

## ORIGINAL ARTICLE

# CORRELATION BETWEEN SERUM FERRITIN LEVELS WITH DIFFERENT HISTOLOGICAL STAGES OF NON-ALCOHOLIC FATTY LIVER DISEASE

FARAH JAHAN UPOMA<sup>1</sup>, ISHA ABDULLAH ALI<sup>2</sup>, FAROQUE AHMED<sup>3</sup>, NISHAT ALI<sup>4</sup>

### Abstract:

**Background:** Non-alcoholic fatty liver disease (NAFLD), a growing global epidemic, encompasses a broad spectrum of stages of disease severity, including simple steatosis, steatohepatitis (NASH), and cirrhosis. Accurate staging is necessary for the treatment and follow-up of the patients. Several non-invasive methods for the objective assessment of disease severity in NAFLD have been tested; serum ferritin is relatively new. Therefore, the objective of the study was to evaluate the correlation between the variation of serum ferritin levels and different histological stages of NAFLD. **Methods:** The study was a cross-sectional observational study conducted at the Department of Medicine and Hepatology, DMCH, from July 2019 to December 2021. All adult patients of NAFLD admitted or attended outdoors in the aforementioned department were approached for inclusion in the study. Assessment of the patients was done by the NAFLD Activity Score (NAS), and fibrosis staging and estimation of serum ferritin were also performed for each patient. Written informed consent was taken from the subject, and ethical issues were addressed. Following data collection, it was analyzed by the SPSS 17. **Results:** Out of a total of 50 patients, 60% were males and 40% were females (M:F=3:2). Mean age was 49.22±9.30 SD (years), ranging from 33 to 65 years. About 46% had class I obesity, 26% were overweight, and 8% had class II obesity. The majority of patients (62%) had NASH, 34% had borderline NASH, and 4% had no signature of NASH in the histopathology. The mean NAFLD activity score (NAS) was 4.98±1.47. Of all, 46%, 40%, and 10% of patients were in fibrosis stages 2, 1, and 3, respectively. Mean serum ferritin was 226.47±155.70 SD (ng/ml) [range: 13.60-722.00 ng/ml with a median value of 186.00 ng/ml]. Serum ferritin level was slightly higher in patients with NASH stage on histopathology than in those with borderline NASH stage and non-NASH stage. Similarly, the mean serum ferritin level was found to increase slightly with increasing fibrosis score. However, in both cases, the relationship is not statistically significant ( $p$ -value>0.05). **Conclusion:** Serum ferritin level is not associated with different histological stages of Non-alcoholic fatty liver disease.

**Keywords:** ferritin level, histological stages, NAFLD.

Date of submission: 08.03.2026 Date of acceptance: 20.04.2026

DOI: <https://doi.org/10.3329/bjm.v37i2.87941>.

**Citation:** Upoma FJ, Ali IA, Ahmed F, Rahman A, Iqbal MJ, Nishat Ali N. Correlation between Serum Ferritin Levels with Different Histological Stages of Non- Alcoholic Fatty Liver Disease . Bangladesh J Medicine 2026; 37(2): 114-120

1. Clinical Associate, Department of General Internal Medicine, Royal Alexandra Hospital, Edmonton, Alberta, Canada.
2. Assistant Professor, Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka, Bangladesh
3. Professor, Department of Hepatology, Dhaka Medical College & Hospital, Dhaka, Bangladesh
4. Specialist, Department of Internal Medicine, Evercare Hospital Limited, Dhaka, Bangladesh

**Correspondence:** Dr. Farah Jahan Upoma, Clinical Associate, Department of General Internal Medicine, Royal Alexandra Hospital, Edmonton, Alberta, Canada. E-mail: farah.jahan@gmail.com

## Introduction

Non-alcoholic fatty liver disease (NAFLD) is a growing global epidemic, which is the most common cause of elevated liver enzymes in Western societies. The prevalence of NAFLD overlaps with related medical epidemics of obesity, DM, and hypercholesterolemia<sup>1</sup>. NAFLD encompasses a broad spectrum of disease severity; these include simple steatosis, steatohepatitis (Non-alcoholic steatohepatitis/ NASH), fibrosis, and cirrhosis. Patients are often found asymptomatic. Accurate staging of NAFLD is necessary to prognosticate patients and identify those who require aggressive treatment of their metabolic syndrome and follow-up for monitoring of disease progression. Guidelines on NAFLD and NASH published in 2012 by the World Gastroenterology Organization suggest that the prevalence of NAFLD has doubled over the last 20 years, making NAFLD & NASH the leading cause of liver disease in Western countries. NAFLD affects more than 20% of the population worldwide. The prevalence of NAFLD in Europe is between 26 & 33%, in the Middle East is 20 & 30%, and up to 25% in America. In Europe, NASH affects 5% population and about 6 million people in the USA<sup>2</sup>. Little is known concerning the prevalence of NAFLD in Bangladesh because of the lack of population-based studies in our country. According to a study done in 2013, females are more affected, with a mean age of 40.8 + 10.2 years. The percentage of overweight, obese I, and obese II patients was 13.2%, 53.9%, and 21.2% respectively<sup>3</sup>.

There is a relationship between NAFLD and the level of serum ferritin. Ferritin is a protein that stores iron, releasing it when the body needs it. The greatest concentrations of ferritin are typically in the cells of the liver (known as hepatocytes) & immune system (known as reticuloendothelial cells); small amounts are secreted into the serum, where it functions as an iron carrier. It acts as a buffer against iron deficiency & iron overload. As NAFLD produces oxidative stress on the liver (an increased amount of iron plays an important role here), to counteract it, more ferritin is released. Other causes of hyperferritinemia are chronic viral hepatitis, massive liver injury due to sepsis, acute hepatitis or toxic injury, autoimmune disorders, etc.

Studying the relationship between these two may be beneficial because, despite promising advancements in non-invasive testing, liver biopsy remains the ultimate pathway for the diagnosis and determination of disease severity. According to the 2012 guidelines American Association for the Study of Liver Disease (AASLD), liver biopsy should be reserved for subjects who will “benefit” from potentially competing diagnoses and children with an unclear diagnosis. For patients of all ages, the NAFLD Activity Score (NAS) is now used

to find out the stages of NAFLD using the biopsy & remains the standard against which non-invasive (serologic and imaging) methods are judged to assess these features. To date, though histology evaluation is “the endpoint” tool, it has some limitations. The major limitation is the invasive nature of the procedure. There may be some complications like pain, minor and major bleeding (0.3%), organ perforation (in case of blind biopsy), even death (0.01%). Most importantly, performing a biopsy on all patients with suspected NAFLD remains controversial, and is not a practical consideration as a “screening” tool<sup>4</sup>.

Therefore, non-invasive methods for the objective assessment of the severity of NAFLD are the focus of intense research. Ultrasound elastography and magnetic resonant elastography may be diagnostic but are not available outside of tertiary care settings. Fibro test, Steato test, and NASH test are all expensive and remain under patents<sup>1, 5, 6, 7, 8</sup>.

In these circumstances, serum ferritin can play a promising role in diagnosing stages of NAFLD. Ferritin has historically been used to predict severe fibrosis in chronic liver disease<sup>9</sup>. Though it is an acute phase protein and can also be induced in the setting of systemic inflammation<sup>10, 11, 12, 13</sup>, studies have shown that inflammation is not the cause of an elevated serum ferritin level in NAFLD<sup>14, 15</sup>. This study aimed to determine the relationship between serum ferritin levels and stages of fatty liver disease.

## Methods

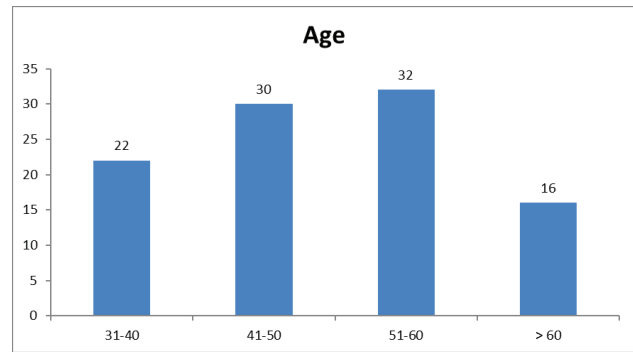
This hospital-based cross-sectional study was conducted at the Department of Medicine and Hepatology, DMCH, from July 2021 to December 2021. All adult patients of NAFLD (diagnosed by ultrasonography), aged more than 30 years of either sex & admitted or attended outdoors in the aforementioned department were approached for the study. A total of 50 cases admitted within the study period, fulfilling the inclusion and exclusion criteria, were included in this study by convenience purposive sampling. Study subjects who were alcoholics, had positive viral markers, and were consuming hepatotoxic drugs were excluded from the study. Assessment of the patients was done by the NAFLD Activity Score (NAS) and fibrosis staging. The total NAS ranged from 0 to 8. A score of 5 – 8 is considered NASH, 3 – 4 is considered borderline positive for NASH, and 0 – 2 is considered not diagnostic of NASH. Blood was collected for serum ferritin level assay, and a biopsy study was done to confirm the histological severity. Liver fibrosis can be divided into 3 stages, which include: Stage 0: No fibrosis; Stage 1: Perisinusoidal or periportal fibrosis; Stage 2: Perisinusoidal and

portal/periportal fibrosis; and Stage 3: Bridging fibrosis. Other necessary laboratory investigations were done and recorded. Informed consent was taken from the subject, and ethical issues were addressed. All the final data were collected in the semi-structured and pretested case record form. These data were analyzed statistically by SPSS 17 to arrive at a definite conclusion about the research question.

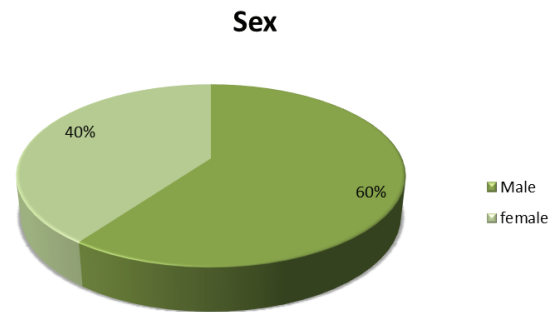
**Results**

A total of 50 cases of NAFLD patients were included in this study. The mean age was 49.22±9.30 years, ranging from 33 to 65 years (Fig. 1). The majority of the patients were male (60%). A male-to-female ratio of 3:2 was found (Fig.: 2). The majority of the patients were housewives (26%), followed in decreasing order by service holders (20%), unemployed (14%), shopkeepers (8%), garments workers (6%), security guards (6%), businessmen (4%), students (4%), school teachers (2%), lawyers (2%), sweepers (2%), day laborers (2%) and drivers (2%) (Fig.: 3). Forty-six percent of patients had a mildly active life, 44% had a sedentary lifestyle, and 10% were moderately active (Fig.: 4). Among all patients, 46% had class I obesity, 26% were overweight and 8% had class II obesity. Only 20% of patients had normal BMI (Fig. 5).

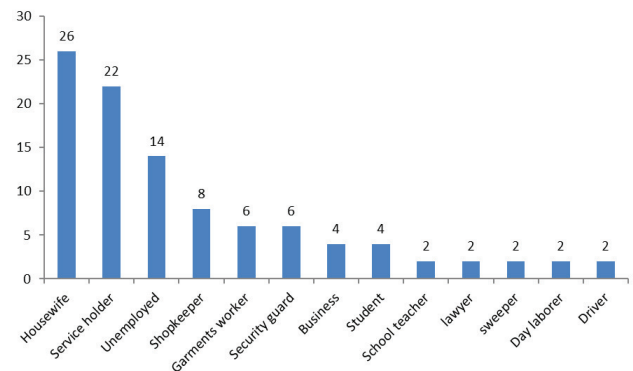
All of the patients in this study underwent a liver biopsy, and histopathological scoring through the NAFLD activity score (NAS) was done in each of them. The mean NAS was 4.98±1.47. A score of 5 – 8 is considered NASH, 3 – 4 is considered borderline or positive for NASH, and 0 – 2 is considered not diagnostic of NASH. The majority of patients (62%) had NASH in this study. Among the rest, 34% had borderline NASH and 4% had no signature of NASH in the histopathology (Table I). One of the important components of NAS is the fibrosis score. In this study, most of the patients (46%) had a score indicating perisinusoidal and portal/periportal fibrosis (stage 2) in histology. Forty percent had either perisinusoidal or periportal fibrosis (score 1) (Table II). Mean serum ferritin was 226.47±155.70 SD (ng/ml). Ferritin ranged from 13.60 ng/ml to 722.00 ng/ml with a median value of 186.00 ng/ml (Table III). Serum ferritin level was slightly higher in patients with NASH stage on histopathology than in those with borderline NASH stage and non-NASH stage. However, differences were not significant (p>0.05), showing that the serum ferritin level was not correlated with NAS histologic stages of liver in NAFLD patients (Table IV). The mean serum ferritin level was found to increase slightly with increasing fibrosis score. However, the differences were not significant (p>0.05), signifying that no clear relationship could be established between serum ferritin level and histologic fibrosis score (Table V).



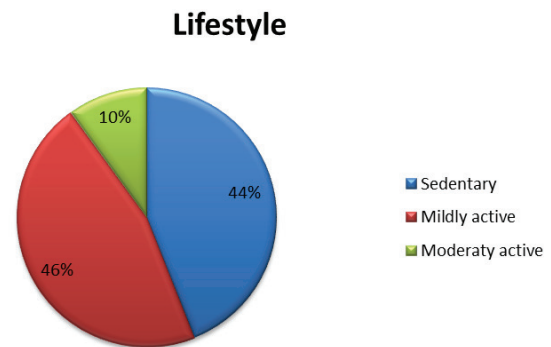
**Fig. 1:** Age group of patients (n=50)



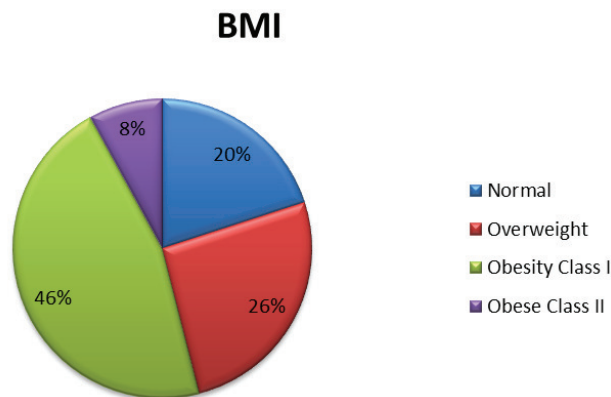
**Fig. 2:** Distribution of patients according to sex (n=50)



**Fig. 3:** Occupation of patients (n=50)



**Fig. 4:** Distribution of patients according to lifestyle (n=50)



**Fig. 5:** Distribution of patients according to BMI (n=50)

**Table I**

NAFLD activity score (NAS) of patients (n=50)

NAS	Frequency	Percentage
Stage		
Non-NASH Score 0 – 2	2	4
Borderline NASH Score 3 – 4	17	34
NASH Score 5 – 8	31	62
Mean±SD	4.98±1.47	

**Table II**

Fibrosis stage of patients (n=50)

Fibrosis score	Frequency	Percentage
None	2	4
Stage 0		
Perisinusoidal or periportal fibrosis	20	40
Stage 1		
Perisinusoidal and portal/periportal fibrosis	23	46
Stage 2		
Bridging fibrosis	5	10
Stage 3		
Mean±SD	1.62±0.72	

**Table III**

Serum ferritin descriptive of patients (n=50)

Serum ferritin (ng/ml)	Value
Mean	226.47
Standard Deviation	155.70
Median	186.00
Maximum	13.60
Minimum	722.00

**Table IV**

Relationship of serum ferritin with NAS histopathology staging of the liver (n=50)

NAS Stage	Serum ferritin (in ng/ml) mean±SD	p-value
Non-NASH	200.50±20.50	
Borderline NASH	200.93±159.23	0.30
NASH	249.38±154.70	

p-value obtained by the independent samples Kruskal-Wallis test

**Table V**

Relationship of serum ferritin with fibrosis score of liver (n=50)

Fibrosis score	Serum ferritin (in ng/ml) mean±SD	p-value
0	200.50±20.50	
1	223.31±164.97	0.67
2	231.71±154.99	
3	270.17±154.09	

p-value obtained by the independent samples Kruskal-Wallis test

**Discussion**

The present study included 50 patients with NAFLD to assess the relationship of serum ferritin with histologic staging of non-alcoholic fatty liver disease.

The mean age of patients was 49.22±9.30 years, ranging from 33 to 65 years. An increasing prevalence of NAFLD was noted through the 4th to 6th decade of life. This finding is consistent with the general finding that NAFLD incidence increases with age<sup>16</sup>. It has been reported that the prevalence of NAFLD increases with age (20% in people younger than age 20) to greater than 40% in those who are older than 60 years of age<sup>17</sup>. In this study, the majority of patients were male (60%) with a male:female ratio of 3:2. This contradicts the result of Alam et al. (2013), who found a female prevalence of 57%<sup>3</sup>. However, it supports the result of an epidemiological review by Lonardo et al. (2018) who reported that NAFLD is more common in men and has been shown to increase in those who are younger to middle-aged aged with a decline noted after the age of 50-60 years<sup>15</sup>. Alam et al.(2013) also concluded that age is not a predictor of NASH from NAFLD<sup>3</sup>.

Query on the occupation of patients revealed a preponderance of jobs requiring less physical activity, and 90% of patients were found to be leading either a sedentary lifestyle (44%) or a mildly active lifestyle (46%). Associations have been shown between a

person's fitness and sedentary behavior with the risk of developing NAFLD and NASH; the severity of NAFLD also intensifies with lower physical activity<sup>17</sup>. Clinical practice guidelines for the management of NAFLD recommend that assessment of physical activity habits should be included as part of a comprehensive NAFLD screening exam<sup>19</sup>.

In this study, only 20% of patients had normal BMI, and among the rest, the majority had class I obesity (46%), followed by 26% overweight and 8% class II obesity. Obesity is an established risk factor for NAFLD alongside dyslipidemia, DM type 2, and metabolic syndrome<sup>20</sup>. Hence, such a high prevalence of overweight NAFLD patients found in this study is not surprising. Also, the findings of these studies corroborate with the findings of Alam et al. (2013), who found normal BMI in 13.5% of patients, overweight in 8.1% of patients, obese type I in 49.3% of patients, and obese type II in 29.1% patients<sup>3</sup>.

All of the patients were found to have fatty liver through an initial USG. After that, all of them underwent histopathology of liver material. The NAFLD activity score (NAS) developed and validated by Kleiner et al. (2005) was calculated using defined criteria, and progression of NAFLD was noted<sup>21</sup>. In this study, 62% of patients progressed to NASH, 34% had borderline, and in 4% NASH was absent. This is nearly consistent with the findings of a study entitled "Prevalence and Predictor of Nonalcoholic Steatohepatitis (NASH) in Nonalcoholic Fatty Liver Disease (NAFLD)". The study found 42.4% positive NASH, 52.6% borderline NASH, and 5.6% NASH was absent. The mean NAS score was  $4.98 \pm 1.47$ . This is higher than the study conducted by Alam et al. (2014), who reported a mean score of  $4.4 \pm 1.4$  in non-obese and  $4.4 \pm 1.1$  in obese individuals<sup>22</sup>.

The mean annual rate of progression in NASH was reported at 40.76% by Younossi et al. (2016) in a global epidemiology study for NAFLD<sup>23</sup>. An explanation for the high prevalence of NASH in the study could be the high prevalence of obesity, which is an established risk factor for NASH<sup>24</sup>. One study found racial differences in the incidence of NASH. The study conducted by Williams et al. (2011) found a higher prevalence of NASH among Hispanics compared to Caucasians (19.4% vs 9.8%)<sup>25</sup>. The people of Bangladesh have a multiracial origin. The effect of race on the progression of NAFLD to NASH from the perspective of the country needs further evaluation through large-scale systematic research.

The fibrosis score is a component of the NAS score. Forty-six percent of patients in this study had stage 2 (perisinusoidal and periportal) fibrosis, 40% had stage

1 fibrosis (perisinusoidal or periportal), and 10% patients had stage 3 fibrosis (bridging). Fibrosis in adult NASH usually starts in acinar zone 3 and has a characteristic "chicken wire" pattern due to the deposition of collagen and other extracellular matrix fibers along the sinusoids of zone 3 and around the hepatocytes. Portal fibrosis without perisinusoidal/pericellular fibrosis has been reported in some cases of morbid obesity-related NASH<sup>26,27</sup>. Bridging fibrosis and cirrhosis may develop in advanced disease<sup>28</sup>. This indicates that approximately 10% of patients in this study were on the verge of developing cirrhosis, needing further attention in the management.

The present study found the mean serum ferritin level well within the normal range:  $226.47 \pm 155.70$  ng/ml. Mean values of serum ferritin showed an increasing trend in advancing histopathologic stage (as determined by NAS) as well as advancing fibrosis. However, the differences in values among stages were not significant. Other studies reported differing results on this aspect. Perikh et al. (2015) in India found that serum ferritin levels predict histologic grading in NAFLD<sup>29</sup>. On the other hand, Chandok et al. (2012) showed that serum ferritin does not have any relationship with the histologic grading of NAFLD<sup>1</sup>. However, a meta-analysis by Du et al. (2017) on this issue showed that the level of serum ferritin was elevated in patients with NAFLD (NAFL and/or NASH) compared with the controls, and compared with NAFL, the level of serum ferritin was increased in NASH<sup>30</sup>. This indicates a positive relationship.

One of the causes of increased serum ferritin in NAFLD patients is a C282Y mutation HFE gene that occurs with variable frequency in NAFLD patients, leading to elevated serum ferritin and transferrin saturation and necessitating a screening for genetic hemochromatosis<sup>31</sup>.

There is strong biological plausibility that serum ferritin may be a useful marker in assessing the stage of NAFLD. In the metabolic syndrome, insulin resistance leads to glucose intolerance, diabetes mellitus, low high-density lipoprotein, elevated uric acid, hypertension, and atherosclerosis. Ultimately, lipid peroxidation promotes steatosis within the liver, and further oxidative stress and alteration in fatty acid metabolism culminate in steatohepatitis and fibrosis through the activation of stellate cells. Through an adaptive response against the oxidative damage elicited by lipid peroxidation, endothelial cells in the liver upregulate the antioxidant molecule heme-oxygenase-1, as well as ferritin. Heme-oxygenase-1 expression correlates with ferritin, and given evidence that heme-oxygenase-1 parallels the severity of disease in NAFLD,

one could speculate that there would be a positive correlation between serum ferritin and the various stages of fatty liver disease <sup>1</sup>.

### Conclusion

In this study, it was revealed that the middle-aged population had suffered from NAFLD with male preponderance. Almost half of the patients lead with sedentary lifestyle with different levels of obesity. Only one-fifth of the patients had a BMI in the normal range. NAFLD activity scores suggest that around two-thirds of the patients had NASH, with fewer having borderline NASH.

### Limitations of the study

It was a single-center study. The study population was relatively small. Long-term follow-up was not included.

### Conflict of Interest:

The authors stated that there is no conflict of interest in this study

### Funding

This research received no external funding.

### Ethical consideration

The study was approved by the Ethical Review Committee of Dhaka Medical College & Hospital, Dhaka, Bangladesh. Informed consent was obtained from each participant or the caregiver of the patient.

### Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis, and interpretation, or all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

### Acknowledgments

The authors were grateful to the staff of the Department of Medicine & Hepatology of Dhaka Medical College & Hospital, Dhaka, Bangladesh.

### References

1. Chandok N, Minuk G, Wengiel M, Uhanova J. Serum ferritin levels do not predict the stage of underlying non-alcoholic fatty liver disease. *J Gastrointest Liver Dis.* 2012; 21(1):53-58
2. The International Liver Congress. Background Media information: NAFLD & NASH: a major public health problem. [Internet] Barcelona: Spain; 2016. Available from: <http://2016.ilc-congress.eu/wp-content/uploads/2016/04/Non-alcoholic-fatty-liver-disease-background.pdf>.
3. Alam S, Alam SMNE, Chowdhury ZR, Alam M, Kabir J. NASH in BD. *World J Hepatology.* 2013; 5(5): 281-287
4. Nalbantoglu I, Brunt EM. Role of liver biopsy in nonalcoholic fatty liver disease. *World Journal of Gastroenterology: WJG.* 2014; 20(27):9026.
5. Ratzu V, Massard J, Charlotte F, Messous D, Imbert-Bismut F, Bonyhay L, et al. Diagnostic value of biochemical markers (FibroTest-FibroSURE) for the prediction of liver fibrosis in patients with non-alcoholic fatty liver disease. *BMC Gastroenterology.* 2006; 6(1):6.
6. Lassailly G, Caiazzo R, Hollebecque A, Buob D, Leteurtre E, Arnalsteen L, et al. Validation of noninvasive biomarkers (FibroTest, SteatoTest, and NashTest) for prediction of liver injury in patients with morbid obesity. *European journal of gastroenterology & hepatology.* 2011; 23(6):499-506.
7. Canbakan B, Senturk H, Tahan V, Hatemi I, Balci H, Toptas T, et al. Clinical, biochemical, and histological correlations in a group of non-drinker subjects with non-alcoholic fatty liver disease. *Acta gastroenterologica Belgica.* 2007; 70(3):277-84.
8. Yoneda M, Thomas E, Sumida Y, Imajo K, Eguchi Y, Hyogo H, et al. Clinical usage of serum ferritin to assess liver fibrosis in patients with non alcoholic fatty liver disease: Proceed with caution. *Hepatology Research.* 2014; 44(14).
9. Kowdley KV, Belt P, Wilson LA, Yeh MM, Neuschwander Tetri BA, Chalasani N, et al. NASH Clinical Research Network. Serum ferritin is an independent predictor of histologic severity and advanced fibrosis in patients with nonalcoholic fatty liver disease. *Hepatology.* 2012; 55(1):77-85.
10. Ferrara F, Ventura P, Vegetti A, Guido M, Abbati G, Corradini E, et al. Serum ferritin as a predictor of treatment outcome in patients with chronic hepatitis C. *The American journal of gastroenterology.* 2009; 104(3):605.
11. Beaton MD, Chakrabarti S, Adams PC. Inflammation is not the cause of an elevated serum ferritin in non-alcoholic fatty liver disease. *Age (yrs).* 2014; 49:12.
12. Crook MA. Hyperferritinaemia: laboratory implications. *Ann Clin Biochem.* 2012; 49: 211-3
13. Britton LJ, Subramaniam VN, Crawford DH. Iron and non-alcoholic fatty liver disease. *World journal of gastroenterology.* 2016; 22(36):8112.
14. Loomba R, Sanyal AJ. The global NAFLD epidemic. *Nat Rev Gastroenterol Hepatol.* 2013; 10: 686-690.
15. Vernon G, Baranova A, Younossi ZM. Systemic review: the epidemiology and natural history of non-alcoholic liver disease and non-alcoholic steatohepatitis in adults. *Aliment Pharmacol Ther.* 2011; 34: 274-285.
16. Patel V, Sanyal AJ, Sterling R. Clinical presentation and patient evaluation in nonalcoholic fatty liver disease. *Clinics in liver disease.* 2016; 20(2):277-92.
17. Benedict M, Zhang X. Non-alcoholic fatty liver disease: An expanded review. *World J Hepatol.* 2017; 9(16):715-732.

18. Lonardo A, Bellentani S, Argo CK, Ballestri S, Byrne CD, Caldwell SH, et al. Epidemiological modifiers of non-alcoholic fatty liver disease: Focus on high-risk groups. *Dig Liver Dis*. 2015; 47: 997-1006
19. Marchesini G, Day ChP, Dufour JF, Canbay A, Nobili V, Ratziu V, et al. EASL-EASD-EASO Clinical Practice Guidelines for the management of non-alcoholic fatty liver disease. *J Hepatol*. 2016;64:1388-1402.
20. 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(6):2005-23.
21. Kleiner DE, Brunt EM, Van Natta M, Behling C, Contos MJ, Cummings OW, et al. Design and validation of a histological scoring system for nonalcoholic fatty liver disease. *Hepatology*. 2005; 41(6):1313-21.
22. Alam S, Gupta U Das, Alam M, Kabir J, Chowdhury ZR, Alam AKMK. Clinical, anthropometric, biochemical, and histological characteristics of nonobese nonalcoholic fatty liver disease patients of Bangladesh. *Indian J Gastroenterol*. 2014; 33(5):452-7.
23. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease—Meta analytic assessment of prevalence, incidence, and outcomes. *Hepatology*. 2016; 64(1):73-84.
24. Rinella ME. Nonalcoholic Fatty Liver Disease: A Systematic Review. *JAMA*. 2015; 313(22):2263-73.
25. Williams CD, Stengel J, Asike MI, Torres DM, Shaw J, Contreras M, et al. Prevalence of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among a largely middle-aged population utilizing ultrasound and liver biopsy: a prospective study. *Gastroenterology*. 2011; 140(1):124-31.
26. Ratziu V, Giral P, Charlotte F, Bruckert E, Thibault V, Theodorou I, et al. Liver fibrosis in overweight patients. *Gastroenterology* 2000; 118: 1117-1123.
27. Dixon JB, Bhathal PS, O'Brien PE. Nonalcoholic fatty liver disease: predictors of nonalcoholic steatohepatitis and liver fibrosis in the severely obese. *Gastroenterology*. 2001; 121: 91-100.
28. Brunt EM, Tiniakos DG. Alcoholic and nonalcoholic fatty liver disease. In: Odze RD, Goldblum JR, eds. *Surgical Pathology of the GI Tract, Liver, Biliary Tract, and Pancreas*. 2nd ed. Philadelphia: Elsevier, 2009: 1007-1014.
29. Parikh P, Patel J, Ingle M, Sawant P. Serum ferritin levels predict histological severity in patients with nonalcoholic fatty liver disease in India. *Indian J Gastroenterol*. 2015; 34(3):200-08.
30. Du SX, Lu LL, Geng N, Victor DW, Chen LZ, Wang C, et al. Association of serum ferritin with non-alcoholic fatty liver disease: a meta-analysis. *Lipids in health and disease*. 2017; 16(1):228.
31. Kowdley KV. The role of iron in nonalcoholic fatty liver disease: the story continues. *Gastroenterology*. 2010; 138:817-819.