Serum Adipsin as a Marker of MAFLD and its Relation with Metabolic Abnormalities

Original Article

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ABSTRACT

Background: Metabolic dysfunction-associated fatty liver disease (MAFLD) is a novel definition for hepatic illness related to recognized metabolic disorders. Approximately 38.77% of people worldwide suffer from MAFLD. It has been documented that serum lipids and insulin resistance (IR) are associated with serum adipsin levels. Our study's objective was to investigate the role of serum adipsin in MAFLD.

Methods: Our prospective case-control study was performed at the hospital of Ain Shams University between October 2023 and April 2024 on two groups: The cases group included 50 MAFLD patients, while the control group included 50 healthy individuals. All cases in both groups underwent a full medical history and physical assessment, as well as a laboratory investigation. The correlation of serum adipsin and MAFLD was statistically analyzed.

Results: The analysis of serum adipsin levels revealed a highly significant difference between the groups of the study. Significant positive associations between adipsin serum levels and BMI, WC (cm), FPG, triglycerides, HBA1c, APRI, FIB-4 score, and ALT in the MAFLD group were found. The ROC analysis demonstrated that a level of serum adipsin >3082.6 ng/mL was the optimal cut-off value for distinguishing between MAFLD and control cases, with an AUC of 0.919.

Conclusion: Serum adipsin levels were significantly greater in MAFLD cases than in controls. Serum adipsin could be used as a prognostic biomarker for MAFLD.

Key Words: Insulin resistance, Metabolic dysfunction-associated fatty liver disease (MAFLD), Metabolic Abnormalities, NAFLD.

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INTRODUCTION

MAFLD is a primary liver steatosis disease associated with insulin resistance and metabolic dysfunction. MAFLD is easier to diagnose and better describes the pathogenesis of the majority of patients previously recognized as having non-alcoholic fatty liver disease (NAFLD)[1].

MAFLD and NAFLD are not equivalent, as certain cases of NAFLD have not been associated with IR, increasing the possibility that they have another unidentified cause of steatosis or have not been appropriately investigated for the etiology. Furthermore, some cases with MAFLD have additional etiology for steatosis, e.g., more alcohol intake or any other secondary cause; however, these factors must be excluded before diagnosing NAFLD[2].

Patients with obesity, diabetes, dyslipidemia, and metabolic syndrome are more likely to have MAFLD since it is only present in those with insulin resistance-associated metabolic dysfunction[3].

The American Association of Clinical Endocrinologists (AACE) in 2003 stated that metabolic syndrome must be linked with either a significant risk of IR or overweight or obesity, plus two or more of the following: elevated serum triglycerides (≥150 mg/dl), HDL-cholesterol (<40 mg/dl in men and <50 mg/dl in women), arterial blood pressure (≥ 130/85 mmHg), or impaired glycemic status[4].

It is difficult to identify NAFLD without first diagnosing steatosis and ruling out all other causes of secondary steatosis. MAFLD is significantly easier to diagnose.

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All that is required is to detect steatosis and metabolic dysfunction related to insulin resistance, for example, type 2 diabetes, obesity, or two of the following conditions: high C-reactive protein (CRP), pre-diabetes, elevated WC, hypertension, hypertriglyceridemia, decreased HDL, or elevated homeostasis model assessment of insulin resistance (HOMA) score[1].

Adipsin is an adipokine that is only made in adipose tissues and released into the bloodstream. It is a homolog of serine protease[5]. Its concentration can, however, change with renal injury-related diseases, leading to a 10-fold increase in adipsin levels because of decreased glomerular filtration[6]. This explains why ***Badawi et al***. found chronic kidney disease to be a risk factor for MAFLD[7].

Adipsin has a major impact on the complement system as a rate-limiting element in the activation of the alternative complement pathway; it is sometimes referred to as complement factor D[8].

Since then, it has been demonstrated that adipsin is essential in ischemia-reperfusion and sepsis models. It produces several molecules, e.g., the anaphylatoxins C3a and C5a, as well as the synthesis of the C5-C9 membrane attack complex. It is unknown, therefore, how adipsin functions are associated with systemic metabolism and energy homeostasis[9].

PATIENTS AND METHODS

Our prospective case-control study was performed in the Internal Medicine Department, Ain Shams University Hospitals, over a six-month period, from October 2023 to April 2024. The study included 100 subjects grouped as follows:

- The group of cases included 50 Egyptian outpatient adults over the age of 18 who had been diagnosed with MAFLD. The liver steatosis was determined by imaging and non-invasive biomarkers, and at least one of the following—1- being overweight or obese, 2- having type II diabetes, and 3- having clinical evidence of metabolic dysfunction—was used to establish the diagnosis of MAFLD[10].

- The group of controls included 50 healthy participants (regarding imaging and non-invasive biomarkers) matched for age and gender with the group of cases.

Patients with chronic hepatic disease other than MAFLD (autoimmune hepatitis, hemochromatosis, Wilson disease, or drug-induced liver disease), those on drug-induced metabolic disorders (steroids, vitamin A, oral contraceptive pills, etc.), and those who declined to take part in the research were all excluded.

After reviewing previous findings of ***Pan et al.***[11] At least 50 patients with MAFLD and 50 healthy controls would provide 100% power at an alpha error of 0.05 by using Power Analysis and Sample Size Software (PASS 15) (Version 15.0.10) for calculation of sample size.

The study gained approval from the Research Ethics Committee (REC) at the Faculty of Medicine, Ain Shams University. An informed consent was signed by all participants after demonstrating the aim of the study, the purpose, and possible complications of each intervention.

All subjects of the study underwent thorough, full medical histories with assessment of all patient complaints and medical comorbidities (diabetes, hypertension, ischemic heart disease, vascular diseases, and renal diseases). A full physical examination was done. Laboratory investigations included CBC, serum bilirubin, albumin, and hepatic transaminases, serum gamma-glutamyl transferase, alkaline phosphatase, prothrombin time, INR, serum creatinine, fasting blood sugar, HbA1C, serum cholesterol, triglycerides, LDL, HDL, and measurement of serum adipsin. A pelvi-abdominal ultrasound was done for all cases.

Hepatic steatosis was assessed by ultrasound using the following criteria: brightness of the parenchyma, contrast between liver and kidney, gallbladder wall definition, bright vessel walls, and deep beam attenuation[12]. The hepatic steatosis grading was assessed as follows: Grade I: increased echogenicity of the liver with visible periportal and diaphragmatic echogenicity; Grade II: increased echogenicity of the liver with invisible periportal echogenicity, without obscurity of the diaphragm; and Grade III: increased echogenicity of the liver with invisible periportal echogenicity with obscurity of the diaphragm[13].

Calculation of FIB-4 was done using this formula: Age ([yr] x AST [U/L]) / ((PLT [10(9)/L]) x (ALT [U/L])(1/2))[14].

Evaluation of AST to platelet ratio index (APRI) was done by this formula: (AST/upper limit of normal) X 100/ platelet count[15].

NAFLD fibrosis score (NFS) was assessed with this formula: NFS = −1.675 + 0.037 × age (years) + 0.094 × body mass index (kg/m2) + 1.13 × (impaired fasting glycemia or diabetes [yes=1, no=0]) + 0.99 × (AST/ALT ratio) − 0.013 × platelets (×109/L) − 0.66 × albumin (g/ dL)[16].

The link between serum adipsin level and the different clinical, laboratory, and imaging data was done to assess the role of serum adipsin in the diagnosis of MAFLD and its relationship with the grade of hepatic steatosis and NFS.

- **Statistical analysis**

The data were coded and analyzed via the SPSS software. Numerical data were displayed as means (with SD) or medians (and ranges). The Chi-Square test, the Student-t, Fisher Exact, and Mann-Whitney tests were used for comparative analysis between the two groups. The Spearman coefficient and Pearson coefficient were used for correlation analysis. ROC curve analysis was used to evaluate the predictivity of serum adipsin level and the best cut-off value with assessment of its sensitivity and specificity. Any *p-value* < 0.05 was considered statistically significant.

RESULTS

The group of cases had a mean age of 49.37 years (± 3.42), ranging from 25 to 65 years, with 37 (65%) females and 13 (35%) males. Diabetes mellitus was found in 18 cases (36%), hypertension in 14 cases (28%), ischemic heart disease in 3 cases (6%), vascular disease in 3 cases (6%), renal disease in 4 cases (8%), and smoking in 18 cases (36%). The mean of BMI (kg/m2) was 28.56 ± 0.53, ranging from 22.62 to 30.09, while the mean of WC (cm) was 108.88 ± 12.45, ranging from 91.2 to 115.5. The group of control had an average age of 48.65 years (± 2.35), ranging from 25 to 65 years, with 30 (60%) females and 20 (40%) males. Smoking was reported in 14 cases (28%). The mean of BMI (kg/m2) was 24.33 ± 1.05 and ranged from 23.47 to 27.83, with a mean of WC (cm) of 89.20 ± 1.55 and ranged from 81.2 to 95.1. No substantially significant differences in age, sex, smoking, or height between the two groups were found, but a highly significant difference in WC and a significant difference in weight and BMI were found, according to the chi-square test, student t-test, and Fisher exact. (Table 1)

Table 1: Comparison between the two studied groups according to demographic data, clinical data and anthropometric criteria.

	Group of cases (*n* = 50)		Group of controls (*n* = 50)		Test of Sig.	*p*
	No.	%	No.	%		
Sex						
Female	37	65.0	30	60.0	χ^2= 0.936	0.462
Male	13	35.0	20	40.0		
Age (years)						
Min. – Max.	25.0 – 65.0		25.0 – 65.0			
Mean ± SD.	49.37 ± 3.42		48.65 ± 2.35		t= 0.813	0.437
Median (IQR)	48.55 (46.54 – 49.52)		48.0 (46.0 – 49.0)			
Smoking	18(36%)		14(28%)		χ^2= 0.332	0.635
Clinical data						
DM	18(36%)		0		NA	NA
HTN	14(28%)		0		NA	NA
Ischemic heart disease	3(6%)		0		NA	NA
Vascular insult	3(6%)		0		NA	NA
Renal diseases	4(8%)		0		NA	NA
Weight (kg)						
Min. –Max.	65.75 – 95.63		65.66 – 74.46			
Mean ± SD.	82.14 ± 4.73		69.87 ± 3.41		t= 2.319*	0.026*
Median (IQR)	82.22 (82.5 – 89.4)		69.74 (67.5 – 74.5)			
Height (cm)						
Min. –Max.	157.2 –176.4		156.4 –176.8			
Mean ± SD.	164.4 ± 5.21		165.1 ± 2.31		t= 1.036	0.412
Median (IQR)	164.2 (160.0 – 168.1)		163.0 (163.5 – 170.9)			
WC (cm)						
Min. –Max.	91.2-115.5		81.2-95.1		FE= 62.138	<0.001*
Mean ± SD.	108.88 ± 12.45		89.20 ± 1.55			
BMI (kg/m2)						
Min. –Max.	22.62 – 30.09		23.47 – 27.83			
Mean ± SD.	28.56 ±0.53		24.33 ± 1.05		t= 2.322*	0.012*
Median (IQR)	28.68 (24.6 – 29.1)		24.33 (24.9 – 26.0)			

χ^2: Chi square test t: Student t-test FE: Fisher Exact *p*: *p value* for comparing between the studied groups

Student t tests were utilized for comparing the two study groups based on laboratory biomarkers. The results showed a highly significant difference in fasting blood glucose (mg/dl) and HbA1c and a significant difference in HDL-C (mg/dl) and triglycerides (mg/dl). However, there was no significant difference regarding cholesterol (mg/dl) and LDL-C (mg/dl). (Table 2)

Table 2: Comparison between the two studied groups according to biochemical parameters.

Biochemical parameters	Group of cases (*n*=50)	Group of controls (*n*=50)	t	*p*
Cholesterol mg/dl				
Min. – Max.	172.81– 223.3	137.61 – 190.35		
Mean ± SD.	191.16 ± 30.88	177.38 ± 9.56	4.194	0.052
Median (IQR)	189.8	177.5		
Triglycerides mg/dl				
Min. – Max.	84.4 – 149.9	73.71 – 118.55		
Mean ± SD.	138.62 ± 53.85	101.31 ± 19.55	7.617	0.041*
Median (IQR)	138.69	103.99		
HDL-C (mg/dl)				
Min. – Max.	40.3-55.51	40.1-53.12	3.651	0.042*
Mean ± SD.	46.86 ± 12.18	48.30 ± 3.74		
LDL-C (mg/dl)				
Min. – Max.	74-118.5	72-118.5	4.632	0.055
Mean ± SD.	105.88 ± 38.34	99.60 ± 15.87		
Fasting blood glucose mg/dl				
Min. – Max.	80.75 – 135.60	77.61 – 131.31		
Mean ± SD.	118.71 ± 4.26	83.28 ± 4.61	3.427*	0.001*
Median (IQR)	119.81(115.91 – 122.02)	82.40(80.20 – 85.91)		
HbA1c				
Min. – Max.	5.1 - 9.6	4.4 - 5.6	8.494*	<0.001*
Mean ± SD.	7.569 ±0.581	5.042 ±0.347		

t: Student t-test

The mean serum adipsin level was 3317.307 ± 186.63 (ng/ml) in the group of cases and 2827.94 ± 115.89 (ng/ml) in the control group. The Mann-Whitney test found a highly substantial difference between the two groups. (Table 3).

Table 3: Comparison between the two studied groups according to Adipsin.

Adipsin	Group of cases(*n*= 50)	Group of controls (*n* =50)	U	*p*
Mean±SD.	3317.307 ± 186.63	2827.94 ± 115.89	16.0*	<0.001*
Median (IQR)	3268.8	2935.7		

U: Mann Whitney test
p: *p value* for comparing between the studied groups
*: Statistically significant at $p \leq 0.05$

In the group of cases, the Spearman coefficient test revealed a highly significant positive association between adipsin and weight, BMI, WC (cm), FPG, and triglycerides. Also, it showed a significant association between adipsin and HbA1c. But no significant association was established regarding age, sex, height, cholesterol, HDL-C, and LDL-C. The Pearson coefficient test revealed a statistically significant positive association between adipsin and NAFLD score, APRI, and fib4 score and a significant association with ALT, but no significant correlation was established with AST and GGT. (Table 4)

Table 4: Correlation between and adipsin and different parameters in the group of cases (MAFLD) (n = 50).

	Adipsin	
	Test	*p*
Age (years)	0.001$^{r\,s}$	0.990
Sex (male)	0.013$^{r\,s}$	0.869
Weight	0.216$^{r\,s}$	0.031
BMI	0.340$^{r\,s}$	0.001
WC (cm)	0.517$^{r\,s}$	< 0.001
Ht (m)	0.086$^{r\,s}$	0.241
APRI score	0.106^{r}	0.022
FIB-4 score	0.133^{r}	0.036
NAFLD score	0.125^{r}	0.017
FPG	0.413$^{r\,s}$	< 0.001
HbA1c	0.156$^{r\,s}$	0.022
Cholesterol	0.333$^{r\,s}$	0.052
Triglycerides	0.419$^{r\,s}$	< 0.001
HDL-C (mg/dl)	0.506$^{r\,s}$	0.061
LDL-C (mg/dl)	0.541$^{r\,s}$	0.084
ALT	0.246^{r}	0.014
AST	0.073^{r}	0.469
GGT	0.074^{r}	0.467

r s: Spearman coefficient r: Pearson coefficient

The ROC analysis demonstrated that Adipsin had a significant diagnostic performance (AUC = 0.919). Adipsin > 3082.6 ng/mL was the most effective cut-off criterion for discriminating patients with MAFLD from the controls (sensitivity = 82.86%, specificity = 100%, and accuracy = 78.9%). (Table 5), (Figure 1)

Table 5: ROC analysis for Sensitivity and specificity of adipsin to detect its relation to MAFLD.

	AUC	Cut off	*p*	95% C.I		Sensitivity	Specificity	accuracy
				LL	UL			
Adipsin	0.919	>3082.6	<0.001*	0.915	1.015	82.86	100.0	78.9

AUC: Area Under a Curve *p value*: Probability value CI: Confidence Intervals

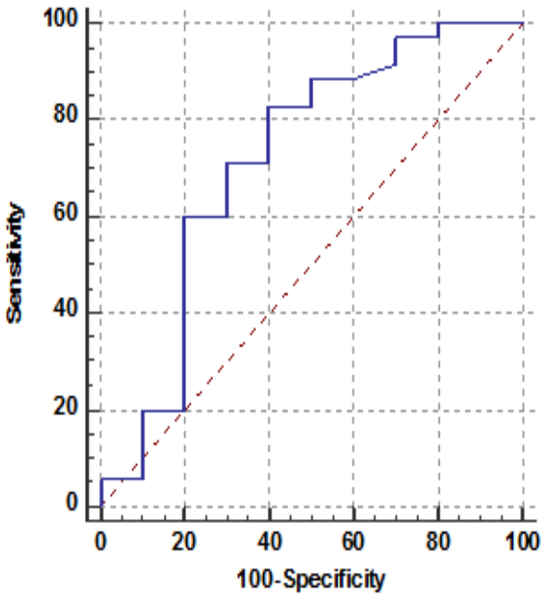

Fig. 1: ROC analysis for Sensitivity and specificity of adipsin to detect its relation to MAFLD.

DISCUSSION

MAFLD is an increasing cause of chronic hepatic illness and associated comorbidities, affecting over 50% of dysmetabolic individuals and around 25% of the general population[17].

In the group of cases in our study, there were 18 (36%) with DM and 14 (28%) with HTN, and that was in agreement with the findings of the meta-analysis done by ***Younossi et al.*** on 8,515,431 cases with the metabolic-associated comorbidities with NAFLD: obesity in 51.34%, arterial hypertension in 39.34%, and type 2 diabetes in 22.51%[3].

MAFLD was linked to a higher risk of incident diabetes (risk ratio [RR] 2.08; 95% CI, 1.72-2.52), chronic kidny disease (RR 1.64; 95% CI, 1.39-1.94), and cardiovascular disease (RR 1.44; 95% CI, 1.15-1.81), according to a recent Chinese cohort study on 6873 people with follow-up for 4.6 years. The observed rates were similar when NAFLD was taken into account as a risk factor instead of MAFLD[18].

In our study, the correlation between MAFLD and other comorbidities was not applicable because the control group cases were selected as healthy without any comorbidities.

According to ***Pan et al.***, the incidence of T2DM was greater in the MAFLD group (11.66%).[11] In NAFLD, metabolic comorbidities are potential factors for the severity of hepatic fibrosis; the more metabolic comorbidities there are, the greater the chance of developing severe liver fibrosis[19].

The incidence of diabetes is twice as high in those with NAFLD as in those without, according to a new meta-analysis that evaluated the growing evidence of NAFLD as a risk factor for diabetes[20].

Our study also revealed that there was a significantly higher increase in HbA1c and fasting blood glucose in the group of cases than in the control one. And that is consistent with A cohort study performed in Korea that evaluated the relationship between NAFLD and diabetic incidence in non-diabetic young people showed that baseline NAFLD was significantly related to a higher risk of acquiring diabetes, particularly in cases of severe fibrosis[21].

Our analysis found a significant increase in WC (108.88 ± 12.45 versus 89.20 ± 1.55) and BMI (28.56 ± 0.53 versus 24.33 ± 1.05) in the group of cases compared to the control one. That was in agreement with ***Qiu et al.***, who found a significantly higher BMI (26.37 ± 2.89 versus 22.63 ± 2.55) and waist circumference (93.75 ± 7.82 versus 81.68 ± 7.85) in the patients compared to the controls[22].

Triglycerides were substantially greater in the group of cases (138.62 ± 53.85), compared to the control one (101.31 ± 19.55), in our study. This was consistent with ***Pan et al.'s*** findings, which showed that the mean TG was 103 (81–125) in the control cases and 151 (105–209) in the MAFLD cases[11].

There were no differences in LDL-C and total cholesterol levels between the two groups of our study. Also, other researchers found no differences in total cholesterol and LDL-C levels between MAFLD cases and control cases or among different subgroups of MAFLD[23,24].

Furthermore, we found that the HDL in the group of cases (46.86 ± 12.18) was substantially less than that of the control one (48.30 ± 3.74). This is consistent with ***Zhang et al.'s*** findings; the HDL-c levels of NAFLD subjects were lower than those of the control cases (31 ± 0.27 in the NAFLD group and 45 ± 0.31 in the non-NAFLD one)[25].

In our study, the mean serum adipsin level was 3317.307 ± 186.63 (ng/ml) in the group of cases and 2827.94 ± 115.89 (ng/ml) in the control one, which was a highly substantial difference. That is also in line with the findings of ***Pan et al.***, which showed that the MAFLD cases had greater serum adipsin levels (3543.00 (3187.94–3972.50) ng/mL) than the non-MAFLD cases (3095.33 (2778.71–3354.77) ng/mL) with a *P value* less than 0.001[11].

In our study, there was a substantial correlation between adipsin and BMI and WC (cm) in the group of patients ($P = 0.001$), which was similar to the findings of ***Pan et al.*** ($P = 0.001$)[11].

There is evidence associating serum adipsin levels to insulin resistance, waist obesity, and serum lipids[26]. Metabolic syndrome in postmenopausal women was found to be related to elevated serum levels of insulin and adipsin, as well as abdominal obesity and dyslipidemia[27].

Our study revealed a significant association between FPG and serum adipsin levels with a *P value* of 0.001, a finding also confirmed by ***Milek et al.*** with a *P value* of 0.025. Furthermore, compared to patients with normal blood glucose, those with type 2 diabetes showed noticeably greater serum adipsin concentrations[28].

However, ***Zhou et al.*** found that patients who had impaired glucose tolerance and type 2 diabetes had decreased serum adipsin levels[29]. Moreover, serum levels of adipsin may be elevated at first due to a compensating mechanism in the early stages of diabetes mellitus and metabolic syndrome but eventually diminish with adipose malfunction; the levels of adipsin were also preferentially low in type 2 diabetes cases with β cell failure[9].

The results of this study were consistent with those of ***Pan et al.***, who also found a highly significant positive connection between adipsin and triglycerides in the group of cases with a *P value* less than 0.001[11].

In the present study, there was also no significant association between serum adipsin levels and LDL-C or total cholesterol levels. These results were in line with the findings reported in ***Qiu et al.*** and other previous studies[22].

Adipsin and APRI, the Fib4 score, and the NAFLD score all showed statistically significant positive correlations in our study, with *p-values* of 0.022, 0.036, and 0.017 in the group of cases, respectively. In contrast to what we found, ***Zhang et al.*** revealed a non-significant correlation between serum adipsin and increased risk of liver fibrosis using FIB-4[25].

The ROC analysis in our study revealed that adipsin had a significant diagnostic performance. The best cut-off value of adipsin for differentiating MAFLD patients from the controls was > 3082.6 ng/mL (AUC of 0.919), with a sensitivity of 82.86%, a specificity of 100%, and an accuracy of 78.9%.

According to ***Pan et al.'s*** study, the optimum adipsin cut-off value to differentiate MAFLD patients from the control group was >3237.85 ng/mL, with a sensitivity of 76.96% and a specificity of 100%[11].

Gu et al. reported that the prediction model of NAFLD remission had an AUC of 0.751 (95% CI: 0.717–0.785) ($p < 0.001$), with 70.9% sensitivity and 97.8% specificity for adipsin[30].

CONCLUSIONS

Adipsin levels are higher in patients with MAFLD, exhibiting a substantial positive connection with weight, BMI, waist circumference (cm), fasting plasma glucose, HbA1c, and triglycerides. For distinguishing patients with MAFLD from the control group, the ideal cut-off value of adipsin is > 3082.6 ng/mL, which exhibits a sensitivity of 82.86%, a specificity of 100%, and an accuracy of 78.9%. Adipsin serum levels may be used as an inflammatory biomarker to predict the prognosis of patients with MAFLD. As a result, measuring adipsin serum levels offers a novel approach for more effective management for high-risk patients.

ABBREVIATIONS

AUC; area under the curve.

APRI; AST to Platelet Ratio.

BMI; body mass index.

CKD; chronic kidney disease.

CVD; cardiovascular diseases.

DM; diabetes mellitus.

T2DM; type 2 diabetes.

FBG; fasting blood glucose.

HbA1c; hemoglobin A1c.

HDL; high-density lipoprotein.

HTN; hypertension.

IR; insulin resistance.

LDL; low-density lipoprotein.

MAFLD; Metabolic associated fatty liver disease.

NAFLD; nonalcoholic fatty liver disease.

TGs; triglyceride.

CI; Confidence Interval.

OR; Odd Ratio.

REC; Research Ethics Committee.

WC; Waist circumference.

FIB-4; Fibrosis-4 Index.

NFS; NAFLD fibrosis score.

DECLARATIONS ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The Research Ethics Committee (REC) at the Faculty of Medicine, Ain Shams University, under the supervision of Prof. Fathy Tash, approved this study with No. FMASU MS 482/2023. 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.

COMPETING INTERESTS

There are no conflicting interests, according to the authors' declaration.

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None

AUTHOR' CONTRIBUTIONS

Ayman E. Elazab collected, followed up the patients, and carried out the requested investigations. Essam M. Bayoumy, Khaled M. Raafat, and Mostafa A. Elfors shared the responsibility of specifying the needed data. All authors shared on analyzing the collected data and authorized the manuscript.

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Not applicable

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مستوي أديبسين بالدم كعلامة على مرض الكبد الدهني المرتبط بالخلل الأيضي وعلاقته بالاضطرابات الأيضية

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الخلفية: مرض الكبد الدهني المرتبط بالخلل الأيضي هو تعريف جديد لمرض الكبد المرتبط بالاضطرابات الأيضية المعروفة. يعاني حوالي ٣٨,٧٧٪ من الأشخاص في جميع أنحاء العالم من مرض الكبد الدهني المرتبط بالخلل الأيضي. وقد تم توثيق أن الدهون في الدم ومقاومة الأنسولين ترتبط بمستويات الأديبسين في الدم. كان هدف دراستنا هو التحقيق في دور الأديبسين في الدم في مرض الكبد الدهني المرتبط بالخلل الأيضي.

الطريقة: أجريت دراستنا المقارنة في مستشفى جامعة عين شمس بين أكتوبر ٢٠٢٣ وأبريل ٢٠٢٤ على مجموعتين: مجموعة الحالات شملت ٥٠ مريضًا مصابًا بمرض الكبد الدهني المرتبط بالخلل الأيضي، بينما ضمت مجموعة الضبط ٥٠ فردًا سليمًا. خضعت جميع الحالات في كلتا المجموعتين لتاريخ طبي كامل وتقييم بدني، بالإضافة إلى فحص معملي. تم تحليل ارتباط الأديبسين في الدم و مرض الكبد الدهني المرتبط بالخلل الأيضي إحصائيًا.

النتائج: كشف تحليل مستويات الأديبسين في الدم عن وجود فرق كبير بين مجموعات الدراسة. تم العثور على ارتباطات إيجابية كبيرة بين مستويات الأديبسين في الدم ومؤشر كتلة الجسم ومحيط الخصر (سم) وسكر الدم الصائم والدهون الثلاثية والهيموجلوبين السكري في مجموعة مرض الكبد الدهني المرتبط بالخلل الأيضي. أظهرت النتائج أن مستوى الأديبسين في الدم <٣٠٨٢,٦ نانوجرام/ مليليتر كان القيمة الحدية المثلى للتمييز بين حالات مرض الكبد الدهني المرتبط بالخلل الأيضي وحالات التحكم، مع منطقه تحت المنحني = ٠,٩١٩.

الخلاصة: كانت مستويات الأديبسين في الدم أعلى بشكل ملحوظ في حالات مرض الكبد الدهني المرتبط بالخلل الأيضي مقارنة بحالات الضبط. يمكن استخدام الأديبسين في المصل كعلامة تشخيصية لمرض الكبد الدهني المرتبط بالخلل الأيضي وتشخيصه.