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Comparative Study of Lipid Profile and Lipoprotein (A) Levels Between Premenopausal and Postmenopausal Women

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ABSTRACT

Background: Hyperlipidemia is one of the most significant risk factors for cardiovascular disease in both men and women. The prevalence of cardiovascular disease is lower in premenopausal women compared to postmenopausal women. The cessation of menstruation is associated with lipid profile abnormalities that contribute to cardiovascular complications.

Methods: This case-control study included 68 women (34 premenopausal, 34 postmenopausal) attending the General Medicine Department of Victoria Hospital and affiliated hospitals of Bangalore Medical College and Research Institute from November 2018 to May 2020. Women with BMI >25, premature menopause (<45 years), on drugs affecting lipid metabolism, or with chronic diseases were excluded. After overnight fasting, blood samples were analyzed for TC, TG, HDL, LDL, VLDL, and Lp(a). Student's t-test and Chi-square tests were used.

Results: The mean age was 52.88±1.32 years in postmenopausal and 39.74±5.13 years in premenopausal women. Postmenopausal women had significantly higher TC (199.91±35.01 vs. 182.97±21.61; p=0.019), TG (178.94±31.49 vs. 106.79±28.09; p<0.001), LDL (159.82±23.64 vs. 112.41±23.07; p<0.001), VLDL (33.47±5.67 vs. 20.59±4.77; p<0.001), TC/HDL (4.79±1.06 vs. 3.24±0.49; p<0.001), LDL/HDL (3.94±0.85 vs. 2.03±0.17; p<0.001), and Lp(a) (35.88±8.72 vs. 9.26±3.94; p<0.001). HDL was significantly lower (42.18±7.03 vs. 54.85±5.29; p<0.001). Lp(a) showed a significant positive correlation with TC, TG, LDL, VLDL, and TC/HDL in postmenopausal women.

Conclusion: The significant increase in all lipid parameters (TC, TG, LDL, VLDL, and Lp(a)) and decrease in HDL levels observed in postmenopausal women are likely due to estrogen deficiency associated with menopause. These findings suggest that postmenopausal women have a significantly altered lipid profile that increases their risk for cardiovascular disease.

Key-words: Lipid profile, Lipoprotein(a), Premenopausal, Postmenopausal, Cardiovascular risk, Estrogen deficiency

INTRODUCTION

Menopause is the permanent cessation of menstruation resulting from loss of ovarian follicular function, diagnosed retrospectively after 12 months of amenorrhea. The average age of menopause is 51 years. After menopause, the ovaries cease to produce significant amounts of estrogen, leading to symptoms and diseases associated with estrogen deficiency that are increasingly important to women's health [1-3]. After menopause, changes in plasma lipid fractions occur following the menopausal transition, leading to an increased incidence of cardiovascular disease. Deposition of fatty plaques on arterial walls (atherosclerosis) is a predisposing factor for coronary artery disease. After attaining menopause, the prevalence of cardiovascular disease (CVD) is mainly due to the influence of endocrine hormones on lipid levels, especially when other risk factors such as blood pressure, blood sugar, and body weight are normal. Estrogens have major beneficial effects on cholesterol metabolism, and the risk of atherosclerosis and cardiovascular disease appears to

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decrease substantially in postmenopausal women with estrogen therapy [4-6].

Lipoprotein(a) [Lp(a)] has been recognized as an independent CVD risk factor. However, despite the independent association of Lp(a) concentrations with increased prevalence of CVD, few studies have assessed its role in menopause-related cardiovascular risk. Postmenopausal women are 4-8 times more likely to die of CVD than of any other disease. Hence, this study was undertaken to evaluate lipid changes in postmenopausal women compared to premenopausal women, to guide the prevention of cardiac complications [7-9]. This study aimed to evaluate and compare lipid profiles and serum lipoprotein(a) levels between pre- and postmenopausal women to assess cardiovascular risks.

MATERIALS AND METHODS

Study Design and Setting- This case-control study was conducted in the General Medicine Department of Victoria Hospital and affiliated hospitals of Bangalore Medical College and Research Institute from November 2018 to May 2020.

Sample Size and Participants- A total of 68 women were enrolled—34 premenopausal and 34 postmenopausal. Women aged ≥50 years with ≥1 year of amenorrhea were considered postmenopausal.

The sample size was calculated using below formula:

$$n = 2(Z\alpha + Z1\text{-}\beta)^2\ \sigma^2/d^2.$$

Inclusion and Exclusion Criteria

Excluded: BMI >25, premature menopause (<45 years), use of drugs affecting lipid metabolism, chronic liver disease, alcohol consumption, smoking, diabetes mellitus, and hypertension.

Data Collection- A detailed history and clinical examination were performed. After overnight fasting (12–24 h), blood samples were collected; in premenopausal women, samples were collected on the 7th day of menstruation.

Biochemical Analysis- Serum TC, TG, and HDL were measured using enzymatic methods. LDL and VLDL were calculated using Friedewald's formula. Lp(a) was also estimated. BMI was calculated from height and weight.

Statistical Analysis- Student's t-test was used to compare lipid parameters between groups. Chi-square test and Pearson's correlation were used for proportions and correlations. A p-value <0.05 was considered statistically significant.

Ethical Considerations- The study received approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants. A detailed history was recorded, and a complete clinical examination was performed.

RESULTS

The mean age of postmenopausal women was 52.88±1.32 years compared to 39.74±5.13 years for premenopausal women (p<0.001). The mean age of menarche was slightly higher for premenopausal women (13.59±1.57 vs. 13.29±1.31; p=0.40). Mean BMI was higher for premenopausal women (20.76±2.60 vs. 19.94±3.45; p=0.27), though the difference was not statistically significant. Out of 68 subjects overall, 45 (66.2%) had normal BMI, with 25 (73.5%) in the premenopausal group and 20 (58.8%) in the postmenopausal group. The percentage of underweight subjects was higher in the postmenopausal group (32.4% vs. 17.6%).

Table 1 shows that TC, TG, LDL, VLDL, TC/HDL, LDL/HDL, and Lp(a) were significantly higher in postmenopausal women compared to premenopausal women. HDL was significantly higher in premenopausal women. All differences were statistically significant (p<0.05).

Table 1: Comparison of Lipid Profile Between Premenopausal and Postmenopausal Women

Lipid Profiles	Premenopausal (n=34) Mean±SD	Postmenopausal (n=34) Mean±SD	Mean diff	p-value
TC (mg/dl)	182.97±21.61	199.91±35.01	-16.94	0.019*
TG (mg/dl)	106.79±28.09	178.94±31.49	-72.14	<0.001*
HDL (mg/dl)	54.85±5.29	42.18±7.03	12.67	<0.001*
LDL (mg/dl)	112.41±23.07	159.82±23.64	-47.41	<0.001*

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VLDL (mg/dl)	20.59±4.77	33.47±5.67	-12.88	<0.001*
TC/HDL	3.24±0.49	4.79±1.06	-1.55	<0.001*
LDL/HDL	2.03±0.17	3.94±0.85	-1.91	<0.001*
Lp(a) (mg/dl)	9.26±3.94	35.88±8.72	-26.61	<0.001*

**Statistically significant*

Table 2 shows that most premenopausal women had TC between 171 and 200 mg/dl (64.7%), TG <100 mg/dl (55.9%), HDL between 46 and 55 mg/dl (61.8%), and LDL between 101 and 130 mg/dl (73.5%). In contrast, most postmenopausal women had TC >200 mg/dl (38.2%), TG between 151 and 200 mg/dl (58.8%), HDL <45 mg/dl (70.6%), and LDL >130 mg/dl (94.1%). All these differences were statistically significant (p<0.05).

Table 2: Distribution of Total Cholesterol, Triglycerides, HDL, and LDL in Premenopausal and Postmenopausal Women

Parameters	Category	Premenopausal (n=34)	Postmenopausal (n=34)	Total	p-value
Total Cholesterol	<140 mg/dl	2 (5.9%)	0 (0.0%)	2 (2.9%)	0.016*
	141-170 mg/dl	6 (17.6%)	9 (26.5%)	15 (22.1%)	
	171-200 mg/dl	22 (64.7%)	12 (35.3%)	34 (50%)	
	>200 mg/dl	4 (11.8%)	13 (38.2%)	17 (25%)	
Triglycerides	<100 mg/dl	19 (55.9%)	0 (0%)	19 (27.9%)	<0.001*
	101-150 mg/dl	13 (38.2%)	7 (20.6%)	20 (29.4%)	
	151-200 mg/dl	2 (5.9%)	20 (58.8%)	22 (32.4%)	
	>200 mg/dl	0 (0%)	7 (20.6%)	7 (10.3%)	
	<45 mg/dl	0 (0%)	24 (70.6%)	24 (35.3%)	
HDL	46-55 mg/dl	21 (61.8%)	10 (29.4%)	31 (45.6%)	<0.001*
	>55 mg/dl	13 (38.2%)	0 (0%)	13 (19.1%)	
	<100 mg/dl	6 (17.6%)	0 (0%)	6 (8.8%)	
LDL	101-130 mg/dl	25 (73.5%)	2 (5.9%)	27 (39.7%)	<0.001*
	>130 mg/dl	3 (8.8%)	32 (94.1%)	35 (51.5%)	

**Statistically significant*

Table 3 shows that 97.1% of premenopausal women had normal VLDL levels, whereas 67.6% of postmenopausal women had elevated VLDL levels (>30 mg/dL). For the TC/HDL ratio, most premenopausal women (85.3%) were in the below-average risk category, while most postmenopausal women (67.6%) were in the average risk category, with 20.6% in the high-risk category. For the LDL/HDL ratio, 97.1% of premenopausal women were in the lower-risk category, while 50% of postmenopausal women were in the average-risk category. Regarding Lp(a), 52.9% of premenopausal women had levels <10 mg/dl, while 76.5% of postmenopausal women had Lp(a) levels >30 mg/dl. All these differences were statistically significant (p<0.001).

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Table 3: Distribution of VLDL, TC/HDL, LDL/HDL, and Lp(a) in Premenopausal and Postmenopausal Women

Parameters	Category	Premenopausal (n=34)	Postmenopausal (n=34)	Total	p-value
VLDL	Normal	33 (97.1%)	11 (32.4%)	44 (64.7%)	<0.001*
	Above 30 mg/dl	1 (2.9%)	23 (67.6%)	24 (35.3%)	
TC/HDL	Lower risk	1 (2.9%)	0 (0%)	1 (1.5%)	<0.001*
	Below average	29 (85.3%)	4 (11.8%)	33 (48.5%)	
	Average risk	4 (11.8%)	23 (67.6%)	27 (39.7%)	
	High risk	0 (0%)	7 (20.6%)	7 (10.3%)	
LDL/HDL	Lower risk	33 (97.1%)	1 (2.9%)	34 (50%)	<0.001*
	Below average	1 (2.9%)	14 (41.2%)	15 (22.1%)	
	Average risk	0 (0%)	17 (50%)	17 (25.0%)	
	High risk	0 (0%)	2 (5.9%)	2 (2.9%)	
Lp(a)	<10 mg/dl	18 (52.9%)	0 (0.0%)	18 (26.5%)	<0.001*
	11-30 mg/dl	16 (47.1%)	8 (23.5%)	24 (35.3%)	
	>30 mg/dl	0 (0%)	26 (76.5%)	26 (38.2%)	

**Statistically significant*

Table 4 shows that in premenopausal women, no significant correlation was observed between Lp(a) and other lipid parameters. However, in postmenopausal women, significant positive correlations were observed between Lp(a) and TC (r=0.67, p<0.001), TG (r=0.41, p=0.01), LDL (r=0.43, p=0.009), VLDL (r=0.38, p=0.02), and TC/HDL (r=0.58, p<0.001).

Table 4: Pearson's Correlation Between Lp(a) and Other Lipid Parameters

Groups	Parameter	TC	TG	HDL	LDL	VLDL	TC/HDL	LDL/HDL
Premenopausal	r value	-0.01	-0.09	-0.11	0.03	-0.13	-0.07	0.07
	p value	0.93	0.57	0.51	0.83	0.46	0.65	0.66
Postmenopausal	r value	0.67	0.41	-0.03	0.43	0.38	0.58	0.27
	p value	<0.001*	0.01*	0.84	0.009*	0.02*	<0.001*	0.11

**Statistically significant*

DISCUSSION

The present study demonstrated significant differences in lipid profiles between premenopausal and postmenopausal women. All parameters (TC, TG, LDL, VLDL, TC/HDL, LDL/HDL, and Lp(a)) except HDL were significantly higher in postmenopausal women. These findings are consistent with several previous studies, including Kilim *et al*. [10], Shenoy *et al*. [11], and Premkumar *et al.* [12], all of which reported similar alterations in lipid profiles following menopause. Estrogen has favorable effects on lipid metabolism through various mechanisms: it increases HDL by increasing the production of apolipoprotein A-I, decreases LDL by increasing the expression of LDL receptors in the liver, and reduces lipoprotein(a) levels. The loss of these protective effects contributes to the adverse lipid profile observed in postmenopausal women [13-15].

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Our study found significantly elevated Lp(a) levels in postmenopausal women compared to premenopausal women (35.88±8.72 vs. 9.26±3.94 mg/dl, p<0.001), which agrees with findings by Anagnostis *et al.* [16]. Lp(a) is structurally like LDL but contains an additional protein, apolipoprotein(a), which shares homology with plasminogen. This structural similarity interferes with fibrinolysis and promotes thrombosis, making Lp(a) an independent risk factor for cardiovascular disease. We also observed significant positive correlations between Lp(a) and other atherogenic lipid parameters (TC, TG, LDL, VLDL, and TC/HDL) in postmenopausal women, suggesting that Lp(a) may contribute to the increased cardiovascular risk in this population through multiple pathways [17-19].

The clinical implications of these findings are significant. Given the substantial alterations in lipid profiles and elevated Lp(a) levels in postmenopausal women, early screening and intervention may be beneficial in reducing cardiovascular risk. Lifestyle modifications, including regular physical activity, maintaining a healthy body weight, and following a heart-healthy diet, should be encouraged. In cases with significantly elevated lipid parameters, pharmacological interventions may be necessary. Additionally, as suggested by Fatma *et al.* [20], identifying predictors of lipid profiles in postmenopausal women and adopting strategies to modulate these mechanisms during the menopausal transition may improve cardiovascular risk profiles in these women. Future studies should focus on the efficacy of various interventions in mitigating the adverse lipid profile changes associated with menopause, particularly elevated Lp(a) levels [21-23].

CONCLUSIONS

Serum dyslipidemia is an important risk factor for cardiovascular disease, and lipoprotein(a) is emerging as a significant lipid risk factor. Our study demonstrated that postmenopausal women have significantly altered lipid profiles compared to premenopausal women, characterized by increased levels of TC, TG, LDL, VLDL, and Lp(a) and decreased HDL levels. Additionally, the TC/HDL and LDL/HDL ratios, which are predictors of cardiovascular risk, were significantly higher in postmenopausal women. We also found that Lp(a) levels were substantially elevated in postmenopausal women and showed significant positive correlations with other atherogenic lipid parameters. This suggests that Lp(a) may play an important role in the increased cardiovascular risk observed in postmenopausal women. These changes in lipid profiles and Lp(a) levels are likely attributable to estrogen deficiency associated with menopause. The findings highlight the importance of monitoring lipid profiles in women transitioning through menopause and implementing appropriate preventive strategies to reduce cardiovascular risk in this population.

CONTRIBUTION OF AUTHORS

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