

## A Comparative Study of the Pathological Changes in Placenta of Pre-Eclamptic Patients and Its Relation to Foetal Outcome

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**Abstract :** The study is to observe pathological changes in the placenta of pre-eclampsia patients and its relation to foetal outcome, which is a comparative study done in the department of obstetrics and gynaecology, SMC, Vijayawada. Pre-eclamptic toxemia is a unique condition of pregnancy, where there are pathophysiological changes in placenta, which leads to ischemic injury to the uteroplacental bed and which affects the foetus adversely.

**Key Words:** Pre-eclampsia, Hypertension, Placenta, IUGR, Foetus.

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### I. Introduction

Pre-eclampsia is a multisystem disorder with uteroplacental insufficiency, characterised by a rise in blood pressure of more than 140/90 after 20 weeks of gestation with proteinuria in a previously normotensive woman. It complicates 10% of pregnancies. It is a leading cause of maternal morbidity and mortality with imposed complications on the foetal growth and development.

The pathophysiological changes in the placental unit are mostly due to endothelial dysfunction and vasospasm. The following changes are described in the placenta.

1. Failure of endovascular invasion of the trophoblast in to the spiral arterioles beyond the deciduo – myometrial junction there by making it a high resistance, high pressure and a low flowunit .Hence there is a reduction of blood flow to the foetus
2. Loss of normal pregnancy refractoriness to the pressor stimuli leading to an increased sensitivity of the vessels to vasoconstrictors. As a consequence of which there is vasospasm due to imbalance between vasodilators and vasoconstrictors
3. Endothelial dysfunction due to release of free radicals, oxidised lipids and inflammatory mediators like cytokines ,tnf alpha and IL-6 antiangiogenic factor, SFlt -1 binds with other factors like VEGE and PLGF, causing endothelial dysfunction

As a result of chronic ischaemic injury to the uteroplacental bed, the placenta undergoes certain gross and microscopic changes. Syncytial degeneration, increased syncytial knots, necrotic changes in villi, basement thickening of trophoblast, fibrinous deposits in placental vessels, cytotrophoblast hyperplasia were noted.

These pathological changes lead to foetal hypoxia, low birth weight, IUGR, IUFD, and other developmental abnormalities.

### II. Aims And Objectives

1. To evaluate the pathological changes in the placenta of pre-eclamptic patients.
2. To analyse and correlate the relationship of pathological changes to foetal outcome.

### III. Material And Methods

This is a comparative study of 30 antenatal women, with pre-eclampsia with or without severe features with an equal number of age matched controls who were normotensive. The changes in placenta, variation in the birth weight and differences in the feto placental index were studied. Antenatal patients with blood pressure more than 140/90 mm of hg with proteinuria were included in the study.

Age matched normotensive non proteinuric antenatal patients for control study was included. Cases with chronic hypertension gestational hypertension, diabetes with hypertension, SLE were excluded.

A detailed case sheet was recorded. BP is recorded in left lateral position in the ward and in sitting posture in outpatient and urine was tested for protein in random clean catch sample. After delivery the foetus is examined for APGAR score, features of prematurity, IUGR, weight of foetus. The placenta is examined in detail, the membranes, cord, foetal surface, maternal surface, any abnormalities in the placenta, weight of placenta and meconium staining were noted in the placenta and umbilical cord also.

The cross section of placenta is examined for infarcts, intervillous thrombus, fibrinose deposits and calcification. 2cms piece of placenta that included foetal and maternal surfaces were selected for fixation, from which 0.5cms was selected for processing in formalin.

Histopathological examination for the presence of calcification, syncitial knots, degeneration, focal syncitial necrosis, cytotrophoblast hyperplasia, basement membrane thickening of trophoblast, fibrinous deposits in vessels were noted, and the results obtained are recorded and compared between the study and control group.

#### IV. Results

Maternal Clinical Features in 60 antenatal patients including both cases and controls (n=30 each)

Clinical Features	Cases (n=30) (%)	Controls (n=30) (%)	P Value
Age (Years)			
17-21	14(46.66)	11(36.66)	0.432
22-26	12(40.00)	14(46.66)	0.604
27-31	4(13.33)	5(16.66)	0.317
Primi Gravida	19(63.33)	11(36.66)	0.038*

\*p value < 0.05 is considered significant

Table: 1

Gestational Age (wks)	Cases (n=30) (%)	Controls (n=30) (%)	P Value
27-32	8(26.66)	0	0.0078*
33-37	11(36.66)	2(6.66)	0.0121*
38-42	11(36.66)	28(93.33)	0.0003*
Previous History of Pre-eclampsia	10(33.33)	1(3.00)	0.0076*

\*p value < 0.05 is considered significant

Table: 2 Fetal Outcome in Cases and Controls

Parameter	Cases (n=30) (%)	Controls (n=30) (%)	P Value
Birth Weight (<2500 Grams)	17(56.66)	4(13.33)	0.0011*
Feto-Placental Index			
3-5	14(46.66)	18(60.00)	0.3007
5-7	16(53.33)	12(40.00)	0.3007

\*p value < 0.05 is considered significant

Table: 3 Gross Morphology of Placenta observed

Features	Cases (n=30) (%)	Controls (n=30) (%)	P Value
Placental Weight (<500 grams)	17(56.66)	4(13.33)	0.0011*
Infarcts	15(50.00)	4(13.33)	0.0055*

\*p value < 0.05 is considered significant

Table: 4 Microscopic features of Placenta

Features	Cases (n=30) (%)	Controls (n=30) (%)	P Value
Calcifications	12(40.00)	5(16.66)	0.0449*
Increased Syncitial knots	18(60.00)	6(20.00)	0.0015*
Fibrinous deposits in the arteries	10(33.33)	2(6.66)	0.0238*
Cytotrophoblastic Hyperplasia	15(50.00)	2(6.66)	0.0005*
Basement Membrane Thickening of Trophoblast	18(60.00)	5(16.66)	0.0005*
Syncitial Degeneration	10(33.33)	1(3.33)	0.0076*
Focal syncitial Necrosis	8(26.66)	1(3.33)	0.0300*
Degeneration of occasional Cytotrophoblasta	9(30.00)	0	0.0038*

\*p value < 0.05 is considered significant

#### II. Statistical Analysis And Observation

1. Among the maternal clinical features compared pre-eclampsia was seen in primigravida and in those with previous history of hypertension.
2. The hypertensive study group was associated with preterm labours p value,<0.05
3. Foetal outcome among the study group showed low to very low birth weight babies p value <0.05
4. Foeto-placental index of the study group and normal controls was not variable with the p value being >0.05

5. Among the gross morphological features, placental weight and infarcts were statistically important with p value, <0.05
6. All the microscopic features compared between the two groups were found to be statistically significant in the study group with p value <0.05. Cytotrophoblastic hyperplasia and basement membrane thickening of trophoblast were found to be highly significant in diseased group.

### III. Discussion

In the present study, the age range of patient with preeclampsia is between 19 -25 years, and pre-eclampsia is more common in primigravida and in cases with previous history of toxemia of pregnancy. This is also observed in another study which reported similar results in hypertensive pregnant women<sup>(8)</sup>. There is increased trend towards preterm deliveries, IUGR, low birth weight compared to normal pregnancy. However there is no significant difference in fetoplacental index as reported<sup>(7)</sup>.

Placental weight was lesser than the normal range of 500 grams. Placental infarcts were common in study group. Syncytial knots were increased in the pre eclampsia group which are formed due to imbalance between the production and shedding of villous trophoblast. Cytotrophoblastic hyperplasia is seen in the diseased group which is related to hypoxia. Syncytial degeneration and focal syncytial necrosis<sup>(12)</sup> was seen pre-eclampsia group. This is due to maximal villous cytotrophoblastic proliferation without syncytial fusion due to hypoxia.

This is supported by a study of Fox et al<sup>(6)(11)</sup>. There is undue thickening of basement membrane of trophoblast which is the result of increased secretion and decreased turnover of basal lamina molecules due to hypoxic injury. This is supported by a study reported by Aparna et al<sup>(9)(11)</sup>

### IV. Conclusion

It is thus concluded that pre eclamptic toxemia is a condition of placental insufficiency mostly seen in primigravida. Birth weight was reduced and IUGR in seen. Reduction in weight of placenta and infarcts on gross examination was observed. Microscopic examination revealed increased Syncytial knots, cytotrophoblastic hyperplasia, basement thickening of trophoblast, syncytial degeneration and focal syncytial necrosis.

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