HISTOPATHOLOGICAL STUDY OF PLACENTAL VILLI IN PRE-ECLAMPSIA- A QUANTITATIVE STUDY

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ABSTRACT

Placenta is a vigorous ,energetic, resourceful organ which shows compensatory changes in response to hypertension .A study was done to find the histopathological changes in placentae of pre-eclamptic mothers in comparison to mothers with normotensive pregnancies.. Histopathological findings like syncytial knot formation, cytotrophoblastic cellular proliferation and fibrinoid necrosis were present in greater amount in placentae from pre-eclamptic mothers .

Key Words: preeclampsia, syncytial knots, cytotrophoblastic cellular proliferation ,fibrinoid necrosis, placenta.

INTRODUCTION

Benirschke[1] observed that physicians generally are uncomfortable with the tasks of examining the placenta. Yet it is a task they should willingly undertake ,submitting this organ to a reasonable knowledgeable look and touch which can provide much insight into prenatal life.

The development of foetus depends on the quality and quantity of maternal blood delivered to the intervillous space of the fetal placenta. Pregnancy complications like hypertension or gestational diabetes are reflected in the placenta in a significant way(both macroscopically and microscopically[2].

Preeclampsia is a multisystem disorder of unknown etiology characterized by development of hypertension to the extent of 140 / 90 mm Hg or more with proteinuria induced by pregnancy after the 20th week in a previously normotensive and non proteinuric patient (International society for the study of hypertension in pregnancy 1988).

Present study has been undertaken to find out the histopathological changes in the placentae of Pre-eclamptic patients and compare them with that of normotensive patients.

MATERIAL AND METHODS

The study was conducted on 60 cases of pregnant women admitted and delivered in the department of Obstetrics and gynaecology, Subharti Medical College Meerut, in which 30 cases were of uncomplicated pregnancy and 30 cases of pre-eclampsia.

Control group, included cases with normal blood pressure without edema or proteinuria. Study group included cases having blood pressure ranging 140/90 mmHg and above, with or without edema,and/or proteinuria. All those Pre-eclamptic cases having any other associated diseases like Diabetes Mellitus ,Jaundice were excluded from the study.

All placentae were collected immediately after delivery from labour room and gynaecology operation theatre, Department of Obstetrics and Gynaecology, SMC ,Meerut. Placentae of each group were kept in separate containers filled with 10% formalin soution.for one week. From each placenta whole thickness tissue blocks were taken from center of the placenta, periphery and from any fibrotic or infarcted area if present.The tissue was subjected to further histological processing. Minimum of three,-3-5 micron thick paraffin sections were cut & stained with hematoxylin & eosin and studied under light microscope. 10 fields in high power were studied in each section.

OBSERVATIONS

A total of 200 villi were counted in each slide and continuous variables for each parameters were expressed as mean ± standard deviation. Unpaired t-test for independent samples was used for continuous variables between two group

On examination under microscope, significant ($p<0.001$) number of syncytial knots (fig.1,3 &4), cytotrophoblastic proliferation (fig. 2&5) and fibrinod necrosis (fig. 1,3 & 6) were observed in the pre-eclamptic group in comparison to control group.

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Histological Features (per HPF)	Control Group (n=30) mean ± S.D.	Study group (n=30) mean ± S.D.	P value
Syncytial knots	19.0 ± 3.5	46.8 ± 5.4	t=23.2 p<0.001
Cytotrophoblast cell proliferation	5.9 ± 1.0	22.2 ± 5.1	t=17.1 p<0.001
Fibrinoid necrosis	2.1 ± 1.0	7.8 ± 0.9	t=54.4 p<0.001

TABLE : HISTOLOGICAL STUDY OF PLACENTAL VILLI

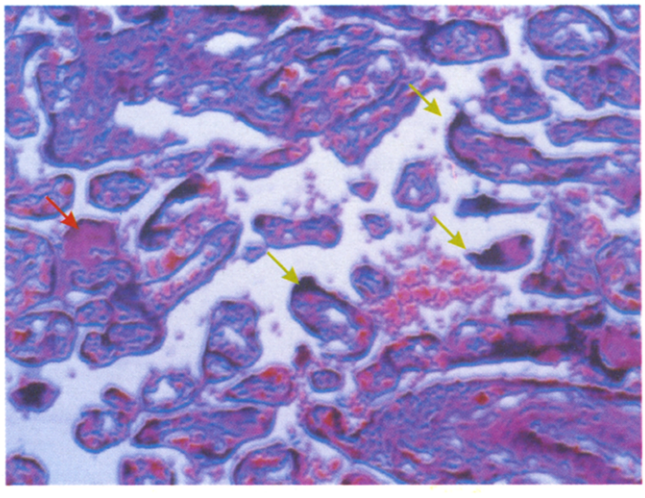

Fig-1: Photomicrograph of placenta (Control Group)- Syncytial knots (green arrows) fibrinoid necrosis (red arrow) (H & E x 100)

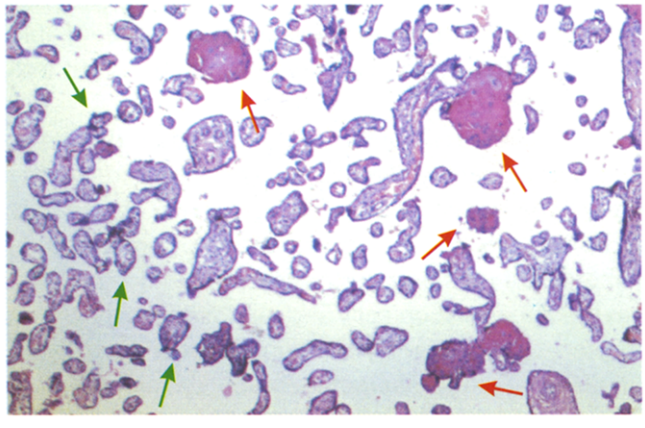

Fig-3: Photomicrograph of placenta (Study Group)- Syncytial knots (green arrows) fibrinoid necrosis (red arrows) (H & E x 100)

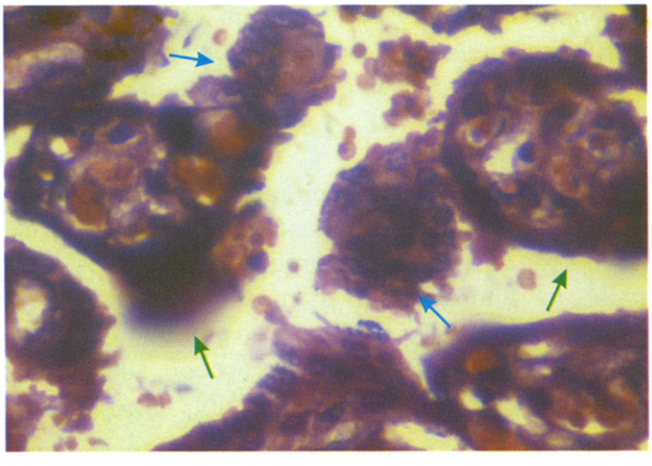

Fig-2: Photomicrograph of placenta (Study Group)- syncytial knots (green arrows) cytotrophoblastic proliferation (blue arrow) (H & E x 400)

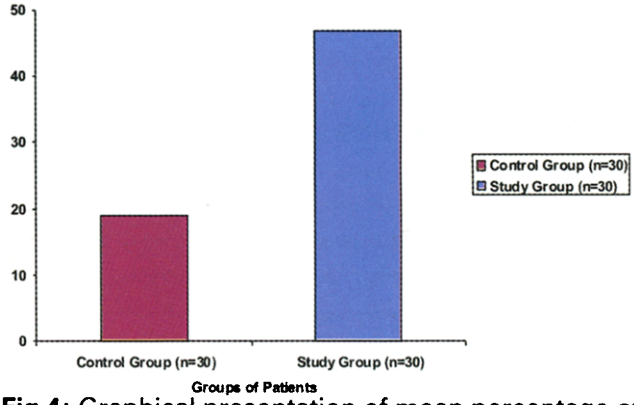

Fig 4: Graphical presentation of mean percentage of syncytial knots

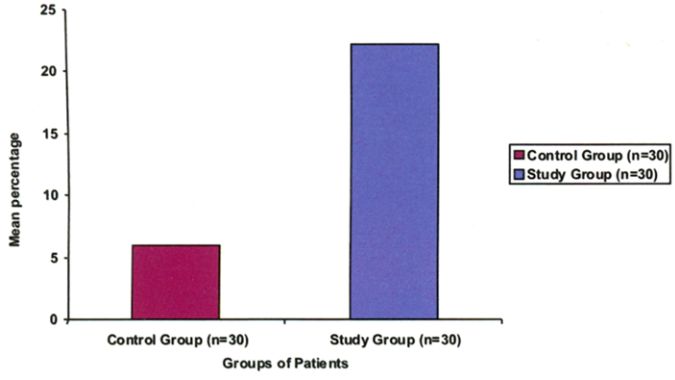

Fig 5: Graphical presentation of mean percentage of cytotrophoblast cell proliferation

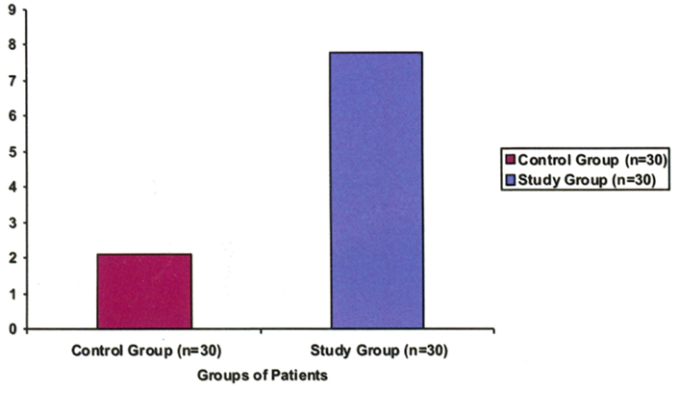

Fig 6: Graphical presentation of mean percentage of fibrinoid necrosis

The study reveals that the mean syncytial knot count in placentae of control and study groups were 19.0+3.5 and 46.8+5.4 respectively .p-value calculated by unpaired t- test was <0.001.The mean cytotrophoblastic cellular proliferation in control and study group was 5.9 +1.0 and 22.2+ 5.1 respectively . p value calculated by unpaired t- test was <0.001. Fibrinoid necrosis observed in placentae of control group was 2.1+ 1.0 and in that of study group was 7.8+ 0.9 . p value calculated by unpaired t-test was <0.001.

DISCUSSION:

Syncytial knots are focal clumps of syncytial nuclei that protrude into the intervillous space from the surface of the villi. Sauramo[3] and Muller et.al[4] suggested that an excessive formation of syncytial knots is a feature of pre- eclampsia. Mallik et.al[5] found that in normal pregnancy the incidence of syncytial knots never exceeded 60% and was found usually to be less than 30%. Masodkar et.al[6] in their study found that 69% of placentae in pre eclampsia group had syncytial knot count more than 30%, justifying the findings of our study. On post mortem examination, Genset[7] reported that stromal fibroses and excessive syncytial knot formation are seen in generalized form as invariable results of overall reduction of foetal perfusion of the placenta. According to study conducted by Majumdar S. et al.[2], the histological findings like syncytial knot formation, cytotrophoblastic cellular proliferation , fibrin plaque formation were present in greater amout in hypertensive placentae.

Wigglesworth[8] suggested that the proliferation activity in the cytotrophoblast is a response to uteroplacental ischaemia. These cytotrophoblastic cells proliferate in an attempt to repair and replace ischaemically damaged syncytiotrophoblast.Results of our study are in accordance with that of Anderson and Mackay[9] who found an apparent increase in the number of cytotrophoblastic cells in pre-eclampsia and the results of their study were similar to results of studies conducted by Jones and Fox[10].

Fibrinoid necrosis appears as a nodular mass of homogenous acidophilic material ,around the periphery of which are a few degenerate syncytial nuclei .The villous fibrinoid necrosis is actually a form of senile amyloidosis , the amyloid being deposited as a result of immune attack on trophoblastic cells with misspecified protein synthesis (Burstein et al)[11]. Mallik et.al[6] reported the incidence of increased fibrinoid necrosis in (80%cases). According to Teasdale[12] and Udainia et al[13],microscopic findings of localized fibrinoid necrosis ,endothelial proliferation of arteries and hyalinization depict the mosaiscism of placenta and probably the aftermath of hypertension,our results are in agreement with the results of above mentioned studies.

CONCLUSION:

On the basis of the above study it has been concluded that there is an improper placental development in preeclampsia. Reduced maternal uteroplacental blood flow leads indirectly to construction of fetal stem arteries and finally results in changes seen in the placenta of pre-eclamptic women. Maternal vasopasm leads to foetal hypoxia, which is responsible for fetal jeopardy.So special precaution can be instituted during antenatal period and labour in subsequent pregnancies to reduce further risk to the mother and foetus.

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