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**Original Research Article**

# Evaluation of Adipsin, Resistin Hormones with Some Biochemical Variables in Obese Women with Endometriosis in Salah Al-Din City

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## Abstract

***Background:*** Endometriosis is a common condition associated with infertility and causes chronic pain in many women. It is defined by the presence of endometrial-like tissue growing outside the uterus and is a chronic inflammatory condition associated with chronic pelvic pain, although the cause and natural history of the disorder remain uncertain. This research study was conducted in the laboratory department of Tikrit Teaching Hospital in Salah al-Din Governorate and some private laboratories affiliated with Al-Dour city for a period from 10/15/2024 to 1/15/2025. The study groups included (70) samples of obese women of childbearing age, aged (18-45) years, and they were distributed into two groups: ***Control group:*** 35 blood samples were collected from healthy women after ensuring their good health. They were randomly selected from the residents of Salah Al-Din city. ***Patient group:*** 35 blood samples were collected from obese women suffering from endometriosis, after confirming their infection following tests for the disease and referring them to a specialist doctor after conducting an ultrasound examination and confirming their infection with endometriosis.
The disease cases were confirmed after being diagnosed by the specialist doctors. After that, blood was taken from both groups (patients and healthy people). Then, blood was collected from the patient and healthy people and separated by centrifugation. Then, the biochemical variables were measured, which included: (Insulin, CRP, FSH, LH, Estradiol, TNF-α, Adipsin, Resistin), The results of the current study showed a significant decrease in the level of (LH, Resistin) with a significant increase in the rest of the groups in the blood serum of both groups and at its level, the probability of $P \leq 0.05$.

**Keywords:** Endometriosis, Resistin, Adipsin, Insulin, Sex hormones.

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## INTRODUCTION

Endometriosis is a common condition associated with infertility and causes chronic pain in many women. It is defined by the presence of endometrial-like tissue growing outside the uterus and is known as a chronic inflammatory condition associated with chronic pelvic pain, Although the cause and natural history of the disorder remain uncertain, hormonal, neurological, and immune factors are all involved in the mechanisms that contribute to the development of symptoms [1].

Endometriosis is a chronic inflammatory disease characterized by immune dysfunction. The immune status of the peripheral and endometrial tissues in women with endometriosis appears to be altered, potentially leading to infertility, early pregnancy failure, and tissue imbalance [2,3]. Clinical studies have shown that the incidence of endometriosis increases with the occurrence of immune diseases, suggesting the possibility of a common pathophysiological pathway between endometriosis and immune diseases [4,5].

The mechanism of endometriosis development is influenced by numerous factors, including immune, endocrine, genetic, and environmental factors. The relative and absolute levels of various immune cells and factors that play key regulatory roles are altered in patients with endometriosis Meanwhile, hormones, which are important microregulators targeting various organs and tissues in the body, play crucial roles in the course of endometriosis. However, immune and endocrine regulatory mechanisms do not operate independently of each other in the development of endometriosis; rather, the immune and endocrine systems share reciprocal regulatory pathways and influence each other [6].

Insulin is a powerful stimulant for ovarian androgen secretion, which occurs via a different insulin

receptor that does not exhibit insulin resistance. Therefore, insulin doubles the effect of LH and also doubles the degree of androgen excess by inhibiting the liver's production of SHBG (a glycoprotein that binds most sex steroids). Thus, free androgen is elevated, excess body weight aggravates underlying hormonal disturbances (increased androgen and insulin levels) and clinical manifestations are evident in women with endometriosis [7].

Diagnosing endometriosis is a challenging topic. Despite extensive research into innovative laboratory tests and advances in imaging techniques, simple, non-invasive diagnostic tests are still lacking. However, given the inflammatory process associated with endometriosis, C-reactive protein (CRP) may be a target for initial screening. The inflammatory process associated with endometriosis is also widespread in the peritoneal space. Therefore, it is hypothesized that there is a difference between the level of C-reactive protein in the peritoneum of women with endometriosis and those without [8].

The most important hormonal event that can be observed in patients with endometriosis is the increase in the level of FSH, which is stimulated by the decline in the level of steroid production during the luteal phase, which is accompanied by an increase in the level of Luteinizing Hormone (LH) from the pituitary gland, which helps in stimulating a number of follicles [9]. There are special receptors for follicle-stimulating hormone on the surfaces of granulosa cells, and in the presence of FSH, the follicles begin to produce limited amounts of androgens [(10)]. In the presence of low concentrations, these androgens reduce the ability to produce estrogen, and thus the environment of the follicle becomes androgenic, leading to the decay of the follicle [11].

The production of estrogen is accompanied by a gradual decrease in the level of FSH, which can be observed during the middle of the follicular phase, which precedes the increase in the diameter of the follicle, which determines the continued growth and development of the primary follicle. The negative feedback mechanism of estrogen at the level of FSH helps in inhibiting all follicles except the follicle containing the egg that will be released. In order to respond to the LH surge, the cells of the granular layer must stimulate the receptors for this hormone. Here again, the importance of estrogen as a basic driver appears, as the increase in its level within the periphery of the follicle itself works to divert the direction of FSH from stimulating receptors for itself, and instead it stimulates the LH receptors [12].

Adipsin is a hormone of white adipose tissue that regulates the differentiation of fat cells and increases fat accumulation, which may be a possible reason for the association between adipsin and metabolic disorders [13]. Adipsin is involved in a variety of biological processes, including blood clotting and complement activation, fertility, the immune system, tissue repair, blood pressure, ferritin degeneration, as well as cell proliferation and apoptosis [14].

Resistin acts as a polypeptide and is involved in many different processes. Multiple tissues in the body have been found to respond to resistin. The mechanism of action may be endocrine and paracrine, but the resistin receptors are still unknown. Interestingly, A recent study has shown that resistin can bind and signal to the receptor (TLR4), thereby stimulating cytokine production in mononuclear cells in response to resistin stimulation [15]. The biological effects of resistin in humans are not fully understood. The primary physiological role of resistin may be to modulate inflammatory and immune responses and autoimmune diseases in human macrophages. Resistin stimulates inflammatory cytokines and enhances the expression of cell adhesion molecules, including vascular cell adhesion molecule and intercellular adhesion molecule, which contribute to chemotaxis and leukocyte recruitment to sites of inflammation [16].

Expression of the TNF-a gene is affected by obesity and its associated health complications [17]. In severe obesity, TNF-a is produced primarily by macrophages that spread to excess adipose tissue, and its levels are associated with the degree of obesity and insulin resistance [18]. High levels of TNF-a in peritoneal fluid contribute to the development of endometriosis by stimulating the production of interleukin-8. Gene and protein expression of IL-8 in endometrial stromal cells is regulated by the expression of type 1 and type 2 TNF-a receptors. This stimulation of IL-8 in endometrial stromal cells leads to the proliferation of endometrial cells, Thus, it facilitates its adhesion to the peritoneal membrane and the development of endometriosis [19]. Through the low level of (LH, resistin) and the high level of diagnostic indicators, the current study aimed to evaluate the level of the hormone (Resistin, Adibasin) with some biochemical variables in obese women suffering from endometriosis in Salah al-Din Governorate.

**Collection of specimens**

This research study was conducted in the laboratory department of Tikrit Teaching Hospital in Salah al-Din Governorate and some private laboratories affiliated with Al-Dour city for a period from 10/15/2024 to 1/15/2025. The study groups included (70) samples of obese women of childbearing age, aged (18-45) years, and they were distributed into two groups:

- **Control group:** 35 blood samples were collected from healthy women after ensuring their good health. They were randomly selected from the residents of Salah Al-Din city.

- **Patient group:** 35 blood samples were collected from obese women suffering from endometriosis, after confirming their infection following tests for the disease and referring them to a specialist doctor after conducting an ultrasound examination and confirming their infection with endometriosis.

The disease cases were confirmed after being diagnosed by the specialist doctors. After that, blood was taken from both groups (patients and healthy people). Then, blood was collected from the patient and healthy people and separated by centrifugation. Then, the biochemical variables were measured, which included: (Insulin, CRP, FSH, LH, Estradiol, TNF-α, Adipsin, Resistin).

**Estimating serum hormone levels**

The hormone assay, which includes (Insulin, FSH, LH, estradiol, TNF-α, adipsin, and resistin), These hormones was measured in serum according to the manufacturer, Monobind Inc., USA, using an ELISA test kit for each measurement.

**Estimating C-reactive protein concentration in serum:**

The CRP-latex test is used to estimate C-reactive protein in human serum through the reaction that occurs between latex, a polystyrene suspension coated with goat IgG anti-human CRP, and latex clots when mixed with serum samples, to estimate the degree of inflammation present in the human body [20].

**Statistical Analysis**

The results were analyzed using the Statistical Package for the Social Sciences (SPSS) using the completely randomized design (CRD) method, using the t-test to analyze the variance between two groups at a probability level of ($P \leq 0.05$).

## RESULTS

**• Estimating of biochemical variables for the samples studied in both groups:**

**Table 1: Shows the mean ± S.D of the biochemical variables for the samples studied in both groups.**

| Groups / Parameter | Mean ± SD | | P-Value |
|---|---|---|---|
| | Control No=35 | Patients No=35 | |
| Insulin (µU/mL) | 10.12 ± 5.93 | 28.83 ± 7.61 | P ≤ 0.001 |
| CRP (mg/L) | 5.81 ± 0.71 | 39.01 ± 11.03 | P ≤ 0.001 |
| FSH (µlU/ml) | 8.18± 0.21 | 12.09± 0.49 | P ≤ 0.001 |
| LH (µlU/ml) | 4.91 ± 0.57 | 1.14 ± 0.31 | P ≤ 0.001 |
| Estradiol (pg/ml) | 7.14 ± 0.61 | 13.34 ± 0.91 | P ≤ 0.001 |
| TNF-α (ng/L) | 60.21±4.31 | 85.12±4.49 | P ≤ 0.001 |
| Adipsin (ng/ml) | 867.37±338.61 | 1456.95±787.81 | P ≤ 0.001 |
| Resistin (ng/ml) | 218. 91±66.67 | 89. 36±24.05 | P ≤ 0.001 |

The results of the current study showed a significant increase in each of (Insulin, CRP, FSH, estradiol, TNF-α, adipsin). With a significant decrease in each of (LH, Resistin) in the blood serum of obese women suffering from endometriosis compared to the control group (healthy people), at a probability level of $P \leq 0.05$, as in the following figures:

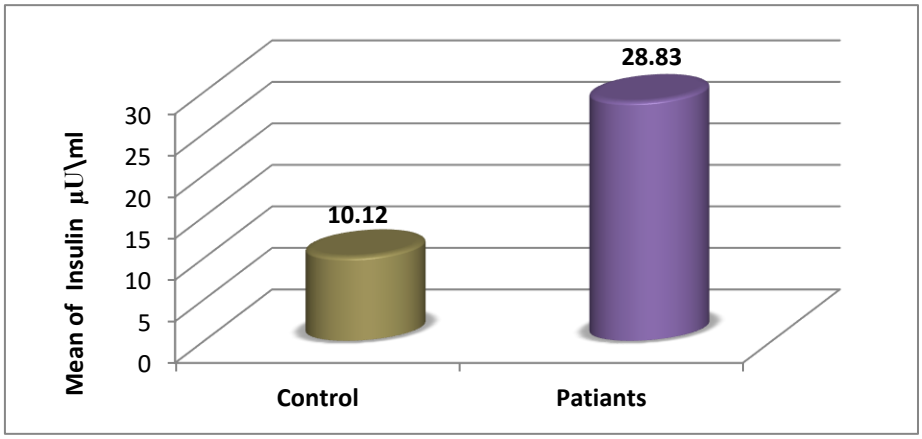


**Figure 1: Insulin level in the group of patients and healthy people**

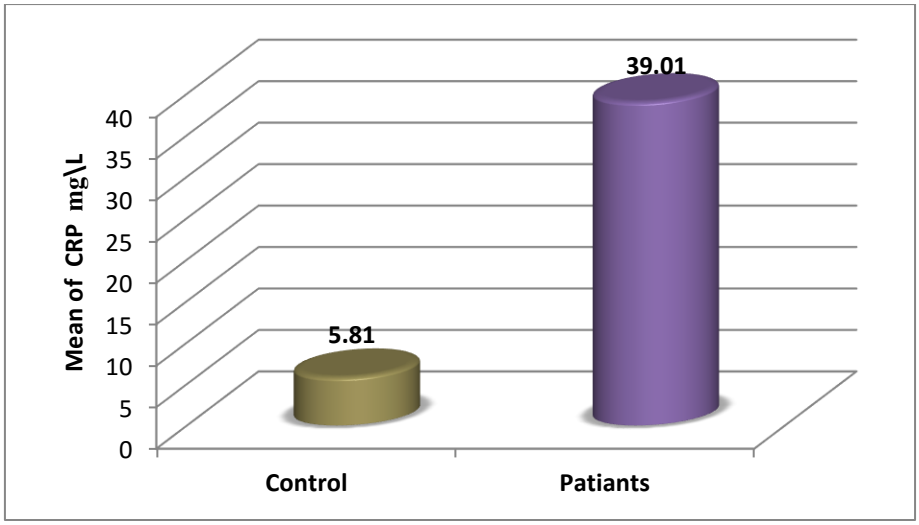


**Figure 2: CRP level in the group of patients and healthy people**

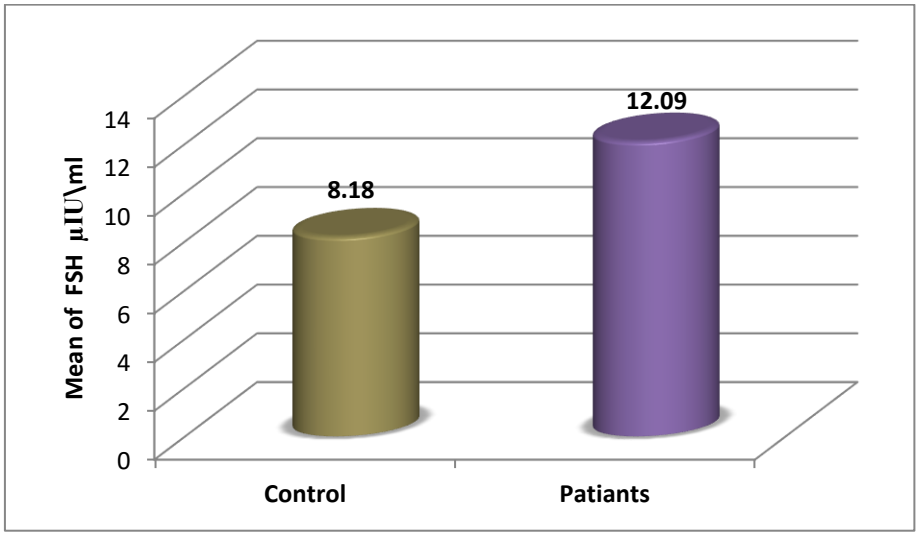


**Figure 3: FSH level in the group of patients and healthy people**

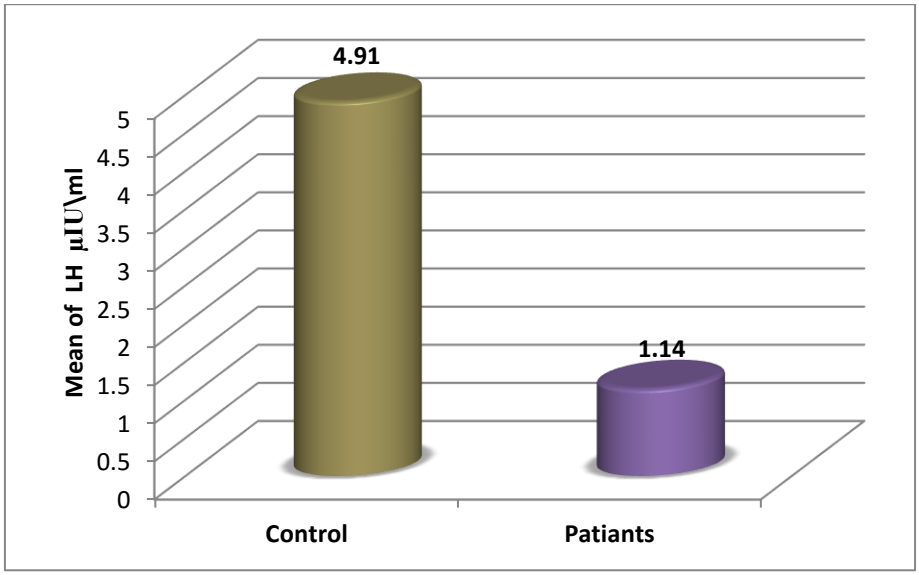


**Figure 4: LH level in the group of patients and healthy people**

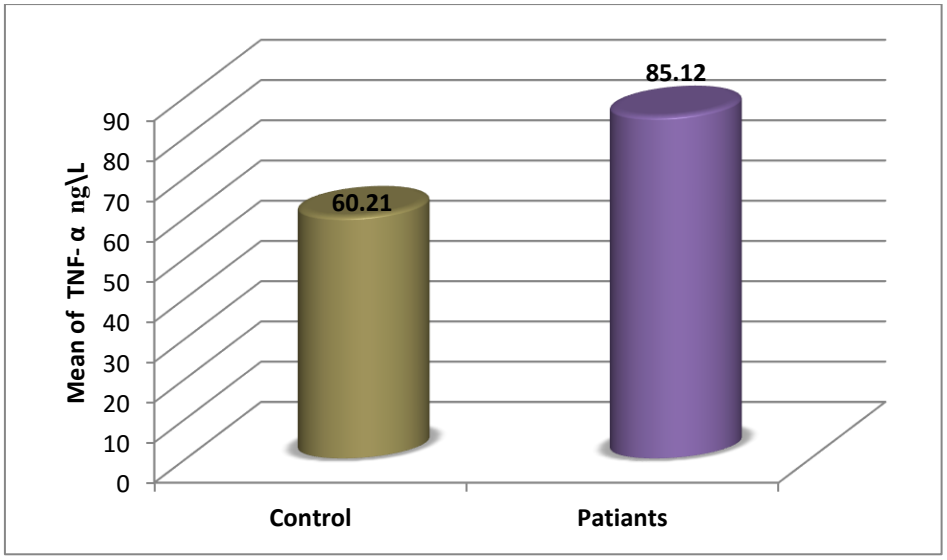


**Figure 5: E2 level in the group of patients and healthy people**

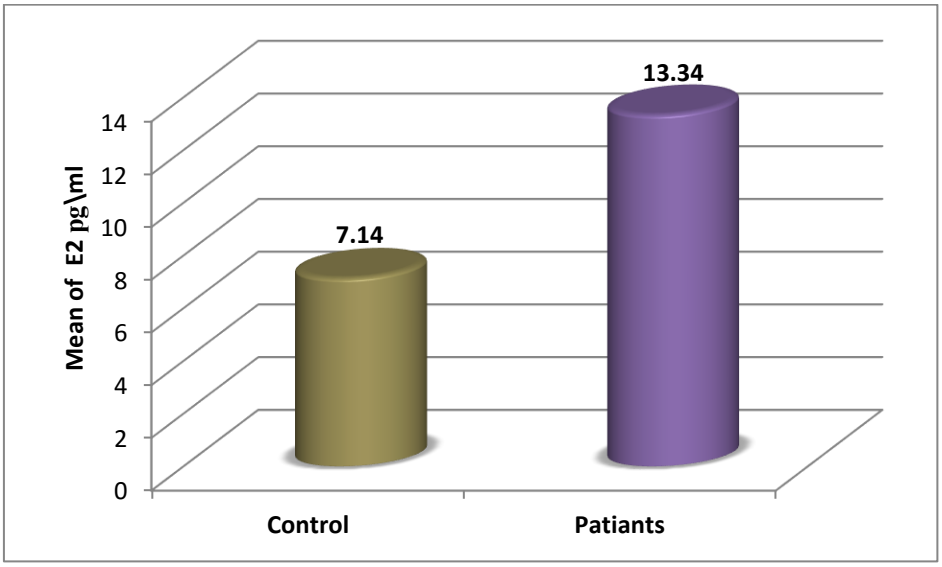


Figure 6: TNF-α level in the group of patients and healthy people

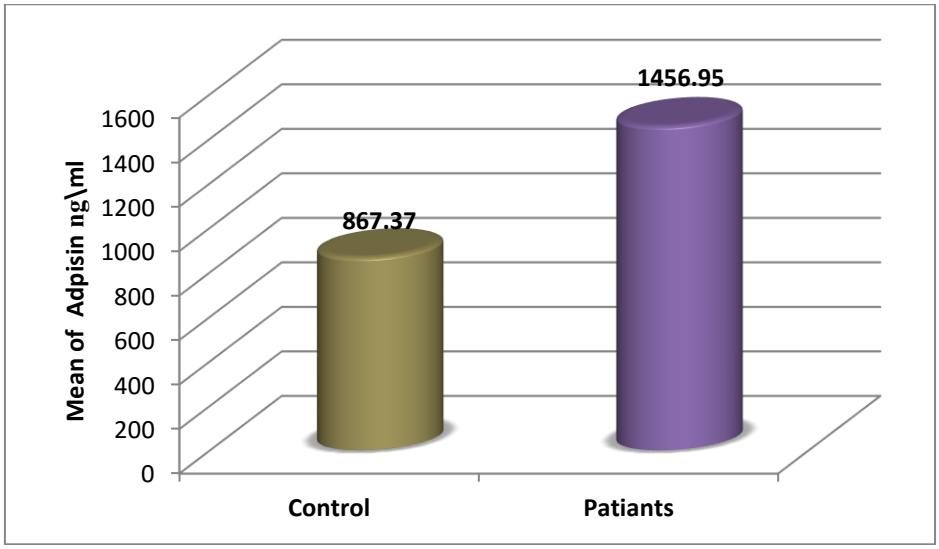


**Figure 7: Adpasin level in the group of patients and healthy people**

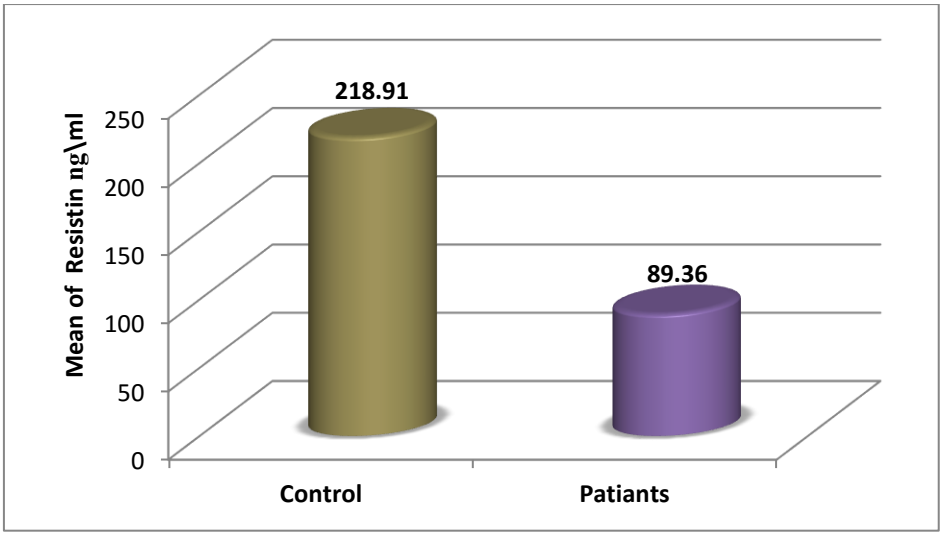


**Figure 8: Resistin level in the group of patients and healthy people**

## DISCUSSION

The etiology of endometriosis is closely related to metabolic factors, but it remains unknown whether the effect of endometriosis on assisted reproductive technology outcomes is partly due to abnormal metabolic markers. A recent study found that endometriosis increases the risk of diabetes, particularly gestational diabetes [21]. The results of the current study are consistent with the results [22], who showed in their research results that the insulin hormone was higher in obese patients suffering from endometriosis compared to the control group, and the reason for the increase was due to disorders associated with insulin resistance. In addition, endometriosis and type 1 diabetes have similar pathophysiological mechanisms, as both are involved in chronic inflammation resulting from immune dysfunction [23,24]. The inflammatory response in endometriosis patients can also affect glucose and lipid metabolism, which can be used as a screening method to aid in the diagnosis of endometriosis [25].

In women with endometriosis, insulin resistance affects both the body's metabolism and the mitotic pathways in insulin-targeted tissues (such as the liver, skeletal muscle, and adipose tissue) and non-traditional target tissues (such as the ovary and pituitary gland) [26]. In addition, hyperandrogenism, lipid deposition, inflammatory cytokines, and other systemic factors are also involved in the IR process of affected peripheral tissues [27].

The result of the increase in CRP was consistent with the result of [28] and [29], as the reason for the increase is due to the number of peritoneal macrophages in women suffering from endometriosis, as the endometrial tissue in the peritoneal cavity is recognized as a foreign entity that must be eliminated. Thus, cells such as retrograde endometrial tissue are killed by peritoneal macrophages and their presence leads to an inflammatory process [30]. A re-evaluation of the historical literature on CRP and endometriosis with a modern understanding of its different isoforms clearly indicates both a pro- and anti-inflammatory function as part of the acute immune response. Furthermore, these isoform-specific activities help explain the large differences in CRP expression, with similar phenotypes, between species [31,32].

The results of the current study of women with Endometriosis showed a significant decrease in the level of LH hormone, while the levels of FSH and E2 hormones increased in them compared to the control group. These results were consistent with the results of [33-35]. The reason for this difference in increase and decrease is believed to be due to changes in the levels of LH and FSH in the serum, peritoneal fluid, and follicular fluid of women with uterine fibroids. These changes cause a disruption in the feedback pathways and thus prevent the normal cyclical changes in the ovary from developing follicles that are less sensitive to luteinizing hormone. This causes ovulation dysfunction and weak embryo implantation, which are among the characteristics or symptoms of endometriosis [36]. Also, in the natural cycle of a fertile woman, the anterior lobe of the pituitary gland secretes nutrient hormones, which are the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH), to stimulate the growth of ovarian follicles. These follicles also provide positive and negative feedback to the pituitary gland, culminating in a surge of luteinizing hormone to trigger ovulation at the optimal time [37].

Testosterone is produced in the ovary mainly by theca cells under the influence of LH, so the low level of LH found in the present results may cause a decrease in testosterone level and the interaction between the theca cells and granulosa cells in the ovary is necessary during follicle development [38]. The granulosa cells then use the androgen produced by the theca cells to produce estrogen under the control of FSH, causing a decrease in

testosterone levels, and an increase in estrogen levels as a result of the increased FSH recorded in the present results [39].

Oxidative stress negatively affects ovarian cells surrounded by endometrial fibroids, as the ovarian tissue adjacent to the endometrial wall differs morphologically from normal ovarian tissue. The density of follicles near the ovarian cortex surrounded by ectopic uterine fibroids is much lower than that near the wall not surrounded by them. Therefore, exposure of the ovarian wall to damage causes oxidative stress Endometriosis-associated uterine fibroids damage the eggs by damaging the DNA of the theca cells, which are the source of androgen production in women, and thus lowering the level of LH [40]. In addition, the production of estradiol (E2) occurs in the follicle by the aromatase enzyme, which is regulated by the follicle-stimulating hormone (FSH) through a process called the two-cell model and the gonadal nutrient response Since the androgen produced by the two-gonadotropin model cells, the luteinizing hormone (LH), is metabolized by this enzyme to estrogen E2 in the granulosa cells, the reason for the high estrogen level in women with endometriosis may be the high level of follicle-stimulating hormone shown by the present results, through increased aromatase activity [41]. Endometrial tissue breakdown with menstruation is a severe inflammatory process in women with ectopic tissue (which creates the potential for widespread inflammation of endometrial cells at ectopic sites as seen in endometriosis which can be activated by elevated estradiol and decreased testosterone which stimulate the activity of inflammatory cytokines [42].

As for TNF-alpha, the results of the current study showed a significant increase in women with endometriosis compared to the healthy group. The results of the current study were consistent with the results of [43] and [44]. The reason for the increase is believed to be due to inflammation, as TNF-alpha is a pro-inflammatory cytokine produced mainly in macrophages and monocytes. It stimulates the immune response by activating a series of pathways for many cytokines and increasing vascular permeability. Thus, by stimulating and increasing macrophage and neutrophil cells at the site of infection, inflammation plays a major role in causing endometriosis [45]. This is done by changing the function of immune cells and increasing the levels of pro-inflammatory cytokines in the peritoneal cavity, endometrium, and blood. These immune changes promote the adhesion of ectopic tissues and the spread of endometrial cells [42]. In addition, macrophages distinguish the inflammatory state, as estrogen plays an important role in stimulating the thickening of the endometrium. This is an important natural process that occurs in all women, but as its level rises, the lining of the uterus grows densely to reach an abnormal size. This provides more tissue from which prostaglandin and other inflammatory markers such as TNF-a can be released Neutrophils and macrophages, cells involved in the primary immune response that induces inflammation, were identified as having higher activity in women with endometriosis. This is consistent with the results of the current study, which showed a significant increase in estrogen levels in women with endometriosis.

The result of the increase in the adipsin hormone in the group of patients with endometriosis was consistent with the result of [46], who showed in his study the possibility of relying on it as a diagnostic indicator that reflects the degree of oxidative stress associated with endometriosis. In addition to the known correlation between obesity, adipose tissue inflammation, and beta cell dysfunction, the molecular link between them has not yet been determined [47].

Adipokines are involved in maintaining a variety of processes such as appetite, satiety, energy expenditure, endothelial function, blood pressure, hemostasis, adipogenesis, insulin sensitivity, energy metabolism in insulin-sensitive tissues, fat distribution, and insulin secretion in pancreatic beta cells [48,49]. Functionally, adipsin plays a critical role in activating the complement system, a key component of innate immunity. This adipsin stimulates and amplifies the alternative complement pathway by inducing the cleavage of its substrate, factor B, to generate complement factor III. The absence of endothelial hyperplasia has been shown to cause inactivation of the alternative pathway [50,51].

The result of elevated resistin in the group of patients with endometriosis was consistent with the results of [52], who showed in their study that inflammation is a fundamental process in the formation and development of the endometrium. Resistin, one of the adipocytokines, has biological properties associated with immune functions. However, its role in the endometrium is unclear. Human resistin is among the inflammatory regulators that regulate inflammatory processes in macrophages and vascular cells. When stimulated with recombinant human resistin, macrophages produce interleukin-12 and interleukin-6 from a pathway mediated by Nuclear Factor Kappa B [53].

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