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Original Research Article

A Study of Correlation between Body Mass Index, Blood Pressure and Lipid Profile Characteristics among Menopausal Women in Nanded, Maharashtra.

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Abstract

Introduction: menopause is the time in women's life when her menstruation stops. It occurs naturally or surgically induced. The average age at menopause in India is 47.5 years as compared to 51 years in other developing countries. Protective effect of estrogen decreases after menopause. There is increase in fat storage with simultaneous lipid deposition in central part of body. The body mass index (BMI) has a major influence on blood pressure and lipid profile and as such is a good predictor of hypertension and hyperlipidemia. Menopause predisposes the woman to the risk of ischemic heart disease.

Aim: This study was carried out to correlate between BMI, blood pressure and blood triglyceride level and low-density lipoprotein (LDL) in postmenopausal women.

Methods: The subjects were in two groups 60 premenopausal women and 60 postmenopausal women. The Parameters studied were BMI, blood pressure and lipid profile (HDL, Total Cholesterol, LDL, VLDL).

Results: we found significant increase in BMI, Blood pressure and total cholesterol, triglycerides, LDL, VLDL and decrease in HDL in postmenopausal women. We found positive correlation between BMI, blood pressure and lipid profile that shows increased risk of cardiovascular diseases in postmenopausal women.

Discussion: Estrogen modulates energy intake by exerting an anabolic effect and exhibiting a lipolytic effect**.** Estrogen reduces collagen accumulation, elastin loss and reduces smooth muscle cell proliferation. It decreases vascular remodelling. Estrogen decreases vascular resistance by production of vasodilators like nitric oxide, cyclic AMP and prostacyclin. Lack of estrogen also causes deranged lipid profile.

Conclusion: Since postmenopausal women lack cardioprotective action of estrogen. There is increased tendency of hypertension, obesity and atherogenic lipid profile in postmenopausal women.

Keywords: premenopausal women, postmenopausal women, blood pressure, body mass index, lipid profile.

Introduction

Menopause is defined as the cessation of menstruation for 12 consecutive months, marking an end of reproductive years [1]. Menopause is often defined as premature (prior to 40 years of age) and early (between 40 and 44 years). Menopause is an estrogen-deficient state, but unlike other hormone deficient states, it is not a disease, it is a normal physiological process. [2]

By the year 2025, 23% of the population will be aged 60 or above. [3] The average age of menopause in Indian women is 47.5 years compared to 51 years in developing countries. [4] Though the age of onset of menopause has remained constant, the life expectancy in women has increased substantially. Currently a woman can expect to live a third of her life after attaining menopause in a state of estrogen deficiency. [5]

Menopause is associated with lipid metabolism disorders due to hormonal changes, such as decreased level of estrogen, progesterone and elevated follicular stimulating hormone, luteinising hormone.

Estrogen causes fat deposition in gluteal and thigh region. After menopause due to estrogen deficiency there is increasing fat mass and tendency to deposit fat in central part of body that is in abdominal region.

Increased the body mass index (BMI) has influence on blood pressure and lipid profile, it is also a good predictor of hypertension and hyperlipidemia. [6]

Hormonal changes in menopausal women causes development of metabolic syndrome which is a group of risk factors for further development of cardiovascular diseases. In menopausal women there is increased sympathetic activity and decreased parasympathetic activity. Release of adrenaline in circulation results in lipolysis further increasing blood lipid level and accelerates risk of coronary artery disease. [7]

This study may also be useful for early diagnosis and primary prevention from cardiovascular disease in postmenopausal women.

Materials and Methods

The present study was carried out in 60 premenopausal and 60 postmenopausal women. The subjects were selected by simple random sampling method from office workers of Dr. Shankarrao Chavhan Govt. Medical college, Nanded and from relatives.

Selection Criteria-

A. Premenopausal women:

1. Age group 25-40 years, with regular menstrual cycle and an average length of 28 days.
2. Subjects in the follicular phase of their menstrual cycle.

B. Postmenopausal women:

1. Age group 45- 60 years.
2. They had completed a period of at least 12 months since their last menstrual period.

Exclusion Criteria:

The following women were excluded from study:

1) Those on oral contraceptive pills or hormonal therapy in any form.
2) Those consuming drugs that alter the cardiovascular functions.
3) Those having any history of diabetes, cardiovascular disease, surgical menopause or history of addiction to tobacco, alcohol, smoking.
4) Those suffering from any other disease or complication.

All the subjects were explained the procedure to alleviate any fear or apprehension. Before starting the procedure, the physical examination of all the subjects was done with the help of proforma and the consent form was signed by the subjects.

The following parameters of cardiovascular sympathetic function tests were recorded in all the subjects:

1. Basal pulse rate (beats/minute).
2. Resting blood pressure (mmHg).

1) Basal pulse rate (beats/minute): **[8]**

- The subject was first asked to take rest for 5 minutes.
- The subject's right forearm was semipronated and the wrist slightly flexed.
- Then the redial pulse was felt with three fingers slightly compressing the vessel against the underlying bone.
- The pulse rate was counted for one full minute and expressed as beats per minute.

2) Resting blood pressure(mmHg): [9]

(systemic arterial blood pressure- systolic blood pressure and diastolic blood pressure).

- The subject was asked to rest for 5 minutes.
- The subject was asked to sit with back supported and legs uncrossed. Based on the circumference of subject's arm, a regular adult cuff was chosen. The cuff of mercury sphygmomanometer was placed on subject's left arm 2.5-3 cm above the antecubital fossa at the heart level.
- First the systolic pressure was measured by the palpatory method. For this, the redial pulse was felt and the cuff was inflated to increase the pressure 30mmHg above the point at which radial pulse disappeared. Then while deflating the cuff 2-3mmHg/ second and simultaneously palpating radial pulse, the point where redial pulse first appeared was taken as systolic blood pressure.
- The systolic and diastolic pressure were then recorded by auscultatory method. For this the cuff was inflated to 30mmHg above the systemic blood pressure as measured by palpatory method. Then while deflating the cuff at the rate of 2-3mmHg/second, the first and last audible sounds were recorded as systolic and diastolic blood pressure respectively. Then the cuff was deflated completely.
- Similarly, one more reading was taken at an interval of 1 minute. Therefore, the mean of the two readings was considered as the resting blood pressure.

1. Anthrometric Parameters:

Various anthropometric parameters of our study were

- **Height [10]** -

We had measured height of patients using standard measuring technique, patient standing erect across wall, with bare feet, legs are straight, arms at sides, and shoulders relaxed, the back of the body touches/has contact with the wall at some point, preferably with heels, buttocks, upper back and head touching the wall by Stadiometer.

- **Weight [10] –**

We had measured weight of patients using standard weighing scale & recorded in kilograms.

- **Body Mass Index (BMI) (Quetelet's Index)[10]** -

Calculation of BMI was done by using formula

$$\mathbf{BMI} = \frac{\mathbf{Wt(kg)}}{\mathbf{Ht^2(m)}}$$

Estimation of serum lipid profile- [11]

Estimation of following parameters was done: total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein (VLDL).

Collection of blood sample [11]

From the women of reproductive age group, blood samples were collected during 6th-10th day of the menstrual cycle, as hormonal level varies with phases of the menstrual cycle. Serum lipid profile levels are more accurate when blood samples are collected 8-10 h after the last meal. Hence instructions were given to the subjects to take dinner at 9-10 p.m. and remain fasting overnight until blood samples were collected in the next day morning. Fasting blood samples were collected in the morning between 7 a.m. and 8 a.m. by venepuncture of antecubital vein, with all aseptic precautions. A test dose of 5 ml of blood was collected with disposable syringe in plain bulb. Clear, unhemolyzed serum was obtained by centrifuging blood at 3000 rpm for 15 min.

Methods of estimation- [11]

Estimation of total cholesterol was done by CHOD-PAP method. [Cholesterol + oxygen --(enzyme cholesterol oxidase)--> cholestenone + hydrogen peroxide. Hydrogen peroxide + 4-aminophenazone + phenol -- (enzyme peroxidase) >colored complex].

Estimation of HDL cholesterol was done by phosphotungstic acid method, of TGs by enzymatic calorimetric method, of LDL-cholesterol by using Friedewald formula and of VLDL-cholesterol by using the formula: [7] VLDL = TG/5.

Also, we studied Casteli risk index I (CRI-I) = TC/HDL-C

Casteli risk index I (CRI-II) = LDL-C/HDL-C

Atherogenic coefficient = (TC-HDL-C)/HDL-C

All the above-mentioned investigations were done by a qualified biochemist who was blinded about the background and BMI of the patient, using the Erba diagnostic kit. The assays were done on the day of collection of a blood sample by using Erba fully autoanalyzer XL 640. The Graph Pad Prism6 software was used for statistical analysis.

Results

Table 1: shows values of BMI and resting systolic blood pressure and diastolic blood pressure in premenopausal and postmenopausal women.

Sr. no.	Components	Premenopausal women (n=60)	Postmenopausal women (n=60)	Z value	P value
1	BMI (kg/m^2) (Mean ±SD)	23 ± 2.27	25.93 ± 2.91	1.96	<0.0001
2	Systolic blood pressure	114±7.369	120.5±7.35	4.68.	<0.0001
3	diastolic blood pressure	75.77±4.94	79.07±5.11	1.611	<0.0001

We found there is significant increase in BMI, resting systolic and diastolic blood pressure in postmenopausal women.

Table 2: shows values of lipid profile characteristics like Total cholesterol, Triglyceride, HDL-C, LDL-C, VLDL, in premenopausal and postmenopausal women.

Sr. no.	Components	Premenopausal women (n=60)	Postmenopausal women (n=60)	Z value	P value
1	Total cholesterol mg/dl (Mean ±SD)	170.5 ± 12,64	228.2 ± 24.64	10.6	<0.0001
2	Triglyceride mg/dl (Mean ±SD)	112.9 ± 24.78	150 ± 22.4	8.6	<0.0001
3	HDL mg/dl (Mean ±SD)	44.9 ± 6.9	36.13 ± 7.34	6.91	<0.0001
4	LDL mg/dl (Mean ±SD)	112.2 ± 20	171.8 ± 26	13.85	<0.0001
5	VLDL mg/dl (Mean ±SD)	25.75 ± 4.835	32.22 ± 7.691	5.38	<0.0001

We found there is significant increase in serum total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C), very low-density lipoprotein (VLDL) in menopausal women.

We found significant decrease in high density lipoprotein cholesterol (HDL-C) in postmenopausal women.

Table 3: Shows values of women Castelli risk index I (CRI -I), Castelli risk index II (CRI -II), Atherogenic coefficient (AC) in premenopausal and postmenopausal women.

Sr. no.	Components	Premenopausal women (n=60)	Postmenopausal women (n=60)
1	CRI – I	3.797	9.261
2	CRI-II	2.4988	4.755
3	Atherogenic coefficient	2.797	5.316

Also, we found there is increase in Castelli risk index I (CRI -I), Castelli risk index II (CRI -II), Atherogenic coefficient (AC) in postmenopausal women as compared to premenopausal women.

Discussion

In present study careful statistical analysis of lipid profile status in premenopausal and postmenopausal women was done. We studied 60 premenopausal women having age between 25 to 40 years with 60 postmenopausal women having age between 45 to 60 years.

The parameters that we studied were BMI, serum total cholesterol, triglyceride, HDL, LDL, VLDL between premenopausal and postmenopausal women.

Estrogen is secreted by the granulosa cells of ovary.

After menopause attrition of primordial follicles occurs and ovarian function gradually become diminished so there is decreased estrogen synthesis.

In our study we found statistically significant increase in BMI ($p<0.05$) in

Postmenopausal women when compared with premenopausal women.

Similar significant increase in BMI in postmenopausal women were noted by Nerella Sharvani et al. The cause for increase in BMI in postmenopausal women may be due to decreased level of estrogen. Estrogen suppresses food intake by acting pro-opiomelanocortin (POMC) neurons and regulates their cellular activity through estrogen receptors (ER). Moreover, there is strong association between leptin level and estrogen level. Leptin modulates energy balance. It exerts a lipolytic effects. Estrogen increases the leptin sensitivity by controlling the expression of leptin specific receptors. [12] After menopause, the ovaries fail completely to produce estrogen, resulting in dysregulation of energy metabolism that may induce an elevation in the total adiposity in the postmenopausal women.

In our study we found elevated resting systolic and diastolic blood pressure in postmenopausal women. Our findings were consistent with findings of Badaruddoza and Hundal et al. [13]

Studies in experimental animals showed that decreased estrogen level reduces collagen accumulation and elastin loss. It also reduces smooth muscle cell proliferation. Reduced arterial compliance in menopausal women may be initially functionally mediated and partially compensated by an increase in arterial diameter.

Later in menopausal women decrease in arterial compliance may become structural. The higher serum cholesterol, smaller high-density lipoprotein fraction, the increased peroxidation of lipoproteins, the endothelial dysfunction and enhanced wall stress resulting from the intermittently functionally elevated systolic blood pressure may set the stage for atherosclerotic lesion. These in turn could irreversibly reduce arterial distensibility and maintain systolic blood pressure on a rising tract. That may lead to increase in systolic blood pressure. [14]

Estrogen decreases vascular resistance by multiple mechanisms. It increases production of vasodilator substances like nitric oxide, cyclic AMP, prostacyclin.

Estrogen decreases endogenous vasoconstrictors like angiotensin II, endothelin-1 and catecholamines. It also increases synthesis of bradykinin and reduces endothelin-1 level. These substances have influence on vascular tone and structure. Estrogen prevents vascular remodelling and decreases total peripheral resistance. Esrogen reduces the circulating levels of homocysteine. This homocysteine contributes to vascular resistance by inducing endothelial cell damage, inhibiting endothelial cell growth and inducing smooth muscle cell growth. [15] So decreased estrogen level in menopausal women may lead to increased diastolic blood pressure.

our study there was statistically significant increase in total cholesterol, triglyceride, LDL and VLDL ($P<0.05$). Similar results were obtained by Madhavi et al. After menopause, as there is loss of ovarian functions and depletion of various ovarian hormones. This results in adverse changes in glucose and insulin metabolism along with derangement in body fat distribution, coagulation process, fibrinolysis and vascular endothelial dysfunction. The major effect of estrogen on lipid metabolism is by its action on regulation of various LDL receptors in liver. Estrogen acts on hepatic LDL receptors and leads to increased clearance of LDL-C there by regulating serum LDL level. Circulating estrogen is a regulator of lipoprotein lipase (LPL). LPL catalyzes the hydrolysis of VLDL to form IDL and later to LDL. After menopause lack of estrogen increases the level of lipoprotein lipase enzyme

which hydrolyses chylomicrons and triglyceride found in VLDL. Estrogen deficiency leads to, down-regulation of LDL receptors. The triglycerides are hydrolyzed to free fatty acids and glycerol by lipoprotein lipase enzyme. All these factors combined together leads to elevated TC, TG, LDL-C, and reduced HDL-C levels in serum of postmenopausal women leading to increased risk of coronary artery disease [16].

In addition to these factors, reduced physical activity and energy expenditure during menopause also play important role in alteration of lipid profile.

Conclusion

In our study we found positive co-relation between increased BMI, increased blood pressure and deranged lipid profile in menopausal women. Obesity, high blood pressure and deranged lipid profile are major risk factors for cardiovascular diseases.

This study provides indication for menopausal women to undergo screening for blood pressure and lipid profile. Also, primary prevention can be done by adopting healthy lifestyle modifications including physical exercise and by doing counselling regarding their diet and eating habits.

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