e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(3); 1946-1948

Original Research Article

Comparative Analysis of Oestrogen Levels and Heart Rate Variability in Premenopausal and Postmenopausal Women

Atreya Tanu[1], Astha Patni[2]

[1]Associate Professor, Department of Physiology, SMS Medical College and Hospitals, Jaipur, Rajasthan, India

[2]Associate Professor, Department of Physical Medicine and Rehabilitation, SMS Medical College and Hospitals, Jaipur, Rajasthan, India

Received: 26-12-2025 / Revised: 25-01-2026 / Accepted: 27-02-2026

Corresponding Author: Atreya Tanu

Conflict of interest: Nil

Abstract:

Background: Menopause induces significant physiological and hormonal changes Menopause causes an imbalance of the autonomic nervous control of the cardiovascular system that shifts toward sympathetic hyperactivity This study compares demographic and physiological parameters, including estrogen levels and heart rate variability (HRV), between premenopausal and postmenopausal women and explores the correlation between estrogen levels and HRV total power.

Methods: A cross-sectional study was conducted involving 60 women: 30 premenopausal and 30 postmenopausal. Demographic, physiological, and biochemical parameters such as age, weight, blood pressure, basal heart rate, body fat percentage, and serum estrogen levels were recorded. HRV was analyzed using spectral power with a focus on total power. Pearson correlation analysis was used to assess relationships between estrogen levels and HRV.

Results: Statistically significant differences were observed between the groups. significantly reduced estrogen levels ($p < 0.001$). HRV total power was significantly reduced in postmenopausal women ($p < 0.001$). A strong positive correlation between estrogen levels and HRV total power was found in both groups ($r = +0.71$ and $+0.66$, respectively), with a combined correlation of $+0.74$ ($p < 0.001$).

Conclusion: Menopause significantly affects autonomic function, menopause shifts the autonomic sympathovagal balance towards sympathetic predominance in post-menopause. The strong positive correlation between estrogen and HRV underscores estrogen's role in cardiovascular autonomic regulation. These findings highlight the importance of hormonal assessment and cardiovascular monitoring in postmenopausal women.

Keywords: Menopause, Estrogen, HRV, Autonomic Function, Total Power, Cardiovascular Health.

DOI: 10.25258/ijcpr.18.3.320

Introduction

Menopause, defined by the permanent cessation of menstruation, is associated with significant physiological and hormonal changes, most notably a marked reduction in estrogen production. Postmenopausal women have alteration in their autonomic status with higher sympathetic and lower vagal tone compared with premenopausal women These hormonal shifts are known to affect cardiovascular health, body composition, and autonomic nervous system regulation [1,2].

Cardio protective role of estrogen is supported by the observation that the excess risk of cardio vascular disease in women who underwent oophorectomy in young adulthood is prevented by estrogen. In addition data shows a significant reduction in the risk of heart disease in women who take estrogen after a non-surgical menopause[7]

Heart Rate Variability (HRV), a non-invasive measure of autonomic nervous system function, reflects the balance between sympathetic and parasympathetic activity [3]. Studies have shown that decreased estrogen levels post-menopause may lead to a decline in HRV, thereby increasing cardiovascular risk [4,5]. This study explores the correlation between estrogen levels and HRV and to provide a comprehensive comparison between premenopausal and postmenopausal women.

Materials and Methods

A cross-sectional study was conducted on 60 women, divided into two groups: 30 premenopausal (age 40-50 years) and 30 postmenopausal(45-55years) of medical profession from medical colleges of Jaipur, selected based on clinical history and menstrual. No history of

chronic illnesses or medication affecting cardiovascular or hormonal status

After detailed inquiry of medical history the subjects with diabetes, hypertension other cardiovascular diseases, subjects on oral contraceptive, hormone replacement therapy and drugs that alter drugs that alter the cardiovascular functions were excluded from the study. Informed written consent was obtained from all participants and the experimental protocol was approved by ethics committee of the college.

Subjects were screened after measuring height, weight, basal blood pressure and basal heart rate. The basal recording of blood pressure was done using phygmomanometer by standard Riva Roci method. The experiment was carried out in the morning in the fasting state.

All the subjects underwent the test for the study in the following order. 1. Blood sample collection- 5 ml of venous blood sample was collected and serum was separated. Samples were stored at 20^0 C until further analysis and serum estradiol asssay was performed using CLIA.

2.Body fat percentage – included the measurement of skin fold thickness at triceps, sub scapular and suprailiac sites. The sum of three skin folds was used in age and gender specific equation to obtain an estimate of body fat percentage.

3. Heart rate variability was recorded by medical analyzer module (based on principle of impedence plethysmography) of NIVOMON, L&T and analysis of signal were done in frequency domain measures. In the frequency domain analysis the power spectrum for the HRV was calculated by the traditional fast Fourier transform (FFT) based method. Data was edited manually for artifacts and ectopic beats. Frequency domain measurement including total variance, high frequency power HF (0.15-0.40 Hz), low frequency power (0.04-0.15hz) and LF/HF ratio was calculated to assess sympathetic/parasympathetic modulation.

Analysis of data –Numerical data are expressed as means± SD (analysis was performed using Microsoft excel software, Microsoft corporation USA, 2003. Data between the study groups were compared using unpaired student t–test. Statistical significance was assigned at $p<0.05$.

Statistical Analysis: Data were presented as mean ± SD. Student's t-test was used for group comparisons. Pearson's correlation coefficient was calculated to assess the relationship between estrogen levels and HRV total power. Significance was set at $p < 0.05$.

Results

Significant differences were noted between the two groups:

Table 1: Comparison of Demographic and Physiological Parameters Between Premenopausal and Postmenopausal Women

Parameter	Premenopausal (n = 30)	Postmenopausal (n = 30)	p-value	Significance
Age (years)	42.83 ± 2.59	50.37 ± 3.22	< 0.001	Highly Significant (**)
Weight (kg)	55.3 ± 4.68	61.9 ± 7.14	< 0.001	Highly Significant (**)
Systolic BP (mmHg)	121.6 ± 4.91	130.8 ± 8.41	< 0.001	Highly Significant (**)
Diastolic BP (mmHg)	78.0 ± 4.30	80.9 ± 2.81	< 0.05	Significant (*)
Basal Heart Rate (BPM)	72.1 ± 3.8	77.8 ± 5.6	< 0.001	Highly Significant (**)
Body Fat Percentage (%)	227.9 ± 19.93	253.93 ± 29.49	< 0.001	Highly Significant (**)
Estrogen (pg/ml)	37.34 ± 6.12	16.03 ± 3.82	< 0.001	Highly Significant (**)

Table 2: Correlation Between Estrogen Levels and Total Power (HRV)

Group	Estrogen Level (pg/ml)	Total Power (ms^2)	Correlation (r)	p-value	Significance
Premenopausal	37.34 ± 6.12	3002.9 ± 2102.4	**+0.71** (strong)	< 0.001	Highly Significant (**)
Postmenopausal	16.03 ± 3.82	791.7 ± 770.3	**+0.66** (moderate)	< 0.001	Highly Significant (**)
Overall (Combined)	—	—	**+0.74** (strong)	< 0.001	Highly Significant (**)

Table I shows subjects characteristics of study groups. It shows that basal systolic and diastolic blood pressure were significantly higher ($p<0.001$) in postmenopausal women. Body fat percentage was also significantly higher ($p<0.001$) among postmenopausal women.

Table 2 shows Estrogen level in postmenopausal women was significantly lower than premenopausal women($p<0.001$). and HRV Total Power: Substantially reduced in postmenopausal women ($p < 0.001$).

Correlation Analysis: Estrogen levels showed a strong positive correlation with HRV total power in

both premenopausal (r = +0.71) and postmenopausal (r = +0.66) groups. The overall correlation was r = +0.74 ($p < 0.001$).

Discussion

This study supports the hypothesis that estrogen plays an important role in maintaining autonomic balance. Lower HRV in postmenopausal women suggests reduced parasympathetic and increased sympathetic tone, consistent with previous findings [6,7].

Estrogen's vasodilatory and anti-inflammatory effects are well documented and are believed to contribute to improved autonomic function and vascular compliance [8,9]. Several recent studies reinforce the association between hormone replacement therapy (HRT) and improved cardiovascular outcomes [10,11].

A large cross-sectional Indian study confirmed that menopausal status significantly impacts HRV and correlates with symptoms of menopause [9]. Further, a recent review emphasized early intervention in the menopausal transition to mitigate cardiovascular risk [10]. Studies also indicate that transdermal estradiol may provide cardiovascular benefits over oral formulations [11].

These findings suggest that regular monitoring of HRV and estrogen levels may serve as effective early indicators for cardiovascular dysfunction in postmenopausal women [12].

Conclusion

Menopause significantly impacts autonomic and cardiovascular function. Reduced HRV and estrogen levels in postmenopausal women underscore the importance of hormonal regulation in autonomic balance. The strong correlation between estrogen and HRV suggests potential for targeted therapies and lifestyle interventions to maintain cardiovascular health during menopausal transition.

References

1. North American Menopause Society. Menopause Practice: A Clinician's Guide. 6th Ed. 2022.
2. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Circulation. 1996; 93(5):1043-1065.
3. Umetani K, Singer DH, McCraty R, Atkinson M. Am J Cardiol. 1998;81(6):799-802.
4. Lopes HF, Martin MG, Nashar K, et al. Clin Exp Hypertens. 2002;24(7-8):447-457.
5. Soares JM Jr, Baracat EC, Maciel GA. Clinics. 2014;69(10):705–709.
6. Muranaka K, Kimura K, Tomita T. Menopause Int. 2015;21(4):146-150.
7. Nudy MN, Aragaki AK, Jiang X, Manson JE, et al. Obstet Gynecol. 2025;145(4):357.
8. Int J Mol Sci. 2025;26(11):5078.
9. Sahu G, Bharshankar J, Shaikh AS. Eur J Cardiovasc Med. 2024. DOI:10.5083/ejcm.
10. Ivatt L, Paul M, et al. Circulation. 2025.
11. Climacteric. 2024. Transdermal vs Oral Estrogen Study.
12. Zhang C, Feng X, et al. Front Endocrinol. 2024;15:149497.