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Study of morphological and histological changes of placenta in normal and high risk pregnancies and effect of pregnancy outcome

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Abstract

Background: High risk pregnancies are a small segment of the obstetric population that produces the majority of maternal and infant mortality and morbidity. Upto 40% of high risk mothers experience placental damage. The pathological changes in the Placenta are not by large specific to a particular disorder and therefore a variety of disorders may show similar changes. This study is an attempt has been made to correlate various placental villous changes to the fetal outcome in normal and specific high risk pregnancies.

Aims and Objectives: To study the morphological and histological changes of placenta in normal and high risk pregnancies and effect of pregnancy outcome.

Materials and Methods: This study was done in Department of Pathology Bidar institute of medical sciences Bidar Karnataka.

The study was done from Oct 2020 to March 2021.

It is a prospective study of morphology and histology of placenta in randomly selected normal and specific high risk pregnancies and its correlation with fetal outcome.

The study was done in 60 patients.

Results: There was no significant correlation with calcification, other morphological features such as both the type of placenta, septal cyst, true knot, with fetal outcome.

Conclusion: Days may not be for off when a definite link can be proved in a very positive way.

Keywords: Morphological, histopathological, placenta, normal, high risk

Introduction

Placenta is a vital lifeline between mother and fetus through which nutrients, oxygen, antibodies and hormones pass ^[1, 2, 3]. Improper placentation and placental function could be potentially danger to the health of the mother and the fetus ^[4, 5]. High risk pregnancies are a small segment of the obstetric population that produces the majority of maternal and infant mortality and morbidity ^[6-9]. Upto 40% of high risk mothers experience placental damage. The pathological changes in the Placenta are not by large specific to a particular disorder and therefore a variety of disorders may show similar changes ^[10, 11]. Final picture is often very complicated and no particular complication of pregnancy produces specific morphological changes within the placenta, which allow one to make a specific morphological diagnosis ^[12]. In this study, an attempt has been made to correlate various placental villous changes to the fetal outcome in normal and specific high risk pregnancies.

Aims and Objectives

To study the morphological and histological changes of placenta in normal and high risk pregnancies and effect of pregnancy outcome

Materials and Methods

This study was done in Department of Pathology Bidar institute of medical sciences Bidar Karnataka.

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Each placenta was washed with tap water and drained off its blood. On initial examination of placenta, any gross abnormality of shape, morphometric measurements like size, weight, measurement of cord length and site of cord insertion were noted. For examination of the membranes, a segment of membrane was cut rolled from the margin and pinned for HPE. Cut surface of cord was examined and number of vessels recorded. Each placenta was then trimmed of membranes, weighed and was then examined for

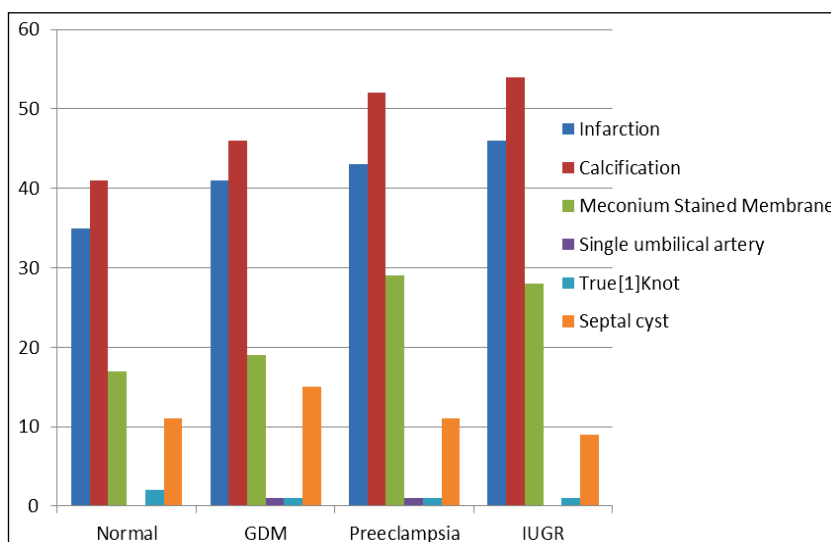
calcification and infarction.

The placenta was dipped in 10% formalin solution for fixation for a period of 48 hrs. They were then cut at every 2 cm interval into vertical strips. 5µ thin sections were cut from each block and stained with haematoxylin and eosin. Two bits from cord were taken. Each section from placenta was examined with light microscope.

Results

Table 1: Macroscopic features of placenta

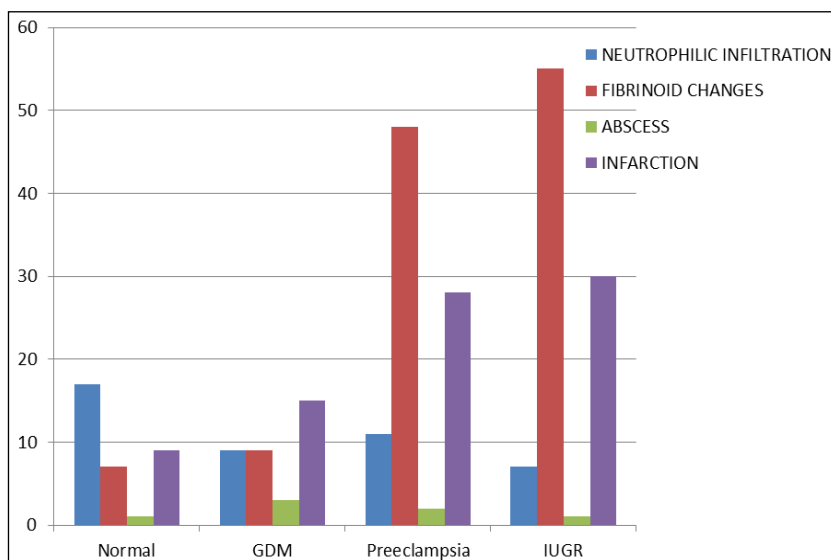
Group	Infarction	Calcification	Meconium Stained Membrane	Single umbilical artery	True Knot	Septal cyst
Normal	35	41	17	-	2	11
GDM	41	46	19	1	1	15
Preeclampsia	43	52	29	1	1	11
IUGR	46	54	28	-	1	09



Graph 1: Macroscopic features of placenta

Table 2: Histological features

Group	Neutrophilic Infiltration	Fibrinoid Changes	Abscess	Infarction
Normal	17	07	1	09
GDM	09	09	3	15
Preeclampsia	11	48	2	28
IUGR	07	55	1	30



Graph 2: Histological features

Outcome

- There was no significant correlation with calcification, other morphological features such as both the type of placenta, septal cyst, true knot, with fetal outcome.
- Neonatal mortality was observed in the 1st week of delivery in cases where we see such histomorphological changes of placenta.

Discussion

Placental pathologic examinations are pointless if they do not provide clinically useful data. One important limitation of placental examination is the inability to make a one to one link between any one placental or uteroplacental vascular lesion and a particular maternal or fetal, neonatal problem. This would be an unrealistic goal, because in no other organs are such relationship the rule. Neither hepatocellular necrosis nor glomerulosclerosis for example, is diagnostic of one and only one hepatic or renal disease; in those diseases individual lesions are considered as part of a greater histopathologic pattern. Finally the pattern is correlated with a variety of clinical data, laboratory data and additional pathologic studies to produce the final clinical pathologic diagnosis. Through examination of the delivered placenta can provide a better picture of the intrauterine environment of the fetoplacental unit than a placental bed biopsy. The ability to routinely identify failure of uteroplacental vascular adaptation, fibrinoid necrosis, atherosclerosis, persistence of endovascular trophoblasts, thrombosis and chronic vasculitis in the basal plate can clarify the nature and mechanisms involved in pregnancy compromise. Many studies are upcoming for placenta screening for high risk pregnancies by using a combination of ultrasound and blood tests to screen high risk pregnant mothers for placental damage. By completing these non-invasive tests, most high risk mothers can be reassured that their placenta is formed and functioning properly, so they can expect a healthy pregnancy. This is an important first step in identifying placental abnormalities in early pregnancy, at a time when a number of interventions can be used to improve outcomes for those with the highest risk. This study will lead the way for future research in placenta screening and help us provide quality care for all mothers.

Conclusion

Many such studies pile up, days may not be far off when a definite link can be proved in a very positive way. This will help to save many babies.

References

1. Benirschke K, Kaufmann P. The pathology of maternal floor infarction. In pathology of human placenta. New York NY-Springer Verlag Inc 1990, 406-411.
2. Benirschke K, Kaufmann P. Pathology of human placenta 3rd edition. Springer Verlag New York 1995.
3. Chrolyn and Chandler. Placental pathology in perinatal diagnosis in Gynecology and Obstetrics. J.B. Lippin Cott. Company. Philadelphia. E/dy^r John J MD. Ph.D. 1992;31(106):1-39.
4. Caspi Soloman, Amnionitis F, Strainmycoplasma T. Am. J Obstet Gynecol 1971;3:1102.
5. Cynthia G, Kaplan. Carolyn Salafia-College of American Pathologists. Conference XIX on examination of placenta. Report of working group on the def of structural changes associated with abnormal functions in maternal/fetal placental unit in 2nd and 3rd trimester.

- Arch. Pathol. Lab. Med 1991;115:709-716.
6. Driscoll SG. Pathology of placenta in intrauterine growth retardation. Ann. Chir, Gynaecol 1987;70:316.
7. Daskalakis G, Marinopoulos Krielesiv, Papapanagiotou A. Placental pathology in women with gestational diabetes. Acta Gynecol Scand 2008;87(4):403-7.
8. Ethches PC. Stewart. The relationship between disorders of umbilical cord and intrauterine growth retardation. Acta. Obstot, Gynecol Scand 1982;72:15.
9. Fox H. Pathology of placenta, London. WB Souder Ltd., 1978. Fox H. Thrombosis of fetal arteries in human placenta, BJOG 1966;73:961-965.
10. Fox H. General pathology of placenta in Fox H. ed.: Obstetrical and Gynecological pathology New York NY: Churchill Livingstone, Inc Z 1987;972:1000.
11. Geoffrey Altshuler. Scott-R-clinicopathological implications of placental pathology clinical obstet and Gynaecol 1996;29:549-570.
12. Gruenwal P. (Ed). The placenta and its maternal supply Line. Lancaster, medical and technical publishing 1975.
13. Hans G Kohler. Pathology of Umbilical cord and fetal membranes in Hainee M. Obstetrical and Gynecological. Path 1987;2:1079-1116.
14. Haust MD. Maternal Diabetes mellitus-effects on the Fetus and placenta, Ch. 8. In Naeye Kissane JM (eds). Prenatal disease, Williams or Wilkins 1981.