

IJMSPR



ISSN : 2394-8973
Vol. 1, No. 3, August 2015

International Journal of Medical Sciences and Pharma Research



www.ijmspr.com

Email: editorijmspr@gmail.com or editor@ijmspr.com



Research Paper

C-REACTIVE PROTEIN IN NON ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

Rajni Dawar^{1*} and Tabassum Yasmin²

*Corresponding Author: **Dr Rajni Dawar** ✉ drrajnidawar@gmail.com

Prevalence of Non Alcoholic Fatty Liver Disease is estimated to be around 9-32% in general Indian Population. It is more common in diabetic and obese individuals. We conducted a case control study in RMCH, a tertiary care hospital with 36 patients of diagnosed NAFLD and 30 age & sex matched controls. BMI was calculated for all subjects. Serum hs-C Reactive Protein levels were estimated by ELISA. It was found that BMI (29.61 ± 4.89 ; 23.66 ± 3.4 , $p < 0.001$) and hs-CRP levels (8.9 ± 1.6 ; 2.3 ± 1.6 , $p < 0.001$) were significantly higher in cases compared to controls. As hs-CRP is an independent factor involved in CAD and metabolic Syndrome, it signifies increased risk of these in NAFLD. We also advocate its use for diagnostic workup & its further evaluation as a marker for NAFLD.

Keywords: NAFLD, C-Reactive Protein, Steatosis, Metabolic Syndrome

INTRODUCTION

Nonalcoholic Fatty Liver Disease (NAFLD) refers to the presence of hepatic steatosis when no other causes for secondary hepatic fat accumulation (e.g., heavy alcohol consumption) are present. It has a wide histopathological spectrum ranging from simple, bland steatosis (NAFL), which is usually associated with a benign prognosis, to non alcoholic steatohepatitis (NASH), which is thought to possess the potential for progress to cirrhosis, and its inherent complications of liver failure and liver cancer.

1. Most patients with NAFLD have few or no symptoms. Patients may complain of fatigue, malaise, and dull right-upper-quadrant

abdominal discomfort. Mild jaundice may be noticed although this is rare. More commonly NAFLD is diagnosed following abnormal liver function tests during routine blood tests. By definition, alcohol consumption of over 20 g/day (about 25 mL/day of net ethanol) excludes the condition. NAFLD is associated with insulin resistance and metabolic syndrome. The exact cause of NAFLD is still unknown. However, both obesity and insulin resistance probably play a strong role in the disease process. The exact reasons and mechanisms by which the disease progresses from one stage to the next are not known.

2. Prevalence of the disease is estimated to be

¹ Department of Biochemistry, LHMC & SSK Hospital, Delhi, India.

² Department of Biochemistry, RMCH, Ghaziabad, India.

around 9-32% in the general Indian population, with a higher incidence rate amongst obese and diabetic patients.

3. The diagnosis of NAFLD requires all of the following: demonstration of hepatic steatosis by imaging or biopsy, exclusion of significant alcohol consumption and exclusion of other causes of hepatic steatosis. Noninvasive investigations to detect steatosis include ultrasonography of abdomen which often reveals a hyperechoic texture or a bright liver because of diffuse fatty infiltration. Ultrasound examinations can accurately identify steatosis with a sensitivity of 94% and a specificity of 84%.
4. C-Reactive Protein (CRP) is one of the major acute phase proteins and is a marker of systemic inflammation. In contrast to regular CRP assays, a high sensitivity CRP (hs-CRP) assay enables the diagnosis of even low grade inflammation. It has important clinical and prognostic implications in cardiovascular disease. However, hs-CRP could be a promising biomarker for screening of NAFLD asymptomatic subjects independently of other metabolic disturbances associated with metabolic syndrome and cardiovascular risk.
5. We conducted a case control study with 36 patients diagnosed with NAFLD and 30 age and sex matched controls from June 2013 to October 2013. The study was approved by the Institutional Ethical Committee. All participants gave written informed consent to participate in the study. Inclusion criteria: The diagnosis was based on ultrasonographic finding of bright liver (the diagnosis of bright liver was based on abnormally intense, high level echoes arising from the hepatic parenchyma, with

amplitude similar to that of echoes arising from the diaphragm). Exclusion criteria: patients with; hepatitis B, hepatitis C infection and other known liver diseases like cirrhosis. Other diseases: diabetes, hypertension, malignancy, hypo or hyperthyroid disease, coronary artery disease and pregnancy. Alcohol consumption, cigarette smoking, use of drugs known to cause fatty liver. Also, subjects with any finding of systemic and local infection on physical examination and subjects with an abnormal urinalysis were excluded as infections increase CRP levels.

Venous blood (5 mL) was collected after overnight fasting in vacutainers without additive, allowed to clot for 30 min at room temperature and centrifuged at 3000 c/m for 5 min. The separated serum was stored into aliquots at -20° C until biochemical analyses. CRP was estimated using ELISA kit from Sigma Aldrich for hs CRP. The results obtained were as shown in Table 1.

The two groups were matched for age. The Body mass index of patients of NAFLD is significantly higher compared to controls. This is indicative of the role of obesity and metabolic syndrome in causing NAFLD. Also hs CRP was found to be significantly higher in cases. The study of Koruk *et al.* reported that increased levels of CRP could be helpful in the diagnostic work-up of patients with fatty liver disease.

6. Moreover, Yoneda *et al.*, added that hs-CRP could be a clinical feature that not only distinguishes NASH from simple nonprogressive steatosis but also indicates the severity of hepatic fibrosis.
7. This association raises the possibility that

BMI and CRP in Two Groups			
	Case(36)	Control(30)	P value
Age	38.10±5.60	37.9 ±6.3	NS
Sex (M:F)	23:13	21:9	
BMI	29.61±4.89	23.66±3.4	P<0.001(Sig)
hs-CRP	8.9±1.16	2.3±1.6	P<0.001 (Sig)

inflammatory processes that accompany NAFLD contribute to the systemic inflammation observed in subjects with obesity. There is no consensus regarding the mechanism for the association between metabolic disorders and chronic subclinical inflammation and several possible explanations have been suggested. These include release of proinflammatory cytokines from adipose tissue; Insulin Resistnace and elevated free fatty acids.

8. These explanation could support the observation of the present study that hs-CRP is significantly elevated in cases. However, as elevated hs-CRP is an independent factor involved in both cardiovascular disease and metabolic syndrome, subjects with NAFLD might be prone to develop cardiovascular complications Therefore hs-CRP concentrations should be further evaluated as a marker of NAFLD.

With the Results obtained in this study we conclude that the high levels of hs-CRP observed in NAFLD patients could suggest that these patients have a higher risk of developing cardiovascular problems and therefore, hs-CRP could be used as an effective marker of NAFLD.

Also obesity becoming a global epidemic and affecting different organ system of the body and evidence of it playing a role in NAFLD, public health

measures to contain this epidemic are required to reduce disease states associated with it.

CONFLICT OF INTEREST

None

ACKNOWLEDGMENT

Dr S Sahai, Assoc. Professor, Radiology, RMCH
Dr A. Manglik, Assistant Professor, Medicine for their help.

REFERENCES

1. Angulo P (2002), "Non Alcoholic fatty Liver Disease", *N England Jour Medicine*, Vol. 346, No. 16, pp. 1221-1229.
2. Adams LA and Angulo P (2006), "Treatment of non alcoholic fatty liver disease", *Postgrad Med J*, Vol. 82, No. 967, pp. 315–22.
3. Kalra S, Vithalani M, Gulati G, *et al* (2013), "Study of prevalence of nonalcoholic fatty liver disease (NAFLD) in type 2 diabetes patients in India (SPRINT)", *J Assoc Physicians India*, Vol. 61, No. 7, pp. 448-53.
4. Saverymuttu S H, Joseph A E and Maxwell J D (1986), "Ultrasound scanning in the detection of hepatic fibrosis and steatosis", *BMJ*, Vol. 292, pp. 13-15.
5. Lizardi-Cervera J, Chavez-Tapia N C, Pérez-Bautista O, Ramos M H and Uribe M (2007), "Association among Creactive protein, Fatty liver disease, and cardiovascular risk", *Dig Dis Sci.*, Vol. 52, No. 9, pp. 2375-2379.
6. Koruk M, Taysi S, Savas M C, *et al.* (2003), "Serum levels of acute phase proteins in patients with nonalcoholic steatohepatitis", *Turk J Gastroenterol*, Vol. 14, No. 1, pp. 12-17.
7. Yoneda M, Mawatari H, Fujita K, *et al.*

(2007), "Highsensitivity C-reactive protein is an independent clinical feature of nonalcoholic steatohepatitis (NASH) and also of the severity of fibrosis in NASH", *J Gastroenterol.*, Vol. 42, No. 7, pp. 573-782.

8. Kerner A, Avizohar O, Sella R, *et al.* (2005), Association between elevated liver enzymes and C-reactive protein: possible hepatic contribution to systemic inflammation in the metabolic syndrome", *Arterioscler Thromb Vasc Biol*, Vol. 25, No. 1, pp. 193-197.



International Journal of Medical Sciences and Pharma Research

Hyderabad, INDIA. Ph: +91-09441351700, 09059645577

E-mail: editorijmspr@gmail.com or editor@ijmspr.com

Website: www.ijmspr.com

