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Assessment of cardiovascular risk determinants in postmenopausal women using raloxifene

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Abstract

Background: Cardiovascular diseases (CVDs) are the leading cause of death worldwide, particularly affecting men and postmenopausal women, where estrogen loss may elevate risk. Hormone replacement therapy (HRT) initially emerged as a potential intervention for CVD risk reduction in the 1970s; however, recent findings advise against HRT for CVD prevention, favouring its limited use for menopausal symptoms. Selective estrogen receptor modulators (SERMs), such as raloxifene, offer a targeted approach by improving lipid profiles without stimulating breast or uterine tissues.

Aim of the study: The study aims to identify and analyze the key cardiovascular risk determinants in postmenopausal women using raloxifene.

Methods: This prospective, case-control study at Bangladesh Medical University in Dhaka included 160 postmenopausal women divided into an osteoporosis group (80 women on daily raloxifene 60 mg) and a control group (80 healthy women advised on diet and exercise). Inclusion required over two years of amenorrhea, while exclusion criteria ruled out systemic diseases, cardiovascular conditions, and recent hormone treatments. Baseline assessments included gynaecological exams, DEXA, mammography, and biochemical markers for cardiovascular risk, measured at baseline and after 12 months. Data were analyzed using SPSS, with a p-value ≤ 0.05 indicating statistical significance.

Result: This study focused on postmenopausal women, with a mean age of 58.84 years in cases and 59.19 years in controls. Both groups had similar menopause durations, mostly natural, and BMI and blood pressure were closely matched. After one year of treatment, the case group showed significant improvements in total cholesterol (222.73 to 198.82 mg/dl, P<0.001), LDL (136.96 to 120.55 mg/dl, P=0.002), Apo A, fibrinogen, and homocysteine levels. Triglycerides and HDL remained stable. The control group had no significant lipid changes, but carotid intima-media thickness increased (P=0.043), indicating vascular changes were absent in the case group.

Conclusion: This study found that raloxifene significantly reduced cardiovascular risk factors in postmenopausal women, including total cholesterol, LDL, homocysteine, and fibrinogen. Unlike controls, the raloxifene group did not show progression in carotid intima-media thickness, indicating potential arterial health protection. These results support raloxifene's role in cardiovascular risk reduction, warranting further study.

Keywords: Cardiovascular risk, postmenopausal women and raloxifene

Introduction

Cardiovascular diseases (CVDs) remain the leading cause of mortality globally, with cancer being the second [1]. According to data from the World Health Organization (WHO) in 2019, approximately 17.9 million people die from CVDs annually, accounting for 31% of global deaths [1]. CVD is a major contributor to morbidity and mortality among both men and women in developed nations. Research indicates that lower levels of estrogen are associated with a higher incidence of coronary artery disease in men [2]. Interestingly, CVD is more prevalent in men than women prior to menopause, but the risk for women rises significantly post-menopause [2]. The heightened risk of CVD in women post-menopause suggests that female hormones, particularly estrogens, have a cardioprotective effect and are crucial to both reproductive and non-reproductive health systems [3]. Hormone replacement therapy (HRT) emerged in the 1970s as a potential solution for managing menopausal symptoms and reducing cardiovascular risk, based on observational studies. However, large-scale randomized controlled trials in the 2000s stirred controversy, leading to the current consensus that HRT should not be used for

primary or secondary prevention of CVD. Instead, its primary use is for managing vasomotor and urogenital atrophic symptoms, with recommendations to limit its use to the lowest effective dose and shortest duration possible [4,5]. In response to these findings, the medical community began exploring alternative treatments. The ideal approach would retain the beneficial effects of estrogen on bones, arteries, and the brain while minimizing adverse effects on the breast and endometrium. This search led to the development of selective estrogen receptor modulators (SERMs) in the late 1990s [6, 7]. These synthetic, non-steroidal compounds selectively bind to estrogen receptors, acting as either agonists or antagonists, depending on the target tissue [8]. Raloxifene, a selective estrogen receptor modulator (SERM), has been primarily used for the prevention and treatment of osteoporosis in postmenopausal women [9]. However, its role in cardiovascular health is becoming increasingly prominent. By selectively acting on estrogen receptors in various tissues, raloxifene mimics some of the beneficial effects of estrogen without stimulating breast or uterine tissues, making it a potential candidate for cardiovascular risk reduction [10, 11]. Studies have demonstrated that raloxifene can positively influence lipid profiles, decreasing total cholesterol and low-density lipoprotein (LDL) levels, which are crucial determinants of CVD risk [12]. Despite the promising effects of raloxifene on cardiovascular risk factors, its impact on the overall cardiovascular health of postmenopausal women requires further investigation. Previous studies have reported conflicting results regarding the efficacy of raloxifene in reducing cardiovascular events [13]. Due to the variability in findings, the potential role of raloxifene in preventing cardiovascular disease among postmenopausal women remains unclear and calls for further in-depth research. Thus, the current study aims to identify and analyze the key cardiovascular risk determinants in postmenopausal women using raloxifene.

Methodology and Materials

This cross-sectional, prospective case-control study was conducted from January 2024 to December 2024 at the Department of Obstetrics and Gynecology, with the support of the Department of Biochemistry and the Department of Radiology at Bangladesh Medical University (BMU), Dhaka, Bangladesh. A purposive sampling approach was employed to recruit 160 postmenopausal women for this study. The study case group consisted of 80 women diagnosed with postmenopausal osteoporosis who started raloxifene hydrochloride (60 mg daily). Simultaneously, 80 healthy postmenopausal women attending the same hospital were selected as the control group. The control group was advised to follow an exercise regimen and maintain a calcium-rich diet.

Inclusion Criteria

- Participants included in the study were postmenopausal women who had experienced amenorrhea for more than two years.

Exclusion Criteria

- Women with systemic diseases that could affect cardiovascular risk such as type 2 diabetes, hypertension, familial dyslipidemia, and obesity were excluded.
- Additionally, smokers, women with known cardiovascular disease, those who had used hormone replacement therapy (HRT), tibolone, antiresorptive agents, statins, or antihypertensive medications in the past year, and those with a history or suspicion of gynecological malignancy, endometrial thickness >4 mm, thromboembolic disease, cerebrovascular disease, or vasomotor symptoms were also excluded.

Before starting treatment, all participants underwent a comprehensive baseline assessment. This included gynecological examination, PAP smear, transvaginal ultrasound (USG), breast examination, mammography, and bone mineral density (BMD) measurement using dual-energy X-ray absorptiometry (DEXA). Additional evaluations included blood pressure measurement, body mass index (BMI) calculation, and laboratory tests assessing liver function, kidney function, thyroid function, and a complete blood count.

Cardiovascular risk factors were assessed through biochemical and radiological evaluations at baseline and after 12 months of raloxifene treatment or lifestyle intervention. Biochemical markers such as total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), lipoprotein (a) [Lp (a)], apolipoproteins A and B (Apo A, Apo B), homocysteine, high-sensitivity C-reactive protein (hs-CRP), and fibrinogen were analyzed. Carotid intima-media thickness (IMT) was measured before and after the study period.

Ethical Considerations

Ethical approval was obtained from the institutional ethics committee. Informed written consent was secured from all participants.

Statistical Analysis

Statistical analysis was conducted using SPSS software (version 26). Continuous variables were presented as mean ± standard deviation (SD) and compared using the unpaired t-test, while categorical variables were expressed as frequencies and percentages and analyzed using the chi-square test. A p-value ≤ 0.05 was considered statistically significant.

Result

The demographic and clinical profiles of the study participants in both the case (N=80) and control (N=80) groups were largely similar, with no statistically significant differences across key variables. Age distribution was well-balanced, with the majority of participants falling within the 51-60 age range. The mean age for the case group was 58.84±8.21 years, while the control group had a similar mean of 59.19±6.53 years. Postmenopausal duration also showed a similar distribution between the groups, with both groups having a mean duration of around 12 years, and the majority of women being postmenopausal for over 11-15 years. Most participants experienced natural menopause (85% in cases and 82.5% in controls), while a smaller proportion had surgical menopause. Body mass index (BMI) and blood pressure levels were also closely matched between the two groups, with a mean BMI of 22.35±1.87 kg/m² in the case group and 21.92±1.73 kg/m² in the control group. Systolic and diastolic blood pressures were similar between the groups, with no significant differences (Table 1). Table 2 focuses on the variations in cholesterol levels and other biochemical markers within the case group, measured at baseline and one year after treatment. Significant improvements were observed in several key parameters post-treatment. Total cholesterol levels dropped from a mean of 222.73±33.42 mg/dl at baseline to 198.82±30.47 mg/dl after one year ($P<0.001$), while LDL cholesterol levels also decreased significantly from 136.96±28.67 mg/dl to 120.55±27.61 mg/dl ($P=0.002$). There were notable reductions in Apo A levels ($P=0.009$) and fibrinogen ($P=0.044$), indicating

improved lipid metabolism and reduced inflammatory responses. Homocysteine levels showed a significant decrease (P=0.048), and hs-CRP levels, although reduced slightly, did not reach statistical significance (P=0.484). Triglyceride levels and HDL cholesterol remained relatively stable, and lipoprotein (a) levels showed no significant changes. Table 3 presents the biochemical data for the control group. Unlike the case group, the control group showed no significant changes in total cholesterol, LDL cholesterol, or other lipid markers over one year. Total cholesterol levels remained nearly unchanged, from 218.22±35.47 mg/dl to 218.65±39.76 mg/dl (P=0.795), and LDL cholesterol levels exhibited a non-significant reduction from 134.76±32.62 mg/dl to 129.85±35.63 mg/dl (P=0.422). Other lipid-related markers, such as Apo A, Apo B, and triglycerides, also showed no significant changes. One notable finding in the control group was a slight but significant increase in carotid intima-media thickness (IMT), from 0.71±0.12 cm to 0.75±0.14 cm (P=0.043).

Table 1: Demographic and clinical profiles of study participants.

Variables	Case (N=80)		Control (N=80)		P value
	N	%	N	%	
Age (year)					
≤45	9	11.25	10	12.50	0.391
46-50	16	20.00	17	21.25	
51-55	19	23.75	20	25.00	
56-60	20	25.00	16	20.00	
>60	16	20.00	17	21.25	
Mean ± SD	58.84±8.21		59.19±6.53		
Postmenopausal period (year)					
≤5	17	21.25	17	21.25	0.869
6-10	16	20.00	14	17.50	
11-15	29	36.25	31	38.75	
>15	18	22.50	20	25.00	
Mean ± SD	12.41±5.65		12.68±5.87		
Menopause type					
Natural	68	85.00	66	82.50	0.996
Surgical	12	15.00	14	17.50	
Mean ± SD					
BMI (kg/m^2)	22.35±1.87		21.92±1.73		0.416
Blood pressure (mmHg)					
Systolic	126±17		125±19		0.482
Diastolic	79±9		82±8		0.658

Table 2: Variations in examined cholesterol levels across case groups.

Variables	Baseline	1 year after treatment	P value
	Mean ± SD		
Total cholesterol (mg/dl)	222.73±33.42	198.82±30.47	<0.001
LDL cholesterol (mg/dl)	136.96±28.67	120.55±27.61	0.002
HDL cholesterol (mg/dl)	64.14±11.89	61.59±13.22	0.582
Triglyceride (mg/dl)	116.54±62.39	118.31±49.65	0.895
Lipoprotein (a) (mg/dl)	25.42±24.66	27.18±29.37	0.589
Apo A (mg/dl)	125.35±23.55	141.41±46.68	0.009
Apo B (mg/dl)	109.25±23.37	104.86±27.11	0.492
Homocysteine (µmol/l)	12.04±2.69	10.74±2.98	0.048
hs-CRP (mg/dl)	0.38±0.43	0.33±0.34	0.484
Fibrinogen (mg/dl)	203.17±109.52	160.97±91.28	0.044
Carotid IMT (cm)	0.81±0.38	0.87±0.44	0.172

Table 3: Variations in examined cholesterol levels across control groups.

Variables	Baseline	1 year after treatment	P value
	Mean ± SD		
Total cholesterol (mg/dl)	218.22±35.47	218.65±39.76	0.795
LDL cholesterol (mg/dl)	134.76±32.62	129.85±35.63	0.422
HDL cholesterol (mg/dl)	62.32±13.75	58.85±15.87	0.165
Triglyceride (mg/dl)	122.79±62.71	133.10±66.56	0.142
Lipoprotein (a) (mg/dl)	34.47±25.19	29.13±31.04	0.412
Apo A (mg/dl)	136.25±25.21	140.87±45.42	0.287
Apo B (mg/dl)	117.27±23.08	108.15±29.24	0.168
Homocysteine (µmol/l)	12.05±2.46	11.28±3.66	0.471
hs-CRP (mg/dl)	0.42±0.44	0.26±0.23	0.124
Fibrinogen (mg/dl)	258.32±191.51	208.58±129.76	0.152
Carotid IMT (cm)	0.71±0.12	0.75±0.14	0.043

Discussion

This study demonstrated that a daily dose of 60 mg raloxifene positively impacted the serum lipid profile and other critical intermediate markers of cardiovascular disease (CVD), consistent with prior research [13-15]. Clinical trials have shown that raloxifene decreases total cholesterol (TC) and low-density lipoprotein (LDL) by 8-12%, with these reductions starting in the third month and continuing for up to four years [13, 15, 16]. In our study, raloxifene significantly reduced TC and LDL levels after one year, while no changes were observed in the control group with comparable baseline levels. This effect is likely related to raloxifene's ability to inhibit LDL oxidation, as demonstrated in vitro [17]. The influence of raloxifene on high-density lipoprotein (HDL) levels has produced mixed results in the literature. For instance, the Euralox trial reported a significant rise in HDL, whereas both the MORE study and research by Walsh et al. (2000) and Delmas et al. (1997) found no significant changes [15, 16]. Similarly, our study did not observe a significant alteration in HDL levels. Regarding triglycerides (TG), the literature remains inconclusive. While Nickelsen et al. reported a decrease in TG levels, Walsh et al. (2000) found no significant effect [15]. In our findings, a slight but statistically insignificant increase in TG levels was observed, consistent with the MORE study [13]. Although raloxifene has been shown to lower lipoprotein (a) [Lp(a)], with Mijatovic et al. (1999) reporting up to a 30% reduction using a 150 mg dose, our study did not observe a significant change in Lp(a). This discrepancy may be due to the lower 60 mg dose used in our study compared to other research [18, 19]. The role of serum concentrations of Apo A and Apo B as predictors of CVD is well-established [20]. Walsh et al. (2000) reported an increase in Apo A and a decrease in Apo B among women using raloxifene [15]. Our study similarly found a significant rise in Apo A levels, while the decrease in Apo B was not statistically significant. Additionally, we observed a notable reduction in fibrinogen levels, a hemostatic factor associated with atherogenesis, which aligns with other studies [19]. While hormone replacement therapy (HRT) has been shown to increase high-sensitivity C-reactive protein (hs-CRP), as reported by Cushman et al. (1999) [21], we did not detect any significant change in hs-CRP levels, consistent with findings from Walsh et al. (2000) [15]. Carotid intima-media thickness (IMT) is a well-established marker of carotid wall structure and atherosclerosis [22]. Numerous studies have confirmed the association between CVD and carotid IMT [23]. Research on HRT and carotid IMT has produced varied outcomes [24, 25]. In our study, the control group showed an increase in carotid IMT after one year, likely due to aging, whereas the raloxifene group did not experience such an increase, suggesting that raloxifene may prevent carotid IMT progression. Excluding confounding factors such as hypertension, obesity, diabetes, and smoking strengthens the evidence for raloxifene's specific effect on carotid IMT. Overall, this study reinforces the view that raloxifene influences several risk factors for atherosclerosis. By evaluating multiple determinants simultaneously in both the treatment and control groups and carefully excluding other risk factors through precise matching, our study enhanced the reliability of these findings. The absence of baseline differences between the groups further supports the robustness of the matching process. As a result, raloxifene appears to exert various anti-atherogenic effects [26-28], reducing TC, LDL, and homocysteine levels similarly to HRT, but without the associated increases in TG and hs-CRP [14, 27]. Additionally, unlike HRT, raloxifene does not adversely affect HDL levels. Multiple clinical studies have highlighted raloxifene's favorable impact on atherosclerotic disease risk factors.

Limitations of the study: A key limitation of this study is its relatively small sample size, which may reduce the generalizability and statistical power of the findings. Additionally, while the strict exclusion criteria effectively controlled for confounding factors, they may have limited the diversity of the study population, potentially restricting the applicability of the results to a broader spectrum of postmenopausal women. The study's relatively short follow-up period does not capture the long-term effects of raloxifene on cardiovascular health, warranting further longitudinal studies with larger cohorts and extended observation.

Conclusion and Recommendations

This study demonstrates that raloxifene significantly improves cardiovascular risk determinants in postmenopausal women by reducing total cholesterol, LDL levels, and homocysteine, while also lowering fibrinogen, a marker of inflammation. Unlike the control group, which showed no significant changes and an increase in carotid intima-media thickness, the raloxifene group did not experience such progression, suggesting a protective effect on arterial health. These findings reinforce raloxifene's potential role in mitigating cardiovascular disease risk factors, though further investigation is needed to fully understand its long-term impact on cardiovascular outcomes.

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