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METABOLIC SYNDROME AND ITS DETERMINANTS IN NON-ALCOHOLIC FATTY LIVER DISEASE PATIENTS: A TERTIARY CARE HOSPITAL ANALYSIS

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Abstract

OBJECTIVE

To investigate the prevalence of metabolic syndrome and its components in NAFLD patients attending a tertiary care centre and to identify the determinants of metabolic syndrome in these patients.

METHODOLOGY

This descriptive cross-sectional study was carried out at Dr. Ruth K.M. Pfau Civil Hospital, Karachi on NAFLD patients (20–60 years old) for duration of ≥6 months. Exclusion criteria were chronic liver disease, autoimmune hepatitis, heavy alcohol users, renal failure and the use of hepatotoxic drugs. Metabolic syndrome was defined according to the NCEP-ATP III criteria. Statistical analyses were conducted using SPSS 26, and $p \leq 0.05$ was defined as the criterion for statistical significance.

RESULTS

The average age of study participants was 43.51 ± 11.58 years. Among the 192 patients, 102(53.1%) were males and 90 (46.9%) were females. Metabolic syndrome (Mets) was reported in 105 (54.68%) patients. The determinants significantly associated (p=0.0001); with high blood pressure (61%), raised blood glucose (62.9%), increased waist circumference (81%), raised serum triglycerides level (78.1%), and low HDL level (88.6%).

CONCLUSION

The most important determinants of Mets, which are known to be highly prevalent even in NAFLD patients, are also confirmed in this study, namely, high blood pressure and increased waist circumference, in addition to hyperglycemia and low HDL levels. The need for early screening, lifestyle change and preemptive management strategies to advance prevention to delay disease onset and associated metabolic complications are emphasized by these findings.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is defined as preferable excess fat in hepatocytes in the absence of substantial ingestion of alcoholic beverages and is actually a continuous liver illness. it is the most prevalent chronic liver disease worldwide, affecting almost one in four individuals and placing a significant economic burden on health systems ([1]. Metabolic syndrome (Mets) consisting of obesity, insulin resistance, hypertension and dyslipidemia is closely related to NAFLD. NAFLD can present a spectrum from simple steatosis (non-progressive form) to non-alcoholic steatohepatitis (NASH) in the form of fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) [2]. Considering the high rate of overlap between the 2 conditions—most patients with NASH also being diagnosed with metabolic syndrome—this indicates a putative metabolic cross talk between the 2 disorders). The existence of a bidirectional epidemiological link between Mets and NAFLD establishes robust clinical links between both pathways, with the strongest link being that patients with metabolic syndrome are at increased risk to develop NAFLD, and it is subsequently predicts the development of metabolic syndrome. In addition, NAFLD is thought to victimise 20%–30% of the study population [3, 4]. The mechanisms by which these factors predispose NAFLD patients to develop metabolic syndrome are not complete comprehended since the high prevalence of metabolic syndrome in NAFLD patients is not only due to unhealthy diet or nutritional discordancy [1] but also closely related to the sedentary life style. Individually conducted studies subsequently combined estimates produced between 50–67% prevalence [5].

Furthermore, several studies reporting on samples of similarly varied demographic make-up have found prevalence rates of metabolic syndrome of 57.6% and 63.84% [7, 8]. These results reinforce the basis of the association between Mets with NAFLD and add novel data on the complex association between these two tightly interrelated human health problems.

Mets is a combination of closely related metabolic derangements [abdominal obesity, insulin resistance, dyslipidemia and hypertension] become one of the most important threats for cardiovascular diseases and type 2 diabetes [5–8]. Comorbidity of metabolic syndrome and NAFLD is common and clearly deserves additional explanation regarding converging pathophysiological pathways as well as combined worsening of natural history of disease progression [9]. Previous studies identify the related factors of metabolic syndrome, in which prevalence of dyslipidemia (60.2%), type II diabetes mellitus (45.7%), abdominal obesity (56.74%), hypertension (38.5%) and insulin resistance (46.0%) [7,10,11].

This indicates the factors explaining the relation of metabolic syndrome with NAFLD in its complexity and clarifies major substances having a role in its coincidence and symptom progression. Mets is closely associated with NAFLD), a likely chronic and progressive hepatic disease with a high threat of some diseases. And NASH incidence also increases with the degree of obesity [11,12]. Obesity-related insulin resistance measured as either continuous variable or by dichotomizing BMI, is universally unmasked with respect to NASH independent of obesity, implicating, again, an integral role in NASH pathogenesis [10-13].

Metabolic syndrome (MetS), a prerequisite for NAFLD to become a public health issue, is a cluster of syndromes associated with metabolic disorders, including obesity, hypertension, insulin resistance, and dyslipidemia [14]. It is not only the risk factor for the development of NAFLD but is also considered a significant risk factor for the incidence of cardiovascular and diabetic complications. This tight association is also aggravated by the scarcity of extensive studies evaluating the incidence and determinants of Mets in the local population of NAFLD patients. In addition, if the diagnosis of metabolic syndrome and other risk factors in NAFLD subjects is the necessary burden for early diagnosis and treatment that enables an earlier intervention by physicians, then possibly, such risk factors should be viewed as advantageous as they serve to rescue morbidity and mortality.

METHODOLOGY

This was a cross-sectional, descriptive study, performed in the department of General Medicine of Dr. Ruth K. M. Pfau Civil Hospital, Karachi. The study included 192 consecutive male and female

participants aged 20 to 60 years old, diagnosed with NAFLD after a clinical assessment, performed at least six months ago from the date of the present study. Those participants with chronic liver disease, autoimmune hepatitis, high alcohol consumption, liver or renal failure, taking hepatotoxic drugs in the past or hemochromatosis were excluded from the study. The written informed consent was taken from all the eligible patients before inclusion in the study. NAFLD diagnosis was ascertained by ultrasound imaging for hepatic steatosis detection, whereby the maximum cut-off alcohol intake limit considered was 20 g/day for females and 30 g/day for males. Patients presented with a range of clinical symptoms including fatigue and abdominal pain, while others were asymptomatic. A significant share of subjects had elevated liver enzymes, and non-neoplastic causes of hepatic steatosis were methodically excluded after extensive clinical evaluation.

According to the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III), at least three of the five following criteria must be met in Mets: waist circumference (WC) of ≥102 cm (men) or ≥88 cm (women); triglyceride level ≥150 mg/dL; high density lipoprotein (HDL) cholesterol level <40 mg/dL (men) or <50 mg/dL (women); blood pressure ≥130/85 mmHg; and fasting glucose level ≥100 mg/dL. Insulin resistance was assessed by the homeostasis model assessment of insulin resistance (HOMA-IR), which was derived from the level of fasting glucose and insulin. Doppler ultrasound had been used to confirm hepatic steatosis with other liver disease exclusion. Waist circumference was assessed in standing patients with a flexible tape at the mid-point between the lower rib margin and the iliac crest.

Blood samples were processed at the hospital's laboratory and baseline metabolic parameters were analyzed and metabolic syndrome was diagnosed as per the NCEP-ATP III guidelines.

The collected data was analyzed through SPSS version 26. Mean ± SD and frequency and percentage were calculated quantitative and qualitative variable respectively. The Chi-square test was used to assess statistical significance; p-values of ≤0.05 were statistically significant.

RESULTS

The sample included 192 study participants, with a mean age of 43.51 ± 11.58 years (95% CI: 41.86–45.15) and a mean BMI of 25.87 ± 3.85 kg/m² (95% CI: 25.32–26.42). Systolic blood pressure 128.31 ±18.65 mmHg (95% CI: 125.65–130.96) and diastolic blood pressure 83.33 ± 11.45 (95% CI: 81.70–84.96). The mean waist circumference was 83.37 ± 5.64 cm (95% CI: 82.57–84.17). The mean blood sugar levels were 118.84 ± 29.05 mg/dL (95% CI: 114.70–122.97). Lipid profile testing revealed a mean total cholesterol of 201.88±37.42 mg/dL (95% CI: 196.55–207.21), mean triglycerides of 195.95±80.03 mg/dL (95% CI: 184.56–207.35), mean HDL of 45.08±6.10 mg/dL (95% CI: 44.21–45.95), and mean LDL of 118.86±23.74 mg/dL (95% CI: 115.48–122.24). Among the participants, 102 (53.1%) were male and 90 (46.9%) were female. Regarding fatty liver severity, 63.0% (n=121) had Grade I fatty liver, 32.8% (n=63) had Grade II, and 4.2% (n=8) had Grade III, as shown in Table I.

A comparison of patient characteristics with and without metabolic syndrome (MetS) showed no significant difference in mean age (43.69 ± 11.62 vs. 43.29 ± 11.60 years, p = 0.813) or gender distribution (51.4% males in the MetS group vs. 55.2% in the non-MetS group, p = 0.605). However, there was a significant association between fatty liver severity and metabolic syndrome (p = 0.036), with Grade III fatty liver more prevalent in the MetS group (6.7% vs. 1.1%). The prevalence of high blood pressure (61.0% vs. 26.4%, p = 0.0001), raised blood glucose (62.9% vs. 25.3%, p = 0.0001), increased waist circumference (81.0% vs. 40.2%, p = 0.0001), elevated serum triglycerides (78.1% vs. 43.7%, p = 0.0001), and low HDL levels (88.6% vs. 44.8%, p = 0.0001) was significantly higher in the MetS group, as presented in Table II.

DISCUSSION

This study aimed to assess the prevalence of metabolic syndrome (MetS) and its determinants in non-alcoholic fatty liver disease (NAFLD) patients. As the result of our study showed that 54.68% of NAFLD subjects had metabolic syndrome, it is not far away with the previous study. Paudel et al. found closely similar prevalence (57.6%) [7] and Fattahi et al. reported a greater prevalence of 65.9% [10].

Whereas a investigation via Gaharwar et al. had a lower prevalence of 51.4%, which is closer the value reported here [20]. These variations could be explained by dissimilarities in genetic predisposition, lifestyle factors, and population characteristics.

In our findings, high blood pressure (61%), raised blood glucose (62.9%), increased waist circumference (81%), raised serum triglycerides (78.1%) and low HDL levels (88.6%) were significantly associated with MetS ($p = 0.0001$). These results parallel other studies. Paudel et al. had a lower prevalence for high blood pressure (51.1%), raised blood glucose (45.7%), waist circumference (67%), raised serum triglycerides (60.2%) and low HDL (62%) [7]. Similarly, Gaharwar et al. stated raised blood glucose (63.8%), increased waist circumference (77.77%), elevated triglycerides (86.1%) and low HDL (94.44%), and met the criteria of metabolic syndrome significantly [20].

NAFLD is tightly linked to metabolic syndrome as previously mentioned. MetS is now regarded as the metabolic nature behind hepatic manifestation of NAFLD, as they share pathophysiological mechanisms, including insulin resistance, obesity, and dyslipidemia [14]. This is in line with findings from other studies where waist circumference was the most common determinant (81%) [18]. An increase in visceral fat deposition is a significant contributor to insulin resistance, which can further promote metabolic dysfunction and hepatic steatosis. Moreover, these metabolic disorders causing inflammation and fibrosis in the liver, and systemic hypertension and dyslipidemia may worsen hepatic inflammation and fibrosis, hence cellular damage and accelerate the progression of NAFLD.

According to recent data, NAFLD may actively contribute to the pathophysiology of metabolic syndrome rather than only being a result of it. Studies indicate that NAFLD itself can induce insulin resistance, worsen lipid metabolism, and increase systemic inflammation, perpetuating the cycle of metabolic dysfunction [15,16]. Moreover, genetic and epigenetic factors may modulate the bidirectional relationship between NAFLD and MetS [17-19]. Future studies should examine these parameters' involvement in the course of the illness, since they were not examined in our study.

This study's emphasis on a well-defined NAFLD cohort, which enables a thorough assessment of MetS prevalence and its drivers, is one of its advantages. Our findings provide region-specific data that can guide clinical interventions and public health strategies for managing NAFLD and its associated metabolic complications.

This research does have several drawbacks, however. Because it is a cross-sectional research, it cannot prove a link between NAFLD and metabolic syndrome. Additionally, since this was a hospital-based study, the findings may not be generalizable to the broader population. Furthermore, lifestyle and dietary factors were not assessed, which could have influenced metabolic syndrome development. Future longitudinal studies should examine these factors, along with genetic predispositions and targeted therapeutic interventions, to better understand the complex interplay between NAFLD and MetS.

CONCLUSION

The most important determinants of metabolic syndrome, which is known to be highly prevalent even in NAFLD patients, are also confirmed in this study, namely, high blood pressure and increased waist circumference, in addition to hyperglycemia and low HDL levels. The need for early screening, lifestyle change and preemptive management strategies to advance prevention to delay disease onset and associated metabolic complications are emphasized by these findings.

Table I: Characteristics of Study Participants (n=192)	
Mean ± SD	**95% CI**
Age in years= 43.51 ± 11.58	41.86---45.15
Body Mass Index in kg/m^2 = 25.87 ± 3.85	25.32---26.42
Systolic Blood Pressure in mmHg = 128.31 ± 18.65	125.65---130.96
Diastolic Blood Pressure in mmHg = 83.33 ± 11.45	81.70---84.96

Waist Circumference in cm= 83.37 ± 5.64	82.57–84.17
Fasting Blood Sugar in mg/dl = 118.84 ± 29.05	114.70–122.97
Total Cholesterol in mg/dl = 201.88 ± 37.42	196.55–207.21
Serum Triglycerides in mg/dl = 195.95 ± 80.03	184.56–207.35
High-Density Lipoprotein in mg/dl = 45.08 ± 6.10	44.21–45.95
Low-Density Lipoprotein in mg/dl = 118.86 ± 23.74	115.48–122.24
Frequency, n (%)	
Gender	
Male	102 (53.1)
Female	90 (46.9)
Grade of Fatty Liver	
I	121 (63.0)
II	63 (32.8)
III	8 (4.2)

Table II: Comparison of Patient Characteristics with and without Metabolic Syndrome (n=192)

Clinical and Metabolic Parameter		Metabolic Syndrome			P-Value
		Yes (n=105)	No (n=87)	95% C. I	
Age in years, Mean ± SD		43.69 ± 11.62	43.29 ± 11.60	-2.924–3.721	0.813
Gender, *n* (%)	Male	54 (51.4)	48 (55.2)	0.486–1.521	0.605
	Female	51 (48.6)	39 (44.8)		
Grade of Fatty Liver, *n* (%)	I	70 (66.7)	51 (58.6)	0.657–1.777	0.036
	II	28 (26.7)	35 (40.2)		
	III	7 (6.7)	1 (1.1)		
High Blood Pressure, *n* (%)	Yes	64 (61.0)	23 (26.4)	2.343–8.051	0.0001
	No	41 (39.0)	64 (73.6)		
Raised Blood Glucose, *n* (%)	Yes	66 (62.9)	22 (25.3)	2.677–9.340	0.0001
	No	39 (37.1)	65 (74.7)		
Increased Waist	Yes	85 (81.0)	35 (40.2)	3.300–12.080	0.0001

Circumference, *n* (%)	No	20 (19.0)	52 (59.8)		
Raised Serum Triglycerides Level, *n* (%)	Yes	82 (78.1)	38 (43.7)	2.455–8.608	0.0001
	No	23 (21.9)	49 (56.3)		
Low HDL Level, *n* (%)	Yes	93 (88.6)	39 (44.8)	4.575–19.889	0.0001
	No	12 (11.4)	48 (55.2)		

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