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Non-Alcoholic Fatty Liver Disease in The First Trimester and Subsequent Development of Gestational Diabetes Mellitus

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Abstract

The clinical importance of non-alcoholic fatty liver disease (NAFLD) in pregnant women has not been adequately established, despite the abundance of data linking it to altered glucose homeostasis. The aim and objectives were to examine the link between NAFLD in the first trimester of pregnancy and gestational diabetes mellitus (GDM). Subjects and methods were 50 singleton pregnant women who received prenatal care in the first trimester at Beni-Sweef General Hospital obstetrics and gynaecology department were included in this cross-sectional. In pregnant women, high-grade steatosis increases the risk of developing gestational diabetes mellitus (GDM). The chance of developing GDM in the middle of pregnancy increases when NAFLD is present in the early stages of pregnancy. To determine and describe the underlying processes causing this connection, further mechanistic research is required.

Keywords: Fatty liver disease, Gestational diabetes mellitus, Insulin resistance, Non-alcoholic.

1. Introduction

Fatty liver disease caused by nonalcoholic steatohepatitis is a common cause of liver failure. As many as 10% to 40% of adults in both Eastern and Western countries are affected by it, according to studies [**1].**
Nonalcoholic fatty liver disease (NAFLD) is defined as the absence of other causes of fat deposition, including excessive alcohol consumption, drugs (such as methotrexate), or viruses, and the presence of more than 5% of hepatocytes with lipid droplets (e.g., hepatitis C) [**2].** Although the molecular pathways that explain these connections remain a mystery. Depending on a person's ethnicity or race, the relationship between NAFLD, obesity, and insulin resistance seems to be more prevalent [**3].**
Obese or overweight pregnant women are known to have a higher risk of unfavorable maternal and fetal/neonatal outcomes [**4].**
It was speculated that NAFLD might increase the risk of gestational diabetes mellitus (GDM). Numerous studies have linked non-pregnant women with a history of GDM with NAFLD [**5].**
Even while recent research has established a link between NAFLD and GDM, there is still a lack of knowledge on this relationship among Asian people, and this has not been well investigated [**6].**

It is generally known that obstetric problems such hypertensive disorders, gestational diabetes mellitus (GDM), caesarean sections (CS), and increased postpartum weight retention are linked to a high pre-pregnancy BMI (PPWR). Miscarriage, heart problems, macrosomia, stillbirth, and neural tube disorders are among the fetal hazards.

The incidence of NAFLD in pregnant women and the results of pregnancies have been the subject of several investigations. An NAFLD diagnosis in the first trimester is linked to a diagnosis of hyperglycemia in the second trimester. NAFLD has also been linked to an increased risk of pregnancy complications such as gestational diabetes, caesarean sections, preeclampsia, and low birth weight. It was shown that pregnant women with NAFLD had a two-fold increased risk of pre-eclampsia and gestational hypertension [**7].**

The purpose of the research was to ascertain how early-onset NAFLD and later-onset gestational diabetes mellitus are related (GDM).

2. Patients and Methods

The Obstetrics and Gynecology Department at Beni-Sweef General Hospital conducted this prospective observational cross-sectional research. 285 women were recruited for the study, we chose 74 pregnant women for the study and excluded the rest, and there were only 50 singleton expectant mothers in total who sought prenatal treatment before 14 weeks of pregnancy who gave consent and completed the study, and all pregnant women have non-alcoholic fatty liver.

2.1 Inclusion Criteria

Ladies who are pregnant and have a straightforward singleton pregnancy in the first trimester who have non-alcoholic fatty liver.

2.2 Exclusion criteria

Pregnant women with known liver conditions, including hepatic steatosis-causing medicines, autoimmune and viral hepatitis, cholangitis, and inborn metabolic problems (corticosteroids, amiodarone, tamoxifen and valproate). pregnant ladies having a history of hepatitis or severe fatty liver from pregnancy. Patients with long-term illnesses such as type 2 diabetes, hypertension during pregnancy, severe heart disease, chronic renal disease, malignancies, and connective tissue diseases and also pregnant women with recent gestational diabetes mellitus.

Exclusion was carried out in accordance with any exclusion criteria that were indicated by a medical history, medical examination, and routine laboratory tests.

2.3 Sample size

50 singleton pregnant women. As prevalence of NAFLD 18.4% (Lee et al., 2019) and the expected number of pregnant females attending to the department at Bani-Sweef General Hospital is 340 so the sample Size is 50. Sample Size was calculated using Open Epi software with confidence limit 5% and design effect 1.

2.4 Methods

A thorough history was obtained, paying particular attention to the following personal information: age, marital status, parity, residence, employment, and any unusual behaviors. **Menstrual history:** with a focus on the timing and regularity of periods and last menstrual period. **Obstetric history:** abortion rate, parity, prior pregnancy result, delivery method, and any postpartum complications. **Past history of any medical condition, such as** "hypertension, diabetes mellitus, and deep venous thrombosis (DVT)," "allergy to certain medicines," and "any prior surgeries, including cesarean section" (CS) **Clinical examination**: Vital signs: Blood pressure, pulse and temperature, weight, height, BMI, abdominal examination for assessment of fundal level and fetal heart sounds.

2.5 Assessment of Fetal Well-Being

Abdominal ultrasound is used to determine the viability of the pregnancy, as well as to determine the gestational age, the fetus's biometric measurements, the location of the placenta, the amniotic fluid index, and the fetal weight estimation.

2.6 Lab Assessment

Complete blood count, liver functions (SGOT, SGPT, Serum albumin, Total bilirubin), Urine Analysis, particularly for proteinuria, kidney function tests, fasting insulin, fasting blood glucose, HA1C, and lipid profile are among the laboratory tests that are performed. Insulin Resistance Homeostasis Model Assessment Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) All subjects' levels of insulin resistance were calculated using the following equation: Fasting insulin concentration (mU/mL) x fasting blood sugar (mg/dL) / 405.

2.7 Diagnosis and Grading of NAFLD

Abdomen Ultrasonography Evaluation: To validate the existence of NAFLD, abdominal ultrasound using GE logic P7 - Convex probe frequency 1-5 MH was used to analyze all of the chosen subjects. Grading of Fatty Liver: Fatty liver was divided into four classes based on the observations of liver brightness, hepatorenal echo contrast, profound attenuation of the ultrasound signal, and vascular blurring.

• **Grade 0 (Normal):** The echogenicity of the liver parenchyma is comparable to or slightly greater than that of the renal cortex and spleen in healthy individuals. With fine low-level echos, the echo texture is also homogeneous.

• **Grade I (Mild steatosis)**: Clear demarcation of the hepatic and portal vein walls, somewhat enhanced liver echogenicity.

• **Grade II (Modest steatosis):** Hepatic and portal vein branches' echogenic walls are obscured by a moderate rise in liver echogenicity. The diaphragm's echogenic line is clearly visible.

• **Grade III (severe steatosis):** Significantly reduced visibility of the hepatic artery walls, diaphragm, and posterior right lobe echogenicity, as well as impaired vision of the diaphragm and liver distinctions. All individuals' BMIs were calculated using the following equation: (Body Weight in Kilograms) Equals BMI (height in meters) [2].

2.8 Evaluation of Pregnancy Outcomes

Results of all chosen participants' pregnancies were noted with reference to the onset of gestational diabetes mellitus.

2.9 Operational design:

All study participants were introduced to the researcher, who then requested their participation after briefly outlining the study's objectives. Everyone who took part in the research was given thorough information about the goal and anticipated benefits. The whole project was conducted with all ethical issues in mind.

2.10 Ethical Committee:

Additionally, authorization from the hospital's ethical committee for conducting human research and institutional review board clearance was acquired.

2.11 Statistical analysis

Version 20 of the Statistical Program for Social Science was used for data analysis (SPSSInc, Chicago, IL, USA). Quantitative variables were described using the mean and standard deviation. In order to convey a sense of quality, we relied on numerical data and percentages. When comparing parametric quantitative variables between two groups, the student t-test was used. The chisquare (X2) test or Fisher's exact test was employed to compare qualitative variables when frequencies dropped below five. In order to evaluate the relationship between two normally distributed variables, Pearson correlation coefficients were utilized. A P value less than 0.05 is

regarded as significant when a variable was not normally distributed.

3. Results

The average age of the group being investigated was 28.36 (±4.05 SD), and 15 (30%) of them had junk food as one of their distinctive habits. Of the group, 38 (76 %) were urban dwellers and 12 (24 %) were rural. Table .2 shows that 50% of them had grade 1 steatosis, and 100% had fatty liver. Table .4 demonstrates that all of the examined patients had fatty liver, with 46 (92%) of the cases having Grade 1 steatosis, 3 (6%) having Grade 2 steatosis, and 1 (2%) having Grade 3 steatosis.

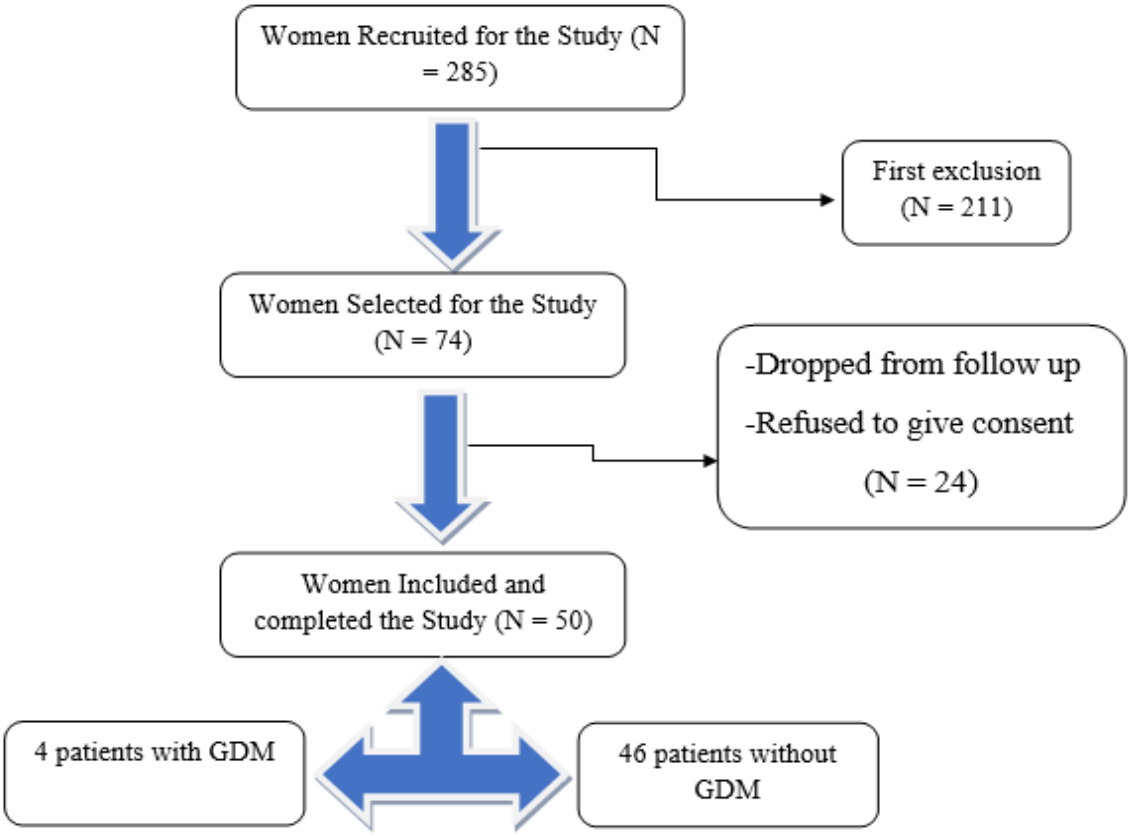

Figure (1): Flowchart of included patients.

Table (1): Demographic data of studied cases.

Demographic data	Studies Cases	
Age (years)		
Range.	22.0 – 35.0	
Mean ± SD.	28.36 ± 4.05	
Residence	No.	%
Urban	38	76.0
Rural	12	24.0
Occupation	No.	%
Housewife	30	60.0
Employee	20	40.0
Special habits	No.	%
Non	35	70.0
Junk food	15	30.0

Table (2): Liver Ultrasound outcome of GDM cases.

Ultrasound outcome	Cases	
Steatosis	No.	%
Grade 1	2	50.0
Grade 2	1	25.0
Grade 3	1	25.0
Presence of fatty liver	No.	%
Yes	4	100.0
No	0	0.0

Table (3): Lab investigations of GDM cases at 24 - 28 weeks.

Lab investigation		GDM
	Mean ± SD.	**Range.**
AST (U/l)	22.25 ± 1.71	20.0 – 24.0
ALT (U/l)	19.75 ± 4.27	15.0 – 25.0
Cholesterol (mmol/l)	4.93 ± 0.64	4.07 – 5.52
HDL-cholesterol (mmol/l)	1.49 ± 0.19	1.23 – 1.66
LDL cholesterol (mmol/l)	2.38 ± 0.30	2.09 – 2.70
Triacylglycerol (mmol/l)	1.64 ± 0.31	1.35 – 1.93
GGT (U/l)	19.25 ± 2.75	16.0 – 22.0
Fasting glucose (mg/dL)	116.0 ± 2.94	113.0 – 120.0
Insulin (mU/mL)	19.05 ± 7.13	11.0 – 25.30
HOMA-IR	5.43 ± 1.97	3.30 – 7.20

Table (4): Liver Ultrasound outcome of studied cases (Steatosis grade and presence of fatty liver).

Ultrasound outcome	Cases	
Steatosis	No.	%
Grade 1	46	92.0
Grade 2	3	6.0
Grade 3	1	2.0
Presence of fatty liver	No.	%
Yes	50	100.0
No	0	0.0

Table (5): Gestational diabetes mellitus prevalence in studied cases.

GDM	Cases	
Yes	4	8.0
No	46	92.0

Table .5 reveals that 4 (or 8%) of the patients under study had gestational diabetes mellitus. Table .6 shows that GDM mean age was 27.75 ± 6.65 years, mean BMI was 31.95 ± 1.79 kg/m^2, 75% of them were urban, 75% were housewife, and 100% were primi gravida. Meanwhile, the mean GA was 12.5 ± 1.91 weeks. Table .7 shows that there was no statistically significant difference in the obstetric history between the groups with and without gestational diabetes. Table .8 shows there was no statistically significant difference in the data between the groups with and without GDM, as shown in the following table. Table .9 shows the data in this table shows that there was a statistically significant difference in BMI between the GDM and non-GDM groups. (Group with GDM had a higher BMI).

Table (6): Demographic data of the GDM cases.

Demographic data	**GDM Cases**	
	Mean ± SD	**Range**
Age (years)	27.75 ± 6.65	22.0 – 34.0
BMI	31.95 ± 1.79	30.4 – 34.0
Gestational age (Weeks)	12.50 ± 1.91	10 - 14
Residence	No.	%
• **Urban**	3	75.0
• **Rural**	1	25.0
Occupation	No.	%
• **Housewife**	3	75.0
• **Employee**	1	25.0
Junk food	3	75.0
Primi-gravida	4	100

Table (7): Comparison between cases with and without GDM regarding Obstetric history.

Obstetric history	No GDM		GDM		Test	P
Gravidity	No.	%	No.	%		
Primi	17	37.0	4	100.0	χ^2=6.0	0.111
2	12	26.1	0	0.0		
3	9	19.6	0	0.0		
≥4	8	17.4	0	0.0		
Parity	No.	%	No.	%		
0	17	37.0	4	100.0	χ^2=6.0	0.111
1	15	32.6	0	0.0		
2	7	15.2	0	0.0		
≥3	7	15.2	0	0.0		
Previous abortion	No.	%	No.	%		
Yes	5	10.9	0	0.0	χ^2=0.483	0.487
No	41	89.1	4	100.0		
Gestational age (Weeks)						
Range.	10.0 – 14.0		10.0 – 14.0		t=0.418	0.908
Mean ± SD.	12.41 ± 1.39		12.50 ± 1.91			

t: Student t-test, χ^2: **Chi-square test**, p: p-value for comparing between the studied groups**,** *: Statistically significant at $p \leq 0.05$

Table (8): Comparison between cases with and without GDM regarding past history.

Past history	No GDM		GDM		χ^2	p
Family history of diabetes	No.	%	No.	%		
Yes	10	20.0	0	0.0	1.087	0.297
No	36	80.0	4	100.0		
History of prior GDM	No.	%	No.	%		
Yes	1	2.0	0	0.0	0.493	0.499
No	45	98.0	4	100.0		

χ^2: Chi-square **test**, p: p-value for comparing between the studied groups, *: Statistically significant at $p \leq 0.05$.

Figure (9): Comparison between cases with and without GDM regarding examination.

Examination	No GDM	GDM	t	P
Systolic BP				
Range.	110.0 – 130.0	110.0 – 130.0	0.691	0.493
Mean ± SD.	118.04 ± 8.33	115.0 ± 10		
Diastolic BP				
Range.	60.0 – 80.0	70.0 – 80.0	1.754	0.086
Mean ± SD.	70.43 ± 7.88	77.50 ± 5.0		
BMI				
Range.	24.30 – 31.70	30.4 – 34.0	2.982	0.004*
Mean ± SD.	28.25 ± 2.41	31.95 ± 1.79		

t: Student t-test, p: p-value for comparing between the studied groups, *: Statistically significant at $p \leq 0.05$.

According to Table 10, GDM patients had higher levels of ALT, GGT, fasting glucose, Insulin, and HOMA-IR than those without GDM, as well as higher levels of HDL-cholesterol, Triacylglycerol, and Triacylglycerol in their blood, compared to those without GDM.

Figure (10): Comparison between cases with and without GDM regarding lab investigation at 24 - 28 weeks.

Lab investigation	No GDM	GDM	T	p
AST (U/l)				
Range.	12.0 – 22.0	20.0 – 24.0	3.040	0.004*
Mean ± SD.	17.11 ± 3.32	22.25 ± 1.71		
ALT (U/l)				
Range.	8.0 – 18.0	15.0 – 25.0	4.059	<0.001*
Mean ± SD.	12.85 ± 3.18	19.75 ± 4.27		
Cholesterol (mmol/l)				
Range.	3.72 – 5.17	4.07 – 5.52	1.902	0.063
Mean ± SD.	4.50 ± 0.42	4.93 ± 0.64		
HDL-cholesterol (mmol/l)				
Range.	1.31 – 2.10	1.23 – 1.66	2.049	0.046*
Mean ± SD.	1.75 ± 0.25	1.49 ± 0.19		
LDL cholesterol (mmol/l)				
Range.	1.60 – 2.66	2.09 – 2.70	1.261	0.213
Mean ± SD.	2.17 ± 0.31	2.38 ± 0.30		
Triacylglycerol (mmol/l)				
Range.	0.80 – 1.69	1.35 – 1.93	2.863	0.006*
Mean ± SD.	1.25 ± 0.26	1.64 ± 0.31		
GGT (U/l)				
Range.	9.0 – 16.0	16.0 – 22.0	5.347	<0.001*
Mean ± SD.	12.57 ± 2.37	19.25 ± 2.75		
Fasting glucose (mg/dL)				
Range.	69.0 – 95.0	113.0 – 120.0	9.921	<0.001*
Mean ± SD.	84.87 ± 6.17	116.0 ± 2.94		
Insulin (mU/mL)				
Range.	6.20 – 15.0	11.0 – 25.30	5.755	<0.001*
Mean ± SD.	10.11 ± 2.47	19.05 ± 7.13		
HOMA-IR				
Range.	1.20 – 3.50	3.30 – 7.20	8.596	<0.001*
Mean ± SD.	2.12 ± 0.57	5.43 ± 1.97		

Steatosis levels differed significantly between groups with and without GDM, according to this table. Pregnant women with high-grade steatosis were more likely to develop gestational diabetes mellitus (GDM). This table shows that there was a statistically significant difference between GDM and no GDM groups as regard Steatosis P<0.001*. Steatosis is a good predictor of GDM with Sensitivity 94.1 %, Specificity 97.1 %, Positive predictive value 98.8 % Negative predictive value 87.8 %.

Figure (11): Comparison between cases with and without GDM regarding Liver ultrasound outcome.

Ultrasound outcome	No GDM		GDM		χ^2	p
Steatosis	No.	%	No.	%		
Grade 1	44	92.0	2	50.0	14.950	0.001*
Grade 2	2	6.0	1	25.0		
Grade 3	0	0.0	1	25.0		
Presence of fatty liver	No.	%	No.	%		
Yes	46	100.0	4	100.0	—	—
No	0	0.0	0	0.0		

χ^2: **Chi-square test**, p: p-value for comparing between the studied groups, *: Statistically significant at $p \leq 0.05$

Figure (12): Validity (AUC, sensitivity, specificity) for steatosis to predict gestational DM.

	AUC	P Value	95% C. I	Sensitivity	Specificity	PPV	NPV
Steatosis to predict GDM	0.922	<0.001*	0.836-1.00	0.941	0.971	0.988	0.872

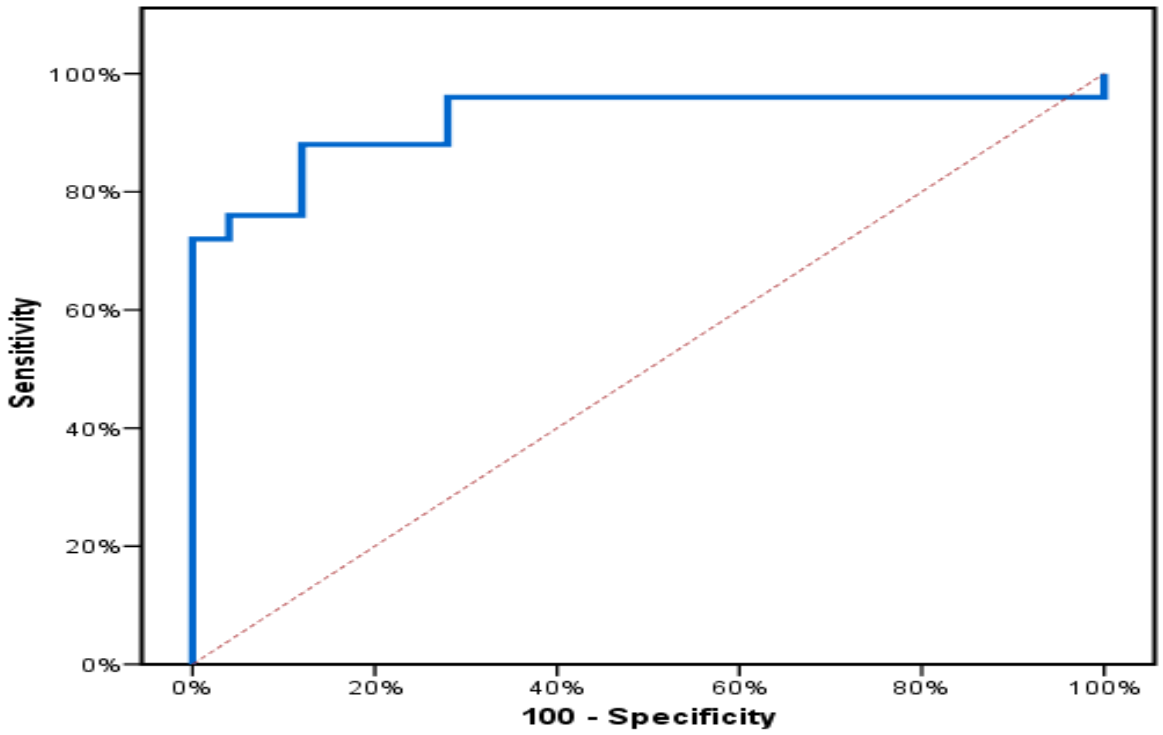

Figure (2): ROC curve for steatosis to predict gestational DM.

4. Discussion

In the absence of any secondary causes of steatosis, an increase in the liver's fat content is referred to as non-alcoholic fatty liver disease (NAFLD). Along with rising rates of obesity, metabolic syndrome (MetS), and type 2 diabetes, NAFLD prevalence is also rising [**8].**

Pregnancy-related gestational diabetes was shown to be associated with early-stage nonalcoholic fatty liver disease (NAFLD) (GDM).

In this study, we investigated the relationship between NAFLD in first trimester and subsequent development of GDM as the study done on 50 women who had NAFLD by antenatal care by doing pelvi-abdominal ultrasound and follow-up later at 24-28 weeks of gestation for development of GDM or not.

We found that 4 women developed GDM and 46 didn't develop GDM. This follow-up up done by fasting glucose and OGTT at 24-28 weeks of gestation.

By the comparison between cases with and without GDM, we found that. GDM and non-GDM groups did not have substantially different maternal ages, despite the fact that advanced maternal age has been associated to an increased risk of developing GDM. Pregnant women who are 40 years of age or older are more at risk for GDM. We may not have been able to show this difference since there were so few women above the age of 40 in our research, which had a mean maternal age of 28.41 3.86 years.

Deng et al. [9] found that Between the GDM and Non-GDM groups, there was no statistically significant difference in maternal age (33.14.9 vs. 32.64.4 years, $p>0.05$).

In this research, we showed that there were statistically significant differences in particular habits between the GDM and Non-GDM groups (junk food consumption was higher in GDM group).

Deng et al. [9] examined the link between gestational diabetes and non-alcoholic fatty liver disease and discovered that women with gestational diabetes (GDM) consumed more fat in their diets than women who did not have the condition.

Davis et al. [10] showed direct associations for high intakes of fried food and soda intake with GDM population.

Lamyian et al., [11] found that Fast food intake was observed to have negative impacts on the prevalence of GDM in women of reproductive age.

According to this study, there was no statistically significant difference in obstetric history between groups with and without gestational diabetes.

Lee et al., [12] found that in research to assess the relationship between non-alcoholic fatty liver disease in the first trimester and the emergence of gestational diabetes. With a p-value of 0.864, he discovered that there was no statistically significant difference in obstetric history between the GDM and no GDM groups.

There was no statistically significant difference in the obstetric history and past history between the groups with and without GDM.

Lee et al., [12] found that There was no statistically significant difference in the past between the GDM and no GDM groups, with a p-value of 0.215.

In this study, regarding examination, there was a statistically significant difference in BMI between two groups (group with GDM had a higher BMI).

GDM and NAFLD have been shown to be linked in previous research. After controlling for BMI, **De Souza et al. [13]** observed that NAFLD in the first trimester of pregnancy was an independent predictor of mid-pregnancy dysglycemia in women. When a pregnant woman's BMI rose, she was more likely to be diagnosed with NAFLD in the early stages of pregnancy in the United States.

According to the lab investigations, GDM patients had higher levels of ALT, GGT, fasting glucose, insulin, and HOMA-IR

than those without GDM. As well as higher levels of HDL-cholesterol, triacylglycerol in their blood compared to those without GDM.

Ajmera et al., [14] found decreased insulin sensitivity, but not insulin secretion, was independently related with NAFLD, as shown by higher baseline HOMA-IR (aOR 1.56, 95% confidence interval 1.2-2.04) and slightly higher baseline triglycerides (aOR 1.05, 95% confidence interval 1.01-1.11) in individuals who developed NAFLD.

By ultrasound we found that steatosis levels differed significantly between groups with and without GDM as women with high grade steatosis were more likely to develop GDM so, steatosis is a good predictor of GDM.

Chai et al., [15] determined that compared to pregnant women without NAFLD, pregnant women with NAFLD had a greater risk of developing GDM (pooled OR 2.9, 95% CI 1.0–8.4, $p < 0.05$,).

Hershman et al., [16] found that GDM risk was greater in women with NAFLD (adjusted relative risk [aRR], 2.78; 95% CI, and 1.25-6.15).

There are some limitations to the present investigation. In the beginning, liver ultrasonography rather than a histological investigation was used to make the diagnosis of NAFLD. However, in asymptomatic pregnant women, the histological diagnosis of NAFLD does not seem to be possible. Particularly in the lower range of hepatic steatosis of 10–15%, liver ultrasonography is a subpar diagnostic tool for NAFLD. Second, since the association between NAFLD and GDM may differ depending on ethnicity, the findings of our investigation should be confirmed in different ethnic groups.

5. Conclusion

In conclusion, early pregnancy NAFLD is linked to a higher risk of gestational diabetes mellitus (GDM) development. To determine and describe the underlying processes causing this connection, further mechanistic research is required.

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Conflicts of interest: No competing interest

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