

# Study of serum lipid profile in premenopausal and postmenopausal women of Aurangabad district

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## Abstract

**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. **Aims and Objectives:** To measure Total cholesterol, Triglycerides, HDL in premenopausal cases were compared to postmenopausal women. **Material 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 from MGM medical college, Aurangabad. Total cholesterol, Triglycerides, HDL were measured by kit method on Analyser. And LDL and VLDL were calculated by Friedwald's formula. Statistical analysis was done by student t-test. Mean values of serum Cholesterol, Triglycerides, HDL, LDL and VLDL in premenopausal cases were compared to postmenopausal women. **Conclusion:** TC and TG is found to be increased in post-menopausal women due to estrogen deficiency when compared to pre-menopausal women and is statistically significant ( $P < 0.05$ ). There was no statistical difference seen in HDL-C in pre and postmenopausal women. ( $P > 0.05$ ) In our study, the VLDL-C and LDL-C was increased in post-menopausal women as compared to premenopausal women and was statistically significant ( $P < 0.05$ ). Due to the change in the lipid pattern and loss of cardioprotective effect of estrogen, post-menopausal women are at increased risk of developing cardiovascular disease. So it is important to educate each and every postmenopausal women to undergo screening for abnormal lipid profile.


**Keywords:** TC: Total cholesterol, TG : Triglycerides, HDL-C : High density lipoprotein cholesterol, LDL-C : Low density lipoprotein cholesterol, VLDL-C : Very low density lipoprotein cholesterol. Friedwald's formula:  $VLDL = \text{Triglycerides} / 5$ ,  $LDL = TC - (HDL-C + VLDL-C)$ .

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## 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. Many workers have shown increased prevalence of coronary heart disease among women who had premature menopause or surgical menopause<sup>1,2</sup>. In these women, plasma cholesterol levels and triglyceride levels are invariably increased after menopause<sup>1,3,4</sup>. A decrease in HDL level in postmenopausal women may predispose them to increased atherosclerotic heart disease<sup>5</sup> besides an increase in LDL level<sup>6</sup>. It has been proposed that estrogen exerts cardioprotective action among pre-menopausal women by maintaining high level of high-density lipoprotein cholesterol (HDL-C) and lowering the low-density lipoprotein cholesterol and triglycerides.<sup>7-10</sup> Lack of estrogen is an essential contributory factor in the derangement of lipid metabolism in post-menopausal women which is associated with increased cardiovascular risk.<sup>11</sup> Currently, post-menopausal women account for more than 30% of the female population at risk for CAD in India.<sup>12</sup>

## MATERIAL 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 from MGM medical college, Aurangabad. Exclusion criteria included obesity, pregnancy, diabetes mellitus, hypertension, hormonal contraception and heavy exercise. Fasting venous samples (5ml) were collected in plain bulb 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 4 degree celsius. Total cholesterol was measured using established enzymatic methods. 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<sup>13</sup>. Triglyceride was isolated enzymatically as described by Henry (1991). VLDL was calculated using the formula  $VLDL-C = TG/5$  (Henry, 1991). Statistical analysis was done using Excel, showing the mean and standard deviation. Comparison of mean was by student t-test.

## RESULTS

Table 1:

	Total Cholesterol	HDL	LDL	VLDL	TG
Premenopausal women(n=25)	202.60±50.50	56.84±22.14	126.09±44.80	25.32±14.90	127.90±70.96
Post Menopausal women(n=25)	246.70±70.14	52.16±16.88	158.52±54.72	34.92±14.25	158.90±80.54
P Values Significance	P<0.05	P > 0.05	P<0.05	P<0.05	P< 0.05

The mean, standard deviation and the P values for total cholesterol, HDL-C, LDL-C, VLDL-C and Triglycerides are shown in Table 1.

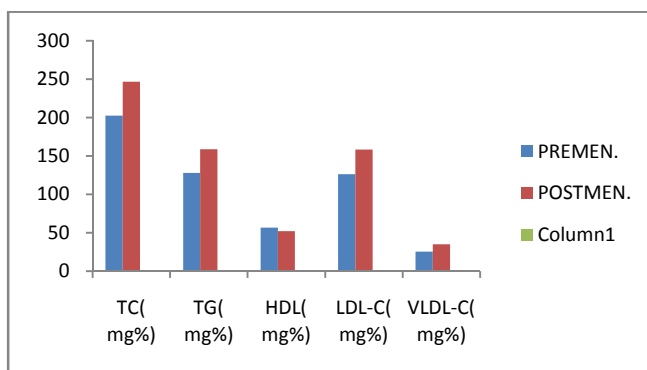


Figure 1: Showing Total Plasma Cholesterol and its Subfractions in Premenopausal and Postmenopausal Women

## DISCUSSION

The hormonal changes associated with menopause i.e low plasma levels of oestrogen and marked increase in LH and FSH levels exerts a significant effect on plasma lipids and lipo-proteins. Oestrogens have a protective effect against cardiovascular system as oestrogen lowers the LDL-cholesterol by acting on LDL-receptors. Apart from maintaining friendly lipid profile, oestrogens changes the vascular tone by increasing nitrous oxide production. It stabilises the endothelial cells, enhances antioxidant effect and alters fibrinolytic protein. All these are cardio-protective mechanisms, which are lost in menopause due to reduced oestrogen levels. This leads to hypercholesterolemia, which is a key factor in the pathophysiology of atherosclerosis. High levels of LDL and low levels of HDL are strongly associated with increased incidence of cardiovascular disease. Hence the present study was undertaken to study the cardiovascular

risk factors in Indian postmenopausal women. The present study was undertaken to evaluate the levels of serum cholesterol and its subfractions in pre- and postmenopausal women. The incidence of cardiovascular disease after menopause may be partly caused by changes in the plasma lipid levels that occur following the menopausal transition.<sup>17-19</sup> Deposition of fatty plaques on arterial walls (arteriosclerosis) is a predisposing factor for coronary heart disease.<sup>20</sup> The increased risk of CAD following menopause is mainly related to the endocrine influences on lipid profile especially when other risk factors such as blood pressure, blood sugar, and body weight are normal. Estrogens have a major beneficial effect on cholesterol metabolism and appear substantially to reduce the risk of atherosclerosis and cardiovascular disease in post-menopausal women.<sup>21</sup> The mean age in the post-menopausal women is  $59.2 \pm 10.2$  years and in the pre-menopausal women is  $34.9 \pm 6.71$  years. The

mean age in post-menopausal women is greater than that of pre-menopausal women. There is difference in age between both groups of women because it is difficult to design studies that can separate the effects of the normal aging process from natural menopause which occurs between 45 and 50 years.<sup>15</sup> The findings in our study are in accordance with other studies done by Kalavathi *et al.*, Muzzio *et al.*, and Matthews *et al.*<sup>15,17,23</sup>. In our study the TC is seen to be increased (246.70±70.14) in post-menopausal women due to estrogen deficiency when compared to pre-menopausal women (202.60±50.50) and is statistically significant. In our study, when compared to pre-menopausal women (127.90±70.96), post-menopausal women were having high TG (158.90±80.54) and were statistically significant ( $P < 0.05$ ). These findings are in accordance with other studies done by Welty and Hallberg and Svanborg.<sup>14,24</sup> In the post-menopausal women, there is increased fat accumulation and increased release of free fatty acids into the circulation, and excessive free fatty acids provide substrate for hepatic TG synthesis.<sup>25</sup> It was observed in our study that the menopausal status is unlikely to alter HDL-C level since no significant differences were found regarding its levels between the pre- and post-menopausal women. The values of HDL-C were found to be 56.84±22.14 and 52.16±16.88 respectively in pre and postmenopausal women. This finding was in accordance with certain previous studies done.<sup>26-29</sup> In our study, post-menopausal women had high levels of LDL when compared to pre-menopausal women. The values of LDL-C were found to be 126.09±44.80 and 158.52±54.72 respectively in pre and postmenopausal women and the difference was statistically significant ( $P < 0.05$ ). These findings are in accordance with other studies.<sup>15,30,31</sup> Lipoprotein lipase is regulated by circulating estrogen. It catalyzes the hydrolysis of VLDL to form intermediate-density lipoprotein and later LDL. Estrogen deficiency after menopause increases the plasma LPL and hepatic TG lipase activity causing plasma LDL to accumulate and also leads to down-regulation of LDL receptors.<sup>23,30,32</sup> In our study, the VLDL was increased in post-menopausal women when compared to pre-menopausal women. The values of VLDL-C were found to be 25.32±14.90 and 34.92±14.25 respectively in pre and postmenopausal women and the difference was statistically significant ( $P < 0.05$ ), and these findings are in accordance with studies done by Swapnali *et al.*, Welty and Matthews *et al.*<sup>30,14,17</sup> Estrogen deficiency in post-menopausal women causes relative enrichment of small VLDL particles with cholesteryl esters either due to the increased catabolism of VLDL with resulting increased number of VLDL remnant particles or increased activity of cholesterol ester transfer protein or both.<sup>16</sup> These small VLDL particles

are highly atherogenic as they contain more cholesteryl esters molecules per particle.<sup>33</sup> The VLDL remnants have a high capacity for interacting with arterial smooth muscle cells.<sup>34</sup>

## CONCLUSION

Menopause leads to changes in lipid profile by elevating TC, TGs, LDL-C, and VLDL-C, thus increasing the risk for cardiovascular disease. Due to the change in the lipid pattern and loss of cardioprotective effect of estrogen, post-menopausal women are at increased risk of developing cardiovascular disease. So it is important to educate each and every postmenopausal woman to undergo screening for abnormal lipid profile. Specific health education strategies are required to prevent the emerging cardiovascular diseases among postmenopausal women. It is important to counsel on proper dietary, social and physical habits. There are many studies showing the beneficial effects of hormone replacement therapy on the lipid profile in post-menopausal women.<sup>36,37</sup> Furthermore, there are several studies which disagree on the beneficial effects of hormone replacement therapy in patients with cardiovascular disease.<sup>38</sup> Predicting the factors affecting the lipid profile in post-menopausal women, adopting strategies to control these mechanisms by modifying the relative risk factors during menopausal transition may improve the cardiovascular risk profile in these women.

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