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Original Research Article

Ferritin in Reproductive Age Group Women and Postmenopausal Women with Non-Alcoholic Fatty Liver Disease: A Comparative Study

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Conflict of interest: Nil

Abstract

Background: Nonalcoholic fatty liver disease, a risk factor for cardiovascular diseases is 25% prevalent in the general population. A higher prevalence of fatty liver leads to hepatic fibrosis.

Aim: To study the levels of serum ferritin in reproductive age group women and postmenopausal women with Nonalcoholic fatty liver disease. This study was conducted in patients visiting Sree Mookambika Institute of Medical Sciences, Kulasekharam, Kanyakumari District.

Methods and Materials: Thirty reproductive age group women and postmenopausal women with Nonalcoholic fatty liver disease and thirty normal reproductive age group women and postmenopausal women participated in the research comprising a total of sixty subjects.

Statistical Analysis: Analysis was performed by using SPSS version 23.0 which determined the percentage, mean and standard deviation. The unpaired sample test was used to study the levels of serum ferritin in reproductive age group women and postmenopausal women with Nonalcoholic fatty liver disease**.**

Results: The mean levels of serum ferritin were higher in reproductive age group women and postmenopausal women with non-alcoholic fatty liver disease, when compared to controls.

Conclusion: When this marker serum ferritin is early identified, intervention can be done earlier, and prevention of coronary heart disease and chronic liver disease can be done.

Keywords: Serum Ferritin, Nonalcoholic Fatty Liver Disease, Coronary Heart Disease, Chronic Liver Disease, Obesity.

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Introduction

The overall incidence of Non-alcoholic fatty liver disease has been growing rapidly and consequently this disorder has emerged as a leading cause of chronic liver disease worldwide. India has witnessed a sharp rise in the incidence of Non-alcoholic fatty liver disease. Prevalence of Non-alcoholic fatty liver disease was 30.77% in a study conducted among adults of a rural community in Haryana, India [1].

The initiating events in Non-alcoholic fatty liver disease are based on the development of obesity and insulin resistance, leading to increased hepatic free fatty acid influx [2]. Stages of Non-alcoholic fatty liver disease are fatty liver, non-alcoholic steatohepatitis, fibrosis and cirrhosis [3]. NAFLD is generally considered as a hepatic manifestation of metabolic syndrome [4]. Serum ferritin has been proposed to be the marker for NAFLD and oxidative stress in different studies. Serum ferritin is known as an acute phase reactant of inflammation [5]. Ferritin is the storage form of iron. Various studies have shown its rise in nonalcoholic fatty liver disease is due to hepcidin. Hepcidin is overexpressed due to stimuli like iron stores, IL-6, endoplasmic reticulum stress that causes an increase in iron retention from macrophages and hepatocytes. In addition, increased hepcidin levels are caused by hepatocyte necrosis and the systemic inflammatory state induced by NAFLD itself [6]. Serum ferritin acts as a marker for NAFLD determination [7]. In postmenopausal women, oestrogen decreases due to the cessation of ovarian function. Iron stores increase due to decreasing menstrual periods [8]. By evaluating the predictor like serum ferritin in

reproductive age group women and postmenopausal women with fatty liver, the incidence of breast cancer, ischemic heart disease, and metabolic syndrome can be predicted. The severity of liver damage leading to chronic liver disease can also be evaluated

Aims and Objectives:

1. To study the levels of serum ferritin in reproductive age group women and postmenopausal women with non-alcoholic fatty liver disease
2. To find factors related to non-alcoholic fatty liver disease in these study subjects and to study association of liver enzymes in these patients and compare with age matched control group

Materials and Methods

The study included thirty reproductive age group women and postmenopausal women with non-alcoholic fatty liver disease and thirty normal reproductive age group women and postmenopausal women who visited Sree Mookambika Institute of Medical Sciences, Kulasekharam, Kanyakumari District. Thirty reproductive age women and postmenopausal women with imaging evidence of fatty liver (USG) and elevated liver enzymes were selected for the study. Thirty controls with no evidence of fatty liver were selected. Patients with Obesity, Diabetes, Hyperlipidemia, Hypertension, pregnant women, patients on Vitamin-E for fatty liver were excluded from the study. After getting clearance from the Institutional Ethical Committee (SMIMS/IHEC No:1/33/2018), study population was selected and examined after informed consent. Venous blood (5ml) was collected from patients around 8.00AM in fasting stage. Serum and plasma were separated. Plasma glucose, triglycerides, alanine transaminase, aspartate transaminase, gamma glutamyl transferase and serum ferritin were calculated. Abdominal ultrasonogram was done for all sixty reproductive age women and postmenopausal women. USG revealed bright hepatic echoes, increased hepatorenal echogenicity, vascular blurring of portal or hepatic vein, enlarged liver due to fat as unique sonographic features in NAFLD patients. Plasma glucose (by Hexokinase method), serum triglycerides, SGOT, SGPT, GGT were estimated in fully automated analyzer and serum ferritin was determined by Chemiluminescence Immunoassay method.

Results

In the excel sheet, data collected was entered. Analysis was performed by using SPSS version 23.0.It determined the percentage, mean and standard deviation. P value of <0.01 is obtained with mean weight of cases and controls. P value of <0.01 is obtained with mean waist circumference and BMI of cases and controls.

Table 1: Comparison of age, weight, waist circumference, BMI between cases and controls

S. No.	Parameter	Cases Mean±SD	Controls Mean±SD	p-value
1.	Age	47.4±14.5	45.9±14.3	>0.05
2.	Weight	61.1±3.2	58.2± 4.1	<0.01
3.	Waist circumference	97.3± 5.5	85.4 ±2.8	<0.01
4.	BMI	31.4 ±2.7	23.3±1.7	<0.01
5.	Systolic B.P.	124.3±10.4	127.0±10.2	>0.05
6.	Diastolic B.P.	79.6 ±6.6	79.0±6.0	>0.05

Mean fasting plasma glucose of study group is higher with a p value of 0.01 than the control group. Mean levels of triglycerides, AST, ALT, GGT were higher in the study group with a p value of <0.01.

Table 2: Comparison of fasting plasma glucose, triglycerides, AST, ALT, GGT between cases and controls

S. No.	Parameters	Cases Mean±S.D.	Controls Mean±S.D.	p-value
1.	Fasting plasma glucose	132.3±5.4	97.9±11.1	<0.01
2.	Triglycerides	165±12.0	114.2±6.6	<0.01
3.	AST(SGOT)	42.8±8.8	18±2.7	<0.01
4.	ALT(SGPT)	47.7±7.6	27.9±2.1	<0.01
5.	GGT	44.07±2.9	34.87±5.1	<0.01

Mean serum ferritin in control group is 11.99±4.1µg/dl whereas in study group, it is 27.07±7.3µg/dl. Mean serum ferritin level in study group is higher than that of the control group with a p value of <0.01.

Table 3: Mean serum ferritin levels between study group and control group

S. No.	Parameter	Cases Mean±S.D.	Controls Mean±S.D.	p-value
1.	Serum Ferritin	27.07±7.3	11.99±4.1	<0.01

The Unpaired sample "t" test revealed the significant difference between the bivariate samples in Independent category. The "p" values were found to be statistically significant at <0.05

Table 4: Comparison of Serum Ferritin µg/dl between groups by Unpaired –"t"test

Groups		N	Mean	S.D.	t-value	p value
Serum Ferritin (µg/dl)	Cases	30	27.07	7.32	9.795	0.0005
	Controls	30	11.99	4.18		

Highly significant at p value <0.01

Table shows mean Serum ferritin between control and study group.

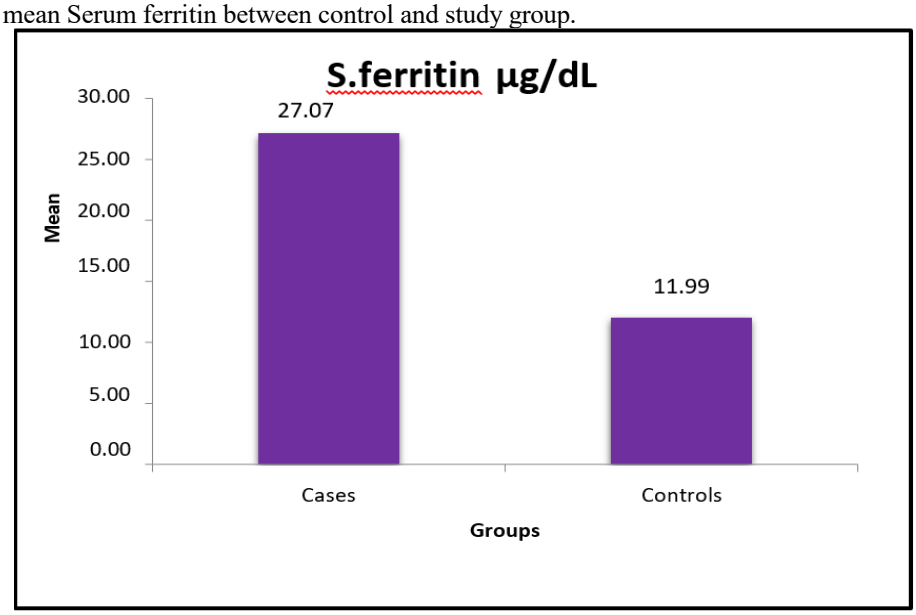

Figure 1: S. ferritin µg/dl

Discussion

The deposition of fat within liver parenchyma is known as Non-alcoholic fatty liver disease. Oxidative stress, insulin resistance and systemic inflammation subscribe to the etiopathogenesis of NAFLD [9].The mean level of serum ferritin was found to be 27.07±7.3µg/dl which is significantly higher than that of the control group (11.99±4.1). The research performed by Modares Mousavi et al (10) and Ponelopi Manousou et al [11] showed similar statistical significance.

Serum ferritin elevation can be caused by hepcidin [12]. Hepcidin, an iron homeostasis regulatory peptide hormone, is overexpressed in NAFLD leading to a decrease in iron intestinal absorption and an increase in iron retention from macrophages and hepatocytes. Thus serum ferritin is increased in reproductive age group women with Nonalcoholic fatty liver disease due to hepcidin. Serum ferritin acts as a marker for NAFLD determination. Ferritin elevation in inflammation is related to leakage of hepatocyte ferritin from damaged cells and release of ferritin from macrophages in inflammation [13].

Ferritin levels are elevated in NAFLD patients due to increased hepatic iron stores. Iron stores rise in postmenopausal women due to cessation of menstruation. In a research by Melanie D Beaton et al, similar results were seen. Aranda et al observed that elevated lipid peroxidation was caused by higher circulating serum ferritin. Due to insulin resistance and chronic liver inflammation, serum ferritin is elevated in NAFLD patients. [14]. Iron is a potent pro-oxidant and induces cellular damage in various tissues of the body resulting in generation of Reactive Oxygen Species. Thus increased serum ferritin concentration correlates with increased oxidative stress in NAFLD patients [15].

Ferritin is involved in NAFLD pathogenesis by promoting apoptosis and inducing signaling cascades related to inflammation, lipid transport and fibrogenesis. [16] It is shown in some studies that increased deposition of hepatic iron catalyzes generation of reactive oxygen species. It is also shown that production of reactive oxygen species causes oxidative stress damage to cells leading to insulin resistance that plays a key role in development of NAFLD. Ferritin increases the risk of insulin resistance through inflammation and oxidative stress [17]. In the state of insulin resistance, the beta oxidation of fatty acids is inhibited, which further promotes the accumulation of fat in the liver. Ferritin leaks from damaged cells into the bloodstream in NAFLD patients. Since Ferritin is an acute phase reactant, it has been assumed that the ferritin elevation is secondary to the inflammation of steatohepatitis. In Framingham heart study, higher ferritin was associated with greater risk of developing NAFLD [18]. During menopause, estrogen decreases due to cessation of ovarian functions. Thereby, iron stores are increased due to decreasing menstrual periods.1µg/L serum ferritin corresponds to 120µg storage iron per kg bodyweight. Body iron storage

is 12mg/kg bodyweight after menopause [19]. Kowdley et al. observed that hyper ferritinemia is common in patients with NAFLD. Serum ferritin, a marker of insulin resistance is a significant predictor of CVD and mortality events. Higher ferritin is linked to increased risk of hyperlipidemia, obesity and diabetes with relative risk of cardiovascular diseases.

Ferritin plays a pro-inflammatory role. Higher ferritin levels indicate it as an acute phase reactant. Higher ferritin levels represent iron overload and are toxic for myocardium and liver Elevated levels of ferritin is linked with increased morbidity and CVS mortality [20]. Liver enzymes AST, ALT and GGT were elevated in the present study .The elevated liver enzymes reflect hepatic inflammation and liver injury. Among liver enzymes, ALT is elevated in cases than controls which is statistically significant and similar findings are seen in a study done by Thirumoorthy Natarajan et al [21]. Various epidemiological studies published recently had shown that elevated levels of ferritin had been correlated with the event of risk factors for CVD.

Conclusion:

NAFLD is due to accumulation of fat in liver. Insulin resistance is mainly concerned with the etiopathogenesis of NAFLD. Serum ferritin is the storage form of iron and it is increased due to oxidative stress. In this present study, it is increased in reproductive age group women and postmenopausal women with Non-alcoholic fatty liver disease.

Insulin resistance causes significant effects on lipid metabolism and causes dyslipidemia which may cause impact on cardiovascular disease in the future. When this marker serum ferritin is early identified, intervention can be done earlier and coronary heart disease and chronic liver disease can be prevented. Simple changes in lifestyle, particularly at the earliest stage such as weight reduction, regular exercise, alteration of dietary habits and pharmacological intervention help to reduce the risk of CVD

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