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Clinicopathological Study of Non-Alcoholic Fatty Liver Disease in Patients with Type 2 Diabetes Mellitus

*B Subhashini[1], E Lakshman[2],D Praveen Kumar[3]
1)Assistant prof, Dept of Pathology, KIMS Narketpally2) Resident , General Medicine, KMC Warangal,3) Resident, General Medicine, KMC Warangal.
Corresponding author-E Lakshman, Resident, General Medicine, KMC Warangal.
Email- drsubhabandar@gmail.com

Abstract:
Introduction: *NAFLD is a group of conditions that have in common the presence of hepatic steatosis (fatty liver), in individuals who do not consume alcohol. It has become the most common cause of chronic liver disease in the United States and in its various forms probably affects more than 30% of the population. Hepatic steatosis may range from a 'benign' indolent deposition of fat to severe lipotoxicity-induced steatohepatitis with necroinflammation known as Nonalcoholic steatohepatitis(NASH).NASH is an overlooked complication of Type 2 diabetes mellitus (T2DM) that if missed may carry serious long-term consequences.*
Objectives of the study*: 1)To study the prevalence of Nonalcoholic fatty liver disease and Nonalcoholic steatohepatitis in patients with Type 2 Diabetes Mellitus and 2) To study the features of NASH by liver biopsy.*
Settings & Design: *Prospective observational study was conducted on a total of 90 patients, aged above 40yrs with type 2 diabetes mellitus.*
Materials & Methods: *Fatty liver by Ultrasonography & various other relevant factors were measured in all study subjects. Based on USG , patients were divided into two groups 1)NAFLD group-altered echo texture of the liver parenchyma and 2)normal liver group . liver biopsy was done in willing NASH patients diagnosed by ultrasound and biopsy slides were examined histopathologically.*
Results: *Incidence of Non-alcoholic fatty liver disease in our study is around 62 (59.7%) of which 37 (55%) are males and 25 (45%) are females. 20 patients diagnosed as NASH by ultrasound underwent liver biopsy*
Conclusion: *The prevalence of NAFLD is high amongst Type2DM patients and considering this risk , NAFLD should be actively sought out and treated in patients with diabetes.*
KEYWORDS*: NonalcoholicSteatohepatitis(NASH) ,NAFLD, obesity, type 2 diabetis mellitus*

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I. Introduction

NAFLD is a group of conditions that have in common the presence of hepatic steatosis (fatty liver), in individuals who do not consume alcohol. NAFLD includes simple hepatic steatosis, steatosis accompanied by minor non-specific inflammation and non-alcoholic steatohepatitis (NASH). Steatosis with or without non-specific inflammation is generally a stable condition without significant clinical problems. In contrast, NASH is a condition in which there is hepatocyte injury that may progress to cirrhosis in 10% to 20% of cases. The main components of NASH are hepatocyte ballooning, lobular inflammation, and steatosis.With progressive disease fibrosis occurs. NASH affects men and women equally and the condition is strongly associated with obesity and other components of the metabolic syndrome such as dyslipidemia, hyperinsulinemia and insulin resistance. It is estimated that more than 70% of obese individuals have some form of NAFLD. Obesity, type 2 diabetes and hyperlipidemia are recognized as risk factors for NAFLD. Insulin resistance is frequently detected in patients with NAFLD, as it is in those without obesity and diabetes. An increasing number of patients have been described with normal body mass index (BMI), although these individuals may have central adiposity and occult insulin resistance.

Nonalcoholic fatty liver disease is a chronic liver condition characterized by insulin resistanceand 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 lipotoxicity-induced steatohepatitis with necroinflammation known as nonalcoholic steatohepatitis (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 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. Fatty liver or hepatic steatosis is characterized by diffuse accumulation of fat in hepatocytes. In obesity, visceral fat accumulation is more important in the pathogenesis of the syndrome than general obesity. In human visceral obesity, white adipose tissue is enriched in macrophages. The presence of macrophages in omental white adipose tissue participates in the cellular mechanisms favouring hepatic fibroinflammatory lesions in obese patients.Mildly inflamed adipose tissue functions as an endocrine organ, releasing several protein signals, amongst others the adipokines involved in energy and glucose metabolism:leptin, adiponectin, resistin, and visfatin. Visceral fat appears to be even directly associated with liver inflammation and fibrosis independent of insulin resistance and hepatic steatosis. Chemokines and cytokines and innate immune processes, occurring both within and outside the liver, are also involved in insulin resistance and end-organ damage in the form of NAFLD

Unfortunately, the liver is not easily accessible and liver biopsy is currently considered the gold-standard to assess possible liver inflammation and degree of ectopic fat accumulation.

Aims and Objectives

1. Main aim of the study is to know the risk factors of non-alcoholic fatty liver disease in patients with type 2 diabetes mellitus .
2. To determine the prevalence of Nonalcoholic fatty liver disease and Nonalcoholic steatohepatitis in patients with Type 2 Diabetes Mellitus.
3. To study the liver biopsy features in patients who were diagnosed as NASH by ultrasonography.
4. Liver biopsy was done in patients who were willing only.

II. Materials And Methods

SOURCES OF COLLECTION OF DATA- The cases for the study were selected from patients with Type 2 diabetes mellitus diagnosed by standard criteria above the age of 40 years who attended **KMC /MGM Hospital ,Warangal.**The study is a PROSEPECTIVE OBSERVATIONAL STUDY, where continuous data was collected which fulfills the inclusion criteria. This study was conducted between august 2015 to august 2017.

Patients satisfying the inclusive criteria were enrolled in this study, after providing written informed consent, a thorough medical history and physical examination was performed for each individual, which included measurements of weight and height. BMI was calculated as a measure of obesity, whereas waist/hip ratio was measured as an index of splanchnic fat accumulation. After an over-night fast, serum samples were obtained from all subjects for liver function tests (Aspartate aminotransferase [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]), fasting blood glucose (FBS), serum insulin level and hemoglobin A1c (HbA1c).

Overweight was defined as a body mass index (BMI) between 25 and 29 kg/m2, and obesity as BMI equal or above 30 kg/ m2. Patients were considered centrally obese if the waist circumference was greater than 80 cm in females and 90 cm in males. Patients with one of the criteria: LDL-C >100 mg/dL, total cholesterol >200 mg/ dL, triglycerides >150 mg/dL, or HDL- C < 40 mg/dL in males and <50 mg/dL in females were considered to have dyslipidemia.

Homeostasis Model Assistant–Insulin Resistance (HOMA-IR) and Quantitative Insulin Sensitivity Check Index (QUICKI) were calculated as measures of insulin resistance and sensitivity using following formula:

HOMA-IR= [fasting insulin (μU/ml) ◊fasting glucose (mmol/l)]/22.5

QUICKI=1/ [log (fasting insulin (μU/ml)) ± log (glucose (mg/dl))]

All subjects underwent abdominal ultrasonography by the radiologist for evidence of fatty liver disease. Based onultrasonographic findings (diffuse increase in echogenicity as compared to that of the spleen or renal cortex).

Inclusion Criteria

- Patients of age above 40 years of either sex with Type 2 diabetes mellitus on oral hypoglycemic drugs.
- Patients willing to give written informed consent

Exclusion Criteria

- Any quantity of alcohol consumption based on careful history
- Usage of drugs known to cause steatosis including Amiodarone, Corticosteroids, Tamoxifen, Methotrexate and high dose Estrogen.

- Significant co-morbidities precluding a liver biopsy (eg. Bleeding Diathesis)
- Positive serological markers of viral or auto immune hepatitis (including HBsAg , HCV , HIV , ANA , anti-smooth muscle antibodies , antiliver / kidney microsomes type 1 antibodies)
- History of jejunoileal bypass or extensive small bowel resection.
- Findings in favour of metabolic liver diseases , including Wilson's disease , hemochromatosis and positive alpha-1 antitrypsin
- Patients with Type 2 diabetes mellitus on insulin therapy.

Preliminary data based on age/name/sex address was entered.
Evaluation of each patient included a proper history , detailed general and systemic examination and evaluation through necessary investigations .
The history included symptoms suggestive of NAFLD – fatigue , malaise , fullness of abdomen , right upper quadrant pain .
In addition to above symptoms details to try and find out possible risk factors was undertaken the history of associated illnesses and drug intake was noted , with emphasis on drugs associated with NAFLD .
A relevant history of duration of diabetes and the drugs was also noted.
In general examination , in addition to the vital parameters , icterus/clubbing/pallor were looked for .
By standard means the weight in kgs and height in meters of all patients was measured .
BMI was calculated by the formula as per QueteletIndex .
B.M.I = Weight in Kgs / (height in meters)2
Systemic examination of respiratory, cardiovascular , gastrointestinal and central nervous system were carried out .
Detailed per abdominal examination for palpability of liver , spleen , their size and consistency was made . Confirmation for absence of tenderness of liver and absence of free fluid was carried out .
The patients were then subjected to necessary investigations .
Investigations included

- Complete blood count
- Renal function test
- Blood sugar level : fasting and post prandial
- **Lipid profile** :
- serum triglycerides
- total cholestrol ,
- S.HDL ,
- S.LDL
- **Liver function tests :**
- SGOT (AST) & SGPT(ALT)
- SGOT/SGPT Ratio
- S.bilirubin- Total/Direct/Indirect
- S.alkaline phosphatases
- S.proteins – Total/Albumin/Globulin
- Serum GGT
- HBsAg/Anti HCV/HIV
- HbA1C
- BT/CT/PT/APTT to rule out bleeding tendencies
- USG – Abdomen

Out of the entire study population 20 cases with elevated serum aminases and USG findings of NAFLD Who were willing were subjected to liver biopsy with informed consent .
Biopsy specimens were collected in formalin . Biopsies were then processed for histopathological examination.
The Grading and Staging of all biopsies were determined based on method proposed by Brunt et al .
Grading and Staging the histopathological lesions of NASH

GRADE 1, MILD

- Steatosis : predominantly macrovesicular , involves <33 upto 66% of the lobules
- Ballooning : occasionally observed ; zone 3 hepatocytes
- Lobular inflammation : scattered and mild acute inflammation and occasional chronic inflammation
- Portal inflammation : none or mild

GRADE 2, MODERATE

- Steatosis : any degree and usually mixed macrovesicular and microvesicular

- Ballooning : obvious and present in zone 3
- Lobular inflammation : polymorphs may be noted associated with ballooned hepatocytes,pericellular fibrosis ; mild chronic inflammation may be seen
- Portal inflammation : none or mild

GRADE 3, SEVERE

- Steatosis : typically >66%(panacinar) ; commonly mixed steatosis . ballooning predominantly zone 3 marked
- Lobular inflammation : scattered acute and chronic inflammation ; polymorphs may appear concentrated in zone 3 areas of ballooning and perisinusoidal fibrosis
- Portal inflammation : mild or moderate

Staging fibrosis in NASH :

Stage 1 : zone 3 perivenularperisinusoidal /pericellular fibrosis, focal or extensive
Stage 2 : as above with focal or extensive periportal fibrosis
Stage 3 : bridging fibrosis , focal or extensive
Stage 4 : cirrhosis

III. Results And Analysis

Study Design: A Correlation clinical observational hospital based clinical study with 90 patients was undertaken to study the predictors of Non-alcoholic fatty Liver disease and Non-alcoholic steatohepatitis in patients with Type 2 Diabetes Mellitus.

Table 1 : Age distribution of patients studied

Age in years	Number of patients	%
41-50	35	38.9
51-60	24	26.5
61-70	15	16.5
71-80	13	14.4
>80	03	3.3
Total	90	100

Mean $\pm$SD : 55.96$\pm$11.65

We studied 90 patients with mean age of 55.96$\pm$11.65 years . 56(60%) subjects were males and 36(40%) subjects were females.

Table 2: Gender distribution of patients studied.

Gender	Number ofPatients	%
Male	54	60.0
Female	36	40.0
Total	90	100.0

Table 3: BMI (kg/m^2) of patients studied

BMI	Number of patients	%
<18.5	04	4.4
18.6-24.9	37	40.7
25.0-29.9	47	51.8
>30	02	2.2
Total	90	100.0

Mean ± SD: 24.90±3.52

Table 4: Waist Circumference (cms) of patients studied.

Waist Circumference	Number of patients	%
<90.0	03	3.3
90-100.0	34	37.7
>100.0	53	58.9
Total	90	100.0

Mean ± SD: 96.36±6.08

Table 5: Duration of Diabetes (yrs) of patients studied.

Duration of Diabetes (yrs)	Number of patients	%
<5	07	7.8
5-10	40	44.5
10-20	41	45.5
>20	02	2.2
Total	90	100.0

Mean ± SD: 11.05±5.43

Table 6 :Glycemic Indices:

Glucose parameters	Number of patients (n= 90)	%	Mean ±SD
FBS (mg/dl)			155.22±63.10
70-130	30	33.3	
>130	60	66.6	
PPBS (mg/dl)			211.53±64.58
<180	39	43.3	
≥180	51	57.7	
HbA1C			9.18±2.42
<7	18	19.9	
7-8	13	14.3	
8-9	15	16.6	
9-10	12	13.3	
>10	32	35.4	

Table 7: Lipid parameters of patients studied

Lipid parameters	Number of Patients(n=90)	%	Mean ± SD
Total cholesterol mg/dl			173.31±46.92
<200	60	66.0	
>200	30	33.0	
Triglycerides (mg/dl)			151.66±96.43
<150	53	58.3	
>150	37	40.7	
HDL (mg/dl)			39.12±13.70
<40	56	61.6	
>40	34	37.4	
LDL (mg/dl)			105.05±40.97
<100	60	66.0	
>100	30	33.0	

Table 8: Fasting Insulin Level (µU/ml) of patients studied.

FastingInsulin Level (µU/ml)	Number of patients	%
<30	75	83.2
30-40	13	14.4
>40	2	2.2
Total	90	100.0

Mean ± SD: 22.23±7.86.

Table 9: Liver Enzymes of patients studied.

Enzymes	Number of patients (n=90)	%	Mean ± SD
SGOT (IU/L)			34.94±18.41

<42	**64**	**71.1**	
>42	**26**	**28.7**	
SGPT (IU/L)			**39±14.84**
<33	**34**	**37.6**	
>33	**56**	**62.0**	
Alkaline Phosphatase (IU/L)			**164.86±63.38**
<125	**30**	**33.3**	
>125	**60**	**66.6**	

Table 10: USG abdomen of patients studied

USG abdomen	Number of patients	%
Normal liver	**28**	**31.1**
NAFLD	**62**	**68.9**
Total	**90**	**100.0**

Table 11: HOMA-IR & QUICKI of patients studied.

HOMA-IR & QUICKI	Number of Patients (n=90)	%	Mean ± SD
HOMA-IR			**8.47±4.45**
<3	**4**	**4.4**	
3-5	**24**	**26.4**	
5-10	**42**	**46.2**	
>10	**20**	**22.0**	
QUICKI			**0.28±0.01**
<0.3	**60**	**66.6**	
>0.3	**30**	**33.4**	

Table 12: Correlation of socio-demographic variables according to USG abdomen.

	USG abdomen		
Variables	**Non-alcoholic fatty liver disease (n=62)**	**Normal Liver (n=28)**	**P value**
Age in yeas	**55.37±10.95**	**57.25±13.22**	**0.482**
Gender			
Male	**37(59.7%)**	**17(60.7%)**	**0.926**
Female	**25(40.3%)**	**11(39.3%)**	
Height (cm)	**5.54±0.19**	**5.57±0.21**	**0.538**
Weight (kg)	**72.32±11.39**	**69.43±10.25**	**0.253**

BMI(kg/m^2)	**25.30±3.62**	**24.03±3.19**	**0.116**
Waist circumference(cm)	**97.75±5.93**	**93.28±5.31**	**0.001****
HIP (cm)	**94.98±7.27**	**91.23±6.60**	**0.022***
Waist/HIP ratio	**1.03±0.07**	**1.03±0.08**	**0.721**

Table 13: Correlation of Clinical variables / USG abdomen

Clinical variables	**USG abdomen**		
	Non-alcoholic fatty liver disease (n=62)	**Normal Liver (n=28)**	**P value**
Duration of DM (yrs)	**10.87±4.99**	**11.46±6.37**	**0.634**
Total cholesterol (mg/dl)	**173.21±46.51**	**173.52±48.63**	**0.977**
Triglycerides (mg/dl)	**157.65±98.68**	**138.41±91.58**	**0.384**
HDL(mg/dl)	**37.73±11.46**	**42.21±17.55**	**0.152**
LDL(mg/dl)	**104.67±42.17**	**105.90±38.92**	**0.896**
SGOT (Iu/L)	**37.95±18.86**	**28.28±15.73**	**0.020***
SGPT (IU/L)	**41.50±14.62**	**33.46±14.05**	**0.017***
ALP (IU/L)	**173.53±70.05**	**145.67±40.09**	**0.053+**

Table 14: Correlation of Insulin resistance parameters / USG abdomen.

	USG abdomen			
Insulin resistance Index	**Non-alcoholicfatty liver disease(n=62)**	**Normal Liver (n=28)**	**P value**	
FBS(mg/dl)	**151.10±58.84**	**164.36±71.94**	**0.359**	
PPBS (mg/dl)	**206.33±64.24**	**223.03±65.07**	**0.259**	
HbA1c(%)	**9.39±2.47**	**8.72±2.26**	**0.225**	
Fasting Insulin (µU/ml)	**22.69±7.66**	**21.21±8.36**	**0.412**	
HOMA-IR	**8.39±4.27**	**8.64±4.91**	**0.815**	
QUICKI	**0.28±0.02**	**0.29±0.02**	**0.686**	

Table 15: Correlation of Anthropometry parameters / USG abdomen.

Anthropometry parameters	**USG abdomen**		
	Non-alcoholic fatty liverdisease (n=62)	**Normal Liver (n=28)**	**P value**
BMI (kg/m^2)			
<18.5	**3(4.8%)**	**1(3.6%)**	**0.187**
18.5-24.9	**21(33.9%)**	**16(57.1%)**	
25.0-29.9	**36(58.1%)**	**11(39.3%)**	
30 & above	**2(3.2%)**	**0(0%)**	
Waist Circumference(cm)			
Male<90 & Female <80	**3(4.8%)**	**2(7.1%)**	

			0.645
Male>90 & Female>80	**59(95.2%)**	**26(92.9%)**	
Hip Circumference (cm)			
Male<100 & Female <105	**54(87.1%)**	**25(89.3%)**	**1.000**
Male>100 & Female>105	**8(12.9%)**	**3(10.7%)**	

Table 16: Correlation of Duration of diabetics / USG abdomen.

	USG abdomen		
Duration of diabetics	**Non-alcoholicfatty liver disease (n=62)**	**Normal Liver (n=28)**	**P value**
<5 years	**10(16.1%)**	**4(14.3%)**	**0.758**
5-10 years	**24(38.7%)**	**9(32.1%)**	
>10 years	**28(45.2%)**	**15(53.6%)**	
Mean ± SD	**10.87±4.99**	**11.46±6.37**	

Table 17: Correlation of glycemic parameters / USG abdomen.

Sugar parameter	**USG abdomen**		
	Non-alcoholicfatty liver disease(n=62)	**Normal Liver(n=28)**	**P value**
FBS (mg/dl)			
≤130	**7(11.3%)**	**4(14.3%)**	**0.688**
>130	**55(88.7%)**	**24(85.7%)**	
PPBS(mg/dl)			
≤180	**37(59.7%)**	**11(39.3%)**	**0.073+**
>180	**25(40.3%)**	**17(60.7%)**	
HbA1c			
<6.5	**10(15.6%)**	**5(17.9%)**	**0.833**
>6.5	**52(83.9%)**	**23(82.1%)**	

Table 18: Correlation of Lipid parameters / USG abdomen.

Lipid parameters	**USG abdomen**		
	Non-alcoholic fatty liver disease (n=62)	**Normal Liver (n=28)**	**P value**
Total Cholesterol (mg/dl)			
<200	**43(69.4%)**	**17(60.7%)**	**0.421**
>200	**19(30.6%)**	**11(39.3%)**	
Triglycerides (mg/dl)			
<150	**36(58.1%)**	**17(60.7%)**	**0.813**
>150	**26(41.9%)**	**11(39.3%)**	
HDL(mg/dl)			
Males<40, Females <50	**48(77.4%)**	**21(75%)**	**0.802**

Males>40, Females >50	**14(22.6%)**	**7(25%)**	
LDL (mg/dl)			
<100	**43(69.4%)**	**17(60.7%)**	**0.421**
>100	**19(30.6%)**	**11(39.3%)**	

Table 19: Correlation of Fasting Insulin Level (µU/ml) / USG abdomen.

Fasting Insulin Level (µU/ml)	**USG abdomen**		
	Non-alcoholic fattyliver disease (n=62)	**Normal Liver(n=28)**	**P value**
<30	**50(80.6%)**	**25(89.3%)**	**0.309**
>30	**12(19.4%)**	**3(10.7%)**	

Table 20: Correlation of insulin index / USG abdomen.

Insulin Index	**USG abdomen**		
	Non-alcoholicfatty liver disease (n=62)	**Normal Liver(n=28)**	**P value**
HOMA IR			
<5.0	**15(24.2%)**	**7(25%)**	**0.934**
>5.0	**47(75.8%)**	**21(75%)**	
QUICKI			
<0.3	**41(66.1%)**	**19(67.9%)**	**0.872**
>0.3	**21(33.9%)**	**9(32.1%)**	

Table 21 : Results of Liver Biopsy

Pathology	**No. of patients**	**%**
Fatty liver	**15**	**75**
NASH	**3**	**15**
Fibrosis	**2**	**10**

Table 22 : Severity wise distribution

Total	**Severity**		
	MILD	**MODERATE**	**SEVERE**
No.of Patients	**7**	**11**	**2**
% of patients	**35%**	**55%**	**10%**

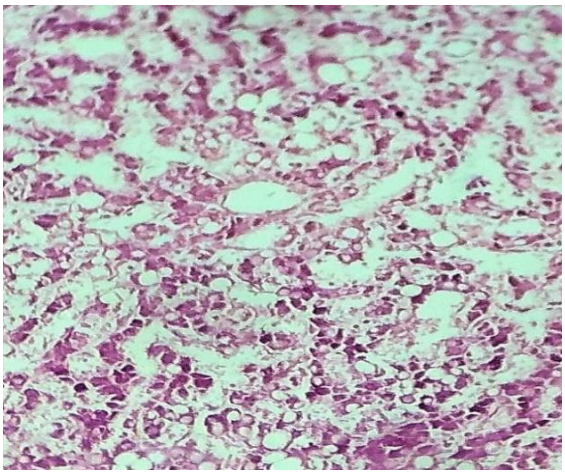

FIG-1)SHOWS FATTY LIVER

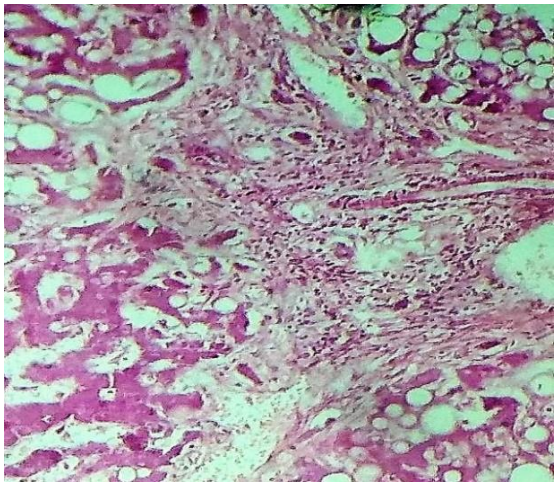

FIG-2)SHOWSSTEATOHEPATITIS

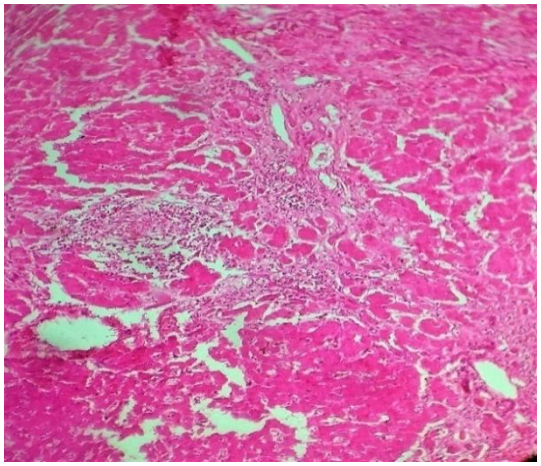

FIG-3)SHOWS LIVER INFLAMMATION AND FIBROSIS

IV. Discussion

NAFLD is a silent serious disease, which is becoming epidemic, such as its association with metabolic syndrome.In our study, there were no significant sex differences for incidence of NAFLD between the two groups .The mean age of patients in both the NAFLD and non-NAFLD groups was 55.37±10.95 and 57.25±13.22, respectively which was not statistically different . We also compared the frequency of NAFLD among different age groups which again did not show any significant differences.Previous studies by (Adams LA et al[11]) have shown that NAFLD can occur at any age, but since its prevalence increases with age, therefore it mostly affects people in their forties to sixties by (Akbar DH et al[12]).

The mean duration of DM was significantly lower in patients with NAFLD (10.87±4.99) as compared to patients without NAFLD (11.46±6.37), which indicated that duration did not have a significance on NAFLD.BMI was significantly higher in patients with NAFLD (25.30±3.62) than those without NAFLD (24.03±3.19). Most patients had abnormally high BMI's and 78% of patients had a BMI >25.0 kg/m2 (Overweight or obese according to NCEP ATPIII Guidelines). In our study particularly noteworthy is the preponderance of Central obesity in our patients with NAFLD. Thus, all but 04 patients (92%) had central obesity[1,2,3]. In our study, the waist/hip ratio was not significantly different between the two groups .
HbA1c was significantly higher with NAFLD group (83.9%) when compared to the non NAFLD group. Hyperglycemia has been reported in 20-75% of adult patients with NAFLD by (van Hoek B et al[15]), which was consistent with our study wherein hyperglycemia was seen in 70.3% of patients in NAFLD group .

Many studies have shown that insulin resistance has a critical role in the pathogenesis of NAFLD. 58 of the 62 patients (96%) fulfilled atleast one criterion for Dyslipidemia as per ATP III guidelines[4,5,6]. All of them(96%) had abnormalities that are characteristic of Insulin Resistance syndrome/ Metabolic Syndrome. (High TG and/or low HDL levels)[7,10]. Other studies have reported similar prevalences (20–80%)[8,13].20 patients out of 62 who underwent liver biopsy 15 patients had fatty liver , 3 had NASH and 2 had fibrosis[9,14].

V. Conclusion

The prevalence of NAFLD is high (68.9%) amongst T2DM patients affecting 59.7% males and 40.3% females.Age of the patient and duration of diabetes did not have a significant difference on the incidence of NAFLD .78% 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 . HbA1C , FBS and PLBS levels in the NAFLD group were significantly higher than the non NAFLD group which showed hyperglycemia increases the risk of developing NAFLD . 96% of the patients in the NAFLD group had dyslipidemia with 41.9% of patients having hypertriglyceridemia.

Serum transaminases were elevated in 58.3% of the NAFLD group with AST:ALT>1

Out of 90 patients that were studied 62 patients were diagnosed with NAFLD based on USG .Of the 62 patients with NAFLD there was a significant impact of BMI ,hyperglycemia, hypertriglyceridemia and insulin resistance on the incidence of NAFLD

The prevalence of NAFLD is high amongst T2DM patients and NAFLD should be actively sought out and treated in patients with diabetes.

Among the 20 patients who underwent liver biopsy fatty liver was seen in 75%, NASH in 15% and fibrosis in 10%. Liver biopsy also plays a role in studying the pathology of liver in NASH diagnosed by ultrasound.

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