Histomorphological Changes in Placentae of Pre-Eclamptic Mothers with Reference to Number of Villi Manifesting Fibrinoid Necrosis

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Abstract

Background: To study the histomorphological changes in placentae of pre-eclamptic mothers and to compare them with placentae of normotensive mothers with reference to number of villi manifesting fibrinoid necrosis

Methods: In this comparative study, one hundred placentae were divided into two groups. Normotensive (N) Group included fifty placentae of mothers having blood pressure 120/80 to 130/80 throughout pregnancy with gestational age between 34-38 weeks. Hypertensive (H) Group included fifty placentae of mothers with blood pressure 140/90 mm of Hg to 160/100 of Hg at two different occasions two hours apart with gestational age 34-38 weeks. Mothers having history of pre-gestational hypertension and diabetes were excluded. After delivery placentae were carefully examined and collected for gross and morphometric study. On microscopic examination terminal villi were recognized as fringing villi containing capillaries and stroma, completely surrounded by blood. Number of villi manifesting fibrinoid necrosis was counted in complete circular cross sections of terminal villi visible in the field. Three fields were randomly selected in each slide from A and B regions.

Results: The number of villi manifesting fibrinoid necrosis was increased in hypertensive group. The quantitative difference between number of villi manifesting fibrinoid necrosis in normal and hypertensive groups was statistically significant.

Conclusion: Increased number of villi manifesting fibrinoid necrosis was observed in (H) group as compared to (N) group that may be the cause or effect of placental hypoxia.

Key Words: Placenta, Pre-eclampsia, Terminal villi, Fibrinoid necrosis

Introduction

Preeclampsia is a disorder of widespread vascular endothelial malfunction and vasospasm that occurs after 20 weeks' gestation and can present as late as 4-6 weeks post partum. It is clinically defined by hypertension and proteinuria, with or without pathologic edema. Pregnancy complications like hypertension or gestational diabetes are reflected in placenta. The placenta is an extremely complex piece of biological equipment primarily responsible for delivery of oxygen to developing fetus and to carry away carbon dioxide from fetal blood. This fetomaternal organ consists of a fetal portion, the chorionic plate and a maternal portion. The decidual plate separated by “the intervillous space”. The fetal part of the placenta is formed by the villous chorion. The chorionic villi that arise from it project into the intervillous space containing maternal blood. The maternal part of the placenta is formed by the decidua basalis, the part of the decidua related to the fetal component of the placenta. The fetal part of the placenta (villous chorion) is attached to the maternal part of the placenta (decidua basalis) by the cytotrophoblastic shell, the external layer of trophoblastic cells on the maternal surface of the placenta. The chorionic villi attach firmly to the decidua basalis through the cytotrophoblastic shell and anchor the chorionic sac to the decidua basalis. Endometrial arteries and veins pass freely through gaps in the cytotrophoblastic shell and open into the intervillous space. the placental septa.

Chorionic villi cover the chorionic sac. As this sac grows, villi associated with decidua capsularis degenerate producing a relatively avascular bare area, the smooth chorion or chorion-laevae. Villi associated with decidua basalis rapidly increase in number, branch profusely and enlarge. This bushy part of chorionic sac is villous chorion or chorion frondosum which form the fetal part of the placenta. The cytotrophoblast within the villi continues to grow.
through the invading syncytiotrophoblast and make contact with decidua basalis forming stem villi, also called anchoring villi. Villi that grow from sides of main stem villi are called terminal villi. It is through the walls of the branch villi that main exchange of materials between blood of mother and embryo takes place. As pregnancy advances, the placental membrane becomes progressively thinner so that blood in many fetal capillaries is extremely close to the maternal blood in the intervillous space. During the third trimester, numerous nuclei in the syncytiotrophoblast aggregate to form multinucleated protrusions called nuclear aggregations or syncytial knots. These aggregations continually break off and are carried from the intervillous space into the maternal circulation. Some knots lodge in capillaries of the maternal lung where they are rapidly destroyed by local enzyme action. Toward the end of pregnancy, fibrinoid material forms on the surfaces of villi. This material consists of fibrin and other unidentified substances that stain intensely with eosin. Fibrinoid material results mainly from aging and appears to reduce placental transfer.

Patients and Methods

Normotensive (N) Group included fifty placentae of mothers having blood pressure 120/80 to 130/80 throughout pregnancy with gestational age between 34-38 weeks. Hypertensive (H) Group included fifty placentae of mothers with blood pressure 140/90 mmHg to 160/100 mmHg at two different occasions two hours apart with gestational age 34-38 weeks. Mothers having history of pre-gestational hypertension and diabetes were excluded. After delivery placentae were carefully examined and collected for gross and morphometric study. After fixation and tissue processing, the block trimming was done with sharp knife till the specimen was just exposed. Tissue was placed in block holder of rotary microtome and 4-5μm thick sections were made. On microscopic study terminal villi were recognized as fringing villi containing capillaries and stroma, completely surrounded by blood. Complete circular cross sections were selected. Number of villi manifesting fibrinoid necrosis was counted in complete circular cross sections of terminal villi visible in the field. Three fields were randomly selected in each slide from A and B regions under X40 magnification and total number of villi manifesting fibrinoid necrosis were noted.

Results

In N group, the mean number of terminal villi manifesting fibrinoid necrosis (Fig.1) in A and B

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Regions of Placenta</th>
<th>N Group (Mean±SE) n=50</th>
<th>H Group (Mean±SE) n=50</th>
</tr>
</thead>
<tbody>
<tr>
<td>Terminal villi manifesting fibrinoid necrosis</td>
<td>A</td>
<td>6.92±0.359</td>
<td>8.36±0.379</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>4.26±0.313</td>
<td>8.96±0.379</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Statistical Significance between N and H groups (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of terminal villi manifesting fibrinoid necrosis</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>p=0.007**</td>
</tr>
<tr>
<td>B</td>
<td>p&lt;0.001***</td>
</tr>
</tbody>
</table>

*** Statistical difference between the two groups is highly significant with p<0.001

Table 3: Data of all the two regions mean number of terminal villi manifesting fibrinoid necrosis in normotensive (n) and hypertensive (h) groups

<table>
<thead>
<tr>
<th>Parameters</th>
<th>N Group(Mean±SE) n=50</th>
<th>H Group(Mean±SE) n=50</th>
<th>Statistical Significance of difference between N &amp; H groups (P-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of terminal villi manifesting fibrinoid necrosis</td>
<td>11.18±1.455</td>
<td>17.32±0.630</td>
<td>p&lt;0.001***</td>
</tr>
</tbody>
</table>

*** Statistical difference between the two groups is highly significant with p<0.001

regions was 6.92±0.359 and 4.26±0.313 respectively (Table 1). The quantitative difference between the mean number of terminal villi manifesting fibrinoid necrosis in A and B region was statistically significant (p< 0.001) (Figure 3). In H group the mean number of terminal villi involved in fibrinoid necrosis (Fig.2) in A
and B regions was 8.36 ± 0.379 and 8.96±0.379 respectively (Table 1)

Discussion
Hypertensive disorder of pregnancy complicates 7-10% of all pregnancies. The dyad of hypertension and proteinuria after 20 weeks of gestation is referred to as pre-eclampsia. It is a major cause of maternal morbidity and mortality during pregnancy. Due to maternal vasospasm, the utero-placental blood flow is decreased in pre-eclampsia. In present study the mean number of terminal villi involved in fibrinoid necrosis was 11.18± 0.455 and 17.32±0.6.30 in N group and H group respectively and was statistically significant (p<0.001).Fibrinoid necrosis of terminal villi is a highly characteristic lesion. The first step in the evolution of this abnormality is the appearance of a small bleb of homogenous strongly PAS positive material in the trophoblast. This substance lies beneath the syncytiotrophoblast and external to basement membrane. Increasing amount of this cellular eosinophilic material eventually form a large fibrinoid nodule bordered by a few attenuated nuclei of syncytiotrophoblast, that virtually replaces the villous stroma. About 3% of the normal placentae show fibrinoid necrosis. Increased deposition is seen in pre-eclampsia. Majmudare also observed a significant increase in number of terminal villi with fibrinoid necrosis with mean diastolic blood pressure 110mm of Hg. Kurdukar et al mentioned same findings in severe preeclampsia. Bukhari observed increased number of terminal villi manifesting fibrinoid necrosis with an increase in diastolic pressure. Narasimha and Vasudeva, also noted significant villous fibrinoid necrosis in 97.82% cases of the pre-eclampsia. This increased deposition of fibrinoid was initially thought to be due to elevated blood pressure but it is now believed that it is due to inappropriate immune response.

References
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