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# Liver Fatty Acid-binding Protein (L-FABP) as a Diagnostic Marker for Non-alcoholic Fatty Liver Disease

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## ABSTRACT

**Background:** Non-alcoholic fatty liver disease (NAFLD) is the leading cause of chronic liver disease worldwide; thus, an early and accurate diagnosis will improve the prognosis therapeutic interventions.

**Aim:** To assess the diagnostic value of liver fatty acid binding protein (L-FABP) for liver injury in non-alcoholic fatty liver disease (NAFLD).

**Patients and methods:** This prospective case-control study was conducted at the Hepatology, Gastroenterology, and Infectious Diseases Clinics, Zagagzig University Hospitals, Egypt on 60 participants divided into three groups: 20 members of the NAFLD in group I had high liver enzymes, 20 members of the NAFLD in group II had normal liver enzymes and 20 members of the healthy control group in group III. L-FABP was measured in all subjects.

**Results:** Regarding L-FABP levels, all of the examined groups showed a significant statistical difference; group I had higher levels than groups II and III, whereas group II had higher levels than group III.

**Conclusion:** The diagnostic biomarker fatty acid-binding liver protein is very useful for NAFLD and a good diagnostic tool of fatty liver injury as its concentrations reflect the level invasion of fat into the liver tissue.

**Keywords:** L-FABP, Diagnosis, NAFLD.

## INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is one of the most frequent causes of chronic liver disease worldwide. The hallmark of NAFLD, which can occur with or without inflammation and fibrosis, is an increase in intrahepatic triglyceride (TG) levels in the absence of alcohol abuse, pharmacological side effects, or viral hepatitis [(1)].

Steatosis alone, which is normally benign, can develop into steatohepatitis, which involves inflammation and fibrosis. Cirrhosis, liver failure, and, in some cases, hepatocellular cancer can then develop as a result of this condition. Diabetes mellitus (DM), metabolic syndrome, and obesity are frequently linked to NAFLD. It is also seen as a component of the metabolic syndrome [(2)].

Due to their expanding influence on global health, non-alcoholic fatty liver disease (NAFLD) and its more severe stage, nonalcoholic Steatohepatitis (NASH), have drawn interest on a global scale. The estimated prevalence of NAFLD worldwide is ~25% and Middle East has the highest levels (32%), South America (31%), USA (24%), Europe (23%), and lowest in Africa (14%) [(3)].

The economic cost of this expanding disease prevalence will rise along with it, and it will be accompanied by an alarming rise in hepatocellular carcinoma cases as well as an increase in the number of individuals requiring liver transplants who have cirrhosis and advanced liver disease. Most HCCs that develop in NASH happen before patients develop cirrhosis and standard cancer screening is carried out [(3)].

A family of 15-kDa proteins is known as FABPs. According to the tissues, nine distinct FABPs have been found and given names. Each member of this family of proteins has been given a name derived from the original tissue that it was separated. L-FABP (liver fatty acid-binding protein), intestinal FABP, heart FABP, and epidermal FABP are significant members of this family [(4)].

Although the kidney and small intestine also contain trace levels of L-FABP, the liver is where it is expressed most frequently. Several biological processes, including intracellular fatty acid transport, cholesterol metabolism, and phospholipid metabolism, are regulated by the protein L-FABP. L-FABP is a key facilitator of hepatic fatty acid oxidation [(5)].

Since serum L-FABP is correlated with fibrosis and activity index scores in hepatitis C patients, it is correlated with liver injury and according to studies on chronic hepatitis C, non-alcoholic steatohepatitis, and non-alcoholic fatty liver disease, it may be a novel diagnostic sign to identify liver injury [(6,7)].

We aimed to assess the diagnostic value of liver fatty acid binding protein (L-FABP) for liver injury in non-alcoholic fatty liver disease (NAFLD).

## PATIENTS AND METHODS

Sixty individuals were separated into three groups of twenty each. The study was carried out in Clinics of Hepatology, Gastroenterology and Infectious disease at Tropical Medicine Department, Zagagzig University Hospitals in Egypt.



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All participants who had steatosis, which is seen in ultrasonography, which can detect non-alcoholic fatty liver disease with or without increased transaminases were covered by the research.

Patients under the age of 18, people using any steatogenic medications (such amiodarone, valproic acid, corticosteroids, and tetracyclines), those who consume alcohol in any amount or have a history of doing so, those with any heart illness (including congestive heart failure or cardiomyopathy), interventional coronary angiography, a history of coronary artery disease, hypertension diabetes mellitus, renal failure, or those with hepatitis C or B, hemochromatosis, Wilson disease or autoimmune hepatitis, those who have polycystic kidney disease, chronic renal disease, or who refuse to give informed consent to participate in the study were not included in it.

**Patients were allocated into three groups as follows:**

**Group I**: 20 patients with proven NAFLD who were not excluded with elevated transaminases (ALT, AST).

**Group II**: 20 NAFLD patients who refused to meet the exclusion criteria with normal elevated transaminases (ALT, AST).

**Group III:** Twenty healthy volunteers were used as the control group, consisting of 11 men and 9 women with ages ranging from 30 to 70 years old and who did not meet the exclusion criteria for NAFLD.

The whole blood count (CBC), liver and lipid profiles, PT, PTT, and INR coagulation profiles, kidney profile, and fasting blood glucose, and viral markers (HBsAg, anti-HCV Ab) were all measured for all participants. Additionally, body blood pressure, height, weight, and the body mass index (BMI) were all measured. A sandwich enzyme-linked immunosorbent test that was created in partnership with Sunshine Biotechnology (China), was used to assess L-FABP.

Abdominal ultrasonography was performed on using machine SonoscapeS11, the participants were examined while fasting for 6 hours at least; survey scanning was done through several projections visualizing different organs in deep suspended inspiration, examination of the liver: size, surface, echogenicity, focal lesions, hepatic veins and the portal vein. A prominent pathologist and gastroenterologist reviewed the images **[8].**

**Definition of NAFLD by abdominal ultrasound as follows**

1. **Grade 0**: no steatosis;
2. **Grade 1 steatosis:** A vivid liver and hepatorenal contrast are seen;
3. **Grade 2 steatosis:** altered diaphragm and intrahepatic vascular shapes, or increased echogenicity.
4. **Grade 3 steatosis:** intrahepatic contours and diaphragm removal, or significantly increased echogenity **[9].**

**Definition of elevated transaminases**

The highest normal limits for alanine aminotransferase (ALT) and aspartate aminotransferase (AST), were 0–35 IU/L for each**[7].**

**Ethics approval:**

Both the Institutional Review Board and the local Ethics Committee at Zagazig University's Faculty of Medicine approved this study. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans. Patient consent was obtained from all patients.

***Statistical analysis***

IBM SPSS 23.0 for Windows (SPSS Inc., Chicago, IL, USA) was used to analyse the data. Quantitative data were compared by one-way ANOVA (F) test, and if the difference was significant, then LSD test was used as a post-hoc test so as to compare each group with each other group. Qualitative data were compared by chi-square ($X^2$) test. Pearson's correlation coefficient (r) test and ROC curves were also used. P value less than 0.05 is considered significant.

**RESULTS**

Table 1 shows a high statistically significant difference among all studied groups as regard weight and BMI, while they were matched in all other characteristics.

**Table 1: General characteristics of the overall studied population**

| | | Group I N=20 N (%) | Group II N=20 N (%) | Group III N=20 N (%) | P value |
|---|---|---|---|---|---|
| **Age (years), Mean ± SD** | | **42.7 ± 7.11** | **43.9 ± 8.45** | **41.8 ± 8.85** | 0.695 NS |
| **BMI (kg/m$^2$), Mean ± SD** | | **30.3 ± 3.82** | **29.4 ± 3.61** | **24.9 ± 3.25** | **<0.001 HS** |
| **Weight (kg), Mean ± SD** | | **86.8 ± 8.28** | **86 ± 8.25** | **71.8 ± 7.67** | **<0.001 HS** |
| **Height (m) Mean ± SD** | | **1.7 ± 0.08** | **1.71 ± 0.06** | **1.8 ± 0.07** | **<0.001 HS** |
| **Gender** | **male** | **13 (65%)** | **14 (70%)** | **14 (70%)** | 0.926 NS |
| | **female** | **7 (35%)** | **6 (30%)** | **6 (30%)** | |

**BMI: Boddy mass index, HS: Highly significant, NS: not significant**

Table 2 shows a high statistically significant difference among all studied groups as regard ALP, FBS, and liver enzymes while there was no significant difference regarding other parameters.

**Table 2: Difference in laboratory investigations among all studied groups**

| | Group I N=20 | Group II N=20 | Group III N=20 | P value |
|---|---|---|---|---|
| | Mean ± SD | | | |
| **Hb (g/dl)** | **13.7 ± 1.29** | **13.2 ± 1.08** | **13.8±0.78** | 0.558 NS |
| **WBCs (x10$^3$/cc)** | **5.88 ± 1.35** | **6.01 ± 1.36** | **5.49 ± 0.95** | 0.389 NS |
| **Platelet (x10$^3$/cc)** | **302.7 ± 66.3** | **297.8 ± 64.5** | **294.2±66.4** | 0.921 NS |
| **INR** | **1.21 ± 0.08** | **1.2 ± 0.08** | **1.17 ± 0.07** | 0.391 NS |
| **ALP** | **79.2 ± 14.9** | **77 ± 12.2** | **63.2 ± 9.13** | **<0.001 HS** |
| **FBS** | **116.4 ± 7.21** | **114.5 ± 6.46** | **88.9 ± 7.11** | **<0.001 HS** |
| **Bilirubin (mg/dl)** | **0.73 ± 0.14** | **0.72 ± 0.1** | **0.73 ± 0.15** | 0.981 NS |
| **AST(IU/L)** | **55.6 ± 12.1** | **20.6 ± 3.43** | **16.9 ± 4.2** | **<0.001 HS** |
| **ALT(IU/L)** | **68.2 ± 12.9** | **27.1 ± 4.53** | **20.7 ± 4.28** | **<0.001 HS** |
| **Albumin (g/L)** | **2.5 ± 0.49** | **2.9 ± 0.37** | **4.5 ± 0.43** | **<0.001 HS** |
| **Urea (mg\dl)** | **17.2 ± 3.77** | **16.8 ± 3.11** | **16.6 ± 2.65** | 0.805 NS |
| **Creatinine (mg/dl)** | **0.92 ± 0.22** | **0.82 ± 0.13** | **0.92 ± 0.21** | 0.152 NS |

HB: hemoglobin, WBCs, white blood cells, PLT: platelet, INR: international normalized ratio, ALP: alkaline phosphatase, FBS: fasting blood sugar, ALT: Alanine aminotransferase, AST: aspartate aminotransferase, HS: Highly significant, NS: Not significant

Table 3 shows a high statistically significant difference among all studied groups as regard lipid profile, as all parameters worsened among cases of group I and II than control group.

**Table 3: Difference in lipid profile among all studied groups**

| | Group I N=20 | Group II N=20 | Group III N=20 | P value |
|---|---|---|---|---|
| | Mean ± SD | | | |
| TC (mg/dl) | 232.7 ± 31.4 | 224.8 ± 34.8 | 139.4 ± 12.8 | <0.001 HS |
| TG (mg\dl) | 209.6 ± 29.9 | 178.2 ± 25.9 | 127.95 ± 29.9 | <0.001 HS |
| HDL (x$10^3$/cc) | 37.3 ± 4.71 | 45.9 ± 6.31 | 56.8 ± 4.02 | <0.001 HS |
| LDL | 160.1 ± 13.8 | 163.9 ± 14.3 | 119.6 ± 12.9 | <0.001 HS |

**HS: Highly significant**

Table 4 shows a high statistically significant difference among all studied groups regarding L-FABP level.

**Table 4: Difference in L-FABP among all studied groups**

| | Group I N=20 | Group II N=20 | Group III N=20 | P value |
|---|---|---|---|---|
| | Mean ± SD | | | |
| L-FABP | 251.2 ± 7.1 | 213.3 ± 7.7 | 123.6 ± 16.1 | <0.001 HS |

**HS: Highly significant**

Table 5 shows a high statistically significant increase in L-FABP level among studied cases with steatosis grade III than cases with grade II and I.

**Table 5: Relation between steatosis grades and L-FABP level among studied cases of group I**

| | Grade I N=8 | Grade II N=7 | Grade III N=5 | P value |
|---|---|---|---|---|
| | Mean ± SD | | | |
| L-FABP | 183.3 ± 17.9$^{AB}$ | 252.1 ± 34.5$^{C}$ | 358.4 ± 28.99 | <0.001 HS |

**HS: Highly significant, A: Significant difference between grade I and II, B: Significant difference between grade I and III, C: Significant difference between grade II and III**

Table 6 shows a high statistically significant increase in LF-ABP level among studied cases with steatosis grade III than cases with grade II and I.

**Table 6: Relation between steatosis grades and L-FABP level among studied cases of group II**

| | Grade I N=12 | Grade II N=6 | Grade III N=2 | P value |
|---|---|---|---|---|
| | Mean ± SD | | | |
| L-FABP | 175.7 ± 11.2$^{AB}$ | 245.8 ± 32.1$^{C}$ | 341.5 ± 20.5 | <0.001 HS |

HS: Highly significant, A: Significant difference between grade I and II, B: Significant difference between grade I and III, C: Significant difference between grade II and III

Table 7 show that L-FABP level higher than 179 was effective in prediction of 80% of NFALD with elevated liver enzymes with specificity of 75% to exclude normal cases and test accuracy was 76.7%.

**Table (7): Data for L-FABP performance as a diagnostic marker of NAFLD with elevated enzymes**

| | ***L-FABP*** |
|---|---|
| **Cut-off** | >179 |
| **AUC (95% CI)** | 0.833 (0.734 -0.933) |
| **P** | <0.001 HS |
| **Sensitivity** | 80 % |
| **Specificity** | 75 % |
| **PPV** | 61.5% |
| **PVN** | 88.2% |
| **Accuracy** | 76.7% |

Table 8 show that L-FABP level higher than 174.5 was effective in prediction of 70% of NFALD cases with normal liver enzymes with specificity of 65.5% to exclude normal cases and test accuracy was 66.7%.

***Table (8): Data for L-FABP performance as a diagnostic marker of NAFLD with normal enzymes***

| | ***L-FABP*** |
|---|---|
| **Cut-off** | >174.5 |
| **AUC (95% CI)** | 0.667 (0.533 -0.801) |
| **P** | 0.036 S |
| **Sensitivity** | 70 % |
| **Specificity** | 65.5 % |
| **PPV** | 50% |
| **PVN** | 81.2% |
| **Accuracy** | 66.7% |

Table 9 show that L-FABP level higher than 157.5 was effective in prediction of 97.5% of NFALD cases with specificity of 100% to exclude normal cases and test accuracy was 98.3%.

**Table (9): Data for L-FABP performance as a diagnostic marker of NAFLD**

| | **L-FABP** |
|---|---|
| **Cut-off** | >157.5 |
| **AUC (95% CI)** | 1.0 (1.0 – 1.0) |
| **P** | <0.001 HS |
| **Sensitivity** | 97.5 % |
| **Specificity** | 100 % |
| **PPV** | 100% |
| **PVN** | 95.2% |
| **Accuracy** | 98.3% |

Table 10 shows that ALT, AST had a highly statistically significant positive correlation with L-FABP among cases of both groups I and II.

**Table (10): Correlation between L-FABP and all clinical *parameters of the studied patients***

| | **Group I N=20** | **Group II N=20** |
|---|---|---|
| | **r (P-value)** | |
| **Creatinine (mg\dl)** | 0.015 (0.96 NS) | 0.45 (0.07 NS) |
| **Urea (mg\dl)** | 0.028 (0.92 NS) | 0.39 (0.15 NS) |
| **bilirubin (mg/dl)** | -0.02 (0.94 NS) | -0.171 (0.833 NS) |
| **Albumin (g/L)** | 0.03 (0.9 NS) | 0.69 (0.008 S) |
| **AST(IU/L)** | **0.737 (<0.001 HS)** | **0.811 (<0.001 HS)** |
| **ALT(IU/L)** | **0.763 (<0.001 HS)** | **0.827 (<0.001 HS)** |
| **INR** | -0.261 (0.635 NS) | -0.183 (0.761 NS) |
| **Hb** | 0.46 (0.06 NS) | 0.05 (0.86 NS) |
| **WBCs** | -0.29 (0.29 NS) | -0.41 (0.12 NS) |
| **Platelets** | 0.145 (0.852 NS) | 0.161 (0.722 NS) |
| **TC** | 0.21 (0.46 NS) | 0.195 (0.49 NS) |
| **TG** | **0.619 (0.002 S)** | **0.544 (0.008 S)** |
| **HDL** | -0.03 (0.93 NS) | -0.168 (0.655 NS) |
| **LDL** | 0.205 (0.46 NS) | 0.282 (0.657 NS) |
| **FBS** | -0.119 (0.504 NS) | -0.175 (0.431 NS) |
| **ALP** | 0.137 (0.533 NS) | 0.183 (0.423 NS) |

**HS: High**ly **significant, S: Significant, NS: not significant**

## DISCUSSION

Our findings revealed a statistically significant difference in weight and BMI between all groups examined, as the two parameters were noticeably higher compared to the control group in the groups with NAFLD. While the age, gender, and height of the three groups were matched. The same outcomes were reported by **Abdulaziz *et al.*** [(10)]. There was a notable statistical difference between the NAFLD group's weight and BMI with mean value (I) and control group (II) with relation to BMI (84.5 ± 9.5), (28.76 ± 4.3) in group I and (74.65 ± 7.44), (23.72 ± 3.04) in group II respectively. The average age of those with NAFLD was 37.74 ±11.7 whereas in wholesome control participants was 36.5 ± 11.31. There

was no statistically significant difference between the two groups. Also, **Akbal *et al.*** [7] stated that there was no age or gender difference between NAFLD patients and controls, and that as compared to the controls, the NAFLD group's BMI was greater. There was male predominance in our study in contrast to both **Abdulaziz *et al.*** [10] **and Akbal *et al.*** [7]; in both groups, women outnumbered men, However, there was no difference between the groups that was statistically significant. some previous studies showed that gender is linked to NAFLD since guys are reported to be more prone to the disease, according to study. However, the male group was contrasted with premenopausal women in those trials, who have high levels of estrogen that shield them from NAFLD [11,12].

Regarding BMI our results agreed with the study of **Loomis *et al.*** [13] that used two distinct EHR databases comprising >2.1 million people and>11,000 incident cases, and showed that future risk of "recorded" NAFLD/NASH is significantly and dramatically inversely correlated with BMI, and **Tang *et al.*** [14] findings point to greater, as a separate dose-dependent risk factor for fatty liver, BMI (overweight/obesity) and it is important to take note of this when considering how to prevent fatty liver by paying attention to ongoing changes in BMI.

Regarding laboratory data, ALP, ALT, AST, and FBS, all of the groups in the current study were statistically significantly different from one another, but there was no other significant difference regarding other tests. Group I and II (cases) were close to each other in levels of ALP and FBS although there was no statistically significant difference between the two groups when compared to group III. Moreover, group I cases had statistically significant higher levels of AST and ALT than the other groups. Furthermore, lipid profile worsened among cases of group I and II than control group as group I cases had statistically significant worsened levels of TG and HDL than the group II and III cases, while regarding TC and LDL levels, both cases groups (II and III) were close with no significant difference.

In accordance with **Abdulaziz *et al.*** [10] comparing the study groups revealed a very statistically significant difference in several biochemical and molecular variables, including total cholesterol, TG, HDL, and LDL ($p < 0.001$). Additionally, there were statistically significant differences in AST, ALT, and FBS between the study groups (p 0.04, p 0.03, and P 0.03, respectively). Furthermore, **Akbal *et al.*** [7] showed that fasting glucose levels in NAFLD patients ($p = 0.014$), TG ($p = 0.006$), AST ($p = 0.004$), ALT ($p < 0.001$) and GGT ($p < 0.001$) levels were greater than those of controls.

NAFLD is characterized by elevated low levels of high-density lipoprotein cholesterol (HDL-C), low levels of TG, and low levels of LDL-C. Standard lipid measurements, particularly the non-HDL to HDL cholesterol ratio and non-HDL cholesterol, have been proposed as independent predictors of incident NAFLD [15,16].

One of the largest studies for assessment a NAFLD cohort's lipoprotein profile on 3362 participants from the Multi-Ethnic Study of Atherosclerosis has revealed that those lipoprotein levels are higher in those with NAFLD than in people without it (n=569) [17].

Our findings were adverse with regard to low HDL levels. **Nigam *et al.*** [18] study revealed that there was no discernible change in HDL levels between 152 healthy controls and 120 NAFLD cases mg/dl (41.31±67.2 vs 41.98±66.54 respectively with p value 0.43).

Regarding fasting blood sugar, insulin is primarily responsible for lowering plasma glucose levels through regulating hepatic glucose metabolism. Insulin encourages glycolysis and glycogenesis for glucose uptake via the Akt/PKB signalling pathway. NAFLD is characterised by the coexistence of hepatic and systemic insulin resistance. Hepatic insulin resistance decreases glucose synthesis in the liver and promotes higher levels of glycogenolysis and gluconeogenesis as well as an increase in cholesterol and triglyceride synthesis, so in addition to dyslipidemia in NAFLD cases, there is also dysglycemia. Systemic insulin resistance is characterized by insulin's failure to effectively lower blood glucose levels [19]. These findings in the present study are similar with findings in previous studies such **Tang *et al.*** [14] **and Fan *et al.*** [20].

Regarding ALT and AST signs of liver damage might be helpful substitutes for NAFLD tests. AST is usually found inside the mitochondria, whereas ALT is found in the hepatocellular cytoplasm. In fact, the most frequent causes of persistently elevated liver enzymes are NAFLD and NASH and they frequently serve as the trigger for additional diagnostic testing [21]. In accordance NAFLD, which can be diagnosed by USG, according to a recent big UK study, is the most frequent cause of abnormal liver biochemistry [22].

In the present study NAFLD cases were compared to healthy controls, however all patients showed increased levels of ALP within normal range. The explanation of this difference could be explained by the increased weight and body mass index among NAFLD cases as previously reported that serum ALP was discovered to be a reliable incidence of severe liver fibrosis in patients with obesity-related NAFLD [23].

In the current study there was a high statistically significant difference among all studied groups regarding L-FABP level as it was higher in NAFLD cases than healthy controls. This was in accordance with **Abdulaziz *et al.*** [10]. They found that the NAFLD group (group I) had statistically significant higher serum levels of L-FABP than the control group (group II), which was (188.6 ± 34.94) (137.7 ± 13.05) respectively. Furthermore, **Akbal**

***et al.*** [7] also reported that L-FABP levels were higher in NAFLD patients than in healthy individuals ($p < 0.001$).

L-FABP level was higher in NAFLD with increased enzyme levels compared to NAFLD cases with normal enzyme levels (251.2 ± 75.1; 213.3 ± 57.7 ng/dl in group I; II respectively) indicating that level of L-FABP is associated with liver cell injury. The molecular weight of L-FABP is small, and liver cells contain it. L-FABP's characteristics cause it to rise even in the presence of mild cell damage. Small proteins circulate more quickly in the blood than large proteins because hepatocytes don't have an interstitial barrier, putting them in direct contact with the blood [24].

Another study identified a link between higher levels of serum L-FABP and the severity of fibrosis and inflammation in those with non-alcoholic steatohepatitis, suggesting that serum L-FABP may function as a non-invasive biomarker for measuring fibrosis and inflammation in those with NAS [25]. Also, **Akbal *et al.*** [7], according to their study, higher serum levels of L-FABP were found in those with non-alcoholic fatty liver disease. L-FABP levels were associated with continued liver damage.

Furthermore, in our study there was significant increase in L-FABP level among studied cases in group I, II with steatosis grade III than cases with grade II and I. so there was a positive relation between level of L-FABP and degree of steatosis. Similar finding was reported by **Adulaziz *et al.*** [10] study. Ultrasound-graded fatty liver and serum L-FABP levels were associated, and this correlation was both clinically and statistically significant in NAFLD patients ($p < 0.001$). Additionally, the Cohen kappa test's concordance correlation coefficient between serum L-FABP and ultrasound in the diagnosis of NAFLD patients was 0.70, indicating a good match between ultrasound and L-FABP in the identification of NAFLD patients. This observation can be the result of excessive lipid infiltration and increasing cellular damage.

The current study showed significant positive correlation with ALT, AST and TG among NAFLD cases of both groups I and II, in accordance with **Abdulaziz *et al.*** [10] who additionally stated that there was an association between L-FABP levels and BMI ($r = 0.289$, $p = 0.015$), AST ($r = 0.350$, $p = 0.003$), ALT ($r = 0.291$, $p = 0.015$), total cholesterol ($r = 0.334$, $p = 0.005$), triglycerides ($r = 0.244$, $p = 0.042$), and LDL ($r = 0.301$, $p = 0.011$). Furthermore, these findings matched with **Akbal *et al.*** [7] who discovered a link between elevated L-FABP levels and BMI, diabetes, AST, ALT, and GGT levels. These findings imply that dyslipidemia, fatty infiltration, and liver cell damage may all increase the serum level of L-FABP.

ROC curve for L-FABP performance as a diagnosis in the current study marker of NFALD with elevated enzymes (group I) shows that L-FABP level higher than 179 ng/L was effective in prediction of 80% of NFALD with elevated liver enzymes with specificity 75% to exclude normal cases and test accuracy 76.7% with AUC=0.833 (0.734 -0.933). While for patient with normal enzymes (group II) level higher than 174.5ng/L was effective in prediction of 70% of NFALD cases with specificity 65.5% to exclude normal cases and test accuracy 66.7%. with AUC=0.667 (0.533 -0.801). This indicates that L-FABP has poor to good diagnostic liver enzyme levels and NAFLD yield. The effectiveness of L-FABP as a diagnostic indicator of NAFLD with or without raised liver enzymes suggested that level higher than 157.5 ng/L was effective in prediction of 97.5% of NFALD cases with specificity 100% to exclude normal cases and test accuracy 98.3% AUC=1. This indicates that this cut value L-FABP is an excellent diagnostic of NAFLD.

## CONCLUSION

Because the levels fatty infiltration in the liver is correlated with the amount of hepatic fatty acid binding protein tissue, they are both great diagnostic biomarkers for NAFLD and a strong diagnostic tool for fatty liver damage. In addition, our study showed a relationship between TG levels, AST levels, and L-FABP levels.

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