ORIGINAL RESEARCH

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Prevalence of impaired glucose tolerance and its association with adverse perinatal outcomes in non-gestational diabetes pregnancies

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Abstract

Objective: Gestational diabetes mellitus (GDM) is characterized by glucose intolerance with onset during pregnancy and is one of the most common metabolic disorders complicating pregnancy. The aim of this study was to evaluate the risk of maternal and neonatal outcomes in non-gestational diabetes pregnancies with abnormal glucose challenge test (GCT) and abnormal glucose tolerance test (GTT) results.

Method: In this retrospective cohort study of 2982 singleton pregnancies, all patients underwent a non-fasting 50 g GCT at 24 to 28 weeks of gestation. A GCT cutoff of ≥ 140 mg/dl was selected. Women with an elevated GCT underwent prompt diagnostic testing with a 3-hour GTT. Subjects were divided into four groups according to GCT and GTT results.

Results: There was an impaired glucose tolerance in 19.2 % of patients and 14.7 % of them had mild glucose intolerance and 4.5 % of them had moderate glucose intolerance. As expected, there was statistically significant difference in fetal macrosomia, neonatal hypoglicemia, PE, primary CS, and preterm birth between secreening negative and GDM patients ($p < 0.0001$). We also observed statistically significant difference in neonatal hypoglicemia ($p = 0.0001$) and PE ($p = 0.0277$) between screning negative and mild glucose intolerance group. Moreover, there was a significant difference in fetal macrosomia ($p=0.0480$) between mild glucose intolerance and moderate glucose intolerance groups.

Conclusion: Compared with screening negative group, mild and moderate glucose intolerance are associated with increased adverse maternal and neonatal outcomes even in the absence of GDM.

Keywords: Gestational diabetes mellitus, Fetal macrosomia, Neonatal hypoglicemia, Cesarean section, Glucose intolerance

INTRODUCTION

Gestational diabetes mellitus (GDM) is characterized by glucose intolerance with onset during pregnancy and common metabolic disorders complicating pregnancy that affect mother and fetus (1). Its prevalence varies among different races and different ethnic groups dependent on their underlying risk of diabetes and approximately 4-17% of all pregnant women are affected by diabetes mellitus (DM) in pregnancy (2, 3).

There are several adverse outcomes for pregnant women and their fetuses associated with GDM. Complications include higher risk for preeclampsia (PE), preterm delivery, operative and cesarean delivery, shoulder dystocia, birth trauma, stillbirth, hydramnios, fetal macrosomia and large for gestational age (LGA) infant, neonatal intensive

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care unit (NICU) admission, perinatal mortality, neonatal respiratory problems, hyperbilirubinemia and hypocalcemia (4–12).

Adequate and efficient screening may prevent these maternal and fetal adverse outcomes. The purpose of GDM screening is to detect asymptomatic individuals. There is no universally accepted approach to screening for GDM nor even agreement on appropriate glucose thresholds at which gestational diabetes is diagnosed (13–16). There are many different strategies for the screening of GDM in pregnancy (17). The American College of Obstetricians and Gynecologists (ACOG) recommends a two-stage approach using cutoff of the Carpenter-Coustan criteria (1). The first step is the glucose challenge test (GCT) and the second step to screen positive patients is the 100-gram, three-hour oral glucose tolerance test (GTT), a diagnostic test for GDM. If two or more of the four values increase in the GTT, the patient is diagnosed with GDM.

Minor degrees of glucose intolerance in pregnancy, defined as mild or moderate glucose-intolerant state, intermediate between normal and GDM. The criteria used to classify glucose tolerance in pregnancy show some differences (18). In studies, these women's metabolic state are referred to impaired glucose tolerance (IGT), insulin resistance, carbonhydrate intolerance, gestational impaired glucose tolerance (G-IGT) and borderline gestational glucose intolerance (BGGI) (18–25).

It is obvious that patients with GDM are at increased risk for adverse obstetric and perinatal outcomes and treatment with close monitoring are required. However, adverse perinatal outcomes of insulin resistant group of patients who have abnormal 1-hour GCT with negative 3-hour GTT and have abnormal 1-hour GCT with one abnormal value on GTT as well as their management during and after pregnancy is controversial (1, 19–22, 26, 27).

This study aimed to investigate the rate of mild and moderate glucose intolerance in non-GDM pregnancies and their relationship with adverse maternal and neonatal outcomes..

METHOD

Study population

A total of 2982 single pregnant women of Turkish ethnic origin, aged between 18-48 years, between January 2013 and December 2016 were included in this retrospective cohort study. All subjects were divided into four groups according to GCT and GTT results; Group I (Screen negative subject, n=2304): GCT value ≤ 140 mg/dl. Group II (mild glucose intolerance, n = 438): GCT value ≥ 140 mg/dl, with normal GTT. Group III (moderate glucose intolerance, n=133): GCT value ≥ 140 mg/dl with one abnormal value on GTT, and Group IV (gestational diabetes mellitus, n=107): GCT ≥ 140mg/dl with two or more abnormal value on GTT, or GCT ≥ 200 mg/dl.

Patients who were diagnosed with multiple pregnancies, pre-gestational diabetes and GDM diagnosed before two step screening at 24-28 weeks of gestations were excluded from the study. Also, women who have negative OGTT results, but receiving diet and/or insulin therapy during follow up due to incident macrosomia and elevated fasting glucose and thus being classified as GDM were excluded. In addition, pregnant women who gave birth before the 20th gestational week and gave birth to babies weighing less than 500 grams were excluded from the study.

All procedures performed were in accordance with the ethical standards of the institutional review board and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Ethical approval was taken from the institutional Ethical Committee of the Balikesir University, School of Medicine (Date:19.10.2016/ registration number: 2016/94).

Glucose testing

All participants underwent a non-fasting 50 g GCT at 24 to 28w of gestation. Those with a GCT value of 200 mg/dl or higher are diagnosed as GDM. A GCT cutoff of ≥ 140 mg/dl was selected. Those with elevated GCT underwent prompt diagnostic testing with a fasting 100 g GTT. Blood samples were drawn 1, 2, and 3 hours after glucose intake. All tests were performed in outpatient clinics, during routine antenatal care. GDM was diagnosed in patients in whom two of the four values in the oral glucose tolerance test were found to be abnormal according to the Carpenter and Coustan criteria (28) (0h, 95 mg/dl; 1h, 180 mg/dl; 2h, 155 mg/dl; and 3h, 140 mg/dl). Pregnant women who did not have GDM on diagnostic testing returned to routine pregnancy follow-up.

Data collection

All data of patients were obtained from medical records. These data include demographic information, pregnancy complications, obstetric history, delivery process and outcomes, as well as neonatal outcomes.

Study outcomes

Maternal and neonatal outcomes were compared among the groups. Maternal outcomes were primary cesarean section (CS) and PE. Neonatal outcomes were fetal macrosomia, stillbirth, neonatal death, and neonatal hypoglicemia.

Gestational weeks were calculated according to the last menstrual period of all patients. If there was a 7-day or more difference between the gestational week calculated according to the fore-aft length distance measured in the first trimester ultrasound and the gestational week calculated according to the last menstrual date, the gestational week calculated by ultrasound was accepted (29). The preeclampsia was diagnosed with the current guideline of ACOG (American College of Obstetricians and Gynecologists). According to this guideline (21), hypertension (140/90 mmHg or higher blood pressure at least twice with an interval of at least 6 hours after 20 weeks of gestation), proteinuria (300 mg in 24-hour urine or ≥1+ with dipstick) were considered as preeclampsia (30). Macrosomia was regarded as birthweight above 4000 g (29). Neonatal Hypoglycemia was defined as neonatal glucose ≤ 1.6 mmol/l during the first 24 h after birth (31). According to international standards, death occurring at or after the 24th week of pregnancy is defined as stillbirth (32).

Statistical analysis

MedCalc Statistical Software version 19.2.1 (MedCalc Software Ltd, Ostend, Belgium; https://www.medcalc.org; 2020) was used for statistical analysis. A p-value of < 0.05 was considered statistically significant. The distribution of evaluated variables in four groups was studied by describing the mean ± standard deviation (SD) or median (minimum-maximum), where applicable. One-way analysis of variance (ANOVA) or Kruskal–Wallis test were used to analyse more than two independent groups. Levene's test was used to analyse variances. When the p value from one-way ANOVA or Kruskal–Wallis test statistics was statistically significant, the Scheffé test or Post-Hoc (Conover) analysis was used to determine which group differed from the others. Odds ratio (OR) and the 95% confidence intervals (CI) were calculated with univariate analysis. The Chi-square test was used to compare categorical data.

RESULTS

A total of 3336 pregnant women were evaluated between the study periods. 581 pregnancies were excluded, 93 (2.8 %) had overt diabetes, 164 (4.9 %) had twin pregnancies and 97 (2.9 %) had GDM diagnosed before 24 weeks of gestation (Figure 1). 2982 pregnant women who underwent GDM screening at 24 to 28 weeks of gestation were included in this study. We found that the total prevalance of GDM was 6.5 % (2.9 % diagnosed before the 24 weeks of gestation and 3.6 % diagnosed with two step screening between 24-28 weeks of gestation). On the other hand, there was an impaired glucose tolerance in 19.2 % of patients and 14.7 % of them had mild glucose intolerance and 4.5 % of them had moderate glucose intolerance. We also found that the rate of the fetal macrosomia, neonatal hypoglicemia, preeclampsia and preterm birth were 5.6 %, 4.2 %, 3.6% and 5.6 %, respectively. Additionally, the rate of the stillbirth was 0.5 % in our studied population. The demographic features of participants was summarized in Table 1.

Table 1 Demographic features of participants

Characteristics	Total patients	
Age (year), mean±SD	26.8 ± 5.6	
Parity, median (min-max)	1 (0-6)	
BMI (kg/m2), mean±SD	24.45 ± 3.63	
Gestational weeks at delivery	39w + 2d	
Newborn Sex		
Female	1488	49.9
Male	1494	50.1
Birth weight (gram) mean±SD	3248.2 ± 525.2	
Mild Glucose intolerance	438	14.7
Moderate Glucose intolerance	133	4.5
Gestational diabetes mellitus	204	6.5
Fetal macrosomia rate, n (%)	167	5.6
Neonatal hypoglicemia rate, n (%)	124	4.2
Preeclampsia rate, n (%)	108	3.6
Delivery type, n (%)		
Vaginal Delivery	1713	57.3
Cesarean Section	872	29.2
Assissted Vaginal Delivery	20	0.7
Primary Cesarean Section	387	12.8
Preterm Birth, n (%)	166	5.6
Stillbirth, n (%)	14	0.5

According to the present results, maternal age was significantly lower in screen negative group. However there were no differences between patients with mild glucose intolerance, moderate glucose intolerance and GDM. Maternal pre-pregnancy BMI were significantly higher in patients with GDM than those patients with screen negative and mild glucose intolerance. There were no differences in parity between the groups (Table 2).

We showed that there was a statistically significant difference between the groups in terms of fetal macrosomia, neonatal hypoglycemia, PE, primary cesarean section and preterm delivery rates ($p < 0.0001$, $p < 0.0001$, $p < 0.0001$, $p < 0.0001$ and $p = 0.0001$, respectively) (Table 2).

Table 2 Demographic and clinical characteristics of groups

Characteristics	Group 1 (n = 2304) Screen Negative		Group 2 (n = 438) Mild Glucose Intolerance		Group 3 (n = 133) Moderate Glucose Intolerance		Group 4 (n = 107)GDM		P value
	n	%	n	%	n	%	n	%	
Age (year), mean±SD	26.2 ± 5.5[a,b,c]		28.7 ± 5.4		28.2 ± 5.9		29.6 ± 6.1		< 0.0001[$]
<25	1118	48.5	125	28.5	43	32.3	33	30.8	
25-30	575	24.9	127	28.9	35	26.3	20	18.9	
30-35	418	18.1	117	26.7	35	26.3	31	28.9	
≥35	193	8.4	69	15.8	20	15.1	23	21.4	
Parity, median (min-max)	1 (0-6)		1 (0-4)		1 (0-5)		1 (0-6)		0.0528[#]
0	849	36.8	148	33.8	41	30.8	33	30.8	
1	1221	52.9	233	53.2	72	54.1	60	56.1	
2	174	7.6	31	7.1	12	9.0	9	8.4	
≥3	60	2.6	26	5.9	8	6.0	5	4.7	
BMI (kg/m2), mean±SD	24.4±3.6[c]		24.5±3.5[e]		25.1±4.1		25.7±3.3		< 0.0001[#]
<18.5	6	0.5	6	2.4	3	4.3	1	2.1	
18.5-25	735	57.6	118	47.4	29	42.0	20	41.7	
>25	536	41.9	125	50.2	37	53.6	27	56.2	
Gestational weeks at delivery									
<37	117	5.1 [c]	22	5.0 [e]	11	8.3	16	14.9	0.0001[#]
37-41	2072	89.9	395	90.2	114	85.7	91	85.1	
>41	115	5.0	21	4.8	8	6.0	0	0.0	
Newborn Sex									
Female	1166	50.6	212	48.4	61	45.9	54	50.5	0.3998[#]
Male	1138	49.4	226	51.6	72	54.1	53	49.5	
Birth weight (gram) mean±SD	3245.1±512.9[c]		3226.7±511.8[e]		3234.7±669.5		3400.8±558.0		0.0004 [#]
<2500	158	6.9	27	6.2	11	8.3	6	5.6	
2500-4000	2035	88.3	384	87.6	109	81.9	86	80.4	
>4000	111	4.8	27	6.2	13	9.8	15	14.0	
Fetal macrosomia, n (%)									
Yes	111	4.8 [b,c]	27	6.2 [d,e]	15	11.3	15	14.0	<
No	2193	93.2	411	(93.8	120	90.3	92	86.0	0.0001[#]
Neonatal hypoglicemia, n(%)									
Yes	63	2.7 [a,b,c]	28	6.4 [e]	14	10.5	19	17.8	<
No	2241	97.3	410	93.6	119	89.5	88	82.2	0.0001[#]
Preeclampsia, n (%)									
Yes	66	2.8 [a,c]	22	5.1 [e]	7	5.3	13	12.1	<
No	2238	97.2	416	94.9	126	94.7	94	87.9	0.0001[#]
Delivery type n (%)									
Vaginal Delivery	1358	58.9	218	49.8	71	53.4	56	52.3	<
Cesarean Section	673	29.2	155	35.4	33	24.8	11	10.3	0.0001[#]
Assissted Vaginal Delivery	12	0.5	2	0.5	2	1.5	4	3.7	
Primary Cesarean Section	261	11.3 [b,c]	63	14.4 [e]	27	20.3 [f]	36	33.6	
Preterm Birth, n (%)									
Yes	117	5.1 [c]	22	5.0 [e]	11	8.2	16	14.9	0.0001[#]
No	2187	94.9	416	95.0	122	91.8	91	85.1	
Stillbirth, n (%)									
Yes	10	0.4	3	0.7	0	0	1	0.9	0.6621[#]
No	2294	99.6	435	99.3	133	100	106	99.1	

ANOVA *Kruskal Wallis test, # Chi-Squared test
Data are presented mean ± SD or median (minimum-maximum)
a. Screen negative group versus mild glucose intolerance group ($p < 0.05$)
b. Screen negative group versus moderate glucose intolerance group ($p < 0.05$)
c. Screen negative group versus GDM group ($p < 0.05$)
d. Mild glucose intolerance group versus moderate glucose intolerance group ($p < 0.05$)
e. Mild glucose intolerance group versus GDM group ($p < 0.05$)
f. Moderate glucose intolerance group versus GDM group ($p < 0.05$)

Our subgroup analysis showed that the rate of fetal macrosomia was significantly lower in screening negative group than in patients with moderate glucose intolerance and GDM (adjusted Odds Ratio (aOR) (95% confidence interval (CI)): 2.14 (1.17-3.91) p = 0.0037 and aOR (95% CI): 3.22 (1.81-5.74) p < 0.0001, respectively). Also, compared with mild glucose intolerance patients, fetal macrosomia rate was significantly higher in patients with GDM (p =0.0064). Neonatal hypoglicemia rate was significantly lower in screen negative group than patients with mild glucose intolerance, moderate glucose intolerance and GDM (aOR (95% CI): 2.43 (1.54-3.84), p = 0.0001, aOR (95% CI): 4.18 (1,72-5,13), p < 0.0001, and aOR (95% CI): 7.68 (4.41-13.38), p < 0.0001, respectively). Moreover, neonatal hypoglicemia rate was significantly lower in patients with mild glucose intolerance than those and with GDM (p = 0.0002, respectively). PE rate was significantly lower in screening negative group than mild glucose intolerance and GDM group (aOR (95% CI): 1.79 (1.09- 2.94), p = 0.0277 and aOR (95% CI): 4.68 (2.49-8.80) p < 0.0001, respectively). Additionaly, PE rate was significantly lower in patients with mild glucose intolerance than those with GDM (p = 0.0133). Primary CS rate was significantly lower in screening negative group than patients with moderate glucose intolerance and GDM (aOR (95% CI): 1.98 (1.25- 3.14), p = 0.0029 and aOR (95% CI): 3.34 (2.16-5.19), p < 0.0001, respectively). Moreover, primary CS rates were significanly different in mild and moderate glucose intolerance groups than GDM (p < 0.0001 and p = 0.0287, respectively) (Table 3). Additionally, preterm birth rates were significantly lower in screening negative and mild glucose intolerance groups than GDM (p < 0.0001 and p = 0.0003, respectively). However, there was no difference in the rate of stillbirth between the groups (p = 0.6621) (Table 3).

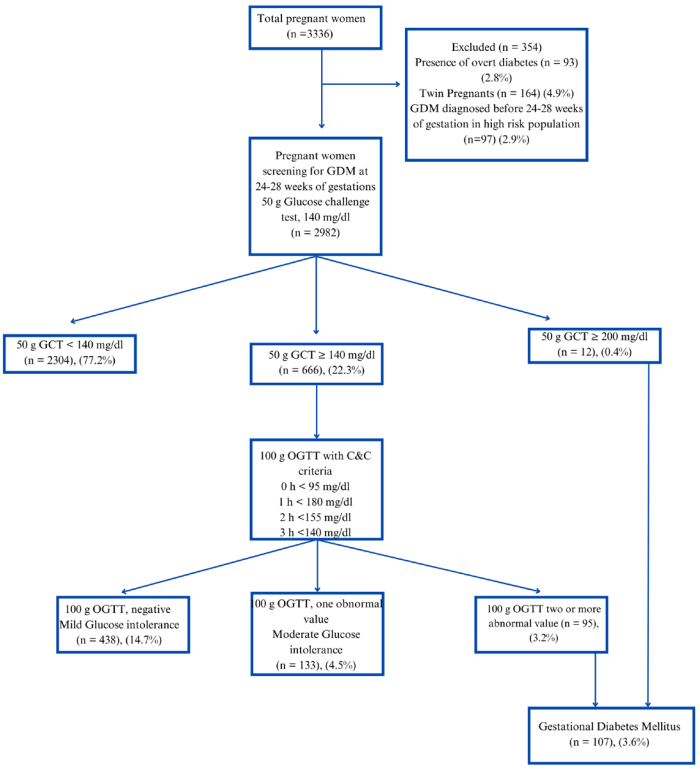

Figure 1. Flow chart showing recruitment of the study women and prevalence of glucose intolerance

Table 3 Comparable analysis of maternal and fetal outcomes of women in screening negative, mild glucose intolerance, moderate glucose intolerance and GDM groups.

Total cohort n=2.982	Group 1 (n = 2304) Screen Negative Group	Group 2 (n = 438) Mild Glucose Intolerance Group	Group 3 (n = 133) Moderate Glucose Intolerance Group	Group 4 (n = 107) GDM
	n aOR (95% CI)	n aOR (95% CI)	n aOR (95% CI)	n aOR (95% CI)
Fetal macrosomia	111/2193 Ref	27/411 1.30 (0.84- 2.00)	13/120 2.14 (1.17-3.91)	15/92 3.22 (1.81- 5.74)
Neonatal hypoglicemia	63/2241 Ref	28/410 2.43 (1.54-3.84)	14/119 4.18 (2.28-7.68)	19/88 7.68 (4.41-13.38)
Preeclampsia	66/2238 Ref	22/416 1.79 (1.09- 2.94)	7/126 1.88 (0.85- 4.19)	13/94 4.69 (2.50-8.80)
Primary Cesarean section	261/1358 Ref	63/218 1.50 (1.10-2.05)	27/71 1.98 (1.25- 3.14)	36/56 3.34 (2.16-5.19)
Preterm Birth	117/2187 Ref	22/416 0.99 (0.62-1.58)	11/122 1.69 (0.88-3.21)	16/91 3.29 (1.87-5.77)

Logistic regression included the outcomes; crude and adjusted (aOR); 95% confidence intervals (CI).
aOR, adjusted odds ratio, CI, confidence interval; GDM, gestational diabetes mellitus.

DISCUSSION

In this retrospective cohort study, we evaluated GDM screening results of pregnant women with mild glucose intolerance, moderate glucose intolerance and GDM. According to our present results, the prevalance of GDM and impaired glucose tolerance were 6.5% and 19.2%, respectively. Pregnant women with impaired glucose tolerance and GDM; demonstrated significantly higher adverse maternal and perinatal outcomes, including increased rate of fetal macrosomia, neonatal hypoglicemia, PE, primary CS and preterm birth.

The prevalence of GDM greatly alterable depending on population characteristics and the diagnostic criteria used. Previous studies demonstrated that the prevalence of GDM varies from 6% to 18% (2, 3) and is rising worldwide in line with increasing trends of maternal obesity, physical inactivity, and maternal age (4, 33). Comperable with these results, the total prevalance of GDM was 6.5% in our study population. Present result may be due to the fact that our participants are relatively young and underweight.

In literature, a number of studies have demonstrated that GDM is associated with increased rates of short and long-term advers maternal and fetal outcomes including fetal macrosomia, shoulder dystocia, birth injury, gestational hypertension, PE, CS, polyhydramnios, preterm birth, neonatal hypoglycemia, neonatal intensive care unit admission and respiratory distress (4–6, 8, 9). Comparable with these results, we found that compared to screening negative group, patients with GDM had increased rate of primary CS, fetal macrosomia, neonatal hypoglicemia, PE and preterm birth. In the present study, to reduce probability of errors, we screened high risk pregnancies with maternal age ≥ 25 years, family history of diabetes, previous macrosomic babies or stillbirth in the first trimester with fasting plasma glucose, random plasma glucose, HbA1c, and 75-g 2-hour OGTT and those patients with diabetes (n = 97, 2.9%) were excluded from the study.

Previous studies have revealed that mild to moderate glucose intolerance is associated with increased rates of adverse pregnancy outcomes such as shoulder distosia, fetal macrosomia, PE, neonatal hypoglicemia, admission of neonatal intensive care unit, preterm birth and cesarean delivery in non-diabetic population (5, 15, 18, 21). Temming et al. compared screening negative patients with screening positive patients who had one abnormal GTT value without GDM had increased risk of pregnancy-induced hypertension (PIH), PE, cesarean delivery, and macrosomia (18). Similarly, Metzger et al. showed a strong correlation between mild or severe hyperglycemia without GDM and increased rates of large for gestational age (LGA), primary CS, shoulder dystocia, PE and elevated cord blood c-peptide levels in Caucasian and Asian women (34). In another study conducted by Dodd et al., it is reported that Australian women with an elevated 1h 50-g GCT and mild glucose intolerance but no GDM on a 2h 75-g GTT had raised risks of shoulder dystocia, PE and neonatal hypoglycemia (21). Landon et al. compared women with a normal 1-hour 50-gram screening test with women with varying degrees of insulin resistance. There were increasing rates of cord blood c-peptide, hypoglycemia, hyperbilirubinemia, LGA, birth trauma and shoulder dystocia across increasing groups with insulin resistance (35). Comperable with these results, we found a linear relationship between presence of mild or moderate glucose intolerance and the rate of pregnancy complications such as primary CS, fetal macrosomia, neonatal hypoglycaemia, PE and preterm birth in non-GDM Turkish pregnants.

However, results of some studies investigating the relationship between GDM and perinatal death were varied. A cohort study conducted by Billionet et al. showed that compared with non-diabetic population, the perinatal mortality was significantly higher in patients with GDM (6). Contrary to these results, some recent large-population based cohort studies demonstrated that perinatal mortality in offsprings from GDM mothers was significantly lower than or similar to the non-diabetic population (9, 36). We found no significant difference between the groups in terms of results for stillbirth. This may be associated with small sample size of participants or exclusion of high risk pregnancies diagnosed as diabetes before 24 weeks of gestations.

In literature, different groups and societies recommend different approaches and criteria for screening and diagnosis of GDM (1, 33, 37–40). Most common known screening strategies are one step 75g 2h test using The International Association of Diabetes and Pregnancy Study Groups (IADPSG) criteria (33) and two step 50g 1h followed for abnormals by 100gr 3h test using C&C criteria (37, 40). In clinical practice, two step aproaches are commonly used in our country for screening and diagnosis of GDM. In order to control blood glucose levels, pregnant women with GDM are recommended a healty diet, oral anti-diabetics or insulin use during pregnancy according to the guideline. However, for other women who is screening negative, screening positive and 100g negative, or screening positive and one abnormal value in GTT, the routine prenatal care is suggested as recommended by the guideline (37, 40). As seen in the results of previous studies and the present study, pregnancy outcomes of patients with negative screening and patients with mild glucose intolerance or moderate glucose intolerance are different and new strategies or approaches are needed to optimize prenatal care in these patient groups.

Limitations of the study

The present study has some limitations. The main limitations were retrospective study design and relatively

small number of patients included in the studied population. Additionally, collection of all data and accounting for all potential confounding variables are not possible and there was some missing data of BMI even though they were included in the analysis.

CONCLUSION

In conclusion, according to the present results, it is obvious that there are two separate groups between screening negative patients and GDM in terms of GDM screening and presence of pregnancy complications. The present study suggests further studies to prevent or minimize pregnancy complications in these groups.

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Peer-Review

Both externally and internally peer reviewed.

Conflict of interest

The authors declare that they have no conflict of interests regarding content of this article.

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Ethical Declaration

Ethical permission was obtained from the Balikesir University Research Ethics Committee for this study with date 19.10.2016 and number 2016/94, and Helsinki Declaration rules were followed to conduct this study.

Author Contribution

Concept: AU, MH, CBBH, CSU, Design: AU, CSU, Supervising: AU, MH. CSU, Financing and equipment: AU, MH, CBBH, CSU, Data collection and entry: AU, CBBH, CSU, Analysis and interpretation: AU, MH, CBBH, CSU, Literature search: AU, MH, CBBH, CSU, Writing: AU, MH, CBBH, CSU, Critical review: AU, MH, CBBH, CSU.

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