Placenta

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Objectives

- Know important information to put in a placental pathology report
- Understand the Amsterdam Placental Workshop Group classifications of placental lesions
- Be able to diagnose maternal vascular lesions, fetal vascular lesions, and inflammatory lesions
- Know what placental lesions are associated with CNS injury
- Know what placental lesions are at high risk of recurrence

References

- Khong et al. Sampling and Definitions of Placental Lesions: Amsterdam Placenta Workshop Consensus Statement. Arch Pathol Lab Med – Vol 140, July 2016. Pp 698-713.
- Redline RW. Classification of placental lesions. Am J Obstet Gynecol. October 2015. Pp S21-8.
- Baergen (ed.) Manual of Pathology of the Human Placenta, 2nd ed. Springer, 2011.

Why examine a placenta?

- Identification of previously unsuspected disease process in mother or infant that requires immediate attention
- Conditions associated with a high probability of recurrence
- Information that can guide management of future pregnancies or influence long-term care of mother or infant
- Diagnoses that provide a specific explanation for an adverse outcome

When to examine a placenta

- 1997 CAP guidelines/criteria
- Maternal indications
  - Systemic disorders (diabetes, htn), premature delivery, peripartum fever/infection, unexplained or excessive bleeding, 'TORCH' infection during pregnancy, severe oligo or poly, unexplained or recurrent pregnancy complication, abruption, thick meconium, non-elective pregnancy termination, hx drug abuse, prolonged ROM, post-dates, severe trauma
- Fetal/neonatal indications
  - NICU admission, stillbirth/perinatal death, 'compromised clinical condition', hydrops, IUGR, LGA, congenital anomalies, multiple gestation
- Placental indications
  - Any gross abnormality, small or large for age

When to examine a placenta

- Following the CAP guidelines, 40-50% of all placentas in a high-risk setting would be examined
  - Optimal?
  - Useful?
  - Improves patient care?
- Individual hospitals/groups should make their own guidelines

Sampling guidelines

- 4 blocks minimum
- 2 cross-sections of umbilical cord
- Membrane roll with marginal parenchyma
- 1 full-thickness disc section near cord insertion
- 2 full-thickness disc sections from central 2/3 of disc

2014 Amsterdam Placental Workshop Group - classifications

- Placental vascular processes
  - Maternal stromal-vascular lesions
  - Fetal stromal-vascular lesions
- Placental inflammatory-immune processes
  - Infectious
  - Immune/idiopathic
- Other placental processes

Maternal stromal-vascular lesions

- Developmental
  - Decidual vasculopathy
- Malperfusion
  - Global/partial
    - Accelerated villous maturation
    - Distal villous hypoplasia
  - Segmental/complete
    - Villous infarcts
- Loss of integrity
  - Abruption

Decidual vasculopathy

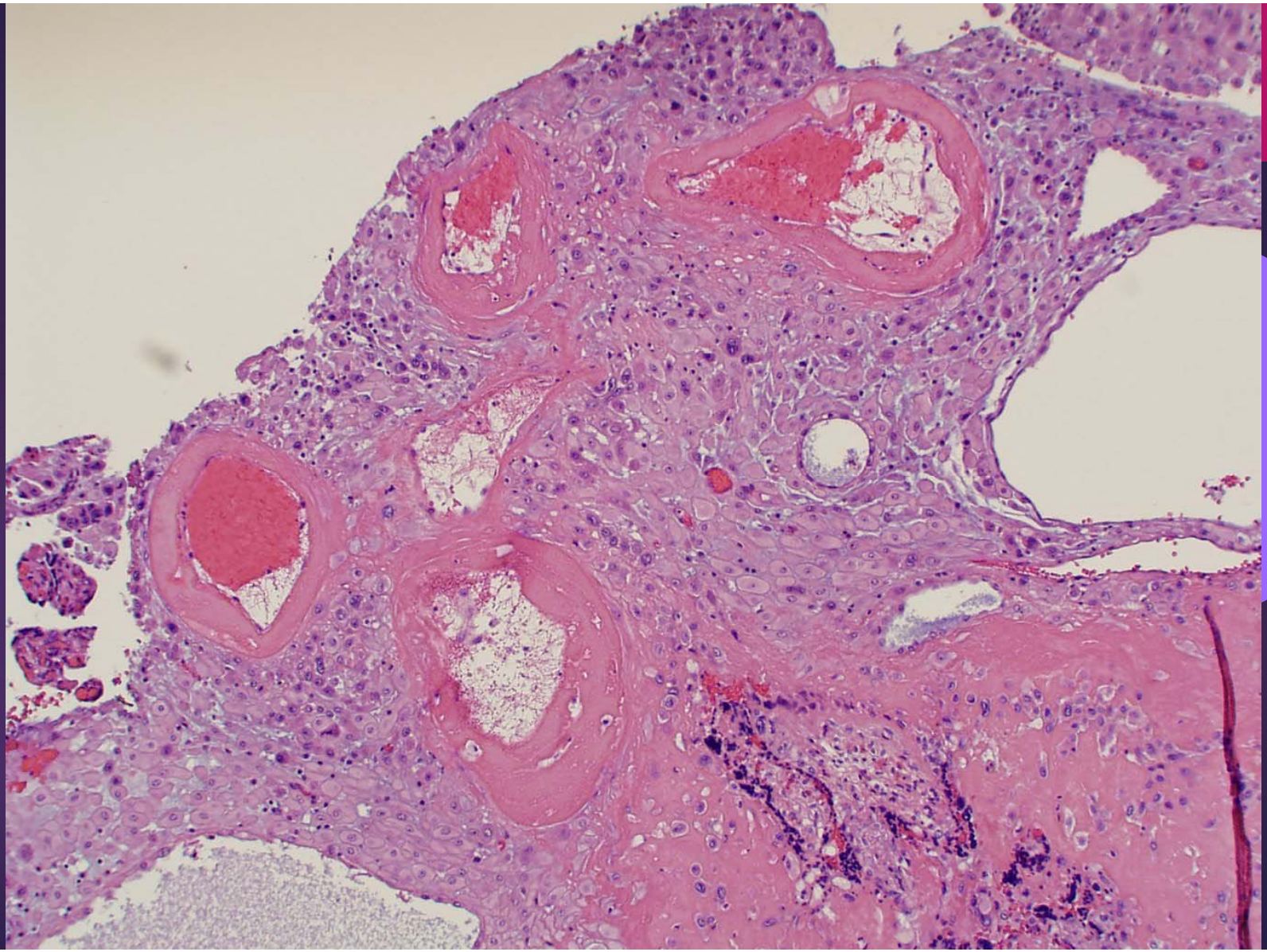
- Spiral arteries must change from high-flow to low-flow system
- Trophoblasts infiltrate arteries and destroy muscular walls
- Complete by 20wk GA

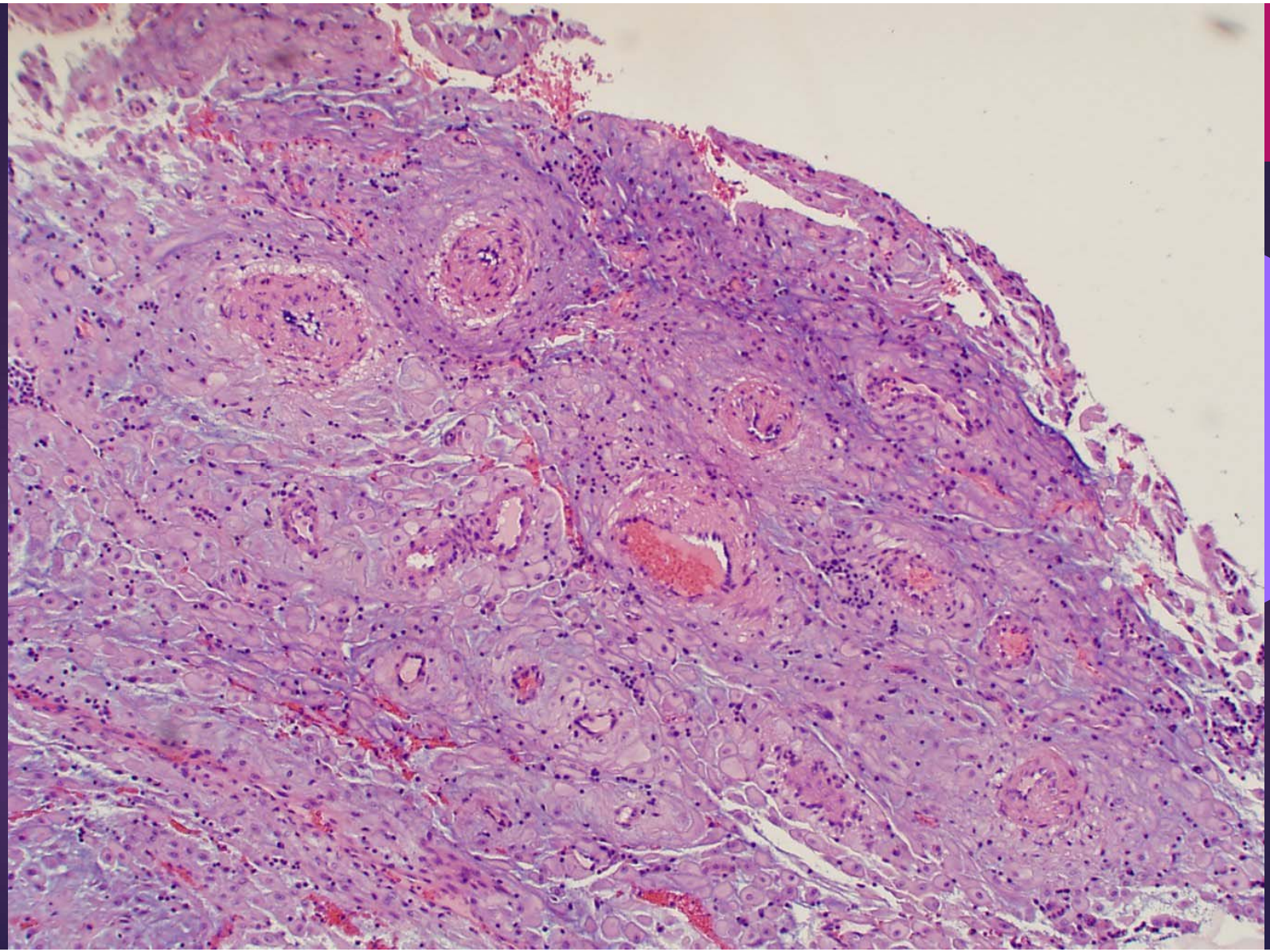
- Defect of extravillous trophoblast differentiation / expansion
- Caused by poorly understood maternal genetic or environmental factors

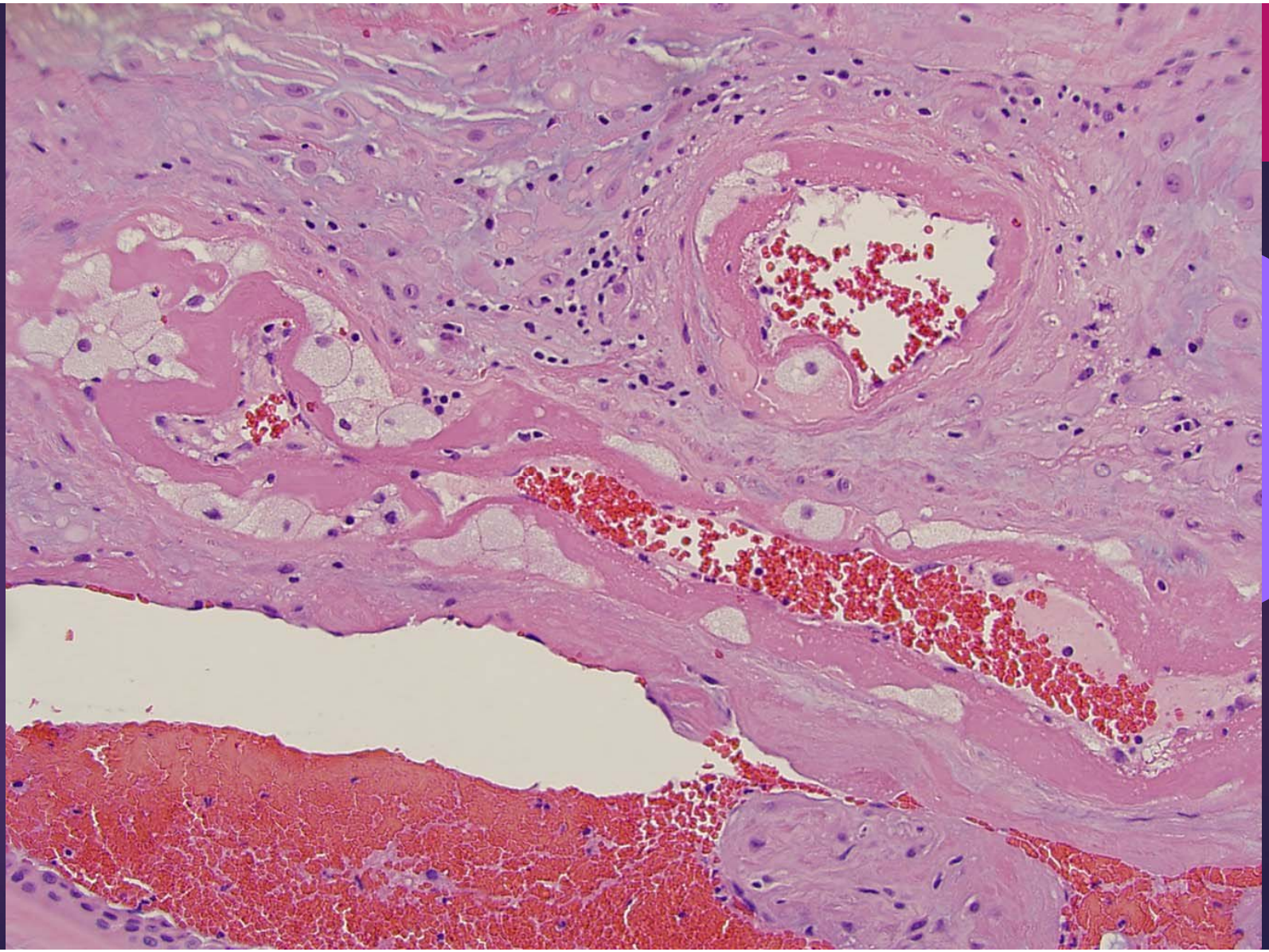
- Clinically – htn, pre-eclampsia

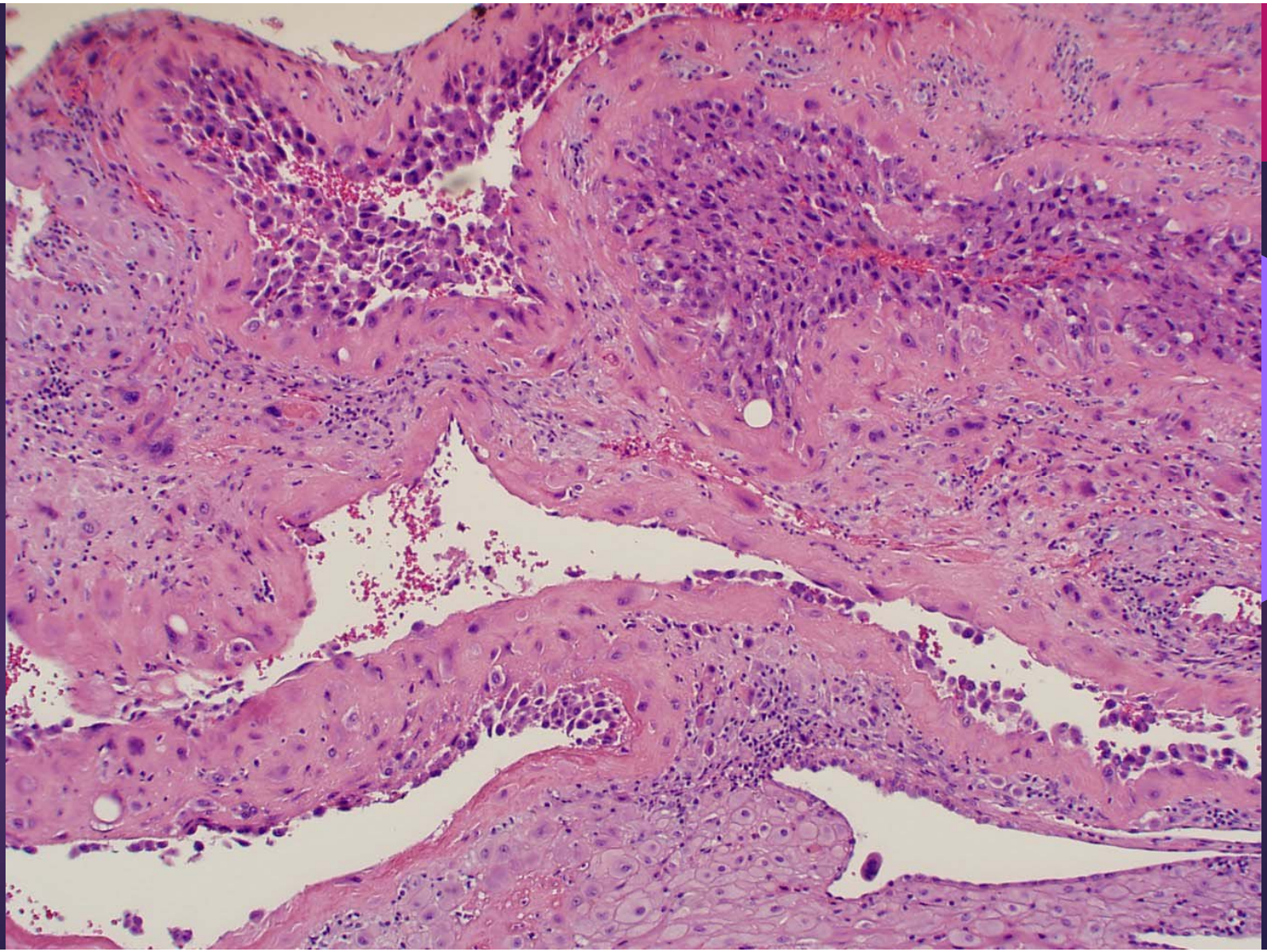
Decidual vasculopathy - histology

- Acute atherosis
- Fibrinoid necrosis +/- foam cells
- Thick-walled vessels (mural hypertrophy, absence of remodeling)
- Perivascular chronic inflammation
- Arterial thrombosis
- Persistence of endovascular trophoblasts









Maternal vascular malperfusion

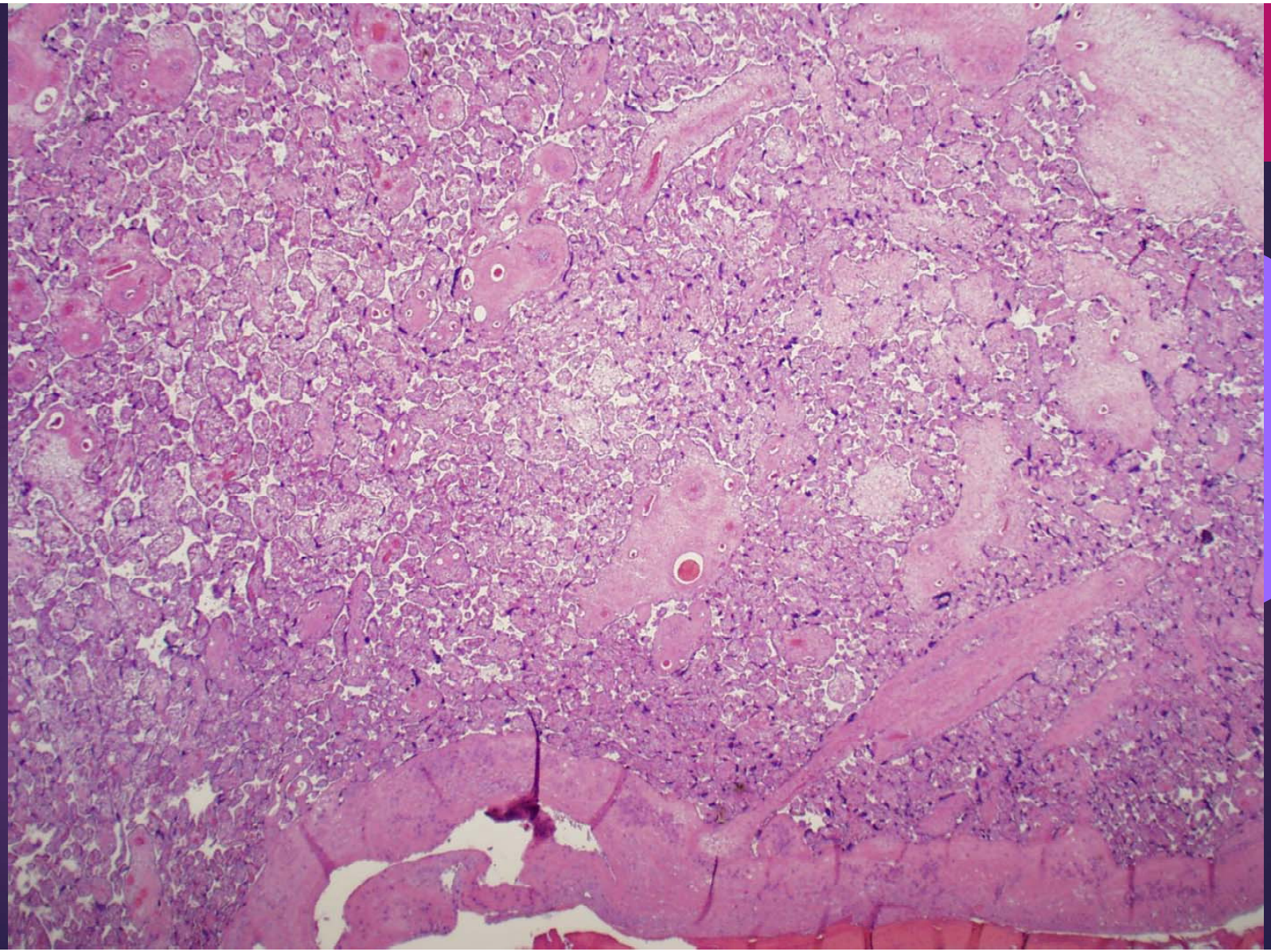
- Abnormal spiral artery flow (not low-velocity, high-volume)
- Global/partial maternal malperfusion
  - Accelerated villous maturation
  - Distal villous hypoplasia
- Segmental/complete maternal malperfusion
  - Villous infarcts

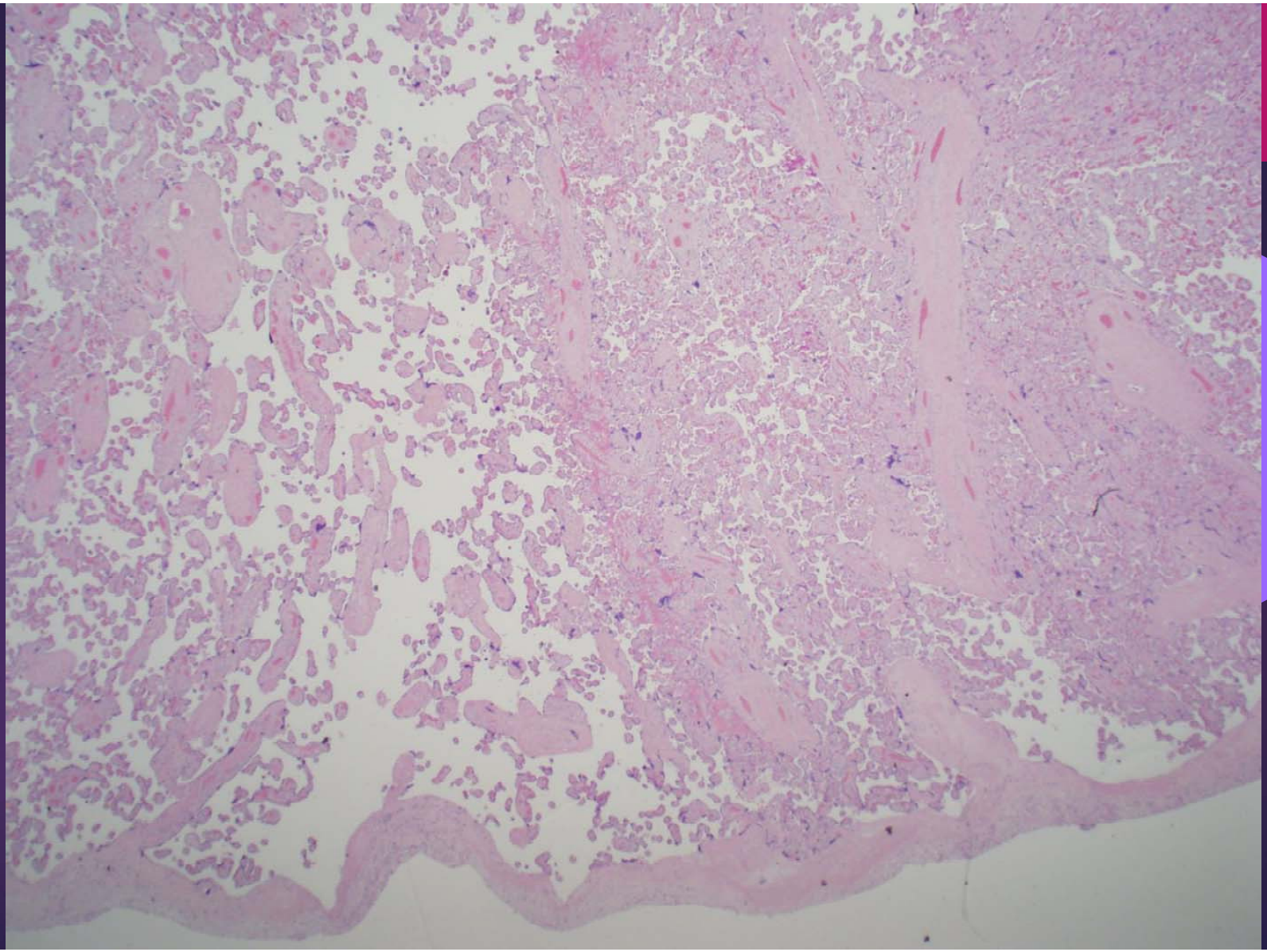
AVM and DVH - histology

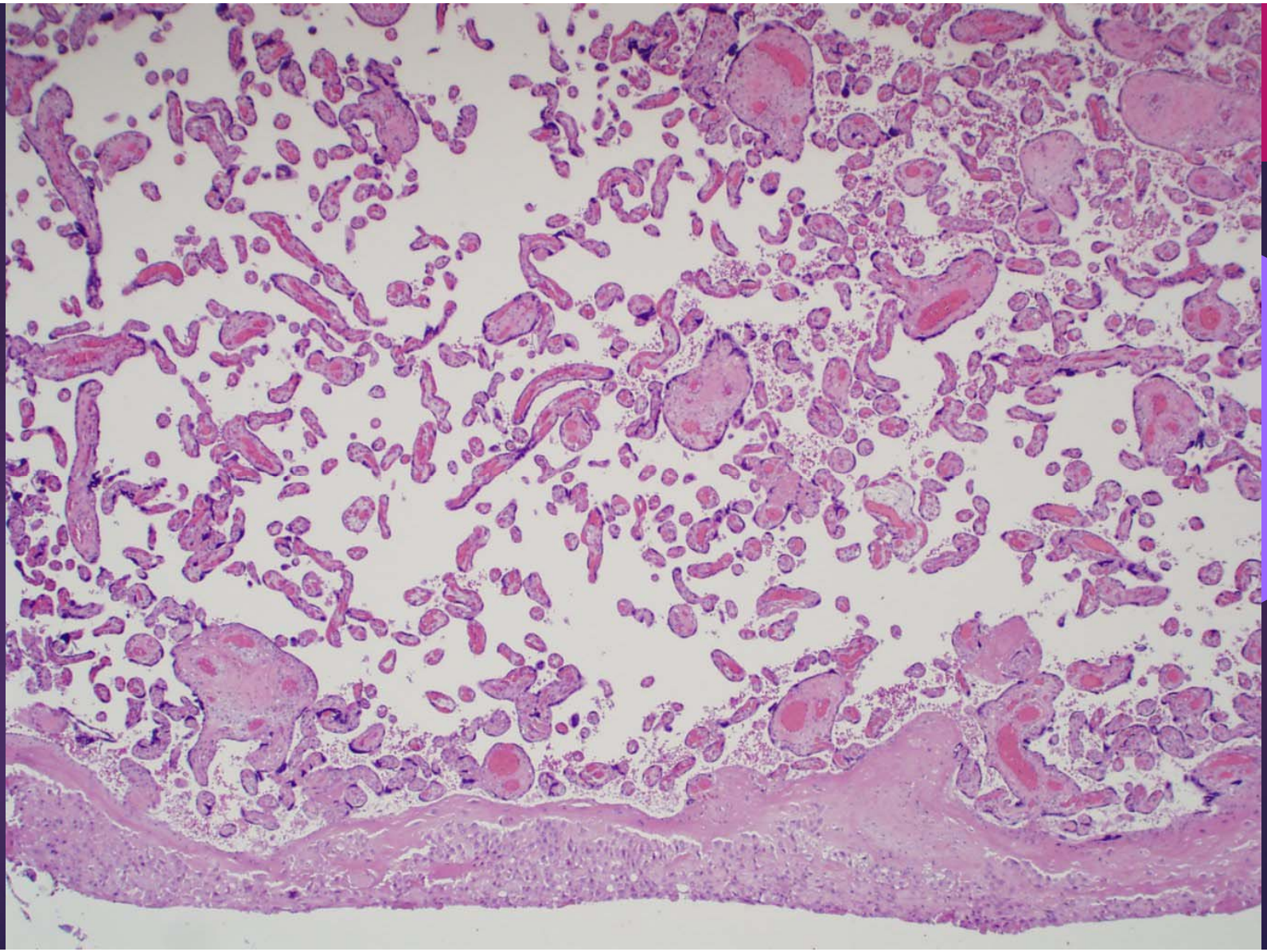
- Small or short, hypermature, villi for the gestation
- Usually accompanied by increased syncytial knots
- Paucity of villi in relation to stem villi (<30% AVM, >30% DVH)

- Can be hard to diagnose at term
  - Syncytial knots in >1/3 villi at term is considered increased

- Do not judge villous maturation near infarcts or in subchorionic region

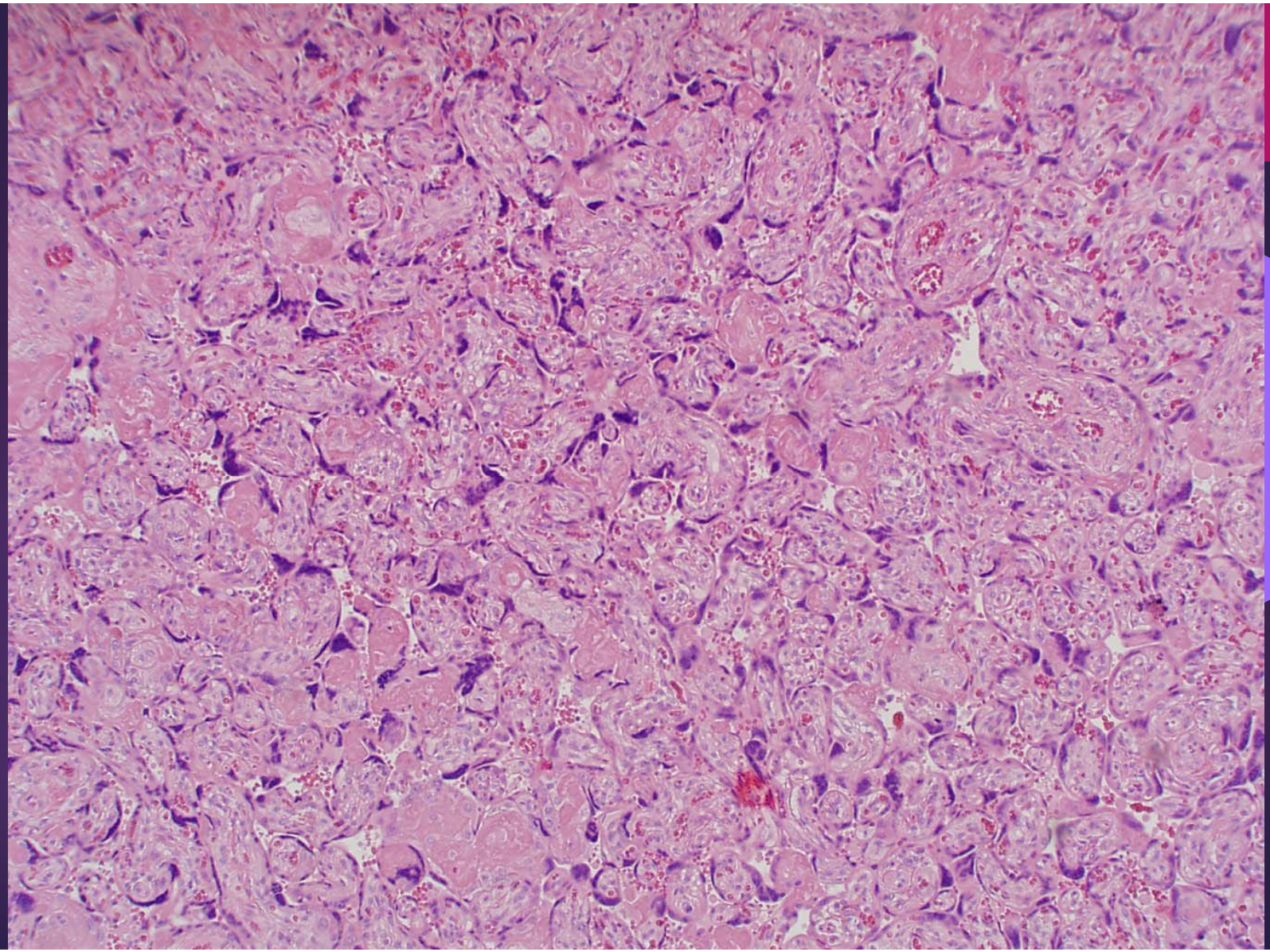


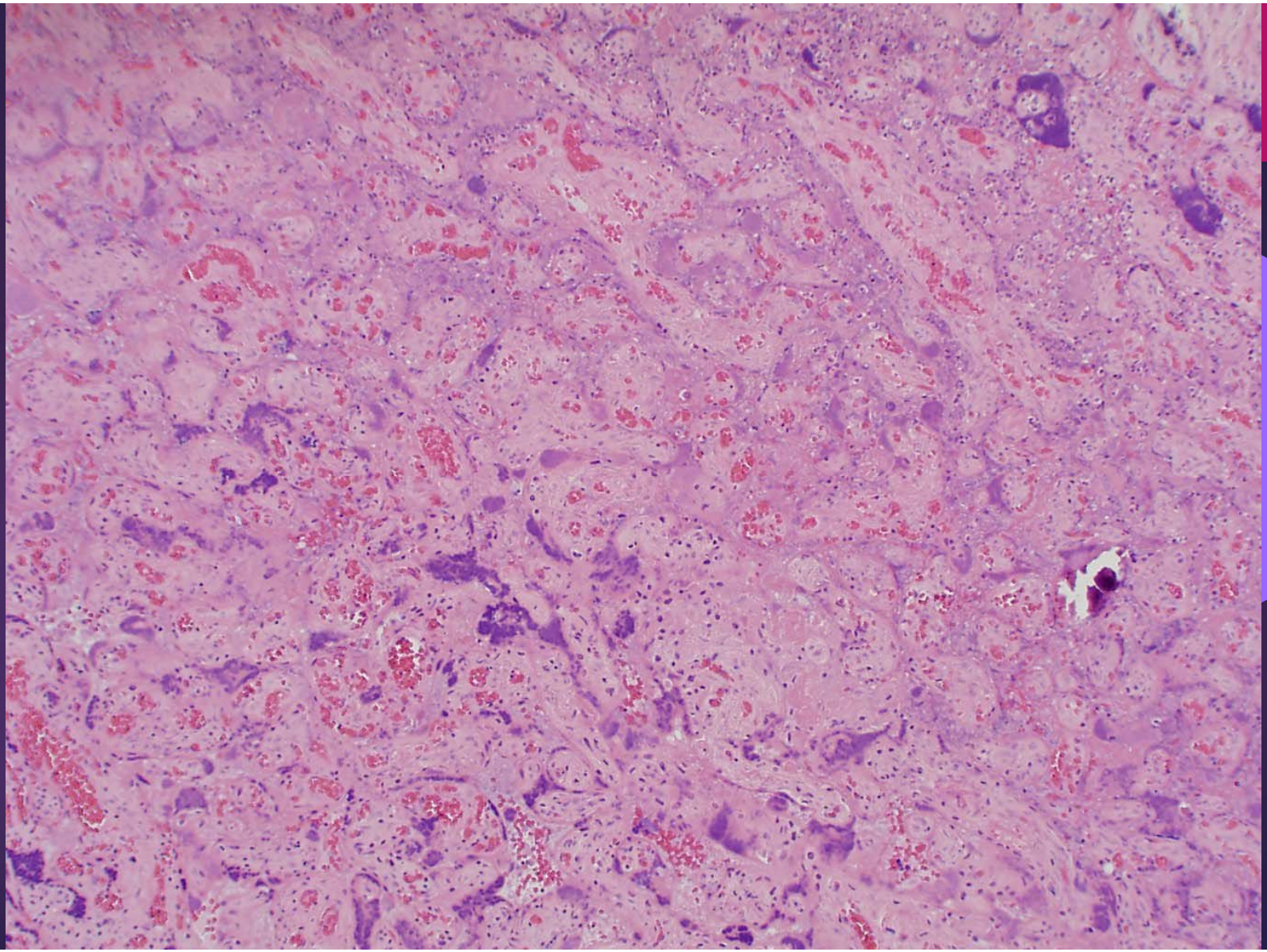


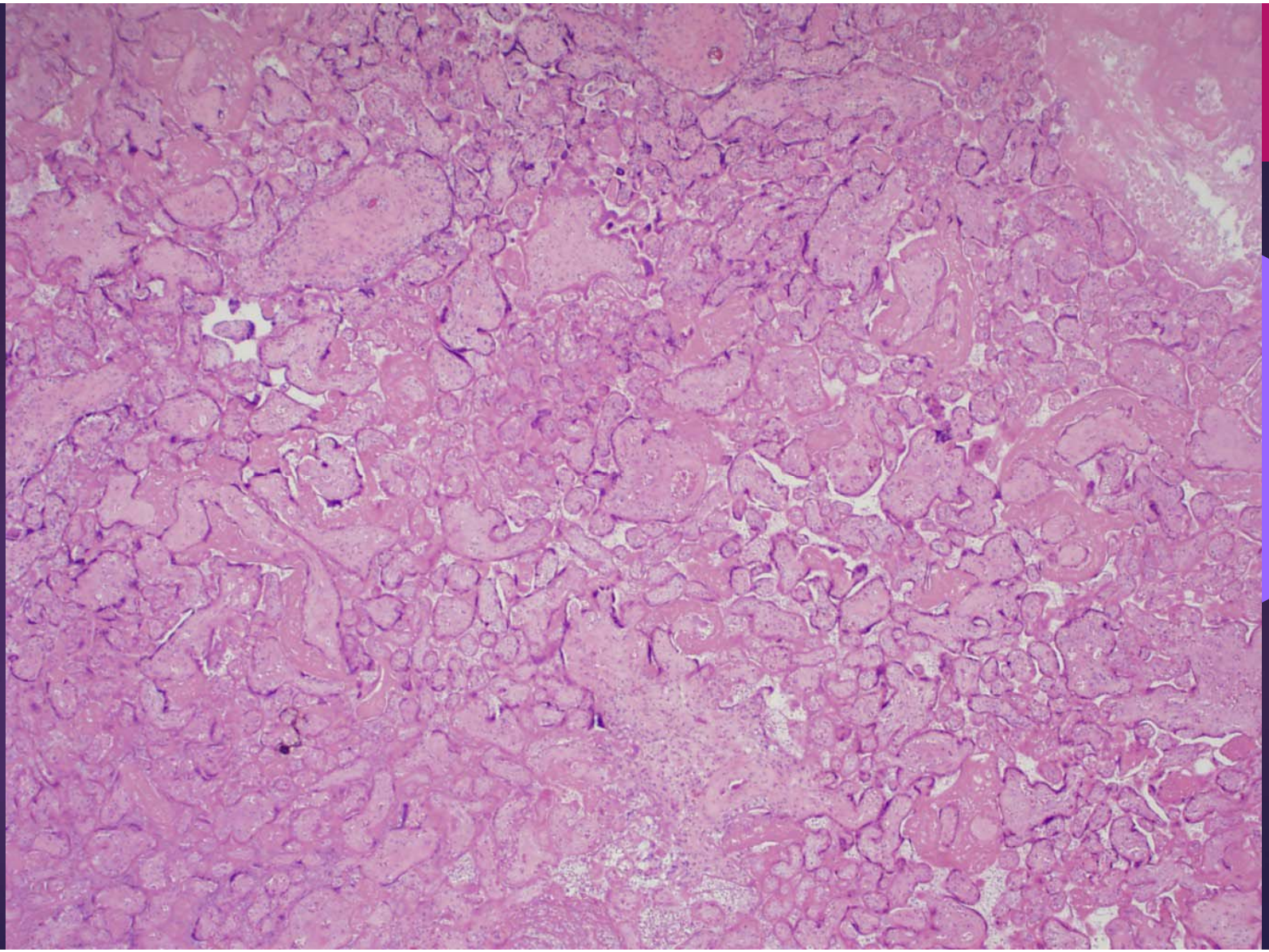


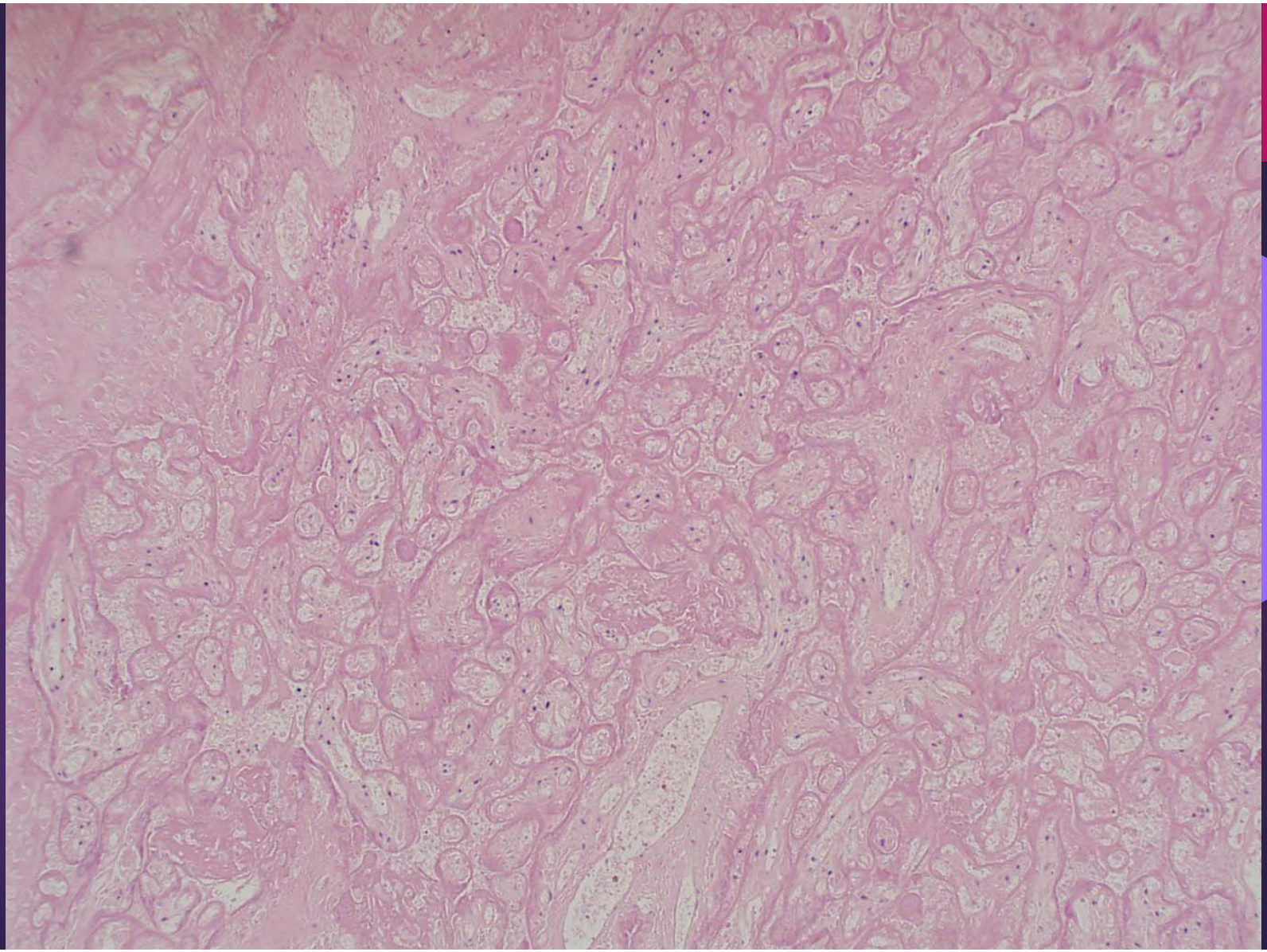
Villous infarcts - histology

- Early
  - Crowding and congestion of villi (agglutination)
  - Early loss of nuclear staining
  - Neutrophils in the intervillous space
- Later
  - Necrotic changes
  - Loss of trophoblast nuclear staining
  - Ghost villi



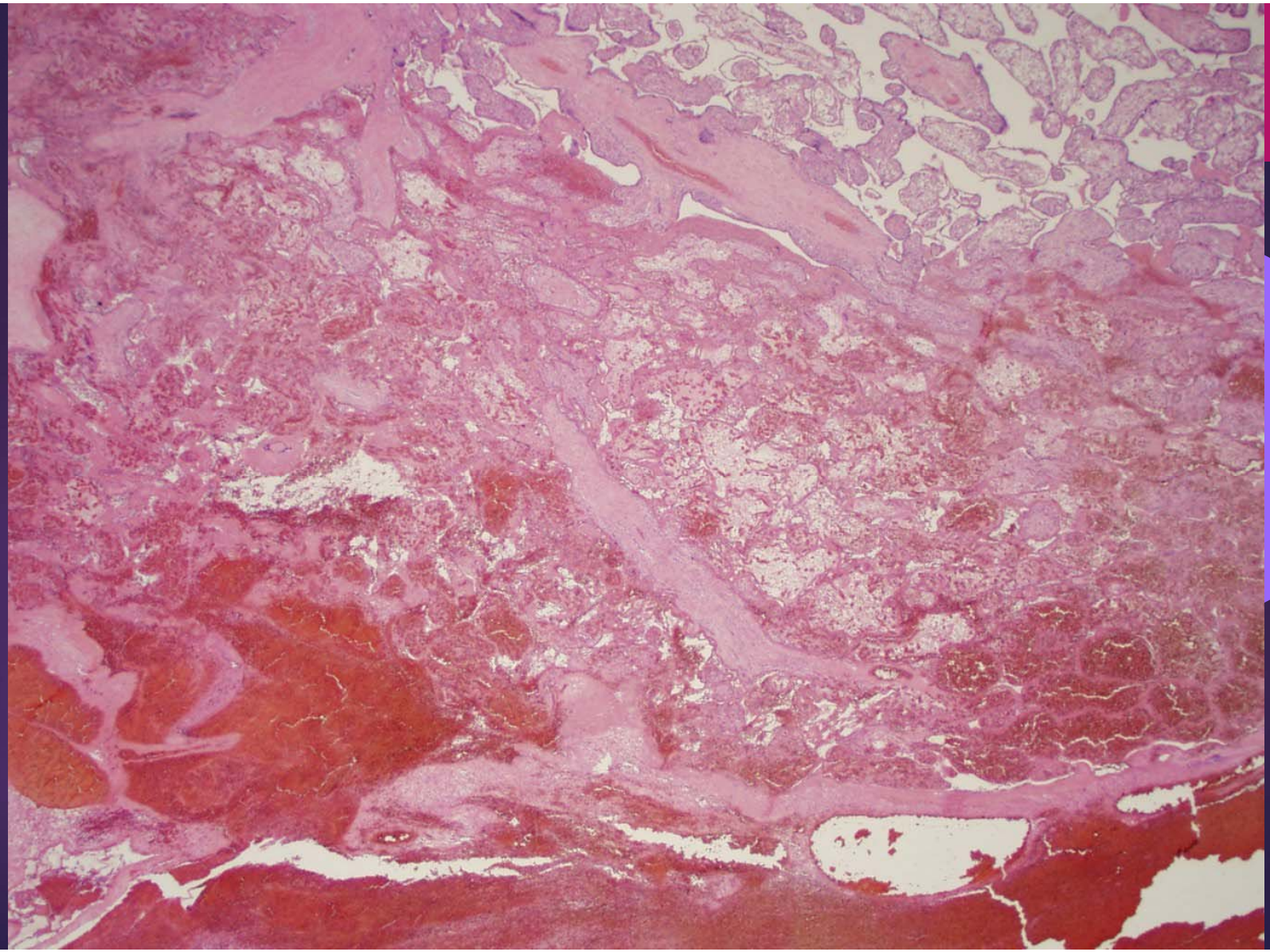


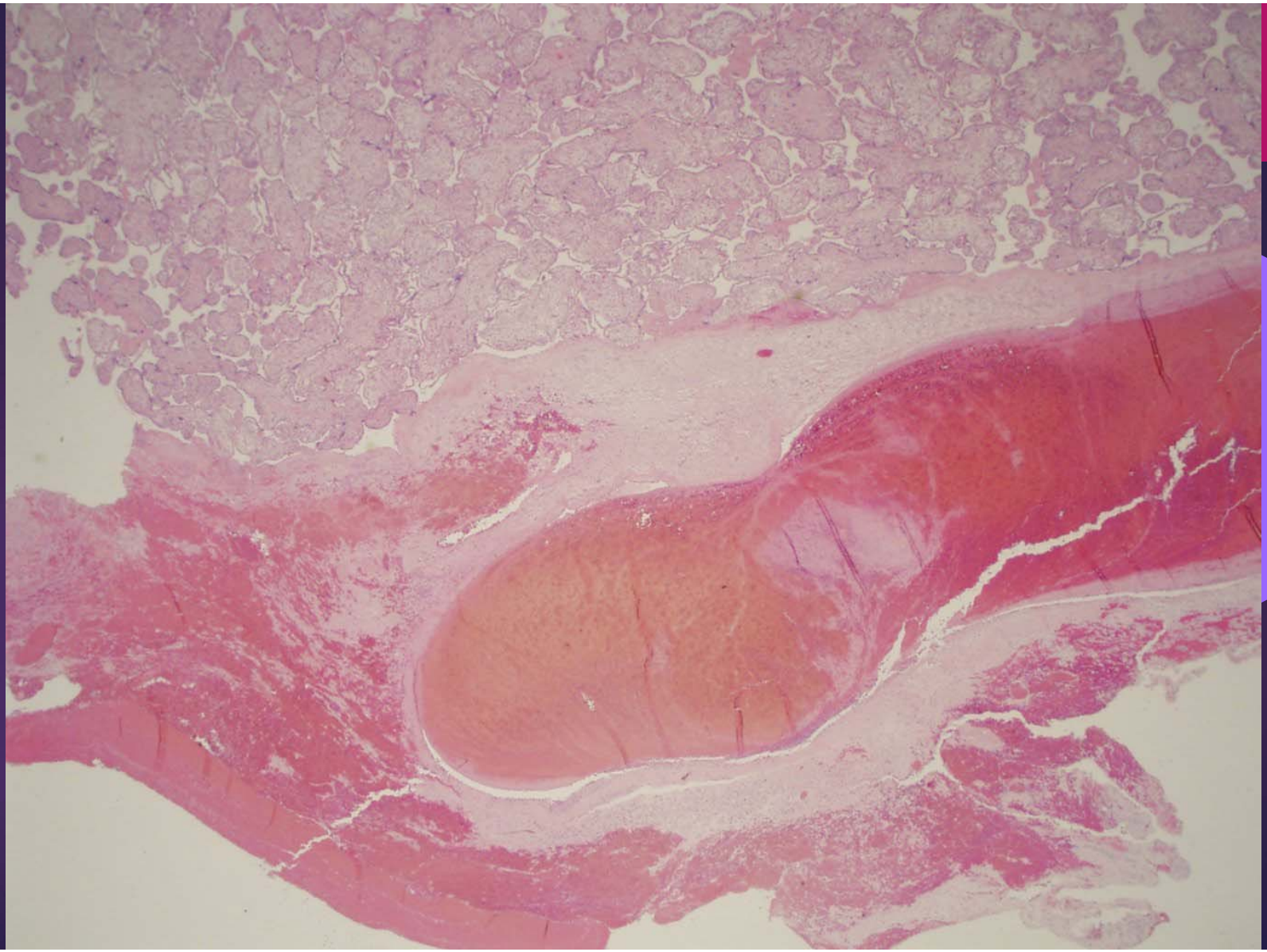


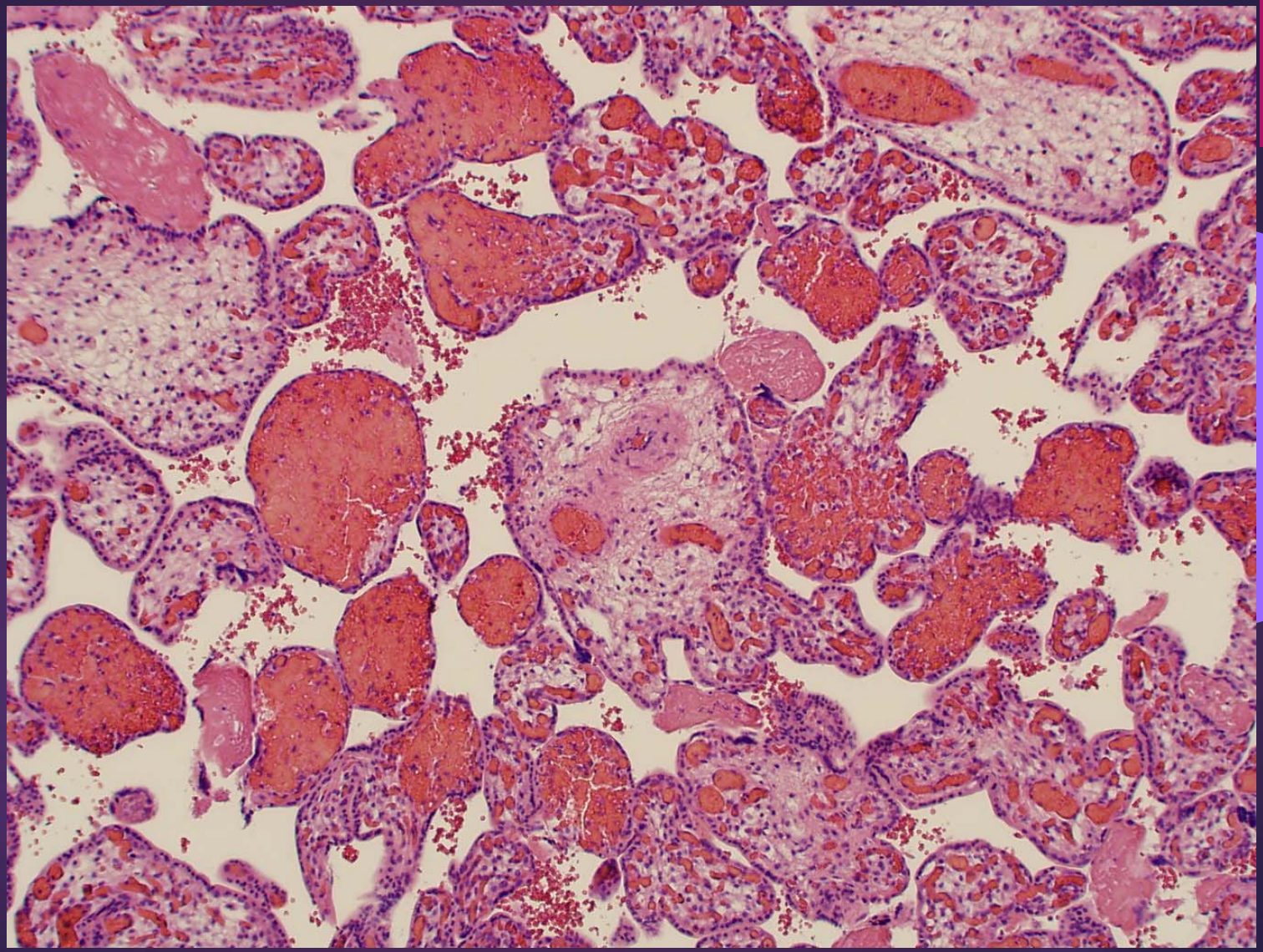


Abruption

- Abruptio placenta
  - Often secondary to arterial maldevelopment in pre-eclampsia
    - Rupture of incompletely remodeled spiral artery
  - Vasoactive drugs (cocaine, nicotine) or sheer stress
  - Central location, high pressure flow
    - Indentation of maternal surface, extension to intervillous space
- Marginal abruption
  - Rupture of maternal veins usually at periphery of placenta
  - Chronic abruption = circumvallate insertion of membranes, hemosiderin








PLACENTA
BABY'S FIRST ROOMMATE
I HEART GUTS!
© i heart guts

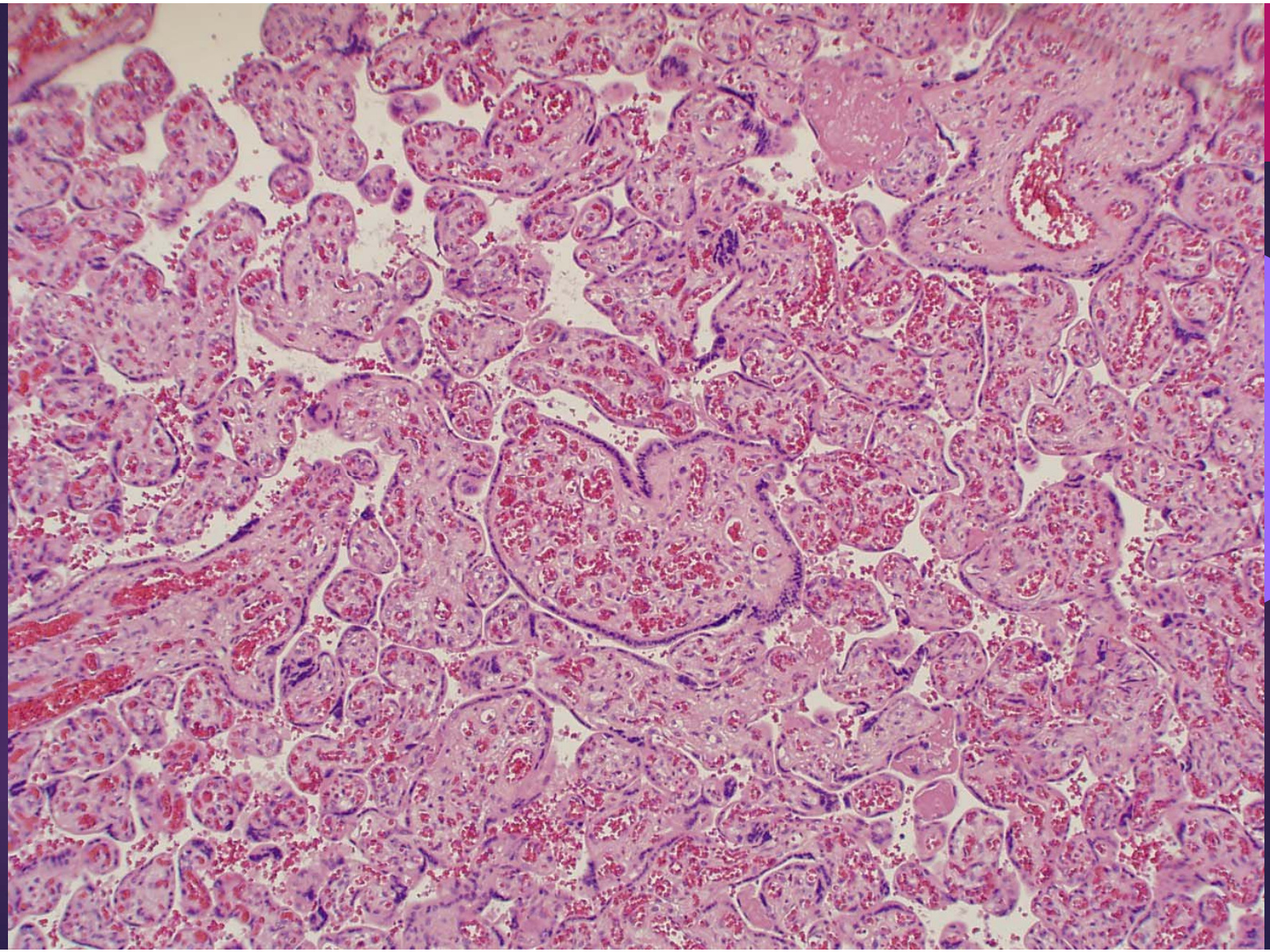
Fetal stromal-vascular lesions

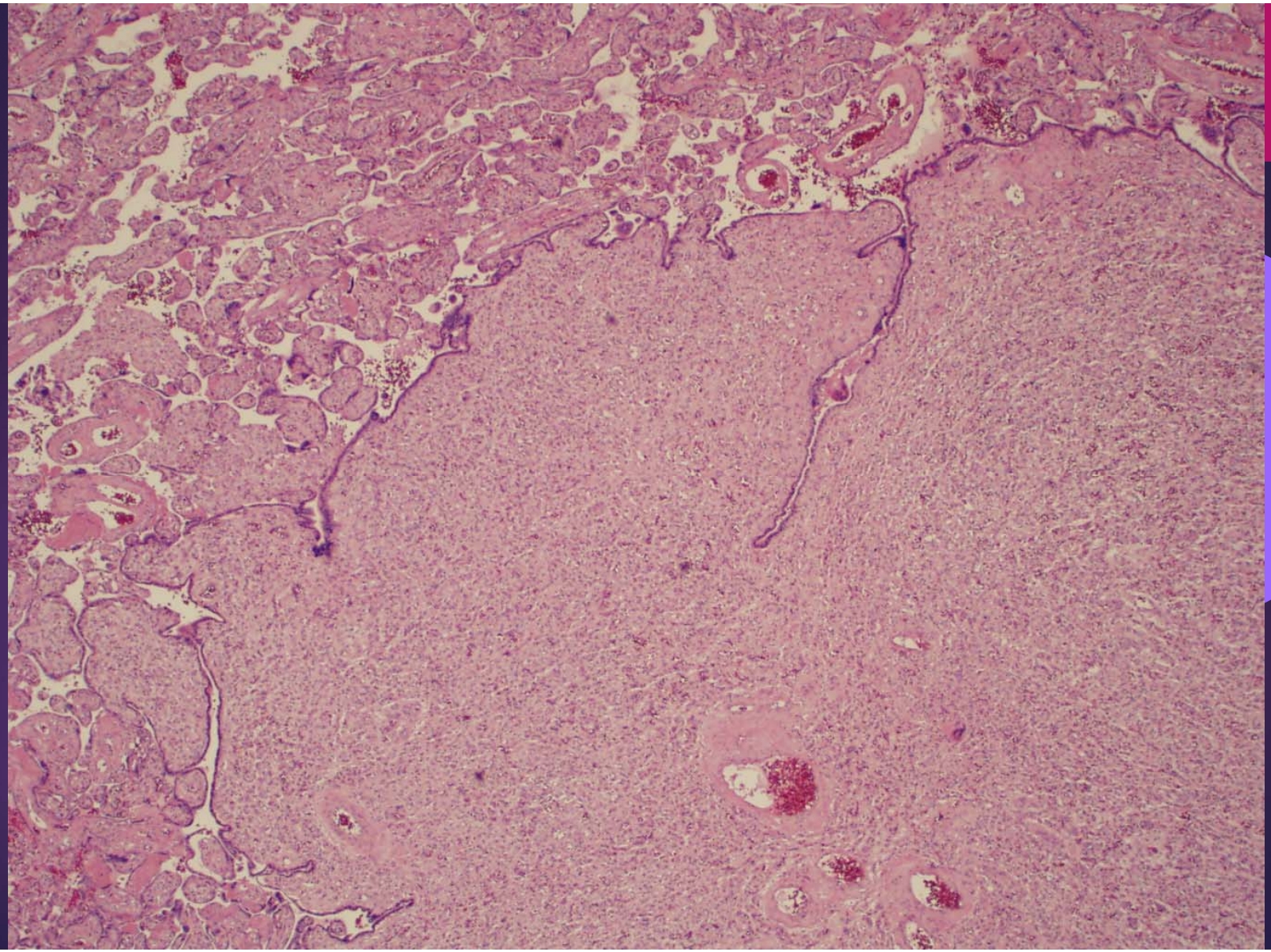
- Developmental
  - Villous capillary lesions
  - Delayed villous maturation
- Malperfusion
  - Global/partial
  - Segmental/complete
- Loss of integrity
  - Fetal or fetomaternal hemorrhage

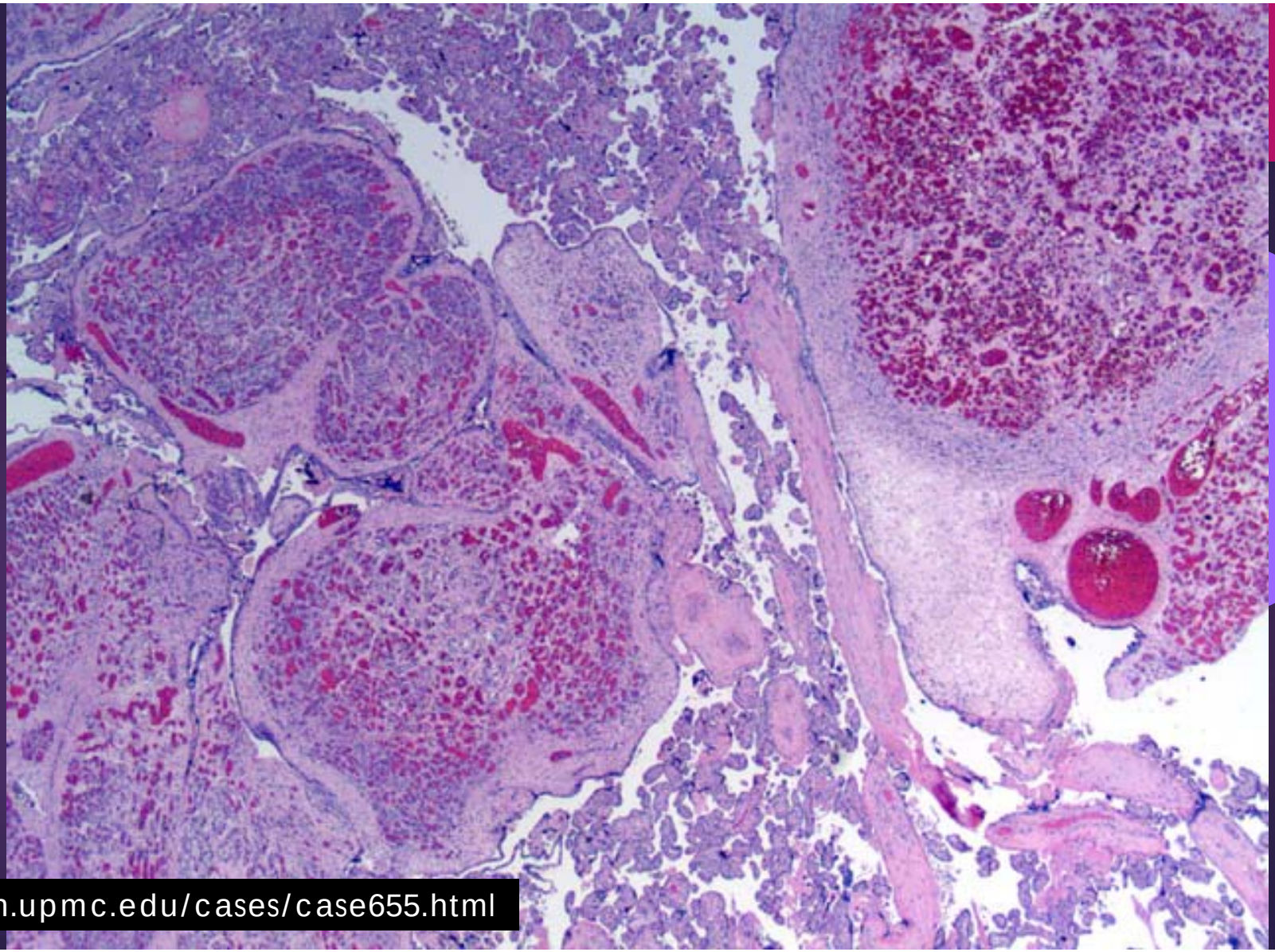
Villous capillary lesions

- Chorangiosis – hypercapillarization of terminal villi
- Chorangioma – benign placental vascular tumor arising in stem villi
- Chorangiomatosis – a more pervasive developmental abnormality involving small vessels at the periphery of immature intermediate villi

- Maternal hypoxia
- Excessive fetal growth factor expression
- Beckwith-Wiedemann







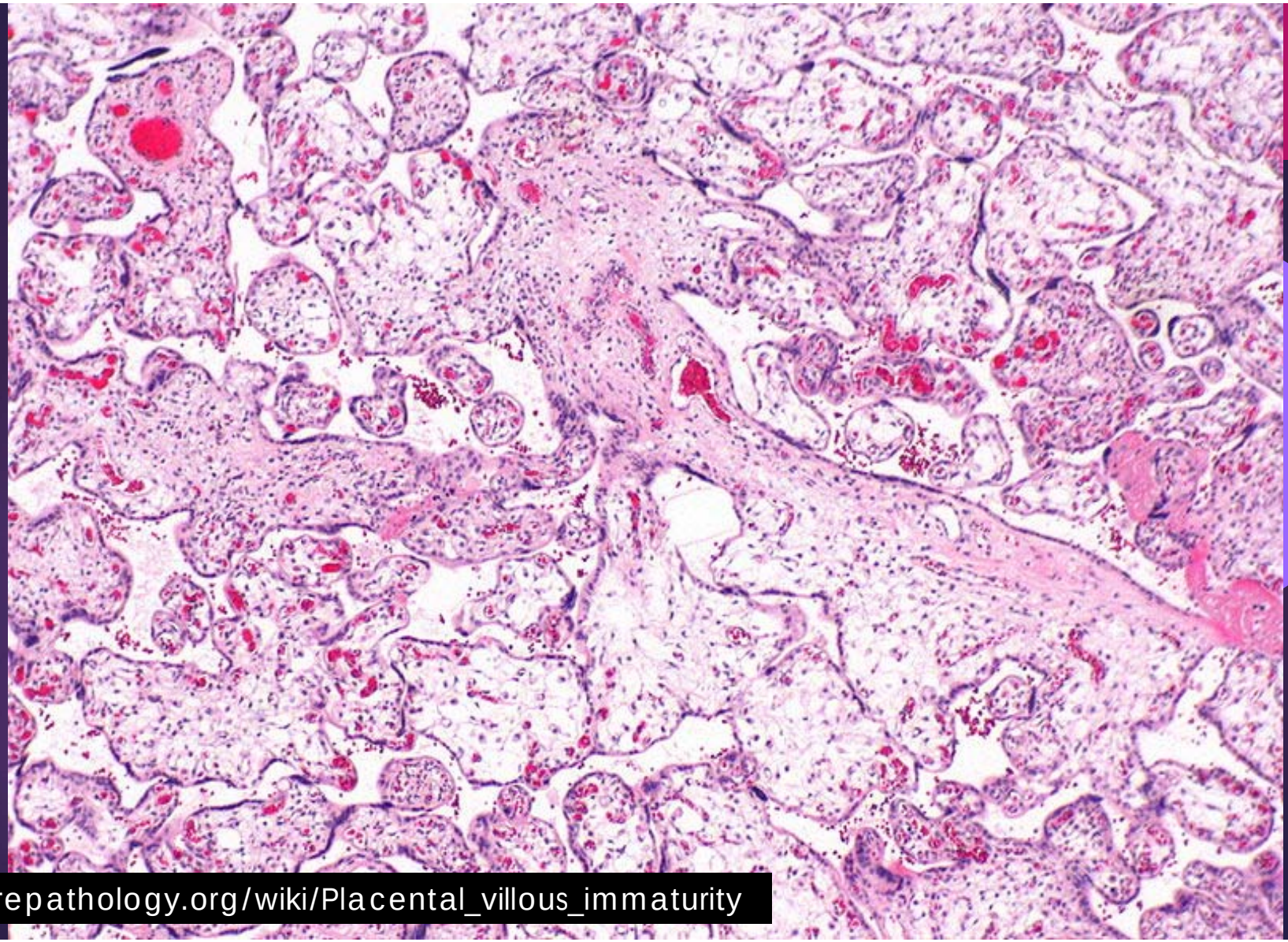
http://path.upmc.edu/cases/case655.html

Delayed villous maturation

- Aka distal villous immaturity

- Usually seen after 36 weeks, rare before 34
- Diabetes, chronic cord obstruction
- Lack of placental reserve increases risk of fetal demise

- Monotonous villous population with centralized capillaries, decreased vasculosyncytial membranes



https://librepathology.org/wiki/Placental_villous_immaturity

Fetal vascular malperfusion

- Preferred term over fetal thrombotic vasculopathy
- Obstruction of fetal blood flow
  - Cord abnormality, hypercoagulability (inherited, diabetes)
- Associated with CNS injury

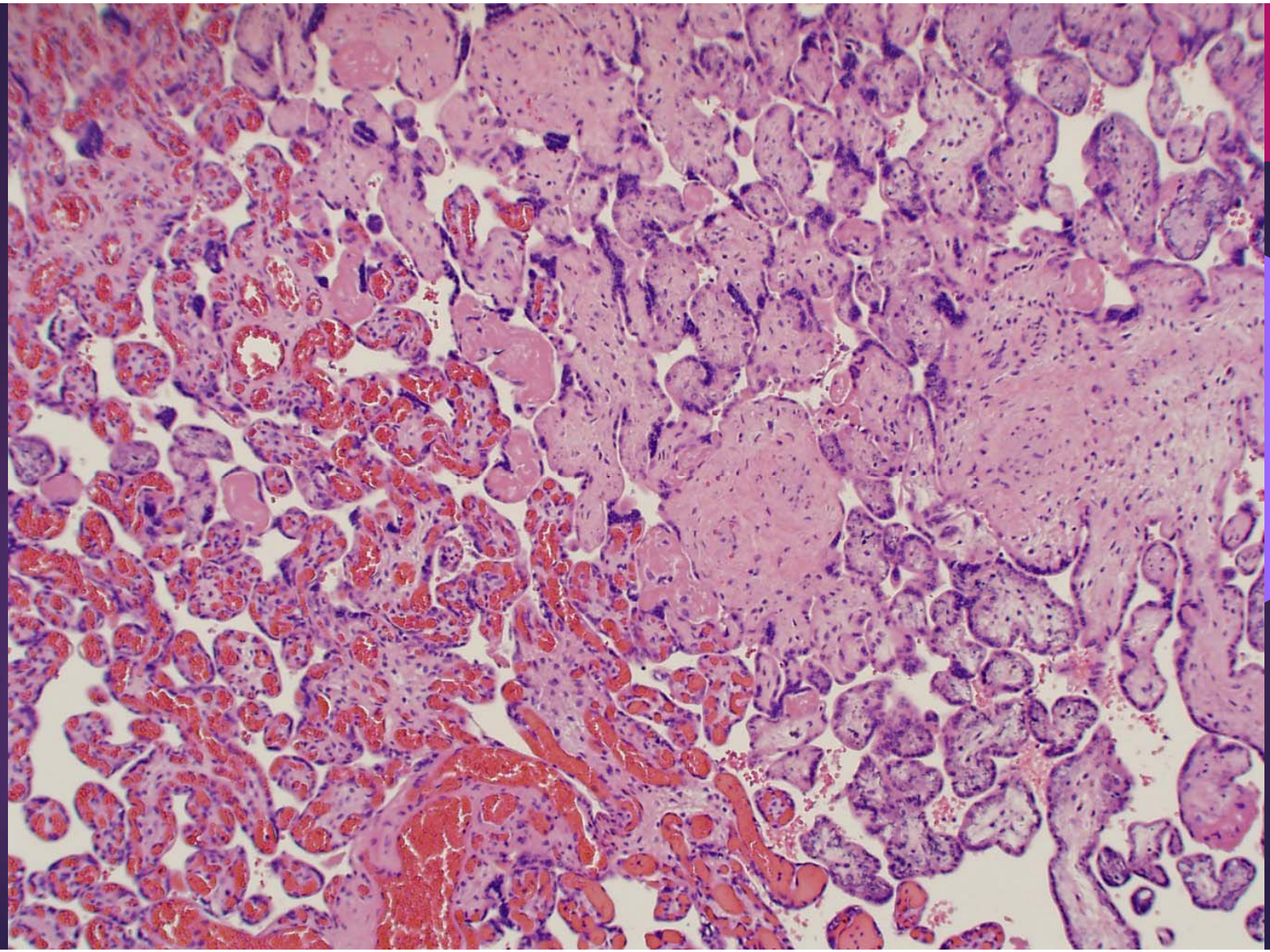
- Global/partial
  - Intermittent, partial obstruction of umbilical cord flow (hypercoiling, stricture, abnormal insertion)
- Segmental/complete
  - Thrombotic occlusion of stem villous vessels

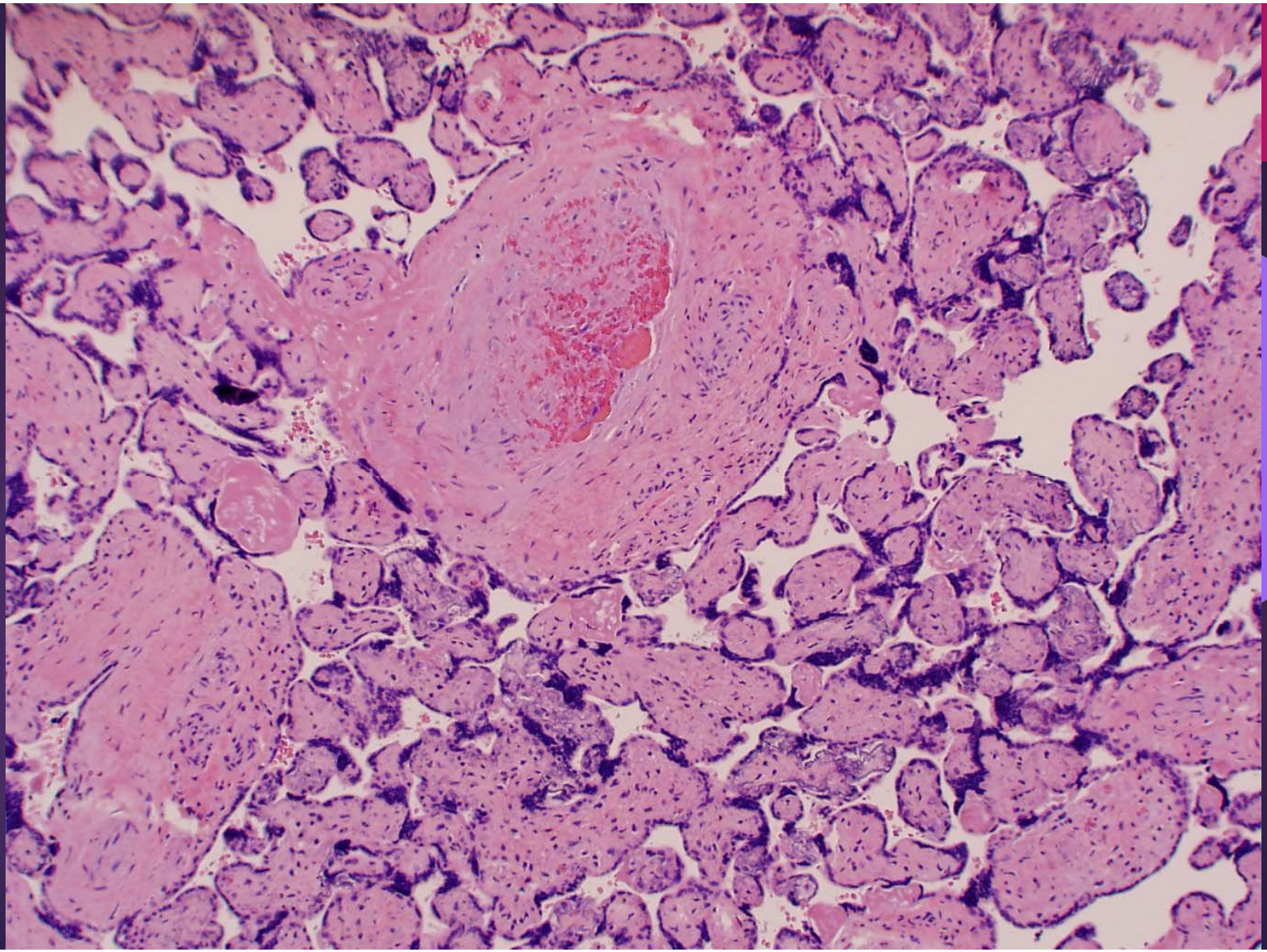
FVM - histology

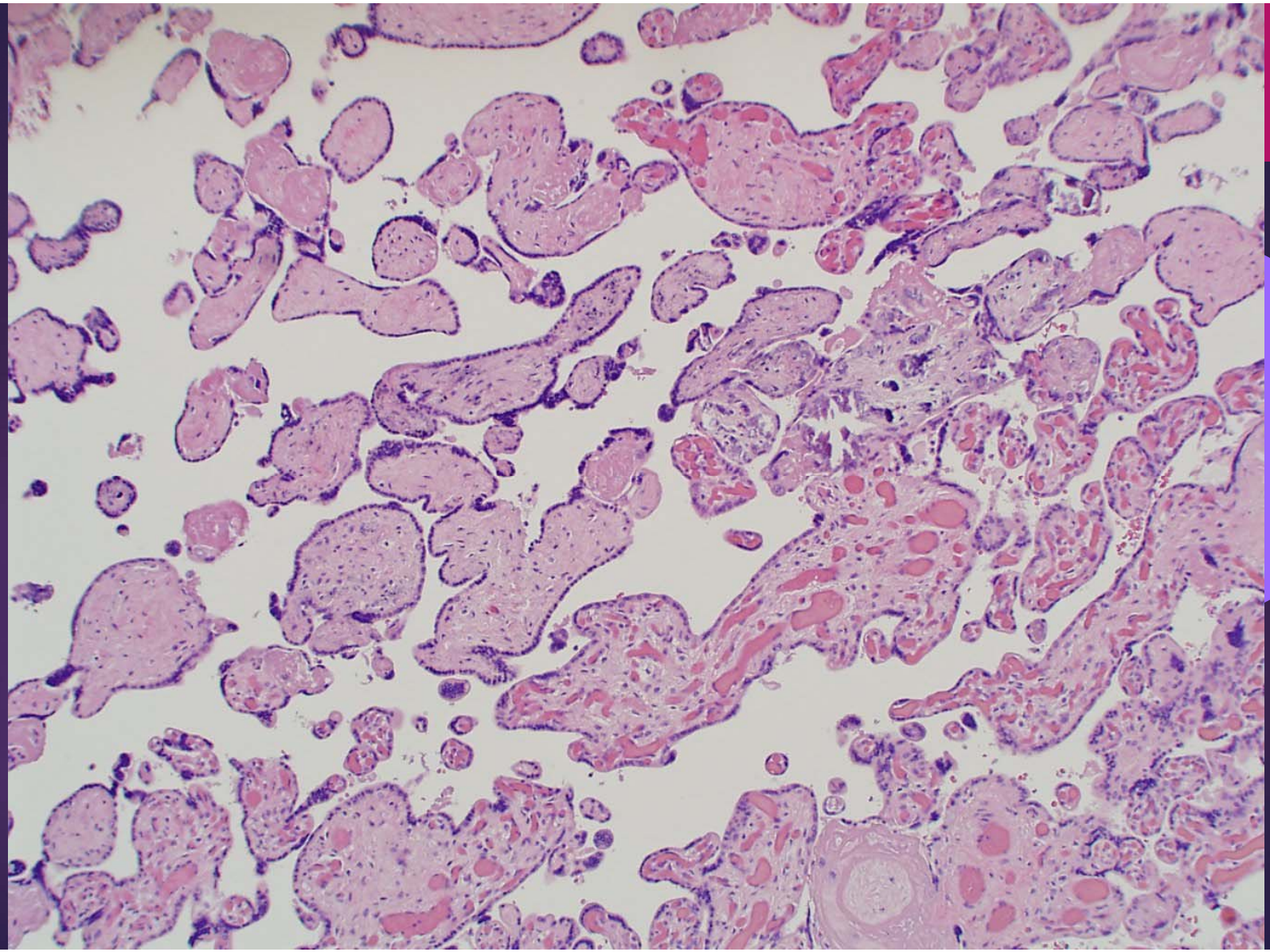
- Thrombosis in fetal vessels
- Segmental avascular villi
- Villous stromal karyorrhexis (preferred term over hemorrhagic endovasculitis)

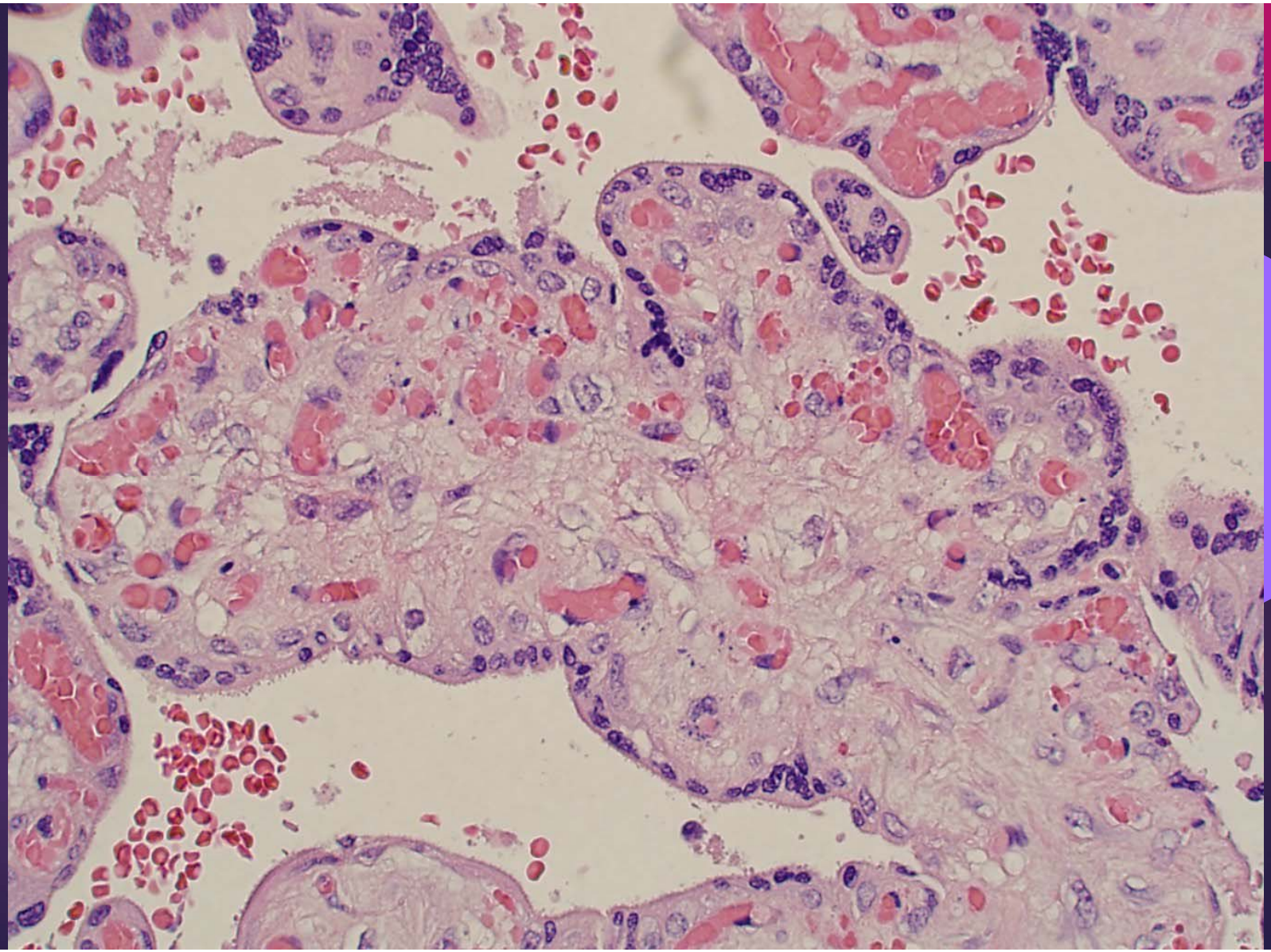
- Global/partial – scattered small foci
- Segmental/complete – larger foci

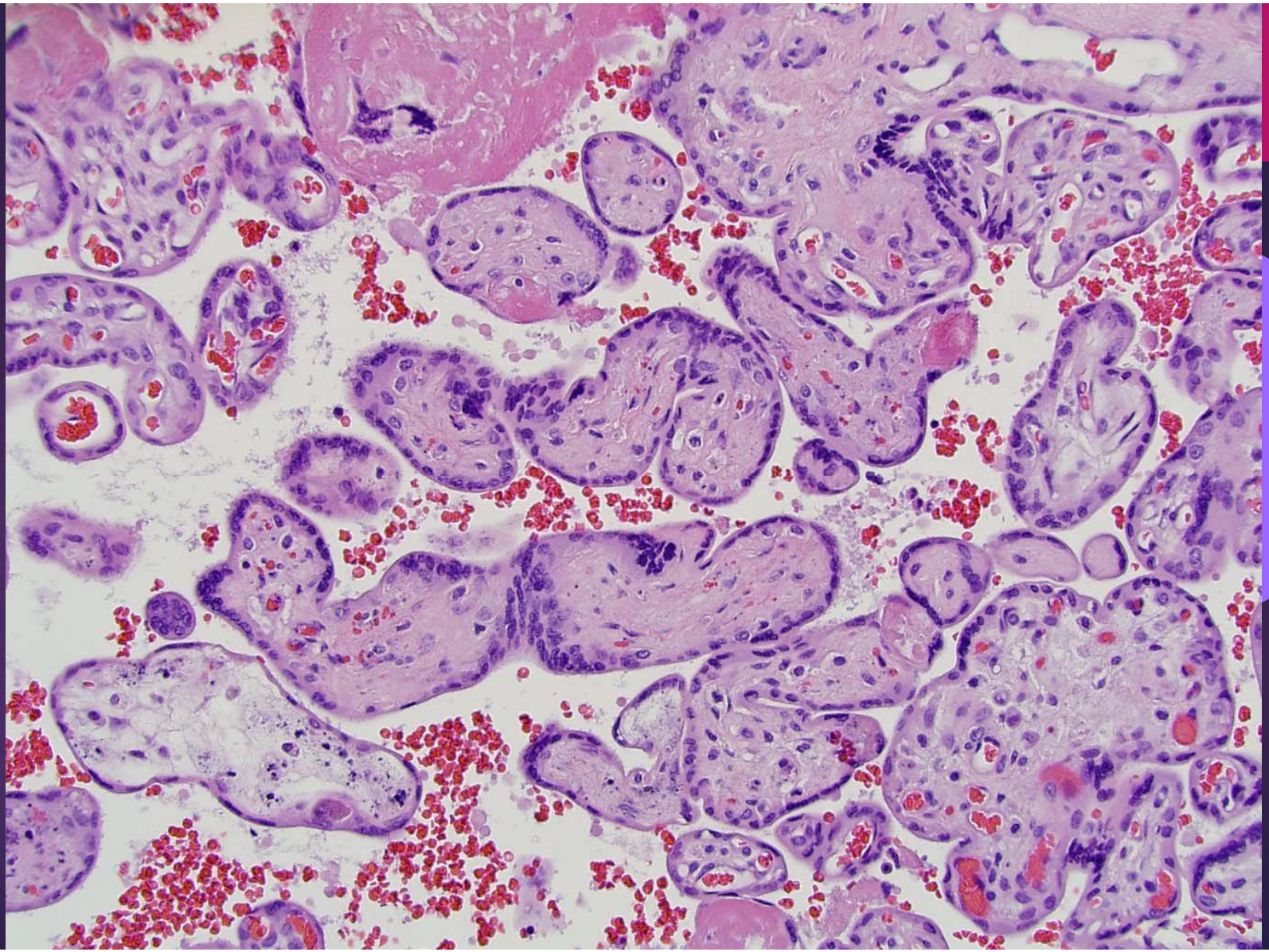
- Difficult diagnosis in stillbirths – look for lesions of varying age

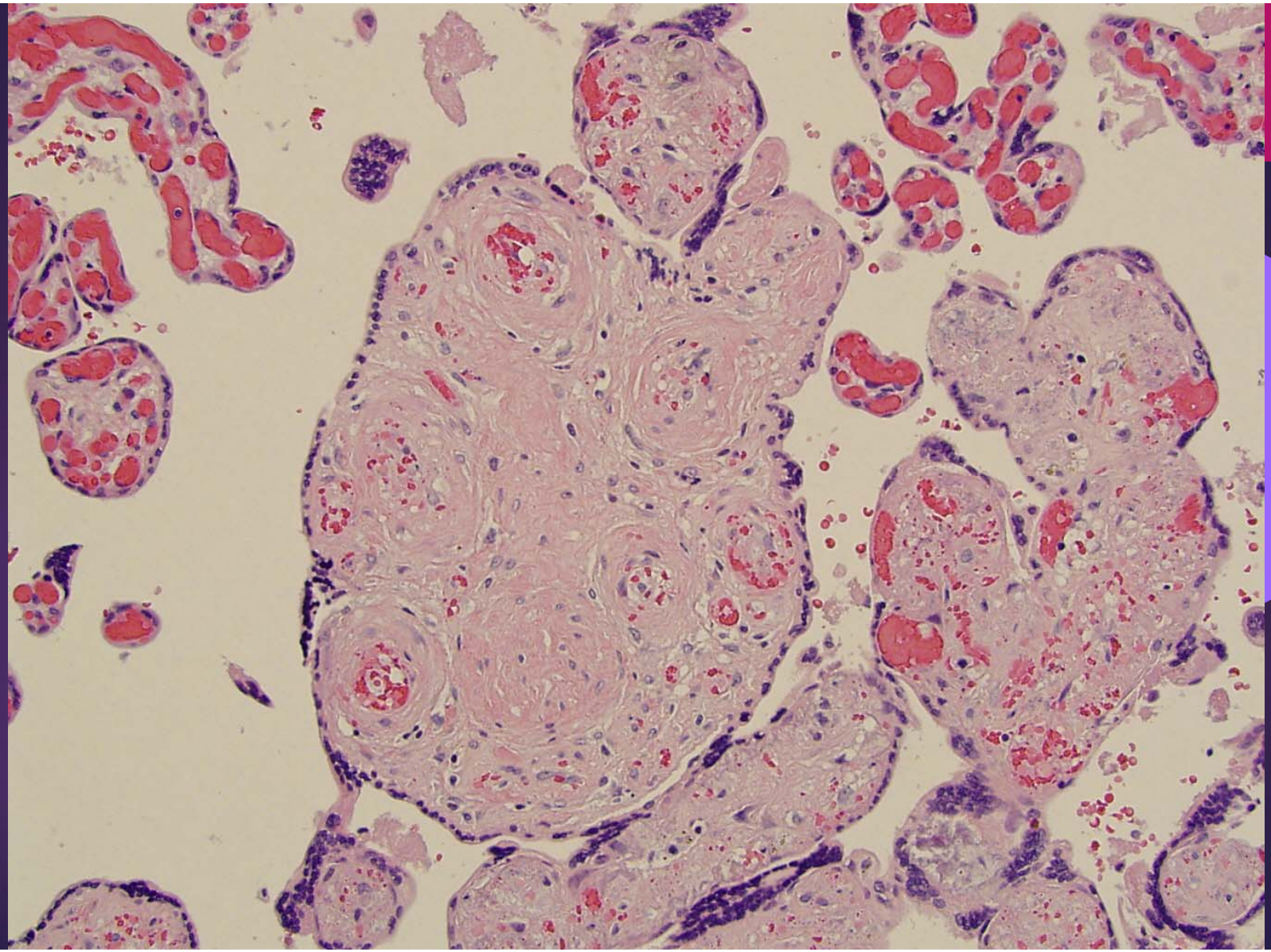






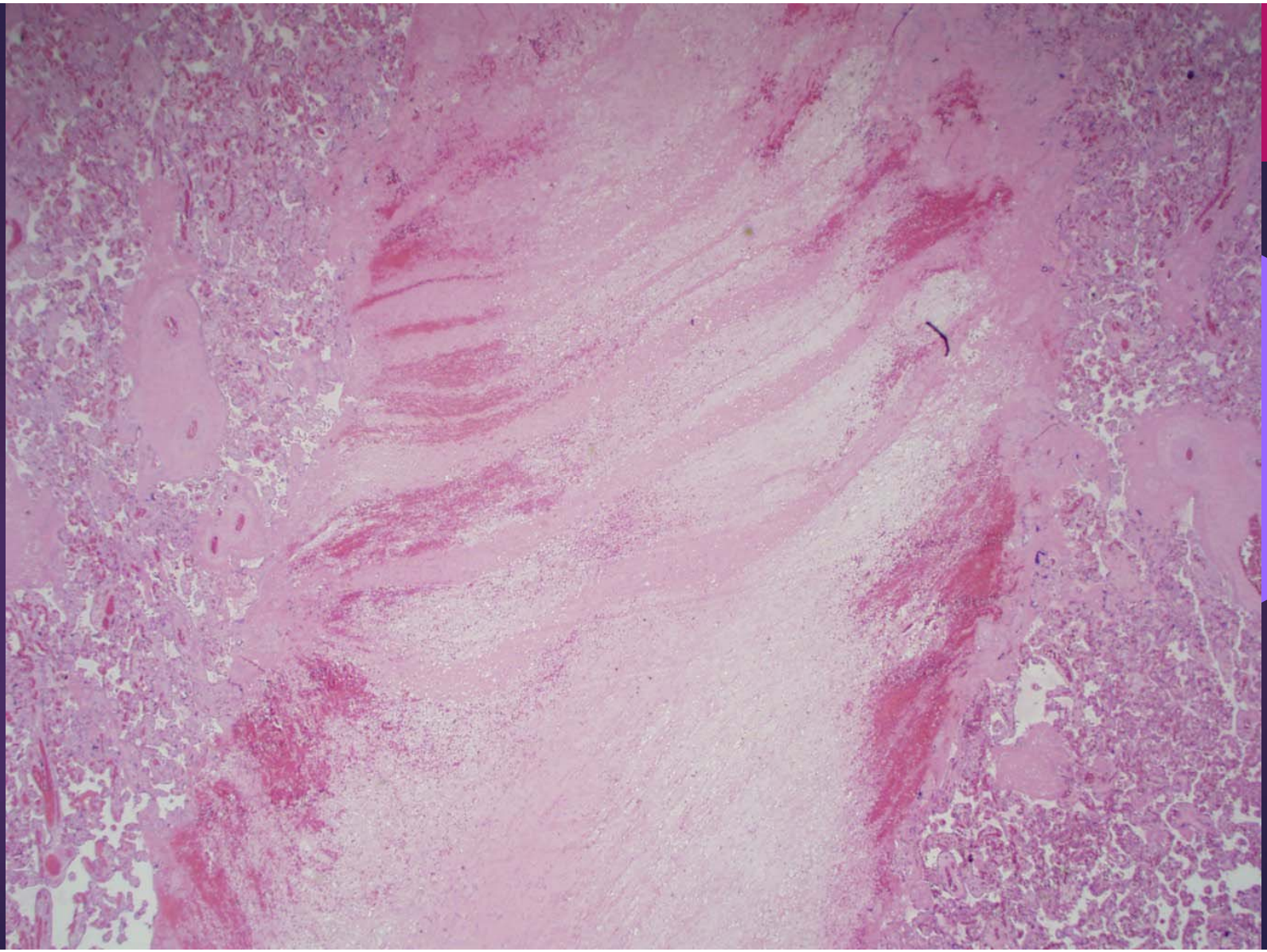






Fetal / fetomaternal hemorrhage

- Fetal – large vessel rupture (e.g. furcate umbilical vessel)
- Fetomaternal – rupture of small vessels in distal villi
  - Intervillous thrombi
  - Increased nRBCs
  - Positive Kleihauer-Betke

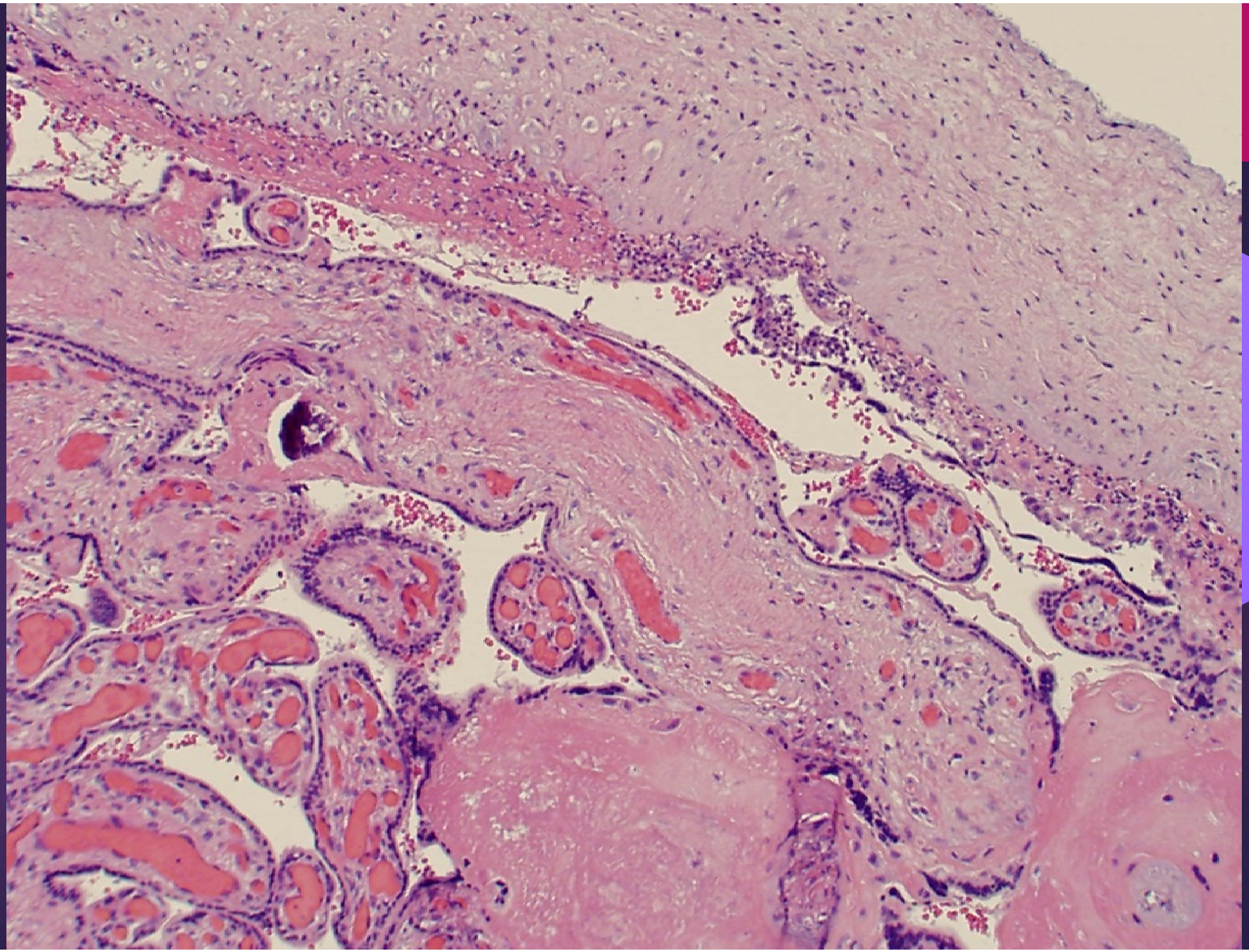


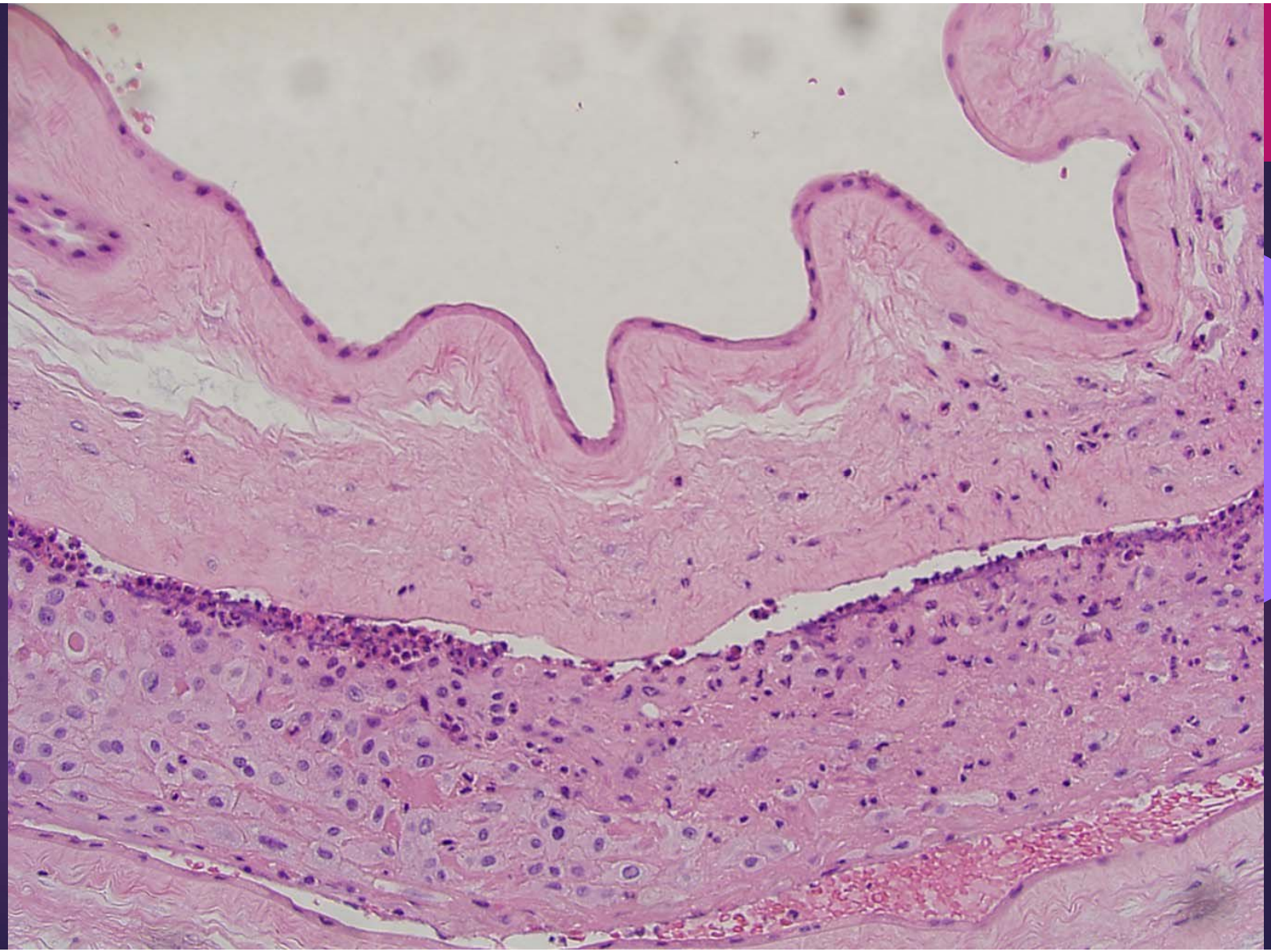
Placental inflammatory-immune processes

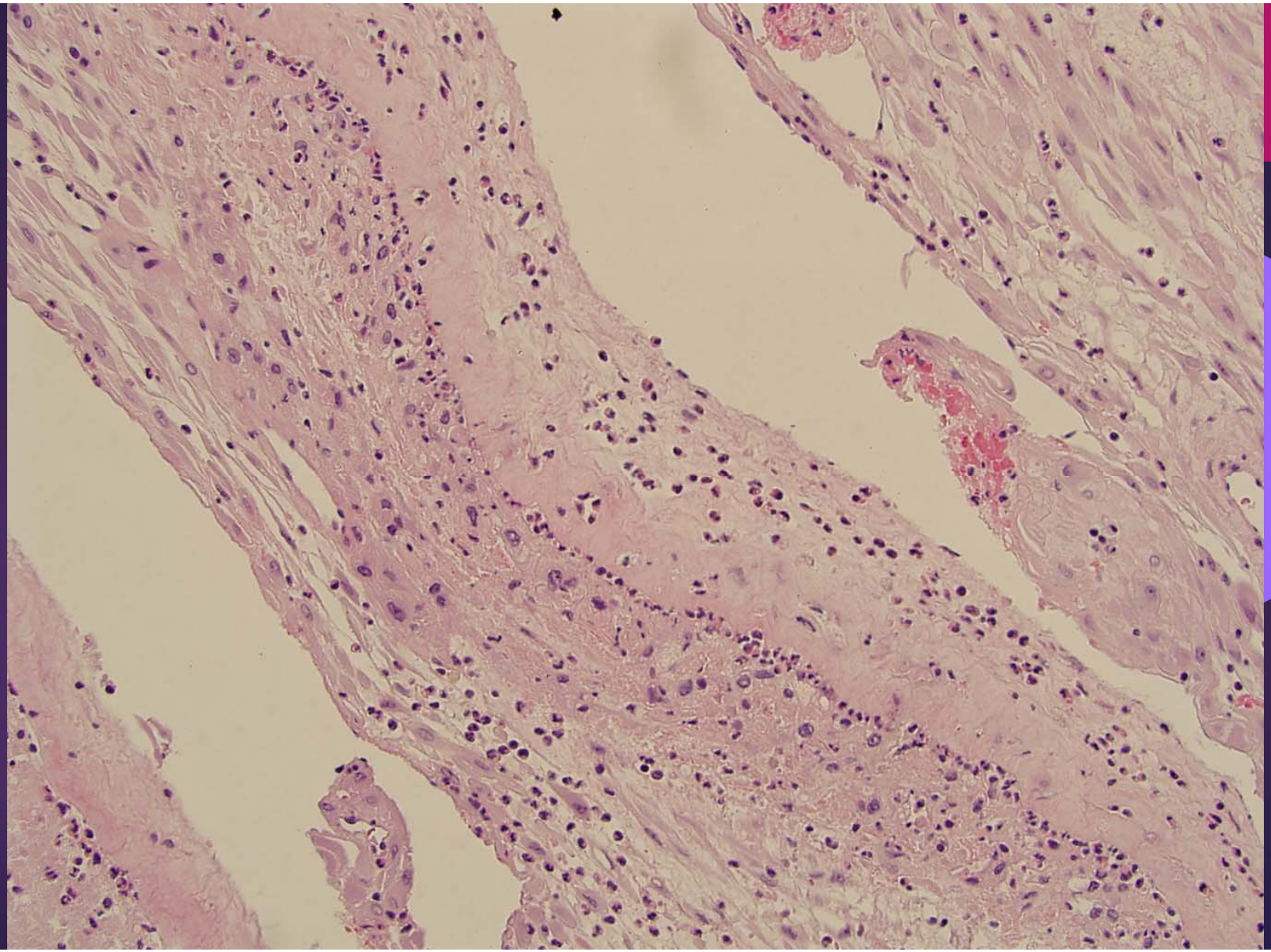
- Infectious lesions
  - Acute
    - Maternal inflammatory response: subchorionitis, chorioamnionitis
    - Fetal inflammatory response: chorionic/umbilical vasculitis
  - Chronic
    - TORCH, malaria, others
- Immune/idiopathic
  - Villitis of unknown etiology
  - Lymphoplasmacytic deciduitis
  - Chronic histiocytic intervillositis

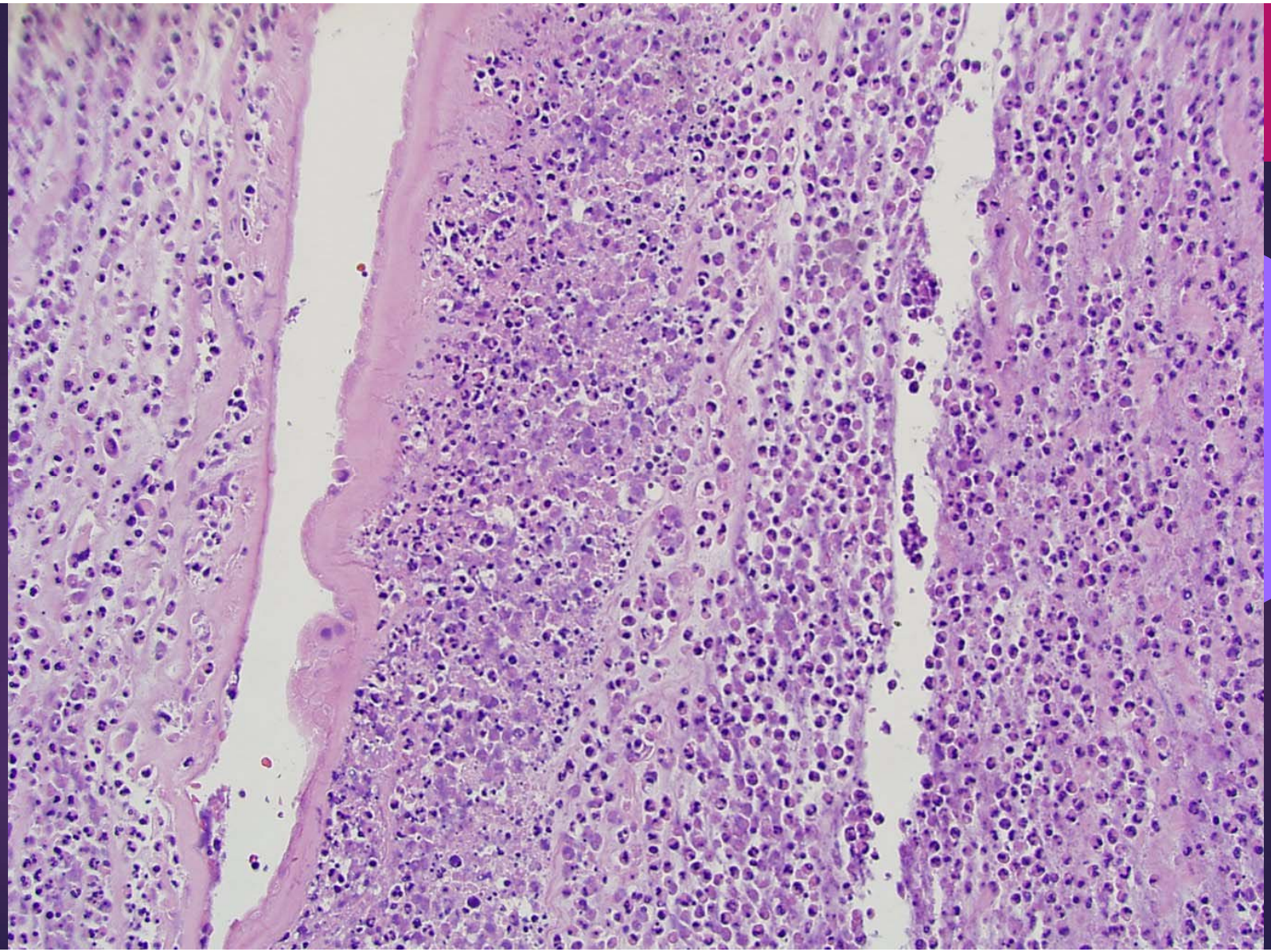
Maternal inflammatory response

- Stage
  - 1 – early – acute subchorionitis and/or acute chorionitis
  - 2 – intermediate – acute chorioamnionitis
  - 3 – late – necrotizing chorioamnionitis
- Grade
  - 1 – mild (not severe)
  - 2 – severe (>30 PMNs/hpf, confluent PMNs, microabscesses)



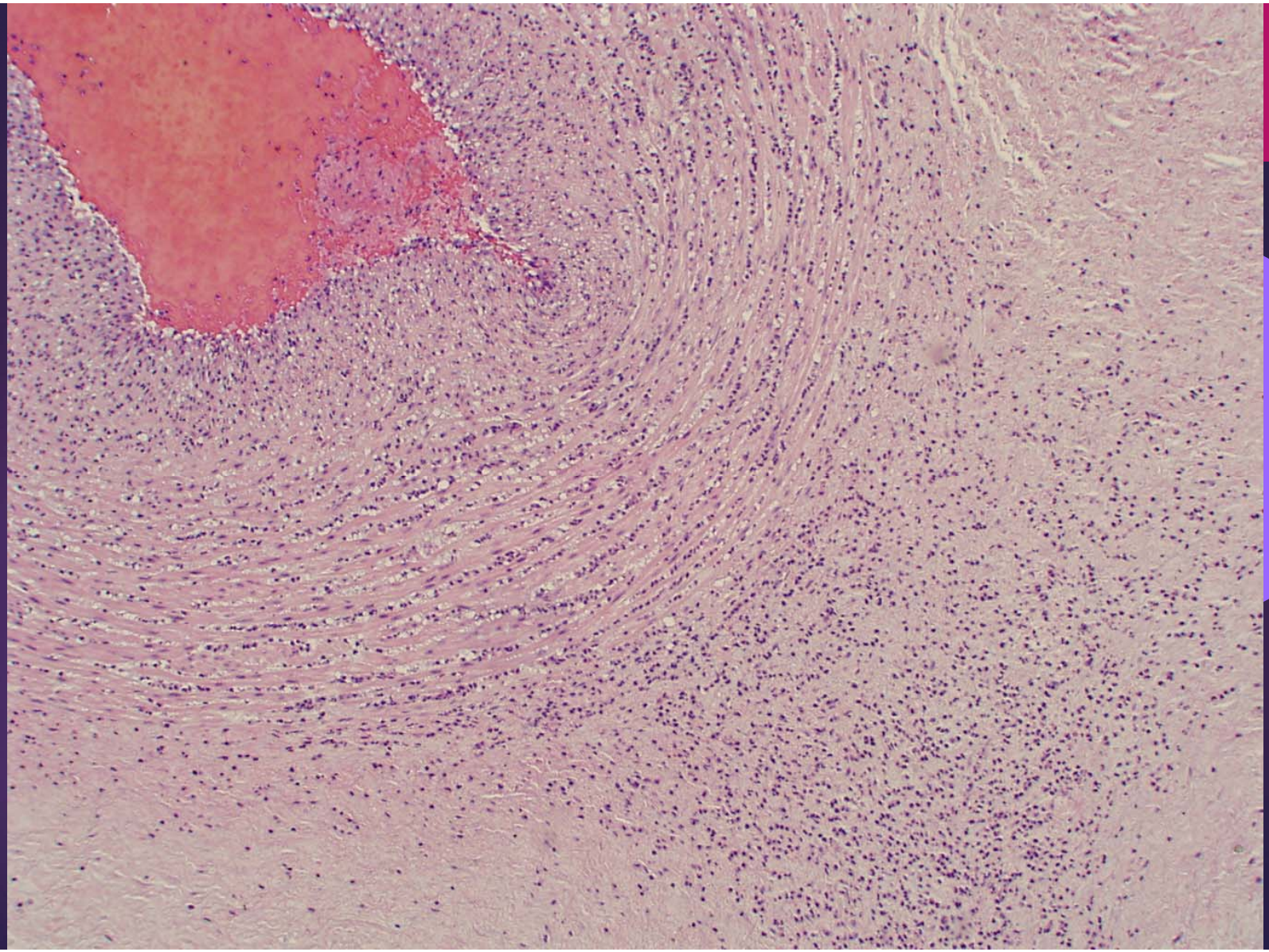


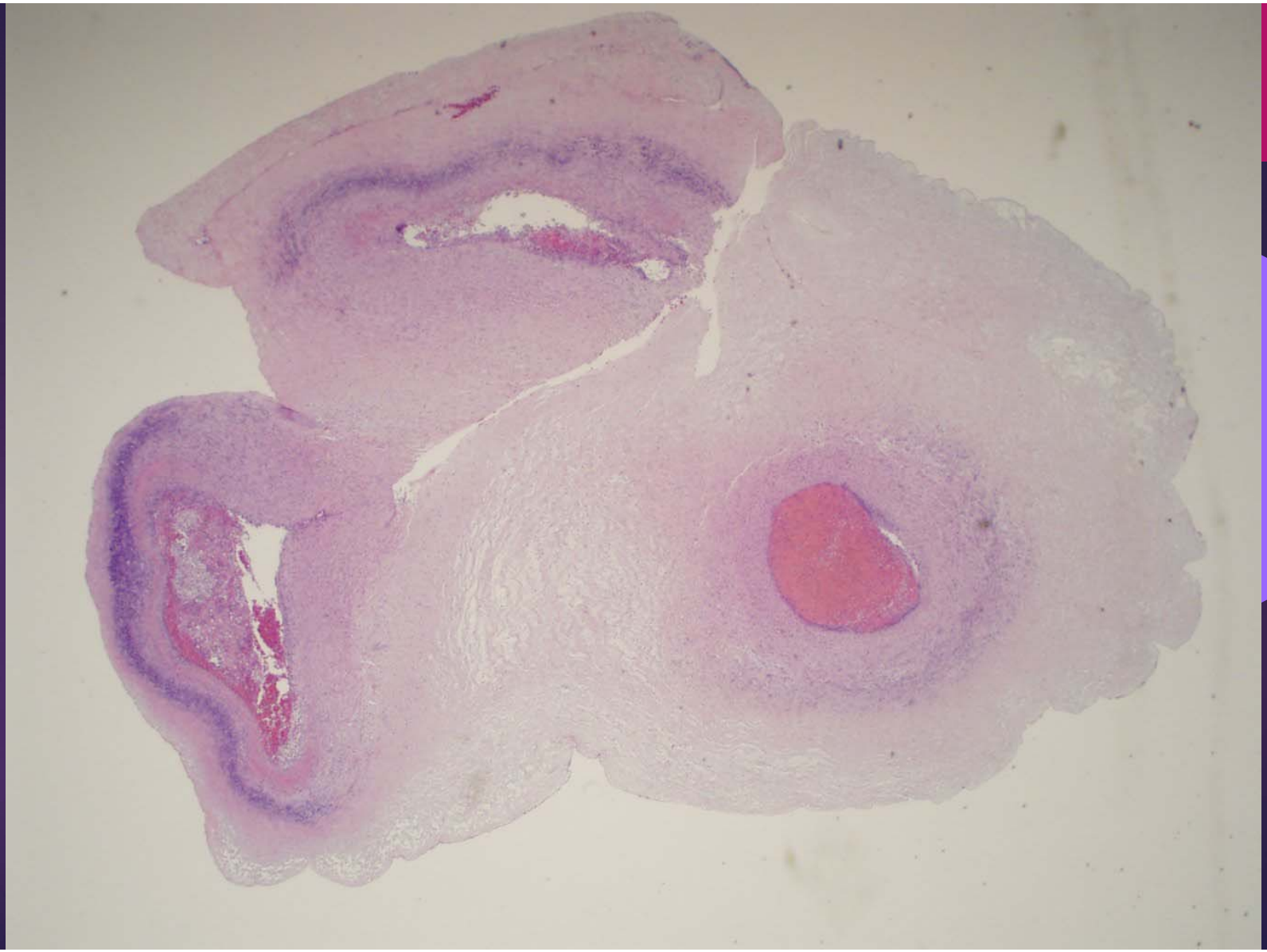


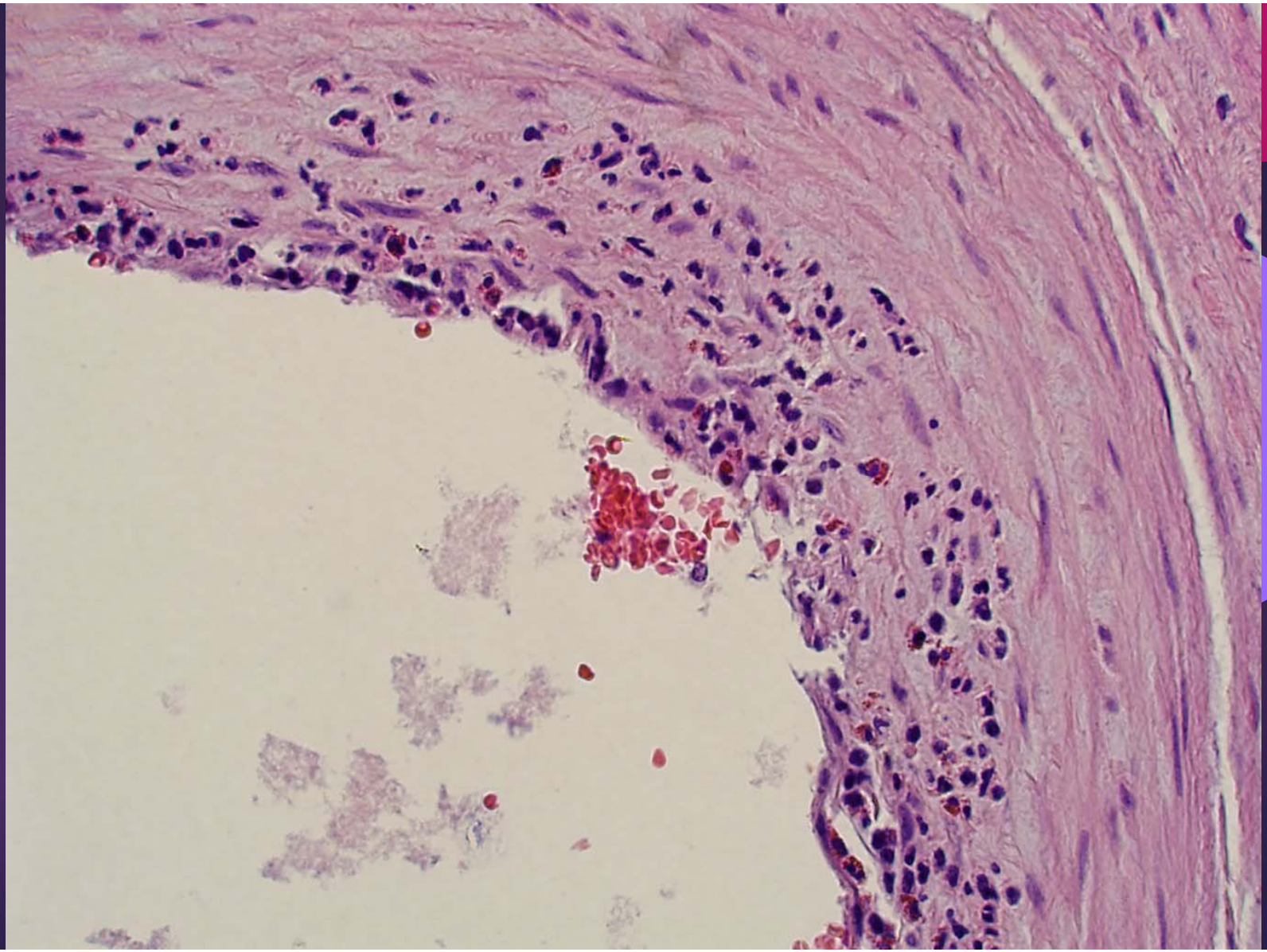


Fetal inflammatory response

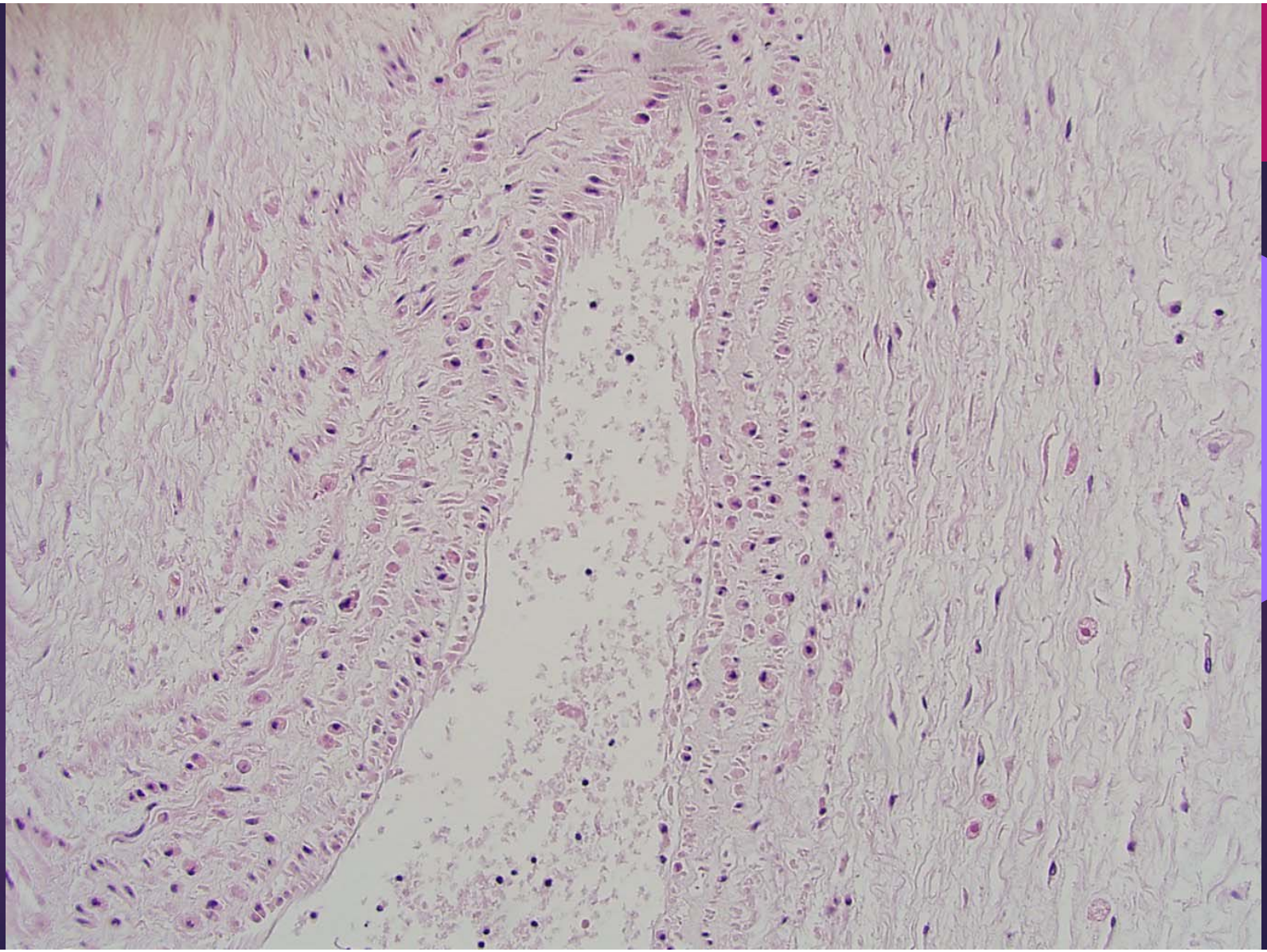
- Stage
  - 1 – early – umbilical phlebitis and/or chorionic plate vasculitis
  - 2 – intermediate – umbilical arteritis
  - 3 – late – necrotizing funisitis and/or concentric umbilical perivasculitis
- Grade
  - 1 – mild (not severe)
  - 2 – severe (near-confluent intramural PMNs with attenuation of vascular smooth muscle)
    - Associated with CNS injury



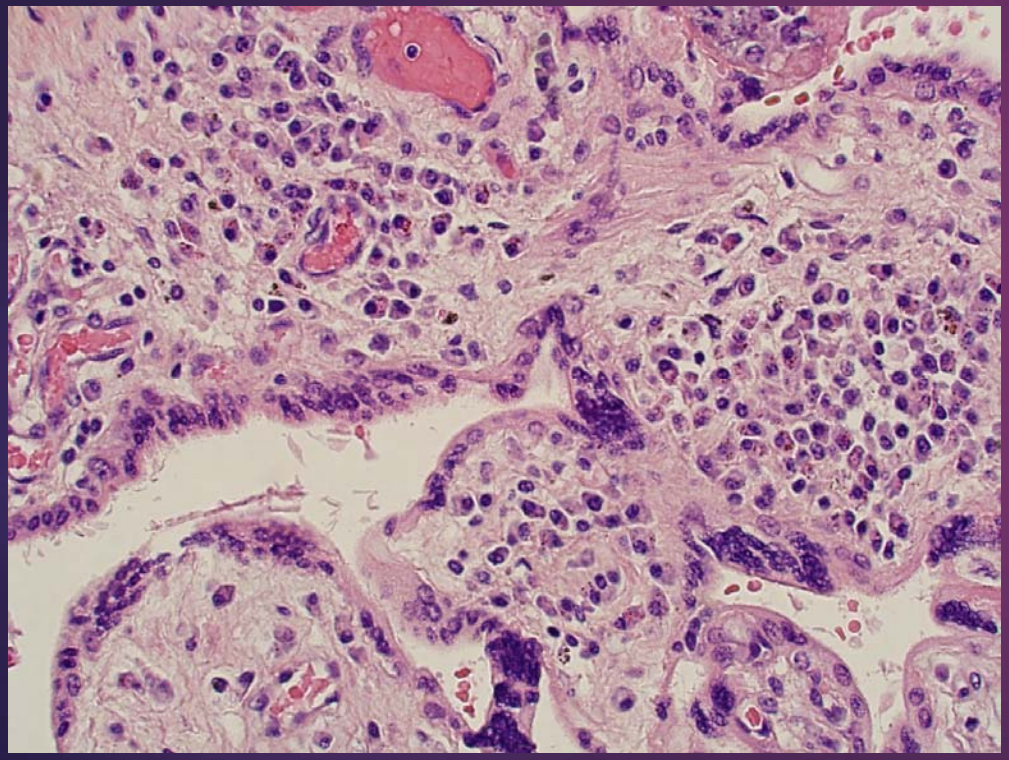


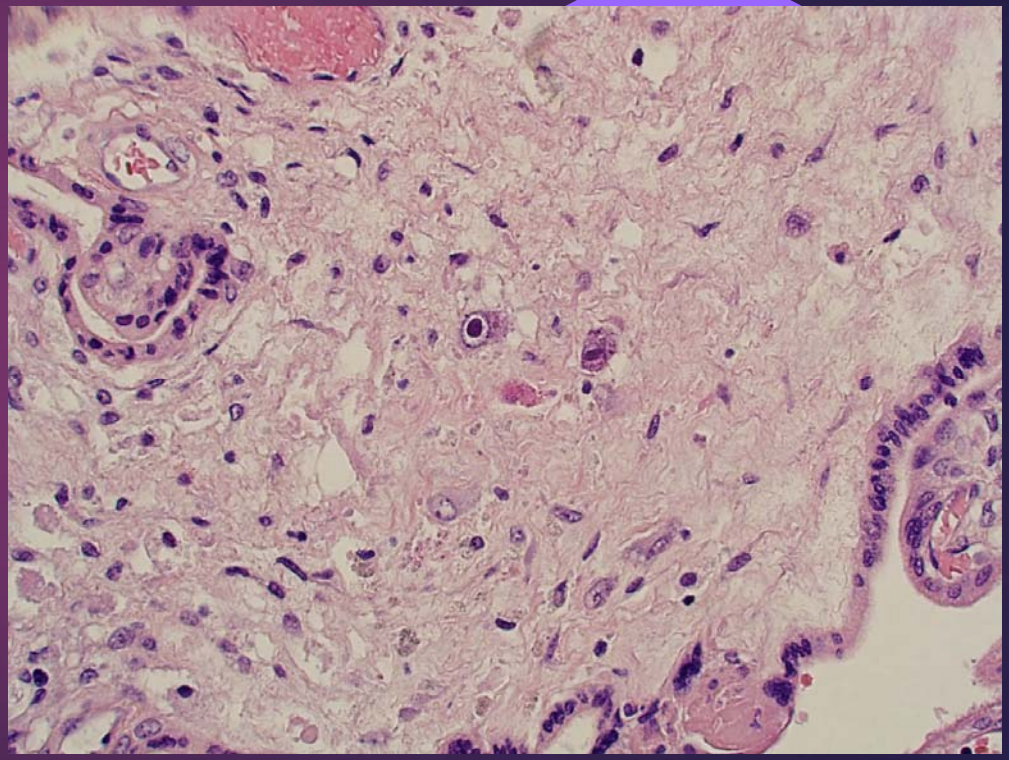


BEWARE!!



CMV villitis





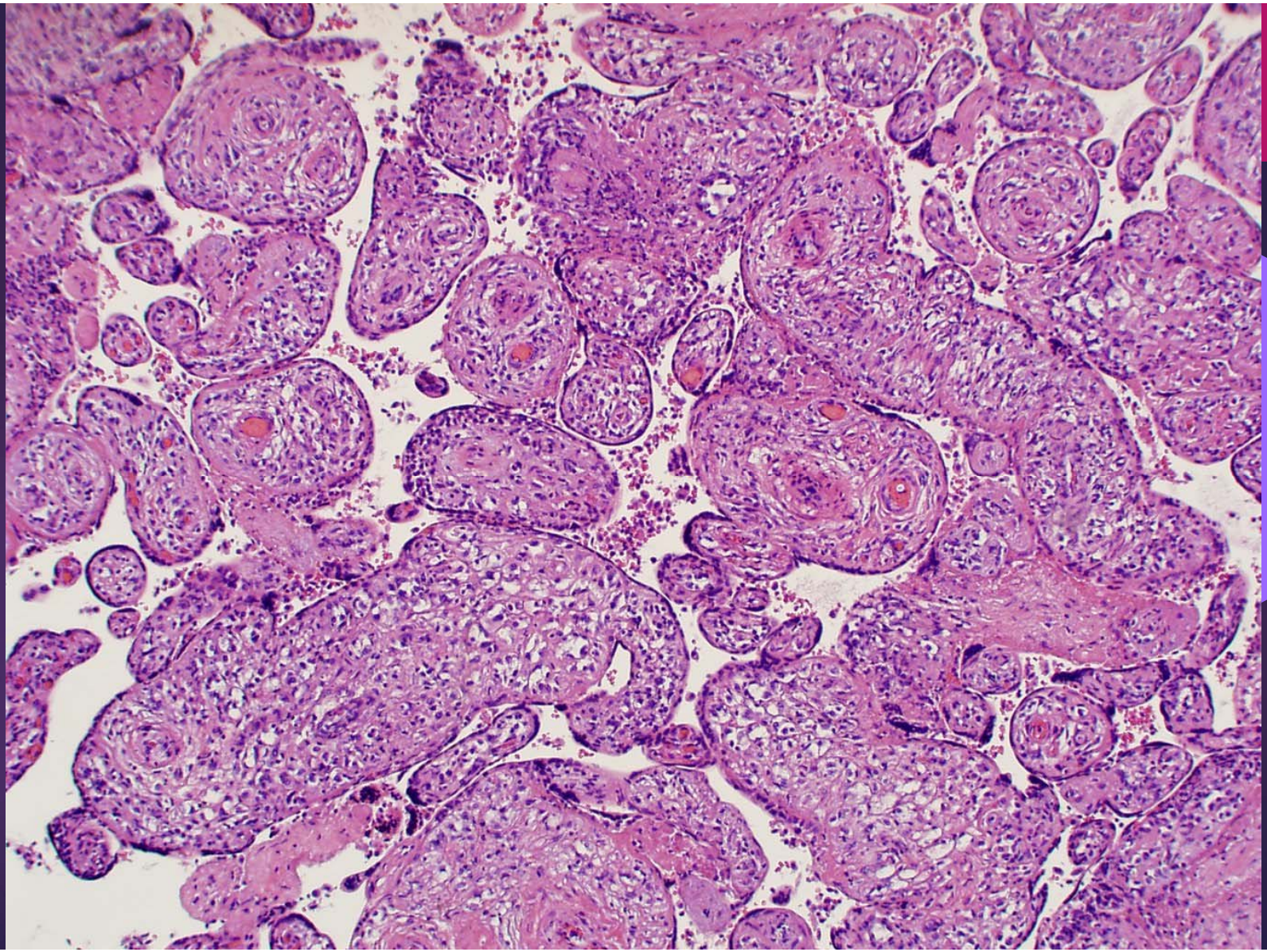
Chronic villitis of unknown etiology

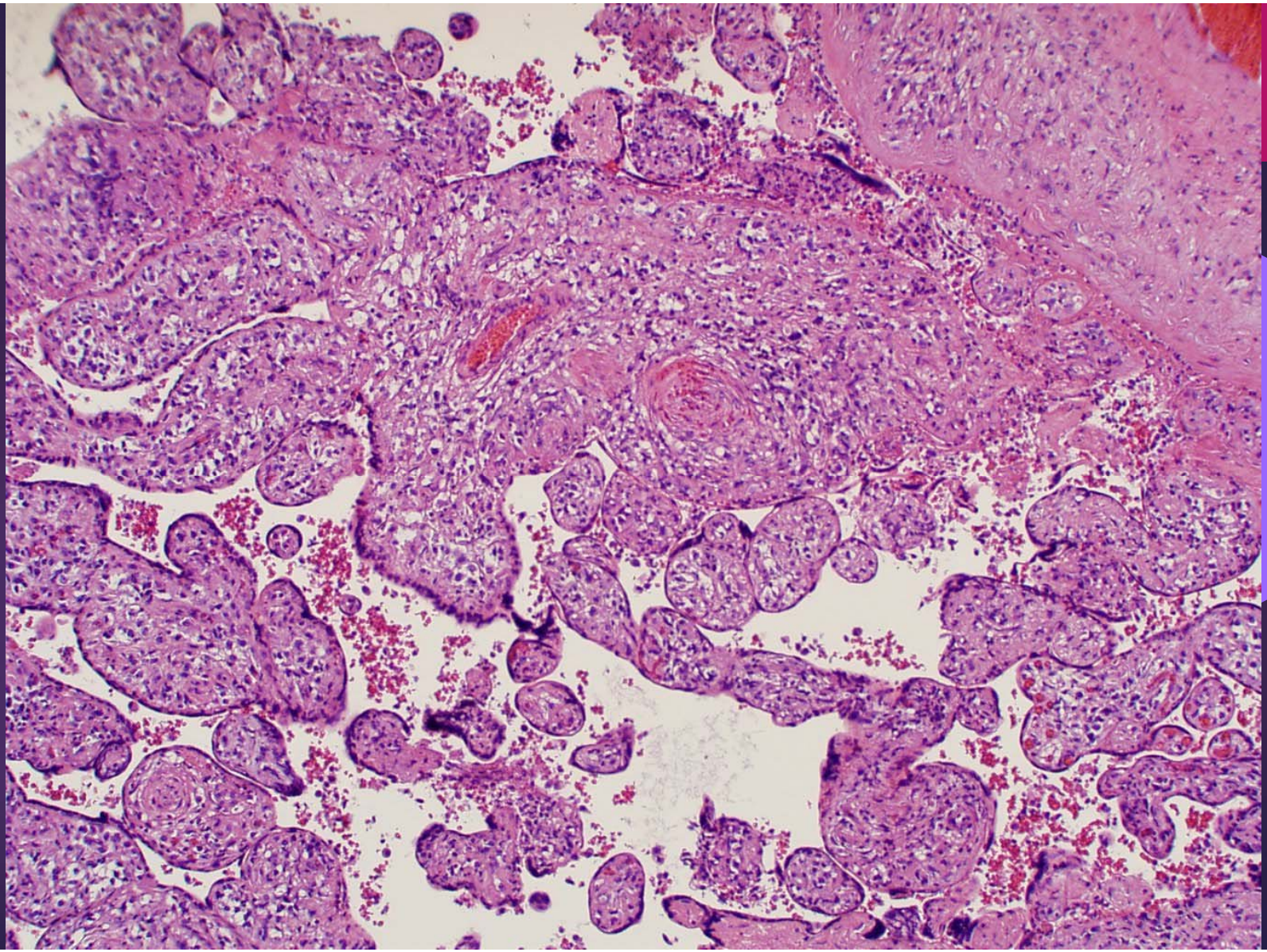
- T-cell mediated disorder targeting distal villi
- Maternal graft-vs-host-type response
- High-grade VUE associated with growth restriction, CNS injury, fetal demise
- 5-10% of term placentas
- Increased incidence and severity in obese women
- Significant recurrence risk (25-50%)

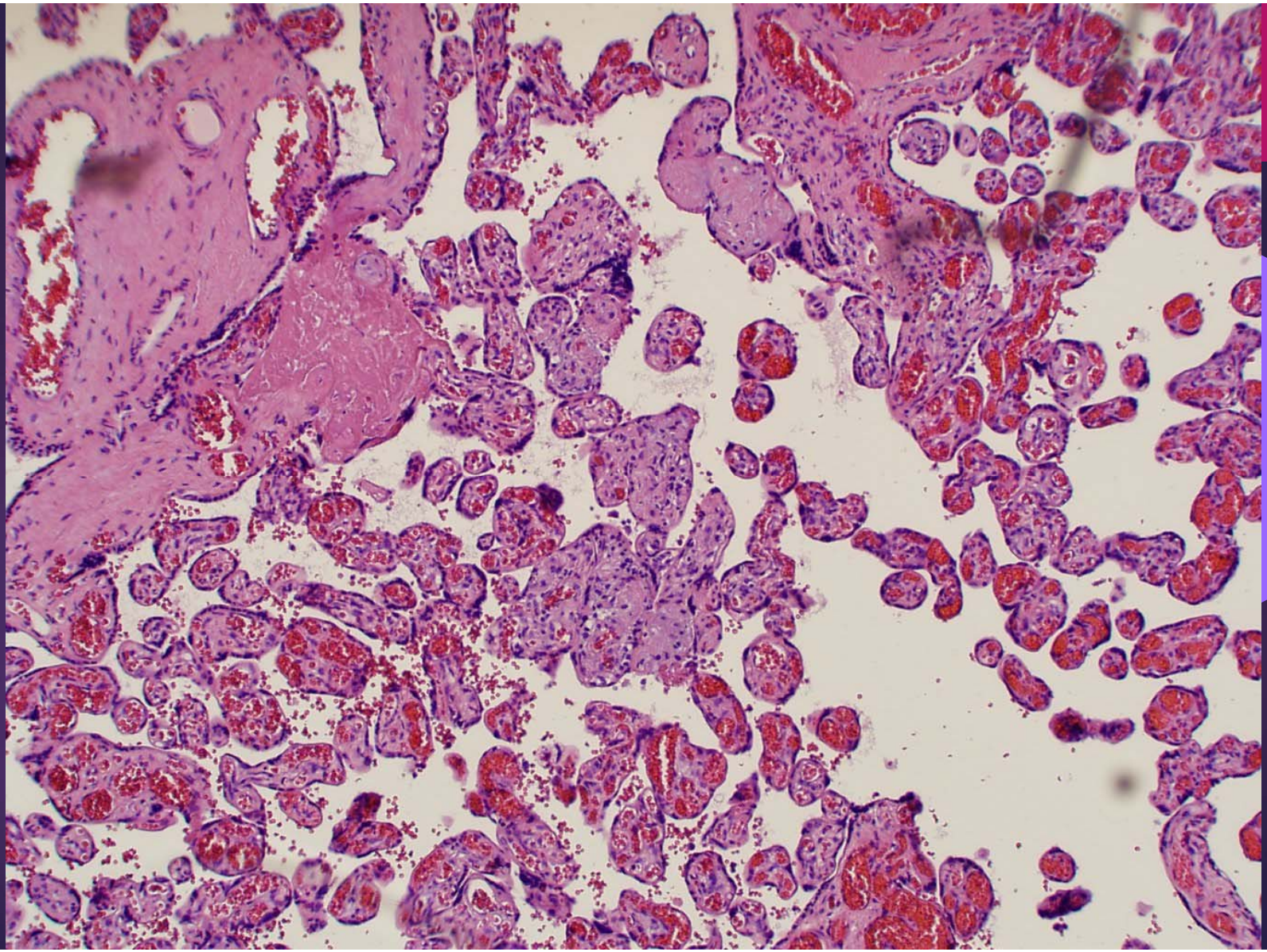
VUE - histology

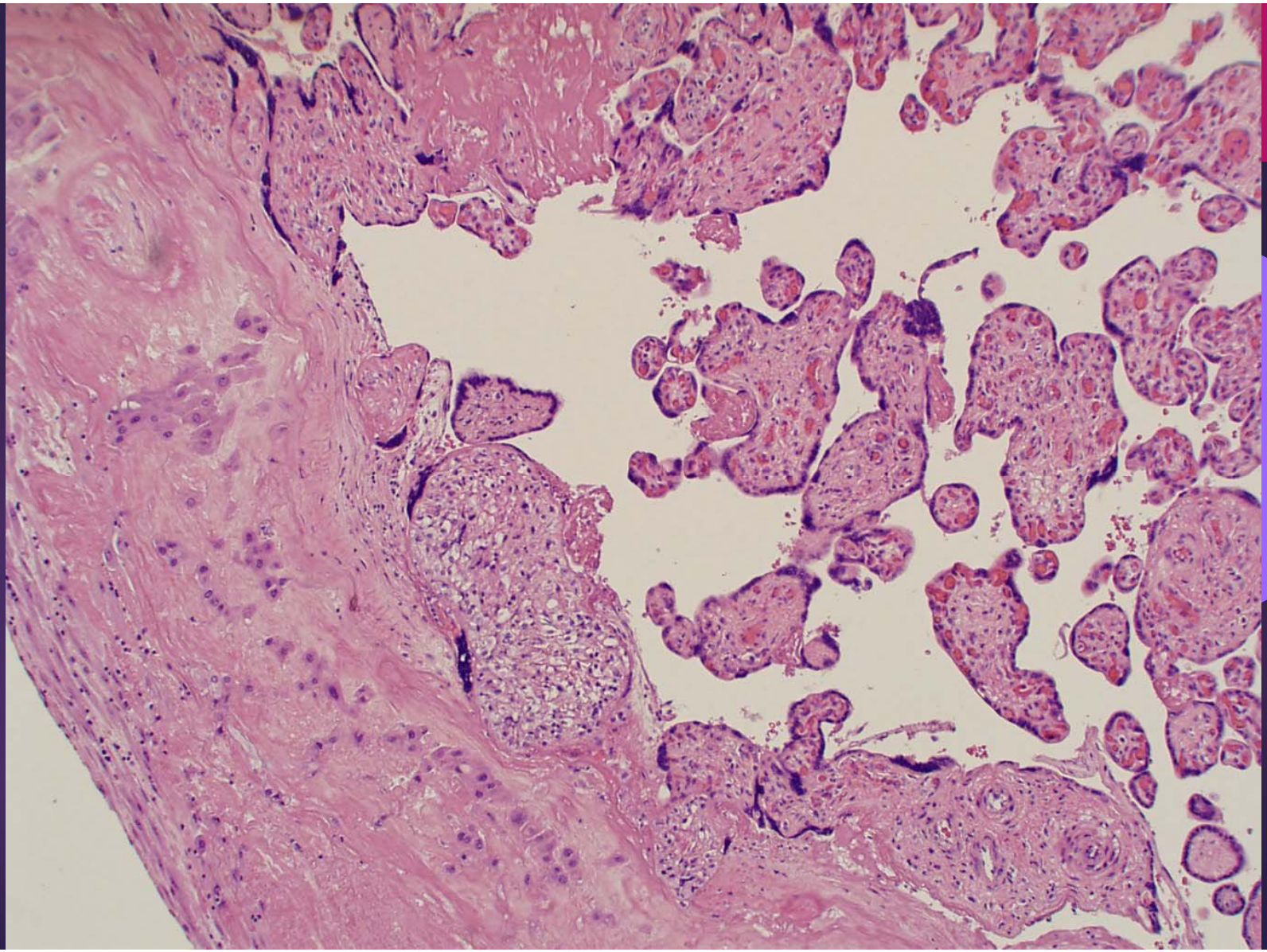
- Lymphohistiocytic inflammation of villi and (sometimes) intervillous space and stem villous vessels
- Low- vs high-grade
- Often basal / parabasal (more frequent with ART)

- Also can see chronic chorioamnionitis, lymphoplasmacytic deciduitis, eosinophilic T-cell vasculitis



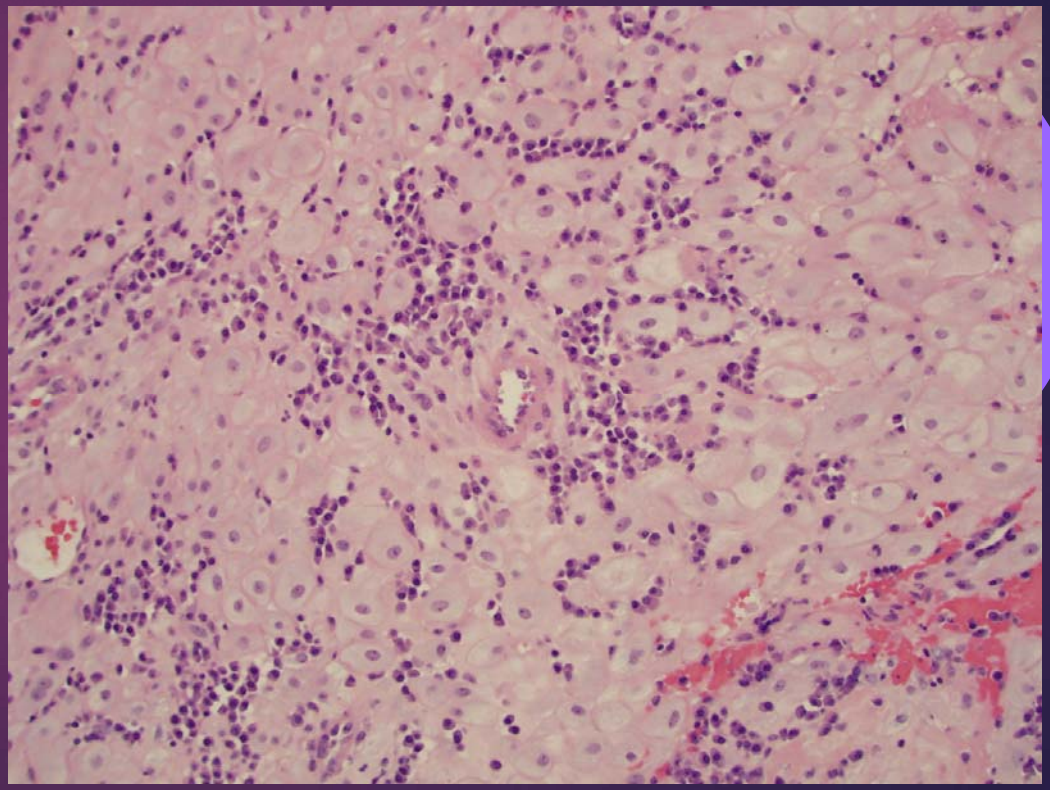






Lymphoplasmacytic deciduitis

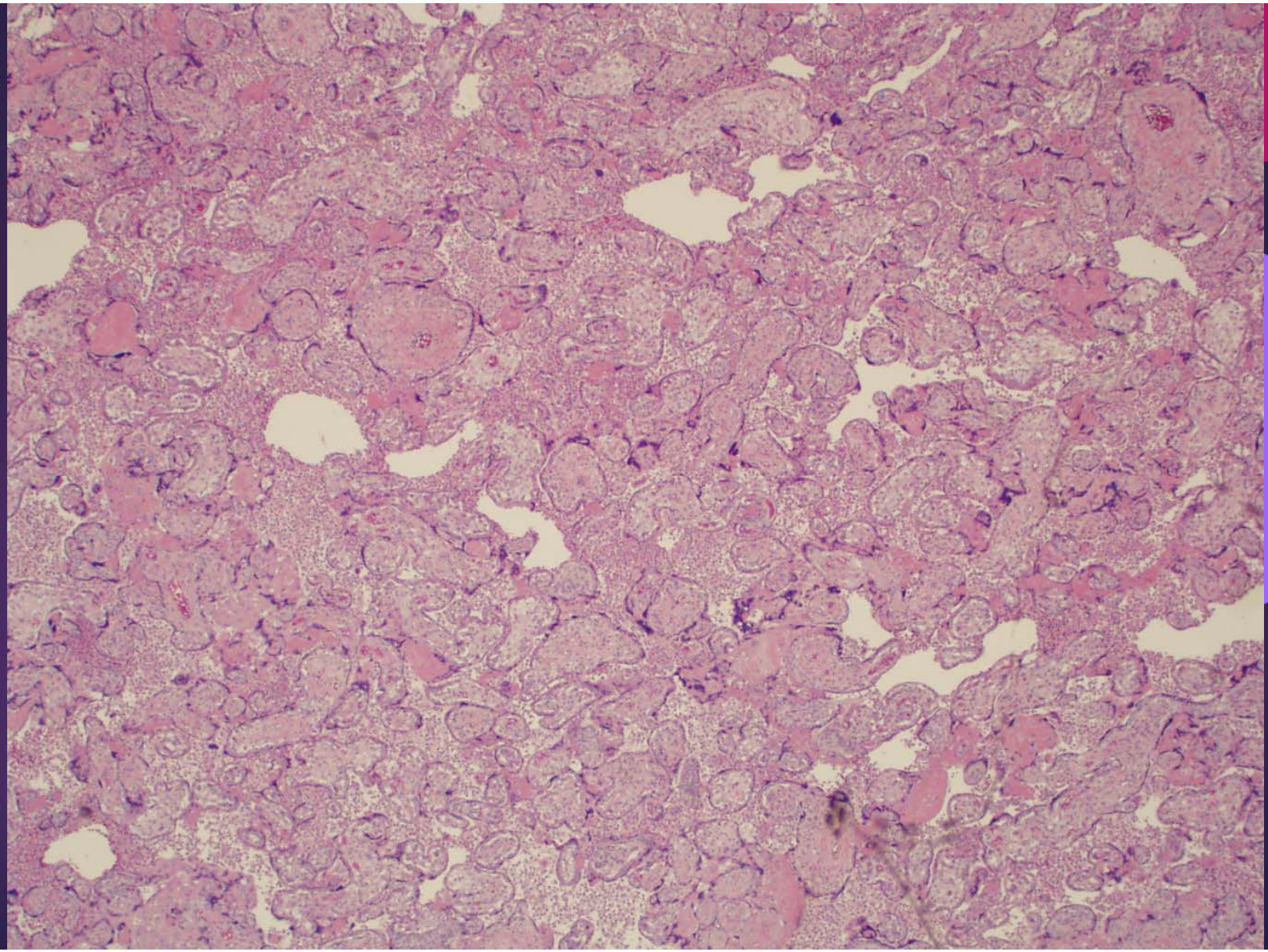
- Infectious vs. autoimmune vs. idiopathic

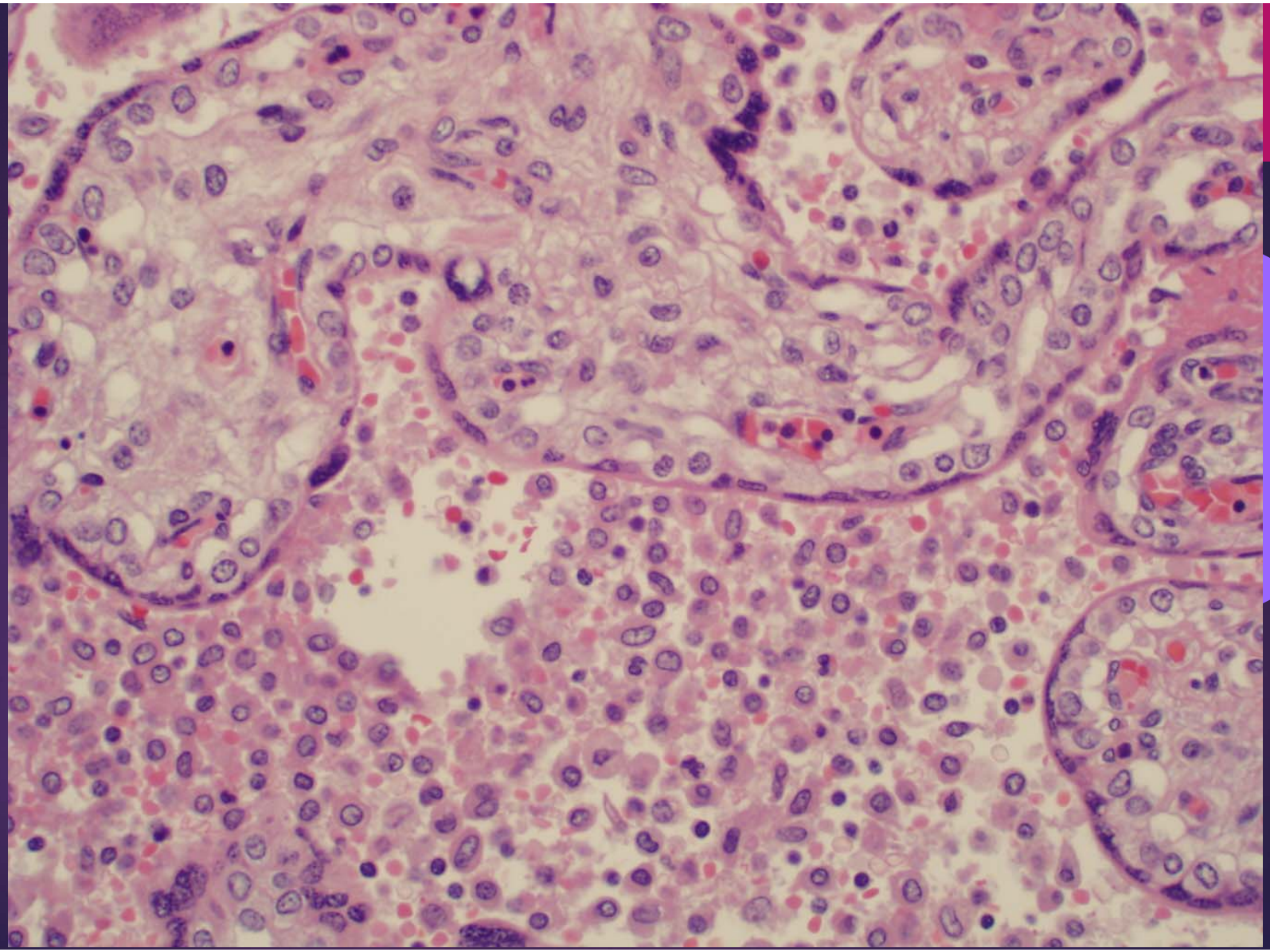


Chronic histiocytic intervillositis

- Rare, idiopathic
- Monomorphic, maternal histiocytic infiltrate in the intervillous space, without accompanying VUE

- Strong association with fetal demise, growth restriction
- Highest recurrence rate of any placental lesion (75-90%), often worse with each subsequent pregnancy





Other placental processes

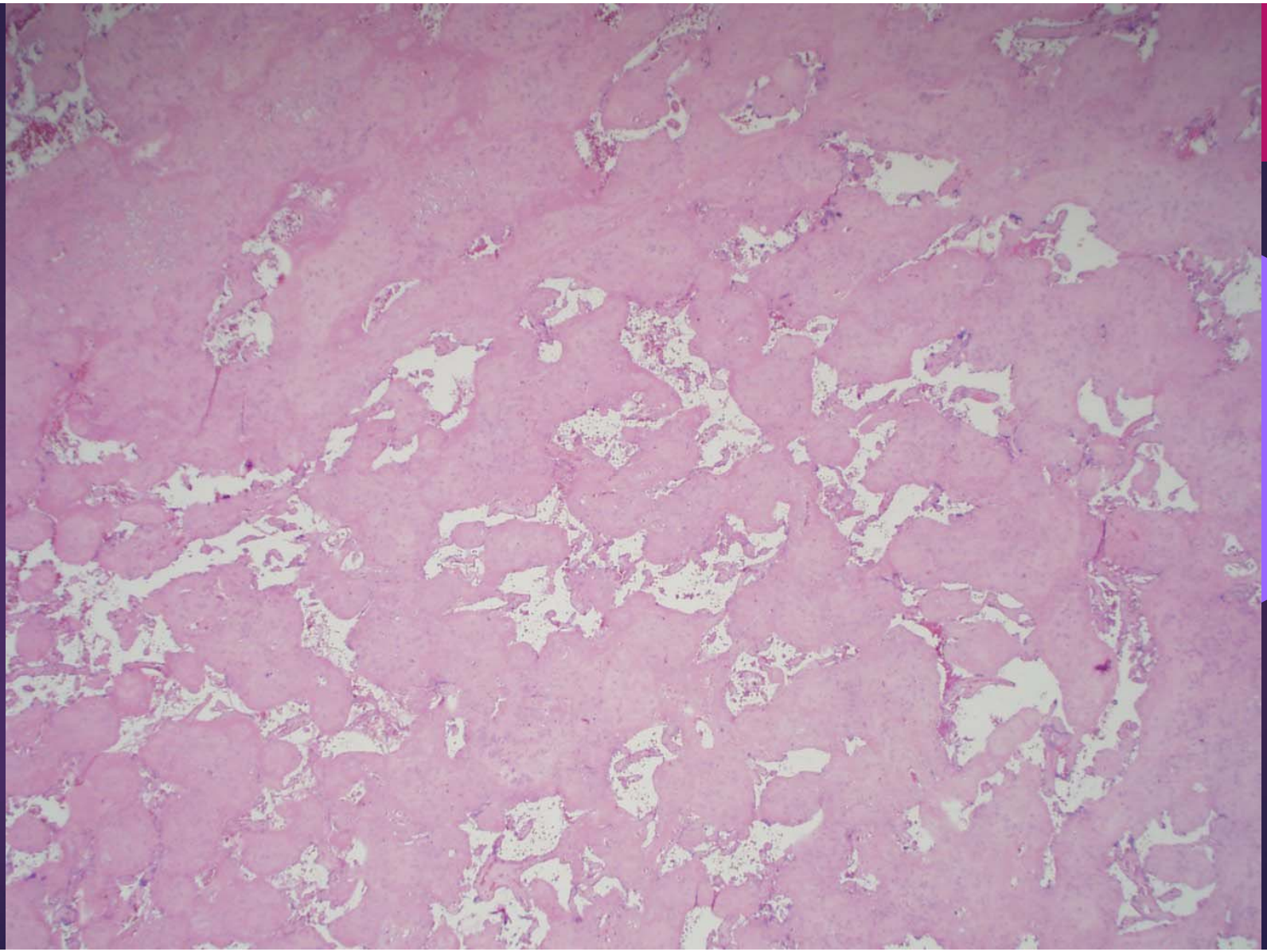
- Massive perivillous fibrin(oid) deposition
- Morbidly adherent placentas (accreta spectrum)

Massive perivillous fibrin(oid) deposition

- Aka maternal floor infarct
- Strong association with adverse outcomes
- Frequent underdiagnosis
- 40-60% recurrence rate

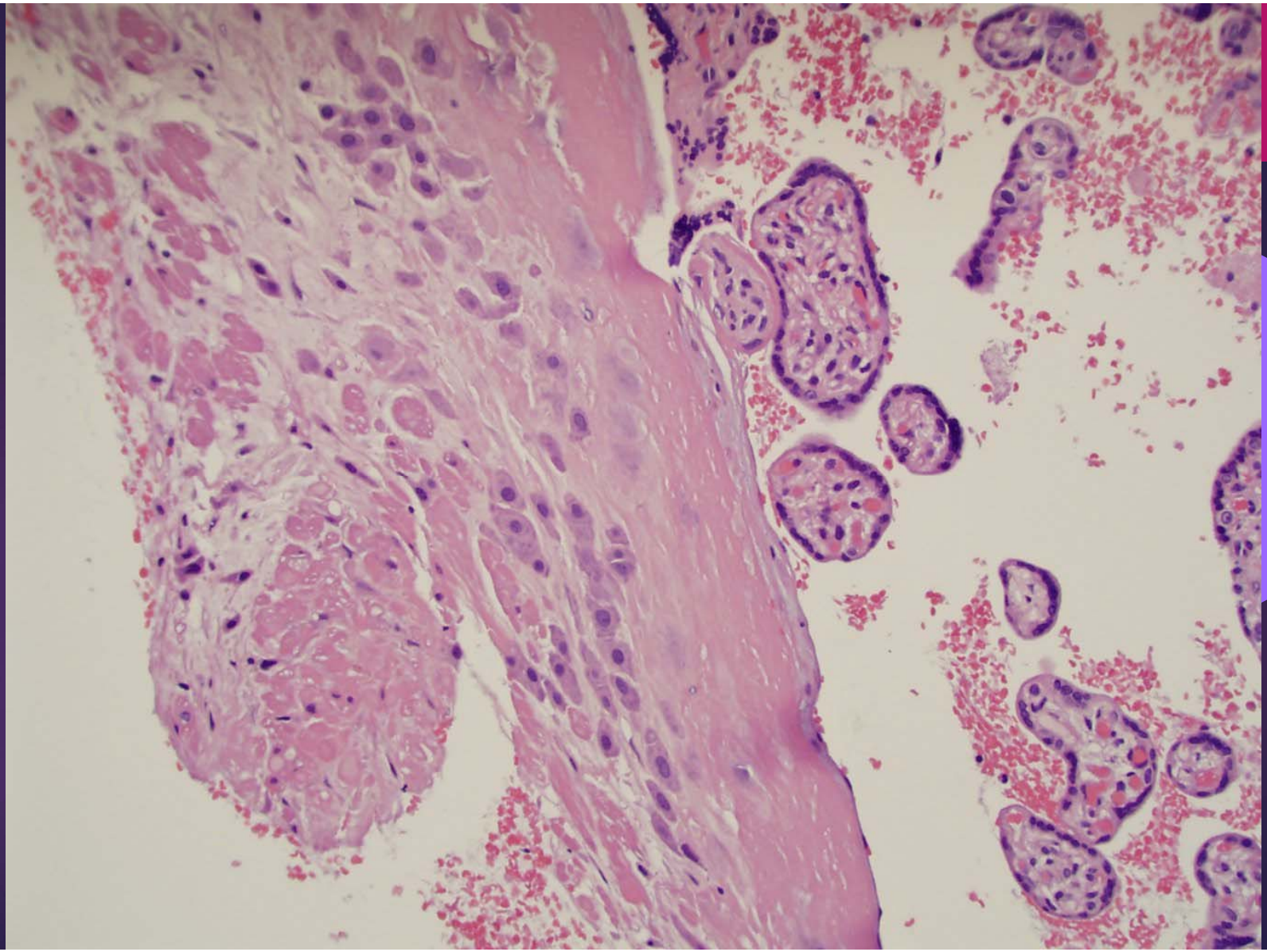
- Large amounts of fibrin and fibrinoid matrix surrounding at least 30% of distal villi

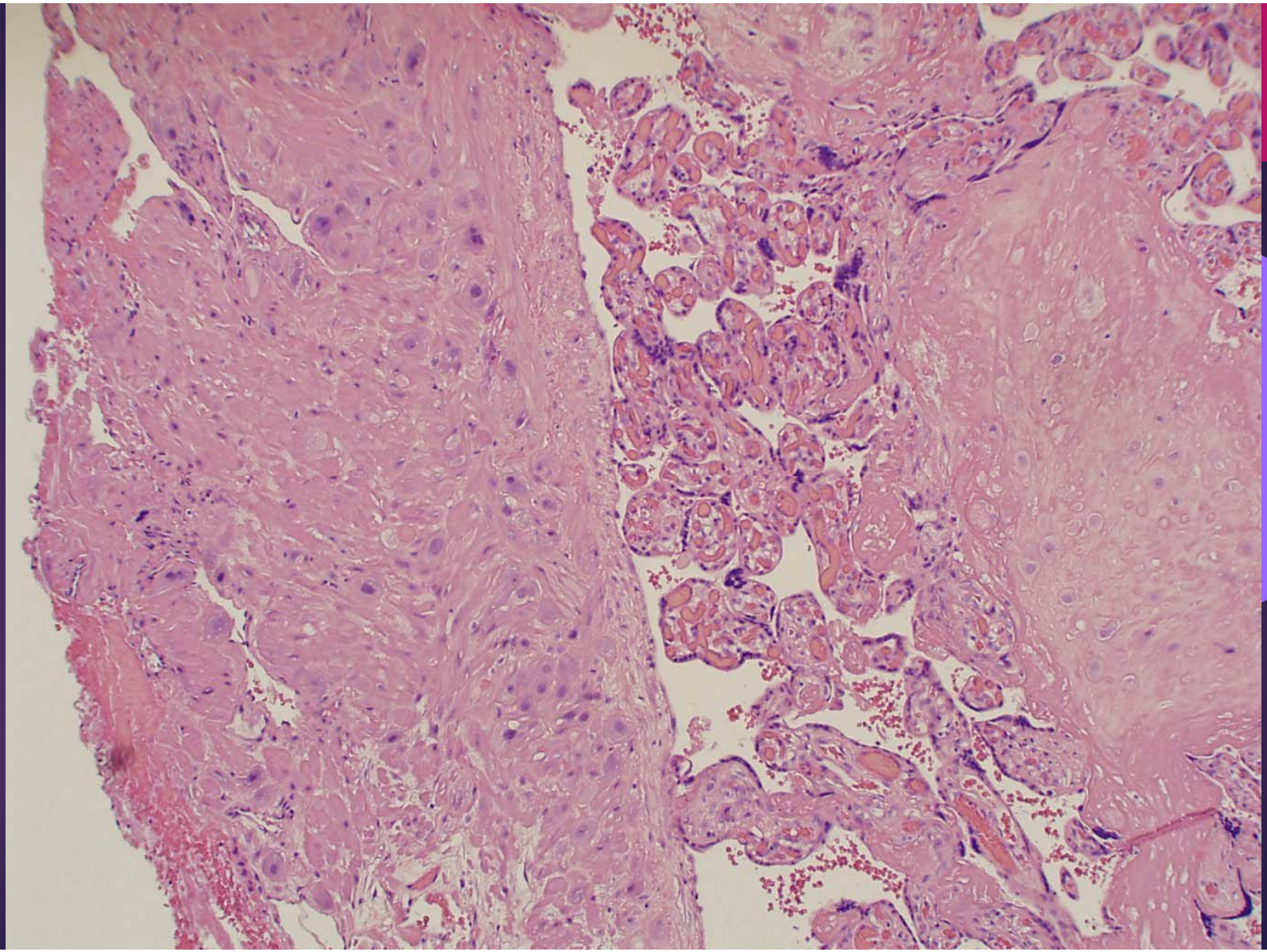
- Etiology unknown, but may be a reaction to diffuse trophoblast damage due to a variety of stressors



Accreta spectrum

- Failure of normal decidua to form, at least focally, because endometrium is deficient and cannot decidualize
- Trophoblast does not stop invading when it should, villi penetrate myometrium
- Usually hx of C-section or curettage
- 25-30% recurrence rate





The End

QUESTIONS?