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Profile of Non Alcoholic Fatty Liver Disease (NAFLD) In Healthy Young Adults.

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Abstract:
Aims and Objectives:
1) *To Study Occurrence of Non Alcoholic Fatty Liver Disease in Healthy Young Adults.*
2) *To Study Profile of subjects with Non Alcoholic Fatty Liver Disease in Healthy Young Adults.*
Materials and Methods:
Study Design: *A cross sectional study.*
Sample Size: *550 cases.*
Data Collection: *24 months (October 2013 To September 2015)*
Written informed consent was taken from the subjects after explaining the nature of the evaluation to them. Each person was subjected to a thorough history and physical examination as per the proforma.
Methodology: *History of Alcohol consumption, Jaundice, Medication, Family history of risk factors was obtained. Blood pressure and anthropometric measurements of the subjects were taken and biochemical investigations like fasting plasma glucose, fasting lipid profile, AST (SGOT) & ALT (SGPT). Ultrasonography of abdomen was done to detect fatty changes in the liver, which was performed by a single experienced radiologist who was blind to the study.*
Results: *Out of 550 subjects, 83(15.09%) had fatty liver. There was statistically significant difference in NAFLD and normal group in Non biochemical factors like blood pressure, BMI, Waist circumference, WHR and Family history of risk factors and Biochemical factors like Fasting plasma Glucose (FPG), SGPT, Total Cholesterol, Triglycerides, LDL, VLDL.*
Conclusion: *The present study concludes that the occurrence of NAFLD in healthy young adults was 15.09%. There is strong strength of association in developing into fatty liver with factors such as Waist-Hip ratio, waist circumference, body mass index (BMI), SGPT, total cholesterol, Triglycerides, LDL and VLDL.*
Keywords: *NAFLD: Non Alcoholic Fatty Liver Disease, Young healthy adults.*

I. Introduction

Nonalcoholic fatty liver disease (NAFLD) is emerging as a major public health problem[1]. It is tightly linked to obesity and thought to be the hepatic manifestation of the metabolic syndrome[2]. NAFLD has been defined as the accumulation of fat in the liver in the absence of recent or ongoing intake of significant amount of alcohol (<20 g per day in men and <10 g in women)[3]. It includes a wide spectrum of liver clinic-pathologic conditions, ranging from pure fatty steatosis (fatty infiltration in >5% of hepatocytes) which is apparently a benign condition to nonalcoholic steatohepatitis (NASH), which may progress to cirrhosis, liver failure, and hepatocellular carcinoma (HCC)[3]. NASH is characterized by biochemical evidence of hepatocellular damage (elevation of aminotransferase levels), histological findings of the type of alcoholic hepatitis (steatosis, lobular inflammatory cell infiltration, Mallory's hyaline, and fibrosis), and no other cause of liver damage[3]. Why do only some people with the metabolic syndrome develop NAFLD? And Conversely, why do only some manifestations of the metabolic syndrome appear in persons with NAFLD? While answer is presently unclear, it could have to do with differential insulin sensitivities of various tissues of the body[4].

The overall prevalence of NAFLD is 15-40% in Western countries while 9-40% in Asian countries[5]. The prevalence of NAFLD in adults has been reported to range from 10% to 24% worldwide and of NASH is 2% - 3% (range 1.2 – 4.8%)[6] NAFLD carries a 2%-50% risk for progressive fibrosis,30% risk for cirrhosis and 5% risk for hepato-cellular carcinoma[7]. 70% of patients with fatty liver have metabolic syndrome and 30% of patients with metabolic syndrome have fatty liver[6]. The prevalence ranges from 54% to 75% in obese adults, higher in boys than in girls and becomes as high as 90% in class III obese individuals[7]. It is described in the 60% of the subjects with mixed hyperlipidemia, and in 83% of those with both mixed hyperlipidemia and an elevated serum alanine aminotransferase (ALT)[3].

Recent data indicates high prevalence NAFLD in adolescents[3]. Cross-sectional studies in patients with NASH have shown that 30-40% of patients have advanced liver fibrosis at the time of presentation, whereas 10-15% have established cirrhosis[3]. Of patients with NASH 15% to 25% progress to cirrhosis and its complications over 10 to 20 years[8]. Some studies report a prevalence of HCC in NAFLD patients of 0% to 0.5% and 0% to 2.8% in NASH patients over a 20-year period[8].

Table: Risk factors of NAFLD[9,10]

Conditions with established association

- Obesity
- Diabetes mellitus
- Insulin resistance
- Dyslipidemia
- Metabolic syndrome
- DRUGS: Estrogens, Tamoxifen, Valprpoic acid, Vitamin A, Methotrxate, Isoniazid, CCB, Nucleoside Analogues.

Conditions with emerging association:

- Hypothyroidism
- Polycystic ovarian syndrome (pcos)
- Obstructive sleep apnea
- Hypopituitarism
- Hypogonadism
- Pancreato-duodenal resection

The clinical importance of primary NAFLD appears to rest on three main observations:

- It commonly occurs in the general population worldwide and among patients presenting with unexplained mild to moderate raised aminotransferase levels.
- It is not a sign or symptom of disease but it is a pathological condition that has the potential to progress to advanced hepatic and extrahepatic disease and to interact with other etiologies of liver disease .
- It may recur following orthotopic liver transplantation and poses a heavy burden of complications in the setting of major extrahepatic and liver-related surgery.

While it is well known that liver biopsy is the gold standard for diagnosing and determining the prognosis of NAFLD based on the severity of liver inflammation and fibrosis, it is hardly as routine a test as the ultrasound of the liver. Various simple serum biomarkers (singly and in combination) and imaging modalities (ultrasound, elastography) have been evaluated over the past decade as surrogates for liver biopsy.

Ultrasound remains the imaging modality of highest interest as it is widely available, non-ionising, and can be easily performed at a low cost.

There is increase in incidence of obesity, diabetes mellitus and hypertension, Insulin resistance in India in last two decades. It is logical to expect increase in incidence of NAFLD in India. There is limited data on the prevalence of NAFLD from India and patient present in late stages when they get sick. Majority of data comes from hospital based studies including small number of patients. Even National Programme are undergoing for prevention of NAFLD and it is proved that the mainstay of treatment is life style modification.

Therefore this study was planned to estimate the occurrence of NAFLD and to study their profile and guide the healthy young adults about preventive measures.

II. Aim And Objective

1) To Study Occurrence of Non Alcoholic Fatty Liver Disease in Healthy Young Adults.
2) To Study Profile of subjects with Non Alcoholic Fatty Liver Disease in Healthy Young Adults.

III. Material and Methodology

Ethics Committee Permission: The study was initiated after clearance from the institutional ethics committee(IEC), DMIMS (DU), Sawangi (M), Wardha.

Study Design: A cross sectional study.

Duration of study: 24 months (October 2013 To September 2015)

Inclusion Criteria:

- Non Alcoholic healthy young adults attending Acharya Vinobha Bhave Rural Hospital, Sawangi(Meghe), Wardha

Exclusion Criteria:

- Any quantity of alcohol consumption based on careful history.
- Known case of Hepatic disease, HBsAg, Anti HCV positivity, Jaundice
- People who did not give consent

IV. Methods

All subjects demographic records (information comprised of sex, age, and address) and history of alcohol use or any kind of addictions, hepatitis was collected.

Informed consent: - Written informed consent was taken from the subjects after explaining the nature of the evaluation to them.

Each person was subjected to a thorough history and physical examination, blood samples for FPG, Fasting Lipid Profile, SGOT, SGPT and USG of abdomen was done as per the proforma.

Ultrasonography study:

- All persons underwent Ultrasound (USG) of the abdomen to detect fatty changes in the liver, which was performed by a single experienced radiologist who was blind to the study.
- It was performed using a high resolution B-mode ultrasonography system (Prosound Alpha 7) manufacturer having an electric linear transducer mid frequency of 3-5MHz.
- Diagnosis of fatty infiltration of liver parenchyma was made by following findings on B-mode ultrasound images with optimum gain settings:

a) Increase in echogenicity of liver compared to renal cortex 'bright liver'
b) Tightly packed echoes
c) Decrease in the amplitude of echoes with depth (posterior beam attenuation)
d) Nonvisualization of echogenic walls of portal vein branches.

- Based on these findings, a quantitative grading system for hepatic steatosis has been proposed:
  - **Grade I:** Simple increase in echogenicity of liver parenchyma.
  - **Grade II :** Obscuration of echogenic walls of portal vein branches due to fatty infiltrarfon.
  - **Grade III :** Obscuration of the diaphragmatic outline by echogenic liver parenchyma.
- Using these criteria, the reported sensitivity of ultrasound in detection of liver steatosis varies from 60% to 94%.

The study subjects were divided into 2 groups:

1) NAFLD : Subjects with USG evidence of fatty changes in liver.
2) Normal : Subjects without any USG evidence of fatty changes in liver.

Anthropometric measurements:

Anthropometric features including weight, height, BMI, waist circumference (WC), hip circumference (HC) were measured by standard method.

V. Observations

Table 1 : Non biochemical risk factors of study population

Baseline Characteristics	NAFLD(N=83)	Normal(N=467)	p-value	Odd's Ratio (CI)
F/H/O Risk Factors	64(77.11%)	296(63.38%)	0.015,S	1.94 (1.12-3.35)
SBP (mm of Hg)	130.09±9.51	116.30±8.83	0.000,S	-
DBP (mm of Hg)	81.22±6.45	73.16±5.61	0.000,S	-
WC (cm)	88.34±10.43	79.40±8.27	0.000,S	6.53 (3.97-10.75)
BMI (kg/m^2)	26.42±4.13	23.22±3.34	0.000,S	4.09 (2.39-6.99)
WHR	1.00±0.14	0.83±0.09	0.000,S	10.92 (6.03-19.77)

Table 2 : Biochemical parameters of study population

Baseline Characteristics	NAFLD (N=83)	Normal (N=467)	p-value	Odd's Ratio (CI)
FPG	101.63±11.33	90.69±10.39	0.0001,S	-
SGOT	30.37±12.03	28.91±12.05	0.11,NS	1.92 (1.09-3.39)
SGPT	32.51±9.77	21.12±11.04	0.04,S	3.50 (1.98-6.19)
SGOT/SGPT	1.41±0.29	1.49±0.45	0.18,NS	0.81 (0.37-1.78)
TC	221.50±38.17	165.05±31.29	0.0001,S	17.46 (10.11-30.15)
TG	188.09±67.96	94±44.57	0.0001,S	22.18 (12.63-38.95)
HDL	41.75±6.04	40.23±6.88	0.060,NS	0.53 (0.27-1.05)

LDL	137.62±36.35	105.66±27.94	0.0001,S	8.99 (4.17-19.37)
VLDL	32.49±14.56	19.37±7.97	0.0001,S	8.33 (3.92-17.70)

Table 3: Family history of risk factors

Risk Factors	Total		□2 -value
	NAFLD	Normal	
Hypertension (HTN)	3 (3.61%)	62 (13.28%)	6.79, p=0.009,S
Diabetes Mellitus (DM)	13 (15.66%)	53 (11.35%)	1.07, p=0.30,NS
Obesity	4 (4.82%)	18 (3.85%)	0.11, p=0.73,p>0.05
Hypertension + DM	2 (2.41%)	40 (8.57%)	4.71, p=0.02,S
Hypertension + Obesity	9 (10.84%)	50 (10.71%)	0.00, p=1.00,NS,p>0.05
DM + Obesity	10 (12.05%)	33 (7.07%)	1.45, p=0.22,NS,p>0.05
HTN + DM + Obesity	23 (27.71%)	40 (8.57%)	11.97, p=0.0005,S,p<0.05
None	19 (22.89%)	171 (36.62%)	4.66, p=0.03,S,p<0.05
Total	83 (100%)	467 (100%)	69.42, p=0.0001,S,p<0.05

VI. Discussion

Total number of subjects enrolled in the study after taking informed consent were 550 out of which 83 had evidence of fatty changes in liver on ultrasonography of abdomen and 467 had no evidence of fatty changes in liver on ultrasonography of abdomen.

1) Age:

In our study the mean age of subjects in NAFLD group was 23.39 ± 3.51 years and normal group was 22.77 ± 2.97 years. There was no statistically significant difference between the two groups (P >0.05). This means the subjects included in study were age matched. Similarly, **Kirovski et al** in their study of 506 subjects the mean age of subjects in NAFLD group was 57.0± 16.4 years and in normal group was 52.2 ± 18.4 years (P= 0.098)[11]. **Asabamaka Onyekwere C et al** in their study of 150 subjects the mean age of subjects in NAFLD group was 52.5 ± 10.9 years and in normal group was 54.0 ± 9.0 (P = 0.4).[12]**Kim et al** in their study of 768 subjects the mean age of subjects in NAFLD group was 53.2 ± 9.8 years and in normal group was 51.3 ± 10.0 years (P = 0.3).[13] In all the studies the subjects were age matched.

2) Gender :

In our study of 550 subjects, 83 subjects were in NAFLD group out of which 62 (71.26%) were males and 21 (24.14%) were females. 467 subjects in normal group out of which 326 (69.81%) were males and 141 (30.19%) were females. There was no statistically significant difference between the two groups (P = 0.36). This means that the subjects in study were sex matched. Similarly, **Das et al** in their study of 224 subjects, 90 subjects were in NAFLD group out of which 63 were male and 27 were female. 134 subjects were in normal group out of which 81 were male and 53 were female (P value – Not Significant).[15]**Bedogni et al** in their study of 598 subjects, 311 subjects were in NAFLD group of out of which 194 were male and 117 were female whereas 287 subjects were in normal group out of which 156 were male and 131 were female (P= 0.56).[16]In all these studies the subjects were sex matched.

3) Family history of (F/H/O) risk factors :

In our study of 550 subjects, 83 subjects were in NAFLD group out of which 64 (77.11%) subjects had family history of risk factors and 19 (22.89%) subjects did not have family history of risk factors.

467 subjects were in normal group out of which 296 (63.38%) had family history of risk factor and 171 (36.62%) did not have family history of risk factors.

This distribution of subjects was on the basis of presence or absence of F/H/O risk factors. The difference in the two groups was statistically significant (P = 0.001). The Odds Ratio was 1.94, which means that risk of developing fatty liver in subjects with positive family history of risk factors is 1.94 times higher.

Furthermore the F/H/O risk factors was studied individually. 83 subjects were in NAFLD group out of which 3 (3.61%) had hypertension , 13 (15.66%) had diabetes mellitus, 4 (4.82%) had obesity, 2 (2.41%) had hypertension + diabetes mellitus, 9 (10.84%) had hypertension + obesity, 10 (12.05%) had diabetes mellitus + obesity, 23 (27.71%) had hypertension + diabetes mellitus + obesity, 19 (22.89%) did not have family history of risk factors.

Out of 467 subjects in normal group, 62 (13.28%) had hypertension, 53 (11.35%) had diabetes mellitus, 18 (3.85%) had obesity, 40 (98.57%) had hypertension + diabetes mellitus, 50 (10.71%) had hypertension + obesity, 33 (7.07%) had diabetes mellitus + obesity, 40 (8.58%) had hypertension + diabetes mellitus + obesity, 171 (36.62%) did not have family history of risk factors.

The difference between the two groups was statistically significant for Hypertension (P=0.009), hypertension + diabetes mellitus (P-0.02), hypertension + diabetes mellitus + obesity (P-=0.0005) and also in those who did not have any family history of risk factors (P=0.03).

The difference between the two groups was statistically not significant for diabetes mellitus (P-0.30), obesity (P -0.73), hypertension + obesity (P-1.00) and diabetes mellitus + obesity (P=0.22).

4) Blood pressure:

In our study the mean systolic blood pressure (SBP) in NAFLD group was 130 .09 ± 9.51 mm of Hg and in normal group was 116.30 ± 8.83 mm of Hg

The mean diastolic blood pressure (DBP) in NAFLD group was 81.22 ± 6.45 mm of Hg and in normal group was 73.16 ± 5.61 mm of Hg.

The difference in the mean systolic blood pressure and diastolic blood pressure of the two groups was statistically significant (P <0.05).

Similarly **Kim et al** in their study showed that the mean SBP in NAFLD group was 134± 18.6 mm of Hg and in normal group was 128.6 ± 18.6. The mean DBP in NAFLD group was 82.1 ± 11.9 mm of Hg and in normal group was 79.1 ± 12.4 (P <0.005).[13] **Bedogni et al** in their study showed that the mean SBP in NAFLD group was 135 ± 20 mm of Hg and in normal group was 130 ± 20 mm of Hg. The mean DBP in NAFLD group was 85± 5 mm of Hg and in normal group was 80 ± 5mm of Hg (P < 0.001, Significant).[16]

5) Obesity: Obesity was assessed by Body Mass Index (BMI), Waist Circumference (WC) and Waist –Hip Ratio (WHR)

A) Body mass index (BMI):

In our study the mean BMI in NAFLD group was 26.42 ± 4.13 kg/m^2 and in normal group was 23.22 ± 3.34 kg/m^2.

The difference in the mean BMI of the two groups was statistically significant (P < 0.0001). The Odds Ratio was 4.09 which means that there is 4.09 times higher risk of developing fatty liver in subjects with higher BMI.

Similarly, **Chen et al** in their study 48.9% subjects in NAFLD group had BMI > 25 kg/m^2 and 51.1% subjects in normal group had BMI > 25 kg/m^2 where 12.7% subjects in NAFLD group had BMI < 25 kg/m^2 and 87.3% subjects in normal group had BMI < 25 kg/m^2 (OR- 6.58)[17] **Kirovski et al** in their study showed that the mean BMI in NAFLD group was 28.7± 5.9 kg/m^2 in normal group was 24.8 ± 3.6 kg/m^2 (P < 0.0001)[11] **Asabamaka Onyekwere C et al** in their study showed that the mean BMI in NAFLD group was 33.9 ± 8.3 kg/m^2 and in normal group was 30 ± 7 kg/m^2 (P = 0.13)[12] **Kim et al** in their study showed that the mean BMI in overweight subjects in NAFLD group was 27.1± 1.3 kg/m^2 and in normal group was 26.5 ± 1.2 kg/m^2. The mean BMI in normal weight subjects in NAFLD group was 23.4 ± 1.3 kg/m^2 and in normal group was 22.6 ± 1.6 kg/m^2 (P < 0.001).[13]

B) Waist circumference (WC) :

In our study the mean of WC in NAFLD group was 88.34 ± 10.43 cm and in normal group was 79.40 ± 8.27 cm.

The difference in the mean WC of the two groups was statistically significant (P < 0.0001). The Odds Ratio was 6.53 which means that there is 6.53 times higher risk of developing fatty liver in subjects with high WC.

Similarly, **Kirovski et al** in their study of 506 subjects the mean WC in NAFLD group was 102.2 ± 13.4cm in normal group was 86.9 ± 13.1cm (P < 0.0001).[11] **Das et al** in their study the mean BMI in NAFLD group was 80.01 ± 12.10cm and in normal group was 75.0 ± 9.01cm. (P <0.05)[15] **Kim et al** in their study the mean WC in subjects in NAFLD group was 87.8 ± 5.9cm in normal group was 81.7 ± 7.0cm (P < 0.001).[13]

C) Waist hip ratio (WHR) :

In our study the mean WHR in NAFLD group was 1.00 ± 0.14 and in normal group was 0.83 ± 0.09.

The difference in the mean WHR of the two groups was statistically significant (P < 0.0001). The Odds Ratio was 10.92 which means that there is 10.92 times higher risk of developing fatty liver in subjects with higher WHR. Similarly, **Kirovski et al** in their study showed that the mean of WHR in NAFLD group was 0.98 ± 0.08 and in normal group was 0.92 ± 0.10 (P < 0.0002).[11] **Kim et al** in their study showed that the mean WHR in NAFLD group was 0.88 ± 0.04 and in normal group was 0.85 ± 0.05 (P < 0.001).[13]

6) Fasting plasma glucose (FPG) :

In our study of 550 subjects, 83 subjects were in NAFLD group with mean FPG of 101.63 ± 11.33 mg/dl and 467 subjects were in normal group with mean of 90.69 ± 10.39 mg/dl.

The difference in the mean FPG of the two groups was statistically significant (P < 0.0001). This shows that there is significant correlation of developing fatty liver with rising trend of sugar levels.

Similarly, **Kim et al** in their study showed that the mean FPG in NAFLD group was 95.3 ± 10.2 mg/dl and in normal group was 91.7 ± 8.6 mg/dl (P < 0.001).[13] **Das et al** in their study showed that the mean FPG in NAFLD group was 86± 25 mg/dl and in normal group was 80 ± 20 mg/dl (P =003). [15] **Bagheri Lankarani et al** of 519 subjects 176 were in NAFLD group out of which 78 (44.3%) had high FPG levels and 98 (55.7%) had normal FPG. 643 subjects were in normal group out of which 89 (13.8%) had high FPG and 554 (86.2%) had normal FPG levels (P < 0.001).[14]

7) SGOT (AST) :

In our study the mean of AST in NAFLD group was 30.37 ± 12.03 IU/L and in normal group was 28.91 ± 12.05 IU/L.

The difference in the mean AST of the two groups was statistically not significant (P < 0.11). The Odds Ratio was 1.92 which means that there is 1.92 times higher risk of developing fatty liver in subjects with higher AST values.

Similarly, **Kirovski et al** in their study showed the mean of AST in NAFLD group was 31.6 ± 15.7 IU/L and in normal group was 28.5 ± 14.0 (P= 0.209).[11]

8) SGPT (ALT) :

In our study the mean of ALT in NAFLD group was 32.51 ± 9.77 IU/L and in normal group was 21.12 ± 11.04 IU/L.

The difference in the mean ALT of the two groups was statistically significant (P < 0.0001). The Odds Ratio was 3.50 which means that there is 3.50 times higher risk of developing fatty liver in subjects with high ALT values.

Similarly, **Kim et al** in their study the mean ALT in subjects with NAFLD group was 32.9 ± 19.1 IU/L and in normal group was 21.6 ± 10.9 IU/L (P < 0.0001).[13] **Kirovski et al** in their study the mean ALT in subjects with NAFLD group was 23.2 ± 22.1 IU/L and in normal group was 15.0 ± 8.2 IU/L (P < 0.001) (OR – 1.02)[11]. **Chen-Chung Fu et al** in their study showed the mean ALT values in NAFLD group was 30.7 ± 31.4 IU/L and in normal group was 15.9 ± 8.8 IU/L (P <0.05)[17]

9) SGOT / SGPT ratio (AST / ALT ratio) :

In our study the mean of AST/ALT ratio in NAFLD group was 1.41 ± 0.29 and in normal group was 1.49 ± 0.45.

The difference in the mean AST/ALT ratio of the two groups was statistically not significant (P= 0.85). The Odds Ratio was 0.81 which was not significant.

Similarly, **Kirovski et al** in their study showed that the mean AST/ALT ratio in subjects with NAFLD group was 1.76 ± 0.79 and in normal group was 2.11 ± 0.94.(P=0.019, OR- 0.39)[11]

10) Fasting lipid profile :

A) Total cholesterol (TC) :

In our study the mean TC in NAFLD group was 221.50 ± 38.17 mg/dl and in normal group was 165.05 ± 38.17 mg/dl and in normal group was 165.05 ± 31.29.

The difference in the mean TC of the two groups was statistically significant (P < 0.05). The Odds Ratio was 17.46 which means that there is 17.46 times higher risk of developing fatty liver in subjects with high TC values.

Similarly, **Kim et al** in their study the mean TC in NAFLD group was 210.2 ± 32.9 mg/dl and in normal group was 119.4 ± 33.2 mg/dl (P < 0.0001).[13] **Kirovski et al** in their study the mean TC in NAFLD group was 197.7 ± 47.4 mg/dl and in normal group was 206.0 ± 49.0 mg/dl (P = 0.303).[11] **Chen – Chung Fu et al** in their study showed the mean TC in NAFLD group was 162 ± 31 mg/dl and in normal group was 150 ± 26 mg/dl.[6] **Bagheri Lankarani et al** in their study of 819 subjects, 176 were in NAFLD group out of which 27 (15.7%) had high TC values and 149 (84.3%) had normal TC values. 463 subjects were in normal group out of which 61 (9.5%) had high total cholesterol values and 582 (90.5%) had normal TC values.[14]

B) Triglyceride (TG) :

In our study the mean of TG in NAFLD group was 188.09 ± 67.96 mg/dl and in normal group was 94 ± 44.57 mg/dl.

The difference in the mean TG of the two groups was statistically significant (P < 0.0001). The Odds Ratio was 22.18 which means that there is 22.18 times higher risk of developing fatty liver in subjects with higher TG values.

Similarly, **Kim et al** in their study showed that the mean TG values in NAFLD group was 207.2 ± 131.7 mg/dl and in normal group was 147.6 ± 111.6 mg/dl (P < 0.001).[13] **Chen-Chung Fu et al** in their study showed that the mean TG values in NAFLD group was 103 ± 55 mg/dl and in normal group was 75 ± 35 mg/dl.[6] **Bagheri Lankarani et al** in their study of 919 subjects showed 176 subjects were in NAFLD group out of which 105 (59.7%) had high TG values and 71 (40.3%) had normal TG values. 643 subjects were in normal group out of which 182 (28.3%) had high TG values and 461 (717%) had normal TG values (P < 0.001)[14].

C) High density lipoprotein (HDL) :

In our study the mean HDL in NAFLD group was 41.75 ± 6.04 mg/dl and in normal group was 40.223 ± 6.8 mg/dl.

The difference in the mean HDL of the two groups was statistically not significant (P < 0.05). The Odds Ratio was 0.53 which was not significant. This may be due to subjects studied were healthy young adults.

However, **Kirovski et al** in their study the mean HDL in NAFLD group was 47 13.1 mg/dl and in normal group was 60.7 ± 19.4 mg/dl (P < 0.0001).[11] **Bagheri Lankarani et al** in their study of 819 subjects, 176 subjects were in NAFLD group out of which 64 (36.4%) had low HDL values and 112 (63.6%) had normal HDL values. 643 subjects were in normal group out of which 237 (36.8%) had low HDL values and 406 (63.2%) had normal HDL values.[14] **Kim et al** in their study showed that the mean HDL in NAFLD group was 43.5 ± 10.8 mg/dl and in normal group was 48.7 ± 12.6 mg/dl (P = 0.002).[13] **Chen Chung Fu et al** in their study showed that the mean HDL values in NAFLD group was 55 ± 10 mg/dl and in normal group was 60 ± 14 mg/dl.[6]

D) Low density lipoprotein (LDL):

In our study the mean LDL in NAFLD group was 137.62 ± 36.35 mg/dl and in normal group was 105.66 ± 27.94 mg/dl.

The difference in the mean LDL of the two groups was statistically significant (P - 0.0001). The Odds Ratio was 8.99 which means that there is 8.99 times higher risk of developing fatty liver in subjects with high LDL values.

Kirovski et al in their study showed that the mean LDL in NAFLD group was 109.8 ± 38.2 mg/dl and in normal group was 105.8 ± 37.9 mg/dl (P= 0.528).[11] **Kim et al** in their study showed that the mean LDL in NAFLD group was 129.4 ± 29.6 mg/dl and in normal group was 122.9 ± 30.7 mg/dl (P= 0.02).[13] **Bagheri Lankarani et al** in their study of 819 subjects. 176 subjects were in NAFLD group out of which 18 (10.2%) had high LDL values and 153 (89.8%) had normal LDL values. 643 subjects were in normal group out of which 40 (6.2%) had high LDL values and 603 (93.8%) had normal LDL values.[14]

E) Very low density lipoprotein (VLDL) :

In our study the mean of VLDL in NAFLD group was 32.49 ± 14.56 mg/dl and in normal group was 19.37 ± 7.97 mg/dl.

The difference in the mean VLDL of the two groups was statistically significant (P - 0.009). The Odds Ratio was 8.33 which means that there is 8.33 times higher risk of developing fatty liver in subjects with high VLDL levels.

However, **Kirovski et al** in their study showed that the mean VLDL in NAFLD group was 41. 0 ± 19.5 mg/dl and in normal group was 39.8 ± 18.4 mg/dl (P= 0.714).[11]

VII. Conclusion

The present study concludes that the occurrence of NAFLD in healthy young adults was 15.09%. There is strong strength of association of developing fatty liver with factors such as Waist-Hip ratio, waist circumference, body mass index (BMI), SGPT, total cholesterol, Triglyceride, LDL and VLDL.

VIII. Limitations

1. The diagnosis of NAFLD was based on liver ultrasonography. Magnetic resonance spectroscopy and liver biopsy are better tools for defining NAFLD and could be considered as "gold standards". Conversely, ultrasonography is by far the most common method of diagnosing NAFLD in clinical practice. It is simple to perform, noninvasive, cost effective and does not entail any radiation hazard, and could also be used in the epidemiological studies.
2. The sample size of our study was relatively less (n=550).

IX. Recommendations

1. Nonalcoholic fatty liver disease (NAFLD) is emerging as a major public health problem and hence it is recommended to consider the possibility of NAFLD in patients presenting with associated conditions and risk factors for NAFLD.
2. Ultrasonography of abdomen and Liver function test must be adviced in all high risk population as NAFLD is asymptomatic in most of the cases.
3. Depending upon ultrasonography of abdomen and Liver function test cases should be evaluated further (Liver biopsy / Elastography) and preventive measures can be implemented.

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