ESTIMATION OF CHOLESTEROL AND URIC ACID LEVEL IN MENOPAUSE

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ABSTRACT

Background: Menopause is a natural physiological process characterized by the permanent cessation of menstruation resulting from the loss of ovarian follicular activity. It commonly occurs between the ages of 45 and 55 years and is associated with progressive hormonal and metabolic changes, particularly a decline in oestrogen production. These hormonal alterations may significantly affect lipid and purine metabolism, thereby increasing the risk of metabolic and cardiovascular disorders.

Aim: This study was designed to evaluate serum cholesterol and uric acid levels in postmenopausal women and compare them with those of premenopausal women in order to determine whether the observed changes are associated with menopause or aging.

Materials and Methods: A comparative cross-sectional study was carried out involving 100 postmenopausal women aged 45–78 years and 50 apparently healthy premenopausal women aged 20–45 years who served as controls. Serum cholesterol levels were determined using the enzymatic cholesterol oxidase-peroxidase method, while uric acid was estimated using the

uricase-peroxidase method. Statistical analysis was performed using Student's *t*-test with level of significance set at $p < 0.05$.

Results: The results showed that serum cholesterol and uric acid levels were significantly higher in postmenopausal women compared with premenopausal controls ($p < 0.05$). However, no statistically significant differences were observed when menopausal women were stratified according to age groups ($p > 0.05$). Similarly, no significant age-related differences were observed among the control group.

Conclusion: menopause significantly influences serum cholesterol and uric acid levels due to hormonal changes associated with oestrogen deficiency. Regular monitoring of these biochemical parameters, together with lifestyle modifications such as healthy diet, physical activity, and weight management, is recommended for postmenopausal women.

Keywords: Menopause, Cholesterol, Uric Acid, Oestrogen, Hyperuricaemia, Postmenopausal Women

INTRODUCTION

Menopause, which is a normal part of aging, marks the end of reproductive years and the termination of cyclic ovarian functions, which are indicated by cyclic menstruation [1]. Menopausal transition, a time when the endocrine, biochemical, and clinical signs of impending menopause start, is what heralds it [2]. Every woman experience this "change of life" in a distinctive way, and it often happens about age 51. Menopause is seen as "natural" and a typical aspect of aging when it happens between the ages of 45 and 55 [2]. However, some women may go through menopause early due to ovarian damage from chemotherapy or surgical procedures like hysterectomy. Premature menopause is defined as menopause that happens before the age of 45, regardless of the reason. Genetics, autoimmune diseases, and medical procedures can all cause premature menopause. Some women will just have the end of their menstrual cycle as a symptom, while others will face more serious mental and physical difficulties, such as memory loss, hot flashes, night sweats, mood swings, hurting joints, and thinning hair. Menopause can also lead to the development of a number of chronic illnesses, including an increased risk of cardiovascular disease, osteoporosis, and gout [3].

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stimulated by the regulating hormones, menopause takes place. Because they lack the hormone oestrogen, menopausal women typically have higher levels of uric acid and cholesterol in their blood [3]

A crucial part of the cell membrane's lipid bilayer is cholesterol. The phospholipid molecules, which are the other main component of the lipid layer of the cell membrane, are positioned between the cholesterol molecules. According to Sembulingam and Sembulingam (2010), cholesterol is responsible for the structural integrity of the cell membrane because it aids in packing the phospholipids, which are soft and oily structures, in the membrane. It is actively required for the manufacture of estrogen because it acts as a precursor to steroids. Menopause causes the synthesis of estrogen to stop, which raises the plasma level of cholesterol because it is no longer needed for synthesis [4].

In primates, including humans, purines and nucleic acids are metabolised primarily to produce uric acid (Yu *et al*., 2023). The enzyme xanthine oxidase converts xanthine into uric acid via a common route. It is further converted into the more water-soluble version of allantoin in the majority of other mammals, but humans are unable to undergo this transformation. Humans are more susceptible to the clinical consequences of hyperuricaemia, such as gout, due to its poor solubility. Man is vulnerable to the clinical consequences of hyperuricaemia, such as gout and kidney impairment, due to its low solubility. Oedema and joint discomfort are signs of gout [5,6]. This study's primary objective is to investigate menopausal cholesterol and uric acid levels.

Recent years have been attempts to look at how menopause affects serum levels of uric acid and cholesterol. Compared to younger women, older women are more likely to have conditions linked to high levels of uric acid and cholesterol. Is this related to menopause or aging?

The aim of this study was to determine the serum levels of uric acid and cholesterol in menopausal women and compare them with those of a control group. Specifically, the study sought to evaluate the effect of menopause on serum uric acid and cholesterol levels and to determine the relationship between menopause duration, serum cholesterol, and uric acid levels.

MATERIALS AND METHODS

Study Population

The study population consisted of women attending Iyi-Enu Mission Hospital. A total of 150 female participants were recruited and categorized into two groups. The test group comprised 100 postmenopausal women aged 45–78 years with a mean age of 62 years, while the control group consisted of 50 apparently healthy premenopausal women aged 20–44 years with a mean age of 33 years. Participants with known histories of dyslipidaemia, gout, liver disease, renal disease, tuberculosis, heart failure, pregnancy, lactation, or harmful alcohol/substance use were excluded from the study to minimize confounding variables.

Sample Size Determination

The sample size for the study was determined using a convenience sample

approach based on the availability of eligible participants who met the inclusion criteria during the study period. A total of 150 participants were considered adequate to provide comparative data between postmenopausal and premenopausal women for the biochemical parameters under investigation.

Sampling Method and Participant Recruitment

A purposive sampling technique was employed for participant recruitment. Eligible postmenopausal women attending the hospital during the study period were consecutively recruited after screening for eligibility criteria. The control group of premenopausal women was selected from hospital staff, students, and other apparently healthy female volunteers within the hospital community. Information about the study objectives and procedures was explained to all participants, and written informed consent was obtained prior to sample collection and data acquisition.

Inclusion Criteria

Only post-menopausal women were selected.

Exclusion Criteria

The exclusion criteria were determined through a combination of medical history assessment, clinical evaluation, and review of participants' medical records. Pregnant women and lactating mothers were identified through self-reporting and clinical history. Participants with liver failure, kidney failure, tuberculosis, or heart failure history were excluded based on documented medical diagnoses, hospital records, current medications, and physician reports where available. History of harmful alcohol intake or substance use was assessed through structured interview questions and self-reported social history during participant recruitment. These measures were taken to minimize confounding factors that could influence the biochemical and clinical parameters under investigation.

Specimen Collection and Processing

Blood samples (4mls) were collected from each subject by venipuncture using sterile needles and syringes after disinfecting the site with methylated spirit. The sample was carefully dispensed into sterile plain containers and labelled appropriately. These were allowed to clot after which they were carefully dislodged with a dropper pipette (avoiding lysis) and were centrifuged at 3000rpm for 10minutes. The sera were frozen until the time of analysis.

Chemicals and Reagents

The following chemicals and reagent were used for this study; 4-aminoantipyrine, phenol, peroxidase, cholesterol esterase, cholesterol oxidase, pipes buffer, hepes buffer, 3,5-Dichloro-2-hydroxyl-bezenesulfonic acid, 4-aminophenazone, peroxidase and uricase. All regents were of analytical grade.

Method of analysis

Cholesterol estimation

The serum levels of cholesterol were determined using the method described by Trinder (1967), with kit assay system (Randox, United Kingdom).

Principle: The principle of this method was based on the enzymatic hydrolysis of cholesterol ester by cholesterol esterase to form cholesterol and fatty acids. The cholesterol is being oxidized in the second stage of the reaction by cholesterol oxidase

to form cholestene -3- one and hydrogen peroxide. The hydrogen peroxide formed reacts in the third stage of the reaction with phenol and 4-Aminoantipyrine in the presence of peroxidase to form a pink quinoneimine whose intensity measured colorimetrically at 500nm is directly proportional to cholesterol concentration.

Procedure: 0.01ml (10µL) of the sample was added to 1.0ml (1000µL) of the reagent. The solution was well mixed and then incubated for 5minutes at 37ºC. The absorbance of the test sample was read colorimetrically at 500nm against water blank. The cholesterol concentration was calculated using the standard formula.

Reference range: < 5.17mmol/L (200mg/dl).

Uric Acid Estimation

The serum level of uric acid was determined using the method described by Fossat *et al* (1980), with kit assay system (Randox United Kingdom).

Principle: The principle of this reaction was based on the conversion of uric acid by uricase to allantoin and hydrogen peroxide which under the catalytic influence of peroxidase, oxidizes 3,5-Dichloro-2-hydroxylbenzenesulfonic acid and 4-aminophenazone to form a red-violet quinoneimine compound whose intensity measured colorimetrically at 520nm is directly proportional to the uric acid concentration in the sample

Procedure: 0.02ml (20µL) of the sample was added to 1.0ml (1000µL) of the reagent. The solution was well mixed and then incubated for 5minutes at 37ºC. The absorbance of the test sample was read colorimetrically at 520nm against water blank. The uric acid concentration was calculated using the standard formula.

Reference range: 2.4 – 5.7mg/dl

Data Analysis

The data obtained from this study were analyzed using appropriate statistical methods. Mean values and standard deviations were calculated for all variables. Comparisons between menopausal women and the control group were performed using the Student's t-test with Statistical Package for Social Sciences (SPSS) Windows version 16.0. Statistical significance was set at a p-value of ≤ 0.05.

RESULTS

The results obtained from this study are presented in Tables 1–3.

Table 1: Shows the mean cholesterol values were significantly higher in menopausal women than the control subjects. Also, the mean uric acid values were significantly higher in menopausal women than the control subjects.

Table 2: also discloses the mean serum cholesterol and uric acid levels of the age groups 45-60 years and 61-78 years in menopausal women, showed no statistically significant difference ($p>0.05$) in the values obtained of age group 45-60 years when compared with age group 61-78 years

Table 3: also discloses the mean serum cholesterol and uric acid levels of the age groups 20-35 years and 36-44 years in control subjects, showed no statistically significant difference ($p>0.05$) in the values obtained of age group 20-35 years when compared with age group 36-44 years.

TABLE 1: Presents the serum concentrations of cholesterol and uric acid in menopausal women compared with the control subjects in the study

Parameters	Menopausal women (n=100)	Control (n=50)	t-value	p-value
CHOL (mg/dl)	216.20 ± 59.71	182.4 ± 39.4	**4.20**	P<0.01
U.A (mg/dl)	10.38 ± 3.49	7.20 ± 3.16	**6.10**	P<0.01

KEY: n = number of subjects, P<0.05 = statistically significant, P>0.05 = not statistically significant, CHOL = Cholesterol and U.A = Uric acid

TABLE 2: shows the age-related distribution of serum total cholesterol and uric acid levels among menopausal women within the age groups of 18–60 years and 61–78 years in the study area

Parameters	Menopausal women 45-60 years (n=45)	Menopausal women 61-78 years (n=55)	t-value	p-value
CHOL (mg/dl)	215.20 ± 63.36	213.70 ± 51.00	**0.131**	0.311
U.A (mg/dl)	9.20 ± 2.34	9.70 ± 2.60	**0.568**	0.582

TABLE 3: Illustrates the age-related distribution of serum total cholesterol and uric acid levels among the control subjects within the age groups of 20–35 years and 36–44 years in the study area.

Parameters	Menopausal women 20-35 years (n=27)	Menopausal women 36-44 years (n=23)	t-value	p-value
CHOL (mg/dl)	196.70 ± 63.36	206.31 ± 48.20	**0.127**	0.220
U.A (mg/dl)	6.91 ± 2.05	7.02 ± 2.10	**0.510**	0.515

DISCUSSION

Menopause is the permanent cessation of menstruation resulting from the loss of ovarian follicular activity and decline in reproductive hormone production. It marks the end of a woman's reproductive phase and is clinically diagnosed after 12 consecutive months of amenorrhea in the absence of other physiological or pathological causes. Menopause commonly occurs between the ages of 45 and 55 years. [12].

The study's findings demonstrate that menopausal women's levels of uric acid and total serum cholesterol were altered. Regardless of age, menopausal women's total cholesterol and uric acid levels were not significantly different from those of premenopausal women (menstruating women) [5;3;6;9]. Menopausal women's elevated levels of uric acid and total cholesterol have been linked to hormonal changes and follicular development failure, where the plasma oestrogen levels that lower LDL while raising HDL and are also in charge of uric acid excretion in the urine are lower than those of premenopausal women [7;11]. Also due to Steatotic liver disease in premenopausal women refers to the accumulation of excess fat in the liver of women who have not yet undergone menopause. It is now broadly classified under the term Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), previously known as non-alcoholic fatty liver disease [4].

CONCLUSION

The findings of this study indicate that menopause is associated with alterations in serum uric acid and total cholesterol levels. However, the results showed no statistically significant difference in these parameters between menopausal and premenopausal women across the studied age groups. The observed variations in uric acid and cholesterol levels among menopausal women have been attributed to hormonal changes, particularly the decline in oestrogen production due to follicular depletion. Since oestrogen plays a role in regulating lipid metabolism and enhancing uric acid excretion, its reduction during menopause may contribute to metabolic changes, even though these changes were not statistically significant in this study.

RECOMMENDATIONS

Menopausal women can lower their cholesterol and uric acid levels by making certain lifestyle changes, such as maintaining a healthy weight by engaging in at least 30 minutes of moderate-to-intense exercise each day; dietary changes, such as eating more fish, whole grains and fibre, less refined sugar, less red meat and high-fat dairy products, more mono- and polyunsaturated fatty acids, and more fruits, vegetables, and legumes; Tran's fats should be avoided or reduced, and saturated fat intake should be less than 7% of daily calories.

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