PREVALENCE OF METABOLIC RISK FACTORS IN NON-ALCOHOLIC FATTY LIVER DISEASE

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ABSTRACT

Objective: To determine the frequency of factors leading to metabolic syndrome among non-alcoholic fatty liver disease (NAFLD) patients at a tertiary care hospital.

Study Design: Descriptive cross sectional study.

Place and Duration of Study: Department of Medicine, Combined Military Hospital, Kharian. Study was carried out over a period of six months from Jan 2015 to Jun 2015.

Material and Methods: A total of 110 patients were included in this study. Past history was taken to rule out alcohol intake, viral and drug induced etiology, to determine the presence of co-morbidities like obesity, type 2 diabetes mellitus, arterial hypertension and dyslipidemia. Physical examination was carried to determine the arterial blood pressure and to determine anthropometric data that is weight, height, body mass index (BMI) and abdominal obesity by measuring waist circumference.

Results: Mean age of the patients was 49.95 ± 8.86 years. There were 72 male patients (65.5%) while 38 (34.5%) patients were female. Different metabolic factors were central obesity in 82 patients (74.5%), raised high density lipoprotein (HDL) in 19 patients (17.3%), raised cholesterol in 87 patients (79.1%), raised blood pressure in 65 patients (59.1%) and raised fasting plasma glucose in 82 patients (74.5%). Mean BMI was 26.31 kg / m^2 ± 2.68, mean waist circumference was 109.82 cm ± 18.41, mean cholesterol was 237.50 ± 48.47mg / dl, mean systolic blood pressure was 148.88mmHg ± 22.10, mean diastolic blood pressure was 90.41mmHg ± 12.25 and mean fasting plasma glucose was 113.28mg / dl ± 22.80. Stratification with regard to age was carried out.

Conclusion: A considerable number of patients with NAFLD had metabolic syndrome. There was a close correlation between NAFLD and metabolic syndrome.

Keywords: IDF criteria, Metabolic syndrome, Non-alcoholic fatty liver disease.

INTRODUCTION

Non alcoholic fatty liver disease (NAFLD) is defined as the deposition of lipid, especially triglycerides (5-10%) in hepatocytes exceeding 5% of total liver weight in the absence of other etiologies of hepatic damage including viruses, alcohol consumption and metabolic diseases[1]. It is increasingly recognized as an important public health problem nowadays. The prevalence of NAFLD in the general population of Western countries is 20-30% and 5-40% in Asian countries[2,3]. Recently a hospital based study in Pakistan has shown a frequency of NAFLD approximately 14%[4].

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NAFLD consists of a wide spectrum of conditions ranging from simple steatosis to non alcoholic steatohepatitis (NASH) which can progress to cirrhosis and hepatocellular carcinoma (HCC). It is reported that almost 10% to 20% of individuals with NAFLD have NASH and 10% to 15% of individuals with NASH progress to cirrhosis[5]. In patients with cirrhotic NASH, HCC and liver failure are the main causes of morbidity and mortality[6].

Available data from clinical, experimental and epidemiological studies describe NAFLD as the hepatic manifestation of metabolic syndrome[7]. The prevalence of hypertension (34.1%), raised fasting plasma glucose (44.3%), raised cholesterol (15.7%), and obesity (60.2%) was also significantly higher in-patients with NAFLD[1]. Approximately 90% of patients with

NAFLD have more than one characteristic feature of metabolic syndrome while about 33% have the complete diagnosis[8]. The number of metabolic syndrome components significantly predicts the development of NAFLD. With only one component of metabolic syndrome, the risk of NAFLD was increased by 2.6 fold[9].

This study aims to assess the frequency of metabolic risk factors like obesity, hypertension, dyslipidemia, and diabetes, based on predefined international diabetes federation (IDF) criteria among adults in NAFLD patients. There is paucity of local studies on the relevant subject and this study will help in early diagnosis and prevention of NAFLD by targeting metabolic risk factors.

Table-I: Stratification for age with regard to central obesity.

Age (Year)	Central obesity		Total
	Yes	No	
25-45	19	13	32
46-60	63	15	78
Total	82	28	110

Chi square = 5.473, *p*-value=0.019.

Table-II: Stratification for age with regard to raised HDL.

Age (Year)	Raised HDL		Total
	Yes	No	
25-45	6	26	32
46-60	13	65	78
Total	19	91	110

Chi square=0.069, *p*-value=0.793

Table-III: Stratification for age with regard to raised cholesterol.

Age (Year)	Raised cholesterol		Total
	Yes	No	
25-45	24	8	32
46-60	63	15	78
Total	87	23	110

Chi square=0.457, *p*-value=0.499

SUBJECTS AND METHODS

A descriptive cross sectional study was conducted at Department of Medicine, Combined Military Hospital, Kharian, over a period of six months from January 2015 to June 2015. Sample size was calculated by using WHO calculator 95% confidence level, population proportion of 15.7% and precision 7%. The sample size turned out 110. Non-probability consecutive sampling technique was used. All indoor and outdoor patients having age between 25 to 60 years diagnosed with NAFLD by ultrasonography at Radiology Department of the study hospital were included in the study. Patients with pregnancy, alcoholic liver disease (alcohol intake daily doses >40 g for men and 20 g for women), known cases of Hepatitis (B and/or C) and autoimmune hepatitis were excluded from the study. Study was started after approval from ethical review committee of the institute. All data were collected in the proforma. All the patients who fulfilled the inclusion criteria were included on the study. Informed consent was taken from the subjects. Basic information regarding demography, past history, physical examination, biochemical parameters and ultrasound abdomen findings were collected through pre-designed proforma. The laboratory investigations were performed including plasma glucose levels, lipid profile and ultrasound findings confirmed by radiologist. Past history was asked to determine the presence of comorbidities like obesity, type 2 diabetes, arterial hypertension and dyslipidemia. Physical

examination was done to determine the arterial blood pressure and to determine anthropometric data including weight, height, BMI, abdominal obesity through waist circumference (at the level of umbilicus with standing posture). All data were analyzed using SPSS software (SPSS version 17.0). Descriptive statistics was used for quantitative and qualitative variables. Qualitative variables like diabetes, hypertension, obesity were measured as frequencies and percentages. For quantitative variables like age, BMI and serum cholesterol, mean and SD was calculated. Effect modifiers like age, gender were controlled by stratification. Post stratification chi-square test was applied. A *p*-value <0.05 was taken as significant.

RESULTS

A total of 110 patients were included in this study. Mean age of the patients was 49.95 ± 8.86 years. There were 72 male patients (65.5%) while 38 (34.5%) patients were females.

In this study, central obesity seen in 82 patients (74.5%), raised low density lipoproteins in 19 patients (17.3%), raised cholesterol in 87 patients (79.1%), raised blood pressure in 65 patients (59.1%) and raised fasting plasma glucose in 82 patients (74.5%).

Mean BMI was 26.31 Kg/m^2 ± 2.68, mean waist circumference 109.82 cm ± 18.41, mean cholesterol was 237.50 mg/dl ± 48.47, mean systolic blood pressure was 148.88 mmHg ± 22.10, mean diastolic blood pressure was 90.41 mmHg ± 12.25 and mean fasting plasma glucose was 113.28mg/dl ± 22.80. Stratification with regard to age is presented in tables-I to V.

Table-IV: Stratification for age with regard to raised blood pressure.

Age (Year)	Raised blood pressure		Total
	Yes	No	
25-45	13	19	32
46-60	52	26	78
Total	65	45	110

Chi square=6.366, *p*-value=0.012

Table-V: Stratification for age with regard to fasting serum glucose.

Age (Year)	Raised fasting serum glucose		Total
	Yes	No	
25-45	20	12	32
46-60	62	16	78
Total	82	28	110

Chi square=3.451, *p*-value=0.063

DISCUSSION

Sedentary lifestyle and poor dietary habits are contributing to a weight gain that is more epidemic in Western society. Recent epidemiological studies suggest an increased risk of cardiovascular disease (CVD) and type-2 diabetes in overweight and obese individuals. Unfortunately, incidence of the metabolic syndrome and NAFLD, which can precede the development of CVD and type-2 diabetes, are also increasing.

The metabolic syndrome, a cluster of metabolic abnormalities with abdominal adiposity and insulin resistance as its central components, affects approximately 25% of the American adult population[10] and is associated with an increased risk of CVD and type-2 diabetes[11]. The same findings are noted in our study regarding obesity and raised plasma glucose levels and are comparable with the study done in American population. It is estimated that about 30% of the general US population has excessive fat accumulation in the liver[12], reaching levels as high as 75%-100% in obese and morbidly obese individuals[13].

Approximately 90% of patients with NAFLD have more than one characteristic feature of metabolic syndrome and about 33% have the complete diagnosis[14], placing NAFLD as the hepatic representation of the metabolic syndrome[15]. In addition, presence of the metabolic syndrome predicts higher risk for the development of NAFLD in both men and women[16]. Risk for development of NAFLD in association with metabolic syndrome is comparable to above mentioned study in our set up. The majority of individuals with NAFLD have no symptoms with a normal physical examination; however, about 2%-6% of adult Americans and 20% of those who are obese may develop steatosis with inflammation, fibrosis, and cirrhosis[17]. Furthermore, there appears to be a close link between the metabolic syndrome, low grade inflammation, and oxidative stress[18].

Although an association between different metabolic abnormalities had been noted for several years, the metabolic syndrome was first publicly described in 1988 by Reaven[19]. Then called Syndrome X, the metabolic syndrome consisted of a cluster of metabolic abnormalities, including obesity (especially abdominal obesity), insulin resistance, impaired glucose metabolism, dyslipidemia, and elevated blood pressure[19]. The current definition of the metabolic syndrome varies depending on the position of different regulating bodies[20].

The metabolic syndrome, in part through glucose intolerance and insulin resistance, is strongly associated with steatosis, fibrosis, and cirrhosis of the liver in severely obese adults[21]. In addition, central fat distribution, fatty liver, and glucose intolerance are noted in mildly obese and in normal weight subjects[22]. Further, numerous studies have demonstrated that obesity, type 2 diabetes, dyslipidemia, hypertension, and insulin resistance are strongly associated with NAFLD[23]. NAFLD also is strongly associated with hepatic, adipose tissue, and whole body reductions in insulin sensitivity, increased rate of gluconeogenesis, impaired insulin response to suppress gluconeogenesis, and impaired fatty acid oxidation[24]. However, the question about whether hepatic insulin resistance is a cause or a consequence of hepatic steatosis is unresolved[25].

To summarize, the metabolic factors noted in our study including central obesity, raised HDL, raised cholesterol, raised blood pressure and raised fasting plasma glucose level are comparable with the findings of majority of the above mentioned studies.

CONCLUSION

A considerable number of patients with NAFLD had metabolic syndrome. There was a close correlation between NAFLD and metabolic syndrome

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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