Association Between Elevated CA15-3 Levels and Sex Hormonal Profile in Women With Breast Cancer: A Case-Control Study in Basrah, Iraq

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ABSTRACT

Breast cancer is among the most common malignant neoplasms affecting women, and a combination of internal and external factors drives its development. The study aims to assess the associations between serum CA15-3 levels and the sex hormonal profile (Estradiol, Progesterone, and Luteinizing Hormone) in a cohort of Iraqi women with breast cancer who are undergoing systemic therapy, and to evaluate the influence of demographic and treatment-related factors on these associations. The investigation was conducted at the Oncology Department of Sadr Teaching Hospital and Zubair General Hospital in Basra Governorate. The study sample consisted of 93 participants, comprising 53 breast cancer patients (case group) and 40 healthy individuals (control group). Serum concentrations of the following parameters were measured: Cancer Antigen 15-3 (CA15-3), Luteinizing Hormone (LH), Estradiol, and Progesterone using Electrochemiluminescence Immunoassay technology on a Cobas e411 analyzer. Researchers used SPSS version 25 for data analysis, applying the Mann-Whitney U test and Spearman's correlation coefficient. The results revealed statistically significant differences in these biochemical variables between the patient and healthy groups. This study demonstrates a significant dysregulation of the sex hormonal profile with elevated LH and diminished estradiol and progesterone in Iraqi breast cancer patients undergoing systemic therapy. The significant positive correlation between CA15-3 and LH introduces a compelling potential interplay between tumor marker and hormone activity.

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1. Introduction

One of the frequent and numerous malignant tumors that affects women is breast cancer. Breast cancer develops and occurs as a result of several internal and external factors [1,2]. Poor lifestyle choices, environmental factors, and social-psychological factors are all linked to its occurrence. It has been demonstrated that 5% to 10% of breast cancers can be attributed to genetic mutations and family history, and 20% to 30% of breast cancers can be attributed to factors that may be modifiable [3,4]. Breast cells are where breast cancer first develops. A collection of cancer cells known as a cancerous tumor is capable of spreading into and destroying nearby tissue. As well as spreading throughout the body, it can. Breast cells occasionally undergo changes that prevent them from growing or behaving normally. Non-cancerous breast conditions, such as atypical hyperplasia and cysts, may result from these changes. Additionally, they may result in benign tumors ,such as intraductal papilloma [5]. According to the Iraqi National Cancer Research Center (INCRC), breast cancer is the most common malignancy among Iraqi women, constituting nearly one-third of all registered female cancers in the country [6]. Surveillance data indicate a concerning rise in the national cancer incidence rate in Iraq from 52 per 100,000 in 2000 to 91.66 per 100,000 in 2019 (Iraqi Cancer Board, 2019). Notably, breast cancer remains the primary cause of cancer-related mortality among Iraqi women, ranking first among all cancers in 2019 with 34.08% of reported cases and a mortality rate of 6.22 per 100,000 [7]. This significant and persistent national burden is starkly reflected in recent international estimates; according to 2022 data from the Global Cancer Observatory (GLOBOCAN), breast cancer constituted approximately one-third (34.1%) of all new cancer cases diagnosed among Iraqi women, accounting for 7,260 new cases annually, thereby solidifying its position as the most prevalent malignancy in this population [8]. In response to this substantial public health challenge, the World Health Organization (WHO) emphasizes early detection strategies to enhance survival outcomes, while also acknowledging the efficacy of pharmacological agents in preventing the disease among high-risk women [7]Epidemiological studies have identified several risk factors for breast cancer, including age, family history, genetic predisposition, age at menarche, parity, duration of lactation, age at menopause, dietary habits, and hormonal levels [9].

CA 15-3 is a tumor marker widely used in the evaluation of breast cancer. It is a glycoprotein secreted by various cell types, with markedly increased production in malignant breast cells. Once released, CA 15-3 enters the bloodstream, allowing for its detection and quantification [10]. Clinically, CA 15-3 is primarily employed as a biomarker for monitoring treatment response and detecting disease recurrence in breast cancer patients. However, its utility in early-stage diagnosis is limited due to insufficient sensitivity during the initial phases of the disease [11,12]. From a hormonal perspective, progesterone and estradiol act through similar receptors and play a fundamental role in breast development, particularly during puberty, pregnancy, and lactation. Recent studies suggest that hormonal stimulation can induce genomic alterations in breast tissue. Elevated progesterone levels during pregnancy have been shown to enhance histone methylation and suppress the expression of tumor suppressor genes [13,14]. Prolonged exposure to

progesterone may therefore disrupt the balance of chromatin methylation and gene regulation, contributing to carcinogenesis. Similarly, variations in circulating estradiol levels have been reported to influence the expression of estrogen-responsive genes in breast tissue at the genomic level [15,16]. The findings revealed that elevated serum CA15-3 is strongly associated with estrogen receptor (ER) status, both in ER+/PR+ and ER+/PR- cases. This highlights its clinical value in advanced stages and higher tumor grades of breast cancer, as well as its potential role in guiding therapeutic decisions. Moreover, a significant association was observed between oral contraceptive or hormonal therapy use and breast cancer, further emphasizing the interplay between CA15-3 and sex hormones in disease progression and treatment planning[17].

In addition, the luteinizing hormone (LH) has been implicated in promoting the invasiveness of breast cancer cells. LH secretion is regulated by gonadotropin-releasing hormone (GnRH), which is released in pulses in response to circulating estrogen and progesterone through a negative feedback mechanism [18,19]. While the role of gonadotropins in ovarian physiology has been well established, more recent evidence indicates that LH receptors (LHRs) are also expressed in extra-gonadal tissues, including both normal and malignant breast tissue [20]. Aggressive tumors demonstrate significantly elevated LH and LHR expression, suggesting that LH overexpression may be a key driver of breast cancer development [21]. Furthermore, experimental studies in animal models have shown that circulating gonadotropin levels are directly associated with tumor progression and that LH regulates the expression of multiple genes involved in the carcinogenicity of breast cancer cells [22]. Serum CA15-3 levels exhibit significant menstrual cycle–dependent variability, with higher concentrations observed during the mid-luteal phase, coinciding with luteinizing hormone (LH) surge and subsequent hormonal changes. This suggests a potential modulatory effect of LH on CA15-3 levels, which should be considered when interpreting this tumor marker, particularly in early-stage breast cancer[23]. Despite the established role of CA15-3 in monitoring breast cancer, its relationship with sex hormones, particularly in Iraqi women undergoing systemic therapy, remains poorly understood. This study aims to fill this gap by evaluating the association between CA15-3 and hormonal profiles in a unique population exposed to distinct genetic and environmental factors.

Aim of the Study

This study aims to assess the associations between serum CA15-3 levels and the sex hormonal profile (Estradiol, Progesterone, and Luteinizing Hormone) in a cohort of Iraqi women with breast cancer who are undergoing systemic therapy, and to evaluate the influence of demographic and treatment-related factors on these associations.

2. Materials and methods

2.1 Study Design

A case-control study was conducted at the Oncology Departments of Sadr Teaching Hospital and Al-Zubair General Hospital in Basrah, Iraq, to investigate potential associations between breast cancer and specific biomarkers. The study population comprised 93 participants, including 53 patients diagnosed with breast cancer (case group) and 40 age- and sex-matched healthy

individuals (control group). Eligible patients were those with a newly confirmed diagnosis of stage II breast cancer who were scheduled to begin or were currently undergoing systemic therapy (chemotherapy or hormonal therapy). Breast cancer patients were recruited during routine follow-up visits or clinical consultations at Sadr Teaching Hospital. Peripheral blood samples were collected from all participants after obtaining written informed consent. The control group was randomly selected from the general population. It included individuals with no prior history of malignancy or any significant medical conditions that could influence biomarker expression or genetic polymorphisms. Matching between cases and controls was performed to minimize potential confounding factors, including age, sex, ethnicity, and other relevant clinical characteristics. The study protocol was reviewed and approved by the Ethics Committee of the Basra Health Directorate on June 11, 2024 (Approval No. 705). All procedures were conducted in strict accordance with the ethical principles of the Declaration of Helsinki.

2.2 Criteria of exclusion

Participants with thyroid disorders, cardiovascular diseases, or renal impairments were excluded from the study. Additionally, individuals under 20 years of age or over 60 years of age were not considered eligible. Pregnant women, as well as those with polycystic disease or other chronic illnesses, were also excluded from the control group. A structured questionnaire was administered to both patients and controls in the hospital during the morning hours, collecting demographic data such as age, place of residence, duration of illness, type of therapy received, and family history.

2.3 Laboratory Tests

Serum concentrations of CA15-3 (Elecsys, REF 03045838122), luteinizing hormone (LH; Elecsys, REF 11732234122), estradiol (Elecsys, REF 06656021190), and progesterone (Elecsys, REF 07092539190) were measured using the fully automated COBAS E411 analyzer.

2.4 Statistical analysis:

For statistical analysis, SPSS version 25 (IBM, Armonk, NY) was used. The findings were extracted using descriptive statistics like mean and standard deviation (SD), and the differences between the quantitative data of the two parameter groups were tested using techniques like the Mann-Whitney test. We used the Spearman correlation coefficient to get the correlation coefficient (r value). P-values below 0.05 were regarded as significant.

3. Results and discussion

As shown in Table 1, the biochemical profiles of the participants revealed highly statistically significant differences between the breast cancer cohort and the healthy controls with respect to serum CA15-3, luteinizing hormone (LH), estradiol (E2), and progesterone.

To assess the impact of age on serum biomarkers, participants were stratified into two age groups (Table 2). The first group included individuals aged ≤44 years, comprising 12 patients and 23 control subjects, while the second group consisted of females aged ≥45 years, including 41 patients and 17 controls. In the younger group, significant differences were observed in CA15-3 ($P = 0.024$), estradiol (E2, $P < 0.001$), and progesterone ($P = 0.004$), whereas luteinizing hormone (LH) showed no significant difference. In the older group, significant differences were detected in CA15-3 ($P = 0.001$), LH ($P < 0.001$), and E2 ($P = 0.002$), while progesterone levels did not differ significantly between patients and controls.

Table -1. The Differences in the Levels of CA15-3 and Hormones between Patients and the Control Group

Parameters	Patients No.=53		Control No.=40		P value
	Mean±SD	Min-Max	Mean±SD	Min-Max	
CA153 (U/mL)	26.40±13.750	5.0-83.0	16.73±5.41	6.0-29.0	0.000*
LH m.IU/ml	30.38±18.69	0.28-74	6.60±4.73	0.19-19.73	0.000*
E2 (pg/mL)	11.36±10.50	5-57.04	61.42±42.17	5-170.20	0.000*
Progesterone (ng/mL)	0.22±0.15	0.05-0.56	3.12±4.06	0.05-13.77	0.001*

Mann-Whitney test, significant ($p < 0.05$), highly significant* ($p \leq 0.001$), very highly significant ($p \leq 0.0001$)

Table 2: Distribution of biochemical markers in the Breast Cancer patient group and control group according to Age.

Parameters	Patient ≤44(Years) Mean ± SD No. (12)	Control≤44(Years) Mean ± SD No. (23)	P. value	Patient≥45(Years) Mean ± SD No. (41)	Control≥45(Years) Mean ± SD No. (17)	P. value
CA153 (U/mL)	23.07± 10.07	16.71± 5.81	0.024*	27.37± 14.61	16.74± 5.23	0.001*
LH m.IU/ml	21.66± 24.27	6.04± 4.37	0.07	32.94± 16.20	7.37± 5.22	0.000*
E2 (pg/mL)	17.09± 16.95	73.52± 39.73	0.000*	9.69± 7.19	45.05± 40.85	0.002*
Progesterone (ng/mL)	0. 23±0.15	4.28±4.13	0.004*	0.21±0.15	1.56±3.50	0.442

Mann-Whitney test, significant ($p < 0.05$), highly significant* ($p \leq 0.001$), very highly significant ($p \leq 0.0001$)

As presented in Table 3, patients were stratified into two groups based on their place of residence: the city center group and the extremity group. No significant differences were observed in any of the measured serum variables between the two groups.

Table -3: Distribution of biochemical markers in the Breast Cancer patient according to Address.

Parameters	The Center Mean ± SD No. (27)	Extremities Mean ± SD No. (26)	P. value
CA153 (U/mL)	25.08±11.45	27.76±15.90	0.776
LH m.IU/ml	35.20±18.57	25.38±17.80	0.086
E2 (pg/mL)	8.92±5.50	13.91±13.60	0.403
Progesterone (ng/mL)	0.26±0.16	0.17±0.12	0.071

Mann-Whitney test, significant ($p < 0.05$), highly significant ($p \leq 0.001$), very highly significant ($p \leq 0.0001$)

According to Table 4, patients were classified into two groups based on the type of treatment administered: hormone therapy and chemotherapy. A significant difference was identified in luteinizing hormone (LH, P = 0.025), whereas the other serum biomarkers showed no statistically significant variations.

Table -4: Distribution of biochemical markers in the Breast Cancer patient according to the Type of Treatment.

Parameters	Chemical Mean ± SD	Hormonal Mean ± SD	P. value
CA153 (U/mL)	27.32±13.64	26.08±14.14	0.565
LH m.IU/ml	40.80±19.09	26.87±16.90	0.025*
E2 (pg/mL)	13.77±12.68	10.56±9.63	0.842
Progesterone (ng/mL)	0.18±0.11	0.23±0.16	0.390

Mann-Whitney test, significant* ($p < 0.05$), highly significant ($p \leq 0.001$), very highly significant ($p \leq 0.0001$)

As shown in Table 5, patients were stratified into two groups based on the duration of their illness: those with a disease duration of less than five years and those with a duration of five years or more. No statistically significant differences were observed in any of the measured serum variables between the two groups.

Table -5 : Distribution of biochemical markers in the Breast Cancer patient according to Duration of Illness.

Parameters	Less than 5 years Mean ± SD	More than 5years Mean ± SD	P. value
CA153 (U/mL)	24.32 ±8.86	30.43±19.88	0.475
LH m.IU/ml	28.12 ±16.53	34.75 ±22.17	0.388
E2 (pg/mL)	11.95 ±9.71	10.22 ±12.12	0.281
Progesterone (ng/mL)	0.23 ±0.15	0.19 ±0.14	0.329

Mann-Whitney test, significant ($p < 0.05$), highly significant ($p \leq 0.001$), very highly significant ($p \leq 0.0001$)

Spearman's correlation analysis revealed significant associations between serum biomarkers in breast cancer patients (Table 6). CA15-3 levels demonstrated a significant positive correlation with luteinizing hormone (LH) ($r = 0.333$, $p = 0.001$). Conversely, CA15-3 levels were significantly inversely correlated with both estradiol (E2) ($r = -0.351$, $p = 0.001$) and progesterone ($r = -0.230$, $p = 0.027$). Furthermore, LH showed significant negative correlations with E2 ($r = -0.411$, $p < 0.001$) and progesterone ($r = -0.288$, $p = 0.005$). A strong, significant positive correlation was observed between E2 and progesterone levels ($r = 0.541$, $p < 0.001$).

Table -6: The Correlations Between CA15-3 and Hormone Variables in Patients

Correlations	r	P. value
CA15-3 vs. LH	**0.333****	**0.001**
CA15-3 vs. E2	**-0.351****	**0.001**
CA15-3 vs. Progesterone	**-0.230***	**0.027**
LH vs. E2	**-0.411****	**0.000**
LH vs. Progesterone	**-0.288****	**0.005**
E2 vs. Progesterone	**0.541****	**0.000**

Spearman's correlation test, r = strength of correlation or correlation coefficient, (-) inverse correlation, (+) proportional correlation. Significant ($*p < 0.05$), highly significant ($**p \leq 0.001$), very highly significant ($***p \leq 0.0001$).

Figures 1 and 6 illustrate positive correlations among the biochemical variables. In contrast, Figures 2, 3, 4, and 5 demonstrate negative correlations among the same biochemical variables.

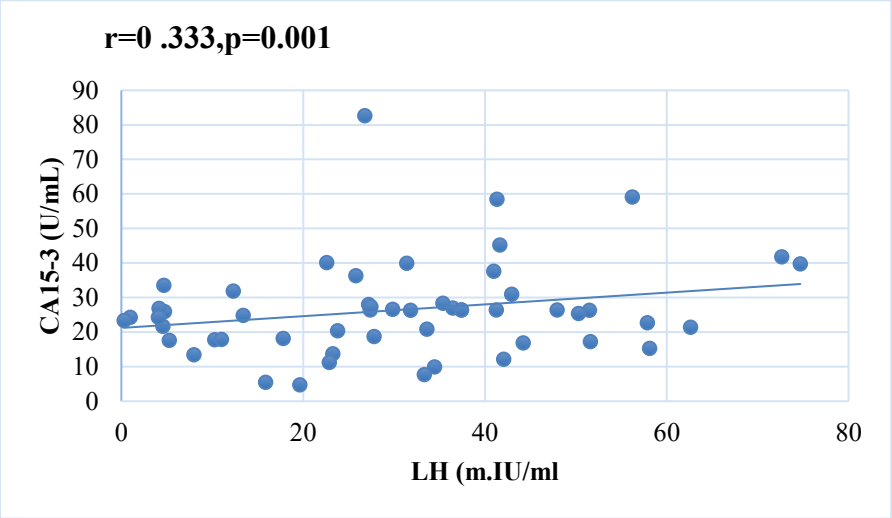

Fig. 1. Correlation between Ca15-3 and LH in Breast Cancer patients

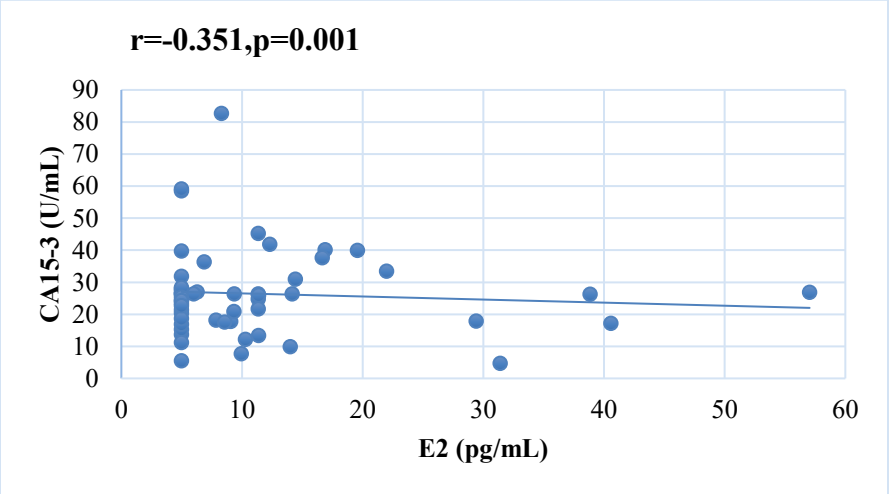

Fig. 2. Correlation between Ca15-3 and E2 in Breast Cancer patients

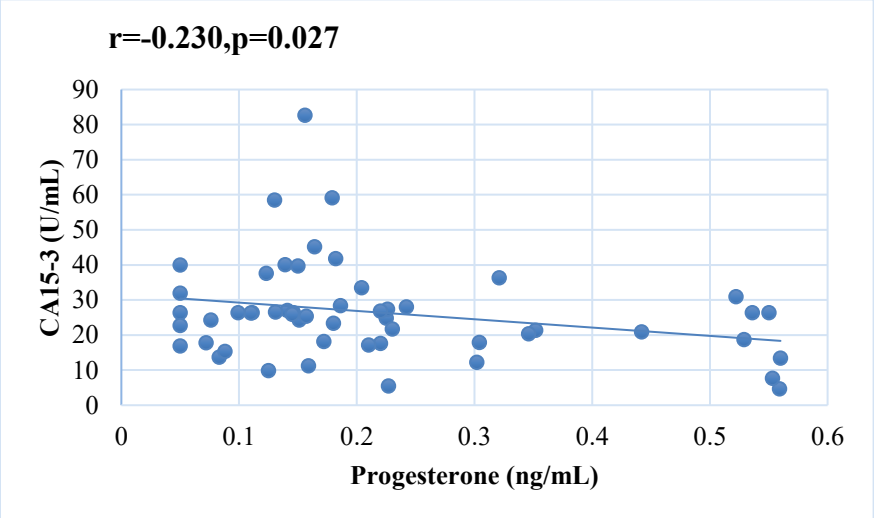

Fig. 3. Correlation between Ca15-3 and Progesterone in Breast Cancer patients

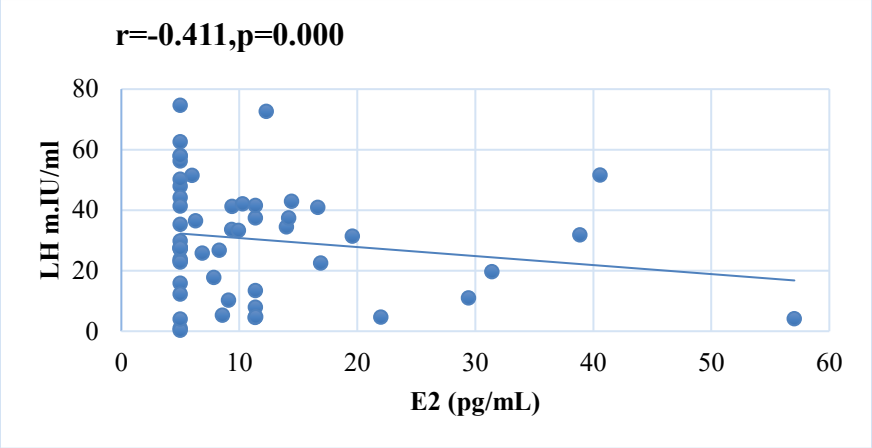

Fig. 4. Correlation between LH and E2 in Breast Cancer patients

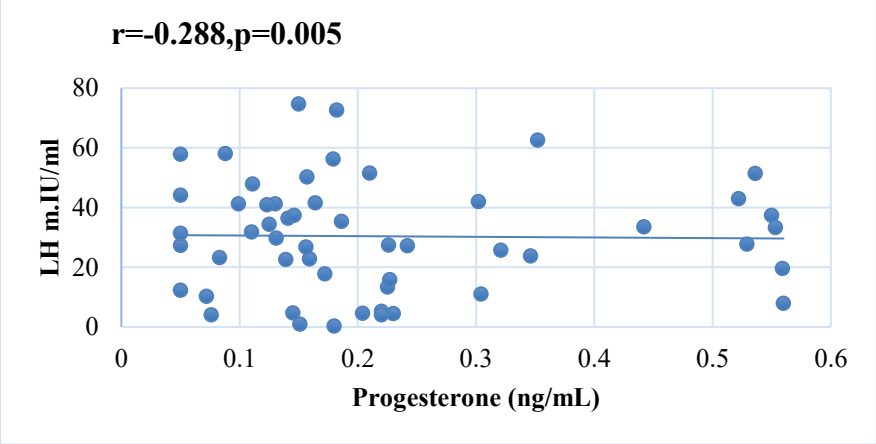

Fig. 5. Correlation between LH and Progesterone in Breast Cancer patients

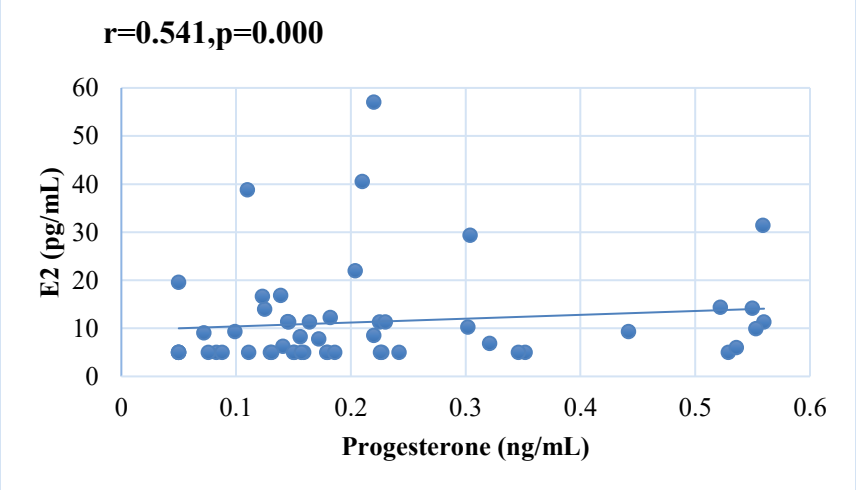

Fig. 6. Correlation between E2 and Progesterone in Breast Cancer patients

Discussion

Breast cancer is the most prevalent malignancy among women and continues to represent the second leading cause of cancer-related mortality worldwide [24]. Biomarkers, defined as biomolecules, function as indicators of pathological processes and provide valuable clinical insights into pharmacological responses to therapeutic interventions [25]. The present case-control study revealed significant alterations in the serum levels of CA15-3 and sex hormones among Iraqi women with breast cancer undergoing systemic therapy, compared to healthy controls. The most salient findings include markedly elevated CA15-3 and LH levels, significantly reduced estradiol and progesterone concentrations, and a notable positive correlation between CA15-3 and LH.

Regarding the tumor marker CA15-3, the present study revealed a highly significant statistical correlation, suggesting its pivotal role in the diagnosis and progression of breast cancer. These findings are consistent with previous research [26]. Moreover, the current results also showed a significant association between CA15-3 levels and age across both age groups. This observation is in agreement with an earlier study reporting that the majority of breast cancer cases (61.4%) occurred among women aged 45–55 years [26].

The hormonal milieu plays a critical role in the development of breast cancer as well as in determining tumor responsiveness to therapy. In the present study, significant alterations in LH concentrations were observed, which are known to promote tumor growth and metastasis, particularly in cells expressing the LH receptor (LHR). LH has been shown to regulate cell migration and invasion by modulating kinases that activate cellular actin [27]. These findings are consistent with previous research, which reported elevated LH concentrations in breast cancer tissues compared to normal tissues [28]. Moreover, significant differences were noted among women over 45 years of age, depending on the type of therapy received. In the present study, the mean LH concentration was 21.66 m.IU/ml with a relatively large standard deviation (24.27 m.IU/ml) in a small subgroup (n = 12). This marked variability may be attributed to interindividual differences among patients, including menopausal status, treatment modalities, and disease stage. In addition, the small sample size magnifies the impact of outlier values, which can substantially increase the SD. The analysis, stratified by treatment type, revealed a significant difference in LH levels between patients receiving chemotherapy and those receiving hormonal therapy. Those receiving chemotherapy had higher LH levels, which is a direct and expected consequence of its more profound ovarian-toxic effects compared to many hormonal therapies [12]. The lack of a significant difference in CA15-3 between these groups, despite different LH levels, suggests that the relationship is complex and may be influenced by other factors such as treatment response and tumor characteristics

About progesterone, a hormone associated with reproductive health, the present study revealed significant differences between cases and controls, indicating that its levels are affected by breast cancer. This observation is in agreement with earlier studies [29]. Similarly, concerning estradiol (a form of estrogen), the current findings demonstrated significant variations in its concentrations, supporting the established notion that estrogen plays a pivotal role in the development of certain types of breast cancer, particularly hormone receptor–positive subtypes. Other studies have also reported differences in blood estradiol levels between patients and healthy controls, which aligns with the results of this investigation [30]. Additionally, a strong

association was found between estrogen levels and age in both groups, while a significant relationship was identified between progesterone levels and women under 44 years of age. The elevated LH levels are likely a compensatory response to the treatment-induced decline in ovarian estrogen and progesterone production, a classic endocrine negative feedback mechanism [19].

A key finding of the present study is the significant positive correlation observed between serum CA15-3 and LH levels ($r = 0.333$, $p = 0.001$). This novel association highlights a potential interaction between tumor activity and the pituitary–gonadal axis in the context of breast cancer management. Recent evidence suggests that LH receptors are expressed in breast tissue, and LH itself may contribute to enhancing tumor invasiveness and progression [27]. Accordingly, the current results imply that elevated LH may not solely represent an endocrine response to therapy, but could also play an active role in modulating tumor behavior, as reflected by increased CA15-3 levels. This observation underscores the need for further investigations into the involvement of gonadotropins in breast cancer pathophysiology.

In addition, the strong negative correlations of CA15-3 with estradiol and progesterone observed in this study further emphasize the state of hormonal suppression induced by therapy and its association with elevated tumor marker levels. These findings are consistent with a previous study [31], which indicated that elevated CA15-3 levels are significantly associated with an increased likelihood of estrogen receptor or progesterone receptor-positive tumors. Moreover, higher CA15-3 levels may serve as a predictive marker for favorable therapeutic outcomes in recurrent breast cancer. Conversely, the strong positive correlation between estradiol and progesterone ($r = 0.541$, $p < 0.001$) in patients was expected, given that both hormones originate primarily from the ovaries and are simultaneously suppressed during chemotherapy. Consistent with experimental models demonstrating the direct tumor-promoting effects of these hormones, estrogen and progesterone have been shown to stimulate breast cancer cell proliferation in vitro by inducing the expression of cyclin G1, a key regulator of the cell cycle [32]. In our clinical cohort, however, the significantly lower concentrations of estradiol and progesterone likely reflect the therapeutic suppression of ovarian function, which aims to counteract these very proliferative signals. Serum CA15-3 levels exhibit significant menstrual cycle–dependent variability, with higher concentrations observed during the mid-luteal phase, coinciding with luteinizing hormone (LH) surge and subsequent hormonal changes. This suggests a potential modulatory effect of LH on CA15-3 levels, which should be considered when interpreting this tumor marker, particularly in early-stage breast cancer.

4. Limitations

This study has several limitations that should be considered: Lack of Baseline Measurements: The main limitation is the absence of data on hormone and CA15-3 levels in patients before starting treatment. This makes it difficult to definitively distinguish between changes caused by the disease itself and those resulting from the side effects of treatment. Lack of Detailed Clinical Data: The unavailability of crucial information, such as tumor stage, grade, and hormone receptor status (ER/PR/HER2), limits our ability to conduct a deeper analysis. These factors could have explained some of the variation observed in the results. Sample Size: The number of

participants in the study, especially when divided into subgroups (by treatment type or age), is somewhat limited, which may affect the statistical power of some comparisons.

5.Conclusion

In conclusion, this study provides evidence of significant dysregulation in the sex hormonal profile of Iraqi women with breast cancer undergoing systemic therapy. Specifically, patients exhibited elevated luteinizing hormone (LH) levels alongside reduced estradiol and progesterone concentrations. Importantly, a significant positive correlation was observed between CA15-3 and LH, suggesting a potential biological interplay between tumor marker activity and gonadotropin regulation. These findings highlight the possibility that LH may play a more active role in breast cancer pathophysiology than previously recognized. Future research should incorporate longitudinal designs with pre-treatment baseline measurements and detailed clinical data (such as tumor stage, grade, and receptor status) to validate these associations and further clarify the mechanisms linking CA15-3 with hormonal alterations.

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الارتباط بين ارتفاع مستويات CA15-3 ونشاط الهرموني الجنسي لدى النساء المصابات بسرطان الثدي: دراسة حالة- شاهد في البصرة، العراق

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الخلاصة:

يُعدّ سرطان الثدي من أكثر الأورام الخبيثة شيوعًا بين النساء، ويعود تطوره إلى تداخل عوامل داخلية وخارجية. تهدف هذه الدراسة إلى تقييم العلاقة بين مستويات المستضد السرطاني CA15-3 في المصل والملف الهرموني الجنسي (الإستراديول، البروجستيرون، والهرمون اللوتيني) لدى مجموعة من النساء العراقيات المصابات بسرطان الثدي والخاضعات للعلاج الجهازي، فضلًا عن دراسة تأثير العوامل الديموغرافية والعلاجية على هذه الارتباطات. أجريت الدراسة في قسم الأورام بمستشفى الصدر التعليمي ومستشفى الزبير العام في محافظة البصرة. شملت العينة 93 مشاركة، توزعن إلى 53 مريضة بسرطان الثدي (مجموعة المرضى) و40 امرأة سليمة (مجموعة الاصحاء). تم قياس تراكيز مصل كل من: المستضد السرطاني 15-3(CA15-3) ، والهرمون اللوتيني(LH) ، والإستراديول، والبروجستيرون باستخدام تقنية المقايسة المناعية الكهروكيميائية (ECLIA) على جهاز .Cobas e411 جرى تحليل البيانات باستخدام برنامج SPSS (الإصدار 25)، ، مع تطبيق اختبار **Mann-Whitney U** ومعامل ارتباط **Spearman**. وأظهرت النتائج وجود فروق ذات دلالة إحصائية في هذه المتغيرات البيوكيميائية بين مجموعتي المرضى والأصحاء. تؤكد هذه الدراسة وجود اضطراب كبير في الملف الهرموني الجنسي لدى المريضات العراقيات بسرطان الثدي الخاضعات للعلاج الجهازي، إذ ارتفعت مستويات LH في حين انخفضت

تراكيز الإستراديول والبروجستيرون. كما يشير الارتباط الإيجابي المعنوي بين CA15-3 و LH إلى احتمال وجود تفاعل بيولوجي جوهري بين الواسم الورمي والنشاط الهرموني.

الكلمات المفتاحية : CA15-3 (المستضد السرطاني 15-3)، الهرمون اللوتيني (LH) , الهرمونات الجنسية, سرطان الثدي