Gestational diabetes mellitus (GDM) is diabetes that is diagnosed during the second or third trimester of pregnancy and is not clearly overt diabetes (1). Diagnosis is defined by severity of carbohydrate intolerance. The upper end of the GDM diagnostic glucose range is the same as would be indicative of diabetes outside of pregnancy, whereas the lower end of the GDM range is only slightly above normal and asymptomatic but still associated with increased risk of fetal morbidity (1,2).

Diabetes during pregnancy is diagnosed by either a one-step approach involving a 75-g oral glucose tolerance test (OGTT) or a two-step approach starting with a 50-g (nonfasting) screen followed by a 100-g OGTT for those who initially screen positive (1). Glycemic goals for patients with a GDM diagnosis are as follows: preprandial ≤95 mg/dL and either 1-hour postprandial ≤140 mg/dL or 2-hour postprandial ≤120 mg/dL.

Hyperglycemia throughout pregnancy carries increased risk for adverse fetal and maternal outcomes (3–8). Treatment of diabetes during pregnancy is aimed at decreasing the risk of perinatal outcomes such as macrosomia, birth trauma, neonatal metabolic abnormalities, and cesarean section (4,9–12). Lifestyle modification is first-line treatment and includes medical nutrition therapy (MNT), exercise, and glucose monitoring (13). Pharmacological therapy generally consists of insulin, glyburide, or metformin, and agents may be used adjunctly to MNT depending on presence and severity of hyperglycemia (13). Insulin is the preferred pharmacological treatment for management of diabetes in pregnancy if lifestyle modification is insufficient in achieving euglycemia (13).

The setting of this review is the diabetes clinic located within the Hall County Health Department (HCHD) prenatal clinic in Gainesville, Ga. The population of Gainesville is ~187,000 and includes a large percentage of Latino immigrants (14). The proportion of Latinos in the diabetes clinic has grown from 20% in the early 1990s to >90% today. The percentage of uninsured individuals, as well as the high percentage of people with diabetes and obesity, in Hall County led to an increase in available funding for safety net providers such as the HCHD.

The clinic provides access to comprehensive, high-quality, affordable prenatal care for low-income, uninsured women. The clinic was formed in the 1970s in response to an increasing number of women lacking prenatal care who presented to the local hospital for delivery. Initially, local physicians donated their time to the clinic and worked with HCHD nursing staff to provide obstetrical care. In the late 1980s, a midwifery program was added to the clinic. In the mid-1990s, The Longstreet Clinic (TLC), a regional multidisciplinary physicians’ practice, joined efforts to operate the obstetrical clinic with the HCHD. As the need for manage-
ment of GDM grew in the late 1990s, a collaboration was formed to allow for the provision of diabetes care by a physician-nurse team. A nurse certified diabetes educator (CDE) began to manage the diabetes care for these patients under the direction of an obstetrician. In 2008, a CDE public health pharmacist began managing the GDM patients working under a collaborative practice agreement with the TLC obstetrician. The pharmacist began a faculty appointment with Philadelphia College of Osteopathic Medicine (PCOM) School of Pharmacy in 2010 and has continued to provide services in the diabetes clinic at the HCHD.

The clinic now functions as a collaboration of the Northeast Georgia Health System, TLC, and the HCHD. Its novel approach has not been duplicated elsewhere. The team of providers includes a TLC obstetrician, a certified nurse midwife (CNM), a pharmacist who is a CDE and is board-certified in advanced diabetes management, and a registered dietitian (RD) with the HCHD. The CNM sees all indigent obstetrics clinic patients, whereas the pharmacist and RD see only those clinic patients who have diabetes. The pharmacist manages the diabetes treatment plan and ongoing diabetes monitoring under a collaborative protocol with the TLC obstetrician. The RD provides MNT education and counseling. Approximately 15–20 patients are receiving care at any given time within the diabetes clinic of the indigent obstetrics clinic.

**Objective**

The objective of this retrospective review was to compare maternal and fetal outcomes between treatment with lifestyle modification (Group A) or lifestyle modification and pharmacological therapy (Group B) in Latina women with GDM.

**Methods**

All patients within the indigent obstetrics clinic at the HCHD are given a 1-hour OGTT at 24–28 weeks' gestation, except for patients who have a family history of diabetes, who have the test before 24 weeks' gestation. At their initial visit, patients are educated about diabetes pathophysiology, how to perform self-monitoring of blood glucose (SMBG) and record their results, glycemic goals, and lifestyle modification, including MNT, physical activity, and weight management. Patients are given a Reli-On blood glucose meter and instructions for its use by the pharmacist, who instructs them to perform SMBG when fasting and 2 hours after each meal. The RD provides MNT education during which patients complete a diet recall and are instructed to limit carbohydrate consumption to 30 g at breakfast, 45 g each at lunch and dinner, and 15 g each at one or two daily snacks.

Follow-up visits occur weekly until glycemic control is achieved; once glucose is within target ranges, follow-up visits are scheduled every other week. At each follow-up visit, patients’ average blood glucose levels at each time of day are calculated, blood glucose is measured, and self-reported nocturia is discussed.

The initial treatment plan for GDM includes lifestyle modification and may progress to the addition of medication such as metformin, glyburide, or insulin (11,14–18). In our clinic, insulin is initiated in patients whose fasting blood glucose (FBG) and/or 2-hour postprandial glucose (PPG) levels are ≥10 mg/dL above the target range after lifestyle modification. A local grant allows the clinic to provide glucose meters, test strips, lancets, insulin, and syringes to patients at each visit as needed at no charge. Glyburide and metformin are feasible pharmacological options for patients at this clinic because both are available for $4.00 per month at local pharmacies.

The clinical pharmacist works with an interpreter to communicate with patients, thereby reducing any language barriers that may exist. The care team assesses patients’ glycemic patterns and literacy level and asks about their work schedules and other patient-specific factors to allow for customization of the selection, frequency, and dosing of any pharmacological agents.

Fetal surveillance consists of monthly serial growth ultrasounds, twice-weekly fetal nonstress testing, and weekly amniotic fluid index (modified biophysical profile) for patients on pharmacotherapy after 32 weeks. The modified biophysical profile consists of a nonstress test and amniotic fluid volume assessment.

Per hospital protocol, infant blood glucose is measured 1 hour after birth. The goal blood glucose from birth to 4 hours of life is >30 mg/dL; after 4 hours of life, it is >40 mg/dL. Infants whose initial blood glucose is <30 mg/dL are transferred to the neonatal intensive care unit. Otherwise, infants’ blood glucose continues to be monitored before feedings until it is stable. If at any time an infant’s blood glucose becomes unstable or an infant becomes symptomatic for hypoglycemia, the provider is contacted for direction of the care plan.

Bilirubin levels are assessed for every infant at 24 hours and again at discharge. A Coombs test is commonly performed in newborns to test for evidence of a reaction between the blood groups of the mother and baby. Blood is taken from the baby’s cord or from the baby after delivery. Antibodies are produced if the baby’s blood group is different from the mother’s blood. If the Coombs test is positive, then baby will be further screened for jaundice and anemia. For infants who are Coombs positive, bilirubin is assessed at 6 hours and then every 12 hours.

A retrospective chart review was performed of 128 Latina patients with GDM (only 5 of whom had a history of GDM) receiving care in the diabetes clinic between March 2012 and March 2014. Data were collected from both paper charts and electronic medical records between August 2014 and May 2015. Baseline
data collected included patient age, ethnicity, BMI at initial visit, A1C at initial visit, and gestational age (in weeks) at diagnosis. Fetal and maternal outcomes assessed are listed in Table 1. Statistical analysis of the data using SPSS Statistics version 22 (IBM, Chicago, Ill.) was performed with Student \( t \) tests (two-tailed) for all continuous data when comparing the two groups. Nominal data were compared by \( \chi^2 \) and Fisher exact tests where appropriate. Statistical significance was considered \( P < 0.05 \). The study was approved by the PCOM institutional review board.

**Results**

Baseline characteristics of patients included in this retrospective review are summarized in Table 2. Group A (lifestyle modification) included 80 patients; Group B (lifestyle modification plus pharmacological therapy) included 48 patients. Maternal age at diagnosis was similar between groups (Group A 32.9 ± 5.5 years; Group B 34.6 ± 4.9 years). The mean age in the mid-30s in both groups was characteristic of women at high risk for GDM (10,19). Mean gestational age at diagnosis was 20.5 ± 9.1 weeks in Group A and 21.6 ± 8.7 weeks in Group B \( (P = 0.49) \). These mean gestational ages are earlier than the standard mean gestational age of 24–28 weeks at diagnosis \( (1) \). Group B had a significantly higher mean BMI than Group A \( (33.6 ± 5.4 \text{ vs. } 31.3 ± 4.8 \text{ kg/m}^2, P = 0.01) \). The difference in mean A1C values was statistically significant between groups \( (5.6 ± 0.5\% \text{ in Group A vs. } 5.9 ± 0.6\% \text{ in Group B, } P < 0.003) \).

Table 3 shows medication use, and Table 4 shows the treatment algorithm the clinic follows.

Maternal outcomes are summarized in Table 5. Maternal glycemic values were calculated based on the difference in average SMBG values for each time of day (i.e., fasting and 2 hours after breakfast and dinner) from the initial GDM clinic visit to the last GDM clinic visit. Any missing blood glucose values were not accounted for in calculation of the average. Maternal glycemic values were significantly different at all three assessed time points (2-hour postprandial breakfast 108.23 ± 13.3 mg/dL in Group A vs. 114.0 ± 11.6 mg/dL in Group B, \( P = 0.02 \); 2-hour postprandial dinner 116.6 ± 14.1 mg/dL in Group A vs. 123.0 ± 15.9 mg/dL in Group B, \( P = 0.02 \); and FPG 89.8 ± 10.9 mg/dL in Group A vs. 99.2 ± 11.0 mg/dL in Group B, \( P < 0.001 \)). There were no significant

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**Table 1. Fetal and Maternal Outcomes Assessed**

| Maternal Outcomes | Group A | Group B | \( P \) |
|-------------------|---------|---------|--------|
| Maternal weight gain |  |  |  |
| Weight gained from initial clinic visit to last visit |  |  |  |
| Hypertension |  |  |  |
| SBP >140 mmHg or DBP >90 mmHg |  |  |  |
| Preeclampsia |  |  |  |
| SBP >140 mmHg, DBP >90 mmHg, or both AND |  |  |  |
| Proteinuria |  |  |  |
| If no proteinuria: |  |  |  |
| Thrombocytopenia |  |  |  |
| Renal insufficiency |  |  |  |
| Maternal glycemic control |  |  |  |
| Fasting |  |  |  |
| 2-hour postprandial (breakfast) |  |  |  |
| 2-hour postprandial (dinner) |  |  |  |
| Maternal A1C (at end of pregnancy) |  |  |  |

| Fetal Outcomes | Group A | Group B | \( P \) |
|----------------|---------|---------|--------|
| LGA (birthweight ≥ 2 SD above the mean) |  |  |  |
| Macrosomia |  |  |  |
| Fetal weight >4,000 g |  |  |  |
| Birth weight |  |  |  |
| Apgar score at 5 minutes |  |  |  |
| Neonatal hypoglycemia |  |  |  |
| Serum glucose <40 mg/dL |  |  |  |
| Premature birth |  |  |  |
| Birth before week 37 |  |  |  |
| Neonatal jaundice |  |  |  |
| Total serum bilirubin level >5 mg/dL |  |  |  |
| Shoulder dystocia |  |  |  |

SBP, systolic blood pressure; DBP, diastolic blood pressure.

**Table 2. Baseline Characteristics in Group A (Lifestyle Modification) and Group B (Lifestyle Modification Plus Pharmacological Therapy)**

|                      | Group A | Group B | \( P \) |
|----------------------|---------|---------|--------|
| Patient age (years)  | 32.9 ± 5.5 | 34.6 ± 4.9 | 0.079 |
| BMI at initial visit (kg/m²) | 31.3 ± 4.8 | 33.6 ± 5.4 | 0.01 |
| A1C at initial visit (%) | 5.6 ± 0.5 | 5.9 ± 0.6 | <0.003 |
| Gestational age at diagnosis (weeks) | 20.5 ± 9.1 | 21.6 ± 8.7 | 0.49 |

Values are given as mean (SD). Bold indicates statistical significance.
between-group differences in caesarean delivery, maternal weight gain, hypertension, or preeclampsia.

Fetal outcomes are summarized in Table 6. There were no significant between-group differences in number of infants who were large for gestational age (LGA) ($P = 0.08$), macrosomia ($P = 0.14$), birth weight ($P = 0.22$), Apgar score at 5 minutes ($P = 0.135$), premature birth ($P = 0.54$), or shoulder dystocia ($P = 0.57$). There were no cases of neonatal jaundice or neonatal hypoglycemia in either group.

**Discussion**

Pregnancy presents many physiological challenges, and diabetes adds to the complexity of this phenomenon (4). Several risk factors are associated with the development of GDM, including elevated A1C (20), higher BMI (21), greater gestational weight gain (22), increasing age (21,22), family history of diabetes (21,22), and ethnicity (21,23). Risk factors during pregnancy, such as elevated A1C (indicative of hyperglycemia), obesity (indicative of insulin resistance), and gestational weight gain, are associated with adverse maternal and fetal outcomes in patients with GDM (4). When multiple risk factors are present in pregnant patients, there is an increased risk for adverse maternal and fetal outcomes such as LGA newborns and macrosomia (4).

Insulin was initiated in 46 (36%) of the patients in this study (Table 3), which was a greater proportion than literature reports of 10–30% (24,25), reflecting the combination of ethnic origin (Latina) and BMI $\geq 25$ kg/m$^2$ in clinic patients. Insulin is the pharmacotherapy treatment of choice for GDM and is regarded as safe for use in maintaining glycemic control in GDM patients (1).

Although this study did not find any statistically significant between-group differences in fetal outcomes, the difference in LGA (1 case [1.3%] in Group A vs. 3 cases [6.3%] in Group B, $P = 0.08$) was trending.

### Table 3. Medication Use in Group B ($n = 48$)

| Medication | $n$ (%) |
|------------|---------|
| Glyburide  | 2 (4.2) |
| Insulin lispro | 3 (6.3) |
| 75% insulin lispro protamine suspension and 25% insulin lispro | 14 (29.1) |
| Human insulin [rDNA origin] isophane + insulin aspart | 5 (10.4) |
| Human insulin [rDNA origin] isophane | 11 (22.9) |
| Insulin aspart | 5 (10.4) |
| 70% insulin aspart protamine suspension and 30% insulin aspart | 3 (6.3) |
| Human insulin [rDNA origin] isophane + insulin lispro | 5 (10.4) |

### Table 4. Treatment Algorithm in the Diabetes Clinic

**Glyburide:**
- If FBG is $\geq 95$ and $\leq 110$ mg/dL and 2-hour PPG is $\geq 120$ and $\leq 140$ mg/dL, consider a trial of glyburide 2.5 mg twice daily 30 minutes before breakfast and dinner.
- If FBG is $\geq 95$ and $\leq 110$ mg/dL, consider glyburide 2.5 mg at bedtime.
- Dose can be adjusted as needed up to 10 mg twice daily.

**Insulin:**
- If FBG is $\geq 110$ mg/dL or 2-hour PPG is $\geq 40$ mg/dL, initiate insulin therapy.

Pattern management is used to determine the type and dosage of insulin. The time of day that hyperglycemia occurs determines which type of insulin is initiated. For example, if a patient has consistently elevated FBG, NPH insulin is initiated at bedtime. For patients with hyperglycemia both while fasting and 2 hours after breakfast and dinner, premixed insulin (either lispro 75/25 or aspart 70/30) is initiated before breakfast and dinner. Give two-thirds of the following total daily doses before breakfast and one-third before dinner:

| Trimester | Dose (units/kg) |
|-----------|-----------------|
| First     | 0.7–0.8         |
| Second    | 0.8–1.0         |
| Third     | 0.9–1.2         |
| Any, with obesity | 1.5–2.0 |

### Table 5. Maternal Outcomes

| Outcome | Group A | Group B | $P$  |
|---------|---------|---------|------|
| Weight gain, mean (SD), lb | 14.3 (11.3) | 16.7 (10.7) | 0.24 |
| Hypertension, n (%)* | 3 (3.8) | 3 (6.3) | 0.75 |
| Pre-eclampsia, n (%) | 6 (7.5) | 2 (4.2) | 0.70 |
| Glycemic values, mean (SD), mg/dL | | | |
| Fasting | 89.8 (10.9) | 99.2 (11.0) | <0.001 |
| 2-hour post-breakfast | 108.23 (13.3) | 114.0 (11.6) | 0.02 |
| 2-hour post-dinner | 116.6 (14.1) | 123.0 (15.9) | 0.02 |
| A1C at end of pregnancy, mean (SD), % | 5.5 (0.4) | 5.9 (1.1) | 0.006 |

*Hypertension was defined as blood pressure $>140/90$ mmHg. Bold indicates statistical significance.
toward significance. Higher rates of LGA and macrosomia are to be expected in insulin-treated women and have been observed in several studies (19,26,27), although the opposite has also been observed (10,28).

Despite the large proportion of overweight or obese Latina women in this patient group, it should be noted that we achieved a lower incidence of LGA (3.125%) than literature reports of 9–28% (19,29–31).

This study was limited by its small sample size and between-group differences in A1C and BMI at baseline, both of which were higher in Group B. Other limitations include the retrospective nature of the study, the homogeneous ethnicity of participants, and the fact that women from only one clinic were evaluated.

**Conclusion**

Excessive morbidity and mortality was reduced in the group receiving medication plus lifestyle modification to the same level as for the women receiving lifestyle modification only. The relative effectiveness of insulin as an adjunct to lifestyle modification should be further evaluated. Additional research is needed to evaluate significant factors useful in improving maternal and fetal outcomes in GDM patients on insulin therapy. With rates of diabetes increasing (32,33), there is likely to be a corresponding continued increase in the number of women who develop GDM.

**Duality of Interest**

No potential conflicts of interest relevant to this article were reported.

**Author Contributions**

S.W.R. researched data and wrote, reviewed, and edited the manuscript. H.S.P. and M.M. contributed to discussion and wrote, reviewed, and edited the manuscript. S.W.R. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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**TABLE 6. Fetal Outcomes**

| Outcome                      | Group A | Group B | P     |
|------------------------------|---------|---------|-------|
| LGA, n (%)                   | 1 (1.3) | 3 (6.3) | 0.08  |
| Macrosomia, n (%)            | 6 (7.6) | 8 (16.67)| 0.14  |
| Birth weight (g), mean (SD)  | 3,361 (530) | 3,490 (607) | 0.22  |
| Apgar score at 5 minutes, mean (SD) | 8.9 (0.3) | 8.7 (1.1) | 0.135 |
| Neonatal hypoglycemia, n (%) | 0 (0)   | 0 (0)   | 0.56  |
| Premature birth, n (%)       | 7 (8.8) | 5 (10.4) | 0.54  |
| Neonatal jaundice, n (%)     | 0 (0)   | 0 (0)   | 0.56  |
| Shoulder dystocia, n (%)     | 2 (2.5) | 1 (2.1)  | 0.57  |

* LGA was defined as birth weight ≥2 SD above the mean.

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