Section: Medicine

Research

CLINICAL PROFILE OF NON- ALCOHOLIC FATTY LIVER DISEASE IN PATIENTS WITH TYPE 2 DIABETESMELLITUS

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Abstract

Background: Non- alcoholic fatty liver disease (NAFLD), which develops in the absence of alcohol abuse, NAFLD is the most common liver disease and the third leading indications for liver transplantation. Non- alcoholic fatty liver disease and type2 diabetes mellitus are common conditions that regularly co-exist and can act synergistically to drive adverse outcomes. The presence of both NAFLD and type2 DM increase the development of complications of diabetes as well as augmenting the risk of more severe NAFLD, including cirrhosis, hepatocellular carcinoma. This study is to estimate the proportion of non- alcoholic fatty liver disease in patients with type2 diabetes mellitus by ultrasonography. **Materials and Methods:** The study was a cross-sectional study done in 70 consecutive patients who were admitted in medical wards of department of General medicine, Government medical college, Kottayam. This study was carried for a period of 1 year. Clinical and biochemical parameters were recorded. NAFLD was diagnosed by ultrasonography. Information was collected through a proforma and data was analysed using SPSS. **Result:** The proportion of NAFLD is high (68.6%) amongst T2DM patients affecting 68.8% males and 31.3% females. Majority of study participants belonged to age group of 41-60 yrs. The youngest age is 41 years and eldest is 86 years with mean age of 56.77 yrs.8.5% of patients hepatomegaly, but no other clinical findings were observed. Majority of the study participants were overweight. 64.6% of the patients in NAFLD group had a BMI > 25 kg/m2 which showed that overweight in combination with T2DM increases the proportion of NAFLD. 91.6% of the patients in the NAFLD group had dyslipidemia with 43.8% of patients having hypertriglyceridemia. Alkaline Phosphatase levels were significantly high in NAFLD group as compared to normal group. **Conclusion:** NAFLD is a common association of type 2 diabetes mellitus. High proportion of NAFLD observed in the current study. The mainstay of NAFLD management, includes health education, diet control and avoid excessive weight gain, NAFLD should be examined among diabetic patients, especially those with abnormal BMI and HDL.

INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD), which develops in the absence of alcohol abuse (<20gm/d in men and <10 gm/d in women) has been recognized as a major health burden.[1] NAFLD is the most common liver disease and the third leading indication for liver transplantation.[2] The clinical implications of NAFLD are derived mostly from its common occurrence in the general population and its potential to progress to cirrhosis and liver failure.[3] Estimates suggest that about 20% to 30% of adults in developed countries have excess fat accumulation in the liver,[4] 50% among people with diabetes, and about 80% in the obese and morbidly obese.[5]

Although research is emerging, it remains uncertain whether diets that are enriched with certain types of food or nutrients are more likely to cause fatty liver than other types of diets.[6] In light of the difficulty

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in reducing weight and maintaining the weight reduction in the long term,[7] changing dietary composition without necessarily reducing calorific intake may offer a more realistic and feasible alternative to treat NAFLD patients. Therefore, exploring the association between specific nutrients and dietary composition and NAFLD is extremely important.

The liver is a central organ and performs multiple metabolic functions for the entire body. Because of its unique location, most blood of the gastrointestinal tract is drained first to the liver, and this organ has a key role in filtering all nutrients that enter the body.

Furthermore, the liver responds to different hormones which are released in response to food intake and reacts to agents released by the intra-abdominal (visceral) fat depots. In case of an excess of food intake, the blood stream to the liver will contain an unusually high concentration of lipids. The liver has an amazing capacity to transform, store and release nutrients, according to the needs of the body. Interestingly, the interplay between the different nutrients and the hormonal and neuronal signals the liver receives affects the fate of individual nutrients. In concert with this, the liver is able to switch rapidly between different modes, e.g. from glucose storage to glucose production.

Disturbances in the delicate metabolic equilibrium of the liver can result in a wide range of whole-body disease and vice versa. The occurrence of the metabolic disease type 2 diabetes mellitus has experienced an extremely rapid increase, affecting currently over 190 million people worldwide. The number of patients is expected to rise to 300 million in 2025.[8] Type 2 diabetes is a highly invalidating disease, especially when taking into account its severe long-term complications: cardiovascular disease, blindness, and kidney failure and impotence. The highly accelerated incidence of type 2 diabetes has been fueled by a tremendous increase in obesity worldwide, resulting from excess calorie intake and lack of physical exercise.

Nonalcoholic fatty liver disease is a chronic liver condition characterized by insulin resistance and hepatic fat accumulation, in the absence of other identifiable causes of fat accumulation, such as Alcohol abuse, Viral hepatitis, Autoimmune hepatitis, Alpha-1 antitrypsin deficiency, medications like corticosteroids and estrogens, and other conditions. Hepatic steatosis may range from a 'benign' indolent deposition of fat to severe lipo toxicity-induced steatohepatitis with necroinflammation [known as nonalcoholic steato hepatitis (NASH)]. NASH is an overlooked complication of Type 2 diabetes mellitus (T2DM) that if missed may carry serious long-term consequences. NASH is frequently associated with fibrosis and approximately of patients develop cirrhosis.

The risk of hepatocellular carcinoma is also increased in patients with T2DM and NASH. Diabetes, Dyslipidemia, Hypertension, and cardiovascular disease (CVD) occur more frequently in individuals with NAFLD. NAFLD may also be associated with a greater risk of renal disease in patients with T2DM. Fatty liver or hepatic steatosis is characterized by diffuse accumulation of fat in hepatocytes. Fatty liver occurring in individuals without a history of significant alcohol intake is termed as non-alcoholic fatty liver disease (NAFLD).

The natural history of NAFLD ranges from pure steatosis to steatohepatitis to cirrhosis and in some patients to hepatocellular carcinoma. NAFLD is strongly associated with obesity, Type-2 diabetes mellitus and Hyperlipidemia. Numerous studiesshowthatitishepaticcomponentofMetabolicSyndromewhosecentral features are peripheral insulin resistance, obesity, hyper insulinemia and hyper triglyceridemia, hypertension.

It has been reported that fatty liver influences the severity of hepatic insulin resistance in Type-2 diabetes mellitus. The hepatic fat content predicts the amount of daily insulin needed to maintain adequate glycemiccontrol.

Objectives

The study is to estimate the proportion of non-alcoholic fatty liver disease in patients with type 2diabetesmellitus. To study the proportion of known risk factors of Non Alchoholic Fatty Liver Disease in patients with in type 2 Diabetes Mellitus.

MATERIALS AND METHODS

A cross-sectional observational hospital based clinical study with 70 patients was undertaken. Patients admitted in the medical wards of Govt. medical college Kottayam. Satisfying the inclusion criteria were selected. Written consent was taken before including the subject to the study. After obtaining the consent, detailed history was taken from the patient or reliable bystander for the assessment of disease status. The history included symptoms suggestive of NAFLD-fatigue, malaise, fullness of abdomen, right upper quadrant pain.

Inclusion Criteria

Patients who are diagnosed with Type 2 Diabetes mellitus

Exclusion Criteria

- Any quantity of alcohol consumption.
- Usage of drugs known to cause steatosis including Amiodarone, Corticosteroids, Tamoxifen, Methotrexate and high -dose Estrogen.
- positive serological markers of viral or auto immune hepatitis (including HBsAg, HCV, ANA).
- History of jejuno ileal bypass or extensive small bowel resection.

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- Metabolic liver diseases, including Wilson's disease and hemochromatosis.

No forthcoming history or asymptomatic were noted. Following this a clinical examination including general examination and systemic examination of gastrointestinal was done. Clinical examination includes hepatomegaly, splenomegaly, jaundice, ascites, xanthelasma, tendon xanthoma were looked. Signs of chronic liver failure features like spidaenaevi, clubbing, palmar erythema, gynacomastia, testicularatrophy, pallor, caput medusa, pigmentation of skin. Bruising was examined. BMI was calculated by the formula as per Quetelet Index. B.M.I = Weight in Kgs / height (in meters)2 After an over-night fast, serum samples were obtained from all subjects for liver function tests (aspartate amino-transferase [AST], alanine aminotransferase [ALT], and alkaline phosphatase), serum lipid profile (total cholesterol, triglycerides, high- density lipoprotein cholesterol [HDL-C] and low-density lipoprotein cholesterol [LDL-C]), and hemoglobin A1C(HbA1c).

All subjects underwent abdominal ultrasonography by the radiologist for evidence of fatty liver disease and itsgraded according to the echogenicity of liver comparing to the echogenicity of periportal area and diaphragm. Data was entered in MS excel spreadsheet. Data was analysed in SPSSv21. Categorical data was represented as frequencies and percentages. Continuous data was represented as mean and standard deviation. Chi square test was used as test of significance for categorical data. Unpaired T test was used as test of significance for continuous data. P value less than 0.05 was considered as statistically significant. Bar diagrams and pie charts were made wherever necessary.

RESULTS

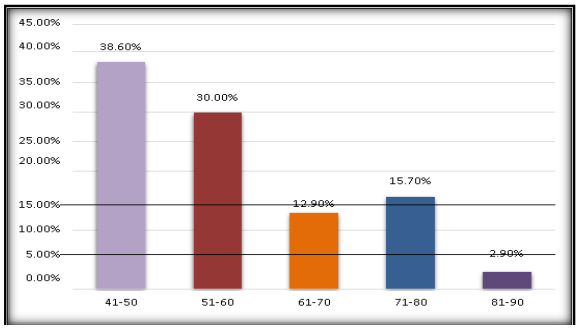

Figure 1: Age distribution

In the present study, majority of study participants were overweight (55.7%). MEAN ± SD: 25.13 ± 3.50

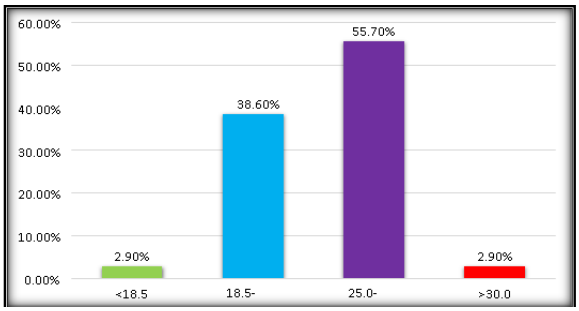

Figure 2: BMI Distribution

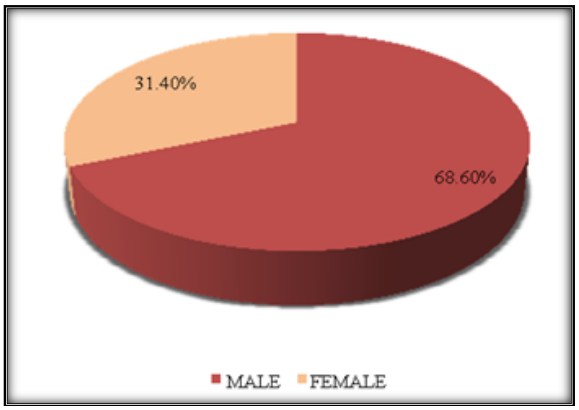

Figure 3: Gender Distribution

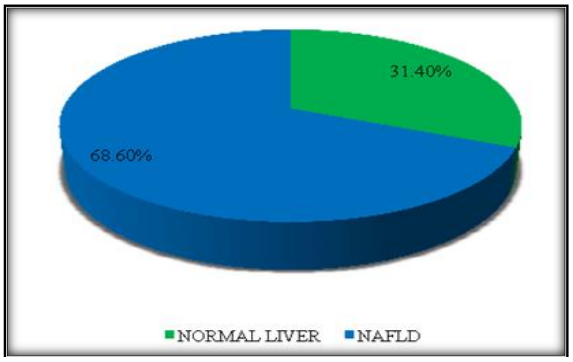

Figure 4: USG Abdomen Showing NAFLD Percentage

In the present study, the youngest age is 41 years and eldest is 86 years with mean age of 56.77 yrs. Majority of study participants belonged to age group of 41-60yearsMEAN ± SD: 56.77 ± 11.37.

Table 1: Duration of Diabetes (After the Detection) of Patients Studied

Duration of Diabetes (Yrs)	Number of Patients	%
<5	6	8.6
5-10	32	45.7
25-10	32	45.7
Total	70	100.0

MEAN ± SD: 10.52 ± 5.17

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Table 2: Glycemic Indices of Patients Studied

Glycemic Indices	Number of Patients	%	MEAN ± SD
HBA1C			
<7.0	19	27.1	
7.1-8.0	11	15.7	
8.1-9.0	9	12.9	
9.1-10.0	5	7.1	
13.4 -10.0	26	37.1	9.26 ± 2.50

Table 3: Lipid Parameters of Patients Studied

Lipid Parameters	Number of Patients	%	MEAN ± SD
Total Cholesterol (MG/DL)			172.21 ± 47.92
<200	52	74.3	
284-200	18	25.7	
Triglycerides (MG/DL)			149.48 ± 98.61
<150	40	57.1	
280-150	30	42.9	
HDL (MG/DL)			40.46 ± 15.38
<40	46	65.7	
117-40	24	34.3	
LDL (MG/DL)			105.54 ± 43.16
<100	34	48.6	
192-100	36	51.4	

Table 4: Liver Enzymes of Patients Studied

Liver Enzymes	Number of Patients	%	MEAN ± SD
SGOT (IU/L)			36.0 ± 17.87
<42	49	70.0	
80-42	21	30.0	
SGPT (IU/L)			37.9 ± 15.69
<33	29	41.4	
72-33	41	58.6	
Alkaline Phosphatase (IU/L)			165.7 ± 62.92
<125	23	32.9	
320-125	47	67.1	

Table 5: Fatty Liver Grades of USG Abdomen of Patients Studied

Fatty Liver Grade	Male N (%)	Female N (%)	Total N (%)
Grade I	16 (57.1)	10 (50.0)	26 (54.2)
Grade II	10 (35.7)	9 (45.0)	19 (39.6)
Grade III	2 (7.2)	1 (5.0)	3 (6.2)
Total	28 (100.0)	20 (100.0)	48 (100.0)

Table 6: Correlation of Socio-Demographic Variables according to USG Abdomen

Variables	USG Abdomen		P Value
	Non-Alcoholic Fatty Liver Disease (n=48)	Normal Liver (n=22)	
Gender			0.962
Male	33 (68.8%)	15 (31.3)	
Female	15 (68.2)	7 (31.8%)	
Height (cm)	1.68 ± 0.06	1.69 ± 0.06	0.439
Weight (Kg)	72.27 ± 11.84	70.36 ± 9.32	0.508
BMI (Kg/M2)	25.44 ± 3.71	24.45 ± 2.96	0.279
Waist Circumference (cm)	98.03 ± 6.30	93.40 ± 5.37	0.004
Hip Circumference (cm)	95.14 ± 7.53	91.59 ± 6.64	0.062
Waist/ Hip Ratio	1.03 ± 0.07	1.02 ± 0.09	0.497

Table 7: Correlation of Clinical Variable with USG Abdomen

Clinical Variables	USG Abdomen		P Value
	Non-Alcoholic Fatty Liver Disease(n=48)	Normal Liver (n=22)	
Duration of DM (Yrs)	10.54 ± 4.98	10.50 ± 5.68	0.975
Total Cholesterol (MG/DL)	174.54 ± 47.33	167.13 ± 49.90	0.552
Triglycerides (MG/DL)	161.64 ± 106.59	122.95 ± 73.82	0.128
HDL (MG/DL)	39.21 ± 13.21	43.18 ± 19.37	0.321
LDL (MG/DL)	104.73 ± 44.46	107.31 ± 41.14	0.818
SGOT (IU/L)	38.50 ± 18.01	30.54 ± 16.67	0.084
SGPT (IU/L)	40.02 ± 15.54	33.27 ± 15.37	0.095
ALP (IU/L)	176.70 ± 67.71	141.68 ± 43.22	0.030

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Table 8: Correlation of Glycemic Parameters Variable with USG Abdomen

Glycemic Status	USG Abdomen		P Value
	Non-Alcoholic Fatty Liver Disease (n=48)	Normal Liver (n=22)	
HbA1c (%)	9.49 ± 2.55	8.74 ± 2.37	0.246

Table 9: Correlation of Anthropometry Parameters with USG Abdomen

Clinical Parameters	USG Abdomen		P Value
	Non-Alcoholic Fatty Liver Disease (n=48)	Normal Liver (n=22)	
BMI (KG/M2)			0.207
<18.5	2 (4.2%)	0 (0.0%)	
18.5-24.9	15 (31.3%)	12 (54.5%)	
25.0-29.9	29 (60.4%)	10 (45.5%)	
>30	2 (4.2%)	0 (0.0%)	
Waist Circumference (cms)			0.305
Male <90& Female<80	3 (6.3%)	3 (13.6%)	
Male >90& Female>80	45 (93.8%)	19 (86.4%)	
Hip Circumference (cms)			0.400
Male <100& Female<105	40 (83.3%)	20 (90.9%)	
Male >100& Female>105	8 (16.7%)	2 (9.1%)	

Table 10: Correlation of Duration of Diabetes with USG Abdomen

Duration of Diabetes	USG Abdomen		P Value
	Non-Alcoholic Fatty Liver Disease(n=48)	Normal Liver (n=22)	
<5 Years	4 (8.3%)	2 (9.1%)	0.994
5-10 Years	22 (45.8%)	10 (45.5%)	
25-10 Years	22 (45.8%)	10 (45.5%)	
MEAN ± SD	10.54 ± 4.98	10.50 ± 5.68	

Table 11: Correlation of Glycemic Parameters with USG Abdomen

Glycemic Parameters	USG Abdomen		P Value
	Non-Alcoholic Fatty Liver Disease (n=48)	Normal Liver (n=22)	
HbA1c			0.876
<6.5	8 (16.7%)	4 (18.2%)	
>6.5	40 (83.3%)	18 (81.8%)	

Table 12: Correlation of Lipid Parameters with USG Abdomen

Lipid Parameters	USG Abdomen		P Value
	Non-Alcoholic Fatty Liver Disease(n=48)	Normal Liver (n=22)	
Total Cholesterol (MG/DL)			0.699
<200	35 (72.9%)	17 (77.3%)	
284-200	13 (27.1%)	5 (22.7%)	
Triglycerides (MG/DL)			0.824
<150	27 (56.3%)	13 (59.1%)	
280-150	21 (43.8%)	9 (40.9%)	
(MG/DL)			0.552
Male <40& Female<50	36 (75.0%)	15 (68.2%)	
Male >40 & Female>50	12 (25.0%)	7 (31.8%)	
LDL (MG/DL)			0.724
<100	24 (50.0%)	10 (45.5%)	
192>100	24 (50.0%)	12 (54.5%)	

DISCUSSION

NAFLD is a silent serious disease, which is becoming epidemic, such as its association with metabolic syndrome. Its etiology is still unknown and further investigations are needed to better understand the pathophysiological processes, and to identify molecular targets for more selective therapies.

A total of 70 diabetic patients were included in the study after applying the selection criteria. Among 70 study participants, 48 (68.6%) were males and 22(31.4%) were females.

Of the 70 diabetics included in this study, 48 (68.6%) of them had ultrasonographical detectable fatty liver.

The overall prevalence of NAFLD in T2DM, in this study was found to be 68.6%, which is higher than different studies conducted in India - Mohan et al it was 56.5% and Kalra S et al it was 54.5%, 20% described by Agal S et al.[9,10,11]

Based on the type of study, the prevalence rates were much higher in those which had histological evidence for NAFLD in comparison to those which were conducted based on biochemical and sonological evidence.

Age Distribution of NAFLD Among Diabetics

In this study, the disease occurrence was found to be predominantly in the fifth decade. In a similar study done in India by Kalra S et al,[10] the prevalence of the disease was found to higher with increasing age

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and commonest in the fifth decade. Similar findings were also observed in the study done by Giovanni et al.[12] A study conducted in Chennai by Viswanathan et al,[13] and in the study by Hardik Patel et al,[14] were found to have a predominant incidence of fatty liver with diabetes in the sixth decade of life. However, in a study by Jaseem Ansari et al,[15] the disease occurrence was found to be predominantly in the fourth decade. However, Prabhakar A et al,[16] reported that prevalence of NAFLD in the younger age group was significantly higher than that in the older age group.

Sex Distribution of NAFLD Among Diabetics

Most of the studies in India have shown a higher prevalence of NAFLD among males than female population.[17] In present study, there were no significant sex differences for incidence of NAFLD between the two groups (p= 0.962). This pattern is also seen in a recent study by Jaseem Ansari et al,[15] and Giovanni et al,[12] where both sexes are affected equally. However, contrary to our findings, in a study by Kalra S et al,[10] higher prevalence rate of disease among females (60%) than in male (53.4%) population was reported. Prabhakar A et al16 also reported high prevalence in females. Hardik Patel et al,[14] reported a high prevalence in males (65.6%).

Duration of T2DM and NAFLD

In present study the mean duration of DM was 10.54 ± 4.98 years in NAFLD group as compared to 10.50 ± 5.68 years in patients without NAFLD (p=0.975), which indicated that duration did not have a significant effect on NAFLD. Similar pattern is also observed by Prashanth et al.[18] However, in the studies done by Kuldeep Chandel et al,[19] Prabhakar A et al,[16] Viswanathan et al,[13] showed that increased duration of diabetes was significantly associated with NAFLD.

Association of obesity with NAFLD in T2DM

Obesity in particular central obesity has been described as one of the strongest risk factors NAFLD and fibrosis.[11]

In present study 64.6% of the patients in NAFLD group had a BMI> 25 Kg/m2 which showed that overweight in combination with T2DM increases the prevalence of NAFLD. BMI was higher in patients with NAFLD (25.44 ± 3.71) than those without NAFLD (24.45 ± 2.96; p=0.279). In a study by Jaseem Ansari et al11, 34% of the total cases were found to have BMI > 25 Kg/m2. Out of which 19% were found to have fatty liver. In a study conducted in Kalra S et al,[10] 54.9% of those patients with obesity enrolled in the study were found to be associated with fatty liver. In a study by Viswanathan et al,[13] 27.6% of the patients with BMI >25% enrolled were found to be associated with NAFLD.

Present study showed significant differences in waist circumference, but no significant differences in BMI, hip circumference, waist to hip ratio among study groups. In a study by Bhatt et al,[20] the waist/hip ratio was significantly different between the two groups (P = 0.0001). Waist/hip ratio denotes abdominal fat distribution. Kuldeep Chandel et al,[19] also reported that NAFLD was significantly associated with high BMI, waist circumference and waist hip ratio.

Kral et al,[21] observed significant correlation between waist/hip ratio and the degree of hepatic steatosis, even in patients with normal BMI.

Similar findings were also reported by Prabhakar A et al, Abdullah A. Alsabaani et al, Jalal MJA et al where high BMI (obesity) is significantly associated with NAFLD.[16,22,23]

Obesity enhances the deposition of increased fat within the hepatocytes leading to a condition of IR (insulin resistance), thus decreasing the fatty acid oxidation. During obesity, fat cell released a number of adipocytokines that enhances the inflammation of the liver. Thus IR considered the key factor in the pathogenesis of NAFLD by increasing the rate of lipogenesis thereby promoting the inhibition of lipolysis.

Association of dyslipidemia with NAFLD in T2DM

In present study - 44 of the 48 NAFLD patients (91.6%) were found to have dyslipidemia. In a study by Kalra S et al,[10] out of 485 patients with dyslipidemia, 311 (59.6%) were found to have NAFLD. In a study by Viswanathan et al,[13] 85.3% of the subjects with dyslipidemia were found to be associated with NAFLD. In a study by Jaseem Ansari et al,[15] 12% of the total cases included were found to have dyslipidemia. Out of the dyslipidemias, 75% were found to have fattyliver.

MV Jalil et al,[24] found that Hypercholesterolemia- 52%, Hypertriglyceridemia- 67%, High LDL- 59% and Low HDL- 27% were significantly higher among subjects with NAFLD-group. In a hospital-based study from North India done by Prashanth et al18 also found almost similar findings. Kuldeep Chandel et al19 also reported that NAFLD was significantly associated with higher triglyceride levels, hypercholesterolemia, low HDLlevels.

Abdullah A. Alsabaani et al,[22] reported from their study that high HDL was a protective factor and low HDL level was a significant factor for the occurrence of NAFLD.

Jalal MJA et al,[23] reported from their study that high triglyceride levels were associated with NAFLD. Nagaraj S et al,[25] also found an association of dyslipidemia with NAFLD.

Association with Liver Enzymes

Contos MJ et al,[26] in his study produce enough evidence to suggest that mild elevation in the liver enzymes may be a marker for significant liver disease.

In a study by Kalra S et al,[10] the mean ALT was found to be higher than mean AST. 34.9% of the patients with NAFLD were found to have at least

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one abnormal amino transferase level out of which 19% had elevation in the ALT and 15.9% had elevation in the AST levels. 65.1% of the patient was found to have elevation of both ALT and AST.
Aparna G et al,[27] observed elevated levels of ALT significantly associated with NAFLD in T2DM. However there have also been studies where there has not been significant correlation between the liver enzymes and NAFLD. In Dallas heart study,[28] 79% of those with hepatic steatosis had normal ALT levels. ALT levels may be normal in the presence of advanced fibrosis or cirrhosis. M Prashanth et al18 also observed similar pattern.
In a study by Jaseem Ansari et al,[15] mean ALT levels were found to be higher than AST levels. 38% of the patients with NAFLD were found to have elevated ALT and 26% of the patients were found to have elevated AST.
Similar findings were also reported by Prabhakar A et al, Vera S. G. Ferreira et al, Nagaraj S et al where elevated liver enzymes were significantly associated with NAFLD.

NAFLD and Ultrasonography

USG is by far the commonest method of diagnosing NAFLD in clinical practice. The sensitivity of diagnosing NAFLD sonologically is found to be 83-90%, Specificity 95-96%.[29]
In present study NAFLD was diagnosed in 68.6% of the study population based on USG finding. In a study by Banerji et al,[30] 63.8% of the patients and in the study by Jaseem Ansari et al,[15] 26% of the patients were found to be associated with increased echogenicity of the liver suggestive of fatty liver.

Association of NAFLD IN T2DM with Glycemic Status

In the present study, HbA1c was found out to be higher with NAFLD group, when compared to the non NAFLD group. The results of the current study suggest that HbA1c levels are not a suitable predictor of NAFLD among DM patients, as we did not observe significant association between them and NAFLD. The same results were reported in previous studies by Poanta L et al,[31] and Madi DR et al.[32] On the other hand, a trend of increased HbA1c level among NAFLD was observed among NAFLD diabetic patients compared with non-NAFLD patients in other studies by Herath HMM et al,[33] and Afolabi et al.[34] However, no significant relation observed HBA1C in present study groups which are also observed by Bhatt et al,[20] M Prashanth et al.[18]
But Kuldeep et al,[19] reported that as glycaemic control worsens the risk of developing NAFLD increases. Also found that raised value of HbA1c correlated significantly with the incidence of NAFLD, with p-value being, 0.001 respectively. These findings were also seen in other studies like, Giorgio Bedogni et al,[35] and also in Giovanni et al.[12]

CONCLUSION

The proportion of NAFLD is high (68.6%) amongst T2DM patients affecting 68.8% males and 31.3%females. 8.5% of the patients had hepatomegaly. but no other clinical findings were observed.
Age of the patient and duration of diabetes did not have a significant difference on the incidence ofNAFLD. 64.6% of the patients in NAFLD group had a BMI > 25 kg/m2 which showed that overweight in combination with T2DM increases the proportion ofNAFLD. Glycemic status was higher among the NAFLD group, but no statistically significant difference observed among study populationgroups.
91.6% of the patients in the NAFLD group had dyslipidemia with 43.8% of patients havinghypertriglyceridemia. Alkaline Phosphatase levels were significantly high in NAFLD group as compared to normalgroup.

Limitations

The diagnosis of NAFLD was based on ultrasonography and was not confirmed by liverbiopsy.
The sample size is small. A larger sample size could have provided greater statistical power and permitted more complete adjustment for potential confounders.

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