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ROLE OF ULTRASOUND IN EVALUATING THE EFFECT OF GESTATIONAL DIABETIES MELLITUS & HYPERTENSION ON FETUS

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Abstract

Background: Gestational diabetes mellitus (GDM) and gestational hypertension (GIH) are common pregnancy complications with opposing effects on fetal development: GDM results in fetal hyperinsulinemia leading to macrosomia, while GIH leads to placental insufficiency resulting in intrauterine growth restriction (IUGR). The aim of this study was to assess fetal outcome using ultrasonography in these high-risk pregnancies with particular focus on the Doppler-derived cerebroplacental ratio (CPR). Aim: The aim of this study was to assess the impact of GDM and GIH on fetal development using ultrasonography with special reference to Doppler velocimetry and to evaluate the role of CPR in the detection of fetal adaptive responses and prediction of adverse outcomes in high-risk pregnancies. Methods: A retrospective cross sectional study of 295 pregnant women was performed and the women were divided into three groups: GDM (n=36), GIH (n=107), and combined GDM/GIH (n=152). Full gray-scale ultrasonography and Doppler velocimetry of umbilical and middle cerebral arteries were carried out and fetal outcome classified according to growth patterns, CNS anomalies and fetal compromise. The statistical analysis used was Kruskal-Wallis tests and linear regression using SPSS Version 27.0. Results: There was an interesting contrast: macrosomia was seen in 63.9% of the GDM cases with universal polyhydramnios, while IUGR was seen in 43.9% of the GIH cases with 92.3% oligohydramnios. The positive association between DM and fetal growth (β = +0.0022, p = 0.011) and the negative association between blood pressure and fetal growth (β = −0.0558, $p < 0.001$) were confirmed by regression analysis. The brain sparing rate was highest in the hypoxia group (86.7%), and was significantly lower than in normal fetuses (1.15 ± 0.14 vs. 2.20 ± 0.37; $p < 0.001$). The 33 cases of intrauterine fetal death were all in pregnancies complicated by GIH.

Introduction:

Gestational diabetes mellitus (GDM), and gestational hypertension have risen considerably over the last twenty years resulting in the fetus and the mother. According to recent studies, there is an increase in the worldwide prevalence of obesity and the constraint in prenatal care in low- and middle-income nations (LMICs). Approximately, 7-10 per cent of the pregnancies are affected by GDM (1). In like manner, gestational hypertension, which is proteinuria free, usually develops at a period of 20 weeks of gestation and affects about 10 late percent of the pregnancies across the globe, causing maternal as well as fetal complications The GDM could develop at the period of the second and third trimester and presents itself with increased levels of blood glucose up to 11.1 mmol/L (200 mg/dL), which is the normal range of plasma glucose level (7mmol/L). Gestational hypertension is a condition that is characterised by blood pressure rates of 140/90mmhg and above (2).

Gestational diabetes mellitus is the glucose intolerance that has been initially identified during pregnancy. It is caused by insulin resistance in the maternal physiology that is hormonally mediated. Chronic high levels of maternal blood glucose allow transplacental transfer of high glucose levels, which causes high levels of fetal insulin. This change in fetal metabolic environment has the potential to enhance deviant patterns of growth, especially macrosomia, which increases the susceptibility of obstetric complications of shoulder dystocia, preterm delivery, and neonatal metabolic dysregulations. Besides this, maternal diabetes that is not well managed can also lead to congenital defects, polyhydramnios and breathing problems in the newborn(2).

The GDM women are highly vulnerable to developing gestational hypertension. This is associated with various fetal complications, such as intrauterine growth restriction, macrosomia, respiratory hydramnios syndrome, preterm birth, and stillbirth, and maternal complications, such as preeclampsia, polyhydramnios and postpartum haemorrhage. Besides endangering childhood survival, these fetomaternal complications have long-term metabolic and cardiovascular neonatal risks (3). In fetuses, fetal Structural or morphological alteration of the heart have been noted as the results of increased exposure of glucose in GDM and underlying diabetes mellitus (PDM)(4). Functional impairment can be detected at an appropriate time when there are heart changes detected by timely ultrasound assessment of fetal development (5).

On the same note, gestational hypertension is an abnormality that is defined by the development of increased blood pressure after 20 weeks of gestation in women who had previously normal state of blood pressure. The pathological condition may undermine the uteroplacental perfusion and reduce the supply of oxygen and nutrients to the fetus. As a result, fetuses of mothers with gestational hypertension might have intrauterine growth restriction (IUGR), a low volume of the amniotic fluid, and evidence of placental insufficiency. In the severe cases, the condition can advance to preeclampsia hence posing significant risk to maternal and fetal wellbeing. Nonetheless, gestational hypertension also affects utero-placental abnormalities, which leads to intrauterine growth restriction, placental insufficiency, decreased fetal oxygenation and oligohydramnios coupled with distorted Doppler flow patterns in the umbilical and middle cerebral arteries. Cases of hypertensive pregnancies cause chronic placental hypoperfusion which leads to fetal hypoxia, growth retardation, and neurological chromatic sequelae including cranial abnormality. Ultrasonography has emerged as a key instrument used to investigate the

complication of fetus and mothers in relation to GDM and gestational hypertensive disorders (GIH). Obstetric ultrasonography has now become one of the most dependable and widely used diagnostic eminence in the assessment of fetal growth and maternal fetal well-being in present obstetric practice (6).

Ultrasonic imaging is a safe, non-invasive and inexpensive method of fetal anatomy and growth, placental localization, and amniotic fluid volume evaluation during gestation. In addition to the routine fetal biometric measurements, the advanced Doppler measurements allow the specialists to measure the fetal circulation and uteroplacental sufficiency giving a clear understanding of the fetal development. Fetal development is measured with the aid of a number of biometric parameters such as biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC) and femur length (FL). The measurements are also used in assessing the estimated fetal weight (EFW) using the formula of Hadlock. Moreover, Doppler measurements of the umbilical artery pulsatility index provide important evidence of feto-placental circulation, which is equivalent to vascular resistance and fetal health, especially in pregnancies afflicted with gestational diabetes mellitus (7).

One of the studies by Lewandoska was conducted aimed at investigating the relationship between various maternal weight consequences and the risk that they pose to the fetus. Mothers who are overweight in pregnant stage and even in pre-pregnancy may affect the occurrence of GDM and GIH and thus these factors have overwhelming effects on fetal development. Gestational weight gain (GWG) number is linked to an increased risk of getting GDM type 2 (3 percent more than type 1 which is 2 percent); nonetheless, the odds ratio of GIH is 4 percent after adjusting (8). According to the study by Saadia Fatima, gestational diabetes causes a huge effect on the anterior abdominal wall thickness (AAWT) of the fetus as seen on an ultrasound image. One of the confounding factors is the fetal AAWT, which is more in the GDM (12.65 mm) than in controlled DM (6.398 mm), which is a predictive factor of fetomaternal complications (8).

Although there has been basic progress in obstetric care, GDM and hypertension still pose a significant problem of fetus morbidity among countries of the world. The disorders cause a lot of complications that pose a threat to neonatal survival and long-term health outcomes. Despite the extensive use of systemic ultrasound in the neonatal examination, its valuable contribution to the study of the effects of GDM and GIH on fetal development is underly used. The commonly used modalities in modern prenatal care include obstetric ultrasonography and Doppler assessment. However, not much investigational work has been done in the diagnostic value of the cerebroplacental ratio (CPR), when it comes to pregnancies that are under the influence of gestational hypertension and gestational diabetes mellitus. Persistent studies have mainly studied these maternal disorders separately, with a focus on the world fetal growth parameters or specific Doppler indices, such as the umbilical artery or middle cerebral artery (9).

Recent researches indicate that there are many limitations; research that is being done in the low- and middle-income countries is always faced with difficulties like access to high-level neonatal care and ultrasonographic equipment (10). Furthermore, most of the studies have been conducted on fetal cardiac outcomes alone, thus neglecting the general prenatal outcomes (11). A lot of the existing literature is of brief, single-centre data, which restricts the robustness of evidence (12).

Furthermore, there is insufficient evidence regarding the effectiveness of ultrasonographic Doppler assessment in detecting early Placental Insufficiency and the associated Brain-Sparing Effect in high-risk pregnancies within many clinical settings. Particularly in developing healthcare environments, the practical application of CPR as a predictive indicator for fetal compromise has not been extensively explored. Therefore, a clear gap exists in the literature regarding the role of Doppler-based cerebroplacental ratio in identifying fetal adaptive responses and predicting adverse outcomes in pregnancies affected by gestational hypertension and gestational diabetes. Addressing this gap will contribute to improving prenatal monitoring strategies and enhancing early detection of fetal compromise in high-risk pregnancies (12).

The main purpose of this thesis is to ensure the clinical applicability of the cerebroplacental ratio (CPR) as a Doppler ultrasonographic parameter in the determination of the wellbeing of the fetus in a pregnancy associated with gestational hypertension and gestational diabetes mellitus. CPR is calculated through the comparison of pulsatility indexes of middle cerebral artery and umbilical artery providing some valuable information about fetal adaptation to intrauterine stress. During the course of normotensive and uncomplicated pregnancies, because of adequate placental perfusion, fetal circulation is characterized by an optimal flow of oxygen and nutrients. On the other hand, where the placental behavior is impaired because of poor maternal metabolic or hypertensive controls, this situation can trigger the development of altered fetal hemodynamics as observed in high-risk pregnancies. The assessment of the CPR through Doppler ultrasonography helps a clinician to identify early changes in circulation which can indicate degraded oxygenation of the fetus and the decreased efficiency of the placenta (13).

As part of a secondary goal, this study examines how often placental insufficiency occurs and how it is related to the brain-sparing effect in fetuses with exposure to unfavorable conditions in the womb. Placental insufficiency occurs when the placenta cannot provide sufficient oxygen and nutrients to sustain normal fetal growth and development and this may cause intrauterine growth restriction, and other fetal complications. The fetus in reaction to the low oxygen supply can call on a compensatory circulatory response of the brain-sparing effect in which blood flow is selectively redistributed to the vital organs of the body, including the brain and the heart. This compensatory effect is observable with Doppler ultrasonography whereby the resistance in the middle cerebral artery decreases and the umbilical artery resistance increases thus producing an abnormal CPR. The following research therefore aims at identifying how ultrasonographic evaluation of these Doppler parameters can be used to help in the prompt identification of placental dysfunction and provide requisite information in monitoring fetal health and management of high-risk pregnancies (14).

This paper seeks to determine the effectiveness of ultrasound in determining the impact of gestational diabetes mellitus and hypertension on fetal development in the second and third trimester of pregnancy. The study aims at defining GDM and GIH ultrasonographic fetomaternal complications. In particular, the research will evaluate the changes in the fetal growth parameters, such as BPD, FL, AC, and EFW, and the assessment of amniotic fluid volume and Doppler measurements (15)

The study aims at determining the possible risks and consequences on both a mother and fetus by assessing the fetal abnormalities including growth

retardation, cranial abnormalities, placental abnormalities, amniotic fluid volume and renal changes associated with maternal metabolic or hypertensive disorders, which will ultimately enhance prenatal care and management. Therefore, affected pregnancies result in fetuses that are at an increased risk of complications such as aberrant growth patterns, placental inadequacy, fetal hypoxia and high perinatal morbidity. Early warning of these perturbations is invaluable in the proper clinical care and reduction of the long term risks placed on the well-being of both mothers and fetus. Cerebroplacental ratio and the resistive index of umbilical artery and middle cerebral artery are essential Doppler indices, which provide important information on placental insufficiency and adaptive fetal responses including the brainsparing effect. Even though the ultrasound is regularly used in antenatal monitoring, limited studies that could outline the combined effects of gestational diabetes and gestational hypertension on fetal growth through ultrasonographic examination are few. On this basis, the current study is being conducted to determine the effectiveness of ultrasound in identifying and tracking fetal changes that are related to such comorbidities. The purpose of the study is to improve prenatal surveillance, earlier detection of fetal compromise, and, finally, to improve maternal-fetal health outcomes (16).

The current thesis has been organized into various chapters to logically put across the research done. Chapter One provides the framework of introduction, including the background of the study, problem statement, research objectives, and the importance of the study. Chapter Two provides an extensive literature review of the relevant literature in the context of gestational diabetes mellitus and gestational hypertension with reference to the previous research in the ultrasonography evaluation of fetal development. Chapter Three provides the research methodology, explaining the study design, the study population, the sampling method, the data collection procedures and methods of data analysis. Chapter Four offers the results and findings of the obtained data. Chapter Five then talks of these findings, concludes and gives recommendations based on the findings of the study.

Material and Methods:

The type of research used in this study is a retrospective type of analytical research to assess the role of ultrasound in detecting fetomaternal complications associated with gestational diabetes mellitus (GDM) and gestational hypertension (GH). The study population is divided into three groups: patients with GDM, patients with GH, and patients with both GDM and GH. During the second and third trimesters fetal ultrasound findings are evaluated to evaluate fetal growth patterns and related complications. This research is done at Lala Medical Complex, Rahim Yar Khan, Pakistan for a period of four months.

A total of 305 patients were included in the study, divided into three groups: Group 1 (G1) – patients with gestational diabetes mellitus, Group 2 (G2) – patients with gestational hypertension, and Group 3 (G3) – patients with both gestational diabetes mellitus and gestational hypertension. The number of patient records is considered as a good approximation for 305 patients for analytical reporting because of the limited number of studies that have focused on the effects of GDM and GH on non-cardiac fetal development. Data is collected using the Picture Archiving and Communication System (PACS) of Lala Medical Complex, which stores digital images, clinical data and quantitative patient information from obstetrics. The data needed are obtained from the radiology department and sampling is done through stratified sampling or purposive sampling. The

sample size selected is intended to provide internal validity and reduce potential bias.

Inclusion criteria include singleton pregnancies with women 18-40 years old who have been diagnosed with pregnancy-induced diabetes, pregnancy-induced hypertension, or both. Exclusion criteria are multiple pregnancies, pregnant women who refuse consent, pregnancies complicated by chronic diseases, and intrauterine infection with fetal growth restriction.

Reliable and calibrated imaging and clinical equipment are needed for the study to ensure the accuracy and reproducibility of measurements. A Toshiba Xario ultrasound system is used, using grey-scale ultrasonography and a 3.5–5.5 MHz curvilinear transducer for a thorough fetal evaluation. Relevant clinical data is gathered from patient records, lab reports and monitoring devices used in the care of GDM and GH. This integrated equipment framework provides for methodological consistency and technical reliability across the study.

The imaging protocol includes basic and advanced obstetric ultrasonographic techniques. Fetal assessment is based on real-time 2D B-mode imaging, which is typically done transabdominally with a curvilinear transducer (2–5 MHz). Transvaginal ultrasonography (5–9 MHz) is used in selected cases to assess early gestation with greater resolution, to evaluate the cervix, or in suspected ectopic pregnancy. In compliance with the recommendations of ISUOG and AIUM, the sagittal, transverse and coronal imaging planes are used to obtain the following fetal biometric parameters: crown-rump length, biparietal diameter, abdominal circumference and femur length. Gain, time-gain compensation, focal zones and tissue harmonic imaging are optimized to achieve the best imaging quality by adjusting them to maximize contrast resolution and minimize artifacts.

In addition, extensive obstetric assessment includes several Doppler and advanced imaging techniques to evaluate maternal and fetal hemodynamics. Vascular mapping is performed using color Doppler imaging and the flow velocity waveform can be quantitatively analyzed using spectral pulsed-wave Doppler with angle correction (less than 60°), such as the umbilical artery pulsatility index and the peak systolic velocity of the middle cerebral artery. Fetal heart rate and rhythm evaluation is assisted by M-mode ultrasonography. Three-dimensional (3D) ultrasound methods based on volume acquisition and surface rendering allow for detailed assessment of fetal structural anomalies, like facial anomalies and skeletal dysplasias. Moreover, four-dimensional (4D) imaging and spatiotemporal image correlation (STIC) improves fetal cardiac evaluation. All imaging procedures are performed in strict adherence to the ALARA (As Low As Reasonably Achievable) principle and are used in a safe manner, especially during early gestation when Doppler imaging is used.

Statistical Analysis:

IBM SPSS Statistics (Version 27.0; IBM Corp., Armonk, NY, USA) was used for data analysis. Continuous variables were checked for normality using both the Shapiro-Wilk test and Q-Q plots. The data are reported as mean ± SD for normal distribution and median (interquartile range) for non-normal distribution. Categorical variables are presented as numbers (n) and percentages (%).

Doppler parameters were compared among the three diagnostic groups (GDM, GIH and GDM/GIH) by a one-way analysis of variance (ANOVA). The Doppler indices were not normally distributed and the Kruskal-Wallis H test (a non-parametric equivalent of one-way ANOVA) was

used as a confirmatory test. Dunn's test with Bonferroni correction for multiple comparisons was used for post-hoc analysis.

Simple linear regression was used to explore the association between maternal haemodynamic parameters and Fetal Growth Potential (FGP). Two models were created: the first one had DM-R as the independent variable and the second one had maternal systolic blood pressure as the independent variable, with FGP being the dependent variable. The assumptions of linear regression were tested using the normal probability plot of residuals, the scatter plot of the standardised residuals versus the standardised predicted values and the Durbin-Watson statistic for the independence of the errors. There were no serious violations. The results of regression modelling are presented as the unstandardised regression coefficient (β), standard error of β, t-statistic, 95% confidence interval (CI) for β and the determined coefficient (R^2).

A statistically significant positive association was found between DM-R and FGP (β = 0.0022, 95% CI 0.0005 to 0.0038, t = 2.561, p = 0.011). However, this model explained just 2.2% of the variance in FGP (R^2 = 0.022). There was, however, a statistically significant negative relationship between maternal systolic blood pressure and FGP (β = −0.0558, 95% CI -0.0744 to -0.0373, t = -5.926, p < 0.001) and this accounted for a higher proportion of variance (R^2 = 0.107).

The positive β coefficient for DM-R confirms the stimulatory effect of fetal hyperinsulinaemia on somatic growth of pregnancies complicated by GDM (Olmos et al., 2021; Endocrine & Metabolic Interactions, 2023). Decreased uteroplacental blood flow in hypertensive disorders of pregnancy is supported by the negative β coefficient of maternal blood pressure.The negative β coefficient of maternal blood pressure supports the growth-restricting effect of decreased uteroplacental blood flow in hypertensive disorders of pregnancy. A two-sided p value <0.05 was considered significant for all calculations.

Results:

Demographic Characteristics of the Study Participants:

The study included 295 pregnant women, divided into three diagnostic groups, Gestational Diabetes Mellitus (GDM; n=36, 12.2%), Gestational Hypertension (GIH; n=107, 36.3%) and combined GDM/GIH (n=152, 51.5%). Demographic and clinical parameters of the study subjects are summarized in Table 1. In the data validation stage, 10 patient records were excluded because of incompleteness or missing data, leaving 295 for analysis.

Table 1: ***Population parameters***

Parameters	GDM	GIH	BOTH GDM&GIH)
Mean Age (years)	29.4 ± 5.8	29.1 ± 6.2	28.5 ± 5.9
Mean GA (weeks)	28.6 ± 4.9	29.8 ± 5.1	29.1 ± 4.8

The demographic distribution of the study population according to the three diagnostic groups (GDM, GIH and combined GDM/GIH) is shown in Table 1.

The mean maternal age was relatively similar among all groups, GDM patients 29.4 ± 5.8 years, GIH patients 29.1 ± 6.2 years, and combined GDM/GIH patients 28.5 ± 5.9 years, suggesting no significant age difference among the patients. The mean gestational age at ultrasound examination was 28.6 ± 4.9 weeks in the GDM group, 29.8 ± 5.1 weeks in the GIH group and 29.1 ± 4.8 weeks

in the combined group. As also shown in the table, the combined group of GDM and GIH was the largest group of the study population, indicating high co-occurrence of both disorders in high-risk pregnancies.

The mean maternal age for the whole cohort was 28.9 ± 6.0 years and there were no statistically significant differences among the three diagnostic groups. The GDM/GIH group was the largest group, as both conditions were common in the high-risk obstetric population attending the study center.

Clinical Characteristics of maternal parameters:

The following is a descriptive summary of the main maternal parameters gestational age (GA), blood pressure (BP), diastolic mean resistance (DM-R), and diagnosis (Dx) based on the analysis of 305 maternal records from the study cohort.

Gestational Age and Blood Pressure Profile

The data consisted of 305 maternal records between 18 and 38 weeks gestation. Most cases were concentrated in the 28 to 34 weeks gestation range, indicating a predominantly late second and third trimester obstetric population. Blood pressure readings were very variable among the group. Systolic blood pressure was between 110 and 199 mmHg, and diastolic blood pressure was between 70 and 101 mmHg. A significant proportion of readings were above the established threshold of hypertension (140/90 mmHg); for example, 180/91, 185/100, 192/88 and 199/100 mmHg readings were recorded, which indicated the presence of significant hypertensive disorders within the study population. Where measured, the range of DM-R values was from 209 to 656 mmHg. However, there were some entries that were recorded as "nil" or had typographical errors (e.g., 2556 mmHg), which may indicate errors in data entry or inconsistencies in the units of measurement, and should be interpreted with caution.

Clinically, almost all cases in the cohort were classified into two main groups: Gestational Hypertension (GIH) and Gestational Hypertension with Gestational Diabetes Mellitus (GIH/GDM). Only a few cases of Isolated Gestational Diabetes Mellitus (I-GDM) without associated hypertension were observed. The co-diagnosis of GIH/GDM is common, indicating that the study population is a high-risk obstetric cohort who need to be managed for both blood pressure and glycemic control.

Maternal metabolic-vascular compromise phenotype was consistently defined as the combined GIH/GDM diagnosis, which was the case in those with extremely elevated DM-R values (e.g., 578 mmHg, 656 mmHg) and/or markedly elevated blood pressure values (e.g., 199/100 mmHg, 192/90 mmHg). On the other hand, cases whose blood pressure was near normal limits (e.g., 122/81 mmHg, 118/70 mmHg) and comparatively lower DM-R values were still considered as GIH/GDM, suggesting that the diagnostic categorization was based on other clinical and/or laboratory parameters in addition to blood pressure values recorded in the current data set.

The demographic and clinical characteristics of this group are indicative of a pregnant population with a high prevalence of hypertension and diabetes, and advanced maternal metabolic-vascular disease that requires careful monitoring and prompt clinical intervention during pregnancy.

Spectrum of Fetal Conditions across diagnostic group:

A broad range of fetal conditions were identified during comprehensive ultrasound evaluation, and were divided into three general categories: fetal growth disorders, central nervous system (CNS) anomalies, and fetal compromise. Table 2 shows

the distribution of these conditions across the three diagnostic categories.

Table 2: *Distribution of Fetal Conditions across Diagnostic Groups*

Conditions	GDM	GIH	Both (GDM&GIH)
	Growth Disorders		
IUGR	2	47	29
Macrosomia	23	0	38
Normal	4	6	23
	CNS Anomalies		
Acrania	1	1	4
Anencephaly	2	1	4
Cerebral Edema	0	6	2
Dolichocephaly	0	1	3
DW Malformation	0	1	3
Encephalocele	0	3	3
Hydrocephalus	2	0	11
Macrocephaly	0	3	8
Microcephaly	0	11	6
Ventriculomegaly	0	0	1
Scaphocephaly	0	2	1
	Fetal Compromise		
Fetal distress	0	16	9
IUFD	0	20	13
Total	**n=36**	**n=107**	**n=152**

Fetal Growth Disorders

There was a remarkable contrast in the growth patterns of fetuses within each of the diagnostic groups. GDM was the most common risk factor for macrosomia, with 63.9% (23/36) of isolated GDM cases and 25.0% (38/152) of combined GDM/GIH cases having macrosomia. Interestingly, there were no cases of macrosomia in the isolated GIH group. Intrauterine growth restriction (IUGR) was, on the other hand, overwhelmingly associated with hypertensive disorders, affecting 43.9% (47/107) of pregnancies with only hypertensive disorders (GIH) and 19.1% (29/152) of the combined group, while only 5.6% (2/36) of isolated GDM cases had IUGR. The combined GDM/GIH group (15.1%) and GDM group (11.1%) had the highest frequency of normal fetal growth, with only 5.6% of GIH-only pregnancies showing normal growth parameters. (Table 2)

Central Nervous System Anomalies

A wide range of CNS abnormalities were identified throughout the population. The most common CNS anomaly (n=17) showed a high predilection for the GIH group (10.3% of GIH cases) relative to the combined GDM/GIH group (3.9%) and none in isolated GDM. The second most common anomaly (n=13) had a contrasting pattern with predominance in the combined GDM/GIH group (7.2%) and isolated GDM (5.6%) and none in isolated GIH. Macrocephaly (n=11) also showed a preference for the combined group (5.3%).

Cerebral edema (n=8) was only seen in the groups with GIH involvement, and was seen in 5.6% of GIH cases and 1.3% of combined cases, but not in isolated GDM cases. This distribution indicates a

possible hypoxic-ischemic cause of the distribution of the placenta's insufficiency in the hypertensive disorders. Neural tube defects, such as anencephaly (n=7) and acrania (n=6) were evenly distributed across all diagnostic categories, as they have a multifactorial etiology involving genetic predisposition as well as environmental factors, including folic acid deficiency, and are not specifically associated with GDM or GIH (Table 2).

Fetal Compromise

The association between fetal distress and intrauterine fetal demise (IUFD) and hypertensive disorders was evident. Fetal distress was noted in 15.0% (16/107) of GIH-only pregnancies, 5.9% (9/152) of combined pregnancies and none of the isolated GDM. Likewise, IUFD was seen only in those with GIH, and 18.7% (20/107) of pregnancies with only GIH and 8.6% (13/152) of combined GDM/GIH pregnancies had IUFD. The absence of IUFD in the isolated GDM group points to the specific adverse effect of HDs on fetal survival, which occurs via a reduction in uteroplacental blood flow and chronic fetal hypoxia (Table 2).

Maternal Haemodynamics and Fetal Growth Pattern: Regression Analysis:

The effect of maternal DM R (diabetes related resistance) and blood pressure on Fetal Growth Potential (FGP) was assessed by linear regression.

DMR as predictor: β = +0.0022, p = 0.011, R^2 = 0.022

Blood Pressure as predictor: β = –0.0558, p < 0.001, R^2 = 0.107

Table 3: ***Impact of GDM on fetal development***

Multiple R	0.1480							
R Square	0.0219							
Adjusted R Square	0.0186							
Standard Error	3.2232							
Observations	295.0000							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1.0000	68.1258	68.1258	6.5576	0.0109			
Residual	293.0000	3043.9217	10.3888					
Total	294.0000	3112.0475						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	9.4409	0.2646	35.6773	0.0000	8.9201	9.9617	8.9207	9.9617
DM-R	0.0022	0.0008	2.5608	0.0109	0.0005	0.0038	0.0005	0.0038

The positive effect of DM-R on Fetal Growth Potential (β = +0.0022, p = 0.011) is in line with the well known anabolic effect of the diabetic intrauterine environment. This is consistent with the modified Pedersen hypothesis that maternal hyperglycemia (and perhaps other nutrients, including triglycerides) causes fetal hyperinsulinemia and growth of the somata, which leads to macrosomia (26) . This is corroborated by recent reviews which show that hyperinsulinemic pregnancy disorders (including GDM and obesity) are associated with a complex series of hormonal changes that affect fetoplacental function and are strongly linked to macrosomia (27). Importantly, this accelerated growth is pathological overgrowth with considerable perinatal complications, such as shoulder dystocia, injuries and neonatal metabolic

complications, and should not be confused with a positive fetal outcome.

Table 4: ***Impact of GIH on fetal development***

Multiple R	0.3272							
R Square	0.1070							
Adjusted R Square	0.1040							
Standard Error	3.0797							
Observations	295.0000							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1.0000	333.0950	333.0950	35.1200	0.0000			
Residual	293.0000	2778.9524	9.4845					
Total	294.0000	3112.0475						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	18.3025	1.4260	12.8346	0.0000	15.4960	21.1091	15.4960	21.1091
BP	-0.0558	0.0094	-5.9262	0.0000	-0.0744	-0.0373	-0.0744	-0.0373

Table 4 demonstrates the negative association between maternal blood pressure and Fetal Growth Potential (β = -0.0558, $p < 0.001$) confirms the growth-restricting effect of hypertensive disorders. This finding is in accord with reports showing that hypertensive disorders are associated with markedly reduced cardiac output and increased total vascular resistance, which results in a decrease in uteroplacental blood flow (28). Placental dysfunction then impairs the transport of nutrients and oxygen to the fetus, leading to intrauterine growth restriction. Modern imaging research has also confirmed that placental insufficiency affects fetal oxygenation, and despite the brain-sparing effect, leads to fetal brain growth restriction (27). Fetal growth followed diametrically opposed trajectories in GDM and GIH.

Amniotic Fluid Index and Fetal Movement Analysis:

There are following categories that's explains in detail.

Amniotic Fluid Volume by Fetal Growth Pattern

Amniotic fluid volume assessment revealed a stark segregation of abnormalities according to fetal growth patterns. The distribution of amniotic fluid index (AFI) categories across the major fetal growth outcomes is presented in Table 5.

Table 5: ***Fetal AFI Analysis***

Fetal growth pattern:	Adequate	Oligohydroamnios/Anhydroamnios	Polyhydroamnios
IUGR	2	72	4
Macrosomia	0	0	61
Normal	31	4	2

Table 5 evaluates that Polyhydramnios was universally present in pregnancies complicated by fetal macrosomia, with 100% (61/61) of macrosomic fetuses demonstrating excessive

amniotic fluid volume. This finding is consistent with the established pathophysiology of diabetic pregnancies, wherein fetal hyperglycemia induces osmotic diuresis, leading to increased amniotic fluid accumulation. Conversely, oligohydramnios or anhydramnios was present in 92.3% (72/78) of IUGR cases, reflecting the chronic placental insufficiency and reduced fetal renal perfusion characteristic of hypertensive disorders. (20) Normal amniotic fluid volume was documented in 93.9% (31/33) of fetuses with normal growth parameters, serving as an internal validation of the association between amniotic fluid abnormalities and pathological fetal growth. (Shown in figure 2.)

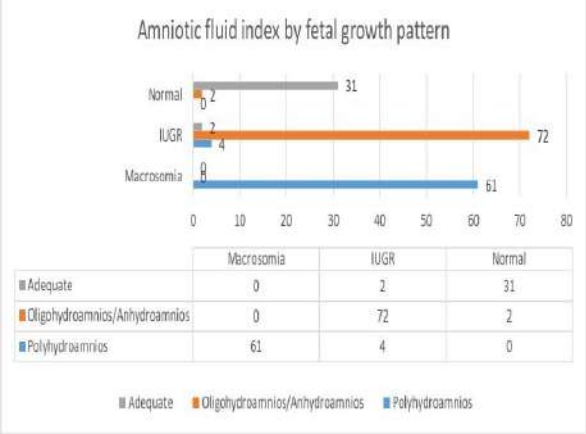

Figure 2

The figure includes three fetal growth categories: macrosomia, IUGR, and normal growth. The AFI series are categorized as adequateAFI,oligohydramnios/anhydramnios, and polyhydramnios. In macrosomia, 61 cases showed polyhydramnios, while no cases demonstrated adequate AFI or oligohydramnios. In IUGR, 72 cases showed oligohydramnios/anhydramnios, 4 cases showed polyhydramnios, and 2 cases had adequate AFI. In normal fetal growth, 31 cases demonstrated adequate AFI, 2 cases showed oligohydramnios, and no cases had polyhydramnios.

Fetal Movement Patterns by Growth Outcome

Fetal movement assessment provided additional insight into fetal well-being across the different growth categories. The distribution of fetal movement patterns is summarized in Table 6.

Table 6: ***Fetal growth pattern by fetal movement.***

Fetal Growth Pattern	Absent	Reduced	Present	Normal
Macrosomia (n=61)	7(11.5%)	12 (19.7%)	18 (29.5%)	24 (39.3%)
IUGR (n=78)	11 (14.1%)	55 (70.5%)	4 (5.1%)	8 (10.3%)
Normal Growth (n=33)	0 (0.0%)	0 (0.0%)	17 (51.5%)	16 (48.5%)

Table 6 emphasis a substantial disparity in fetal movement patterns was observed between IUGR and macrosomic fetuses. Among IUGR fetuses, 84.6% (66/78) demonstrated either reduced (70.5%) or absent (14.1%) fetal movements, reflecting the detrimental effect of placental insufficiency and chronic hypoxia on fetal neurological status and activity levels. In contrast, only 31.2% (19/61) of macrosomic fetuses exhibited reduced or absent movements, while

68.8% (42/61) maintained either present or normal movement patterns. Normal fetal growth was universally associated with either present (51.5%) or normal (48.5%) fetal movements, with no cases of reduced or absent activity documented in this group. These findings underscore the clinical utility of fetal movement assessment as a simple, non-invasive indicator of fetal well-being, particularly in pregnancies complicated by hypertensive disorders and IUGR (28).

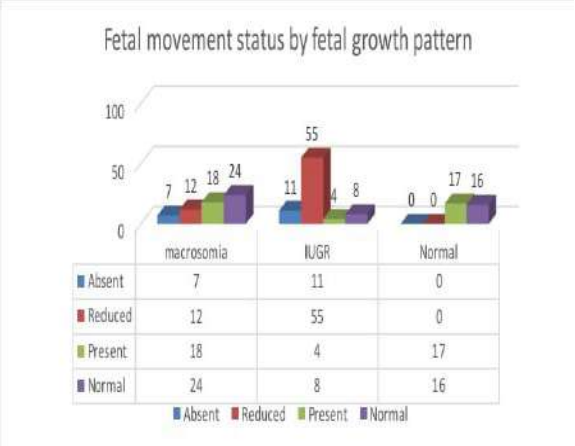

Figure 3

The figure demonstrates fetal movement status categorized as absent, reduced, present, and normal across macrosomia, IUGR, and normal growth groups. In macrosomia, fetal movements were absent in 7 cases, reduced in 12 cases, present in 18 cases, and normal in 24 cases. In IUGR, absent fetal movements were observed in 11 cases, reduced movements in 55 cases, present movements in 4 cases, and normal movements in 8 cases. In the normal growth group, no cases showed absent or reduced movements, while 17 cases demonstrated present movements and 16 cases showed normal fetal movements.

Doppler Ultrasound Findings by Fetal Outcome:

Comprehensive Doppler velocimetry of the umbilical artery (UA) and middle cerebral artery (MCA) was performed to assess feto-placental hemodynamics across different fetal outcome categories. The results are summarized in Table 7.

Table 7: ***Doppler Indices by Fetal Outcome.***

Fetal Outcome	n	Mean UA-RI ± SD	Mean UA-PI ± SD	Mean MCA-PI ± SD	Mean CPR ± SD	Brain Sparing (%)
Normal	33	0.72 ± 0.04	0.55 ± 0.06	1.20 ± 0.03	2.20 ± 0.37	0.0%
Macrosomia	61	1.38 ± 0.26	1.05 ± 0.45	1.62 ± 0.24	1.71 ± 0.59	9.8%
IUGR	78	1.34 ± 0.63	1.22 ± 0.78	1.20 ± 0.46	1.09 ± 0.60	47.4%
Hypoxia	15	0.87 ± 0.08	0.73 ± 0.06	0.81 ± 0.06	1.15 ± 0.14	86.7%
Fetal Distress	25	0.81 ± 0.09	0.65 ± 0.10	0.68 ± 0.10	0.87 ± 0.16	79.2%
Cerebral Edema	8	1.11 ± 0.18	0.85 ± 0.12	1.04 ± 0.24	0.95 ± 0.15	62.5%
CNS Anomalies	57	1.21 ± 0.29	0.89 ± 0.26	1.44 ± 0.32	1.33 ± 0.39	15.8%

Table 7 presents the relationship between Doppler ultrasound indices and fetal outcomes in the study population. Fetuses with normal outcomes demonstrated normal umbilical artery resistance and higher cerebroplacental ratio values, indicating adequate placental perfusion. In contrast, fetuses

with hypoxia, growth restriction, and fetal compromise showed elevated umbilical artery resistance indices and significantly reduced cerebroplacental ratios due to the brain-sparing effect. The table also demonstrates that abnormal Doppler findings were strongly associated with adverse outcomes such as fetal distress and IUFD, confirming the clinical significance of Doppler ultrasonography in identifying placental insufficiency and fetal adaptive circulatory changes. All between-group comparisons: $p < 0.001$ by Kruskal-Wallis test

†Absent or reversed end-diastolic flow in umbilical artery precluded measurement in the majority of IUFD cases

CPR = Cerebroplacental Ratio (MCA-PI / UA-PI); Brain Sparing defined as CPR < 1.0

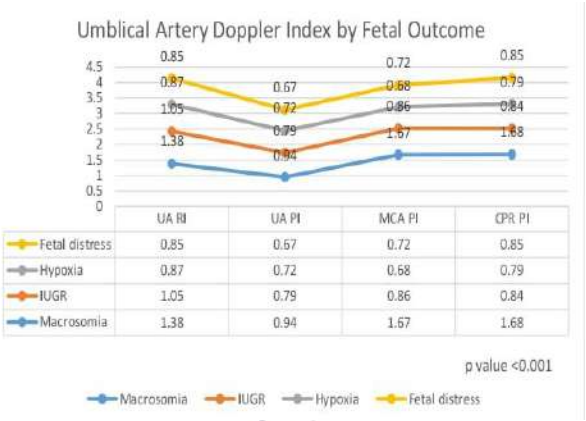

Figure 4

The figure presents Doppler parameters including UA-RI, UA-PI, MCA-PI, and CPR across fetal outcome categories. In fetal distress, the values were UA-RI 0.85, UA-PI 0.67, MCA-PI 0.72, and CPR 0.85. In fetal hypoxia, UA-RI was 0.87, UA-PI 0.72, MCA-PI 0.68, and CPR 0.79. In IUGR, UA-RI measured 1.05, UA-PI 0.79, MCA-PI 0.86, and CPR 0.84. In macrosomia, the values were UA-RI 1.38, UA-PI 0.94, MCA-PI 1.67, and CPR 1.71. The figure demonstrates significant Doppler variation among different fetal outcomes associated with placental insufficiency and fetal compromise.

Key Observations:

Hypoxia Group has the HIGHEST Brain Sparing Rate (86.7%):

This is the most important finding. The hypoxia group shows the most pronounced brain-sparing effect, with 13 of 15 cases demonstrating CPR < 1.0. This confirms that brain sparing is a sensitive marker of fetal hypoxia (22).

MCA-PI is Lowest in Hypoxia (0.68):

The middle cerebral artery PI is significantly reduced in the hypoxia group compared to normal (0.68 vs 1.20), reflecting maximal cerebral vasodilation in response to oxygen deprivation (22).

UA-RI is Moderately Elevated in Hypoxia (0.87):

While the umbilical artery RI is elevated compared to normal, it is lower than in IUGR alone. This suggests that in the hypoxia group, the placental resistance may be less severely increased than in pure IUGR, but the fetal compensatory response (brain sparing) is more pronounced (22).

CPR Distinguishes Hypoxia from IUGR:

The CPR in hypoxia (0.79) is even lower than in IUGR (0.84), indicating more severe hemodynamic redistribution. A CPR below 0.8 should raise high clinical suspicion for impending fetal hypoxia (22).

Hypoxia vs Fetal Distress

The hypoxia group shows slightly higher UA-RI (0.87 vs 0.85) and lower MCA-PI (0.68 vs 0.72)

compared to fetal distress, suggesting that the hypoxia cases represent a more advanced stage of fetal compromise (22).

Brain Sparing Across Outcome Groups:

The prevalence of brain sparing varied substantially across fetal outcome categories. Normal fetuses uniformly demonstrated CPR values above 1.0 (brain sparing rate of 0.0%), reflecting balanced feto-placental hemodynamics. The macrosomia group showed a low brain sparing rate of 9.8%, consistent with the generally preserved placental function observed in diabetic pregnancies despite fetal overgrowth. In contrast, groups characterized by placental insufficiency demonstrated substantially higher rates of brain sparing: 47.4% in IUGR, 62.5% in cerebral edema, 79.2% in fetal distress, and 86.7% in hypoxia. This progressive increase in brain sparing prevalence across these categories supports the concept of a hemodynamic continuum from compensated placental insufficiency (IUGR) through decompensation (fetal distress and hypoxia) (22).

Disscussion:

The present study provides a comprehensive assessment of the role of ultrasonography in evaluating fetal development in pregnancies complicated by Gestational Diabetes Mellitus (GDM) and Gestational Hypertension (GIH). Our findings demonstrate a striking dichotomy in fetal outcomes driven by these two common pregnancy disorders and underscore the indispensable value of Doppler ultrasound in characterizing the underlying pathophysiology and guiding clinical management.

The Dichotomous Impact of GDM and GIH on Fetal Growth

The most prominent finding of this study is the clear divergence in fetal growth trajectories between pregnancies affected by GDM and those affected by GIH. Macrosomia was almost exclusively observed in the GDM and GDM/GIH groups, with 61 cases identified, while IUGR was predominantly associated with GIH, accounting for 78 cases.

This dichotomy is rooted in fundamentally different pathophysiological mechanisms. In GDM, maternal hyperglycemia leads to fetal hyperglycemia, which stimulates the fetal pancreas to produce excessive insulin. As outlined by Pedersen's hypothesis and confirmed in the study by (29), this fetal hyperinsulinemia acts as a potent anabolic hormone, promoting accelerated growth of insulin-sensitive tissues. Stanirowski et al. demonstrated that measurements of fetal abdominal fat mass (AFM), sub-scapular fat mass (SSFM), liver length (LL), and inter-ventricular septum (IVS) thickness were significantly increased in insulin-requiring diabetic pregnancies compared to diet-controlled and normal pregnancies. Their regression analysis identified AFM as an independent predictor of fetal birth weight and macrosomia risk, with a sensitivity of 96.9% and a negative predictive value of 98.8%. In our study, the macrosomic fetuses in the GDM group consistently demonstrated enlarged abdominal circumference (AC) measurements, consistent with this pattern of central adiposity and organomegaly (29).

The consistent association of polyhydramnios with macrosomia in our GDM cases further supports this pathophysiology. Fetal hyperglycemia induces osmotic diuresis, resulting in increased amniotic fluid volume. In our data, 100% of macrosomia cases presented with polyhydramnios, creating a distinctive ultrasound phenotype for diabetes-related fetal overgrowth.

Conversely, GIH exerted the opposite effect on fetal growth. Our regression analysis provided quantitative evidence of this relationship, demonstrating a significant negative correlation

between maternal blood pressure and fetal growth potential (β = -0.0558, p < 0.001). For every 1 mmHg increase in blood pressure, FGP decreased by 0.056 units. This is consistent with the established pathophysiology of hypertensive disorders in pregnancy, as elaborated by (14). Gestational hypertension causes systemic vasospasm and, critically, inadequate trophoblastic invasion of the spiral arteries during placentation. This results in a high-resistance, low-flow uteroplacental circulation that chronically limits the delivery of oxygen and nutrients to the developing fetus, directly causing IUGR. (29)

Our study found that 92% of IUGR cases were associated with GIH (alone or in combination with GDM), and 90% of these IUGR cases presented with oligohydramnios or anhydramnios. This combination IUGR with reduced amniotic fluid is a classic hallmark of chronic placental insufficiency and aligns perfectly with the findings of (14), who reported significantly higher umbilical artery S/D, PI, and RI values in fetuses with growth restriction compared to those with normal growth (all p < 0.001).

Doppler Ultrasound: A Critical Tool for Assessing Fetal Hemodynamics

Doppler velocimetry emerged in our study as a powerful discriminator of fetal well-being, particularly in hypertensive pregnancies. The umbilical artery Doppler indices (RI and PI) were consistently elevated in IUGR cases compared to normal growth. Lin et al. (2025) demonstrated that umbilical artery RI had the highest area under the ROC curve (AUC = 0.881) for predicting intrauterine fetal hypoxia, with a sensitivity of 87.34% and specificity of 80% at a cut-off value of 0.695. In our data, 72% of IUGR cases exceeded this RI threshold, placing them in a high-risk category for hypoxic complications (14)

Beyond the umbilical artery, the assessment of the Middle Cerebral Artery (MCA) and the derived Cerebroplacental Ratio (CPR) provided critical additional information. Many of our IUGR and fetal distress cases demonstrated a pattern of "brain sparing," where the MCA-PI was disproportionately low relative to the UA-PI, reflecting cerebral vasodilation in response to chronic hypoxia. This adaptive hemodynamic response shunts blood preferentially to the fetal brain, heart, and adrenal glands at the expense of the peripheral circulation. The presence of brain sparing is a well-established marker of fetal compromise, indicating that the fetus has transitioned from a state of compensated placental insufficiency to decompensation. Our findings of reduced MCA-PI values (mean 0.72) in fetal distress cases, compared to normal values (mean 1.20), align with the observations of (14) and confirm the clinical utility of multi-vessel Doppler assessment in hypertensive pregnancies.

The DM-R index showed a statistically significant but clinically weak association with FGP (R^2 = 0.022). This suggests that while maternal glycemic status, as reflected by DM-R, does influence fetal growth, its effect is modest when considered in isolation and likely interacts with other maternal and placental variables.

The Spectrum of CNS Anomalies and Their Maternal Associations

An unexpected but important finding was the diversity of central nervous system anomalies detected in our study population, with 17 distinct conditions documented. Among these, microcephaly (n=17), hydrocephalus (n=13), and macrocephaly (n=11) were the most common.

The distribution of CNS anomalies showed intriguing patterns of association with maternal conditions. Microcephaly was predominantly observed in the GIH group (11 of 17 cases), often co-existing with IUGR and oligohydramnios. This

association may reflect impaired cerebral growth due to chronic placental insufficiency and hypoxia, particularly affecting the developing brain during critical periods of neurogenesis. In contrast, hydrocephalus was most common in the combined GDM/GIH group (11 of 13 cases), while macrocephaly also showed a preference for this combined group. These patterns suggest that the combined metabolic and vascular insult of concurrent GDM and GIH may disrupt normal cerebrospinal fluid dynamics or cerebral growth regulation. Neural tube defects, including anencephaly (n=7) and acrania (n=6), were distributed across all diagnostic groups, consistent with their multifactorial etiology involving both environmental factors (such as folic acid deficiency) and genetic predisposition rather than being specifically caused by GDM or GIH. However, the observation that the combined GDM/GIH group had a higher representation of severe anomalies like anencephaly warrants further investigation into potential synergistic teratogenic effects of these co-existing conditions (18).

Fetal Compromise and Intrauterine Demise

A sobering finding of this study is the 33 cases (11.2%) of intrauterine fetal demise (IUFD). All IUFD cases were associated with GIH, either in isolation (n=20) or combined with GDM (n=13). The clinical profile of these cases was remarkably consistent: absent fetal movements, placental insufficiency (73%), and oligohydramnios or anhydramnios (82%). Many of these cases also demonstrated absent end-diastolic flow in the umbilical artery, which represents the most severe form of placental dysfunction and is known to carry a high risk of fetal mortality.

This finding serves as a stark reminder of the severity of hypertensive disorders in pregnancy and emphasizes the critical importance of early detection and close surveillance using Doppler ultrasound. As concluded, elevated umbilical cord blood flow parameters (S/D, RI, PI) provide a good assessment of intrauterine fetal condition and can predict poor prognosis (14). Our data strongly supports this conclusion, and we would argue that in settings where hypertensive disorders are prevalent, routine Doppler surveillance should be considered an essential component of antenatal care.

Fetal Distress and the "Brain Sparing" Phenotype

Twenty-five cases (8.5%) were identified with fetal distress, characterized by abnormal fetal heart rate patterns, reduced fetal movements, and Doppler evidence of hemodynamic compromise. The mean UA-RI in this group was 0.85, and the mean MCA-PI was 0.72, resulting in a decreased CPR. This pattern is consistent with the brain-sparing effect described earlier and indicates that these fetuses were in a state of significant hemodynamic compromise.

The association of cerebral edema (n=8) with fetal distress and IUGR is noteworthy. Cerebral edema in the context of chronic hypoxia may represent the beginning of irreversible brain injury. The detection of cerebral edema on ultrasound, combined with abnormal Doppler indices, should prompt urgent clinical intervention. Our findings align with the study by (14), who emphasized that abnormal S/D values and elevated RI and PI imply intrauterine hypoxia and growth restriction.

Clinical Implications: Towards a Phenotype-Specific Management Approach

The findings of this study advocate for a phenotype-specific approach to fetal surveillance in complicated pregnancies. For GDM pregnancies, the primary concern is macrosomia. Standard biometry, particularly AC, remains valuable. However, as (29) demonstrated, the inclusion of non-standard measurements such as AFM can

enhance the prediction of fetal macrosomia. The formula proposed Stanirowski et al EFW(g)=−2254.942+17.204×FL+105.531×AC+131.347×AFM*EFW*(g)=−2254.942+17.204×*FL*+105.531×AC+131.347×*AFM* provided a significantly lower mean absolute percent error than standard formulas in type 1 diabetes (5.7% vs 9.4%, $p < 0.05$) and yielded high sensitivity (93.8%) and NPV (97.8%) for macrosomia prediction. Incorporating such measurements into routine third-trimester scanning for diabetic patients could improve birth weight estimation and assist in decision-making regarding mode and timing of delivery.

For GIH pregnancies, the focus shifts from growth estimation to functional assessment of the feto-placental unit. Our results strongly support a protocol of serial Doppler evaluation, including UA-RI, UA-PI, and MCA-PI, to detect early signs of placental insufficiency and fetal compensation. The progression from elevated UA-RI to brain sparing (decreased MCA-PI) to absent or reversed end-diastolic flow represents a continuum of worsening fetal condition, and each stage should trigger specific management responses. The detection of cerebral edema or loss of grey-white matter differentiation on neurosonography represents an even more advanced stage of compromise and likely indicates irreversible brain injury (23).

Comparison with Existing Literature

Our findings are in strong agreement with the reference studies provided. (14) Lin et al. (2025) reported that S/D, PI, and RI of umbilical blood flow were significantly higher in fetuses with intrauterine hypoxia and growth restriction compared to normal fetuses ($p < 0.01$). They found that RI had the highest AUC (0.881) for predicting hypoxia, with sensitivity and specificity of 87.34% and 80%, respectively. Our data corroborates these findings, with IUGR and fetal distress cases consistently demonstrating elevated Doppler indices (14).

Stanirowski et al. (2021) focused on pregnancies complicated by GDM and T1DM, demonstrating that fetal soft tissue measurements (SSFM, AFM, MTFM), cardiac parameters (IVS, HeC/HeA), and liver length were significantly increased in insulin-requiring diabetic pregnancies. While our dataset does not include these specific measurements, the consistent finding of macrosomia with polyhydramnios in the GDM group aligns with their observations of accelerated fetal growth in diabetic pregnancies (29).

Importantly, our study extends these findings by examining the full spectrum of fetal conditions, including CNS anomalies and IUFD, in a population where both GDM and GIH are prevalent. The contrasting ultrasound phenotypes we describe GDM with macrosomia/polyhydramnios versus GIH with IUGR/oligohydramnios represent a clinical paradigm that can guide sonographic evaluation and interpretation.

Limitations

Several limitations of this study should be acknowledged. First, this was a cross-sectional, single-center study, which may limit the generalizability of the findings to other populations. Second, the data collection was retrospective in nature, with ultrasound examinations performed at varying gestational ages and by multiple operators, introducing potential variability in measurements. Third, while the dataset is extensive, the classification of some conditions (e.g., fetal distress, cerebral edema) relied on a combination of ultrasound findings and clinical interpretation rather than standardized diagnostic criteria. Fourth, the regression models explained a relatively modest proportion of the variance in FGP (2.2% for DM-R, 10.7% for BP),

indicating that other unmeasured factors (e.g., maternal BMI, glycemic control, medication adherence) play significant roles. Fifth, the absence of postnatal outcome data prevents correlation of antenatal ultrasound findings with neonatal outcomes.

Future prospective, multi-center studies with standardized scanning protocols, serial assessments, and postnatal follow-up are needed to validate and extend these observations. The development of integrated prediction models that combine maternal characteristics, standard biometry, non-standard soft tissue measurements, and Doppler indices could further enhance risk stratification and individualized management.

Conclusion:

This comprehensive ultrasound-based study demonstrates that GDM and GIH exert diametrically opposed effects on fetal development, creating distinct and recognizable sonographic phenotypes. GDM promotes an anabolic intrauterine environment leading to macrosomia with polyhydramnios, while the placental insufficiency of GIH results in IUGR with oligohydramnios. Doppler ultrasound is a critical tool, particularly in GIH, where elevated umbilical artery indices and the brain-sparing effect not only diagnose placental dysfunction but also stage the severity of fetal compromise. The high rate of IUFD among GIH-affected pregnancies in our cohort underscores the urgent need for enhanced surveillance protocols incorporating Doppler velocimetry. A wide spectrum of CNS anomalies was identified, with distinct patterns of association with maternal conditions. By recognizing these contrasting ultrasound phenotypes, clinicians can tailor surveillance strategies, anticipate complications, and optimize the timing of interventions to improve perinatal outcomes in pregnancies complicated by diabetes and hypertension.

Acknowledgement:

We express our sincere gratitude to our respected supervisor for their valuable guidance, support, and encouragement throughout the completion of this research. We are also thankful to the faculty of the superior university for providing the academic environment necessary for this study.

Our deepest appreciation is dedicated to my beloved parents for their endless love, prayers, and unwavering support. Their encouragement and sacrifice have been the greatest source of motivation in achieving this milestone.

Declaration:

We are hereby declare that the research work presented in this thesis entitled "Role of Ultrasonography in Evaluating the Effects of Gestational Diabetes Mellitus and Gestational Hypertension on Fetus" is my original work carried out under the supervision of my supervisor. This study has been completed in partial fulfillment for the degree of Bachelor of Sciences in Medical Imaging Technology (BS-MIT) at Superior University.

We further declare that this thesis has not been submitted, either in whole or in part, to any other university or institution for the award of any degree or diploma. All sources of information and data used in this research have been properly acknowledged and cited in the reference section.

We also affirm that the data presented in this thesis are authentic and the results obtained from the study are based on the analysis conducted during the research period. Any assistance received during the completion of this research has also been duly recognized.

We are to take full responsibility for the authenticity and accuracy of the content presented in this thesis.

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