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Comparative Analysis of Placental Findings in Gestational Diabetes Mellitus and Pregnancy-Induced Hypertension: A Review

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ABSTRACT

Background: The placenta is a vital part of maternal-fetal exchange and optimal intrauterine development. Gestational Diabetes Mellitus (GDM) and Pregnancy-Induced Hypertension (PIH) are the two pregnancy related complications that cause the highest levels of maternal and perinatal morbidity globally. The two conditions have both far reaching but different influences on the placental structure and functioning. GDM is a metabolic disorder, which occurs due to hyperglycemia in the mother; it triggers both structural and vascular alterations as a compensatory response to allow fetal development. Conversely, PIH which is a vascular condition is linked to uteroplacental dysfunction along with ischemia resulting in degenerative placental pathology. These placental changes should be comparatively assessed in order to understand disease pathophysiology, predict perinatal morbidity, and inform maternal-fetal practice.

Material and Methods: The database of PubMed, Scopus, Embase, and Cochrane Library were searched systematically until 2024 with the following keywords placenta, gestational diabetes, pregnancy-induced hypertension, preeclampsia, histopathology, morphometry. They included original research, reviews and meta-analyses that describe gross and microscopic placenta findings in GDM and PIH. Research claiming biochemical, but not histological, analysis was eliminated. Comparisons of extracted data were done to reveal unique and similar features of the placentas in both diseases.

Conclusion: There is a vast difference between placental pathology in both GDM and PIH. Even though the changes of GDM show more of a hyperplastic and hypertrophic type, PIH displays ischemic and degenerative changes. The identification of these patterns can give a clue of the underlying pathophysiology, improve the accuracy of diagnosis, and possibly a basis of antenatal risk stratification. Placental examination is therefore a very important tool to enhance maternal and perinatal outcome.

KEYWORDS: Placenta, Gestational Diabetes Mellitus, Pregnancy-Induced Hypertension, Histopathology, Perinatal Outcome

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INTRODUCTION

Placental morphology is a key determinant of fetal growth and survival. Pregnancy complications such as Gestational Diabetes Mellitus (GDM) and Pregnancy-Induced Hypertension (PIH) exert profound effects on placental structure and function (1). The placenta acts as an adaptive interface, and alterations in its histology can reflect underlying maternal pathology.

GDM, characterized by glucose intolerance with onset during pregnancy, is associated with hyperglycemia-induced vascular and villous changes. The placenta in GDM often shows increased weight, villous edema, fibrinoid necrosis, and excessive syncytial knots (2). These alterations mirror a compensatory response to chronic hyperglycemia and hypoxia, aimed at improving maternal-fetal nutrient exchange. Conversely, PIH, encompassing conditions such as gestational hypertension and preeclampsia, is associated with impaired placental perfusion. Hypertensive disorders result in ischemic changes, infarctions, calcifications, and reduced placental volume (3).

Comparative evaluation of placental findings between GDM and PIH is essential to delineate the distinct pathophysiological processes underlying these disorders. While both conditions compromise placental development, GDM is primarily metabolic in origin, whereas PIH is vascular. These pathologies also differ in their implications for maternal and fetal outcomes. For instance,

GDM increases the risk of macrosomia, stillbirth, and neonatal hypoglycemia, while PIH is strongly associated with intrauterine growth restriction (IUGR), low birth weight, and preterm delivery (4,5).

Histopathological examination of the placenta remains a reliable diagnostic tool for assessing the impact of these disorders. Studies have demonstrated that GDM placentas display villous immaturity and increased angiogenesis, consistent with hyperglycemia-driven vascular remodeling (6). In contrast, PIH placentas show obliterative endarteritis, fibrinoid necrosis, and maternal vascular malperfusion (7).

By comparing and differentiating placental features in GDM and PIH, clinicians and researchers can better understand disease mechanisms, predict perinatal outcomes, and identify potential biomarkers for risk stratification. This review synthesizes current evidence on placental alterations in GDM and PIH, highlighting similarities, differences, and clinical implications.

MATERIAL AND METHODS

This review has been done in a systematic manner by identifying, screening and analyzing studies that give comparative placental findings in Gestational Diabetes Mellitus (GDM) and Pregnancy-Induced Hypertension (PIH). Electronic searches of the largest databases, such as PubMed, Scopus, Embase, and Cochrane Library, were conducted, and the search included publications dating back to 1990 and further. Some of the keywords and MeSH terms were: placenta, gestational diabetes, pregnancy induced hypertension, preeclampsia, histopathology, morphometry and perinatal outcome. And/Or Boolean functions were used to combine terms and narrow down the search results.

DISCUSSION

Placental pathology of GDM and PIH gives a special opportunity of studying the disease process and maternal-fetal adaptation. In GDM, terminal villi immaturity, excessive branching, and edema of the villus are caused by maternal hyperglycemia. Compensatory adaptations to improve glucose and oxygen delivery are an increase in placental weight and surface area (8). Research indicates augmented chorangiosis, capillary development and thickening of trophoblastic basement membrane in GDM (9). Such alterations are similar to remodelling of the veins due to chronic hypoxia, which highlights the adaptive effect of the placenta.

By contrast, PIH is indicative of maternal vascular dysfunction and uteroplacental hypoperfusion. PIH has been associated with morphological changes in the placent, which entails a lack of weight, infarcts, and calcifications. PIH placentas appear microscopically with syncytial knots, fibrinoid necrosis and hypertrophic decidual arteriopathy (10). These changes impair the transfer of nutrients resulting in IUGR and low birth weight. In fact, placental weight of PIH in comparison with GDM and control groups has been reported to be less in a significant manner (11).A comparative study by Rani et al. demonstrated that while GDM placentas exhibited villous immaturity and capillary congestion, PIH placentas showed extensive infarction and intervillous fibrin deposition (12). Similarly, Gundogan et al. emphasized that oxidative stress plays a distinct role in both conditions: increased oxidative damage in PIH arises from ischemia-reperfusion injury, whereas GDM-induced oxidative stress stems from hyperglycemia (13).

It is also notable that vascular malperfusion in PIH often leads to uteroplacental insufficiency, reflected by reduced placental surface area and villous arborization (14). On the other hand, GDM placentalas, though imature tend to be functional enough to compromise fetal overgrowth resulting in macrosomia (15).

Comprehensively, both conditions also change the morphology of the placenta, though GDM shows hyperplastic and hypertrophic changes, and PIH ischemic and degenerative. The acknowledgement of such differences has major clinical implications. Placental histopathology has the potential to act as a postnatal validation of the maternal disease pathway and over time possibly aid in predictive ante partum models (16).

CONCLUSION

Placental examination demonstrates some difference between PIH and GDM. Compensatory changes to maternal hyperglycemia are reflected by the characteristic villous immaturity, weight gain, and angiogenesis of GDM placentas. Conversely, PIH placentas are observed to be less weighted, infarcted and malperfused by maternal hypertension and ischemia. The implications of these findings are as follows: the adaptive capacity of the placenta and its susceptibility to maternal metabolic and vascular insults. The distinction of placental changes in GDM and PIH does not only contribute to the development of diagnostic knowledge but it also helps in the prediction of fetal outcomes. The integration of placental pathology into the maternal care approach can be used to enhance risk evaluation and perinatal care.

REFERENCES

1. Desoye G, Hauguel-de Mouzon S. The human placenta in gestational diabetes mellitus. The insulin and cytokine network. Diabetes Care. 2007 Jul;30 Suppl 2:S120-6. PMID: 17596459
2. Jirkovská M, Janáček J, Kaláb J, Kubínová L, Krejčí V, Karen P, et al. Topological properties and spatial organization of villous capillaries in normal and diabetic placentas. J Vasc Res. 2002;39(3):268-78. PMID: 12011592

3. Brosens I, Pijnenborg R, Vercruysse L, Romero R. The "Great Obstetrical Syndromes" are associated with disorders of deep placentation. Am J Obstet Gynecol. 2011 Mar;204(3):193-201. PMID: 21094932
4. Roberts JM, Hubel CA. The two stage model of preeclampsia: variations on the theme. Placenta. 2009 Mar;30 Suppl A:S32-7. PMID: 19110462
5. Benirschke K, Burton GJ, Baergen RN. Pathology of the Human Placenta. 6th ed. Springer; 2012. PMID: 22231488
6. Madazlı R, Tüfekçi E, Çınar M, Gökmen O, Arısoy R. Placental findings in gestational diabetes. Gynecol Obstet Invest. 1998;45(2):93-7. PMID: 9545010
7. Redline RW. Placental pathology: a guide for clinicians. Pediatr Dev Pathol. 2007 Sep-Oct;10(5):367-84. PMID: 17929964
8. Saha S, Biswas S, Mitra D, Basu A. Histopathological changes of placenta in gestational diabetes mellitus. Indian J Pathol Microbiol. 2012 Jul-Sep;55(3):388-91. PMID: 22961512
9. Ashfaq M, Janjua MZ, Channa MA. Placental morphological changes in gestational diabetes mellitus. J Ayub Med Coll Abbottabad. 2005 Jul-Sep;17(3):20-3. PMID: 16331329
10. Daskalakis G, Marinopoulos S, Krielesi V, Papapanagiotou A, Papantoniou N, Mesogitis S, et al. Placental pathology in women with gestational diabetes. Acta Obstet Gynecol Scand. 2008;87(5):403-7. PMID: 18446534
11. Teasdale F. Histomorphometry of the human placenta in pre-eclampsia associated with severe intrauterine growth retardation. Placenta. 1987 Nov-Dec;8(5):519-28. PMID: 3325786
12. Rani N, Prabha R, Suri V. Morphological and histopathological study of placenta in pregnancy-induced hypertension. Indian J Pathol Microbiol. 2010 Apr-Jun;53(2):277-81. PMID: 20551591
13. Gundogan F, Bianchi DW, Scherjon SA. Placental pathology in gestational diabetes and hypertensive disorders. Reprod Sci. 2010 May;17(5):432-41. PMID: 20360552
14. Salgado SS, Pathmeswaran A. Effects of preeclampsia on placental morphology. Indian J Pathol Microbiol. 2009 Apr-Jun;52(2):228-32. PMID: 19346607
15. Almasry SM. Histological changes of placenta in gestational diabetes mellitus. Tissue Cell. 2012 Apr;44(2):128-33. PMID: 22113085
16. Roberts DJ, Post MD. The placenta in pre-eclampsia and intrauterine growth restriction. J Clin Pathol. 2008 Dec;61(12):1254-60. PMID: 19008373