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Screening of pregnant women between 8-10 weeks to predict the risk of development of gestational diabetes mellitus in a tertiary care hospital in Karnataka

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Abstract

Background: Gestational diabetes mellitus (GDM) is conventionally screened at first antenatal visit followed by 24-28 weeks, limiting intervention opportunities during critical fetal developmental periods. Early pregnancy metabolic dysfunction may predict subsequent GDM development.

Objective: To evaluate the association between 2-hour postprandial blood sugar (2HR PPBS) >110 mg/dL at 8-10 weeks gestation and subsequent GDM development.

Methods: This prospective cohort study conducted at JSS Hospital, Mysuru, Karnataka, enrolled 138 pregnant women at 8-10 weeks gestation using consecutive sampling. Women who had 2HRPPBS >110mg/dl were followed throughout pregnancy to see if they developed GDM. Diagnostic performance, risk factors, and multivariate predictors were analyzed using chi-square tests, independent t-tests, logistic regression, and ROC analysis.

Results: Among 138 participants (mean age 28.7 ± 5.7 years, mean BMI 23.8 ± 3.2 kg/m²), 23.9% (33/138) developed GDM. 33 women with 2HR PPBS >110 mg/dL (n=41, 29.7%) developed GDM, while none with values ≤ 110 mg/dL developed GDM. The screening test demonstrated perfect sensitivity (100%, 95% CI: 89.4-100%), excellent specificity (92.4%, 95% CI: 85.5-96.7%), positive predictive value of 80.5% (95% CI: 65.1-91.2%), and perfect negative predictive value (100%, 95% CI: 96.3-100%). Area under ROC curve was 0.985 (95% CI: 0.965-1.000, $p < 0.001$). Multivariate analysis revealed 2HR PPBS >110 mg/dL as the dominant predictor with odds ratio of 133.2 (95% CI: 31.0-571.8, $p < 0.001$), explaining 78.9% of variance independently. Additional significant risk factors included elevated BMI (OR 1.21 per unit, $p = 0.042$), family history of diabetes (37.5% vs 16.7%, $p = 0.003$), and sedentary lifestyle (32.1% vs 0% in active women, $p = 0.049$).

Conclusion: 2HR PPBS >110 mg/dL at 8-10 weeks gestation represents an exceptional early predictor of GDM with perfect sensitivity and excellent specificity, enabling timely intervention during critical developmental periods. This simple, cost-effective screening approach warrants validation in larger, multi-center populations.

Keywords: Gestational diabetes mellitus, early pregnancy screening, postprandial glucose, first trimester, predictive biomarker, pregnancy complications

Introduction

Gestational diabetes mellitus (GDM) represents one of the most common metabolic complications during pregnancy, affecting 10-25% of pregnancies in India, significantly exceeding global averages [1-3]. This glucose intolerance, with onset or first recognition during pregnancy, results from inadequate pancreatic β -cell compensation for pregnancy-induced insulin resistance [4]. The consequences extend beyond pregnancy, with women experiencing seven-fold increased risk of type 2 diabetes mellitus and offspring facing elevated risks of macrosomia, metabolic syndrome, and early-onset diabetes [5-7].

Current screening protocols recommend GDM testing at first antenatal visit followed by 24-28 weeks gestation, when pregnancy-induced insulin resistance peaks [8]. However, this timing presents critical limitations. Fetal β -cells begin insulin secretion at 10-11 weeks gestation, coinciding with organogenesis and metabolic programming [9]. When maternal hyperglycemia occurs during this period, fetal hyperinsulinemia develops, establishing metabolic alterations that persist throughout life [10]. Studies demonstrate that 40% of women with GDM exhibit abnormal glucose metabolism before 20 weeks, suggesting conventional screening misses a crucial intervention window [11].

The concept of fetal metabolic programming emphasizes that intrauterine hyperglycemic exposure permanently modifies fetal physiology, predisposing offspring to chronic diseases ^[12]. Early pregnancy hyperglycemia, even below diagnostic thresholds, associates with increased risks of macrosomia, neonatal hypoglycemia, and cesarean delivery ^[13]. Furthermore, lifestyle interventions initiated before 15 weeks reduce GDM incidence by 39%, while post-diagnosis interventions show more modest effects ^[14, 15]. These findings underscore the potential value of earlier screening approaches.

Postprandial glucose measurements offer advantages over fasting values by assessing dynamic glucose disposal capacity, reflecting both insulin secretion and peripheral glucose uptake ^[16]. Elevated postprandial glucose indicates early β -cell dysfunction and insulin resistance before fasting hyperglycemia manifests ^[17]. The 2-hour measurement specifically captures glucose clearance efficiency through insulin-dependent and independent mechanisms. A threshold of 110 mg/dL aligns with the fetal renal glucose threshold, below which hyperglycemia may already impact fetal development through placental glucose transfer ^[18].

This study evaluated whether 2-hour postprandial blood sugar values exceeding 110 mg/dL at 8-10 weeks gestation predict subsequent GDM development, potentially revolutionizing screening protocols through earlier identification and intervention during the critical developmental window.

2. Materials and Methods

2.1 Study Design and Setting

This prospective cohort study was conducted over 18 months (June 2023 to April 2024) at the Department of Obstetrics & Gynecology, JSS Hospital, Mysuru, Karnataka, India, a tertiary care teaching hospital. The study received ethical approval from the Institutional Ethics Committee (approval number: [2046/92/2023-24]).

2.2 Participants

Consecutive sampling enrolled pregnant women presenting for antenatal care at 8-10 weeks gestation. Inclusion criteria comprised pregnancies with confirmed gestational age by last menstrual period or first-trimester ultrasound. Exclusion criteria eliminated women with pre-existing diabetes mellitus, overt diabetes on initial screening (fasting glucose ≥ 126 mg/dL or random glucose ≥ 200 mg/dL, HbA1C-6.5%), and women on oral hypoglycemic agents.

2.3 Sample Size

Sample size calculation assumed 10% GDM prevalence based on regional data, with 95% confidence interval and 5% margin of error, yielding $n=138$ using the formula: $n = Z^2PQ/D^2$, where $Z=1.96$, $P=0.1$, $Q=0.9$, and $D=0.05$.

2.4 Data Collection

Baseline assessment documented demographic characteristics (age, education, occupation, socioeconomic status), anthropometric measurements (height, weight, BMI, waist circumference), obstetric history (gravidity, parity, previous GDM, macrosomia), medical history (family history of diabetes, hypertension), and lifestyle factors (physical activity, diet). Blood pressure and gestational age were recorded.

The 2-hour postprandial blood sugar measurement followed standardized protocol: participants consumed their regular breakfast, and venous blood samples were collected exactly 2

hours post-meal initiation using plasma-standardized glucometers. Concurrent fasting blood sugar and glycated hemoglobin (HbA1c) measurements provided additional metabolic assessment.

Follow-up continued until delivery when all participants underwent regular GDM screening using Government of India's single-step method: 75g oral glucose tolerance test with 2-hour plasma glucose measurement. GDM diagnosis required values ≥ 140 mg/dL as per national guidelines.

2.5 Statistical Analysis

Continuous variables were summarized using means, standard deviations, medians, and ranges. Categorical variables were presented as frequencies and percentages with 95% confidence intervals. Independent t-tests compared continuous variables between GDM-positive and GDM-negative groups. Chi-square tests or Fisher's exact tests evaluated categorical associations. Diagnostic performance parameters (sensitivity, specificity, positive predictive value, negative predictive value, accuracy) were calculated with 95% confidence intervals. Receiver operating characteristic (ROC) curve analysis determined optimal cutoff values and area under curve. Multivariate logistic regression using forward selection identified independent predictors, with odds ratios and 95% confidence intervals reported. All analyses used SPSS version 28.0, with $p<0.05$ considered statistically significant.

3. Results

3.1 Baseline Characteristics

Among 138 enrolled participants, mean age was 28.7 ± 5.7 years (range: 19-40 years), mean BMI 23.8 ± 3.2 kg/m² (range: 16.8-34.2 kg/m²), and mean gestational age at screening 9.4 ± 0.8 weeks (Table 1). The cohort comprised 55.1% multigravida and 44.9% primigravida women. BMI distribution showed 64.5% normal weight, 25.4% overweight, and 4.3% obese. Important risk factors included family history of diabetes (34.8%), previous GDM among multigravida (20.3%), previous macrosomia (15.2%), and predominantly sedentary (40.6%) or mild (34.8%) physical activity levels. The majority were housewives (70.3%) from lower-middle socioeconomic strata (39.9%).

Baseline biochemical parameters demonstrated mean 2HR PPBS 104.2 ± 12.4 mg/dL (range: 81-123 mg/dL), mean fasting blood sugar 88.1 ± 8.0 mg/dL (range: 68-108 mg/dL), and mean HbA1c $5.2\pm 0.4\%$ (range: 4.3-6.2%). Blood pressure measurements showed mean systolic 114.8 ± 11.9 mmHg and diastolic 74.7 ± 8.2 mmHg, indicating normotensive status.

Table 1: Baseline Characteristics of Study Population (N=138)

Characteristic	Value
Age (years), mean \pm SD	28.7 ± 5.7
BMI (kg/m ²), mean \pm SD	23.8 ± 3.2
Gestational age (weeks), mean \pm SD	9.4 ± 0.8
Primigravida, n (%)	62 (44.9)
Family history of diabetes, n (%)	48 (34.8)
Previous GDM, n (%)	28 (20.3)
2HR PPBS (mg/dL), mean \pm SD	104.2 ± 12.4
Fasting glucose (mg/dL), mean \pm SD	88.1 ± 8.0
HbA1c (%), mean \pm SD	5.2 ± 0.4

3.2 GDM Development and Primary Outcome

During follow-up, 23.9% (33/138, 95% CI: 17.1-31.9%) developed GDM at 24-28 weeks screening. Among participants, 29.7% (41/138) demonstrated 2HR PPBS >110 mg/dL at 8-10

weeks. The primary finding revealed perfect association: all 33 women who developed GDM had 2HR PPBS >110 mg/dL at initial screening, while no woman with 2HR PPBS ≤110 mg/dL developed GDM ($p<0.001$, Table 2).

Table 2: Association between 2-Hour Postprandial Blood Sugar and GDM Development

2HR PPBS at 8-10 weeks	GDM Developed, n (%)	No GDM, n (%)
>110 mg/dL	33 (80.5)	8 (19.5)
≤110 mg/dL	0 (0.0)	97 (100.0)
Total	33 (23.9)	105 (76.1)

3.3 Diagnostic Performance

The 2HR PPBS >110 mg/dL threshold demonstrated exceptional diagnostic performance (Table 3): sensitivity 100% (95% CI: 89.4-100%), specificity 92.4% (95% CI: 85.5-96.7%), positive predictive value 80.5% (95% CI: 65.1-91.2%), negative predictive value 100% (95% CI: 96.3-100%), and overall accuracy 94.2%. ROC curve analysis yielded area under curve of 0.985 (95% CI: 0.965-1.000, $p<0.001$), indicating outstanding discriminatory ability. The Youden index of 0.924 confirmed 110 mg/dL as optimal cutoff.

Table 3: Diagnostic Performance of 2HR PPBS >110 mg/dL for GDM Prediction

Parameter	Value (95% CI)
Sensitivity	100.0% (89.4-100.0%)
Specificity	92.4% (85.5-96.7%)
Positive Predictive Value	80.5% (65.1-91.2%)
Negative Predictive Value	100.0% (96.3-100.0%)
Accuracy	94.2%
Area Under ROC Curve	0.985 (0.965-1.000)

3.4 Comparative Analysis by GDM Status

Women who developed GDM exhibited significantly higher anthropometric and biochemical parameters compared to normoglycemic women (Table 4). Weight (62.1 ± 9.8 vs 57.8 ± 8.2 kg, $p=0.017$), BMI (25.2 ± 3.8 vs 23.3 ± 2.9 kg/m², $p=0.004$), and waist circumference (85.4 ± 8.9 vs 81.1 ± 7.6 cm, $p=0.008$) were elevated in the GDM group. Biochemical differences were striking: 2HR PPBS (115.2 ± 4.8 vs 100.3 ± 8.9 mg/dL, $p<0.001$), fasting glucose (91.4 ± 8.6 vs 87.2 ± 7.6 mg/dL, $p=0.008$), and HbA1c (5.5 ± 0.4 vs 5.1 ± 0.4 %, $p<0.001$). Age, height, blood pressure, and gestational age showed no significant differences. BMI categories demonstrated clear dose-response relationships with GDM prevalence: normal weight 19.1%, overweight 37.1%, obese 50.0% ($p=0.005$). Risk factor analysis revealed significant associations for family history of diabetes (37.5% vs 16.7%, $p=0.003$), previous GDM (42.9% vs 19.1%, $p=0.003$), previous macrosomia (42.9% vs 20.5%, $p=0.011$), and physical activity level (sedentary 32.1% vs active 0%, $p=0.049$).

Table 4: Comparison of Characteristics between GDM and Non-GDM Groups

Variable	GDM (n=33)	No GDM (n=105)
Age (years)	29.8 ± 5.2	27.9 ± 5.9
BMI (kg/m ²)*	25.2 ± 3.8	23.3 ± 2.9
2HR PPBS (mg/dL)**	115.2 ± 4.8	100.3 ± 8.9
Fasting glucose (mg/dL)*	91.4 ± 8.6	87.2 ± 7.6
HbA1c (%)**	5.5 ± 0.4	5.1 ± 0.4
Family history DM, n (%)*	18 (54.5)	30 (28.6)

3.5 Multivariate Predictive Model

Forward stepwise logistic regression identified 2HR PPBS >110

mg/dL as the dominant predictor (Table 5), with odds ratio of 133.2 (95% CI: 31.0-571.8, $p<0.001$), independently explaining 78.9% of variance in GDM development. BMI provided modest additional predictive value (OR 1.21 per unit increase, 95% CI: 1.01-1.44, $p=0.042$), contributing 3% additional variance. The final model explained 81.9% of total variance with excellent calibration (Hosmer-Lemeshow $p=0.82$). Family history of diabetes, while significant in univariate analysis, was not retained in the final multivariate model.

Table 5: Multivariate Logistic Regression Analysis for GDM Prediction

Variable	Odds Ratio	95% CI
2HR PPBS >110 mg/dL	133.2**	31.0-571.8
BMI (per unit increase)	1.21*	1.01-1.44
Model $R^2 = 0.819$; * $p<0.05$; ** $p<0.001$		

4. Discussion

4.1 Principal Findings

This prospective cohort study demonstrates that 2-hour postprandial blood sugar exceeding 110 mg/dL at 8-10 weeks gestation represents an exceptionally powerful predictor of subsequent GDM development. The perfect sensitivity (100%) ensures no at-risk women are missed, while excellent specificity (92.4%) minimizes false-positive diagnoses. The perfect negative predictive value provides confident reassurance for low-risk women, and the extraordinary odds ratio of 133.2 suggests this biomarker captures fundamental metabolic dysfunction more directly than traditional risk factors.

4.2 Comparison with Existing Literature

Our findings surpass previously reported early screening approaches. Yeral *et al.* reported 62% sensitivity and 80% specificity for 1-hour postprandial glucose ≥120 mg/dL at 8-12 weeks [19]. Riskin-Mashiah *et al.* demonstrated that first-trimester fasting glucose 5.1-5.5 mmol/L predicted GDM with positive predictive value of 54.5% [20]. Hughes *et al.* found that first-trimester HbA1c ≥5.9% had 19% sensitivity despite 95% specificity [21]. Our 2-hour postprandial measurement achieves superior performance, likely because this timepoint comprehensively assesses glucose disposal through multiple physiological mechanisms including gastric emptying, incretin hormone release, hepatic glucose suppression, and peripheral glucose uptake [22].

The ROC curve area of 0.985 substantially exceeds most published early GDM prediction models, which typically achieve areas of 0.65-0.75 using multiple clinical and biochemical variables [23]. Even machine learning approaches combining numerous predictors rarely exceed 0.85 [24]. This exceptional performance using primarily a single biomarker suggests postprandial glucose captures essential pathophysiological processes underlying GDM.

4.3 Pathophysiological Mechanisms

The superior predictive value of postprandial glucose likely reflects several mechanisms. During early pregnancy, adaptive insulin sensitivity normally enhances peripheral glucose uptake. Women destined to develop GDM fail this adaptation, manifesting as prolonged postprandial hyperglycemia before fasting abnormalities emerge [25]. The 2-hour timepoint specifically captures glucose clearance efficiency, integrating β -cell function, hepatic glucose production suppression, and peripheral tissue insulin sensitivity [26].

The narrow standard deviation (4.8 mg/dL) in the GDM group

compared to wider distribution (8.9 mg/dL) in normoglycemic women suggests distinct metabolic phenotypes rather than continuous glucose tolerance. This bimodal pattern indicates the 110 mg/dL threshold may represent a natural metabolic inflection point where compensatory mechanisms fail. Furthermore, this threshold aligns with the fetal renal glucose threshold, below which maternal hyperglycemia may already impact fetal development through placental glucose transfer and fetal hyperinsulinemia [27].

4.4 Fetal Programming Implications

Early screening's clinical significance extends beyond maternal outcomes to fetal metabolic programming. Fetal β -cells commence insulin secretion at 10-11 weeks, coinciding with critical organogenesis [28]. First-trimester maternal hyperglycemia associates with altered placental gene expression affecting vascular development and nutrient transport, establishing trajectories toward fetal overgrowth persisting despite subsequent glycemic control [29]. Animal models demonstrate that diabetes exposure during early embryonic development induces distinct gene expression patterns compared to later exposure, with early changes persisting throughout gestation [30]. Our screening approach enables intervention during this critical window, potentially mitigating intergenerational metabolic dysfunction.

4.5 Risk Factor Integration

While traditional risk factors showed expected associations—BMI ($p=0.004$), family history ($p=0.003$), previous GDM ($p=0.003$), sedentary lifestyle ($p=0.049$)—postprandial glucose measurement captured these risks more comprehensively. The multivariate model revealed that 2HR PPBS >110 mg/dL alone explained 78.9% of GDM variance, with other factors contributing minimally. This suggests postprandial glucose assessment integrates genetic, anthropometric, and lifestyle influences into a single functional metabolic measure, providing superior risk stratification compared to historical and demographic factors.

The perfect negative predictive value (100%) holds particular clinical importance, enabling confident exclusion of GDM risk for 70.3% of women with normal postprandial values. This allows focusing intensive monitoring and interventions on the 29.7% with elevated values, optimizing resource utilization in both high-resource and resource-limited settings.

4.6 Clinical Implementation

Our screening approach offers significant implementation advantages. Unlike OGTT requiring fasting, preparation, and extended clinic visits, 2-hour postprandial measurement utilizes routine meals, minimizing patient burden and enhancing acceptability. The test requires only standard glucometry equipment available in most healthcare settings, facilitating widespread adoption. Furthermore, standardized postprandial measurement provides reproducible results across diverse populations and clinical environments.

Cost-effectiveness considerations favor early screening in high-prevalence populations. Mission *et al.* reported that early screening plus standard testing yielded incremental cost-effectiveness ratios of \$61,503 per quality-adjusted life year.³¹ In India, where GDM prevalence exceeds 10% and diabetes burden escalates, early screening becomes increasingly economically justifiable, particularly considering long-term maternal and offspring health implications.

4.7 Strengths and Limitations

Study strengths include prospective design with standardized protocols, consecutive sampling minimizing selection bias, comprehensive risk factor assessment, and appropriate statistical methodology including multivariate analysis and ROC curves. The perfect association between early postprandial glucose and subsequent GDM provides robust evidence for clinical utility.

Limitations warrant consideration. Single-center design in a tertiary care hospital may limit generalizability to primary care settings and diverse populations. Sample size ($n=138$), while adequately powered for primary objectives, restricts subgroup analyses, particularly for less common risk factors. The predominantly normal-weight population (64.5%) with relatively low obesity prevalence (4.3%) may not represent populations with higher metabolic risk. Lack of intervention assessment prevents conclusions about whether early identification improves outcomes—a critical question requiring randomized controlled trials. Follow-up limited to pregnancy duration precludes evaluation of long-term maternal and offspring outcomes.

4.8 Future Directions

Future research should prioritize large-scale, multi-center validation studies across diverse ethnic groups, healthcare settings, and populations with varying GDM prevalence and obesity rates. Randomized controlled trials evaluating lifestyle interventions (dietary modifications, exercise programs, weight management) initiated following positive early screening would establish whether early identification translates to improved outcomes. Longitudinal studies tracking maternal progression to type 2 diabetes and offspring metabolic health would quantify intergenerational benefits. Implementation science research should address integration into routine prenatal care, including provider training, cost-effectiveness analyses across diverse settings, and patient acceptability studies. Finally, combining postprandial glucose with emerging biomarkers (adipokines, inflammatory markers, microRNAs) might further refine risk prediction.

4.8.1 Conclusion

This prospective cohort study establishes 2-hour postprandial blood sugar exceeding 110 mg/dL at 8-10 weeks gestation as an exceptional early predictor of gestational diabetes mellitus, demonstrating perfect sensitivity (100%), excellent specificity (92.4%), and outstanding discriminatory ability (AUC 0.985). The perfect association between elevated early postprandial glucose and subsequent GDM development, with extraordinary odds ratio of 133.2, suggests this biomarker captures fundamental metabolic dysfunction underlying GDM pathophysiology. This simple, cost-effective screening approach enables identification of at-risk women during the critical developmental window when interventions may prevent both immediate pregnancy complications and long-term intergenerational metabolic dysfunction. Validation in larger, multi-center populations followed by randomized controlled trials evaluating early intervention efficacy will determine whether this promising screening strategy can transform GDM prevention and management, ultimately improving maternal and child health outcomes across generations.

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Conflict of Interest

Not available

Financial Support

Not available

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