Placental Dx	Subcategories	Associated Findings	Caused By	Future Risk	Baby prognosis
Acute Chorioamnionitis (ACA)	**Severe**: involves umbilical cord starting with vein, if all 3 vessels involved = umbilical panvasculitis, if extends to wharton jelly = necrotizing funisitis or perivasculitis	opaque appearance, yellow/cream colored chorionic plate, yellow umbillical cord	E coli, GBS, anaerobic, bacterial vaginosis, UTI, long labor, 1st pregnancy, posterm, PROM & PPROM	recurrence ↑ if: (1) cervical shortening surgery, (2) prior HX chorioamnionitis, (3) HX UTI, (4) HX vaginal colonization by ascending organisms; recurs in 10-25%	↑ adverse outcome: fetal vasculitis, prematurity, meconium & neo sepsis; **Morbitities:** neo sepsis, respiratory complications, NEC, neurodisability grade 2 w/ CP
Fetal Vascular malperfusion (FVM)	**Severe**: avascular villi (gross); **global/partial:** vascular ectasia, fibrin, ischemic terminal chorionic villi**; segmental/complete:** thrombi in large chorionic plate or fetal stem villi, chorionic or stem vessel obliteration, avascular villi or villi stromal-vascular karyorrhexis	abnormalities of umbilical cord, chorangiosis	trisomy 21	generally small recurrance risk,↑risk if HX thromboembolic disease	significant fetal stress; **global/partial:** assoc. w/ neonatal encephalopathy, **global and segmental:** assoc w/ nonreassuring fetal HR, IUFD, IUGR/FGR, chronic fetal monitoring abnormalities, neonatal coagulopathies, fetal cardiac abnormalities; **high grade**: CP, neonatal encephalopathy
Maternal Vascular Malperfusion (MVM)	**Severe**: chr perivasculitis, fibrinoid necrosis ±acute atherosis; g**lobal/partial MVM:** accelerated villous maturation; s**egmental/complete MVM** =villous infarction	small placenta for GA, ↑fetal/placental weight ratio, thin umbillical cord, villous infarction, decidual vasculopathy/arteriopathy, poor spiral artery remodeling in preeclampsia	diabetes, preeclampsia, HTN, renal disease, connective tissue d/o, lupus anticoagulant d/o	**severe** recurs10-25%	Depends on gestational age & maternal illness, as well as perinatal complications
Villitis of unknown etiology (VUE)	**High-grade chronic**: ≥10 involved villi/ focus (patchy or diffuse w >30% terminal villi involved), perivillois fibrin deposition; only made when infection excluded; more common late pregnancy & can be normal	avascular villi, ↑perivillous fibrin deposition, focal or patchy lymphocytes & histiocytes in chorionic villi w/ intervening normal placental parenchyma	either response to unidentified pregnancy or autoimmune immunological mechanism	**high-grade:** recurs 15-55%	94% have live birtn & seen in 5-15% of normal 3rd Δ pregnancies; **high-grade:** increased risk adverse neurologic outcomes, associated w/ IUGR/FGF 2-66%; risk for recurrent pregnancy loss
Rare Lesions:					
Chronic histiocytic intervillositis (CHI)	Maternal CD68+ histoicytes/fibrin in intrervillous space**; High grade**:>50% intervillous space by histiocytes/fibrin; **Low grade**: 5-50% intervillous space by histiocytes/fibrin	diffuse infiltrate maternal histiocytes in intervillous space, variable perivillous fibrin deposition	uncommon, idopathic, maternal SLE, APS), ↑mat Alk Phos, neo alloimmune thrombocytopenia	1st Δ miscarriage; poor reproductive outcomes; recurs 67-100% w/o treatment if illnesses listed	↑IUGR/FGR (72%) & IUFD (38%)
Maternal floor infarction (MFI) & massive perivillous fibrinoid deposition (MPVFD)	**MFI Severe**: Fibrin encasing basal villi over entire maternal surface; **MFI Moderate**: transmural from maternal to fetal ≥50% of villi; **MFI Mild**: transmural 25-50% of villi	**MPVFD**: 25-50% of intervillous space w/fibrin placental parenchyma (fetal to maternal surface); atrophy; sclerosis;	uncommon, idiopathic, infection, autoimmune dz, ↑mat serum AFP, LCHAD, coagulopathies (APS), dysregulated angiogenic/ antiangiogenic factors, fetal renal dysgenesis, fetal rejection	recurs ≤40%	↑IUGR/FGR 10-80%, IUFD 15%, adverse neurologic outcomes

Placenta Accreta Spectrum (PAS)	accreta is placental villi embed into myometrium; increta deeper into myometrium; percreta deeper into uterine serosa; Dx not made on placenta alone, needs myometrial tissue	Accreta @50% hysterectomy, increta & percreta @ 80%; Grade 1: w/o uterine wall thinning; Grade 2: >25% wall thickness; Grade 3: ≤25% wall thickness or disruption of serosa	previous uterine surg, previous pregnancies, maternal age, lower uterine implantation	recurs 25-30%	↑Risk for preterm birth, complications if hemorrhage

Pathological Finding	Definition
Acute chorioamnionitis (ACA)	**Severe**: involves umbilical cord starting with vein, if all 3 vessels involved = umbilical panvasculitis, if extends to wharton jelly = necrotizing funisitis or perivasculitis
Fetal vascular malperfusion (FVM)	associated with fetal growth restriction (FGR), associated with trisomy 21; recurrence risk; **Severe**: avascular villi (gross); **Global/partial**: vascular ectasia, fibrin, ischemic terminal chorionic villi; **Segmental/complete**: thrombi in large chorionic plate or fetal stem villous, chorionic or stem vessel obliteration, avascular villi or villous stromal-vascular karyorrhexis
Delayed Villous Maturation (DVM)	↓tertiary villus formation, ↓vasculosyncytial membrane formation, & if more severe, ↑bullous villi; etiology could be biochemical (ex. maternal diabetes), circulatory (FVM, fetal cardiac malformations, fetal low colloid osmotic pressure, or high placental weight), or chromosomal (Trisomy 21); more common in early onset FGR
Chorangiosis	villous stromal abnormality; villous hypervascularity in terminal chorionic villi; from longstanding, low-grade hypoxia, asst w/ FGR/IUGR, diabetes, gestational HTN pregnancy, ↑altitude
Maternal vascular malperfusion (MVM)	most frequent placental abnormality in hypertensive disorders (HTN) & renal disease; severe: chr perivasculitis, fibrinoid necrosis ±acute atherosis; global/partial MVM: accelerated villous maturation; segmental/complete MVM =villous infarction
Decidual Arteriopathy	abnormality seen in maternal vascular malperfusion (MVM), placental injury pattern of maternal decidual vessels related to altered uterine and intervillous blood flow
Chorionic villi	chorionic villi sprout from chorion in order to maximize area of contact w/ maternal blood
Accelerated Villous Maturation	abnormal villous branching pattern when diffuse, is associated w/ in utero hypoxic conditions
Villous Developmental defects	hypoplasia, dysmaturity, capillary dysplasia due to underperfusion or malperfusion of intervillous space by maternal blood
Placental remodeling	regression of villi starts near end of 1st trimester and gradually extends until those vill covering the deep pole in contact with the placenta bed remain, i.e., discoid placenta
Spiral Arterial Remodeling	1st & into 2nd trimester, it extends as far as inner 1/3 of myometrium including the spiral artery in junctional zone, it dilates mouth of artery to reduce pulsatility & it decreases smooth muscle to prevent spontaneous vasoconstriction; deficient remodeling seen in FGR & preeclampsia
Atherosis of Spiral Arteries	failure of spiral artery remodeling in placental basal plate; ↓velocity of maternal blood into intervillous space; smooth muscle cells retained & perfusion intermittent; fibrinoid arterial wall necrosis; accumulating foam cells & lumen narrowing; seen in FGR & preeclampsia
Loss of integrity of Fetal Vasculature	fetal hemorrhage (lge vessel rupture), cord accident, fetomaternal hemorrhage (small vessel rupture), villous edema
Villitis of unknown etiology (VUE)	pattern of placental injury, oxidative stress due to ischemia-reperfusion of intervillous space; Infiltrated maternal CD8+ T Lymphs in chorionic villi, proliferation fetal macrophages w/o infection; avascular villi, hperivillous fibrin deposition, focal or patchy lymphocytes & histiocytes in chorionic villi w/ intervening normal placental parenchyma**; High-grade chronic**: ≥10 involved villi/ focus (patchy or diffuse w >30% terminal villi involved); 10-25% recurrence, perivillois fibrin deposition
Chronic chorioamnionitis + VUE or chronic deciduitis	alloimmune etiology more likely, foacal/patchy, lymphohistiocytic, no plasma cells, no hemosiderin, may recur
Infectious villitis - viral	diffuse, histiocytic, villous plasma cells, hemosiderin, viral inclusion bodies, HX infection, rare to have autoimmune disease
Maternal floor infarction (MFI)	fibrinoid material involving at least 3 mm of parenchyma adjacent to the maternal floor on a single slide
Maternal floor infarction & massive perivillous fibrinoid deposition (MPVFD)	extended lesion parabasal villous necrosis, fibrin, thrombosis, hematoma; gross involving >50% parenchyma, microscopic involving >50% by fibrinoid material on single slide, involving intervillous space from fetal to maternal surfaces; combining parabasal villous necrosis, fibrin deposition, thrombosis, hematoma; uncommon, may recur ≤40% of pregnancies; associated w/ infection, autoimmune disease, high maternal serum alpha-fetoprotein, LCHAD & gene mutations, maternal coagulopathies APA, dysregulated angiogenic/antiangiogenic factors, fetal renal dysgenesis & maternal antifetal rejection-like, IUGR, IUFD, poor neurodevelopmental outcomes; discordant twins; placental Coxackie virus
Intervillous Thrombosis	focal coagulation of maternal blood inside intervillous space; isolated small are of no clinical significance; more pervasive seen in FGR
Breus' mole	massive subchorial thrombosis involving ≥50% of chorionic plate, seen in FGR
Infarct	villous necrosis due to obstruction of uteroplacental artery, isolated small thromboses are of no clinical significance; larger infarcts w/ fibrin seen in FGR & preeclampsia
Perivillous Fibrinoid Material (PFM)	patchy is common, likely non-pathologic; increased is 25–50% of parenchyma of single slide w/ fibrinoid involvement
Chronic histiocytic intervillositis (CHI)	maternal CD68+ histoicytes/fibrin in intrervillous space; High grade:>50% intervillous space by histiocytes/fibrin; Low grade: 5-50% intervillous space by histiocytes/fibrin; diffuse infiltrate maternal histiocytes in intervillous space, variable perivillous fibrin deposition
Placenta Accreta Spectrum (PAS)	accreta is placental villi embed into myometrium; increta deeper into myometrium; percreta deeper into uterine serosa; Dx not made on placenta alone, needs myometrial tissue; previous uterine surg, previous pregnancies, ↑maternal age, lower uterine implantation

By Clinical Presentation:	Findings	Caused By
Preeclampsia (preE)	(1) inadequate extravillous trophoblast migration, (2) VUE, (3) chronic deciduitis, (4) chronic chorioamnionitis	Maladaptation of immune response to pregnancy
preE	(1) unconverted placental bed spiral arteries, (2) Intraluminal endovascular trophoblast 3rd Δ, (3) ↑ multinucleate trophoblast cells in basal plate	↑extravillous trophoblast migration
preE	Acute atherosis	Absence of physiological vascular change
preE	(1) uteroplacental thrombosis, (2) infarction, (3) abruption	Acute atherosis
preE	(1) accelerated villous maturation (↑syncytial knots), (2) membrane chorionic microcysts, (3) Laminar membrame necrosis, (4) Diffuse decidual leukocytoclastic necrosis	↑uteroplacental vascular perfusion
preE	(1) persistent villous cytotrophoblast, (2) fetal nRBCs in villous vessels, (3) chorangiosis, (4) meconium effects	intervillous hypoxia
Chronic Hypertension	↑fetal vascular ectasia, ↓hypoxic lesions vs preE, ↓thrombotic lesions vs preE	
Antiphospholipid Syndrome (APS)	vascular thrombosis, early pregnancy loss, lupus anticoagulant, ant cardiolipin ab, β-2 glycoprotein-1 ab	
Diabetes	heavier, thickened basement membrane, villous immaturity in both GDM & T1DM, syncytial knots, ↑villous surface areas, hypovascular villi, hemorrhage, fibrin, ↑nonparechymal tissue, ↑capillaries/ terminal villi, ↑branching of peripheral villi	
Obesity	placental weight >90th, ↓placental efficiency, chronic villitis, fetal thrombosis, normoblastemia	
Fetal Growth Restriction (FGR)	global/partial maternal malperfusion, accelerate maturation	↓uteroplacental perfusion/MVM
FGR	CHI, VUE, MPVFD	Inflammatory conditions
FGR	↑diffusion distance for exchange	FVM, villous maturation d/o
FGR		Genetic chromosomal abnormalities, placental mosaicism
Trisomy 21	large, irregular hypovascular villi or relatively normal villi w/ few abnl villi & focal hypervascularity, DVM	
Spontaneous Preterm Birth	acute chorioamnionitis	
Preterm Fetal Death	global/partial FVM, cord accident, abruptio placenta	
Spontaneous Preterm Birth	mild global/partial MVM, accelerated maturation. Marginal abruption	
Preterm Fetal Death	global/partial MVM, accelerated maturation	
Preterm Fetal Death	accelerated maturation, global/partial FVM, cord accident, abruptio placenta	
Term Fetal Death	Abruptio placenta, cord accident, fetomaternal hemorrhage	
Term Fetal Death	global/partial FVM, DVM	
FGR	impaired placental vascularization, altered growth hormones & immunologic milieu	Infection (rubella, malaria, zika)
SARS CoV-2	Chronic villitis in 26%, perivillous fibrin 33%; if stain+for virus also had CHI, MPFD, chronic villitis & CD20+ B cells	