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Original Research Article

Study of Non-Alcoholic Fatty Liver Disease in Andhra Pradesh Population

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Abstract:

Background: Non-alcoholic fatty liver disease is a benign steatosis disease that leads to steatohepatitis, cirrhosis of the liver, and hepatic neoplasia. In the majority of cases of such disease, patients remain asymptomatic despite progressive liver disease. Hence, clinical manifestations and biochemical profiles are ruled out.

Method: 95 NAFLD Patients were studied for USG, lipid profile, HbA_1C, routine blood examination, blood pressure recorded by sphygmomanometer, and ECG recorded (if necessary) to rule out cardiac co-morbidities.

Results: 19 (20%) were in grade I, 44 (46.3%) had in grade II, and 32 (33.6%) had in grade III, NAFLD. In the BMI study, 59 (62%) had 22.8 to 23.2, 36 (37.8%) were 23.3 to 242, 35 (36.2%) were pre-diabetic, 60 (63.1%) were diabetic, 25 (26.3%) were norma-tensive, 70 (73.6%) were hypertensive, 71 (74.7%) were hyperlipidemic, 26 (27.3%) had IHD, and 4 (4.20%) had MI.

Conclusion: The prevalence of 3rd grade NAFLD among type II DM and dyslipidaemia is alarming. Hence, high-grade NAFLD must be treated efficiently to avoid morbidity and mortality because the liver is the largest metabolic centre of the body.

Keywords: USG, grades of NAFLD, dyslipidaemia, type II DM, hypertensive.

Introduction

NAFLD is a common benign finding in ultrasonography studies, but it is associated with obesity, type II DM, dyslipidaemia, and hypertension. NAFLD includes patients with simple steatosis, steatohepatitis (non-alcoholic steato-hepatitis). NAFLD has a higher risk of progressing to liver cirrhosis or hepatocellular carcinoma (HCC) [1].

The term NASH was introduced by Ludwia et al. in 1980 and described the histological changes indistinguishable from alcoholic hepatitis with no or insignificant (less than 20g / day) alcohol intake [2]. Some of the patients develop hepatic oxidative stress and the recruitment of various cytokines, leading to hepatic inflammation and/or fibrosis, thus setting the stage for future complications such as cirrhosis and hepatocellular carcinoma (HCC). From a pathogenesis point of view, NAFLD is caused by the intake of some drugs, surgery, or total parental nutrition [3].

The prevalence of NAFLD is estimated to be 20% to 30% and 90% in obese subjects globally [4]. It is also observed that simple steatosis with no inflammation or fibrosis is associated with liver-related mortality; hence, an attempt is made to evaluate the grades of NAFLD and associated clinical manifestations with the biochemical profile.

Material and Method

95 (Ninety-five) patients who visited the medicine department of Kurnool Medical College, Kurnool, Andhra Pradesh, and were studied.

Inclusion Criteria: Patients aged between 20 to 65 years with symptoms of hepatic steatosis, cirrhosis of the liver, and diabetic mellitus were selected for study.

Exclusion Criteria: Alcoholic, hemochromatosis, hydatid cyst, presence of HBSAg, and immunocompromised patients were excluded from the study.

Method: Every patient underwent a USG, routine blood examination, lipid profile, HBA1c, and BMI. A detailed history of every patient was recorded. The ECG was recorded (if required). Blood pressure was recorded with a sphygmomanometer.

The duration of the study was from June 2023 to December 2023.

Statistical analysis: Various grades of fatty liver, clinical manifestations, and biochemical profiles were classified by percentage.

The statistical analysis was carried out using SPSS software. The ratio of males and females was 2:1.

Observation and Results

Table 1: Study of grade of non-alcoholic fatty liver: 19 (20%) grade-I, 44 (46.3%) grade-II, and 32 (33.6%) grade-III.

Table 2: Clinical manifestations of non-alcoholic fatty liver

- Body mass index: 59 (62.1%) had 22.8 to 23.2, and 36 (37.8%) had 23.1 to 24.2.
- Status of type II DM: 35 (36.8%) were pre-diabetic, and 60 (63.1%) were diabetic.
- Status of Blood Pressure: 25 (26.3%) were normotensive, and 70 (73.6%) were hypertensive.
- 71 (74.7%) were hyperlipidemic, 26 (27.3%) had ischemic heart disease (IHD), and 4 (4.20%) had myocardial infarction (MI).

Table 3: Mean Value of Biochemical Profile – 224 (± 5.8) total cholesterol, 249 (± 9.8) triglyceride, 42.6 (± 2.4) HDL, 130 (± 10.3) LDL, 52.4 (±3.4) AST, 65.2 (± 4.6) ALT, 10.4 (± 26) ALP, 3.46 (±0.10) S. albumin, 0.92 (±0.68) Total bilurubin, 134 (10.4%) Fasting Blood sugar, 9.12 (± 3.2) HbA1c

Table 1: Study of grades of Non-Alcoholic Fatty liver

Sl. No	Grades of NAFLD	No. of patients (95)	Percentage (%)
1	Grade-I	19	20
2	Grade-II	44	46.3
3	Grade-III	32	33.6

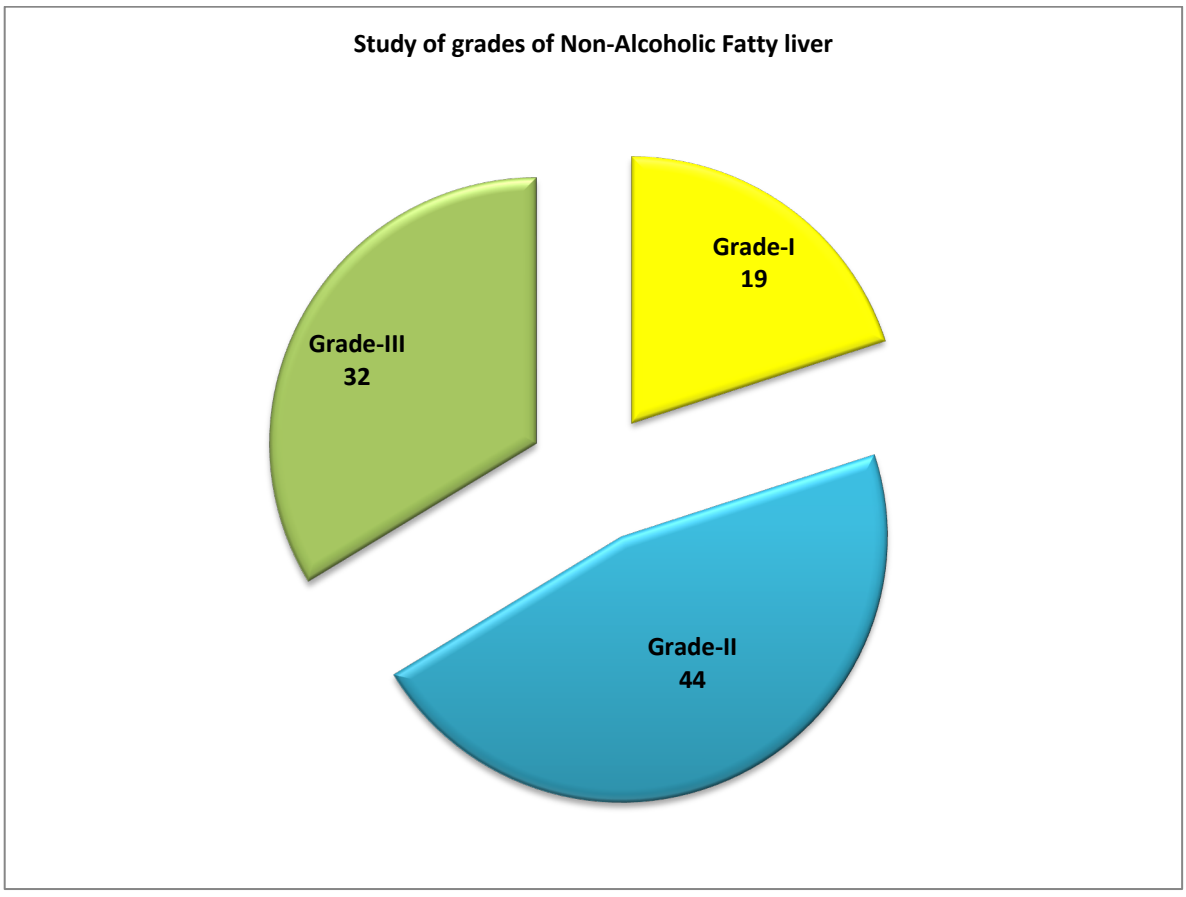

Figure 1: Study of grades of Non-Alcoholic Fatty liver

Table 2: Clinical manifestations of Non-Alcoholic fatty liver

Sl. No	Clinical Manifestation	No. of Patients (95)	Percentage (%)
1	Body Mass Index (BMI) a-22.8 to 23.2	59	62.1
	b-23.3 to 24.2	36	37.8
2	Status type-II DM		36.8

	a – Pre-diabetic	35	
	b – Diabetic	60	63.1
3	Status of Blood Pressure a – Normatensive	25	26.3
	b – Hypertensive	70	73.6
4	Hyper-lipidemic	71	74.7
5	Ischemic Heart Disease (IHD)	26	27.3
6	Myocardial Infarction (MI)	4	4.20

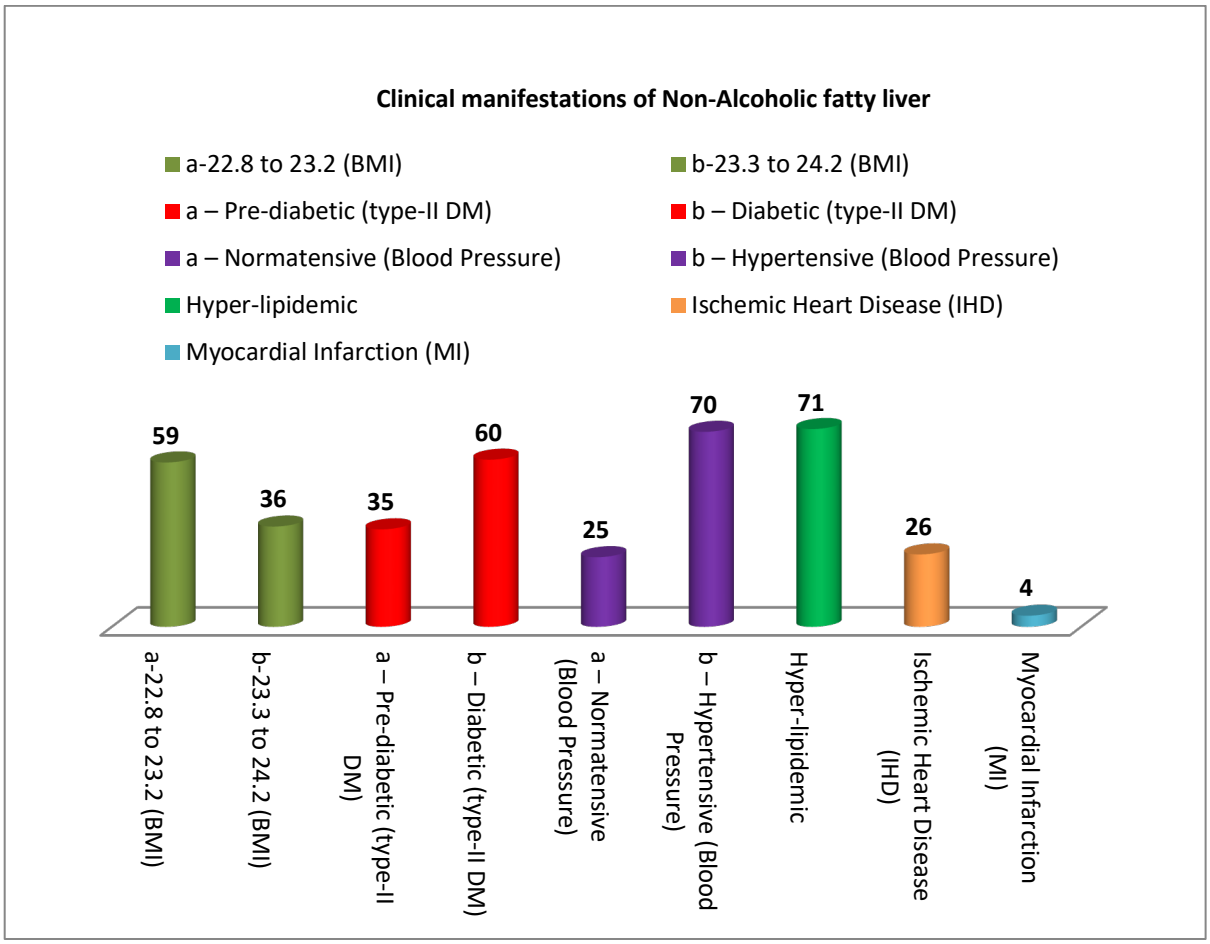

Figure 2: Clinical manifestations of Non-Alcoholic fatty liver

Table 3: Biochemical profile of Non-Alcoholic Fatty liver

Sl. No	Biochemical profile	Mean Value (±SD)
1	Total Cholesterol	224 (± 5.8)
2	Triglyceride	249 (± 9.8)
3	HDL	42.6 (± 2.4)
4	LDL	130 (±10.3)
5	AST	52.4 (± 3.4)
6	ALT	65.2 (±4.6)
7	ALP	10.4 (±2.6)
8	Serum Albumin	3.46 (±0.10)
9	Total Biliurubin	0.92 (±0.68)
10	Fasting Blood Sugar	134 (±10.4)
11	HA1C	9.12 (±3.2)

ALP = Alkaline Phosphatise, ALT = Alanine amino
LDL = Low Density Lipoprotein Transfarase, HbA1C = Haemoglobin A1c
AST = Aspirate Amino transfarase lipoprotein

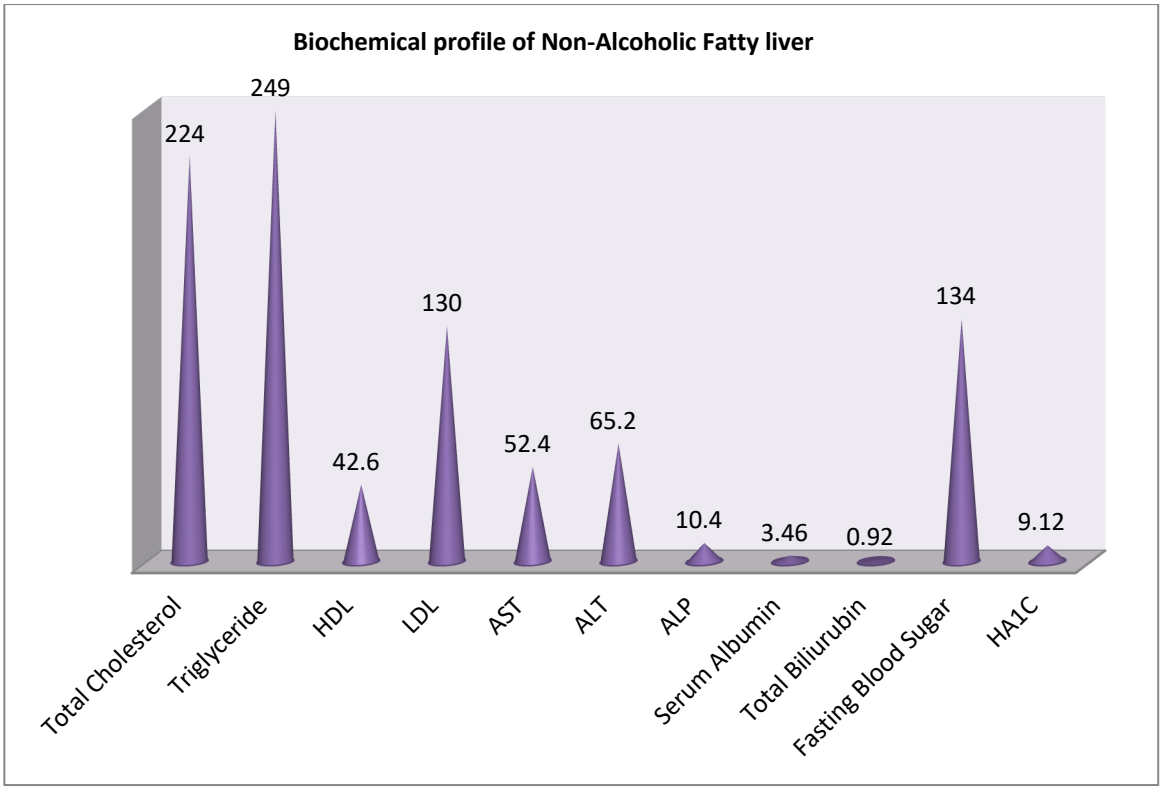

Figure 3: Biochemical profile of Non-Alcoholic Fatty liver

Discussion

In the present study of non-alcoholic fatty liver disease in Andhra Pradesh Population was 19 (20%) grade-I, 44 (46.3%) grade-II, 32 (33.6%) grade-III (Table-1). The clinical manifestation were body mass index – 59 (62%) had 22.8 to 23.2, 36 (37.8%) had 23.3 to 24.2, Status of type-II DM was 35 (36.2%) were pre-diabetic, 60 (63.1%) were diabetic, status of blood pressure was 25 (26.3%) were normotensive, 70 (73.6%) were hypertensive, 71 (74.7%) were hyperlipidemic, 26 (27.3%) were IHD, 4 (4.20%) had MI (Table-2).

The Bio-chemical profile was – 224 (±5.8) was mean values total cholesterol, 249 (±9.8) Triglyceride, 42.6 (±2.4) HDL, 130 (±10.3) LDL, 52.4 (±3.4) AST, 65.2 (±4.6) ALT, 10.4 (±2.6) ALP, 3.46 (± 0.10) serum albumin, 0.92 (± 0.68) Total Bilurubin, 134 (± 10.4) Fasting blood sugar, 9.12 (±3.2) HbA1C (Table-3). These findings are more or less in agreement with previous studies [5,6,7].

NAFLD is associated with metabolic syndrome, which is characterised by insulin resistance, HTN, Cholesterol abnormality, increased risk of blood clotting, type-II DM, obesity, elevated serum triglyceride, and reduced HDL which has greater risk of heart diseases, stroke and liver related diseases [8]. Although, the exact cause of NAFLD is still unclear but it is associated with variations in lipid metabolism [9]. It is also reported that NAFLD is the common cause of chronic liver diseases or chronic viral hepatitis [10]. Histological spectrum of NAFLD has no pathological changes which can definitively distinguish NAFLD from alcoholic liver diseases thus accurate alcohol history is essential to alcoholic liver disease. Insulin resistance factor is believed to be a significant role that leads to increased lipolysis in peripheral adipose tissue and increased uptake of fatty acids by hepatocytes.

The end result is an increase in fatty acids and triglycerides in the hepatocytes leading to steatosis. Hence insulin resistance is almost universal factor in patients with NAFLD and is related to an imbalance between pro-insulin (adiponectin) and anti-insulin cytokine (TNF-α) [11] [12].

It is also reported that, high prevalence of NAFLD, is due to rapid industrialization, sedentary lifestyle, obesity-DM, and junk-food intake in developing countries.

Summary and Conclusion

Present study of NAFLD is associated with obesity, diabetes and metabolic syndrome, which are the major causes of morbidity and mortality because simple steatosis carries a benign prognosis but, in the majority of cases, will have hepatocellular carcinoma.

Although liver biopsy remains the gold standard for disease assessment, the development of risk scores and biomarker panels has But this demands further pathophysiological, genetic, nutritional, environmental, and hormonal studies because the exact pathogenesis of NAFLD is still unclear.

Limitation of study: Due to the tertiary location of the research centre, the small number of patients, and the lack of the latest techniques, we have limited findings and results. This research paper has been approved by the ethical committee of Kurnool Medical College, Kurnool, Andhra Pradesh.

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