Review Article

"A REVIEW OF MORPHOMETRICAL, HISTOLOGICAL AND RADIOLOGICAL EXAMINATION OF PLACENTA IN TOXAEMIA OF PREGNANCY"

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Abstract: The placenta is a unique organ, short lived by design. Its existence is essential for the survival of human embryo/foetus in the intra uterine environment. As the placenta is the direct link between mother and foetus, the examination of placenta gives a clear idea of what had happened with it, when it was in the mother's womb and what is going to happen with the foetus in future. Now a days, hypertensive disorders complicating pregnancy (Toxaemia of pregnancy) are common and forming a deadly triad along with haemorrhage and infection.Maternal hypertension is diagnosed in 7% of all deliveries; is associated with 22% of all perinatal deaths and 30% of all maternal death. A number of microscopic abnormalities in the villi like decreased villous vascularity, basement membrane thickening, stromal fibrosis, cytotrophoblastic cell proliferation, syncytial knot formation and villous fibrinoidnecrosis, These are thought to represent a response, often a compensatory in nature to the disturbances in blood flow. It has been emphasized that the most striking changes are cytotrophoblastic cell proliferation and thickening of basement membrane.

Key words: Placenta, Pregnancy induced hypertension (PIH), Pre-eclampsia(PE), Eclampsia, Intra uterine growth retardation(IUGR).

Introduction: The placenta is a unique organ, short lived by design. Its existence is essential for the survival of human embryo/foetus in the intra uterine environment. The placenta performs diversity of functions, ranging from anchoring the fertilized ovum, preventing its rejection by the maternal immune system to enabling the transport of nutrients and wastes between the mother and the embryo/foetus[1]. The placenta was first recognized by an early Egyptian named Realdus Columbus in 1559. The word placenta comes from the Latin word Plakos means "cake" or from Greek Plakoenta meaning "flat, slab-like", referring to its round and flat appearance in humans[2,3].

The placenta is the most accurate record of infant's prenatal experiences. Generally physicians are uncomfortable with the task of examining the placenta, they should willingly undertake it because submitting this organ to a knowledgeable look and touch can provide much insight in to

prenatal life[4].Structural and functional derangement of placenta evokes a considerable interest, as this may be the only yardsticks to measure adequacy of the foetal environment[4].

As the placenta is the direct link between mother and foetus, the examination of placenta gives a clear idea of what had happened with it, when it was in the mother's womb and what is going to happen with the foetus in future[5]. Since the substance required for foetal metabolism comes from the mother's blood and foetal catabolites are passed back in to the mother's circulation through the placenta and umbilical cord, examination of placenta is very important to know what had happened to foetus in the gestational period[5].

Now a days, hypertensive disorders complicating pregnancy (Toxaemia of pregnancy) are common and forming a deadly triad along with haemorrhage and infection. Pre-eclampsia (PE) is considered to be severe if one or more of the following criteria are present: when systolic blood pressure of 160 mmHg or higher or diastolic blood pressure 110mmHg or higher on two occasions at least 6 hours apart while the patient is in semi sitting position, proteinuria of 5grms or higher in a 24hrs urine specimen or at least +2 protein on stick test, oliguria, cerebral or visual disturbances, pulmonary edema or cyanosis, epigastric or upper quadrant pain, impaired liver function, thrombocytopenia and foetal growth restriction[6]. Conflicting findings have been reported regarding the placental abnormalities, both gross and microscopic in hypertensive pregnancies. Pregnancy induced hypertension (PIH) is a cause of large number of maternal deaths and thereof foetal deaths. Maternal hypertension is diagnosed in 7% of all deliveries; is associated with 22% of all perinatal deaths and 30% of all maternal death[7].In some mysterious way, in certain women, the presence of chorionic villi with or without a foetus incites vasospasm and hypertension[8].As a consequence of this vasospasm; villi in these placentas are subjected to a reduced maternal utero-placental blood flow[9].

A number of microscopic abnormalities exist in the villi like decreased villous vascularity, basement membrane thickening, stromal fibrosis, cytotrophoblastic cell proliferation, syncytial knot formation and villous fibrinoidnecrosis[10].These are thought to represent a response, often a compensatory in nature to the disturbances in blood flow. It has been emphasized that the most striking changes are cytotrophoblastic cell proliferation and thickening of basement membrane [11].

The present review study concentrates on morphological, histological and radiological findings of placenta in toxaemia of pregnancy (pregnancy induced hypertension).

Morphometrical Examination: In Morphometrical examination of placenta, various workers have mentioned parameters such as placental weight, surface area, volume, foeto-placental weight ratio, insertion of umbilical cord and birth weight of the neonates.

According to Damania et al.,(1989)[12]and Mohan (1989)[13] the placental weight is reduced significantly in PIH mothers, and

the average weight of placenta is reduced proportionately with degree of hypertension. In the study of Das et al.,(1996)[14], Collins et al.,(2005)[15]and Majumdar et al.,(2005)[16] noticed weight of placenta was significantly lower in PIH group due to placental insufficiency.

In his study Collins et al.,(2005)[17]and Rath G et al.,(2000)[18] have found the placental weight and size were directly proportional to the birth weight of neonates.

Vaibhav et al.,(2014)[19]in their study mentioned the average weight of babies delivered from PIH and normal mother as 2279.14+/-418.08grams, 2651.18+/-392.00gramsrespectively. They also mentioned the placental weight in PIH & normal mothers as 306.45+/-78.74grams, 408.14+/-54.78grams respectively. With reference to these values it is inferred that placental & foetal weight is decreased significantly to the increased degree of hypertension. These values support the concept that placental weight & foetal weight are directly proportional to each other.

Pasaricha Navbir(2012)[20] observed that 80% of eclamptic placentas had weight less than 400grams. 60% of moderate pre-eclamptic placentas have weight less than 400grms while 40% of mild preeclamptic mothers having placental weight less than 400grms. These results support the hypothesis mentioned by various workers that placental weight goes on decreasing with increasing hypertension due to reduced placental blood flow. According to Vaibhavet al., (2014)[19]placental surface area is also reduced in PIH mother as compared with normal mother. He observed the mean placental surface area in normal as 221.99+/-50.00sqcm, and in toxaemia of pregnancy 182.84+/-56.71sqcm.

Nagu et al.,(2013)[21]mentioned that placental surface area in normal mother was 280+/-72sqcm, and in hypertensive mother 211+/-56sqcm. Various workers have mentioned that placental surface area is significantly decreased in toxaemia of pregnancy as compared with normotensive mother[15,16,18].

On the other hand wide variations in the incidence of placental infarction have been reported by Fox and Langely.,et el,(1970)[8] ranging from 34% in women with mild preeclampsia and 60% in severe form of the disease.

Salvatore (1963)[22] has noted the incidence of red and white infarcts 34.3-66.6% and40.6-65.5respectively in PIH placenta. With reference to these values he has stated that infarcted areas are more in severe form of eclampsia as compared to normal placenta. Other parameters such as placental volume and foeto-placental ratio show significant differences. According to Vaibhavet al.,(2014)[19] foeto-placental ratio in normal mother is 6.49+/-0.55 and in hypertensive mother is 7.75+/-1.8. According to Nagu et al., (2013)[21] average foeto-placenta ratio in normal mother was5.94+/-0.86 and in hypertensive mothers is 6.02+/-0.36. These results showing that foeto-placental ratio is significantly increasing in PIH mothers than normotensive mothers.

Histologial Examination: In histological examination of placenta in PIH, changes like decreased villous vascularity,

basement membrane thickening, stromal fibrosis, cytotroblastic cell proliferation, syncytial knot formation and villous fibrinoid necrosis have been reported[5].

Syncytial knots are consistently increasing with increased gestational age can be used to evaluate villous maturity. A study conducted by Sodhi et al.,(1990)[23] revealed that increased syncytial knots are associated with conditions of uteroplacental malperfusion and are important in placental examination.

Sodhis et al, (1990)[23], Heazella AEP (2007)[24] and Kristinal (2009)[25] reported that increased number of syncytial knots in placentas of mothers with hypertension disorders lead to foetal growth restriction (FGR) induced by hypoxia.

Syncytial knot counts were found to be significantly higher in mild, moderate preeclampsia and eclampsia as compared with control normotensive group in Kalra et al.,(1985)[26]study.Increased number of villi syncytial knots formation is attributed to decreased foetal perfusion of the villi reported in Bhatia et al (1985)[27] and Masodkar et al(19885)[28] study.

Fox (1970)[8] in his study demonstrated that 66.7% (20/30) cases of hypertensive pregnancies had excess syncytial knot counts, in them 3cases 18.75% had neonatal asphyxia and 5 cases (35%) had low birth weight neonates. The other parameters such as vasculosyncytial membrane also show significant differences in normal & hypertensive mother. Fox (1970)[8] also found a low vasculosyncytial membrane count (4.5-5.3%) in placenta from preeclampsia group & considered this as a manifestation of villous regression.

Kher and Zawar(1981)[29], Mathews et al.,(1973)[30]and Mirchandani (1970)[31] have also commented upon association of vasculosyncytial membrane deficiency and poor foetal outcome observed in their studies. The other histological findings such as basement membrane thickening, stromal fibrosis, cytotrophoblastic cell proliferation, fibrinoid necrosis were also increased in mild,moderate preeclmpsia and in eclampsia when compared with control group reported in Mirchandani JJ (1974)[31], Sen DK(1974)[32]study.

Mirchandani et al.,(1979)[31] and Send DK (1974)[32] in their study noted that undue thickening of basement membrane and fibrinoid necrosis have been considered as manifestation of antigen and antibody reaction in the body.

Radiological Findings: In Radiological examination of placenta by Russel JGB(2005)[33], Spirit BA(1982)[34], Sarkar et al., (2007)[35] and Narasimha(2011)[36] have noticed that calcification of placenta from 1mm to biggest of 5mm were seen significantly more frequently in Prime gravidas with hypertension.

The mean number of calcified areas seen on gross as well as microscopy were more in PIH than in normal placenta; in a study by Sarkar M(2007)[35]. The presence of preterm placental calcification is a predictor of poor uteroplacental outcome which needs closer monitoring for maternal and foetal well-being as stated by Narasimha et al.,(2011)[36].

Conclusion: Thus from the above literature, it can be concluded that:

1. The birth weight of the babies and placental weight in pregnancy induced hypertension are reduced.
2. The mean surface area and volume of placenta is also decreased in PIH group than normal group pregnancies.
3. The foeto-placental weight ratio is increased in PIH.
4. Number of infarcts in placenta is also increased in PIH due to reduced blood flow.
5. Findings like syncytial knots & hypovascular villi were more in PIH group due to reduced villous blood flow.
6. Immunological reactions of placental tissue in PIH gwere attributed to be the cause of increased stromal fibrosis &fibrinoid necrosis .
7. The number of calcified area in the placenta are also more in PIH.

As per the above literature, we can also derive a conclusion that the patients with PIH have increased chances of ischemic damage in the placental tissue along with maldevelopment of terminal villi. These findings may account for impaired diffusion of gases and nutrients to the foetus resulting in low birth weight, preterm deliveries and intra uterine growth retardation.

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