

A COMPARATIVE STUDY OF SERUM LIPID PROFILE BETWEEN PREMENOPAUSAL AND POSTMENOPAUSAL WOMEN IN KOTA, RAJASTHAN, INDIA

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ABSTRACT

Dyslipidemia in menopause is a known feature in women, which may lead to significant increase in the development of coronary heart disease (CHD). The present study was aimed at comparing the level of total serum cholesterol and their subfractions, in premenopausal with that of their postmenopausal counterparts.

Fifty (50) apparently healthy, nonpregnant females (25 premenopausal and 25 postmenopausal) were recruited for the study. Serum total cholesterol and their subfractions - high-density lipoproteins (HDL), low-density lipoproteins (LDL), very low-density lipoproteins (VLDL) and triglycerides (TG) were estimated using enzymatic and established mathematical methods. There was no significant difference in the total serum cholesterol and triglyceride between the two groups. There was however, a significant reduction of HDL and VLDL in the postmenopausal group ($P < 0.005$) and a significant increase in the level of LDL in the postmenopausal group ($P < 0.005$). The elevated LDL and the reduction of cardio protective HDL and VLDL is an indication that menopause is an independent risk factor for developing cardiovascular disease.

KEYWORDS: Menopause, Dyslipidemia, Cholesterol

INTRODUCTION

Menopause means permanent cessation of menstruation at the end of reproductive life due to loss of ovarian follicular activity. The effect of the hormonal changes associated with menopause on the serum lipid levels play important role in most cardiac disorders associated with menopause. Up to the age of 50 years, the prevalence of coronary artery disease (CAD) among women is lower than among men, but the incidence rises significantly after the menopause. The incidences of coronary heart disease have been observed to be increased in postmenopausal women.

Hypercholesterolemia is a key factor in the pathophysiology of atherosclerosis (Igweh et al, 2003). High levels of LDL and low levels of HDL are strongly associated with the risk of CAD (McNamara et al 1992). Smaller LDL particles (LDL-III) are considered more atherogenic than larger more buoyant species because of their increased susceptibility to oxidation (Dejagar et al, 1993) and their increased residence time in plasma (Rainwater, 2000). Plasma triglycerides concentration also has a determinative influence on the concentration of small dense LDL particles in normal population (Dejagar et al, 1993, Mendelsohn, 1999). After menopause, there is loss of ovarian function. This results in adverse changes in glucose and insulin metabolism, body fat distribution, coagulation, fibrinolysis and vascular endothelial dysfunction (Spencer et al, 1977). There is also derangement of lipoprotein profile independent of age (Bales, 2000). A number of changes that occur in the lipid profile after menopause are associated with increased cardiovascular disease risk. Lack of estrogen is an essential factor in this mechanism. Apart from maintaining friendly lipid profile, estrogen

changes the vascular tone by increasing nitrous oxide production. It stabilizes the endothelial cells, enhances antioxidant effects and alters fibrinolytic protein (Taddec et al, 1996). All these are cardioprotective mechanisms, which are lost in menopause.

MATERIALS AND METHODS

A group of 50 women, 25 premenopausal aged between 25-45 years and 25 postmenopausal aged between 55-65 years were studied after an informed consent and ethical clearance from the ethical committee of Government medical college, Kota. Exclusion criteria included obesity, pregnancy, diabetes mellitus, hypertension, hormonal contraception and heavy exercise. Fasting venous samples (10ml) were collected in heparinized bottles. This however, was done on the 7th day of the L.M.P. for the premenopausal group. Sample was centrifuged and plasma separated and stored in plastic tubes at 4⁰ c.

Total cholesterol was measured using established enzymatic methods of Allain et al (1974). HDL-C was isolated by HDL-C precipitant method (Lopes-virella et al, 1977). LDL was calculated with a formula from total cholesterol and triglycerides (Friedwald, 1972). Triglyceride was isolated enzymatically as described by Henry (1991) and the expert panel on detection, evaluation and treatment of high blood cholesterol in adults (Expert Panel, 1988). VLDL was calculated using the formula VLDL-C = TG/2.825mmol/L (Henry, 1991).

Statistical analysis was done using Excel, showing the mean and standard deviation.

Comparison of mean was by student t-test.

RESULTS

The mean, standard deviation and the P values for total cholesterol, HDL-C, LDL-C VLDL-C and Triglycerides are shown in Table 1. There was no significant difference in the total cholesterol and the triglycerides between the two groups (P>0.005). However there was significant reduction in HDL and VLDL-C fraction in the postmenopausal group and a significant increase in the value of LDL-C (P<0.005).

Table 2 shows the HDL/LDL ratio between the two groups.. The mean age for premenopausal women was 36.7±4.74 and that for post menopausal women was 60.9±9.9

Table 1: Showing Total Plasma Cholesterol and its Subfractions in Pre and Postmenopausal Women

	Total Cholesterol	HDL	LDL	VLDL	TG
Premenopausal women(n-25)	4.30±0.68	1.37±0.020	2.7±0.74	0.28±0.04	1.12±0.43
Post Menopausal women(n-25)	4.51±0.74	1.23±0.21	3.03±0.69	0.25±0.04	1.3±0.73
P Values	0.117	0.005	0.039	0.004	0.137
Significance	P>0.05	P<0.05	P<0.05	P<0.05	P>0.05

Table 2: HDL/LDL Ratio in Pre and Postmenopausal Women

	HDL	LDL	HDL/LDL
Premenopausal women	1.37	2.7	1:2
Post Menopausal women	1.23	3.03	1:3

DISCUSSIONS

There are variations in lipid levels obtained in different individuals based on race, age, sex, obesity, exercise,

smoking, alcohol, diet, diseases like hypertension, chronic liver and renal diseases (Gordon et al, 1987, Jarikre et al, 2000). However, in this study we tried to exclude the co founding variables and results obtained can be taken as the standard normal. The premenopausal values are similar to values obtained in other studies in different parts of the world (Ajose et al, 2002, Igweh et al, 2003). However, there is paucity of literature in postmenopausal values, but studies on total cholesterol alone done in Ibadan showed no significant change (Otolorin et al, 1989), as was the case in our study.

Studies in some centers in developed countries showed higher values with dyslipidemia being taken as cholesterol > 240mg% (6.21mmol/l) and occurs in 25-33% of women (Grundty, 1994, Gordon et al, 1989).

As a result of racial, genetic and technical differences, it has been suggested that our own cut off level should be adjusted to suit our peculiar circumstances. In determining the risk of cardiovascular disease, the absolute cholesterol value is not the most important factor; rather the concentrations of the various subclasses of cholesterol.

In the present study there was no significant differences in the total cholesterol level but there was significant reduction in the cardio protective HDL-C and VLDL-C and significant increase in the atherosclerotic LDL-C. This is in agreement with findings in other studies (Jenson et al, 1990, Eder et al, 1982, Igweh et al, 2003).

It has also been estimated that for any 1mg/dl (0.026mmol/ml) increase in HDL-C, there is a 3% decrease in risk of coronary artery disease and a 4.7% decrease in the risk of mortality from cardiovascular disease (Okonofua 1990).

In this study this protective effect was lost by five fold. Again the HDL/LDL ratio was increased in the post menopausal group and it has been shown that HDL/LDL ratio is a significant predictor for the development of arteriosclerosis.

There is no doubt from this study that the changes that occur in the lipid profile after menopause is not friendly for the cardiovascular health of our women. It is generally believed that postmenopausal symptoms are less in our women than their caucasian counterparts. This may not be true because several studies in the past have shown psychological, physical, biochemical, hormonal and vasomotor parameters that are not of great variance with what is obtained elsewhere (Grundty, 1994, Mandel et al, 2002, Pirway et al, 2002). The problem is that the harsh climatic and poor socioeconomic environment overwhelms our women that they hardly ever complain about menopausal symptoms unless it is severe.

The elevated LDL and the reduction in the cardio protective HDL and VLDL is an indication that menopause is an independent risk factor for developing cardiovascular disease in our environment.

Several studies have shown the beneficial effects of hormonal replacement therapy on the lipid profile of menopausal women (Abbot et al, 1988, Stampfer et al, 1991, Stampfer, 1991). However, controversy exists as to whether these changes culminate to reduced risk of cardiovascular heart disease. Observational studies over the years have touted the beneficial effects of hormone replacement therapy (HRT) in preventing coronary heart disease in post menopausal women (Hulley et al, 1998).

More recent studies have however, cast some doubts on the beneficial effects of HRT especially in patients with established cardiovascular disease (Cheng, 2000). Further studies are needed in this area. It should be noted that our post menopausal women have unfriendly lipid profile; it is thus important to note this and devise means of correcting the dyslipidemia since the use of HRT and lipid lowering drug is still controversial. It is important to counsel on proper dietary, social and physical habits.

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