Abstract: A study of one hundred placentae was done to find out the morbid and histological changes of placentae of hypertensive mothers in comparison to those of mothers with uncomplicated pregnancies. As placenta is the mirror of maternal and foetal status, it reflects the changes due to maternal hypertension.

This study was carried out on fifty mothers with uncomplicated pregnancy and fifty mothers with pregnancy induced hypertension (PIH).

It was found that mothers with moderate to severe PIH had smaller, irregular placentae with marginal insertion of umbilical cord with deviation in respect of foci of calcification, infarction and histological features of vascular insufficiency like thrombosis, infarction etc. Histological findings like cytotrophoblastic cellular proliferation, syncytiotrophoblastic proliferation, fibrin plaque formation etc. were present in greater amount in hypertensive placentae. Babies of such mothers were mostly small for date, few of them had birth asphyxia. The changes in the placentae may be the cause / effect or both of hypertension in pregnancy of mothers who were normotensive.

Key Words: Hypertension, gestation, placenta, histology.

Introduction:

Placenta is the most accurate record of the infants' prenatal experience. After delivery if the placenta is examined minutely it provides much insight into the prenatal health of the baby and the mother.

Pregnancy complications like hypertension or gestational diabetes are reflected in the placenta in a significant way (both macroscopically and microscopically). It has been recorded that the maternal utero-placental blood flow is decreased in pre-eclampsia (Browne & Veall, 1953; Bewly et al, 1991) because there is maternal vasospasm (Landesmon, 1954). Reduced maternal utero-placental blood flow leading indirectly to constriction of foetal stem arteries (Stock et al, 1980) has been associated with the changes seen in the placentae of pre-eclamptic women. Maternal vasospasm leads to foetal hypoxia. According to Thomson, et al (1969) foetal hypoxia is not uncommon near term and accordingly it may lead to foetal distress and foetal death. In recent years, it has been revealed that there is a clear relationship between confined placental mosaicism and foetal growth retardation (Kalousek & Langlosis, 1994). Naeye and Friedman (1979) calculated that 70% of the excess foetal deaths in women with hypertension are due to large placental infarcts, markedly small placental size and that histopathological changes related to confined placental mosaicism may be associated with inadequate placentation and hence with retroplacental ischaemia (Fox, 1994).

Present study has been undertaken to record the hitherto unpublished data on the morphology, morphometry and histology of placenta from mothers with pregnancy induced hypertension and correlate the findings with the birth weights of new born babies, Apgar score and congenital anomalies (if any) of babies of middle class mothers of West Bengal with pregnancy induced hypertension.

Materials And Methods

Fifty mothers with uncomplicated pregnancy and fifty mothers with pregnancy induced hypertension were selected from indoor patients of Gynaecology and Obstetrics Department of S.S.K.M Hospital (Institute of Post Graduate Medical Education & Research, Kolkata).

The age range of these mothers varies from 20 years to 38 years, primi and para 2 to 3 and they belonged to Bengali middle class family; income per family being Rs. 5000/- to 10, 000/- per month.

Among these hypertensive mothers, 30 mothers had pregnancy induced hypertension without proteinuria and oedema; whereas 20 mothers had pre-eclampsia (hypertension was associated with oedema or proteinuria or both). These mothers were normotensive before pregnancy.

Mothers were examined clinically (for height, weight, blood pressure, pulse, anaemia, jaundice etc.) along with recording of their medical history (history of past illness, history of previous child birth etc). Their investigation reports were checked (blood sugar, urea, creatinine, haemoglobin levels, urine for albumin, pus cells along with ophthalmoscopic examinations). Mothers with hypertension had their blood pressure ranging from 140/90 mm. of Hg and...
After delivery placentae were collected for morbid and histopathological studies. The size, shape, surface area, weight of placentae were noted along with the inspection of marginal veins for any thrombus; the number of cotyledons, condition of membranes, presence of infarction, calcification and site of insertion of umbilical cord were noted.

The newborn babies were inspected for congenital anomalies, Apgar score etc. Their birth weights were noted and the foeto-placental weight ratio was calculated in each case.

Tissues were taken from the following placental sites for histopathological studies:

i. Near the implantation of the umbilical cord.
ii. Margins – 12, 3, 6, 9 o-clock positions.
iii. Centre of the placenta.
iv. Fibrotic area if any.
v. Infarcted area, if any.
vi. Umbilical cord at placental junction and cut end.

Observation and Result
Placental Morphometric Study
Mean birth weight of new born babies: - (MBW)
MBW was 2.8 Kg ± 0.32 Kg in the control group and it was 2.04 ± 0.48 Kg in the hypertensive group. Babies of mothers with poorly controlled pregnancy induced hypertension were mostly small for date. Birth weights of 6 such babies were from 950 gm to 2.13 Kg. Apgar score was below 7 in 15 babies of study group and in two such babies it was 2 and 3 only. Apgar score of the control group was within normal limits Table I.

Thus it was noted that in placental insufficiency due to PIH the mean birth weight of new born babies, mean placental weight, mean placental area and mean placental volume were significantly (p>0.01) in the hypertensive group than in the control normal group. These findings corroborate with the studies of other workers Damania (1989), Fox, (1994) and Kalousek (1994). In this study it was also found that newborn babies of mothers with poorly control late PIH were small for date and few of them had birth asphyxia; some of them were born prematurely. According to Yousonszai and Haworth (1969), placental weight and size were directly proportional to the birth weight of babies. Rath in 1994 stated that in hypertension arrangement of the intracotyledonous vasculature is altered; resulting in low birth weight of the babies.

The gross anatomic features of placentae e.g infarcted areas, calcified areas and marginal insertion of the umbilical cord in the study group show significant increase in value (p>0.01) when compared to that of the control group. This is in concurrence with the findings above.

Stromal Pathology
Thus the stromal and villous histopathological changes in the placenta like stromal fibrosis, medial coat proliferation of medium sized blood vessels and mean number of calcified and hyalinized areas per low power field were significant (p>0.01) in the hypertensive group than in the control group which might be the cause or effect of hypertension Table IV.

Discussion:
The study reveals that the foetal weight is significantly less in the hypertensive group than the control group and the morphometry of placenta i.e weight, surface area and volume show significantly lower values in the study group than the control normotensive group. These findings corroborate with the studies of other workers Damania (1989), Fox, (1994) and Kalousek (1994). In this study it was also found that newborn babies of mothers with poorly controlled PIH were small for date and few of them had birth asphyxia; some of them were born prematurely. According to Yousonszai and Haworth (1969), placental weight and size were directly proportional to the birth weight of babies. Rath in 1994 stated that in hypertension arrangement of the intracotyledonous vasculature is altered; resulting in low birth weight of the babies.

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Histological Study Of Placental Villi
Thus, to sum up the histological study of the placenta, it had been noticed that on examination under microscope, significant (p>0.01) number of syncytial knots, (Fig. 2) cytotrophoblastic cellular proliferation, (Fig. 3) fibrinoid necrosis, endothelial proliferation, calcified and hyalinised villous spots (fig 5) were observed per low power field in the hypertensive group in comparison to control group.

Gross Anatomy Of Placenta
Thus the mean number of infarcted areas, calcified (Fig. 1) statistically areas and marginal insertion of umbilical cord had significant (p>0.01) values in hypertensive group in comparison to the control group. However, the number of cotyledons show lower values and is not significant. The findings may indicate cause / effect of pregnancy induced hypertension.

Histological Study Of Placental Villi
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of Fox (1967) and Udainia et al (2004) who had observed a similar increase in the incidence of placental infarction with severity of toxaemia. Also, Pretorius (1996) reported cases of marginal insertion of placenta in about 42% cases of pregnancy induced hypertension.

The histology of placenta of hypertensive mothers also shows significant increase in syncytial knot formation, cytotrophoblastic cellular proliferation, proliferation of endothelial lining of capillaries, stromal fibrosis, calcification and hyalinisation of villi in comparison to the control group. On post-mortem examination, Genset (1992) reported that stromal fibrosis and excessive syncytial knot formation are seen in generalized form as invariable results of overall reduction of foetal perfusion of the placenta.

On histological observation of placentae, evidence of cytotrophoblastic cellular hyperplasia and patchy necrosis of the villous syncytiotrophoblastic cells are obvious in the study group in comparison to the control group. This is also very much in accordance with the previous studies conducted by Jones and Fox (1980).

It is already known that the physiology of the uterine vascular pattern, notably of the spiral arteries in patients (who were normotensive in pre-pregnant stage) is remarkably modified following decidual implantation of blastocyst and during placentation. This modification is enhanced; as confirmed in the present study; in certain abnormal clinical situations, notably pre-eclamptic toxaemia and eclampsia Rushton (1984) in which hypertension is a major sign.

A significant increase in the cytotrophoblastic cellular proliferation and syncytial knot formation in the placental villi may indicate a disturbance in the hormonal factors which may probably lead to altered morphometry of placenta resulting in pregnancy induced hypertension in the mother and to low birth weight babies. Role of marginal insertion of umbilical cord in the placenta has also been implicated in the induction of hypertension Pretorius,(1996); Di Salvo, (1998) and Rath G, (2000).

Microscopic findings of localised fibrinoid necrosis, endothelial proliferation of arteries and hyalinisation depict the mosaicism of placenta and probably the aftermath of hypertension Teasdale, (1980) and Udainia

Fig. 2- Areas of Syncytial Knot formation, H & E X 100

Fig. 3: Areas of medial coat proliferation of medium sized blood vessels, H & E X 100.

Fig. 4- Calcified areas, H & E X 100.

Fig. 5- Hyalinised areas, H & E X 100.

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### Table I - Placental Morphometric Study

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Hypertensive Group</th>
<th>Statistical Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mean birth wt. of babies in kg</td>
<td>2.8 ± 0.32</td>
<td>2.04 ± 0.48</td>
<td>Significant</td>
</tr>
<tr>
<td>2. Mean Placental wt. in grams</td>
<td>485.85 ± 47.31</td>
<td>399.10 ± 90.31</td>
<td>Significant</td>
</tr>
<tr>
<td>3. Mean Placental Area in sq.cm.</td>
<td>265.15 ± 65.24</td>
<td>202.59 ± 58.37</td>
<td>Significant</td>
</tr>
<tr>
<td>4. Mean Placental volume in cc</td>
<td>612.98 ± 213.26</td>
<td>375.99 ± 173.61</td>
<td>Significant</td>
</tr>
<tr>
<td>5. Mean Foeto-placental weight ratio</td>
<td>5.89 ± 65.24</td>
<td>6.23 ± 65.24</td>
<td>Significant</td>
</tr>
</tbody>
</table>

### Table II- Gross Anatomy Of Placenta

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Hypertensive Group</th>
<th>Statistical Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mean Number of Cotyledons per placenta</td>
<td>17 + 2</td>
<td>16 + 2</td>
<td>Not significant</td>
</tr>
<tr>
<td>2. Mean Infarcted area in placentae in number</td>
<td>3.77 + 1.87</td>
<td>16.5 + 4.6</td>
<td>Significant</td>
</tr>
<tr>
<td>3. Marginal insertion of umbilical cord (in percentage)</td>
<td>5.2 + .01</td>
<td>20 + 3</td>
<td>Significant</td>
</tr>
<tr>
<td>4. Mean calcified areas in placentae in number</td>
<td>4.125 + 1.15</td>
<td>33.3 + 3.15</td>
<td>Significant</td>
</tr>
</tbody>
</table>

### Histological Study

#### Table - III - Study Of Placental Villi

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Hypertensive Group</th>
<th>Statistical Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mean No. of areas of Syncytial knot formation per *lpf</td>
<td>9.92± 3.04</td>
<td>27.1±3.24</td>
<td>Significant</td>
</tr>
<tr>
<td>2. Mean number of areas of cytotrophoblastic cellular proliferation/lpf</td>
<td>6 ± 1.54</td>
<td>18.82± 3.39</td>
<td>Significant</td>
</tr>
<tr>
<td>3. Mean number of areas of Fibrinoid necrosis/lpf</td>
<td>3.13± 1.87</td>
<td>11.3 ± 2.3</td>
<td>Significant</td>
</tr>
<tr>
<td>4. Mean number of areas of hyalinised villi /</td>
<td>2.33 ± 0.97</td>
<td>11.1 ± 1.82</td>
<td>Significant</td>
</tr>
</tbody>
</table>

### Table - IV - Stromal Pathology

<table>
<thead>
<tr>
<th>Placental Pathology</th>
<th>Control Group</th>
<th>Hypertensive Group (per lpf)</th>
<th>Statistical Significance (per lpf)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mean No. of Areas of Stromal Fibrosis/lpf</td>
<td>2.95 ± 1.65</td>
<td>10.9 ± 3.25</td>
<td>Significant</td>
</tr>
<tr>
<td>2. Mean number of areas of Medial coat proliferation of medium sized blood vessels/lpf</td>
<td>3.8 ± 1.5</td>
<td>13.1 ± 1.91</td>
<td>Significant</td>
</tr>
<tr>
<td>3. Mean no. of calcified areas/lpf</td>
<td>3.62 ± 1.5</td>
<td>12.6 ± 2.01</td>
<td>Significant</td>
</tr>
<tr>
<td>4. Mean no. of hyalinised areas/lpf</td>
<td>3.9 ± 1.26</td>
<td>13.45 ± 1.211</td>
<td>Significant</td>
</tr>
</tbody>
</table>
et al, (2004). Again the mosaicism of the placenta probably leads to placental insufficiency and ultimately to foetal growth retardation, Zacutti (1992), thus creating a vicious cycle.

Acknowledgement

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References