ASSOCIATION OF NON ALCOHOLIC FATTY LIVER WITH TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is by far the commonest cause of chronic liver disease in the developed countries. In type 2 diabetes mellitus (T2DM), NAFLD has even more aggressive course and can result in early onset chronic liver disease. Although biopsy remains the gold standard for diagnosing NAFLD, many noninvasive tests such as liver ultrasound can give a clue about the severity of the disease. This study was conducted to determine NAFLD prevalence in patients with T2DM using liver ultrasound and determine its association with the body mass index and other biochemical markers (such as liver transaminases, glycated hemoglobin HbA1c, and lipid profile).

Methods: This cross-sectional study was carried out at Azadi General Teaching Hospital from January to September 2019. All the involved patients were known to have T2DM. After being consented, their body mass index (BMI) was determined, and patients were classified into mild, moderate, and severe fatty liver based on ultrasonographic criteria. Then, the biochemical blood measurements were performed by a standard laboratory procedure to determine their lipid profile, liver transaminases, and glycated hemoglobin levels.

Results: One hundred thirty diabetic patients were involved in the study. Around 55% were overweight, and 34% were obese. Fatty liver was seen in 53.7% (74 patients). Among these, mild, moderate and severe NAFLD was seen in 79.9%, 17.7% and 4.35%, respectively. Fatty liver diabetics had a mean BMI of 32.09% vs. 27.59% for patients with non-fatty liver. The average mean HbA1c, triglyceride and GPT levels in fatty liver and non fatty liver diabetics were 8.37 % vs. 7.82 %, 200mg/dl vs. 150mg/dl and 24.4 IU/L vs. 20.4 IU/L, respectively.

Conclusion: The overall prevalence of NAFLD among type 2 diabetes mellitus patients is significantly high. Elevated GPT, triglyceride and HbA1c levels may correlate with the development of NAFLD in diabetic patients.

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Keywords: Diabetes mellitus type 2, Glycated hemoglobin, Lipid profile, Liver transaminases, Non-alcoholic fatty liver disease.

Non-alcoholic fatty liver disease (NAFLD) is a growing global public health problem; about a third of adults might be affected in developed countries. The disease incorporates clinically and histologically different non-alcoholic entities; fatty liver (NALF, steatosis hepatitis) and steatohepatitis (NASH-characterised by hepatocyte ballooning and lobular inflammation ± fibrosis), which might lead to cirrhosis and, eventually, end-stage liver disease and rarely to hepatocellular cancer[1].

The majority of patients with NAFLD are

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asymptomatic and typically discovered when abnormal liver functions are obtained on routine laboratory evaluation. In particular, the liver enzymes alanine aminotransferase and aspartate aminotransferase are elevated. However, the level of these enzymes does not reliably predict the degree of inflammation and cirrhosis in all cases of NAFLD because their levels may not be increased in all patients with the disease[2].

Imaging techniques, such as liver ultrasonography or MRI, can give insight into the extent of hepatic involvement in NAFLD but also do not differentiate effectively between NAFL and NASH[3]. Additional noninvasive measures of liver inflammation and fibrosis are under investigation, including levels of circulating cytokeratin-18 fragments, measures of a pool of fibrosis markers, and transient elastography as a measure of liver stiffness[3,4]. However, histological analysis of tissue obtained by liver biopsy will remain the definitive diagnosis of NAFLD, which can assess the degree of liver inflammation and fibrosis[4].

The is an increase in the Prevalence of NAFLD worldwide and about 34%-46% of the obese population in developed countries have NAFLD.5 It is well known that the Prevalence of NAFLD is strongly related to several risk factors, including obesity, metabolic syndrome, insulin resistance and type 2 diabetes[6,7]. There is a robust association between NAFLD and diabetes risk. The chance of developing diabetes is increased approximately 5-fold in the presence NAFLD[8,9]. This association could be explained by dyslipidemia, insulin resistance and hepatic triglyceride (TG) accumulation in NAFLD and defective B-cell in type 2 diabetes mellitus[7]. Non-alcoholic fatty liver disease and its complications are responsible for mortality among a proportion of type 2 diabetic patients.10 NAFLD appears to enhance the risk for type 2 DM. In turn, type 2 diabetes may contribute to NAFLD progression[11].

There is a high likelihood that those patients with NAFLD who had also type 2 DM are more prone to get progressive forms of the disease and are at higher risk of developing the end stage liver disease than those who do not have diabetes[12,13]. Although cardiovascular disease is the major cause of excess morbidity and mortality in type 2 diabetes, hepatic failure may also be a threat to patients with type 2 diabetes and NAFLD[13,14]. Therefore, it is important for physicians to be aware of the high likelihood that their patients with T2DM have NAFLD, as this is another potential complication that requires attention.

The availability, easy access, noninvasiveness and low cost of ultrasonography have made it the most widely used tool for routine screening for NAFLD. Its sensitivity of ultrasonography ranges from as low as 60% to as high as 94%[5,15].

Although the performance of liver ultrasound for the diagnosis of NAFLD is much better than the determination of plasma levels of amino-transferase, it still underperforms when compared with gold standard liver biopsy.16 The use of semi-quantitative scores based on different echographic parameters may somehow improve the outcome but still has low performance when the hepatic triglyceride content is 12.5%[17]. Vibration controlled

transient elastography (FibroScan) or magnetic resonance elastography can be used to assess the severity of fibrosis If available[18,19]. Both these modalities have a strong correlation with the histologic findings and may avoid the demand of doing liver biopsy in a large number of patients.

Objectives: To determine the frequency of NAFLD in type 2 diabetic patients and its association with biochemical parameters.

PATIENTS AND METHOD

A cross-sectional study design was used for this research, in which 138 adult patients of both sexes who were diagnosed as having type 2 diabetes mellitus were enrolled during the period between January 2019 and September 2019. These patients were those who attended the Diabetic center for follow-up and those who were admitted to the Azadi Teaching Hospital, Duhok, Kurdistan Region (Iraq) for management. After taking informed consent from each, abdominal ultrasound was done for evaluation.

The following patients were excluded from the study: those with type I DM, pregnant ladies, hepatitis and other liver diseases patients, those on hepatotoxic medications, history of alcohol consumption and those with the serious concomitant disease.

After classifying patients into fatty and non-fatty liver disease, they were then evaluated by measuring the body mass index (BMI), Glycosylated hemoglobin (HbA1c), total cholesterol, triglycerides (TG), low density lipoprotein (LDL) and high density lipoprotein (HDL), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase.

For this study, the patient was considered as having type 2 DM depending on fasting plasma glucose if it was ≥ 126 mg/dl or random if > 200 mg/dl. Other than these, if the patient had a diagnosis report from a physician or he/she is on the treatment for diabetes was also considered for diagnosis. Regarding BMI, if it was between 23 and 25 kg/m2, the patient was considered overweight and obese if equal to or more than 25kg/m^2.

An experienced sonographer carried out an ultrasound examination of the liver. Steatosis diagnosed based on liver brightness (evident sonographic contrast between hepatic and renal parenchyma), further supported by high posterior attenuation and reduced diaphragm and vessel wall distinction.

The severity of steatosis was graded as follows: Non, Score 0 (when the echotexture of the liver is normal). Mild, Score I (when there is a slight and diffuse increase in fine echoes in hepatic parenchyma with normal visualization of the diaphragm and portal vein wall). Moderate Score II (when there is a moderate and diffuse increase in fine echoes with impaired visualization of the intrahepatic vessel borders and diaphragm). Severe Score III (diffuse increase in fine echoes with poor or non-visualization of the intrahepatic vessel, diaphragm and the posterior aspect of the right lobe). Ultrasonography has a sensitivity of 89% and a specificity of 93% in detecting moderate-to-severe hepatic steatosis.

STATISTICAL ANALYSIS

The descriptive purposes of the study were presented in mean and standard deviation

or frequency and percentage, including age in mean and St. deviation and gender and BMI in frequency and percentage. The prevalence of non-alcohol fatty liver was determined in frequency and percentage. Disease duration was presented in the median and interquartile range due to non-normality. The biochemical parameters were presented in mean and standard deviation and prevalence of their normal ranges in frequency and percentage.

The association of general and biochemical parameters with non-alcoholic fatty liver was examined in Pearson Chi-squared and ANOVA One-way, Kruskal Wallis tests. The association of patients' characteristics with non-alcoholic fatty liver was examined in independent t-test, Pearson Chi-squared test, or Mann-Whitney U-test.

The level of biochemical parameters in patients with and without alcoholic fatty liver was examined in an independent t-test. The P-value of less than 0.05 was used to reject the null hypothesis. The statistical calculations were performed by Statistical Package for Social Sciences version 24 (SPSS 24; IBM Corp; USA).

RESULTS

The majority of the enrolled patients in this study were overweight (34.8%) and obese (55%), and most of them with NAFLD have mild degree fatty changes (42.8%) (Table.1). More than 70% of patients have an HbA1c > 7, which means that they are uncontrolled cases of type 2 DM and about 50% of them have significant elevation at TG level (Table 2). The P values were significant statistically for BMI, TG, SGOT and SGPT levels in regard to the degree of fatty liver, whether mild, moderate, or severe, as shown in (Table 3). In contrast, the only statistically significant measures were BMI and TG when comparing patients with no fatty change with those who had NAFLD, P-value 0.001(Table 4).

Table 1: General information and prevalence of non-alcoholic fatty liver in type 2 diabetic patients

Characteristics (n=138)	Distribution	Frequency
Age (Range: 30-84 years); Mean/SD	53.96	SD 11.14
Gender; F (%)		
Male	39	28.3
Female	99	71.7
BMI (Range: 18.5-42.5); Mean/SD	30.48	4.89
Normal; F (%)	14	10.1
Overweight	48	34.8
Obese	76	55.1
Disease duration/years (median/Interquartile range)	6.0	Int. range 8.0
Non-Alcoholic Fatty Liver, F (%)		
No fatty changes	64	46.4
Mild	59	42.8
Moderate	14	10.1
Severe	1	0.7

Comment: Duration of type 2 diabetes duration was presented in median and interquartile range due to non-normality.

Table 2: Biochemical parameters of patients with type 2 diabetes mellitus

Characteristics (n=138)	Mean	Std. Deviation	Minimum	Maximum
HbA1c (%)	8.16	1.74	5.50	12.60
Uncontrolled	98	71.0		
Controlled	40	29.0		
Serum GPT (IU/L)	22.54	7.65	11.00	44.00
Abnormal	13	9.4		
Normal	125	90.6		
Serum GOT (IU/L)	20.54	6.24	11.00	36.00
Abnormal	9	6.5		
Normal	129	93.5		
TC (mg/dL)	171.35	38.29	91.00	269.00
Abnormal	34	24.6		
Normal	104	75.4		
TG (mg/dL)	174.11	78.58	63.00	369.00
Abnormal	78	56.5		
Normal	60	43.5		
LDL (mg/dL)	96.88	33.95	26.00	195.00
Abnormal	96	69.6		
Normal	42	30.4		
HDL (mg/dL)	41.29	9.13	21.00	64.00
Abnormal	66	47.8		
Normal	72	52.2		

The normal values of the biochemical parameters were presented in frequency and percentage.

Table 3: Association of general and biochemical parameters with non-alcoholic fatty liver in T2DM patients

Characteristics (n=138)	Non-alcoholic Fatty Liver				P-Value
	No Fatty Changes	Mild	Moderate	Severe	
Age (year)	55.83 ± 11.87	52.80 ± 10.11	51.79 ± 10.52	34.00	0.102
Gender					
Male	20 (31.3)	17 (28.8)	2 (14.3)	0 (0.0)	0.565
Female	44 (68.8)	42 (71.2)	12 (85.7)	1 (100.0)	
BMI	27.59 ± 3.58	31.53 ±4.50	33.97 ± 3.30	37.70	<0.001
HBA1C (%)	7.82 ± 1.39	8.31 ±1.85	8.89 ± 2.27	6.30	0.090
Serum GPT (mg/dL)	20.47 ± 7.87	23.27 ±7.19	25.00 ± 9.87	80.00	<0.001
Serum GOT (mg/dL)	18.41 ± 4.91	18.92 ±5.04	23.55 ± 8.34	42.00	<0.001

Characteristics (n=138)	Non-alcoholic Fatty Liver				P-Value
	No Fatty Changes	Mild	Moderate	Severe	
TC (mg/dL)	167.33 ± 38.37	175.88 ±41.24	171.57 ± 29.48	173.00	0.689
TG (mg/dL)	151.31 ± 59.18	199.95 ±96.84	209.57 ± 95.03	79.00	0.004
LDL (mg/dL)	95.10 ± 30.77	96.93 ±36.11	100.21 ± 30.88	62.00	0.715
HDL (mg/dL)	40.19 ± 8.68	41.61 ± 8.82	42.64 ± 10.77	51.00	0.485
Diabetes duration (year)	5.0 ± 8.0	7.0 ± 7.0	6.5 ± 8.50		0.965

ANOVA One-way was performed for all statistical analyses except the Kruskal Wallis test for disease duration and Pearson Chi-squared for gender.

Table 4: Association of patients' characteristics in patients with and without non-alcoholic fatty liver

Characteristic (n=138)	Study Groups		P-Value (Two-Sided)
	No Fatty Changes	Non-Alcohol Fatty Liver	
Age	55.83 ± 11.87	52.35 ± 10.28	0.070*
Gender			
Male	20 (31.3)	19 (25.7)	0.468**
Female	44 (68.8)	55 (74.3)	
BMI	27.59 ± 3.58	32.09 ± 4.40	<0.001*
Duration of DM	5.0 ± 8.0	6.5 ± 7.0	0.154***
HBA1C	7.82 ± 1.39	8.39 ± 1.93	0.048*
Serum GPT	20.47 ± 7.87	24.40 ± 10.25	0.015*
Serum GOT	18.41 ± 4.91	20.11 ± 6.55	0.109*
TC	167.33 ± 38.37	175.03 ± 38.85	0.247*
TG	151.31 ± 59.18	200.14 ± 96.32	<0.001*
LDL	95.10 ± 30.77	97.08 ± 34.99	0.724*
HDL	40.19 ± 8.68	41.94 ± 9.17	0.258*

* Independent t-test, ** Pearson Chi-squared test, and *** Mann-Whitney U-test were performed for statistical analysis.

DISCUSSION:

In this observational cross sectional study, the prevalence of NAFLD and the grading of fatty liver in type 2 diabetes mellitus patients is investigated. A significant increase in the Prevalence of NAFLD was observed in this group of patients. This high prevalence indicated the importance of management and early evaluation of NAFLD in type 2 diabetes mellitus patients.

Up to fifty-three percent of the study participants had NAFLD along with type 2 diabetes mellitus. The value was higher than the findings of studies done by Portillo-Sanchez et al. (2015)[20], Adams et al. (2010)[21], and a study done in Nigeria 22 where the respective prevalence were 49.5%, 34.4%, and 16.7%. This could be due to a lack of liver checking habits and low attention given by the health sector on fatty liver disease.

The gender distribution of the present study showed that more females were affected by fatty liver diseases than males. However, the difference was not significant at p≤0.05. In a recent study, Yi et al. demonstrated that the Prevalence of NAFLD in men is higher than in females in type 2 diabetes mellitus patients,23 however the report of NAFLD among different sexes is not conclusive. Some reports confirm a high prevalence in women, while recent studies came up with even distribution[24].

Obesity was reported as the risk factor for NAFLD. In many research findings, a fatty liver disease among type 2 diabetic patients was significantly associated with BMI.20,25 In our findings, 35% (48 patients) of the participants were overweight, had BMI in range (25-29.9) and 55 % (76 patients) were obese, having BMI > 30, and the majority of the patients who had fatty liver were among the obese group also the severity of fatty changes in the term of grading were significantly more in those with higher BMI (p<0.001), this indicates the significant role of obesity in the disease progression.

It is expected that patients with NAFLD have higher liver function test abnormalities than individuals who do not have NAFLD in diabetic patients.26 It is scientifically proved that alanine aminotransferase (ALT) is more predictive of liver fat accumulation among the liver enzymes and correlate with liver fat independent of obesity.[27] The results of this study showed that ALT correlates with the severity of fatty liver (p ≤0.001), which means that it is significantly associated with fatty liver diseases. Although the value of ALT is within the normal range, its value is higher among those with moderate and severe fatty liver than normal type 2 diabetic patients. Research outputs in many other study areas showed that serum ALT levels are normal in patients with NAFLD. Hence, elevated ALT does not necessarily mean serious hepatic damage.[25]

Triglyceride is one of the main factors affecting NAFLD in the present study. The mean value of TG among type 2 diabetic patients with fatty liver was higher than the laboratory means results of normal and patients (p0.004).

NAFLD is highly bonded with TG accumulation in the hepatocytes. This store may arise from different sources, including the intestine (through absorption) and the liver (synthesis). The high level of glucose or insulin will activate some transcription factors resulting in increased hepatic de novo lipogenesis. Finally, excessive lipolysis will form steatosis[9,28,29].

Up to 70% of the patients enrolled in the study were uncontrolled cases of type 2 DM, the mean HbA1c 8.16, but there was no significant correlation between the severity of fatty liver and the level of HbA1c as shown in our results (p0.090). In a meta-analysis done by Amiri-Dash Atan N. et al.[30], they found that the subgroup

analysis of HbA1c in the Prevalence of NAFLD is lower than the pooled Prevalence of NAFLD in type 2 diabetes mellitus patients, as it is suggested that there is an unusual relationship between HbA1c and NAFLD[30].

The duration of diabetes did not show any significant statistical association with the degree of severity of fatty liver in our study.

In conclusion, the overall Prevalence of NAFLD among type 2 diabetes mellitus patients is significantly high, and it implies more care in these groups of patients to prevent NAFLD.

We recommend doing more research across our country to know the pathogenesis and identify more effective treatment options because Non-alcoholic fatty liver diseases are the major risk factors for developing cardiovascular diseases, stroke, peripheral vascular disease, chronic kidney disease, cirrhosis and liver cancer, among type 2 diabetic patients[31].

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پوختە

هەڤبەندی دناڤبەرا نەخوشیا جەرگێ دوهنی یا نەکحولی دگەل نەخوشییا شەکرێ جورێ 2

پێشەکی: نەخوشیا جەرگێ دوهنی یا نەکحولی دهێتە هژمارتن مشەترین ئەگەر بو نەخوشیێن جەرگی یێن دومدرێژ ل وەلاتێن پێشکەفتی. ئەڤ جورە نەخوشییا جەرگی دژوارترە دەما دگەل نەخوشییا شەکرێ جورێ 2 بیت و زویتر نەخوشیێن دومدرێژ بو جەرگی دروستدکەت. دەستنیشانکرنا نەخوشیا جەرگێ دوهنی یا نەکحولی بوەرگرتنا نمونەکی ژ جەرگی رێکا ستاندەردە بو دەستنیشانکرنێ لێ هندەك رێکێن بسانەهی تر هەنە وەکی سونەرا جەرگی کو دشێت دژواربیا نەخوشیێ دیارکەت. ئەڤ ڤەکولینە هاتە کرن ژبو دیارکرنا رێژا ڤێ نەخوشیێ دناڤ نەخوشێن شەکرێ جورێ 2 دا بکارئینانا سونەرا جەرگی هەروەسا دیارکرنا هەڤبەندیا ڤێ نەخوشیێ دگەل رێبەرێ سەنگا لەشی و هندەك پیڤەرێن دی یێن کیمیایی.

رێکێن ڤەکولینێ: ئەڤ ڤەکولینا پارچەیی هاتە ئەنجامدان لنەخوشخانا ئازادی یا فێرکرنێ دناڤبەرا کانونا دووێ و ئیلونا 2019ێ. هەمی نەخوشێن بەشدار ئەو بوون یێن نەخوشیا شەکرێ جورێ 2 هەی و بو هەمیان رێبەرێ سەنگا لەشی هاتە پیڤان. نەخوشیا جەرگێ دوهنی یا نەکحولی لدەڤ ڤان نەخوشان هاتە دابەشکرن بو سڤك، ناڤەند و دژوار لدویڤ سونەرا جەرگی و تاقیکرنێن خوینی بو هاتنە کرن وەك رێژا جورێن دوهنی و تاقیکرنێن کارێ جەرگی.

ئەنجام: سەد وسیە نەخوشێن شەکرێ هاتنە بەشدارکرن دڤێ ڤەکولینێ دا. نێزیکی 55٪ ژ وان سەنگا لەشێ وان یا زێدە بوو و 34٪ د قەلەو بوون. جەرگێ دوهنی هاتە دیتن لدەڤ 53.7٪ ژ وان (74 نەخوش) و دابەشکرنا وان بو سڤك و ناڤەند و دژوار بڤی شێوەی بوو، 79.9٪، 17.7٪ و 4.35٪ لدویڤ ئێك. تێکرایێ رێبەرێ سەنگا لەشی لدەڤ نەخوشێن شەکرێ یێن جەرگێ دوهنی هەی 32.09 بوو بەرامبەر 27.59 یێن جەرگێ دوهنی لدەڤ نەبیت. تێکرایێ HbA1c و دوهنێن سیانی و GPT دناڤبەرا نەخوشێن جەرگێ دوهنی هەی و یێن نەیی بڤی شێوەی بوو 8.37 بەرامبەر 7.82 و 200 مگم/دل بەرامبەر 150 مگم/دل و 24. یەکە/ل بەرامبەر 20.4 یەکە/ل لدویڤ ئێك.

دەرئەنجام: بگشتی رێژا جەرگێ دوهنی دناڤ نەخوشێن شەکرێ دا بشێوەکێ بەرچاڤ یا زێدەبوو. بلندبوونا ئاستێ HbA1c و دوهنێن سیانی و GPT دبیت پەیوەندی دگەل دروستبوونا جەرگێ دوهنی هەبیت لدەڤ نەخوشێن شەکرێ.

الخلاصة

ارتباط مرض الكبد الدهني غير الكحولي مع داء السكري من النوع ٢

الخلفية والأهداف: يعتبر مرض الكبد الدهني غير الكحولي هو السبب الأكثر شيوعًا لمرض الكبد المزمن في البلدان المتقدمة في مرضى السكري من النوع ٢، ياخذ مرض الكبد الدهني غير الكحولي مسار أكثر عدوانية ويمكن أن يؤدي إلى ظهور مرض مزمن في الكبد في وقت مبكر .على الرغم من أن الخزعة لاتزال المعيار الذهبي لتشخيص مرض الكبد الدهني غير الكحولي، إلا أن العديد من الاختبارات الاخرى مثل فحص الكبد بـالموجات فوق الصوتية يمكن أن يعطي فكرة عن مدى خطورة المرض. أجريت هذه الدراسة لتحديد تواتر مرض الكبد الدهني غير الكحولي في المرضى الذين يعانون مرضى السكري من النوع ٢ من باستخدام الموجات فوق الصوتية للكبد وتحديد الارتباط بمؤشر كتلة الجسم وعلامات الكيمياء الحيوية الأخرى) مثل ترانساميناسات الكبد، الهيموغلوبين السكري HbA1c وملف الدهون.

طرق البحث: أجريت هذه الدراسة المقطعية في مستشفى آزادي التعليمي في الفترة من يناير إلى سبتمبر ٢٠١٩. وكان من المعروف أن جميع المرضى المعنيين لديهم مرضى السكري من النوع ٢. بعد الموافقة، تم تحديد مؤشر كتلة الجسم (BMI) وتم تصنيف المرضى إلى كبد دهني خفيف ومعتدل وشديد بناًء على معايير التصوير بالموجات فوق الصوتية، ثم أجريت اختبارات دم لتحديد ملف تعريف الدهون لديهم، وانزيمات الكبد ومستويات الهيموغلوبين السكرية.

النتائج: شارك في الدراسة مائة وثلاثون مريضًا بالسكري، كان حوالي ٥٥٪ منهم يعانون من زيادة الوزن و٣٤٪ يعانون من السمنة المفرطة، وشوهد مرض الكبد الدهني غير الكحولي في ٧.٥٣٪ (٧٤ مريضًا)، وشوهد مرض الكبد الدهني غير الكحول بنسبة خفيفة ومعتدلة وشديدة في ٩.٧٩٪ و٧.١٧٪٣٥.٤٪ على التوالي. كان لدى مرضى الكبد الدهني متوسط مؤشر كتلة الجسم يبلغ ٠٩.٣٢ مقابل ٥٩.٢٧ للمرضى الذين لا يعانون من الكبد الدهني. وكان متوسط مستويات HbA1c والدهون الثلاثية ومعدلات GPT بين مرضى الكبد الدهني والمرضى الذين لا يعانون من الكبد الدهني ٣٧.٨ مقابل ٨٢.٧ و٢٠٠ ملج/ دل مقابل١٥٠ ملجم/ دل و٤.٢٤وحدة/ لتر مقابل ٤.٢٠ وحدة /لتر على التوالي.

الاستنتاج: معدل انتشار تغيرات الكبد الدهنية بين مرضى السكري من النوع ٢. ارتفاع انزيم GPT، مستويات الدهون الثلاثية والهيموكلوبين السكري قد تهيئ مرضى السكري لمرض الكبد الدهني غير الكحولي.