Comparison of obstetric and perinatal outcomes in women with diabetes at Steve Biko Academic Hospital

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Background. Diabetes and obesity in pregnancy have been associated with increased rates of adverse maternal and neonatal outcomes compared with women with normoglycaemia and normal weight.
Objective. To investigate the effect of diabetes and pre-pregnancy obesity on obstetric and perinatal outcomes.
Methods. This study included women with pregestational diabetes types 1 (T1DM) and 2 (T2DM), gestational diabetes (GDM) and normoglycaemia, who received care at the Steve Biko Academic Hospital antenatal clinic between 2017 and 2022. The women were followed up until delivery. Data collected included obstetric history and care, diabetes, obstetric and perinatal outcomes.
Results. A total of 183 women were recruited: 13 (7.1%) with T1DM, 65 (35.5%) with T2DM, 39 (21.3%) with GDM and 66 (36.1%) normoglycaemic controls. Women with T2DM and GDM were older ($p<0.01$) and more likely to have a history of chronic hypertension ($p=0.025$) compared with controls. Women with GDM were more likely to be obese than their T1DM counterparts ($p=0.036$). T1DM and T2DM were associated with higher rates of preterm delivery than controls ($p=0.002$). The frequency of GDM was significantly higher in women with obesity ($p=0.039$). The frequency of caesarean section before the onset of labour was higher in women with a weight ≥80 kg compared with women with a weight <80 kg ($p=0.015$).
Conclusion. Diabetes in pregnancy is associated with adverse obstetric and perinatal outcomes. Therefore, adequate glucose control should be accompanied by preconceptual weight optimisation to reduce adverse outcomes during pregnancy.

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Diabetes mellitus is a common pregnancy complication that poses a serious health threat to maternal and child health.[1] Diabetes in pregnancy (DIP) can be classified as pregestational type 1 (T1DM) or 2 (T2DM) diabetes; T1DM or T2DM first diagnosed during pregnancy; or gestational diabetes mellitus (GDM), a milder form of carbohydrate intolerance that first develops during pregnancy, with glucose homeostasis usually restored within 6 weeks after delivery. Globally, ~16.7% (21.1 million) of live births are affected by DIP. Among these, pregestational T1DM and T2DM account for 10.6% of cases, T1DM and T2DM first detected in pregnancy account for 9.1% of cases and GDM accounts for 80.3% of cases.[1] South Africa (SA) is a low-to-middle-income country (LMIC) with high rates of DIP. Recent studies reported that the prevalence of GDM varied from 9.1% to 25.6%, depending on the diagnostic criteria.[2]

All types of DIP are associated with an increased risk of short- and long-term adverse outcomes for mother and child (Table 1), especially when glycaemic control is suboptimal. The severity and frequency of these adverse outcomes are higher in women with pregestational diabetes compared with GDM. Achieving adequate glycaemic control and appropriate gestational weight gain is critical to prevent pregnancy complications and adverse outcomes.[3]

Obesity is considered a major risk factor for DIP, with an increasing number of epidemiological studies supporting this association.[4] In addition, obesity has also been reported to independently increase the risk of maternal and fetal adverse outcomes.[5] In SA, the estimated prevalence of obesity in women of reproductive age is 35.2%,[6] highlighting the potential negative effects of obesity on both maternal and child health. Studies have shown that an increase in both maternal weight and body mass index (BMI) before and during pregnancy is associated with adverse pregnancy outcomes.[7–9] However, in resource-limited settings where measuring weight is more practical, the use of maternal weight instead of BMI to assess the risk of adverse outcomes related to weight during pregnancy might be a more viable option. This is substantiated by a study conducted by Wolfe *et al.*[10]

This study aimed to investigate the effect of DIP and obesity on obstetric and perinatal outcomes in women attending the diabetic antenatal clinic at a tertiary hospital in Tshwane, South Africa.

Table 1. Short- and long-term outcomes of DIP

Short-term	Long-term
Maternal	
Pre-eclampsia, preterm birth, CS, miscarriage,* obstructed labour, PPH	Worsening of diabetic retinopathy and nephropathy*, diabetes mellitus, cardiovascular diseases
Neonatal	
Congenital anomalies,* respiratory distress syndrome, jaundice, neonatal hypoglycaemia macrosomia, NICU admission	Adiposity/obesity, diabetes mellitus, cardiovascular risk, cognitive impairment

CS = caesarean section; PPH = postpartum haemorrhage; NICU = neonatal intensive care unit.
*specific to pregestational diabetes.

Methods

We conducted a prospective study including women with pregestational T1DM or T2DM, GDM and normoglycaemia (negative oral glucose tolerance test (OGTT)) who attended the high-risk antenatal clinic at Steve Biko Academic Hospital (SBAH), Tshwane, Pretoria, between May 2017 and March 2022. The study was approved by the University of Pretoria Health Science Research Ethics Committee (ref. no. 191/2016 and 41/2021). This study is part of a larger study investigating epigenetic mechanisms in women with DIP. At SBAH, the diabetes antenatal clinic manages referrals from local endocrine and internal medicine or antenatal clinics in the cluster. The referring clinics use the risk factor-based selective screening approach,[11] which includes screening for risk factors, such as family history of diabetes mellitus, previous GDM, advanced maternal age, obesity and previous adverse pregnancy outcome, including congenital abnormality, recurrent miscarriages, delivery of a stillborn child, delivery of a baby ≥4 000 g in a previous pregnancy or persistent glycosuria.[12]

Women were included in the study if they had singleton pregnancies, were aged between 18 - 40 years, were of black African ethnicity, were at ≤28 weeks' gestation and were HIV negative. DIP was categorised as TIDM if diagnosed prior to pregnancy or if first diagnosed in pregnancy and was confirmed by the presence of positive antibodies or the occurrence of diabetic ketoacidosis, which is determined in consultation with an endocrinologist. A T2DM diagnosis was made if it was identified prior to pregnancy or if overt diabetes was diagnosed during pregnancy (fasting plasma glucose level ≥7.0 mmol/L, random plasma glucose or 2-hour plasma glucose ≥11.1 mmol/L on the OGTT; or glycated haemoglobin (HbA1c) ≥6.5%). GDM was diagnosed if carbohydrate intolerance was first diagnosed during pregnancy according to the International Association of the Diabetes and Pregnancy Study Groups (IADPSG) criteria at 24 - 28 weeks' gestation (fasting plasma glucose level 5.1 - 6.9 mmol/L or 1-hour plasma glucose ≥10 mmol/L or 2-hour plasma glucose 8.5 - 11.0 mmol/L after a 2-hour 75-g oral glucose tolerance test (OGTT)).[13] Women were recruited as normoglycaemic controls if they had a negative OGTT. Women were followed up until delivery. Data collected included demographics, anthropometric measures, obstetric history and care, diabetes care and fetal outcomes, according to standard clinical care. Gestational age (GA) was determined using early ultrasound when available; otherwise, it was determined based on menstrual history or late ultrasound. Maternal weight at the first antenatal visit was recorded as pre-pregnancy weight was not available. Due to missing height measurements, maternal BMI data were limited. Consequently, both weight and BMI were collected. Obesity was defined as a BMI ≥30 kg/m^2 as per Institute of Medicine guidelines.[14] Dietary counselling and education on diabetes were provided by a trained dietician. Post diagnosis, some women with GDM were initiated on metformin in consultation with specialists, while others were started on a low-carbohydrate diet (dependent on OGTT levels). Women were counselled on maintaining glycaemic targets, including fasting/pre-prandial glucose levels of ≤5.3 mmol/L and 2-hour post-prandial glucose levels of ≤6.7 mmol/L. All women with DIP monitored their glucose levels at home. Women were required to test their glucose with an On-Call Plus glucometer (On Call, Mexico) at least five times a day, at various times during the week: 30 minutes before each meal (fasting), 2 hours after each meal (post-prandial), at bedtime and 02h00. Poor glycaemic control was defined as having >25% of glucose values outside the recommended range based on home glucose monitoring.

Obstetric and perinatal outcomes included GA at delivery (weeks), onset of labour, route of delivery, birthweight (g), neonatal outcome and Apgar score at 5 minutes. GA at delivery was categorised into preterm (≤37 weeks) and term delivery (>37 weeks). Fetal growth was classified as small for gestational age (SGA) if fetal growth <10th centile and large for gestational age (LGA) if fetal growth >90th centile. Birthweight was defined as low birthweight (501 - 2 500 g), normal weight (2 500 - 4 000 g) and macrosomia (>4 000 g).

Statistical analysis

Data were captured in Microsoft Office Excel 2010 (Microsoft Corp., US) and analysed using STATA 17 (Stata Corp., US). Baseline characteristics were summarised using descriptive statistics. A skewness-kurtosis test was performed to assess normality. Continuous variables are presented as the median and interquartile range (IQR), while categorical variables are expressed as counts and percentages. Continuous data were compared using the Kruskal-Wallis test, followed by Dunn's *post hoc* multiple comparisons test. Categorical data were compared using Pearson's chi-squared (χ^2) test with the Bonferroni *post hoc* test. For counts less than 5, Fisher's exact test was used. Statistical significance was defined as $p<0.05$.

Results

The general characteristics of the population according to diabetes type are summarised in Table 2. A total of 183 women were recruited, including 13 (7.1%) with T1DM, 65 (35.5%) with T2DM, 39 (21.3%) with GDM and 66 (36.1%) who were classified as normoglycaemic. Women with T2DM and GDM were older ($p<0.01$), had higher BMI ($p<0.05$) and had a history of chronic hypertension ($p=0.025$) compared with the control group. Obesity results were based on 74.31% of BMI data. Women with GDM had a significantly higher frequency of obesity compared with women with T1DM (80.6% v. 36.4%) but were not different to women with T2DM and controls. Women with T1DM and T2DM had significantly higher glycated haemoglobin (HbA1c) compared with those with GDM ($p<0.001$). At enrollment, more women with T1DM were on insulin treatment compared with those with T2DM (76.9% v. 15.0%; $p<0.01$), while more women with T2DM were on metformin compared with women with GDM (53.3% v. 26.5%; $p<0.05$). At delivery, 67.2% of women with T2DM and 18.2% of women with T1DM were managed with a combination of metformin and insulin compared with 15.2% of women with GDM ($p<0.001$; Table 2).

Obstetric and perinatal outcomes were compared in the combined diabetic group (T1DM, T2DM, GDM) compared with controls. Overall, the frequency of preterm delivery was higher in the diabetic group compared with the controls (51.5% v. 17.1%; $p<0.001$). However, no between-group differences were noted in the other obstetric and perinatal outcomes. Next, the diabetic group was stratified into T1DM, T2DM and GDM and controls. Obstetric and perinatal outcomes in the sub-groups were compared with the control group (Table 3). The frequency of preterm birth was higher in women with T1DM (66.7% v. 17.1%; $p<0.05$) and T2DM (54.4% v. 17.1%; $p<0.05$). However, there was no significant difference between the GDM and control groups. No between-group differences were observed in the other obstetric and perinatal outcomes.

Next, the effect of obesity and weight on obstetric and perinatal outcomes was investigated. The frequency of GDM was significantly higher in women with obesity compared with women without obesity (30.5% v. 11.5%; $p=0.036$) (Table 4). The frequency of caesarian section (CS) performed before the onset of labour was

Table 2. Participant characteristics according to glucose tolerance

Variable	Normoglycaemic controls (*n*=66)	T1DM (*n*=13)	T2DM (*n*=65)	GDM (*n*=39)	*p*-value
Age (years), median (IQR)	31 (27 – 36)*	29 (27 – 32)	35 (30 – 37)*	35 (32 – 38)*	<0.001
BMI (kg/m²), median (IQR)	32.3 (28.2 – 39.7)†,‡	24.5 (23.1 – 33.6)	31.6 (27.6 – 38.1)‡	37.8 (31.2 – 42.2)†	<0.001
Obesity (≥30 kg/m²), *n* (%)	23 (60.5)	4 (36.4)*	30 (55.6)	25 (80.6)*	0.036
Weight (kg), median (IQR)	82.2 (71.1 – 94.5)*,†	69.9 (60.4 – 85.0)‡,†	84.7 (72.4 – 98.9)‡	98.1 (81.9 – 112.0)*,‡†	<0.001
Weight (≥80 kg), *n* (%)	30 (49.2)	4 (30.8)	39 (60.9)	27 (69.2)	0.034
Weight (≥120 kg), *n* (%)	5 (8.2)	0 (0.0)	2 (3.1)	4 (10.3)	0.332
GA at recruitment (weeks), median (IQR)	22.0 (19.0 – 24.0)*	17.0 (15.0 – 21.0)*†	21.0 (16.0 – 25.0)†	26.0 (24 – 27.0)†	<0.001
HbA1c (%), median (IQR)	4.9 (5.2 – 5.5)†	9.7 (9.0 – 11.0)*†	7.6 (6.3 – 9.3)*†	5.7 (5.4 – 6.1)†	<0.001
Poor obstetric history§, *n* (%)	32 (61.5)	2 (16.7)	19 (31.1)	10 (27.0)	<0.001
History of hypertension in pregnancy, *n* (%)					
Yes	4 (6.1),*†	2 (15.4)	23 (35.9)*	12 (30.8)†	<0.001
Medication at enrollment, *n* (%)					
Insulin		10 (76.9)*	9 (15.0)*	0 (0.0)*	<0.001
Metformin		0*	32 (53.3)*	9 (26.5)*	
Diet		0*	7 (11.7)*	24 (28.7)*	
Metformin + insulin		3 (23.1)	12 (20.0)	1 (2.9)	
Medication at delivery, *n* (%)					
Insulin		9 (81.8)*	5 (8.6)*	0*	<0.001
Metformin		0*	5 (8.6)	13 (39.4)*	
Diet		0*	2 (3.4)*	15 (16.7)*	
Metformin + insulin		2 (18.2)*	39 (67.2)*	5 (15.2)*	

T1DM = type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; GDM = gestational diabetes mellitus; BMI = body mass index; IQR = interquartile range; GA = gestational age.

*, †, ‡Similar superscripts denote statistical significance among groups.

§Poor obstetric history was based on 78.79% of obstetric history data.

Table 3. Obstetric and perinatal outcomes in sub-groups of diabetes compared with normoglycaemic controls

	Normoglycaemic controls (*n*=66)	T1DM (*n*=13)	T2DM (*n*=65)	GDM (*n*=39)	*p*-value
Preterm delivery (≤37 weeks), *n* (%)	6 (17.1)*†	6 (66.7)*	31 (54.4)*†	13 (41.9)	0.002
Onset of labour, *n* (%)					
Induction of labour‡	7 (21.9)	2 (28.6)	13 (26.0)	8 (27.6)	0.356
CS	8 (25.0)	1 (14.3)	20 (40.0)	13 (44.8)	
Spontaneous onset of labour	17 (53.1)	4 (57.1)	17 (34.0)	8 (27.6)	
Route of delivery, *n* (%)					
Normal vaginal delivery	16 (47.1)	3 (30.0)	16 (28.1)	13 (40.6)	0.514
Elective CS	7 (20.6)	2 (20.0)	18 (31.6)	10 (31.3)	
Emergency CS	11 (32.4)	5 (50.0)	23 (40.4)	9 (28.1)	
Fetal growth,§ *n* (%)					
SGA (<10th centile)		0	4 (7.3)	2 (6.3)	0.339
AGA		6 (66.7)	32 (58.2)	25 (78.1)	
LGA (>90th centile)		3 (33.3)	19 (34.5)	5 (15.6)	
Birthweight (g), *n* (%)					
501 – 2 500	7 (20.0)	1 (10.0)	14 (24.1)	5 (15.6)	0.659
2 500 - 4 000	26 (74.3)	9 (90.0)	40 (69.0)	27 (84.4)	
>4 000	2 (5.7)	0	4 (6.9)	0	
Stillbirth, *n* (%)	0	1 (10.0)	4 (6.9)	1 (3.1)	0.227
Apgar score at 5 min, *n* (%)					
<7	2 (5.7)	1 (10.0)	5 (8.9)	4 (12.9)	0.743

T1DM = type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; GDM = gestational diabetes mellitus; CS = caesarean section; BMI = body mass index; SGA = small for gestational age; AGA= appropriate for gestational age; LGA = large for gestational age; min = minute.

*†Similar superscripts denote statistical significance among groups.

‡Induction of labour by either medical, mechanical or surgical means (or a combination thereof) was used to achieve labour. Failed inductions were considered if a patient was not in active labour within 24 hours.

§No fetal growth data for controls because of a lack of postnatal follow-up.

higher in women weighing ≥80 kg compared with women weighing <80 kg (45.6% v. 26.0%; $p<0.05$). The frequency of T1DM was lower in the ≥80 kg weight category compared with the <80 kg weight category (13.6% v. 3.6%; $p<0.05$), while the frequency of GDM was higher in the ≥80 kg compared with the <80 kg weight category (27.9% v. 12.1%; $p<0.05$) (Table 5). The frequency of spontaneous onset of labour was higher in the <80 kg weight category compared to the ≥80 kg and <120 kg weight category (52% v. 28.6; $p<0.05$). The rate of low Apgar scores at 5 minutes was significantly higher in the ≥120 kg group compared with the ≥80 and <120 kg groups (33.3% v. 4.2%; $p<0.05$) (Table 5).

Discussion

Literature has shown that diabetes and obesity in pregnancy are associated with adverse pregnancy outcomes for both the mother and child. Therefore, this study aimed to investigate the effect of diabetes and obesity in pregnancy on adverse obstetric and perinatal outcomes. The main findings of the study are *i*) higher rates of preterm delivery in women with T1DM and T2DM compared with the control group, *ii*) higher frequency of GDM in women with obesity compared with women without obesity, *iii*) higher risk of CS before the onset of labour in women who weighed more than 80 kg compared with women who weighed less than 80 kg and *iv*) lower rates of spontaneous onset of labour and higher rates of low Apgar scores in women who weighed more than 120 kg compared with women who weighed between 80 kg and 120 kg.

Our study showed that T1DM and T2DM were associated with higher rates of preterm delivery compared with the control group. These findings are consistent with those of previous studies that also reported elevated rates of preterm delivery in women with pregestational T1DM and T2DM compared with women with GDM and the control group.[15–17] In contrast, a systematic review reported deaths that showed higher or similar risks of preterm delivery in women with GDM compared with women with pregestational diabetes.[18] The optimal timing of delivery for women with DIP is contentious. Some recommendations suggest that in women with pregestational diabetes, especially those with vascular complications or suboptimal glycaemic control, early delivery (before 38.5 weeks' gestation) is the better option.[19] However, a 2018 Cochrane systematic review that aimed to determine the optimal timing of delivery for women with pregestational diabetes concluded that there were insufficient data to adequately determine the timing of delivery due to the lack of published trials.[20] Accordingly, the clinical decision regarding the timing of delivery in women with diabetes depends on several maternal and fetal factors, as well as the associated risk of adverse outcomes. Surprisingly, our study did not show differences in other obstetric and perinatal outcomes among the diabetes groups. This may be attributed to early delivery. Therefore, in our population, early delivery might be a better option to reduce adverse outcomes that may occur at term delivery.

The higher frequency of GDM in women with obesity, compared with their non-obese counterparts, is evidence that obesity is an independent risk factor for the development of GDM.[5] A meta-analysis including 20 studies reported that women who were overweight (2.1-fold), obese (3.6-fold) or severely obese (8.6-fold) had a significantly higher risk of developing diabetes compared with normal-weight pregnant women.[4] Furthermore, the Hyperglycaemia and Adverse Pregnancy Outcome (HAPO)

Table 4. Effect of obesity on obstetric and perinatal outcomes

	Non-obese (*n*=54)	Obese (*n*=82)	*p*-value
Preterm delivery (≤37 weeks), *n* (%)	21 (52.5)	24 (45.3)	0.491
Onset of labour, *n* (%)			
Induction of labour*	7 (19.4)	14 (31.1)	0.094
CS	11 (30.6)	19 (42.2)	
Spontaneous onset of labour	18 (50.0)	12 (26.7)	
Route of delivery, *n* (%)			
Normal vaginal delivery	14 (35.0)	18 (33.3)	
Elective CS	10 (25.0)	20 (37.0)	0.409
Emergency CS	16 (40.0)	16 (29.6)	
Normoglycaemic control, *n* (%)	15 (28.8)	23 (28.0)	0.036
T1DM, *n* (%)	7 (13.5)	4 (4.9)	
T2DM, *n* (%)	24 (46.2)	30 (36.6)	
GDM, *n* (%)	6 (11.5)†	25 (30.5)†	
Fetal growth, *n* (%)			
SGA (<10th centile)	3 (10.0)	3 (6.4)	0.759
AGA	19 (63.3)	29 (61.7)	
LGA (>90th centile)	8 (26.7)	15 (31.9)	
Birthweight (g), *n* (%)			
501 – 2 500	7 (17.1)	9 (16.4)	1.000
2 500 – 4 000	32 (78.0)	44 (80.0)	
>4 000	2 (4.9)	2 (3.6)	
Stillbirth, *n* (%)	3 (7.3)	3 (5.5)	1.000
Apgar score at 5 min, *n* (%)			
<7	4 (10.0)	5 (9.4)	1.000

CS = caesarean section; T1DM = type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; GDM = gestational diabetes mellitus; SGA = small for gestational age; AGA= appropriate for gestational age; LGA = large for gestational age; min = minute.
*Induction of labour by either medical, mechanical or surgical means (or a combination thereof) was used to achieve labour. Failed inductions were considered if a patient was not in active labour within 24 hours.
†Similar superscripts denote statistical significance.

study reported that a high maternal BMI is associated with an increased risk of adverse outcomes, independent of glycaemic status.[21] Factors such as advanced maternal age, high rates of diabetes and obesity have contributed to increasing rates of GDM.[3] In 2021, the International Diabetes Federation (IDF) estimated that GDM affected 80.5% of live births, while the prevalence of GDM is estimated to be 14.1% in Africa.[1] Studies have shown that physical activity and weight loss prior to conception significantly reduced the risk of developing GDM.[22,23] This emphasises the importance of preconception health for women of reproductive age who are either overweight or obese. Initiatives to encourage weight loss prior to pregnancy and to maintain appropriate gestational weight gain to reduce the risk of developing GDM and subsequent adverse outcomes are recommended.

The increased risk of CS before labour onset in women who weigh more than 80 kg compared with women who weigh less than 80 kg, and the reduced likelihood of spontaneous labour onset in women who weigh more than 120 kg, further demonstrates the negative impact of maternal weight on obstetric outcomes. Abdominal operative delivery in women with obesity is known to present significant problems such as anaesthetic difficulties, infections and greater blood loss, which pose a risk to both the mother and neonate.[24] Brost *et al.*[8] reported that even after controlling for confounders, any increase in maternal weight and BMI before and during pregnancy was associated with an increased risk of CS. They reported that for each incremental BMI unit (1 kg/m^2) increase, there was an approximate 7.8% rise in the likelihood of CS. This complication is thought to be due to an increase in pelvic soft tissue, resulting in the narrowing of the birth canal, leading to difficulty in delivery.[24] A study conducted in Norway reported that European/Central Asian women who were overweight or obese were at an increased risk of elective CS compared with Norwegian women without overweight and obesity, while sub-Saharan African women who were overweight or obese had the highest risk for emergency CS compared with normal-weight women from Norway.[7] A study by Wolfe *et al.*[10] reported that calculating maternal BMI offers no advantage over simply using maternal weight in the initial risk assessment of outcomes related to maternal weight. This practice should be considered for risk assessment of pregnant women instead of BMI, especially in busy, resource-limited settings.

Increased rates of low Apgar scores in women weighing more than 120 kg compared with women weighing between 80 and 120 kg are consistent with studies that showed negative effects of higher maternal weight and BMI on neonatal outcomes. There is evidence that the 5-minute Apgar score is a good predictor and indicator of infant survival and low Apgar scores at either 1, 5 or 10 minutes are associated with long-term neurological disabilities in infants.[25] A study conducted in Pakistan reported that increasing maternal BMI was strongly associated with low Apgar scores at birth and NICU admissions.[9] Another study conducted in Germany found that women with obesity had a higher percentage of giving birth to neonates with a low Apgar score at 1 minute; however, no differences in Apgar scores were observed at 5 and 10 minutes among different BMI groups.[26] Since evidence has shown that Apgar scores are crucial indicators of neonatal and subsequent infant outcomes, knowledge of risk factors, especially modifiable risk factors such as maternal weight, that are associated

Table 5. Effect of stratified weight on outcomes

	Weight <80 kg (*n*=70)	Weight ≥80 kg - <120 kg (*n*=101)	Weight ≥120 kg (*n*=11)	*p*-value
GA at delivery, *n* (%)				
≤37 weeks	27 (50.0)	27 (37.5)	3 (50.0)	0.353
>37 weeks	27 (50.0)	45 (62.5)	3 (50.0)	
Onset of labour, *n* (%)				
Induction of labour*	11 (22.0)	17 (27.0)	2 (40.0)	0.040
CS	13 (26.0)	28 (44.4)	3 (60.0)	
Spontaneous onset of labour	26 (52.0)†	18 (28.6)†	0 (0.0)	
Route of delivery, *n* (%)				
Normal vaginal delivery	21 (38.9)	25 (34.2)	1 (16.7)	0.398
Elective CS	11 (20.4)	24 (32.9)	3 (50.0)	
Emergency CS	22 (40.7)	24 (32.9)	2 (33.3)	
Fetal growth, *n* (%)				
SGA (<10th centile)	3 (9.4)	3 (5.3)	0	0.454
AGA	23 (71.9)	36 (63.2)	3 (50.0)	
LGA (>10th centile)	6 (18.8)	18 (31.6)	3 (50.0)	
Birthweight (g)				
501 – 2 500	13 (23.6)	14 (18.9)	0	
2 500 – 4 000	41 (74.5)	55 (74.3)	6 (100.0)	0.391
>4 000	1 (1.8)	5 (6.8)	0	
Neonatal outcome, *n* (%)				
Alive	52 (94.5)	71 (95.9)	6 (100.0)	0.803
Stillbirth	3 (5.5)	3 (4.4)	0 (0.0)	
Apgar score at 5 min, *n* (%)				
<7	7 (13.0)	3 (4.2)‡	2 (33.3)‡	0.025
≥7	47 (87.0)	69 (95.8)§	4 (66.7)§	

GA = gestational age; CS = caesarean section; SGA = small for gestational age; AGA= appropriate for gestational age; LGA = large for gestational age; min = minute; AGA = appropriate for gestational age, min = minute.
*Induction of labour by either medical, mechanical or surgical means (or a combination thereof) was used to achieve labour. Failed inductions were considered if a patient was not in active labour within 24 hours.
†, ‡, §Similar superscripts denote statistical significance among groups.

with a low Apgar score, is important in reducing associated neonatal adverse outcomes.

The relationship between obesity and diabetes and their effect on pregnancy outcomes has been established. Globally, non-communicable diseases such as diabetes and obesity are negatively associated with maternal and perinatal health. A study by Rosenberg *et al.*[27] suggested that diabetes and excess maternal weight can adversely affect maternal and delivery outcomes through two different pathways. The first pathway involves the contribution of diabetes and excess weight to the development of pre-eclampsia, which can trigger preterm delivery and CS. The second pathway pertains to the increased risk of macrosomia in neonates born to women with either diabetes, obesity or both. Babies with macrosomia often contribute to labour dystocia, which can result in an increased prompt for CS 4delivery.

The strength of our study lies in its ability to demonstrate the negative effect of maternal diabetes and obesity on obstetric and perinatal outcomes.

The limitations of the study include a small sample size and restriction to a specific ethnic group, namely black African ethnicity, which restrict the generalisability of our findings. A larger study that includes multiple ethnicities is needed to further validate our results. Also, the study had limited maternal BMI data due to missing height measurements. Therefore, we reported on both BMI and weight, as weight is easily obtained. Nevertheless, despite the low number of BMI measurements, we still observed the effects of both BMI and weight on obstetric and perinatal outcomes. Lastly, hypertension was not categorised into chronic, gestational or preeclampsia.

Conclusion

This study showed that pregestational diabetes is associated with high rates of preterm birth and obesity is associated with the development of GDM, high rates of CS and low Apgar scores at 5 minutes. Adequate glycaemic control and weight loss prior to pregnancy, as well as appropriate gestational weight gain, have been shown to reduce the risk of adverse pregnancy outcomes. Therefore, clinicians should prioritise pre-pregnancy glycaemic control and weight optimisation. Additionally, pregnant women with DIP should be advised about the importance of glycaemic control to reduce adverse pregnancy outcomes. Pregnant women with obesity should be counselled on the importance of appropriate gestational weight gain to prevent the development of GDM. Good antenatal care and education are essential to reduce adverse pregnancy outcomes for mothers with diabetes and obesity.

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