**Supplementary table 1. Placental pathology definitions**

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| Accelerated villous maturation | Defined as the presence of small or short hypermature villi for gestational period and usually accompanied by an increase in syncytial knots and considered to reflect maternal vascular malperfusion (24, 48). With earlier and more severe type of maternal vascular malperfusion, premature placentas show distal villous hypoplasia, characterized by missing branching of mature intermediate and terminal villi (24). The histologic indicator for accelerated villous maturation is an alternating pattern of villous crowding and paucity in a low power examination (45). |
| Delayed villous maturation | Defined by the presence of a monotonous villous population (at least 10 such villi) with centrally placed capillaries and decreased vasculosyncytial membranes, recapitulating the histology of early pregnancy (20, 26, 30, 71). The maturity level is compared to that expected for gestational age (26). |
| Maturational arrest | Defined as the presence of primitive mesenchymal, embryonic, and loose reticular stroma with Hoffbauer cells (26). Capillaries are few with only rare contact to the surface epithelium and thereby considerably reduced vasculosyncytial membranes and the stem villi are narrow with reduced vascular α-smooth muscle actin positive cells (26). |
| Placental histologic patterns suggestive of hypoxia | Lesions demonstrating histologic patterns suggestive of hypoxia included nucleated red blood cells, hypercapillarized villi, intravillous hemorrhage, massive perivillous fibrinoid deposition, and laminar necrosis of the decidua capsularis. Nuclear red blood cells, hypercapillarized villi and intravillous hemorrhage represent compensatory change due to fetal hypoxia; while massive perivillous fibrinoid deposition and laminar necrosis represent cellular hypoxia. |
| Nucleated red blood cells | Defined by the presence of increased circulating nucleated red blood cells identified by the presence of hyperchromatic, round nuclei with dense chromatin and polychromatic pink-gray cytoplasm. |
| Hypercapillarization or hypervascularity of villi | Characterized by the presence of an increased number of vascular channels, greater than five but less than ten, in terminal villi, which were differentiated from congestion or dilation (23, 120). |
| Intravillous hemorrhage | Defined by the presence of extravasated red blood cells in the chorionic villous stroma (123, 124). |
| Laminar necrosis of the placental membranes | Defined by the presence of a band of coagulative necrosis at the choriodecidual interface of the placental membranes, involving at least 10% of the membrane roll (77, 120, 130). |
| Massive perivillous fibrinoid deposition | Defined by the presence of extensive deposition of fibrinoid material in the intervillous space encasing and strangulating the chorionic villi (126). Three patterns of massive perivillous fibrinoid deposition have been described: classic, borderline and transmural. *Classic* - encasement of basal villi by fibrinoid along the entire maternal floor and of 3 ≥ mm thickness on at least one slide. *Borderline* massive perivillous fibrinoid deposition-involvement of 25%–50% of villi on at least one slide with transmural or nearly transmural distribution. *Transmural* massive perivillous fibrinoid deposition-transmural perivillous fibrinoid extension, with encasement of ≥ 50% of villi on at least one slide. |
| Maternal vascular malperfusion (MVM) | MVM diagnosis represents a constellation of findings involving the villous parenchyma and/or involving maternal decidual vasculature (23, 24, 66, 113).  *Villous Changes*  Villous Infarct(s): dead villous tissue in which the trophoblastic nuclei are clustered and necrotic with clumped partly dispersed DNA (recent), or the intervillous space is collapsed, the villi are crowded together, and there is complete disappearance of nuclear DNA.  Increased Syncytial Knots: aggregates of syncytiotrophoblasts nuclei along proximal stem villi or at one or more poles of distal villi.  Villous Agglutination: clusters of adherent distal villi agglutinated by fibrin and/or bridging syncytial knots.  Increased Intervillous Fibrin: abnormal amounts of intervillous fibrinoid either coat proximal stem villi or are eccentrically adherent to (or within, following re-epithelialization) distal villi.  Distal villous hypoplasia: paucity of villi in relation to the surrounding stem villi. The villi are thin and relatively elongated-appearing, and syncytial knots are increased. The features seen in lower two-thirds and involve at least 30% of one full-thickness parenchymal slide.  *Vascular Lesions*  Persistent Muscularization of Basal Plate Arteries: Persistence of smooth muscle cells in the wall of a large spiral artery in the basal plate.  Mural Hypertrophy of Decidual Arterioles: Thickening of the muscular wall of arterioles in the extraplacental membranes  Acute Atherosis of Basal Plate Arteries and/or Decidual Arterioles: Eosinophilic fibrinoid necrosis of arterial smooth muscle and presence of subendothelial or medial foam cells (macrophages) in maternal arteries |
| Fetal vascular malperfusion (FVM) | Fetal vascular malperfusion is a term applied to a group of placental lesions indicating reduced or absent perfusion of the villous parenchyma by the fetus (22, 24, 25, 30, 45, 60). Findings consistent with fetal vascular malperfusion (FVM)  *Villous Changes*  Early: Villous stromal-vascular karyorrhexis (three or more foci of two or more terminal villi showing karyorrhexis of fetal cells with preservation of surrounding trophoblast)  Late: Hyalinized avascular villi, either as exclusively small foci or variable sized foci  Severe (high grade): Consistent with fetal thrombotic vasculopathy with an average of 15 or more affected villi per slide  *Vascular Lesions*  Thrombi large fetal vessels: Organized blood clot, occlusive or non-occlusive, of any age (defined by 2 or more of the following: Fibrin strands, glassy texture with slight hematoxylin blush, adherence to endothelium) compromising the lumen of fetal vessels in the chorionic plate or proximal origin of the villous tree.  Intimal fibrin deposition, large fetal vessels: Fibrin or fibrinoid deposition (subendothelial or intramuscular) within the wall of large fetal vessels  Fibromuscular sclerosis, intermediate-sized fetal vessels: Abnormal fetal stem vessels caused by loss of fetal circulation, resulting in thickening of the muscular wall of vessels with progressive obliteration of lumen and a loss of endothelial lining. |
| Chronic Placental Inflammation | Chronic inflammatory lesions of the placenta are characterized by the infiltration by  lymphocytes, plasma cells and/or macrophages, which may be a result of infections (viral, bacterial, parasitic) or may be of immune origin (maternal anti-fetal rejection). Chronic villitis (when the inflammatory process affects the villous tree), chronic chorioamnionitis (which affects the chorioamniotic membranes), and chronic deciduitis (which involves the decidua basalis) are the three types described (24, 30, 45, 103).  *Villitis of unknown etiology* defined as a destructive chronic inflammatory lesion of the chorionic villi characterized by an inﬁltrate of maternal T lymphocytes in the villous stroma and an elevated number of fetal macrophages (Hoffbauer cells) (102).  Low grade: Presence of chronic inflammation affecting fewer than 10 contiguous villi in any one focus; Focal (one slide, fewer than 10 villi) or multifocal (more than one slide)  High grade: Presence of multiple foci, on more than one section, at least one of which shows inflammation affecting more than 10 contiguous villi; Patchy (multiple foci, with at least one focus with 10 or more contiguous villi) or diffuse (more than 30% of all distal villi are involved)  Basal villitis: Predominantly involving anchoring villi and adjacent terminal villi  Proximal villitis: Involving distal villi or proximal stem villi  *Chronic Deciduitis with plasma cells*: A diffuse lymphocytic infiltration of the decidua or any infiltrate accompanied by plasma cells |
| Acute Chorioamnionitis and Funisitis | Acute inflammatory lesions of the placenta are defined by diffuse infiltration of neutrophils and include acute chorioamnionitis, funisitis, and chorionic vasculitis representing a host response (maternal or fetal) to a chemotactic gradient in the amniotic cavity (24, 25, 30, 45, 101, 108) *Maternal Inflammatory Response*  Stage 1: Acute subchorionitis/acute chorionitis with accumulations of neutrophils in the subchorionic zone and/or in the chorionic trophoblast layer of the extraplacental membranes  Stage 2: Acute chorioamnionitis with more than a few scattered neutrophils in the chorionic plate and membranous connective tissues and/or in the amnion  Stage 3: Necrotizing or subacute chorioamnionitis with a marked neutrophilic infiltrate with degenerating neutrophils (karyorrhexis), thickened eosinophilic amniotic basement membrane, and focal amniotic epithelial necrosis  Grade 1: Not severe as defined  Grade 2: Severe: confluent polymorphonuclear leukocytes or with subchorionic microabscesses  *Fetal Inflammatory Response*  Stage 1: Umbilical phlebitis/chorionic vasculitis with neutrophils identified in the wall of any chorionic plate vessel or in the umbilical vein  Stage 2: Umbilical arteritis with neutrophils seen in one or both umbilical arteries and/or the umbilical vein  Stage 3: Necrotizing funisitis with neutrophils, cellular debris, and/or mineralization present in a concentric band, ring, or halo around one or more umbilical vessels  Grade 1: Not severe as defined  Grade 2: Severe: near-confluent intramural polymorphonuclear leukocytes with attenuation of vascular smooth muscle |