Taking a Closer Look - Pathology and the Placenta

A Review of Delayed Villous Maturity

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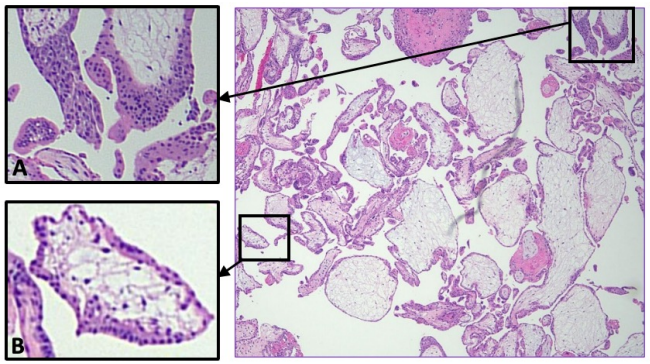
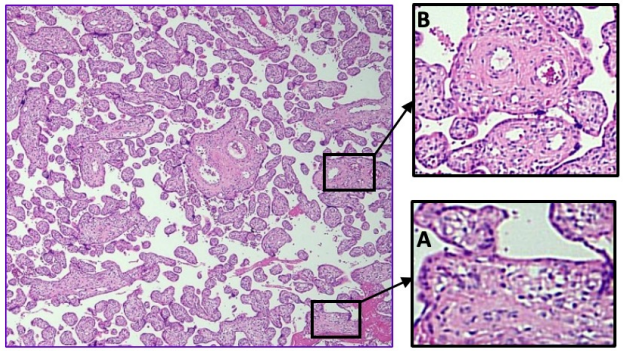
Introduction

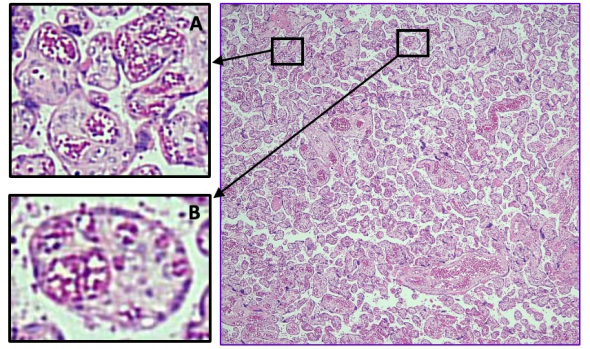
The placenta is a critical regulator of the fetal environment. It facilitates gas and nutrient exchange, whilst also acting as an endocrine organ, secreting hormones vital to maintaining gestation. Adequate development and function are fundamental for a successful pregnancy. Notably, placental pathology is present in 73.8% of pathological pregnancies.[1] However, placental histopathology is often overlooked in the discussion of obstetric pathology. Taking a closer look at the placenta enables a deeper understanding of the pathological basis for many gestational complications, allowing a broader understanding of the clinical implications.



Placental tissue from late first trimester (8/40) demonstrates villi with a polar collection of trophoblastic proliferation (A). The villi are composed of loose vasoformative stroma surrounded by a bilayer syncytiotrophoblast - cytotrophoblast wall (B).

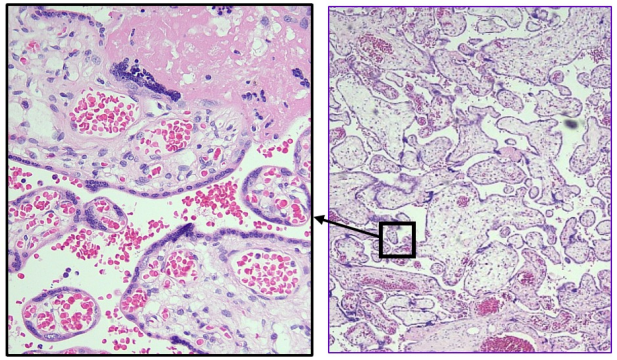
Placental tissue from late second trimester (26/40). Note the proximal mesenchymal villi from the first trimester have become collagenised stem villi (A) with muscular arteries and veins supplying the distal villous tree (B).





Placental tissue from the late third trimester (38/40) demonstrates densely packed terminal villi with minimal stroma. The cytotrophoblasts have retracted from the villi wall, forming syncytial knots, leaving a relatively acellular, discontinuous syncytiotrophoblast wall (A). Capillaries are predominantly peripheral and fuse with the wall to form the vasculosyncytial membrane (B). This morphology optimises surface area, reduces diffusion distance and enhances oxygen transfer.

Delayed Villous Maturity in a term placenta (37/40). The villi have not reduced in diameter and are not as numerous nor densely packed. Increased stroma and stromal cellularity remain. There is poor vasculosyncytial membrane formation; the villi wall is continuous, and capillaries have not moved to the periphery.



Back to Basics

The placenta, embryologically of fetal origin, comprises parenchyma, chorion, amnion and umbilical cord. It interfaces with the maternal decidua.

When the embryonic blastocyst implants approximately 6 to 7 days post-fertilisation, the outer trophoblast cells contact the endometrium. The trophoblast differentiates into the inner cytotrophoblast which grows into the outer syncytiotrophoblast to form villi like projections known as primary chorionic villi. The outer syncytiotrophoblast invades the endometrium and spiral arteries of the maternal decidua, facilitating flow of maternal blood into intervillous spaces. Subsequently, extra embryonic mesoderm infiltrates the primary chorionic villi to form secondary chorionic villi. Mesenchyme then forms the fetal blood and vessels found within tertiary villi.

Immature villi are large with polar collections of trophoblastic proliferation. As villi mature, they become smaller and denser, increasing surface area. Cytotrophoblasts pull away from the double layered villi wall to form syncytial knots, creating a discontinuous wall. The reduced diffusion distance contributes to a 30-fold increase in placental oxygen diffusion capacity.[2]

Delayed Villous Maturity

Delayed Villous Maturation results in distal villi with increased diameter for gestational age, associated with increased stromal cellularity and connective tissue. A continuous cytotrophoblast layer with decreased syncytial knots remains. Capillary density is variable with centrally placed capillaries and poor vasculosyncytial membrane formation.

Macroscopically, this can present as a large for gestational age placenta with a decreased fetoplacental weight ratio, secondary to accelerated but comprised placental growth.[3] This measure has been recognised as an indicator of placental efficiency.[4] Furthermore, it may be pale secondary to reduced vascularisation and increased stroma. Often associated with a thick or hypercoiled umbilical cord.

Implications

Delayed villous maturation is noted in 2 to 6% of pregnancies and is most strongly associated with diabetes, prediabetes, obesity, and excess weight gain.[3,5-10] It is associated with a 2.3% incidence of fetal death.[8]

Ultimately, the increased distance required for maternofetal exchange compounded with the significantly reduced surface area, creates a substantial functional deficit in nutrient and gas exchange. While this typically remains clinically silent, it leads to a decreased fetal tolerance to hypoxia and acute stress. Thus, increased risk of fetal and early neonatal mortality, with some studies quoting a tenfold risk of recurrent stillbirth.[6,8]

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