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Original Article

Prediction of Independent Risk Factors for Non-Alcoholic Fatty Liver Disease Based on Regression Analysis

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Abstract

Background: *Nonalcoholic Fatty Liver Disease (NAFLD) is a common liver disease globally, but there are no optimal methods for its prediction or diagnosis. Diagnostic markers for NAFLD are still needed for screening individuals at risk. The present cross-sectional study proposes a non-invasive tool for NAFLD screening. To establish a risk prediction model of NAFLD by Multivariable logistic regression analysis, to identify the independent risk factors for NAFLD and thus provide management strategies for preventing this disease.*

Materials and methods: *This cross-sectional study was conducted inthe Department of Biochemistry in collaboration with Institute of Nuclear Medicine and Allied Sciences (INMAS) Chittagong Medical College Hospital (CMCH) for a period of one year from June-2017 till June- 2018. One hundred and fifty (150) subjects aged between 18 to 60 years were enrolled using a non-probability consecutive sampling method. The variables of interest were Age, Body Mass Index (BMI) Central Obesity, Fasting Blood Glucose/Sugar (FBS) Fasting Plasma Insulin (FPI) Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) AST, ALT Total Cholesterol (TC) Triglycerides (TG) and High-density Lipoprotein Cholesterol (HDL-c). NAFLD subjects were identified through ultrasonography and Insulin Resistance (IR) was evaluated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR).*

Results: *It revealed that after multivariate adjustment, only Age, BMI, FBS, FPI and HOMA-IR >2.6 have significant predictive value for NAFLD. Respondent who had age more than 40 years, BMI ≥25kg/mt^2, Central obesity, FBS ≥100 mg/dl, FPI >15mIU/L and HOMA-IR >2.6 were significantly more likely to have NAFLD than their counterpart.*

Conclusion: *The predictive indicators can have a certain effect on the early screening and the timely prevention of the progress of related complications. As a result, introducing the predictive indicators is useful for the prediction of NAFLD individuals.*

Key words: Body Mass Index (BMI), Fasting Plasma Insulin (FPI); Homeostatic Model Assessment of Insulin Resistance (HOMA-IR); Multivariable logistic regression analysis; Non Alcoholic Fatty Liver Disease (NAFLD).

INTRODUCTION

Non-Alcoholic Fatty Liver Disease (NAFLD) is defined as the accumulation of fat in the liver in the absence of secondary factors like excessive alcohol consumption.[1] It is commonly associated with non-alcoholic steatohepatitis (NASH), hepatic fibrosis, liver cirrhosis and Hepatocellular Carcinoma (HCC).[2–4] NAFLD affects approximately 25% of the global population, contributing to a significant health burden.[5,6] The liver-specific and all-cause mortality rates among NAFLD patients are 0.77 and 11.77 per 1,000 person-years, respectively.[6] While the exact causes of

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NAFLD remain unclear and are multifactorial, its prediction becomes essential so as to prevent its progression to NASH and HCC. Although there are no approved pharmacological treatments and since it is reversible in its early stages, measures such as lifestyle modification, including caloric restriction, dietary modifications and increased physical activity, have proven effective in preventing the progression of the disease.[7, 8] These interventions are particularly beneficial in the early phases of the disease, highlighting the importance of early detection as a public health priority.

Currently, there are neither published nor established clinical markers that can reliably predict NAFLD. Although Liver biopsy remains the gold standard for diagnosis, its invasive procedure carries potential risks rather complications, and is costly.[9,10] In contrast, Ultrasonography (USG) is a non-invasive and more commonly used method for diagnosing NAFLD.[11] However, ultrasonography, along with other imaging techniques like Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) is expensive and impractical for routine health check-ups or large-scale population screening. Given the limitations of liver biopsy and imaging techniques, there has been growing interest in identifying non-invasive clinical variables measurable from peripheral blood to diagnose NAFLD [12–14].

Several previous studies have focused on creating NAFLD risk prediction models using non-invasive measures [15–18]. The most commonly used variables in these models are biochemical markers such as HDL-c, TC, TG, FPI and ALT. However, many predictive models rely on one or two biomarkers that are not typically included in routine health examinations, such as serum insulin or FPI.[19] Additionally, these indicators often require complex calculations and do not account for lifestyle factors, such as dietary habits and physical activity frequency, which are important for assessing the impact of interventions.[8] Anthropometric Indicessuch as BMI, Waist Circumference (WC) and Waist Hip Ratio (WHR) also play role in predicting NAFLD. An ideal non-invasive test should be simple, readily accessible, low-cost, and effective, offering clear results that can easily identify the individuals at high risk for NAFLD. Such a test would enable large-scale screening and preventive health programs for broader populations. Consequently, this study aims to develop an optimal model for predicting and monitoring NAFLD.

The aim of this study was to develop an easily applicable prediction model for identifying individuals at high risk for NAFLD, using known risk factors such as age, obesity, lifestyle, and biochemical indicators. While prediction models have been successfully developed for various clinical diseases and tumors, their application to NAFLD remains relatively uncommon. Therefore, the model presented in this study could contribute to the creation of an early warning and prediction system for NAFLD, specifically tailored to the Bangladeshi population.

MATERIALS AND METHODS

A cross-sectional study was conducted at the Department of Biochemistry in collaboration with the Institute of Nuclear Medicine and Allied Sciences (INMAS) Chittagong Medical College and Hospital (CMCH). The study included 150 subjects, aged between 18 and 60 years, and was carried out over a period of one year, from June 2017 to June 2018 by non-probability consecutive sampling method. Both male and female subjects were evaluated sonographically and categorized as NAFLD cases (n=80) and non-NAFLD controls (n=70). Subjects were excluded if they tested positive for hepatitis B virus surface antigen or anti-hepatitis C virus antibody, or if they had a history of liver cirrhosis, acute or chronic hepatitis, alcohol abuse, type 2 diabetes mellitus or pregnancy.

Data were collected using a pre-tested structured questionnaire that included all relevant variables of interest, after obtaining informed and written consent from the subjects. Standing height and Weight was measured using stadiometer. The height was recorded to the nearest 5 mm and Weight was recorded to the nearest 0.5 Kg. BMI of the subjects were calculated using the formula, BMI = Weight (Kg)/ Height (m^2). WC was measured to the nearest 0.5 cm with a soft non-elastic measuring tape and was taken between the lower border of the 12th rib and the highest point of the iliac crest on the mid-axillary line at the end of normal expiration.

Following standard phlebotomy procedures and ensuring all aseptic precautions, 5 ml of fasting venous blood was drawn from the median cubital vein between 8 and 9 a.m. The blood was collected into clean, dry test tubes and allowed to clot. After centrifugation, the serum was separated and transferred into Eppendorf tubes, which were then immediately transported to the Biochemistry Laboratory for analysis.

FBS was determined by enzymatic Glucose Oxidase method. FPI assay was a two-site sandwich immunoassay using direct chemiluminescent technology which used constant amounts of two antibodies. IR was calculated from FBS and FPI values by HOMA-IR i.e. HOMA-IR = [(FBS mmol/L) X (FPI μIu/L)] ÷ 22.5. Fasting TC was measured using a polychromatic endpoint technique. Fasting TG, HDL-c and LDL-c were measured by bichromatic endpoint technique.AST and ALT reagent is used to measure AST and ALT activity respectively by an enzymatic rate method.

Statistical analyses were performed using SPSS for Windows version 20.0. Statistical inference was based on a 95% Confidence Interval (CI) with a p-value ≤0.05 considered statistically significant. The summarized data were presented in tabular form. The Student's t-test was used for comparing quantitative or continuous variables, while the Chi-square test was applied for qualitative or categorical variables. Predictive risk factors for NAFLD were identified through Binary Logistic Regression Analysis.

RESULTS

Complete clinical profiles, sonographic data, and serum samples were available for 150 subjects, consisting of 80 NAFLD cases (53%) and 70 non-NAFLD controls (47%). The mean age of the NAFLD group (35.05 ± 1.05 years) was significantly higher than that of the control group (26.53 ± 1.02 years). Among the 80 NAFLD cases, 66.3% were in the 30-49 years age group [Table I]. Central obesity was observed in 90% of the NAFLD group, with a higher prevalence in females. Both male and female subjects with NAFLD had significantly higher WC and WHR compared to the non-NAFLD controls [Table II]. Additionally, the BMI of NAFLD subjects was significantly higher, with 81.3% of NAFLD patients classified as obese.

FBS, FPI and HOMA-IR were significantly higher in the NAFLD cases ($p < 0.05$). Serum TG was also significantly higher in the NAFLD group, while HDL-c levels were significantly higher in the control group ($p < 0.05$). No significant difference was observed in the mean LDL values between the two groups. Additionally, serum ALT and AST levels were significantly elevated in the NAFLD cases compared to the controls ($p < 0.05$) [Table III].

Of the 80 NAFLD cases, 90% (n = 72) had IR(HOMA-IR value >2.6), proving to be more prevalent and being 15.23 times more likely to have IRin NAFLD ($p < 0.001$). Obese individuals with NAFLD were 4.53 times more likely to have IR. A significant association was found between NAFLD, BMI, WC and HOMA-IR (Table IV).

After adjusting for age, BMI, FBS, TGand LDL, the independent risk factors for NAFLD were identified. Multivariate analysis revealed that Age, increased BMI, central obesity, and FBS were the only significant predictors of NAFLD. Respondents aged over 40 years, with a BMI 25 kg/m², and FBS 100 mg/dl were significantly more likely to have NAFLD compared to their counterparts (Table V).

Table I Age distribution among the NAFLD cases (n = 80)

Age in years	NAFLD cases	Percentage (%)
<30	22	27.5%
30-39	28	35%
40-49	25	31.25%
50-59	4	5%
60	1	1.25%
Total	80	100%

Table II Distribution of Respondents according to their waist circumference

Waist circumference (cm) Category	Case (n=80) n	%	Control (n=70) n	%	Test statistics p value
Normal	8	10.0	70	100.0	p< 0.001
Central obesity	72	90.0	0	0.0	
Gender	Mean ±SEM	Mean ±SEM	p value		
Male	95.83±1.59	72.59±0.69	p<0 .001		
Female	96.11±1.03	66.37 ±0.41	p< 0.001		

Table III Comparison of fasting plasma glucose, fasting serum insulin level, HOMA-IR, Fasting Lipid Profile and Liver enzymes amongst the study population (n=150)

Variables	Cases(n=80) (Mean ± SEM)	Controls(n=70) (Mean ± SEM)	p value
Fasting blood glucose (mmol/l)	5.63 ± 0.67	5.45 ± 0.6	p = 0.02
Fasting serum insulin (mIU/L)	19.00 ± 0.61	12.8 ± 0.73	p < 0.00001
HOMA-IR	4.77 ± 0.16	3.13 ± 0.19	p < 0.0001
Total Cholesterol (mg/dl)	174.64 ± 3.82	163.39 ± 4.10	p = 0.04
Serum TG (mg/dl)	203.01 ± 9.65	177.94 ± 6.48	P = 0.032
Serum HDL (mg/dl)	34.94 ± 0.94	36.61 ± 0.79	p = 0.035
Serum LDL (mg/dl)	101.94 ± 2.58	99.91 ± 2.85	p = 0.61
Serum ALT (U/L)	42.15 ± 1.37	38.87 ± 1.18	p = 0.037
Serum AST (U/L)	23.29 ± 0.76	21.43 ± 0.73	p = 0.039

Table IV Association between Predictive Variables and insulin resistance status (HOMA-IR)

Groups	Category of HOMA-IR HOMA-IR >2.6	HOMA-IR ≤2.6	Total	Odds ratio (95% CI)	p value & test statistic
NAFLD Category (n=150)					
NAFLD (Cases)	72 (90%)	08 (10%)	80	15.23 (6.34-36.59)	$\chi^2 = 46.05$ p < 0.001
Non-NAFLD (Controls)	26 (37%)	44 (63%)	70		
BMI category (kg/m^2) (n=80)					
Non-obese (BMI<25)	13 (77%)	04 (23%)	17	4.53 (1.32-9.82)	χ^2= 4.39 p=0.03
Obese (BMI ≥ 25)	59 (94%)	04 (06%)	63		
Waist hip ratio (n= 80)					
Normal	03 (60%)	02 (40%)	05	7.67 (2.03-10.45)	χ^2= 5.33 p = 0.02
Increased	69 (92%)	06 (08%)	75		

Table V Risk factors associated with NAFLD derived from binary logistic regression analysis

Variables	Range	Odds ratio	95% CI	p value
Age	>40 years versus ≤40 years	18.51	41.86-113.75	<0.001
BMI	≥ 25kg/m^2 versus <25kg/m^2	21.85	14.86-33.75	<0.001
Central obesity	Central obese versus normal	1.34	1.01-12.56	0.044
HTN	Present versus absent	1.27	0.093-6.01	0.859
FBS	Elevated versus normal	4.39	1.09-17.59	0.037
FPI	>15 mIU/L versus ≤15mIU/L	2.03	1.02-14.59	0.047
HOMA-IR	>2.6 versus ≤2.6	19.25	1.3-173.59	0.005
TG	High versus normal	1.25	0.34-4.65	0.739
HDL	Normal versus low	0.75	0.09-6.07	0.787
ALT	High versus normal	1.25	1.02-4.65	0.039
AST	High versus normal	1.01	0.34-4.65	0.629

DISCUSSION

NAFLD, as a metabolic disease affecting multiple systems, is strongly associated with a higher incidence of extrahepatic complications, including cirrhosis, coronary heart disease and CKD.[20] Its growing prevalence has become a significant public health challenge and poses a serious threat to health and public safety.[21,22] Despite its increasing significance, the exact pathogenesis of NAFLD remains unclear. Contributing factors such as viral infections, autoimmune liver diseases, oxidative stress, IR, genetic predisposition, and disturbances in intestinal flora have all been implicated in the development NAFLD.[23] Current research is focusing on identifying the factors influencing NAFLD, developing predictive risk models, pinpointing high-risk groups, and advancing early diagnosis and prevention strategies.[24] The development of an effective prediction model could play a crucial role in accurately forecasting the progression of NAFLD, thereby enabling timely monitoring and intervention in high-risk populations.Therefore, it is the key to establish the forecasting model to select easily available and accurate prediction indicators.

In this study, a NAFLD risk prediction model was developed using logistic regression, incorporating various factors such as physical examination results and laboratory investigations. Clinical profiles, sonographic data, and serum samples were available for 150 subjects, consisting of 80 NAFLD cases (53%) and 70 non-NAFLD controls (47%). The mean age of the NAFLD group was significantly higher, with 66.3% of cases falling within the 30-49 years age group. Central obesity was observed in 90% of the NAFLD group, with a higher prevalence among females. Both male and female subjects with NAFLD exhibited significantly higher BMI, WC and WHR. Additionally, FBS, FPI and HOMA-IR, Serum ALT, AST, and TG were significantly elevated in the NAFLD group. On the other hand, HDL-c levels were significantly higher in the control group. A significant association was identified between NAFLD, BMI, WC, and HOMA-IR. After adjusting for Age, BMI, FBS, TG, and LDL, multivariate analysis revealed that Age, increased BMI, central obesity, and FBS were the primary independent predictors of NAFLD. Subjects aged over 40 years, with a BMI ≥25 kg/m² and FBS ≥100 mg/dl were significantly more likely to have NAFLD compared to their counterparts.

The results from this study indicate that high BMI and age are independent risk factors for NAFLD, which aligns with findings from previous studies aimed at developing NAFLD risk models. This association may be linked to lower levels of physical activity, as other investigations have shown a positive correlation between sedentary behavior and the development of NAFLD.[25] A recent cross-sectional study focusing on younger individuals also confirmed that physical activity is independently related to the extent of hepatocyte damage and the risk of NAFLD in the general population.[26]

These findings emphasize the importance of maintaining an active lifestyle to reduce the risk of NAFLD. Previous studies have highlighted dyslipidemia as a key factor in the development of NAFLD.[20] In the present study TG and LDL-c levels were higher in the NAFLD group but were not identified as independent risk factors for the condition. However, numerous studies have shown that excessive lipid accumulation contributes to the production of Reactive Oxygen Species (ROS) which disrupt the redox balance by inducing oxidative stress. This, in turn, activates proinflammatory signaling pathways that exacerbate hepatocyte damage.[27,28] Additionally, high TG levels can lead to IR, which subsequently results in hyperglycemia. Elevated blood glucose then stimulates insulin secretion, promoting the synthesis of TG and LDL-C in hepatocytes, creating a vicious cycle that further exacerbates lipid accumulation in the liver.[29] ALT is a key enzyme predominantly found in hepatocytes and the results of this study support its potential use as a predictive biomarker for the onset of NAFLD. A recent study showed that elevated ALT levels can reduce insulin sensitivity in hepatocytes, negatively affecting glucose metabolism and fat accumulation, thus exacerbating the development of NAFLD.[30] Furthermore, several studies have suggested that polymorphisms in the Microsomal Triglyceride Transfer Protein (MTTP) gene impair the transport of fats out of the liver, leading to the retention of TG within hepatocytes. This genetic defect may contribute to the pathogenesis of NAFLD.[31, 32]

LIMITATION

One limitation of this study includes not assessing NAFLD by Liver biopsy and the absence of MTTP gene analysis, which could have provided additional insights into the genetic factors influencing NAFLD development.

CONCLUSION

The predictive indicators can have a certain effect on the early screening and the timely prevention of the progress of related complications. As a result, introducing the predictive indicators is useful for the prediction of NAFLD individuals.

RECOMMENDATIONS

Further prospective multicenter study in large scale, application of Liver biopsy for NAFLD diagnosis and MTTP gene analysisis necessary to better understand the biochemical strategy of inflammatory markers for the development of NAFLD.

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DISCLOSURE

All the authors declared no competing interest.

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