Mohammed *et. al.,* Babcock Univ. Med. J.2026 9(S1):244-251
https://doi.org/10.38029/babcockuniv.med.j..v9iS1.1641
eISSN: 2756-4657

RESEARCH ARTICLE OPEN ACCESS

Association between oxidative stress markers and antioxidant vitamins C and D in Iraqi women with breast cancer: A cross-sectional study

Mohammed NR[1ID], Hilal NN[1ID], Abdul-aziz MM[2ID]

[1]*Department of Clinical Biochemistry, College of Medicine, University of Tikrit, Tikrit, Iraq*
[2]*Department of Surgery, College of Medicine, University of Tikrit, Iraq*

Submitted: 15th April 2026
Accepted: 7th June 2026
Published: 31st October 2026
ID: Orcid ID

Abstract
Objective: To evaluate the relationship between oxidative stress (measured by malondialdehyde, MDA) and antioxidant vitamins C and D in the incidence and progression of breast cancer among Iraqi women.
Methods: This cross-sectional study was conducted at Kirkuk Oncology Centre from December 2025 to March 2026. It included 90 women with histologically confirmed breast cancer (stages I–IV) and 45 age-matched healthy controls. Fasting serum samples were analysed for MDA, superoxide dismutase (SOD), glutathione peroxidase (GPx), vitamin C and vitamin D using ELISA and the Roche Cobas system.
Results: Breast cancer patients had significantly lower mean vitamin C (1.00 ± 0.20 vs. 1.32 ± 0.23 mg/dL, p<0.001) and vitamin D (13.19 ± 4.35 vs. 23.26 ± 5.18 IU/L, p<0.001) levels than controls, and significantly higher MDA (26.27 ± 6.34 vs. 18.20 ± 5.76 ng/mL, p<0.001). Vitamin D levels decreased progressively from stage I (14.9 ± 4.1) to stage IV (10.37 ± 3.85) (p=0.006). A moderate negative correlation was observed between vitamin D and MDA (r = –0.45, p=0.009). Vitamin C levels did not differ significantly across cancer stages.
Conclusion: Elevated oxidative stress and reduced antioxidant vitamins C and D are associated with breast cancer in Iraqi women. Vitamin D shows an inverse relationship with cancer stage, suggesting a possible role in disease progression. Prospective studies are needed to establish causality.

Keywords: Breast cancer; Malondialdehyde; Vitamin D; Vitamin C; Oxidative stress

Plain English Summary

Women with breast cancer often have higher levels of harmful molecules called free radicals (oxidative stress) and lower levels of protective nutrients such as vitamins C and D. This study measured these substances in 90 Iraqi women with breast cancer and 45 healthy women. The results showed that women with breast cancer had much lower vitamin C and D levels and higher levels of a marker of oxidative damage (MDA) than healthy women. As breast cancer became more advanced, vitamin D levels dropped further. This suggests that maintaining adequate vitamin D might be important for women with breast cancer, although more research is needed to confirm whether low vitamin D directly contributes to cancer progression.

Introduction

Breast cancer is the most diagnosed malignancy and the leading cause of cancer-related death among women worldwide (1). According to the World Health Organisation, approximately 2.3 million new cases were diagnosed globally in recent years (1, 2). In Iraq, breast cancer incidence has been rising, and a concerning

Correspondence:
Mohammed Nawar R
Department of Clinical Biochemistry, College of Medicine
University of Tikrit, Tikrit
Iraq
nawaralmosawi9@gmail.com

© BUMJ. 2026 Open Access This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated.

proportion of women present with advanced-stage disease. A study from Baghdad reported that nearly 47% of women had stage III or IV disease at diagnosis, with a peak age in the 40s – about two decades younger than the typical Western patient (3, 4). These patterns highlight delays in diagnosis and the need for region-specific research on risk factors and biomarkers.

All risk factors for breast cancer can be broadly divided into genetic factors (e.g., family history, benign proliferative lesions) and external factors (diet, lifestyle, hormonal contraceptives, and exposure to oxidative stress) (5). Among these, oxidative stress plays a dual role in breast cancer: chronically elevated reactive oxygen species (ROS) promote DNA damage, proliferation, angiogenesis, and epithelial–mesenchymal transition, whereas very high ROS levels can trigger cancer cell death (6, 7). Breast cancer cells typically show increased oxidative damage to lipids, proteins, and DNA, coupled with adaptive upregulation of antioxidant defences (8).

Malondialdehyde (MDA), a stable end-product of lipid peroxidation, is widely used as a biomarker of oxidative stress. Numerous studies have reported higher MDA levels in breast cancer patients than in healthy controls (9). Antioxidant vitamins, particularly C and D, may counteract oxidative damage. Vitamin C scavenges ROS and regenerates other antioxidants such as vitamin E and glutathione (10, 11). Vitamin D, beyond its classical role in calcium homeostasis, modulates the expression of antioxidant and stress-response genes, inhibits proliferation, induces apoptosis, and reduces angiogenesis in breast cancer models (12, 13). Epidemiological evidence suggests that low circulating vitamin D is associated with increased breast cancer risk and worse outcomes (14, 15).

However, data from Iraqi populations remain limited. Most Iraqi patients are diagnosed at a younger age and later stage than their Western counterparts, which may alter the relationship between oxidative stress markers and disease progression. This study therefore aimed to evaluate the association between serum levels of MDA, vitamin C, vitamin D, and other antioxidants (SOD, GPx) in Iraqi women with breast cancer across different clinical stages, compared with healthy controls.

Materials and Methods

Study design and setting

This hospital-based cross-sectional study was conducted at the Kirkuk Oncology Centre, Kirkuk, Iraq, from 1 December 2025 to 31 March 2026. The study protocol was approved by the Scientific Committee of the College of Medicine, University of Tikrit (approval number TUC-M-2025-089, dated 15 November 2025), and all participants provided written informed consent.

Participants

A total of 90 women with histologically confirmed breast cancer were enrolled consecutively during routine follow-up visits. Diagnosis was made according to World Health Organisation criteria, and tumour staging (I–IV) was performed using the American Joint Committee on Cancer (AJCC) system. Exclusion criteria were metastatic disease at diagnosis (except for stage IV patients enrolled as advanced cases), concurrent malignancy, renal or hepatic dysfunction, coronary artery disease, and current use of high-dose antioxidant supplements.

Forty-five healthy women, frequency-matched by age (±5 years), were recruited from women attending the outpatient clinic for routine check-ups. They had no personal history of breast cancer or other malignancies, no acute or chronic inflammatory diseases, and no current use of antioxidant vitamins. All controls underwent clinical breast examination; mammography was not routinely performed because of resource limitations, but any woman with a breast lump or suspicious finding was excluded.

Sample collection and biochemical analysis

Fasting venous blood samples (5 mL) were collected in the morning (8:00–11:00 a.m.) after an overnight fast. Samples were allowed to clot for 10–15 minutes at room temperature, then centrifuged at 3000 rpm for 10 minutes. Serum was separated into three Eppendorf tubes and stored at –20 °C until analysis (maximum storage: 4 weeks). All samples were processed in duplicate.

MDA (malondialdehyde) was measured using a commercial ELISA kit (Cloud-Clone Corp., Houston, TX, USA; cat# CEB851Ge) according to the manufacturer's instructions. The intra-assay coefficient of variation (CV) was <8%, and the inter-assay CV <10%.

SOD (superoxide dismutase) and GPx (glutathione peroxidase) were measured by ELISA (Elabscience, Wuhan, China; cat# E-EL-R1429 for SOD, cat# E-EL-0029 for GPx).

Vitamin C was measured by the 2,4-dinitrophenylhydrazine method using a Roche Cobas c311 analyser (Roche Diagnostics, Mannheim, Germany).

Vitamin D (25-hydroxyvitamin D) was measured by electrochemiluminescence immunoassay (ECLIA) on a Roche Cobas e411 analyser (Roche Diagnostics). The assay detects both D2

and D3 forms. Results were reported in IU/L (conversion factor: 1 IU/L = 2.5 nmol/L).
All assays were performed blinded to the participant group.

Statistical analysis
Data were analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro–Wilk test and were presented as mean ± standard deviation (SD). Comparisons between patients and controls were performed using independent t-tests (or the Mann–Whitney U test for non-normal distributions). Comparisons across cancer stages (I–IV) used one-way ANOVA followed by Tukey's post hoc test for multiple comparisons. Correlations between biomarkers were assessed using Pearson's (or Spearman's) correlation coefficient. A two-tailed p-value <0.05 was considered statistically significant. No formal sample size calculation was performed because of the exploratory nature of the study; the sample size was based on consecutive enrolment during the study period.

Results

Demographic and clinical characteristics
The mean age of breast cancer patients was 47.5 ± 9.1 years, and that of controls was 45.3 ± 7.0 years (p=0.114). The majority of patients (43.3%) were aged 50–60 years, followed by 40–50 years (27.8%). Table 1 summarises the sociodemographic characteristics. Patients were less likely to be employed (24% vs. 58%, p<0.001) and more likely to live in urban areas (79% vs. 78%, p<0.001) compared with controls. These differences reflect the underlying population distribution rather than matching failures.

Table 1: Sociodemographic characteristics of breast cancer patients and healthy controls

Variable	Patients (n=90) n (%)	Controls (n=45) n (%)	p-value*
Age <40 years	55 (61)	31 (68)	0.114
Age ≥40 years	35 (39)	14 (32)	
Employed	22 (24)	26 (58)	<0.001
Housewife	68 (76)	19 (42)	
Rural residence	19 (21)	10 (22)	<0.001
Urban residence	71 (79)	35 (78)	
Married	62 (69)	30 (67)	<0.001
Unmarried	28 (31)	15 (33)	
Upper-lower economic status	67 (74)	29 (64)	<0.001
Low economic status	33 (36)	16 (36)	

*Chi-square test; p-values reflect differences in distributions, but age was matched (p=0.114 for age comparison)

Comparison of oxidative stress and antioxidant markers between groups
Table 2 presents the serum levels of all measured biomarkers. Breast cancer patients had significantly lower vitamin C (mean difference –0.32 mg/dL, 95% CI –0.41 to –0.23, p<0.001) and vitamin D (mean difference –10.07 IU/L, 95% CI –11.9 to –8.2, p<0.001) compared with controls. MDA was significantly higher in patients (mean difference 8.07 ng/mL, 95% CI 5.9 to 10.2, p<0.001). Both SOD and GPx were also lower in patients, though the difference was modest for SOD (p=0.022) and more pronounced for GPx (p<0.001).

Table 2: Biomarker levels in breast cancer patients and healthy controls

Biomarker	Patients (n=90) Mean ± SD	Controls (n=45) Mean ± SD	Mean difference (95% CI)	p-value*
Vitamin C (mg/dL)	1.00 ± 0.20	1.32 ± 0.23	–0.32 (–0.41, –0.23)	<0.001
Vitamin D (IU/L)	13.19 ± 4.35	23.26 ± 5.18	–10.07 (–11.9, –8.2)	<0.001
MDA (ng/mL)	26.27 ± 6.34	18.20 ± 5.76	8.07 (5.9, 10.2)	<0.001
SOD (ng/mL)	151.8 ± 18.9	160.0 ± 20.9	–8.2 (–15.2, –1.2)	0.022
GPx (U/mL)	0.80 ± 0.27	1.06 ± 0.25	–0.26 (–0.36, –0.16)	<0.001

*Independent t-test

Biomarkers across breast cancer stages
Table 3 shows the one-way ANOVA results for biomarker levels according to cancer stage. Vitamin D decreased progressively from stage I (14.9 ± 4.1 IU/L) to stage IV (10.4 ± 3.9 IU/L), with a significant overall F-test (p=0.006) (Figure 1). Post-hoc analysis revealed significant differences between stage I and stage IV (p=0.003) and between stage II and stage IV (p=0.02). Vitamin C levels did not differ significantly across stages (p=0.332). MDA showed significant variation (p<0.001), with the

highest mean in stage II (28.5 ± 5.9 ng/mL) and slightly lower values in stages III and IV, though still elevated compared with controls (Figure 2). SOD was not significantly different across stages (p=0.262). GPx differed significantly (p=0.006), with lower values in advanced stages.

Table 3: One-way ANOVA of biomarkers across breast cancer stages

Biomarker	Stage I (n=20)	Stage II (n=35)	Stage III (n=19)	Stage IV (n=26)	F	p-value
Vitamin C (mg/dL)	1.05 ± 0.22	0.98 ± 0.18	1.02 ± 0.21	0.97 ± 0.20	1.155	0.332
Vitamin D (IU/L)	14.9 ± 4.1	14.1 ± 4.0	12.8 ± 4.2	10.4 ± 3.9	4.41	0.006
MDA (ng/mL)	24.0 ± 5.8	28.5 ± 5.9	26.1 ± 6.2	25.8 ± 6.5	6.827	<0.001
SOD (ng/mL)	155 ± 19	149 ± 18	152 ± 20	153 ± 19	1.356	0.262
GPx (U/mL)	0.92 ± 0.25	0.78 ± 0.26	0.79 ± 0.28	0.75 ± 0.27	4.426	0.006

Data are mean ± SD. Tukey's post-hoc: Vitamin D: stage I vs. IV p=0.003, stage II vs. IV p=0.02; MDA: stage I vs. II p=0.008, stage I vs. III p=0.04, stage I vs. IV p=0.05; GPx: stage I vs. IV p=0.005

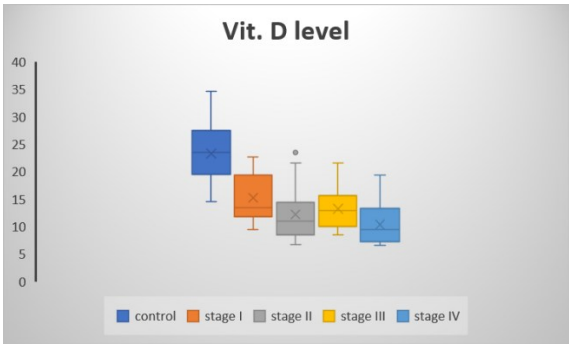

Figure 1: Serum vitamin D levels

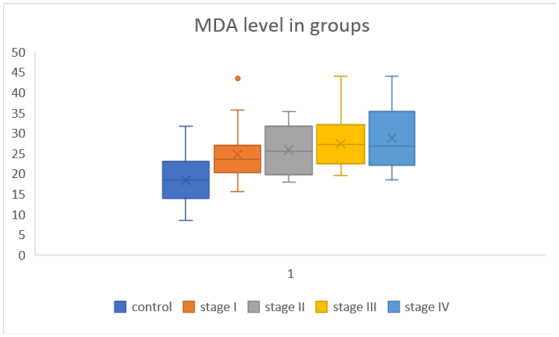

Figure 2: Serum MDA levels

Serum vitamin D levels decreased progressively from healthy controls (23.3 IU/L) to stage I (14.9), stage II (14.1), stage III (12.8), and stage IV (10.4 IU/L). Serum MDA levels were highest in stage II (28.5 ng/mL), followed by stage III (26.1), stage IV (25.8), and stage I (24.0); all were higher than controls (18.2 ng/mL)

Correlations among biomarkers

Pearson correlation analysis in the patient group (Table 4) revealed a moderate negative correlation between vitamin D and MDA (r = –0.45, p=0.009) (Figure 3). A negative correlation was also observed between MDA and SOD (r = –0.38, p=0.007). No significant correlations were found between vitamin C and other markers, or between age/BMI and the measured biomarkers, except for a weak positive correlation between BMI and vitamin D (r = 0.25, p=0.019).

Table 4: Correlation matrix for biomarkers in breast cancer patients (n=90)

Variable	Age	BMI	Vit C	Vit D	MDA	SOD	GPx
Age	1	–0.056	0.101	0.039	–0.027	0.046	0.006
BMI		1	–0.158	0.247*	–0.103	0.122	–0.033
Vit C			1	0.036	–0.021	0.018	–0.128
Vit D				1	–0.45**	0.054	0.15
MDA					1	–0.38**	–0.071
SOD						1	0.085

GPx	1

*p<0.05; **p<0.01. Correlation coefficients are Pearson's r

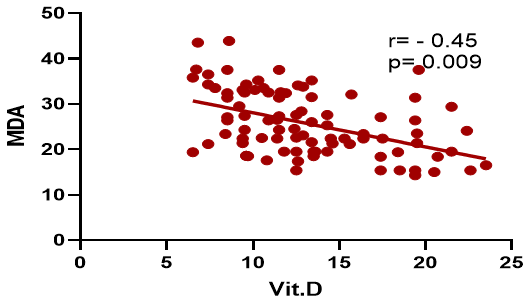

Figure 3: Scatter plot showing a negative linear relationship between serum vitamin D (IU/L) and MDA (ng/mL) in breast cancer patients (r = –0.45, p=0.009)

Discussion

This cross-sectional study of Iraqi women with breast cancer provides three main findings. First, breast cancer patients have significantly lower serum levels of antioxidant vitamins C and D and higher levels of the oxidative stress marker MDA compared with age-matched healthy controls. Second, vitamin D levels decline progressively with increasing cancer stage, whereas vitamin C levels do not show a stage-dependent pattern. Third, there is a moderate negative correlation between vitamin D and MDA, suggesting that lower vitamin D status is associated with greater lipid peroxidation.

Oxidative stress in breast cancer

The elevated MDA levels observed in our patients are consistent with a large body of evidence indicating increased lipid peroxidation in breast cancer (9, 16). MDA is a genotoxic aldehyde that can form adducts with DNA, potentially contributing to mutagenesis and tumour progression. The highest MDA levels in our study occurred in stage II disease, with a slight decline in stages III and IV. This pattern has been reported previously and may reflect several phenomena: advanced tumours may outgrow their blood supply, creating hypoxic regions with altered redox balance; alternatively, aggressive cancers might upregulate certain antioxidant defences that reduce measurable MDA (6, 17). We also observed lower SOD and GPx activities in patients, suggesting a compromised antioxidant enzyme system. These enzymes are often downregulated or inactivated by oxidative stress, creating a vicious cycle of further damage (8).

Vitamin C and breast cancer

Our finding of lower vitamin C in breast cancer patients aligns with meta-analyses showing an inverse association between dietary vitamin C intake and breast cancer risk (10, 18). Vitamin C's antioxidant properties are well established: it scavenges superoxide and hydroxyl radicals, protects DNA from oxidative damage, and regenerates vitamin E and glutathione (11). However, the lack of a significant difference in vitamin C levels across cancer stages suggests that while vitamin C may influence susceptibility, it may not strongly affect disease progression once cancer is established. Clinical trials of high-dose intravenous vitamin C in combination with chemotherapy have shown mixed results, though preclinical studies indicate that pharmacological vitamin C can enhance immunotherapy responses (19). The modest reduction in serum vitamin C in our patients (mean 1.00 mg/dL) is unlikely to represent severe deficiency (which is <0.2 mg/dL) but may still indicate suboptimal antioxidant reserve.

Vitamin D as a stage-dependent biomarker

The progressive decline in vitamin D from stage I to stage IV is one of the most striking findings of this study. Patients with stage IV disease had a mean vitamin D level of only 10.4 IU/L (equivalent to ~26 nmol/L), which is well below the threshold for vitamin D deficiency (<50 nmol/L or 20 ng/mL). This inverse association between vitamin D and breast cancer stage has been reported in several large observational studies (14, 15, 20). The biological plausibility is strong: the vitamin D receptor (VDR) is expressed in normal breast epithelium and in many breast cancers. Active vitamin D (1,25-dihydroxyvitamin D) regulates genes involved in cell cycle arrest (p21, p27), apoptosis (BAX, BCL-2), and inhibition of angiogenesis (VEGF) (12, 13). Vitamin D also modulates oxidative stress by upregulating antioxidant enzymes and reducing pro-oxidant iron availability (21, 22). The negative correlation between vitamin D and MDA observed here supports the hypothesis that vitamin D deficiency exacerbates oxidative damage, which in turn may fuel cancer progression.

Nevertheless, causality cannot be determined from a cross-sectional design. It remains possible that advanced cancer causes vitamin D depletion through poor nutritional intake, reduced sun exposure, or altered metabolism. Prospective studies and randomised trials of vitamin D supplementation are needed to test whether correcting deficiency improves breast cancer outcomes. A recent meta-analysis of randomised trials suggested that vitamin D supplementation does not reduce cancer incidence, but most trials used low doses and had short follow-up (23).

Correlation between vitamin D and MDA

The moderate negative correlation (r = –0.45) between vitamin D and MDA indicates that lower vitamin D is associated with higher lipid peroxidation. This aligns with experimental data showing that vitamin D reduces oxidative stress by suppressing NADPH oxidase activity, enhancing glutathione synthesis, and reducing labile iron (21, 22). The correlation was not extremely strong (r = –0.45), which is expected given that oxidative stress is influenced by many factors beyond vitamin D, including diet, smoking, obesity, and genetic polymorphisms.

Comparison with other Iraqi studies

Our finding that Iraqi breast cancer patients have low vitamin D is particularly relevant given the high ambient sunshine in Iraq. Paradoxically, vitamin D deficiency is common in Middle Eastern countries because of cultural practices that limit sun exposure (covering clothing, indoor lifestyle) and darker skin pigmentation (24). A previous study from Baghdad reported that 78% of breast cancer patients had vitamin D deficiency, with lower levels associated with higher tumour grade (3). Our data extend these observations by showing a stage-dependent decline. To our knowledge, this is the first Iraqi study to simultaneously measure MDA and antioxidant vitamins in breast cancer patients across all stages.

Study limitations and future directions

Several limitations must be acknowledged. First, the cross-sectional design precludes causal inference; we cannot determine whether low vitamin D contributes to cancer progression or vice versa. Second, the study was conducted at a single centre in Kirkuk, which may limit generalisability to other Iraqi regions or ethnic groups. Third, we did not measure dietary intake of vitamins or sun exposure, both of which influence vitamin D status. Fourth, the control group was not screened by mammography, so subclinical breast cancer cannot be excluded, though this is unlikely given the low prevalence in this age group. Fifth, vitamin C is unstable and may degrade during storage at –20 °C; although we processed samples rapidly, some loss may have occurred. Sixth, we did not measure parathyroid hormone or calcium, which could help interpret vitamin D status. Seventh, the sample size was based on convenience rather than a formal power calculation, and some subgroup analyses (e.g., stage I vs. stage III) may have been underpowered.

Future studies should adopt a prospective cohort design, measuring vitamin D and oxidative stress markers before cancer diagnosis and following patients longitudinally. Randomised controlled trials of vitamin D supplementation in women with early-stage breast cancer, with endpoints of disease-free survival and quality of life, would be valuable. Additionally, research on genetic variants in the VDR and vitamin D metabolic pathway in Iraqi populations could identify high-risk groups.

Conclusion

This study demonstrates that Iraqi women with breast cancer have significantly higher levels of oxidative stress (MDA) and significantly lower levels of antioxidant vitamins C and D compared with healthy controls. Serum vitamin D levels decline progressively from stage I to stage IV, and vitamin D shows a moderate negative correlation with MDA, suggesting a link between vitamin D deficiency and increased lipid peroxidation. Vitamin C, although lower in patients, does not vary by cancer stage. These findings support the potential role of vitamin D as a biomarker of breast cancer progression and raise the hypothesis that maintaining adequate vitamin D status might slow disease advancement. However, because of the cross-sectional design, causality remains unproven. Large-scale prospective studies and clinical trials are urgently needed, particularly in Middle Eastern populations where vitamin D deficiency is endemic, and breast cancer presents at a younger age.

List of Abbreviations

AJCC: American Joint Committee on Cancer
BMI: Body mass index
CV: Coefficient of variation
DNA: Deoxyribonucleic acid
ECLIA: Electrochemiluminescence immunoassay
ELISA: Enzyme-linked immunosorbent assay
GPx: Glutathione peroxidase
MDA: Malondialdehyde
ROS: Reactive oxygen species
SOD: Superoxide dismutase
SPSS: Statistical Package for the Social Sciences
VDR: Vitamin D receptor

WHO: World Health Organisation

Declarations

Ethics Approval and Consent to Participate

This study was approved by the Scientific Committee of the College of Medicine, University of Tikrit (approval number TUC-M-2025-089, dated 15 November 2025). All procedures performed were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent was obtained from all individual participants included in the study.

Consent for Publication

All authors gave consent for the publication of this work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data Availability

The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

MNR worked on the conceptualisation, investigation, data curation, writing the original draft and visualisation.

HNN worked on the methodology, validation, formal analysis, supervision, writing the review and editing.

AMM worked on the resources, clinical assessment, patient recruitment, writing the review and editing.

Acknowledgements

The authors thank the staff of the Kirkuk Oncology Centre, especially the nursing and laboratory teams, for their assistance with patient recruitment and sample collection. We also thank all the women who participated in this study for their time and cooperation.

References

1. Freihat O, Sipos D, Kovacs A. Global burden and projections of breast cancer incidence and mortality to 2050: a comprehensive analysis of GLOBOCAN data. Frontiers in Public Health. 2025 Oct 30;13:1622954. https://doi.org/10.3389/fpubh.2025.1622954
2. Bowen DJ, Fernandez Poole S, White M, Lyn R, Flores DA, Haile HG, Williams DR. The role of stress in breast cancer incidence: risk factors, interventions, and directions for the future. International Journal of Environmental Research and Public Health. 2021 Feb 15;18(04):1871. https://doi.org/10.3390/ijerph18041871
3. Al-Hashimi MM. Trends in breast cancer incidence in Iraq during the period 2000-2019. Asian Pacific journal of cancer prevention: APJCP. 2021 Dec;22(12):3889. https://doi.org/10.31557/APJCP.2021.22.12.3889
4. Iraqi Ministry of Health. Annual Cancer Registry Report 2024. Baghdad: Ministry of Health; 2024.
5. Patro VJ, Sheela E, Srinavya M, Jyothika A, Sailaja M. A review on pathophysiology of breast cancer. Int J Pharm Sci Drug Anal. 2023;3(1):94-97.
6. Mdkhana B, Goel S, Saleh MA, Siddiqui R, Khan NA, Elmoselhi AB. Role of oxidative stress in angiogenesis and the therapeutic potential of antioxidants in breast cancer. European Review for Medical & Pharmacological Sciences. 2022 Jul 1;26(13).
7. Kirtonia A, Sethi G, Garg M. The multifaceted role of reactive oxygen species in tumorigenesis. Cellular and Molecular Life Sciences. 2020 Nov;77(22):4459-83. https://doi.org/10.1007/s00018-020-03536-5
8. Nass N, Sel S, Ignatov A, Roessner A, Kalinski T. Oxidative stress and glyoxalase I activity mediate dicarbonyl toxicity in MCF-7 mamma carcinoma cells and a tamoxifen resistant derivative. Biochimica et Biophysica Acta (BBA)-General Subjects. 2016 Jun 1;1860(6):1272-80. https://doi.org/10.1016/j.bbagen.2016.03.006
9. Saha SK, Lee SB, Won J, Choi HY, Kim K, Yang GM, Abdal Dayem A, Cho SG. Correlation between oxidative stress, nutrition, and cancer initiation. International Journal of Molecular Sciences. 2017 Jul 17;18(7):1544. https://doi.org/10.3390/ijms18071544
10. Zhang D, Xu P, Li Y, Wei B, Yang S, Zheng Y, Lyu L, Deng Y, Zhai Z, Li N, Wang N. Association of vitamin C intake with breast cancer risk and mortality: a meta-analysis of observational studies. Aging (Albany NY). 2020 Sep 29;12(18):18415. https://doi.org/10.18632/aging.103769
11. Gęgotek A, Skrzydlewska E. Antioxidative and anti-inflammatory activity of ascorbic acid. Antioxidants. 2022 Oct 7;11(10):1993. https://doi.org/10.3390/antiox11101993

12. Carlberg C. Vitamin D and its target genes. Nutrients. 2022 Mar 24;14(7):1354. https://doi.org/10.3390/nu14071354
13. Welsh J. Vitamin D and breast cancer: mechanistic update. Journal of Bone and Mineral Research Plus. 2021 Dec 1;5(12):e10582. https://doi.org/10.1002/jbm4.10582
14. Atoum M, Alzoughool F. Vitamin D and breast cancer: latest evidence and future steps. Breast Cancer: Basic and Clinical Research. 2017 Dec 19;11:1178223417749816. https://doi.org/10.1177/1178223417749816
15. Ooi LL, Zhou H, Kalak R, Zheng Y, Conigrave AD, Seibel MJ, Dunstan CR. Vitamin D deficiency promotes human breast cancer growth in a murine model of bone metastasis. Cancer Research. 2010 Mar 1;70(5):1835-44. https://doi.org/10.1158/0008-5472.CAN-09-3194
16. Hu F, Wu Z, Li G, Teng C, Liu Y, Wang F, Zhao Y, Pang D. The plasma level of retinol, vitamins A, C and α-tocopherol could reduce breast cancer risk? A meta-analysis and meta-regression. Journal of Cancer Research and Clinical Oncology. 2015 Apr;141(4):601-14. https://doi.org/10.1007/s00432-014-1852-7
17. Shashni B, Nishikawa Y, Nagasaki Y. Management of tumor growth and angiogenesis in triple-negative breast cancer by using redox nanoparticles. Biomaterials. 2021 Feb 1;269:120645. https://doi.org/10.1016/j.biomaterials.2020.120645
18. Sram RJ, Binkova B, Rossner Jr P. Vitamin C for DNA damage prevention. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis. 2012 May 1;733(1-2):39-49. https://doi.org/10.1016/j.mrfmmm.2011.12.001
19. Magrì A, Germano G, Lorenzato A, Lamba S, Chilà R, Montone M, Amodio V, Ceruti T, Sassi F, Arena S, Abrignani S. High-dose vitamin C enhances cancer immunotherapy. Science Translational Medicine. 2020 Feb 26;12(532):eaay8707. https://doi.org/10.1126/scitranslmed.aay8707
20. de La Puente-Yagüe M, Cuadrado-Cenzual MA, Ciudad-Cabañas MJ, Hernández-Cabria M, Collado-Yurrita L. Vitamin D: And its role in breast cancer. The Kaohsiung Journal of Medical Sciences. 2018 Aug 1;34(8):423-7. https://doi.org/10.1016/j.kjms.2018.03.004
21. Bajbouj K, Sahnoon L, Shafarin J, Al-Ali A, Muhammad JS, Karim A, Guraya SY, Hamad M. Vitamin D-mediated anti-cancer activity involves iron homeostatic balance disruption and oxidative stress induction in breast cancer. Frontiers in Cell and Developmental Biology. 2021 Nov 8;9:766978. https://doi.org/10.3389/fcell.2021.766978
22. Tahmasebi M, Veissi M, Hosseini SA, Jamshidnezhad A. Effect of vitamin D supplementation on inflammatory markers and total antioxidant capacity in breast cancer women using a machine learning technique. Exploration of Targeted Anti-tumor Therapy. 2023 Oct 30;4(5):1059. https://doi.org/10.37349/etat.2023.00180
23. Manson JE, Cook NR, Lee IM, Christen W, Bassuk SS, Mora S, Gibson H, Gordon D, Copeland T, D'Agostino D, Friedenberg G. Vitamin D supplements and prevention of cancer and cardiovascular disease. New England Journal of Medicine. 2019 Jan 3;380(1):33-44. https://doi.org/10.1056/NEJMoa1809944
24. Bassil D, Rahme M, Hoteit M, Fuleihan GE. Hypovitaminosis D in the Middle East and North Africa: prevalence, risk factors and impact on outcomes. Dermato-endocrinology. 2013 Apr 1;5(2):274-98. https://doi.org/10.4161/derm.25111