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RESEARCH ARTICLE OPEN ACCESS

Expression and prognostic significance of MMP9, RANKL, CXCL12, and CXCR4 in Iraqi women with breast cancer

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Abstract
Objective: Breast cancer remains a global health burden. The tumour microenvironment plays a key role in invasion and metastasis, partly through the activity of molecules such as MMP9, RANKL, and the CXCL12/CXCR4 axis.
Methods: Blood samples from the periphery were taken from 60 women with breast tumours (30 with primary breast cancers and 30 with benign lesions). The RNA was extracted and then the gene expression levels of MMP9, RANKL, CXCL12 and CXCR4 were measured by real-time PCR. The statistical analysis was performed with SPSS version 26. Comparisons between the groups were determined by Student's t-test, and $p < 0.05$ was the threshold for statistical significance.
Results: The mRNA expression levels of MMP9, RANKL, CXCL12, and CXCR4 were significantly higher in the cases of primary breast cancer compared to those having benign lesions with a *p-value* less than 0.001 for all markers. The mean ± SD expression of CXCL12 was 2.45 ± 1.31 in the cancer group and 0.78 ± 0.75 in the benign group. Along with this, the expression of all four markers was increased in a stepwise fashion with tumour stage and they were significantly correlated with large tumour size (≥3 cm), high histological grade, and lymph node metastasis.
Conclusion: The Iraqi women with breast cancer showed elevated levels of MMP9, RANKL, CXCL12, and CXCR4 expression in tumours, as well as their correlation with advanced clinicopathological features indicates that they may serve as prognostic biomarkers.

Keywords: Breast cancer, Prognosis, benign tumour, stage, MMP9, RANKL, CXCL12, CXCR4

Plain English Summary

This study looked at four genes, MMP9, RANKL, CXCL12, and CXCR4, that may influence how breast cancer grows and spreads. We compared their activity in women with breast cancer and women with benign breast lumps. We found that all four genes were much more active in women with breast cancer. Their activity also increased as the cancer became more advanced, especially in women whose tumours were larger or had spread to the lymph nodes. These findings suggest that these genes may help doctors

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understand how aggressive a tumour is. In the future, they may also help in developing new tests or treatments for breast cancer.

Introduction

Breast cancer (BC) is recognised as the most common form of cancer diagnosed in women globally, and it still accounts for a large share of cancer-related deaths. Recent GLOBOCAN estimates for 2022 place the number of newly diagnosed cases at around 2.3 million worldwide, with an ongoing increase in case numbers in numerous low and middle-income regions (1).

In Iraq, breast cancer is the leading malignancy among women, and its role in female cancer mortality and incidence is significant. This situation underlines the need for enhanced molecular and prognostic research in Iraqi populations as a matter of national public health.

The invasion and metastasis of tumours are the results of complicated interactions between the tumour and the surrounding environment, one of the main factors being the proteolytic remodelling of the extracellular matrix (ECM). The gelatinase matrix metalloproteinase-9 (MMP-9) is implicated in the breakdown of the components of the basement membrane and thus promotes the spread of tumour cells; its high levels, both in the tumour and in circulation, are often linked to the presence of metastases in the lymph nodes and to an unfavourable prognosis in the case of breast cancer (2, 3).

Though the receptor activator of nuclear factor-κB ligand (RANKL)–RANK signalling pathway has been primarily investigated in bone biology and osteoclastogenesis, its connection to breast tumour biology is undeniable. RANKL signalling not only enhances mammary epithelial proliferation and migration but also aids in the establishment of pro-metastatic niches. Moreover, it has been involved in the mechanistic explanation of breast cancer progression and bone metastasis. These characteristics make the RANKL–RANK pathway a biomarker of great interest and a possible therapeutic target at the same time (4, 5).

Chemokine signalling through CXCL12 (stromal cell-derived factor-1) and its main receptor CXCR4 regulates the movement and localisation of cells; in breast cancer, the CXCL12–CXCR4 pathway facilitates tumour cell survival, invasion and organ-specific metastasis and was drawn as a prognostic marker as well as a therapeutic target in recent translational research (6, 7).

Although extensive mechanistic and clinical data exist from many regions, molecular evidence from Iraq remains limited. Previous local reports have shown increased expression of markers such as MMP9 and alterations in the CXCL12/CXCR4 pathway in breast cancer. However, no comprehensive studies have evaluated the circulating peripheral-blood mRNA levels of these markers alongside detailed clinicopathological correlations in Iraqi women (8, 9).

Materials and Methods

Study Design

The current case-control research encompassed 60 Iraqi females as patients who went to the Oncology Teaching Hospital and Al-Yarmouk Teaching Hospital in Baghdad from January to October 2023. The patients were classified into two groups: one containing 30 women with histopathologically confirmed primary breast carcinoma (no surgery or treatment was done prior) and another with 30 women having benign breast lesions (like fibroadenoma or fibrocystic changes). The benign and malignant groups were matched by age and menopausal status to minimise confounding. The mean age in the benign group was 47.1 ± 8.9 years, compared with 48.6 ± 9.4 years in the cancer group, and the proportions of pre- and post-menopausal women were similar (18/12 vs. 17/13, respectively). No significant differences were observed between the two groups ($p > 0.05$).

Sample Collection and Processing

Peripheral venous blood, with a volume of five millilitres (5 *mL*), was aseptically drawn from each participant and collected into EDTA tubes before any surgery, biopsy, or treatment. The samples were placed on ice and directly sent to the molecular biology laboratory, where they were processed within two hours of collection. The whole-blood samples were divided, and their portions were kept *at −80 °C* until the extraction of RNA was done.

RNA Extraction and Real-Time PCR

The GeneJET RNA Purification Kit (Thermo Fisher Scientific, USA) was used to extract total RNA from 200 µL of whole blood, taking into account the manufacturer's instructions. A NanoDrop spectrophotometer (Thermo Fisher Scientific) was used to measure the RNA purity and concentration at 260/280 nm.

The cDNA synthesis was accomplished with the RevertAid First Strand cDNA Synthesis Kit (Thermo Fisher Scientific). Quantitative real-time PCR was then performed to assess the gene

expression of MMP9, RANKL, CXCL12, and CXCR4 using SYBR Green Master Mix (Applied Biosystems, USA) on an Applied Biosystems StepOne system.

The β-actin gene served as the endogenous control. Relative gene expressions were calculated using the $2^{-\Delta\Delta Ct}$ method, in which ΔCt = (Ct_target − Ct_β-actin). Each reaction was performed three times to obtain more accurate results.

Inclusion and Exclusion Criteria

Inclusion criteria consisted of adult female patients (>18 years) whose primary breast malignancy had been confirmed by histopathology or those with benign breast tumours confirmed by needle aspiration cytology or biopsy. Patients who had undergone prior chemotherapy, radiotherapy or hormonal therapy were excluded, as well as those with co-existing malignancies, chronic inflammatory conditions, autoimmune diseases, or recent infections that might influence circulating RNA expression profiles.

Statistical Analysis

SPSS version 26 (IBM Corp., Armonk, NY, USA) and GraphPad Prism version 9 were the software packages used to conduct all the statistical analyses. The continuous variables were presented as mean ± standard deviation (SD). The differences between groups were tested by applying either the Student's t-test or the one-way ANOVA, depending on the situation.

Pearson's correlation or Chi-square tests were used to evaluate the correlations between gene expression levels and clinicopathological parameters (tumour size, grade, lymph node status, and hormonal receptors).

In the case of expression comparisons, a *p-value* of less than 0.05 was taken as evidence of statistical significance.

The cut-off points that separated "high" from "low" gene expression were set at the median expression value for each gene within the breast cancer group.

Results

General Characteristics of the Study Population

The research involved 60 female participants who were randomly allocated to two groups of 30 each: one group consisted of patients diagnosed with primary breast cancer, while the other group patients with benign breast lesions. The two groups were comparable regarding age and menopausal status ($p > 0.05$). Among the patients with diagnosed cancer, 43.3% were rated as stage I, 36.7% as stage II, and 20% as stage III. Lymph node metastasis was observed in 46.7% of the breast cancer patients. Table 1 presents the clinicopathological features in summary.

Descriptive characteristics of women included in the study. The benign group consisted mainly of fibroadenoma and fibrocystic lesions. No significant age or menopausal status differences were observed between groups ($p > 0.05$).

Table 1: Clinicopathological characteristics of the study population (n = 60)

Variable	Benign group (n = 30)	Breast cancer group (n = 30)	*p*-value
Age (years), mean ± SD	47.1 ± 8.9	48.6 ± 9.4	0.56
Menopausal status (Pre/Post)	18/12	17/13	0.80
Tumour size <3 cm / ≥3 cm	–	12 / 18	–
Histological grade (I / II / III)	–	8 / 13 / 9	–
Lymph node metastasis (Yes / No)	–	14 / 16	–

MMP9 Expression

MMP9 mRNA expression was found to be markedly elevated in the primary breast cancer patients (mean ± SD: 2.87 ± 1.24) as compared to those with benign breast conditions (0.91 ± 0.63; $p < 0.001$). There was a gradual increase in the MMP9 expression with the progress of the clinical stage, and its level was significantly higher in the tumours that were ≥3cm, grade II–III carcinomas, and in lymph node metastasis cases (Table 2; Figure 1, Figure 2). Expression of *MMP9* was significantly higher in malignant breast lesions and increased with tumour size, histological grade, and lymph node involvement ($p < 0.05$).

Table 2: Comparison of MMP9 mRNA expression between groups and clinicopathological variables

Variable	Category	MMP9 (Mean ± SD)	*p*-value
Benign vs. Malignant	Benign	0.91 ± 0.63	<0.001
	Malignant	2.87 ± 1.24	
Tumour size	<3 cm	2.34 ± 0.97	0.013

	≥3 cm	3.21 ± 1.18	
Grade	I–II	2.41 ± 1.05	0.027
	III	3.16 ± 1.26	
Lymph node metastasis	Negative	2.26 ± 0.91	0.009
	Positive	3.25 ± 1.14	

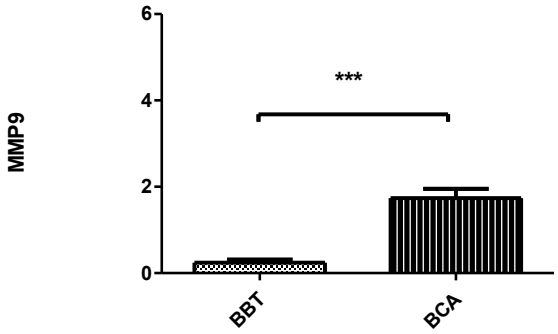


Figure 1: The MMP-9 mRNA fold expression in breast cancer and benign breast tumour patients

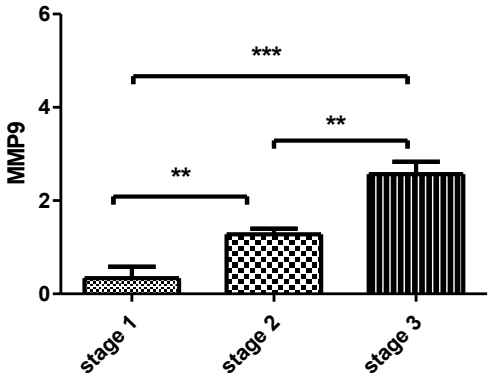


Figure 2: The MMP9 fold expression in different Breast Cancer stages

RANKL Expression

Breast cancer patients exhibited a pronounced increase in RANKL expression (2.52 ± 1.12) when compared to the benign group (0.85 ± 0.59; p < 0.001). Stage III tumours had the highest expression levels, demonstrating a distinct increase with the advancement of stage. Besides that, RANKL expression was also significantly linked with larger tumours, higher histological grades and lymph node positivity (Table 3; Figure 3; Figure 4). *RANKL* expression demonstrated a progressive rise with increasing tumour size, histological grade and nodal metastasis.

Table 3: Comparison of RANKL mRNA expression between groups and clinicopathological variables

Variable	Category	RANKL (Mean ± SD)	*p*-value
Benign vs. Malignant	Benign	0.85 ± 0.59	<0.001
	Malignant	2.52 ± 1.12	
Tumour size	<3 cm	2.11 ± 1.03	0.041
	≥3 cm	2.78 ± 1.17	
Grade	I–II	2.29 ± 0.91	0.034
	III	2.95 ± 1.22	
Lymph node metastasis	Negative	2.18 ± 0.88	0.018
	Positive	2.84 ± 1.19	

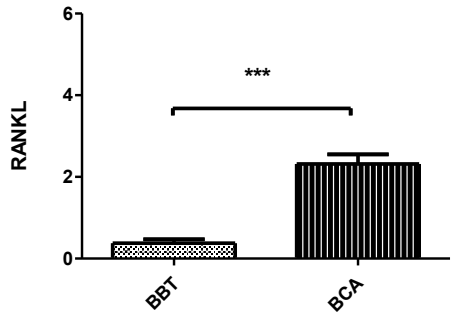


Figure 3: RANKL mRNA fold expression in breast cancer and benign breast tumour patients

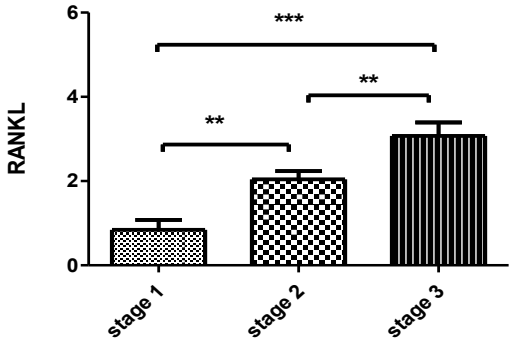


Figure 4: Comparison of the Fold of RANKL expression against Breast Cancer stage

CXCL12 Expression

The expression of CXCL12 was noticeably greater in the malignant group (2.45 ± 1.31) compared to the benign group (0.78 ± 0.75; $p < 0.001$). Moreover, the expression levels were found to correlate positively with tumour stage and grade, and to be higher in patients with nodal metastasis. Thus, the results suggest that the upregulation of CXCL12 is intimately associated with tumour progression (Table 4). *CXCL12* expression was significantly higher in breast cancer and correlated with indicators of disease progression.

Table 4: Comparison of CXCL12 mRNA expression between groups and clinicopathological variables

Variable	Category	CXCL12 (Mean ± SD)	*p*-value
Benign vs. Malignant	Benign	0.78 ± 0.75	<0.001
	Malignant	2.45 ± 1.31	
Tumour size	<3 cm	1.98 ± 0.86	0.021
	≥3 cm	2.64 ± 1.28	
Grade	I–II	2.12 ± 1.04	0.033
	III	2.69 ± 1.17	
Lymph node metastasis	Negative	2.03 ± 0.87	0.019
	Positive	2.73 ± 1.25	

CXCR4 Expression

In the breast cancer group, the CXCR4 mRNA expression was significantly increased (2.68 ± 1.17) in comparison to the benign group (0.95 ± 0.61; $p < 0.001$). The pattern of CXCR4 expression increased with tumour stage and grade and was conspicuously higher in the cases with positive nodes (Table 5, Figure 5, Figure 6). *CXCR4* levels were significantly elevated in malignant breast lesions and increased with tumour stage, grade, and lymph node involvement.

Table 5: Comparison of CXCR4 mRNA expression between groups and clinicopathological variables

Variable	Category	CXCR4 (Mean ± SD)	*p*-value
Benign vs. Malignant	Benign	0.95 ± 0.61	<0.001
	Malignant	2.68 ± 1.17	
Tumour size	<3 cm	2.21 ± 0.89	0.024
	≥3 cm	2.93 ± 1.15	
Grade	I–II	2.36 ± 0.93	0.031
	III	2.81 ± 1.18	
Lymph node metastasis	Negative	2.32 ± 0.96	0.015
	Positive	2.87 ± 1.11	

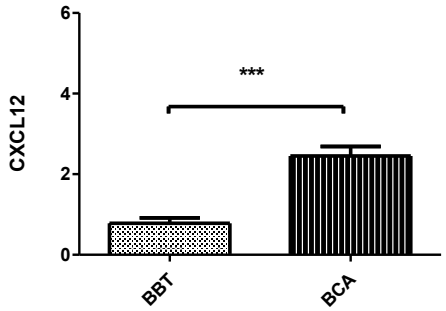


Figure 5: CXCL12 mRNA fold expression in breast cancer and benign breast tumour patients

As shown in Figure 6, CXCL12 was expressed stage-dependently, with the lowest levels found in Stage 1 and increasing to higher levels from Stages 2 to 3.

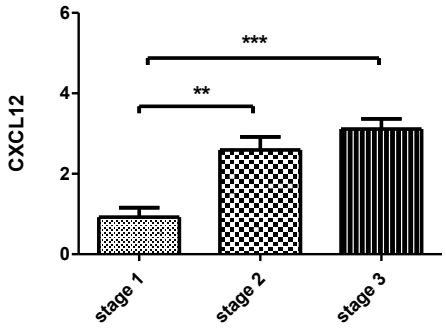


Figure 6: The fold of CXCL12 expression against Breast Cancer stage

Summary of Associations

The higher expression levels of all four biomarkers were consistently linked to the more aggressive characteristics of the tumour, such as larger tumour size, higher histological grade, and lymph node metastasis. The patient's age, menopausal status, or hormone receptor status did not show any notable correlation with gene expression.

Table 6 shows the most important clinicopathological features in breast cancer patients that are statistically significantly correlated with the expression of MMP9, RANKL, CXCL12, and CXCR4.

Table 6: Summary of significant associations between biomarkers and clinicopathological parameters

Biomarker	Tumour Size (≥3 cm)	Histological Grade (High)	Lymph Node Metastasis	*p*-value (overall)
MMP9	↑	↑	↑	<0.001
RANKL	↑	↑	↑	<0.001

CXCL12	↑	↑	↑	<0.001
CXCR4	↑	↑	↑	<0.001

↑ Indicates a statistically significant increase in mean expression relative to the reference subgroup.

Figure 7 demonstrates a markedly higher CXCR4 expression in BC patients compared to benign breast tumour patients.

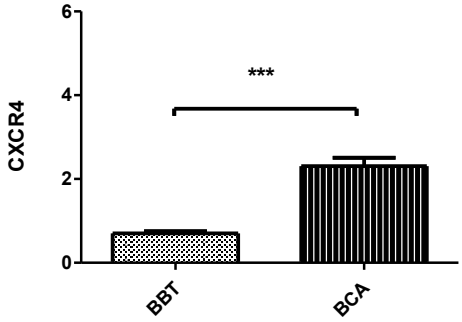


Figure 7: The mRNA expression of CXCR4 in a sample of breast cancer and benign breast tumour patients

Expression of CXCR4 increases progressively at increasing stages of cancer, lowest in stage 1 and highest in stage 3 in women with BC (Figure 8).

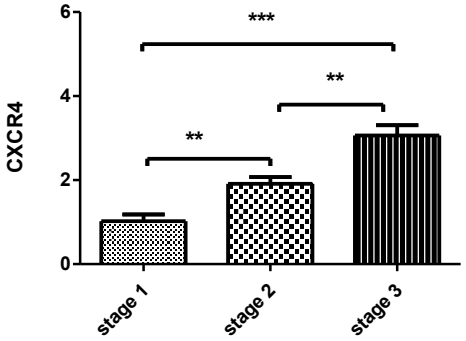


Figure 8: CXCR4 Fold expression against Breast Cancer stage.

Discussion

In the case-control study of Iraqi women with breast tumours, we found that MMP9, RANKL, CXCL12 and CXCR4 mRNA were upregulated consistently in the peripheral blood of patients with primary breast cancer when compared to benign lesions, and the expression of each marker was higher in the case of larger tumour size, higher histological grade and lymph node metastasis. Thus, these results imply that the markers' expression indicates tumour aggressiveness and suggest a systemic relationship between tumour biology and host factors.

The results we obtained for MMP-9 match the findings of several recent investigations that reported high levels of MMP-9 expression related to greater tumour cell invasiveness and poorer prognosis in breast cancer groups, MMP-9 being involved in lymphatic spread and in the occurrence of adverse clinicopathological features (10). Conversely, the rise in RANKL expression with tumour stage and node involvement agrees with studies that have identified the RANKL-RANK axis as a signalling pathway that might support not only the migration of cancer cells but also their stemness and the formation of the metastatic niche in breast cancer (5). Moreover, the concordant increase of CXCL12 and CXCR4 in our patient group is in line with the vast literature indicating that the CXCL12-CXCR4 chemokine axis not only

facilitates the attraction of breast cancer cells to certain sites, but also their survival and organ-specific metastasis, and is often associated with the disease's progression in human studies (6, 7).
The associations described in this report are consistent with biological processes. MMP-9 is an enzyme that breaks down extracellular matrix proteins and basement membranes, thereby making it easier for cancer cells to invade and find their way to lymphatic and blood vessel channels; besides, angiogenesis and growth factors released from the matrix are also the results of its enzymatic action, which further aids tumour maturation and development. RANKL can activate NF-κB and the pathways that follow, leading to increased migration and supporting cancer stem-like characteristics, thus making it easier for the cancer to spread. Tumour cells migrate toward metastatic sites by following concentration gradients of CXCL12, which guide their movement and promote their survival once they reach target organs. Therefore, the co-occurrence of increased MMP9, RANKL, and CXCL12/CXCR4 expression depicts the joint actions of mechanisms that are enabling the spread of cancer and the growth of secondary tumours.
The findings taken as a whole provide two important points that are clinically relevant. Firstly, the blood mRNA levels of MMP9, RANKL, CXCL12 and CXCR4 may be used as a non-invasive measure that combines the histopathological staging in the stratification of patients according to the risk of aggressive disease; their patterns of expression at different stages suggest that they may be useful in the prognosis or monitoring of the disease. Secondly, the findings encourage the use of these pathways as targets for treatment; for instance, RANKL inhibitors and CXCR4 antagonists are actively being studied as methods to reduce metastasis or to deal with resistance; however, the full integration of these approaches into standard clinical practice will hinge on the results of the prospective validation and interventional studies.

Strengths of the present study
The present research fills an existing void in molecular-epidemiological data from Iraq by analysing four different but complementary markers in the same group of patients and relating their varying levels in the blood to the already established clinical and pathological characteristics. Matching for age and menopause status in the study design, as well as the use of triplicate Quantitative real-time PCR measurements, also contribute to the internal reliability of the findings.

Study limitations.
Inevitably, there are several limitations to this research that must be recognised. First, the sample size is relatively small and the subjects are all from one centre, which poses a risk of limited extrapolation and lower power for statistical tests of subgroups. The study examined mRNA levels in the blood but did not do parallel tests for protein levels or tissue validation (using immunohistochemistry or ELISA), which would have made it easier to draw conclusions about functional pathway activation. The last limitation is that the cross-sectional nature of the study does not allow for causation to be inferred and does not evaluate the prognostic value of the markers for the long-term outcomes of recurrence or survival. These limitations should be seen as precursors for the design of larger, more robust, and longer-term prospective studies.

Recommendations and future directions.
Future research must substantiate these conclusions in larger, multicentre groups with longitudinal follow-up to assess if the initial or progressive alterations in the expression of the markers can forecast either recurrence or survival. The combination of mRNA data together with protein measurements and imaging or histopathological correlates would elucidate the connection between circulating signatures and intratumoural biology. Lastly, it would be possible to estimate the benefit of marker-guided therapy in terms of clinical outcomes through translational studies testing pathway-directed interventions (for instance, antagonists of CXCR4 or RANKL blockade) in patients with high expression profiles.

Conclusion
The investigation reveals that MMP9, RANKL, CXCL12, and CXCR4 have shown a significant increase in the peripheral blood of Iraqi females suffering from breast cancer when compared to those suffering from benign breast lesions. Their affirmative association with tumour size, histological grade, and lymph node metastasis indicates that these biomarkers may serve as prognostic indicators of disease severity. Nevertheless, large-scale and longitudinal studies are still needed to confirm their clinical and predictive value.

List of Abbreviations

BC: Breast cancer
MMP-9: Matrix metalloproteinase-9
RANKL: Receptor activator of nuclear factor-kappa B ligand
CXCL12: C-X-C chemokine ligand 12
CXCR4: C-X-C chemokine receptor type 4
HER-2: Human epidermal growth factor receptor
PR: Progesterone receptor
ER: Estrogen receptor

Declarations

Ethics approval and consent to participate

The Baghdad / Al-Karkh Health Department granted official approvals in compliance with administrative protocols for the collection of blood samples, biopsy, and patient data. Written informed consent was obtained from all participants. The study protocol was approved by the Institutional Ethics Committee of the Baghdad / Al-Karkh Health Directorate (Approval No. 1598/14-1-2024).

Consent for Publication

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

Availability of Data

The data and materials associated with this research will be made available by the corresponding author upon reasonable request.

Conflict of Interest

All of the authors declare that they have no competing interests.

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Authors' contributions

Zamin Al-Sarray: Data Collection, Laboratory Analysis, Statistical Analysis, Interpretation of Results, Drafting and Writing the Manuscript.
Izzat Al-Rayahi: Supervision, Guidance on Methodology, Conceptualisation, Experimental Design, Critical Review and Editing of the Manuscript, Final Approval of the Version to Be Published.
Hiba Naser: Co-supervised the study, assisted in experimental design, and participated in manuscript preparation and final approval.

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