

Original Research Article

Study of nonalcoholic fatty liver Telangana population

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ABSTRACT

Background: Among 83 patients of both sexes aged between 25 to 65 adults had NAFLD with metabolic syndrome were studied.

Methods: U.S.G. biochemical study included total cholesterol, AST, ALP, S, Albumin total Bilirubin, FBS, HbA1c and blood pressure was recorded.

Results: Among 16(19.2%) had grade-I (mild steatosis) 38(45.7%) had grade-II (Moderate steatosis), 29(34.9%) had grade-III (severe steatosis), The clinical manifestation were 49(59%) had BMI 22.8 to 23.2, 34(40.9%) had BMI 23.3 to 24.2. D.M status was 33(39.7%) were pre-diabetic, 50(60.2%) were diabetic mellitus. 19(22.8%) were normotensive, 64(77.1%) were hypertensive, 63(75.9%) were hyperlipidemic, 23(27.7%) had IHD. 4(4.81%) had MI. Mean value of total cholesterol was 223 ± 9.2 , Triglyceride 24.8 ± 13.3 , HDL 42.3 ± 2.5 , LDL 128 ± 13.8 , AST 52.8 ± 3.6 , ALT 67.2 ± 6.8 , ALP 107 ± 11.8 , S. Albumin 3.50 ± 0.12 , Total bilirubin 0.93 ± 0.10 , FBS $13. \pm 12.2$, HB A/c 9.10 ± 402 .

Conclusions: The present study of NAFLD was performed by combination of radiological and laboratory techniques, greatly reducing the requirement for invasive biopsy and reduce the morbidity and mortality.

Keywords: Diabetes mellitus, Metabolic syndrome, Non-alcoholics fatty liver disease, Non-alcoholics steatohepatitis

INTRODUCTION

Non-Alcoholic Fatty Liver Disease (NAFLD) encompasses a spectrum of disease ranging from simple steatosis, to inflammatory steatohepatitis (SASH) with increasing levels of fibrosis and ultimately cirrhosis of liver NAFLD closely associated with obesity and insulin resistance and is now recognized to represent the hepatic manifestation of the metabolic syndrome. NASH term was coined by ludewing et al, in 1980.¹

The prevalence of NAFLD has risen rapidly in parallel with the dramatic in population level of obesity and diabetics, which is quite common in India and abroad.^{2,3} Despite of recent advances in elucidation the complex metabolic and inflammatory pathways involved in

NAFLD, the pathogenesis of steatosis and progression to steatohepatitis and fibrosis/ cirrhosis is not fully understood.^{4,5} Hence attempt was made to study the clinical manifestation and biochemical parameters of NAFLD patients to evaluate the causes of NAFLD.

METHODS

Among 83 patients often visiting to medicine Department of Medi Citi Institute of Medical Sciences Ghanpur Medchal 501401. Telangana were selected for study.

USG (Ultrasonography) of the abdomen and biochemical investigation total cholesterol, triglyceride HDL, LDL, AST, ALT, ALP, serum albumin total bilirubin. FBS, HBAIC and blood pressure to rule out the grades of Non-

alcoholic fatty liver, BMI of the patients were also studied to correlate the NAFL disease with BMI.

Inclusion criteria

The patients aged between 25 to 65 years having the symptoms hepatic steatosis, cirrhosis of liver with diabetes mellitus were included

Exclusion criteria

Alcoholic haemochromatosis, hydatid cyst, presence of HBS Ag, HIV positive were excluded from the study.

Statistical analysis

The grades of NAFLD was numbered with percentage BMI, pre-diabetic, diabetic hypertensive, normotensive, hyperlipidemic, IHD, MI patients were classified with percentage, mean value of Biochemical investigation was studied. The ratio of male and females were 2:1. The duration of study was about 3 years (April 2015-June 2018).

RESULTS

Among 16(19.2%) had grade-I (mild steatosis) 38(45.7%) had grade-II (moderate steatosis) 29(34.9%) had grade-III (Severe steatosis).

Table 1: Grades (types) of NAFLD.

Particulars	No of patients	Percentage %
Grade-I(mild steatosis)	16	19.2
Grade-II (moderate steatosis)	38	45.7
Grade-III (severe steatosis)	29	34.9

Grade II (moderate steatosis) was highest 38(45.7%) and Grade-I (mild steatosis) was least steatosis 16(19.2%) was observed in the grades of NAFLD (Table 1).

Clinical manifestations of NAFLD. 1-BMI status-49(59%) had 22.8 to 23.2, 34(40.9%) had 23.3 to 24.2. 2-D.M status-33(39.7%) were pre-diabetic, 50(60.2%) were diabetic 3-status of BP-19(22.8%) were normotensive, 64(77.1%) were hypertensive 4-63(75.9%) were Hyperlipidemic, 5-23(27.7%) had IHD, 6-4(4.81%) had history of MI.

BMI status was from 22.8 to 24.2. Diabetic mellitus study Pre-diabetic were 33 while diabetic was 50. Hypertensive were 64 while norma tensive were 19, IHD 23, MI 04 were observed (Table 2).

Mean value of total cholesterol was 223±9.2, Triglyceride 248±13.3, HDL 42.3±2.5, LDL 128±13.8, AST-52.8±3.6, ALT 67.2±6.8, ALP 107±11.8, S. albumin 3.50±0.12. Total bilirubin 0.93±0.10, Fasting Blood sugar - 130±12.2, HbA1c 9.10±4.2 (Table 3).

Table 2: Clinical manifestation of NAFLD patients.

Particulars	No of patients	Percentage
BMI		
a-22.8 to 23.2	49	59%
b-23.3 to 24.2	34	40.9%
Diabetic mellitus status		
a-pre-diabetic	33	39.7%
b-Diabetic	50	60.2%
Status of blood pressure		
a-Norma tensive	19	22.8%
b-Hypertensive	64	77.1%
Hyper lipidemic	63	75.9%
IHD	23	27.7%
MI	04	4.81%

IHD= Ischemic Heart Disease
MI= Myocardial Infarction

Table 3: Bio-chemical study of NAFLD Patients.

Particulars	Mean value (SD±)
Total cholesterol	223±9.3
Triglyceride	248±13.3
HDL	42.3±2.5
LDL	128±13.8
AST	52.8±3.6
ALT	67.2±6.8
ALP	107±11.8
S. Albumin	3.50±0.12
Total Bilirubin	0.93±0.10
Fasting Blood sugar	130±12.2
HbA1c	9.10±4.2

ALP= Alkaline Phosphatase, ALT= Alanine amino Transferase
LDL= Low Density Lipoprotein, HDL= High Density lipoprotein, AST= Aspirate Amino transferase, HbA1c= Hemoglobin Alc.

DISCUSSION

In the present study of NAFLD in Telangana population. The grades or types of NAFLD was 16(19.2%) were grade-I (mild steatosis), 38(45.7%) were grade-II (moderate steatosis), 29(34.9%) were grade-III (Severe steatosis) (Table 1).

The clinical manifestation of NAFLD patients were 1-BMI- 49(59%) 22.8 to 23.2, 34(40.9%) had 23.3 to 24.2. 2-D.M status was 33(39.7%) were pre-diabetic, 50(60.2%) were diabetic, 3-status of BP was 19(22.8%) were norma tensive 64(77.1%) were hypertensive. 63(75.9%) were hyperlipidemic, 23(27.7%) had IHD 4(4.81%) had MI (Table-2) the biochemical profile was Mean values total cholesterol 223±9.2, triglyceride 248±13.3, HDL 42.3±2.5, LDL 128±13.8, AST 52.8±3.6, ALP 107±11.8, S. Albumin-3.50±0.12, Total bilirubin was 0.93±0.10 FBS= 130±12.2, HbA1c= 9.10±4.02 (Table 3). These findings were more or less in agreement with previous studies.⁶⁻⁸

NAFLD is associated with metabolic syndrome which is characterized by insulin resistance, HTN, cholesterol abnormality, increased risk of blood clotting, DM type-II, obesity, elevated serum triglyceride, reduced HDL which has greater risk of heart disease, stroke and liver related diseases.⁹ associated with differences in lipid metabolism.¹⁰ Exact cause of NAFLD is still unclear, the probable reasons could be nutritional status, or it could be drug induced steatosis Drug induced agents steatosis include glucocorticoids amiodarone, synthetic estrogens and highly active anti-retroviral drugs. Steatosis (NAFLD) is frequently associated with hepatitis-c particularly genotype-3 and endocrine disorders in females such as Polycystic Ovarian Syndrome (PCOD), hypopituitarism, and hypothyroidism.¹¹ It is reported that, NAFLD is the common cause of chronic liver diseases, chronic viral hepatitis.

The insulin resistance factor is believed to be key factor, that leads to increased lipolysis in peripheral adipose tissue and increased uptake of fatty acids by hepatocytes. The end results are 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 cytokines (TNF- α).

It is also reported that rise of prevalence of NAFLD in developing countries is related to rapid industrialization, sedentary lifestyle, obesity, DM, junk food intake etc.

CONCLUSION

The present study of NAFLD in patients of Telangana Population will be useful to physician endocrinologist, radiologist, correlate the findings of bio-chemical techniques to treat such patients efficiently without liver biopsy study however in the severity of disease and not responding to the treatment biopsy of the liver is unavoidable Hence biopsy method is popularly called as gold method. But this study demands further nutritional, hormonal, genetic, biochemical, histopathological study because exact pathogenesis of steatosis is still un-clear.

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REFERENCES

1. Ludwig J. Steatohepatitis: Mayo Clinic experiences with a hitherto unnamed disease. In Mayo Clin Proc. 1980;55:434-8.
2. Vuppalanchi R, Chalasani N. Nonalcoholic fatty liver disease and nonalcoholic steatohepatitis: Selected practical issues in their evaluation and management. Hepatol. 2009;49(1):306-17.
3. Gupta P, Amarapurkar D, Agals S, Baijal R, Kulshreshta P, Pramanik S, et al. Non Alcoholic fatty liver disease in type-II diabetes mellitus. J Gastroenterol Hepatol. 2004;19(8):854-8.
4. Petta S, Muratore C, Craxi A. Non-alcoholic fatty liver disease pathogenesis: the present and the future. Dige Liver Dis. 2009;41(9):615-25.
5. Dowman JK, Tomlinson JW, Newsome PN. Pathogenesis of non-alcoholic fatty liver disease. QJM: An Int J Medi. 2009;103(2):71-83.
6. Cheung O, Kapoor A, Puri P, Sistrun S, Luketic VA, Sargeant CC, et al. The impact of fat distribution on the severity of nonalcoholic fatty liver disease and metabolic syndrome. Hepatol. 2007;46(4):1091-100.
7. Duseja A. Non-alcoholic fatty liver disease in India- a lot done, yet more required! Ind J Gastroenterol. 2010;29(6):217-25.
8. Agarwal SR, Malhotra V, Sakhuja P, Sarin SK. Clinical, biochemical and histological profile of non-alcoholic steatohepatitis. Indi J Gastroenterol: offici J Indi Soci Gastroenterol. 2001;20(5):183-6.
9. Day CP. From fat to inflammation. Gastroenterol. 2006;130(1):207-10.
10. Browning JD, Szczepaniak LS, Dobbins R, Nuremberg P, Horton JD, Cohen JC, et al. Prevalence of hepatic steatosis in an urban population in the United States: impact of ethnicity. Hepatol. 2004;40(6):1387-95.
11. Loria P, Carulli L, Bertolotti M, Lonardo A. Endocrine and liver interaction: the role of endocrine pathways in NASH. Nature Reviews Gastroenterol Hepatol. 2009;6(4):236.

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