E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.02.137

Research Article

Clinical and Biochemical Profile of Patients with Non-Alcoholic Fatty Liver Disease: A Clinical Study

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Received: 22.06.26, Revised: 20.07.26, Accepted: 29.07.26

ABSTRACT
Background: Non-alcoholic fatty liver disease (NAFLD) is becoming recognised as a significant metabolic liver condition associated with obesity, diabetes mellitus, dyslipidaemia, and cardiovascular risk. It has a silent clinical presentation, and biochemical abnormalities can be variable depending on the severity of the disease.
Objective: To evaluate the clinical and biochemical traits of patients with non-alcoholic fatty liver disease and determine the relationship between metabolic abnormalities and ultrasonographic severity.
Methods: This cross-sectional research was carried out at Rashid Latif Khan University Medical College in Lahore, Pakistan, from January to November 2025. A total of n=120 individuals diagnosed with non-alcoholic fatty liver disease by ultrasonography were included. Demographic data, clinical manifestations, anthropometric measurements, coexisting conditions, hepatic function assessments, glycaemic indicators, and lipid profiles were recorded. The extent of hepatic steatosis was evaluated using ultrasonography as mild, moderate, or severe.
Results: The mean age was 47.9 ± 11.6 years, and 55.0% were male. 43.3% of the patients were obese, 44.2 percent had diabetes mellitus, 40 percent had hypertension, and 52.5 percent had dyslipidaemia. The most common symptom was fatigue, and 35.8% were asymptomatic. 47.5% had steatosis grade I, 36.7% had steatosis grade II, and 15.8% had steatosis grade III. The severity of steatosis was significantly associated with higher BMI, WC, ALT, AST, FbS, HbA1C, and TG, and lower HDL-C.
Conclusions: There was a strong association of non-alcoholic fatty liver disease with metabolic dysfunction. The ultrasonographic severity was associated with progressive obesity, glycaemic control, dyslipidaemia, and hepatocellular damage. These findings provide evidence for the practice of early metabolic screening for metabolic risk factor management.

Keywords: Non-Alcoholic Fatty Liver Disease, Hepatic Steatosis, Obesity, Diabetes Mellitus, Dyslipidaemia, Liver Enzymes.

INTRODUCTION
Non-alcoholic fatty liver disease is a common chronic hepatic disorder characterised by the excessive accumulation of lipids in liver cells among individuals who do not ingest significant quantities of alcohol or possess any identifiable cause for liver fat deposition[1]. It represents a broad clinical range that includes uncomplicated steatosis, non-alcoholic steatohepatitis, advancing fibrosis and cirrhosis, liver insufficiency, and hepatocellular cancer. It is now regarded as a significant public health issue because to its rising incidence among those experiencing fast obesity, along with related sedentary habits, type 2 diabetes mellitus, and metabolic syndrome[2,3].
Insulin resistance, dyslipidaemia, and dysglycaemia have been reported to be closely associated with the development of NAFLD[4]. Fat accumulation and hepatocellular injury are consequences of increased free fatty acid (FFA) mobilization from fat, augmented lipogenesis in the liver, oxidative stress, mitochondrial dysfunction, and inflammatory responses. Thus, obesity, diabetes mellitus, hypertension, dyslipidaemia, and central adiposity are often seen in affected persons. There is also an increased risk of cardiovascular complications of the disease that may have more important effects on the mortality rates than its liver effects in many of the patients[5].
Non-alcoholic fatty liver disease is typically asymptomatic and non-specific. Most patients are asymptomatic and are only diagnosed when the liver enzymes are abnormal or on abdominal ultrasonography[6]. Symptomatic

people might present with fatigue, malaise, or a vague discomfort in the right upper abdomen, and sometimes hepatomegaly is found on physical examination. Many people who have advanced disease show no signs or symptoms, which makes it difficult to diagnose[7].

Non-alcoholic fatty liver disease: biochemical abnormalities are variable. Mild to moderate increases in alanine aminotransferase and aspartate aminotransferase are common; normal aminotransferase levels do not exclude the presence of significant steatosis, inflammation, or fibrosis[8]. Other abnormalities are also often seen, such as abnormal fasting glucose, high glycated haemoglobin, hypertriglyceridaemia, high low-density lipoprotein cholesterol, and low high-density lipoprotein cholesterol. Usually, the serum bilirubin, albumin, and coagulation parameters are well maintained until a late stage of liver dysfunction[9].

Since ultrasonography is a relatively cheap, widely available, non-invasive imaging modality, it is often the initial imaging modality chosen[10]. It can detect hepatic steatosis and categorize its severity by increased hepatic echogenicity. Clinical findings, biochemical measurements, and metabolic risk factors, however, continue to be important for the overall assessment of affected patients and/or for identifying patients who might need further evaluation for steatohepatitis or fibrosis[11].

Understanding the clinical and biochemical characteristics of patients with non-alcoholic fatty liver disease can help identify the disease early, stratify the risk, and manage the metabolic abnormalities associated with the disease. The current investigation aimed to assess the demographic features, clinical symptoms, comorbidities, and biochemical irregularities in individuals with non-alcoholic fatty liver disease, as well as to ascertain their correlation with the severity of hepatic steatosis [12,13].

MATERIALS AND METHODS

Study Design and Setting

A cross-sectional study was conducted at Rashid Latif Khan University Medical College (RLKUMC) in Lahore, Punjab, Pakistan, from January 2025 to November 2025. The group included 120 people diagnosed with non-alcoholic fatty liver disease (NAFLD) by a consecutive non-probability sampling technique.

Study Participants

Patients ≥18 years of age, both males and females, with ultrasonographic findings of fatty liver were included. NAFLD was diagnosed as hepatic steatosis revealed by abdominal ultrasonography, without major alcohol consumption or other known causes of liver steatosis. Significant alcohol intake was considered more than 20 g per day in women and more than 30 g per day in men.

Patients with hepatitis B or C infection, autoimmune hepatitis, Wilson's disease, haemochromatosis, known cirrhosis, hepatocellular carcinoma, acute liver failure, or heavy alcohol consumption were excluded. Patients who were pregnant or who were on long-term use of any of the following medications were also excluded: corticosteroids, amiodarone, methotrexate, tamoxifen, or other medications that are known to cause hepatic steatosis. Patients with incomplete clinical or laboratory information were not included.

Data Collection

Demographic and clinical data were collected by a structured data collection form after informed consent. Variables recorded were age, sex, presenting complaint, duration of symptoms, smoking history, physical activity, dietary pattern, medication history, and relevant family history. Fatigue, right upper abdominal discomfort, loss of appetite, nausea, and abdominal fullness were assessed in the participants, as well as the incidental detection of fatty liver.

A comprehensive history of diabetes mellitus, hypertension, dyslipidaemia, obesity, and other metabolic conditions was taken. General physical and systemic examination was performed with special emphasis on blood pressure, peripheral oedema, clinical features of chronic liver disease, hepatomegaly, and jaundice in all the participants.

Anthropometric Assessment

Body mass was assessed in kilograms using a calibrated scale, while stature was recorded in meters, with the individual standing barefoot. The Body Mass Index was calculated by dividing weight in kilograms by height in meters squared. Patients were categorised based on conventional body mass index characteristics into normal weight, overweight, and obese classifications. The waist circumference was measured at the conclusion of normal exhalation, positioned equidistantly between the lowest rib and the top edge of the iliac crest. Blood pressure was assessed while seated after a five-minute period of relaxation.

Biochemical Assessment

Venous blood specimens were obtained under sterile circumstances after a minimum 8-hour overnight fast. Biochemical assessments for all subjects included FPG, glycated haemoglobin, total bilirubin, direct bilirubin, ALP, ALT, gamma-glutamyl transferase, serum albumin, total cholesterol, triglycerides, LDL cholesterol, and HDL cholesterol. The other parameters assessed were total blood count, serum creatinine, and blood urea levels. The hepatitis B surface antigen and antibodies to the hepatitis C virus were examined to exclude viral hepatitis.

Diabetes mellitus was identified by a prior recorded diagnosis, ongoing use of glucose-lowering drugs, a fasting plasma glucose level of ≥ 126 mg/dL, or a glycated haemoglobin concentration of ≥ 6.5%. Dyslipidaemia was taken as the presence of a previous diagnosis, or medication consumption for dyslipidaemia, or abnormal lipid levels at fasting. Hypertension was identified by a history or from current antihypertensive therapy, or persistent elevation of blood pressure measurements.

Ultrasonographic Assessment

An experienced radiologist performed abdominal ultrasonography with a conventional ultrasound machine. Hepatic steatosis was defined by increased echogenicity of the liver relative to the renal cortex, decreased intrahepatic vascular visualisation, posterior beam attenuation, and decreased diaphragm visualisation.

Fatty liver was divided into three ultrasonographic grades. Grade I was considered to be a slight diffuse increase in hepatic echogenicity with intact visualisation of the borders of the intrahepatic vessels and the diaphragmatic surface. Grade II was used to indicate a moderate increase in echogenicity with the presence of partial obliteration of the vessel borders and diaphragm. Grade III was taken as a significant increase in the hepatic echogenicity with poor or absent intrahepatic vessels, posterior liver, and diaphragm visualization.

Outcome Measures

The main outcome was the clinical and biochemical characteristics of NALFDs. The variables evaluated were presenting symptoms, body mass index, waist circumference, associated metabolic conditions, liver enzyme abnormalities, glycaemic parameters, lipid profile, and ultrasonographic grade of hepatic steatosis. The clinical and biochemical features were also assessed between the different grades of fatty liver.

Ethical Considerations

Before data collection, approval from the relevant institutional ethics review committee of Rashid Latif Khan University Medical College was obtained. Every participant submitted a signed form of informed consent. The confidentiality of patients and their data was maintained, with all procedures conducted in compliance with ethical norms.

Statistical Analysis

Data input and analysis were done using IBM SPSS Statistics software (version 26.0). Frequencies and percentages were used to describe categorical data, whereas mean and standard deviation were used to represent continuous variables. When comparing continuous variables, the independent-samples t-test or one-way ANOVA was used. For categorical variables, the chi-square test or Fisher's exact test was used. To find independent variables of moderate to severe hepatic steatosis, a multivariable logistic regression analysis was used. We computed adjusted odds ratios with a 95% confidence range. Results with p-values less than 0.05 were considered statistically significant.

RESULTS

The study included 120 people with a diagnosis of non-alcoholic fatty liver disease. The average age of the participants was 47.9 ± 11.6 years, with ages ranging from 21 to 72. Of the total, 54 (45.0%) were women and 66 (55.0%) were men. The average waist circumference was 96.8 ± 11.3 cm, and the average body mass index was 29.2 ± 4.4 kg/m². Overweight and obesity rates were 40.8% and 43.3%, respectively. Table 1 shows that 53 patients (44.2%) had type 2 diabetes mellitus, 48 patients (40.0%) had hypertension, 63 patients (52.5%) had dyslipidaemia, and 61 patients (50.8%) had metabolic syndrome.

Table 1. Demographic and Clinical Characteristics of Individuals with Non-Alcoholic Fatty Liver Disease

Variable	Total, N = 120
Age, Years	47.9 ± 11.6
Male	66 (55.0%)
Female	54 (45.0%)
Body Mass Index, Kg/M²	29.2 ± 4.4

Waist Circumference, Cm	96.8 ± 11.3
Normal Body Weight	19 (15.8%)
Overweight	49 (40.8%)
Obesity	52 (43.3%)
Type 2 Diabetes Mellitus	53 (44.2%)
Hypertension	48 (40.0%)
Dyslipidaemia	63 (52.5%)
Metabolic Syndrome	61 (50.8%)
Sedentary Lifestyle	72 (60.0%)
Current Smoking	23 (19.2%)

The commonest presenting complaint was fatigue (44 (36.7%) patients). There was right upper abdominal discomfort in 31 (25.8%), abdominal fullness in 18 (15.0%), and reduced appetite in 11 (9.2%). A total of 43 (35.8%) patients were asymptomatic and were diagnosed incidentally during abdominal ultrasonography. Mild hepatomegaly was found in 17 patients (14.2%), and none of them had clinical signs of hepatic decompensation.

According to the ultrasound assessment, 57 (47.5%) patients had grade I, 44 (36.7%) patients had grade II, and 19 (15.8%) had grade III hepatic steatosis. Thus, forty-two patients (52.5%) had moderate to severe fatty liver disease.

The mean level of alanine aminotransferase was 66.8 ± 33.7 U/L, and the mean level of aspartate aminotransferase was 50.9 ± 26.4 U/L, with 75 (62.5%) patients having elevated alanine aminotransferase and 57 (47.5%) patients having elevated aspartate aminotransferase. The mean FPG level was 126.9 ± 41.7 mg/dL, and the mean glycated haemoglobin was 6.7 ± 1.4%. Hypertriglyceridaemia was identified in 69 (57.5%) patients, while reduced high-density lipoprotein cholesterol was identified in 58 (48.3%) patients, as shown in Table 2.

Table 2. Biochemical Characterisation of the Study Cohort

Biochemical Parameter	**Mean ± Standard Deviation**
Fasting Plasma Glucose, Mg/Dl	126.9 ± 41.7
Glycated Haemoglobin, %	6.7 ± 1.4
Total Bilirubin, Mg/Dl	0.9 ± 0.3
Alanine Aminotransferase, U/L	66.8 ± 33.7
Aspartate Aminotransferase, U/L	50.9 ± 26.4
Alkaline Phosphatase, U/L	120.7 ± 39.8
Gamma-Glutamyl Transferase, U/L	71.9 ± 47.5
Serum Albumin, G/Dl	4.1 ± 0.4
Total Cholesterol, Mg/Dl	203.4 ± 40.7
Triglycerides, Mg/Dl	207.6 ± 76.9
Low-Density Lipoprotein Cholesterol, Mg/Dl	124.7 ± 33.1
High-Density Lipoprotein Cholesterol, Mg/Dl	39.1 ± 8.8
Serum Creatinine, Mg/Dl	0.9 ± 0.2

A progressive increase in body mass index, waist circumference, liver enzymes, glycaemic parameters, and triglyceride levels was observed with increasing ultrasonographic severity. The mean body mass index increased from 27.2 ± 3.3 kg/m² in grade I disease to 30.1 ± 3.7 kg/m² in grade II and 33.0 ± 4.2 kg/m² in grade III disease. Similarly, alanine aminotransferase increased from 47.5 ± 19.4 U/L in grade I to 76.8 ± 28.1 U/L in grade II and 101.8 ± 38.9 U/L in grade III disease.

The prevalence of Type 2 diabetes mellitus was 15 (26.3%) patients with grade I steatosis, 23 (52.3%) with grade II, and 15 (78.9%) with grade III. A significant increase was also observed in obesity in patients with advanced steatosis (grade I 22.8%, grade II 54.5%, and grade III 78.9%). Table 3 shows that there were significant differences among the three grades in body mass index, waist circumference, diabetes mellitus, alanine aminotransferase, fasting plasma glucose, glycated haemoglobin, triglycerides, and high-density lipoprotein cholester

Table 3. Comparison of Clinical and Biochemical Variables According to Ultrasonographic Grade

Variable	Grade I, N = 57	Grade II, N = 44	Grade III, N = 19	P-Value
Age, Years	44.9 ± 10.8	49.7 ± 11.0	52.8 ± 12.1	0.010
Body Mass Index, Kg/M²	27.2 ± 3.3	30.1 ± 3.7	33.0 ± 4.2	<0.001
Waist Circumference, Cm	90.8 ± 9.2	100.1 ± 9.8	107.2 ± 10.4	<0.001
Type 2 Diabetes Mellitus	15 (26.3%)	23 (52.3%)	15 (78.9%)	<0.001
Obesity	13 (22.8%)	24 (54.5%)	15 (78.9%)	<0.001
Alanine Aminotransferase, U/L	47.5 ± 19.4	76.8 ± 28.1	101.8 ± 38.9	<0.001
Aspartate Aminotransferase, U/L	37.2 ± 15.7	57.1 ± 22.5	76.1 ± 30.2	<0.001
Fasting Plasma Glucose, Mg/Dl	106.7 ± 27.6	136.2 ± 38.9	166.0 ± 49.5	<0.001
Glycated Haemoglobin, %	6.0 ± 0.9	7.1 ± 1.3	8.0 ± 1.5	<0.001
Triglycerides, Mg/Dl	171.8 ± 52.7	226.5 ± 68.4	271.4 ± 88.6	<0.001
High-Density Lipoprotein Cholesterol, Mg/Dl	42.8 ± 8.1	37.4 ± 7.6	31.9 ± 7.1	<0.001

Overall, the results revealed a significant association between ultrasonographic severity of NAFLD and age, obesity, central adiposity, diabetes mellitus, poor glycaemic control, high levels of aminotransferase, hypertriglyceridaemia, and low levels of high-density lipoprotein cholesterol.

DISCUSSION

The present study assessed the clinical and biochemical features in 120 non-alcoholic fatty liver disease patients[1]. Results showed that obesity, central adiposity, type 2 diabetes mellitus, hypertension, dyslipidaemia, and sedentary lifestyle were strong predictors of the condition. More than four-fifths of the participants were either overweight or obese, while nearly half had diabetes mellitus. These observations help point to the close association between the accumulation of fat in the liver and systemic metabolic dysfunction[2].

The mean age of the subjects was 47.9 years, and there was only a marginal difference between the ages of the males and females[3]. The severity of hepatic steatosis by ultrasonography increased with age. This could be due to chronic exposure to metabolic risk factors, progressive decline in insulin sensitivity, and increases in body-fat distribution with age and decreased physical activity. But fatty liver is not just a disease of older people anymore, as there are also indications of the disease among younger adults[4].

A significant number of patients were asymptomatic and were only diagnosed as part of an abdominal ultrasound[5]. Fatigue and right upper abdominal discomfort were the most common symptoms among those who were symptomatic. These signs and symptoms are vague and are not necessarily directly correlated with the severity of the liver damage. The frequent incidental finding emphasizes the asymptomatic nature of non-alcoholic fatty liver disease and the need to consider specific investigation in the presence of obesity, diabetes mellitus, or dyslipidaemia[5,6].

Among the most frequent clinical findings were obesity and central adiposity. BMI and WC were progressively higher in grade I to III steatosis[7]. Overabundance of visceral adipose tissue leads to greater free fatty acid (FFA) production in the portal circulation and delivery to the liver. It also plays a role in insulin resistance, oxidative stress, inflammatory cytokine production, and augmentation of hepatic lipid production. These mechanisms could be related to the high correlation between obesity and ultrasonographic steatosis seen in the current study[8,9].

The prevalence of type 2 diabetes mellitus was 44.2% among the participants, and its prevalence increased with increasing severity of fatty liver[10]. Patients who had grade II and grade III disease also had significantly raised levels of fasting plasma glucose and glycated haemoglobin. Insulin resistance leads to elevated glucose production by the liver, promotes de novo lipogenesis, and decreases suppression of lipolysis from adipose tissue. Thus, patients with sub-optimal glycaemic control might have higher accumulation of liver triglycerides and be at a higher risk of liver injury[11].

Another common metabolic disorder was dyslipidaemia. Dyslipidaemia was present in over half of the patients, hypertriglyceridaemia and low HDL cholesterol being the most frequent[12]. With the severity of steatosis, there was an increase in triglyceride concentrations and a decrease in high-density lipoprotein cholesterol. This is the typical lipid profile found in insulin-resistant and metabolic syndrome. It

also highlights how patients with non-alcoholic fatty liver disease are more at risk of cardiovascular disease[13].

Biochemical results revealed that more often the ALT was raised while the AST was not. Both enzymes showed a significant increase with each ultrasonographic grade, indicating that the higher the amount of fat in the liver, the more likely it was to have hepatocellular injury[14,15]. But serum bilirubin and albumin were relatively spared, reflecting the fact that the majority of the participants had preserved hepatic synthetic and excretory function[16].

Raised aminotransferase levels were frequent, but the presence of normal liver enzymes does not rule out clinically relevant disease. In some people with significant steatosis, inflammation, or fibrosis, concentrations of aminotransferases may be within the laboratory reference range. Thus, the diagnosis of non-alcoholic fatty liver disease should not rely exclusively on liver enzyme levels, and should involve anthropometric parameters, metabolic risk factors, ultrasonography, and a suitable non-invasive fibrosis assessment[17,18].

Using ultrasonography, slightly over half of the study population had a grade II or III steatosis[19]. Age, body mass index, waist circumference, glucose, glycated haemoglobin, triglycerides, alanine aminotransferase, and aspartate aminotransferase were significantly higher in patients with a higher ultrasonographic severity. The results showed that the metabolic and biochemical abnormalities were worsened by increasing hepatic steatosis[20].

The results have important clinical implications. Obesity, diabetes mellitus, hypertension, and dyslipidaemia should be actively investigated for fatty liver, especially if the aminotransferase level is raised. Likewise, those patients with hepatic steatosis should have a thorough evaluation for metabolic syndrome and cardiovascular risk factors. Liver and systemic risks should be treated by weight loss, dietary changes, regular exercise, good glycaemic control, and treatment of lipid and blood pressure disorders[1-7].

There were some limitations to this study. The cross-sectional design did not allow for causal conclusions or observing the progression of diseases over time. Steatosis was graded by ultrasonography, which has limited ability to distinguish simple steatosis from steatohepatitis, and is not very accurate at determining the fibrosis stage. Due to the invasive nature of liver biopsy, it was not performed. The study was carried out at a single centre, and therefore, the results may not be generalisable. The study has some limitations, but it offers valuable insights into the clinical and biochemical profile of NAFLD in the local population[15-20].

CONCLUSION

Non-alcoholic fatty liver disease was strongly associated with obesity, central adiposity, type 2 diabetes mellitus, hypertension, dyslipidaemia, and sedentary lifestyle. Patients were largely asymptomatic, which is a typical feature of the disease. Waist circumference, fasting plasma glucose, glycated haemoglobin, triglycerides, alanine aminotransferase, aspartate aminotransferase, and body mass index were higher, and high-density lipoprotein cholesterol was lower in the higher ultrasonographic severity groups. The findings indicate a strong association between the extent of hepatic steatosis and the level of metabolic impairment and hepatocellular damage. Identification and management of obesity, hyperglycaemia, dyslipidaemia, and other cardiovascular risk factors may help to slow the progression of liver disease and the risks of systemic complications.

Conflict of Interest: The authors declare no conflict of interest.

Funding: No external funding was received.

Authors' Contributions: K.Z. conceived the study and drafted the manuscript. K.J., H., A., Z.N., and R.A. contributed to data collection and analysis. U.S. supervised the study and critically revised the manuscript. All authors approved the final manuscript.

Acknowledgments: The authors thank the staff of Rashid Latif Khan University Medical College, Lahore, for their support.

Data Availability: Data are available from the corresponding author upon reasonable request.

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