EJPMR, 2026, 13(1), 330-333 **Research Article** **Impact Factor 7.065**

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

ISSN (O): 2394-3211
ISSN (P): 3051-2573
Coden USA: EJPMAG

THE INTERPLAY OF PITUITARY GONADOTROPINS AND PROLACTIN IN THE PATHOGENESIS AND ACTIVITY OF RHEUMATOID ARTHRITIS: A CROSS SECTIONAL STUDY DESIGN

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DOI: https://doi.org/10.5281/zenodo.18094180

How to cite this Article: Rohitash Kumar*, Vyas R. K. (2026). The Interplay of Pituitary Gonadotropins and Prolactin In The Pathogenesis and Activity of Rheumatoid Arthritis: A Cross Sectional Study Design. European Journal of Pharmaceutical and Medical Research, 13(1), 330–333.

Article Received on 05/12/2025 **Article Revised on 25/12/2025** **Article Published on 01/01/2026**

ABSTRACT

Background: Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory disorder predominantly affecting middle-aged individuals, leading to joint pain, disability, and systemic complications. Early diagnosis and evaluation are essential to prevent disease progression and associated metabolic disturbances. **Objectives:** To evaluate and compare selected biochemical parameters in newly diagnosed patients with rheumatoid arthritis and age- and gender-matched healthy controls **Methods:** The present observational cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Departments of General Medicine and Physical Medicine and Rehabilitation at Sardar Patel Medical College and Associated Group of Hospitals, Bikaner, Rajasthan. A total of 80 subjects aged 45–60 years were enrolled and divided into two groups: Group A comprising 40 newly diagnosed cases of rheumatoid arthritis diagnosed according to the Modified 2010 ACR-EULAR classification criteria, and Group B comprising 40 age- and gender-matched healthy controls. Patients with autoimmune diseases other than RA, metabolic and endocrine disorders, chronic systemic illnesses, malignancy, obesity (BMI >30), smokers, those on hormone therapy, corticosteroids, lipid-lowering drugs, vitamins or minerals, and those already receiving anti-rheumatic treatment were excluded. After obtaining informed consent, 7–8 mL of venous blood was collected under aseptic precautions. Serum was analyzed for the required biochemical parameters. **Results:** The biochemical parameters analyzed showed significant differences between newly diagnosed rheumatoid arthritis patients and healthy controls, indicating early metabolic and inflammatory alterations associated with rheumatoid arthritis. **Conclusion:** This study was designed to evaluate biochemical alterations in newly diagnosed rheumatoid arthritis patients by comparing them with healthy controls, providing insight into early disease-related changes.

KEYWORDS: RA, FSH, LH, PRL.

INTRODUCTION

Rheumatoid arthritis is a chronic autoimmune disease characterized by systemic inflammation, primarily affecting the synovial joints and leading to progressive joint destruction and functional disability. Its global prevalence is estimated to be between 0.5% and 1%, predominantly affecting women aged 25 to 55 years, suggesting a potential link between sex hormones and disease susceptibility.[1] Specifically, aberrant levels of follicle-stimulating hormone and luteinizing hormone, key regulators of reproductive function, have been implicated in the pathogenesis and progression of RA.[2] This review aims to synthesize existing research concerning the association between FSH and LH levels and the risk of developing rheumatoid arthritis, as well as their influence on disease activity and severity. Understanding these hormonal roles could offer crucial insights into the underlying mechanisms of RA and pave the way for novel diagnostic and therapeutic strategies.[3] Given that women are disproportionately affected by RA, often at a younger age, the investigation into female hormonal factors, such as FSH and LH, becomes

particularly pertinent.[4] Elevated FSH levels, for instance, have been identified as a potential risk factor for RA and are positively correlated with disease activity.[5] This hormonal dysregulation, particularly the elevated gonadotropin levels observed in postmenopausal patients, may contribute to the differential disease manifestations and severity seen across various reproductive stages.[6]

MATERIALS AND METHODS

This observational cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Departments of General Medicine and Physical Medicine and Rehabilitation at Sardar Patel Medical College and Associated Group of Hospitals, Bikaner, Rajasthan.

Patients aged 45–60 years presenting with joint pain were screened and clinically diagnosed with rheumatoid arthritis (RA) according to the Modified 2010 American College of Rheumatology/European League Against Rheumatism (ACR-EULAR) classification criteria. Only newly diagnosed RA patients were included.

Patients aged 30–45 years, individuals with premature ovarian failure, bilateral ovariectomy, malignancy, or those receiving hormone replacement therapy, contraceptives, corticosteroids (>7.5 mg/day), or anti-rheumatic drugs were excluded. Additional exclusion criteria included smoking, history of other autoimmune diseases, diabetes mellitus, endocrine disorders, tuberculosis, cardiovascular, hepatic or renal disease, obesity (BMI >30 kg/m²), use of lipid-lowering agents, vitamin or mineral supplementation, and recent trauma.

A total of 80 participants were enrolled and divided into two groups: Group A comprised 40 newly diagnosed RA patients, while Group B included 40 age- and gender-matched healthy controls.

After obtaining informed consent, 7–8 mL of venous blood was collected under aseptic conditions. Samples were allowed to clot at 37 °C for 30 minutes and were then centrifuged at 3000 rpm for 15 minutes and the separated serum was used for biochemical analysis of the study parameters.

Statistical Analysis

Sample Size

Sample size estimation was performed using Epi Info statistical software. Based on data from a reference study by Xianhui Zhang *et al.* (2022)[2] the mean follicle-stimulating hormone (FSH) level in patients with rheumatoid arthritis was 57.58 ± 15.94, compared to 43.11 ± 19.46 in the control group.

With an alpha error of 5%, beta error of 20% (power of 80%), and assuming a two-sided test, the minimum required sample size was calculated to be 35 participants in each group. To account for an anticipated data loss or dropout rate of approximately 10%, the sample size was rounded up to 40 participants per group for the present study.

The sample size was calculated using the formula:

$$n = \frac{(Z_{\alpha/2} + Z_{\beta})^2 \times 2\sigma^2}{d^2}$$

Where σ represents the pooled standard deviation and d denotes the expected difference in mean FSH levels between the two groups

Data Analysis

Data were collected using a pre-structured proforma and analyzed using Microsoft Excel and Epi Info software. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Group comparisons were performed using unpaired Student's *t*-test or one-way ANOVA for continuous variables, and Chi-square or Fisher's exact test for categorical variables. A *p*-value <0.05 was considered statistically significant

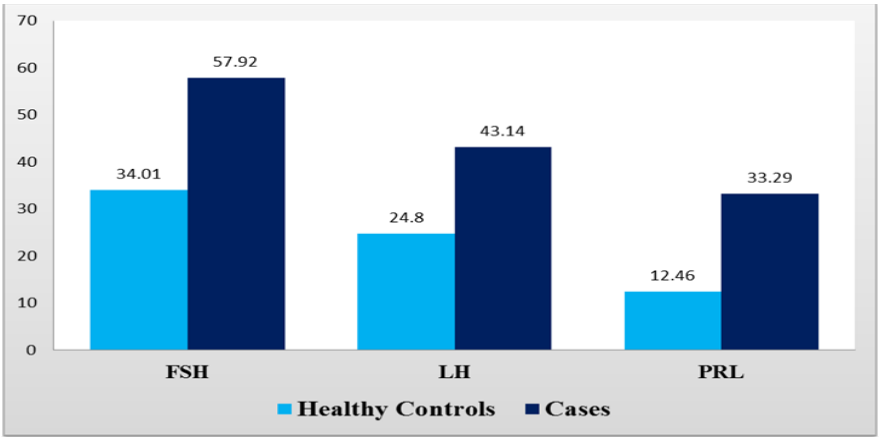
OBSERVATIONS

In our study mean age of participants was comparable between the two groups, with healthy controls at 50.10 ± 10.54 years and cases slightly older at 52.16 ± 11.35 years. Analysis of demographic profile revealed that a majority of RA cases hailed from rural areas (25), with the remaining 15 from urban regions. Similarly, among healthy controls, rural participants (22) slightly outnumbered urban participants (18), reflecting a predominance of rural representation in both groups. In our study we measured the level of three hormones FSH, LH and PRL in rheumatoid arthritis patients. Here, Hormonal assessment revealed highly significant differences between RA cases and healthy controls. FSH was substantially higher in cases (57.92 ± 10.26 mIU/ml) versus controls (34.01 ± 7.34 mIU/ml; t = 11.98, P < 0.0001). Similarly, LH levels were elevated in RA patients (43.14 ± 8.39 mIU/ml) compared to controls (24.80 ± 6.28 mIU/ml; t = 11.07). PRL levels were also markedly increased in the RA group (33.29 ± 8.23 ng/ml) versus controls (12.46 ± 4.08 ng/ml; t = 14.34).

Table 1: Statistical analysis of serum FSH, LH and prolactin in both the studied groups.

S.No	Parameters	Healthy Control Group (Mean ± S.D.)	RA Group (Mean ± S.D.)	T- test	P value
1.	FSH (mIU/ml)	34.01 ±7.34	57.92 ± 10.26	11.98	P<0.0001
2.	LH (mIU/ml)	24.80 ±6.28	43.14 ± 8.39	11.07	
3.	PRL (ng/ml)	12.46 ±4.08	33.29 ± 8.23	14.34	

Note: $p < 0.0001$ = Highly significant.



Graph: 1
Graphical representation of mean FSH, LH and PRL in both groups.

DISCUSSION

Consistent to our results **Buckman et al.**[7] reported that rheumatoid arthritis was less recorded among the younger age group as compared to the older age group with a mean age of 51.25 years in their study. **Lee et al**[8] reported mean age of 51.8 years among the study population with female preponderance (around 86% were females in their study (**Buckman et al**[7]).

As we found that peak age of RA in women around menopause is accompaniedby a sharp increase in serum follicle-stimulating hormone (FSH) levels. Another study by **Zhang et al**[2] also reported that high FSH levels are a possible risk factor for RA and are positively associated with disease activity.

Kass AS et al.[9] also stated that the increases in LH and FSH correlated positively with an increase in key pro-inflammatory cytokines including TNFα, interleukin 1 beta, IL-2, IL-2R, IL-8, and many other pro-inflammatory cytokines. That is probably the reason behind the worsening of rheumatoid arthritis in the post-partum period and menopause. We also evaluated the correlation between these hormones and repotted that FSH and LH were mildly positively correlated ($r = 0.28$), suggesting a linked increase in these gonadotropins. FSH also showed a mild positive correlation with PRL ($r = 0.26$), whereas LH demonstrated a moderate positive correlation with PRL ($r = 0.35$), highlighting interdependent hormonal alterations in RA patients. **Erb et al.**[10] reported that hormone prolactin is known to have a number of pro-inflammatory actions and may play a role in the pathogenesis and persistence of several autoimmune diseases, including rheumatoid arthritis.

Limitations of the study & Recommendations

Limitations of our study are small sample size and non measurement of other markers of inflammation & Cardiovascular risk factors in patients with Rheumatoid Arthritis. Moreover, a large cross-sectional study needs to be done to conclude the fact.

ACKNOWLEDGEMENTS

It is our proud privilege to express profound sense of gratitude & sincere thanks to all the participants and specially Head of the Institute of sardar patel medical college for their support to make this study successful which has been completed with logical and fruitful conclusion.

REFERENCES

1. Lashkari, M., Noori, A., Hajiimanouchehri, F., Oveisi, S. and Kazemifar, A.MM. 2014. Determination of specificity and sensitivity of anti-RA 33 in diagnosis of early rheumatoid arthritis. Global Journal of Health Science, 6(4): 292.
2. Zhang X, Qiao P, Guo Q, Liang Z, Pan J, Wu F, et al. High Follicle- Stimulating Hormone Level Associated With Risk of Rheumatoid Arthritis and Disease Activity. Front Endocrinol (Lausanne) 2022; 13: 862849.
3. Namavari et al., 2024, Menopausal state and rheumatoid arthritis: a systematic review and meta-analysis, BMC Rheumatol. 2024 Sep 30; 8(1): 48. doi:10.1186/s41927-024-00418-2.
4. Israel, T., Saleh, H.A., Ahmed, S.F. et al. Anti-Müllerian hormone (ovarian reserve) in rheumatoid arthritis patients: correlation with disease activity. Egypt Rheumatol Rehabil, 2024; 51: 28. https://doi.org/10.1186/s43166-024-00255-8
5. Novella-Navarro M, Plasencia-Rodríguez C, Nuño L, Balsa A. Risk Factors for Developing Rheumatoid Arthritis in Patients With Undifferentiated Arthritis and Inflammatory Arthralgia. Front Med (Lausanne). 2021 Jun 15; 8: 668898. doi: 10.3389/fmed.2021.668898. PMID: 34211986; PMCID: PMC8239127
6. Alpízar-Rodríguez D, Finckh A. Environmental factors and hormones in the development of rheumatoid arthritis. Semin Immunopathol. 2017 Jun; 39(4): 461-468. doi: 10.1007/s00281-017-0624-2. Epub 2017 Apr 27. PMID: 28451785.

7. Buckman Tonnies Abeku et al. Demographic, Clinical Profile of Rheumatoid Arthritis Patients and Their Association with Disease Severity in Ghana Int J Rheumatol. 2024 Jan 12; 2024: 6639079.
8. Lee Y., Cui J., Lu B., et al. Pain persists in DAS28 rheumatoid arthritis remission but not in ACR/EULAR remission: a longitudinal observational study. Arthritis Research and Therapy. 2011; 13(3): R83. doi: 10.1186/ar3353.
9. Kass AS, Lea TE, Torjesen PA, Gulseth HC, Forre T. The association of luteinizing hormone and follicle-stimulating hormone with cytokines and markers of disease activity in rheumatoid arthritis: a case-control study. Scand J Rheumatol, 2010; 39(2): 109-17.
10. Erb N, Pace A, Delamere J, Kitas J. Control of unremitting rheumatoid arthritis by the prolactin antagonist cabergoline. Rheumatology, 2001; 40(2): 237-239.