Section: Biochemistry

Original Research Article

STUDY OF SERUM URIC ACID LEVELS IN PRE AND POSTMENOPAUSAL WOMEN WITH HEPATIC STEATOSIS ATTENDING A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Hepatic steatosis, 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 uric acid in pre and postmenopausal women with hepatic steatosis. This study was conducted in patients visiting Sree Mookambika Institute of Medical Sciences, Kulasekharam, Kanyakumari District. **Methods and Materials:** Thirty premenopausal and postmenopausal women with hepatic steatosis and thirty normal premenopausal 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 uric acid in pre and postmenopausal women with hepatic steatosis. **Results:** The mean levels of serum uric acid were higher in pre and postmenopausal women with hepatic steatosis, when compared to controls. **Conclusion:** When this marker serum uric acid is early identified, intervention can be done earlier and coronary heart disease can be prevented.

INTRODUCTION

Hepatic steatosis is a condition where excess fat builds up in the liver. In recent days, hepatic steatosis has become one of the world"s leading causes of chronic liver disease. Obesity, metabolic abnormalities like insulin resistance, hypertension, dyslipidemia, atherosclerosis and systemic micro-inflammation are often associated with hepatic steatosis.[1] The burden of disease has raised from 15% in 2005 to 25% in 2010 due to increasing rates of obesity.[2] Currently, the burden of disease has raised to 35% in 2025.According to WHO criteria, hepatic steatosis is defined as the presence of fat in liver either on imaging or on liver histology after the exclusion of secondary causes of fat accumulation in the liver [significant alcohol consumption, certain medications and other medical conditions].[3] As hepatic steatosis is potentially progressive towards hepatic fibrosis and its associated complications, the need for predictive factors of fatty liver disease is mandatory.[4] Among the different serum markers considered, serum uric acid has emerged as a possible predictor of severity of liver damage in fatty liver disease.[5]

Uric acid is the end product of purine metabolism. Various studies have shown that insulin resistance is involved in the pathogenesis of hepatic steatosis. Hyperuricemia is the reflection of insulin resistance in hepatic steatosis.[6] Estrogen causes increased renal clearance of uric acid.[7] Lack of estrogen causes hyperuricemia in postmenopausal women. By evaluating the predictor serum uric acid in pre and postmenopausal women, the incidence of breast cancer, ischemic heart disease, 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 uric acid in pre and postmenopausal women with hepatic steatosis
2. To find factors related to hepatic steatosis 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 premenopausal and postmenopausal women with hepatic steatosis and thirty normal premenopausal and postmenopausal

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women who visited Sree Mookambika Institute of Medical Sciences, Kulasekharam, Kanyakumari district. Thirty premenopausal 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 and 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 uric acid were calculated. Abdominal ultrasonogram was done for all sixty premenopausal 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 hepatic steatosis patients. Plasma glucose was estimated by Hexokinase method in fully automated analyzer. Serum triglycerides, SGOT, SGPT, GGT &serum uric acid were also estimated in fully automated analyzer.

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.	Parameters	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 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

The mean serum uric acid in control group is 4.8±0.9mg/dl, whereas the mean serum uric acid in the research group is 7.9±1.6mg/dl. Thus, in the research group, the mean serum uric acid is higher than the control group with a p value of <0.01.

Table 3: Mean Serum Uric Acid levels between control group and study group

S.no.	Parameter	Cases Mean±S.D.	Controls Mean±S.D.	p-value
1.	Serum Uric acid	7.9±1.6	4.8±0.9	<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.

Comparison of Serum Uric acid mg/dl between groups by Unpaired –"t"test

Table 4: Unpaired –"t" test"s comparison of serum uric acid levels between cases and controls

Groups		No.	Mean	S.D.	t-value	p value
Serum Uric acid (mg/dl)	Cases	30	7.97	1.69	8.864	0.0005
	Controls	30	4.86	0.9		

Highly significant at p <0.01 level

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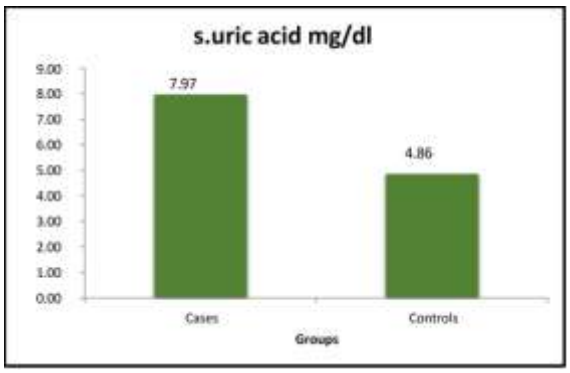

Table shows mean Serum Uric acid between control and study group.

DISCUSSION

The deposition of fat within liver parenchyma is known as Hepatic steatosis. Nutritional, metabolic and genetic factors cause the occurrence of Hepatic Steatosis. Oxidative stress, insulin resistance and systemic inflammation subscribe to the etiopathogenesis of Hepatic steatosis.[8] This research was conducted to evaluate if serum uric acid is increased in premenopausal and postmenopausal women with hepatic steatosis.

The corresponding biochemical parameters such as fasting plasma glucose, serum triglycerides, liver enzymes such as AST, ALT and GGT were also estimated in this study. In the present study, the mean AST in cases was 42.8±8.8IU/L which is higher than that of the control group (18±2.7). In a study done by Chengfu Xu, this statistically significant difference was noted.[9] The mean ALT in cases was 47.7±7.6 IU/L which is higher than that of the control group (27.9±2.1). This is statistically significant and similar findings are seen in a study done by Kathy A. Osborne.[10]

The mean GGT in cases was 44.07±2.9IU/L and in control group it was 34.87±5.1 which is statistically significant. The similar findings are seen in study presented by Harsharan Kaur et al.[11] The elevated liver enzymes reflect hepatic inflammation and liver injury.

Uric acid is a product of metabolic breakdown of purine nucleotides and it is a normal component of urine. Xanthine oxidase is an enzyme that catalyzes the formation of uric acid. Uric acid is an antioxidant and is a strong reactive oxygen species & peroxynitrite scavenger.[12] Uric acid also plays a key role in neuroprotection.[13] Zhang et al found that hyperuricemia is a prevalent finding in patients with Hepatic steatosis.[14]

The mean serum uric acid levels (7.9±1.6mg/dl) were found to be comparatively higher in cases than in controls (4.8±0.9). Equal statistical significance was reported in a study by S.Petta et al.[15] Different studies have shown a direct correlation of serum uric acid levels with Hepatic steatosis.

Uric acid causes insulin resistance by reducing nitric oxide bioavailability. It impairs endothelial function by inducing intracellular oxidative stress. Uric acid also affects adipocytes with direct pro-inflammatory and prooxidative effects that play an important role in the development of insulin resistance whereas insulin resistance inhibits uric acid excretion.[16] Hyperuricemia is due to insulin resistance in Hepatic steatosis patients. Thus Li et al found that serum uric acid is a marker for Hepatic steatosis.[17] Similar results were also found in previous study done by Jeffrey C Sirota et al.[18]

Estrogen causes increased renal clearance of uric acid. Estrogen falls in postmenopausal women due to cessation of ovarian function. In postmenopausal women, lack of estrogen induces hyperuricemia.[19]

National Health and Nutrition Examination Survey[NHANES-I] performed in US in 1975 explained a significant association between hyperuricemia and CVD mortality. This study explained that 1mg/dL increase of serum uric acid caused 48% elevation of CAD risk.[20]

CONCLUSION

Hepatic steatosis is due to accumulation of fat in liver. Insulin resistance is mainly concerned with the etiopathogenesis of Hepatic steatosis. Serum uric acid is the product of purine metabolism and causes insulin resistance by reducing nitric oxide bioavailability, whereas insulin resistance decreases excretion of uric acid and thereby causes hyperuricemia. In this present study, it is increased in pre and postmenopausal women with hepatic steatosis.

Insulin resistance causes significant effects on lipid metabolism and causes dyslipidemia which may cause impact on CVD in the future. When this marker serum uric acid is early identified, intervention can be done earlier and coronary heart 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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