

Original article

Development of a risk prediction model for postpartum onset of type 2 diabetes mellitus, following gestational diabetes; the lifestyle InterVention in gestational diabetes (LIVING) study



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SUMMARY

Aims: This study aimed to develop a prediction model for identifying a woman with gestational diabetes mellitus (GDM) at high risk of type 2 diabetes (T2DM) post-birth.

Methods: Utilising data from 1299 women in the Lifestyle Intervention IN Gestational Diabetes (LIVING) study, two models were developed: one for pregnancy and another for postpartum. Key predictors included glucose test results, medical history, and biometric indicators.

Results: Of the initial cohort, 124 women developed T2DM within three years. The study identified seven predictors for the antenatal T2DM risk prediction model and four for the postnatal one. The models demonstrated good to excellent predictive ability, with Area under the ROC Curve (AUC) values of 0.76 (95% CI: 0.72 to 0.80) and 0.85 (95% CI: 0.81 to 0.88) for the antenatal and postnatal models, respectively. Both models underwent rigorous validation, showing minimal optimism in predictive capability. Antenatal model, considering the Youden index optimal cut-off point of 0.096, sensitivity, specificity, and accuracy were measured as 70.97%, 70.81%, and 70.82%, respectively. For the postnatal model, considering the cut-off point 0.086, sensitivity, specificity, and accuracy were measured as 81.40%, 75.60%, and 76.10%, respectively.

Conclusions: These models are effective for predicting T2DM risk in women with GDM, although external validation is recommended before widespread application.

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Table 1 One-page tabular summary of the paper

Key elements	Antenatal model (model 1)	Postnatal model (model 2)
Study population	Pregnant women with gestational diabetes mellitus (GDM)	Pregnant women with GDM
Purpose of the model	To predict the risk of type 2 diabetes (T2DM)	To predict the risk of (T2DM)
Length of time to predict(t1)	2–3 years after delivery	2–3 years after delivery
Study area	19 urban hospitals located in India, Sri Lanka, and Bangladesh.	19 urban hospitals located in India, Sri Lanka, and Bangladesh.
Type of data	Prospectively collected data	Prospectively collected data
Samples size (total/event)	1299/124	1299/124
Missing data handling	Multiple imputation	Multiple imputation
Predictor selection	Statistical results, Clinicians consultation and experience, Literature search	Statistical results, Clinicians consultation and experience, Literature search
Statistical analysis	Logistic regression by R	Logistic regression by R
Variables included in the model	1. Antenatal Fasting Plasma Glucose (FPG) level 2. Antenatal 2-hour-Oral Glucose Tolerance test (2h - OGTT) 3. History of recurrent GDM 4. GDM Insulin treatment 5. Parity 6. History of the irregular menstrual cycle 7. Family history of diabetes mellitus	1. Antenatal 2h- OGTT 2. Postnatal FPG level 3. Postnatal 2h-OGTT 4. Postnatal Body mass index (BMI)
Coefficients of the variables	Risk of T2DM = $-10.0757 + 0.7086$ antenatal FPG + 0.3656 antenatal 2h-OGTT + 0.3190 history of recurrent GDM + 0.5100 Insulin treatment during pregnancy + 0.3526 parity + 1.0922 history of irregular menstrual cycle + 0.0972 family history of diabetes mellitus	Risk of T2DM = $-15.3625 + 0.3008$ Antenatal 2h-OGTT + 1.0033 Postnatal FPG + 0.5581 Postnatal 2h-OGTT + 0.0359 post-natal BMI
Area under the curve (AUC)	0.76 (95% CI: 0.72 to 0.80)	0.85 (95% CI: 0.81 to 0.88)
Mode of internal validation	Cross-validation and bootstrapping	Cross-validation and bootstrapping
Optimism corrected AUC during cross-validation	75.29	84.40
Optimism corrected AUC during bootstrapping	75.08	84.33
AUC optimism	0.0187–0.0219	0.0074–0.0089
Brier Score	0.078	0.07
Optimism corrected Calibration slope	0.9491–0.9432	0.9790–0.9816
Optimal cut-off point (Sensitivity, Specificity, Accuracy)	0.096 (70.97, 70.81, 70.82)	0.086 (81.4, 75.6, 76.1)
Mode of model presentation	Coefficients	Coefficients

1. Introduction

Gestational Diabetes Mellitus (GDM) is the most common pregnancy metabolic complication worldwide, affecting an estimated 18 million women annually [1]. Globally, GDM affects 15–25% of pregnant women [2] and is a significant cause of maternal and child mortality [3]. The burden is higher in South Asian communities, of which more than 25% of pregnancies have GDM [1]. The prevalence of GDM is increasing globally due to factors including changes in diagnostic criteria, lifestyle, and rising obesity [4].

GDM increases the risk of long-term postnatal complications. According to recent reports, 12.3%–60% of pregnant women with GDM will develop one form of glucose intolerance within early to 15 years postnatal, varying in different populations [5,6]. This risk increases to 70% 28 years after pregnancy [7,8]. A meta-analysis in 2018 showed that women with GDM have a greater than seven-fold risk of developing postnatal glucose intolerance at any time after childbirth compared to those who did not develop GDM in pregnancy [9,10].

For postpartum type 2 diabetes mellitus (T2DM) screening, although fasting plasma glucose level (FPG) and Haemoglobin A1c (HbA1c) are suggested by some guidelines, an oral glucose tolerance test (OGTT) remains the gold standard in most guidelines and countries at 4–12 weeks postnatal to facilitate early intervention [11–13]. Although primarily used to establish a diagnosis of T2DM or other glucose abnormalities, the OGTT alone cannot forecast

future risks. In addition, despite numerous efforts [14], a low uptake of postnatal T2DM screening among women who had GDM has been reported across many regions [15] due to various factors, including fear of diabetes diagnosis, low health prioritisation, lack of clinician guidance, and inefficient screening systems [16,17]. Therefore, it is vital to incorporate antenatal information into a predictive model to assess postnatal T2DM risk. This approach would assist in initiating early preventive measures for women starting from the pregnancy phase.

Existing models for predicting postnatal glucose intolerance, including T2DM, have been limited by inadequate sample sizes, ambiguous application of inclusion and exclusion criteria, absence of reporting and/or managing missing data, improper handling of continuous and categorical variables, and the employment of univariable analysis to select predictors [18]. Furthermore, they failed to evaluate or disclose relevant model performance metrics, neglected to account for model overfitting and unwarranted optimism in model performance, and did not address the need for internal and external validation. Indeed, greater compliance with standard reporting guidelines would better understand the high risk of bias inherent in these models [19,20].

This study aimed to develop two robust models for the prediction of postnatal T2DM among women with prior GDM in South Asian populations (India, Sri Lanka, and Bangladesh): one model will use only antenatal predictors, and the second model will include a mix of antenatal and postnatal predictors. The Lifestyle Intervention in Gestational Diabetes (LIVING) study had Clinical

Trials Registry of India Identifier CTRI/2017/06/008744, Sri Lanka Clinical Trials Registry Identifier SLCTR/2017/001, and [ClinicalTrials.gov](https://www.clinicaltrials.gov) Identifier NCT03305939.

2. Methods

2.1. Study population

The predictive model was developed using prospectively collected data from the LIVING study, a randomised controlled trial that offered a lifestyle intervention program to prevent T2DM or prediabetes to women who had previously experienced GDM [21]. The detailed methods for this study have been previously reported [21,22]. The study included women diagnosed with GDM between the 24th and 34th weeks of their pregnancies across 19 urban hospitals in India, Sri Lanka, and Bangladesh. These diagnoses were determined using an OGTT based on the criteria set by the International Association of Diabetes and Pregnancy Study Groups (IADPSG). According to the IADPSG guidelines, a diagnosis of GDM can be made using a 2-h, 75-g OGTT if any of the following results are observed: a fasting glucose level (FPG) of ≥ 5.1 mmol/L (92 mg/dl), a reading of ≥ 10.0 mmol/L (180 mg/dl) at the 1-h mark, or a 2-h reading of ≥ 8.5 mmol/L (153 mg/dl) [23]. Considering the variance in GDM screening procedures across different centers, some of which conduct tests before the 24-week mark, additional criteria were also introduced. After delivery, these individuals underwent a subsequent OGTT. The study excluded individuals with over 2 hours of travel time to the hospital, those without a home-based mobile phone, individuals who used steroids during pregnancy (except for the advancement of fetal lung maturation), and those anticipated to move residence within the next three years.

Since the effect of lifestyle intervention on the worsening of glycemic status (25.5% vs 27.1%; hazard ratio, 0.92 [95% CI, 0.76–1.12]; $P = 0.42$) [22], and secondary outcomes, including weight, were not significantly different in intervention and control groups, we used both groups for prediction model development using predictors collected during pregnancy and in the postnatal period. The sensitivity analysis of how the intervention impacts primary and secondary outcomes, compared to the usual care group, is detailed in the primary reports of the study [22]. The Transparent Reporting of a Multivariable Prediction model for Individual Prognosis or Diagnosis (TRIPOD) checklist was followed [24].

2.2. Outcome

The primary outcome of interest was the onset of T2DM, as defined by the American Diabetes Association (ADA), over a postnatal period of approximately three years. The diagnosis was confirmed using an OGTT for nearly 98% of participants. If the OGTT was unavailable, the diagnosis was made based on the FPG. If that, too, was unavailable, the HbA1c level was utilised. Based on ADA criteria [25], individuals were classified as T2DM if they had an FPG ≥ 126 mg/dl (≥ 7.0 mmol/L), 2h-OGTT ≥ 200 mg/dl (≥ 11.0 mmol/L), or an HbA1c level $\geq 6.5\%$.

2.3. Predictors

A comprehensive list of demographic details, co-existing medical conditions, family medical history, vital signs recorded at study registration postpartum, and anthropometric measurements predictors considered for inclusion in the prediction model is presented in the supplementary file. Candidate predictors were selected a priori based on our systematic review [18], statistical strength of association with the outcome variable, and clinical

relevance based on consultation with clinicians. Obstetricians, gynecologists, and endocrinologists reviewed all significant and non-significant variables for iterative discussion and input regarding the variables' clinical relevance and practical applicability.

2.4. Sample size determination

With 1299 participants patients and 124 events (T2DM post-birth), the LIVING study provided a sufficient sample size to develop a prediction model with approximately 30 predictors. We performed the sample size calculation for model development using the 'pmsampsize' package [26], which is grounded on criteria suggested by Riley et al. [27–29]. Since the primary outcome is binary, the type was set as "b". With about 30 candidate predictor parameters proposed for the new model and a c-statistic of 0.91 from the existing study [30], the T2DM prevalence was taken as 9.5% [22]. Consequently, using the command "pmsampsize (type = "b", c-statistic = 0.91, parameters = 30, prevalence = 0.095)" and presuming an acceptable difference of 0.05 in apparent and adjusted R-squared, as well as a margin of error of 0.05 in intercept estimation, the minimum sample size required for new model development based on these inputs was 1122. It includes 107 events (presuming an outcome prevalence = 0.095 and an event per predictor (EPP) = 107/30 (3.56)). This demonstrates that the sample size used for our prediction model development was sufficiently robust for the development process.

2.5. Data cleaning and pre-processing

The existence and patterns of missing values were thoroughly evaluated using the "VIM" package in R [31]. This package provides a robust method for visually inspecting missing and imputed values in the data. Working under the assumption that the data were 'missing at random,' we utilised the Multiple Imputation by Chained Equations (MICE) for multiple imputations. The imputation model incorporated 1h-OGTT, 2h-OGTT, and HbA1c during this process. A total of 10 imputed datasets were created. Subsequently, a prediction model was built for each of these imputed datasets. The results from these models were then combined using Rubin's rules to produce a single, cohesive prediction model.

2.6. Statistical analysis

All analyses were conducted using R programming language for statistical computing and graphics version 4.2.3. Statistically, using the "glmnet" package in R, least absolute shrinkage and selection operator (Lasso) logistic regression was applied to determine the strength of association with the outcome and to select relevant variables. Subsequently, a multivariate logistic regression model was developed by considering the results of the Lasso regression model and other aforementioned selection criteria.

2.7. Internal validation

After developing the prediction model, 10-fold cross-validation and 1000 times bootstrapping were conducted to assess the prediction performance. The discrimination and calibration of the final model were assessed by calculating performance measurement metrics such as Area under the Receiver Operating Characteristic (ROC) Curve (AUC), sensitivity, specificity, and Brier score. Calibration metrics and calibration plots of observed and predicted probabilities of postnatal T2DM were generated. Decision curve analysis and clinical impact analysis were conducted to explore the net benefit of the models.

3. Results

3.1. Baseline characteristics

This study included 1299 participants, with 124 developing T2DM by the end of the follow-up period. The average age of participants was 30.1 years, with a standard deviation (SD) of 5.0 years. Of the participants, 39.6% had an education beyond secondary school, and 18.8% were employed. Previous instances of GDM, not considering the index delivery, were reported by 7.4%. The prevalence of family history of diabetes mellitus was 48.0%, hypertension 37.5%, and cardiovascular disease 15.3%, while personal medical histories revealed 9.6% had hypertension, 0.8% had heart disease, and 0.9% had stroke. Fifty percent of women had two pregnancies, and GDM was diagnosed on average at 26.6 weeks (SD of 5.6 weeks) of pregnancy. One hundred sixty-three (12.5%) women were diagnosed with GDM before the 24th week of gestation and were prescribed medication, and 12.2% of women required insulin to manage GDM. When considering body mass index (BMI), 21.8% were classified as obese (BMI ≥ 30 kg/m²), while 40.0% were in the overweight category (BMI 25–29.9 kg/m²). A history of irregular menstrual cycles was reported in 22.2% of women (Table 2).

3.2. Model development

In simple regression, most of the associations were either significant (p-value <0.05) or marginally significant (p-value = 0.06). After using Lasso, the chosen antenatal factors were 2h-OGTT level, FPG, the need for insulin treatment due to GDM, and a history of irregular menstrual cycles. The selected postnatal factors included 2h-OGTT, FPG, and HbA1c. Based on the results of lasso and clinical relevance assessment, finally, we included seven predictors for model 1 (antenatal FPG, antenatal 2h-OGTT, history of recurrent GDM, insulin treatment during pregnancy, parity, history of irregular menstrual cycle, and family history of diabetes mellitus) and four predictors for model 2 development (antenatal 2h-OGTT, postnatal 2h-OGTT, postnatal FPG, and BMI).

Including the LIVING trial intervention status into the models was done in sensitivity analyses, and this variable provided no changes to the model performances (see supplemental file).

3.3. Performance of the prediction model

The AUC of the ROC curve for model 1 was 0.76 (95% CI: 0.72 to 0.80) (Fig. 1). Employing the optimal cut-off point based on the Youden Index (0.096), model 1 exhibited a sensitivity of 70.97%, a specificity of 70.81%, and an accuracy of 70.8%. The model calibration, depicted in Figure 2, was excellent, with a mean absolute error of 0.012 and a mean squared error of 0.00036. The Brier score was 0.07.

Model 2 produced an AUC of 0.85 (95% CI: 0.81 to 0.88), and taking 0.086 as an optimal cut-off point, it showed a sensitivity of 81.4%, specificity of 75.6% and accuracy of 76.1% (Fig. 1). It also had an excellent calibration, as shown in the calibration plot in Fig. 2, with a Brier score of 0.07.

The tenfold cross-validation internal validation process showed minimal optimism in AUC in both models: 0.01 for model 1 and 0.07 for model 2, indicating minimal model overfitting. Moreover, during the 1000 bootstrap internal validation rounds, the model revealed only slight optimism in AUC 0.02 for model 1 and 0.008 for model 2, further emphasising the lack of overfitting. The decision curve analysis, as illustrated in Fig. 3, further validates the superior clinical utility of our developed model when compared to the ‘treat all’ and ‘treat none’ decision options. Similarly, the clinical impact

Table 2
Sociodemographic status and characteristics of study participants.

	Total N = 1299
Age, years (mean, SD)	30.1 (5.0)
Religion (n, %)	
Buddhist	218 (16.5)
Christian	133 (10.1)
Hindu	541 (41.0)
Muslim	391 (29.7)
Other	5 (0.4)
Sikh	30 (2.3)
Education (n, %)	
Secondary school or below	795 (60.3)
Higher than secondary school	523 (39.7)
Employment (n, %)	
Unemployed	1070 (81.2)
Employed	248 (18.8)
Graida (median, IQR)	2.0 (1.0–3.0)
Prior history of GDM (n, %)	
No	1, 220 (92.6)
Yes	98 (7.3)
GDM requiring insulin treatment (n, %)	
No	1140 (87.8)
Yes	159 (12.2)
Family history of diabetes in first degree relatives (n, %)	
No	688 (52.0)
Yes	630 (48.0)
Family history of hypertension (n, %)	
No	824 (62.5)
Yes	494 (37.5)
Body weight, kg (mean, SD)	63.4 (11.9)
Body mass index, kg/m² (mean, SD)	26.6 (4.7)
Body mass index classification (n, %)	
Underweight	36 (2.7)
Healthy weight	468 (35.4)
Overweight	527 (40.0)
Obesity	287 (21.8)
History of irregular menstrual cycle (Yes, %)	289 (22.2)
Waist circumference, cm (mean, SD)	89.4 (11.7)
Systolic blood pressure, mmHg (mean, SD)	112.6 (11.2)
Diastolic blood pressure, mmHg (mean, SD)	74.5 (9.0)
Fasting plasma glucose during pregnancy, mg/dl (mean, SD)	5.4 (0.6)
Glucose 1 h post OGTT during pregnancy, mg/dl (mean, SD)	9.9 (1.7)
Glucose 2 h post OGTT during pregnancy, mg/d (mean, SD)	8.1 (1.6)

BMI classification: underweight (<18.5kg/m²); normal weight (≥ 18.5 to < 25 kg/m²); overweight (≥ 25.0 to < 30 kg/m²); obese (>30.0kg/m²); SD: Standard Deviation, IQR: Interquartile Range, GDM: Gestational Diabetes Mellitus.

analysis emphasises that both models have a better clinical impact (supplemental file).

4. Discussion

We have developed robust prediction models for T2DM among women with a history of GDM using a multicenter dataset from high-risk South Asian women. Our models can be applied either during pregnancy or immediately after delivery. By using glucose levels and additional maternal characteristics collected during and immediately post-childbirth, our models can effectively identify women at high risk of developing T2DM. The two models include the antenatal model, which includes seven available antenatal predictors and achieves good performance (AUC = 0.76, Brier score = 0.07), and the postnatal model, which includes a mix of four antenatal and postnatal variables and achieves excellent predictive performance (AUC = 0.85, Brier score = 0.07).

Antenatal and postnatal blood glucose levels, insulin treatment for GDM, parity, a history of irregular menstrual cycles, recurrent GDM, a family history of diabetes mellitus, and postnatal BMI were selected as potential predictors of postnatal T2DM. This is appropriate given the contribution/relationship between these factors

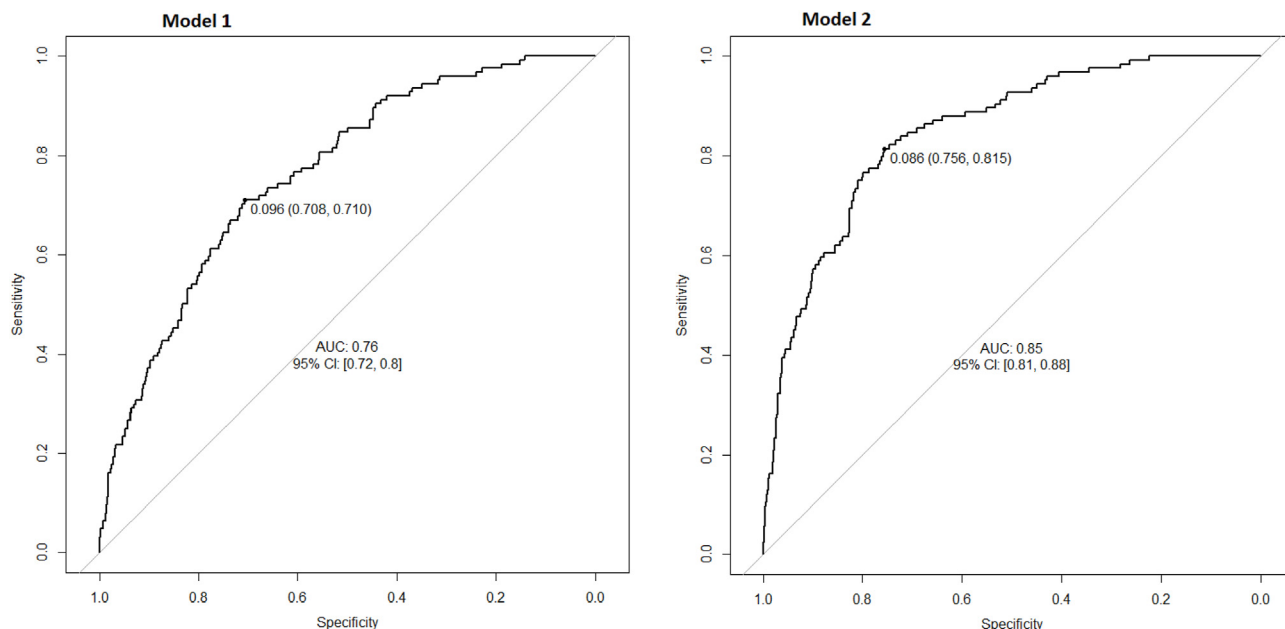


Fig. 1. Discrimination of the two antenatal and postnatal models Represented by the Area Under the Receiver Operating Characteristic Curve. **model 1** (during pregnancy): “Risk of T2DM after 2–3 years = $-10.0757 + 0.7086$ antenatal FPG + 0.3656 antenatal 2h-OGTT + 0.3190 history of recurrent GDM + 0.5100 Insulin treatment during pregnancy + 0.3526 parity + 1.0922 history of irregular menstrual cycle + 0.0972 family history of diabetes mellitus”, **model 2** (after delivery): “Risk of T2DM after 2–3 years = $-15.3625 + 0.3008$ pregnancy 2h-OGTT + 1.0033 postnatal FPG + 0.5581 postnatal 2h-OGTT + 0.0359 BMI”.

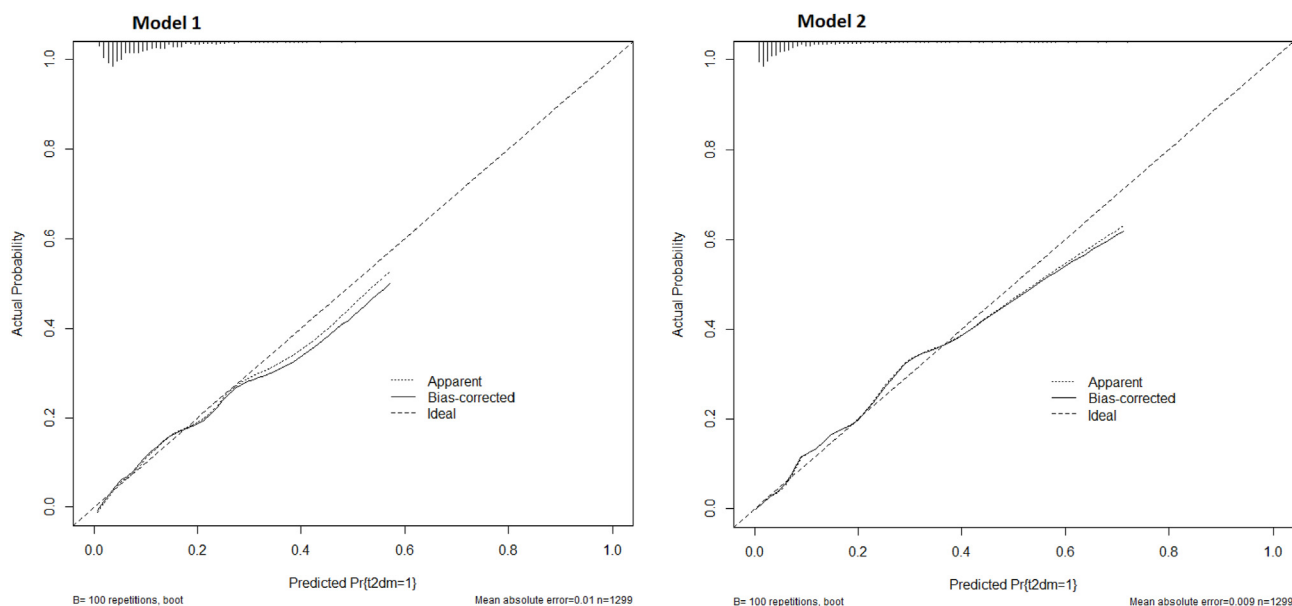


Fig. 2. Calibration plots of the antenatal and postnatal models. Both Model 1 and Model 2 exhibit good calibration, closely following the diagonal line. However, there is a slight overconfidence in Model 1 beginning at predicted probabilities of 0.3 and in Model 2 starting at 0.4.

and the known pathophysiology of T2DM. Blood glucose levels, including FPG and 2h-OGTT, are crucial glycemic control and insulin resistance markers. Elevated FPG may indicate potential beta-cell dysfunction and a higher risk of diabetes onset [32]. The 2h-OGTT measures how effectively the body processes glucose after intake. Elevated OGTT values suggest insulin resistance and may precede T2DM [33,34]. A history of recurrent GDM can also signal underlying metabolic challenges and an increased risk of glucose intolerance either due to ongoing issues with insulin function or production [35]. Furthermore, when GDM requires insulin

treatment, it may signify greater glucose intolerance or worse beta-cell function [36]. When considering parity, the number of times a woman has given birth may correlate with older age or metabolic shifts. Indeed, a study of 2552 women from Tehran suggested that higher parity was associated with T2DM after adjustment for a range of potential risk factors [37]. A family history of diabetes suggests a genetic vulnerability to T2DM, pointing to potential inherited issues with insulin resistance or beta-cell function. Genetics significantly influence T2DM risks, with specific genetic traits related to insulin and glucose metabolism inherited across

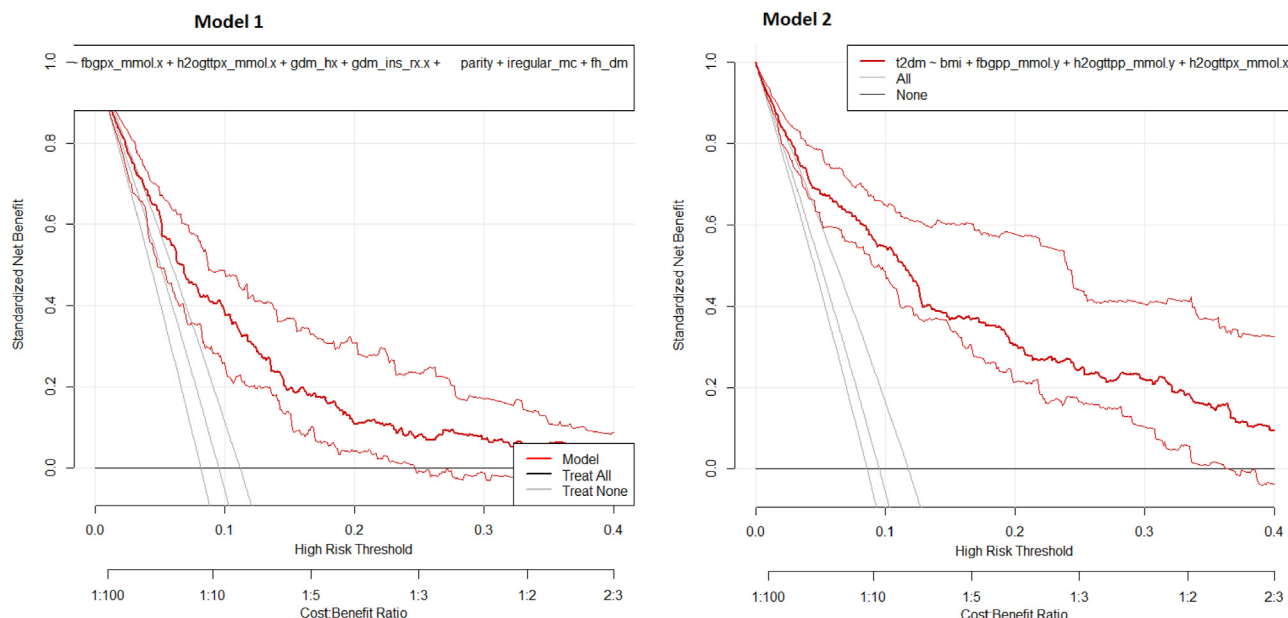


Fig. 3. Decision curve analysis of the antenatal and postnatal models. The Decision curve shows the clinical “net benefit” when comparing the outcomes of treating all versus treating none. By utilizing a model, it identifies the span of probabilities where a particular model offers the most value for clinical choices. As a result, both models present a greater net advantage over the alternative approaches of treating all or treating none.

generations [38,39]. The link between obesity and T2DM is complex, stemming from changes in β cell function due to increased adiposity, variations in adipose tissue function, and insulin resistance [40].

The first model developed used a combination of seven of the aforementioned variables: antenatal FPG, antenatal 2h-OGTT level, insulin treatment for GDM, parity, a history of irregular menstrual cycles, a history of recurrent GDM, and a family history of diabetes mellitus. While most of the variables included in our model have been utilised in previously reported prediction models for T2DM, it was not expected to see a history of irregular menstrual cycles included. The AUC for model 1 was 0.76 (95% CI 0.72, 0.80). However, when the history of irregular menstrual cycles was excluded, the predictive capability dropped to an AUC of 0.73 (95% CI 0.68, 0.77). This underscores the significance of including the history of irregular menstrual cycles in predicting T2DM. Prior research has reported a strong association between irregular menstrual cycles and increased risk of GDM and T2DM [41,42], possibly because irregular menstrual cycles are a feature of polycystic ovary syndrome (PCOS). Nevertheless, the frequency of collecting and reporting data on irregular menstrual cycles or associated PCOS in women with GDM needs to be clarified. Future research and care should prioritise assessment of this history during pregnancy in order to improve the prediction of T2DM.

Many women with a history of GDM often skip postpartum T2DM screening. Lack of awareness and personal factors such as limited education and low self-confidence may contribute to this trend [16]. Moreover, the absence of recommendations or referrals from clinicians for screening, inadequate emphasis on risk communication, inappropriate testing facilities or screening methods, and the lack of an integrated reminder system and procedures for documenting and sharing GDM history further hamper adequate postnatal screening [17,43]. Clinical and socio-demographic circumstances, such as increased diabetes risk and perinatal depression, also complicate the situation [44]. Based on antenatal factors, model 1 may be used during pregnancy to identify women at increased risk for developing T2DM

postpartum. Our approach is practical as it utilises historical data and glucose tests taken between 24 and 28 weeks for GDM diagnosis. Its main advantage is in delivering early risk stratification, allowing for prompt preventive measures during pregnancy. This model may also be more applicable in settings with poor uptake of postpartum OGTT screening [45,46]. Thus, aside from increasing awareness efforts to increase postnatal screening for T2DM, this model provides the opportunity to identify women who have GDM who would benefit from preventive approaches to reduce future risk of T2DM.

We also developed additional models with and without including postnatal HbA1c and postnatal 2h-OGTT. Consequently, a model comprising solely three variables: antenatal 2h-OGTT, postnatal FPG level, and postnatal BMI, yielded an AUC of 0.79 (95% CI, 0.75–0.83). Incorporating postnatal HbA1c into this model enhanced its performance, elevating the AUC to 0.82 (95% CI, 0.78–0.85). Substituting postnatal HbA1c with postnatal 2h-OGTT improved the model's predictive performance, with the AUC reaching 0.85 (95% CI, 0.81–0.88). This indicates that postnatal 2h-OGTT is a more effective predictor of future T2DM risk compared to postnatal HbA1c. Therefore, we have developed a prediction model that can be used after delivery using only four readily available variables and with better predictive capability: antenatal 2h-OGTT, postnatal FPG, postnatal 2h-OGTT, and postnatal BMI. Early postnatal stratification of at-risk women using easily accessible predictors will allow further targeted efforts to implement strategies to prevent the future development of diabetes for those at the highest risk. By combining antenatal OGTT results with postnatal BMI measurements, rather than solely relying on postnatal glucose status screening, we can more accurately predict which women are at risk of developing T2DM within the next three years. This comprehensive approach aids women and their physicians in making well-informed decisions and thus optimises current prevention efforts. Identifying high-risk women also helps focus and mobilise finite resources to a smaller population segment in contrast to universal intervention, which may not be feasible in low-resource settings.

Considering the differences in pregnant populations across different settings and countries, the measurement of predictors and outcome variation, and the change of populations and measurements over time, we highly recommend external validation and continuous updating across diverse geographical locations.

4.1. Strengths and limitations of the study

The development of our prediction model, based on readily available antenatal and postnatal characteristics from multi-country and multicenter data, makes it suitable for low-resource settings. However, despite the sample size calculation showing that the sample size was adequate to develop a prediction model based on the predetermined parameters, the relatively small sample size remains a limitation. Indeed, a larger sample size would enhance the prediction model's generalisability and give more precise results. Due to its developmental stage, external validation is advised further to ensure the reliability and generalisability of the results. Two proxy measures of socioeconomic status (education and employment) were collected from the sample. However direct information on household income was sought, but in these samples, most women were not aware of their exact income status, and underreporting of income is common. Information on whether women with GDM had received an oral glucose-lowering agent (metformin or glibenclamide) was sought in the original LIVING study. However, no specific distinction was made between the two medicines, which was a limitation. Furthermore, only 25 participants were recorded as receiving such medicines, which is likely to change in future studies as particularly metformin use has increased. Therefore, we expect metformin will be investigated as a potential predictor in future research.

5. Conclusion

We developed prediction models that can be used during pregnancy or the postnatal period to predict the risk of T2DM among South Asian women with a history of GDM. The model based on pregnancy variables can predict those at the highest risk for dysglycaemia postpartum and may help streamline OGTT follow-up postpartum, given the poor uptake and cost burden. Both models may assist in targeted prevention strategies. However, the developed model should be validated externally to improve predictive performance and to test generalisability.

Author contributions

YB, JE, and HT participated in the conceptual design of the study. YB and JE completed the statistical analysis and wrote the original draft. The remaining authors were involved in clinical relevance variable selection, interpretation, and reviewing with the intellectual input to the manuscript.

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Data availability statement

All data access requests can be forwarded to DSC@georgeinstitute.org.

Conflict of interest

The authors declare no conflict of interest.

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Appendix A. Supplementary data

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