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Relation between BMI and Non alcoholic fatty liver risk

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Abstract

Background: *Nonalcoholic fatty liver disease (NAFLD) is the most ordinary liver disease worldwide. The growing body of evidence has demonstrated that BMI is risk factor for cardiovascular disease, and the pathological processes included insulin resistance, oxidative stress, and systemic inflammation, which are all considered as important risk factors for the development or progression of NAFLD. Limited evidence is available regarding the association between BMI and NAFLD.* ***Aims and Objectives:*** *The aim of this study was to investigate the association of BMI with non-alcoholic fatty liver disease.* ***Materials and Methods:*** *A cross-sectional observational study carried out in the Department of Biochemistry, Sylhet MAG Osmani Medical College, Sylhet in collaboration with the Department of Hepatology, Sylhet MAG Osmani Medical College, Hospital during the period between July 2017 and December 2018. Fifty cases of NAFLD and 50 age and sex matched healthy subjects were selected. BMI of both case & control were measured.* ***Results:*** *The mean BMI of the non-alcoholic fatty liver disease was 26.08 ± 1.41 (range 23.44-30.10) kg/m^2; whereas the mean BMI of the control subjects was 23.89 ± 2.31 (range 17.82-30.10) kg/m^2. The mean BMI of non-alcoholic fatty liver disease was significantly higher than that of control subjects ($t=5.718$; $p<0.001$).* ***Conclusion:*** *It may be concluded that BMI is strongly associated with non-alcoholic fatty liver disease.*

Keywords: *BMI, Nonalcoholic fatty liver disease*

Introduction

Nonalcoholic fatty liver disease (NAFLD) is the most prevalent causes of chronic liver disease worldwide. It is characterized by increased fat accumulation in the hepatocytes of the liver parenchyma (1). Increasingly sedentary lifestyles and altering dietary patterns mean that the frequency of adiposity and insulin resistance has accelerated worldwide, and so fat collection in the liver is a familiar finding during abdominal imaging probes and on liver biopsy.

Several studies have suggested that a high BMI triggers risk factors for lifestyle-related diseases such as type 2 diabetes, cardiovascular diseases, cancers, and high UA (2). Indeed, a study by De Oliveira et al. has shown that individuals with BMI ≥25 kg/m^2 showed greater chances of high UA levels [odds ratio (OR) = 2.28 (1.13–4.6)] (3). Consequently, this study indicates that elevated BMI levels are closely related to high UA levels, and in order to prevent high UA, one should pay attention to BMI level change.

Hyperuricemia is associated with the NAFLD development, even after adjusting for potential confounders including metabolic syndrome features and insulin resistance (4,5). Regarding the prospective role, hyperuricemia has pro-inflammatory, pro-oxidant and insulin resistance properties in adipose tissue (6).

Therefore metabolic derangement based on insulin resistance is the most acknowledged cause of fat accumulation (7), and metabolic syndrome is considered to be a crucial mediator from simple over-nutrition to severe body lesion by promoting inflammation (6), and are hypothesized to explain the complex pathgenesis and progression of non-alcoholic fatty liver disease (8).

The proportion of nonalcoholic fatty liver disease is higher in people with type 2 diabetes (60%-70%), and in those who has high BMI compared to the general population (9,10,11). It seems that insulin resistance (IR) related to obesity is central to the pathogenesis of NAFLD. Moreover, oxidative stress and cytokines are main contributing factors, together resulting in steatosis and progressive liver damage in genetically susceptible individuals. Key histological components of NASH are steatosis, hepatocellular ballooning, and lobular inflammation (7).

Materials and Methods

This cross-sectional observational study was carried out in the Department of Biochemistry, Sylhet MAG Osmani Medical College, Sylhet in collaboration with the Department of Hepatology, Sylhet MAG Osmani Medical College, Hospital, during the period from July 2017 to December 2018 with a view to compare BMI between non-alcoholic fatty liver disease and healthy subjects. In this study 50 cases of NAFLD and another 50 age and sex matched healthy subjects were selected and grouped as group-A and group-B respectively. Informed written consent was taken before interview. Anthropometric measurements including height, weight, waist circumference and blood pressure were recorded. BMI was considered as weight in kilogram divided by the height in meter square. Fatty liver was diagnosed based on the findings of abdominal ultrasonography without alcohol consumption, viral or autoimmune liver disease. Fasting plasma glucose and fasting insulin were collected for biochemical analysis.

Result

The mean age of the participants of non-alcoholic fatty liver disease (Group-A) was 40.04 ± 9.37 years and control subjects (Group-B) was 40.90 ± 12.37 years. The mean age of the participants did not differ significantly between two groups (t=0.519; p=0.605) (Table-I).

The mean BMI of the non-alcoholic fatty liver disease was 26.08 ± 1.41 (range 23.44-30.10) kg/m^2; whereas the mean BMI of the control subjects was 23.89 ± 2.31 (range 17.82-30.10) kg/m^2. The mean BMI of non-alcoholic fatty liver disease was significantly higher than that of control subjects (t=5.718; p<0.001) (Table-I).

Table-I: Distribution of the participants according to baseline characteristics

Parameters	Case (n=50)	Control (n=50)	Test value	p-value
Age in years Mean ± SD	40.04 ± 9.37	40.90 ± 12.37	t=0.519	*p=0.605
Sex				
Male	28 (56.0%)	32 (64.0%)	p=0.667	p=0.414
Female	22 (44.0%)	18 (36.0%)		
BMI in Kg/M^2 Mean ± SD	26.08 ± 1.41	23.89 ± 2.31	t=5.718	*p<0.001

*unpaired 't' test and †Chi-Square (χ2) test were employed to analyze the data. P<0.05 was the level of significance.

Distribution of patients by serum insulin level:

The median serum insulin level (µIU/ml) was 9.36 (inter quartile range, 5.94-14.05) in non-alcoholic fatty liver disease and was 4.69 (inter quartile range, 3.44-6.72) in control subjects. The serum insulin level of the non-alcoholic fatty liver disease was significantly higher compared to control subjects (p<0.001) (Figure-1).

* Mann-Whitney U test was employed to analyze the data.

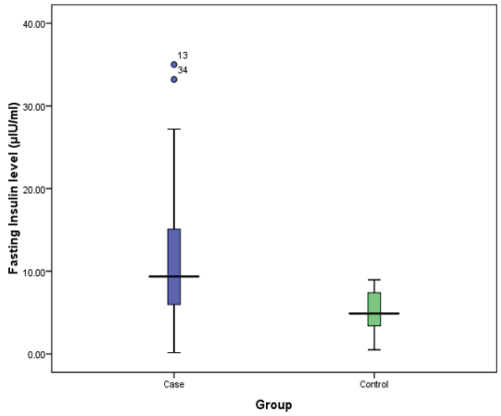

Figure 1. Distribution of patients by serum insulin level

Distribution of patients by fasting plasma glucose level:

The mean fasting plasma glucose level (mg/dl) was 130.54 ± 41.06 (range, 75.0-195.0) in non-alcoholic fatty liver disease and was 87.94 ± 17.38 (range, 66.0-161.0) in control subjects. The mean serum glucose level of the non-alcoholic fatty liver disease was significantly higher compared to control subjects (t=6.810; p<0.001) (Figure-2).

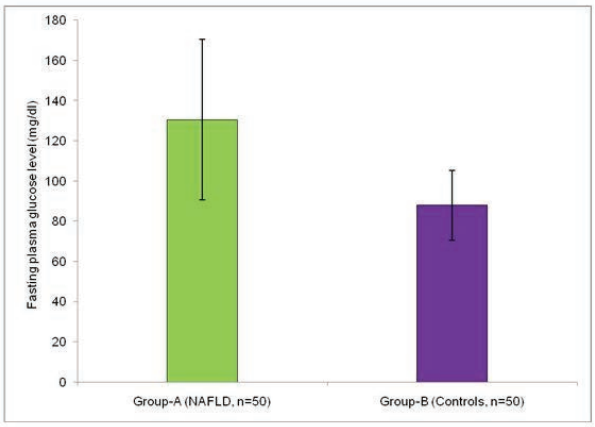

Figure 2. Distribution of patients by fasting plasma glucose level

* unpaired 't' test was employed to analyze the data.

Comparison of HOMA and insulin resistance between NAFLD and control subjects:

The mean HOMA level (mg/dl) was 3.29 ± 1.97 (range, 0.45-7.65) in non-alcoholic fatty liver disease and was 1.07 ± 0.40 (range, 0.38-1.95) in control subjects. The mean HOMA level of the non-alcoholic fatty liver disease was significantly higher compared to control subjects (t=7.803; p<0.001).

The frequency of insulin resistance and sensitive subjects were 56.0% and 44.0% in the group of NAFLD whereas none was insulin resistance and all were insulin sensitive in the without NAFLD group. Insulin resistance was significantly more in non-alcoholic fatty liver disease compared to control subjects (χ 2=38.889; p<0.001).

Table II. Comparison of HOMA and insulin resistance between NAFLD and control subjects

Parameters	Case (n=50)	Control (n=50)	Test value	p-value
HOMA	3.29 ± 1.97	1.07 ± 0.40	t=7.803	*p<0.001
Insulin				
Resistance	28 (56.0%)	0 (0.0%)	p=38.889	p<0.001
Sensitive	22 (44.0%)	50 (100.0%)		

*unpaired t test and Chi-square ($\chi 2$) Test were employed to analyze the data.

Discussion

Nonalcoholic fatty liver disease (NAFLD) representing a range of conditions from simple steatosis, nonalcoholic steatohepatitis to cirrhosis, is the most prevalent liver disease worldwide. NAFLD has been received as a major health compulsion and the prevalence is increasing year by year. In western countries, NAFLD has also become one of the most liver illnesses, affecting 20% to 40% of the general population. NAFLD is considered as a multifactorial chronic disease that is associated with genetic, environmental, and metabolic factors (12).

This study revealed that the mean BMI was 26.08 ± 1.41 kg/m^2 in non-alcoholic fatty liver disease and was 23.89 ± 2.31 kg/m^2 in control subjects. The mean BMI of non-alcoholic fatty liver disease was significantly higher than that of control subjects (p<0.001). This result was consistent with the study of Mohan et al., (2014) (13) where they observed the he mean BMI (kg/m^2) for NAFLD was 27.01 ± 3.53 and for controls (no fatty liver) was 23.91 ± 3.11. Elevated BMI was seen in NAFLD with a statistical significance (p < 0.05). This result was consistent with the study of Li et al., (2009) that body mass index (kg/m^2) was 25.9 ± 2.8 in non-alcoholic fatty liver disease and 22.5 ± 2.8 in control subjects; difference was significant (p<0.001) (14).

NAFLD subjects in the Bangladeshi population have previously been shown to have insulin resistance; however, the underlying causes of this defect are high BMI and hyperuricemia. In this settings, we found significantly higher (p<0.001) levels of HOMA in NAFLD subjects (3.29 ± 1.97) compared to the controls (1.07 ± 0.40). This result was consistent with the study of Hossain et al., (2018)(15) where they found that HOMA level was significantly higher (p=0.001) in NAFLD subjects (2.21 ± 1.01) compared to the controls (1.79 ± 0.51). This observation was also in agreement with a number of studies (16,17).

Conclusion

This study revealed that the mean BMI, serum insulin level, HOMA and insulin resistance were significantly higher in non-alcoholic fatty liver disease compared to control subjects. From the findings of this study, it may be concluded that high BMI has strong association in non-alcoholic fatty liver disease.

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