Evaluation of Fatty Liver and its Correlation with Liver Enzymes, BMI, and HbA1c Using Ultrasonography

Prashanth G Patil[1]

[1]Assistant Professor, Department of Radiology, Assistant Professor, Department of Radiology, M S Ramaiah Medical College, Bangalore, India.

Abstract

Background: The present study is a comprehensive evaluation of the role of non-invasive ultrasound imaging in diagnosing and assessing the severity of fatty liver disease, a condition that is increasingly recognized as a significant public health issue due to its association with metabolic disorders like obesity, diabetes, and cardiovascular diseases. This prospective cross-sectional study aimed to evaluate the prevalence of diabetes in patients with fatty liver, to determine the correlation between ultrasound grading of fatty liver and liver function tests and to explore the relationship between ultrasound grading of fatty liver and Body Mass Index (BMI). **Methodology:** This study focused on the utility of ultrasound, a non-invasive, cost-effective, and widely available diagnostic tool, in detecting fatty liver and correlating its severity with biochemical markers including liver enzymes ALT, AST, GGT, BMI, and HbA1c levels. The study involved 56 participants who were selected using a simple random sampling method. The inclusion criteria required participants to be aged 18 years or older, to have been referred for abdominal ultrasonography, and to have undergone liver function tests, fasting and random blood sugar tests, and HbA1c measurements. **Results:** The study's findings revealed important insights into the prevalence and severity of fatty liver disease in the study population. The mean age of participants was 55.31 years, with a mean BMI of 26.9 kg/m², indicating that most participants were overweight or obese. Liver enzyme levels varied widely, with ALT levels ranging from 10 to 500 U/L, AST from 10 to 560 U/L, and GGT from 13 to 1760 U/L. The mean HbA1c level was 7.7%, reflecting a high prevalence of diabetes among the participants. **Conclusion:** In conclusion, this study demonstrates the utility of ultrasonography as a non-invasive and effective tool for detecting and grading fatty liver disease. The significant correlations observed between fatty liver severity, liver enzyme levels, and BMI provide valuable insights into the pathophysiology of the disease and its association with metabolic dysfunction. Although no significant correlation was found between fatty liver grades and blood glucose levels, the high prevalence of diabetes among participants highlights the complex interplay between these metabolic conditions.

Keywords: BMI, Fatty Liver, HbA1c, ALT, GGT.

INTRODUCTION

Fatty liver disease, characterized by excessive fat accumulation in the liver, is commonly associated with both alcoholic and non-alcoholic etiologies. The disease can progress from simple steatosis to more severe conditions such as cirrhosis or liver failure, which shows the importance of early detection and management. Traditional diagnostic methods, such as liver biopsy, are considered the gold standard for diagnosing fatty liver. However, these methods are invasive, often painful, and not suitable for routine monitoring due to the risk of complications. Therefore, this study focused on the utility of ultrasound, a non-invasive, cost-effective, and widely available diagnostic tool, in detecting fatty liver and correlating its severity with biochemical markers including liver enzymes (ALT, AST, GGT), BMI, and HbA1c levels.

Address for correspondence*

Dr. Prashanth G Patil
Assistant Professor, Department of Radiology,
M S Ramaiah Medical College, Bangalore, India.

Non-alcoholic fatty liver disease (NAFLD) refers to a condition wherein excess fat accumulates in the liver of people with no history of significant alcohol consumption. Fat molecules are deposited in the form of triacylglycerols (TAGs) in hepatocytes. Hepatic steatosis refers to fatty change in hepatocytes and is largely a benign condition, while, not in a small number of patients, it may trigger an immune response and lead to non-alcoholic steatohepatitis (NASH) followed by cirrhosis and cancer. NAFLD is alarmingly increasing around the globe. The estimated global prevalence of NAFLD ranges from 6.3-33% among the general population, varying among and within populations. The prevalence is highest among obese (57%) and diabetic (90%) populations. A rising trend of prevalence of NAFLD has been observed in line with obesity, at a rate of 25%. Sedentary lifestyle, dyslipidemia and metabolic syndrome are also well documented risk factors for NAFLD, along with other risk factors, such as hepatitis B and C virus infection, Wilson's disease, and chronic blood and kidney diseases.

High blood glucose levels non-enzymatically form glycosylated hemoglobin (HbA1C) as an irreversible reaction. Once formed, HbA1C remains in circulation for 2-3 months; hence, it has been identified as the marker for diabetes diagnosis and control. According to the American Association of Clinical Endocrinologists, an acceptable level of HbA1C in diabetics is <6.5% and reflects good metabolic control, although, tight control is recommended to avoid increased risk of hypoglycemia, but the level of <6.5% is considered as acceptable in this study. Obesity and diabetes have been reported as strong predictors of NAFLD.[1,2,3,4] This study aimed to evaluate the prevalence of diabetes in patients with fatty liver, to determine the correlation between ultrasound grading of fatty liver and liver function tests and to explore the relationship between ultrasound grading of fatty liver and Body Mass Index (BMI).

METHODS

This study focused on the utility of ultrasound, a non-invasive, cost-effective, and widely available diagnostic tool, in detecting fatty liver and correlating its severity with biochemical markers including liver enzymes (ALT, AST, GGT), BMI, and HbA1c levels. The study involved 56 participants who were selected using a simple random sampling method. The inclusion criteria required participants to be aged 18 years or older, to have been referred for abdominal ultrasonography, and to have undergone liver function tests, fasting and random blood sugar tests, and HbA1c measurements. Exclusion criteria included the presence of chronic liver diseases such as Hepatitis B or C, hepatocellular carcinoma, or Wilson's disease, as well as a history of hepatotoxic drug use or refusal to provide informed consent.

Data collection involved recording patients' demographic information, anthropometric measurements (age, height, weight), and biochemical measurements (fasting blood sugar, random blood sugar, HbA1c, ALT, AST, GGT). Ultrasonography was performed using a SAMSUNG UGEO H60 with a 3-5 MHz convex probe. The grading of fatty liver was based on the echogenicity of liver parenchyma relative to the kidney cortex, along with other sonographic features such as increased liver contrast, vascular blurring, and attenuation of echogenic levels in deeper liver areas. The study employed statistical analysis using IBM SPSS version 25, with descriptive statistics such as frequencies, percentages, means, and standard deviations used to present the data. The Student’s t-test was applied to compare mean differences between groups, with a p-value of <0.05 considered statistically significant.

RESULTS

The study's findings revealed important insights into the prevalence and severity of fatty liver disease in the study population. The mean age of participants was 55.31 years, with a mean BMI of 26.9 kg/m², indicating that most participants were overweight or obese. Liver enzyme levels varied widely, with ALT levels ranging from 10 to 500 U/L, AST from 10 to 560 U/L, and GGT from 13 to 1760 U/L. The mean HbA1c level was 7.7%, reflecting a high prevalence of diabetes among the participants.

The analysis of fatty liver grading revealed significant differences based on alcohol consumption, gender, presence of hepatomegaly, and echogenicity observed in ultrasound imaging. Among the participants, non-alcoholics were more likely to have Grade 1 fatty liver (93.3%) compared to alcoholics (63.6%), with higher grades more commonly observed among alcoholics. Gender analysis showed that females had a higher prevalence of Grade 1 fatty liver (93.1%) compared to males (81.5%), though higher grades were more frequently seen in males. The presence of hepatomegaly was associated with higher grades of fatty liver, although this association did not reach statistical significance. Importantly, increased echogenicity on ultrasound was strongly correlated with higher grades of fatty liver, highlighting the effectiveness of ultrasound as a diagnostic tool.

The study also explored the relationship between fatty liver grading and liver function tests, specifically ALT, AST, and GGT levels. The results indicated a significant increase in AST levels with higher grades of fatty liver (p=0.006), suggesting that AST may be a particularly useful marker for assessing liver inflammation and disease progression. While ALT and GGT levels also increased with the severity of fatty liver, these changes did not reach statistical significance in this study (Table 1-15). In addition to liver function tests, the study examined blood glucose levels, including HbA1c, fasting blood sugar, and random blood sugar, in relation to fatty liver grades. Interestingly, no significant correlation was found between these glucose parameters and the severity of fatty liver as graded by ultrasound. This finding suggests that while diabetes is prevalent in patients with fatty liver, the severity of fatty liver may not directly correlate with blood glucose levels, indicating the need for further research to clarify this relationship. Finally, the study found a significant association between BMI and fatty liver grading, with higher grades more common in overweight and obese individuals. This finding supports the well-established link between obesity and fatty liver disease and highlights the importance of weight management in preventing and treating this condition.

Table 1: Descriptive Statistics of Study Participants

Parameter	N	Minimum	Maximum	Mean	Std. Deviation
Age (years)	55	25	85	55.31	13.312
ALT	56	10.0	500.0	48.805	76.456
AST	56	10	560	48.71	78.892
GGT	56	13	1760	164.16	294.927
HbA1c	56	4.90	16.40	7.7362	2.58532
Fasting Blood Sugar	56	73	557	168.98	85.846
Random Blood Sugar	56	103	600	233.29	101.815
Height (cm)	56	130	173	158.79	9.745
Weight (kg)	56	30	98	69.00	13.388

Explanation: This table provides an overview of the baseline characteristics of the study participants, including their age, liver enzyme levels (ALT, AST, GGT), HbA1c, blood sugar levels, and anthropometric measurements (height and weight). The data are presented as the minimum, maximum, mean, and standard deviation values for each parameter.

Table 2: Distribution of Fatty Liver Grading by Alcohol Consumption

Fatty Liver Grading	Alcoholic	Non-alcoholic	Total	P value
Grade 1	7 (63.6%)	42 (93.3%)	49	0.00
Grade 2	3 (27.3%)	3 (6.7%)	6	
Grade 3	1 (9.1%)	0 (0.0%)	1	
Total	11 (100%)	45 (100%)	56	

Explanation: This table shows the distribution of fatty liver grades among alcoholic and non-alcoholic participants. The P value indicates the statistical significance of the difference in fatty liver grading between the two groups.

Table 3: Distribution of Fatty Liver Grading by Gender

Fatty Liver Grading	Female	Male	Total	P value
Grade 1	27 (93.1%)	22 (81.5%)	49	0.00
Grade 2	2 (6.9%)	4 (14.8%)	6	
Grade 3	0 (0.0%)	1 (3.7%)	1	
Total	29 (100%)	27 (100%)	56	

Explanation: This table presents the distribution of fatty liver grades by gender. The P value indicates the statistical significance of the difference in fatty liver grading between female and male participants.

Table 4: Distribution of Fatty Liver Grading by Hepatomegaly

Fatty Liver Grading	No	Yes	Total	P value
Grade 1	38 (88.4%)	11 (84.6%)	49	0.00
Grade 2	5 (11.6%)	1 (7.7%)	6	
Grade 3	0 (0.0%)	1 (7.7%)	1	
Total	43 (100%)	13 (100%)	56	

Explanation: This table shows the distribution of fatty liver grades in participants with and without hepatomegaly. The P value indicates the statistical significance of the difference in fatty liver grading based on the presence of hepatomegaly.

Table 5: Distribution of Fatty Liver Grading by Echogenicity

Fatty Liver Grading	Increased	Total	P value
Grade 1	49 (87.5%)	49 (87.5%)	0.00
Grade 2	6 (10.7%)	6 (10.7%)	
Grade 3	1 (1.8%)	1 (1.8%)	
Total	56 (100%)	56 (100%)	

Explanation: This table presents the distribution of fatty liver grades based on increased echogenicity observed in imaging studies. The P value indicates the statistical significance of the relationship between fatty liver grading and echogenicity.

Table 6: Liver Function Tests by Fatty Liver Grading

Parameter	N	Minimum	Maximum	Mean	Std. Deviation	P value
ALT	56	10.0	500.0	48.805	76.4560	0.34
AST	56	10	560	48.71	78.892	0.006
GGT	56	13	1760	164.16	294.927	0.083

Explanation: This table provides liver function test results (ALT, AST, GGT) for the study participants, stratified by fatty liver grading. The P values indicate the statistical significance of the differences in liver enzyme levels across different fatty liver grades.

Table 7: Blood Glucose Levels by Fatty Liver Grading

Parameter	N	Minimum	Maximum	Mean	Std. Deviation	P value
HbA1c	56	4.90	16.40	7.7362	2.58532	0.941
Fasting Blood Sugar	56	73	557	168.98	85.846	0.954
Random Blood Sugar	56	103	600	33.29	101.815	.115

Explanation: This table presents the blood glucose levels (HbA1c, fasting blood sugar, and random blood sugar) for the study participants, categorized by fatty liver grading. The P values indicate the statistical significance of the differences in blood glucose levels across different fatty liver grades.

Table 8: Distribution of Fatty Liver Grading by Body Mass Index (BMI)

Fatty Liver Grading	Normal	Overweight	Obese I	Obese II	Obese III	Severe Thinness	Total	P value
Grade 1	19 (95.0 %)	16 (84.2%)	8 (83.3 %)	5(80.0%)	1 (100 %)	0 (0.0%)	49	0.00
Grade 2	1 (5.0 %)	2 (10.5%)	1 (16.7 %)	1 (20.0 %)	0 (0.0 %)	1 (100 %)	6	
Grade 3	0 (0.0 %)	1 (5.3%)	0 (0.0 %)	0 (0.0%)	0 (0.0 %)	0 (0.0%)	1	
Total	20 (100 %)	19 (100%)	9(100%)	6 (100 %)	1 (100 %)	1 (100 %)	56	

Explanation: This table shows the distribution of fatty liver grades by BMI categories. The P value indicates the statistical significance of the differences in fatty liver grading across different BMI categories.

Table 9: Distribution of liver status in participants

Alcohol Consumption	Normal	Hepatomegaly	Total	P value
Alcoholic	7 (63.6%)	4 (36.4%)	11	0.245
Non-alcoholic	36 (80%)	9 (20.0%)	45	
Total	43 (76.8 %)	13 (23.2 %)	56	

Explanation: This table presents the distribution of hepatomegaly in alcoholic and non-alcoholic participants. The P value indicates the statistical significance of the difference in hepatomegaly prevalence between the two groups.

Table 10: Gender Distribution among participants

Gender	Alcoholic	Non-alcoholic	Total	P value
Female	2 (18.2%)	27 (60.0%)	29	0.011
Male	9 (81.8%)	18 (40.0%)	27	
Total	11 (100%)	45 (100%)	56	

Explanation: This table shows the gender distribution of alcoholic and non-alcoholic participants. The P value indicates the statistical significance of the difference in gender distribution between the two groups.

Table 11: Distribution of echogenicityamong participants

Echogenicity	Alcoholic	Non-alcoholic	Total	P value
Increased	11(19.6%)	45 (80.3.0%)	56	0.00
Total	11(19.6%)	45 (80.3%)	56	

Explanation: This table presents Echogenicity for participants, categorized by alcohol consumption. The P values indicate the statistical significance of the differences in echogenicity between alcoholic and non-alcoholic participants.

Table 12: Liver Function Tests among participants

Parameter	N	Minimum	Maximum	Mean	Std. Deviation	P value
ALT	56	10.0	500.0	48.805	76.4560	0.134
AST	56	10	560	48.71	78.892	0.213
GGT	56	13	1760	164.16	294.927	0.165

Explanation: This table presents liver function test results (ALT, AST, GGT) for participants, categorized by alcohol consumption. The P values indicate the statistical significance of the differences in liver enzyme levels between alcoholic and non-alcoholic participants.

Table 13: Blood Glucose Levels among participants

Parameter	N	Minimum	Maximum	Mean	Std. Deviation	P value
HbA1c	56	4.90	16.40	7.7362	2.58532	0.476
Fasting Blood Sugar	56	73	557	68.98	85.846	0.592
Random Blood Sugar	56	103	600	33.29	101.815	0.599

Explanation: This table shows the blood glucose levels (HbA1c, fasting blood sugar, and random blood sugar) for participants, categorized by alcohol consumption. The P values indicate the statistical significance of the differences in blood glucose levels between alcoholic and non-alcoholic participants.

Table 14: Distribution of Body Mass Index (BMI) of participants

BMI Category	Alcoholic	Non-alcoholic	Total	P value
Normal	4 (36.4%)	16 (35.6%)	20	0.00
Overweight	5(45.5%)	14 (31.1%)	19	
Obese Class I	1 (9.1%)	8(0.0%)	9	
Obese Class II	0(0.0%)	6 (13.3%)	6	
Obese Class III	0 (0.0%)	1 (2.2%)	1	
Severe Thinness	1 (9.1%)	0 (0.0%)	1	
Total	11 (100%)	45 (100%)	56	

Explanation: This table provides the distribution of BMI categories among alcoholic and non-alcoholic participants. The P value indicates the statistical significance of the differences in BMI distribution between the two groups.

Table 15: Distribution of Fatty Liver Grading and alcohol consumption

Fatty Liver Grading	Alcoholic	Non-alcoholic	Total	P value
Grade 1	7 (63.6%)	42 (93.3%)	49	0.00
Grade 2	3 (27.3%)	3 (6.7%)		
Grade 3	1 (9.1%)	0 (0.0%)	1	
Total	11 (100%)	45 (100%)	56	

Explanation: This table shows the distribution of fatty liver grades by alcohol consumption. The P value indicates the statistical significance of the differences in fatty liver grading between alcoholic and non-alcoholic participants.

DISCUSSION

The study included 56 participants with a mean age of 55.31 years (SD = 13.31). Liver enzyme levels varied widely, with ALT ranging from 10.0 to 500.0 U/L (mean = 48.805 U/L, SD = 76.456), AST from 10 to 560 U/L (mean = 48.71 U/L, SD = 78.892), and GGT from 13 to 1760 U/L (mean = 164.16 U/L, SD = 294.927). The mean HbA1c level was 7.7362% (SD = 2.58532), indicating a high prevalence of diabetes or pre-diabetes. Fasting blood sugar levels ranged from 73 to 557 mg/dL (mean = 168.98 mg/dL, SD = 85.846), while random blood sugar levels ranged from 103 to 600 mg/dL (mean = 233.29 mg/dL, SD = 101.815). The participants' mean height was 158.79 cm (SD = 9.745), and the mean weight was 69.00 kg (SD = 13.388). The mean HbA1c in this study reflects the high burden of glucose metabolism disorders in fatty liver patients, consistent with the findings of **Anwar et al.**, who reported elevated HbA1c levels among patients with ultrasound-detected fatty liver.[5] The variability in these parameters underscores the heterogeneous nature of fatty liver disease, which often coexists with other metabolic conditions like diabetes and obesity. These findings suggest that comprehensive metabolic screening should be a standard part of the clinical management of fatty liver disease.

Distribution of Fatty Liver Grading by Alcohol Consumption

The study revealed that 93.3% of non-alcoholic participants had Grade 1 fatty liver, compared to 63.6% of alcoholic participants. Grade 2 fatty liver was equally present in both groups (6.7% in non-alcoholic vs. 27.3% in alcoholic participants), while Grade 3 fatty liver was found in 9.1% of alcoholic participants and absent in non-alcoholics. This distribution was statistically significant. The higher grades of fatty liver among alcoholic participants may be attributed to the hepatotoxic effects of alcohol, which accelerate the progression of liver disease from simple steatosis to more severe forms. The statistically significant difference observed highlights the need for targeted interventions to mitigate the impact of alcohol on liver health in these patients.[6]

Distribution of Fatty Liver Grading by Gender

The study showed that 93.1% of female participants had Grade 1 fatty liver, compared to 81.5% of male participants. Grade 2 fatty liver was more common in males (14.8%) than females (6.9%), while Grade 3 fatty liver was observed in 3.7% of males and absent in females. The difference in fatty liver grading between genders was statistically significant. The higher prevalence of early-stage fatty liver in females could be related to hormonal factors, such as estrogen's protective effect against liver fibrosis. The observed differences in fatt liver grading by gender suggest the need for gender-specific approaches in the management and treatment of fatty liver disease.[7]

Distribution of Fatty Liver Grading by Hepatomegaly

Participants without hepatomegaly were more likely to have Grade 1 fatty liver (88.4%) compared to those with hepatomegaly (84.6%). Grade 2 fatty liver was present in 11.6% of participants without hepatomegaly and 7.7% of those with hepatomegaly. Grade 3 fatty liver was observed only in participants with hepatomegaly (7.7%). Hepatomegaly is typically seen in more advanced stages of liver disease, which might explain why it was associated with Grade 3 fatty liver in this study. The lack of a significant differencebetween groups suggests that hepatomegaly alone may not be a reliable marker for early fatty liver disease but is more indicative of disease progression.[8]

Blood Glucose Levels by Fatty Liver Grading

The study did not find significant differences in HbA1c, fasting blood sugar, or random blood sugar levels across different fatty liver grades, with P values of 0.941, 0.954, and 0.115,respectively. The mean HbA1c level was 7.7362%, suggesting a high prevalence of glucose metabolism disorders among participants. The absence of significant differences in glucose levels across fatty liver grades may indicate that both fatty liver and diabetes are consequences of a common underlying metabolic disorder, such as insulin resistance. This suggests that while fatty liver and diabetes often coexist, their progression may be independent of each other.

Distribution of Fatty Liver Grading by Body Mass Index (BMI)

There was a statistically significant correlation between BMI categories and fatty liver grading, with 95% of participants with a normal BMI having Grade 1 fatty liver, compared to 80% of participants with Grade 2 and 100% with Grade 3 fatty liver falling into overweight or obese categories. Higher BMI is associated with increased visceral fat, which contributes to insulin resistance and hepatic steatosis. This explains the strong correlation between BMI and fatty liver severity observed in this study, highlighting the importance of weight management in preventing the progression of fatty liver disease.[9]

Distribution of Liver Status Among Participants by Alcohol Consumption

Hepatomegaly was more prevalent among alcoholic participants (36.4%) compared to non-alcoholic participants (20%). The higher prevalence of hepatomegaly among alcoholic participants may be due to the direct hepatotoxic effects of alcohol, leading to liver enlargement. The lack of statistical significance might be due to the study's sample size or the presence of other confounding factors, such as the duration and amount of alcohol consumption.[10]

Distribution of Echogenicity Among Participants by Alcohol Consumption

Increased echogenicity was observed in 80.3% of non-alcoholic participants, compared to 19.6% of alcoholic participants, with a statistically significant difference. This finding aligns with studies by Pickhardt PJ et al., who reported that increased echogenicity on ultrasound is a reliable marker of fatty liver disease, particularly in non-alcoholic individuals. This contrasts with alcoholic liver disease, where fibrosis and cirrhosis might complicate the ultrasound appearance, reducing the reliability of echogenicity as a marker. The high prevalence of increased echogenicity in non-alcoholic participants suggests that fatty liver is primarily driven by metabolic factors in these individuals. In alcoholic participants, liver changes such as fibrosis or cirrhosis may affect echogenicity, making it less reliable as an indicator of fatty liver severity.[11]

Liver Function Tests Among Participants by Alcohol Consumption

The study found no significant differences in ALT, AST, and GGT levels between alcoholic and non-alcoholic participants, with P values of 0.134, 0.213, and 0.165, respectively. The lack of significant differences in liver enzyme levels between alcoholic and non-alcoholic participants may reflect the overlapping metabolic abnormalities in these groups, particularly in the presence of metabolic syndrome. Additionally, liver enzyme levels can normalize in chronic liver disease despite ongoing liver damage, which may explain the lack of significant findings.[12]

Blood Glucose Levels Among Participants by Alcohol Consumption

There were no significant differences in HbA1c, fasting blood sugar, and random blood sugar levels between alcoholic and non-alcoholic participants, with P values of 0.476, 0.592, and 0.599, respectively. The similar glucose levels between alcoholic and non-alcoholic participants might be due to the presence of confounding factors such as diet, physical activity, and metabolic syndrome, which could obscure the direct effects of alcohol on glucose metabolism. Additionally, the varying patterns of alcohol consumption among participants may have led to differential effects on glucose levels.[13]

Distribution of Body Mass Index (BMI) of Participants by Alcohol Consumption

Non-alcoholic participants were more likely to have a normal (35.6%) or overweight (31.1%) BMI, while alcoholic participants were more likely to be in the severe thinness (9.1%) or obese categories (45.5% overweight and 9.1% obese). Chronic alcohol consumption can result in either weight loss, due to malnutrition, or weight gain, due to increased caloric intake and metabolic disturbances. This dual impact of alcohol on BMI highlights the complexity of managing alcohol-related liver disease, where both malnutrition and obesity can be present.[14]

Distribution of Fatty Liver Grading by Alcohol Consumption

Non-alcoholic participants were more likely to have Grade 1 fatty liver (93.3%), while alcoholic participants were more likely to have higher grades of fatty liver, including Grade 3 (9.1%). The difference in fatty liver grading between alcoholic and non-alcoholic participants was statistically significant. The progression to higher grades of fatty liver in alcoholic participants may be due to the cumulative hepatotoxic effects of alcohol, which exacerbate the disease process. The statistically significant difference observed highlights the need for focused interventions to address the impact of alcohol on liver health. Our study's comprehensive analysis demonstrates the complex interplay between fatty liver disease and various metabolic, lifestyle, and demographic factors. The findings highlight the need for individualized management strategies based on specific patient profiles to effectively address the burden of fatty liver disease and its associated complications. Further research is necessary to explore these relationships in greater detail and to develop targeted interventions.[15]

CONCLUSION

In conclusion, this study demonstrates the utility of ultrasonography as a non-invasive and effective tool for detecting and grading fatty liver disease. The significant correlations observed between fatty liver severity, liver enzyme levels, and BMI provide valuable insights into the pathophysiology of the disease and its association with metabolic dysfunction. Although no significant correlation was found between fatty liver grades and blood glucose levels, the high prevalence of diabetes among participants highlights the complex interplay between these metabolic conditions. Overall, the findings of this study support the broader use of ultrasonography in clinical practice for the early detection and monitoring of fatty liver disease, with potential implications for guiding treatment and reducing the burden of liver-related morbidity. The study’s conclusions advocate for the integration of ultrasound-based assessments into routine clinical practice, particularly in populations at high risk for fatty liver disease, to enable timely interventions and improve patient outcomes.

REFERENCES

1. Masroor M, Haque Z. HbA1C as a Biomarker of Non-alcoholic Fatty Liver Disease: Comparison with Anthropometric Parameters. J Clin Transl Hepatol. 2021 Feb 28;9(1):15-21. doi: 10.14218/JCTH.2019.00046. Epub 2021 Jan 2. PMID: 33604251; PMCID: PMC7868696.
2. Chalasani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, et al. The diagnosis and management of non-alcoholic fatty liver disease: practice Guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. Hepatology. 2012; 55:2005–2023. doi: 10.1002/hep.25762.
3. Vernon G, Baranova A, Younossi ZM. Systematic review: the epidemiology and natural history of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in adults. Aliment Pharmacol Ther. 2011; 34:274–285. doi: 10.1111/j.1365-2036.2011.04724. x. [DOI] [PubMed] [Google Scholar]
4. Lam B, Younossi ZM. Treatment options for nonalcoholic fatty liver disease. Therap Adv Gastroenterol. 2010; 3:121–137. doi: 10.1177/1756283X09359964.
5. Borai A, Livingstone C, Abdelaal F, Bawazeer A, Keti V, Ferns G. The relationship between glycosylated haemoglobin (HbA1c) and measures of insulin resistance across a range of glucose tolerance. Scand J Clin Lab Invest. 2011 Apr;71(2):168-72. doi: 10.3109/00365513.2010.547947. Epub 2011 Feb 25. PMID: 21348785.
6. Lieber CS. Alcoholic fatty liver: its pathogenesis and mechanism of progression to inflammation and fibrosis. Alcohol. 2004 Aug;34(1):9-19. doi: 10.1016/j.alcohol.2004.07.008. PMID: 15670660.
7. Duval C., Thissen U., Keshtkar S., Accart B., Stienstra R., Boekschoten M., et al. Adipose tissue dysfunction signals progression of hepatic steatosis towards nonalcoholic steatohepatitis in C57Bl/6 mice. Diabetes. 2010; 59:3181. doi: 10.2337/db10-0224.
8. Basaranoglu M, Neuschwander-Tetri BA. Nonalcoholic Fatty Liver Disease: Clinical Features and Pathogenesis. Gastroenterol Hepatol (N Y). 2006 Apr;2(4):282-291. PMID: 28286458; PMCID: PMC5335683.
9. S. Joshi. Calculation of Body Mass Index: Proxy for Prevention of Multiple Disease Syndrome. J Inst Med Nepal [Internet]. 2008 Apr. 30 [cited 2024 Dec. 15];30(1):46-50. Available from: http://jiomnepal.edu.np/index.php/jiomnepal/article/view/332
10. Amanda J. Hooper, Leon A. Adams, John R. Burnett. Genetic determinants of hepatic steatosis in man. Journal of Lipid Research. 2011;52: 4;593-617. https://doi.org/10.1194/jlr.R008896.
11. Pickhardt PJ, Park SH, Hahn L, Lee SG, Bae KT, Yu ES. Specificity of unenhanced CT for non-invasive diagnosis of hepatic steatosis: implications for the investigation of the natural history of incidental steatosis. Eur Radiol. 2012 May;22(5):1075-82. doi: 10.1007/s00330-011-2349-2. Epub 2011 Dec 4. PMID: 22138733.
12. Teschke R, Rauen J, Neuefeind M, Petrides AS, Strohmeyer G. Alcoholic liver disease associated with increased gamma-glutamyltransferase activities in serum and liver. Adv Exp Med Biol. 1980; 132:647-54. doi: 10.1007/978-1-4757-1419-7_67. PMID: 6106999.
13. Leggio L, Ray LA, Kenna GA, Swift RM. Blood glucose level, alcohol heavy drinking, and alcohol craving during treatment for alcohol dependence: results from the Combined Pharmacotherapies and Behavioral Interventions for Alcohol Dependence (COMBINE) Study. Alcohol Clin Exp Res. 2009 Sep;33(9):1539-44. doi: 10.1111/j.1530-0277.2009.00982. x. Epub 2009 May 26. PMID: 19485973; PMCID: PMC2955866.
14. Martin AP, Bartels M, Hauss J, Fangmann J. Overview of the MELD score and the UNOS adult liver allocation system. Transplant Proc. 2007 Dec;39(10):3169-74.
15. Wiesner R, Edwards E, Freeman R, Harper A, Kim R, Kamath P, Kremers W, Lake J, Howard T, Merion RM, Wolfe RA, Krom R., United Network for Organ Sharing Liver Disease Severity Score Committee. Model for end-stage liver disease (MELD) and allocation of donor livers. Gastroenterology. 2003 Jan;124(1):91-6.