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ORIGINAL ARTICLE

Multifactorial Etiology of Uterine Fibroids: A Comparative Study on Endocrine Imbalance, Oxidative Stress Markers, and Vitamin D3 Status

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ABSTRACT

Uterine fibroids are the most common tumor among women of reproductive age, with genetic, inflammatory, hormonal, and other related factors playing a significant role in their development. A comparative study included 55 patients with uterine fibroids and 30 healthy women as a control group. These samples were collected in collaboration with obstetrics and gynecology consultants at hospitals in Diyala and Duhok, northern Iraq, as well as from outpatient clinics in both cities. The results showed significantly higher levels of testosterone and estradiol in patients with uterine fibroids (1.23 ± 0.03 and 13.23 ± 0.32) compared to the control group (0.37 ± 0.13 and 8.12 ± 0.49), while progesterone levels were lower in patients with fibroids (0.11±0.027) compared to healthy women (0.55±0.013). The results showed elevated follicle-stimulating hormone (FSH) and decreased luteinizing hormone (LH) and anti-Müllerian hormone (AMH) levels in patients (12.43, 4.92, and 1.47) compared to healthy controls (8.54, 7.43, and 4.39) respectively. The results also showed a significant increase in the oxidative stress index (MDA) and leptin levels in patients (16.23 ng/mL, and 18.48 nmol/L) compared to healthy controls (10.49ng/mL and 15.23 nmol/L) respectively. Regarding thyroid hormones, the results indicated elevated T3 levels (3.21 vs. 1.4 nmol/L) and decreased T4 (57.83 vs. 154) and thyroid-stimulating hormone (TSH) levels (1.65 vs. 3.2). Vitamin D levels were also significantly lower in the fibroid group 15 ng/mL compared to 55 ng/mL healthy controls. These findings suggest that uterine fibroids are a systemic metabolic-endocrine disorder. Therefore, their management should be approached within the context of a broader interaction between malnutrition, systemic oxidative stress, and hormonal imbalances as contributing factors to the disease.

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1- INTRODUCTION

The high prevalence of uterine fibroids and their strong association with estradiol and progesterone has been well documented [1]. An imbalance in the hormones is known to support cell growth and tumor development since these hormones play a key role in the proliferation of leiomyoma cells [2, 3]. Women with miscarriages and fibroids generally have lower pre-ovulatory estradiol levels and greater progesterone deficiency compared to those with preserved fertility [4]. It is believed that estrogen dominance accompanied by weak progesterone leads to

hyperplasia and apoptosis inhibition, a process observed in the pathogenesis of some endometrial cancers [5]. Other studies have also proposed that some surgical therapies, for example laparoscopic myomectomy, may modify the levels of some sex hormones in women with uterine fibroids. Despite the fairly extensive information available regarding the relationship between uterine fibroids and sex hormones, this bond is intricate and not well defined [6, 7]. Many factors influence the concentration of hormones, including the stage of menstrual cycle, presence of other medical conditions (e.g., PCOS), and extrinsic or environmental factors. For instance, it is known that some compounds (e.g., per- and polyfluoroalkyl compounds (PFAS)) can change sex hormones in pregnant women [8, 9]. These compounds may change the levels of progesterone, testosterone, and estradiol levels differently depending on fetal sex (female or male) [10], the increased incidence of fibroids in women is thought to be related to hormonal and vitamin imbalances, as well as abnormalities in the FSH/LH ratio and ovarian response [11], high levels of FSH and LH are found in most women with PCOS [12], AMH hormone levels show a similar association with secondary amenorrhea. Studies have also investigated leptin hormone levels in women with uterine fibroids, finding them to be elevated in those with unexplained infertility. Malonaldehyde (MDA) is a key indicator of oxidative stress, a factor associated with a wide range of reproductive problems and fibroid development [13].

Thyroid hormones namely T3, T4 and TSH play a crucial role in many physiological processes including reproduction. A Chinese study showed a relationship between thyroid nodules, functioning thyroid and uterine fibroids. Women with chronic uterine fibroids were reported to have significantly higher TSH, T3 and T4 levels than healthy women in the young reproduction group, which raises a possible link between thyroid function and reproductive disorders [14,15]. In addition to thyroid hormones, vitamin D3 has been extensively studied as a potential risk factor and therapeutic agent for uterine fibroids. Blood levels of vitamin D that are not sufficient have been associated with a higher risk of fibroids in several studies. Similarly, it has been indicated to improve DNA damage in human uterine fibroids [16]. The objective of this study is to evaluate the relationship between uterine fibroids with sex hormones, thyroid hormones and oxidative stress through measuring MDA level and vitamin D3.

2- MATERIALS AND METHODS

2.1 Study Participants

The case-control study included 30 serum samples taken from fertile women aged 20-40 years with a history of childbirth and no gynecological conditions such as fibroids or ovarian cysts, weighing between 65 and 88 kg (as control group). In addition, 55 serum samples were taken from women diagnosed with uterine fibroids, aged 22-40 years, who had given birth and weighed between 65 and 80 kg. These samples were collected in collaboration with obstetrics and gynecology consultants at hospitals in Diyala and Duhok, northern Iraq, as well as from outpatient clinics in both cities.

2.2 Collection of samples

Blood samples (4–5 mL) were obtained from the antecubital vein through gel tubes during the menstrual cycle. Serum was separated from the coagulated blood by centrifuging at 4000 rpm for 15 minutes. Serum was divided into two Eppendorf tubes and remained frozen at −20°C until analyzed. People with control or patients were chosen from the same ages (20–40 years) and weights so that comparisons between groups could be made within homogeneous ranges.

2.3 Measurement of Sex Hormones

Serum levels of FSH, LH, AMH, Testosterone, Estrogen, Progesterone, Leptin and MDA were measured by ELISA technique. Analyses were performed with Biotek ELISA Reader (USA) according to standard protocols and were carried out on commercial reagent kits from BT Laboratory; Accusing (All Reagents, Shijiazhuang, Hebei Province, China) [17].

2.4 Leptin Measurement

Serum leptin concentration was measured using ELISA (bt laboratory, China). The sandwich ELISA protocol was used to quantify serum leptin (LEP). This technique relies primarily on specific antibodies pre-fixed to the wells of ELISA plates to capture leptin antigens present in the samples. The color was collected by adding the substrate solution to the wells after incubation with biotin-binding secondary antibodies and horseradish streptavidin-peroxidase (HRP) conjugates. The results were read at a wavelength of 450 nm using an ELISA reader; the color intensity was directly proportional to the leptin concentration in each sample [18].

2.5 Malondialdehyde (MDA) Level Estimation

The concentration of malondialdehyde (MDA) in patient and healthy samples was measured using sandwich ELISA technology according to the manufacturer's protocol. The absorbance in each well was measured at a wavelength of 450 nm [19].

2.6 Measurement of Thyroid Hormones and Vitamin D3

Thyroid hormone levels (T3, T4, and TSH) and vitamin D3 in serum were measured using an enzyme-linked immunosorbent assay (ELISA), according to the manufacturer's protocol) Mitr /Italy) [20].

2.7 Statistical Analysis

Data were analyzed using SPSS software (Version 26.0). Means ±SE were used to express descriptive statistics. The Shapiro-Wilk test was performed to check the normality of data distribution. An independent samples t-test was performed to compare between patients and control group. Biochemical and hormonal parameters were analyzed using ANOVA followed by Tukey's Honestly Significant Difference (HSD) post-hoc test for pair-wise comparisons. Statistical significance was defined as $P \leq 0.05$ and the difference at a level of high significance when $P \leq 0.01$.

3- RESULTS AND DISCUSSION

Table 1 shows hormone levels associated with uterine fibroids (Testosterone, Estradiol, and Progesterone) in women with uterine fibroids syndrome compared to healthy women. The results showed that testosterone levels were significantly higher ($p < 0.01$) in women with uterine fibroids, reaching 1.23 ng/dL compared to 0.37 ng/dL in healthy women. Similarly, estradiol levels were elevated in women with the condition, reaching 13.23 pg/mL compared to 8.12 pg/mL in healthy women. In contrast, the results showed a significant decrease in progesterone concentrations in women with fibroids, reaching 0.11 ng/mL compared to 0.55 ng/mL in healthy women.

Table (1): Levels of Certain Sex Hormones in Women with Uterine Fibroids Syndrome (n=55) and the Healthy Control Group (n=30 (Mean ± SE)

Groups	Progesterone (ng/ml)	Estradiol (pg/ml)	Testosterone (ng/dl)
Healthy	0.55±0.013a	8.12±0.496b	0.37±0.13a
Disease-infected	0.11±0.027b	±0.32a13,23	±0.031b1,23
LSD%	0,15	3,43	0,0021

Hormone levels, such as FSH, were significantly higher in the patient compared healthy group ($P<0.01$), while LH levels were lower than in patient women. Furthermore, AMH analysis revealed a marked decrease in the blood of the affected group compared to the control group (Figure 1). AMH is a good indicator of ovarian reserve (the number of remaining eggs). Therefore, the markedly lower AMH levels in this group confirm a reduced ovarian reserve compared to healthy women.

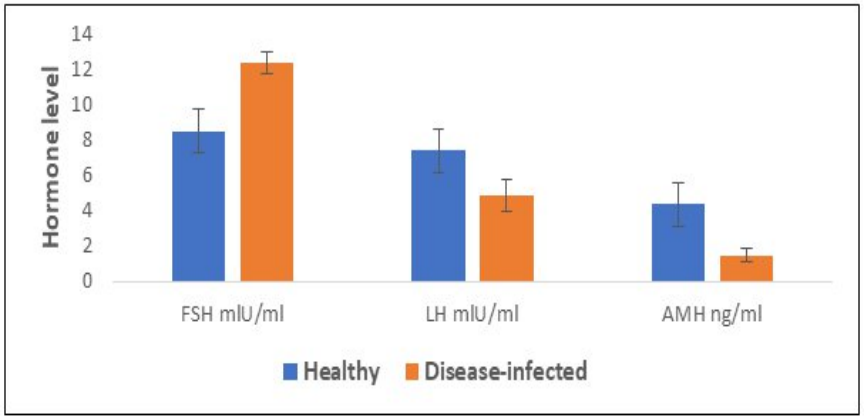

Fig (1): Illustrate the levels of sex hormones FSH, LH, and AMH in women diagnosed with uterine

3.1 Leptin and MDA Levels

Figure 2 shows leptin and MDA levels in women with uterine fibroids compared to a healthy control group. The mean serum leptin level in women with fibroids was 16.231 ng/mL, while in healthy women it was 10.490 ng/mL ($p<0.0001$). Furthermore, the MDA level was 18.48 ng/mL in the fibroid group, significantly higher than in the healthy control group (15.23 ng/mL).

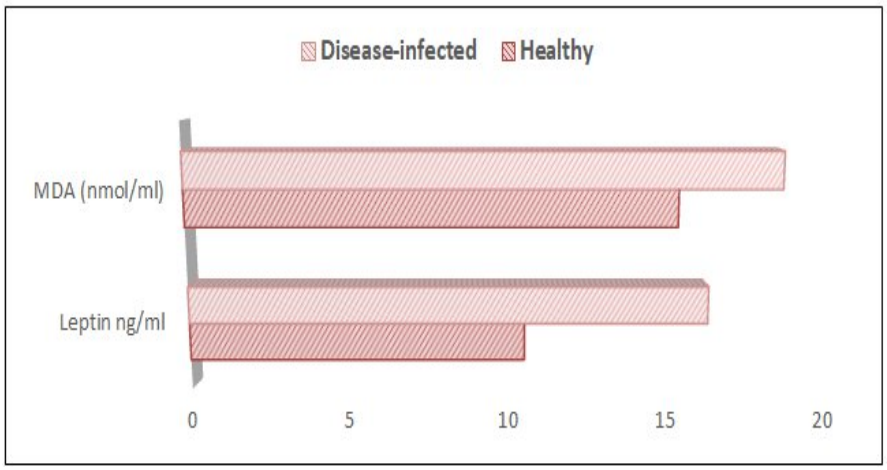

Fig (2): Levels Leptin hormone (ng/ml) and MDA level (nmol/L)

3.2 Thyroid Hormone and Vitamin D3 Levels

Table 2, shows the thyroid hormone levels (T3, T4, TSH) between women with uterine fibroids and healthy women. The results showed that women with uterine fibroids had higher levels of T3 (3.21±0.015) compared to healthy (1.4±0.01). T4 levels differed significantly between the fibroid group and the healthy control group, with an average level of 57.83 nmol/L in the fibroid group compared to 154.22 nmol/L in the healthy control group. Additionally, TSH (thyroid-stimulating hormone) levels were lower in the fibroid group (1.65 IU/L) compared to the healthy control group (3.2 IU/L). These results indicate elevated T3 with decreased T4, suggesting a more complex regulatory dysfunction.

Table (2): Thyroid Hormones and Vitamin D3 Levels in Women with Uterine Fibroids

Groups	D3 (ng/ml)	TSH	T4	T3
Healthy	55±0.042	3,2±0.	154,22±0.06	1,4±0.01
Disease-infected	15±0.011	1,65±0.082	57,83±0.022	3,21±0.015

The current study showed a significant decrease in vitamin D3 levels (15 ng/ml in fibroids patients and 55 ng/ml in healthy women). Normal values are considered to be >20 ng/ml, and levels below this threshold are associated with fibroids. Androgens (specifically testosterone) are thought to play a role in the development of uterine fibroids [21]. Sex-linked steroid hormones: Most reports to date indicate that estrogen is the predominant signaling hormone responsible for uterine fibroid growth. This pathological process may occur either via androgen receptor pathways or through the local conversion of androgens to estrogens within the fibroid structure, supporting recent observations that steroid hormones contribute to fibroid development [22]. Estradiol is the most relevant natural estrogen, and the biological actions of estradiol are carried out in human body through stimulation of fibroid cell proliferation with at the same time inhibition of apoptosis signaling pathways which contributes to an increment in tumor volume. These increased estradiol levels seen in women with fibroids further support earlier observations that local hyperestrogenism (absolute or relative to progesterone) contributes to the maintenance of these lesions. These hormonal changes may result from persistently increased systemic secretion, altered local metabolism in the endometrial tissues or elevated expression of estrogen receptors within the lesions.

Recent studies indicate that progesterone is a key factor in promoting the growth of fibroids. This study observed lower blood progesterone levels in the fibroid group compared to healthy individuals [23]. Estrogen in permissive

amounts suppresses the mutagenic effects of progesterone while acting independently of a relative deficiency of progesterone. Aberrant progesterone receptor signaling may contribute; fibroid cells typically exhibit altered localization of steroid receptors or altered downstream pathways resulting in hypersensitivity to low levels of hormone and/or loss of normal function of the steroid [24].
The results of this study currently support targeted hormonal therapies, including gonadotropin-releasing hormone (GnRH) agonists/inhibitors (to suppress sex hormones), selective progesterone receptor modulators (SRRMs), and aromatase inhibitors, for the treatment of uterine fibroids. The results show a marked difference in hormonal composition (along with other pathological conditions) in women with uterine fibroids compared to healthy women. This suggests reduced ovarian function. In this case, FSH secretion from the pituitary gland increases to stimulate follicle growth if the ovaries are more resistant to it. Based on this mechanism, we hypothesize that women with fibroids either have a reduced ovarian reserve or an innate predisposition to premature reproductive aging compared to healthy women. The finding could be due to mechanical compression of the fibromas or effects on adjacent ovarian tissue in terms of vasculature, or it may reflect common systemic and genetic factors that simultaneously trigger both follicle depletion and the expansion of fibroids [25,26].

Fibroid effects on the ovary may also be mechanical or vascular in nature, which prevents blood flow and alters the local hormonal milieu that hinders normal functioning of the ovary. Potential common molecular biological mechanisms ranging from inflammation to oxidative stress or common growth factors through which the progression of fibroids relates with diminished ovarian reserve may exist [27,28]. A decrease in luteinizing hormone (LH) levels may indicate a change in hormonal feedback on the hypothalamic-pituitary-adrenal (HPA) axis. This decrease may create a hormonal environment unfavorable to normal ovarian function. Alternatively, it may simply reflect a general hormonal pattern of ovarian aging or the presence of fibroids. Taken together, these indicators suggest that uterine fibroids may be associated with decreased fertility or accelerated aging of the gonads. This is accompanied by elevated FSH and decreased AMH, indicating an alteration in both the ovarian environment and function [29]. The thyroid pattern of the present investigation (high T3, low T4 and low TSH) can represent a compensatory increased conversion of the prohormone T 4 to active hormone in peripheral tissues; however, this abnormal thyroid hormone pattern is not pathologically dissimilar from that described as "non-thyroid syndrome" or NTIS as it reflects an adaptive metabolic shift to less efficient thyroid hormone metabolism to chronic stress.
The current study indicated that women with uterine fibroids have higher levels of leptin and MDA, evidence of significant metabolic disturbances and oxidative stress. These data support our previous findings regarding a favorable environment in the uterus for fibroid growth [7] and are consistent with other studies we have conducted on hormone levels and ovarian reserve indices [8]. These mechanisms, taken together, can help us develop more effective preventive and therapeutic strategies against uterine fibroids [30, 31]. The primary roles of vitamin D include modulating immune responses, sustaining bone integrity, and preserving mineral homeostasis [32]. The findings of the current study are consistent with those of Ivanova *et al*. [33] and Ashkar *et al*. The results of this study support the association between low vitamin D3 levels and elevated serum oxidative stress index (MDA) levels [14] in women with uterine fibroids. In contrast, the typical presentation of hyperthyroidism is low TSH levels due to negative feedback mechanisms in the pituitary gland (which likely corresponds to low T4 and high T3 levels) [34, 35]. The strong association between uterine fibroids and the deficiency of metabolites of vitamin D is probably one of more profound influences on the etiology and development of these tumors than other molecular mechanisms or biomarkers.

4- CONCLUSION

These results substantiate the notion that uterine fibroids are a systemic metabolic endocrine disorder rather than a localized gynecological disease. The hormone disorders found (increased estrogen, decreased progesterone and increased androgen) cannot be separated from systemic changes like increased MDA and high leptin. The results also revealed elevated T3 and decreased T4 and TSH, the latter being a new marker in the pathophysiology of uterine fibroid. The exact stratification of vitamin D deficiency is an urgent clinical problem because, as a modifiable risk factor, it requires immediate attention. In conclusion, these observations require a basic re-evaluation of the design of early treatment using an integrative, multifaceted treatment strategy that addresses circulatory and tissue endocrine interactions, systemic oxidative stress and micronutrient deficiency. These findings reinforce the need for large-scale longitudinal studies to clarify the proximal mechanism of this complex disease, followed by focused, multicenter clinical trials.

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