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Serum ferritin and high-sensitivity C-reactive protein levels in patients with alcoholic, non-alcoholic liver disorders and in healthy controls

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Abstract

Background: The liver is the most essential internal organ, and it aids in food digestion, metabolism, toxin clearance, and the elimination of toxic substances from the body. Viruses, excessive alcohol consumption, severe type 2 diabetic mellitus (T2DM), hyperlipidemia, and obesity are among the variables linked to liver damage. Long-term liver damage causes cirrhosis, which can develop to liver failure, which is a life-threatening condition.

Objective of the study: the levels of serum ferritin and high-sensitivity C-reactive protein in patients with alcoholic and non-alcoholic liver diseases.

Materials and methods: The investigation was conducted on 100 DN subjects of both sex and aged 20 or more and 100 age and sex matched healthy control subjects. Ferrittin and hs-CRP of each subject was measured.

Results: the present investigation shows that the Ferrittin and hs-CRP were elevated significantly in AFLD and NAFLD individuals as compared to controls.

Conclusion: This study concluded that the Ferrittin and hs-CRP could be marker for early recognition of AFLD and NAFLD.

Keywords: hs-CRP, Ferrittin

Introduction

Alcoholism is the most frequent and widespread drug addiction problem (National Institutes on Alcohol Abuse and Alcoholism, 2016), and it is a condition that affects people all over the world. Excessive alcohol consumption leads to fatty liver, alcohol hepatitis, and cirrhosis. In India, men who consume 40-80 ml of alcohol (wine) per day for 15 years can get fatty liver, while those who consume 160 ml per day develop cirrhosis, and 15% of alcoholics suffer alcoholic liver disease. Other factors, such as nutrition and immunology, play a significant influence in liver disease.

C-reactive protein (CRP) is a plasma protein produced in the liver that elevates in the bloodstream in response to inflammation caused by a variety of conditions[1]? CRP is also known as high sensitive C-reactive protein (hs-CRP) or ultra-sensitive protein (US-CRP). Inflammation is usually an issue, but it can also be a sign of a host infection, kidney failure, arthritis, or pancreatitis. Patients

with high CRP levels are more likely to develop liver damage (Robin Donovan et al. 2015). hs-CRP has been implicated in AFLD and NAFLD in just a few studies.

CRP levels rise during chronic inflammation and play a key role in metabolic syndrome, insulin sensitivity, and endothelial dysfunction. HsCRP is a promising noninvasive predictive marker for cirrhosis and NAFLD that may help with liver transplantation.

Ferritin is a 24-protein subunit intracellular globular protein complex (450kDa) that forms a nonocage with numerous metal-protein interactions[2]. When the ferritin level is high, it indicates how much iron is stored in the body. Ferritin is an iron storage protein found in prokaryotes and eukaryotic cells, as well as in living organisms. Ferritin is a sign of iron deficiency anaemia because it functions as a buffer for iron deficiency and iron overload. Ferritin is an acute phase protein that is elevated in inflammation, liver necrosis, and alcohol misuse (Bell et al, 1995) as well as NASH (National Institute of Health) (Bonkoysky et al, 1999)[3].

The liver is the most essential internal organ, and it aids in food digestion, metabolism, toxin clearance, and the elimination of toxic substances from the body. Viruses, excessive alcohol consumption, severe type 2 diabetic mellitus (T2DM), hyperlipidemia, and obesity are among the variables linked to liver damage. Long-term liver damage causes cirrhosis, which can develop to liver failure, which is a life-threatening condition[4]. According to the Globe Health Organization (WHO), India has a 23-death rate of liver disease per 100,000 people, and the world has a 27-death rate.

Cirrhosis of the liver is a slowly progressive condition in which healthy tissue is replaced by irreversible scar tissue, resulting in reduced liver function, obstructed blood flow, and slowed nutritional, hormone, and medicine absorption. Alcohol abuse, hepatitis B, hepatitis C, non-alcoholic fatty liver disease (NAFLD), and metabolic syndrome are all prominent causes of liver cirrhosis[5].

Fatty liver (steatosis) is fat accumulation in the liver that accounts for 5 to 10% of the liver's weight and can lead to fatty liver disease. There are two forms of fatty liver disease: alcoholic fatty liver disease (AFLD), which is caused by excessive alcohol consumption[6], and nonalcoholic fatty liver disease (NAFLD), which is caused by excessive fat accumulation in the liver and is linked to obesity and metabolic syndrome[7,8].

Alcoholism is a widespread condition over the world, with serious medical consequences. Excessive alcohol consumption causes liver damage, inflammatory cytokine release, oxidative stress, lipid peroxidation response, and acetaldehyde toxicity. These can result in liver inflammation, apoptosis, and finally liver cell fibrosis[9]. The general Indian population has a prevalence rate of 25-40% for alcoholic liver disease[10]. Alcoholism affects 140 million individuals worldwide, according to the WHO.

Chronic liver disease caused by nonalcoholic fatty liver disease (NAFLD) can progress to end-stage liver disease. NAFLD hastens the development of fibrosis, cirrhosis, and finally liver failure[11]. The buildup of triglycerides (TG) in hepatocytes is a sign of NAFLD[12]. Although the pathophysiology of NAFLD is unknown, it is known that obesity, diabetes, and metabolic syndrome are the key risk factors linked to the development of nonalcoholic fatty liver disease[13]. NAFLD is the most common illness in both developed and developing countries[14]. The prevalence rate of NAFLD in the Indian general population is roughly 9-32 percent, with a greater incidence rate in diabetes and obese patients.

The current topic is the levels of serum ferritin and high-sensitivity C-reactive protein in patients with alcoholic and non-alcoholic liver diseases, as well as healthy controls, are being investigated.

Materials and methods

In the current study, 300 people were involved in the investigation. 100 male and female patients with alcoholic fatty liver disease (AFLD) and 100 patients with non-alcoholic fatty liver disease (NAFLD) were chosen as study group subjects from the OPD (outpatient department) of the medical department of Index Medical College and Research Center, Indore. Clinical examination, information of the patients' clinical history, and analysis of relevant biochemical investigations were used to diagnose patients with AFLD and NAFLD.

A total of 100 healthy control individuals, both males and females, attended a normal health check-up as outpatients. They were chosen based on a lack of medical history of any ailment. Each individual provided a 12-hour fasting blood sample in a simple, EDTA, and fluoride container. After collection, the sample was centrifuged and the serum was stored at 4 degrees Celsius. CMIA (ELISA) was used to calculate serum ferritin, and Immunoturbidimetric was used to calculate high-sensitivity C-reactive protein (hs-CRP).

Results

Table 1: Shows statistical analyzes projected that the Ferrittin and hs-CRP of study group found to be significantly lower. This was observed that the average (mean ± SD) Ferrittin and hs-CRP concentration that was found in the control group was 92.16 ± 40.85 and 2.75 ± 0.87 and in the test group, it was (485.25 ± 210.69 and 7.11 ± 2.63) and (379.72 ± 114.42 and 7.97 ± 2.71). The SOD and CAT level was found to be significantly low in comparison to that in the healthy subjects (control group), with a p value of < 0.001.

Table 1: Comparison of Ferrittin and hs-CRP of controls and patients of two groups

Parameter	Group	Mean ± SD	P-value
Ferrittin	Group-1 (Control)	92.16 ± 40.85	<0.001
	Group-2 (ALFD)	485.25 ± 210.69	
	Group-3 (NALFD)	379.72 ± 114.42	
Hs-CRP	Group-1 (Control)	2.75 ± 0.87	<0.001
	Group-2 (ALFD)	7.11 ± 2.63	
	Group-3 (NALFD)	7.97 ± 2.71	

Values in Mean ± SD

* p<0.001 (highly significant)

ALFD- alcoholic fatty liver disease

NAFLD-non-alcoholic fatty liver disease

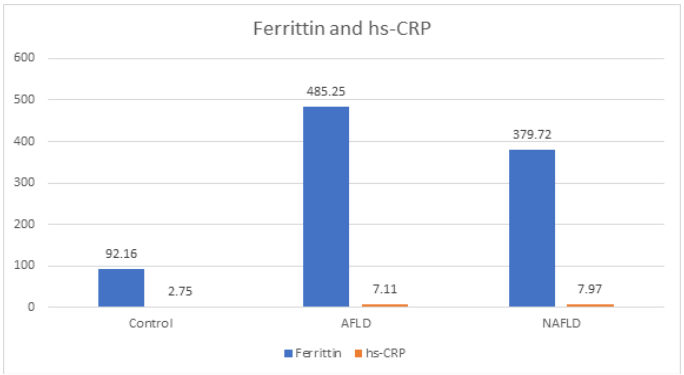

Figure 1: Comparison of Ferrittin and hs-CRP of controls and patients of two groups

Discussion and Conclusion

In the present study, the mean level of Ferrittin and hs-CRP were found to be significantly increased (p>0.001) in AFLD and NAFLD group in comparison to control group. These findings were concordant with the results of

the studies, which were previously done by Kogi so T et al (2009)[15], Riquelme A et al., (2009)[16], (Vanbiervliet G et al., 2006)[17] has discovered that serum hs-CRP levels rise in both alcoholic and nonalcoholic fatty liver disease. C-reactive protein is a biomarker for AFLD that decreases with liver fibrosis and is associated with excessive alcohol consumption. Jeong DW et al., (2014)[18] and Polyzosal P et al (2012)[19] has discovered that serum ferritin levels rise in both alcoholic and nonalcoholic fatty liver disease. have discovered that alcoholic liver disease patients have higher serum ferritin levels than healthy controls. Alcohol intake leads to higher ferritin levels, alcohol dependence, and the lack of iron overload. Serum ferritin levels in NAFLD patients may be linked to systemic inflammation and iron overload storage. Serum ferritin is a reflector of body iron reserves in diabetic and obese patients with nonalcoholic fatty liver disease.

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