

ORIGINAL RESEARCH

Fetomaternal Medicine

Quality of glycemic control and pregnancy outcomes among patients with gestational diabetes at the Kenyatta National Hospital, Kenya

Salome N. Noreh^{1*}, Joseph W. Gichuhi¹, Alfred Mokomba²

¹ Department of Obstetrics and Gynecology, University of Nairobi, Nairobi, Kenya.

² Department of Obstetrics and Gynecology, Kenyatta National Hospital, Nairobi, Kenya.

*Correspondence: salomenoreh@gmail.com

Received: 10 May 2021; Revised: 10 September 2021; Accepted: 28 September 2021; Available online: October 2021

Copyright © 2021, The authors. Published by JOGECA. This is an open access article under the terms of the Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted reuse, distribution, and reproduction in any medium provided the original author(s) and the source are properly cited.

Abstract

Background: The prevalence of diabetes and gestational diabetes is rising worldwide. If poorly managed, diabetes in pregnancy has a far-reaching negative impact on the mother and newborn.

Objective: To determine the quality of glycemic control and pregnancy outcomes among patients with gestational diabetes in Kenyatta National Hospital, Nairobi, Kenya.

Methods: A retrospective descriptive cohort study design was employed. The study setting was Kenyatta National Hospital, Nairobi, Kenya. The study population was women with diabetes in pregnancy. The study period was May 2011 to November 2019. A sample size of 258 diabetic pregnant patients was reached; 230 and 28 were in the exposed and unexposed groups with the average third trimester fasting blood sugar levels of ≥ 5.3 mmol/L and < 5.3 mmol/L, respectively. Data were analyzed using the IBM statistical package for social sciences (SPSS) version 23. A p-value of < 0.05 was considered statistically significant.

Results: The prevalence of poor glycemic control (average third-trimester fasting blood sugar level ≥ 5.3 mmol/L) was 89.1%. There was a higher incidence of macrosomia (25.7% vs. 21.4%; p-value 0.627), stillbirths (17.9% vs. 3.6%; p-value 0.058) and preterm birth (43.9% vs. 21.4%; p-value 0.025) among patients with gestational diabetes with poor glycemic control compared to those with good glycemic control. Among mothers with poor glycemic control, the pre-existing diabetes patients experienced significantly worse outcomes of stillbirths (20.9% vs. 0%; p-value 0.004) and preterm births (47.2% vs. 24.2%; p-value 0.014) than the gestational diabetes patients.

Conclusion: Poorly controlled diabetes in pregnancy increases the risk of adverse pregnancy outcomes, including macrosomia, stillbirths, and preterm births, with the poorly controlled pre-existing diabetic women experiencing significantly worse outcomes of stillbirths and preterm births than the poorly controlled gestational diabetes patients.

Keywords: gestational diabetes, insulin, macrosomia, stillbirths, preterm births

Introduction

Gestational diabetes mellitus is hyperglycemia recognized for the first time during pregnancy, with

fasting blood sugar of 5.1-6.9mmol/L, 1-hour post 75g glucose load ≥ 10 mmol/L, or 2-hour post 75g glucose load 8.5-11mmol/L (1). Overt or pre-existing diabetes in pregnancy is hyperglycemia detected by

testing at any time during pregnancy that meets the criteria for diagnosis of diabetes in the nonpregnant state, with fasting plasma glucose ≥ 7.0 mmol/L or 2-diabetes (1). The prevalence of diabetes and gestational diabetes is rising worldwide. According to the International Diabetes Federation (IDF), the global estimate of gestational diabetes prevalence in 2017 was 14% (18.4 million live births). Africa contributed 9.5% of all cases (2).

Gestational diabetes mellitus occurs due to the inadequate multiplication of the beta cells in response to the increased demand for insulin due to the elevated insulin-resistant somatotrophic hormones (prolactin, growth hormone, placental lactogen, placental growth hormone). Hence, maternal hyperglycemia occurs with increased transplacental glucose transport. Transplacental glucose transport is modulated by the corticotropin-releasing hormone (3-5). Maternal hyperglycemia leads to fetal hyperglycemia and hyperinsulinemia once the fetal pancreas becomes functional in the second trimester. Fetal hyperinsulinemia increases central fat deposition in the abdominal and interscapular areas, resulting in fetal macrosomia, a significant indicator of fetal hyperglycemia (6). The greater the maternal fasting blood sugar level, the higher the risk of macrosomia and increased probability of shoulder dystocia (7).

A fasting plasma glucose value ≥ 5.6 mmol/L is associated with a five-fold macrosomia risk than a fasting blood sugar value < 4.2 mmol/L (8). Fetal macrosomia heightens the risk of cesarean and instrumental delivery, shoulder dystocia, perineal and bladder trauma, and postpartum hemorrhage in the mother. The risk of fresh stillbirths, nerve injuries, bone fractures, fetal hypoxia is also increased. The baby has a high likelihood of delayed motor development, gaining weight early in life, and an increased risk of getting type II diabetes later in life (9,10). Treatment of gestational diabetes mellitus reduces preeclampsia, perinatal death, shoulder dystocia, birth weight, clavicular or humeral fracture, nerve injury, newborn unit admissions, and labor induction (11). The risk of fetal death around birth, newborn hypoglycemia, birth injuries, preterm deliveries, preeclampsia, cesarean delivery, and labor induction is also significantly reduced (10). According to the 2017 Kenya National Diabetes Guidelines, when treating gestational diabetes mellitus, the aim is to achieve a fasting blood sugar < 5.0 mmol/L and < 5.3 mmol/L for pre-existing diabetes patients (12). Therefore, this study sought to determine the quality of glycemic control and pregnancy outcomes among patients with gestational diabetes in a national referral hospital in Kenya.

Methods

hour 75g oral glucose tolerance test (OGTT) ≥ 11.1 mmol/L, or random plasma glucose ≥ 11.1 mmol/L associated with signs and symptoms of

Study design and setting

A retrospective descriptive cohort study design was used. The study setting was Kenyatta National Hospital (KNH), Kenya's largest referral, teaching, and research hospital. It has an 1800 bed capacity and handles approximately 12000-18000 deliveries annually.

Study population

The study population was patients with gestational diabetes mellitus attended at KNH. Patients with gestational diabetes were identified by screening pregnant women who attended antenatal clinics (ANC) with fasting blood sugar levels and OGTT results.

Data collection and management

Gestational diabetes mellitus was diagnosed with fasting blood glucose 5.1-6.9 mmol/L, 1-hour post 75g oral glucose load ≥ 10 mmol/L, or 2-hour post 75g oral glucose load 8.5-11 mmol/L. For the pre-existing diabetes patients, the fasting blood glucose level was < 5.3 mmol/L. An obstetric ultrasound for fetal monitoring was done. Post-delivery, the blood sugar of babies born to diabetic mothers was measured and recorded in the mother's file. Consecutive sampling was done on patients based on an average of at least two third trimester fasting blood sugar levels between 27 and 37 gestational weeks; the fasting blood sugar in the exposed and unexposed patients was < 5.3 mmol/L and ≥ 5.3 mmol/L, respectively. Diabetic mothers with multiple gestations and preterm birth were excluded in this study. The endpoint of the study was delivery and the study period was May 2011 to November 2019.

Data analysis

A sample size of 258 was achieved; 230 and 28 in the exposed and unexposed groups, respectively. Data were analyzed using the IBM statistical package for social sciences (SPSS) version 23. A p-value of < 0.05 was considered statistically significant. The results were presented in figures and tables.

Ethical consideration

This study's protocol was reviewed and approved by the Kenyatta National Hospital/University of Nairobi Ethics Research Committee (KNH/UoN ERC) (registration number P470/06/2029). Compliance with relevant research and human subjects' regulations was observed.

Results

The mean age of the study population was 32.2 (SD=0.3). The majority, 54.7% (n=141), were between 31-40 years. The mean age for the poor and good glycemic control was 32.2 (SD=5.4) and 32.4 (SD=4.2), respectively (Table 1). A majority, 89.1% (n=230) had poor glycemic control (Figure 1). The average third trimester fasting blood sugar level was 8.27mmol/L (Figure 2). Gestational diabetes patients with poor and good glycemic control were likely to have a history of macrosomia (24.3% vs. 32.1%, p-value 0.37), a history of pregnancy loss (39.6% vs. 28.6%, p-value 0.259), a family history of diabetes mellitus (23.1% vs. 32%, p-value 0.327), a body mass index of ≥ 30 (55.8% vs. 70%, p-value 0.507). A majority, (68.3% vs. 57.1%, p-value 0.237), were on insulin, whereas, a minority (35.7% vs. 21.4%, p-value 0.134) were on oral hypoglycemics (Table 1).

Gestational diabetes patients with poor and good glycemic control were likely to have preterm birth (43.9% vs. 21.4%, p-value 0.025), postpartum hemorrhage (28.9% vs. 44.4%, p-value 0.098), and perineal trauma (15.3% vs. 22.2%, p-value 0.403) (Table 2).

Gestational diabetes patients with poor and good glycemic control were likely to have macrosomia (25.7% vs. 21.4%, p-value 0.627), stillbirths (17.9% vs. 3.6%, p-value 0.058), and neonatal hypoglycemia (27% vs 28.6%, p-value 0.876) (Table 3).

Among gestational diabetes patients with poor glycemic control, the pre-existing and gestational diabetes patients, 58.9% vs. 63.6% (p-value 0.703) had a parity ≥ 2 , a history of macrosomia (22.3% vs. 36.4%, p-value 0.082), a history of pregnancy loss (38.1% vs. 48.5%, p-value 0.258), a history of gestational diabetes (1% vs. 15.2%, p-value 0.001), a family history of diabetes mellitus (23% vs. 24%, p-value 0.914), a body mass index ≥ 30 (51.6% vs. 76.9%, p-value 0.093), had a first antenatal visit < 24 weeks gestation (46.6% vs. 63.6%, p-value 0.131), and had more than four ANC visits (57.4% vs. 87.1%, p-value 0.002). A majority, 69.5% vs. 60.6% (p-value 0.307) were on insulin, whereas, 32.5% vs. 54.5%, p-value 0.014, were on oral hypoglycemics (Table 4).

Among patients with poorly controlled pre-existing and gestational diabetes, the incidence of preterm birth was 47.2% vs. 24.2%, p-value 0.014, postpartum hemorrhage (24% vs. 59.4%, p-value < 0.001), perineal trauma (15.3% vs. 15.2%, p-value 0.982) (Table 5).

Among patients with poorly controlled pre-existing and gestational diabetes, the incidence of macrosomia was 24.9% vs. 30.3% (p-value 0.509), stillbirths (20.9% vs. 0% p-value 0.004), neonatal

hypoglycemia (27.4% vs. 25%, p-value 0.795) (Table 6).

Discussion

A comparison of the sociodemographic data and clinical characteristics of patients with gestational diabetes with poor and good glycemic control was not statistically significant. The prevalence of poor glycaemic control was 89.1% in women with diabetes in pregnancy, not in keeping with the 2018 Kenya National Diabetes Guidelines (12). The incidence of preterm birth was 43.9% and 21.4% in gestational diabetes patients with poor and good glycemic control, respectively. This could be attributed to the higher probability of large gestational age babies in the higher fasting blood glucose level, leading to high preterm labor and birth incidences. The incidence of stillbirths was 20.9% in the poorly controlled pre-existing diabetes. This could be attributed to placental insufficiency in the poorly controlled pre-existing diabetes patients who also had a higher average third-trimester fasting blood sugar level of 9.1mmol/L. Low maternal hyperglycemia stimulates vascular proliferation in response to a lower hypoxia level, ensuring maternal and fetal exchange. However, further increase in glycemic levels inhibits villous angiogenesis, interfering with maternal-fetal exchange and increasing the risk of perinatal mortality (13).

The incidence of postpartum hemorrhage was 24% and 59.4% (p-value 0.001) in the poorly controlled pre-existing and gestational diabetes patients, respectively. This could be due to the higher incidence of fetal macrosomia among the gestational diabetes patients, leading to overdistention of the uterus, with a predisposition for uterine atony and postpartum hemorrhage in the third stage of labor. The incidence of preterm birth was 47.2% and 24.2% (p-value 0.014) in poorly controlled pre-existing and gestational diabetes patients, respectively. This could be due to the longer period of fetal exposure to hyperglycemia in the pre-existing diabetes patients, leading to larger for gestational age babies, with distention of the uterus inducing preterm labor, resulting in preterm birth. In a Saudi Arabian study, the incidence of preterm birth was 33.3% in type 2 diabetes patients, 25% in type 1, and 21.1% in gestational diabetes (14), similar to this study's findings.

The incidence of macrosomia was 25.7% and 21.4% (p-value 0.627) in mothers with poor and good glycemic control, respectively. This was not consistent with the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study, which found that higher fasting plasma glucose values were related to a greater risk of macrosomia (8). The incidence of stillbirths was 17.9% and 3.6% (p-value 0.058) in

poor and good glycemic, respectively. In Uganda, the incidence of stillbirth was 4.1% (15). In another study that examined gestational diabetes mellitus in early pregnancy with good glycemic control, the incidence of stillbirth was 1.8% in those with type 2 diabetes

and 3.4% in those with gestational diabetes diagnosed at less than 12 weeks gestation (16). The incidence of neonatal hypoglycemia was 27% and 28.6% (p-value 0.876) in mothers with poor and good

Table 1: Clinical characteristics in pregnant women with poor versus good glycemic control in a national referral hospital

	Poor (≥ 5.3 mmol/L)	Good (< 5.3 mmol/L)	Total	p-value
Age				
Below 21	2 (0.9)	0 (0.0)	2 (0.8)	
21-30	92 (40)	10 (35.7)	102 (39.5)	
31-40	124 (53.9)	17 (60.7)	141 (54.7)	
>40	12 (5.2)	1 (3.6)	13 (5.0)	
Mean	32.2	32.4	32.2	
Parity				
< 2	93 (40.4)	14 (50.0)	107 (41.5)	0.33
≥ 2	137 (59.6)	14 (50.0)	151 (58.5)	
History of macrosomia				
Yes	56 (24.3)	9 (32.1)	65 (25.2)	0.37
No	174 (75.7)	19 (67.9)	193 (74.8)	
History of pregnancy loss				
Yes	91 (39.6)	8 (28.6)	99 (38.4)	0.26
No	139 (60.4)	20 (71.4)	159 (61.6)	
History of Gestational DM				
Yes	7 (3.0)	1 (3.6)	8 (3.1)	1
No	223 (97.0)	27 (96.4)	250 (96.9)	
Family history of diabetes				
Yes	50 (23.1)	8 (32.0)	58 (24.1)	0.33
No	166 (76.9)	17 (68.0)	183 (75.9)	
Body Mass Index				
Below 30	34 (44.2)	3 (30.0)	37 (42.5)	0.51
30 and above	43 (55.8)	7 (70.0)	50 (57.5)	
Treatment Patient on Insulin				
Yes	157 (68.3)	16 (57.1)	173 (67.1)	0.24
No	73 (31.7)	12 (42.9)	85 (32.9)	
Oral hypoglycemics				
Yes	82 (35.7)	6 (21.4)	88 (34.1)	0.13
No	148 (64.3)	22 (78.6)	170 (65.9)	

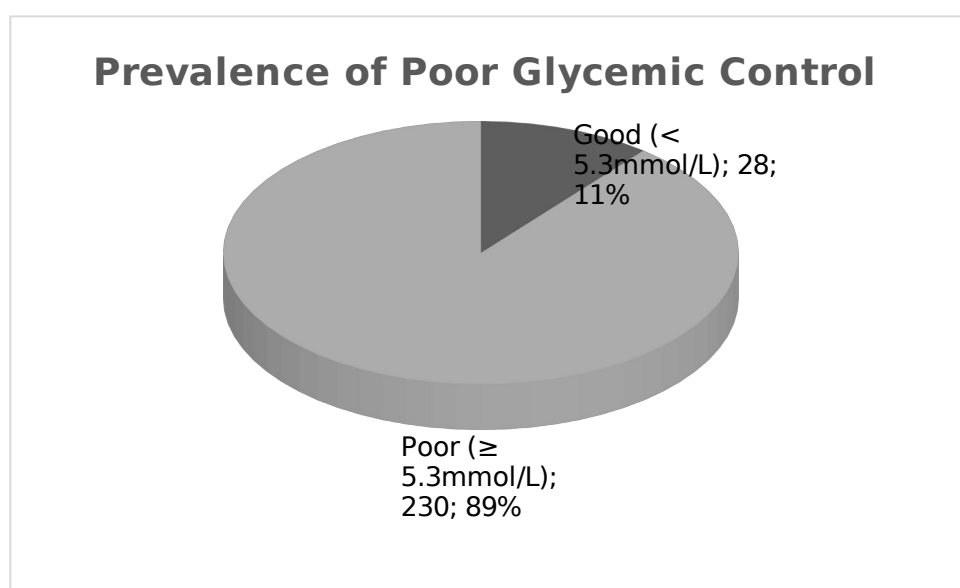


Figure 1: Pie chart showing the prevalence of poor glycemic control

Table 2: Maternal outcomes in pregnant women with poor versus good glycemic control in a national referral hospital

	Poor (≥ 5.3 mmol/L)	Good (< 5.3 mmol/L)	Total	p-value
Gestational age at delivery				
< 37	93 (43.9)	8 (21.4)	101 (43.9)	0.03
≥ 37	104 (52.8)	25 (75.8)	129(56.1)	
Presence of PPH				
Yes	47 (28.9)	19 (44.4)	66 (28.9)	0.1
No	149 (76)	13 (40.6)	162 (71.1)	
Perineal trauma				
Yes	30 (15.3)	5 (22.2)	35 (15.3)	0.4
No	166 (84.7)	28 (84.8)	194 (84.7)	

Table 3: Neonatal outcomes in patients with gestational diabetes with poor and good glycemic control in a national referral hospital

	Poor (≥ 5.3 mmol/L)	Good (< 5.3 mmol/L)	Total	p-value
Birth weight				
Less than 4kg	171 (74.3)	22 (78.6)	193 (74.8)	0.63
≥ 4 kg	59 (25.7)	6 (21.4)	65 (25.2)	
Baby alive				
Yes	188 (82.1)	27 (96.4)	215(83.7)	0.06
No	41 (17.9)	1 (3.6)	42 (16.3)	
Presence of neonatal hypoglycemia				
Yes	38 (27)	6 (28.6)	44 (27.2)	0.88
No	103 (73)	15 (71.4)	118 (72.8)	

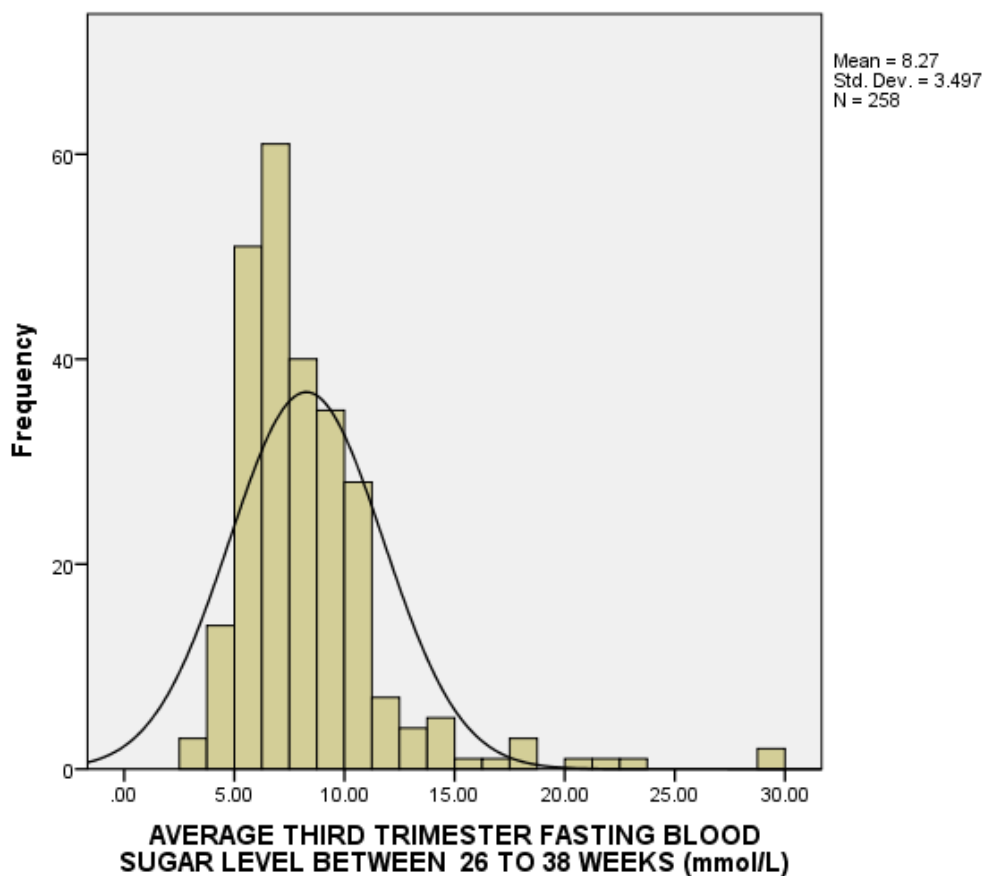
**Figure 2:** Bar chart showing the distribution of the third-trimester fasting blood sugar levels

Table 4: Clinical characteristics of mothers with poor glycemic control in a national referral hospital

	Poorly Controlled Pre-existing Diabetes	Poorly Controlled Gestational Diabetes	Total	p-value
Parity				
< 2	81 (41.1)	12 (36.4)	93 (40.4)	0.7
≥ 2	116 (58.9)	21 (63.6)	137 (59.6)	
History of macrosomia				
Yes	44 (22.3)	12 (36.4)	56 (24.3)	0.08
No	153 (77.7)	21 (63.6)	174 (75.7)	
History of pregnancy loss				
Yes	75 (38.1)	16 (48.5)	91 (39.6)	0.26
No	122 (61.9)	17 (51.5)	139 (60.4)	
History of Gestational DM in an earlier pregnancy				
Yes	2 (1.0)	5 (15.2)	7 (3.0)	0
No	195 (99.0)	28 (84.8)	223 (97.0)	
Family history of diabetes				
Yes	44 (23.0)	6 (24.0)	50 (23.1)	0.91
No	147 (77.0)	19 (76.0)	166 (76.9)	
Body Mass Index				
Below 30	31 (48.4)	3 (23.1)	34 (44.2)	0.09
30 and above	33 (51.6)	10 (76.9)	43 (55.8)	
Gestational age at first antenatal visit (weeks)				
< 24	81 (46.6)	14 (63.6)	95 (48.5)	0.13
≥ 24	93 (53.4)	8 (36.4)	101 (51.5)	
Number of ANC visits				
< 4	81 (42.6)	4 (12.9)	85 (38.5)	0
≥ 4	109 (57.4)	27 (87.1)	136 (61.5)	
Treatment Patient on Insulin				
Yes	137 (69.5)	20 (60.6)	157 (68.3)	0.31
No	60 (30.5)	13 (39.4)	73 (31.7)	
Oral hypoglycemics				
Yes	64 (32.5)	18 (54.5)	82 (35.7)	0.01
No	133 (67.5)	15 (45.5)	148 (64.3)	

Table 5: Maternal outcomes in patients with poorly controlled pre-existing and gestational diabetes

	Poorly Controlled Pre-existing Diabetes	Poorly Controlled Gestational Diabetes	Total	p-value
Gestational age at delivery				
< 37	93 (47.2)	8 (24.2)	101 (43.9)	0.01
≥ 37	104 (52.8)	25 (75.8)	129(56.1)	
Presence of PPH				
Yes	47 (24)	19 (59.4)	66 (28.9)	<0.001
No	149 (76)	13 (40.6)	162 (71.1)	
Perineal trauma				
Yes	30 (15.3)	5 (15.2)	35 (15.3)	0.98
No	166 (84.7)	28 (84.8)	194 (84.7)	

Table 6: Neonatal outcomes in patients with poorly controlled pre-existing and gestational diabetes

	Poorly Controlled Pre-existing Diabetes	Poorly Controlled Gestational Diabetes	Total	p-value
Birth weight				
Less than 4kg	148 (75.1)	23 (69.7)	171 (74.3)	0.51
≥ 4kg	49 (24.9)	10 (30.3)	59 (25.7)	
Baby alive				
Yes	155 (79.1)	33 (100)	188 (82.1)	0
No	41 (20.9)	0 (0.0)	41 (17.9)	
Presence of neonatal hypoglycemia				
Yes	31 (27.4)	7 (25.0)	38 (27.0)	0.8
No	82 (72.6)	21 (75.0)	103 (73.0)	

glycemic control, respectively. This differed in trend from the findings of the HAPO study, which found a linear relationship between maternal blood sugar levels and neonatal hypoglycemia (8). The incidence of postpartum hemorrhage was 28.9% and 44.4% (p-value 0.098) in gestational diabetes patients with poor and good glycemic control, respectively. This compared unfavorably to a Cameroonian study that reported an incidence of 3.2% (17). The incidence of perineal trauma was 15.3% and 22.2% in mothers with poor and good glycemic control, respectively (p-value 0.403). Studies in Uganda and Cameroon reported 53.1% and 13% of perineal trauma, respectively (13,17). The incidence of neonatal hypoglycemia was 27.4% and 25% (p-value 0.795) in patients with poorly controlled pre-existing and gestational diabetes, respectively. In Saudi Arabia, the neonatal hypoglycemia incidence was 33.3% in type 2 diabetes, 41% in type 1, and 15.4% in gestational diabetes (14), relatively similar to this study.

Study strengths and limitations

The inaccessibility of data due to poor storage of patient diagnosis records and computer system failure was a significant limitation in this study. Body mass index is not routinely calculated and recorded at patient visits in the study hospital. Thus, a high body mass index was an important confounder in this study.

Conclusion

There is a higher incidence of macrosomia, stillbirths, and preterm birth among gestational diabetes patients with poor than those with good glycemic control. Among mothers with poor glycemic control, those with pre-existing diabetes patients experienced significantly worse outcomes of stillbirths and preterm births than those with poorly controlled gestational diabetes. Poorly controlled pregnancy diabetes increases the risk of adverse pregnancy outcomes such as macrosomia, stillbirths, and preterm birth, with poorly controlled pre-existing diabetes patients sometimes experiencing worse outcomes than those with poorly controlled gestational diabetes.

Recommendations

Health care workers and antenatal mothers should be educated on risk factors for pregnancy diabetes, the importance of early-onset and regular antenatal follow-up, timing, screening for diabetes in pregnancy, and symptoms to watch out for in diabetes mellitus. The glycemic targets to aim for in the control of diabetes mellitus should also be disseminated to patients and health care providers managing gestational diabetes patients. Early diagnosis and management of diabetes in pregnancy

are essential to avoid adverse fetal and maternal outcomes.

Acknowledgement

The authors wish to acknowledge the Kenyatta National Hospital antenatal and labor ward nurses for their assistance in this study.

Declarations

Conflict of interests

The authors declare no conflicts of interest.

Funding

This study was self-funded.

References

- Hod M, Kapur A, Sacks DA, et al. The International Federation of Gynecology and Obstetrics (FIGO) Initiative on gestational diabetes mellitus: A pragmatic guide for diagnosis, management, and care. *Int J Gynaecol Obstet.* 2015;131 Suppl 3:S173-S211. doi:10.1016/S0020-7292(15)30033-3
- Cho NH, Shaw JE, Karuranga S, et al. IDF Diabetes Atlas: Global estimates of diabetes prevalence for 2017 and projections for 2045. *Diabetes Res Clin Pract.* 2018;138:271-281. doi:10.1016/j.diabres.2018.02.023
- Thomson M. The physiological roles of placental corticotropin releasing hormone in pregnancy and childbirth. *J Physiol Biochem.* 2013;69(3):559-573. doi:10.1007/s13105-012-0227-2
- Nielsen JH. Beta cell adaptation in pregnancy: a tribute to Claes Hellerström. *Ups J Med Sci.* 2016;121(2):151-154. doi:10.3109/03009734.2016.1165776
- Jayabalan N, Nair S, Nuzhat Z, et al. Cross Talk between Adipose Tissue and Placenta in Obese and Gestational Diabetes Mellitus Pregnancies via Exosomes. *Front Endocrinol (Lausanne).* 2017;8:239. Published 2017 Sep 27. doi:10.3389/fendo.2017.00239
- Lowe LP, Metzger BE, Dyer AR, et al. Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study: associations of maternal A1C and glucose with pregnancy outcomes. *Diabetes Care.* 2012;35(3):574-580. doi:10.2337/dc11-1687
- Athukorala C, Crowther CA, Willson K; Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) Trial Group. Women with gestational diabetes mellitus in the ACHOIS trial: risk factors for shoulder dystocia. *Aust N Z J Obstet Gynaecol.* 2007;47(1):37-41. doi:10.1111/j.1479-828X.2006.00676.x
- HAPO Study Cooperative Research Group, Metzger BE, Lowe LP, et al. Hyperglycemia and adverse pregnancy outcomes. *N Engl J Med.*

- 2008;358(19):1991-2002.
doi:10.1056/NEJMoa0707943
9. Campbell S. Fetal macrosomia: a problem in need of a policy. *Ultrasound Obstet Gynecol.* 2014;43(1):3-10. doi:10.1002/uog.13268
 10. Poolsup N, Suksomboon N, Amin M. Effect of treatment of gestational diabetes mellitus: a systematic review and meta-analysis. *PLoS One.* 2014;9(3):e92485. Published 2014 Mar 21. doi:10.1371/journal.pone.0092485
 11. Cheung NW, Oats JJ, McIntyre HD. Australian carbohydrate intolerance study in pregnant women: implications for the management of gestational diabetes. *Aust N Z J Obstet Gynaecol.* 2005;45(6):484-485. doi:10.1111/j.1479-828X.2005.00481.x
 12. Ministry of Health. Guidelines for the screening and management of Diabetic Retinopathy in Kenya Guidelines for Screening and Management of Diabetic Retinopathy MINI REPUBLIC OF KENYA STRY OF HEALTH [Internet]. [cited 2021 Sep 10]. Available from: <http://www.health.go.ke/wp-content/uploads/2017/11/Guidelines-for-Screening-and-Management-of-Diabetic-Retinopathy-in-Kenya.pdf>
 13. Calderon IMP, Damasceno DC, AMorin RL, Costa RAA, Brasil MAM, Rudge MVC. Morphometric study of placental villi and vessels in women with mild hyperglycemia or gestational or overt diabetes. *Diabetes Res Clin Pract.* 2007;78:65-71.
 14. Buhary BM, Almohareb O, Aljohani N, et al. Glycemic control and pregnancy outcomes in patients with diabetes in pregnancy: A retrospective study. *Indian J Endocrinol Metab.* 2016;20(4):481-490. doi:10.4103/2230-8210.183478
 15. Nakabuye B, Bahendeka S, Byaruhanga R. Prevalence of hyperglycaemia first detected during pregnancy and subsequent obstetric outcomes at St. Francis Hospital Nsambya. *BMC Res Notes.* 2017;10(1):174. Published 2017 May 2. doi:10.1186/s13104-017-2493-0
 16. Sweeting AN, Ross GP, Hyett J, et al. Gestational Diabetes Mellitus in Early Pregnancy: Evidence for Poor Pregnancy Outcomes Despite Treatment. *Diabetes Care.* 2016;39(1):75-81. doi:10.2337/dc15-0433
 16. Sweeting AN, Ross GP, Hyett J, et al. Gestational Diabetes Mellitus in Early Pregnancy: Evidence for Poor Pregnancy Outcomes Despite Treatment. *Diabetes Care.* 2016;39(1):75-81. doi:10.2337/dc15-0433
 17. Djomhou M, Sobngwi E, Noubiap JJ, Essouma M, Nana P, Fomulu NJ. Maternal hyperglycemia during labor and related immediate post-partum maternal and perinatal outcomes at the Yaoundé Central Hospital, Cameroon. *J Health Popul Nutr.* 2016;35(1):28. Published 2016 Aug 22. doi:10.1186/s41043-016-0065-x