Section: General Medicine

Original Research Article

STUDY OF NON-ALCOHOLIC FATTY LIVER DISEASE

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Abstract

Background: Non-alcoholic fatty liver disease is predominantly observed in type-II diabetes and obese populations. Most of them are asymptomatic despite progressive liver disease; hence, associated clinical manifestations have to be ruled out. **Materials and Methods:** 50 (fifty) NAFLD Patients were studied for USG lipid profile, HbA1C, routine blood examination, blood pressure recorded by sphygmomanometer, and ECG recorded (if necessary) to rule out cardiac co-morbidities. **Result:** 10 (20%) were grade I, 23 (46%) had grade II, 17 (34%) had grade III NAFLD, BMI 23.3 to 23.2 in 31 (62%), 23.3 to 24.2 in 19 (38%) 18 (36%) were pre- diabetic, 36 (64%) were diabetic, 13 (26%) were normotensive, 37 (74%) had HTN, 14 (28%) had IHD, and 2 (4%) had MI and elevated biochemical profiles. **Conclusion:** In the present pragmatic study, it is observed that 3rd grade NAFLD among type II DM and hyperlipidemia have a high risk of morbidity and mortality. Such patients must be treated efficiently.

INTRODUCTION

The liver is the largest metabolic center of the body. Non-alcoholic fatty liver disease is nothing but an excess accumulation of triglyceride in hepatocytes. A simple steatosis, which is often clinically stable in non-alcoholic steatohepatitis (NASH) which may progress into cirrhosis of the liver.[1] Non-alcoholic fatty liver disease is more significant in the diabetic and obese population. Non-alcoholic fatty liver disease (NAFLD) includes simple steatosis, which is more advanced. It has a higher risk of progression of cirrhosis of the liver or hepatocellular carcinoma. The metabolic syndrome with insulin resistance develops hepatic steatosis due to increased lipolysis and increased delivery of fatty acids from adipose tissue to the liver.[2] Some NAFLD patients with hepatic steatosis develop oxidative stress and recruitment of various cytokines, leading to hepatic inflammation and/or fibrosis.[3] It is presumed that NAFLD may have a larger burden of viral hepatitis.

Sedentary lifestyle, Urbanization and intake of fat-rich food, along with a higher inherited tendency of type-II diabetes mellitus, enhance metabolic syndrome or insulin resistance, and its manifestation leads to NAFLD.[4] Hence an attempt is made to evaluate the associated clinical manifestations to rule out the cause of NAFLD so that further complications can be prevented.

MATERIALS AND METHODS

50 (fifty) patients who visited the medicine department of Surabhi Institute of Medical Sciences, Mittapally Village, Siddipeth (Mandal and district), Telangana-502375 were studied.

Inclusive Criteria

Patients aged between 20 to 65 years with symptoms of hepatic steatosis, cirrhosis of the liver, and diabetic mellitus who gave their consent in writing were selected.

Exclusion Criteria

Alcoholics, those with hemochromatosis, hydatid cysts, the 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 December 2024 to February 2025.

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.

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RESULTS

[Table 1] Study of grade of non-alcoholic fatty liver (NAFLD): 10 (20%) grade-I, 23 (46%) grade-II, and 17 (34%) grade-III.

[Table 2] Clinical manifestations of NAFLD

- Body mass index: 31 (62%) had 22 to 24.2, and 19 (38%) had 23.3 to 24.2.
- Status of type II DM: 18 (36%) were pre-diabetic, 32 (64%) were diabetic.
- Status of Blood Pressure: 13 (26%) were normatensive, and 37 (74%) were hypertensive.
- Hyperlipidemic: 37 (74%) were, Ischemic heart disease (IHD) 14 (28%) myocardial infarction 2 (4%).

[Table 3] Mean value of Biochemical profile of non-alcoholic fatty liver (NAFLD): 222 (± 4.6) total cholesterol, 248 (± 8.8) triglyceride, 42.5 (± 2.6) HDL, 128 (± 9.8) LDL, 52.2 (±3.3) AST, 66.3 (± 3.5) ALT, 10.2 (± 2.5), 3.45 (± 0.11) serum albumin, 0.90 (±0.65) Total bilurubin, 133 (10.2%) Fasting Blood sugar, 9.10 (± 3.3) HbA1c.

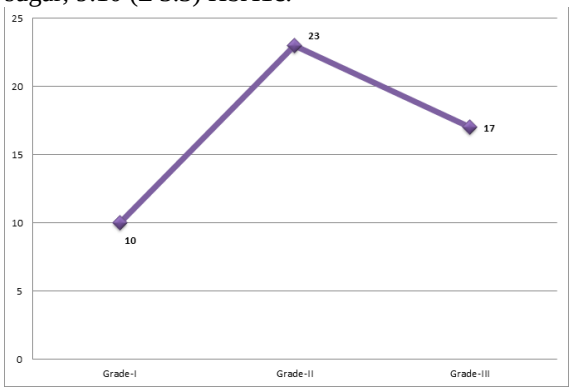

Figure 1: Study of grades of Non-Alcoholic Fatty liver

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

Sl. No	Grades of NAFLD	No. of patients (50)	Percentage (%)
1	Grade-I	10	20
2	Grade-II	23	46
3	Grade-III	17	34

Table 2: Clinical manifestations of Non-Alcoholic fatty liver

Sl. No	Clinical Manifestation	No. of Patients (50)	Percentage (%)
1	Body Mass Index (BMI) a-22.8 to 23.2	31	62
	b-23.3 to 24.2	19	38
2	Status type-II DM a – Pre-diabetic	18	36
	b – Diabetic	32	64
3	Status of Blood Pressure a – Normatensive	13	26
	b – Hypertensive	37	74
4	Hyper-lipidemic	37	74
5	Ischemic Heart Disease (IHD)	14	28
6	Myocardial Infarction (MI)	2	4

Table 3: Biochemical profile of Non-Alcoholic Fatty liver

Sl. No	Biochemical profile	Mean Value (±SD)
1	Total Cholesterol	222 (± 4.6)
2	Triglyceride	248 (± 8.8)
3	HDL	42.5 (± 2.6)
4	LDL	128 (±9.8)
5	AST	52.2 (± 3.3)
6	ALT	66.3 (±3.5)
7	ALP	10.2 (±2.5)
8	Serum Albumin	3.45 (±0.11)
9	Total Biliurubin	0.90 (±0.65)
10	Fasting Blood Sugar	133 (±10.2)
11	HA1C	9.10 (±3.3)

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

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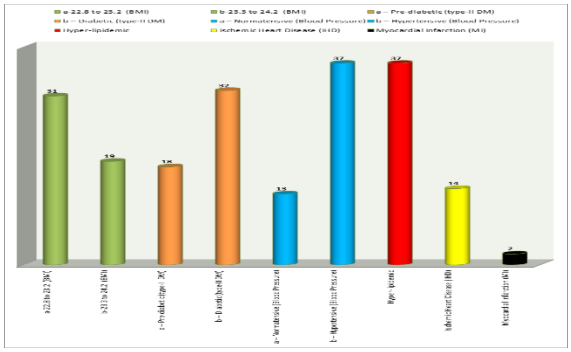

Figure 2: Clinical manifestations of Non-Alcoholic fatty liver

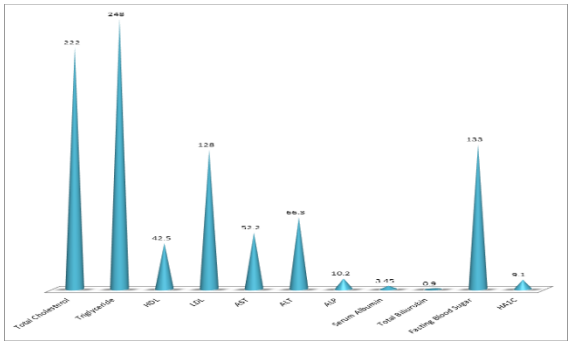

Figure 3: Biochemical profile of Non-Alcoholic Fatty liver

DISCUSSION

In the present study of NAFLD in the Telangana population, out of fifty patients, 10 (20%) had grade-I, 23 (46%) had grade-II, and 17 (34%) had grade-III NAFLD [Table 1]. The clinical manifestations were BMI 22 to 23.2 in 31 (62%), 23.3 to 24.2 in 19 (38%), 18 (36%) were pre-diabetic, 32 (64%) were diabetic, 13 (26%) were normotensive, 37 (74%) were hypertensive (HTN), 14 (28%) had ischemic heart disease, and 2 (4%) had myocardial infarction [Table 2]. An elevated biochemical profile was noted in NAFLD patients [Table 3]. These findings are more or less in agreement with previous studies.[5-7]

NAFLD is associated with metabolic syndrome, which is characterized by insulin resistance, HTN, cholesterol abnormality, increased risk of blood clotting, type-II DM, obesity, elevated serum triglyceride, and reduced HDL, which has a greater risk of heart diseases, stroke, and liver-related diseases.[8] Although the exact cause of NAFLD is still unclear, 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] The histological spectrum of NAFLD has no pathological changes that can definitively distinguish NAFLD from alcoholic liver diseases; thus, accurate alcohol history is essential to alcoholic liver disease.

Insulin resistance factor is believed to play 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 a universal factor in patients with NAFLD and is related to an imbalance between pro-insulin (adiponectin) and anti-insulin cytokine (TNF-a).[11,12]

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

CONCLUSION

The 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 remote location of the research center, the small number of patients, and the lack of the latest techniques, we have limited findings and results.

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