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Research Article

Liver Fatty Acid-Binding Protein as a Diagnostic Marker for Non-Alcoholic Fatty Liver Disease

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Abstract

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as NAFLD, is the most common cause of chronic liver disease in the developed world. It represents a spectrum of diseases starting from metabolic dysfunction to cirrhosis and hepatocellular carcinoma. Liver biopsy remains the gold standard for diagnosis of MASLD. However, liver biopsy has significant limitations, including pain, risk of severe complications, sampling error, cost and patient unwillingness to undergo invasive testing. Therefore, it is preferable to use effective noninvasive methods in clinical practice for identifying the disease, tracking its processes, and monitoring treatment effects.

Aim of the Work: The aim of the current study was to investigate the diagnostic value of Liver Fatty Acid-Binding Protein (L-FABP) as a noninvasive marker for metabolic-dysfunction associated steatotic liver disease (MASLD).

Patients and Methods: We enrolled in this study 80 consecutive MASLD patients presented to the GIT clinic and internal medicine clinic at Ain Shams University Hospitals between November 2021 and November 2022. Patients were diagnosed with ultrasonography and elastography. The control group consisted of 20 healthy control subjects matched for age and gender. Serum levels of L-FABP were determined by enzyme-linked immunosorbent assay. L-FABP was measured in patients and controls as well as laboratory and imaging modalities for diagnosis.

Results: L-FABP levels in MASLD patients were higher than in the control group. According to ROC curve analysis, we have found that LFABP was diagnostic of MASLD at a Cutoff value >145 ng/L with sensitivity of 88.7%, specificity of 85%, PPV of 95.9, NPV of 65.4 and Accuracy of 90.7%.

Conclusion: Serum L-FABP could be considered as a reliable non-invasive marker for detection of MASLD with a significant positive correlation between its serum level and degree of steatosis and fibrosis.

Keywords: Liver Fatty Acid-Binding Protein, Non-Alcoholic Fatty Liver Disease

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Introduction

MASLD is the most common cause of chronic liver disease in the developed world and will probably emerge as the leading cause of end-stage liver disease in the coming decade [1].

It has far-reaching implications beyond the liver, including an elevated risk of cardiovascular disease (CVD), obstructive sleep apnea, osteoporosis, chronic kidney disease, and extrahepatic malignancies such as carcinomas of the colon, stomach, pancreas, uterus, and breast.[2].

It encompasses a wide spectrum of histological and clinical manifestations, ranging from simple steatosis to steatohepatitis, fibrosis and cirrhosis [3]. The global

prevalence of MASLD is currently estimated to be 24% [4]. But the highest rates are reported from South America (31%) and the Middle East (32%), followed by Asia (27%), the USA (24%) and Europe (23%), whereas MASLD is less common in Africa (14%) [4].

Fatty acid-binding proteins (FABPs) are a family of small and highly conserved lipid chaperone molecules with highly varied functions [5]. Different members of the FABP family exhibit unique patterns of tissue expression and are expressed most abundantly in tissues involved in active lipid metabolism in hepatocytes, adipocytes and cardiac myocytes, where fatty acids are prominent substrates for lipid biosynthesis, storage or breakdown, the respective FABPs make up between 1% and 5% of all soluble cytosolic proteins [6]. In the hepatic lobule, L-FABP is expressed in hepatocytes in a declining gradient from portal to central location [7]. L-FABP is a protein involved in multiple biologic functions, such as intracellular fatty acid transport, cholesterol and phospholipid metabolism, which plays an important facilitative role in hepatic fatty acid oxidation. Some studies reported that L-FABP level is increased in MASLD [8].

Liver biopsy remains the gold standard for diagnosis of MASLD [9]. However, liver biopsy has significant limitations, including pain, risk of severe complications, sampling error, cost and patient unwillingness to undergo invasive testing [10].

Therefore, it is preferable to use effective noninvasive methods in clinical practice for identifying MASLD, tracking disease processes, and monitoring treatment effects [11]. Many studies demonstrated that serological markers are beneficial as noninvasive tools for diagnosis of MASLD[12]. This study was conducted to determine whether serum L-FABP has diagnostic value as a marker for MASLD.

Patients and Methods

This study included 100 subjects divided in 2 groups: Group I: included 80 patients diagnosed to have nonalcoholic fatty liver disease (MASLD) by Fatty liver index (FLI) score, abdominal ultrasound and transient elastography (Fibroscan® with Controlled Attenuation Parameter (CAP)), Group II: included 20 healthy control subjects.

Patients showing clinical or laboratory evidence of acute or chronic decompensated liver disease were excluded from the study. Likewise,,

patients with chronic liver disease due to HCV, HBV, autoimmune hepatitis or hereditary liver disease were also excluded. Patients known to have chronic kidney disease, alcoholic patients pregnant ladies as well as any patient on drugs causing secondary steatosis as corticosteroids, amiodarone, tetracycline, valproic acid were excluded from the study.

All participants were subjected to the following:

1. Full history taking including age, sex, alcohol intake, diabetic history, medical history and drug history.

2. Physical examination including:

Blood pressure measurement, measuring body mass index (BMI) by dividing weight (kg) by height (m2), measuring waist circumference (cm) midway between costal margin and iliac crest.

3. Laboratory assessment by the following:

Aspartate amino transferase (AST), alanine amino transferase (ALT), gamma-glutamyl-transferase (GGT), complete blood count (CBC), serum albumin level, coagulation profile, serum creatinine level, lipid profile (including Cholesterol, LDL, HDL and triglycerides levels), random blood sugar, viral markers (HCV antibodies, HBsAg), anti-nuclear antibody (ANA), serum ferritin.

4. Transient elastography (Fibroscan) with controlled attenuation parameter for assessment of liver steatosis:

While the patient is in the supine position, a probe of a real time scanner (Fibrosense®) was placed sub-costally in the right mid-clavicular line in a sagittal plane showing a good liver window. One experienced operator in the National Hepatic Institute was responsible for scanning all cases to minimize errors.

5. Abdominal ultrasound:

All abdominal ultrasound examinations were performed by the same specialist. The severity of MASLD on ultrasound was graded as follows: grade 1 (mild), defined as a slight diffuse increase in liver echogenicity in the hepatic parenchyma with normal visualization of the diaphragm and portal veins; grade 2 (moderate), defined as a moderately diffuse increase in liver echogenicity with a slightly impaired visualization of the diaphragm and portal veins; and grade 3 (severe), defined as a marked increase in liver echogenicity with poor or no visualization of the diaphragm and portal veins.

6. Fatty Liver Index (FLI):

The FLI was calculated according to a previously published report by Bedogni et al. 2006 [37]: FLI = [e0.953×loge (TGs) + 0.139×BMI + 0.718×loge (GGT) + 0.053×waist circumference-15.745)] / [1+ e0.953×loge (TGs) + 0.139×BMI + 0.718×loge (GGT) + 0.053×waist circumference-15.745] ×100, with TGs measured in mmol/l, GGT in U/l, and waist circumference in cm.

III. Statistical analysis:

Data was collected, coded, translated to English to facilitate data manipulation and double entered into Microsoft Access and data analysis was performed using SPSS software version 18 under windows 10. Simple descriptive analysis in the form of numbers and percentages for qualitative data, and arithmetic means as central tendency measurement, standard deviations as measure of dispersion for quantitative parametric

data, and inferential statistic test. Statistical presentation and analysis of the present study was conducted, using the mean, standard deviation, student t- test, Chi-square, Linear Correlation Coefficient and Analysis of variance [ANOVA] tests by SPSS V17. The level $P \leq 0.05$ was considered the cut-off value for significance.

Results

The study included 80 consecutive patients with MASLD (Group I) and 20 apparently healthy subjects (Group II) matched for age and sex.

Mean age of patients in group I was 46 years ± 11 (22 – 76 years old) (with a male to female ratio of 7:13, while age ranged from 21 – 55 years (42 ± 8) in the control group with male cases representing 55% (table1).Patients with MASLD (group 1) had higher blood pressure, body weight and waist circumference with statisticaly significant difference. They also had higher incidence of Diabetes and hypertension (table 1).

Table 1: Demographic investigations in group I & II

		Group			**Chi-Square**	
		Group I (80 person)		**Group II (20 person)**		
		Number	**%**	**Number**	**%**	**X^2**	**P-value**
Gender	**Male**	28	35.00	11	55.00	2.690	0.101
	Female	52	65.00	9	45.00		
DM	**Yes**	38	47.50	0	0.00	15.323	<0.001*
	No	42	52.50	20	100.00		
Hypertension	**Yes**	29	36.25	0	0.00	10.211	0.001*
	No	51	63.75	20	100.00		
T-Test						**T**	**P-value**
Age (years)	**Range**	22 - 76		21 - 55		1.388	0.168
	Mean ±SD	46.250 ± 11.354		42.500 ± 8.140			
BMI (kg/m2)	**Range**	25 - 43.7		21 - 28		10.330	<0.001*
	Mean ±SD	32.718 ± 3.720		23.765 ± 2.107			
Waist (cm)	**Range**	80 - 107		65 - 90		17.451	<0.001*
	Mean ±SD	99.588 ± 5.532		74.650 ± 6.426			
Systole (mmHg)	**Range**	110 - 150		110 - 130		5.937	<0.001*
	Mean ±SD	130.250 ± 9.308		117.250 ± 5.955			
Diastole (mmHg)	**Range**	70 - 95		70 - 85		5.566	<0.001*
	Mean ±SD	82.875 ± 5.779		75.000 ± 5.130			

Table 2: Comparison of routine investigations between group I & group II

		Group I		**Group II**		**T**	**P-value**
CHOL (mg/dl)	**Range**	69 - 302		124 - 184		4.785	<0.001*
	Mean ±SD	206.475 ± 44.946		157.450 ± 16.256			
TG (mg/dl)	**Range**	23 - 332		82 - 136		3.564	0.001*
	Mean ±SD	157.638 ± 64.693		105.450 ± 17.221			
HDL (mg/dl)	**Range**	30 - 47		56 - 69		-26.505	<0.001*
	Mean ±SD	39.038 ± 3.458		62.250 ± 3.683			
LDL (mg/dl)	**Range**	137 - 183		110 - 130		16.630	<0.001*
	Mean ±SD	157.575 ± 9.930		119.250 ± 5.320			
Creat (mg/dl)	**Range**	0.08 - 2.9		0.12 - 2.9		0.193	0.848
	Mean ±SD	0.817 ± 0.630		0.787 ± 0.637			
Urea (mg/dl)	**Range**	9 - 39		11 - 37		-1.219	0.226
	Mean ±SD	24.100 ± 7.459		26.300 ± 6.140			
T. Bil (mg/dl)	**Range**	0.04 - 1.32		0.04 - 1.14		-0.112	0.911
	Mean ±SD	0.719 ± 0.236		0.726 ± 0.261			
D. Bil (mg/dl)	**Range**	-0.03 - 0.56		0.07 - 0.48		-0.039	0.969
	Mean ±SD	0.264 ± 0.120		0.266 ± 0.105			
ALB (g/dl)	**Range**	3.1 - 5.8		3.4 - 5.8		-0.321	0.749
	Mean ±SD	4.365 ± 0.559		4.410 ± 0.571			
INR	**Range**	0.9 - 1.1		0.9 - 1.1		-0.362	0.718
	Mean ±SD	1.024 ± 0.068		1.030 ± 0.073			
AST (U/l)	**Range**	18 - 68		8 - 35		7.159	<0.001*
	Mean ±SD	38.638 ± 9.251		22.45 ± 8.13			
ALT (U/l)	**Range**	21 - 75		8 - 35		9.64	<0.001*

	Mean ±SD	45.200	±	9.596	22.85	±	7.78		
ALP (U/l)	**Range**	33	-	107	33	-	87	0.983	0.328
	Mean ±SD	68.788	±	17.994	64.600	±	12.279		
FBS (mg/dl)	**Range**	65	-	180	79	-	107	3.589	0.001*
	Mean ±SD	121.213	±	33.704	93.850	±	8.647		
HbA1c (%)	**Range**	3.5	-	10	4.3	-	5.9	3.886	<0.001*
	Mean ±SD	6.558	±	1.748	5.020	±	0.462		
WBCs (10^9/l)	**Range**	3.1	-	10	2.9	-	8.8	1.076	0.285
	Mean ±SD	6.188	±	1.710	5.725	±	1.757		
Hb (g/dl)	**Range**	9.9	-	15.8	10.9	-	14.7	1.226	0.223
	Mean ±SD	13.414	±	1.354	13.005	±	1.245		
PLTs (10^9/l)	**Range**	193	-	487	307	-	450	-0.651	0.516
	Mean ±SD	362.938	±	51.907	371.100	±	42.021		
GGT (U/l)	**Range**	11	-	76	14	-	48	8.596	<0.001*
	Mean ±SD	52.025	±	12.878	25.700	±	9.189		
RBCs (10^{12}/l)	**Range**	3.6	-	5.4	3.7	-	5.3	0.763	0.447
	Mean ±SD	4.568	±	0.448	4.480	±	0.500		
Ferritin (ugm/l)	**Range**	13	-	95	18	-	32	7.254	<0.001*
	Mean ±SD	55.300	±	17.943	25.900	±	4.128		

Fasting blood glucose, glycated haemoglobin, total cholesterol, LDL & triglycerides were higher in patients than controls while and HDL was lower. Liver enzymes AST, ALT & GGT were also significantly higher in patients than in the control group. However, no significant difference in bilirubin, serum albumin or alkaline phosphatase (ALP) was found between the 2 groups (table 2).

Moreover, Fatty liver index (FLI) and LFABP there were higher in group I than group II as shown in (table 3).

Table 3: Comparison of FLI and LFABP between group I & group II

		Group I			Group II			T	P-value
Fatty Liver Index	**Range**	60	-	96	4	-	29	26.853	<0.001*
	Mean ±SD	81.075	±	10.244	15.700	±	7.270		
LFABP (ng/mL)	**Range**	106.73	-	387.27	133	-	175	4.994	<0.001*
	Mean ±SD	195.885	±	47.721	141.900	±	13.167		

LFABP was found to be statistically correlated to HDL, AST, FBS, HbA1C, waist circumference, steatosis level, fatty liver index and fibrosis score, and not correlated to other labs as shown in (table 4).

Table 4: Correlation of LFABP to routine investigations

Correlations		
	LFABP	
	R	**P-value**
Age	-0.153	0.176
BMI	0.152	0.180
CHOL	0.021	0.854
TG	0.073	0.519
HDL	0.251	0.025*
LDL	-0.105	0.353
Creat	0.112	0.321
Urea	-0.008	0.947
T. Bil	-0.044	0.697
D. Bil	0.053	0.640
ALB	0.119	0.294
INR	-0.109	0.337
AST	-0.239	0.032*
ALT	-0.210	0.062
ALP	-0.071	0.529
FBS	0.242	0.030*
HbA1C	0.224	0.046*

WBCs	0.177	0.116
Hb	0.065	0.567
PLTs	0.022	0.849
GGT	0.098	0.385
RBCs	-0.040	0.725
Steatosis	0.733	<0.001*
Systole	-0.179	0.112
Diastole	-0.214	0.057
Waist	0.435	<0.001*
Ferritin	0.022	0.846
Fatty Liver Index	0.286	0.010*
Fibrosis score	0.553	<0.001*
NAFLD F. score	0.137	0.224

According to ROC curve analysis , LFABP at a cutoff value of >145 ng/ml showed a sensitivity of 88.75% and specificity of 85% for diagnosis of MASLD. table (5) and figure (1).

Table 5: ROC curve analysis of LFABP

ROC curve between Group I and Group II						
	Cutoff	**Sens.**	**Spec.**	**PPV**	**NPV**	**Accuracy**
LFABP (ng/ml)	**>145**	**88.75**	**85.00**	**95.9**	**65.4**	**90.7%**

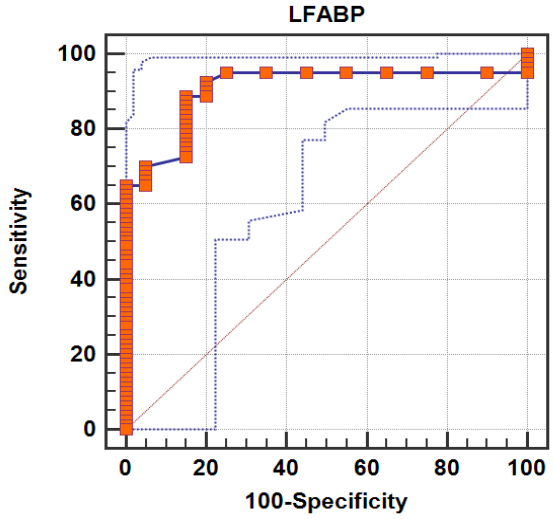

Figure 1: Sensitivity & Specificity of LFABP.

Correlation of LFABP to steatosis grades:
Patients with hepatic steatosis were divided by ultrasound in to 3 grades according to their steatosis appearance (G1,2,3): G1: involved 17 patients (21.25%), G2: involved 31 patients(38.75%), G3: involved 32 patients (40%)They were also divided by fibroscan with CAP in to 3 grades according to their steatosis score (S1,2,3) with S1: involving 15 patients, S2: 29 patients and S3: 36 patients.We have found that LFABP was positively correlated to steatosis grades by elastography (r=0.733, p value<0.001*), and the level of LFABP was increased as steatosis grade increased as in table (7,

Table 6: Comparison of LFABP to steatosis grades

		LFABP		**ANOVA**	
		Number of patients	**LFABP measurement (Mean ± SD)**	**F**	**P-value**
Steatosis Grade	**S1**	15 (18.75%)	138.635 ± 16.091	37.603	<0.001*
	S2	29 (36.25%)	185.815 ± 26.458		
	S3	36 (45.00%)	227.851 ± 44.009		

Comparison of LFABP to fibrosis grades:

Patients were additionally divided by fibro scan in to 3 grades according to their fibrosis score (F0,1,2): F0: involved 14 patients (17.50%), F1: involved 30 patients (37.50%), F2: involved 36 patients (45.00%).

LFABP was positively correlated to fibrosis grades (r=0.553, p value <0.001*) as shown in table ().

Table 7: Comparison of LFABP to fibrosis grades

		LFABP			ANOVA	
		Number of patients	**LFABP measurement (Mean ± SD)**		**F**	**P-value**
Fibrosis Grade	**F0**	14	156.547	± 25.498	24.782	<0.001*
	F1	30	175.883	± 33.841		
	F2	36	227.851	± 44.009		

Discussion

MASLD is the most common cause of chronic liver disease in the developed world ***Ando and Jou*** [1] and represents a spectrum of diseases with some patients developing cirrhosis and hepatocellular carcinoma [3]**.** Liver biopsy remains the gold standard for diagnosis of MASLD [9]**.** However, liver biopsy has significant limitations, including pain, risk of severe complications, sampling error, cost and patient unwillingness to undergo invasive testing [10]. Therefore, it is preferable to use effective noninvasive methods in clinical practice for identifying MASLD, tracking disease processes, and monitoring treatment effects [11]. Many studies demonstrated that serological markers are beneficial as non-invasive tools for diagnosis of MASLD [12]. L-FABP has a low molecular weight and is present in liver cells. The properties of L-FABP lead to its elevation even with a small amount of cell injury. Since hepatocytes are in direct contact with the blood and have no interstitial barrier, small proteins appear sooner in the circulation than large proteins. For these reasons, L-FABP could be a promising biochemical marker for early detection of liver cell injury [13]. The aim of this study was to assess the diagnostic value of L-FABP in the detection of MASLD.

In the present study, there was a strong relationship between MASLD and body mass index (BMI), in which BMI was markedly elevated in MASLD group (32.7 kg/m^2 ± 3.7) than control group (23.7 kg/m^2 ± 2). This association was established in multiple studies, for example the study of ***Parente et al.*** [14] who reported that preperitoneal fat is a non-invasive marker of increased risk of severe non-alcoholic fatty liver disease in patients with type 2 diabetes and the study of ***Zhou et al.*** [15] who reported a strong relationship between MASLD and visceral fatty obesity. ***Eslami et al.*** [16] additionally reported the use of BMI as a simple and practical predictive factor for the MASLD onset, with a cutoff level of 25.5 kg/m^2.

In our study, there was a significant difference in the lipid profile between the two groups, in which total cholesterol, LDL & triglycerides were higher in the MASLD group than Control group, and HDL was lower in the MASLD group than control group. This finding agrees with the research by ***Younossi et al.*** [17] who reported a strong relationship between MASLD and combined dyslipidemia, ***Harrison et al.*** [18] who reported that hypercholesterolemia was found in 44% of patients with MASLD. and ***Chan et al.*** [19] reported that hypertriglyceridemia has frequenly reported in MASLD. The pathogenesis of dyslipidemia in MASLD is not well understood, but it is likely related to hepatic overproduction of the very low-density lipoprotein particles and dysregulated clearance of lipoproteins from the circulation. Dyslipidemia in MASLD is a strong risk factor for cardiovascular disease [20].

AST and ALT are enzymes present in the hepatocytes and are well known markers of hepatocellular injury. Also, the glycoprotein gamma glutamyltransferase (GGT) is located on membranes of cells with high secretory or absorptive activities and is significantly increased in hepatobiliary diseases [21]**.** In the studied participants, AST, ALT and GGT levels were elevated in the MASLD group more than control group as evident by abundant research, for example, Armstrong et al. who reported that MASLD was the most common cause of abnormal liver biochemistry [22]**,**Debmalya et al. who concluded that MASLD was significantly associated with higher ALT and GGT [23] in addition to ***Fuji et al.*** [24] who reported a strong relation between MASLD and elevated GGT.

. Fatty liver disease is known to be strongly associated with elevated blood glucose. Diabetes mellitus was observed in 38 patients in the MASLD group (47.5%) matching the opinion of several authors as ***Assim et al.*** [25] who stated that MASLD is highly prevalent in patients with type 2 diabetes mellitus**,** the study of ***Antonio et al.*** [26] who reported strong correlation between type 2 DM and MASLD and the study of ***Chen et al.*** [27] who reported a positive association between type2 DM status and prevalence of hepatic steatosis and fibrosis. Additionally, the study of ***Marieke et al.***[28] reported that fatty liver was prevalent in 20% of patients with Type 1 DM and 76% of patients with Type 2 DM. Furthermore, ***Teruki et al.*** [29] reported that MASLD is a risk factor for T2DM**.** MASLD and Type2 DM have similar risk factors, and epidemiology and pathophysiology which further emphasize their connections [30]**.**

Regarding hypertension,36.25 % of our MASLD patients were hypertensive. Likewise, many studies reported that MASLD contributed independently to the development of hypertension and vice versa. Studies by ***Yang et al.*** [31] and ***Gerui et al.*** [32] reported a strong relationship between MASLD and Hypertension. The mechanism underlying this interaction might be insulin

resistance or inflammation with the activation of the sympathetic nervous system and renin-angiotensin system [33].

L-FABP and MASLD

The current study revealed a statistically significant elevation in serum concentration of L-FABP levels in MASLD group (195.8 ng/mL ± 47.7) than control group (141.9 ng/mL ± 13). Similar research by ***Akbal et al.*** [34] and ***Badawy et al.*** [35] reported that serum L-FABP levels were elevated in MASLD patients.

In the present study, Roc curve analysis of L-FABP as a diagnostic test of MASLD suggested that at the cut off value >145 ng/mL differentiated MASLD patients from healthy individuals with a sensitivity, specificity, positive and negative predictive values of 88.7%, 85%, 95.9% and 65.4% respectively. The study by ***Akbal et al.*** [34] also reported that LFABP can be used to differentiate MASLD from healthy controls at a cut-off value of 222.54 ng/mL for L-FABP (80% sensitivity and 80% specificity), positive and negative predictive values of L-FABP were 82% and 81%, respectively and when the cut-off value was 284 ng/mL, L-FABP had 73% sensitivity and 100% specificity, positive and negative predictive values for L-FABP were 100% and 79%, respectively [34].

Additionally, the study by ***Badawy et al.*** [35] reported that in the receiver operating curve (ROC), the area under curve (AUC) for L-FABP was 0.839 at a cut-off point 151.1 ng/mL with sensitivity, specificity, positive and negative predictive values were 83.3%, 71.8%, 31.3% and 96.6% respectively while at a cut-off point 189.5 ng/mL the AUC was 0.950 with sensitivity, specificity, positive and negative predictive values were 90%, 85%, 95.4% and 70.8% respectively which was statistically significant ($p < 0.001$) [35].

In the current study, we have found that LFABP was correlated to AST in agreement with the studies by ***Akbal et al.*** [34] and ***Badawy et al.*** [35] who have reported statistically significant correlations between LFABP and AST, ALT and GGT.

On the other hand, the study by ***Chang et al.*** [36] reported no significant association between LFABP and AST, ALT or GGT,and we reported no statistically significant correlations of LFABP with ALT and GGT in this study.

We additionally reported a positive correlation of LFABP to high density lipoprotein (HDL); however, no statistical correlation to cholesterol, LDL or Triglycerides. Similarly, ***Chang et al.*** [36] and ***Hassaan et al.*** [38] have reported that no significant correlation has been found between LFABP and all lipid profile (Cholesterol, LDL, HDL & triglycerides).

Although LFABP was positively correlated to waist circumference, it was not correlated to BMI in our study ***Hassaan et al.*** [38] has also reported negative correlation of LFABP to BMI. However, previous studies by ***Chang et al.*** [36]***and Akbal et al.*** [34]have reported positive correlation between LFABP and BMI.

In the current study, we have found that LFABP was positively correlatedto the degree of steatosis of fatty liver detected by ultrasound and fibroscan and the degree of fibrosis by elastography ($p < 0.001$). ***Badawy et al.*** [35] also who reported similar correlation between LFABP and ultrasound grades in detection of MASLD.

Finally,.LFABP was also positively correlated to Fatty liver index (FLI) score with statistical significance ($p < 0.001$). This means that there is a significant correlation between LFABP and fibroscan in identification of fibrosis grades.

Our findings

Conclusion

Serum L-FABP could be considered as a reliable non-invasive marker for detection of MASLD with a significant positive correlation between its serum level and degree of steatosis and fibrosis.

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