

ORIGINAL ARTICLE

Do Non Alcoholic Fatty Liver Disease Patients Have more Severe Metabolic Syndrome

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ABSTRACT

Background and goal: Alcohol-unrelated fatty liver disease (NAFLD) is a frequent type of chronic liver disease (CLD) that is rapidly spreading around the world. The study's primary goal is to evaluate the severity of metabolic syndrome in people with nonalcoholic fatty liver disease.

Materials and methods: From January to July 2019, Shalimar Medical and Dental College in Lahore, Pakistan, conducted this cross-sectional study. Through the use of non-probability sampling, the data was gathered. 100 patients with hepatic cirrhosis provided the data. The participants in this study ranged in age from 20 to 60. For the serum examination of liver enzymes, blood was drawn.

Results: A correlation between the severity of the metabolic syndrome and NAFLD was found in this study, which included data from 100 individuals with biopsy-proven NAFLD. The patients were 45.67 3.56 years old on average. Compared to the normal control (11,3.42), the ALT levels in the patients were 258.291.73, 79.6628.63, and 50.73 8.4 correspondingly. Patients with cirrhosis, alcoholic liver disease, and viral hepatitis all had considerably higher aspartate aminotransferase levels. The significant frequency elevated aminotransferases and of NAFLD in IR-obese adolescents, as well as the potential consequences for their health, are concluded. The strong connections between NAFLD and MetS encourage the screening for additional MetS co-morbidities.

INTRODUCTION

The most prevalent type of chronic liver disease (CLD) is non-alcoholic fatty liver disease (NAFLD), which is rapidly spreading throughout the world. Alcohol consumption is the common factor associated with fatty liver disease (FLD), although it is recently reported that FLD is not direct associated with alcohol consumption. Alcohol intake, diabetes mellitus (DM), and hyperlipidemia are important risk factors for NAFLD [1]. When dyslipidemia and endocrine resistance interact with NAFLD, the metabolic syndrome is present. Non-alcoholic steato hepatitis (NASH), also known as non-alcoholic liver disease (NALD) (NASH), is eventually related to liver cirrhosis (LC) [2]. The incidence and grade of NAFLD grading and its incidence differs widely with the population screening. The incidence of histologically defined NAFLD was 20 percent and 51 per cent in two separate research involving prospective liver donors [3]. Based on the population survey the incidence of NAFLD in South America is 31%, 32% in the Middle East, 23% reported in USA and 24% in Europe. A community-based incidence is 2008 reported 37.5% NAFLD in Sri Lankan female and recent research in 2017 reported 8.7% prevalence in adolescent of NAFLD in Sri Lankan population [4]. Apparently, due to different genetic makeup and environmental associated factors the Asian population having NAFLD had lower Basic Metabolic Index (BMI) than those in western countries [5]. The study's primary goal is to assess the severity of metabolic syndrome in people with nonalcoholic fatty liver disease.

MATERIAL AND METHODS

From January to July 2019, Shalimar Medical and Dental College in Lahore, Pakistan, conducted this cross-sectional study. Through the use of non-probability sampling, the data was gathered. 100 patients with hepatic cirrhosis provided the data. The participants in this study ranged in age from 20 to 60. For the serum examination of liver enzymes, blood was drawn. The Reitman and Frankel method was used to measure the enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST). It was determined the Gamma Glut amyl Transferees (GGT). SPSS for Windows version 17.0 was used to conduct the statistical analysis. Results are shown as mean (plus or minus standard deviation). Student t-tests for continuous variables were used to compare variables between two groups. Statistically significant p values were those below 0.05. Using a scale of 0 to 4, a pathologist who was blind to the patient's details graded the liver

biopsies for fibrosis, steatosis, and inflammation. All biopsies were stained with Masson's Trichrome for additional fibrosis assessment, and data were presented as mean percentages. Percentage fibrosis was calculated in triplicate by microscopy and image analysis. Using SPSS version 17.0, all of the data were gathered and examined.

RESULTS AND DISCUSSION:

The severity of the metabolic syndrome and NAFLD were found to be related in this study, which included data from 100 individuals with biopsy-proven NAFLD. The patients were 45.67 3.56 years old on average. Compared to the normal control (11,3.42), the ALT levels in the patients were 258.291.73, 79.6628.63, and 50.73 8.4 correspondingly. Patients with cirrhosis, alcoholic liver disease, and viral hepatitis all had considerably higher aspartate aminotransferase levels. The levels were, respectively, 157.8067.8, 16454.35, and 6212.17 as compared to the usual control (133.54). Patients with cirrhosis, alcoholic liver disease, and viral hepatitis all had significantly higher levels of alkaline phosphatase. Patients with cirrhosis, alcoholic liver disease, and viral hepatitis all had considerably elevated levels of gamma glutamine trans peptidase.

An AST: ALT ratio of 2:1 or above is a sign that should raise the clinician's suspicion of alcohol-related liver damage. Another sensitive but non-specific marker for hepatic injury that cannot be utilised to exclusively detect alcohol-related hepatic insult is gamma-glut amyl transferees (GGT). The best treatment for NAFLD at the moment is gradual weight loss brought on by dietary changes and exercise [6, 7]. The suggested weight loss rate is one pound per week, as losing weight too quickly could make NAFLD worse. Although the best diet to treat NAFLD has not yet been determined, the significance of IR implies that low glycemic diets may be helpful. However, the majority of patients, particularly adolescents, have little success changing their lifestyles, which has sparked interest in pharmacologic treatments for NASH. Small sample size, open-label approach, and lack of a placebo control, and a brief follow-up period have, however, hampered investigations to date [8]. Antioxidant treatment experiments were prompted by the discovery of oxidative stress's part in the pathophysiology of NAFLD. ALT returned to normal in a short unrestricted pilot project involving 11 NASH-afflicted children who underwent a 2-4 month vitamin E treatment [9]. Through the NASH CRN TONIC study, additionally, vitamin E immunotherapy is now

being researched. A modest randomised trial combining vitamin E and C therapy that is combined, in which every participant received a customised diet and more exercise, failed to show any additional effect above lifestyle measures alone [10]. Betaine, a metabolite of choline that increases levels of Sadenosylmethionine, was shown to ameliorate aminotransferases, steatosis, and necrotic inflammation In a short pilot investigation, in adult NASH patients [11].

CONCLUSION

It is established that obese adolescents with IR have a significant prevalence of NAFLD and increased aminotransferases, which has serious consequences for their health. The tight connections from NAFLD to MetS promote screening for further MetS co-morbidities.

REFERENCES

- 1 Brunt EM, Janney CG, Di Bisceglie AM, et al. Nonalcoholic steatohepatitis: a proposal for grading and staging the histological lesions. *Am J Gastroenterol.* 1999;94:2467–74.
- 2 Nobili V, Marcellini M, Devito R, et al. NAFLD in children: a prospective clinicalpathological study and effect of lifestyle advice. *Hepatology.* 2006;44:458–65.
- 3 Harrison SA, Ramrakhiani S, Brunt EM, et al. Orlistat in the treatment of NASH: a case series. *Am J Gastroenterol.* 2003;98:926–30.

- 4 Sabuncu T, Nazligul Y, Karaoglanoglu M, et al. The effects of sibutramine and orlistat on the ultrasonographic findings, insulin resistance and liver enzyme levels in obese patients with non-alcoholic steatohepatitis. *Rom J Gastroenterol.* 2003;12:189–92.
- 5 Dixon JB, Bhathal PS, Hughes NR, O'Brien PE. Nonalcoholic fatty liver disease: Improvement in liver histological analysis with weight loss. *Hepatology.* 2004;39:1647–54.
- 6 Kral JG, Thung SN, Biron S, et al. Effects of surgical treatment of the metabolic syndrome on liver fibrosis and cirrhosis. *Surgery.* 2004;135:48–58.
- 7 Luyckx FH, Desai C, Thiry A, et al. Liver abnormalities in severely obese subjects: effect of drastic weight loss after gastroplasty. *Int J Obes Relat Metab Disord.* 1998;22:222–6.
- 8 Tiikkainen M, Hakkinen AM, Korshennikova E, et al. Effects of rosiglitazone and metformin on liver fat content, hepatic insulin resistance, insulin clearance, and gene expression in adipose tissue in patients with type 2 diabetes. *Diabetes.* 2004;53:2169–76.
- 9 Marchesini G, Brizi M, Bianchi G, et al. Metformin in non-alcoholic steatohepatitis. *Lancet.* 2001;358:893–4.
- 10 Nair S, Diehl AM, Wiseman M, et al. Metformin in the treatment of non-alcoholic steatohepatitis: a pilot open label trial. *Aliment Pharmacol Ther.* 2004;20:23–8.
- 11 Bugianesi E, Gentilcore E, Manini R, et al. A randomized controlled trial of metformin versus vitamin E or prescriptive diet in nonalcoholic fatty liver disease. *Am J Gastroenterol.* 2005;100:1082–90.