HHS Public Access

Author manuscript

J Perinatol. Author manuscript; available in PMC 2016 July 21.

Published in final edited form as:

J Perinatol. 2016 May ; 36(5): 347–351. doi:10.1038/jp.2015.216.

Predicting Failure of Glyburide Therapy in Gestational Diabetes

Lorie M. Harper, MD, MSCI[1], **Angelica V. Glover, MD**[1], **Joseph R. Biggio, MD**[1], and **Alan Tita, MD, PhD**[1]

[1]The University of Alabama at Birmingham, Center for Women's Reproductive Health, Department of Obstetrics and Gynecology, Birmingham, AL

Abstract

Objective—We sought to develop a prediction model to identify women with gestational diabetes (GDM) who require insulin to achieve glycemic control.

Study Design—Retrospective cohort of all singletons with GDM treated with glyburide 2007–2013. Glyburide failure was defined as reaching glyburide 20 mg/day and receiving insulin. Glyburide success was defined as any glyburide dose without insulin and >70% of visits with glycemic control. Multivariable logistic regression analysis was performed to create a prediction model.

Results—Of 360 women, 63 (17.5%) qualified as glyburide failure and 157 (43.6%) glyburide success. The final prediction model for glyburide failure included prior GDM, GDM diagnosis ≤26 weeks, 1-hour GCT ≥228 mg/dL, 3-hour GTT 1-hour value ≥221 mg/dL, ≥7 post-prandial blood sugars >120 mg/dL in the week glyburide started, and ≥1 blood sugar >200 mg/dL. The model accurately classified 81% of subjects.

Conclusions—Women with GDM who will require insulin can be identified at initiation of pharmacologic therapy.

Introduction

In 2000, Langer et al published a randomized control trial comparing glyburide and insulin for the treatment of gestational diabetes (GDM).[1] They demonstrated that glyburide and insulin achieve similar levels of maternal glycemic control. Given that glyburide is significantly easier to use and less expensive than insulin, glyburide has become a first-line therapy for many clinicians for the treatment of GDM. The use of glyburide to treat GDM has increased dramatically since 2001, increasing from 7.4% of prescriptions to treat GDM to 64.5% in 2011.[2] However, several retrospective studies have found an increased risk of macrosomia or large for gestational age infants in women receiving glyburide compared to

Corresponding Author: Lorie M. Harper, The University of Alabama at Birmingham, Department of Obstetrics and Gynecology, 1700 6th Ave South, Ste 10270, Birmingham, AL 35233, Phone: 205-975-0515, Fax: 205-975-4375, ; Email: lmharper@uabmc.edu

Presented as a poster at The Pregnancy Meeting, Society for Maternal Fetal Medicine, February 6, 2015, San Diego, CA.

Conflict of Interest: The authors have no conflicts of interest.

those receiving insulin.[3, 4, 5] Other retrospective studies have found a higher incidence of preeclampsia in women treated with glyburide compared to insulin.[6, 7]

These increased adverse perinatal outcomes in women treated with glyburide may be due to delays in glycemic control in those who ultimately fail glyburide therapy and require treatment with insulin. This situation arises in 5–20% of women who require medical therapy for GDM.[1, 8, 9, 10] Delay of insulin initiation may result in weeks of hyperglycemia and subsequently increased risks of adverse perinatal outcomes.

We therefore we aimed to develop a prediction model for glyburide failure based on factors known at the time of initiation of medical therapy for GDM in order to optimize therapy for women requiring medical therapy. We hypothesized that failure of glyburide therapy can be predicted at the time that pharmacotherapy for GDM is initiated.

Materials and Methods

We conducted a retrospective cohort study of all singletons diagnosed with GDM and treated with glyburide at the University of Alabama at Birmingham Jan 1, 2007–Dec 31, 2013. Institutional review board approval was obtained.

Subjects were identified from the searchable electronic medical record system using a diagnosis of gestational diabetes and diabetes; a diagnosis of gestational diabetes was confirmed on review of medical records. The protocol for diagnosing gestational diabetes is to perform a 1-hour glucose tolerance test on all women; if ≥135 mg/dL a 3-hour glucose tolerance is performed. Subjects were considered to have gestational diabetes if a 3-hour glucose tolerance test was performed and met Carpenter-Coustan criteria,[11] if a 1-hour glucose challenge test was ≥200 mg/dL, or if fasting blood sugar was ≥120 mg/dL. Trained personnel (obstetricians and medical students) reviewed medical records to abstract detailed patient data using standardized data collection forms. Abstracted data included maternal age, ethnicity, socioeconomic status, self-reported prepregnancy height and weight, comorbid medical conditions, obstetric history, blood sugar testing, gestational weight gain, prenatal blood sugar logs, delivery details, and perinatal outcomes.

At UAB, women with gestational diabetes are managed by Maternal-Fetal Medicine specialists. Women are screened at the recommended time of 24–28 weeks estimated gestation. Women with risk factors for gestational diabetes, such as obesity or a prior history of gestational diabetes may be screened earlier at the discretion of the provider. All women diagnosed with gestational diabetes receive individualized nutrition counseling and are advised to check their blood sugars fasting and 2-hours after each meal. Women are typically seen at 1 week intervals until glycemic targets are reached, and then may have visits every other week until 34–36 weeks. Glycemic targets are fasting blood sugar <95mg/dL and 2-hour post-prandial blood sugars <120 mg/dL. Women are started on a pharmacologic agent for gestational diabetes if >50% of fasting or post-prandial blood sugars are elevated. Since 2007, glyburide has been the first-line agent at UAB for gestational diabetes. At UAB, glyburide is typically started at 2.5–5 mg/day (in single or divided dosing) and increased by 2.5–5 mg/day. However, use of glyburide and its dosing

was at the discretion of the attending physician; some patients may have been initiated on other pharmacologic agents such as metformin or insulin as the first line.

Subjects were included in this analysis if they were diagnosed with GDM and received glyburide for glycemic control. Subjects were excluded from this analysis if comorbid conditions other than chronic hypertension were present (eg systemic lupus erythematosus, HIV, hepatitis), any fetal anomaly was diagnosed, or if presentation for prenatal care was late (>26 weeks).

Glyburide failure was defined as reaching 20 mg/day of glyburide and then receiving insulin therapy. Glyburide success was defined as those treated with any dose of glyburide without receiving insulin and who had demonstrated glycemic control at >70% of visits. As the median number of visits after starting pharmacotherapy was 6 (interquartile range 4–9); a marker of 70% allowed for 1 visit with poor control and/or medication adjustment. Additionally, at least 3 fasting and 3 post-prandial blood sugars had to be recorded to be considered in the glyburide success group. The group that was neither a glyburide failure or success, i.e. those that were continued on glyburide without receiving insulin and had fewer than 70% of prenatal visits without blood sugar control, were classified as poorly controlled on glyburide and excluded from analysis.

We determined factors that were significantly associated with glyburide failure in a univariate analysis. Only factors known at the time glyburide was initiated were considered in the analysis. Factors considered included maternal age, prepregnancy body mass index, race, education, insurance, prior pregnancy history including a history of GDM in a prior pregnancy, gestational age at diagnosis, gestational weight gain, one-hour and three-hour glucose challenge test results, and blood sugar log records the week prior to starting glyburide. The Liu method[12] was used to identify an optimal cut point for continuous variables.

Groups were compared using Student’s t-test or chi-square test as appropriate. Clinically relevant covariates for initial inclusion in multivariable models were selected using the results of the stratified analyses, and factors were removed in backwards stepwise fashion, based on significant changes in the exposure adjusted odds ratio or significant differences between hierarchical models using the likelihood ratio test. The cutoff point was selected to minimize false-positive diagnoses; in other words, specificity for correctly identifying glyburide failure was more important than sensitivity for identifying glyburide failure. Receiver operator characteristics curves were created to estimate the predictive value of the final model. The internal validity of the model was then assessed using bootstrapping over 5,000 replications. All analyses were performed using STATA SE, version 13.0 (College Station, TX).

Results

Of 1,212 women identified with GDM, 145 were excluded for being unable to confirm a diagnosis of GDM with laboratory testing, 82 for major medical comorbidities, 29 for fetal anomalies, 61 for late prenatal care and 493 for being managed by diet therapy alone,

leaving 402 subjects for analysis. Of these 402subjects, only 39 were initiated directly on insulin (for suspicion of pregestation diabetes) and 3 on metformin, leaving 360 subjects with A2 GDM initiated on glyburide after diet failure (Figure 1). Of the 360 subjects, 63 (17.5%) were classified as glyburide failure and 157 (43.6%) as glyburide success. The remaining 140 (38.9%) were managed on glyburide alone without meeting the success criteria of >70% of prenatal visits with blood sugar control.

Those successfully managed on glyburide and those failing glyburide were similar with regards to maternal age, race, nulliparity, chronic hypertension, prepregnancy body mass index, prior cesarean, and prior macrosomic infant (Table 1). Women who failed glyburide therapy were less likely to have public health insurance and more likely to smoke or to have had GDM in a prior pregnancy. With regards to pregnancy and GDM diagnosis, women who failed glyburide therapy were diagnosed at an earlier gestational age, had higher 1-hour glucose challenge test values, higher fasting and one-hour values on the three-hour glucose tolerance test (Table 2). On blood sugar logs on the week prior to initiating glyburide, women who failed glyburide had more fasting blood sugars >100 mg/dL, more post-prandial values >120 mg/dL, and more blood sugars >200 mg/dL. Gestational weight gain until initiation of glyburide was similar between groups.

We then built a multivariate model to predict glyburide failure versus glyburide success. Factors significant in the final model are demonstrated in Table 3 and Box 1 and include GDM in a prior pregnancy, gestational age at diagnosis ≤26 weeks, a 1 hour glucose challenge test value ≥228 mg/dL, a 1-hour value on the three hour glucose tolerance test ≥221, 7 or more 2-hour postprandial blood sugars >120 mg/dL on blood sugar log for the week prior, 1 or more blood sugar >200 mg/dL on blood sugar log for the week prior. This model classified women as glyburide failure if the calculated risk of failure based on the equation in Box 1 was ≥75%. As each factor was added to the model, the percentage of women correctly classified increased, as did the sensitivity and specificity. The final model correctly classified 80.9% of women, with a sensitivity of 39.7% and a specificity of 97.5%. The area under the curve for this model was 0.86 (Figure 2). Over 5,000 iterations, the sensitivity of the model ranged from 32.3–40.3% and the specificity ranged from 97.4–99.4%. The area under the curve for the model over 5,000 iterations ranged from 0.86–0.87, demonstrating that this model is robust.

Because early gestational age at diagnosis and a 1 hour glucose challenge test value >200 mg/dL are frequently considered markers of pregestational diabetes, we re-evaluated the prediction model excluding subjects with either of these markers. Of the 98 subjects remaining, 13 failed glyburide therapy and 85 achieved good glucose control on glyburide. The model predicts glyburide failure with 30.8% sensitivity and 98.8% specificity and an area under the curve of 0.87.

When this model was applied to all 360 women started on glyburide (including the 140 women in the poorly controlled on glyburide category), 40 (11%) women were predicted to have a glyburide failure. Of these 40, 25 (62.5%) failed glyburide and 4 (10%) were poorly controlled on glyburide. If this model were applied prospectively, only 11 (3.1%) women would have been falsely predicted as glyburide failure.

Discussion

The majority of women who will reach a maximum dose of glyburide without achieving glycemic control can be identified at the time glyburide is initiated. We developed a prediction model with a specificity of 97.5% for those who will fail glyburide therapy and require insulin therapy. This prediction model classified only 11% of our subjects as glyburide failure with a low false positive rate.

The trade-off of the model having a high specificity is a relatively low sensitivity of only 40%. However, we feel that a sensitivity of 40% of women who will fail glyburide being identified by the model is a vast improvement over initiating a patient on glyburide and waiting several weeks to determine success or failure of treatment. Additionally, we purposely elected to build a model with an emphasis on specificity for several reasons. First, patients prefer glyburide to insulin as it is easier and less painful. Secondly, glyburide is significantly less expensive than insulin and does not require additional visits for diabetic education. Therefore, we felt a model that is more specific (fewer predictions of glyburide failure when glyburide may succeed) was preferable to a model that was more sensitive.

Several factors included in the model may actually be associated factors associated with pregestational diabetes, such as extremely elevated fasting values, a one hour glucose challenge test >200 mg/dL, a prior pregnancy complicated by gestational diabetes, or an early gestational age at diagnosis. However, because these women were diagnosed during pregnancy and had no known history of diabetes outside of pregnancy, we included them in the cohort. As many women first diagnosed during pregnancy may be considered for a trial of oral medications, particularly as they are unlikely to be familiar with insulin, this is representative of clinical practice. Indeed, these patients are those who will most likely benefit from aggressive treatment of blood sugars by direct initiation on insulin without a trial of glyburide. Additionally, when we applied the model to a cohort of women excluding markers of pregestational diabetes, the model continued to perform robustly, with a specificity of 98.8%.

Several prior studies have examined risk factors for requiring insulin therapy for glycemic control in GDM. In a prospective study, Chmait et al determined that the the most important markers of glyburide failure were gestational age (<30 weeks) and mean blood glucose values (fasting blood sugar >110 mg/dL, 1-hour post-prandial values >140 mg/dL) at initiation of glyburide.[8] However, the chosen criteria in this study were only 65% specific for identifying glyburide success or failure. This would lead to a falsely identifying glyburide failure in almost 1/3 of patients in the population, potentially using the more expensive and more cumbersome insulin in women who could achieve glycemic control with glyburide. Another study to identify predictors glyburide failure versus glyburide success confirm the importance of gestational age at glyburide initiation, but found that gestational at diagnosis prior to 25 weeks (as opposed to 30 weeks) was associated with an 8-fold increase in the risk of glyburide failure.[9] Other important predictors were maternal age and multiparity. Rochon et al also sought to identify factors predictive of glyburide failure.[10] In this study, glyburide failure was defined as anybody who was started on glyburide and switched to insulin, not just women who failed to achieve glycemic control on

a maximum dose of glyburide and switched to insulin. Unlike other studies, gestational age at diagnosis was not significantly associated with glyburide failure. Factors associated with glyburide failure included a glucose challenge test value greater than 200 mg/dL and a fasting blood sugar value greater than 95 mg/dL.

Our study has several strengths. First, we had a large number (n=63) of women who failed glyburide therapy. This is approximately 3–5 times larger than other studies we identified in the literature of women who failed glyburide therapy. This enabled us to investigate multiple factors involved in glyburide failure simultaneously. Additionally, we had detailed patient level data that included GDM testing results and blood sugar logs for the entire pregnancy. This detailed data enabled us to consider a multitude of factors that may be important in determining glyburide failure, including blood sugar values the week prior to glyburide initiation. Furthermore, we considered only factors known at the time glyburide was initiated in the prediction model. Although information obtained later in pregnancy may also be associated with glyburide failure, this information is not available to clinicians at the time of the decision to initiate glyburide or insulin; consequently, it does not belong in a clinically useful prediction model. We used an empiric method (the Liu method)[12] to define optimal thresholds of continuous variables to use in our prediction model, rather than outcomes considered clinically important traditionally (i.e. a glucose challenge test of 200 mg/dL or gestational age prior to 20 weeks). Consequently, we optimized the sensitivity and specificity of our model. Finally, a stringent definition of glyburide failure (i.e. reaching a dose of 20 mg/day of glyburide and initiation on insulin) and glyburide success (i.e. >70% of visits with glycemic control) was used, limiting misclassification bias due to provider judgment or patient preferences for glyburide versus insulin.

One potential weakness of this study is that we excluded subjects who were poorly controlled on glyburide when developing our prediction model, thus excluding 39% of the cohort. We did this for several reasons. First, the goal of initiating glyburide is to achieve glycemic control. Thus, the ideal comparison group to failing glyburide therapy is a group of women who actually achieved glycemic control with glyburide. Also, in a retrospective study, it is unclear if the excluded group represents glyburide success or failure. Although we collected significant amounts of detailed patient-level information (including weekly blood sugar logs and medication doses), we did not collect data on information such as patient refusal of insulin or patient's access to medication. Therefore, some may have had improved blood sugar control with simply higher doses of glyburide, others may have actually been recommended to start insulin but refused or delivered before it could be initiated. Therefore, exclusion of this group likely avoided some misclassification bias. When this group of women was included and the model applied, the model continued to perform well with a low positive rate. Finally, although our clinical practice typically gives every patient a trial of glyburide, some patients (less than 10%) were initiated on insulin without first receiving glyburide therapy. Their exclusion may have impacted the model by excluding women most likely to fail glyburide therapy, although this may be preferred as it mimics what happens in clinical practice. Women who were initiated directly on insulin without a trial of glyburide were more likely to have had a prior pregnancy complicated by GDM than those given a trial of glyburide, but were otherwise similar with respect to age,

prepregnancy BMI, and race (data available upon request). Therefore, we do not feel that their exclusion significantly altered our results.

In sum, we have developed a prediction model based on factors known at the time of glyburide initiation to predict those who will ultimately fail glyburide therapy and require insulin. This model has excellent specificity in order to avoid initiating insulin in a patient who could achieve glycemic control with glyburide therapy. Application of this model prospectively should be investigated to determine if starting women at high risk of glyburide failure directly on insulin can improve perinatal outcomes.

Acknowledgments

Dr. Harper is supported by K12HD001258-13, PI WW Andrews, which partially supports this work.

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Box 1

Logistic Regression Equation for Prediction of Glyburide Failure

$$\text{Predicted probability of glyburide failure} = \frac{exp\,(w)}{1+exp\,(w)} \text{ where}$$

where w=−3.864063 +1.589964*(GDM in a Prior Pregnancy) + 0.6698857*(Gestational Age at Diagnosis ≤26 weeks) +1.059195*(Glucose Challenge Test ≥228 mg/dL)
+ 1.1380658* (on 3-hour Glucose tolerance test, a 1-hour value ≥221 mg/dL)
+ 1.471988*(≥7 Post-Prandial Blood Sugars >120 on Log for the Week Prior)
+ 1.312223*(≥1 Blood Sugars >200 on Log for the Week Prior)

For each variable, replace variable name with 1 if “yes” or 0 if “no”

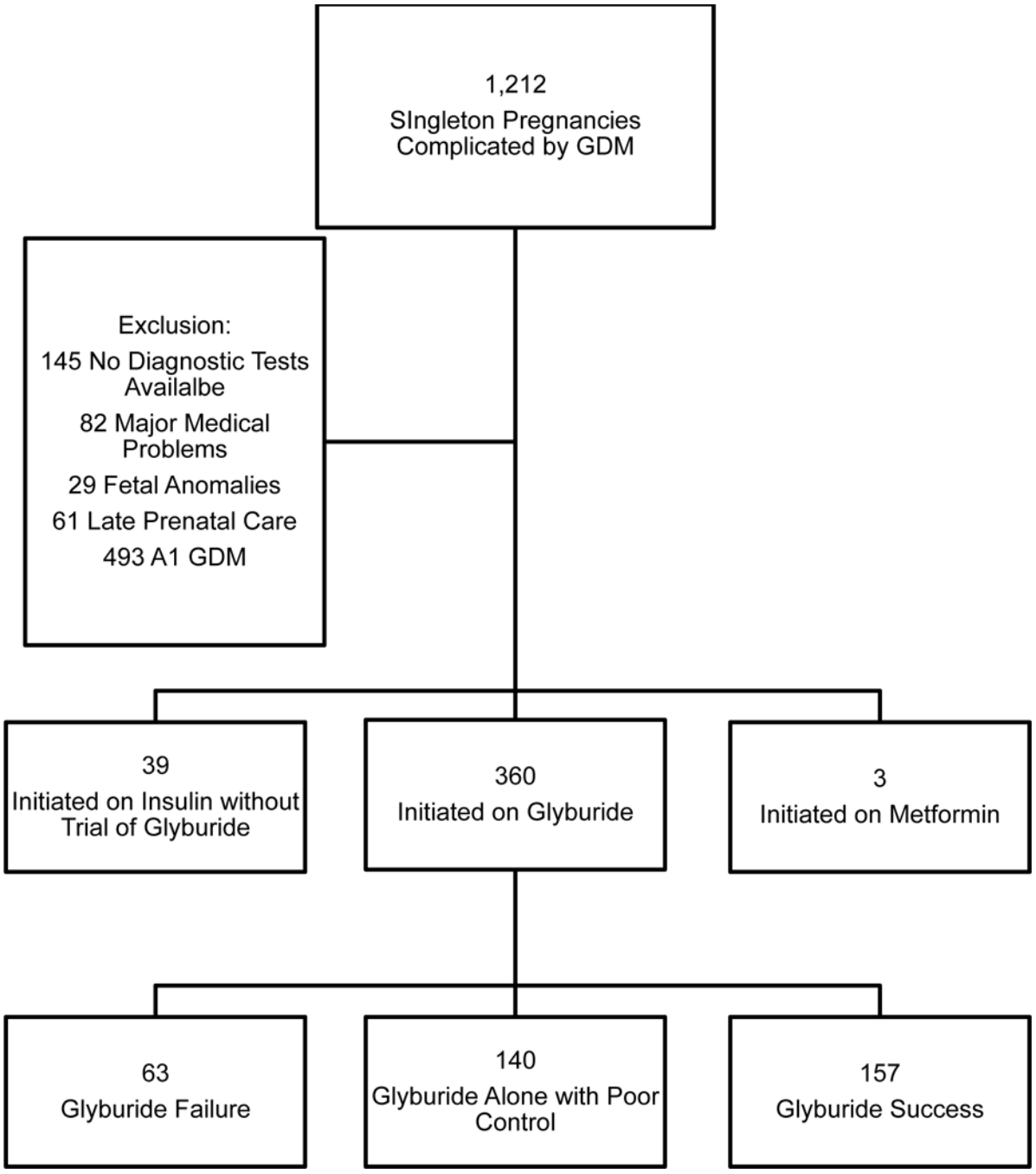

Figure 1.
Flow Diagram of Patients in Cohort

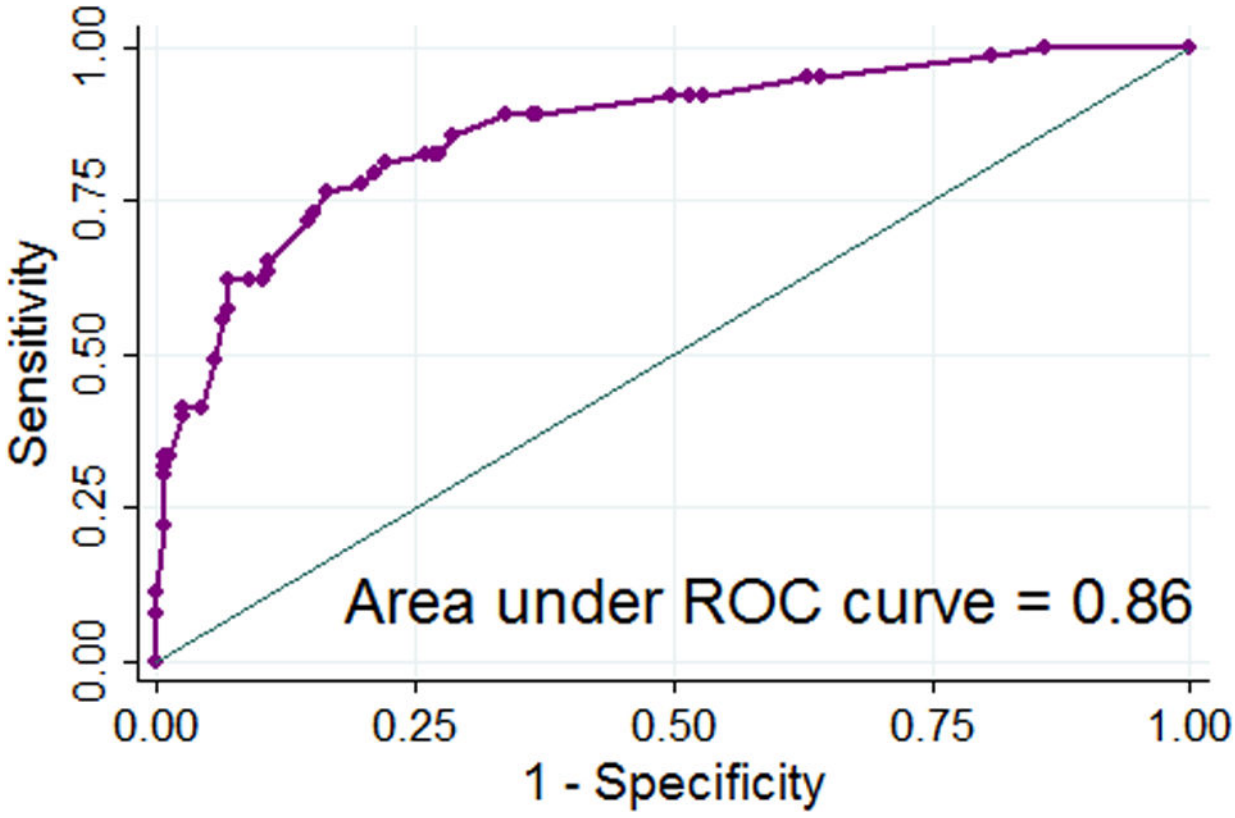

Figure 2.
Receiver Operator Characteristics Curve for Prediction Model of Glyburide Failure

Table 1

Maternal Characteristics Associated with Glyburide Failure

	Glyburide Successful	Glyburide Failure	p
	N=157	**N=63**	
Maternal Age (years)	29.7 ± 5.8	30.1 ± 5.1	0.51
Race			0.38
Black	80 (55.6%)	33 (52.4%)	
White	15 (10.4%)	12 (19.0%)	
Hispanic	47 (32.6%)	16 (25.4%)	
Other	2 (1.4%)	2 (3.2%)	
Nulliparity	47 (29.9%)	14 (22.2%)	0.25
Chronic Hypertension	23 (14.7%)	11 (17.5%)	0.6
Prepregnancy BMI (kg/m^2)	35.0 ± 8.6	35.4 ± 8.2	0.78
Obese (Unknown BMI in 7 subjects)	115/153 (75.2%)	44/60 (73.3%)	0.78
Tobacco Use	17 (10.8%)	15 (23.8%)	0.01
Public Health Insurance	125 (79.6%)	40 (63.5%)	<0.01
Prior Gestational Diabetes	22 (14.0%)	30 (47.6%)	<0.01
Prior Macrosomic Infant	21 (13.4%)	7 (11.1%)	0.65
Prior Cesarean	41 (26.1%)	19 (30.2%)	0.54
Number of Visits after Glyburide Initiation	6 (4–8)	9 (7–14)	<0.01

Data presented as mean ± standard deviation or n(%)

Table 2

Pregnancy Characteristics Associated with Glyburide Failure

	Glyburide Successful	Glyburide Failure	p
	N=157	N=63	
Gestational Age at Diagnosis (weeks)	25.7 ±3.7	19.6 ± 7.8	<0.01
Gestational Weight Gain until Glyburide Started	7.4 ± 6.9	6.8 ± 8.8	0.6
1-Hour GCT Level	189 ± 33	228 ± 66	<0.01
1-Hour GCT >200 mg/dL	59 (37.6%)	43 (68.3%)	<0.01
3-Hour GCT Results			
Fasting	106 ± 15	138 ± 52	<0.01
1-Hour	201 ± 28	218 ± 31	0.02
2-Hour	178 ± 28	188 ± 42	0.25
3-Hour	140 ± 27	143 ± 42	0.75
Blood Sugars on Diet			
Number of Fastings >100 mg/dL	2 (1–3)	4 (1–6)	<0.01
Number of Post-Prandial >120 mg/dL	3 (1–6)	8 (5–11)	<0.01
Number of Blood Sugars >200 mg/dL	0 (0–0)	0 (0–1)	<0.01

Data presented as mean ± standard deviation, n(%), or median (interquartile range)

Table 3

Odds Ratios, Sensitivities, and Specificities for Components of Prediction Model

	Odds Ratio (95% CI)	Correctly Classified Cumulative	Sensitivity Cumulative	Specificity Cumulative
GDM in Prior Pregnancy	4.90 (2.0–11.8)	71.40%	0%	100%
Gestational Age at Diagnosis ≤26 weeks	1.95 (0.87–4.38)	71.40%	0%	90.50%
1-Hour Glucose challenge Test ≥228 mg/dL	2.88 (1.29–6.45)	77.70%	28.60%	97.50%
On 3-hour Glucose tolerance test, a 1-hour value ≥221 mg/dL	3.12 (1.21–8.07)	77.70%	27.00%	98.10%
≥7 Post-Prandial Blood Sugars >120 on Log for the Week Prior	4.36 (1.83–10.4)	80.90%	36.50%	98.70%
≥1 Blood Sugars >200 on Log for the Week Prior	3.7 (1.36–10.16)	80.90%	39.70%	97.50%
Final Model		80.90%	39.70%	97.50%

Percentages given for Correctly Classified, Sensitivity, and Specificity are for models built using the variable in the row as well as all rows above. For example, numbers given for gestational age at diagnosis ≤26 weeks include gestational age as well as GDM in prior pregnancy.