STUDY OF HUMAN PLACENTAE ASSOCIATED WITH PRE-ECLAMPSIA AND ESSENTIAL HYPERTENSION IN RELATION TO FOETAL OUTCOME

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SUMMARY

The study of comparison of pathology of placentae from pregnancies complicated by pre-eclampsia, eclampsia and essential hypertension with that of uncomplicated pregnancies have been the subject of considerable scrutiny because of contradictory placental findings, put forward by different authors from time to time. In the present study, a total of 100 placentae from singleton pregnancies are analysed, which includes 22 from eclampsia, 7 from severe pre-eclampsia, 30 from mild per-eclampsia, 9 from women with essential hypertension and 32 from normotensive pregnancies.

On morphometric study no characteristic pathological changes were detected in the placentae of both complicated and uncomplicated pregnancies. On histology placentae from both severe pre-eclampsia and eclampsia showed increased incidence of red and white infarction, fibrin deposition in the intervillous space, decidual haematoma, congestion, stromal density and accentuated maturation of the chorionic villi. Placentae from pregnancies with essential hypertension without superimposed pre-eclampsia appeared normal except increased incidence of white infarction, fibrin deposition in the intervillous space, stromal density and congestion of villi microscopically.

Placental pathology of babies with birth weight below 2500 gm (13%) did not reveal any characteristic changes as compared to those above 2500 gms (87%), indicating that the effect of poor utero-placental circulation producing a small foetus remains obscure. It appeared that placental pathology is more likely an effect, rather than a cause, of diminished foetal growth. No definite conclusion could be drawn from this study on the findings of the placental changes with stillbirth (11.8%).

Introduction

The aetiology of pregnancies complicated by pre-eclampsia, eclampsia and essential hypertension is not yet known but a number of deductions can be made from the available evidence. Various factors were claimed to be associated with either an abnormality of trophoblast itself or of the maternal adaptation to the presence of trophoblast.

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Bartholomew and Cohin (1961), Fox (1967), Salvatore (1968), Abitol *et al* (1976), Bond and Scoff (1985), have considered placenta to be responsible for the causation of pre-eclampsia. Placental ischemia could occur via different mechanisms such as failure of normal adaptation of the spiral arteries occurring in early pregnancy. In other stipulations, the excessive placental mass or selerotic uterine vessels could result in placental ischemia.

In view of the above and prevalence of pregnancies complicated by pre-eclampsia, eclampsia and essential hypertension, we carried out a wider and careful pathological study of 100 placentae both from uncomplicated and complicated pregnancies. The results obtained from this study were reviewed and critically analysed to find out any correlation between the placental changes in uncomplicated and complicated pregnancies and their relationship with that of the foetal outcome.

Material and Methods

A total of 100 placentae both from normal and pregnancies complicated by pre-eclampsia, eclampsia, and hypertension were studied, from August, 1984 to November, 1986. The present series consisted of 22 cases of eclampsia, 7 cases of severe pre-eclampsia, 30 cases of mild pre-eclampsia, 9 cases of hypertension and 32 cases of normal pregnancy.

A detailed history of each patient was taken on admission and all cases were personally watched and observed carefully before, during and after the delivery.

Placental study

Placenta expressed gently so as to remove its blood content and then washed in tap water. Placenta was then studied.

A. *Macroscopically*

Size, Shape, Thickness, Weight, Attachment of Cord, Number of Cotyledons, Any Infarction (White and Red), Calcification, Haemorrhage, Any Abnormality, Cyst etc. were studied.

After macroscopic studies several fragments of each placenta were removed from the centre and peripheral part of the maternal and foetal surfaces. Tissues were fixed in 10 per cent formalin, then subjected for histological examination.

The fixed tissues were processed through the ascending grade of alcohol (50 percent, 70 percent, 90 percent and absolute alcohol), xylol and then melted in paraffin (56 percent) for 2 hours in each and finally paraffin blocks were made. From the paraffin block several microsections were cut in microtome at 3 to 5 micron and fixed over the slides. They were then stained by Harris haematoxylin eosin stain as per standard laboratory procedure.

B. *Microscopically*

After mounting in D.P.X. they were seen at first in low power, then in high power for microscopic view, where following important points were noted after studying 500 villi (approx.). These were red infarct, white infarct, decidual haematoma, increased fibrin deposition, congestion of villi, premature villi, mature villi, hypermature villi, endarteritia, increased stromal density, and syncytial knot.

Terminology

The lack of standardization in terminology is a factor which has added to the confusion surrounding this subject. In

the present study, the following terms have been used.

(a) *Red infarction*

This corresponds to the most recent type of placental infarct. Macroscopically —it appears as red thickened haemorrhagic area and microscopically shows presence of more R.B.C. but less fibrin.

(b) *White infarction*

This corresponds to the old type of placental infarction. Microscopically firm to hard white fibrin plaques are found (Fox, 1963).

Macroscopically: mostly fibrous but less R.B.C.s.

White and Red infarct are represented as follows:

(a) Few, scattered or discrete—+
(b) Moderate degree —++
(c) Massive degree —+++

The chorionic villi near the maternal surface of the placenta were classified in groups (according to Salvatore 1968) as follows:

(a) *Premature villi*: are those villi of the placenta found at the sixth and seventh months of normal pregnancy. The villi are not too mature. The 'nuclear knots' are not prominent and contain few nuclei. The nuclei of the syncytium are basophilic and in general are alinged and dispersed throughout the syncytial layer. The stromal tissue has few collagen fibrin and the capillaries are not close to the syncytium.

(b) *Mature villi*: are the typical villi of the normal term placenta, the typical hyperchromatic 'nuclear knots' are not prominent. The villi are small with collagenous stroms.

(c) *Hypermature villi*: are the hypersenile villi. They are very small, with a thin layer of syncytium devoid, in general, of nuclei. 'Nuclear knots' are conspicuous, some are polypoid and some are pyknotic. The capillaries approximate the thin layer of syncytium.

The interpretation of the villi is based upon the presence of highest number (more than 75 percent) of the particular villi.

Despite the difficulty of estimating the amount of fibrin deposition in the intervillous space of the placenta and the stromal density of the villi, such studies were done in relation to the term placenta. The vascular congestion of villi was studied in portions of placenta removed from the areas far from the red and white infarct.

For the convenience of study following classification were done:

Mild pre-eclampsia: B.P.—140-160/90-110 mm Hg. with Traces (+) of (i) oedema or proteinuria or (ii) oedema + proteinuria.

Severe pre-eclampsia: B.P.—161 and above/all and above mm of Hg with moderate or marked (++ or +++) degree of oedema + proteinuria.

Eclampsia: Pre-eclampsia + convulsion.

Hypertension: High blood pressure without oedema and proteinuria.

Normal: Normal control group.

Results and Observation

Seventy six percent cases in the present series were between 15-25 years of age. 51 percent of cases were primi-gravidae, and 28 percent cases had one previous pregnancy. 83 percent of cases studied were of term pregnancy.

It is evident from the Table I that the placental weight in the range of 451-500

TABLE I

Macroscopic Changes in Normal, Pre-eclampsia, Eclampsia and Essential Hypertension

Macroscopic	Normal (N—32)	Mild Pre-eclampsia (N—30)	Severe Pre-eclampsia (N—7)	Eclampsia (N—22)	Essential hypertension (N—9)
1. Weight in Grams 150-300	—	3 (10%)	1 (14.28%)	5 (22.73%)	2 (22.2%)
301-450	14 (43.7%)	17 (56.66%)	4 (57.14%)	17 (77.27%)	6 (66.7%)
451-500	15 (46.9%)	10 (33.34%)	2 (28.58%)	—	1 (11.1%)
501 and above	3 (9.4%)				
2. Infarction Red	14 (43.15%)	24 (80%)	6 (85.71%)	11 (50%)	6 (66.7%)
White	—	5 (16.7%)	3 (42.86%)	21 (95.46%)	—
3. Calcification	4 (12.5%)	9 (30%)	4 (57.14%)	13 (59.09%)	9 (44.4%)

gms was found to be more in normal pregnancies (46.9%) and mild pre-eclamptic (33.3%) cases as compared to severe pre-eclamptic (28.58%) and hypertensive (11.1%) cases.

On the other hand, it is interesting to note that 77.27% of eclamptic and 66.7% of hypertensive cases, the placentae were in the range of 301-450 gms. This incidence is much higher as compared to severe pre-eclamptic (56.14%), mild pre-eclamptic (56.17%) and normal (43.7%) cases.

Red infarction was found to be more common in severe pre-eclamptic (85.71%) and mild eclamptic (80%) cases as compared to normal (43.15%), eclamptic (50%) and hypertensive (66.7%) cases.

White infarction was found to be absent in placentae of both normal and hypertensive cases. But it was found to be more common in eclamptic cases (95.46%) as compared to severe pre-eclamptic (42.86%) and mild pre-eclamptic (16.7%) cases.

Calcification were observed was more often in eclampsia (59.09%), severe pre-eclampsia (57.14%), hypertension (44.4%) cases, as compared to normal (12.5%) and mild pre-eclampsia (30%) cases respectively.

Microscopic placental changes in normal and complicated pregnancies

It has been observed from Table II that white infarction of placenta in severe pre-eclampsia and eclampsia was were found in 71.42% and 54.54% of cases respectively. The red infarction was found in 42.86% of severe pre-eclampsia and 59.09% of eclampsia cases.

The decidual haematoma, as compared to normal was found to be increased in eclampsia (31.82%) and severe pre-eclampsia (28.58%). The increased fibrin deposition in severe pre-eclampsia and eclampsia were found in 71.42% and 50.01% cases respectively. The congestion of villi was found to be increased in eclampsia (77.73%) and severe pre-eclampsia (42.86%).

It has been also observed that mature villi (90.7%) was the common findings in normal term placentae. At the same time, it is interesting to note that 59.09% of eclampsia and 57.14% of severe pre-eclampsia showed hypermature villi. Pre mature villi as compared to normal, was also found to be increased in hypertension (22.2%), severe pre-eclampsia (14.28%) and eclampsia (18.18%) (Table II).

In severe pre-eclampsia and eclampsia the increased stromal density was found in 71.42% and 59.09% of cases respectively and 44.4% in hypertension cases. Endarteritis was found to be increased in eclampsia (28.58%). Syncytial knot was also found to be increased in severe pre-eclampsia (28.59%) and eclampsia (18.18%). In hypertension 44.4% showed syncytial knot (Table II).

Foetal outcome

Eighty-seven percent of the babies from the present series were found to be above 2,500 gms in weight, only 13% of babies were less than 2,500 gms in weight —they were in cases of eclampsia (22.73%) in hypertension (22.22%), in mild pre-eclampsia (13.3%) and normal control group (6.25%) (Table III). Placental pathology from birth weight below 2500 gms did not reveal any characteristics pathological changes as compared to birth weight above 2500 gms.

TABLE II

Showing Microscopic Changes in Placentae

Microscopic Changes	Normal (N—32)	Mild pre-eclampsia (N—30)	Severe pre-eclampsia (N—7)	Eclampsia (N—22)	Hypertension (N—9)
Red infarction	9 (28.1%)	8 (26.7%)	3 (42.46%)	13 (59.09%)	1 (11.1%)
White infarction	14 (43.75%)	12 (40%)	5 (71.42%)	12 (64.54%)	6 (66.7%)
Decidual haematoma	—	2 (6.6%)	2 (28.58%)	7 (31.82%)	1 (11.1%)
Increased fibrin deposition	1 (3.1%)	13 (43.3%)	5 (71.42%)	11 (50.01%)	6 (66.7%)
Congestion of villi	8 (25%)	5 (16.7%)	3 (42.86%)	16 (72.73%)	4 (44.4%)
Premature villi	2 (6.2%)	1 (3.3%)	1 (14.28%)	4 (18.18%)	2 (22.2%)
Mature villi	29 (90.7%)	22 (73.4%)	2 (28.58%)	5 (22.73%)	6 (66.7%)
Hypermature villi	1 (3.1%)	7 (23.3%)	4 (57.14%)	13 (59.09%)	4 (11.1%)
Increased stromal density—	—	11 (36.7%)	5 (71.42%)	13 (59.09%)	4 (44.4%)
Endarterities	—	1 (3.3%)	2 (28.58%)	8 (36.37%)	—
Syncytial knot	3 (9.4%)	4 (13.3%)	2 (28.58%)	4 (18.18%)	4 (44.4%)

TABLE III

Showing Analysis of Weight of the Baby in the Present Series

Analgesis of birth weight	Normal (N—32)	Mild pre-eclampsia (N—30)	Severe pre-eclampsia (N—7)	Eclampsia (N—22)	Hypertension (N—9)
Below 2,500 gms.	2 (6.25%)	4 (13.3%)	—	5 (22.73%)	2 (22.22%)
Above 2,500 gms.	30 (95.75%)	26 (86.7%)	7 (100%)	17 (77.27%)	7 (77.78%)

11.8% of cases was found to be still birth, of which eclampsia was the major causes of death (75%) (Table IV).

TABLE IV
Showing Incidence of Birth in the Present Series

Still birth	Normal cases (32)	Pre-eclampsia and eclampsia (68)
Number	Nil	8
Percentage	Nil	11.8

It is also observed that placentae in cases who had still birth do not reveal any characteristic feature as compared to the placentae in similar cases with live births.

Discussion

The exact aetiology of pregnancies complicated by pre-eclampsia and eclampsia is not known. Many authors (Bartholomew *et al* 1961, Went Worth 1967 and Salvators, 1968) considered placenta to be responsible for the causation of pre-eclampsia and eclampsia. Macroscopically no characteristic changes were detected in the placenta except red infarction in 95.46% cases of eclampsia and 42.86% cases of severe pre-eclampsia. Microscopically it was found that as compared to normal, the placentae from pre-eclampsia and eclampsia were showing an increased incidence of red and white infarcts, fibrin deposition in the intervillous space, appearance of decidual haematoma, congestion of capillaries, proliferative endarterities, stromal connective tissue density of the villi, and occurrence of accentuated maturation of the chorionic villi. These present series result confirm those of Salvatore (1968), Barthelomew and Colvin (1938), Little (1960) and Zeek and Assali (1950).

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