Evaluation of Triglyceride-Glucose Index as a Marker of Hepatic Steatosis and Fibrosis in Egyptian Patients with NAFLD

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Article

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) grows in frequency, being a significant health burden. A liver histopathology is uncommonly used due to its risks and invasive nature. In order to assess and diagnose NAFLD patients, a trustworthy indicator is needed. An easy and fast method to determine if someone is insulin resistant is to use the triglyceride-glucose (TyG) index.

Aim of the Work: Our goal of this research was to examine the relationship between the onset of NAFLD and the TyG index. Methods: Our observational cross-sectional analysis at the hepatology outpatient clinic at Al Sahel Teaching Hospital, from January 2022 to July 2022, was conducted on 100 subjects, divided after history, laboratory results, and ultrasound, into 50 cases with NAFLD and 50 healthy controls.

Results: In this study, there was an insignificant difference between both groups as regard age or sex, but the difference was significant between both groups as regard BMI, ALT, fasting blood sugar, TGs, TyG-index, and HOMA IR. Moreover, a noteworthy positive correlation was observed between NAFLD fibrosis score with HOMA-IR and TyG-index, as well as a substantial correlation was noticed between the TyG index and LSM in NAFLD.

Conclusion: Our research emphasizes that, in cases of NAFLD, the TyG index might be considered a distinct risk element for both liver fibrosis and hepatic steatosis. The TyG index is low-cost to test and simple to calculate when compared to other IR markers.

Key Words: Hepatic steatosis, insulin resistance, NAFLD, triglyceride-glucose index, .

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INTRODUCTION

NAFLD includes lipid deposition in the hepatic tissue and progression to more steatosis-associated hepatitis and fibrosis, then cirrhosis, where hepatocellular carcinoma. (HCC) could develop on top. NAFLD stands for nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH). With steatosis, more than 5% of the parenchyma of the liver, without proof of hepatocyte injury, is defined as NAFL. While NASH is a process of necroinflammation, it is described by histologic criteria^[1].

Without a doubt, the rising incidence of obesity and the concurrent rise in the prevalence of NAFLD are owing in part to calorie intake and physical inactivity^[2].

Approximately 60% of those with fatty livers exhibit hyperglycemia and dyslipidemia. Fat deposition within the hepatic parenchyma led to increased endoplasmic reticulum and mitochondrial stress and impaired mitophagy, which is associated with lipotoxicity. metabolic dysfunction that may occur as a result of increased hepatic triacylglycerol (TAG), induced insulin resistance, dyslipidemia, cardiovascular disease, and progression to NASH, cirrhosis, and HCC^[3].

Hepatic free fatty acids (FFAs) are derived from diet, lipogenesis, and adipose tissue lipolysis. Subsequently, FFAs undergo β -oxidation, esterification into TAG, and lipoprotein assembly for potential secretion or storage as lipid droplets. This is followed by an additional hepatocellular injury. Hepatic steatosis is also influenced by hereditary and environmental factors^[4]. The liver histopathological assessment is the ideal diagnostic tool for detecting NAFLD; nevertheless, because of its invasiveness and the uneven distribution of fat throughout the liver, sampling errors frequently arise^[5].

As a reliable predictor of the onset of NAFLD, the triglyceride-glucose index could have an enormous impact on this disease's diagnosis, prognosis, and treatment^[6].

The TyG index is linked to NAFLD through insulin resistance. Insulin resistance may bring on a metabolic and cellular stress response, resulting in TG's lipotoxic effects on hepatocytes. Insulin resistance and the dysfunction of the islet β -cell may develop with hyperglycemia^[7].

The TyG index, which may be estimated using this equation [FPG (mg/dL) \times fasting TG (mg/dL)/2], has been extensively established in relation to the hyperinsulinaemic-euglycaemic clamp (HEC), and it is also more stable and simpler to perform than HOMA^[8].

AIM OF THE WORK

Our goal of this research was to examine the relationship between the TyG index in NAFLD Egyptian patients and hepatic steatosis and fibrosis.

PATIENTS AND METHODS

In our cross sectional observational research, 100 Egyptian subjects were categorized into:

- Group (1) included 50 NAFLD patients diagnosed by trans-abdominal ultrasonography and NAFLD fibrosis score to assess the fibrosis.
- Group (2) included 50 healthy control subjects.

Depending on a research performed by *Guo et al.* in 2020, our sample size was determined using NCSS PASS $11.0^{[9]}$.

The cases that were included were gathered between January and July of 2022 from the Al Sahel Teaching Hospital's outpatient clinics for hepatology and gastroenterology.

The scientific ethics committee gave its clearance for this study to be carried out, and each case included provided informed consent. This study excluded patients with other liver disease or cirrhosis using ultrasound and laboratory testing, patients with malignancy within the gastrointestinal tract, patients using steatogenic drugs (methotrexate, amiodarone, tamoxifen, valproate, or corticosteroids), patients consuming alcohol, and patients using lipid-lowering agents in addition to weight-loss medications.

Every study participant underwent a thorough medical history, a full physical assessment, and a BMI calculation, which was based on the following formula: weight per square of height kg/m2. According to the conventional WHO classification, four groups were determined based on this calculation: underweight if less than 18.5 kg/m2, normal if between 18.5 and 24.9 kg/m2, overweight if between 25 and 29.9 kg/m2, and obese if equal to or more than 30 kg/m2. A laboratory examination including CBC, hepatic profile (ALT, AST, serum albumin, serum bilirubin, prothrombin time (P.T) and I.N.R), serum creatinine, blood urea, serum lipids (cholesterol, LDL, HDL, and triglycerides), fasting blood glucose level, and HbA1c.

Using the homeostatic model assessment for IR (HOMA-IR) and the subsequent formula, the degree of IR was determined:^[10]

[HOMA-IR = [fasting insulin (mU/L) x fasting blood glucose (mg/ dL)]/405]

The TyG index was measured with this subsequent formula:^[11]

 $TyG = Ln [TG (mg/dl) \times FPG (mg/dl)/2]$

Abdominal ultrasonography evaluated the liver, spleen, ascites, and abdominal masses, as well as the portal, hepatic, and splenic vessels, for their patency and diameter in all patients.

Through the use of ultrasonography, NAFLD was diagnosed. The following method was used to determine the NAFLD fibrosis score depending on six factors, age, BMI, albumin, platelet count, state of serum glucose, and AST/ALT ratio:^[12]

[1.675 + 0.037 x age (years) + 0.094 x BMI(kg/m2) + 1.13 x impaired fasting glucose/diabetes (yes = 1, no = 0) + 0.99 x AST/ALT ratio - 0.013 x platelet (x 109/L) - 0.66 x albumin (g/dL) (It is used to categorize the possibility of fibrosis into three groups: low probability (< -1.5), intermediate probability (> -1.5 to < 0.67), and high probability (> 0.67)).] The Fibro-Touch FT100 was used for transient elastography, the hepatic fibrosis was measured applying the liver stiffness measurement (LSM), and the extent of hepatic steatosis was assessed applying the fat attenuation parameter (FAP).

Based on the FAP value, the severity of NAFLD was categorized as subsequent: 240 dB/m \leq FAP < 265 dB/m for mild cases, 265 dB/m \leq FAP < 295 dB/m for moderate cases, and FAP \geq 295 dB/m for severe cases.

Cases with steatosis were categorized based on LSM measures as: LSM greater than 7.3 kPa as liver fibrosis, and LSM equal to or less than 7.3 kPa as non-liver fibrosis.

• Statistical analysis:

For the analysis, the SPSS statistical program (USA, Chicago, IBM Inc., IL) was used to assess, code, and investigate the data. Independent sample t-test was applied to assess of the standard deviation and mean of the numerical variables. Pearson's correlation coefficient was used for the correlation analysis. The frequency of the categorical parameters has been reported. A two-tailed *P*

value eual to or less than 0.05 was statistically considered as a significant value.

RESULTS

We studied a set of 100 cases, categorized into two groups: group (1) included 50 cases, and group (2) included 50 cases. According to gender, the studied cases included 41 males with a percentage of 41%, while the females were 59 with a percentage of 59%, distributed as 21 males (percentage of 42%) and 29 females (percentage of 58%) with an average age of 39.24 \pm 4.39 years in group (1) and 20 males (percentage of 40%) and 30 females (percentage of 60%) whose average age was 40.18 \pm 5.49 years in group (2).

The comparison between the two groups of the study according to BMI and laboratory findings revealed that there was statistically significant higher mean value of ALT in group (1) was 27.62 ± 13.38 comparing to 22.90 ± 6.11 for group (2). and also showed statistically significant higher mean value of BMI, FBS (mg/dL), TGS (mg/dl), TyG-index and HOMA–IR in group (1) comparing to group (2). (Table 1)

 Table 1: Comparison between the two groups of the study according to BMI and laboratory findings.

Anthropometric data and blood pressure	Group (1) (<i>n</i> =50)	Group (2) (<i>n</i> =50)	Test value	P-value
BMI [wt/ (ht)2]	31.53±4.85	29.35±4.89	7.372	<0.001**
ALT (U/L)	27.62±13.38	22.90±6.11	2.269	0.025*
AST (U/L)	28.56±6.31	26.14±4.80	1.501	0.203
Albumin	3.98±0.32	4.23±0.31	-1.972	0.093
Total Bilirubin (mg/dl)	1.16±0.42	1.02±0.36	1.717	0.089
FBS (mg/dL)	111.70±39.30	71.60±16.64	6.644	<0.001**
HbA1C (IU/ml)	5.68±0.76	5.66±0.54	0.13	0.897
Fasting insulin	10.71 ± 1.98	10.28 ± 1.80	1.128	0.262
TGS (mg/dl)	194.02±54.50	165.42±38.19	3.039	0.003*
LDL (mg/dl)	145.28 ± 19.47	139.24±15.98	1.696	0.093
HDL (mg/dl)	43.74±7.59	46.22±6.19	-1.791	0.076
TyG-index	9.19±0.50	8.64±0.33	6.593	<0.001**
HOMA–IR	2.89±1.15	1.70±0.74	2.142	0.007*

Using: t-Independent Sample t-test for Mean±SD;

p-value >0.05 is insignificant

In group (1) there were 48% of patients with low probability of NAFLD fibrosis score, 34% with intermedite probability and 18% with high probability. (Figure 1)



NAFLD fibrosis score: Interprettion

Fig. 1: NAFLD fibrosis score distribution among patients of group (1).

Based on the fibrosis grade, group (1) patients included 30% with F0 to F1, 22% with F2, 34% with F3, and 14% with F4. (Figure 2)



Fibrosis grde

Fig. 2: Fibrosis grade distribution among patients of group (1).

Regarding the severity of steatosis among the patients in Group (1), there were 24% with a mild score, 44% with a moderate score, and 32% with a high score. (Figure 3)



Fig.3: Severity of Steatosis distribution among the group (1).

When correlating the NAFLD fibrosis score of patients in group (1) with the TyG index and HOMA-IR, we found that a substantially significant positive correlation existed with both the TyG index and the HOMA-IR. (Table 2)

 Table 2: Correlation between triglyceride-glucose index and

 HOMA-IR with NAFLD fibrosis score in patients of group (1).

Group (1)	NAFLD fibrosis score		
	Rs	<i>p</i> -value	
TyG-index	0.797	<0.001**	
HOMA-IR	0.622	<0.001**	

Using: Pearson's correlation coefficient

p-value >0.05 NS; **p-value* <0.05 S; ***p-value* <0.001 HS

When correlating TyG index with liver steatosis and fibrosis for cases in group (1), it was revealed that a significant positive correlation existed with LSM (kPa), while there was an insignificant correlation with UAP. (Table 3)

 Table 3: Correlation between triglyceride-glucose index and hepatic steatosis and fibrosis in nonalcoholic fatty liver disease.

	Triglyceride-glucose index		
Group (1)	Rs	p-value	
LSM (kPa)	0.535	<0.001**	
UAP (dB/m)	0.054	0.708	

Using: Pearson's correlation coefficient

p-value >0.05 NS; **p-value* <0.05 S; ***p-value* <0.001 HS

The analysis of the ROC curve proved the high degree of discrimination provided by the TyG index between the two groups, where the area under the ROC curve (AUC) = 0.821 with a standard error (SE) of 0.0388 (95%). Confidence interval: 0.731–0.890. Z score = 6.271, p<0.001. Cutoff point >8.94 with sensitivity = 72% and specificity = 82%. (Figure 4)



Fig. 4: Receiver Operating Characteristics (ROC) analysis for TyG-index to discrimination between patients and control.

DISCUSSION

Since NAFLD is a burden health problem, it is critical to comprehend its etiology, risk factors, and early management. Furthermore, for the early detection of the risk of developing NAFLD, a simple and reliable diagnostic test is required^[13].

Thus, our study was carried out to evaluate the relationship between TyG index and NAFLD-related hepatic steatosis and fibrosis.

Regarding the age and sex, our analysis demonstrated that, non-significant differences existed between the groups of the study.

Research by *Guo et al.* examined 4784 cases of NAFLD that had an ultrasonography diagnosis. They demonstrated that there were insignificant age differences between the two groups, which is consistent with our findings. However, they actually demonstrate that there were significant variations in sex between the two groups, with more participants with liver fibrosis being male, which ran counter to our findings^[9].

A study on a number of adult asymptomatic instances was carried out in 2020. In 100 cases, ultrasonography revealed the presence of NAFLD. Anthropometric, biochemical, and general data were evaluated. According to their findings, NAFLD patients were statistically significant when they were older^[14].

According to our research, the blood pressure, albumin, AST, total bilirubin, CBC parameters, HbA1C, fasting insulin, LDL, and HDL were insignificantly different between the two groups. However, group (1) showed statistically substantially higher BMI, ALT, FBS, and TG levels.

Patients with NAFLD showed a considerable increase in their waist circumference and BMI, according to *Ali et al.* Obese patients had a greater prevalence of NAFLD (78%), compared to overweight patients (16%), while it was found in about 6% of individuals with a normal BMI. NAFLD patients showed statistically insignificant levels of ALT and AST^[14].

The aim of the research performed in 2021 by *Naguib et al.* was to evaluate the metabolic risk variables related to

the onset of NAFLD. Comparisons between the control and the NAFLD groups revealed significantly higher levels of WC, BMI, HbA1c, ALT, and GGT in the NAFLD group^[15].

Significant differences in ALT and AST levels were discovered by *Guo et al.* between the two groups in their study^[9].

Our research revealed that in the NAFLD group, the TyG-index and HOMA-IR were statistically substintially higher, with a cut-off level of 8.94 for the TyG-index for prediction of the possibility of NAFLD. Also, a substintially significant positive correlation existed between the NAFLD fibrosis score and both TyG-index and HOMA-IR.

Ling et al. carried out a thorough meta-analysis to assess the link between the TyG index and the chance of developing NAFLD. They suggested that the TyG index may be a novel potential factor for fatty liver. With 105,365 cases total, their study comprised four cohorts and eight cross-sectional studies, involving 28,788 NAFLD patients. Furthermore, a higher association between NAFLD and the TyG index existed with females compared to males in the subgroup analysis^[16].

Li et al. studied the validity of the TyG index for the assessment of T2DM cases who are at risk for NAFLD. Consistent with our findings, they demonstrated that, when compared to other glycemic and lipid indicators in T2DM, the TyG index has a more reliable prediction for NAFLD to arise. In a stratified analysis, patients who were younger females with lower HDL had a higher correlation between an elevated TyG index and NAFLD^[17].

In order to evaluate the risk variables for non-diabetic patients with NAFLD, *Naguib et al.* employed a logistic regression analysis. Meanwhile, there was a strong correlation between the incidence of the development of NAFLD and HOMA-IR^[15].

According to *McGill et al.*, ALT—the main screening test for acute hepatitis—was similarly linked to NAFLD; yet, in a ROC study, the TyG index's area under the curve for NAFLD diagnosis was greater than that of FPG, ALT, and TG. These suggested that the TyG index is more valid to assess NAFLD^[18].

Lee et al. analyzed the TyG index and its association with NAFLD, as well as its efficacy in diagnosing NAFLD in Korean people when compared to the HOMA-IR. There

were 4,986 subjects included. The results showed that the TyG index was more reliable than HOMA-IR as an indicator for NAFLD^[19].

In our study, regarding the correlation between hepatic steatosis and fibrosis with the TyG index in NAFLD patients, a substentially significant positive correlation existed between the triglyceride-glucose index and liver stiffness measurement (LSM), while there was no significant correlation with the ultrasound attenuation parameter (UAP).

A study conducted in 2020 on the link between the TyG index and the degree of hepatic steatosis and fibrosis in patients with NAFLD found a positive correlation between the TyG index and liver fibrosis. Additionally, the TyG index outperformed HOMA-IR.^[9].

CONCLUSIONS

A good indicator of the development of NAFLD is the TyG index. Lifestyle modification and monitoring may be necessary if the TyG index is higher than 8.7 in order to stop the progression of NAFLD and maybe hepatic fibrosis. Therefore, our results in this study lend validity to the widespread application of the TyG index to evaluate NAFLD. In light of our findings, we reinforce our conclusion with the recommendation for more expansive patient populations and longer follow-up times.

ABBREVIATIONS

NAFLD; Nonalcoholic fatty liver disease.

BMI; body mass index.

FAP; fat attenuation parameter.

FFAs; free fatty acids.

IR; insulin resistance.

NASH; non-alcoholic steatohepatitis.

FPG; fasting plasma glucose.

HCC; hepatocellular carcinoma.

HEC; hyperinsulinaemic-euglycaemic clamp.

HOMA; homeostasis model assessment.

HOMA-IR; homeostasis model assessment of insulin resistance.

LSM; liver stiffness measurement.

NAFL; non-alcoholic fatty liver.

NFS; NAFLD fibrosis score.

AUC; area under the curve.

T2DM; type 2 diabetes mellitus.

TAG; Triacylglycerol.

TG; Triglycerides.

TyG; triglyceride-glucose.

UAP; ultrasound attenuation parameter.

WC; waist circumference.

ROC; Receiver operating characteristic.

DECLARATIONS

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The Research Ethics Committee (REC) at the Faculty of Medicine, Ain Shams University, approved this study with No. AMASU MSO 24/2024, under the supervision of Prof. Fathy Tash, The REC does not release the names of its members, per the regular operating procedures of both the institution and the REC. For data analysis, informed approval was provided by each participant.

CONSENT FOR PUBLICATION

Inapplicable.

AVAILABILITY OF DATA AND MATERIALS

The editorial board can obtain the data upon request.

CONFLICT OF INTERESTS

There are no conflicts of interest.

FUNDING

None

AUTHOR' CONTRIBUTIONS

Badr A. M. collected and followed up the patients, carrying out the requested investigations. Naguib A. M., Mahmoud H. A., Morsy M. S., and Elfors M. A. shared in following up the patients and analyzing the collected data. The manuscript was authorized by all authors.

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REFERENCES

- 1. Lebeaupin C, Vallée D, Hazari Y, *et al*, (2018). Endoplasmic reticulum stress signalling and the pathogenesis of non-alcoholic fatty liver disease. Journal of hepatology, 69(4), 927-947.
- Ciardullo S, Sala I & Perseghin G, (2020). Screening strategies for nonalcoholic fatty liver disease in type 2 diabetes: Insights from NHANES 2005–2016. Diabetes Research and Clinical Practice, 167, 108358.
- 3. German M.N, Lutz M.K, Pickhardt P.J, Bruce R.J & Said A. (2020). Statin use is protective against hepatocellular carcinoma in patients with nonalcoholic fatty liver disease: A case-control study. Journal of Clinical Gastroenterology, 54(8), 733-740.
- 4. Ullah R, Rauf N, Nabi G, *et al*, (2019). Role of nutrition in the pathogenesis and prevention of non-alcoholic fatty liver disease: recent updates. International journal of biological sciences, 15(2), 265.
- 5. Jayakumar S, Middleton M.S, Lawitz E.J, et al, (2019). Longitudinal correlations between MRE, MRI-PDFF, and liver histology in patients with non-alcoholic steatohepatitis: Analysis of data from a phase II trial of selonsertib. J. Hepatol. 70, 133–141.

- 6. Song K, Park G, Lee H.S, *et al*, (2022). Comparison of the triglyceride glucose index and modified triglyceride glucose indices to predict nonalcoholic fatty liver disease in youths. The Journal of pediatrics, 242, 79-85.
- 7. Zheng R, Du Z, Wang M, Mao Y & Mao W. (2018). A longitudinal epidemiological study on the triglyceride and glucose index and the incident nonalcoholic fatty liver disease. Lipids in Health and Disease, 17(1), 1-9.
- Pandyarajan V, Gish R.G, Alkhouri N & Noureddin M. (2019). Screening for nonalcoholic fatty liver disease in the primary care clinic. Gastroenterology & hepatology, 15(7), 357.
- **9. Guo W, Lu J, Qin P, et al, (2020).** The triglycerideglucose index is associated with the severity of hepatic steatosis and the presence of liver fibrosis in nonalcoholic fatty liver disease: a cross-sectional study in Chinese adults. Lipids in Health and Disease, 19(1), 1-9.
- **10.** Salgado A, Carvalho L, Oliveira A, *et al*, (2010): Insulin resistance index (HOMA-IR) in the differentiation of patients with nonalcoholic fatty liver disease and healthy individuals. Arquivos de gastroenterologia, 47, 165-169.
- **11.** Jin J, Cao Y, Wu L, *et al*, (2018): Triglyceride glucose index for predicting cardiovascular outcomes in patients with coronary artery disease. Journal of thoracic disease, 10(11): 6137.
- 12. Angulo P, Hui J, Marchesini G, *et al*, (2007): The NAFLD fibrosis score: a noninvasive system that identifies liver fibrosis in patients with NAFLD. Hepatology, 45(4): 846-854.
- **13. Makri E, Goulas A, Polyzos S.A(2021).** Epidemiology, Pathogenesis, Diagnosis and Emerging Treatment of Nonalcoholic Fatty Liver Disease. Arch Med Res. 2021 Jan;52(1):25-37. doi: 10.1016/j. arcmed.2020.11.010. Epub 2020 Dec 14. PMID: 33334622.
- 14. Ali M, Kamal G, Younis M, and Zaghloul A (2022): Diagnostic performance of serum steatosis biomarkers in Prediction of Non-Alcoholic Fatty Liver Disease in Adult Asymptomatic Egyptians. SVU-International Journal of Medical Sciences, 5(2): 51-63.

- **15.** Naguib H and Kassab H. (2021): Potential relation between non-alcoholic fatty liver disease and glycemic and metabolic parameters in subjects without diabetes. Egyptian Liver Journal, 11(1): 1-7.
- **16.** Ling Q, Chen J, Liu X, *et al*, (2023). The triglyceride and glucose index and risk of nonalcoholic fatty liver disease: A dose–response meta-analysis. Frontiers in Endocrinology, 13, 1043169.
- 17. Li W, Wang Y, He F, *et al*, (2022). Association between triglyceride–glucose index and nonalcoholic

fatty liver disease in type 2 diabetes mellitus. BMC Endocrine Disorders, 22(1), 1-7.

- **18.** McGill M.R. (2016): The past and present of serum aminotransferases and the future of liver injury biomarkers. EXCLI J.15:817–28.
- **19.** Lee S.B, Kim M.K, Kang S, *et al*, (2019). Triglyceride glucose index is superior to the homeostasis model assessment of insulin resistance for predicting nonalcoholic fatty liver disease in Korean adults. Endocrinology and Metabolism, 34(2), 179-186.

تقييم مؤشر الدهون الثلاثية والجلوكوز كعلامة على تليف الكبد الدهني لدى المرضى المصريين المصابين بمرض الكبد الدهني غير الكحولي

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الخلفية: يزداد معدل الإصابة بمرض الكبد الدهني غير الكحولي، وهو يشكل عبنًا صحيًا كبيرًا. نادرًا ما يتم استخدام فحص الأنسجة الكبدية بسبب مخاطره وطبيعته الغازية. من أجل تقبيم وتشخيص مرضى الكبد الدهني، هناك حاجة إلى مؤشر جدير بالثقة. تتمثل إحدى الطرق السهلة والسريعة لتحديد ما إذا كان شخص ما مقاومًا للأنسولين في استخدام مؤشر ثلاثي الجليسريد- الجلوكوز. كان هدفنا من هذا البحث هو فحص العلاقة بين ظهور مرض الكبد الدهنى غير الكحولى ومؤشر ثلاثي الجليسريد- الجلوكوز. كان هدفنا من هذا

الطريقة: تم إجراء تحليلنا المقطعي الرصدي في العيادة الخارجية لأمراض الكبد في مستشفى الساحل التعليمي، من يناير ٢٠٢٢ إلى يوليو ٢٠٢٢، على ١٠٠ مريض، تم تقسيمهم بعد التاريخ المرضي ونتائج المختبر والموجات فوق الصوتية، إلى ٥٠ حالة مصابة بمرض الكبد الدهني غير الكحولي و٥٠ حالة صحية.

النتائج: في هذه الدراسة، كان هذاك فرق غير مهم بين المجموعتين فيما يتعلق بالعمر أو الجنس، ولكن الفرق كان كبيرا بين المجموعتين فيما يتعلق بمؤشر كتلة الجسم، وALT، وسكر الدم الصائم، والدهون الثلاثيه، ومؤشر ثلاثي الجليسريد- الجلوكوز، ومؤشر مقاومة الانسولين. علاوة على ذلك، لوحظ وجود ارتباط إيجابي جدير بالملاحظة بين درجة تليف الكبد و مؤشر مقاومة الانسولين مع مؤشر ثلاثي الجليسريد- الجلوكوز، وكذلك لوحظ وجود ارتباط كبير بين مؤشر ثلاثي الجليسريد- التلوف الكبد م مؤشر الكحولي.

الخلاصة: يؤكد بحثنا أنه في حالات مرض الكبد الدهني غير الكحولي، يمكن اعتبار مؤشر ثلاثي الجليسريد- الجلوكوز عنصر خطر مميز لكل من تليف الكبد وتدهن الكبد. يعتبر مؤشر ثلاثي الجليسريد- الجلوكوز منخفض التكلفة للاختبار وسهل الحساب عند مقارنته بعلامات مقاومة الأنسولين الأخرى.