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Determination of Interleukin-6, Tumor Necrosis Factor-α, and Malondialdehyde in Breast Cancer Patients from Kirkuk

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Abstract:

Breast cancer (BC) is one of the most common malignancies in women and a leading cause of cancer-related death worldwide, and in Iraq, it accounts for nearly one-third of female cancer cases. Chronic inflammation and oxidative stress contribute to its development. The aim of this study was to compare patients to healthy controls regarding serum levels of interleukin 6 (IL-6), tumor necrosis factor alpha (TNF-α), and malondialdehyde (MDA) in different age groups and their possible role in diagnosis and prognosis in BC. Serum levels of IL-6, TNF-α, and MDA were evaluated among 48 breast cancer patients with an age range of 25-64 years and compared with those of 47 matched healthy individuals. We collected blood samples in Kirkuk between November 2025 and February 2026. measured IL-6 and TNF-alpha using ELISA, while we determined them using the Thiobarbituric acid reactive substances (TBARS) method, and data were analyzed with Student's t-test in SPSS. The results showed that IL-6 reaches 12.47 ± 3.82 pg/mL in patients compared to 4.21 ± 1.35 pg/mL in controls, and TNF-α reaches 8.93 ± 2.47 pg/mL in patients versus 3.15 ± 1.02 pg/mL in controls, with significant differences for both markers. MDA also increases to 3.85 ± 0.94 µmol/L in patients compared to 1.42 ± 0.38 µmol/L in controls, showing a highly significant difference. Regarding age groups, we found that patients within the age group (45-64 years) show the highest values for all markers, with clear statistical

differences across age categories, especially for MDA. As a conclusion, these findings indicated an increased inflammation and oxidative stress in breast cancer patients, and can be used in detection, monitoring, and risk assessment, especially in older patients. As a recommendation, larger, longitudinal, and multicenter surveys are required to validate these findings, establish cutoff values, and explore therapeutic targeting of these pathways.

Keywords: Breast cancer, Interleukin-6 (IL-6), Tumor Necrosis Factor-alpha (TNF-α), Malondialdehyde (MDA)

Introduction:

Breast cancer (BC) is the most life-threatening malignancy among women and remains a leading cause of cancer-associated death globally. According to the GLOBOCAN assessment for 2022, around 2.3 million new cases and approximately 670,000 deaths have been recorded, accounting for 11.6% of all cancer cases and 6.9% of cancer-associated deaths [1]. In Iraq, breast cancer ranks first among women's cancers and accounts for nearly one-third of all recorded cases, surpassing other types such as bronchial cancer [2,3]. Recent national and regional reports show a steady increase in the incidence rate. For example, a study in Karbala recorded 342 cases of breast cancer out of 1737 cancers, and the majority of the cases were non-prevalent and positive for hormone receptors [4]. In the Kurdistan region, an increase in the number of patients has been indicated, with invasive ductal carcinoma being the most common histological type, and the predominant molecular type being Luminal A [5]. In Sulaymaniyah, 630 cases were reported in 2025 alone, reflecting the continuing burden on public health systems [6]. Early detection of the disease is critical to reduce mortality rates. The World Health Organization (WHO) confirms that screening, together with appropriate therapeutic intervention, improves survival rates [7]. In Iraq, a national early detection initiative was launched in 2001 and subsequently expanded across various provinces; however, early diagnosis rates remain unsatisfactory. Evidence suggests that only a small proportion of tumors are identified in the early stages, although many cases can be effectively managed if detected early [8]. In particular, there are several factors, such as age or family history of BC, reproductive factors, and hormonal influences, that play some role in BC etiology. Age is an important factor as incidence rates increase with age [9]. Chronic inflammation and BC are strongly linked.

The tumor microenvironment represents a source of cytokines that are instrumental for tumor growth, angiogenesis, and cell proliferation [10,11]. IL-6 and TNF-α are the best characterized pro-inflammatory cytokines. IL-6 contributes to immune regulation and is often found elevated in several solid tumors, including BC, where it correlates with a bad prognosis and treatment resistance [12,13]. It signals via the JAK/STAT3, MAPK, and PI3K/Akt-mediated pathways, which promote cell survival and proliferation [14,15]. In addition, this review demonstrates that IL-6sustains stem cell-like features in cancer cells and enhances metastatic potential [16-18]. TNF-α is a major mediator, and its high levels correlate with disease aggravation and poor survival. Pooled analyses indicate that higher levels of TNF α are associated with poor clinical outcomes[16,19]. Its effects are mainly mediated by NF-κB and JNK pathways, which induce inflammation, DNA damage, and survival of tumor cells [20,21]. Oxidative stress is another important factor in the development of cancer and is closely related to inflammation. It arises as a result of an imbalance between the production of reactive oxygen species and antioxidant defenses [22]. MDA is a widely used indicator of the process of lipid peroxidation and reflects the extent of oxidative damage in cells [23]. Elevated levels of MDA indicate increased membrane damage and are associated with DNA mutations and genomic instability, which are key steps in cancer formation [24,25]. Recent studies have pointed to MDA as a potential biomarker in breast cancer, together with other oxidative stress markers [26]. Based on these mechanisms, IL-6, TNF-α, and MDA have been studied as non-invasive serum biomarkers that might help in the early detection, disease monitoring, and prognosis of patients with breast cancer [27,28]. However, studies that evaluate these markers together in Iraqi patients are limited, especially in Kirkuk city. The aim of this study is to measure these markers and compare patients with healthy controls to look at age-related differences and their possible role in diagnosis and prognosis in BC.

Material and methods:

Study Population

This case-control study included 48 women diagnosed with breast cancer and 47 apparently healthy women as controls. Participants were enrolled from hospitals and oncology centers in Kirkuk Governorate from November

2025 to February 2026. Histopathological confirmation of the diagnosis of breast cancer was done by a specialist oncologist. Individuals with the above-mentioned diseases, as well as those with chronic inflammatory diseases, autoimmune disorders, liver diseases, diabetes mellitus, or other malignancies, were excluded from the study.

Blood Collection and Serum Preparation

Venous blood samples (5 mL) were obtained from all subjects after informed consent was acquired. Blood was drawn in plain tubes and allowed to clot for 30 min at room temperature, followed by centrifugation at 3,000 rpm for 10 min. The isolated serum was portioned into aliquots and frozen at-20°C until use.

Determination of serum IL-6 and TNF-α levels:

Serum IL-6 andTNF-α levels were measured with commercial enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems, USA, Cat# D6050 for IL-6 and Cat# DTA00D for TNF-α). The assays were performed according to the manufacturer's protocol. Using a microplate reader(BioTek, USA), the absorbance was read at 450 nm. Concentrations were determined from standard curves and expressed in pg/mL.

Determination of serum MDA levels

MDA levels were determined as a marker of lipid peroxidation using the thiobarbituric acid reactive substances (TBARS) assay. Briefly, 0.5 mL serum was combined with 1.0 mL of 20% trichloroacetic acid (TCA) and 1.0 mL of 0.67% thiobarbituric acid (TBA). The reaction mixture was heated at 95 ºC in a water bath for 30 min and cooled to room temperature. The samples were spun at 3000 rpm for 10 min to pellet the precipitated proteins. The supernatant absorbance was read at 532nm by using a UV–Visible spectrophotometer. MDA levels were calculated with the help of a standard calibration curve using 1,1,3,3-tetraethoxypropane standards and represented as µmol/L.

Ethical Approval

The study protocol was approved by the Ethical Committee of the College of Science, University of Tikrit. Written informed consent was obtained from all participants before sample collection.

Statistical Analysis

Statistical analysis was performed using SPSS 25.0 (IBM Corp., Armonk, NY, USA). Continuous data are expressed as mean± standard deviation (SD). Data distribution normality was evaluated with the Shapiro-Wilk test. Between two groups, comparisons were conducted by Student's independent t-test (normally distributed data) or Mann-Whitney U test (non-normally distributed data). A p-value of<0.05 was deemed to be statistically significant.

Results:

This case-control study included 95 participants: 48 women with breast cancer and 47 healthy women as controls. The average age of the breast cancer patients was 48.5 ± 7.2 years, and that of the control subjects was 47.8 ± 6.9 years. Serum levels of IL-6, TNF-α, and MDA were evaluated in all subjects and between the groups.

IL-6 levels in breast cancer patients vs. healthy controls

IL-6 levels were significantly elevated in breast cancer patients (12.47 ± 3.82 pg/mL) compared to healthy controls (4.21 ± 1.35 pg/mL; $p < 0.01$), indicating a clear elevation of this pro-inflammatory cytokine in breast cancer patients. These results are presented in Table 1 and Figure 1.

Table 1.IL-6 Levels in breast cancer patients and the control group

Group	IL-6 (pg/mL) Mean ± SD	p-value
Breast cancer patients (n = 48)	12.47 ± 3.82	< 0.01
Healthy controls (n = 47)	4.21 ± 1.35	

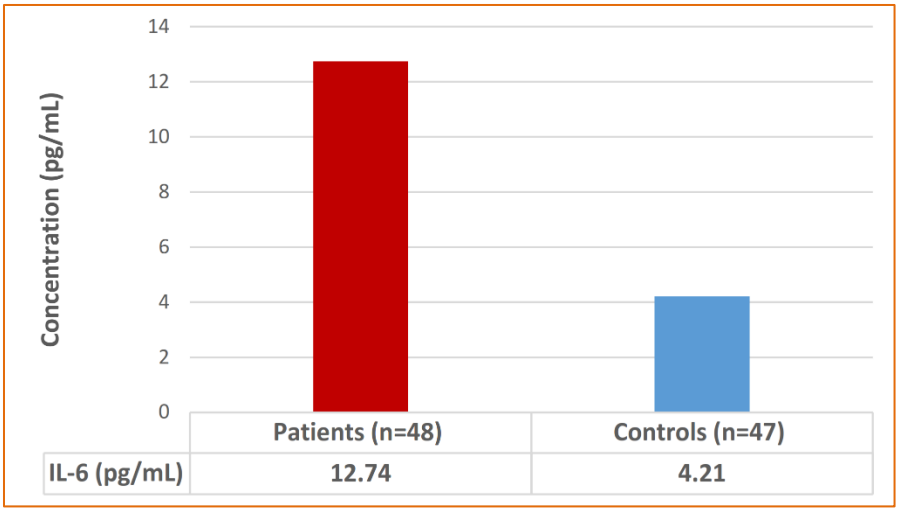

Figure 1. IL-6 Levels in breast cancer patients and controls

TNF-α levels in breast cancer patients vs. healthy controls

The serum TNF-α level in the breast cancer patients was also significantly higher than that in the healthy controls. Patients' TNF-α mean value was 8.93 ± 2.47pg/mL, while the mean value in the control group was 3.15 ± 1.02 pg/mL. There was a significant difference between the two groups ($p < 0.01$), demonstrating the enhancement of the systemic inflammatory reaction in the breast cancer group. The results are shown in Table 2 and Figure 2.

Table 2:TNF-α Levels in Breast Cancer Patients and Control Group

Group	TNF-α (pg/mL) Mean ± SD	p-value
Breast cancer patients (n = 48)	8.93 ± 2.47	< 0.01
Healthy controls (n = 47)	3.15 ± 1.02	

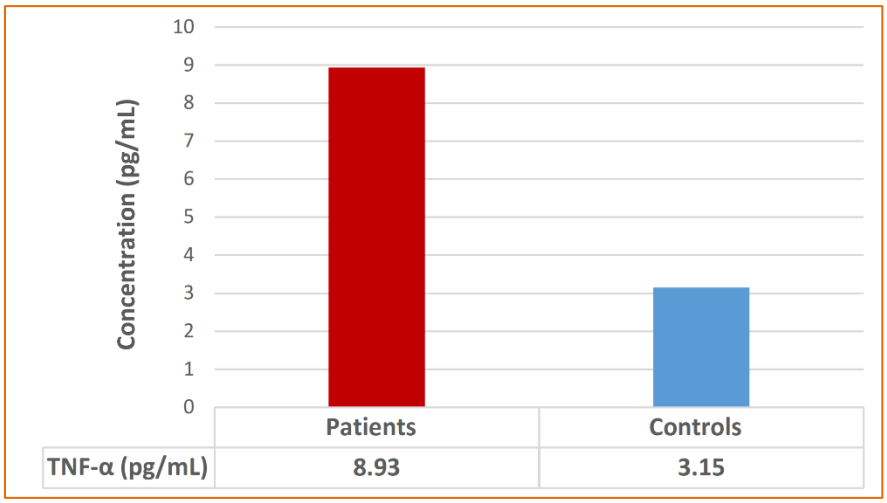

Figure 2. TNF-α Levels in breast cancer patients vs. healthy controls

Age-Dependent Differences in IL-6 and TNF-α Levels

Age-stratified analysis showed that the levels of IL-6 andTNF-α were significantly

increased in breast cancer patients compared with controls in all age groups. Among the 20–34 years group ofpatients, the levels of both cytokines were significant higher in patients than controls ($p < 0.05$). In the age groups more clearly were 35–44 and45–64 years, and the differences were greater ($p < 0.01$). The highest mean values for both IL-6 andTNF-α were found in the 45–64 years group, suggesting a possible progressive increase of inflammatory activity with age in breast cancer patients. These results aresummarized in Table 3 and shown in Figure 3.

Table 3.IL-6 and TNF-α Levels by age group

Biomarker	Age Group (years)	Patients Mean ± SD	Controls Mean ± SD	p-value
IL-6 (pg/mL)	20–34	10.24 ± 3.15	3.98 ± 1.22	< 0.05
	35–44	12.08 ± 3.54	4.11 ± 1.30	< 0.01
	45–64	14.85 ± 4.02	4.52 ± 1.48	< 0.01
TNF-α (pg/mL)	20–34	7.62 ± 2.11	2.95 ± 0.88	< 0.05
	35–44	8.79 ± 2.34	3.08 ± 0.95	< 0.01
	45–64	10.23 ± 2.86	3.42 ± 1.10	< 0.01

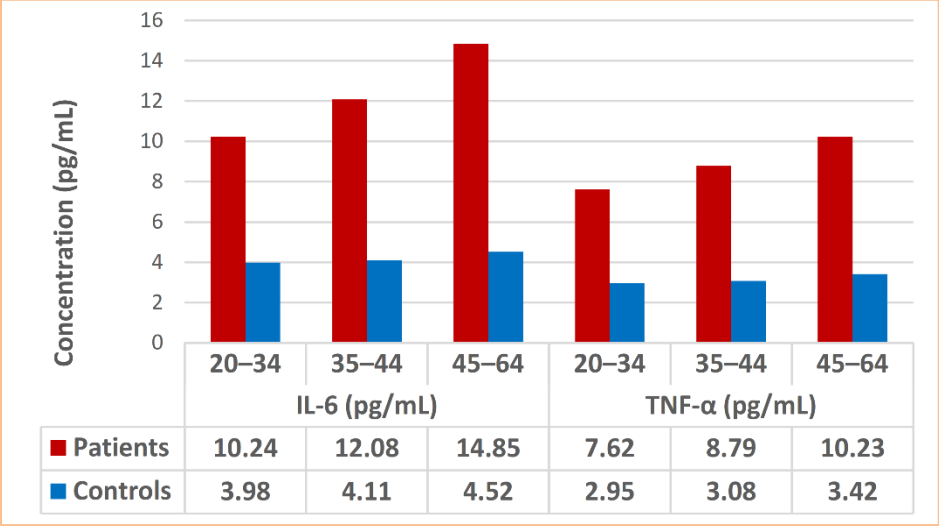

Figure 3. Age-stratified IL-6 and TNF-α levels in patients and controls

MDA levels in patients vs. controls

Serum MDA levels were found to be raised in breast cancer patients when compared to healthy controls. The mean MDA levels were 3.85 ± 0.94 µmol/L in the patients and 1.42 ± 0.38 µmol/L in the controls. This was extremely significant ($p < 0.001$), indicating enhanced lipid peroxidation and oxidative stress in the breast cancer patients. The findings are shown in Table 4 and Figure 4.

Table 4. MDA levels in breast cancer patients and the control group

Group	MDA (µmol/L) Mean ± SD	p-value
Breast cancer patients (n = 48)	3.85 ± 0.94	< 0.001
Healthy controls (n = 47)	1.42 ± 0.38	

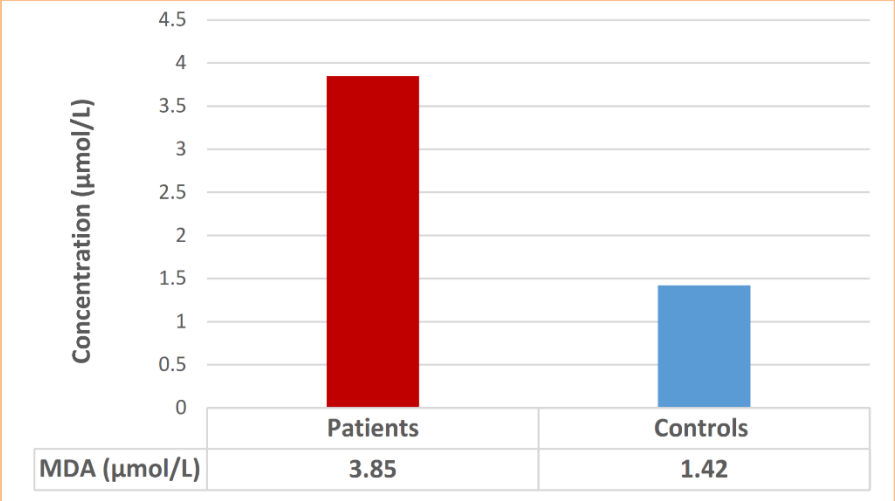

Figure 4. MDA Levels in sera of breast cancer patients and the control group

Age-dependent differences in MDA levels

The age-stratified analysis of MDA concentrations revealed a consistent elevation in breast cancer patients relative to controls in all age groups. Among those aged 20–34 years, MDA was significantly higher in patients (3.21 ± 0.85 µmol/L) than in controls (1.28 ± 0.35 µmol/L; $p < 0.01$). Higher MDA levels were also observed in patients versus controls in the 35–44 years category by +2.39 µmol/L ($p < 0.01$). The most marked increment was in the 45–64 years age group, in which MDA level was as high as 4.52 ± 1.02 µmol/L in patients versus 1.58 ± 0.42 µmol/L in controls, with a mean difference of +2.94 µmol/L ($p < 0.001$). This suggests that oxidative stress was higher in

elderly breast cancer patients, as summarized in Table 5 and Figure 5.

Table 5. Age-based variations in MDA levels

Age Group (years)	Patients (µmol/L) Mean ± SD	Controls (µmol/L) Mean ± SD	Difference (Δ)	p-value
20–34	3.21 ± 0.85	1.28 ± 0.35	+1.93	< 0.01
35–44	3.78 ± 0.91	1.39 ± 0.37	+2.39	< 0.01
45–64	4.52 ± 1.02	1.58 ± 0.42	+2.94	< 0.001

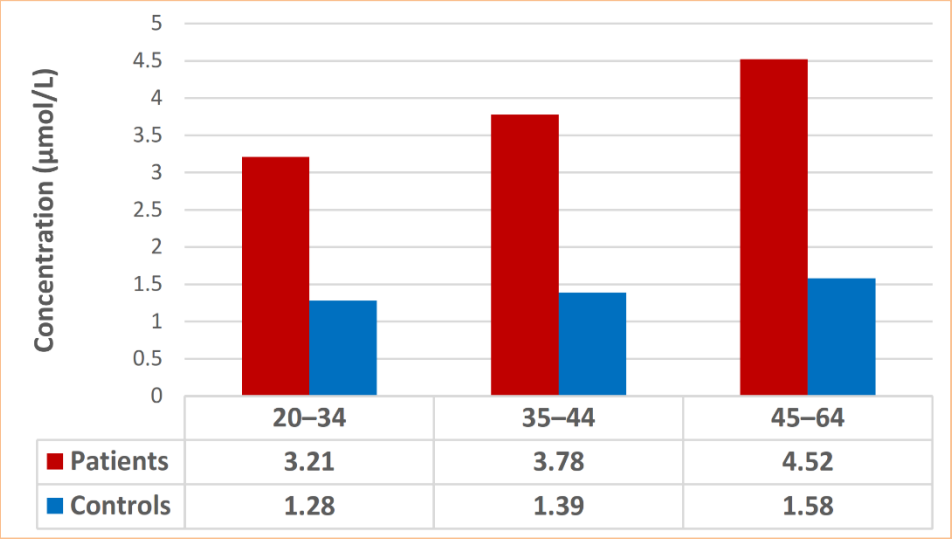

Figure 5. Age-based variations in MDA levels in patients and controls

Discussion

The present study demonstrated significantly elevated serum levels of IL-6, TNF-α, and MDA in breast cancer patients compared with healthy controls. These findings support the growing evidence that chronic inflammation and oxidative stress play important roles in the pathogenesis and progression of breast cancer. Our results also showed that all three biomarkers increased consistently across different age groups, with the highest levels observed in older patients aged 45–64 years. This age-related pattern may have important clinical significance, especially in Iraq, where breast cancer is frequently diagnosed at advanced stages[2,3].

Interleukin-6 (IL-6) as a biomarker in breast cancer

IL-6 levels in breast cancer patients (12.47 ± 3.82pg/mL) were more than threefold elevated compared to healthy women. This was a statistically significant difference and may indicate enhanced inflammatory activity in relation to the progression of breast cancer. A recent meta-analysis by De La Cruz-Vargas et al. (2025) with a sample size of >3, 000 patients revealed that increased IL-6 levels were predictive of an almost threefold (HR= 2.87) risk of death [19]. Of note, our results are comfortably in the upper band of that meta-analysis for aggressive/met patients. In terms of its pro-inflammatory cytokine role, IL-6 may contribute to tumorigenesis through various interconnected signaling cascades. Activates the JAK/STAT3 pathway, which increases proliferation and inhibits apoptosis of tumor cells via the upregulation of anti-apoptotic proteins Bcl-2 and survivin [15,16]. Recently, Przanowska et al. (2026) described a novel role for IL-6 in chemoresistance: In estrogen receptor-positive (ER+)breast cancer, IL-6 signaling in pericyte cells that surround blood vessels contributes to tumor cells' survival after chemotherapy [17]. This observation suggests that modulation of IL-6 may not only have an anti-inflammatory effect on therapeutic efficacy. Similarly, Werner-Klein et al. (2020) demonstrated that IL-6trans-signaling may indeed be responsible for the re-awakening of dormant disseminated tumor cells, a number of years post initial treatment, to cause late recurrences [16]. From a diagnostic standpoint, IL-6 showed good performance for differentiating patients from controls in our cohort. However, it should be recognized that IL-6 is not specific for breast cancer and may be elevated in other chronic inflammatory conditions, such as autoimmune diseases and sepsis [30].

Therefore, its usefulness as a single biomarker may be limited, but it may have value as one component of a biomarker panel.

Tumor necrosis factor-alpha (TNF-α) as a biomarker

TNF-α levels in the breast cancer patients (8.93± 2.47 pg/mL) were about three folds higher than the control group suggesting the presence of enhanced systemic inflammatory responses in these patients. This is inline with the systematic review by Qodir et al. (2025) who showed consistentlyhigher levels of TNF-α in breast cancer patients especially in those with the lymph node metastasis and metastasis [18]. TNF-α effects on cancerare dual. At low and chronic doses, it may induce tumor progression byinducing NF-κB and AP-1 transcription factors which control the expression of genes that are involved in cell survival, angiogenesis and invasion [20,21]. Nevertheless, tumor cell death can be triggered in the presence of highlevels of TNF-α under certain conditions. Thisdual action can possibly explain why TNF-α neutralization by itself may not be effective in all cases and why the biological context matters. The significant elevation of TNF-α in older patients may be related to "inflammaging," which refers to a chronic low-grade inflammatory state associated with aging, cellular senescence, mitochondrial dysfunction, and accumulated oxidative damage [31]. Inflammaging may also create a tumor-favorable microenvironment and may partly explain the increased breast cancer risk after menopause [9]. The interaction of TNF-α with the tumor microenvironment adds further complexity. TNF-α derived from tumor-associated macrophages (TAMs) and cancer-associated fibroblasts (CAFs) may induce epithelial-to-mesenchymal transition (EMT), thereby increasing cancer cell motility and invasiveness [32]. Some studies indicate that increasedTNF-α is associated with E-cadherin loss of E-cadherin and increased vimentin expression, which are markers of EMT [33]. However, whether these molecular changes are present in our patients remains unknown and should be investigated in future studies.

Malondialdehyde (MDA) and oxidative stress

A significant difference was observed in serum MDA levels between breast cancer patients and healthy controls (4.85 ± 1.94 μmol/L vs. 1.42 ± 0.38 μmol/L, $p < 0.001$). Such elevation may be attributed to increased lipid peroxidation due to overproduction of reactive oxygen species (ROS), which overtake the antioxidant defense activity of the body. Besides being a biomarker for

oxidative stress, MDA could play a role in causing DNA damage and genomic instability. It can also interact with DNA to form exocyclic adducts, namely M1G, which trigger G→T and G→C transversions and are commonly found as mutated bases in the p53 gene. A strong pattern was seen for all the age groups, 3.21µmol/L and 4.52 µmol/L for the 20–34 years and 45–64 years age groups, respectively, for mean MDA, suggesting an age-dependent oxidative damage[25,34]. Ayob and Mydin (2025) also found that MDA levels rise with tumor stage and grade [26]. It is worth mentioning that none of the youngest patients in our cohort had MDA levels less than twice that of their age-matched healthy controls, suggesting that oxidative stress may take hold early in the course of cancer and may exist long before clinical manifestations. Prospective studies are needed to determine whether elevated MDA levels may predict breast cancer risk in apparently healthy women. Multiple factors may increase oxidative stress in breast cancer patients, including genetic variations in antioxidant enzymes (e.g., SOD2, GPx), diet, environmental toxins, and persistent infections that lead to inflammation. In Iraq, war-related pollutants, burning oil fields, and psychological stress may further increase oxidative damage. Future studies should also measure other oxidative stress markers, such as 8-hydroxy-2'-deoxyguanosine (8-OHdG) for DNA oxidation and protein carbonyls for protein oxidation to provide a more comprehensive evaluation [35-37].

Age-dependent variations

The clinical importance of our finding of the highest levels of all three biomarkers in the 45-64 years age group is twofold: First, this is the age group that most of the breast cancer patients in Iraq, as well as in many other countries, belong to [2,9]. The biological underpinning of this age-related rise is multifactorial. Immune senescence can also be involved via enhanced activity of autoimmune and self-reactive T cells, as well as diminished functionality of regulatory immune cells. Under the effects of age progression, the immune system experiences immunosenescence, which is defined as the gradual deterioration of the immune response in the adaptive arm and the simultaneous up-regulation of the innate immune cells, which can secrete high amounts of IL-6 and TNF-α [31,38]. And yet mitochondria are becoming dysfunctional, increasing ROS production and decreasing ATP synthesis. The Nrf2signaling pathway, which regulates antioxidant enzyme expression, may also become downregulated during aging, leading

to a cycle of oxidative stress and inflammation [39]. Interestingly, also the group of patients aged 20-34 in our study demonstrated a significant increase of all studied biomarkers in comparison to controls, although the absolute values were lower than in the group of older patients. These results indicate that inflammatory and oxidative stress responses are not merely byproducts of aging, but might actively contribute to breast cancer development in younger and older patients. These markers may be monitored to give supportive information for young women with a family or genetic risk (such as BRCA mutations) [40].

Comparison with regional and international studies

The findings of the present study are mainly in line with those from the other regions of the world, but there are some differences. Kocyigit et al. showed comparable levels of IL-6 and TNF-α in breast cancer patients, but the MDA level in these patients was moderately decreased (about 2.8 μmol/L). As opposed to this, Sharma et al. have also found elevated MDA levels (5.2 μmol/L) in stage III/IV breast cancer. These discrepancies may be attributable to differences in the distribution of tumor stage and in diet habits, environmental exposure, and ethnicity. Among all, perhaps only a handful of studies in Iraq have examined these three markers in combination. The Baghdad study by Al-Hilli and Kadhim (2023) documented raised IL-6 and TNF-α in breast cancer [41-46], but did not measure MDA. Hence, the current investigation might be one of the earliest works that has allowed integrated analysis of IL-6, TNF-α, and MDA in the breast cancer patients from the Kirkuk province. A multicenter study is highly desirable to replicate these findings because there are environmental and lifestyle disparities among the Iraqi governorates.

Clinical Implications and Future Directions

The elevated IL-6, TNF-α, and MDA levels observed in breast cancer patients may have several clinical applications. These biomarkers may help in identifying inflammatory and oxidative stress status in individuals at high risk, such as women with a family history of breast cancer or a BRCA mutation. Combining these biomarkers into a panel may provide better sensitivity and specificity than using each marker individually [28]. In addition, these markers may have prognostic value, as patients with markedly elevated levels may require closer

follow-up and more intensive management. Serial measurements during and after therapy may also assist in monitoring treatment response and early detection of disease recurrence [47]. However, several limitations should be considered before these biomarkers can be applied routinely in clinical practice. Standardization of cutoff values is required, and pre-analytical factors such as sample collection time, storage conditions, and hemolysis may influence the results. Furthermore, these biomarkers may be elevated in several non-malignant inflammatory conditions and therefore should be used as supportive rather than independent diagnostic markers. In resource-limited countries such as Iraq, where routine mammography screening may be limited, serum biomarkers may provide supportive and relatively inexpensive tools for disease monitoring and risk assessment.

Limitations of the study

We would like to highlight some limitations of the present study. The case-control design limits the ability to establish causal relationships;therefore, it cannot be determined whether elevated IL-6 and MDA levels preceded breast cancer development or occurred as a consequence of the disease. The sample size was relatively modest, which may have limited the statistical power for subgroup analyses, such as comparisons according to molecular subtype or tumor stage. In addition, information regarding treatment history was not available, and some patients may have received chemotherapy or radiotherapy before sample collection, which could potentially influence biomarker levels [48]. Finally, additional potentially relevant biomarkers, including C-reactive protein (CRP), interleukin-10 (IL-10), and total antioxidant capacity (TAC), were not evaluated in the current study. Future investigations should address these limitations to provide a more comprehensive understanding of inflammatory and oxidative stress biomarkers in breast cancer.

Future research directions

However, a longitudinal study is needed to determine whether levels of IL-6, TNF-α, and MDA at baseline can be used to predict future risk of breast cancer in healthy women. Animal- or cell-based mechanistic studies could potentially also describe the role of these markers in particular steps of metastasis. Registered and ongoing clinical trials using an anti-IL-6 mAb (tocilizumab) or TNF-α antagonists (etanercept) in combination with standard chemotherapy in other malignancies may be extrapolated to

breast cancer [17,49]. In line with these results, because of an increasing burden of breast cancer in Iraq, we recommend the building of pilot national screening programs that would encompass biomarker analyses.

CONCLUSION

The findings of the current study indicated an increased inflammation and oxidative stress in breast cancer patients, and can be used in detection, monitoring, and risk assessment, especially in older patients.

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تقدير مستويات الإنترلوكين-6، وعامل نخر الورم-ألفا، والمالونداياليديهايد لدى مريضات سرطان الثدي في كركوك

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الخلاصة:

سرطان الثدي هو واحد من أكثر الأورام الخبيثة شيوعاً بين النساء، وسبب رئيسي للوفيات المرتبطة بالسرطان في جميع أنحاء العالم، وفي العراق يمثل ما يقرب من ثلث حالات السرطان لدى النساء. يساهم الالتهاب المزمن والإجهاد التأكسدي في تطور هذا المرض. في هذه الدراسة، قمت بتقييم المستويات المصلية لكل من الإنترلوكين-6، وعامل نخر الورم ألفا، والمالونداياألدهيد لدى 48 مريضة بسرطان الثدي تتراوح أعمارهن بين 25 و64 سنة، ومقارنتها مع 47 فرداً أصحاء من نفس الفئة العمرية. قمت بجمع العينات في كركوك بين سبتمبر 2024 وفبراير 2025. قمت بقياس *IL-6* و *TNF-alpha* باستخدام تقنية *ELISA*، بينما حددت *MDA* باستخدام طريقة *TBARS*، ثم حللت البيانات باستخدام اختبار *t* للطلاب في برنامج *SPSS*. أظهرت النتائج أن *IL-6* بلغ 12.47 ± 3.82 بيكوغرام/مل لدى المريضات مقارنة بـ 4.21 ± 1.35 بيكوغرام/مل في المجموعة الضابطة، وبلغ *TNF-alpha 8.93 ± 2.47* بيكوغرام/مل لدى المريضات مقابل 3.15 ± 1.02 بيكوغرام/مل في المجموعة الضابطة، مع وجود فروق ذات دلالة إحصائية لكلا المؤشرين. كما ارتفع *MDA* إلى 3.85 ± 0.94 ميكرومول/لتر لدى المريضات مقارنة بـ 1.42 ± 0.38 ميكرومول/لتر في المجموعة الضابطة، مما أظهر فرقاً شديد الدلالة. عند فحص الفئات العمرية، وجدت أن المريضات اللواتي تتراوح أعمارهن بين 45 و64 سنة أظهرن أعلى القيم لجميع المؤشرات، مع وجود فروق إحصائية واضحة عبر الفئات العمرية، خاصة بالنسبة لـ *MDA*. تشير هذه النتائج إلى زيادة الالتهاب والإجهاد التأكسدي لدى مريضات سرطان الثدي، وتدعم استخدام هذه المؤشرات في الكشف والمتابعة وتقييم المخاطر، خاصة لدى المريضات الأكبر سناً.

الكلمات المفتاحية: سرطان الثدي، إنترلوكين-6 (IL-6)، عامل نخر الورم ألفا (TNF-α)، مالوندايالديهايد (MDA)