Microanatomical analysis of hypertensive placentae - a retrospective case control study

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Abstract

Background: Placenta is regarded as the byproduct of the birth process, but actually it is the mirror of maternal and foetal status. It often reflects the progress of many maternal conditions during pregnancy and is known to undergo changes both structurally and functionally in hypertensive pregnancy due to hypoxia.

Aim: To compare the histopathological changes in hypertensive placentae with that of normal placentae.

Materials and methods: A retrospective case control study was carried out on human placentae with 50 as study and 50 as control group. Placentae of study group were divided into three categories depending upon severity of hypertension. Tissues were microscopically studied for various histopathological changes. Results: Among severe pregnancy induced hypertension cases (PIH), 77.77% showed red infarction while 70.6% of eclampsia showed white infarction. 76.47%, 64.7% and 58.82% of eclampsia cases showed syncytial knots, fibrinoid necrosis and stromal fibrosis respectively. 70.6% of eclampsia cases showed cytotrophoblastic proliferation and calcification separately. Hyperplasia of tunica media was found in 20%, 50% and 58.82% of mild PIH, severe PIH and eclampsia respectively.

Conclusion: Infarction, cytotrophoblastic cellular proliferation, syncytial knots, fibrinoid necrosis, stromal fibrosis, calcification and tunica media hyperplasia were seen with increased frequency in the study group as compared to control group. In study group, eclamptic placentae showed all the microscopic changes in significant number. This study of microscopic changes occurring in hypertensive placentae can be used to enhance our knowledge about the severity and progress of the disease. This can equip us to effectively manage the hypertensive pregnancies.

Key Words: hypertension, pregnancy, histological changes, syncytial knots.

Introduction

Placenta is a vital organ for maintaining pregnancy and promoting normal foetal development. It is the window through which understanding of maternal dysfunction as well as the impacts on foetal wellbeing can be observed. Hypertension complicating pregnancy is common and form one of the deadly triad along with haemorrhage and infection that results in large number of maternal deaths and there off foetal deaths. Pregnancy induced hypertension (PIH), a multisystemic disorder peculiar to pregnancy is characterized by gestational hypertension, proteinuria and activation of coagulation cascade is associated with many abnormalities in renal and hepatic dysfunction. Abnormality of placenta is responsible for Preeclampsia. It has been recorded that the maternal uteroplacental bloodflow is decreased in preeclampsia. Pregnancy complicated by hypertension is commonly associated with placental insufficiency. The main feature of abnormal placentation is inadequate trophoblastic invasion of the maternal spiral arteries. This result in persistence of muscular and elastic tissues of tunica media of spiral arteries. As a result, vessels fail to dilate and remain responsive to vasomotor influences that lead to high resistance low flow chorio-decidual circulation. Consequently there is reduction in uteroplacental perfusion with placenta being ischemic as gestation progresses. This results in morphological and histological changes in placenta.
Despite the significant achievements made by India in healthcare, maternal mortality continues to be significantly high 212 per one lakh live births, out of which 5% is due to pregnancy induced hypertension. The present study can help us to widen our knowledge about the expected complication of hypertensive pregnancies in relation to the severity of disease.

**Materials and Methods**

100 placentae were collected from department of Obstetric & Gynaecology and studied at Anatomy department of M.K.C.G Medical college, Berhampur after obtaining due consent from the patients. Out of 100 placenta, 50 from normal term pregnancy without any complications with normal BP were taken as control group and the rest 50 that formed the study group belonged to patients having PIH. In PIH only those cases of more than 20 weeks gestation having BP>140/90 mm of Hg and above with or without edema and/or proteinuria were included. Study group had been categorized into 3 group- mild PIH [15], severe PIH [18] and eclampsia [17].

After preserving the specimens in 10% formal saline over a period for 24-48 hours, the specimens were sectioned. Bits were taken from 1. near the implantation site of umbilical cord, 2. margins of placenta-12,3,6,9 O'clock positions, 3. centre of placenta, and 4. any pathological area.

Tissues were processed and stained with haematoxylin and eosin by routine procedure. All observations from case and control group were compared statistically.

**Results**

Among severe PIH, 77.77% of cases showed red infarction while 70.6% of eclampsia showed white infarction. 76.47%, 64.7% and 58.82% of eclampsia cases showed syncytial knots, fibrinoid necrosis and stromal fibrosis respectively. 70.6% of eclampsia cases showed cytotrophoblastic proliferation and calcification separately. Hyperplasia of tunica media was found in 20%, 50% and 58.82% of mild PIH, severe PIH and eclampsia respectively.

**Discussion**

The foetus, placenta and mother constitute vital triad in relation to foetal outcome of any pregnancy and examination of placenta plays a vital role in understanding the pregnancy related problem. Evaluation of placenta is extremely important in attempting to understand the pathophysiology of PIH. But there is no single gross or microscopic change that could be considered pathognomonic of the disease process. More over there is great inter-observer variability concerning the different entities of hypertensive placentae, necessitating careful gross and histopathological examination along with clinico-pathological correlation.

Histologically there are two [red and white] types of infarction according to the severity of hypertension. Wentworth found red infarction in 67% of severe PIH and white infarction in 73% eclamptic cases out of 676 hypertensive placenta's studied. In the present study 77.77% placentae of severe PIH had red infarction and 70.6% placentae showed white infarction which is very much in accordance with the above study. The rising percentage of placenta showing infarction with increase in severity of hypertension is probably due to thrombotic occlusion of maternal utero-placental blood vessels in hypertension.

Cytotrophoblastic cellular proliferation serves as a rough guide to severity and duration of ischemia and is seen in response to ischemic damage of placenta. It was seen in 69% placental villi as observed by Jones and Fox. Aparna Narimha observed that 86% cases in study group had cytotrophoblastic cellular proliferation as compared to 20% in control group. The present study also showed increasing number of placentae having cytotrophoblastic cellular proliferation as the severity of hypertension increases [Table1].

Excessive syncytial knots formation was seen in generalised form as an invariable result of overall reduction of foetal perfusion of placenta. It is an indication of excessive aging due to either postmaturity or a disease state causing placental insufficiency.
Hypertension in pregnancy causes placental hypoxia leading to loss of large number of parenchymal cells, which causes appearance of syncytial knots and synthesis of fibrous tissue in their place\(^1^6\). In present study, number of placentae in study group showing syncytial knots increases with severity of hypertension like 20%- mild PIH, 55.55%- severe PIH and 70.6%- eclampsia.

Fibrinoid necrosis of spiral artery [tunica intima and media of spiral artery] is virtually a pathognomonic lesion of preeclampsia\(^1^7\). It has been considered to be the result of increased blood pressure\(^1^8\). In contrast, some researchers suggested an immune reaction may be the causative factor\(^1^9\). Mirchandani et al\(^2^0\) found 80% of toxaemic placentae showing increased fibrinoid necrosis when compared with normal placentae. In the present study, 56% of hypertensive placentae had fibrinoid necrosis which is lower than the findings of above author. But this value is much higher in comparison to control group[4%].

There was increased incidence of fibrotic placentae in pregnancies complicated by preeclamptic toxemia\(^2^1\). The factors responsible for formation of stromal fibrosis were a normal aging process and a reduced uteroplacental blood flow\(^2^5\). The present study also showed increased incidence of stromal fibrosis in hypertensive placentae\([Table 1]\).

Calcification is regarded as an evidence of placental senescence or degeneration\(^2^2\). However Chen et al\(^2^3\) stressed that in high risk pregnancy the preterm calcification is a predictor of poor uteroplacental blood flow. 56% of placentae had calcification in study group which is much higher than in control group [12%].

Hyperplasia of tunica media of spiral arteriole is secondary to development of hypertension and act as protective mechanism against high pressure\(^2^4\). There occurs significant increase in tunica media hyperplasia in hypertensive placentae\(^2^5\). The present study confirmed this by showing increasing number of placentae having medial coat proliferation with increase in severity of hypertension [Table 1].

**Conclusion**

Our study revealed that there happened definitive histological changes like infarction, cytotrophoblastic cellular proliferation, syncytial knots, fibrinoid necrosis, stromal fibrosis, calcification and hyperplasia of tunica media of spiral artery in more number of hypertensive pregnancies according to their severity when compared

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<tr>
<th>MICROSCOPIC CHANGES</th>
<th>CASE</th>
<th>CONTROL</th>
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<tr>
<td></td>
<td>Mild PIH</td>
<td>Severe PIH</td>
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<td>Infarction 1</td>
<td>11</td>
<td>73.33</td>
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<td>Infarction 2</td>
<td>1</td>
<td>6.67</td>
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<tr>
<td>Cytotrophoblastic cellular proliferation</td>
<td>5</td>
<td>33.33</td>
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<tr>
<td>Syncyntial Knots</td>
<td>3</td>
<td>20</td>
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<td>Fibrinoid necrosis</td>
<td>6</td>
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<td>Stromal fibrosis</td>
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<tr>
<td>Calcification</td>
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<td>Tunica media hyperplasia</td>
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with control group. Microscopic examination of placentae in our study can be treated as an addition to the studies made by eminent authors in this field. This study is confined with microscopic changes but still there is a scope for the study of molecular changes that can occur because of hypertensive insult to the placentae. Though the contribution of present study is very small, it helps to sustain our constant effort to study the spectrum of the mysterious structural changes of hypertensive placentae.
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