DOI: https://doi.org/10.53350/pjmhs020231712356

ORIGINAL ARTICLE

Biochemical Profiling of Inflammatory and Metabolic Markers in Reproductive-Age Women with Endometriosis

HANANA HAMEED[1], AAISHA QADIR[2], TARIQ HUSSAIN[3], WAQAS QURSHI[4], ARIFA INAYAT[5], NOSHEEN SIKANDAR BALOCH[6]
[1]*Associate Professor, Gynecology and Obstetrics Department, Bolan Medical College, Quetta, Pakistan*
[2]*Assistant professor Biochemistry, Pak Red Crescent Medical and Dental College, Kasur, Pakistan*
[3]*Senior Demonstrator, Sheikh Zayed Medical College, Rahim Yar Khan, Pakistan*
[4]*Assistant Professor Physiology, ABWA Medical College Khurrianwala, Pakistan*
[5]*Associate Professor, Gynecology and Obstetrics Department, unit 3, civil hospital, Quetta, Pakistan*
[6]*Associate Professor Department of Obstetrics and Gynaecology/ Oncology Bolan Medical College Quetta, Pakistan*
Correspondence to: Hanana Hameed, Email: drhananah@gmail.com

ABSTRACT

Background: Endometriosis is a chronic gynecological disorder characterized by the ectopic presence of endometrial tissue, commonly presenting with pelvic pain, dysmenorrhea, and infertility. Increasing evidence suggests that endometriosis is a systemic disease involving chronic inflammation and metabolic dysfunction, yet data from South Asian populations remain limited.

Objective: To assess the biochemical profile of inflammatory and metabolic markers in reproductive-age women with endometriosis and compare them with healthy controls.

Methods: This cross-sectional study was conducted from May 2022 to February 2023 at Bolan Medical Complex Hospital and the Gynecology and Obstetrics Department, Unit 3, Civil Hospital Quetta. A total of 120 women aged 18–45 years were enrolled, including 80 women with laparoscopically confirmed endometriosis and 40 healthy controls. Serum levels of hs-CRP, IL-6, TNF-α, fasting insulin, glucose, lipid profile, and HOMA-IR were measured. Statistical analysis was performed using SPSS version 26.0.

Results: Inflammatory markers were significantly elevated in women with endometriosis: hs-CRP (6.72 ± 1.18 vs. 3.25 ± 0.94 mg/L), IL-6 (7.94 ± 2.43 vs. 3.37 ± 1.12 pg/mL), and TNF-α (13.12 ± 3.45 vs. 8.24 ± 2.21 pg/mL) (all $p < 0.001$). Metabolic disturbances were also noted, with increased fasting insulin, glucose, and HOMA-IR values. Triglycerides were elevated and HDL-C levels were significantly reduced in the endometriosis group. Correlation analysis showed positive associations between disease severity and both inflammatory markers and insulin resistance.

Conclusion: Endometriosis in reproductive-age women is associated with systemic inflammation and metabolic dysfunction, with a clear correlation between biochemical abnormalities and disease severity. These findings support the need for a multidisciplinary approach in the diagnosis and management of endometriosis, integrating metabolic and inflammatory monitoring.

Keywords: Endometriosis, inflammation, metabolic dysfunction, HOMA-IR, cytokines, reproductive-age women, hs-CRP, IL-6, TNF-α.

INTRODUCTION

Endometriosis is a chronic, estrogen-dependent, inflammatory disease defined by the presence of functional endometrial glands and stroma outside the uterine cavity, most commonly affecting the ovaries, pelvic peritoneum, and surrounding reproductive structures[1]. It is one of the most frequent gynecological conditions among women of reproductive age, with an estimated global prevalence ranging from 10% to 15%, and higher rates observed among women presenting with pelvic pain or infertility. Clinically, endometriosis manifests as severe dysmenorrhea, chronic pelvic pain, dyspareunia, and varying degrees of subfertility, making it a major contributor to physical, emotional, and reproductive morbidity in affected individuals[2].

Historically, endometriosis was perceived as a localized pelvic disease primarily driven by retrograde menstruation and estrogen dominance. However, accumulating research now positions endometriosis as a complex, systemic disorder with profound implications on immune function, inflammatory regulation, oxidative stress, and metabolic homeostasis[3]. The ectopic endometrial tissue in endometriosis displays heightened proliferative activity, increased resistance to apoptosis, and the capacity to elicit a persistent inflammatory microenvironment characterized by immune cell infiltration, cytokine overexpression, and angiogenesis. This pro-inflammatory state is not restricted to the lesions but has systemic manifestations, reflected in altered serum levels of various inflammatory markers[4].

Several studies have reported elevated concentrations of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and acute-phase proteins like high-sensitivity C-reactive protein (hs-CRP) in the serum and peritoneal fluid of women with endometriosis[5]. These cytokines not only sustain the inflammatory cascade but are also implicated in nociceptive sensitization, which contributes to chronic pain. Simultaneously, endometriosis has been associated with components of metabolic dysfunction, including insulin resistance, dyslipidemia, hyperinsulinemia, and altered glucose metabolism. These metabolic derangements may be both a consequence of chronic inflammation and a contributing factor to disease progression, forming a vicious cycle that further complicates clinical outcomes[6].

The interplay between systemic inflammation and metabolic dysfunction in endometriosis is increasingly recognized in the scientific literature, giving rise to the emerging concept of "immunometabolic endometriosis." This paradigm shift underscores the need for a holistic diagnostic approach that goes beyond the identification of pelvic lesions to include assessment of systemic biomarkers. Biomolecular profiling of these patients may not only aid in early diagnosis but also provide insights into individualized management strategies, such as anti-inflammatory and insulin-sensitizing therapies[7, 8].

Despite growing international research, there is a critical lack of region-specific data on the biochemical alterations in South Asian women with endometriosis[9]. In countries like Pakistan, diagnostic delays are common, and the disease burden remains underrecognized due to socio-cultural factors, limited access to diagnostic laparoscopy, and insufficient integration of biochemical evaluations into gynecological workups. As a result, many patients remain undiagnosed or mismanaged, leading to prolonged suffering and compromised fertility outcomes[10, 11].

The current study aimed to investigate the biochemical profile of inflammatory and metabolic markers in reproductive-age women diagnosed with endometriosis compared to healthy age-matched controls. The specific aim was to measure serum levels

Received on 27-04-2023
Accepted on 28-11-2023

of hs-CRP, IL-6, TNF-α, fasting insulin, glucose, lipid profile, and insulin resistance using the HOMA-IR index, and to explore their correlation with disease severity. By establishing a biochemical signature associated with endometriosis in this population, this study seeks to enhance the understanding of the systemic nature of the disease and support the development of integrative diagnostic and therapeutic frameworks that target both inflammatory and metabolic pathways[12].

MATERIALS AND METHODS

This cross-sectional analytical study was conducted over a ten-month duration, from May 2022 to February 2023, at two leading tertiary care hospitals in Quetta, Pakistan: the Gynecology and Obstetrics Department, Unit 3, Civil Hospital Quetta, and the Department of Gynecology, Bolan Medical Complex Hospital, Quetta. Ethical approval was obtained from the respective institutional review boards, and informed written consent was secured from all participants prior to data collection.

Study Design and Population: A total of 120 women aged between 18 and 45 years were enrolled in the study. Of these, 80 women had laparoscopically confirmed endometriosis, while 40 healthy age-matched women with no evidence of endometriosis on clinical or sonographic examination served as controls. The diagnosis of endometriosis in patients was confirmed through both visual inspection during diagnostic laparoscopy and histopathological verification. Participants in both groups were selected using purposive sampling from outpatient and inpatient gynecological admissions.

Inclusion and Exclusion Criteria: Women were included in the study if they were within the reproductive age range (18–45 years), had regular menstrual cycles (25–35 days), and were not on any hormonal medications. Women in the patient group were required to have a laparoscopic confirmation of endometriosis. The control group included women attending the gynecology outpatient department for unrelated, non-inflammatory conditions and had no personal or family history of endometriosis. Women were excluded if they had a diagnosis of polycystic ovary syndrome (PCOS), diabetes mellitus, thyroid disorders, autoimmune diseases, cardiovascular disease, chronic inflammatory conditions, or any malignancy. Additionally, women who were pregnant, menopausal, or had used anti-inflammatory or hormonal medications within the last three months were excluded to avoid confounding biochemical alterations.

Sample Collection and Biochemical Analysis: Venous blood samples (5 mL) were collected from all participants during the early follicular phase of their menstrual cycle (days 2–5), after an overnight fast of 8–12 hours. Blood samples were drawn aseptically and centrifuged at 3000 rpm for 10 minutes. The serum was separated and immediately stored at –80°C until further analysis. All biochemical evaluations were carried out in the central diagnostic laboratory of Bolan Medical Complex Hospital Quetta using standardized protocols and quality control procedures.

Inflammatory markers assessed included high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). hs-CRP was measured using a high-sensitivity turbidimetric immunoassay, while IL-6 and TNF-α levels were determined through enzyme-linked immunosorbent assay (ELISA) techniques using commercially available kits. Metabolic parameters included fasting blood glucose, fasting serum insulin, and a full lipid profile. Glucose levels were measured using the glucose oxidase-peroxidase method, and serum insulin was evaluated using a chemiluminescent immunoassay. Lipid profile testing included serum total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglyceride concentrations, assessed by enzymatic colorimetric assays.

To evaluate insulin resistance, the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was calculated for each participant using the formula:

$$\text{HOMA-IR} = \frac{\text{Fasting Insulin}(\mu U/mL) \times \text{Fasting Glucose}(mg/dL)}{405}$$

This index served as a surrogate marker for identifying metabolic imbalance and insulin resistance in the study participants.

Statistical Analysis: All statistical analyses were performed using IBM SPSS software version 26.0. Continuous variables were assessed for normality using the Shapiro-Wilk test. Normally distributed data were expressed as mean ± standard deviation (SD), while non-normally distributed data were reported as medians with interquartile ranges. Between-group comparisons were performed using the independent sample t-test for normally distributed variables and the Mann-Whitney U test for non-parametric data. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test. Correlations between serum biochemical markers and the severity of endometriosis (staged I to IV according to the revised American Society for Reproductive Medicine classification) were evaluated using Pearson or Spearman correlation coefficients, depending on data distribution. A p-value of less than 0.05 was considered statistically significant in all analyses.

RESULTS

This study included n=120 women of reproductive age, of whom 80 were diagnosed with endometriosis and 40 served as healthy controls. The demographic, clinical, inflammatory, and metabolic characteristics of both groups were analyzed. The data revealed significant differences in inflammatory cytokines and metabolic markers between the two groups, indicating a systemic immunometabolic disturbance in patients with endometriosis.

Demographic and Baseline Characteristics: The mean age of participants in the endometriosis group was 32.6 ± 5.4 years, whereas in the control group it was 31.8 ± 4.9 years, showing no significant difference (p = 0.44). The mean body mass index (BMI) was slightly higher in the endometriosis group (26.9 ± 2.7 kg/m²) compared to controls (25.8 ± 2.5 kg/m²), but the difference was not statistically significant (p = 0.06). The average duration of menstrual flow and cycle length were comparable in both groups. Nulliparity was more prevalent in the endometriosis group (65%) than in controls (25%), which is consistent with the infertility burden often associated with the disease. Family history of endometriosis was positive in 23.7% of patients, while none of the control participants reported a similar history.

Table 1: Demographic and Baseline Characteristics of Study Participants

Characteristic	Endometriosis (n = 80)	Control (n = 40)	p-value
Age (years)	32.6 ± 5.4	31.8 ± 4.9	0.44
BMI (kg/m²)	26.9 ± 2.7	25.8 ± 2.5	0.06
Menstrual Cycle Length (days)	28.3 ± 2.1	27.9 ± 1.9	0.28
Menstrual Flow Duration (days)	4.9 ± 1.2	4.7 ± 1.1	0.42
Nulliparity (%)	65%	25%	<0.001
Positive Family History (%)	23.7%	0%	<0.001

As seen in Table 1, the majority of women with endometriosis were nulliparous and had a family history suggestive of genetic predisposition, factors known to increase the risk and severity of the disease.

Inflammatory Marker Profile: Serum levels of inflammatory markers were significantly elevated in women with endometriosis. The mean hs-CRP level was 6.72 ± 1.18 mg/L in the endometriosis group versus 3.25 ± 0.94 mg/L in controls (p < 0.001), indicating a state of systemic inflammation. Similarly, IL-6 levels were nearly twice as high in patients (7.94 ± 2.43 pg/mL) compared to controls (3.37 ± 1.12 pg/mL; p < 0.001). TNF-α levels were also significantly higher in affected women (13.12 ± 3.45 pg/mL vs. 8.24 ± 2.21 pg/mL; p < 0.001). These findings reflect a persistent and

exaggerated pro-inflammatory response in patients with endometriosis.

Table 2: Comparison of Inflammatory Markers Between Groups

Marker	Endometriosis (n=80)	Control (n=40)	p-value
hs-CRP (mg/L)	6.72 ± 1.18	3.25 ± 0.94	<0.001
IL-6 (pg/mL)	7.94 ± 2.43	3.37 ± 1.12	<0.001
TNF-α (pg/mL)	13.12 ± 3.45	8.24 ± 2.21	<0.001

Table 2 clearly demonstrates that endometriosis is not just a localized gynecological disorder but a condition with measurable systemic inflammatory activity.

Metabolic Profile and Insulin Resistance: The metabolic profile of the participants further supported the systemic nature of endometriosis. Women with the disease showed significantly elevated fasting insulin levels (15.7 ± 4.1 µU/mL) compared to the control group (9.6 ± 2.8 µU/mL; $p < 0.001$). Fasting glucose was also higher in the patient group (98.1 ± 9.3 mg/dL) than controls (91.4 ± 8.2 mg/dL; $p = 0.002$). Consequently, the calculated HOMA-IR values were markedly elevated in women with endometriosis (3.80 ± 0.94) versus controls (2.17 ± 0.63; $p < 0.001$), confirming significant insulin resistance. Dyslipidemia was also evident. Serum triglycerides were higher (171.6 ± 32.4 mg/dL vs. 138.7 ± 28.6 mg/dL; $p < 0.001$) and HDL-C levels were lower (39.1 ± 5.2 mg/dL vs. 47.2 ± 6.8 mg/dL; $p < 0.001$) in the endometriosis group.

Table 3: Comparison of Metabolic Markers and HOMA-IR Between Groups

Parameter	Endometriosis (n=80)	Control (n=40)	p-value
Fasting Insulin (µU/mL)	15.7 ± 4.1	9.6 ± 2.8	<0.001
Fasting Glucose (mg/dL)	98.1 ± 9.3	91.4 ± 8.2	0.002
HOMA-IR	3.80 ± 0.94	2.17 ± 0.63	<0.001
Triglycerides (mg/dL)	171.6 ± 32.4	138.7 ± 28.6	<0.001
HDL-C (mg/dL)	39.1 ± 5.2	47.2 ± 6.8	<0.001
Total Cholesterol (mg/dL)	181.4 ± 26.1	173.3 ± 21.5	0.082
LDL-C (mg/dL)	116.7 ± 20.9	111.4 ± 19.2	0.178

Table 3 provides clear evidence that women with endometriosis have significantly altered metabolic parameters. These abnormalities, particularly insulin resistance and reduced HDL-C, may contribute to the chronic inflammation and hormonal dysregulation associated with the disease.

Association Between Biochemical Markers and Disease Severity: Among the 80 women diagnosed with endometriosis, staging based on the revised American Society for Reproductive Medicine (rASRM) criteria revealed that 28 (35%) had minimal to mild disease (stage I–II), while 52 (65%) had moderate to severe disease (stage III–IV). Serum hs-CRP was significantly correlated with disease severity ($r = 0.48$, $p = 0.001$), as was IL-6 ($r = 0.44$, $p = 0.003$) and HOMA-IR ($r = 0.40$, $p = 0.005$). These findings suggest that as the clinical stage of endometriosis advances, the systemic inflammatory burden and insulin resistance also increase proportionally.

In summary, the results of this study reveal a consistent pattern of elevated systemic inflammation and metabolic dysregulation in reproductive-age women with endometriosis. The strong correlation between inflammatory/metabolic markers and disease severity underscores the need for a holistic approach to endometriosis management, incorporating not only gynecological assessment but also metabolic evaluation and inflammatory status monitoring.

DISCUSSION

The present study aimed to evaluate the biochemical profile of inflammatory and metabolic markers in reproductive-age women with endometriosis and to compare these findings with healthy controls. The results clearly demonstrate that endometriosis is associated with a distinct pattern of systemic inflammation and metabolic disturbances[13]. Women diagnosed with endometriosis exhibited significantly elevated levels of inflammatory cytokines including hs-CRP, IL-6, and TNF-α, as well as higher fasting insulin levels, increased HOMA-IR indices, elevated triglycerides, and decreased HDL-C levels. These findings reinforce the growing understanding that endometriosis is not merely a localized gynecological condition but a complex systemic disease with widespread immunometabolic implications[14].

The marked elevation of hs-CRP, IL-6, and TNF-α in patients with endometriosis supports the concept of persistent, low-grade systemic inflammation. IL-6 and TNF-α are potent pro-inflammatory cytokines known to play a central role in modulating immune responses, angiogenesis, and pain pathways in endometriosis[15]. The increased hs-CRP levels observed in our study are consistent with previous international research that links chronic pelvic inflammation with increased hepatic production of acute-phase proteins. These inflammatory markers not only reflect ongoing immune activation but may also participate in the propagation of the endometriotic lesions and in the exacerbation of pain symptoms commonly reported by patients[16].

Equally significant is the metabolic component revealed in our results. Women with endometriosis showed substantial evidence of insulin resistance, demonstrated by elevated fasting insulin and HOMA-IR values. This is of particular concern as insulin resistance can contribute to hormonal imbalances, including hyperestrogenism, which promotes the survival and proliferation of ectopic endometrial tissue. Dyslipidemia, characterized by elevated triglycerides and reduced HDL-cholesterol levels in our study population, further supports the involvement of metabolic dysfunction in the pathophysiology of endometriosis. These metabolic alterations not only have implications for disease progression but may also predispose affected women to long-term cardiometabolic risks[17].

The correlation analysis revealed a significant positive association between disease severity and levels of hs-CRP, IL-6, and HOMA-IR, suggesting that the extent of systemic inflammation and insulin resistance increases with the advancement of endometriosis. This provides valuable insight into the potential of using biochemical markers as non-invasive indicators of disease activity, which could assist in disease staging and monitoring response to treatment, particularly in settings where access to laparoscopic diagnosis is limited[18].

Our findings also reinforce the clinical observation that a substantial proportion of women with endometriosis are nulliparous, highlighting the association between the disease and infertility. Furthermore, the presence of a positive family history in a subset of patients suggests a possible genetic predisposition and underlines the importance of early screening and family-based risk assessment[19].

Although the current study provides robust evidence supporting the systemic nature of endometriosis, it is not without limitations. The cross-sectional design restricts causal interpretations. Additionally, being a hospital-based study from a specific region, the findings may not be generalizable to the broader population[20]. Other potential confounders such as diet, physical activity, and stress levels were not controlled for and may have influenced metabolic and inflammatory parameters. Nevertheless, the strength of this study lies in its dual-institutional approach, the inclusion of a substantial sample size, and the comprehensive biochemical analysis conducted under standardized conditions[21].

Given these findings, there is a strong rationale to consider an integrative management approach in women with endometriosis, one that targets both inflammation and metabolic dysfunction. Pharmacological interventions such as insulin sensitizers (e.g., metformin), anti-inflammatory agents, and lifestyle modifications including dietary changes and physical activity may have a potential role in modulating disease activity and improving quality of life[22].

CONCLUSION

This study provides compelling evidence that endometriosis in reproductive-age women is associated with significant systemic inflammatory and metabolic disturbances. Elevated levels of hs-CRP, IL-6, and TNF-α, along with increased insulin resistance and dyslipidemia, characterize the biochemical profile of affected individuals. These alterations were found to correlate with disease severity, suggesting their potential role in disease progression and clinical symptomatology. The findings emphasize the need to recognize endometriosis as a systemic immunometabolic disorder rather than a purely pelvic disease. Early detection and comprehensive management targeting both inflammatory and metabolic pathways may improve clinical outcomes and long-term health in women suffering from this debilitating condition.

Funding: No funding was received for this research project.

Conflicts of Interest: The authors declare that there are no conflicts of interest related to this study.

Ethical Approval: Ethical approval was obtained from the appropriate Institutional Review Board prior to the commencement of the study.

Informed Consent: Written informed consent was obtained from all participants before their inclusion in the study.

Authors' Contributions:

H.H. contributed to study design and overall manuscript preparation.

A.Q. was involved in data acquisition and literature review.

T.H. conducted statistical analysis and data interpretation.

W.Q. handled manuscript formatting and critical revisions.

A.I. provided supervision and clinical insight.

N.S.B. reviewed the final draft and approved the version to be submitted.

Acknowledgements: The authors sincerely acknowledge the support of all participants and the institutional staff for their assistance in this research.

REFERENCES

1. Kokot I, Piwowar A, Jędryka M, Sołkiewicz K, Kratz EM. Diagnostic significance of selected serum inflammatory markers in women with advanced endometriosis. International journal of molecular sciences. 2021;22(5):2295.
2. Murgia F, Angioni S, D'Alterio MN, Pirarba S, Noto A, Santoru ML, et al. Metabolic profile of patients with severe endometriosis: a prospective experimental study. Reproductive Sciences. 2021;28:728-35.
3. Rafi U, Ahmad S, Bokhari SS, Iqbal MA, Zia A, Khan MA, et al. Association of inflammatory markers/cytokines with cardiovascular risk manifestation in patients with endometriosis. Mediators of inflammation. 2021;2021(1):3425560.
4. Vicente-Munoz S, Morcillo I, Puchades-Carrasco L, Payá V, Pellicer A, Pineda-Lucena A. Pathophysiologic processes have an impact on the plasma metabolomic signature of endometriosis patients. Fertility and Sterility. 2016;106(7):1733-41. e1.
5. Irungu S, Mavrelos D, Worthington J, Blyuss O, Saridogan E, Timms JF. Discovery of non-invasive biomarkers for the diagnosis of endometriosis. Clinical Proteomics. 2019;16:1-16.
6. Mu F, Harris HR, Rich-Edwards JW, Hankinson SE, Rimm EB, Spiegelman D, et al. A prospective study of inflammatory markers and risk of endometriosis. American journal of epidemiology. 2018;187(3):515-22.
7. Kokot I, Piwowar A, Jędryka M, Kratz EM. Is there a balance in oxidative-antioxidant status in blood serum of patients with advanced endometriosis? Antioxidants. 2021;10(7):1097.
8. Kusum K, Raj R, Rai S, Pranjali P, Ashish A, Vicente-Muñoz S, et al. Elevated circulatory proline to glutamine ratio (PQR) in endometriosis and its potential as a diagnostic biomarker. ACS omega. 2022;7(17):14856-66.
9. Yao Z, Zhang Y, Yan J, Yan L. Deciphering biomarkers of endometriosis by proteomic analysis of eutopic endometrium in infertile patients. Journal of Gynecology Obstetrics and Human Reproduction. 2021;50(5):102043.
10. Vicente-Muñoz S, Morcillo I, Puchades-Carrasco L, Payá V, Pellicer A, Pineda-Lucena A. Nuclear magnetic resonance metabolomic profiling of urine provides a noninvasive alternative to the identification of biomarkers associated with endometriosis. Fertility and sterility. 2015;104(5):1202-9.
11. Starodubtseva N, Chagovets V, Borisova A, Salimova D, Aleksandrova N, Chingin K, et al. Identification of potential endometriosis biomarkers in peritoneal fluid and blood plasma via shotgun lipidomics. Clinical Mass Spectrometry. 2019;13:21-6.
12. Yang T, Zhao J, Liu F, Li Y. Lipid metabolism and endometrial receptivity. Human reproduction update. 2022;28(6):858-89.
13. Turathum B, Gao E-M, Grataitong K, Liu Y-B, Wang L, Dai X, et al. Dysregulated sphingolipid metabolism and autophagy in granulosa cells of women with endometriosis. Frontiers in Endocrinology. 2022;13:906570.
14. Cordeiro FB, Cataldi TR, do Vale Teixeira da Costa L, de Souza BZ, Montani DA, Fraietta R, et al. Metabolomic profiling in follicular fluid of patients with infertility-related deep endometriosis. Metabolomics. 2017;13:1-12.
15. Braga DP, Montani DA, Setti AS, Turco EGL, Oliveira-Silva D, Borges Jr E. Metabolomic profile as a noninvasive adjunct tool for the diagnosis of Grades III and IV endometriosis-related infertility. Molecular reproduction and development. 2019;86(8):1044-52.
16. Krasnyi AM, Sadekova AA, Smolnova TY, Chursin VV, Buralkina NA, Chuprynin VD, et al. The levels of ghrelin, glucagon, visfatin and glp-1 are decreased in the peritoneal fluid of women with endometriosis along with the increased expression of the CD10 protease by the macrophages. International Journal of Molecular Sciences. 2022;23(18):10361.
17. Gajbhiye R, Bendigeri T, Ghuge A, Bhusane K, Begum S, Warty N, et al. Panel of autoimmune markers for noninvasive diagnosis of minimal–mild endometriosis: a Multicenter Study. Reproductive sciences. 2017;24(3):413-20.
18. Vouk K, Ribič-Pucelj M, Adamski J, Rižner TL. Altered levels of acylcarnitines, phosphatidylcholines, and sphingomyelins in peritoneal fluid from ovarian endometriosis patients. The Journal of steroid biochemistry and molecular biology. 2016;159:60-9.
19. Hudson QJ, Perricos A, Wenzl R, Yotova I. Challenges in uncovering non-invasive biomarkers of endometriosis. Experimental Biology and Medicine. 2020;245(5):437-47.
20. Angioni S, Saponara S, Succu AG, Sigilli M, Scicchitano F, D'Alterio MN. Metabolomic characteristics in endometriosis patients. Endometriosis Pathogenesis, Clinical Impact and Management: Volume 9: Frontiers in Gynecological Endocrinology. 2021:9-17.
21. Cao X-L, Song J-Y, Sun Z-G. Quantitative label-free proteomic analysis of human follicle fluid to identify novel candidate protein biomarker for endometriosis-associated infertility. Journal of Proteomics. 2022;266:104680.
22. Gleason JL, Thoma ME, Zukerman Willinger N, Shenassa ED. Endometriosis and uterine fibroids and their associations with elevated C-reactive protein and leukocyte telomere length among a representative sample of US women: data from the National Health and Nutrition Examination Survey, 1999–2002. Journal of Women's Health. 2022;31(7):1020-8.

This article may be cited as: Hameed H, Qadir A, Hussain T, Qurshi W, Inayat A, Baloch NS: Biochemical Profiling of Inflammatory and Metabolic Markers in Reproductive-Age Women with Endometriosis. Pak J Med Health Sci, 2023;17(12):356-359.