

Association Between Maternal Glycemic Status and Urinary Infection Markers During Pregnancy: A Prospective Observational Study

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Research Article

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Abstract

Purpose

The aim of this study was to evaluate the relationship between maternal glycemia and urinary infection markers during pregnancy and to investigate the effect of glycemic variations in pregnant women with a negative oral glucose tolerance test (OGTT) on urine culture positivity, Group B Streptococcus (GBS) colonization, and asymptomatic bacteriuria (ASB).

Methods

This prospective observational study included 2,017 pregnant women who underwent a 75-g OGTT between 28 and 32 weeks of gestation and had a urine culture evaluated during the same visit. Gestational diabetes mellitus (GDM) was diagnosed according to the criteria of the International Association of Diabetes and Pregnancy Study Groups. The primary outcome was a positive urine culture; secondary outcomes were the presence of GBS and ASB. The independent effects of fasting plasma glucose (FPG), 1-hour, and 2-hour glucose values were evaluated in the OGTT-negative subgroup using multivariate logistic regression analysis.

Results

OGTT positivity was determined to be 30.5%. Urine culture positivity was significantly higher in the OGTT-positive group (23.5% vs. 15.6%; OR 1.67; 95% CI 1.32–2.11; $p < 0.001$). The prevalence of GBS and ASB was also higher in OGTT-positive pregnant women. In the OGTT-negative subgroup, each 10 mg/dL increase in FPG levels was found to be the strongest independent predictor for all outcomes (aOR 1.39 for culture positivity; aOR 1.35 for GBS; aOR 1.38 for ASB; all $p < 0.001$). In the ROC analysis, the discriminatory performance of glucose parameters was modest (AUROC 0.56–0.60).

Conclusion

The presence of GDM is associated with an increase in urinary infection markers during pregnancy. However, the risk of infection is not limited to pregnant women diagnosed with GDM; even in pregnant women with a negative OGTT, elevated glucose levels—particularly FPG—have been found to be associated with infection markers. These findings suggest that glycemic status during pregnancy may be better assessed as a continuous risk spectrum rather than based on categorical thresholds.

INTRODUCTION

GDM is a common metabolic disorder that develops against a background of increased insulin resistance during pregnancy and has significant effects on maternal and neonatal outcomes[1].

Pregnancy-specific hormonal changes (e.g., human placental lactogen, progesterone, and cortisol) reduce peripheral insulin sensitivity, thereby creating a predisposition to relative hyperglycemia[2]. This metabolic environment is characterized by increased oxidative stress and low-grade inflammation and may negatively affect host defense mechanisms[3]. The literature indicates that hyperglycemia disrupts neutrophil chemotaxis and phagocytosis, modulates the cellular immune response, and may delay bacterial clearance in the urinary tract[4] .

Urinary tract infections during pregnancy, particularly ASB and GBS colonization, are significant infectious conditions associated with maternal and neonatal morbidity. Untreated ASB has been linked to pyelonephritis, preterm birth, and low birth weight; GBS colonization, meanwhile, is one of the primary risk factors for early-onset neonatal sepsis[5]. However, studies examining the relationship between maternal glycemic status and these infection markers have not yielded consistent results. In addition to studies reporting an increased risk of infection in the presence of GDM, there are also data that fail to identify a significant association[1, 6]. This heterogeneity is thought to stem from differences in study populations, definitions of infection, and the fact that glycemic status was assessed categorically in most studies[7] .

The current approach to diagnosing GDM is based on fasting, 1-hour, and 2-hour plasma glucose levels measured during a 75-g oral glucose tolerance test[8]. The diagnosis of GDM is based on threshold values established by the IADPSG[9]. However, the current diagnostic approach largely reduces glycemic status to a binary classification based on diagnostic thresholds[10]. Furthermore, OGTT parameters are biological variables with continuous distributions, and the clinical implications of glycemic variations falling below diagnostic thresholds have not been sufficiently characterized. In the current literature, the evaluation of OGTT components as continuous variables and, in particular, the examination of the relationship between glucose levels and infection markers in OGTT-negative pregnant women is limited. This may contribute to the heterogeneity observed in the results reported in the literature.

In this study, the association between glucose levels measured during an OGTT in pregnancy and positive urine culture, Group B Streptococcus (GBS) colonization, and asymptomatic bacteriuria (ASB) was evaluated. Additionally, the independent effect of glycemic variations below diagnostic thresholds in OGTT-negative pregnant women on these outcomes was analyzed. It is believed that this approach will contribute to a more detailed characterization of the relationship between maternal glycemic status and infection risk.

MATERIALS AND METHODS

This study was designed as a prospective observational study conducted at a tertiary care center. Pregnant women who underwent a 75-g oral glucose tolerance test (OGTT) between 28 and 32 weeks of gestation as part of routine antenatal care and had a urine culture evaluated during the same visit were consecutively enrolled.

Inclusion criteria were defined as having undergone an OGTT between 28 and 32 weeks of gestation, having a urine culture result available during the same visit, and having complete clinical and laboratory data. Cases with pre-existing diabetes mellitus, multiple pregnancies, preeclampsia, and other high-risk pregnancy conditions, as well as those with known chronic kidney disease or urinary tract anomalies, were excluded from the study. Cases with missing data were not included in the analyses.

All participants underwent a standard 75-g OGTT after at least an 8-hour fast. Plasma glucose levels were measured at baseline, 1 hour, and 2 hours. The diagnosis of gestational diabetes mellitus (GDM) was made according to the criteria of the International Association of Diabetes and Pregnancy Study Groups (IADPSG). Meeting any of the following threshold values was considered sufficient for diagnosis: fasting plasma glucose ≥ 92 mg/dL, 1-hour glucose ≥ 180 mg/dL, or 2-hour glucose ≥ 153 mg/dL.

The primary outcome was defined as a positive urine culture. A positive urine culture was defined as the growth of Group B Streptococcus (GBS) or other bacterial pathogens. Secondary outcomes were GBS positivity and the presence of asymptomatic bacteriuria (ASB). ASB was defined as a positive urine culture result in the absence of urinary tract symptoms.

The following were evaluated as independent variables: OGTT result, fasting plasma glucose (FPG), and 1-hour and 2-hour plasma glucose levels. Potential confounding variables were identified as maternal age, gestational week, and parity. The age variable was categorized in the analyses (16–25, 26–35, and ≥ 36 years).

Statistical Analysis

All analyses were performed using two-sided tests, and $p < 0.05$ was considered statistically significant. Statistical analyses were conducted using SPSS (version 25.2, IBM Corp.) and Python-based analysis tools.

The analyses were planned in two phases. The primary outcome was positive urine culture, while the secondary outcomes were Group B Streptococcus (GBS) positivity and the presence of asymptomatic bacteriuria (ASB). The explanatory variables were OGTT results, fasting plasma glucose (FPG), and 1-hour and 2-hour plasma glucose levels. Age group, gestational week, and parity were included in the models as potential confounding variables.

The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test and histogram analyses. Variables that did not follow a normal distribution were presented as median and interquartile range (IQR). Categorical variables were expressed as counts and percentages.

The association between OGTT positivity and outcomes in the entire population was assessed using the Pearson chi-square test, and results were reported as odds ratios (OR) with 95% confidence intervals (CI).

Multivariate logistic regression analyses were performed to assess the independent effect of fasting plasma glucose, 1-hour, and 2-hour glucose levels on outcomes in the OGTT-negative subgroup. All models were adjusted for age group, gestational week, and parity. Effect sizes were reported as the adjusted odds ratio (aOR) and 95% confidence interval for every 10 mg/dL increase in glucose levels.

The discriminatory performance of glucose parameters was assessed using ROC curve analysis, and the area under the curve (AUC) was calculated. Missing data were excluded using a list-based method.

RESULTS

Characteristics of the study population

2,017 pregnant women were included in the analysis. Of the cases, 625 (31.0%) were in the 16–25 age group, 990 (49.1%) in the 26–35 age group, and 402 (19.9%) in the 36 years and older group. The mean gestational week was 30.0 ± 2.1 . The median fasting plasma glucose (FPG) was 73 mg/dL (interquartile range [IQR] 65–83), the median 1-hour glucose was 118 mg/dL (IQR 92–149), and the median 2-hour glucose level was 127 mg/dL (IQR 117–148). The OGTT positivity rate was 30.5% ($n = 616$), and the urine culture positivity rate was 18.0% ($n = 363$). Among secondary outcomes, GBS positivity was 16.2% ($n = 326$), and ASB positivity was 16.0% ($n = 322$). Demographic and clinical characteristics are summarized in Table 1.

Table 1
Basic characteristics of the study population (N = 2017).

Variable	Value
Total number of cases	2017
Age group 16–25, n (%)	625 (31.0)
Age group 26–35, n (%)	990 (49.1)
Age group ≥ 36, n (%)	402 (19.9)
Gestational week, mean ± SD	30.0 ± 2.1
Gravidity, mean ± SD	2.51 ± 1.14
Parity, mean ± SD	0.93 ± 1.00
Fasting plasma glucose, mg/dL	74.8 ± 13.1; median 73 (IQR 65–83)
1-hour glucose, mg/dL	121.3 ± 31.8; median 118 (IQR 92–149)
2-hour glucose, mg/dL	132.0 ± 21.9; median 127 (IQR 117–148)
Positive OGTT, n (%)	616 (30.5)
Positive urine culture, n (%)	363 (18.0)
GBS positive, n (%)	326 (16.2)
ASB positive, n (%)	322 (16.0)

Urinary outcomes associated with OGTT positivity in the entire population

Urine culture positivity, defined as the primary outcome, was found to be 23.5% (145/616) in OGTT-positive cases and 15.6% (218/1401) in OGTT-negative cases. This difference was statistically significant ($\chi^2 = 17.92$; $p < 0.001$), and OGTT positivity was found to be associated with a 1.67 odds ratio (95% CI 1.32–2.11) for culture positivity.

When secondary outcomes were examined, GBS positivity was 21.1% (130/616) in the OGTT-positive group and 14.0% (196/1401) in the OGTT-negative group ($p < 0.001$; OR 1.65; 95% CI 1.29–2.10). ASB positivity was calculated as 19.5% (120/616) in the OGTT-positive group and 14.4% (202/1401) in the OGTT-negative group ($p = 0.005$; OR 1.44; 95% CI 1.12–1.84). The findings are presented in Table 2.

Table 2
Association between OGTT positivity and primary and secondary outcomes in the entire population.

Outcome	OGTT(+) n/N (%)	OGTT(−) n/N (%)	p	OR (95% CI)
Culture positivity	145/616 (23.5)	218/1401 (15.6)	< 0.001	1.67 (1.32–2.11)
GBS positivity	130/616 (21.1)	196/1401 (14.0)	< 0.001	1.65 (1.29–2.10)
ASB positivity	120/616 (19.5)	202/1401 (14.4)	0.005	1.44 (1.12–1.84)

Urinary outcomes associated with glycemic variables in the OGTT-negative subgroup

In the OGTT-negative subgroup (n = 1401), urine culture positivity was 15.6% (n = 218), GBS positivity was 14.0% (n = 196), and ASB positivity was 14.4% (n = 202). In this subgroup, fasting plasma glucose (FPG), 1-hour, and 2-hour glucose levels were higher in culture-positive cases compared to culture-negative cases; the difference was statistically significant for all three variables (p < 0.001, p < 0.001, and p = 0.002, respectively; Mann–Whitney U). The same trend was observed in GBS- and ASB-positive cases (Table 3). The most pronounced median difference was observed in 1-hour glucose.

Table 3
Comparison of glucose values between positive and negative cases in the OGTT-negative subgroup (median and IQR).

Outcome	Glucose	Positive: median (IQR)	Negative: median (IQR)	p
Culture	FPG	75 (68–82)	72 (64–79)	< 0.001
Culture	1-hour	116 (90–138)	99 (89–134)	< 0.001
Culture	2-hour	124 (118–134)	122 (112–130)	0.002
GBS	FPG	75 (68–82)	72 (64–79)	0.001
GBS	1- hour	115 (90–136)	99 (89–135)	< 0.001
GBS	2nd hour	123 (117–134)	122 (112–130)	0.008
ASB	FPG	75 (68–82)	72 (64–79)	< 0.001
ASB	1-hour	116 (90–138)	99 (89–134)	< 0.001
ASB	2- hour	123 (117–134)	122 (112–130)	0.008

Multivariate analysis and strongest predictor

In the OGTT-negative subgroup, each glucose variable was first included individually in a logistic regression; then, FPG, 1-hour, and 2-hour glucose levels were included together in the same model and

adjusted for age group, gestational week, and parity. All results are presented per 10 mg/dL increase. In the univariate model, all three glucose parameters were significant for all three outcomes (Table 4).

In the multivariate model, FPG remained independently significant for all three outcomes: aOR 1.39 (95% CI 1.19–1.62; $p < 0.001$), aOR 1.35 for GBS positivity (95% CI 1.15–1.59; $p < 0.001$), and aOR 1.38 for ASB positivity (95% CI 1.18–1.62; $p < 0.001$). The 1-hour glucose level also maintained its independent significance, though the effect size was smaller (aOR 1.11 for culture; aOR 1.08 for GBS; aOR 1.13 for ASB). When 2-hour glucose, FPG, and 1-hour values were included in the model together, none of the three outcomes remained significant ($p \geq 0.41$). Age group showed a significant effect toward older age (aOR approximately 1.80–1.90; $p < 0.001$), while gestational week and parity did not retain their independent effects.

Table 4
Logistic regression results in the OGTT-negative subgroup (per 10 mg/dL increase; the multivariate model was adjusted for age group, gestational week, and parity).

Outcome	Glucose	OR* (95% CI) – univariate	aOR† (95% CI) – multivariate	p (aOR)
Culture	FPG	1.32 (1.14–1.53)	1.39 (1.19–1.62)	< 0.001
Culture	1-hour	1.11 (1.06–1.17)	1.11 (1.04–1.19)	0.002
Culture	2-hour	1.18 (1.07–1.31)	1.05 (0.91–1.20)	0.497
GBS	FPG	1.30 (1.11–1.52)	1.35 (1.15–1.59)	< 0.001
GBS	1-hour	1.09 (1.04–1.15)	1.08 (1.01–1.16)	0.022
GBS	2-hour	1.16 (1.05–1.30)	1.06 (0.92–1.23)	0.413
ASB	FPG	1.31 (1.13–1.53)	1.38 (1.18–1.62)	< 0.001
ASB	1- hour	1.12 (1.06–1.17)	1.13 (1.05–1.21)	< 0.001
ASB	2-hour	1.16 (1.04–1.29)	1.01 (0.88–1.16)	0.911

When discriminatory performance was evaluated, the areas under the ROC curve (AUROC) for individual glucose values remained at a modest level (Table 5, Fig. 1). The 1-hour glucose value yielded the highest AUROC values for culture and ASB positivity (0.605 and 0.606, respectively). However, in the multivariate model, the effect size of FPG was significantly higher than that of the other two glucose values; although the AUROC values were similar, this supports the finding that FPG is the strongest independent predictor when other parameters are held constant.

Table 5
Individual discriminatory power (AUROC) of each glucose variable in the OGTT-negative subgroup.

Outcome	FPG – AUROC	1-hour – AUROC	2-hour – AUROC
Culture positivity	0.577	0.605	0.566
GBS positivity	0.571	0.592	0.559
ASB positivity	0.576	0.606	0.558

DISCUSSION

In this prospective observational study, the presence of GDM was shown to be significantly associated with positive urine culture, GBS colonization, and ASB. Additionally, it was found that elevated glucose levels were independently associated with infection markers even in pregnant women with a negative OGTT[1]. In particular, the fact that FPG maintained its independent significance for all outcomes even after adjusting for age, gestational week, and parity suggests that glycemic risk during pregnancy should be evaluated as a continuous biological spectrum, not limited solely to diagnostic thresholds. Furthermore, the persistence of the association between glucose parameters and culture positivity even in the OGTT-negative subgroup supports the potential impact of subclinical glycemic variations on maternal susceptibility to infection.

Gestational diabetes mellitus is a complex metabolic disorder resulting from the exacerbation of physiologically increased insulin resistance during pregnancy and is closely associated with maternal-fetal morbidity[11–13]. Currently, the diagnosis of GDM is commonly based on at least one of the following plasma glucose values exceeding the thresholds during a 75-g OGTT: fasting (≥ 92 mg/dL), 1-hour (≥ 180 mg/dL), and 2-hour (≥ 153 mg/dL) levels[14]. However, OGTT parameters should not be evaluated solely as categorical diagnostic values but should also be considered as biological indicators reflecting the persistence of maternal metabolic burden[15]. It is known that hyperglycemia impairs neutrophil chemotaxis, phagocytosis, and the cellular immune response; glucose causes functional impairment in immune cells via the polyol pathway and advanced glycation end products[4, 16, 17]. Therefore, disruption of glycemic balance is considered one of the key mechanisms that may increase susceptibility to infection during pregnancy[1, 18].

ASB and GBS colonization during pregnancy are among the significant infectious conditions associated with maternal and neonatal morbidity[19, 20]. In particular, it has been shown that ASB, when left untreated during pregnancy, increases the risk of developing pyelonephritis by up to 30%; furthermore, it has a strong association with preterm birth, low birth weight, and various obstetric complications[21–23]. Similarly, maternal GBS colonization is recognized as one of the primary risk factors for early-onset neonatal sepsis, and therefore, routine antenatal screening is recommended in many international guidelines[24]. However, the relationship between maternal glycemic status and ASB and GBS colonization remains a subject of debate in the literature. While some studies report that the prevalence

of ASB and GBS is significantly higher in diabetic pregnant women and that impaired glycemic control (such as high HbA1c) triggers this risk, there are also recent series that have not identified a statistically significant association between the presence of GDM and these infections[6, 25–26] .

This heterogeneity in the current data is thought to stem from differences in study designs, a lack of standardization in infection definitions, and the fact that glycemic status is often assessed as a categorical classification rather than a continuous biological variable in most studies[27–29] .

In this study, we went beyond classifying glycemic status solely based on diagnostic thresholds and evaluated OGTT parameters as continuous variables. It is particularly noteworthy that the increase in FPG levels in OGTT-negative pregnant women was found to be independently associated with positive urine culture, GBS colonization, and ASB. This finding suggests that the risk of infection is not limited to overt hyperglycemia alone, and that glycemic variations below the diagnostic threshold may also be biologically significant. The modest AUROC values in ROC analyses indicate that glucose parameters alone are not strong discriminatory tests; however, the aim of this study was to characterize the biological relationship between maternal glycemic status and infection markers rather than to develop a new screening test.

It is noteworthy that FPG remained the strongest independent predictor in all multivariate models. This suggests that fasting glucose may better reflect not only the transient postprandial response but also the basal metabolic and inflammatory state. It has been demonstrated that chronic or persistent mild hyperglycemia exerts deleterious effects on neutrophil chemotaxis, phagocytosis, and the oxidative burst response; furthermore, it can lead to dysfunction in cellular immune functions via polyol pathway activation, advanced glycation end-products (AGEs), and oxidative stress[4]. These mechanisms are thought to play a role in the fundamental cellular processes that increase the risk of infection in individuals with diabetes. In particular, it has been shown that polyol pathway activation and the accompanying NADPH consumption may impair neutrophil function by reducing intracellular antioxidant capacity[16]. Furthermore, the fact that fasting glucose is more closely associated with hepatic insulin resistance and basal metabolic dysregulation suggests that the effect of FPG on maternal immune homeostasis may be more pronounced compared to post-load glucose values.

In conclusion, the presence of GDM is associated with an increase in urinary infection markers during pregnancy. However, this association is not limited to pregnant women who meet diagnostic criteria; even in OGTT-negative pregnant women, elevated glycemic levels, particularly FPG, show an independent association with positive urine culture, GBS colonization, and ASB. These findings support the need to assess glycemic risk during pregnancy as a continuous biological spectrum rather than categorical thresholds and underscore the potential role of subclinical glycemic variations in maternal susceptibility to infection.

Strengths and limitations

The study's main strengths include its prospective design, large sample size, and the evaluation of both the OGTT and urine culture during the same visit. This approach minimized temporal variability, allowing for a more reliable assessment of the association. Additionally, the separate analysis of the OGTT-negative subgroup enabled the evaluation of subthreshold glycemic effects, which have been limited in the literature.

However, the study has some limitations. First, the study was conducted at a single center, and the generalizability of the results to other populations may be limited. Additionally, culture positivity was assessed based on microbiological data, and a correlation with clinical infection could not be demonstrated in all cases. The modest AUROC values obtained in the ROC analyses suggest that glucose parameters alone are not strong discriminatory markers. However, the primary aim of the study was to assess the biological and clinical significance of the relationship between glycemic status and infection risk rather than to develop a discriminatory screening model.

CONCLUSION

In this prospective observational study, the presence of GDM was shown to be significantly associated with positive urine culture, GBS colonization, and asymptomatic bacteriuria. The most important finding of our study is that the risk of infection is not limited to pregnant women with a categorical diagnosis of GDM; even in pregnant women with a negative OGTT, elevated glycemic levels show an independent association with infection markers.

In particular, the identification of fasting plasma glucose as the strongest independent predictor in all multivariate models supports the decisive role of basal glycemic homeostasis in maternal susceptibility to infection. These findings suggest that glycemic status during pregnancy should be evaluated not merely based on diagnostic threshold values, but within the framework of a continuous biological risk spectrum. It should be noted that even glycemic variations below diagnostic thresholds may contribute to infection risk.

Although the discriminatory performance of glycemic parameters is limited, the relationship between maternal glycemic load and urinary infection markers appears to be biologically and clinically significant. Larger, multicenter prospective studies are needed to better understand the underlying mechanisms of this relationship and its impact on clinical outcomes.

Declarations

Ethics approval and consent to participate

Ethics approval for this study was obtained from the Clinical Research Ethics Committee of Gazi Yaşargil Training and Research Hospital, University of Health Sciences (Approval No: 784, Date: 12/05/2025). As Silvan Dr. Yusuf Azizoğlu State Hospital does not have an independent clinical research ethics committee, approval was obtained from the authorized regional ethics committee. The study was

conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Consent for publication

Not applicable.

Conflicts of interest

The authors declare no conflicts of interest.

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Author Contribution

GK: Study concept, data collection, data analysis, and manuscript preparation. YB: Statistical analysis, methodological guidance, and critical revision. KU: Data collection, literature review, and critical revision of the manuscript. All authors have read and approved the final version of the manuscript.

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Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

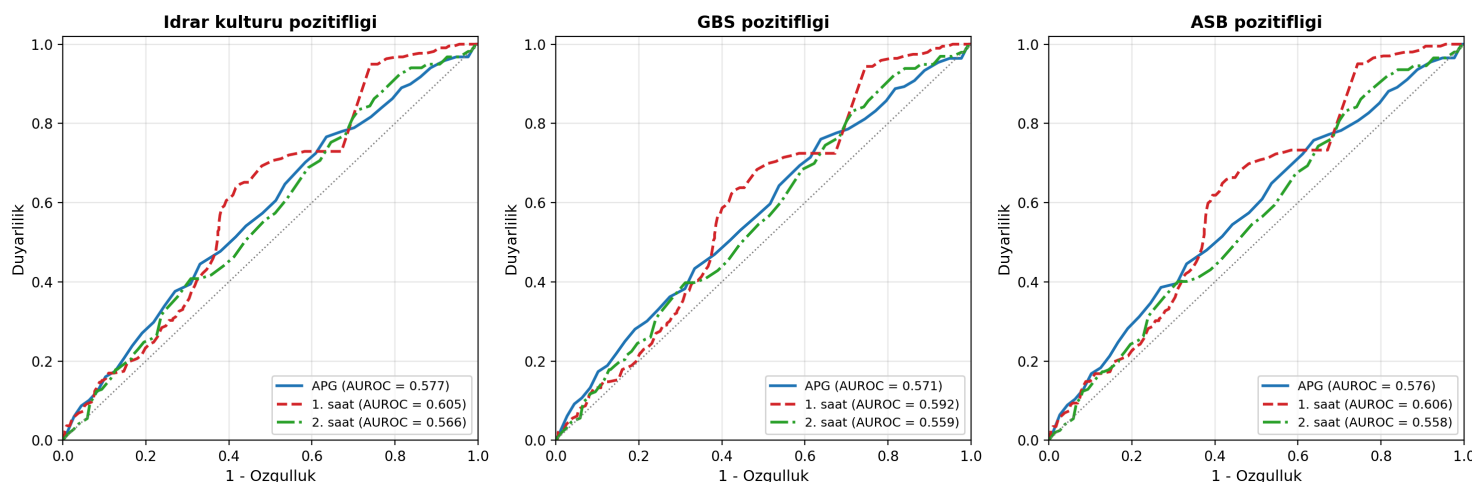


Figure 1

1a. ROC curves for urine culture positivity.

1b. ROC curves for GBS positivity.

1c. ROC curves for ASB positivity.

ROC curves for urine culture, GBS, and ASB positivity based on fasting plasma glucose (FPG), 1-hour, and 2-hour glucose values in the OGTT-negative subgroup. In each panel, the blue solid line represents FPG, the red dashed line represents 1-hour glucose, and the green dashed-dotted line represents 2-hour glucose.