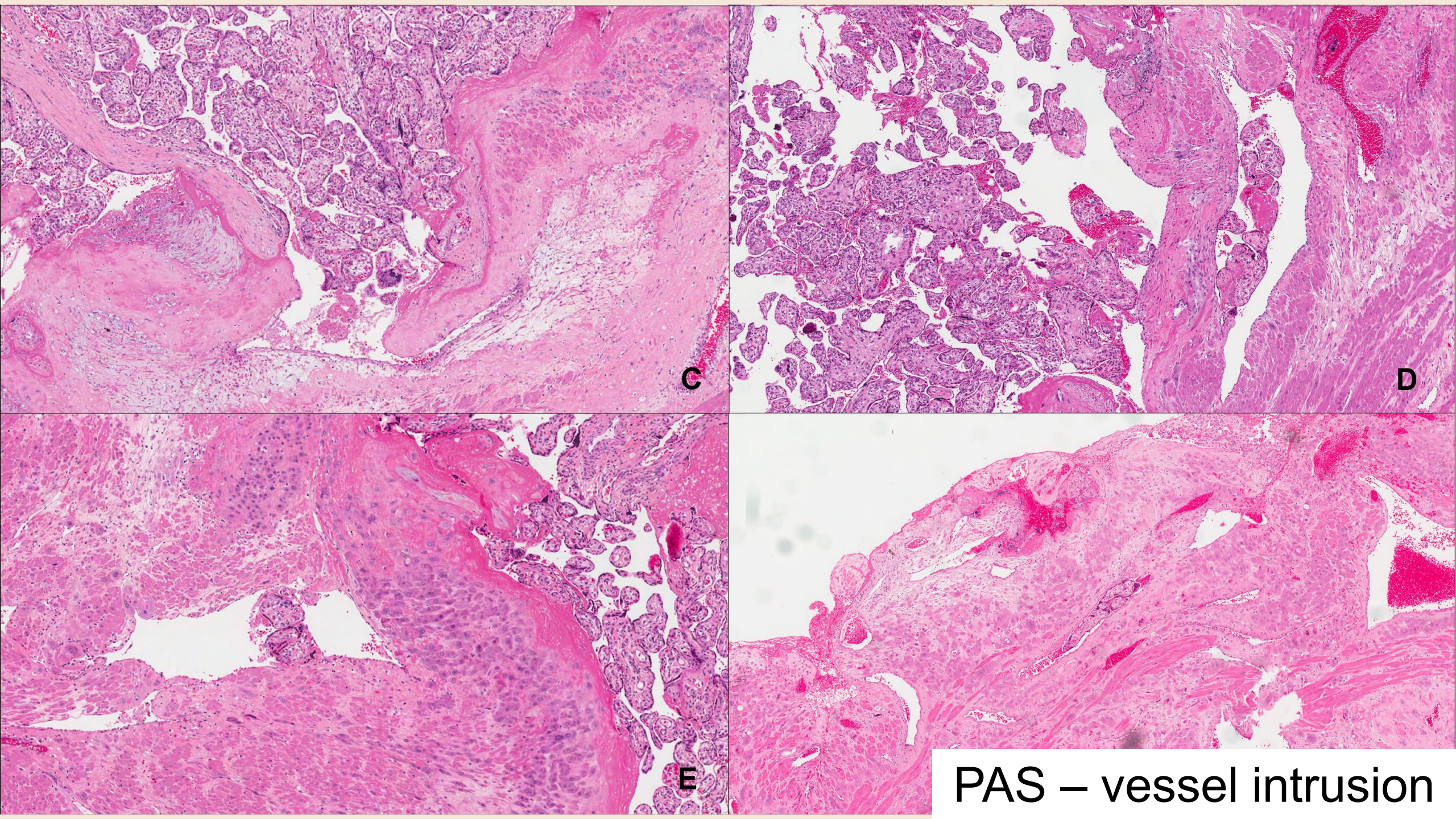
- DIAGNOSTIC CRITERIA
  - *Chorionic villi adjacent to myometrial fibers*
    - Directly in contact or with an intervening layer of fibrinoid
  - *Absence of intervening decidua*
- Other findings
  - *Villous intrusion into myometrial vessels*
  - *Abundant extravillous trophoblast within myometrium*

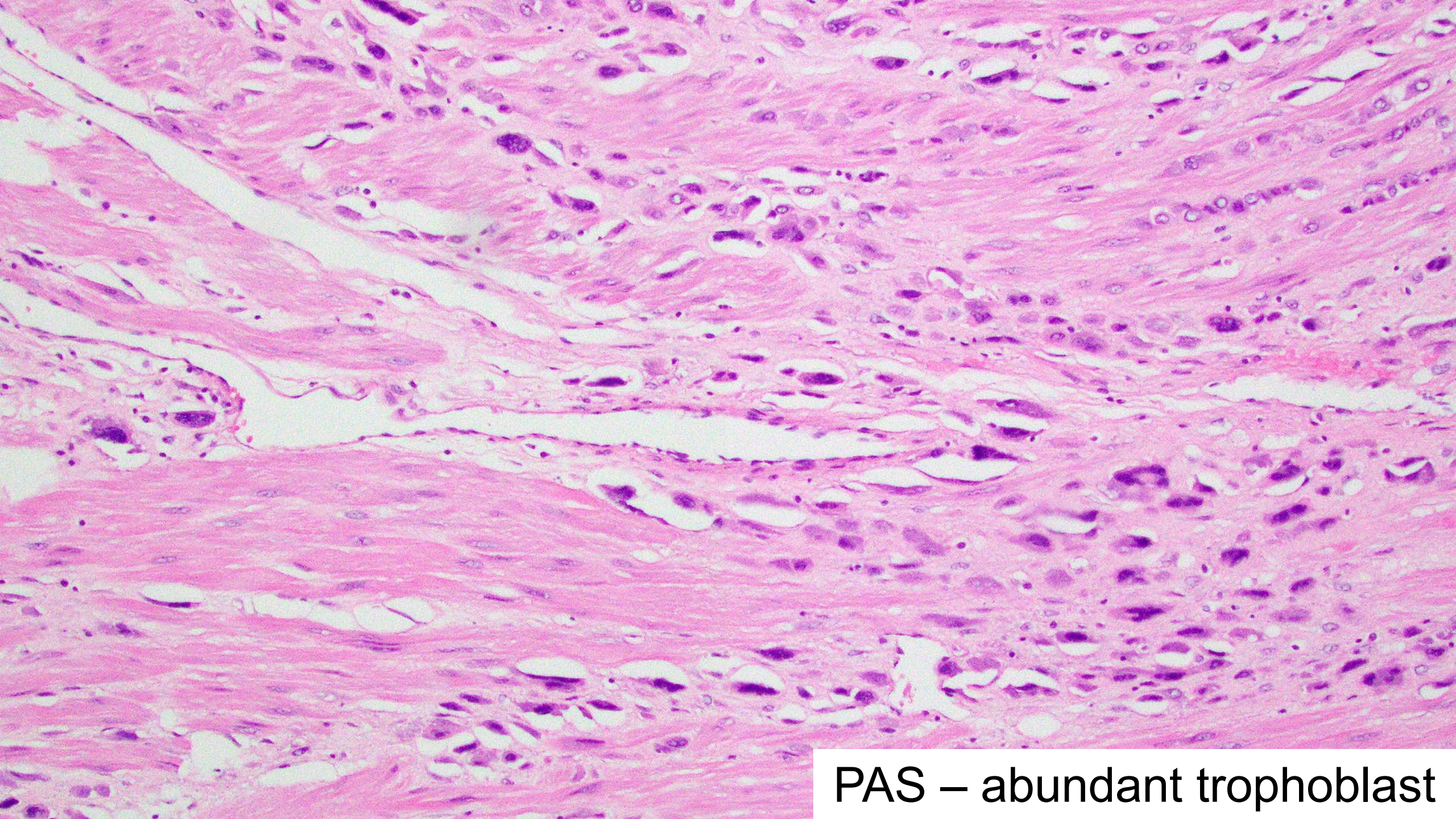
Placenta 2008;29(7):639–45
Int J Gynecol Pathol 2016;35(6):497–508



PAS – no decidua


PAS – no decidua


C
D
E
PAS – vessel intrusion


PAS – abundant trophoblast

Placenta Accreta Spectrum Pathologic Grading			
Clinical Grading (FIGO[81])—Histologic Criteria		Pathologic Grading (Hecht and Coworkers[80])—Criteria	
Grade 1	Accreta - Histology shows extended areas of absent decidua between villous tissue and myometrium; placental villi attached directly to the superficial myometrium	Grade 1	Noninvasive: grossly adherent placenta. Myometrial cross sections show a smooth placental–myometrial interface and uniform myometrial thickness without thinning
Grade 2	Increta - Histology shows placental villi within the muscular fibers and sometimes in the lumen of the deep uterine vasculature	Grade 2	Superficial invasion: irregular placental–myometrial interface with preservation of at least 25% of the myometrial wall thickness relative to the uninvolved myometrium

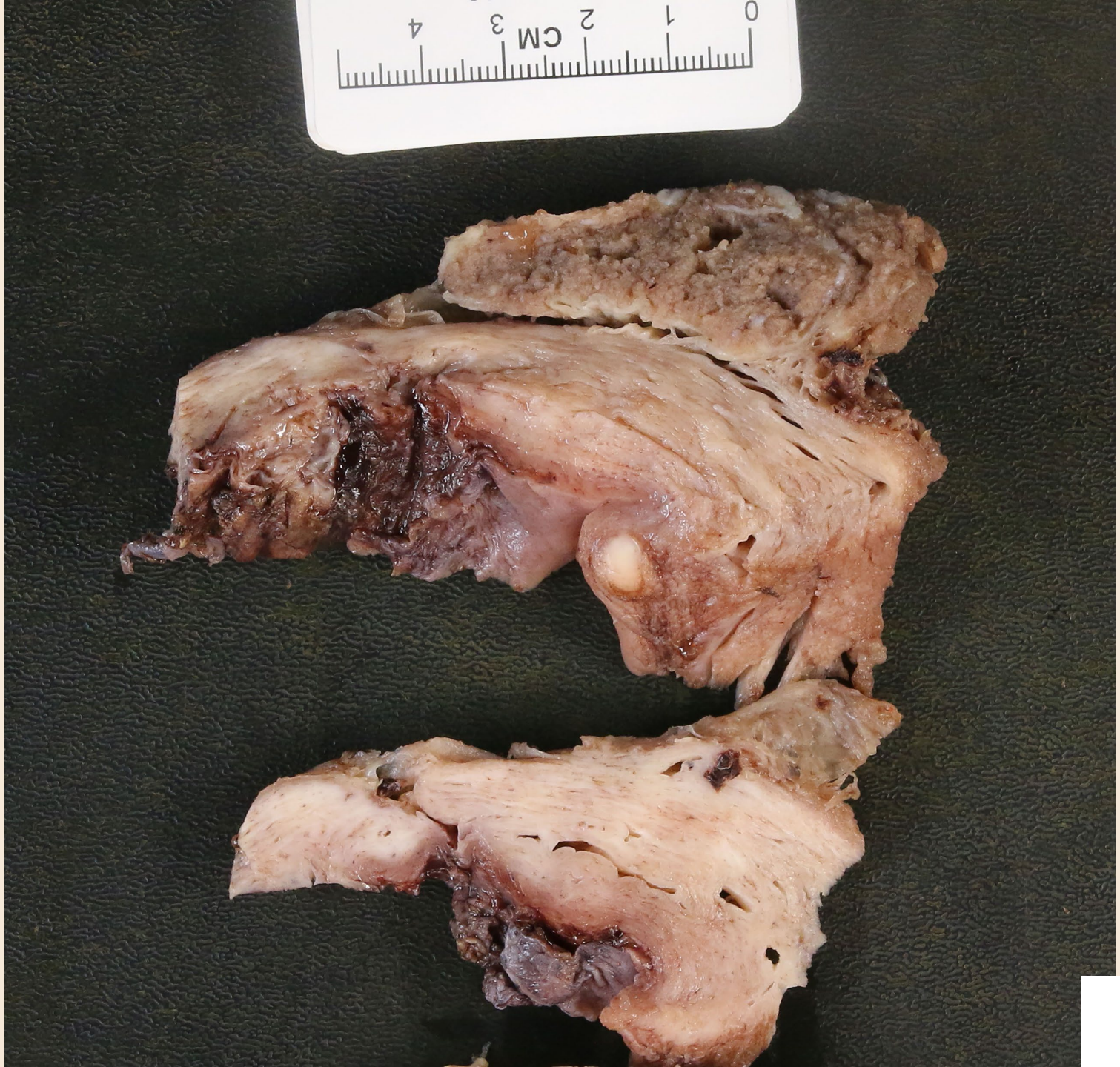
Surg Pathol Clin 2022;15(2):175-196

Int J Gynaecol Obstet 2019;146(1):20–4 Mod Pathol 2020;33(12):2382–96

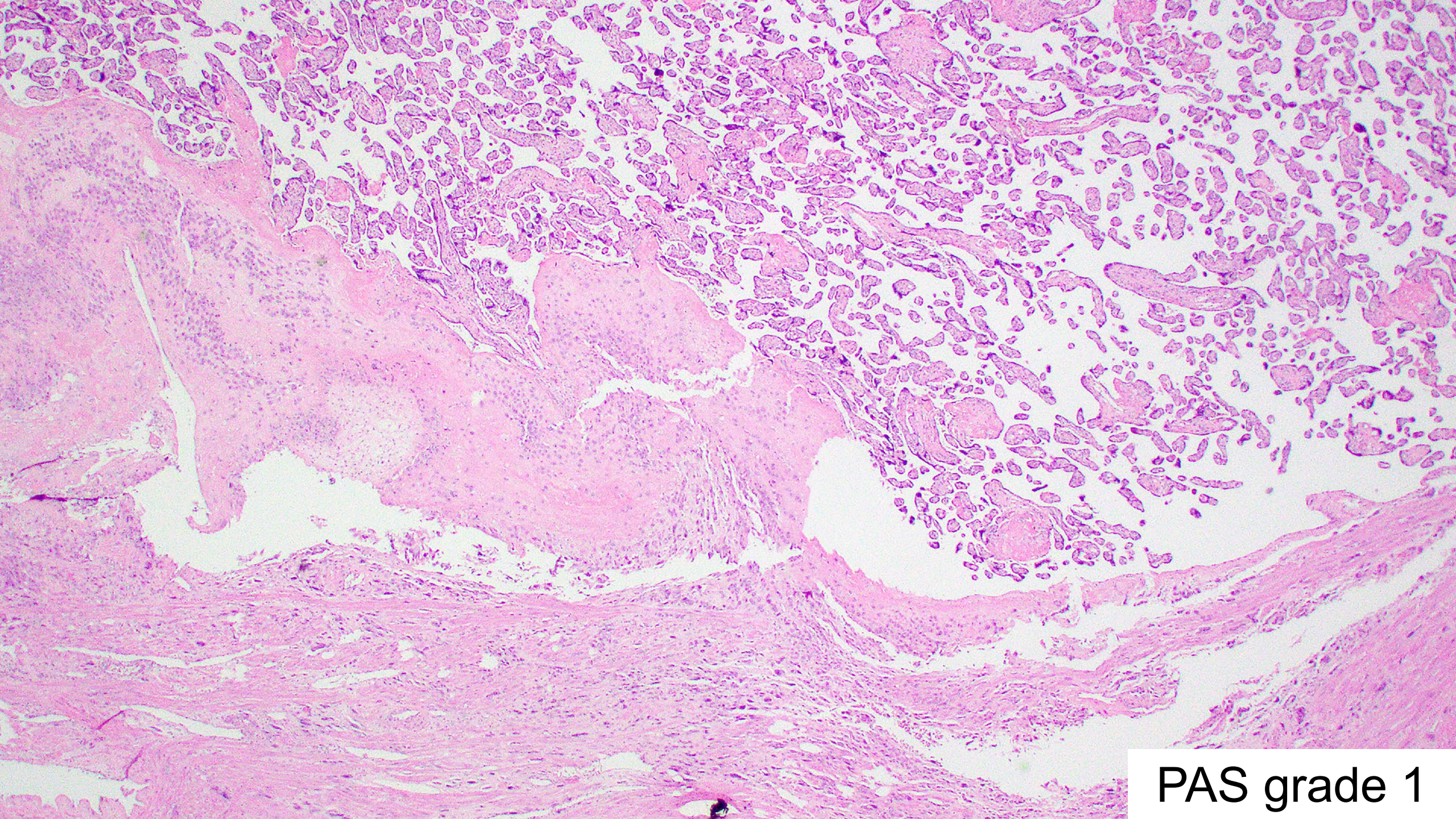
Grade 3	3a: Percreta - Histology shows villous tissue within or breaching the uterine serosa	Grade 3	3a: Deep invasion: Irregular placental–myometrial interface with preservation of <25% of the myometrial wall thickness relative to the uninvolved myometrium. The serosa is intact.
	3b: Percreta - Histology shows villous tissue breaching the uterine serosa and invading the bladder wall tissue or urothelium		3d: Deep invasion with disruption of the serosa
	3c: Percreta - Histology shows villous tissue breaching the uterine serosa and invading pelvic tissues/organs (with or without invasion of the bladder)		3e: Placental invasion into adjacent organs (most commonly bladder) or extrauterine fibroadipose tissue, confirmed by microscopy

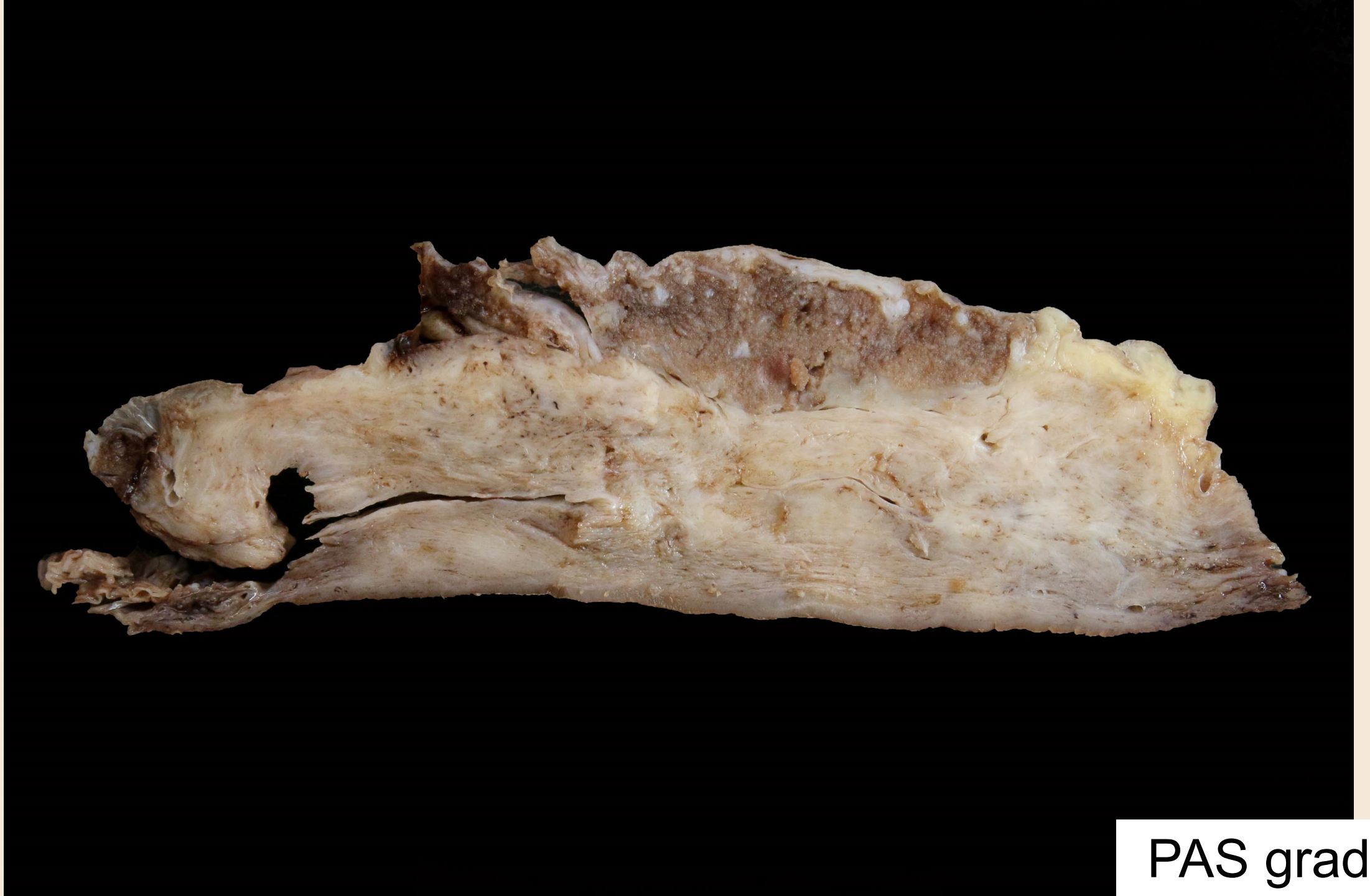
Surg Pathol Clin 2022;15(2):175-196

Int J Gynaecol Obstet 2019;146(1):20–4 Mod Pathol 2020;33(12):2382–96

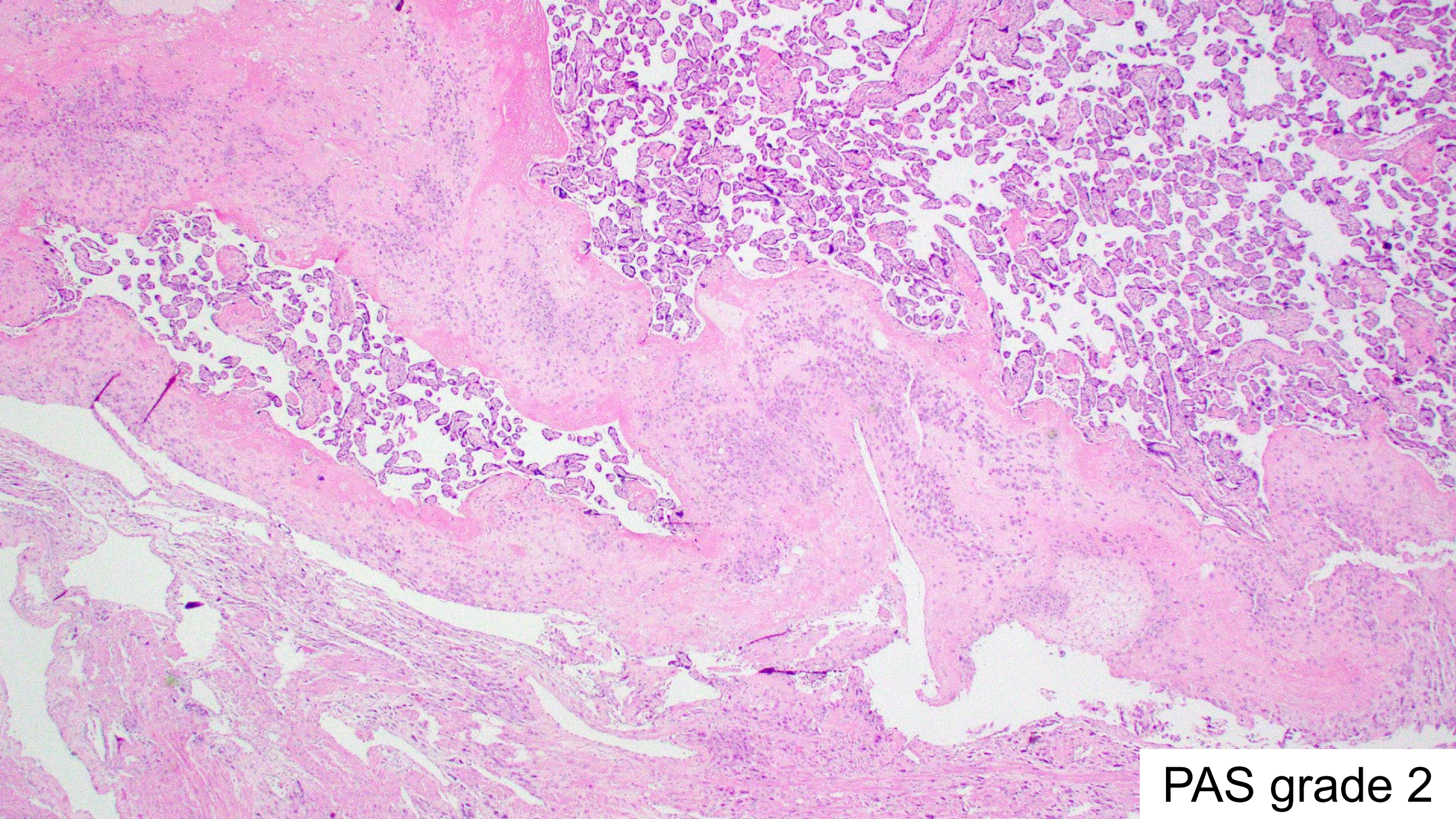


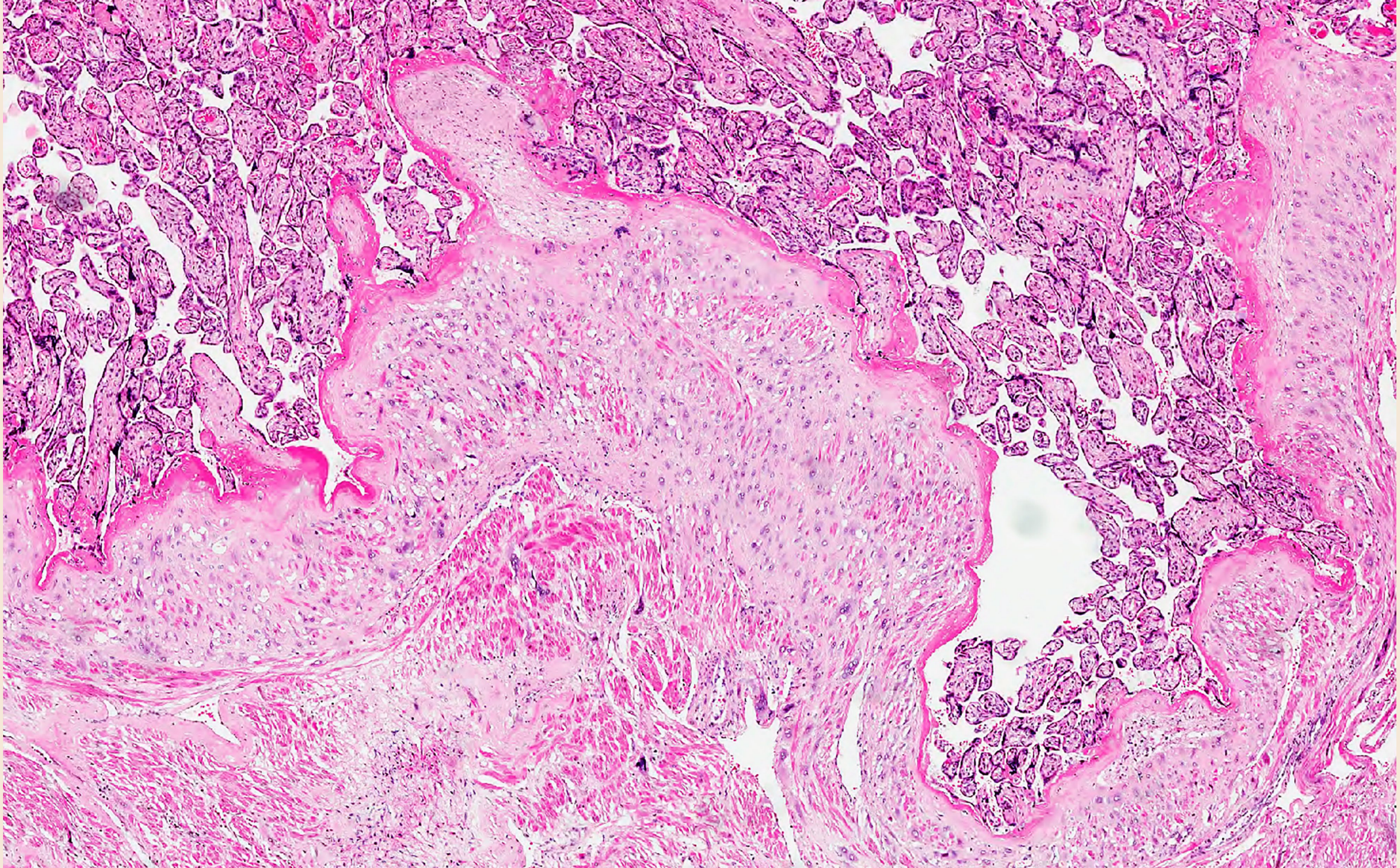
PAS grade 1

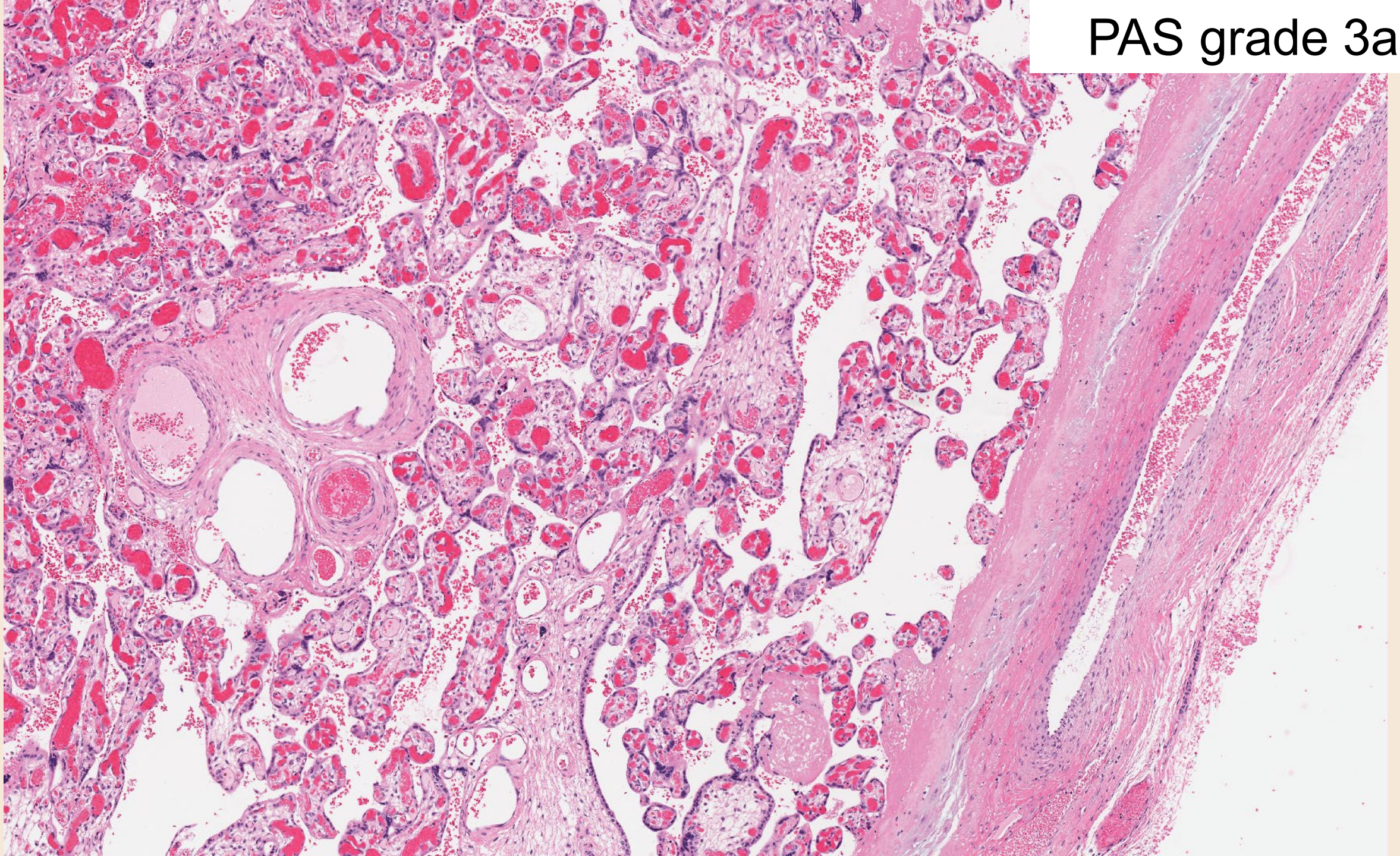

PAS grade 1

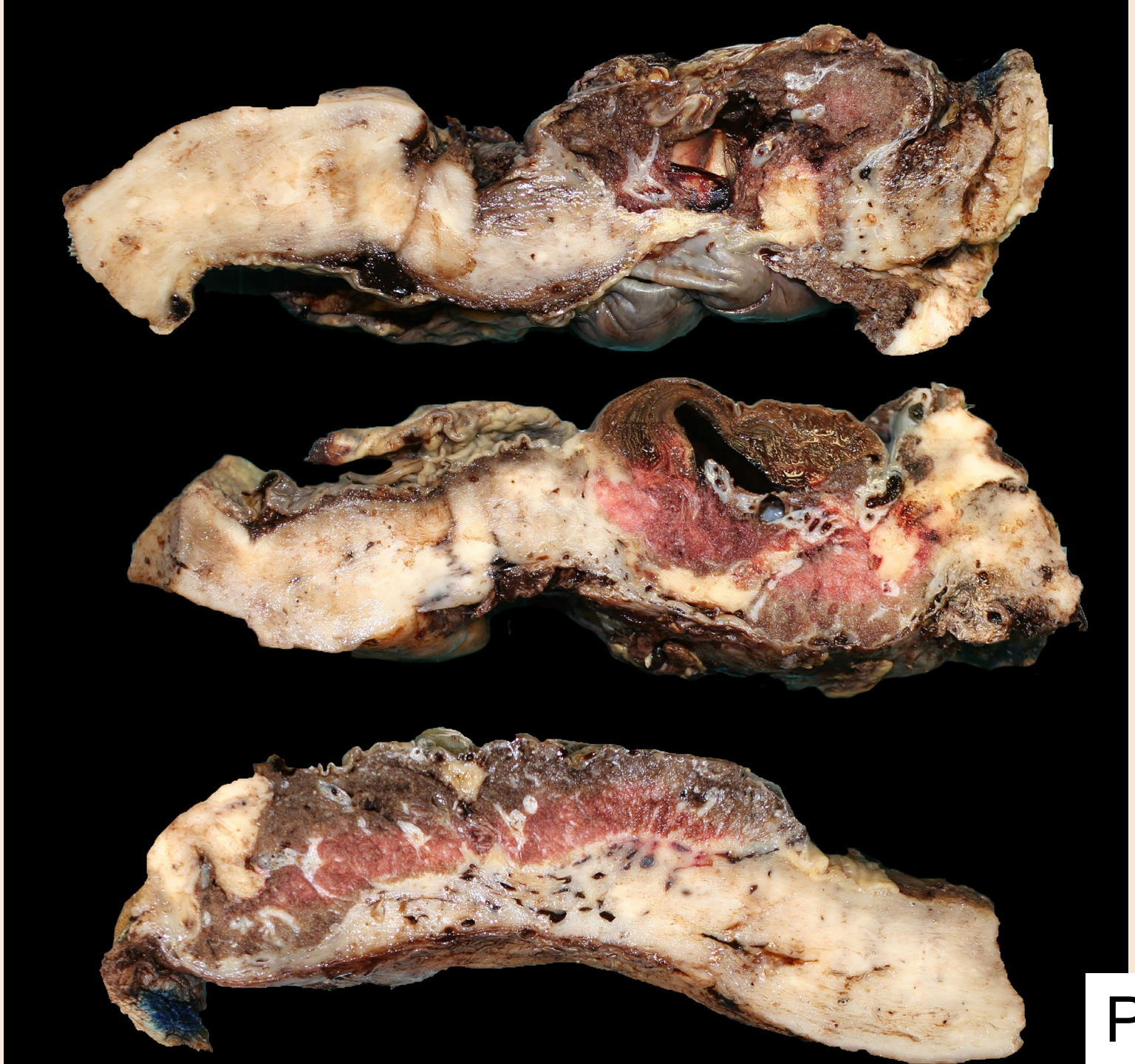


PAS grade 2


PAS grade 2

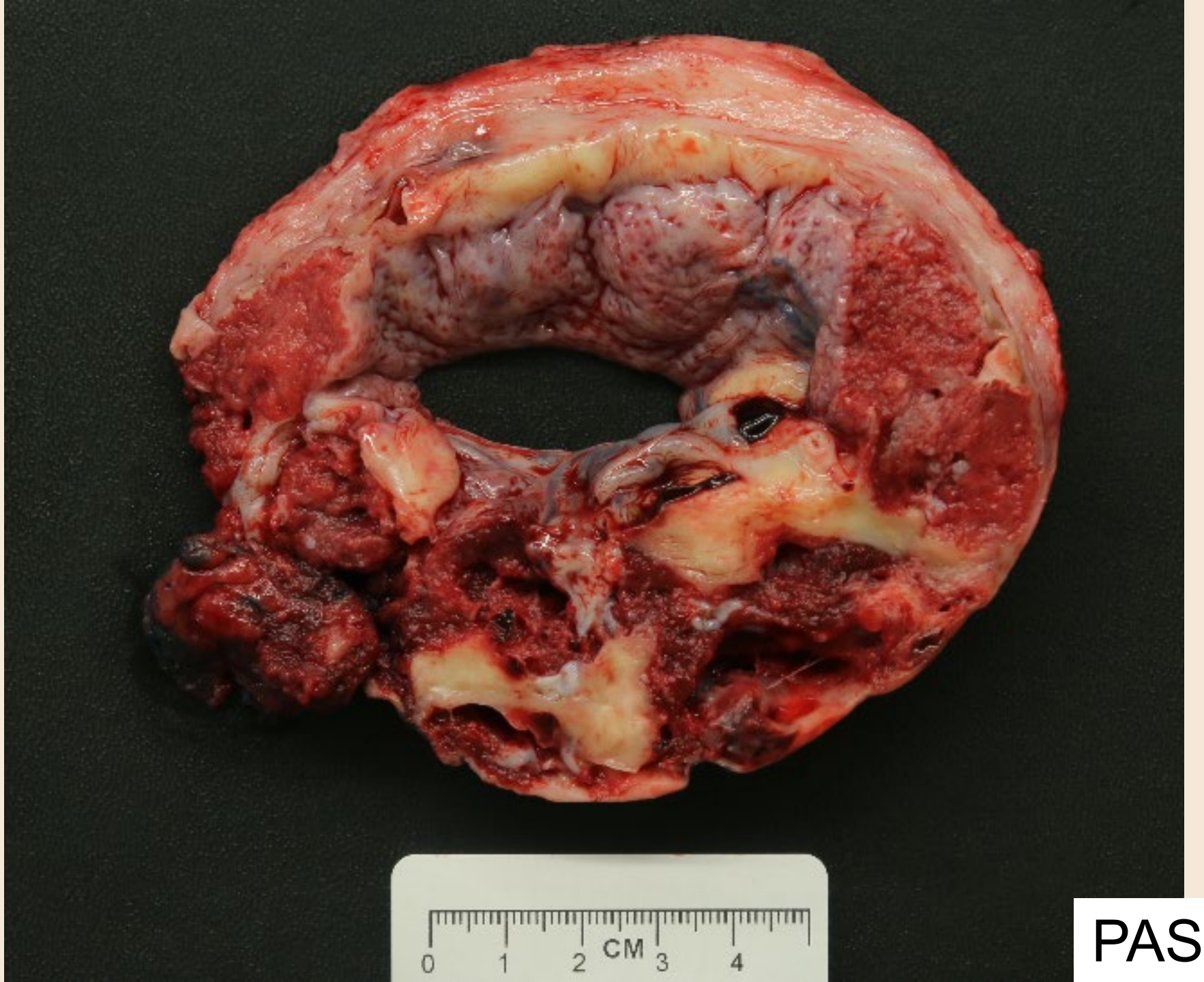



PAS grade 3a

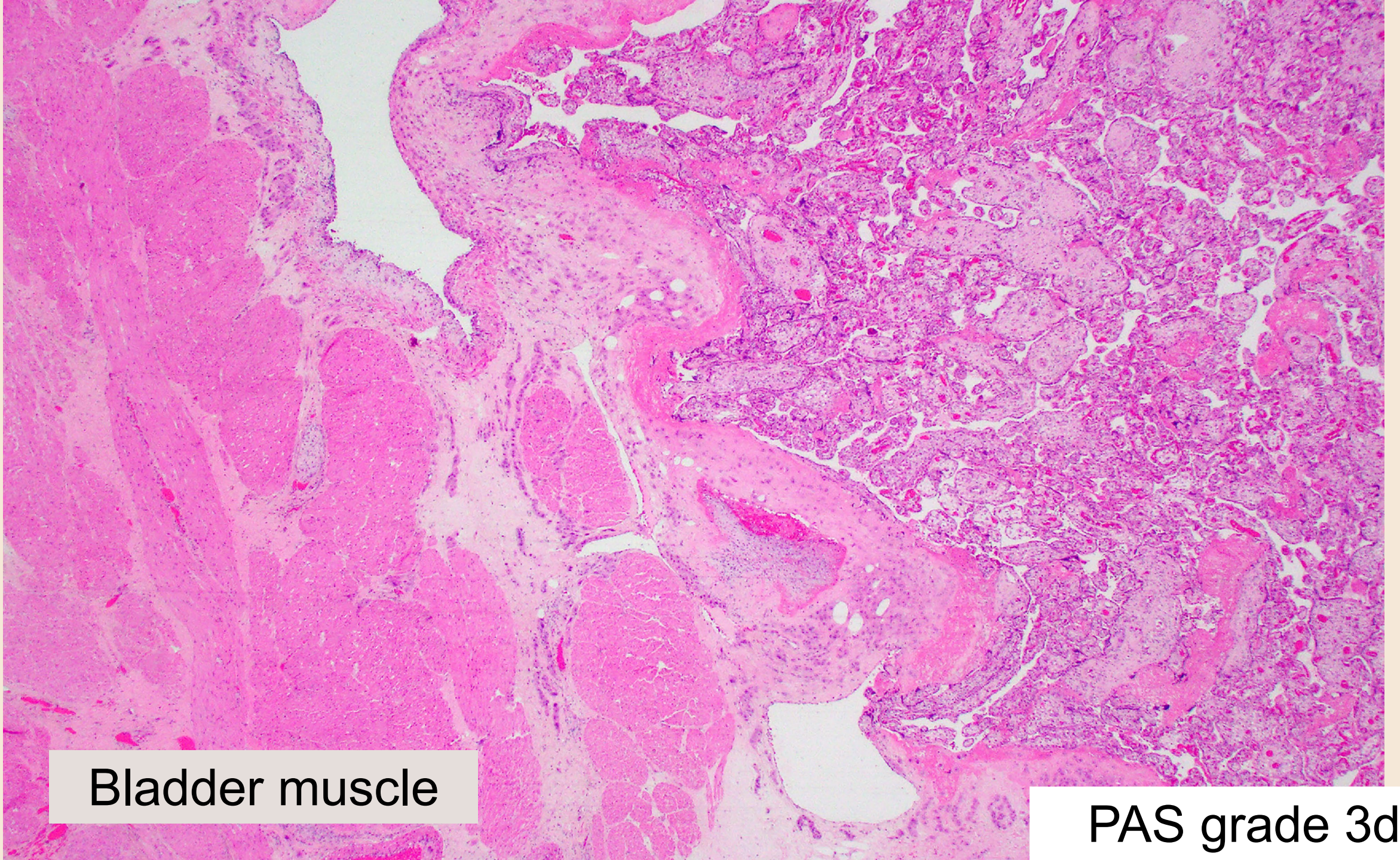


PAS grade 3d

PAS grade 3d



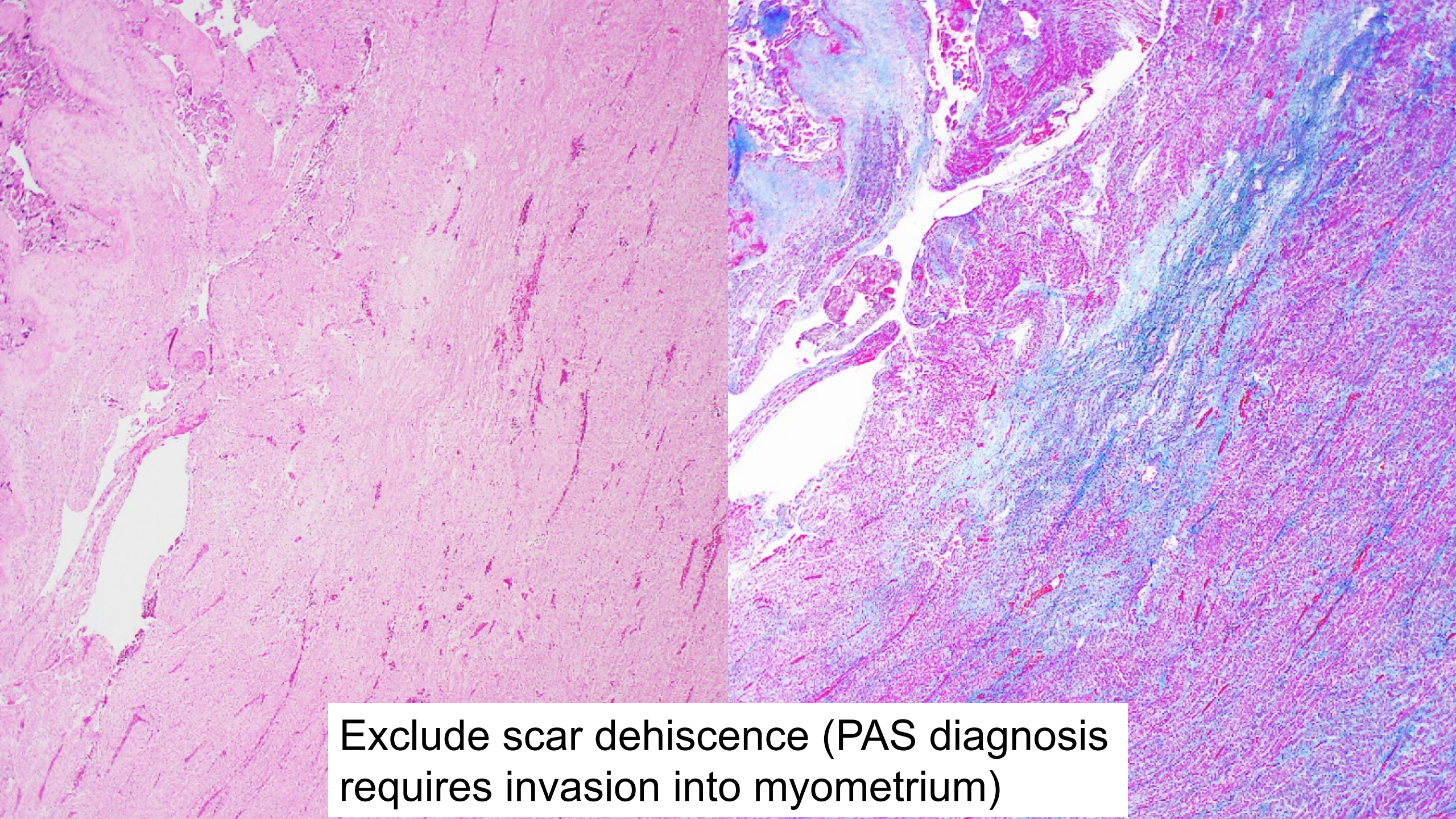
PAS grade 3d


Bladder muscle
PAS grade 3d

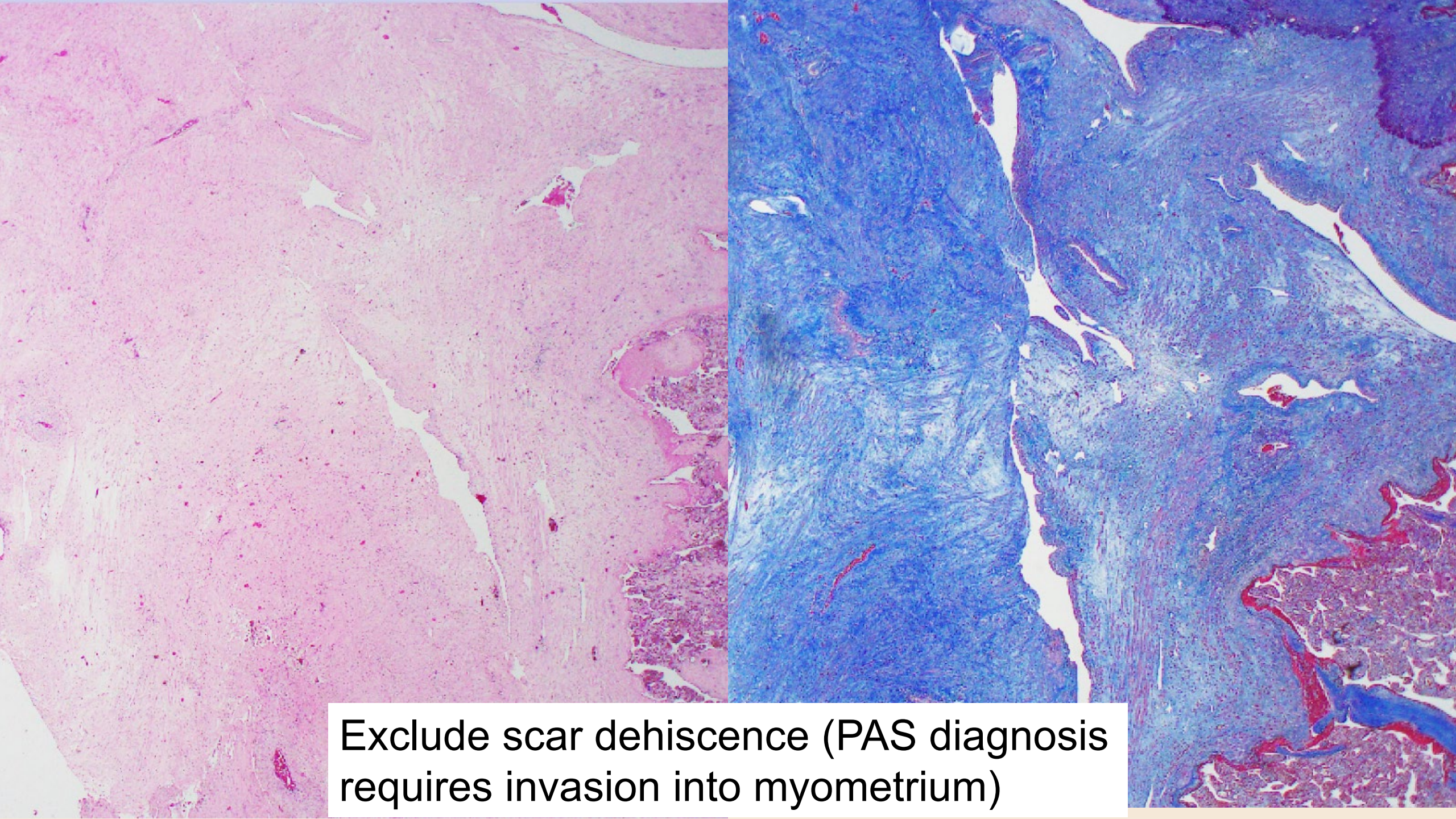
PAS OR C-SECTION SCAR DEHISCENCE?

- Features that suggest dehiscence
  - *History of previous C-section delivery*
  - *Placenta bulging on anterior lower uterine segment*
  - *“Uterine window” seen intraoperatively*
  - *Thinned uterine wall composed of scar tissue*
- Distinction is not always easy
- PAS and dehiscence overlap (and probably collaborate)

Mod Pathol 2020;33(12):2382-2396
Surg Pathol Clin 2022;15(2):175-196
Obstet Gynecol 2020;135(5):1104–11



Exclude scar dehiscence (PAS diagnosis requires invasion into myometrium)

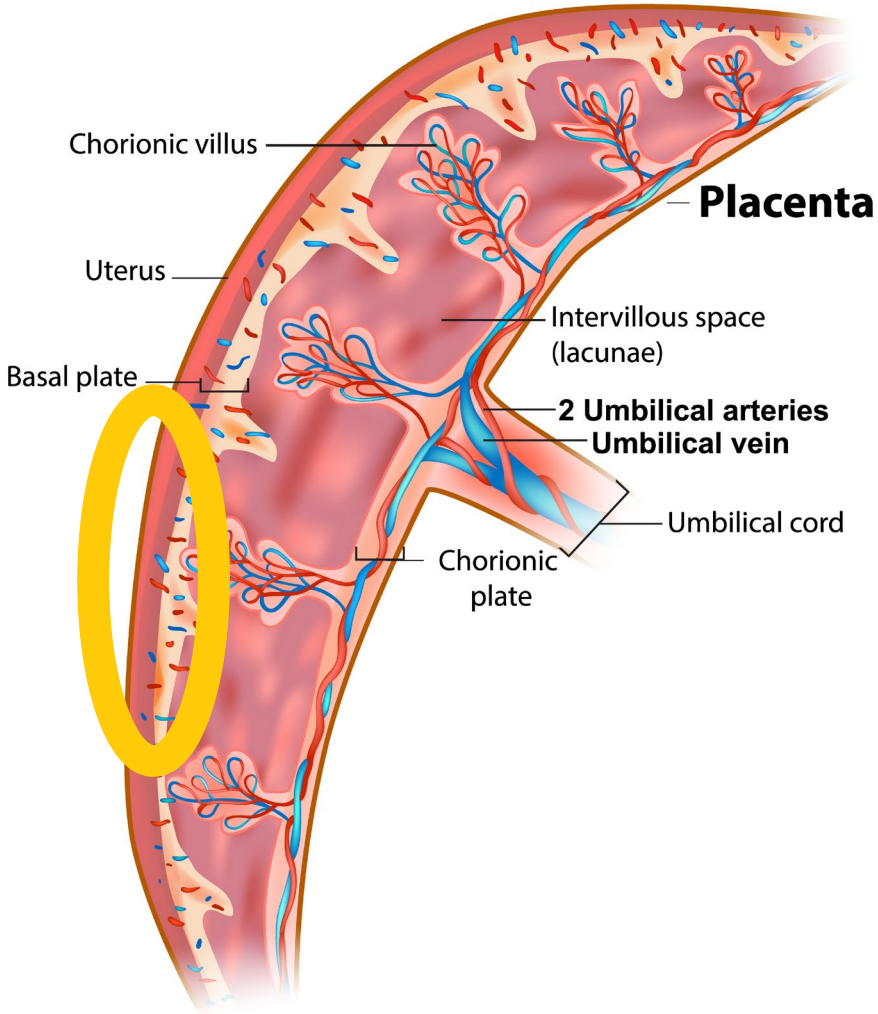


Exclude scar dehiscence (PAS diagnosis requires invasion into myometrium)

PLACENTAL LESIONS B

- Placental lesions associated with recurrence risk
  4. *Basal plate myometrial fibers*
  5. *Massive perivillous fibrin deposition*
  6. *Chronic histiocytic intervillositis*
  7. *Chronic villitis of unknown etiology*

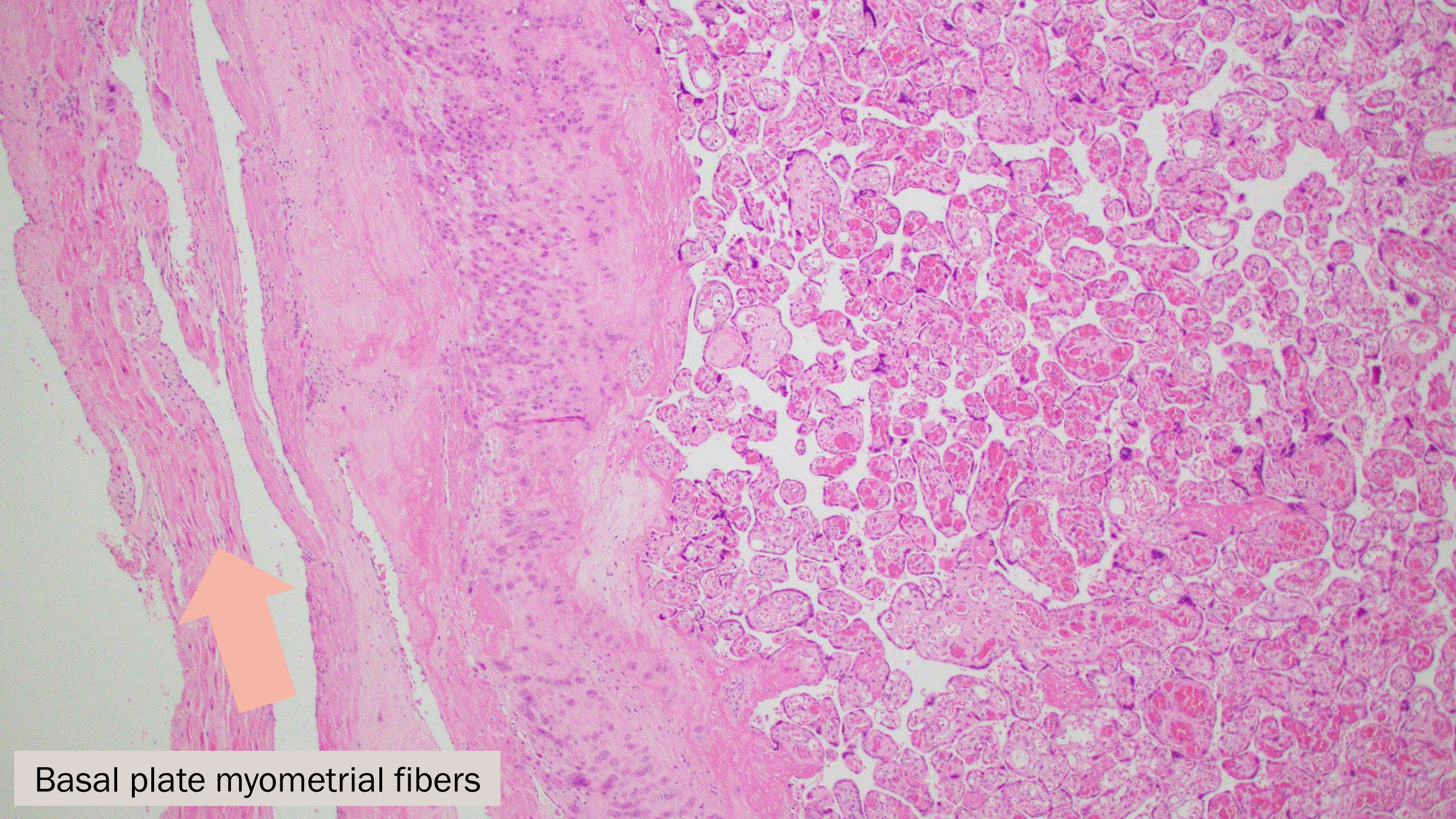
4. BASAL PLATE MYOMETRIAL FIBERS

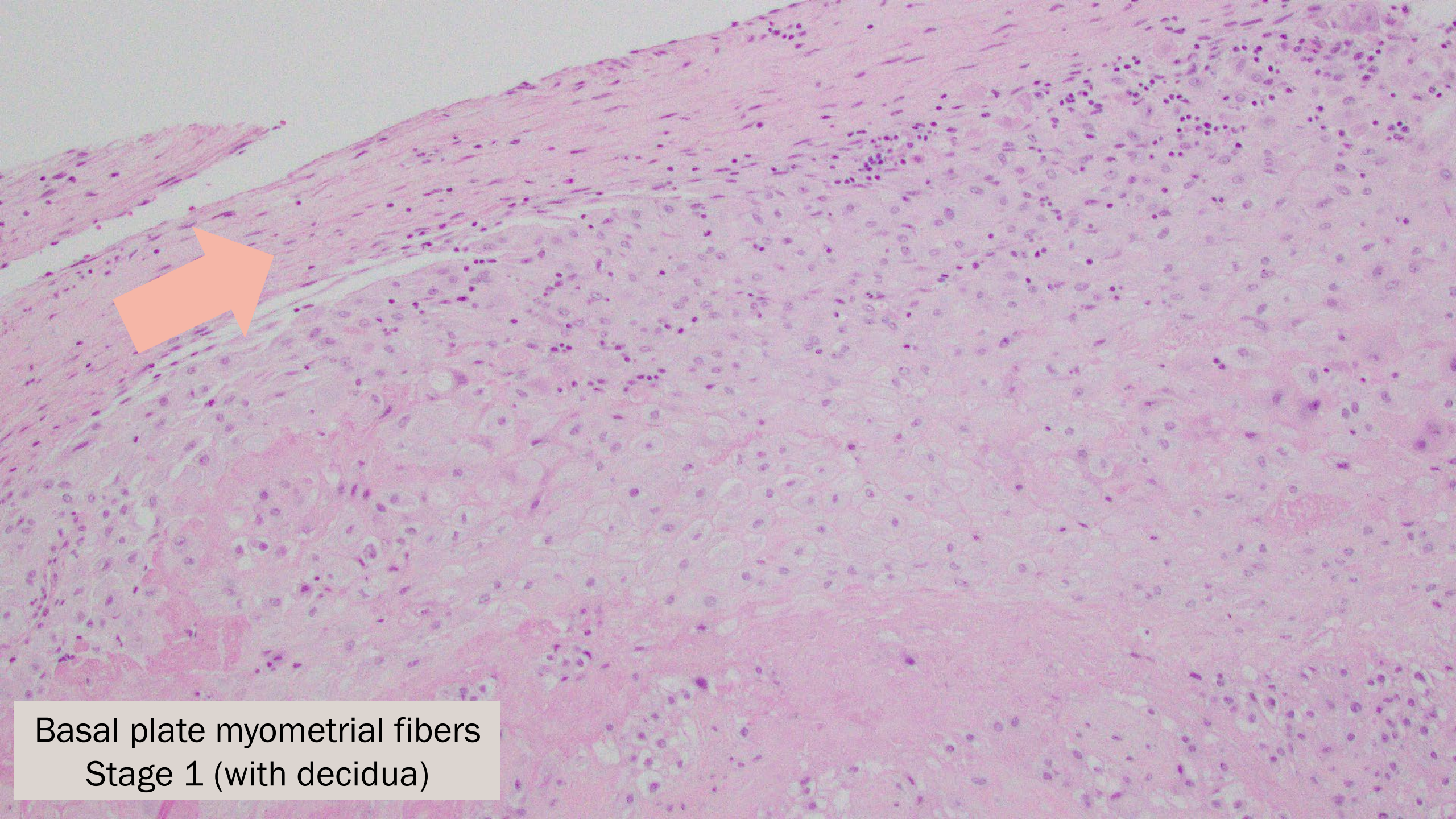


BPMF—Reporting Parameters

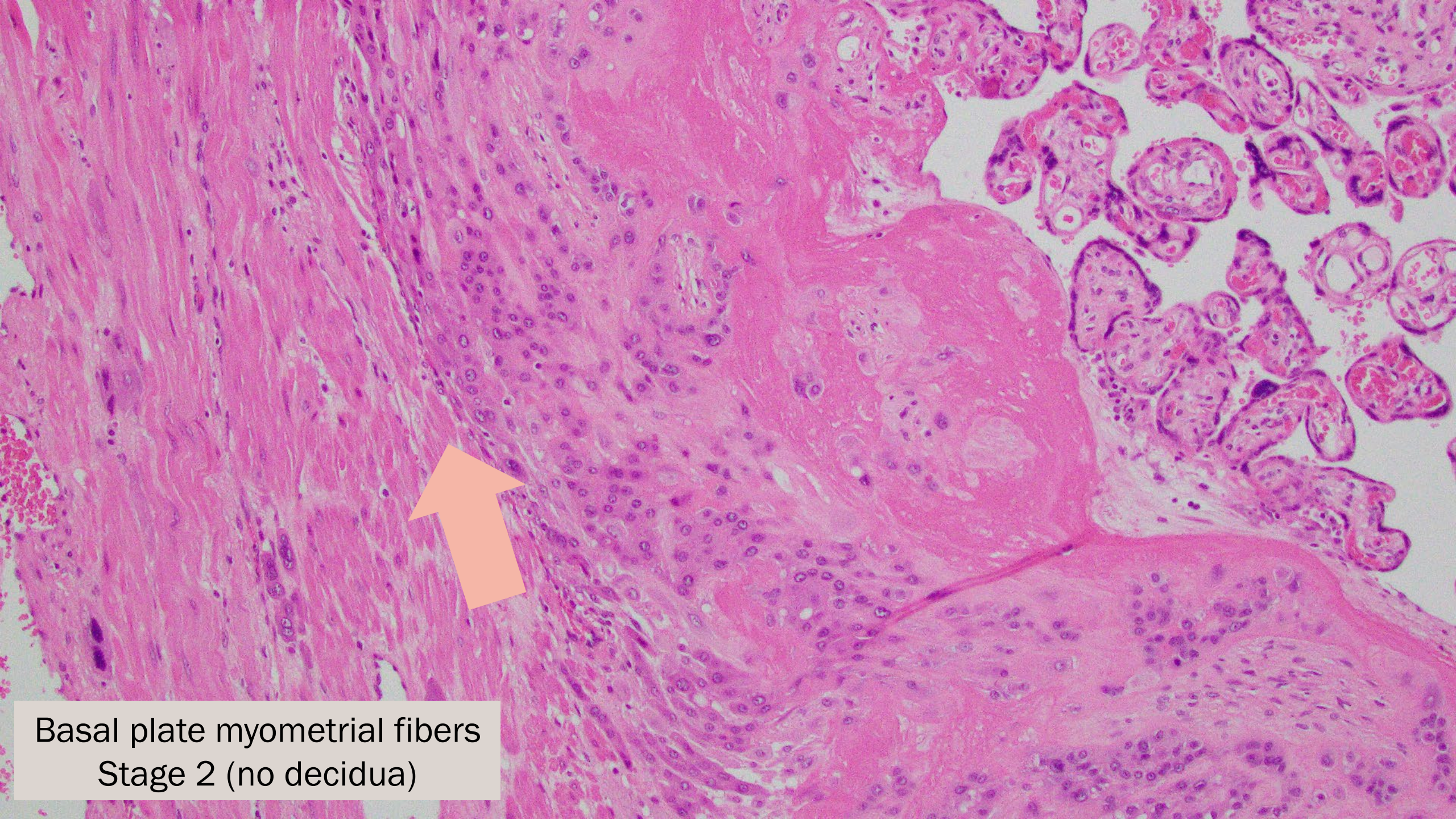
Parameter	Definition
Stage	Stage 1: With accompanying decidua Stage 2: Without decidua
Size (in mm)	Linear dimension along the basal plate of the largest focus
Number of foci	In all sections including basal plate

Mod Pathol 2020;33(12):2382–96
Surg Pathol Clin 2022;15(2):175-196


Basal plate myometrial fibers



Basal plate myometrial fibers
Stage 1 (with decidua)



Basal plate myometrial fibers
Stage 2 (no decidua)

4. BASAL PLATE MYOMETRIAL FIBERS

- Stage 2 associated with placenta accreta spectrum in subsequent gestations
- Stage 1 is of uncertain significance

BPMF—Reporting Parameters

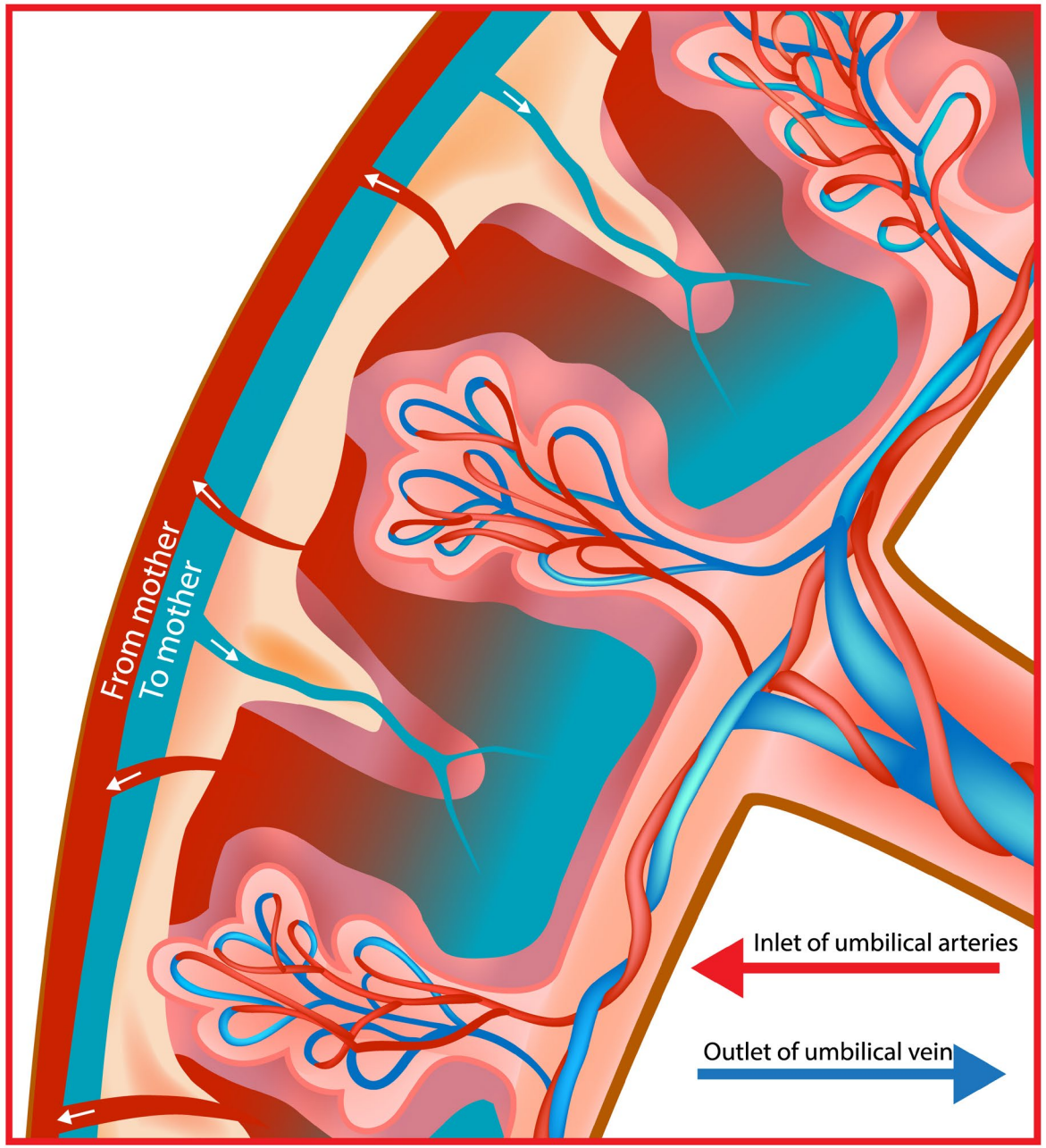
Parameter	Definition
Stage	Stage 1: With accompanying decidua Stage 2: Without decidua
Size (in mm)	Linear dimension along the basal plate of the largest focus
Number of foci	In all sections including basal plate

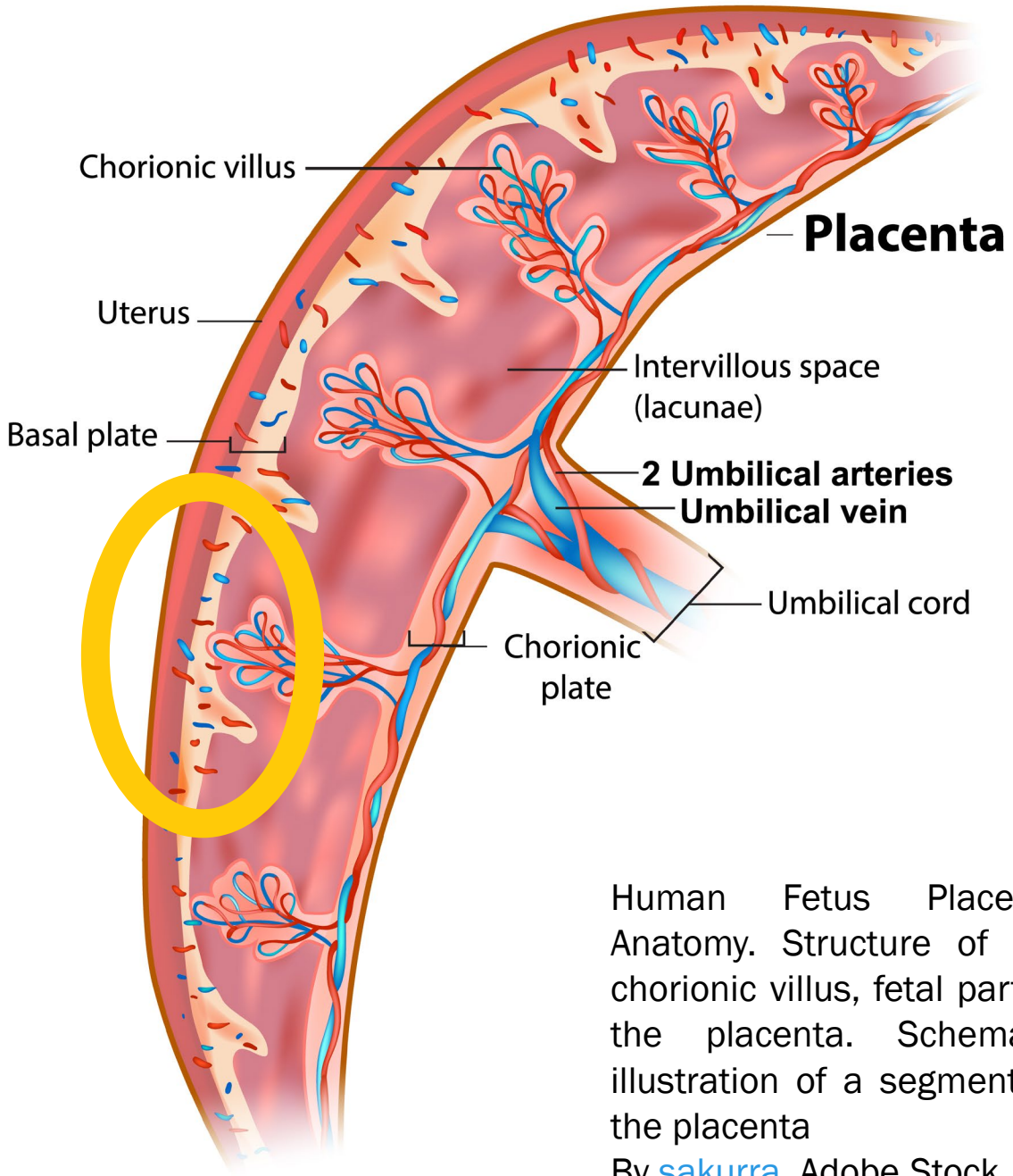
Placenta 2015;36(12):1419–24
Am J Clin Pathol 2001;116(5):703–8.

5. MASSIVE FIBRIN DEPOSITION

- Maternal floor infarction
  - *At least 25% of basal plate surface*
  - *≥ 3 mm in thickness (from basal plate)*
- Massive perivillous fibrin deposition
  - *≥ 50% of villi in one slide*
  - *≥ 30% of the gross placental volume*
- Most accept these entities as part of the same phenomenon

Redline R, Boyd T, Roberts D. Placental and Gestational Pathology. Cambridge. 2019. Chapter 17. Page 181

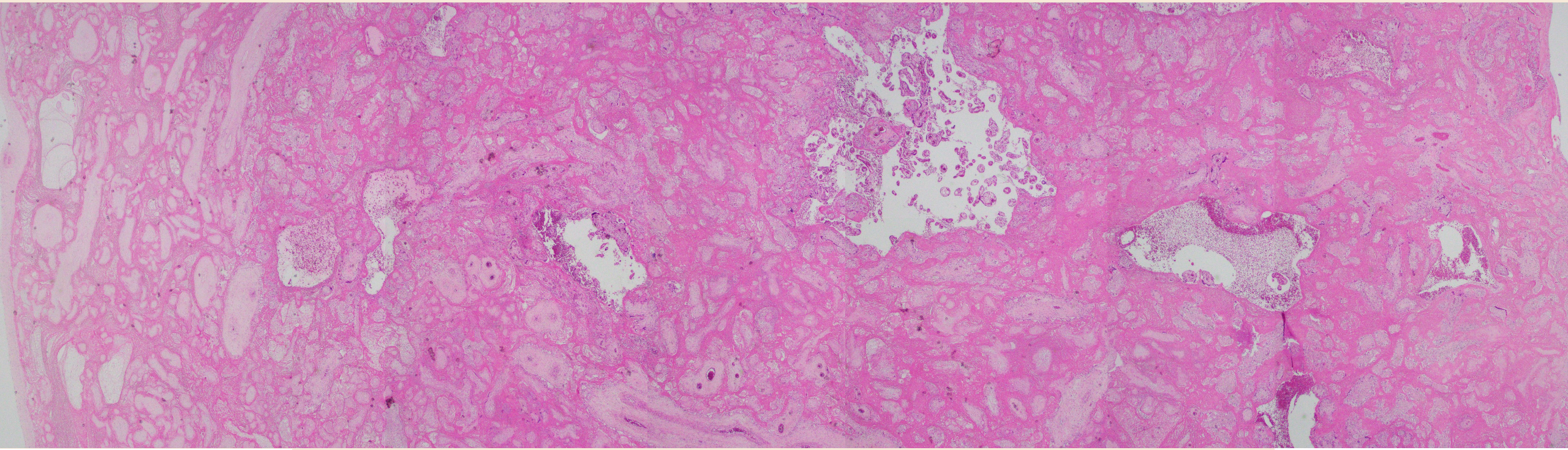




Human Fetus Placenta Anatomy. Structure of the chorionic villus, fetal part of the placenta. Schematic illustration of a segment of the placenta

By sakurra Adobe Stock

MASSIVE FIBRIN DEPOSITION

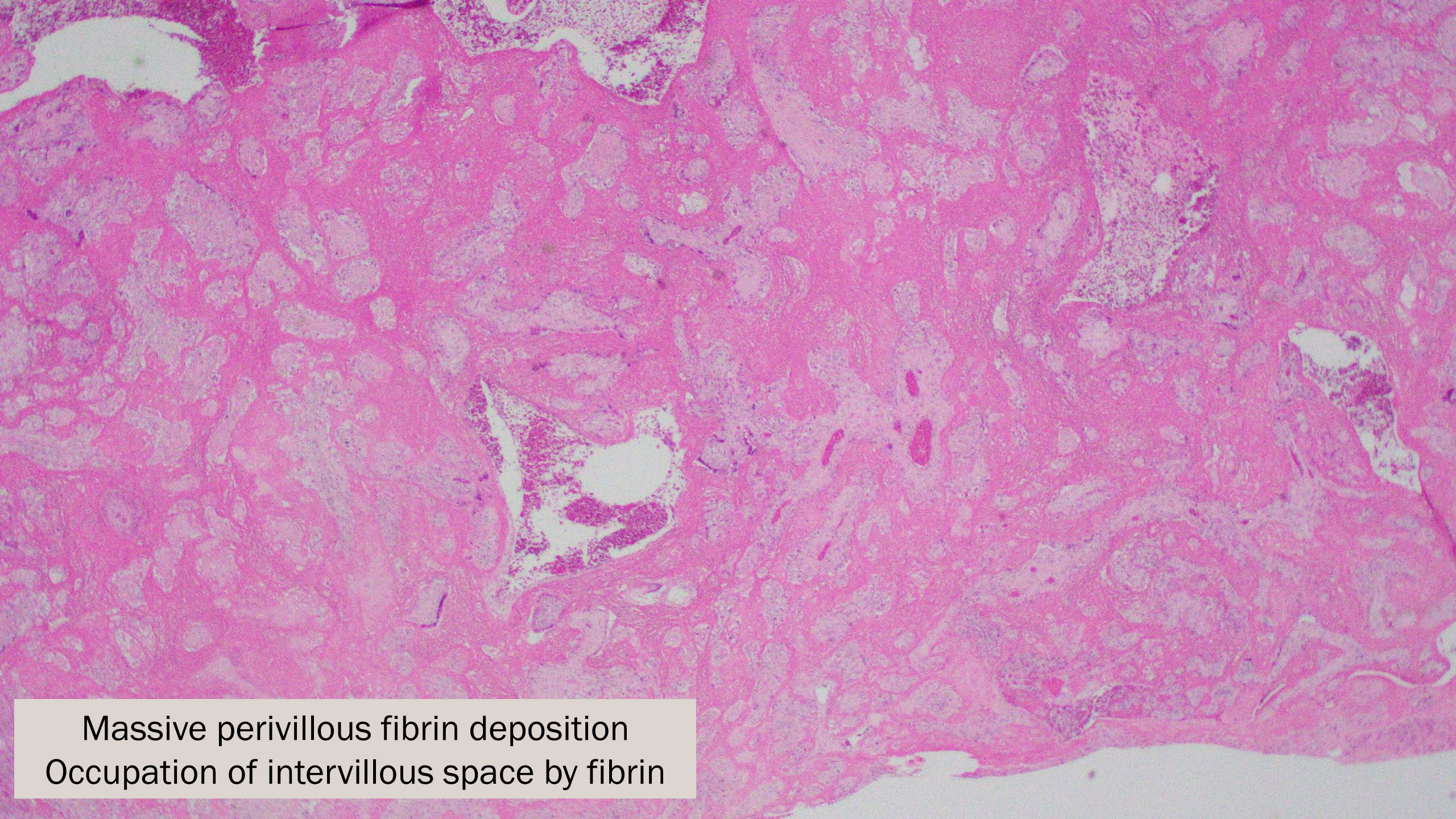


Chorionic plate

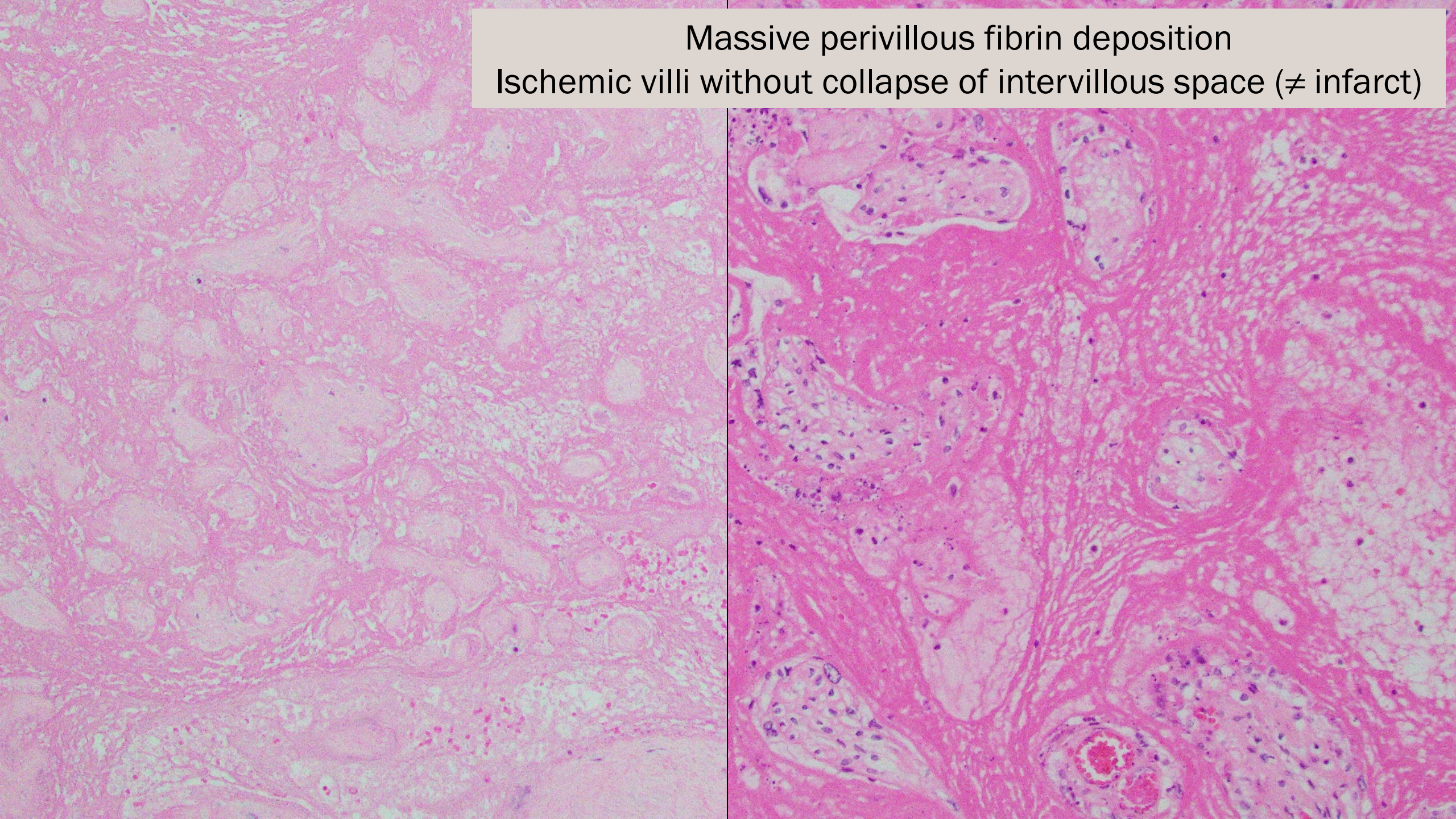
Basal plate

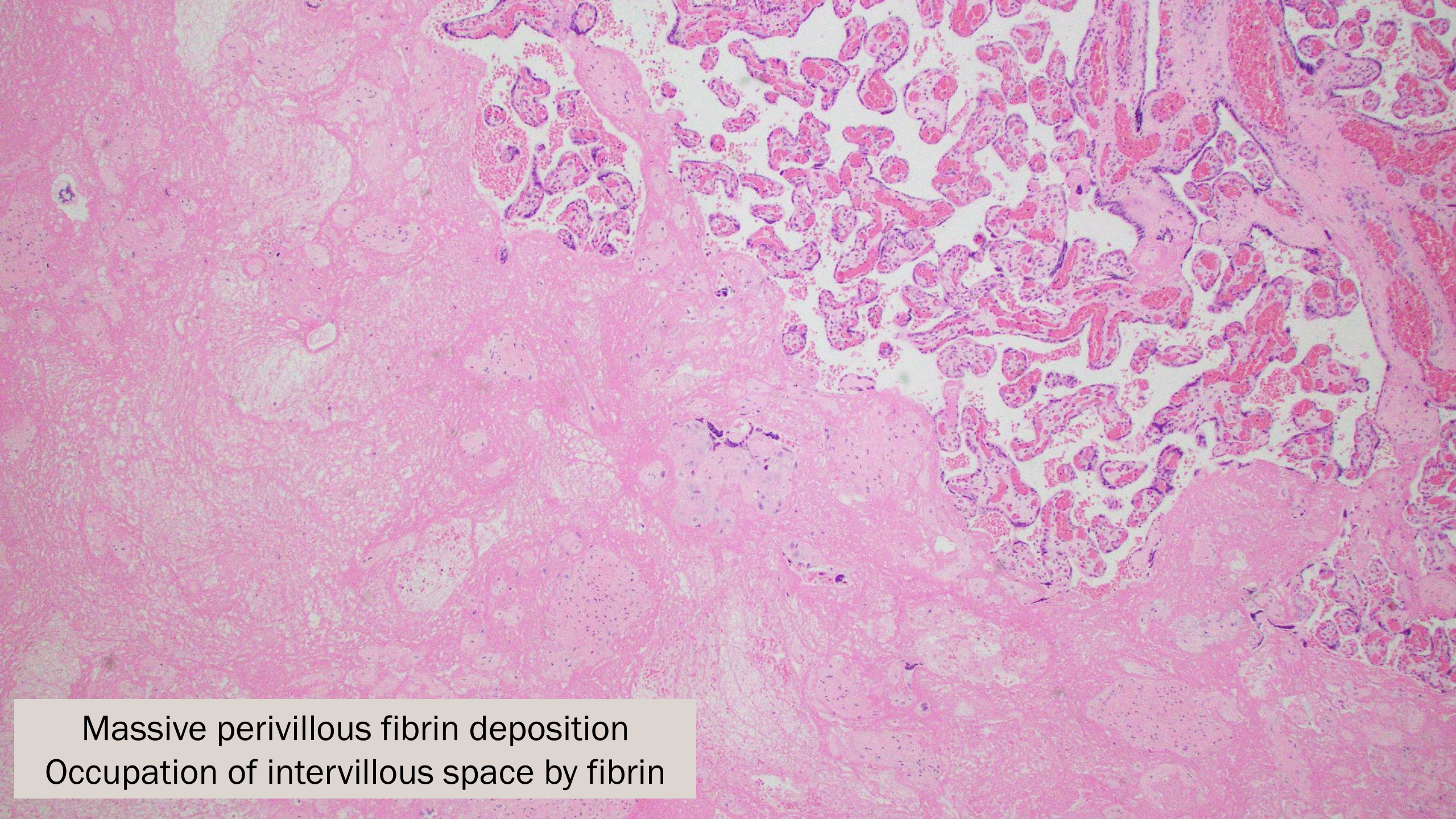
Perivillous fibrin involves basal plate

Not confined to the chorionic plate (transmural in this case)


Massive perivillous fibrin deposition
Occupation of intervillous space by fibrin

Massive perivillous fibrin deposition
Ischemic villi without collapse of intervillous space (≠ infarct)





Massive perivillous fibrin deposition
Occupation of intervillous space by fibrin

5. MASSIVE FIBRIN DEPOSITION

- Rare (incidence <0.1%)
- Unknown pathogenesis
- Association with coagulopathy
  - *Protein S deficiency*
  - *Antiphospholipid syndrome*
  - *Protein C resistance*
- Others: Multigravidity, hypertension

Pediatr Dev Pathol 2008;11:424-9
Clin Obstet Gynecol 2006;49:885-94
Pediatr Dev Pathol 2002;5:159-64
BJOG 2002;109:570-3

5. MASSIVE FIBRIN DEPOSITION

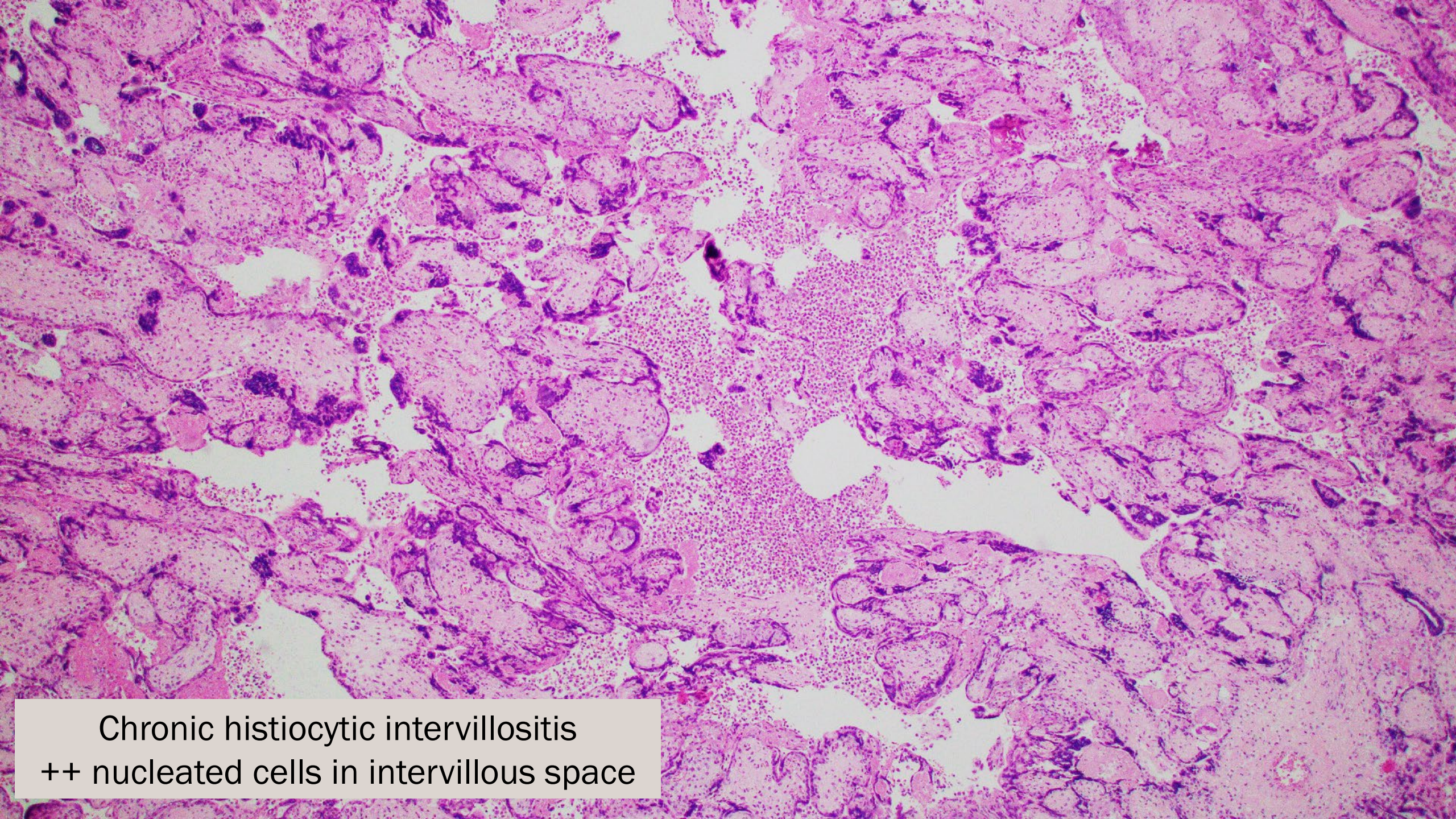
- Frequent pregnancy loss (often 2nd-3rd trimester): ~40%
- Other adverse events
  - *Intrauterine growth restriction ~69%*
  - *Premature delivery ~58%*
- Can develop rapidly (3-4 weeks)
- Recurrence rate 12-78%
- Documented success with pravastatin Tx

Am J Obstet Gynecol 1990;163:935-8
J Matern Fetal Neonatal Med 2016;29:855-62
Pediatr Dev Pathol 2002;5:159-64
Surg Pathol Clin 2013;6:101-14

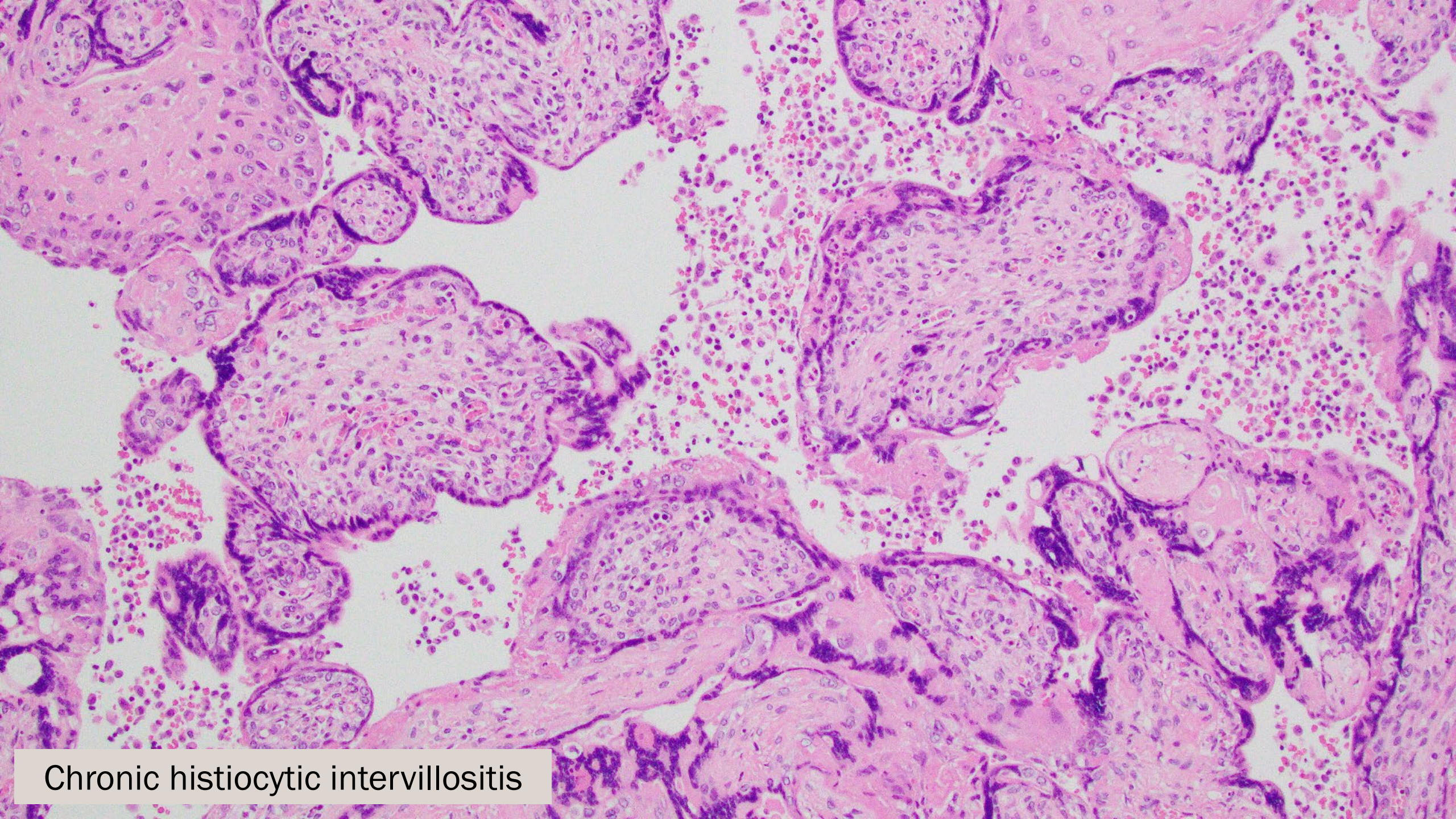
6. CHRONIC HYSTIOCYTIC INTERVILLOSITIS

- Also rare (<1%) and also idiopathic
- CD68 / PU1 positive macrophages and T cells (1:1 CD4/CD8) occupying the intervillous space
- Strong association with maternal autoantibodies and autoimmune disease
  - *Adaptive immune response to paternally derived antigens*
- Other associations: pre-eclampsia, MVM, aneuploidy

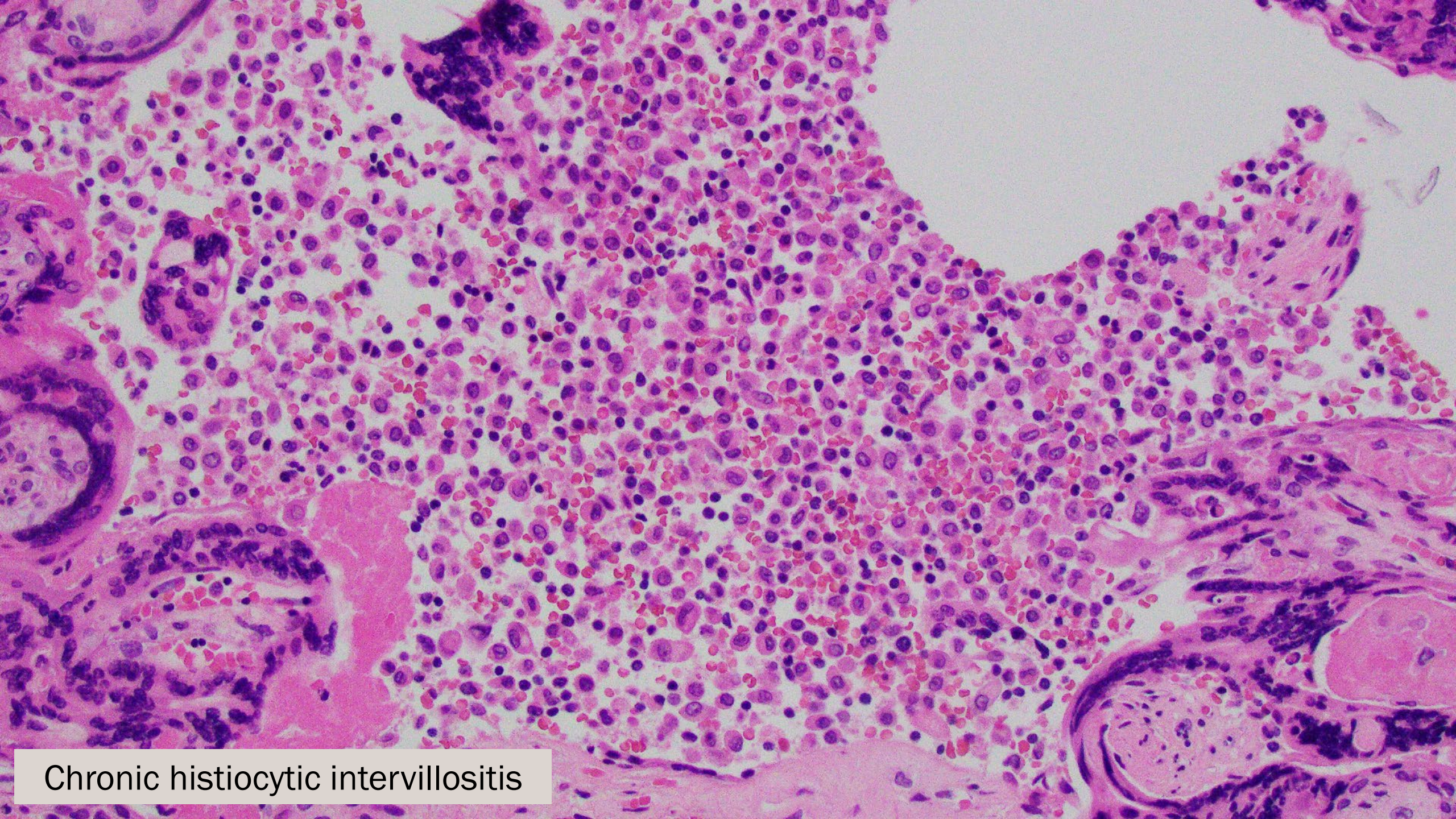
BMJ Case Rep 2017;doi:10.1136/bcr-2016-217886
Arch Gynecol Obstet 2015;291:1229-36
Placenta 2013;34:149-54
Autoimmunity 2015;48:40-5



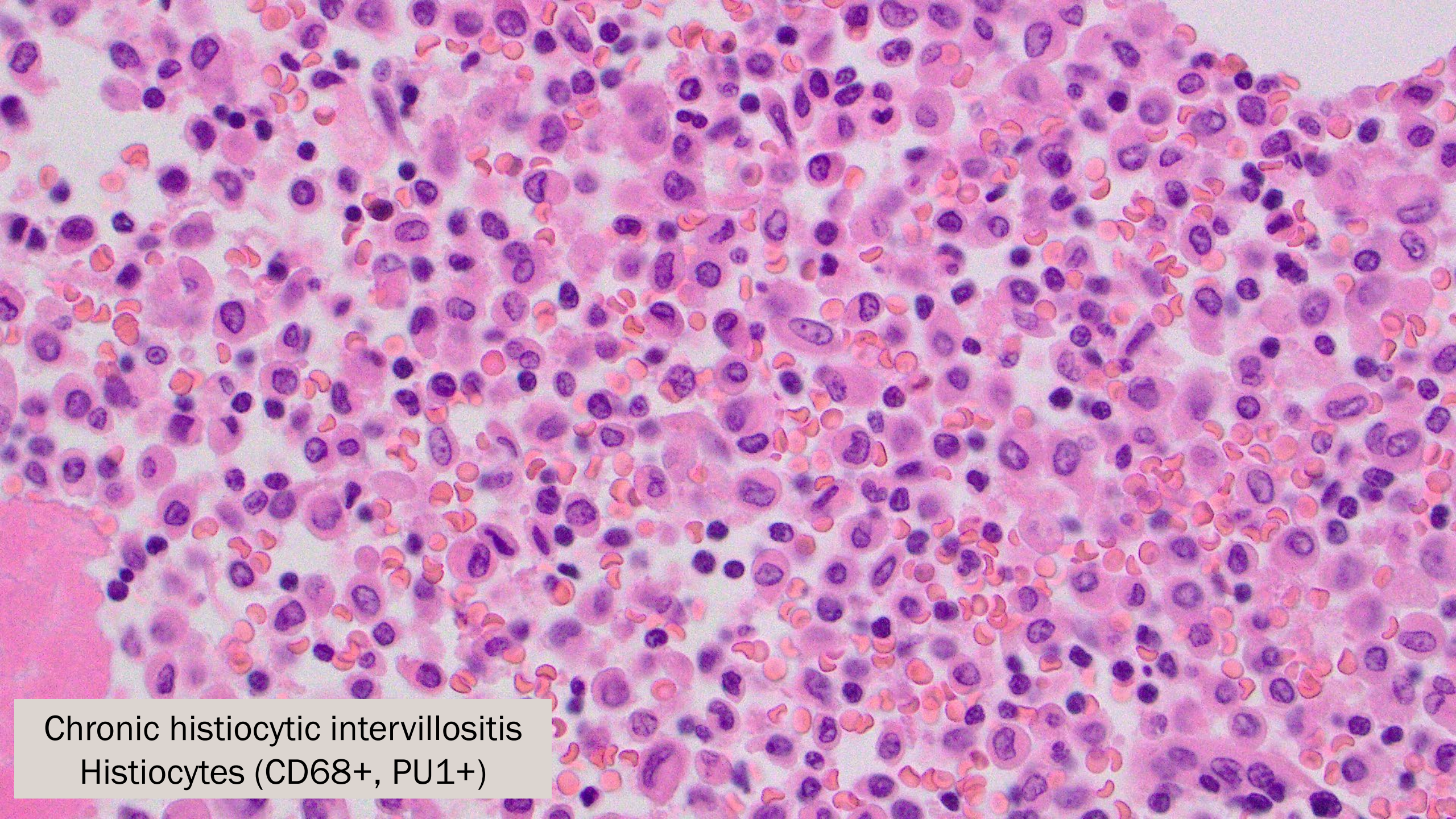
Chronic histiocytic intervillositis
++ nucleated cells in intervillous space



Chronic histiocytic intervillositis



Chronic histiocytic intervillositis


Chronic histiocytic intervillositis
Histiocytes (CD68+, PU1+)

6. CHRONIC HYSTIOCYTIC INTERVILLOSITIS

- Overall perinatal mortality is 80%
- After viability (22 weeks)
  - *Stillbirth ~46%*
  - *Intrauterine growth restriction ~62%*
- Recurrence rate 67-100%
- Immune modulating therapy decreases recurrence to ~50%
  - *Corticosteroids, aspirin, LMW heparin, hydroxychloroquine*

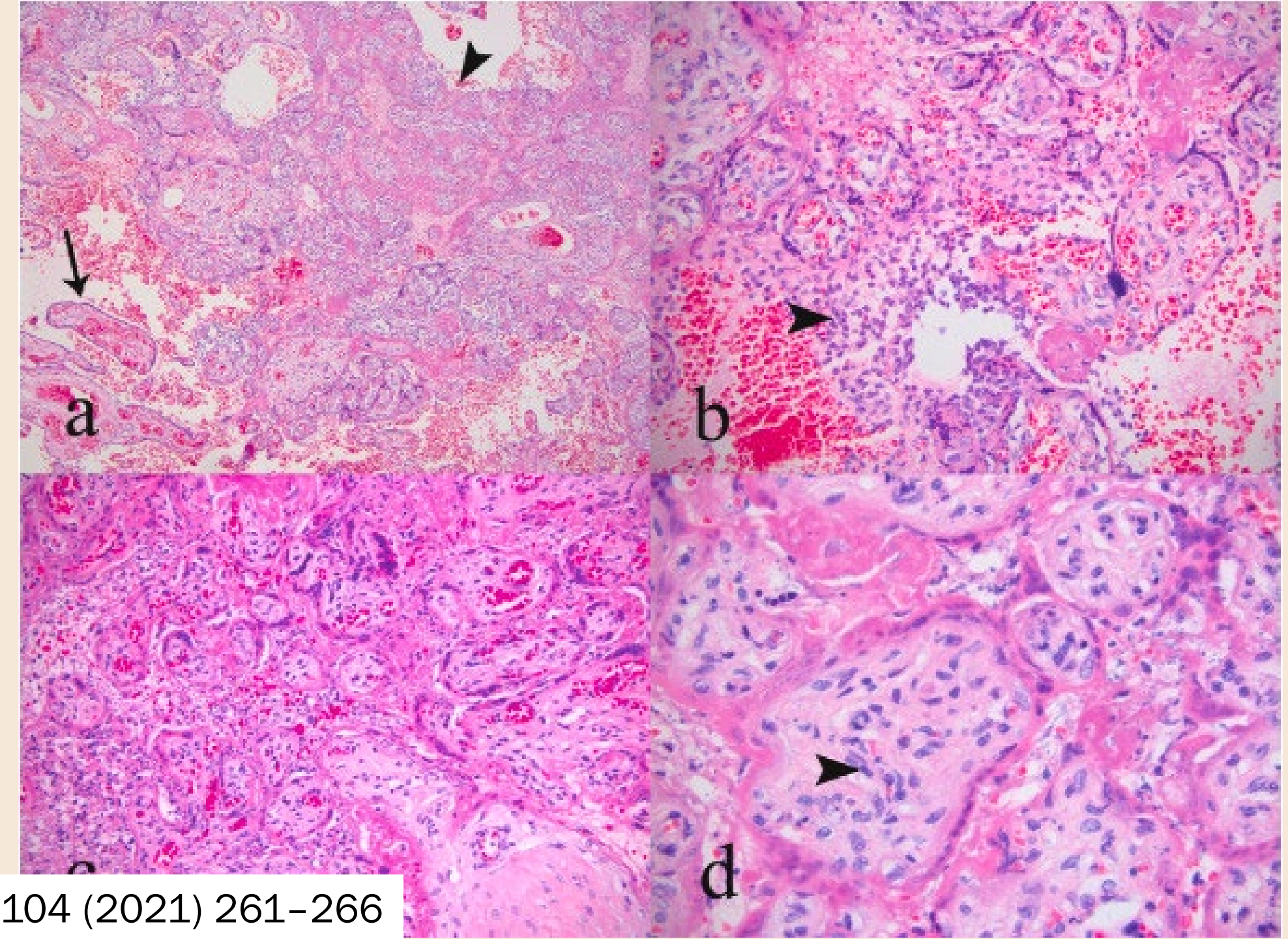
Placenta 2011;32:140-5
BMJ Case Rep 2017;doi:10.1136/bcr-2016-217886
Placenta 2010;31:1106-10
Autoimmunity 2015;48:40-5

DIFFERENTIAL DIAGNOSIS

- Malarial infection during pregnancy
  - *Villous trophoblast necrosis*
  - *Acute inflammation*
  - *Fibrin admixed with black hematozoin pigment*
  - *Trophozoites within red blood cells*
- SARS-2CoV placentitis
  - *Villous trophoblast necrosis*
  - *Extensive perivillous fibrin deposition & intervillous space collapse*

Redline R, Boyd T, Roberts D. Placental and Gestational Pathology. Cambridge. 2019. Chapter 17. Page 181

Mod Pathol 2021;34(9):1704–9
Arch Pathol Lab Med 2021;145(11):1341–9

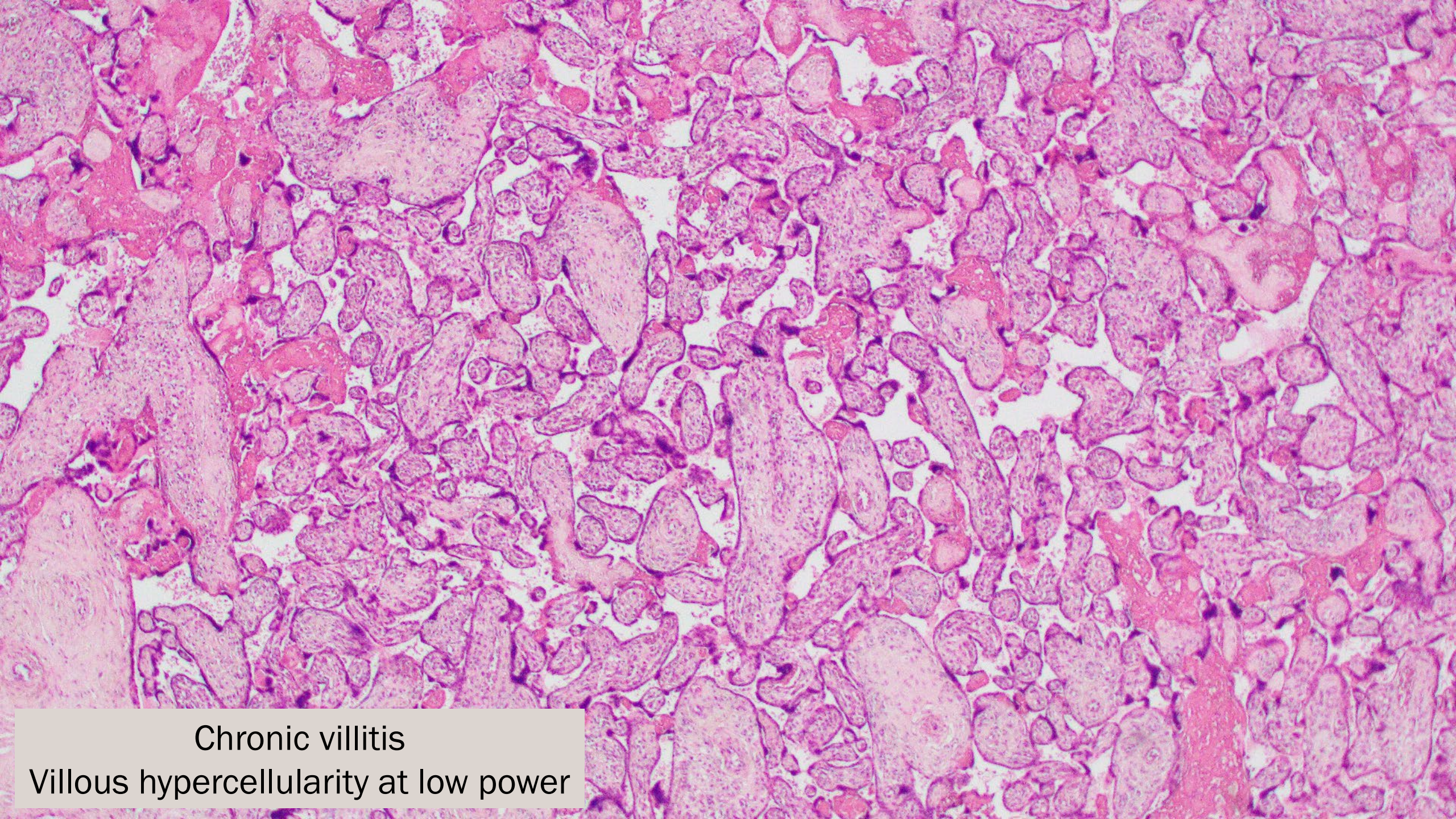


Placenta 104 (2021) 261–266

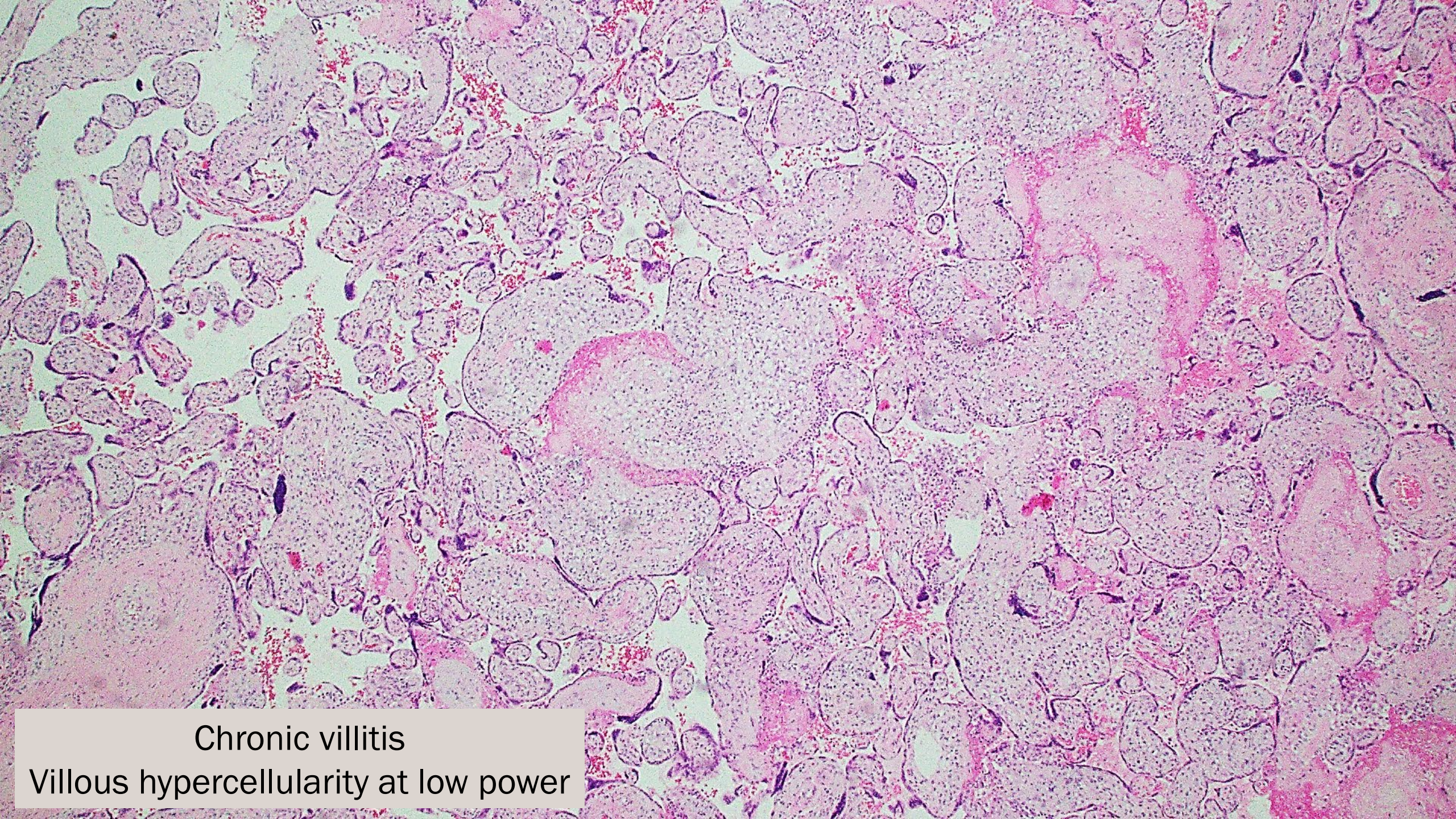
7. VILLITIS OF UNKNOWN ETIOLOGY

- Chronic (lymphoplasmacytic) infiltrate in chorionic villi *in the absence of an identifiable infectious agent*
- Often seen along with
  - *Eosinophilic / T-cell chorionic vasculitis (ETCV)*
  - *Chronic deciduitis (plasma cells in decidua)*
  - *Chronic chorioamnionitis (lymphocytes in membranes)*
  - *Chronic intervillositis*

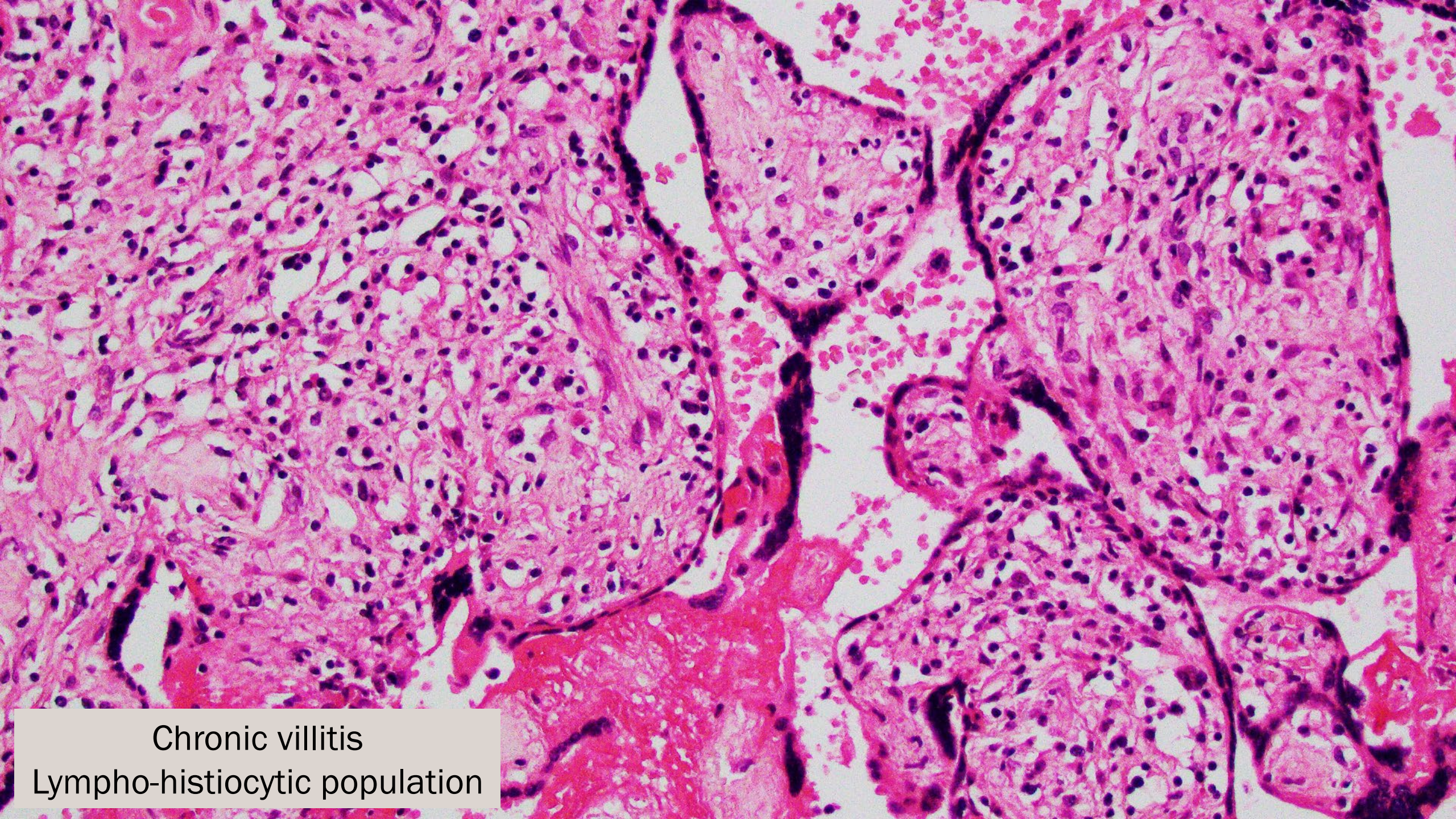
Surg Pathol Clin 2022;15(2):175-196

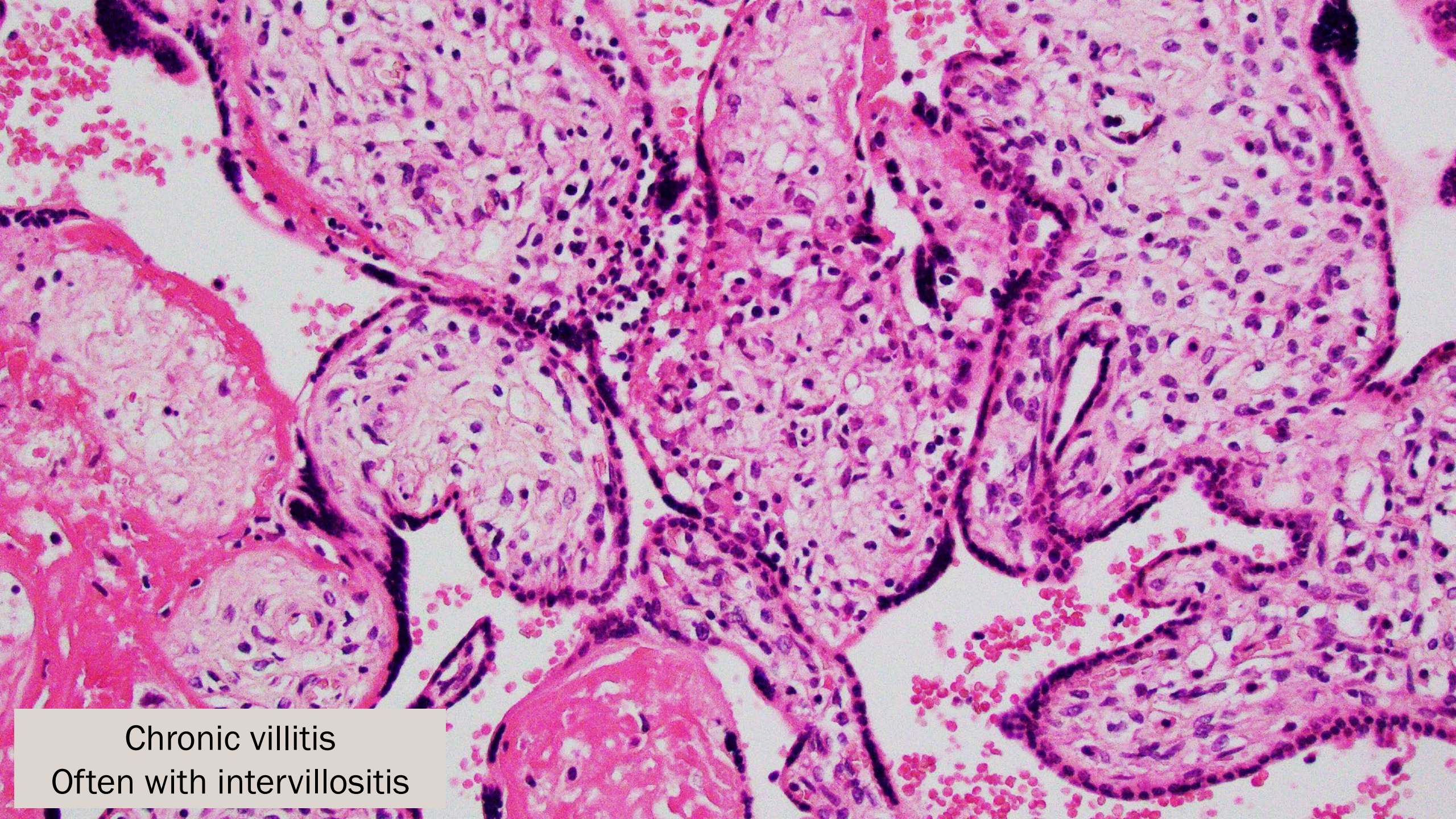


Chronic villitis
Villous hypercellularity at low power



Chronic villitis
Villous hypercellularity at low power


Chronic villitis
Lympho-histiocytic population


Chronic villitis
Often with intervillositis

7. VILLITIS OF UNKNOWN ETIOLOGY

- Maternal inflammatory response against fetal allo-antigens
  - ↑↑↑ *incidence in egg donor pregnancies and in multigravid women*
  - *Recurrence rate 15-50%*
- Maternal and/or fetal immune response to infection
  - *Seasonal variation in incidence*
  - *Viral detection in 65% VUE placentas vs 35% controls*
    - HSV in 50% VUE vs 10% controls
  - *Chronic villitis is often the hallmark of hematogenous infection (TORCH, COVID-19)*

Hum Pathol 2007;38(10):1439–46
Placenta 2021;112:135–40
Placenta 2021;107:24–30
Pediatr Dev Pathol 2020;23(4):253–9

7. VILLITIS OF UNKNOWN ETIOLOGY

- Low-grade = >1 foci*, each less than 10 contiguous villi
  - *Focal = Villitis seen in one slide only*
  - *Multifocal = Villitis seen in more than one slide*
- High-grade = >1 foci*, at least one ≥10 contiguous villi
  - *Patchy = ≤30% of all distal villi involved*
  - *Diffuse = >30% of all distal villi involved*

* For one focus, use VUE-NOS

Arch Pathol Lab Med 2016;140(7):698-713

VUE - ISSUES

- Low-grade VUE is common in placentas from normal gestations
- Romero *et al*
  - *30% had 1+ chronic inflammatory lesion(s)*
  - *Chronic deciduitis (20%), low-grade VUE (19%) & chronic chorioamnionitis (13%)*
  - *Versus high-grade VUE (1%) & intervillositis (<1%)*

J Perinat Med 2018;46(6):613–30

DDX – INFECTIOUS VILLITIS

- It often is diffuse / multifocal
- Obliteration of villous vessels (downstream AVV)
- Unusual infiltrate
  - *Histiocytic* *Giant cells* *Neutrophilic*
  - *Plasma cell* *Granulomatous*
- Trophoblast necrosis
- Nucleated red blood cells in fetal circulation
- If you're lucky, viral inclusions or trophozoites IHC

Redline R, Boyd T, Roberts D. Placental and Gestational Pathology. Cambridge. 2019. Chapter 17. Page 181

PLACENTAL LESIONS C

- Placental lesions that require an immediate intervention
  8. *Ascending intrauterine infection*
  9. *BONUS CASE*

9. ASCENDING UTERINE INFECTION

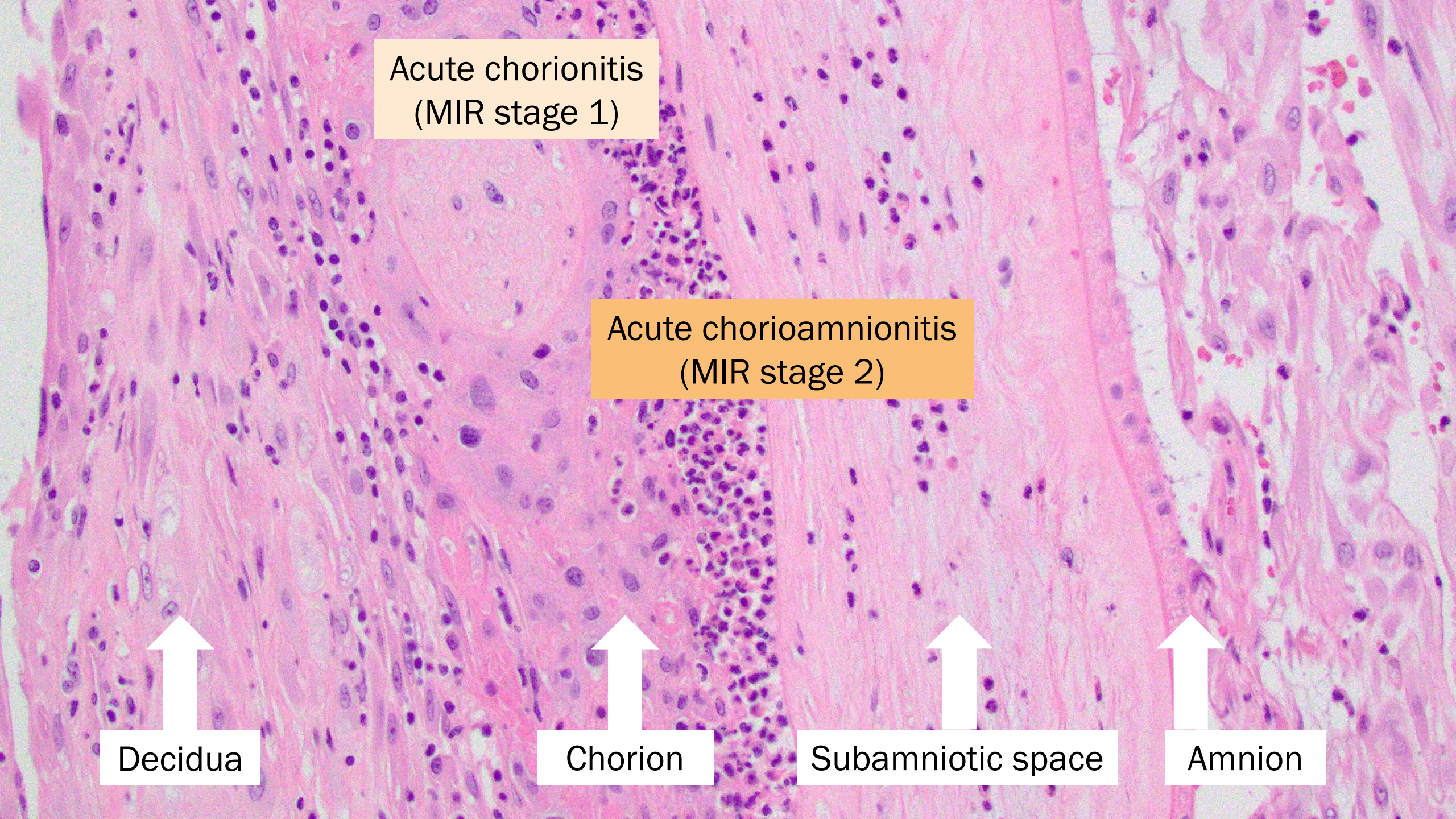
- Inflammation of placental structures closer to the amniotic sac (chorionic membranes & plate, umbilical cord)
- Amsterdam endorses a stage / grade classification schema
  - *Stage is predominantly determined by the anatomic compartment(s) involved*
  - *Grade is determined by the severity of the inflammation*

Arch Pathol Lab Med 2016;140(7):698-713

ASCENDING INFECTION - STAGING AND GRADING

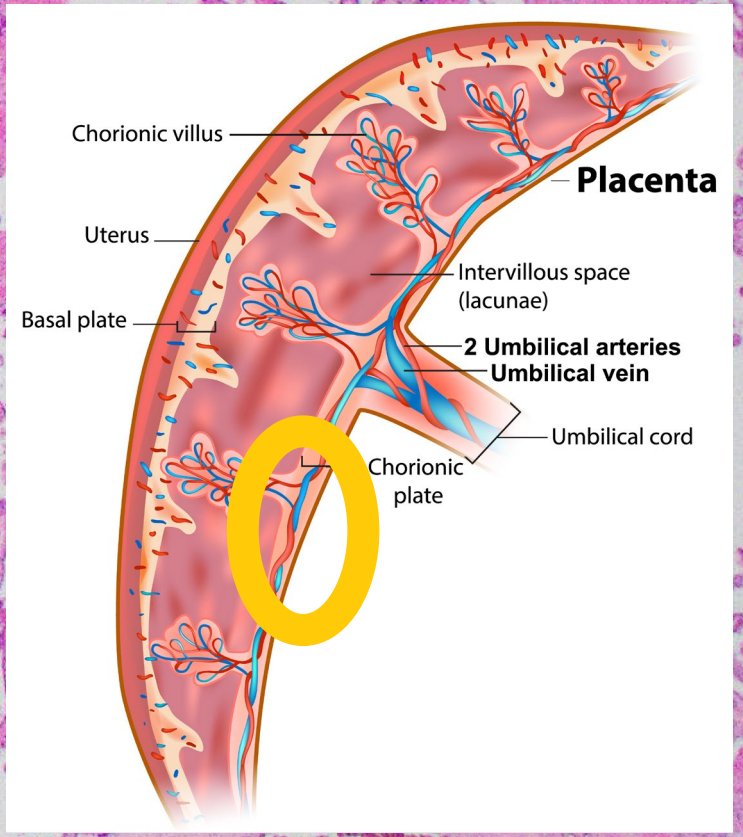
Stage	Grade
Maternal Response	
1 - Acute subchorionitis or chorionitis	1 - Not severe as defined
2 - Acute chorioamnionitis	2 - Severe (confluent neutrophils or subchorionic microabcesses)
3 - Necrotizing chorioamnionitis	
Fetal Response	
1 - Chorionic vasculitis or umbilical phlebitis	1- Not severe as defined
2 - Umbilical vein and one or more arteries	2 - Severe (confluent intramural neutrophils with smooth muscle loss)
3 - Necrotizing funisitis	

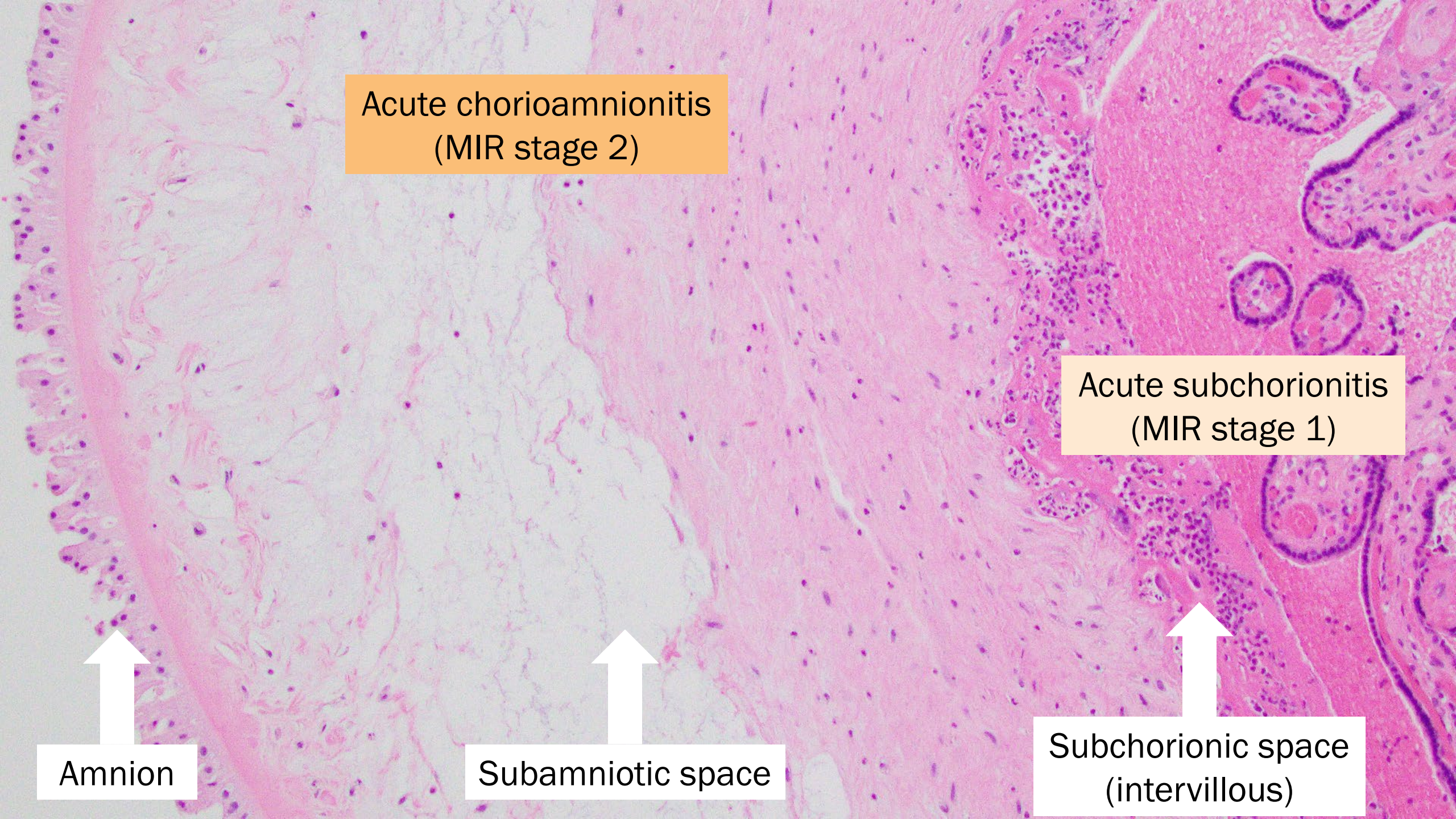
Arch Pathol Lab Med 2016;140(7):698-713

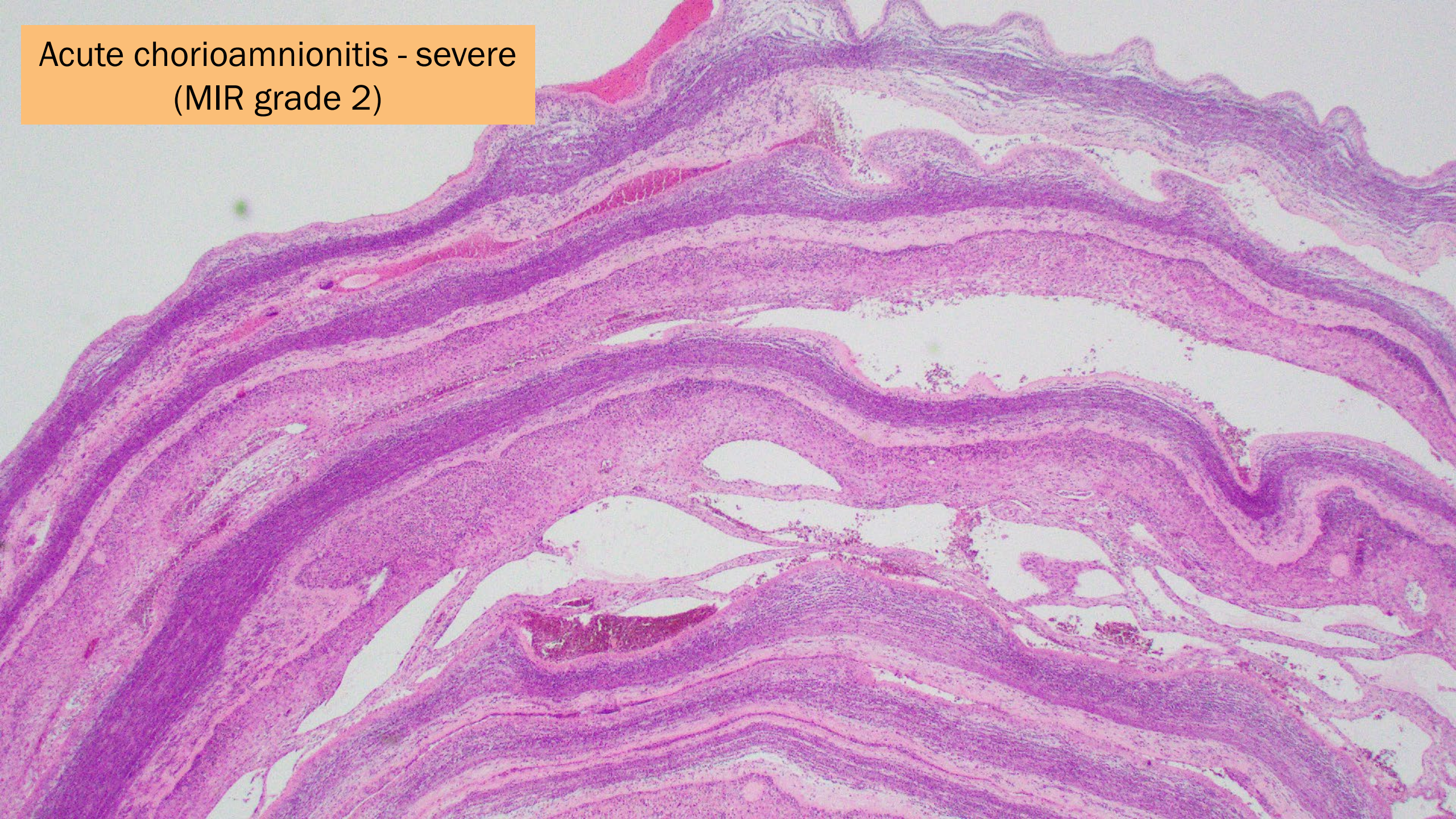

Acute chorionitis
(MIR stage 1)
Acute chorioamnionitis
(MIR stage 2)
Decidua
Chorion
Subamniotic space
Amnion

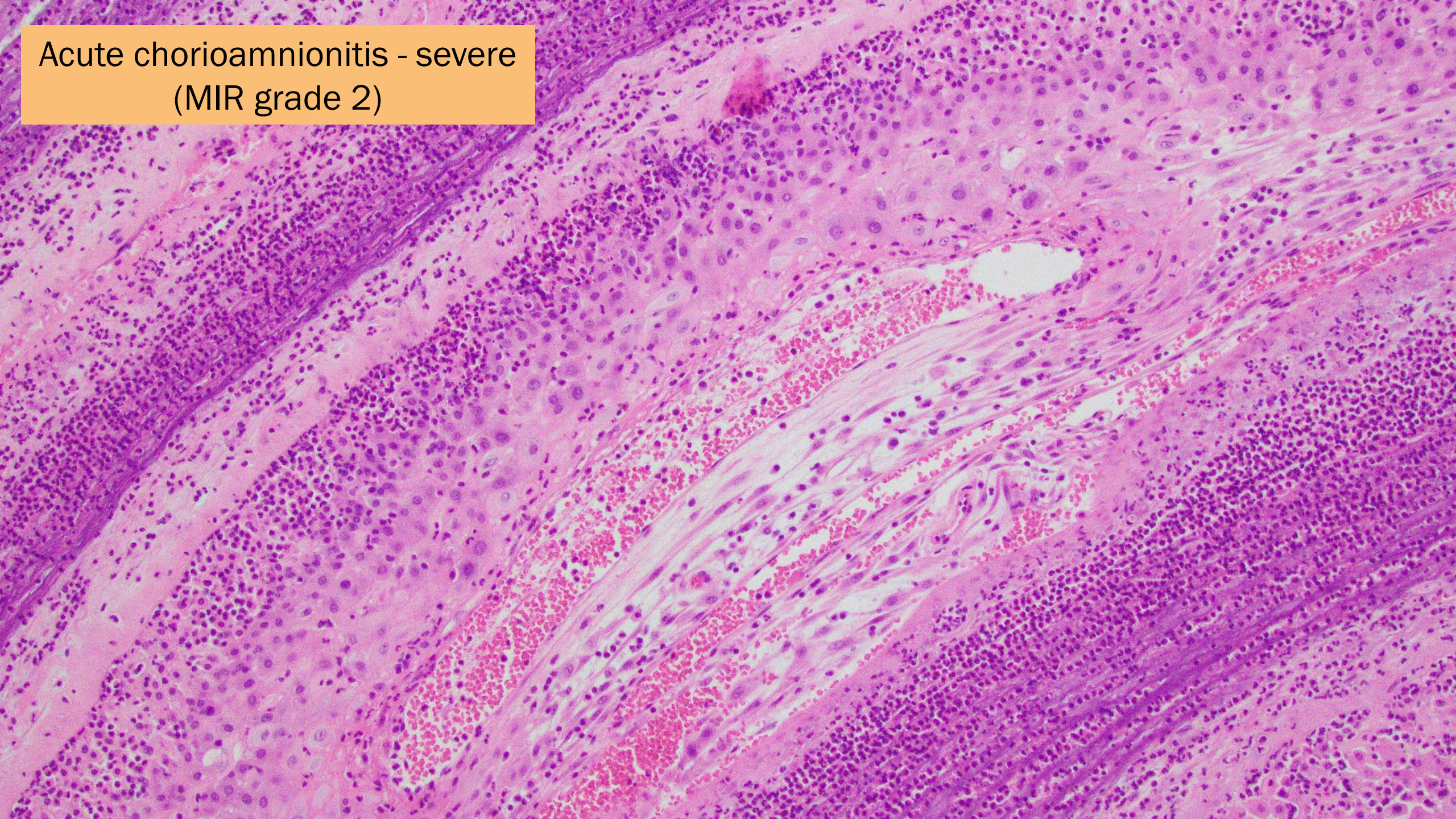
Chorionic plate (fetal side)

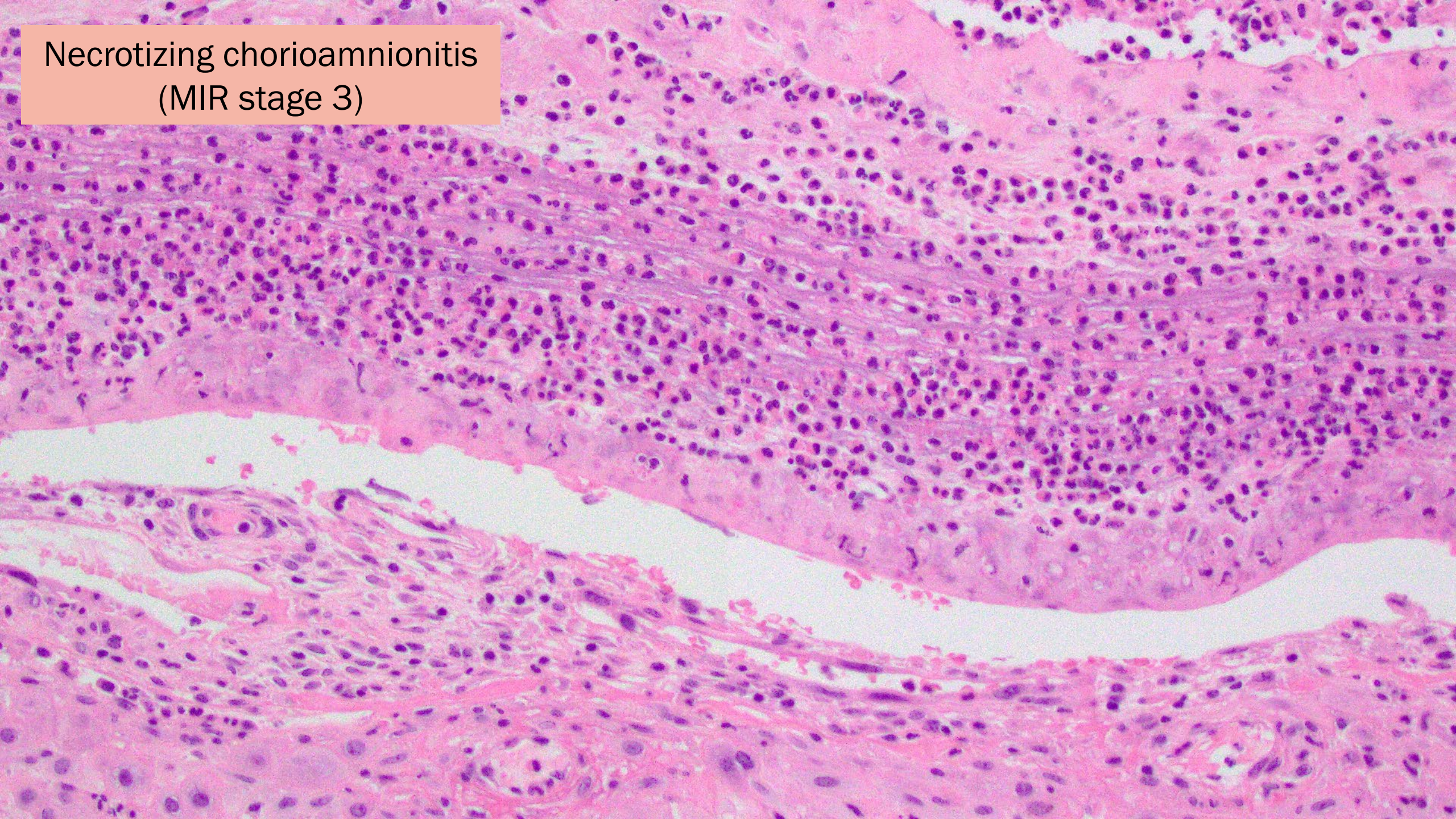
Parenchyma

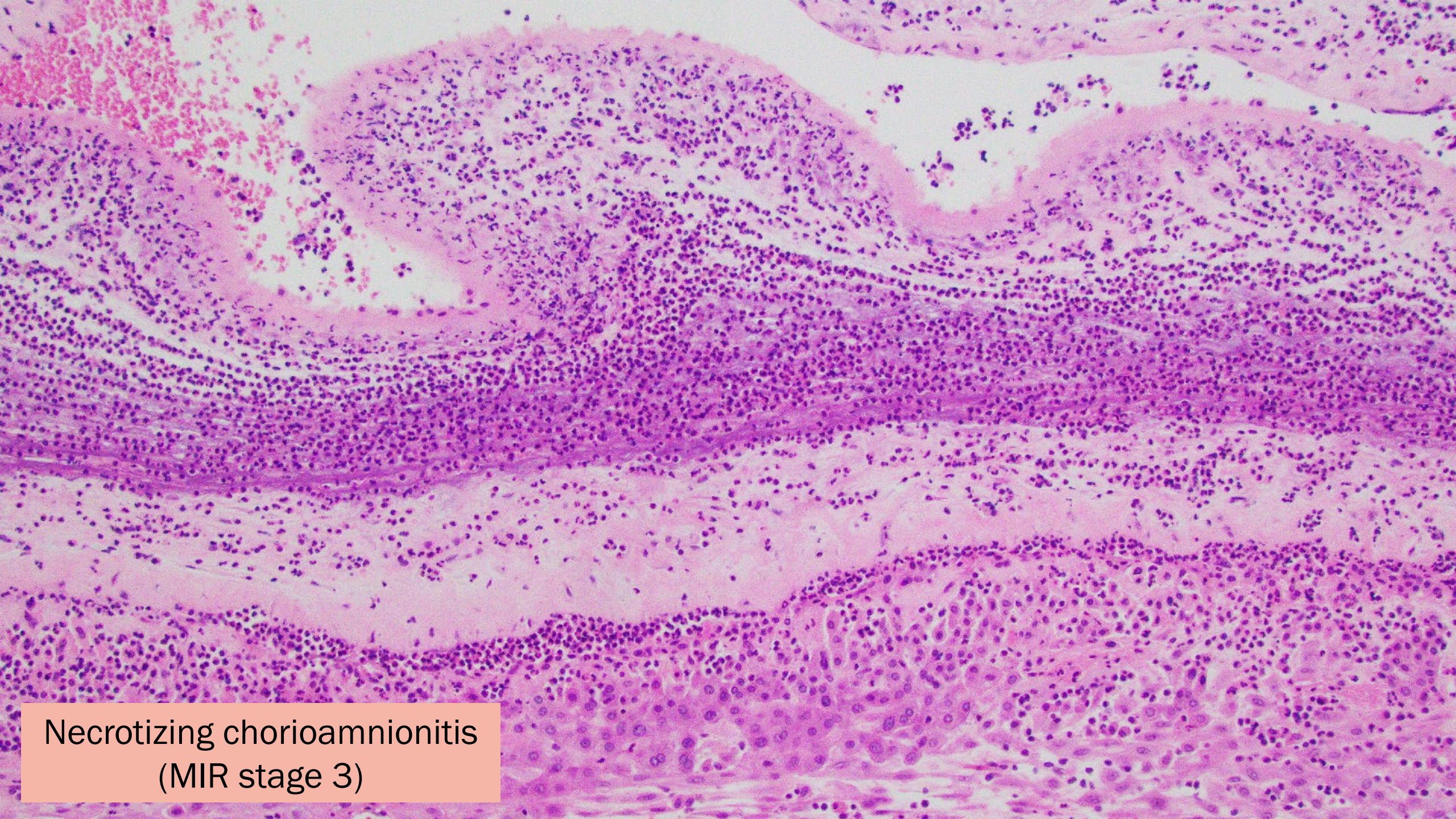


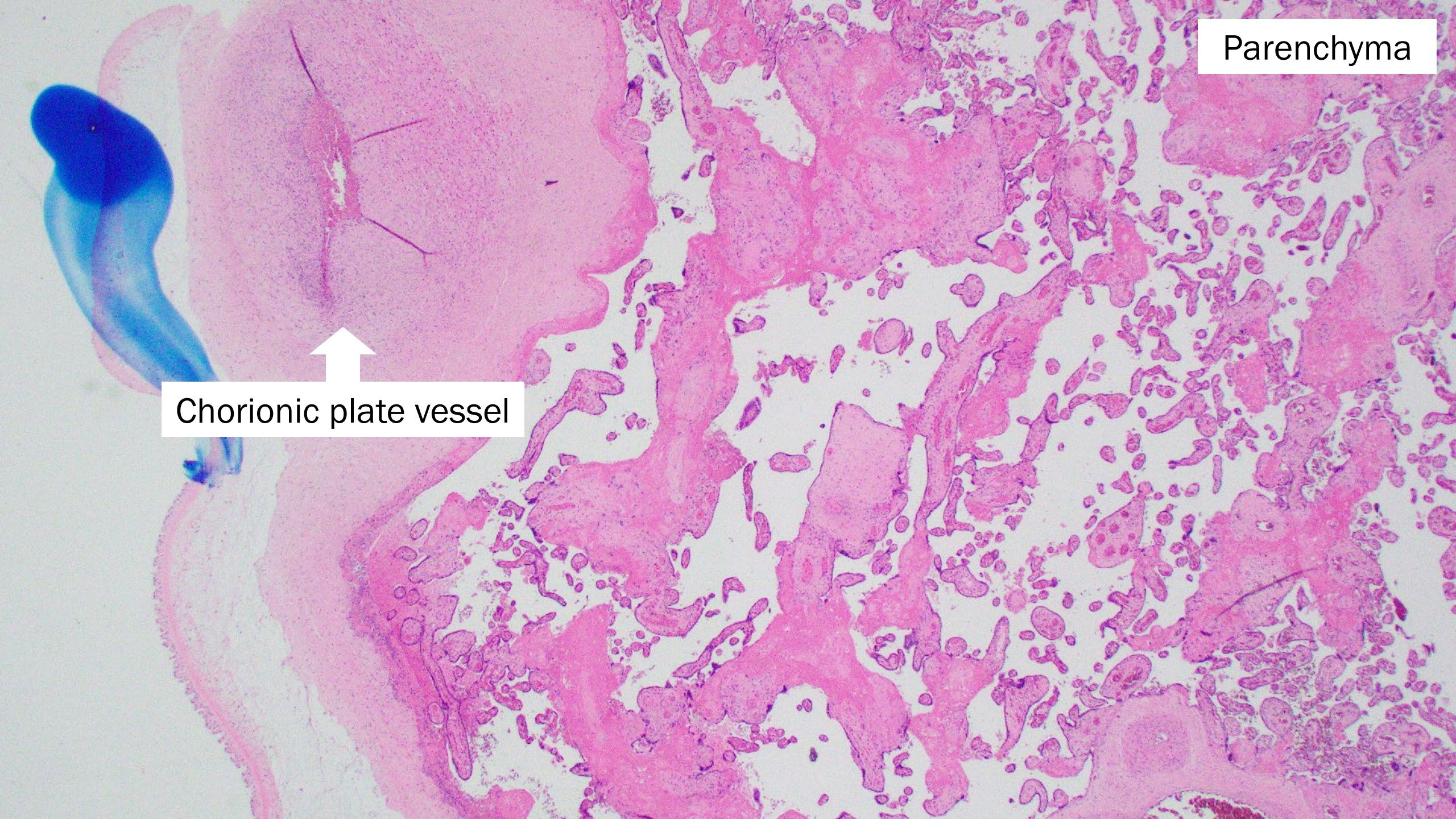

Acute chorioamnionitis
(MIR stage 2)
Acute subchorionitis
(MIR stage 1)
Amnion
Subamniotic space
Subchorionic space
(intervillous)

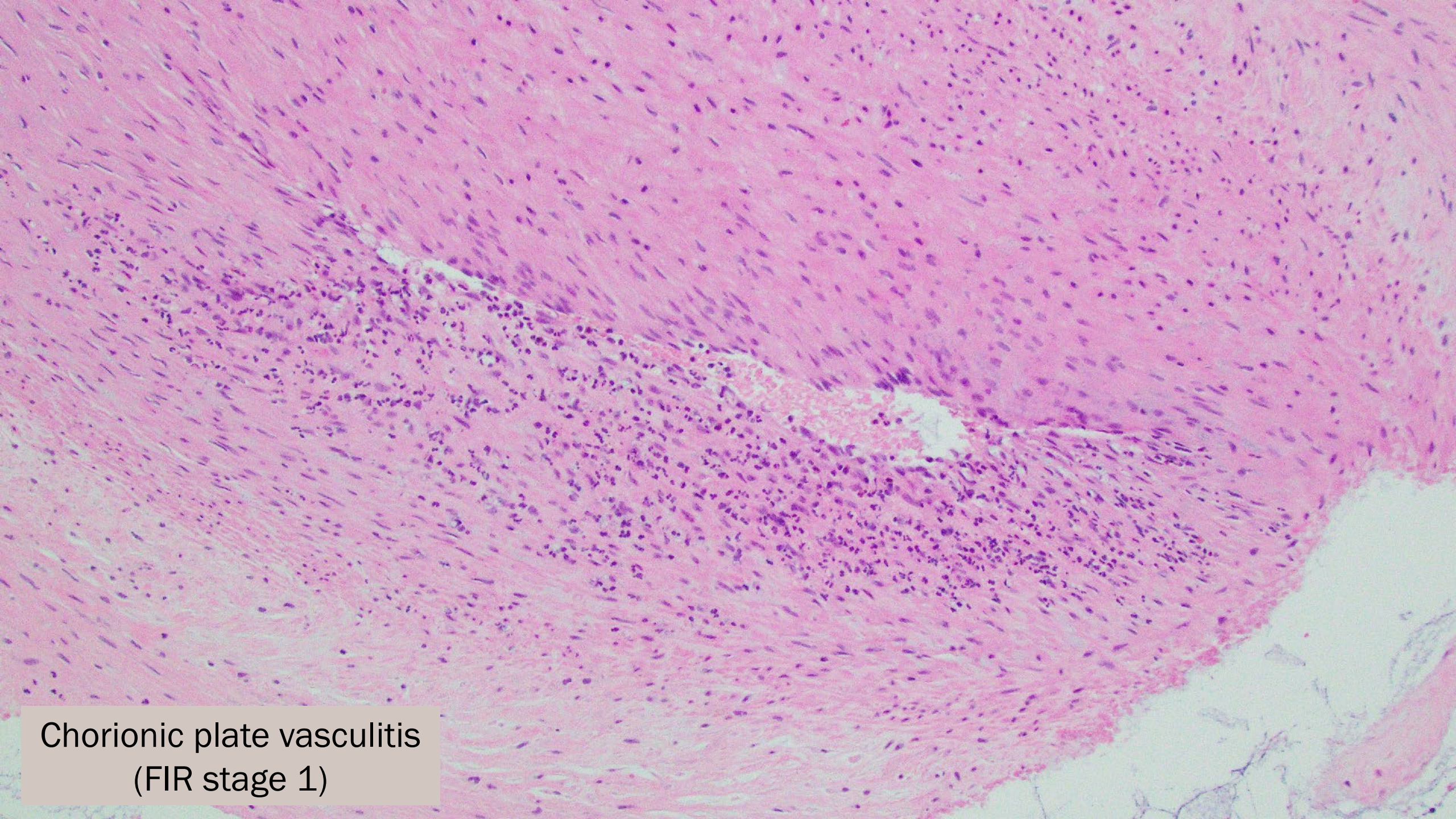

Acute chorioamnionitis - severe
(MIR grade 2)

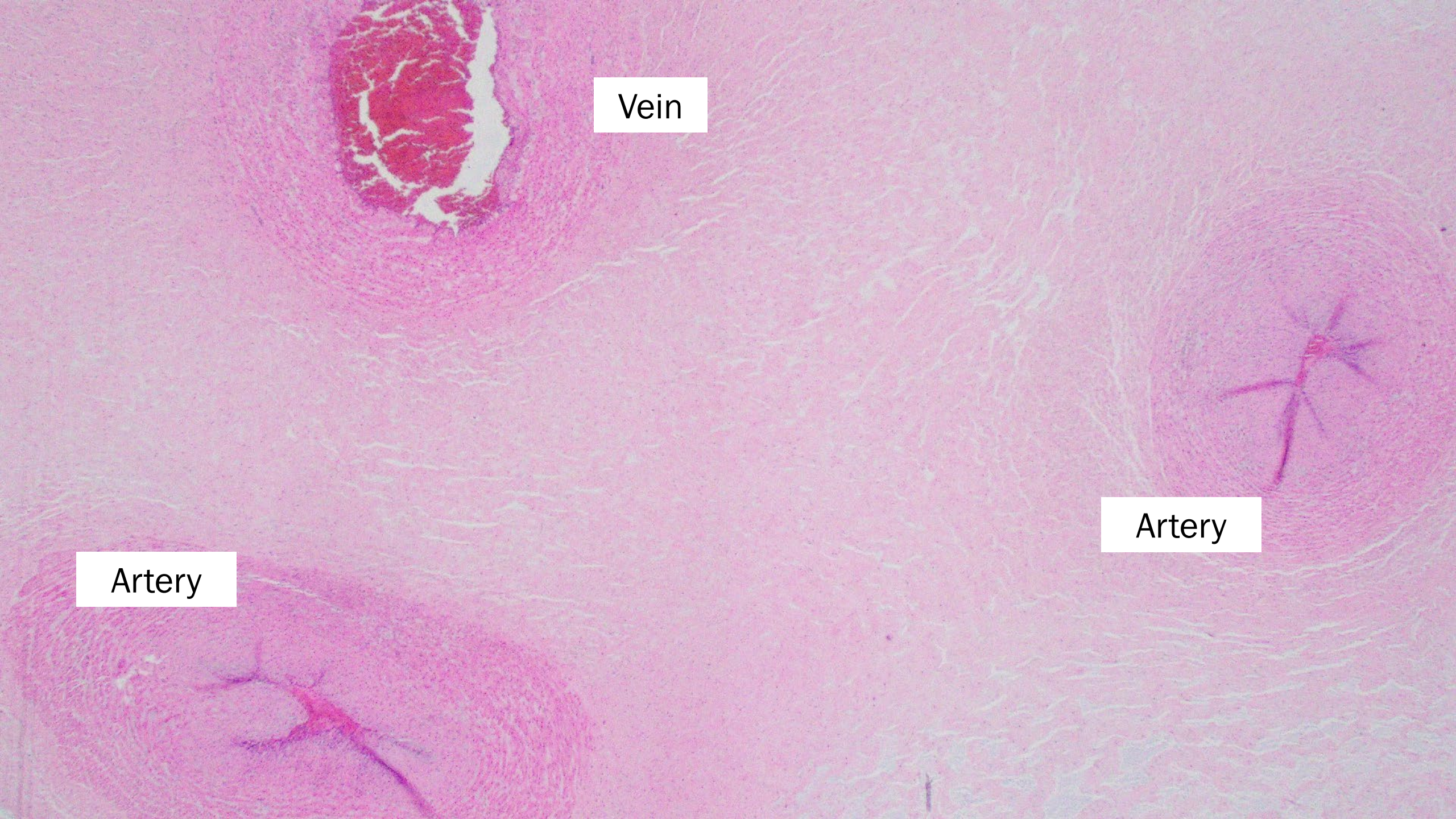

Acute chorioamnionitis - severe
(MIR grade 2)

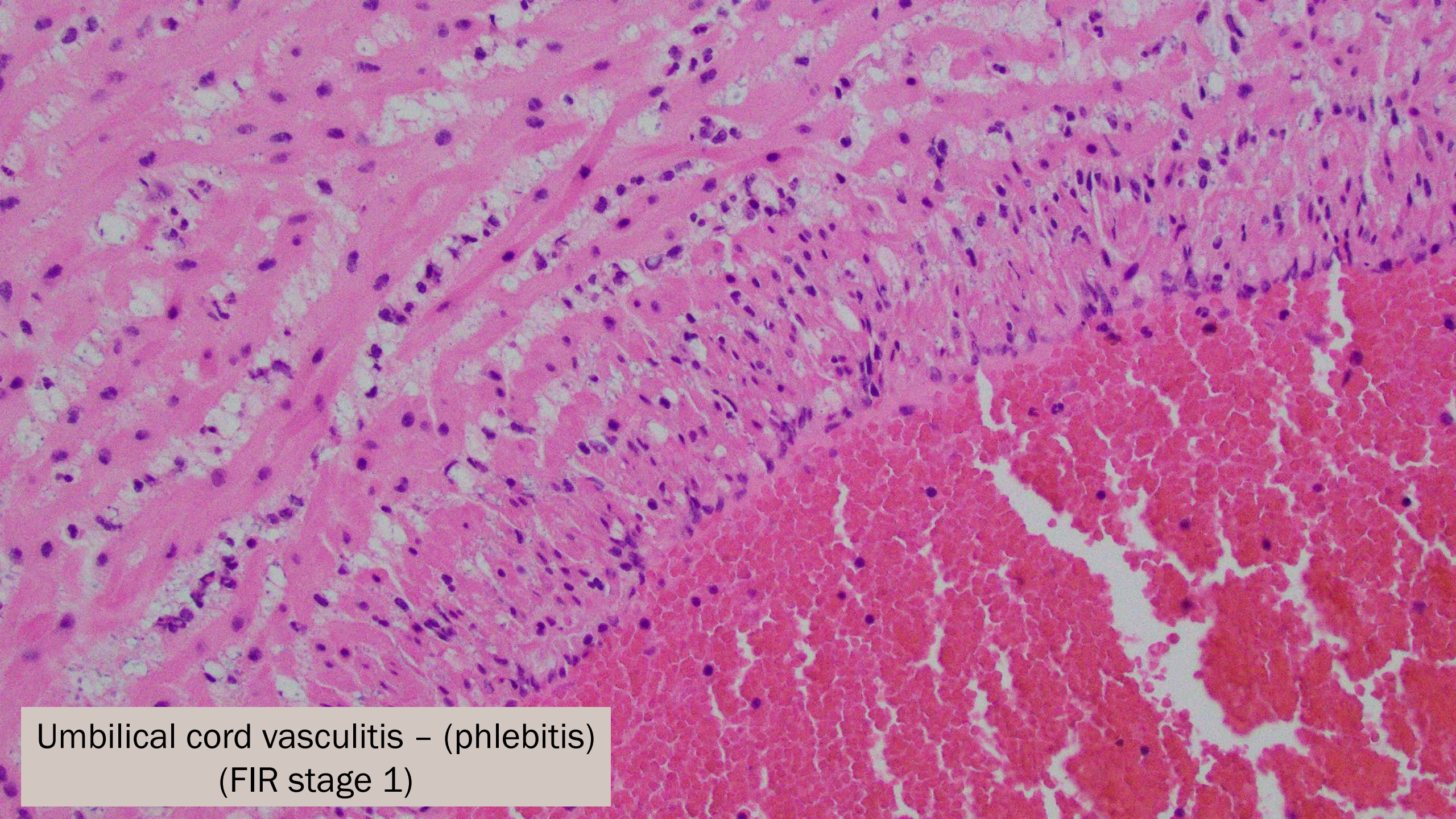

Necrotizing chorioamnionitis
(MIR stage 3)


Necrotizing chorioamnionitis
(MIR stage 3)

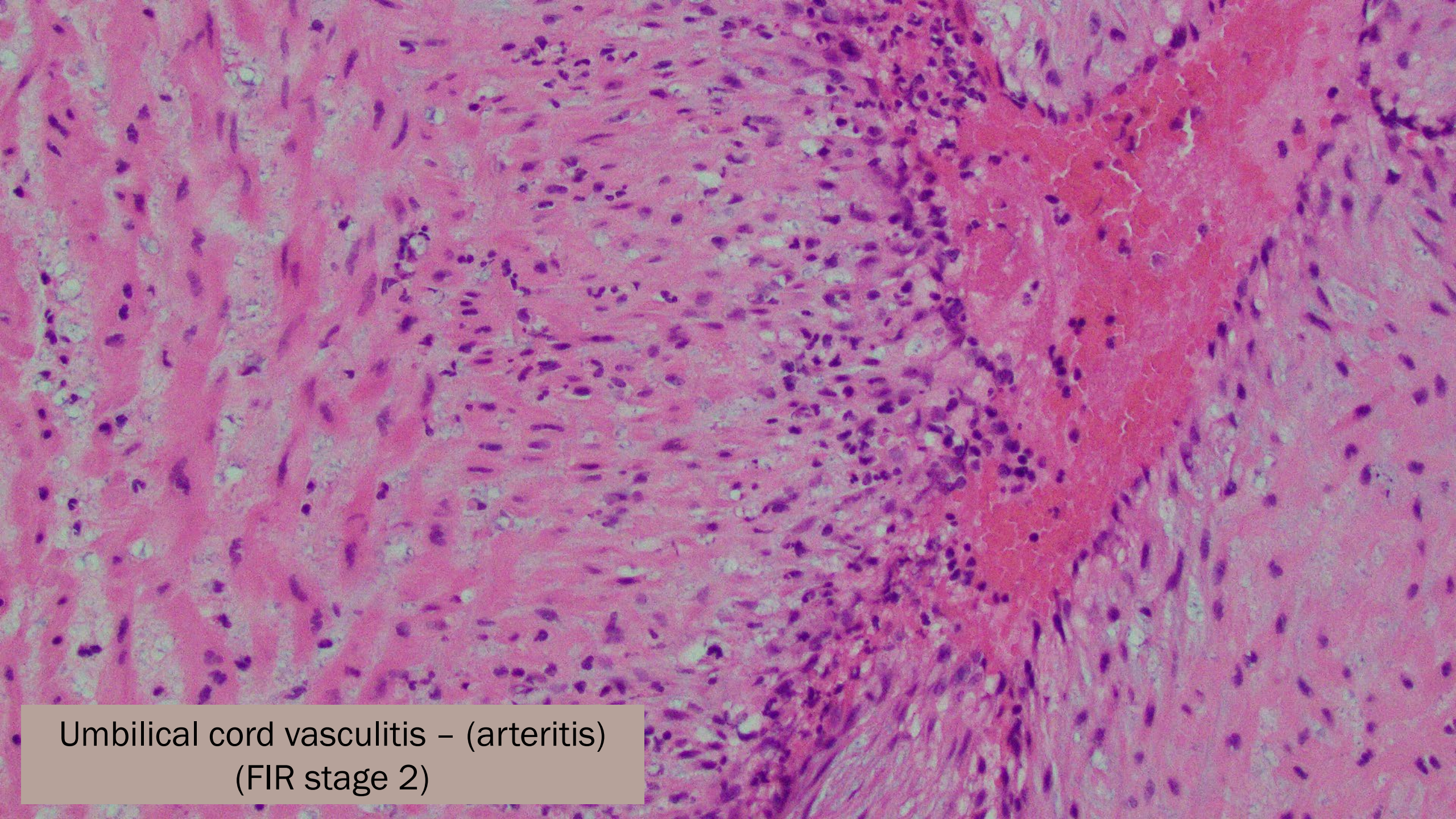

Parenchyma
Chorionic plate vessel


Chorionic plate vasculitis
(FIR stage 1)

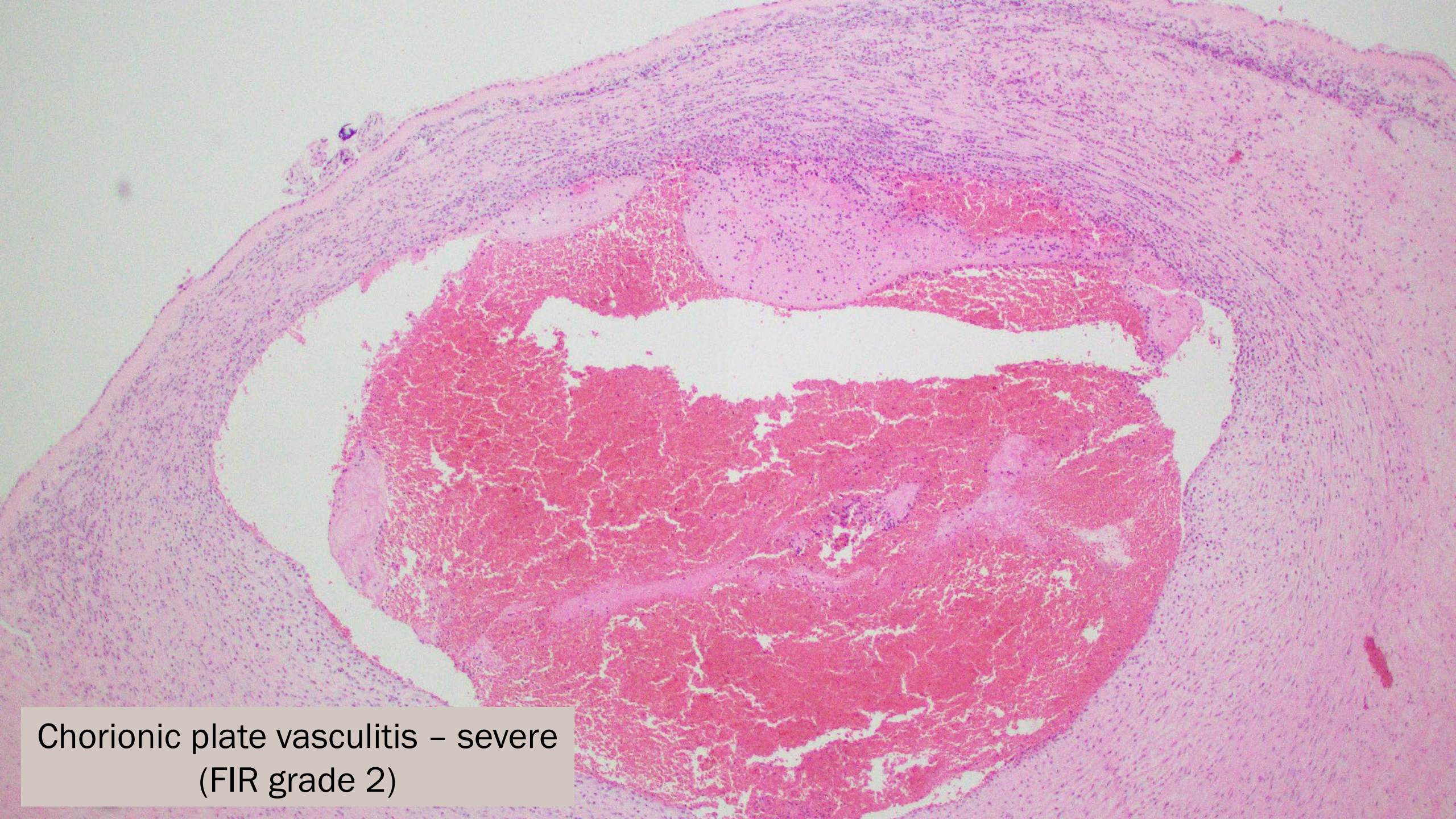

Vein
Artery
Artery



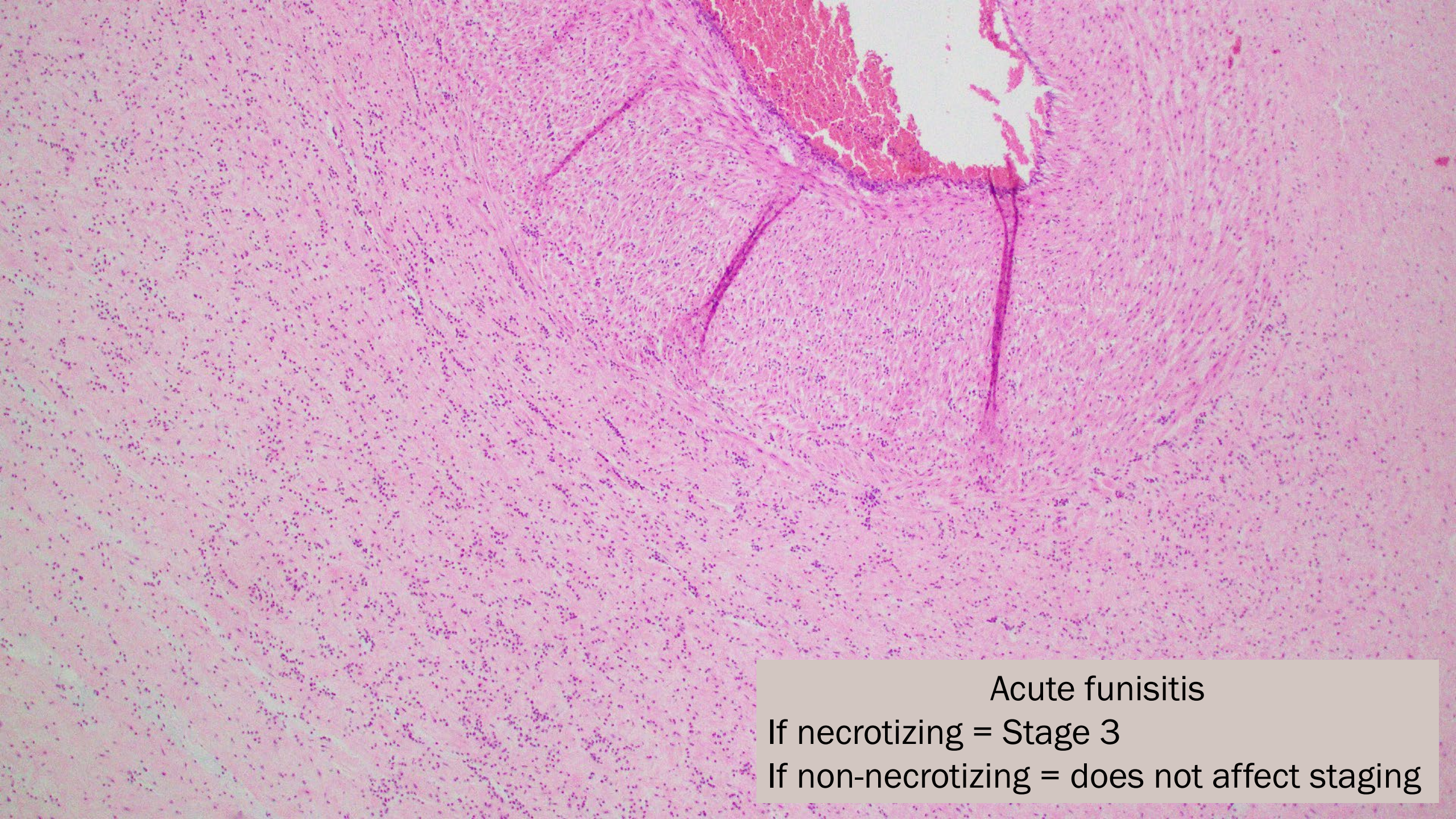
Umbilical cord vasculitis – (phlebitis)
(FIR stage 1)



Umbilical cord vasculitis – (arteritis)
(FIR stage 2)



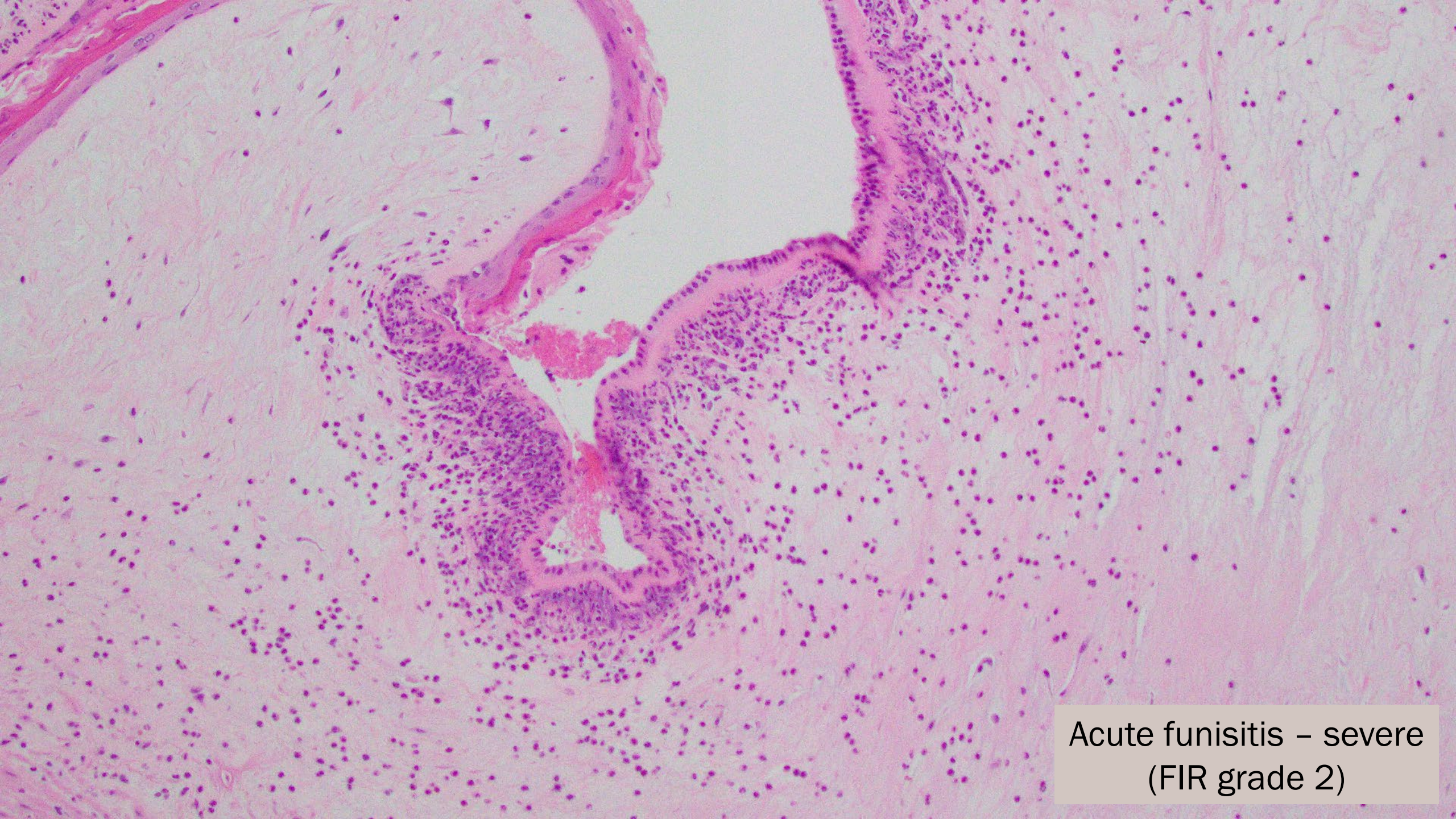
Chorionic plate vasculitis – severe
(FIR grade 2)



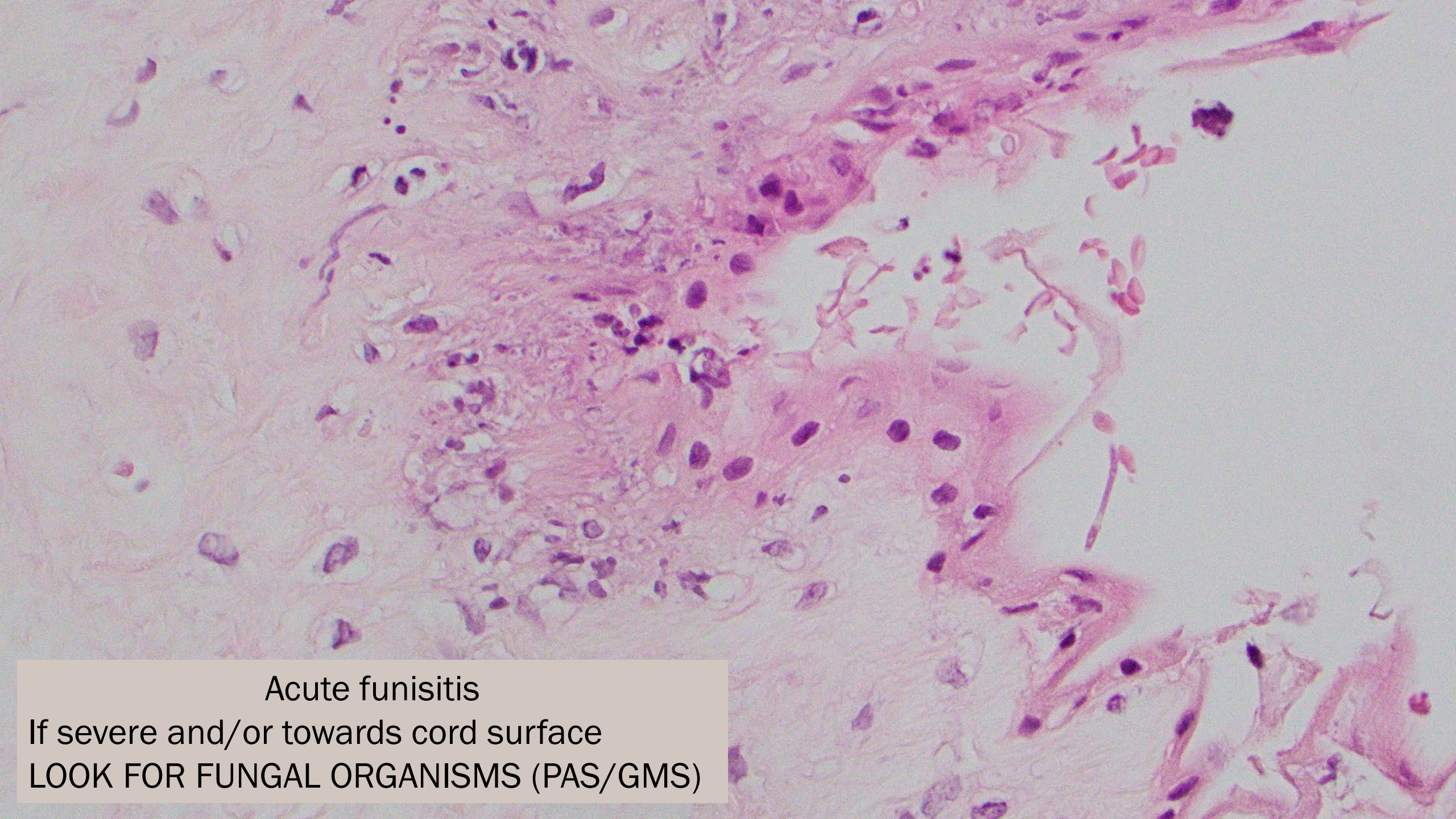
Acute funisitis

If necrotizing = Stage 3

If non-necrotizing = does not affect staging



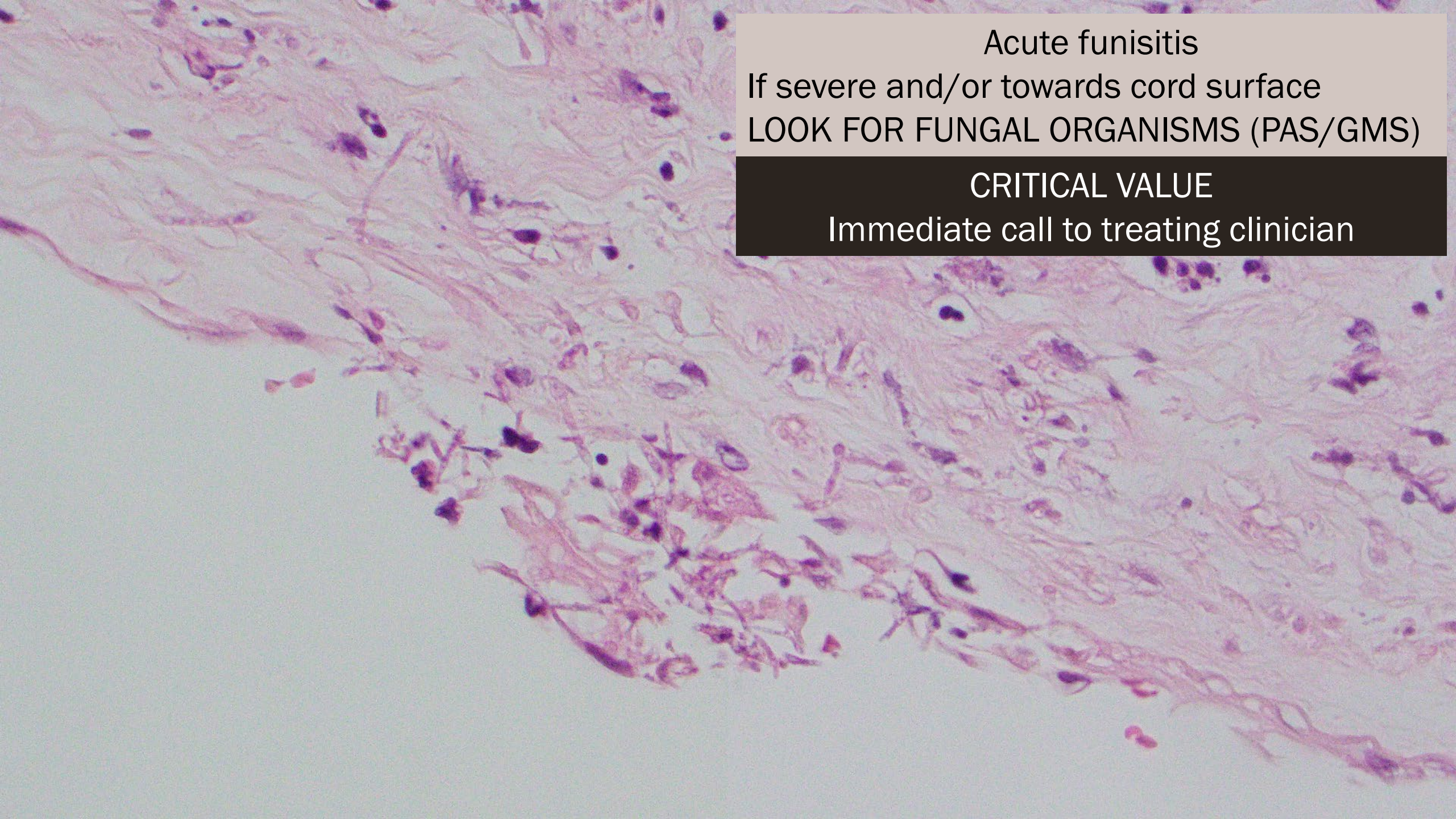
Acute funisitis – severe
(FIR grade 2)



Acute funisitis

If severe and/or towards cord surface

LOOK FOR FUNGAL ORGANISMS (PAS/GMS)



Acute funisitis

If severe and/or towards cord surface

LOOK FOR FUNGAL ORGANISMS (PAS/GMS)

CRITICAL VALUE

Immediate call to treating clinician

ASCENDING INFECTION - ISSUES

- Correlation between inflammation stage/grade and neonatal sepsis and outcomes
- Stage 1-2 / grade 1 inflammation in pregnancies without clinical or microbiological evidence of amniotic fluid infection

IS IT REALLY RELEVANT?

- Stage 3 / grade 2 inflammation is, in turn, rare (<1%)

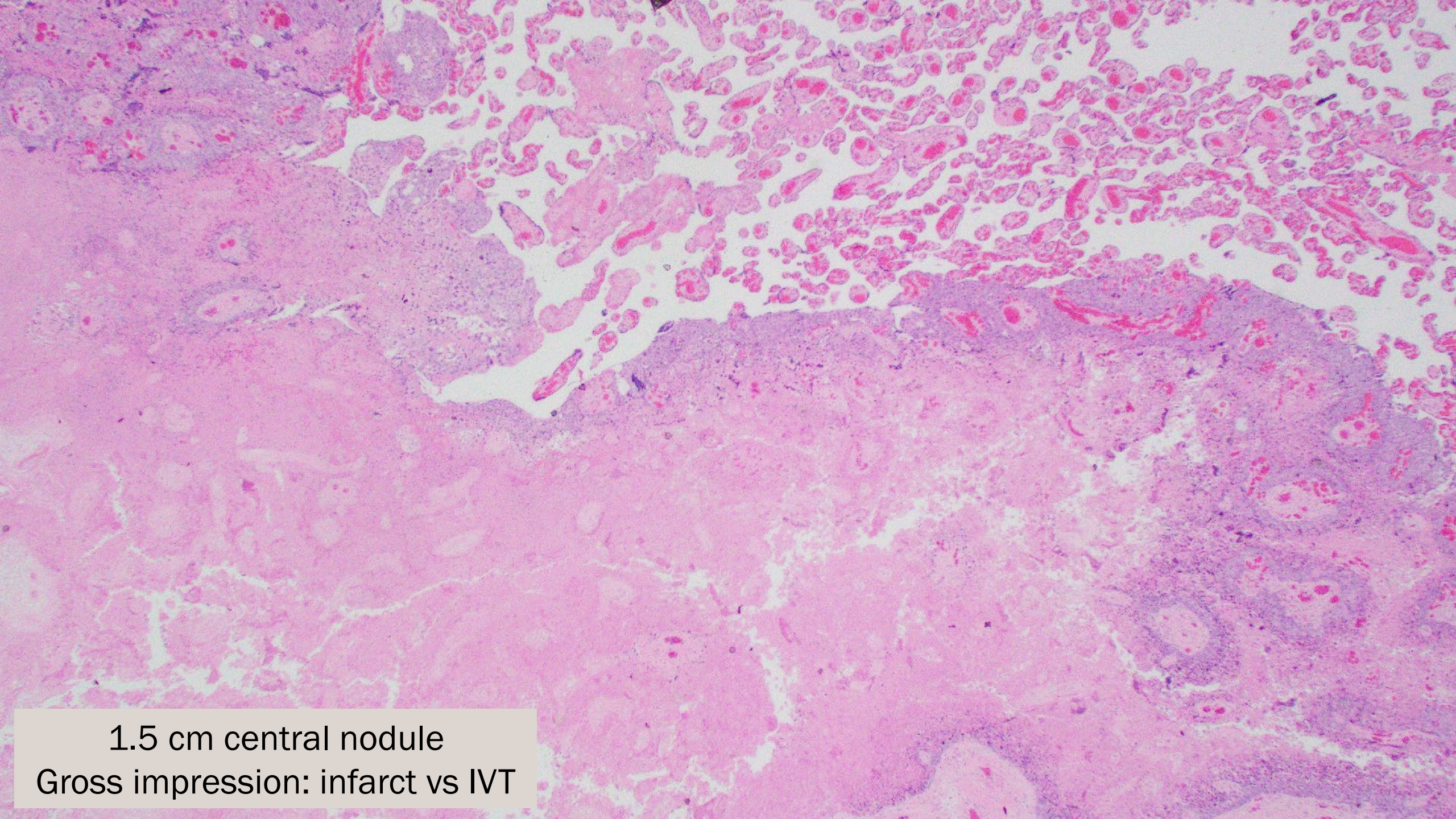
Acta Biomed 2020;91(3):e2020061.
Placenta 2015;36(12):1490–3

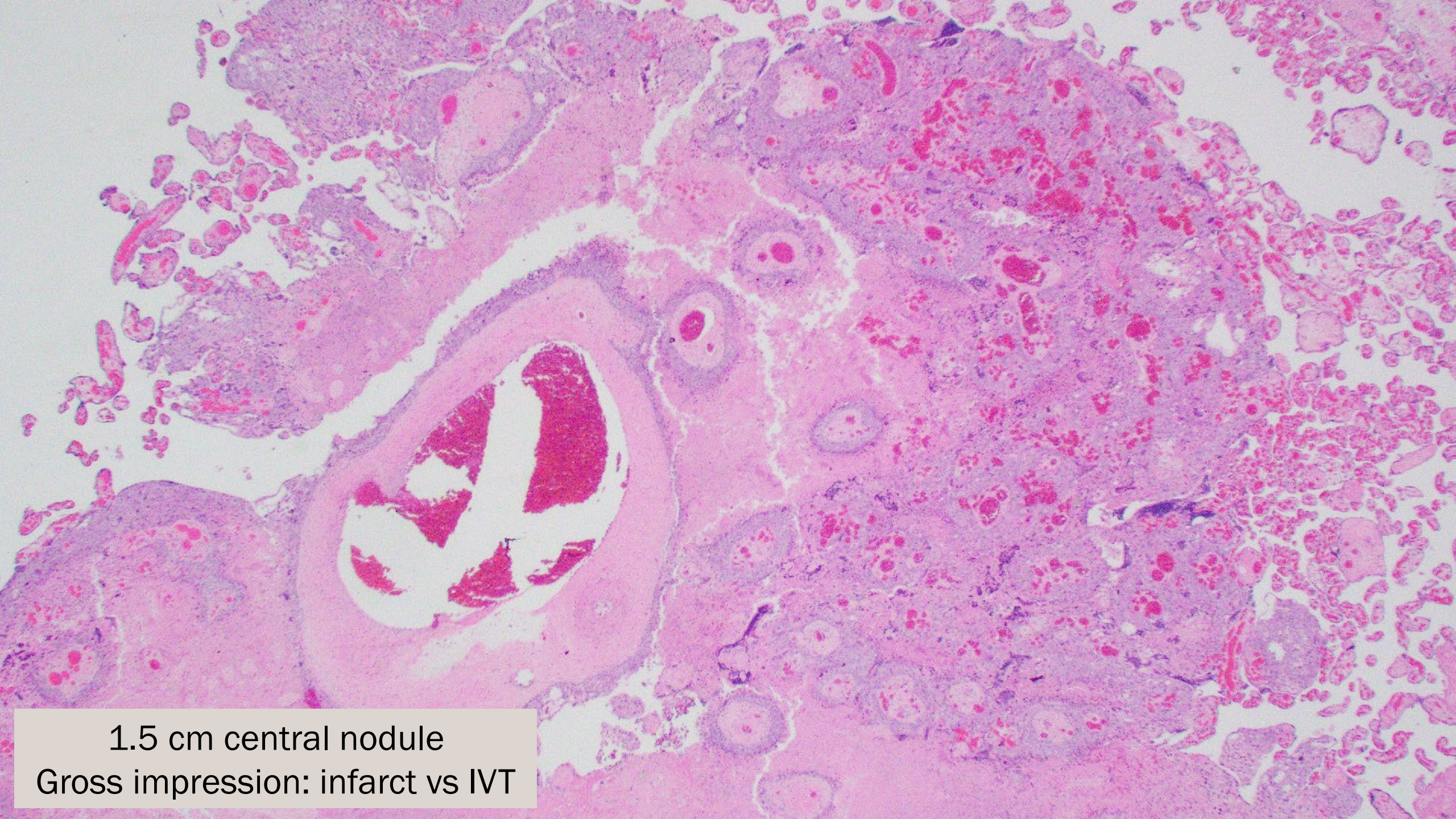
J Perinat Med 2018;46(6):613–30
PLoS One 2012;7(3):e31819

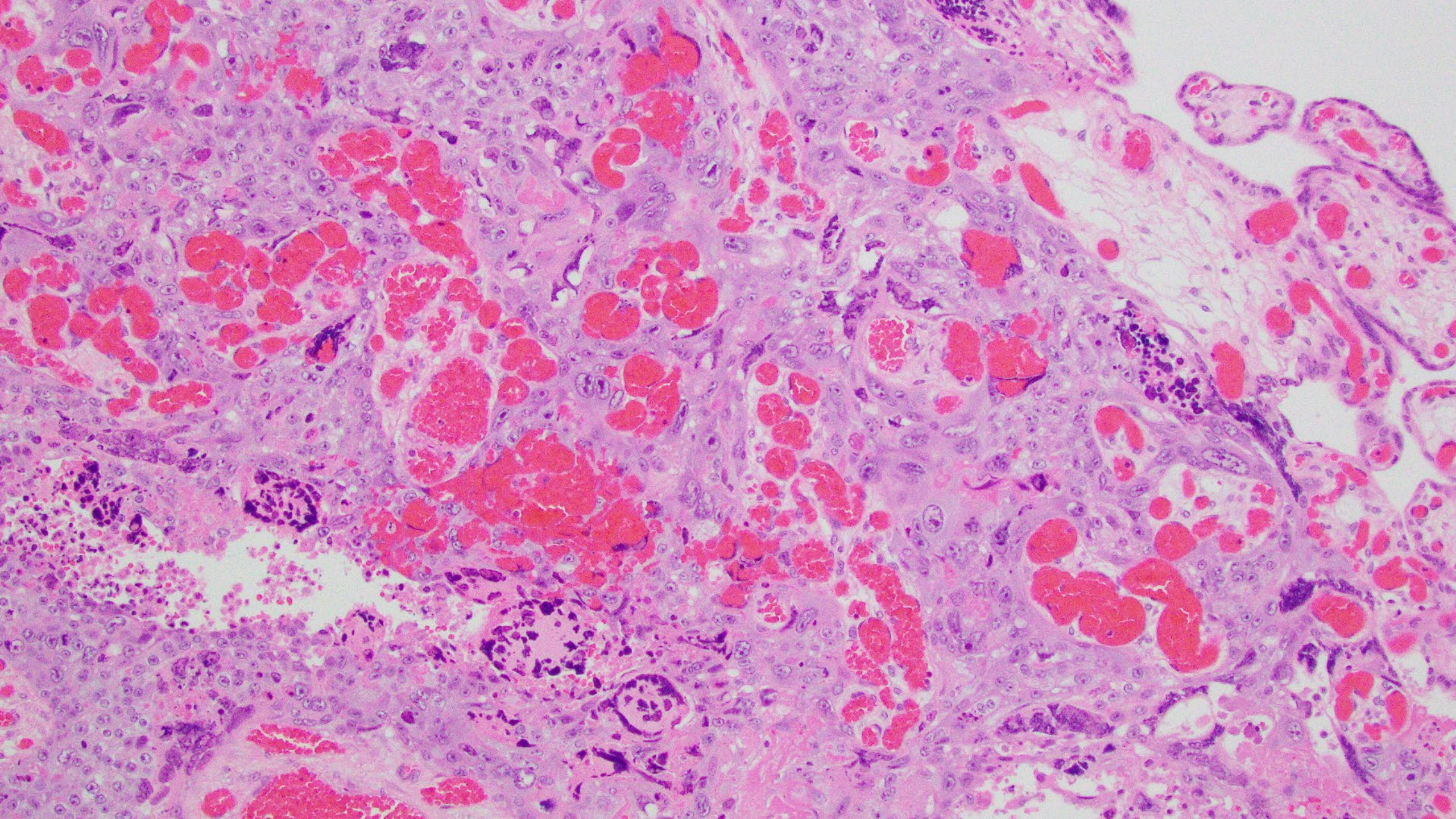
PLACENTAL LESIONS C

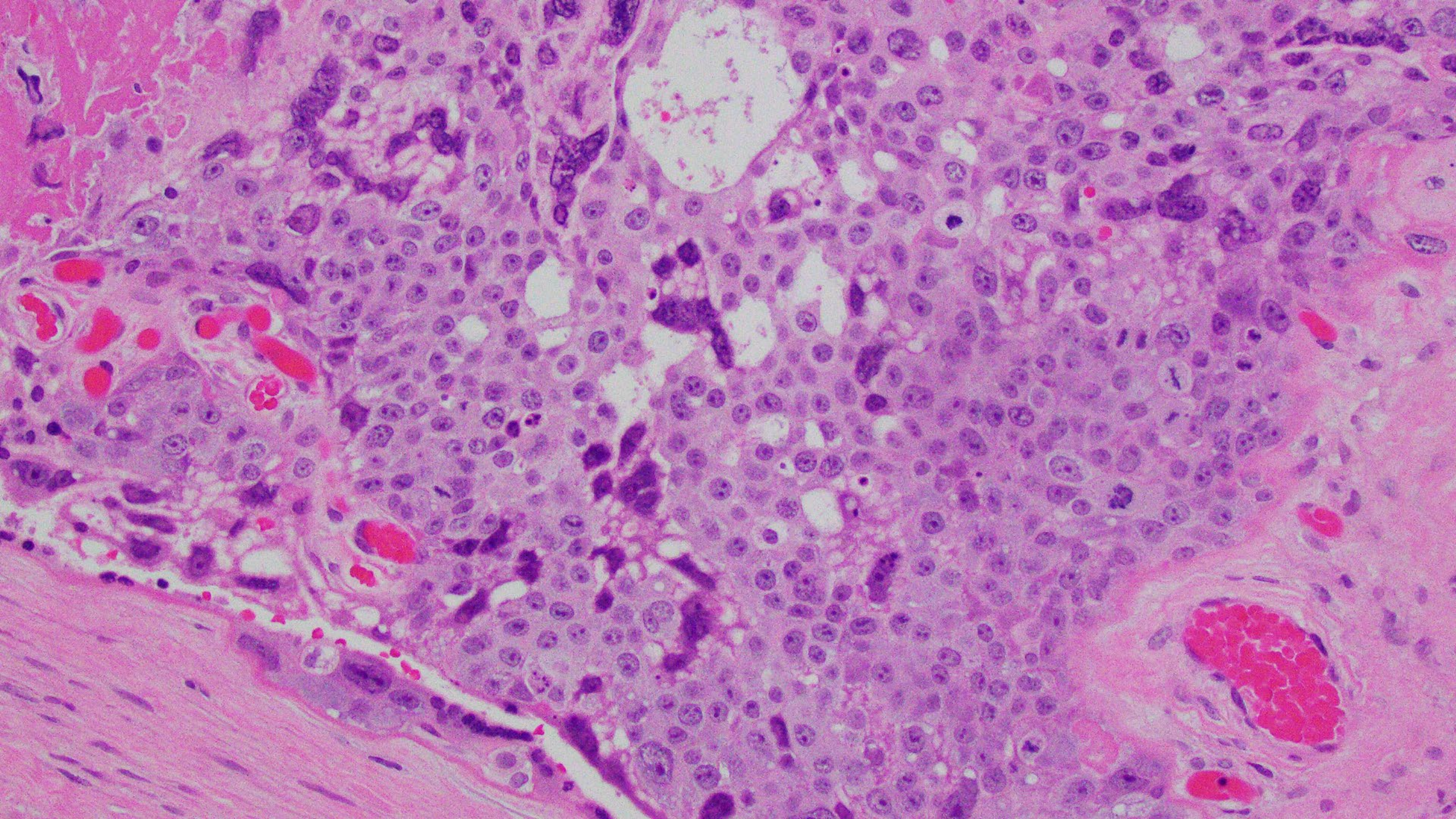
- Placental lesions that require an immediate intervention

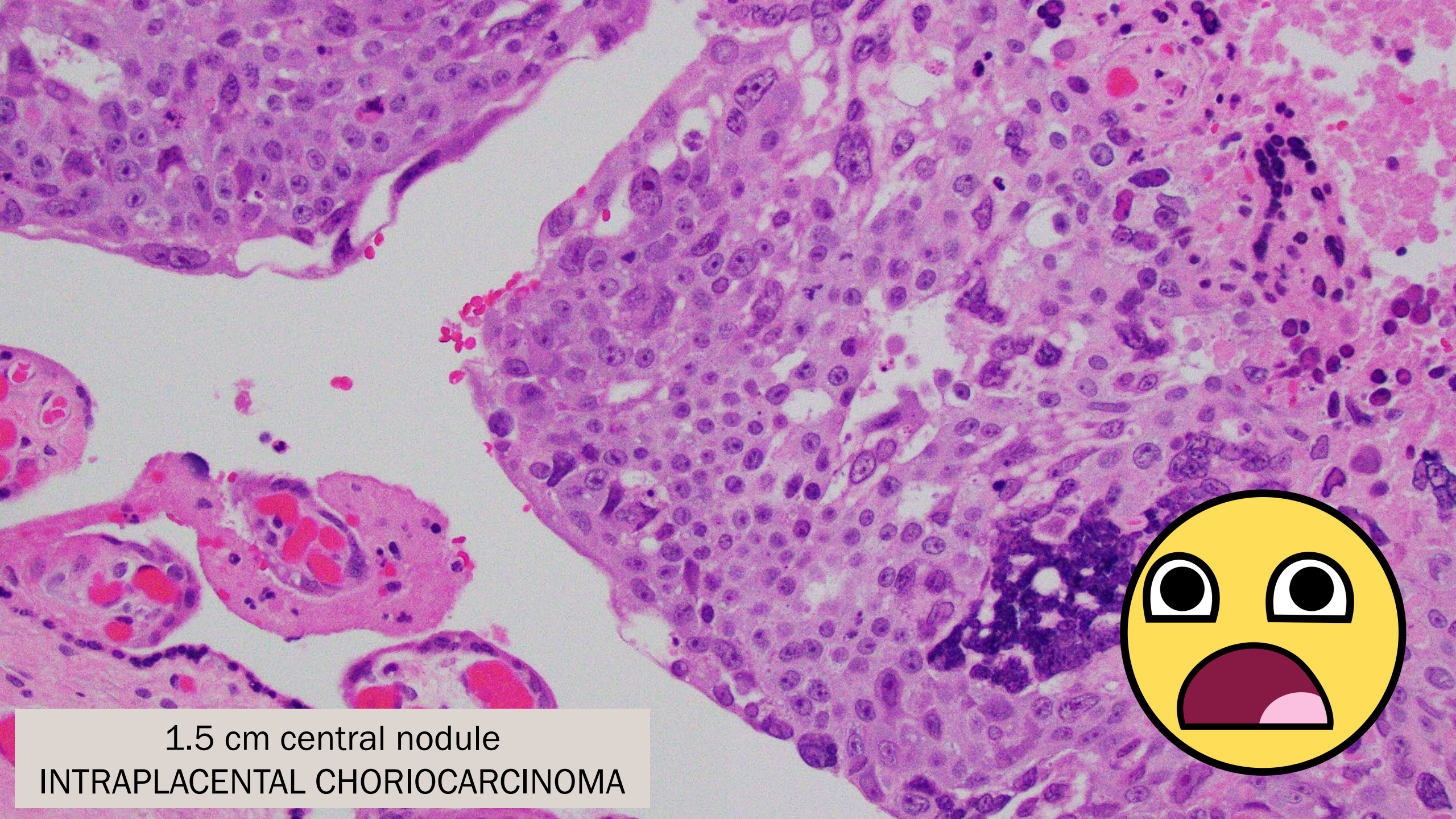
BONUS!


1.5 cm central nodule
Gross impression: infarct vs IVT


1.5 cm central nodule
Gross impression: infarct vs IVT







1.5 cm central nodule
INTRAPLACENTAL CHORIOCARCINOMA

MALIGNANCY IN PLACENTA

- Extra-placental (usually there is previous history)
  - *Increased sampling (7 parenchyma sections)*
- Intraplacental choriocarcinoma
  - *Exceedingly rare phenomenon*
  - *Associated with chorionic villi*
  - *Biphasic and highly atypical population*
- ALWAYS sample nodules / masses / discrete lesions

SUMMARY

- Certain placental lesions matter
  - *Explain obstetric outcomes*
  - *Have recurrence risk*
  - *Require intervention*
- Staging and grading systems have helped narrow the important lesions (e.g. HG-VUE, stage 2 BPMF)
- Use consensus terminology (Amsterdam)

SUMMARY

- *Sample every single grossly identifiable lesion*
- For every gross lesion, DOCUMENT
  - *Size in cm*
  - *Location*
  - *% of the disc parenchyma involved*

THANK YOU!

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