Comparative histological studies on the non cellular component of normal and antepartum haemorrhagic human placenta

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Abstract:
A qualitative histological observation of two main non cellular components of human placenta were made in normal as well as in the placenta with history of antepartum haemorrhage. Forty placentae of each group were studied. The histological studies were done after staining with H&E, Masson’s Trichrome and MSB to see the difference of these deposits in each group. Fibrin and Fibrinoid were found both in normal placenta and placenta with history of APH. These deposits were found to be more intensified in the APH group which probably hampered the growth of the foetus.

Key-words : Fibrinoid, Nitabuch’s layer, Rohr’s striae, Langhans layer

Introduction: The placenta through its unique anatomical association with the mother accomplishes the vital functions for the survival of the foetus. The first reference to any lesion now referred to as infarct was made by Mauriceau [1]. Fibrinoid always played a role in placental pathology. The Fibrinoid layer of basal plate was called Rohr’s stria [2 & 3]. Maternal disorders and diseases bring a change in both morphological and histological level and therefore brings a change in both the cellular and non cellular components of the placenta. The two noteworthy non cellular components are fibrinoid and fibrin. The aim of the study was to observe the variation of the non-cellular component between normal placenta and placenta of patients with history of hypertension and APH and to correlate them. The static villous lesion in normal placenta (3rd trimester) were the encasement of villi with fibrin, disappearance of syncytial cells and reactivation of cytotrophoblastic cell [4]. With further fibrin deposition the chorionic villi were separated from maternal blood. Increasing in the deposition of excess fibrin have been mentioned by many workers time to time [5,6] Fibrinoid was a product of degeneration of both decidual and trophoblastic cells or through transformation of the ground substance in which these cells are embedded [7]. Nitabuch’s layer is fibrinoid and makes almost continuous demarcation between foetal and maternal tissues and can be recognized histologically. An increase in fibrin
deposition of placenta was found in normal gestation during last six months of pregnancy. The maternal and foetal cells were separated in the basal plate by a fibrinoid deposit. Fibrin was a fibrous protein, precipitated from fibrinogen of blood and fibrinoid was a secretion of trophoblast or a product of degeneration of trophoblast [8]. Fibrinoid layer of Nitabuch was considered as immunological “no mans land” walling off the foetus from its mother [7,9]. Perivillous fibrin deposition occurred in nearly every full term placenta (22% in uncomplicated pregnancies) [10]. The aim of the study was to observe the variation of the non-cellular component between normal placenta and placenta of patients with history of hypertension and APH and to correlate them. The static villous lesion.

Materials and Methods: The present study was carried out with 40 placentae of normal group and 40 with history of antepartum haemorrhage. All the tissue samples as fresh as possible were collected from the labour room after parturition with requisite permission from the hospital authority. Placental tissue 1.5cm in diameter were harvested along with basal deciduas. Histological studies were carried out after proper fixation of the tissue in 10% formalin. Dehydration, clearing, embedding, sectioning and deparaffinisation of section and staining with H & E, Masson’s Trichrome and M.S.B. (Martius, Scarlet Blue) method were done as per standard procedure. Histological features were studied by light microscopy and qualitative difference between the two groups were recorded.

Observation and Results: In the normal group some of the villi showed perivillous fibrin deposits which were stained red by Masson’s Trichrome (Fig-1). Intervillous fibrin deposits was also scanty. Some extravillous trophoblasts were found entrapped in fibrin. Rohr’s striae, a typical fibrin deposit was seen facing intervillous space (Fig - 2). The basal plate was a region where genetically dissimilar cells i.e. trophoblasts and decidual cells were found which were separated by a layer of fibrinoid, was the Nitabuch’s layer. In APH placenta, excessive perivillous fibrin and intervillous fibrin deposition was very frequent and was a notable feature. Perivillous, intervillous deposition of fibrin were seen as red deposit by Masson’s Trichrome stain and M.S.B. stain (Fig 3 & 4). Some of the villi were sclerosed and hyalinised with structural distortion. These were referred to as “Ghost Villi” within the fibrin plaques. In these group the villi stroma appeared structure less and was poorly stained (Fig - 5). There were some areas which indicated placental infarction and were marked by overcrowding of villi, obliteration of intervillous space and degeneration of syncytiotrophoblast. There was extensive perivillous fibrin deposition noted near the basal plate (Fig - 6). Basal plate in some cases showed the typical a) Rohr’s fibrinoid b) Layer of syncytiotrophoblast c) Nitabuch’s fibrinoid layer and d) Decidual cells. Many trophoblastic cells were encased by fibrin or surrounded by fibrillar material. Increased numbers of cytotrophoblast cells entrapped in fibrin were seen to be X cell which were present in isolation or in groups.

Discussion: Presence of fibrin deposits in the perivillous space was detected in the form of red stained material by Masson’s Trichrome staining in normal placenta. Increased fibrin deposition was found in normal gestation during last six month of pregnancy [4,7,8,10,]. Rohr’s striae as has been detected in this investigation faced intervillous space.
Rohr’s striae was reported to be superficial and faced intervillous space [11]. Nitabuch’s layer was also found in the basal plate of the normal placenta. Fibrinoid layer of Nitabuch was considered as immunological “no mans land” walling of the foetus from its mother [7,9]. Excessive deposition of fibrin in the intervillous and perivillous spaces was noted in this study by Masson’s Trichromme and M.S.B. stain. Excess deposition of fibrin in patients suffering from hypertension was noted earlier [6]. Typical Ghost Villi were found in some places where the villi were sclerosed and hyalinised with structural distortion. Some areas were marked by overcrowding of villi, obliteration of intervillous space and degeneration of syncytiotrophoblast were also noted in the APH placentae in this study. Layers of Rohr’s fibrinoid and Nitabuch’s fibrinoid could also be detected in APH placenta. A number of extravillious cytotrophoblastic cells either in isolation or in groups entrapped in fibrin were detected in the study were the X cells. Extravillious cytotrophoblastic cells (X cells of wilkin) were found encapsulated by fibrin [8]. It may therefore be concluded that deposition of non cellular component in excess amount in APH placentae acts as a barrier and interferes with the process of metabolic interaction between mother and foetus which leads to its improper growth and development.

References:


