http://doi.org/10.33279/jkcd.v15i01.684

Orginal Article

HYPERTRIGLYCERIDEMIA AS A RISK FACTOR FOR NON–ALCOHOLIC FATTY LIVER DISEASE

Sarwat Abbasi[1], Saadia Sadiq[2], Sara Jadoon[3], Salma Shazia[4], Haroon ur Rashid[5], Amjad Farooq[6], Zahoor Ahmed[7]

[1]Department of Biochemistry, Ayub Medical College, Abbottabad, Khyber Pakhtunkhwa, Pakistan
[2]Department of Biochemistry, Abbottabad International Medical College, Abbottabad, KPK, Pakistan
[3]Department of Anatomy, Ayub Medical College, Abbottabad, Khyber Pakhtunkhwa, Pakistan
[4]Department of Forensic Medicine, Ayub Medical College, Abbottabad, Khyber Pakhtunkhwa, Pakistan
[5]Department of Pathology Women Medical College, Abbottabad, Khyber Pakhtunkhwa, Pakistan
[6]Department of Surgery, Ayub Medical Institute, Abbottabad, Khyber Pakhtunkhwa, Pakistan
[7]Department of Biochemistry, Khyber Medical College, Peshawar, Khyber Pakhtunkhwa, Pakistan

ABSTRACT

Objectives: *To determine increased triglyceride levels as a potential cause of development of non–alcoholic fatty liver disease.*

Materials and Methods: *This cross sectional study was conducted at Ayub Medical Institute, Abbottabad. A well designed questionnaire was designed. Subjects between 40–60 years age group were selected after informed consent and all the data was saved confidentiality. Data accumulated and examined with the help SPSS version 22.*

Results: *Among patients with hepatic steatosis 28 (56.0%) have normal serum triglyceride levels while 22 (44.0%) have high triglyceride levels. Among individuals with no fatty infiltration 41 (82.0%) have normal triglyceride level while only 9 (18.0%) have elevated serum triglyceride levels. 27 (54.0%) obese patients and 17 (34.0%) overweight individuals have fatty liver disease hence giving a outstanding p-value of <0.001 with chi-square test between NAFLD and BMI.*

Conclusion: *Hypertriglyceridemia was found as a risk factor for non–alcoholic fatty liver disease (NAFLD).*

Key words: *Hepatic disorder, Hypertriglyceridemia, NAFLD*

Cite as: Abbasi S, Sadiq S, Haleem N, Shazia S, Rashid HU, Farooq A, Ahmed Z. Hypertriglyceridemia as a risk factor for non–alcoholic fatty liver disease. Journal of Khyber College of Dentistry Mar 2025, Vol. 15, No. 1. http://doi.org/10.33279/jkcd.v15i01.684

INTRODUCTION

Hypertriglyceridemia is established contributing element for cardiac abnormalities. Poor eating habits causes high level of fats in the form of triglycerides not only in blood but also increases its storage throughout the body including liver. Fatty liver is usually asymptomatic unless it may lead to liver damage. High triglycerides and high weight are allied with NAFLD. Non–alcoholic fatty liver disease is one of the prevalent illnesses that's prevalence and incidence is continuously being increasing. Lipid accumulation in the hepatocytes is the pathology found in the disease and it suggest a deep relationship between altered lipid metabolism and non–alcoholic fatty liver disease[1]. Dyslipidemia, obesity and non-insulin dependent diabetes (NIDDM) keep documented role in pathogenesis of the disease[2,3]. Dyslipidemia is a risk factor for cardiovascular complications. Liver and circulatory system should be carefully observed and controlled non–alcoholic fatty liver disease. Triglyceride is the principal form that gathered in hepatocytes and is responsible for

Correspondence:
Dr. Sara Jadoon
Assistant Professor
Department of Anatomy, Ayub Medical College, Abbottabad, Khyber Pakhtunkhwa, Pakistan
Email: sarashafaq@hotmail.com

Date Submitted: April 2024
Date Revised: June 2024
Date Accepted: September 2024

lipotoxicity and hepatic injury. Patients show high atherogenic lipoprotein profile with high serum triglycerides level, increased level of low density lipoprotein and apolipoprotein–B (ApoB) level with low level high density lipoprotein[4,5]. It is documented that there is increased production of very low density lipoproteins by the liver cells with less removal from the circulation. However exact pathogenesis of dyslipidaemia in NAFLD is unclear[6].

Risk factors for cardiovascular disease because of hypertriglyceridemia including increased thickness in the walls of coronary ducts with its calcification, malfunctions of endothelial cells of the pericardial adipose tissue and sensitivity C–reactive protein hypersensitivity are increased in NAFLD patients[7]. The relation between NAFLD and cardiovascular disease is suggestive to be due to inflammation, insulin resistance, dyslipidemia, abnormal lipid metabolism, fat distribution and endothelial dysfunction[8].

Obesity increases serum triglyceride levels and is associated with increased cholesterol levels. Several studies reported high quantity of triglyceride appeared to elevate the chance of augmenting NAFLD in high body mass index subjects[9]. In addition obesity and hypertriglyceridemia may be mediated by insulin resistance insulin resistance has been found to have special role in pathogenesis, evolution and progression of NAFLD[10,11].

MATERIALS AND METHODS

This cross-sectional study was conducted at the tertiary care hospital, Ayub Medical Institute (AMI), Abbottabad, utilizing the institute's well-equipped advanced technical laboratory to ensure standardized data collection and analysis. The study included patients aged 40 to 60 years diagnosed with non-alcoholic fatty liver disease (NAFLD) based on ultrasonographic findings. Participants were randomly recruited from the general population visiting AMI, Abbottabad. The inclusion criteria consisted of individuals within the specified age range diagnosed with fatty liver through ultrasonography. Exclusion criteria included patients with a history of alcohol consumption, those diagnosed with other hepatic diseases such as hepatitis or HIV, and pregnant women.

Participants were divided into two groups: Group A included 50 individuals aged 40 to 60 years diagnosed with ultrasonographically confirmed fatty liver, excluding other hepatic pathologies, while Group B consisted of 50 individuals of the same age group with normal hepatic ultrasound findings (i.e., without fatty liver). The study was conducted following approval from the Institutional Ethical Review Committee, and informed consent was obtained from all participants to ensure confidentiality and anonymity of data.

Abdominal ultrasonography was performed by a specialist radiologist to confirm fatty liver, which was identified based on diffuse increased echogenicity of the liver, poor visibility of intrahepatic duct structures, and mild to moderate hepatomegaly with a blunt hepatic edge. Anthropometric measurements were recorded, including height, measured in a standing position using a vertical calibrated scale, and weight, measured with a calibrated physical balance. BMI was calculated using the formula: weight (kg) divided by height (m^2).

Biochemical analysis involved the collection of a 5 mL venous blood sample from each participant following an overnight fast. The samples were centrifuged at 3000 revolutions per minute for 10 minutes to obtain serum. Serum triglyceride levels were measured using the Innoline diagnostic kit via the enzymatic splitting method. Data analysis included descriptive statistics to summarize baseline characteristics. The chi-square test was applied for categorical data analysis, while the independent t-test was used to assess differences between groups and evaluate the association between hypertriglyceridemia and NAFLD. A p-value of less than 0.05 was considered statistically significant.

RESULT

A total of 100 subjects, 50 NAFLD patients and 50 normal individual having normal liver study were taken in the present research with the age between 40–60 years. Both male and female individuals were selected randomly. Different causative factors for the incidence and progression of NAFLD was investigated. Serum triglyceride level was measured between the normal values of 0.4–2.0mmol/L. 22(44.0%) of the subjects with NAFLD has high serum triglyceride levels while 6 (12.0%) have no fatty infiltration but have hypertriglyceridemia. Statistically by applying Chi–square test p–value was found highly significant giving the evidence of association of hypertriglycer-

idemia with NAFLD (Table–1). BMI of the study subjects measured by calculating weight and height of the individuals. Based on BMI study participants were grouped into normal, overweight and obese. Significant association was found between obesity and NAFLD (p<0.001) as shown in Table –2. Individuals with high BMI have hypertriglyceridemia which then affects hepatocytes and get accumulated in hepatic tissue. Table–3 shows the association of BMI with triglyceride levels. Statistically a highly significant p–value (<0.001) was observed between serum triglyceride level and BMI.

DISCUSSION

Research study carried out shows evidence of hypertriglyceridemia and obesity as strong risk factors for NAFLD[12]. NAFLD is emerging problem worldwide and is considered as a common cause of hepatocellular carcinoma[13,14]. Obese but otherwise metabolically healthy individuals are more exposed to NAFLD progression in contrast to low weight individuals[15]. Sedentary life style, eating habits increases the frequency of metabolic syndrome and increase in triglyceride levels. The relation between obesity, dyslipidemia and metabolic syndrome develop insulin resistance in peripheral tissues which increases hepatic flux of fatty acids from diet, increases lipolysis & resistance to antilipolytic effect of insulin from peripheral tissue. Hepatic accumulation of triglycerides is caused by the transport of free fatty acid into liver[16]. Obesity is considered as a vital element for NAFLD; however, studies gives an evidence of NAFLD in lean persons[17].

However, Hypertriglyceridemia is found responsible for cardiovascular complications as evidenced by previous studies that documented cardiovascular events as the chief source of death amongst NAFLD subjects yet hepatic tissue is also affected by high triglyceride level in the body[18,19]. Our current study shows significant association between hypertriglyceridemia and NAFLD which is consistent with several other studies. Lonardo A. et al., confirms hypertriglyceridemia as peril cause of NAFLD[20]. Souza MR et al documented 20-80% of NAFLD patients have dyslipidemia[21]. Hu Y et al and Grander C et al also confirm the risk of developing the disease in patients with high triglyceride level and high BMI[22,23]. Degree of hypertriglyceridemia also suggests the severity of NAFLD[24,25]. In addition, overweight and obesity are found responsible for the prevalence of dyslipidemia[26]. It is also documented that obesity and inactivity of adult males have high circulating triglyceride concentration[27].

LIMITATIONS

Our present study only considered the association between hypertriglyceridemia and non–alcohol-

Table 1: Association between Serum Triglyceride levels and NAFLD (n=100)

Triglyceride Level		Normal	High	Total	p–value
NAFLD	Yes	28 (56%)	22 (44%)	50 (100%)	<0.001
	No	44 (88%)	6 (12%)	50 (100%)	
Total			72 (72%)	28 (28%)	

Table 2: Association between NAFLD and BMI

Parameters		BMI			Total	p–value
		Normal	Over-weight	Very Obese		
NAFLD	Yes	6 (12%)	17 (34%)	27 (54%)	6 (12%)	< 0.001
	No	38 (76%)	12 (24%)	0 (0%)	38 (76%)	
Total		44	44 (44%)	29 (29%)	27 (27%)	

Table 3: Association between Serum Triglyceride and BMI (n=100)

Parameters	BMI			Total	p–value
	Normal	Over-weight	Very Obese		
Triglyceride Levels	37 (67.3%)	18 (32.7%)	0 (0%)	55 (100%)	< 0.001
	6 (21.4%)	9 (32.1%)	7 (25%)	28 (100%)	
Total	44 (44%)	29 (29%)	27 (27%)	100 (100%)	

Normal triglyceride level=0.4-2.0mmol/l, p-value ≤0.05 is significant

ic fatty liver disease.

RECOMMENDATIONS

NAFLD association can also been studied in future with cholesterol levels which is also risk factors for cardiovascular diseases.

CONCLUSION

Elevated triglyceride level mediated with obesity is found to be a marker of NAFLD. Hypertriglyceridemia is an established risk factor for developing cardiovascular abnormalities. Fatty infiltrations in the hepatic tissue make individuals vulnerable to serious consequences like liver cirrhosis and hepatocellular carcinoma. Sedentary life styles, eating habits, lack of physical activity, emotional stress and other environmental factors are connected with progression of the illness. Prevention of disease need lifestyle changes along with early observation and therapy of the disease.

REFERENCES

1. QQ Zhang, LG Lu. Nonalcoholic Fatty Liver Disease: Dyslipidemia, Risk for Cardiovascular Complications, and Treatment Strategy; J Clin Transl Hepatol. 2015 Mar; 3(1): 78–84.
2. Kalra S, Vithalani M, Gulati G, Kulkarni CM, Kadam Y,Pallivathukkal J, et al. Study of prevalence of nonalcoholic fatty liver disease (NAFLD) in type 2 diabetes patients in India (SPRINT) J Assoc Physicians India. 2013;61: 448–453.
3. Sahebkar A, Chew GT, Watts GF. New peroxisome proliferator-activated receptor agonists: potential treatments for atherogenic dyslipidemia and non-alcoholic fatty liver disease. Expert OpinPharmacother.2014; 15:493–503.
4. Alshamiri M, Ghanaim MMA, Barter P, Chang KC,Li JJ, Matawaran BJ ,et al. Expert opinion on the applicability of dyslipidemia guidelines in Asia and the Middle East. Int J Gen Med. 2018; 11:313–22.
5. Loria P,Marchesini G,Nascimbeni F,Ballestri S,Maurantonio M, Carubbi F, et al. Cardiovascular risk, lipidemic phenotype and steatosis.A comparative analysis of cirrhotic and non-cirrhotic liver disease due to varying etiology. Atherosclerosis.2014; 232(1):99–109.
6. Chatrath H, Vuppalanchi R, Chalasani N.Dyslipidemia in patients with nonalcoholic fatty liver disease 2012 Feb; 32(1):22-9.doi: 10.1055/s-0032-1306423.Epub 2012 Mar 13.
7. Speliotes EK, Massaro JM, Hoffmann U, Vasan RS,-Meigs JB, Sahani DV,et al.Fatty liver is associated with dyslipidemia and dysglycemia independent of visceral fat: the Framingham Heart Study.Hepatology.2010; 51: 1979–1987.
8. Liu H, Lu HY.Nonalcoholic fatty liver disease and cardiovascular disease. World J Gastroenterol.2014; 20: 8407–8415.
9. Xing J; Guan X; Zhang Q; Chen S; Wu S; Sun X. Triglycerides Mediate Body Mass Index and Nonalcoholic Fatty Liver Disease: A Population-Based Study. (2021 March 29) 14 (2): 190–196.
10. Badawi A, Klip A, Haddad P, Cole DE, Bailo BG, El-Sohemy A, et al. Type 2 diabetes mellitus and inflammation: prospects for biomarkers of risk and nutritional intervention. Diabetes Metab Syndr Obes.2010 May; 3:173–86.
11. Bray GA, Clearfield MB, Fintel DJ, Nelinson DS. Overweight and obesity: the pathogenesis of cardiometabolic risk. Clin Cornerstone. 2009; 9 (4):30–40.
12. Chalasani N, Younossi, Lavine J, Diehl AM, Brunt EM, Cusi K, et al; American Association for the Study of Liver Diseases; American College of Gastroenterology; American Gastroenterological Association. The diagnosis and management of non-alcoholic fatty liver disease: Practice guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association .Am J Gastroenterol. 2012 Jun;107(6):811–26.
13. Yu J, Shen J, Sun TT, Zhang X, Wong N. Obesity, insulin resistance, NASH and hepatocellular carcinoma. Semin Cancer Biol. 2013 Dec; 23(66 Pt B):483–91.
14. Dyson J, Jaques B, Chattopadyhay D, Lochan R, Graham J, Das D, et al Hepatocellular cancer: the impact of obesity, type 2 diabetes and a multidisciplinary team. J Hepatol.2014 Jan; 60 (1):110–7.
15. Lonardo A, Mantovani A, Lugari S,Targher G. Epidemiology and pathophysiology of the association between NAFLD and metabolically healthy or metabolically unhealthy obesity. Ann Hepatol. 2020; 19(4):359–66.
16. Klop B, ElteJW, Cabezas MC. Dyslipidemia in obesity; mechanism and potential targets. Nutrients.2013 Apr 12;5(4):1218-40.
17. Huh Y, Cho YJ, Nam GE.Recent Epidemiology and Risk Factors of Nonalcoholic Fatty Liver Disease.J Obes Metab Syndr. 2022 Mar 30; 31(1):17-27.
18. Söderberg C, Stål P, Askling J, Glaumann H, Lindberg G, Marmur J, et al. Decreased survival of subjects with elevated liver function tests during a 28-year follow-up. Hepatology.2010; 51:595–602.
19. Ekstedt M, Franzén LE, Mathiesen UL, Thorelius L, Holmqvist M, Bodemar G, et al. Long-term follow-up of patients with NAFLD and elevated liver enzymes. Hepatology.2006; 44: 865–873.
20. Lonardo A, Loria P, Leonardi F, Borsatti A, Neri P, Pul-

virenti M, et al. Fasting insulin and uric acid levels but not indices of iron metabolism are independent predictors of non-alcoholic fatty liver disease.A case -control study. Dig Liver Dis. 2002; 34(3):204–11.

21. Souza MR, Diniz MF, Medeiros JE, Araujo MS. Metabolic syndrome and risk factors for non-alcoholic fatty liver disease. Arq Gastroenterol.2012; 49:89–96.
22. Hu Y, Liu J, Dong , Xu Y,Leng S, Wang G. Clinical Study of Serum Homocysteine and Non-Alcoholic Fatty Liver Disease in Euglycemic Patients.Med Sci Monit.2016 Nov; 22:4146–51.
23. Grander C, Grabherr F, Moschen AR, Tilg H. Non-Alcoholic Fatty Liver Disease:Cause or Effect of Metabolic Syndrome.Visc Med. 2016 Oct; 32 (5):32934.
24. Hsiao PJ, Kuo KK, Shin SJ, Yang YH, Lin WY, Yang JF, et al. Significant correlations between severe fatty liver and risk factors for metabolic syndrome. J Gastroenterol Hepatol.
25. Tutunchi H, Saghafi-Asl M, Asghari-Jafarabadi M, Ostadrahimi A .The relationship between severity of liver steatosis and metabolic parameters in a sample of Iranian adults.BMC Res Notes. 2020; 13(1):218.
26. Nogueira-de-Almeida CA, Mello ED. Correlation of body mass index Z-scores with glucose and lipid profiles among overweight and obese children and adolescents. J Pediatr.2018;94 (3):308–12
27. Dejavitte RAS, Enes CC, Nucci LB. Prevalence of metabolic syndrome and its associated factors in overweight and obese adolescents. J Pediatr Endocrinol Metab.2020; 33(2):233–9.2007; 22(12):2118-23.

CONFLICT OF INTEREST
Authors declare no conflict of interest.
GRANT SUPPORT AND FINANCIAL DISCLOSURE
None declared.

AUTHORS' CONTRIBUTION

The following authors have made substantial contributions to the manuscript as under:

Conception or Design:	SA, SS, NH, SS, HUR, AF, ZA
Acquisition, Analysis or Interpretation of Data:	SA, SS, NH, SS, HUR, AF, ZA
Manuscript Writing & Approval:	SA, SS, NH, SS, HUR, AF, ZA

All the authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.