Original Research Article

Prevalence of non-alcoholic fatty liver disease (NAFLD) in type 2 diabetic patients in correlation with coronary artery disease

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Abstract

Introduction: Non-alcoholic fatty liver disease (NAFLD) is a condition characterized by excess accumulation of fat in the liver occurring in people who consume little or no alcohol. This study was aimed at estimating the prevalence of fatty liver in type 2 diabetic patients using ultrasonography and to also to study the correlation between coronary risk factors, coronary artery disease and fatty liver.

Materials and methods: This was a cross-sectional study conducted on 150 patients above the age group of 30yrs who were already diagnosed of type 2 diabetes mellitus and on treatment for T2DM were recruited according to inclusion criteria

Results: In the present study it was observed that 54 % of the diabetic patients had fatty liver and 46 % had no fatty liver disease on ultrasonography. Out of the 81 diabetic patients who had fatty liver, grade I fatty liver or mild steatosis was observed in 69.1 % cases while 44.9% of cases with no fatty liver disease and 49.4% of cases with non-alcoholic fatty liver disease and were in the age group of 51- 60 yrs with male predilection. 88 patients out of 150 were hypertensive, in patients with no fatty liver disease 59.4 % patients presented with hypertension and 58% hypertensive patients with non-alcoholic fatty liver disease. Mean BMI, waist circumference, HBA1c and liver enzymes (AST and ALT) were significantly higher in patients with fatty liver than in patients without fatty liver. The mean total cholesterol levels (178.18), the mean triglyceride levels (154.9) and mean LDL cholesterol (103.8) were significantly higher in patients with fatty liver when compared to those without fatty liver. Mean HDL cholesterol in patients with fatty liver was 43.3 which were significantly lower than

mean HDL cholesterol of 46.21 in patients without fatty liver. The prevalence of metabolic syndrome (66.7%) and CAD (59.3%) were significantly higher in the NAFLD subgroup.
Conclusion: The prevalence of NAFLD in type 2 diabetics is very high. NAFLD is associated with higher prevalence of metabolic syndrome and coronary artery disease. There is clustering of traditional coronary risk factors in patients with NAFLD.

Key words

Non-alcoholic fatty liver disease, Type 2 diabetes, Metabolic syndrome.

Introduction

Non-alcoholic fatty liver disease (NAFLD) is a condition characterized by excess accumulation of fat in the liver occurring in people who consume little or no alcohol. The amount of excess fat deposited in the liver in this condition usually exceeds 5-10% by weight and is recognized by accumulation of triglyceride within the cytoplasm [1]. NAFLD encompasses a wide spectrum of liver abnormalities ranging from steatosis that is Nonalcoholic fatty liver (NAFL) to non-alcoholic steatohepatitis (NASH),which can progress to end-stage liver disease (fibrosis to cirrhosis and possibly hepatocellular carcinoma), a reason why this entity long considered an incidental finding has received increasing attention [2].

NAFL is defined as the presence of hepatic steatosis with no evidence of hepatocellular injury in the form of ballooning of the hepatocytes or no evidence of fibrosis. The risk of progression to cirrhosis and liver failure is minimal. NASH is defined as presence of hepatic steatosis and inflammation with hepatocyte injury with or without fibrosis. This can progress to cirrhosis, liver failure and rarely liver cancer. NASH cirrhosis means the presence of cirrhosis with current or previous histological evidence of steatosis or steatohepatitis [2].

Although the mechanisms underlying liver disease progression remain unclear, insulin resistance and oxidative stress play an important role in NAFLD development and progression. Insulin resistance is almost a universal finding in patients with NAFLD and it is considered the hepatic manifestation of the metabolic syndrome which includes central obesity, hyperglycemia, low HDL (high-density lipoprotein), hypertension and hypertriglyceridemia. The incidence of NAFLD remains unknown because no prospective studies have been conducted. Although NAFLD has been reported worldwide, it is difficult to determine the true prevalence because of problems in interpreting data from various studies due to referral bias, population heterogeneity, study design, imaging modality used and use of liver biopsies [3]. Population-based studies provide better estimates of the prevalence of NAFLD in general population, but few such studies have been reported to date. Based on those studies the prevalence of NAFLD in the general population western countries is 15-30% [4].

In the last two decades, NAFLD has been increasingly recognized as the most common chronic liver disease in Western countries and an increasing indication for liver transplantation and a possible cause of hepatocellular carcinoma, but until recently was thought to be uncommon in the Asia-Pacific region because it was considered a disorder of affluence, and in this region the burden of viral hepatitis is huge. But rising trend is observed in developing countries as well because of the changes in the socio demographics and lifestyle with economic prosperities [5].

The prevalence of the major risk factors for NAFLD like type 2 diabetes mellitus, obesity, dyslipidemia and metabolic syndrome is increasing globally and the Asia-Pacific region is at forefront of the current pandemic. So the overall prevalence of NAFLD is also likely to increase progressively in the coming years. On

the basis of studies available till date the estimated prevalence of NAFLD in the general population across Asia using ultrasonography varies from 5-40% [6]. The community prevalence of NAFLD in India varies from 5%-28% [7]. Several studies done till date on NAFLD and Coronary artery disease(CAD) have shown that steatosis is associated with an increased prevalence and incidence of CAD and cardiovascular mortality [8]. Moreover the common cause of death in patients with NAFLD was CAD followed by extra hepatic malignancy and finally cirrhosis and its complications [9].

Both NAFLD and CAD share the classic common risk factors like age, physical inactivity, dyslipidemia, obesity, T2DM and hypertension. The new risk factors for CAD include markers for inflammation (e.g. C - reactive protein, lipoprotein A), homocystine and markers of fibrinolytic and homeostatic function (e.g. fibrinogen, tissue plasminogen activator and plasminogen activator inhibitor 1). These markers are also associated with NAFLD [10, 11].

NAFLD is a growing public health problem worldwide. The clinical impact of NAFLD on CAD risk deserves particular attention in view of the implications for screening and surveillance strategies in the growing number of NAFLD patients. NAFLD patients should not only be candidates for aggressive treatment of their liver disease, but also for aggressive treatment of underlying CAD risk factors, because many patients with NAFLD will have major CAD events and die prior to the development of advanced liver disease. This study was aimed at estimating the prevalence of fatty liver in type 2 diabetic patients using ultrasonography and to also to study the correlation between coronary risk factors, coronary artery disease and fatty liver.

Materials and methods

This was a cross-sectional study conducted in Osmania General Hospital, Hyderabad. The study period extended from November 2013 to July 2015.

A Total of 150 patients above the age group of 30yrs who were already diagnosed of type 2 diabetes mellitus and on treatment for T2DM were recruited from Osmania Medical College and hospital for the present study.

Inclusion criteria

Patients above the age of 30yrs who is already diagnosed as type 2 diabetes mellitus and on treatment for it and Patients on drugs like statins were included.

Exclusion criteria

Patients with significant alcohol consumption (> 21 drinks per week in men and > 14 drinks per week in women over a period of 2 years), Hepatitis B positive status, Hepatitis C positive status, Chronic liver disease due to any cause, Grossly deranged transaminases due to any cause (more than three times the upper limit of the normal), History of ingestion of hepatotoxic drugs like anti tubercular therapy, methotrexate, amiodarone and all other possible drugs on fibrates.

Consecutive type 2 diabetic patients were taken. Detailed history was taken regarding alcohol consumption and their medications were noted to see if they were on any hepatotoxic drugs. Hepatitis B and C status was evaluated. After the initial evaluation if patients were satisfying the exclusion criteria they were included in the study for further evaluation. Then a detailed history was taken regarding duration of type 2 diabetes and history of hypertension. The presence of coronary artery disease (CAD) was assessed from ECG changes, past history of CAD or any intervention that the patient has undergone like coronary angiogram or revascularization procedure like angioplasty. Their records were gone through for ECHO and TMT reports. IHD medication that patients were on was noted.

NAFLD if present was classified based on standard ultrasonographic criteria as Grade I fatty liver or mild steasosis, Grade II fatty liver or

moderate steasosis and Grade III fatty liver or severe steasosis.

Physical examination was done with particular emphasis on height, weight and waist circumference. Laboratory investigations included HbA1C, fasting lipid profile and serum aminotransferases.

Ultrasound abdomen was done for all the 150 patients to detect fatty changes in the liver, performed by single radiologist using high – resolution B mode ultrasonography system and if the fatty change was present, it was graded as mild, moderate or severe fatty liver by the radiologist.

Parameters like age, sex, hypertension, body mass index (BMI), waist circumference (WC), Glycosylated hemoglobin (HbA1c), fasting lipid profile (FLP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), presence or absence of CAD and metabolic syndrome were analyzed across the two groups (no fatty liver and fatty liver) to see if any correlation exists between them and the presence of fatty liver. And also in the CAD group, the above parameters were compared across fatty liver and no fatty liver group. While analyzing the fasting lipid profile among no fatty and fatty liver group, subjects who were on statins and fibrates were excluded.

Data analysis

The data obtained was analyzed using SPSS software version 20.0. Descriptive results are expressed as mean and SD of various parameters in different groups. Appropriate statistical tests were used to assess the significance of difference in mean values of different parameters in between groups. P value less than 0.05 was considered as significant and p value less than 0.001 was considered highly significant.

Results

In the present study, it was observed that 54 % of the diabetic patients had fatty liver and 46 % had no fatty liver disease on ultrasonography. Out of the 81 diabetic patients who had fatty liver, grade I fatty liver or mild steasosis was observed in 69.1 %cases, grade II fatty liver or moderate steasosis was observed in 27.2 %cases and grade III fatty liver or severe steasosis was observed in 3.7% cases (**Table – 1**).

Table - 1: Prevalence and grading of Fatty liver disease in study population.

Variable	Present	%
Fatty liver disease	81	54
No fatty liver disease	69	46
Grade I	56	69.1
Grade II	22	27.2
Grade III	3	3.7

In the present study, it was observed that 44.9% of cases with no fatty liver disease and 49.4% of cases with non alcoholic fatty liver disease and were in the age group of 51- 60 yrs. The mean age group in patients with no fatty liver disease was 51.15 yrs and in patients with non alcoholic fatty liver disease was 49.67 yrs there was no significant difference in the mean age between two groups $p>0.05$.

In the present study, it was observed that in patients with no fatty liver disease68.1 % were males, 31.9% were females compared to 70.4 % males and 29.6% females in patients with non alcoholic fatty liver disease. There was no significant difference in gender distribution observed in two groups $p >0.05$ (**Table – 2**).

The BMI in patients with and without fatty liver was further classified based on consensus guidelines for Asian Indians as underweight (<18 kg/m^2), normal (18 – 22.9 kg/m^2), overweight (23 – 24.9 kg/m^2), obese (> 25 kg/m^2). It was observed that 34. 8% patients with normal BMI had fatty liver disease, 75 % of patients with overweight BMI had fatty liver disease and 65.4% patients with obese BMI had fatty liver disease. In the present study 37.3 % cases presented with mild steatosis, 14.7% presented with moderate steatosis and 2 % cases presented

with severe steatosis. The present study significantly demonstrates the occurrence and correlation of fatty liver disease and its severity in higher BMI patients p<0.001 (**Table – 3**).

Table - 2: Demographic Distribution of subjects.

Age group in Years	**No Fatty liver**		**Fatty liver**	
	No.	**%**	**No.**	**%**
30– 40	10	14.5	9	11.1
41- 50	19	27.6	30	37
51- 60	31	44.9	40	49.4
61- 70	9	13	2	2.5
Total	**69**	**100**	**81**	**100**
Mean ± SD	**51.15 ± 9.08**		**49.67 ± 7.25**	
T value	**1.04**		**p = 0.29**	
Male	47	68.1	57	70.4
Female	22	31.9	24	29.6
Total	**69**	**100**	**81**	**100**
P2 value	**0.89**	**p value**		**0.45**

Table - 3: Grading FLD as per BMI.

BMI	**No fatty liver Disease**	**Fatty liver (No. of cases)**		
		Mild	**Moderate**	**Severe**
Normal	43	23	0	0
Overweight	8	24	0	0
Obese	18	9	22	3
P^2 **value**	74.76	**p value** <0.001		

In the present study, it was observed that the mean BMI in patients with CAD and fatty liver was 25.27 kg/m^2 which were significantly higher than mean BMI 21.86kg/m^2in patients with CAD and without fatty liver. The mean difference was statistically significant with p value = 0.001.Mean waist circumference in patients with CAD and fatty liver was 105.37 cm which was significantly higher than mean waist circumference 95.96 cm in patients with CAD and without fatty liver. The mean difference was statistically significant with p value = 0.001. The mean HbA1c in patients with CAD and fatty liver was 10.45 % which was significantly higher than mean HbA1c of 9.27 % in patients with CAD and without fatty liver, which indicates a significantly poor glycemic control in patients with CAD and fatty liver. The mean difference was statistically significant with p value 0.001 (**Table – 4**).

In the present study it was observed that the mean AST and ALT levels in patients with CAD and fatty liver were significantly higher than mean AST and ALT levels of 30.23 IU/L and 32.5 IU/L respectively in patients with CAD and without fatty liver suggesting a significantly increase in liver enzymes in patients with CAD and fatty liver. The mean difference was statistically significant with p value < 0.001 (**Table – 5**).

In the present study it was observed that the mean total cholesterol in patients with CAD and fatty liver was 188.1 compare to mean total cholesterol of 166.72 in patients with CAD and

without fatty liver there was no statistical significance in the mean cholesterol between 2 groups (p=0.07), mean triglycerides in patients with CAD and fatty liver was 168.87 which was significantly (p=0.006) higher than mean triglycerides of 149.07 in patients with CAD and without fatty liver, mean HDL cholesterol in patients with fatty liver and CAD was 43.5 which was significantly (p=0.011) lower than mean HDL cholesterol of 47.4 in patients with CAD and without fatty liver, mean LDL cholesterol in patients with fatty liver and CAD was 110.8 compared to mean LDL cholesterol of 99.1 in patients with CAD and without fatty liver there was no statistical significance in the mean LDL cholesterol between 2 groups (p=0.08), which indicates a significant higher levels of serum triglycerides and a lower protective HDL cholesterol in patients with CAD and fatty liver.

Table - 4: BMI, waist circumference, HBA1cand fatty liver disease in CAD patients.

Variable	**No Fatty liver**	**Fatty liver**
	Mean ± SD	**Mean ± SD**
BMI	23.38 ± 2.8	25.27 ± 2.45
t value	**3.73**	**p = 0.001**
Waist circumference	95.96 ± 13.53	105.37 ± 8.96
t value	**3.58**	**p = 0.001**
HBA1c	9.27 ± 1.34	10.45 ± 1.4
t value	**3.48**	**p = 0.001**

Table - 5: Liver enzymes and fatty liver disease in CAD patients.

Variable	**No Fatty liver**	**Fatty liver**	**T value**	**p value**
	Mean ± SD	**Mean ± SD**		
SGOT/AST	30.23 ± 7.68	55.33 ± 12.69	9.1	<0.001
SGPT/ALT	32.5± 8.3	61.89 ± 15.05	9.2	<0.001
Total Cholesterol	176.38 ± 20.21	188.1 ± 29.9	1.78	0.07
Triglycerides	149.07 ± 31.64	168.87 ± 27.34	2.81	0.006
HDL- Cholesterol	47.4 ± 7.18	43.5 ± 5.53	2.61	0.011
LDL- Cholesterol	99.1 ± 20.5	110.8 ± 29.84	1.77	0.08

There was a significant higher occurrence of CAD in patients with fatty liver compared to patients without fatty liver p =0.008 (**Table – 6**). Thus presence of CAD suggests significantly higher occurrence of fatty liver disease and its severity p<0.001. There was no statistically significant difference in distribution of hypertensive patients was observed among two groups p >0.05.

Discussion

Prevalence of non-alcoholic fatty liver disease in type 2Diabetics

Several studies have shown a positive relationship between abnormal glucose tolerance and NAFLD. In a study done by AK Agarwal [12] where 124 diabetic patients were evaluated for the presence of fatty liver, the prevalence was found to be 57.2%. The presence of fatty liver in this study was estimated using ultrasonography. A study done in Italy by Targher, et al. [13] where 2839 diabetic patients with available liver ultrasound data were studied, 1974 (69.5%) patients were detected to have NAFLD. The prevalence of fatty liver in type 2 diabetic patients in the current study as detected by ultrasound abdomen was 54%. Most of the patients in the present study had mild fatty liver.

This was comparable to the prevalence of fatty liver estimated in type 2 diabetic patients in other studies. Various other studies from India have shown the prevalence of NAFLD in diabetics to vary from 57% to 64% [14]. This suggests that fatty liver is very common in type 2 diabetic patients and ultrasound is the most common imaging modality used.

Table - 6: CAD, Hypertension, metabolic syndrome in fatty liver disease.

CAD	**No Fatty liver**		**Fatty liver**	
	No.	**%**	**No.**	**%**
Absent	43	62.3	33	40.7
Present	26	37.7	48	59.3
Total	**69**	**100**	**81**	**100**
P^2 **value**	**6.94**	**p value**		**0.008**
Hypertension				
Non HTN	9	34.6	11	22.9
HTN	17	65.4	37	77.1
Total	**26**	**100**	**48**	**100**
P^2 **value**	**1.17**	**p value**		**0.209**
Metabolic syndrome				
Absent	9	34.6	6	12.5
Present	17	65.4	42	87.5
Total	**69**	**100**	**81**	**100**
P^2 **value**	**5.1**	**p value**		**0.027**

Non-alcoholic fatty liver disease and age

There was no correlation between age and prevalence of fatty liver in our study and similar results were found in studies done by AK Agarwal, et al. [12] and Hui, et al. [15]. But in contrary to these studies, studies done by Targher, et al. [13], Frith, et al. [16] showed that prevalence of fatty liver increased with increasing age. The reason why the prevalence of NAFLD was more among the older age group as observed in these studies was that the risk factors for NAFLD such as hypertension, obesity, diabetes and hyperlipidemia were significantly more among older patients. It has also been observed that older age also increases the risk of developing related problems such as severe hepatic fibrosis, hepatocellular carcinoma, but contrary to this study done by Hui, et al. note no significant differences in age between individuals with progressive NAFLD and those who did not progress. The relationship between age, NAFLD and fibrosis remains unsettled. The association between age and high prevalence of NAFLD as well as the higher stage of fibrosis and cirrhosis may be related to the duration of the disease rather than to age itself.

Gender and non-alcoholic fatty liver disease

The prevalence of fatty liver was more among males (70.4%) when compared to females (29.6%) in the current study. In study by AK Agarwal, et al. [12] prevalence of NAFLD was marginally higher in men (58.1%) as compared to females (56%) and in study by Targher, et al. [13] also the prevalence of NAFL was 71.15% in men and 60.8% in women respectively. Role of gender in NAFLD is controversial. NAFLD was initially thought to be more common in women, but this notion lacks empirical support. Recent studies including studies from Asian countries show that men outnumber women in NAFLD prevalence. Female hormones seem to have a protective effect as the disease is more prevalent in post-menopausal when compared to pre-menopausal women [17].

Non-alcoholic fatty liver disease and hypertension

The prevalence of hypertension was more among fatty liver group in the present study. Similar results were also observed in the studies done by Ak Agarwal, et al. [12] and Targher, et al. [13]. Patients with fatty liver tend to have hypertension may be as a part of metabolic syndrome.

Non-alcoholic fatty liver disease and body mass index

The mean BMI in the present study was more among fatty liver group when compared to those without fatty liver and the difference in means between the two groups was statistically significant indicating the association between abnormal BMI and fatty liver. The NAFLD subgroup had higher prevalence of overweight and obesity (71.6%) when compared to non-NAFLD group (37.6%). Similar results were observed in the other studies done by Ak Agarwal, et al. [12] and Targher, et al. [13]. Obesity is associated with an increased risk of nonalcoholic fatty liver disease (NAFLD). Steatosis, the hallmark feature of NAFLD, occurs when the rate of hepatic fatty acid uptake from plasma and de novo fatty acid synthesis is greater than the rate of fatty acid oxidation and export (as triglyceride within very low-density lipoprotein). Therefore, an excessive amount of intrahepatic triglyceride (IHTG) represents an imbalance between complex interactions of metabolic events. In obese individuals, there is increased supply of FFA to the liver from the diet, adipose tissue and also through de novo lipogenesis. All these serve to promote hepatic steatosis. Obesity also predisposes to insulin resistance. There is decrease in whole body insulin sensitivity with effect particularly on hepatic, skeletal muscle and adipose tissue. Peripheral IR in the skeletal muscle affects a large portion of the total glucose uptake leading to hyperglycemia. In the adipose tissue, IR impairs the anti-lipolytic action of insulin leading to increased release of FFA. The mechanism behind this phenomenon is complex and is related to an excess of circulating FFA, an abundance of pro-inflammatory cytokines with a relative or absolute deficiency of anti-inflammatory cytokines, which is governed by the hormonally active adipose tissue. The role of obesity in the pathogenesis of NAFLD is supported by results of interventional trials that have shown that weight loss, physical exercise, reduction of sedentary life style and dietary changes improve hepatic steatosis and should be considered the first line therapy for the management of NAFLD [18, 19].

Non-alcoholic fatty liver disease and waist circumference

The mean waist circumference was more among the subjects with fatty liver in both sexes in the present study when compared to those without fatty liver. The difference in means between the groups was statistically significant indicating association between waist circumference and fatty liver. Similar observation was seen in study done by Ak Agarwal [12]et al also.

Non-alcoholic fatty liver disease and blood sugar

The mean HbA1C levels were more among fatty liver group when compared those without fatty liver. Ak Agarwal, et al. [12] and Targher, et al. [13] also have shown in their studies that the mean HbA1C levels were higher in patients with fatty liver than those without. Insulin resistance is the key factor in the development of hepatic steatosis due to the important action of insulin on skeletal muscle, adipocytes and the liver- all important organs in maintaining glucose and lipid homeostasis. Insulin resistance leads to hyperglycemia, impaired glucose tolerance and increased risk of developing type 2 diabetes mellitus. NAFLD is strongly associated with type 2 diabetes mellitus. Though type 2 diabetes, central obesity, dyslipidemia and hypertension are the risk factors for the development of fatty liver, NAFLD can however also precede the development of this comorbidities [20].

Non-alcoholic fatty liver disease and liver enzymes

The mean values of liver enzymes (AST and

ALT) were higher in NAFLD subgroup in studies done by AK Agarwal, et al. [12] and Targher, et al. [13] and this association was statistically significant. But contrary to these studies in our study though the median values of liver enzymes were higher among NAFLD subgroup, the association was not statistically significant. There are studies providing evidence that normal ALT levels provide little diagnostic or prognostic value when assessing patients for NAFLD, as in study done by Targher, et al. [13] more than four-fifths (86%) of the patients with NAFLD had normal ALT levels, Browning, et al. [4] 79% patients with hepatic steatosis had normal ALT levels. Therefore serum ALT levels appear to be insensitive markers for NAFLD. Indeed, it is known that the full histological spectrum of NAFLD may be present among patients with normal liver enzymes, which therefore cannot be reliably used to exclude the presence of more advanced stages of NAFLD. Studies which have taken liver enzymes to estimate the prevalence of fatty liver underestimated it because of the above reason. This might be another reason for the difference in results among the studies.

Non-alcoholic fatty liver disease and Hypercholesterolemia

The mean total cholesterol levels in the present study were higher in patients with fatty liver when compared to those without it and this difference in mean was statistically significant. Studies have shown varying results. In study by AK Agarwal, et al. [12] the difference in mean total cholesterol levels were not statistically significant among the two groups. Lipid abnormalities can be noted in up to 75% of patients with NAFLD [21]. Severe hypertriglyceridemia and mixed hyperlipidemia increase the risk of developing NAFLD by 5-6 times [21]. Patients with hypercholesterolemia less commonly develop NAFLD. Hepatic lipid homeostasis is disordered in NAFLD. However, whether dyslipidemia is a cause or a consequence of NAFLD is not clear.

Non-alcoholic fatty liver disease and Hypertriglyceridemia

The mean triglyceride levels were higher in patients with fatty liver when compared to those without fatty liver in the present study and the difference in means was statistically significant. AK Agarwal, et al. [12] and Targher, et al. [13] also had shown significant correlation between hypertriglyceridemia and NAFLD. Hepatic steatosis is a manifestation of excessive triglyceride accumulation in the liver and the fundamental defect is an imbalance between import and export of fat to and from the liver. This can occur from the excessive importation of free fatty acids form adipose tissue, from diminished hepatic export of FFA secondary to reduced synthesis or secretion of VLDL, or from impaired beta oxidation of FFA. The major sources of triglycerides are from fatty acids stored in adipose tissue and fatty acids newly made within in the liver through de novo synthesis [21]. The central mechanisms for all these metabolic alterations are insulin resistance (IR), which usually develops in the setting of an inappropriate diet, sedentary lifestyle, obesity etc.

Non-alcoholic fatty liver disease, HDL and LDL levels

The mean HDL cholesterol was significantly more among no fatty liver group in the present study, the current study also demonstrate correlation between LDL cholesterol and fatty liver. Studies have shown varying results [12, 13]. The exact difference in the results from study to study couldn't be explained.

Metabolic syndrome and non-alcoholic fatty liver Disease

The prevalence of metabolic syndrome was significantly higher in the NAFLD subgroup as compared to those who did not have NAFLD in the present study and this association was statistically significant. Similar observations were seen in studies by AK Agarwal, et al. [12] and Targher, et al. [13]. Currently NAFLD is considered an integral component of metabolic syndrome. It is considered as the hepatic manifestation of metabolic syndrome. NAFLD

and metabolic syndrome are two intertwined diseases sharing the same factor in their pathogenesis; insulin resistance [22]. The metabolic syndrome is a clustering of risk factors that greatly increases an individual's probability for developing atherosclerotic cardiovascular disease, type 2 diabetes mellitus and chronic kidney disease. The predominant underlying risk factors appear to be abdominal obesity, atherogenic dyslipidemia, hypertension, elevated plasma glucose. It is also considered a prothrombotic state and a proinflammatory state.

Non-alcoholic fatty liver disease and coronary artery Disease

The prevalence of CAD was more among fatty liver group (59.3%) than those without fatty liver (37.7%) which was significant statistically. In study by AK Agarwal, et al. [12] also the prevalence of CAD was more among NAFLD group (60.5%) when compared to non-NAFLD group (45.25%). Using multivariate logistic regression analysis it was found that NAFLD is an independent risk marker for CAD with significant p value. In study done by AK Agarwal, et al. [12] it was found that NAFLD was a significant independent predictor of CAD and in study by Targher, et al. [13] it was found that NAFLD was independently associated with CVD with significant p value in both the studies. Several epidemiological studies suggest that NAFLD role is not limited as a marker of CVD, but indicates its active involvement in its pathogenesis. AK Agarwal, et al. [12] and Targher, et al. [10] showed a significant increase of carotid intima-media thickness (IMT) in the presence of NAFLD. Lin, et al. [11] showed that patients with NAFLD were more likely to have CAD compared to patients without NAFLD, independent of obesity and other risk factors. Targher, et al. [8] showed an increased prevalence of CAD in patients with type 2diabetes mellitus and NAFLD as compared with diabetic patients without NAFLD.

Conclusion

The prevalence of NAFLD in type 2 diabetics is very high. NAFLD is associated with higher prevalence of metabolic syndrome and coronary artery disease. There is clustering of traditional coronary risk factors in patients with NAFLD. When patient is detected to have fatty liver the other components of metabolic syndrome like diabetes, obesity, dyslipidemia, hypertension should be looked in for as a majority of them have one or more components of metabolic syndrome.

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