A STUDY OF HISTOPATHOLOGICAL CHANGES OF PLACENTA IN PRE ECLAMPSIA AND PERINATAL OUTCOME

Vijayalakshmi B¹, Sunitha Kitteli²

HOW TO CITE THIS ARTICLE:

Vijayalakshmi B, Sunitha Kitteli. "A Study of Histopathological Changes of Placenta in Pre Eclampsia and Perinatal Outcome". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 67, August 20; Page: 11667-11673, DOI: 10.14260/jemds/2015/1682

ABSTRACT: BACKGROUND: Placenta is multifunctional organ strategically positioned between the uterus and the mother. It functions as an organ of transportation, respiration, execration of waste products endocrine and barriers to the extent where these function dependent on histological development at molecular and biological levels. **OBJECTIVE:** To study the histological changes of placenta with severity of pre-eclampsia (Mild and severe) and perinatal outcome. **METHODS:** 200 placentas of hypertensive patients (100 mild pre-eclampsia, 100 severe eclampsia were studied histologically for subchorinic fibrin, Retro placental hematoma, Infraction, clarification these placental findings were correlated with fetal outcome. **RESULTS:** The presence of infraction, retro placenta with Infraction and retro placental hematomas. **CONCLUSION:** The hypertensive disorders of pregnancy adversely influence the histopathology of the placenta. The pathological changes observed in placenta of patients with Hypertensive disorder of pregnancy like retro placental hematoma and infection adversely influence the perinatal outcome however none of these pathological changes of placenta were specific to hypertensive disorders of pregnancy.

KEYWORDS: Placenta, infraction, Retroplacental hematoma, Calcification, Subchornic fibrin, Fetal outcome.

INTRODUCTION: In recent years placenta has drawn attention as valuable indicator for maternal and fetal wellbeing and disease. Histologically the placenta of hypertensive disorder of pregnancy have high incidence of infarction, retro placental haematoma, sub chorionic fibrin areas. Fetal outcome is adversely influenced by these pathological changes occurring in placenta due to hypertensive disorder of pregnancy. As patho physiology lies in placenta most attention is drawn to examine the placenta in recent years as it give valuable indication for maternal and fetal disease many of disorders of pregnancy which are associated with high perinatal morbidity and mortality are accompanied by this pathological changes in placenta¹ most obstetricians and pediatricians would agree that the examination of placenta often helps to explain an abnormal neonatal outcome. Despite the understand and appreciation of placental disease, great resistance still exists in performing placental examination routinely therefore the placenta has remained neglected and mysterious organ. So detailed histological study of placenta was done to know significant changes that occur in case of pre-eclampsia.

OBJECTIVE OF THE STUDY:

- To study histological changes of placenta in case of pre-eclampsia.
- To correlate this histological changes of placenta with severity of pre-eclampsia (Mild and severe) and perinatal outcome.

MATERIAL AND METHODS: a total of 200 placenta of pre-eclampsia (Mild 100, severe 100) cases between 34 to 42 weeks of gestation studied for histopathological changes from December-2011 to September-2013 in VIMS, OBG Dept. Ballari.

Inclusion Criteria: All pregnant women admitting to the labor room with gestational age >34 weeks with pre-eclampsia (Mild, severe).

Exclusion Criteria:

- 1. Twin pregnancy.
- 2. Gestational diabetes, Rh negative pregnancy, pregnant women with heart disease, other medical disorders.
- 3. Chronic hypertension.
- 4. Placenta previa.
- 5. Pregnant women with auto immune disorders (SLE).
- 6. Eclampsia.

Statistical Analysis: Various parameters in the study group was analyzed by ANOVA and by students test categorical data was analyzed by Chi- Square Test.

Investigations:

- 1. Urine: Sugar, Albumin, Microscopy.
- 2. Blood: Hb %, Blood Grouping and Rh typing.
- 3. HIV, HBsAg.
- 4. Blood Urea, and uric acid, Serum Creatinine.
- 5. Platelet Count, Liver Enzymes.
- 6. Fundoscopy.

Abnormality	Mild	Severe			
Diastolic Blood Pressure	< 100 mmHg	110 mmHg or Higher			
Protein urea	Trace to 1+	Persistent 2+, more			
Headache	Absent	Present			
Visual Disturbance	Absent	Present			
Upper abdominal pain	Absent	Present			
Oliguria	Absent	Present			
Serum Creatinine	Normal	Elevated			
Thrombocytopenia	Absent	Present			
Liver enzyme elevation	Minimal	Marked			
Fetal Growth restriction	Absent	Obvious			
Pulmonary oedema	Absent	Present			
Diagnostic Criteria indicating mild and severe pre-eclampsia					

RESULTS: A total no of 200 cases (100 mild and 100 severe) cases were studied for histopathological changes which include:

- **a. Syncytial Knots:** In the present study we found significantly higher syncytical knot counts about 66.67% cases and 18.75% Infants had neonatal as PHYSCIA and 35% had low birth weight. In case number of vilri showed syncytial knots in conditions like,² Maqueo et al,³ et at,⁴ Mathews et al,⁵ Masodkar et al.⁶ Mehrotra et at.⁷
- **b. Fibrinoid Necrosis:** Present study revealed statically significant areas of fibrioid necrosis, mild 4.45±3.31 and severe 6.74±5.21. This finding similar to Muzumade S. et at 2005). We observed statically significant association between NICU admission and fibrinoid necrosis in case group. This is similar to pasricha navbir study.
- **c. Hyalinisation of Villi:** We observed increased incidence of Hyalinisation of Villi in severe preeclampsia cases compared to mild pre-eclampsia this findings are similar to study by Mujumadar et at(2005) wed did not find any association between its occurrence and fetal outcome.
- **d. Calcification:** These was increased incidence of calcification in present study in server PF group 35% compared to mild PF 13%. This is similar to study by aparna narasimha¹ DS vasydeva² (2011) in present study we observed in case in mean calcified areas per LPF (Low power field) in severe PF 10.55±3.01 Compared to mild PF 7.46±1.09 and 1.20±1.42.

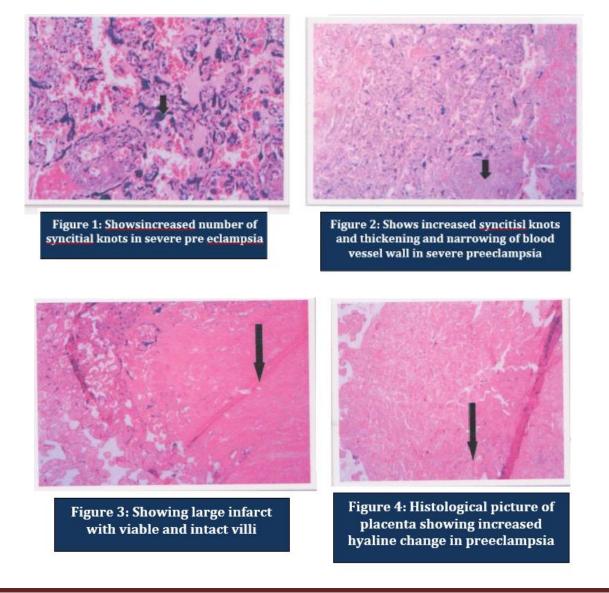
This observation is statically significant and was comparable to study by pushpa goswami (2011). 10

e. Infraction: Placental infraction of more than 5% surface area was considered pathological. In the present study infraction was seen in 48% of severe PF compared to 25% of mild PF. This is in comparison with MASODKAR ET AT'S 40.4%¹¹ and UDAINIA ET AT (2004).¹²

Studies		Mean no. of syncytial Knots/IPF	Mean no. of Fibrinoid necrosis/IPF	Mean No. of Hyalinised Villi/IPF	
Present Study	Mild PF	31.90±11.0	4.45±3.31	9.37±1.25	
	Sevre PF	46.7±11.81	6.74±5.21	13.04±3.23	
	P. Value	0.001	0.001	0.001	
Meymdas et al (2005)	Hypertensive	27.1±3.24	11.3±2.3	11.1±1.82	
	P. Value	0.001	0.001	0.001	
Parricha Navbir (2012)	Mild PF	27.36±6.90	7.79±3.33		
	Severe PF	40.20±9.78	10.8±2.53		
Comparison of Histopathological changes in different Studies					

DISCUSSION: Placenta is a vital organ maintaining pregnancy and promoting fetal development, which functions as fount upon which developing fetus derives its nutritional substances and obtain its metabolic and immunological requirements the underlying pathological types can be best identified by clearly defined histology.¹³ Although dissenting opining have been propounded in the examination is currently well accepted. The etiology and pathological Mechanism are still not understood however it in generally accepted that the placenta in the key organ in the pathogenesis of

disease. One important mechanism is defective remodeling of the spiral arteries, known as deciduas arteriopathy, that could contribute to the hypoxic environment and there by placental insufficiency which is found in pregnancies with pre eclampsia.^{14,15} This results in histopathological changes in placenta and fetal hypoxia leading to IUGR and preterm delivery and fetal death. Maternal morbidity remains great with pre eclampsia, which continued to be one of the leading causes for the admission of pregnant women to intensive care units. Furthermore, fetal mortality and morbidity in considerable, related to the effects of the disease on the fetus as well us prematurity although the study of placenta is retrospective in nature, yet it provides a reflection of hazards the fetus has been subjected to during its growth and development. In the earlier studies of the effect of maternal disease on placenta, gross abnormality of the placenta has received undue attention and undeserved status. It is difficult to define the normal placental finding and differentiate it from the abnormal, because of the structural complexity and rapid evolution of the placenta. FOX (1968) suggested that placental pathology is quantitative rather than qualitative BENRICHEAND FOX (1975), stressed the significance of placental findings only when there has a bearing on the fetal outcome.^{16,17}



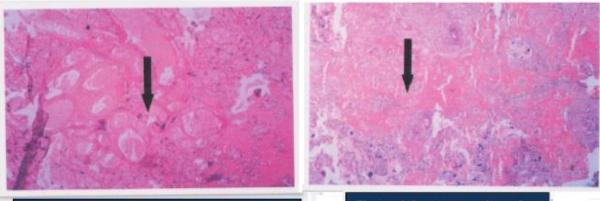


Figure 5: Showing haemorrhagic changes with multiple microthrombi of blood vessels

Figure 6: low power view of an infarct area with goust villi

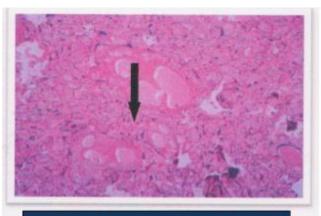


Figure 7-low power view of fibrin microthrombosis of the arterioles



Figure 8- showing a large focal area of calcification with partial clearing

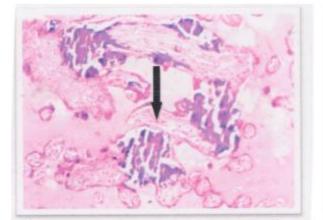


Figure 9: Shows high power view of microcalcification in preeclampsia

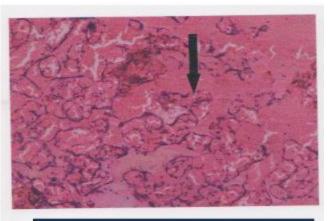


Figure 10: Showing haemorrhagic necrosis

CONCLUSION: Patients with pregnancy induced hypertension have increased chance of ischemic damage to placental tissue along with maldevlopment of terminal villi. These finding may account form impaired gas and nutrient transfer in this disorder, there by resulting in low birth weight babies. In essence most of the important placental changes represent placental ischemia, secondary to reduced blood flow. The three main gross lesions which were observed were placental infracts, retro placental haematoma, and calcification and there incidence was higher in hypertensive disorder of pregnancies. Placental clarification was seen more commonly in this study. The pathological changes like incidence of infraction, calcification was higher in severe PE compared to mild PE. The other histopathological changes like fibrinoid necrosis, hyalinization of villi were significantly increased in severe PE and fetal outcome was poor if the severity of the disease increased.

REFERENCES:

- 1. Bandana Das, D. Dutta, S. Chakraborthy, P. Nath: Placental Morphology in Hypertensive disorder of pregnancy and its correlation with fetaloutcome. J. Obstetand Gynecol India, 1996; 46 (1): 40-46.
- 2. Feye-Petersen OM HD, Joshi VV Handbook of Placental Pathology. Second ed. United Kingdom: Taylor and Francis; 2006.
- 3. Mawueo M, Azuela JC, Manuel Dosal de la Vega, Placental Pathology in eclampsia and preeclampsia, Obstet Gynacol, 1964; 24:350.
- 4. Kalra VB, Aggarwal A, Sareen PM, Histopathological changes in placenta in toxemia of pregnancy, J Obstet Gynacol, 1985; 35-86.
- 5. Mathews R, Aikat M, Aikat BK, Morphological studies of placenta in abnormal pregnancies, Indian J Pathol Microbiol, 1973; 16:15.
- 6. Masodkar AR, Kalamkar LR, Patki PS, Histopathology of Placenta and its correlation with fetal outcome, J Obstet Gynacol, India, 1985; 35-294.
- 7. Mehrotra VG, Mukherjee K, Pande M, Gurtu M, The histological Study of Placenta in normal and abnormal pregnancy, J Obstet Gynacol, India, 1972; 22-248.
- 8. Pushpa Goswami, Hemalatha, Samreen Memon, Lal Baksh Khaskhelli, JLUMHS Septemberdecember 2012; Vol-11.
- 9. Mirchandani JJ, Malik GB, Chitra S. Correlation of Fetal outcome with some pathological changes of Placenta. J Obstet Gynacol, India, 1979; 29:1131-9.
- 10. Udainia A, Bhagwat SS, Mehta CD, Relation between placental surface area infraction and fetal distress in pregnancy induced hypertension with its clinical relevance. J Anat Soc India 2004, 53; 1:27-30.
- 11. Redline RW, Wilson-Costello D, Borawski E, Fanaroff AA, Hack M. Placental Lesions associated with neurologic impairment and cerebral palsy in very low-birth weight infants. Arch pathol Lab Med. 1998; 122 (12):1091-8.
- 12. ACOG practice bulletin. Diagnosis and management of pre-eclampsia and eclampsia Number 33, January 2002. Obstet Gynecol. 2002; 99: 159-67.
- 13. Wang Y., Lewis, D.F., Gu, Y., Zhang, Y., Alexander, J. S and Granger, D.N. (2004) Placental Trophoblast Derived Factors Diminish Endothlial Barrier Function. Journal of Clinical Endocrinology and Metabolism, 89:2421-2428.
- 14. Fox H. Pathology of Placenta Major Problem in pathology. Vol. 7, Philadelphia: WBW aunderscompany Ltd. 1978.

J of Evolution of Med and Dent Sci/ eISSN- 2278-4802, pISSN- 2278-4748/ Vol. 4/ Issue 67/ Aug 20, 2015 Page 11672

15. Benrischke K, Kaufmann P. Pathology of the human placenta 4th ed. New York: Springer Verlang, 2000.

AUTHORS:

- 1. Vijayalakshmi B.
- 2. Sunitha Kitteli

PARTICULARS OF CONTRIBUTORS:

- 1. Associate Professor, Department of Obstetrics and Gynaecology, Vijayanagar Institute of Medical Sciences, Bellary, Karnataka.
- 2. Senior Resident, BIMS, Belagavi.

FINANCIAL OR OTHER COMPETING INTERESTS: None

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Vijayalakshmi B, H. No. 10/2 ward No. 19, Behind Govt. School Patel Nagar, Ballari-583101. E-mail: vijaya.b.yadav72@gmail.com

> Date of Submission: 11/08/2015. Date of Peer Review: 12/08/2015. Date of Acceptance: 14/08/2015. Date of Publishing: 18/08/2015.