

Excerpts from

THE PLACENTA: TO KNOW ME IS TO LOVE ME

A reference guide for gross placental examination

Placental Indication: Maternal Floor Infarction

By Doris Schuler-Maloney, M.S.

<http://showcase.netins.net/web/placenta>

The maternal surface, basal plate of the placenta, is the base of the intervillous space defined by most of the following layers (rarely are they all present):

- 1) an innermost layer of patches of degenerating syncytiotrophoblast with normally absent cytotrophoblast;
- 2) rudimentary, incomplete basal lamina under the syncytiotrophoblast patches, overlying an inconsistent layer of collagen fibers and intermingled fibroblasts;
- 3) highly variable layer of cytotrophoblasts, patches of Rohr's fibrinoid, loosely arranged connective tissue admixed with decidua, remnants of encased villi and "buried" cell columns;
- 4) variably present Nitabuch's fibrinoid;
- 5) variable amount of decidua and other endometrial components including the spiral arteries. It is the most important and intimate contact zone between fetal and maternal tissues.

The term maternal floor infarction (MFI) is a misnomer, since ischemic necrosis/infarction is not a feature of this entity. Recall, a placental infarct results from localized interruption of the maternal blood supply, resulting in regional collapse of the intervillous space causing crowding/touching of neighboring villi. In contrast to infarcts elsewhere in the body, placental infarcts never undergo "organization" and they never show fibrous tissue ingrowth nor neovascularization.

Rather, in cases of MFI, the well formed, spongy cotyledons of the maternal surface are either flattened and obliterated or corrugated/exaggerated with prominent septation, due to thick, stiff and often yellow fibrin deposition which encases the deep villi (extensive perivillous fibrin), many of which are sclerotic and avascular. This fibrin may be present through the rest of the parenchyma in a "net-like" fashion, referred to as *gitterinfarct*. Although the placenta is typically small and firm, aside from increased X-cells, septal and subchorionic cysts, often there are no other lesions.

The pathogenesis of MFI is unknown. The excessive fibrin deposits are thought to develop quickly in the second and third trimesters, effectively reducing blood flow into the intervillous space, commonly resulting in fetal growth retardation, with a 26 fold increased risk for intrauterine fetal death. The children who survive are at increased risk for neurological abnormalities due to impaired uteroplacental blood flow.

MFI may recur in subsequent pregnancies and has been associated with long duration latent herpes simplex virus type 2 endometritis. It is not associated with abruption.

Gross Placental Features

Classically, the maternal surface is smooth, stiff and pale gray-yellow, without septation, or has an exaggerated cerebriform appearance due to prominent septation. The maternal surface change may be partial or complete and the parenchyma may show severe "net-like" parenchymal fibrin - *gitterinfarct*. There may be an unusually long umbilical cord, increased number of septal cysts and occasionally many subchorial cysts.

Microscopic Features

Besides the classic extensive fibrin deposition along the maternal surface which engulfs villi which atrophy and extensive perivillous fibrin throughout the parenchyma, MFI is typically associated with normal uteroplacental vasculature and shows no villous ischemic necrosis or intervillous space collapse. There is no associated inflammation.

From:

Schuler-Maloney D and Lee S: The Placenta: To Know Me Is To Love Me. A reference guide for gross placental examination; 1998, published by DSM PathWorks, Inc., St. Mary's, Iowa; <http://showcase.netins.net/web/placenta>.