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Original Research Article

A study of non-alcoholic fatty liver disease in patients with type 2 diabetes mellitus and its correlation with cardiovascular risk

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ABSTRACT

Background: NAFLD (non-alcoholic fatty liver disease) encompasses a variety of disorders of lipid metabolism ranging from NAFL, NASH to cirrhosis and rarely HCC. A great deal of evidence suggests that the metabolic syndrome predicts incident cardiovascular disease (CVD), so it is possible to hypothesize that NAFLD patients might portend a greater CVD risk and that NAFLD itself might confer a CVD risk above that associated with individual metabolic syndrome risk factors.
Methods: 55 T2DM patients were included in current study conducted from August 2019 to September 2021 for assessment of NAFLD using USG abdomen, NAFLD fibrosis score and FIBROSCAN. ASCVD score was used for correlation between CVD risk and NAFLD.
Results: Out of 55 patients 42 (76.4%) were having fatty liver based on USG abdomen while 13 (23.4%) patients were having no fatty liver. As far as steatosis is concerned mean CAP (dB/m) was 245±50.89 out of which 24 (43.6%) were having no or minimal steatosis (S0), while 31 (56.4%) were having significant steatosis with 11 (20%), 9 (16.4%) and 11 (20%) having S1, S2 and S3 grade of steatosis respectively. 15 patients out of 55 were of F0 grade while 19 (34.5%), 9 (16.4%), 7 (12.7%) and 5 (9.1%) were of grade F1, F2, F3 and F4 respectively. There was a positive statistically significant ($p \leq 0.001$) association of ASCVD risk score with NAFLD fibrosis score and fibroscan: E (KPa).
Conclusions: Our study found that the prevalence of NAFLD was quite higher in patients with T2DM based on both USG abdomen (76.4%) and transient elastography (steatosis-56.4% and fibrosis-65.5%) and a statistically significant association between fibrosis and ASCVD score with higher fibrosis associated with higher 10 years ASCVD score.

Keywords: Non-alcoholic fatty liver disease, NAFL, Non-alcoholic steatohepatitis

INTRODUCTION

NAFLD encompasses a variety of disorders of lipid metabolism ranging from NAFL (non-alcoholic fatty liver), NASH (non-alcoholic steatohepatitis) to cirrhosis and rarely HCC in very severe form. It is defined as hepatic steatosis in >5% of hepatocytes according to histological analysis or by proton density fat fraction or >5.6% as assessed by proton magnetic resonance spectroscopy (MRS) or quantitative fat/water selective magnetic resonance imaging (MRI) with no secondary cause for steatosis.[1] NAFLD is the most common cause of abnormal liver function test among western countries[2] and is commonly associated with components of metabolic syndrome supporting the idea of it being a hepatic manifestation of the syndrome. As obesity and insulin resistance has been strongly associated with NAFLD, the prevalence of NAFLD is higher in T2DM

patients than in general population and around 70-75% of T2DM patients have some form of NAFLD.[2] There is a 5-fold risk of developing T2DM in a patient of NAFLD.[3] A great deal of evidence suggests that the metabolic syndrome predicts incident cardiovascular disease (CVD), so it is possible to hypothesize that NAFLD patients might portend a greater CVD risk and that NAFLD itself might confer a CVD risk above that associated with individual metabolic syndrome risk factors.[4] If these data holds true, the identification of NAFLD in type 2 diabetes may help in CVD risk prediction with important management implications.[3] Also this would help us manage actively this subgroup of diabetic patients with NAFLD and reduce CVD related complications. This study was primarily done to study the prevalence of NAFLD in patients with T2DM using ultrasound abdomen, NAFLD fibrosis score and transient elastography.

METHODS

This hospital based cross-sectional study has been carried out from August 2019 to September 2021 with a sample size of 55 subjects in department of medicine, KPS institute, GSVM medical college, Kanpur, Uttar Pradesh, India.

Inclusion criteria

The study group consisted of patients with T2DM with age greater than 18 years and excluding other causes of chronic liver disease like viral infection (hepatitis B and hepatitis C), history of intake of hepatotoxic drugs (e.g. ATT, valproate), patients with significant alcohol intake and patients of congestive heart failure. The patients underwent a detailed history including past, treatment and personal history and a thorough clinical examination. The findings were logged in a specially prepared proforma. The cases were subjected to following investigations: ECG, Hb, TLC, DLC, ESR, urine routine and microscopy, HbA1c, random blood sugar, SGPT/SGOT, serum bilirubin total and differential, kidney function test,serum total protein, albumin and globulin, USG whole abdomen, transient elastography. The patients were then graded in grades of fatty liver (grade I, grade II and grade III) based on USG abdomen and hepatic steatosis based of CAP value of transient elastography. Patients were categorized for liver fibrosis using E value of transient elastography. Cardiovascular risk was assessed using 10-year ASCVD risk.

Statistical analysis

Data analysis was done using statistical package for social science (SPSS version 10.5) package. The collected data was summarized in the form of mean±standard deviation (SD) and range for measurable data and frequency and percentage for qualitative data. Comparisons between the various study groups were done using different statistical tests. Association between variables was considered statistically significant if $p<0.05$.

RESULTS

Out of the 55 cases 26 (47.3%) were male and 29 (52.7%) were female. Gender distribution among groups is shown in (Figure 1). Age distribution of the subjects has been shown in the Figure 2. 9.1% of the participants had age: 31-40 years. 29.1% of the participants had age: 41-50 years. 32.7% of the participants had age: 51-60 years. 25.5% of the participants had age: 61-70 years. 3.6% of the participants had age: 71-80 years.

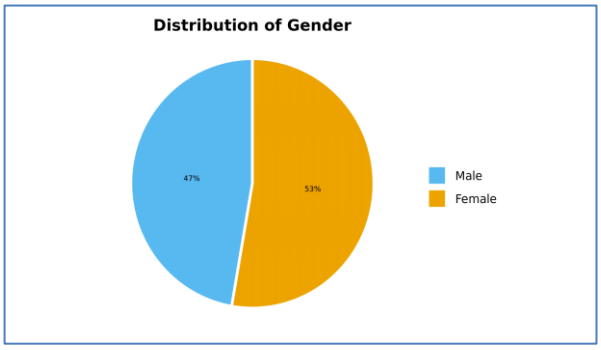

Figure 1: Gender distribution of study subjects.

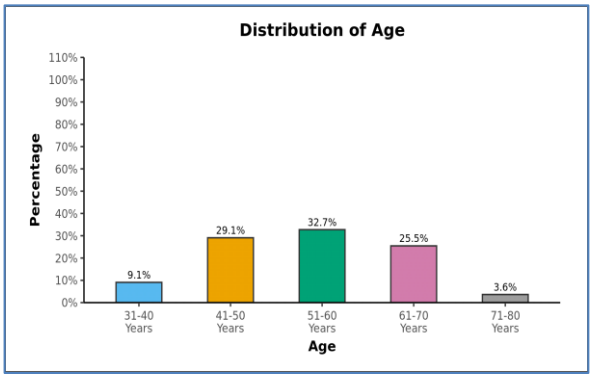

Figure 2: Age distribution of study subjects.

Out of 55 patients 42 (76.4%) were having fatty liver (grade 1-25, grade 2-15, grade 3-2) based on ultrasound abdomen while 13 (23.4%) patients were having no fatty liver. As far as steatosis is concerned mean CAP (dB/m) was 245+50.89 out of which 24 (43.6%) were having no or minimal steatosis (S0-CAP <237 dB/m), while 31 (56.4%) were having significant steatosis with 11 (20%), 9 (16.4%) and 11 (20%) having S1 (CAP-237-259 dB/m), S2 (259-291 dB/m) and S3 (>291 dB/m) grade of steatosis respectively. The mean E (kpa) of the study population was 9.05±7.24. Patients were graded for liver fibrosis using E (kpa) value obtained from transient elastography into F0 (E <5.5 kpa), F1 (5.5-8 kpa), F2 (8-10 kpa), F3 (10-16 kpa) and F4 (>16 kpa). 15 patients out of 55 were of F0 grade (no fibrosis) while 19 (34.5%), 9 (16.4%), 7 (12.7%) and 5 (9.1%) were of grade F1 (mild fibrosis), F2 (moderate fibrosis), F3 (severe fibrosis) and

F4 (cirrhosis) respectively. ASCVD (10 year) risk score was calculated using an online calculator to assess the cardiovascular risk of the patients. Mean ASCVD risk score of the study population was 11.16±11.58.

Table 1: Descriptive data of the study population.

USG	Observation
USG grade (N%)	
Normal	13 (23.6)
Grade 1	25 (45.5)
Grade 2	15 (27.3)
Grade 3	2 (3.6)
Fatty Liver (yes)	42 (76.4)
Fibroscan: CAP (mean±SD)/median (IQR)/range	245.82±50.89/246.00 (218-279.5)/100-343
Steatosis grade N (%)	
S0	24 (43.6)
S1	11 (20.0)
S2	9 (16.4)
S3	11 (20.0)
Fibroscan: E (KPa) (mean±SD)/median (IQR)/range	9.05±7.24/6.40 (5.40-9.20)/2.90-47.10
Fibrosis grade N (%)	
F0	15 (27.3)
F1	19 (34.5)
F2	9 (16.4)
F3	7 (12.7)
F4	5 (9.1)
ASCVD (10 year) risk score (%) (mean±SD)/median (IQR)/range	11.16±11.58/7.50 (2.50-14.95)/0.80-46.

DISCUSSION

Several shared pathophysiological pathways link NAFLD and T2DM to increased cardiovascular risk including proatherogenic lipid alteration, increase in thrombosis factors, insulin resistance, low-grade inflammation and microbiome alteration.[5]

Kalra et al conducted a study to determine frequency and risk factors in type 2DM patients. Out of 924 patients (335 females/569 males) in an age group of 15-85 years were identified as having NAFLD. In our study as well the prevalence of fatty liver was quite high than in general population. 42 (76.4%) participants (21 males/21 females) had fatty liver (grade 1, 2 and 3) on USG while 13 (23.6%) had no fatty liver. Also the average HBa1c of the fatty liver group was higher (9.14±2.56) than that of the non-fatty liver group (8.76±3.540) although this difference wasn't significant statistically. There was no significant difference in terms of ASCVD score in both these groups (fatty liver and non fatty liver) with ASCVD risk score of the fatty liver and non-fatty liver group as 10.43 (SD-11.25) and 13.53 (SD-12.78) respectively. Toung et al conducted a cross-sectional design in T2DM adults who attended Dai Phuoc Ho Chi Minh Polyclinic and Polyclinic of Pham Ngoc Thach University of Medicine and found in their study that the prevalence of NAFLD in T2DM patients based on FibroScan was 73.3%. The prevalence of significant fibrosis (≥ F2), advanced fibrosis (≥F3) and cirrhosis (F4) was 13.0%, 5.9% and 3.6%, respectively. The prevalence of steatosis in our study population was 56.4% (31/55) based on transient elastography. 11 (20%), 9 (16.4%) and 11 (20%) out of 55 study subjects had steatosis of grade S1 (CAP-237-259 dB/m), S2 (259-291 dB/m) and S3 (>291 dB/m) respectively based on CAP. The mean value of Fibroscan: E (KPa) of our participants was 9.05±7.24. 15 (27.3%) of the participants had fibrosis grade: F0. 19 (34.5%) of the participants had fibrosis grade: F1. 9 (16.4%) of the participants had fibrosis grade: F2. 7 (12.7%) of the participants had fibrosis grade: F3. 5 (9.1%) of the participants had fibrosis grade: F4. There was a moderate positive correlation between ASCVD risk score (%) and fibroscan: E (KPa) and this correlation was statistically significant (rho=0.47, p≤0.001). For every 1 unit increase in ASCVD risk score (%), the fibroscan: E (KPa) increases by 0.19 units. Conversely, for every 1 unit increase in fibroscan: E (KPa), the ASCVD risk score (%) increases by 0.48 units.

Limitations

The limitation of the present study was a relatively small sample size. Future studies have to be directed with larger sample size. Also since the study was conducted at a tertiary centre which may not be a representative of the general population.

CONCLUSION

Current study revealed that the prevalence (76.4%) of NAFLD was quite higher in patients with T2DM based on USG abdomen. Mean HbA1c of the patients with fatty liver was higher than that of the non-fatty liver group. The prevalence of steatosis (56.4%) and fibrosis (65.5%) as measured by transient elastography was also higher in our study population showing the facts that patients with T2DM are at increased risk of developing NALFD. Cardiovascular risk assessed by ASCVD risk score was also significantly associated with NAFLD.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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