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Morphological and Histological Features of Placenta in Women with Gestational Diabetes Mellitus and Hypertension and its Association with Perinatal Outcome: A study Protocol

Farzana Jailani Manchankop[1], Dr. Ravindra Vedpathak[2], Dr. Nishigandha Sadamate[3], Dr. Rakesh Kumar Jha[4], Dr. Shyambabu Prasad Rauniyar[5]

[1]PhD Scholar, Department of Anatomy, Jawaharlal Nehru Medical College, Datta Meghe institute of Higher Education and Research, Sawangi (M), Wardha, Email: farzana.farajm@gmail.com
[2]Associate professor, Department of Anatomy, Jawaharlal Nehru Medical College, Datta Meghe institute of Higher Education and Research, Sawangi (M), Wardha, Email: vedbhagy@gmail.com
[3]Associate professor, Department of Anatomy, Jawaharlal Nehru Medical College, Datta Meghe institute of Higher Education and Research, Sawangi (M), Wardha, Email: sadamatenishi@gmail.com
[4]Assistant professor, Department of Biochemistry, Jawaharlal Nehru Medical College, Datta Meghe institute of Higher Education and Research, Sawangi (M), Wardha, Email: jhaarakesh1993@gmail.com
[5]Assistant professor, Department of Anatomy, Datta Meghe Medical College, Wanadongri, Datta Meghe institute of Higher Education and Research, Sawangi (M), Wardha, Email: shyambaburauniyar@gmail.com

ABSTRACT

The placenta is a key fetomaternal organ, and its structural changes mirror maternal disease and influence neonatal outcomes. Gestational diabetes mellitus (GDM) and hypertensive disorders of pregnancy (HDP) commonly alter placental morphology and histology, leading to adverse perinatal outcomes. This prospective observational study will analyze 90 placentae (30 GDM, 30 HDP, 30 controls) for gross parameters (weight, size, infarcts, calcifications) and histological features (villous immaturity, syncytial knots, fibrinoid necrosis, chorangiosis, villous hypoplasia). GDM is expected to correlate with macrosomia and NICU admissions, while HDP is linked to growth restriction, preterm birth, and low Apgar scores.

KEYWORDS: Placenta, GDM, HDP, Morphology, Histology, Perinatal outcome

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INTRODUCTION

The placenta is a transient but vital fetomaternal organ that mediates nutrient, gaseous, and waste exchange, while also reflecting maternal health and pathological alterations during pregnancy. Its morphology and histology undergo dynamic changes in response to maternal metabolic and vascular conditions, directly influencing perinatal outcomes. 1

Gestational diabetes mellitus (GDM) and hypertensive disorders of pregnancy (HDP), including gestational hypertension and preeclampsia, are common complications associated with significant maternal and perinatal morbidity and mortality. GDM, characterized by maternal glucose intolerance, leads to hyperglycemia and hyperinsulinemia in the fetus, whereas HDP is marked by vascular dysfunction and placental hypoperfusion.2

Morphologically, GDM placentas are often enlarged and thickened with villous immaturity and intervillous congestion, while hypertensive placentas tend to be smaller, irregular, and show infarctions and calcifications.3 Histologically, GDM is associated with villous immaturity, increased syncytial knots, fibrinoid necrosis, and thickened basement membranes, whereas HDP shows villous hypoplasia, accelerated maturation, and spiral artery atherosis. 4

These pathological changes contribute to adverse outcomes—fetal macrosomia and birth trauma in GDM, and intrauterine growth restriction, preterm birth, and perinatal asphyxia in HDP. Hence, systematic evaluation of placental morphology and histology is essential for understanding disease mechanisms, predicting neonatal outcomes, and guiding perinatal care.

AIM AND OBJECTIVES

Aim

To evaluate the gross Morphological and Histological Features of Placenta in Women with Gestational Diabetes Mellitus and Hypertension and its Association with Perinatal Outcome

Objectives

1. To study gross placental changes (weight, size, thickness, infarcts, calcifications, cord insertion) in GDM and HDP versus normal pregnancies.

2. To analyze histological alterations (villous immaturity, syncytial knots, fibrinoid necrosis, chorangiosis, infarction, distal villous hypoplasia) in GDM and HDP.
3. To compare and differentiate placental findings between GDM and HDP.

REVIEW OF LITERATURE

The placenta is the key interface between maternal and fetal circulation; structural and vascular abnormalities reflect maternal metabolic and vascular disease, mediating adverse perinatal outcomes. Both GDM and HDP cause distinct gross and histopathological changes that correlate with fetal growth, preterm birth, neonatal complications, and perinatal mortality. 2,5

Placental changes in GDM:

Grossly, placentas are often heavier and thicker, sometimes with larger lobulation and surface area. Microscopically, findings include villous immaturity, villous edema, chorangiosis, intervillous fibrin deposition, and variable syncytial knot changes. These alterations are linked with macrosomia, neonatal hypoglycemia, and higher NICU admissions, though severity varies with glycemic control and treatment.6

Placental changes in HDP/preeclampsia:

Placentas are usually smaller with reduced surface area, infarcts, and retroplacental hematoma. Histological hallmarks include maternal vascular malperfusion (MVM): decidual arteriopathy, infarcts, fibrinoid necrosis, syncytial knots, and villous hypoplasia. These correlate with FGR, preterm birth, low Apgar scores, and increased neonatal morbidity.7

Mechanistic links:

In GDM, hyperglycemia and altered insulin signaling affect villous maturation, vascularization, and nutrient transfer, predisposing to macrosomia and metabolic dysregulation. 8 In HDP, abnormal spiral artery remodeling leads to placental hypoxia and oxidative stress, producing MVM lesions and impaired fetal growth. 9

Evidence and gaps:

Studies consistently associate GDM placental changes with macrosomia and metabolic complications, and HDP lesions with FGR and preterm birth. 10 However, comparative studies evaluating GDM, HDP, combined pathology, and controls are limited. Variability in sampling, definitions, and lack of standardized scoring hinder comparability. Few studies stratify findings by treatment or disease control, leaving gaps in understanding disease–placenta–outcome relationships. 3,4,11

MATERIAL AND METHODS

Study Design: Prospective, comparative, observational study.

Place of study: The Study will be conducted in the Department of Anatomy.

Study population: Women delivering at Acharya Vinobha Bhave Rural Hospital during the study period. Placentae will be collected immediately after delivery for gross and histological evaluation, processed and reported per **Amsterdam/RCPath/CAP** guidance.

GROUPS

Group I (GDM): Placenta from women diagnosed with GDM using standard criteria (75-g OGTT per **WHO 2013/IADPSG**, or as per **ADA Standards**) .

•**Group II (Hypertension in Pregnancy):** Placenta from women with pregnancy-induced hypertension, pre-eclampsia, or chronic hypertension per **ACOG/ISSHP** definitions.

•**Group III (Controls):** Placenta from normotensive, non-diabetic women with uncomplicated term pregnancies.

Study period: 3 years.

Sample size: Total 90 placenta (30 GDM, 30 hypertensive, 30 controls) for comparative analysis.

Placental handling & histology: Placentae will undergo immediate grossing (weight, size, thickness, infarcts, calcification, cord insertion) and standardized histologic sampling (cord, membranes, full-thickness blocks) per Amsterdam 2016 and RCPath/CAP guidelines, with reporting based on standardized lesion terminology (e.g., MVM). 12

METHODOLOGY

Inclusion criteria

1. Pregnant women aged 18–45 years.

2. Singleton pregnancy.
3. Gestational age ≥ 28 weeks at delivery.
4. Women diagnosed with Gestational Diabetes Mellitus (GDM) as per standard guidelines.

Exclusion criteria

1. Pre-gestational diabetes mellitus.
2. Chronic hypertension predating pregnancy.
3. Major fetal congenital anomaly.
4. Intrauterine fetal demise before labor.

EXPECTED OUTCOME

Placentas in GDM are anticipated to show increased weight, thickness, villous immaturity, chorangiosis, and fibrin deposition, correlating with macrosomia, neonatal hypoglycemia, and higher NICU admissions. In HDP/preeclampsia, placentas are likely smaller with infarcts, syncytial knots, and vascular malperfusion, linked to growth restriction, low APGAR, preterm birth, and NICU admissions. Combined GDM with hypertension may demonstrate mixed pathology with the poorest perinatal outcomes. 7

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