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Visceral and subcutaneous fat, muscle mass, and liver volume as noninvasive predictors of the progress of non-alcoholic fatty liver disease

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Abstract

Background The term "non-alcoholic fatty liver disease" (NAFLD) refers to a range of disorders caused by lipid accumulation in the liver. High abdominal fat levels can cause adipocytes to become more lipolytic, releasing free fatty acids into the portal venous system. In this study, we aimed to use the analysis of visceral fat, subcutaneous fat, muscle mass, and liver volume to evaluate the severity of fatty liver in NAFLD.

Results This study enrolled 130 patients with non-alcoholic fatty liver disease. The mean age of studied patients was 51.38 ± 11.11 years, ranging between 25 and 65 years. Of the studied patients, 60 (46.2%) patients were males and 70 (53.8%) were females. The mean body mass index was 41.23 ± 7.83 (kg/m^2). Based on the radiological assessment of those patients, patients with grade III fatty liver had significantly higher total fat volume, visceral fat volume, subcutaneous fat volume, fat rate in the body, visceral fat volume rate, psoas muscle volume, and psoas muscle ratio in comparison with those with grade I and grade II fatty liver. Liver enzymes significantly correlated with total fat volume, visceral fat volume rate, psoas muscle volume, psoas muscle ratio, and liver volume.

Conclusions The degree of fatty liver severity among patients with NAFLD was positively correlated with the amount of subcutaneous, visceral fat, and muscle mass. Also, both liver transaminases had a significant positive correlation with the amount of total and visceral fat, psoas muscle mass, and liver volume.

Keywords Non-alcoholic fatty liver, Visceral fat, Subcutaneous fat, Psoas muscle

Background

One of the most prevalent global causes of chronic liver disease is non-alcoholic fatty liver disease (NAFLD). Cirrhosis and hepatocellular carcinoma are examples of end-stage liver disorders that can develop from severe forms of NAFLD and non-alcoholic steatohepatitis (NASH). Therefore, it is necessary to look at risk factors related to hepatic fat deposition and determine the progress of this disease [1].

NAFLD, cardiovascular disease, and diabetes are all more common in people with substantial obesity [2]. In obesity, it seems that fat distribution is more crucial than the total mass of fat [3]. When there is excess abdominal fat, a disproportionately upper body fat distribution increases the risks for metabolic consequences of obesity, including fatty liver. The majority of "metabolically obese" patients who are of average weight have some

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increase in fatty tissue mass and insulin resistance, which is most likely caused by a rise in visceral fat [4].

As a result, persons with relatively low body mass indexes (BMIs) might experience a huge rise in abdominal visceral fat, while those with high BMIs may have extremely low levels of this type of fat. It was proposed that liver fat deposition can be predicted clinically by visceral obesity. Additionally, computed tomography (CT) evaluation of the amount of visceral fat has been associated with the degree of fatty liver. Only a few previous studies investigated the relationship between NAFLD and fat distribution in the body [5–7].

This work was designed to find whether there are relationships among the severity of NAFLD, the amount of visceral and subcutaneous fat, psoas muscle volume, and liver volume, and also, to assess the relations among the increase in liver enzymes in NAFLD, the visceral and subcutaneous fat volumes, the psoas muscle volume, and liver volume.

Methods

Study setting and design

This prospective cross-sectional study was done at Assiut University Hospital in the period from September 2019 to June 2021.

Inclusion criteria

We included adult patients of both genders with fatty liver who came for any other medical problem and were incidentally diagnosed by abdominal ultrasound examination to have fatty liver disease.

Exclusion criteria

We excluded all patients with other liver diseases including hepatitis C and hepatitis B, acute viral hepatitis, and autoimmune hepatitis. Also, patients who have a history of chronic drug usage affecting the liver were excluded from this study.

Ethical consideration

The Ethics Committee of the Faculty of Medicine approved this research, which was carried out by the Declaration of Helsinki's tenets.

All participants completed signed informed permission after being told of the study's goal. The research procedure was registered on *clinicaltrials.gov* with NCT04240145.

Clinical and laboratory examination

All patients were subjected to thorough history taking and clinical examination with the calculation of BMI. Laboratory investigations were included; liver function tests including liver transaminases, lipogram, and hepatitis serology were investigated in all patients.

Radiological assessment

Ultrasound examination

An abdominal ultrasound examination was done using Logiq E9 for all patients by a radiologist with 22 years of experience. Fatty liver grading was done as follows: Grade 1: Diffuse increase in the liver echogenicity. Grade 2: The increased liver echogenicity veils the echogenic walls of the intrahepatic portal veins. Grade 3: The diaphragmatic outline is obscured by the increased hepatic echogenicity [8].

CT examination

All patients underwent CT scan examination using a 16 multidetector CT scanner (GE BrightSpeed 16, USA). Non-contrast CT examination of the abdomen was done starting from the top of the diaphragm to the symphysis pubis. A dynamic post-contrast CT examination was then done. Iohexol (Omnipaque 350 mg of iodine/mL; GE Health Care) was injected at a rate of 3 to 5 mL/sec in a total of 100 to 150 mL of volume. The following CT parameters were employed to obtain dynamic data: acquisition in 1 s gantry rotation time, 100 kvp, 250 mA, and 5 mm reconstructed section thickness. Using contrast agent bolus tracking, the arterial phase was typically attained 15 to 25 s after administration. Between 40 and 60 s after injection, the portal phase was recorded. After 180 s, the delayed venous phase was acquired [9].

Post-processing

Data analysis was performed using commercial software (Synapse 3D software, Fujifilm Healthcare, USA) including the assessment of the visceral and subcutaneous fat amount, liver volume, and psoas muscle mass. A radiologist with 21 years of experience in abdominal imaging analyzed the CT scan data, blinded to the clinical and ultrasonographic findings.

Measurement of the subcutaneous and visceral fat

In the pre-contrast CT scan, fat was identified as the pixels ranging from − 250 to − 50 Hounsfield units. The subcutaneous fat was described as the extraperitoneal fat between the muscles and the skin. Visceral fat was defined as the intraperitoneal portion that had the same density as subcutaneous fat (Fig. 1) [10].

Subcutaneous fat volume (SFV), abdominal volume, and visceral fat volume (VFV) were measured from the top of the diaphragm to the symphysis pubis bone level. The fat rate in the body was estimated by dividing the

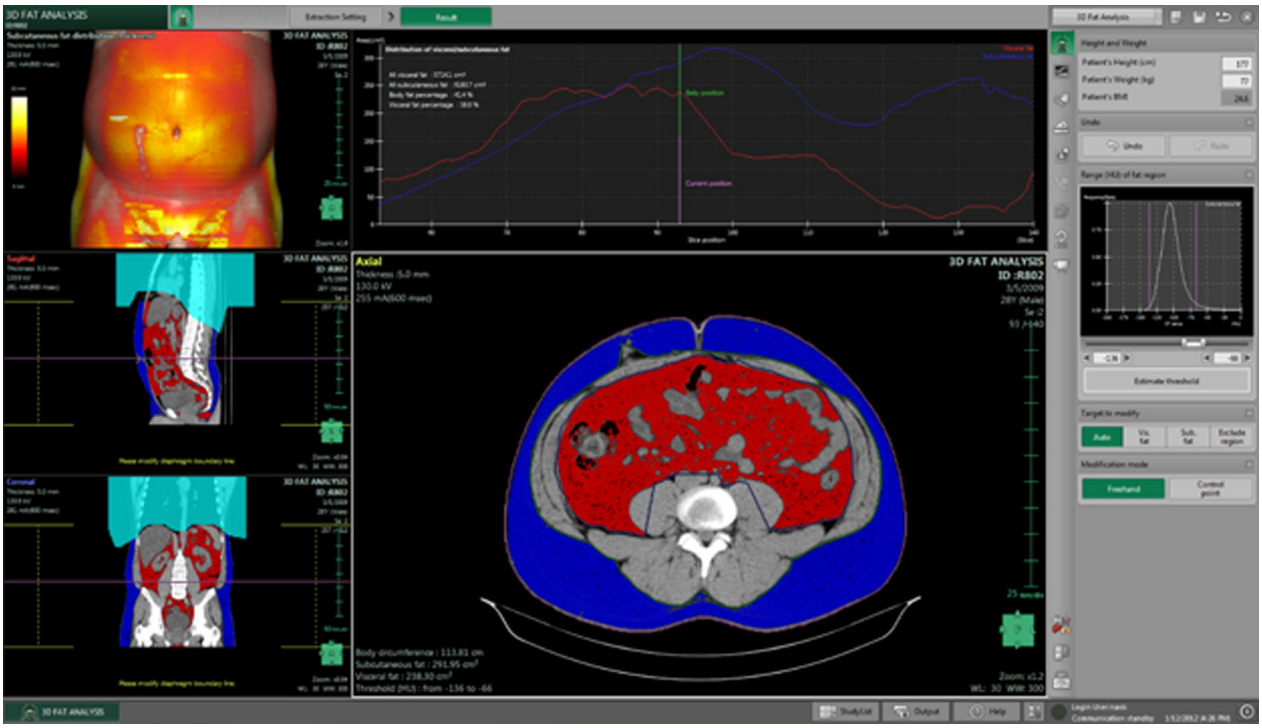

Fig. 1 Calculation of the subcutaneous fat volume (SFV) (blue) and visceral fat volume (VFV) (red). Subcutaneous fat is the extraperitoneal fat between the muscles and the skin. Visceral fat is the intraperitoneal portion that had the same density as subcutaneous fat

sum of subcutaneous and visceral fat volumes by the volume of the entire body. To calculate the visceral-to-subcutaneous (V/S) rate, VFV was divided by SFV. The visceral fat volume rate was calculated by dividing VFV by abdominal cavity volume [11].

Measurement of the liver volume

CT volumetry was done during the portal venous phases. The hepatic boundaries were traced to keep out neighboring structures, blood vessels, and liver fissures. A virtual hepatectomy plane is depicted on each slice on the axial scans, following the falciform ligament in the left lateral segmentectomy and to the right of the middle hepatic vein in the right hemihepatectomy [4, 5]. The volumes of all cuts are added together to determine the total liver volume. (Fig. 2) [12, 13].

Psoas muscle assessment

Psoas muscle volume was automatically calculated (Fig. 3); then, the psoas muscle volume was divided by the height of the patient to estimate the psoas muscle ratio [14].

Generation of the report

After the whole measurement had been calculated, an automatic report was then generated documenting the name, age, sex, weight, and height of the patient. The measurements that appeared in the report were: visceral fat volume, subcutaneous fat volume, fat rate in the body, visceral fat volume rate, BMI (basal metabolic index), 3D V/S rate (visceral-to-subcutaneous fat rate), and psoas major muscle volume. The obesity index of BMI was also graded as too thin, normal, or too fat (Fig. 4). The liver volume report was generated separately.

Statistical analysis

Data were summarized by the mean and standard deviation for numerical data and by percentage/frequency for categorical data. All statistical tests were done by IBM SPSS version 20. A comparison of visceral and subcutaneous fat, liver volume, and psoas muscle mass among different grades of fatty liver was made by one-way ANOVA test. Pearson correlation determined the correlation between visceral and subcutaneous fat, liver volume, and psoas muscle mass with liver enzymes. A 95% degree of confidence was maintained. If the *P* value was less than 0.05, it was significant.

Results

Baseline data of the enrolled patients ($n = 130$)

The mean age of the studied patients was 51.38 ± 11.11 years, and 53.8% of patients were females.

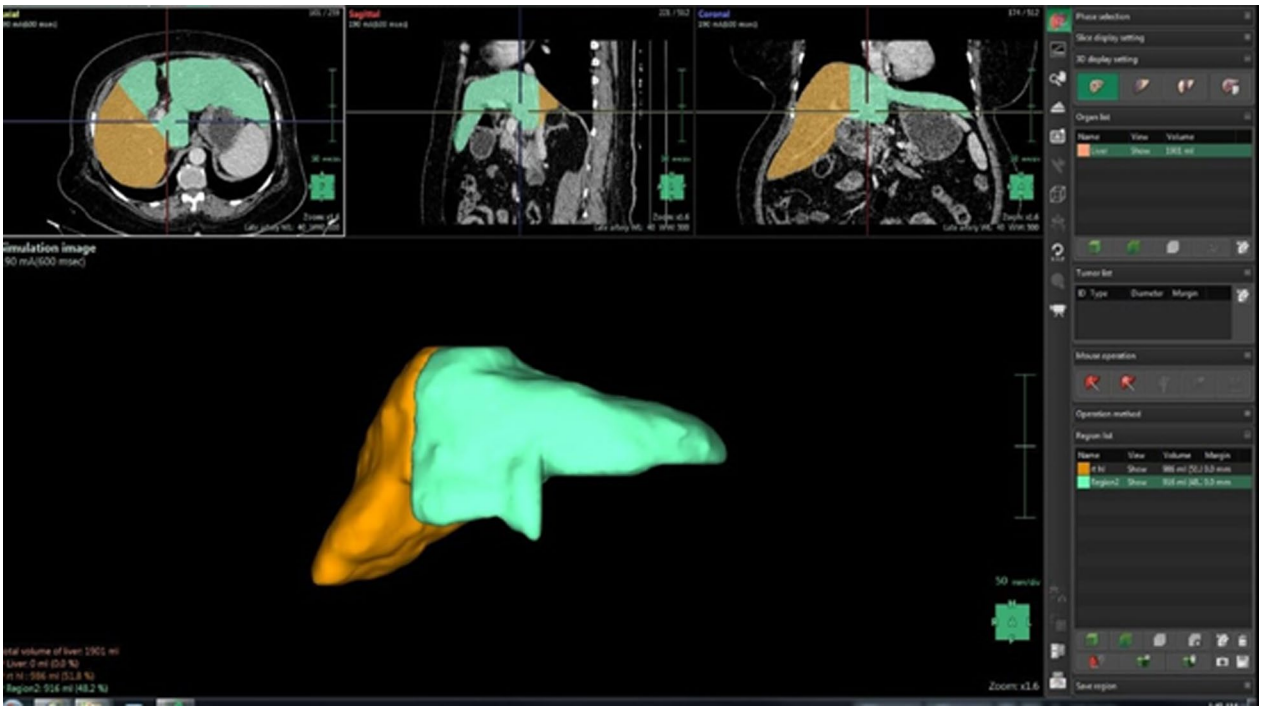

Fig. 2 Liver volumetry was done in the portal venous phase. The left lobe is colored green and the right lobe is colored yellow

Fig. 3 Psoas muscle volume. Both psoas muscles are shown in green color

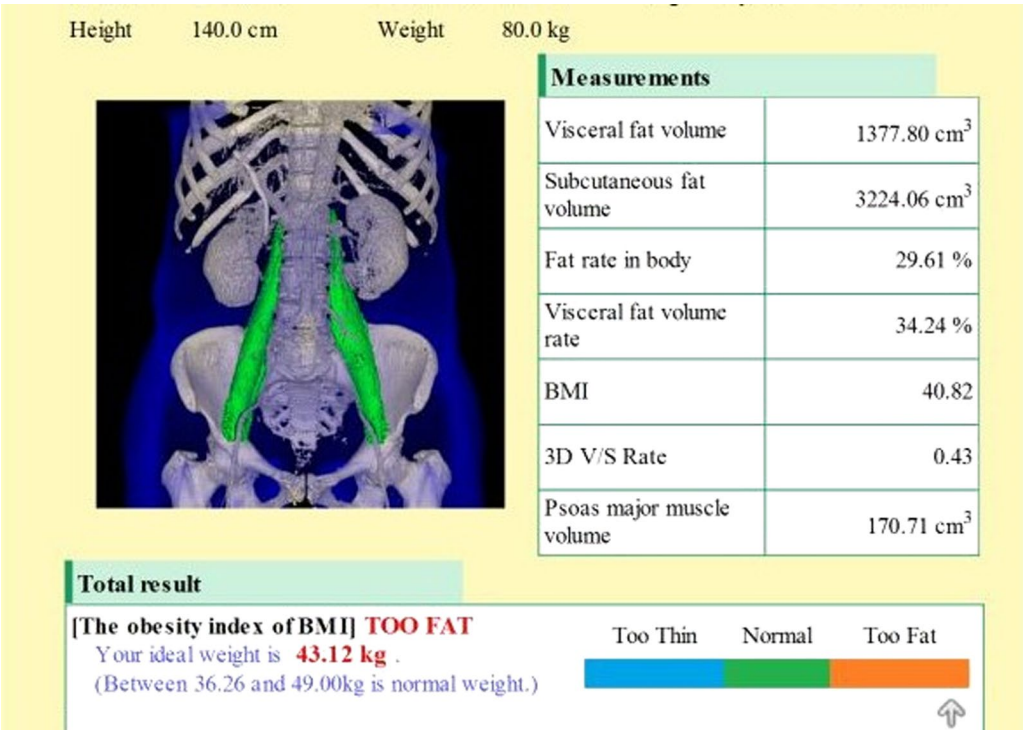

Fig. 4 Automatically generated report of the visceral and subcutaneous fat and psoas muscle volume

Table 1 Demographic data of enrolled patients ($N=130$)

Demographic data	No	Percent (%)
Age (years)		
< 20	2	1.54
20–	58	44.61
40–	68	52.31
60+	2	1.54
Sex		
Male	60	46.2
Female	70	53.8
Body mass index (kg/m^2)		
< 35	3	2.31
35–	61	46.92
40–	62	47.69
45+	4	3.08
Comorbidity		
Diabetes mellitus	50	38.5
Hypertension	40	30.8
Other comorbidities	20	15.4

Table 2 Liver enzymes and lipid profile among the studied patients ($N=130$)

Laboratory test	Mean (SD)
Alanine transaminase (u/L)	41.49 ± 21.77
Aspartate transaminase (u/L)	36.80 ± 18.18
Cholesterol (mg/dl)	197.13 ± 25.56
Triglyceride (mg/dl)	269.26 ± 80.93
Low-density lipoproteins (mg/dl)	105.74 ± 25.01
High-density lipoproteins (mg/dl)	32.80 ± 8.34

Table 3 Radiological grade of fatty liver among the studied patients ($N=130$)

Radiological grades of fatty liver	No. of cases	Percent (%)
Grade I	60	46.2
Grade I	42	32.3
Grade I	28	21.5

The mean body mass index was 41.23 ± 7.83 (kg/m^2). Fifty patients (38.5%) were diabetics and forty patients (30.8%) were hypertensive. The demographic data of the enrolled patients are given in Table 1. Liver enzymes and lipid profiles among the studied patients are given in Table 2.

Radiological grade of fatty liver among the studied patients ($n=130$)

Based on the ultrasonographic assessment of the studied patients, it was found that 60 (46.2%), 42 (32.3%), and 28 (21.5%) patients had grade I, grade II, and grade III fatty liver, respectively (Table 3).

Visceral and subcutaneous fat, psoas muscle mass, and liver volume among the studied patients ($n=130$)

Different parameters of visceral and subcutaneous fat are summarized in Table 4. The mean total fat volume was 466.09 ± 98.46 (cm^3), psoas muscle volume was 289.98 ± 104.24 (cm^3), psoas muscle ratio was 1.58 ± 0.45 (cm^3/cm), and liver volume was 2062.90 ± 555.95 (cm^3).

Correlation between fatty liver grade, visceral and subcutaneous fat, psoas muscle mass, and liver volume (Table 5)

Patients with grade III fatty liver had a significantly higher total fat volume, visceral fat volume, subcutaneous fat volume, visceral-to-subcutaneous fat rate, fat rate in the body, visceral fat volume rate, psoas muscle volume, and psoas muscle ratio than those with grade I and grade II ($P=0.03$, $P=0.03$, $P=0.04$, $P=0.02$, $P=0.03$, $P=0.03$, $P<0.001$, $P<0.001$, respectively). It was noticed that grade I and grade II had insignificant differences regarding those parameters. There is no significant difference in the liver volume among each grade of fatty liver.

Correlation between liver transaminases, visceral and subcutaneous fat, psoas muscle mass, and liver volume (Table 6)

Aspartate transaminase (AST) had a significant positive correlation with total fat volume ($P=0.02$), visceral fat volume rate ($P<0.001$), liver volume ($P=0.04$), psoas muscle volume ($P<0.001$), and psoas muscle ratio ($P<0.001$).

Alanine transaminase (ALT) had also a significant positive correlation with total fat volume ($P=0.01$), visceral fat volume rate ($P<0.001$), psoas muscle volume

Table 4 Visceral and subcutaneous fat, psoas muscle mass, and liver volume among the studied patients

Variable	Mean ± SD
Total fat volume (cm^3)	466.09 ± 98.46
Visceral fat volume (cm^3)	1566.84 ± 1621.85
Subcutaneous fat volume (cm^3)	5073.26 ± 4708.60
Visceral-to-subcutaneous fat rate (%)	0.42 ± 0.19
Fat rate in the body (%)	36.34 ± 15.12
Visceral fat volume rate (%)	24.42 ± 12.01
Psoas muscle volume (cm^3)	289.98 ± 104.24
Psoas muscle ratio (cm^3/cm)	1.58 ± 0.45
Liver volume (cm^3)	2062.90 ± 555.95

Table 6 Visceral, subcutaneous fat, liver volume, psoas muscle mass, and liver volume based on liver enzymes

Variable	AST	ALT
Total fat volume (cm^3)	0.22 (0.02)*	0.43 (0.01)*
Visceral fat volume (cm^3)	0.13 (0.09)	0.13 (0.29)
Subcutaneous fat volume (cm^3)	0.19 (0.12)	0.17 (0.08)
Visceral-to-subcutaneous fat rate (%)	0.15 (0.11)	0.14 (0.09)
Fat rate in the body (%)	0.21 (0.33)	0.10 (0.53)
Visceral fat volume rate (%)	0.78 (< 0.001)*	0.76 (< 0.001)*
Psoas muscle volume (cm^3)	0.81 (< 0.001)*	0.49 (< 0.001)*
Psoas muscle ratio (cm^3/cm)	0.84 (< 0.001)*	0.51 (< 0.001)*
Liver volume (cm^3)	0.28 (0.04)*	0.36 (< 0.001)*

Data presented as *r* (correlation strength) and *P* (significance of correlation)

*If *P* value was less than 0.05, it was significant

ALT, alanine transaminase; AST, aspartate transaminase

Table 5 Visceral and subcutaneous fat, psoas muscle mass, and liver volume based on fatty liver's grade

Variable	Grade I ($n=60$)	Grade II ($n=42$)	Grade III ($n=28$)	*P* value
Total fat volume (cm^3)	482.01 ± 92.72	460.67 ± 103.98	540.10 ± 102.78	0.03
Visceral fat volume (cm^3)	1045.16 ± 538.87	1634.77 ± 476.46	4030.07 ± 538.87	0.03
Subcutaneous fat volume (cm^3)	3074.01 ± 214.45	3987.24 ± 508.34	7750.14 ± 307.13	0.04
Visceral-to-subcutaneous fat rate (%)	0.34 ± 0.18	0.41 ± 0.13	0.52 ± 0.25	0.02
Fat rate in the body (%)	35.80 ± 14.15	34.33 ± 18.01	49.38 ± 15.19	0.03
Visceral fat volume rate (%)	25.10 ± 11.97	20.80 ± 12.20	36.77 ± 12.38	0.03
Psoas muscle volume (cm^3)	260.87 ± 101.49	244.52 ± 106.57	396.82 ± 99.16	< 0.001
Psoas muscle ratio (cm^3/cm)	1.40 ± 0.73	1.63 ± 0.35	1.83 ± 0.20	< 0.001
Liver volume (cm3)	2020.64 ± 525.54	2015.18 ± 666.19	2221.18 ± 434.89	0.57

Data expressed as mean (SD). *P* value was significant if < 0.05

($P < 0.001$), psoas muscle ratio ($P < 0.001$), and liver volume ($P < 0.001$). We did not find a significant correlation between liver transaminases and the other parameters.

Discussion

Both the prevalence and the degree of NAFLD are substantially associated with obesity. It is widely recognized that abdominal obesity, particularly high visceral adipose tissue, plays a significant role in the development of metabolic disorders and NAFLD. NAFLD can proceed to steatohepatitis, liver cirrhosis, and hepatocellular failure. It is also linked to metabolic syndrome, type 2 diabetes, insulin resistance, cardiovascular disorders, and hepatoma [15–17].

Although the pathophysiology of NAFLD is complicated, obesity is a major risk factor for its occurrence. The adipocytes' ability to release free fatty acids into the portal venous system is activated by the high levels of abdominal fat. This may subject the liver to a significant amount of fat, resulting in NAFLD [18–20].

Only a few studies have examined the effect of the amount of body fat and its distribution in the development of NAFLD. We postulated that body fat distribution shown by the visceral-to-subcutaneous fat (V/S) ratio would indicate the risk for the development of NAFLD and its severity.

In the current study, we aimed to assess the relationship between the severity of fatty liver in NAFLD and the amount of visceral fat, muscle mass, and liver volume. Also, to evaluate the correlations among the increase in the liver enzymes in NAFLD, visceral and subcutaneous fat, muscle mass, and liver volume.

Based on the radiological assessment of our patients, we found that 60 (46.2%) had grade I fatty liver, 42 (32.3%) had grade II fatty liver, and 28 (21.5%) patients had grade III fatty liver. We used abdominal ultrasonography to grade fatty liver, a simple widespread noninvasive modality. It is now replacing liver biopsy in NAFLD grading because it avoids any hazards that can occur during or after liver biopsy [5].

We found that the patients with grade III fatty liver had significantly higher total fat volume, visceral fat volume, subcutaneous fat volume, visceral-to-subcutaneous fat rate, fat rate in the body, and visceral fat volume rate in comparison with those with grade I and grade II fatty liver.

Our results are based on the fat volume rather than the fat areas used in the previous studies. For more accurate results, we calculated the fat rate in the body by dividing the sum of subcutaneous and visceral fat volumes by the volume of the entire body, the V/S rate by dividing VFV by SFV, and the visceral fat volume rate by dividing VFV by abdominal cavity volume.

In line with our findings, some previous studies found that the amount of visceral fat was significantly correlated with the degree of fatty liver, the degree of liver inflammation, as well as hepatic fibrosis [5, 19–24]. Additionally, the amount of visceral and subcutaneous fat and muscle was significantly higher in patients with grade II and III compared to those with grade I fatty liver [25].

These results were confirmed histologically by Eguchi et al. who reported that the amount of visceral fat was correlated with the progress of NASH, as evaluated by the histological grade. Therefore, from the onset of fat deposition in hepatocytes to the emergence of NASH, visceral fat accumulation continually affects the histological abnormalities in NAFLD [23].

Few previous papers studied fat distribution in the body as a risk factor in the development of NASH. One of these studies reported that the visceral-to-subcutaneous fat (V/S) ratio is more important than the visceral fat area as a metabolic risk factor in the progress of NASH and liver fibrosis [26]. However, the contribution of subcutaneous fat in NAFLD is debatable because some research found no association between SFV and NAFLD [27], while others found an opposite association [28].

In our study, we found that the amount of visceral fat is more important than the amount of subcutaneous fat in the detection of the severity of NAFLD which is similar to the results of the previous research. We found the V/S rate the most significant parameter ($P = 0.02$) followed by VFV, VFV rate, total fat volume, fat rate in the body ($P = 0.03$, each), and lastly SFV ($P = 0.04$).

The current research also studied the correlation between the increase in liver transaminases in NAFLD and the amount of visceral and subcutaneous fat. Recent epidemiological studies have demonstrated that the rise of liver enzymes is closely linked to liver fat content, especially in patients with a very high incidence of obesity and NAFLD [29–31].

In this study, we found that both transaminases had a significant positive correlation with total fat volume and visceral fat volume rate. Similar to our results, some authors found a correlation between visceral fat deposition and moderate elevation of the liver transaminases [29, 32]. On the other hand, a previous study revealed no significant correlation between liver transaminase and visceral fat area [25]. However, to our knowledge, no previous work handled the correlation between liver enzymes and visceral fat volume rate in which we used the fat volume rather than the fat area.

Regarding the psoas muscle mass, we found that patients with grade III fatty liver had a significantly higher psoas muscle volume and psoas muscle ratio than those with grade I and grade II. We also found that liver transaminases significantly correlated with psoas

muscle volume and psoas muscle ratio. In this study, we used psoas muscle volume which is more accurate than the psoas muscle area used in the previous studies. Our results are consistent with Chen et al. [33] and Nachit et al. [34] who found that NAFLD is strongly correlated with muscle fat rather than muscle mass. Although other studies reported that sarcopenia (decreased appendicular muscle mass) is associated with NAFLD [35, 36], they did not use muscle mass in grading NAFLD.

Regarding the liver volume, we found a strong positive correction between it and the liver transaminases but the difference in the liver volume among the different grades of NAFLD was insignificant. Only recent research studied the liver volume obtained by magnetic resonance imaging in NAFLD and reported that there is a strong correlation between liver volume, transaminases, and high risk of mortality [37].

The main limitations of this study are the lack of histopathological correlation, a single-center study, and lastly no long-term follow-up of those patients. Yet this is the first study in our locality that discussed the relation between NAFLD and visceral and subcutaneous fat and its correlations with liver transaminases.

Conclusions

The amount of subcutaneous and visceral fat deposition, as well as appendicular muscle mass in patients with non-alcoholic fatty liver disease, was increasing with advances in the disease grades. Also, liver enzymes had a significant positive correlation with the amount of total and visceral fat, psoas muscle mass, and liver volume. Multicenter future studies are warranted to confirm these findings.

Abbreviations

NAFLD	Non-alcoholic fatty liver disease
NASH	Non-alcoholic steatohepatitis
CT	Computed tomography
SFV	Subcutaneous fat volume
VFV	Visceral fat volume
V/S	Visceral-to-subcutaneous fat
AST	Aspartate transaminase
ALT	Alanine transaminase

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Author contributions

OM, GM, WA, and HA designed the research. OM, GM, and HA performed the research and wrote the manuscript. OM, GM, MA, and HMA analyzed the collected data. MA and HMA revised the data and manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

Data are available upon request with the corresponding author.

Declarations

Ethics approval and consent to participate

The study was conducted after approval of the Ethical Committee of the Faculty of Medicine (approval number 17100712) and after clinical trial approval (NCT04240145). Informed written consent was obtained from each participant.

Consent for publication

All patients included in this study gave written informed consent to publish the data contained in this study.

Competing interests

The authors declare that they have no competing interests.

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