

Assessment of Estradiol and C-Reactive Protein in Menopause Women

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ABSTRACT

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Background: A woman enters menopause when her menstrual cycles permanently cease. Estradiol has a significant impact on inflammatory and immunological functions. **Objective:** The purpose of this study was to compare menopausal women's levels of estradiol and inflammatory markers (C-reactive protein) to those of healthy women as a control group. **Methodology:** Samples were taken from female volunteers throughout a three-month period, and forty-five (45) menopausal women were compared with (45) healthy women as controls, whose ages ranged from 45 to 55. **Results:** There was a significant difference in BMI across the groups in this study ($p = 0.039$). Patient total cholesterol, triglyceride SBP, and DBP were significantly higher than those of controls ($p < 0.001$), while HDL was significantly lower than that of controls ($p = 0.011$). Significant differences were observed in hormonal and inflammatory markers: estradiol ($p \leq 0.001$) and CRP ($p \leq 0.001$). **Conclusion:** A prominent indicator of early cardiovascular disease in menopausal women is dyslipidemia, elevated inflammatory markers (C-reactive protein), and decreased estrogen levels.

Keywords: C-reactive protein, Estradiol, lipid profile.
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INTRODUCTION

The time of a woman's life when her menstrual cycles permanently cease is known as menopause (1). Menopause is a natural aspect of aging for women; it is not a sickness nor a pathology. It is brought on by a drop in the ovaries' production of estrogen, which causes symptoms like mood swings, vaginal dryness, hot flashes, and irregular periods during the menopausal transition (2). For many women, weight gain is linked to the menopausal transition. In both normal-weight and obese people, body weight also rises with age (3). In certain studies, these weight and lipid alterations during the transition were unrelated to age, but in others, they were not. It has been investigated that this weight increase during the menopausal transition may be a significant contributor to midlife body weight (4). One type of estrogen, the most well-known and potent steroid hormone, is estradiol. Because it controls the development and maintenance of female sex traits, estradiol is frequently referred to as the "female hormone". One of the three structurally similar estrogens is estradiol (5). Both men and women contain estrogens, but women of reproductive age often have much larger quantities of these chemicals, which aid in the development of female secondary sexual features, such as breasts, and also contribute to the endometrium's thickening (6). Low levels of estradiol affect older women's quality of life and are risk factor for disease. After menopause, increased bone re-absorption may result from an estrogen shortage (7). The liver produces a chemical called C-reactive protein (CRP) in reaction to inflammation. It is a crucial sign of the

body's inflammation (8). CRP is a vascular inflammatory marker for endothelial dysfunction linked to coronary heart disease and diabetes mellitus risk (9). The easiest way to identify people with systemic inflammation is to use CRP, a sensitive biomarker and acute-phase protein (10). Reduced estrogen levels, which are associated with anti-inflammatory qualities, can raise C-reactive protein (CRP) levels, resulting in low-grade systemic inflammation (11). The purpose of this study was to compare menopausal women's levels of estradiol and inflammatory markers (C-reactive protein) to those of healthy women as a control group.

METHODOLOGY

Forty-five (45) menopausal women and forty-five (45) healthy women were compared between the groups. The control group's age range was 45–55 years. Over the course of three months, samples were taken from female subjects. Weight, height, age, body mass index (BMI), systolic and diastolic blood pressure (SBP and DBP) were the most common demographic information. The Cobas c111 in Germany automatically measured clinical data and other laboratory measurements, including lipid profiles (total cholesterol, triglycerides, high density lipoprotein, very low-density lipoprotein, and low-density lipoprotein), while the Cobas e411 measured estradiol and the Nycocard device measured CRP.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics Version 28.0. Continuous variables were expressed as means $\pm$ standard error (SE). The independent-samples t-test was used to compare the two groups. Discrimination of the two groups by CRP and estradiol levels was analyzed by receiver operating characteristic (ROC) curve. Statistical significance was defined as $p < 0.05$.

RESULTS

As shown in Table 1, age, weight, and height were comparable between the menopausal and control groups (all $p < 0.05$), indicating that these characteristics were unlikely to explain the observed biochemical differences. BMI was higher in the menopausal group (26.07 ± 0.52) than in controls (24.43 ± 0.58), and the difference reached statistical significance ($p = 0.039$). This represents an approximately 6.7% higher mean BMI in the menopausal group.

Table (1): Distribution of patients and control according to age, weight, height and BMI.

Parameter	Patients (N=45), mean $\pm$ SE	Control (N=45), mean $\pm$ SE	<i>p</i> -value
Age (Years)	47.978 $\pm$ 0.478	48.911 $\pm$ 0.464	0.165
Weight (Kg)	72.233 $\pm$ 1.414	73.556 $\pm$ 1.276	0.489
Height (cm)	166.778 $\pm$ 1.018	164.178 $\pm$ 1.06	0.080
BMI	26.07 $\pm$ 0.52	24.43 $\pm$ 0.58	0.039

Data are presented as mean $\pm$ SE. Group comparisons were performed using the t-test. Statistical significance was defined as $p < 0.05$.

Table (2) demonstrates a consistent adverse cardiometabolic pattern in the menopausal group. Total cholesterol, triglycerides, and LDL-C were higher by approximately 13.1%, 34.8%, and 35.6%, respectively, whereas HDL-C was approximately 22.0% lower than in controls (TC, TG, and LDL-C: $p < 0.001$; HDL-C: $p = 0.011$). CRP was approximately 2.8-fold higher (8.93 ± 0.23 vs 3.18 ± 0.16 mg/L; $p < 0.001$), indicating substantially greater systemic inflammatory activity. The simultaneous increase in BMI and CRP is important because adipose tissue can release pro-inflammatory mediators. Estradiol was approximately 41.3% lower (35.36 ± 0.64 vs 60.24 ± 0.80 pg/mL; $p < 0.001$). Systolic and diastolic blood pressures were also higher in the menopausal group (15.30 ± 0.28 vs 12.42 ± 0.07 and 8.76 ± 0.10 vs 7.83 ± 0.06 ,

respectively; both $p < 0.001$, in the units recorded). Collectively, these differences suggest concurrent dyslipidemia, low-grade systemic inflammation, reduced estradiol, and higher blood pressure after menopause.

Menopausal women had less favorable lipid profiles with higher levels of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) and lower levels of high-density lipoprotein cholesterol (HDL-C) (all $p < 0.001$). The greatest relative changes from the menopausal women's baseline values occurred with TG and LDL-C. Decreased levels of estrogen can influence how the liver metabolizes fats, alters LDL receptors, and change fat distribution.

Table (2): Biochemical parameters in the studied groups

Parameter	Patients (N=45), mean ± SE	Control (N=45), mean ± SE	<i>p</i> -value
TC (mg/dL)	204.000 ± 5.5842	180.356 ± 1.1721	<0.001
TG (mg/dL)	149.889 ± 7.2033	111.178 ± 2.5608	<0.001
HDL (mg/dL)	42.316 ± 4.5624	54.267 ± 0.5341	0.011
LDL (mg/dL)	116.693 ± 4.8605	86.036 ± 1.3887	<0.001
CRP (mg/L)	8.933 ± 0.2344	3.178 ± 0.1598	<0.001
Estradiol (pg/ml)	35.356 ± 0.6358	60.244 ± 0.7972	<0.001
SBP mmHg	15.300 ± 0.2844	12.422 ± 0.0745	<0.001
DBP mmHg	8.756 ± 0.0962	7.833 ± 0.0550	<0.001

Data are presented as mean ± SE. Group comparisons were performed using the t-test. Statistical significance was defined as $p < 0.05$.

ROC analysis, as shown in Figures (1, 2, and 3), showed excellent separation between the two groups. CRP provided complete discrimination at a cut-off > 6.50 mg/L, with 100% sensitivity, 100% specificity, and an AUC of 1.000 (95% CI: 1.000–1.000; $p < 0.001$). Lower estradiol also showed excellent discrimination at a cut-off ≤ 48.50 pg/mL, with 97.8% sensitivity, 100% specificity, and an AUC of 0.998 (95% CI: 0.993–1.000; $p < 0.001$) (Table 3). Although these estimates indicate very high diagnostic performance in this sample, external validation is required because the cut-offs and accuracy was derived from the same relatively small cohort.

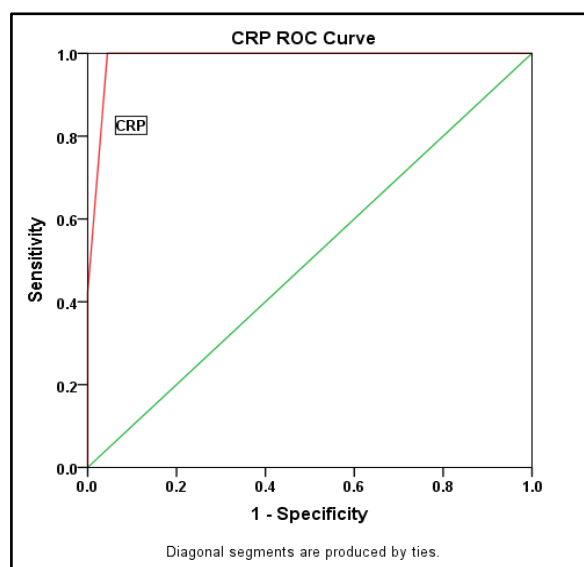


Figure (1): Receiver operating characteristic (ROC) curve of CRP in the studied groups.

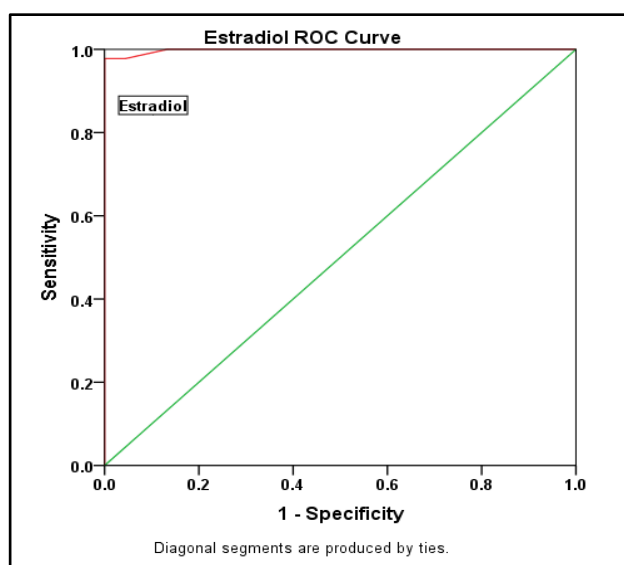


Figure (2): Receiver operating characteristic (ROC) curve of estradiol in the studied groups.

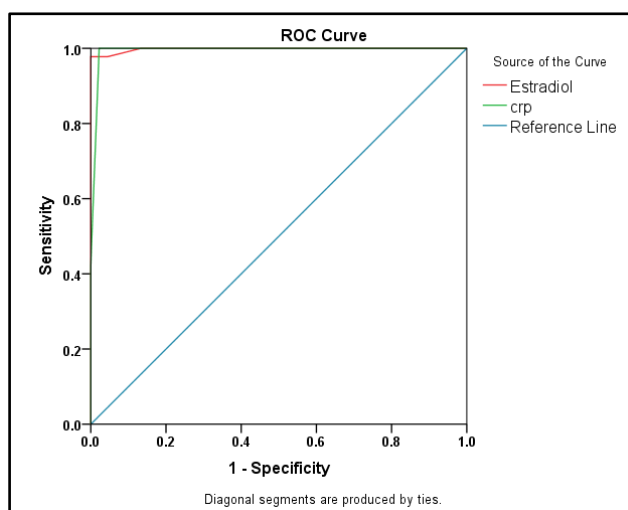


Figure (3): Receiver operating characteristic (ROC) of CRP and estradiol in the studied groups.

Table (3): Receiver operating characteristic (ROC) analysis of the studied markers

Marker	Sensitivity %	Specificity %	95% CI (AUC)	Area $\pm$ SE	Cut-off	<i>p</i> -value
CRP	100.0	100.0	1.000–1.000	1.000 $\pm$ 0.000	> 6.50 mg/L	<0.001
Estradiol	97.8	100.0	0.993–1.000	0.998 $\pm$ 0.002	< 48.50 pg/mL	<0.001

ROC *p*-values test whether the AUC differs from 0.50. Statistical significance was defined as $p < 0.05$. The *t*-test is not applicable to ROC performance indices.

DISCUSSION

The immunological and inflammatory systems are significantly impacted by estrogen. Increased inflammatory responses to infection and a higher incidence of autoimmune disorders in postmenopausal women are indicators of the function of estrogens (12). Results indicated that there was a significant rise in BMI between groups, which was aligned with a previous study (13). Women frequently see an increase in their body mass index (BMI) during and after menopause, especially in their truncal (abdominal) fat, which is linked to elevated systemic inflammation. Reduced estrogen levels, increased visceral adipose tissue, and the release of pro-inflammatory cytokines are all associated with inflammation. Increased inflammation raises the risk of cardiovascular disease, metabolic problems, and other illnesses (13). Particularly in postmenopausal women, excess adipose tissue causes increased aromatase activity, which raises systemic estrogen levels. Endometrial hyperplasia and carcinogenesis can result from the proliferative effects of estrogen on the endometrial lining when progesterone is not present to counteract it (14).

These effects can occur outside of the regular menstrual cycle. Declining estrogen levels can raise total cholesterol, LDL cholesterol, triglycerides, and apolipoproteins while reducing HDL cholesterol and its health benefits, which is why lipid problems are common in menopausal women. Because of these changes, it is essential to manage cardiovascular disease through lifestyle changes such as regular exercise and quitting smoking (15).

According to Table (2) patients had significantly higher levels of total cholesterol, triglycerides, and low-density lipoprotein than controls. In contrast, there was a significant drop in HDL cholesterol in patients compared with controls, which is consistent with findings from other studies (16,17). Regardless of the impact of age, it is generally acknowledged that postmenopausal women seem to be more vulnerable to atherosclerotic cardiovascular events, which may be explained by an altered lipid profile. Angina, myocardial infarction, and cerebral infarction are all thought to be caused by atherosclerosis, which appears to be directly linked to the decline of lipid metabolism, particularly hypercholesterolemia. LDL-C values were considerably greater during menopause than before, which was consistent with the study by MacLennan (18). A healthy lipid profile is maintained in part by estrogen. Changes in lipid metabolism may result from its reduction during menopause. Monitoring and controlling lipid levels at this period of life are essential since these changes may raise the risk of cardiovascular disease (19).

Higher HDL concentrations are associated with a reduced incidence of heart disorders, while lower HDL is associated with a high incidence of coronary heart diseases, according to numerous results by various researchers (20). Postmenopausal women have higher blood fatty acid release and fat storage, and these excess free fatty acids provide a stronger substrate for the liver's triglyceride synthesis (21). Following menopause, women who are deficient in estrogen effectively accumulate density lipoprotein components containing cholesterol esters. These remnants interact with smooth arterial muscle cells quite effectively. These lipoproteins are known to be a risk factor for cardiovascular disorders on their own. Women's cardiovascular risk will be reduced after menopause if these risk factors that affect their lipid panel are addressed (22). Menopausal women's elevated blood C-reactive protein (CRP) levels could be a sign of systemic inflammation linked to a drop in estradiol levels. Menopausal women's elevated hypertension in menopausal women is brought on by a combination of lifestyle factors that frequently accompany aging and menopause, such as weight gain, decreased physical activity, and increased stress, as well as hormonal changes, especially decreased estrogen levels, which stiffen and increase blood flow resistance in arteries (23, 24).

Dysregulated microRNAs and signaling contribute to altered inflammatory biological regulation associated with increased systemic inflammation, reflected by elevated CRP levels (25). The risk of cardiovascular disease during and after menopause is increased by these physiological and lifestyle changes, which also raise blood pressure readings. Regular blood pressure checks and lifestyle modifications, like eating a balanced diet, exercising frequently, and managing stress, are essential for controlling this elevated risk. Estradiol and CRP have a complex interaction as well. Reduced estradiol levels in menopause can damage tissue health, which can promote inflammation in the vaginal organs and urinary system. Nevertheless, systemic inflammation, which is not always directly related to estradiol levels, may be indicated by high CRP levels in the blood. Monitoring CRP and estradiol levels can be helpful when evaluating menopausal health, but a complete diagnosis and treatment of menopausal symptoms require careful assessment and further testing by medical professionals (26). Numerous bodily systems may be impacted by this inflammation. Lower levels of libido, arousal, and sexual enjoyment are observed in premenopausal women with elevated levels of CRP and IL-6 (27). This is because inflammation can interfere with arousal and sexual desire, causing sexual reluctance as a defense mechanism to lessen the risk of infection.

Persistent inflammation can cause endothelial dysfunction and impair nitric oxide's vasodilator action, which is necessary for eliciting sexual stimulation (28). The study data corroborates the notion that a number of concurrent events contribute to the postmenopausal increase in CRP and decrease in estradiol. First, both aging and conventional risk factors like pulse pressure and dyslipidemia were significant predictors of increases in CRP. Second, there was a strong positive correlation between age-adjusted

CRP levels and sex hormones such E1, E2, FEI, T, and FAI. Although this correlation was somewhat attenuated by additional adjustment for conventional risk factors, the relationship was still highly statistically significant. Thirdly, the relationships were once more slightly diminished but still significant when the degree of modification was extended to body build indicators (29). According to Table 3 and Figure 3, aging, changes in the sex hormone profile, and changes in body composition all contribute to the increased CRP sensitivity following menopause. Nonetheless, a sizable amount of the CRP variance cannot be accounted for (30). Future research must determine whether this unexplained portion of the CRP variance represents the chronic low-grade vascular inflammation thought to hasten the progression of atherosclerosis or whether "total CRP", regardless of its source, is the best alternative for evaluating the inflammatory potential (31). The finding that elevated CRP was associated with higher estrogenic plasma concentrations is an interesting one. Therefore, our data provided fresh insight into the menopause hypothesis and methods to "correct" for ovarian function loss with HRT, given the null results of recent major randomized trials on the impact of HRT (Hormone Replacement Therapy) on primary and secondary prevention of cardiovascular disease. By lowering the amount of circulating estrogens and androgens, the drop in estrogenic and androgenic sex steroid levels after menopause may represent a natural defense mechanism that lowers the risk of inflammation (32).

CONCLUSION

Menopausal women in this study had higher BMI, an adverse lipid profile, higher CRP and blood pressure, and lower estradiol than controls. These concurrent abnormalities indicate a less favorable cardiovascular-risk profile, but longitudinal and adjusted analyses are needed to establish independent associations and clinical predictive value.

ETHICAL APPROVAL

The research was approved by the Research Ethics Committee of College of Dentistry, Al-Mustansiriyah University (No. 853, dated 03/12/2025).

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تقييم الإستراديول والبروتين التفاعلي-سي لدى النساء في سن اليأس

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الخلاصة

للإسترايول تأثير كبير على الوظائف الا.تدخل المرأة مرحلة انقطاع الطمث عندما تتوقف دورتها الشهرية نهائيًا : خلفية البحث
 بروتين سي (تهدف هذه الدراسة إلى مقارنة مستويات الإسترايول وعلامات الالتهاب :الهدف من البحث .لتهابية والمناعية
 أخذت عينات من : المواد وطرق العمل. لدى النساء في سن اليأس بمستويات النساء الأصحاء كمجموعة ضابطة(التفاعلي
 ، امرأة سليمة كمجموعة ضابطة) امرأة في سن اليأس بـ (45) وقورنت خمس وأربعون (45،متطوعات على مدار ثلاثة أشهر
 .تراوحت أعمارهن بين 45 و55 عام ٢
 النتائج: كان مؤشر كتلة الجسم أعلى بصورة معنوية لدى مجموعة النساء في سن اليأس مقارنةً بالمجموعة الضابطة ($p = 0.039$). كما
 كانت تراكيز الكوليسترول الكلي والدهون الثلاثية وLDL، إضافة إلى ضغط الدم الانقباضي والانبساطي، أعلى لدى مجموعة سن اليأس
 جميعها ($p > 0.001$)، في حين كان HDL أقل معنويًا ($p = 0.011$). وارتفع CRP بصورة واضحة، بينما انخفض الإسترايول لدى
 مجموعة سن اليأس كلاهما ($p < 0.001$)
 الاستنتاج: من المؤشرات البارزة لأمراض القلب والأوعية الدموية المبكرة لدى النساء في سن اليأس ارتفاع مستوى شحميات الدم،
 وارتفاع العلامات الالتهابية (البروتين التفاعلي سي)، وانخفاض مستويات هرمون الإستروجين.

الكلمات المفتاحية: البروتين التفاعلي-سي، إسترايول، ملف الدهون.