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RESEARCH ARTICLE

Serum NOD–Like Receptor Protein 3 (NLRP3) Level as a Potential Diagnostic Biomarker in Iraqi Women with Breast Cancer

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Abstract

Introduction: Breast cancer (BC) is the leading cause of cancer-related mortality among women. The type of BC is determined by the specific cells in the breast that undergo malignant transformation. BC can originate in various region of the breast. Nod–Like Receptor Protein 3 (NLRP3) is the most well-studied inflammasome, expressed mainly by myeloid lineage cells. The study aimed to assess NLRP3 serum levels in Iraqi women with BC, evaluating its diagnostic and prognostic value, and investigated its correlation with pathological features. **Materials and Methods:** A case-control study was consisted of 90 Iraqi women newly diagnosed with breast cancer. A control group of 90 healthy women, selected to match the patients in age, was also included. Serum NLRP3 levels was measured using enzyme-linked immunosorbent assay (ELISA). Additionally, lipid profiles levels were assessed. Statistical analysis was performed using SPSS software. **Results:** Serum NLRP3 levels were significantly higher in the breast cancer patient group compared to the healthy control group (6.149 ± 0.643 vs. 2.666 ± 1.200 ng/mL, $P < 0.0001$). NLRP3 levels were particularly elevated in advanced stages of the disease. Lipid levels, including triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C), were also elevated in patients, while high-density lipoprotein cholesterol (HDL-C) levels were decreased compared to healthy women. Elevated serum NLRP3 levels showed a significant positive correlation with age but non-significant with other parameters. NLRP3 illustrated remarkable diagnostic efficacy in breast cancer patients, with a NLRP3 cutoff value of 5.380 ng/ml, a sensitivity of 93.3% and a specificity of 93.3% (AUC 0.959, 95% confidence interval: 0.922-0.995; $P < 0.001$). **Conclusion:** Elevated levels of the NLRP3 in women with breast cancer have been identified as a significant predictor of disease prognosis and a valuable diagnostic tool, suggesting a possible role in disease progression. NLRP3 may be used as a potential biomarker and immunotherapeutic target in breast cancer patients.

Keywords: Breast cancer- NLRP3- Metastasis

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Introduction

Breast cancer is one of the most common cancers among women. It is a complex disease, comprising different types with varying biological characteristics and clinical presentations [1]. The inflammatory cytokines contribute to the development of systemic low-grade inflammation, and aberrant NLRP3 activation can drive a chronic inflammatory state in the body to modulate the pathogenesis of inflammation-associated diseases. Therefore, targeting NLRP3 or other signaling molecules downstream, such as caspase-1, IL-1β or IL-18, has the potential for great therapeutic benefit. However, NLRP3 inflammasome–mediated inflammatory cytokines play dual roles in mediating human disease. While they are detrimental in the pathogenesis of inflammatory and metabolic diseases, they have a beneficial role in numerous infectious diseases and some cancers [2]. NLRP3 activation within tumor cells in estrogen receptor-negative tumors, such as triple-negative BC (TNBC), promotes pre-metastatic niche formation and distant metastasis through IL-1β autocrine/paracrine loops [3]. NLRP3

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inflammasome can also cause apoptosis in macrophages, suggesting that NLRP3 inflammasome may mediate JNK-induced apoptosis, and the apoptotic function of NLRP3 inflammasome may be a reason for the conflicting results reported [4]. NLRP3 is activated primarily in innate immune cells, including macrophages, and by a variety of stimuli including pathogens and danger signals [5]. We also have shown that a high level of NLRP3 expression in cancer cells promotes more aggressive tumor features, such as enhanced proliferation and migration, compared to cancer cells that express a lower level of NLRP3 [6]. A secondary signal, such as adenosine 5′-triphosphate, is required to trigger the polymerization and formation of an active NLRP3 complex [7]. NLRP3 is a crucial pattern recognition receptor and plays pivotal roles in host defense against microbial pathogens. NLRP3 assembles a multi-protein complex termed inflammasome, which comprise the following molecules-NLRP3, apoptotic speck protein containing a caspase recruitment domain and Caspase-1 [8]. In previous studies, it was demonstrated that high glucose induced NLRP3 inflammasome activation in Tohoku Hospital Pediatrics-1 macrophages, thereby further aggravating insulin resistance [9, 10]. This clinical study evaluated the role of the NLRP3 protein in breast cancer patients, paving the way for innovative treatment strategies and improved treatment outcomes.

Materials and Methods

A total of 180 women aged 35 to 65 years were enrolled in the present study, and it was conducted from December 2024 to April 2025. The total participants were categorized into two groups: Ninety individuals newly diagnosed with breast cancer encompassing [all stages: stage I (n=23), stage II (n=27), stage III (n=24), and stage IV (n=16)]. Diagnosed were made by mammography and histopathological examination at the Hospital of Oncology and Hematology in Najaf Al-Ashraf, Iraq. Additionally, ninety healthy participants, matched by age served as controls. This study excluded individuals who were pregnant, afflicted chronic diseases, autoimmune disorders, microbial infections, smokers, using oral contraceptives or any hormonal medications, and undergoing surgery. Prior to sample collection for the study, ethical principles in accordance with Declaration of Helsinki were adhered. Patients granted both verbal and written consent before the collection of sample. The study methodology, as along with the subject information and consent form, received approval from a local Ethics Committee following clearance from the ethics commission (IRB) of the Faculty of Science, University of Kufa, Iraq (No. 37037, 4/11/2024). Additionally, approved by the hospital's administration the medical records encompass, demographic data, medical history, history of exposure, signs and symptoms, and laboratory findings.

Subsequently, Five milliliters of blood from each individual's veins were obtained. Gel tubes were utilized to transport the blood subsequent to a breast cancer diagnosis and prior to initiation of any treatment. The blood is centrifuged at 3000 rpm for ten minutes to separate the sera into four tubes (Eppendorf tubes). It is subsequently stored at -8°C until the examination. The serum concentrations of total cholesterol (TC), triglyceride (TG), and high-density lipoprotein cholesterol (HDL-C) were quantified using an enzymatic colorimetric approach with a commercial kit from Emclab (Germany). Level of low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) were calculated using Friedewald's formula. Serum NLRP3 was measured using enzyme-linked immunosorbent assay (ELISA) kits (Melsin, China. Assay range 1.25-40 ng/mL). Anthropometric measurements including age, weight (kg), height (cm). Furthermore, the body mass index was calculated by dividing weight in kilograms by the square of individual's in meters: BMI = (weight in kg) / (height in meters^2) [11].

Statistical Analysis

The Kolmogorov-Smirnov test was employed to analyze the distribution types of the results group. The student t-test was utilized to evaluated differences in scale variables across diagnostic categories. The analysis of variance (ANOVA) test was employed to assess the differences in scale variables among diagnostic groups. The results were presented as (mean ± standard deviation) for normally distributed values. The Pearson's correlation coefficients (r) assess the relationship between the scale variable and the parametric parameter to determine their correlation another variable. Statistical significance for all hypothesis tests was determined with p-values below 0.05 (two-tailed). Receiver operating characteristic (ROC) curves were utilized to assess the diagnostic efficacy of the evaluated biomarkers in diagnosing the disease. The cut-off values of the concentrations yield optimal sensitivity and specificity based The data was compiled and analyzed by using on the area under the curve (AUC). IBM's Statistical Package for Social Sciences, version 27 (SPSS, Chicago, Illinois, USA).

Results

The baseline characteristics of 90 breast cancer patients diagnosed for the first time, compared with 90 healthy women in the study group, are detailed in Table 1, which summarizes various clinicopathological parameters of the patients presented as (mean ± SD). The mean age at breast cancer diagnosis was years (range, 35-65 years). No significant difference was found between the study groups regarding mean age and BMI ($p < 0.2745$ and $p < 0.5823$, respectively). The NLRP3 level was significantly higher in the BC group compared to the control group (6.149 ± 0.643 vs. 2.666 ± 1.200 ng/mL, $P < 0.0001$; Table 1), respectively. In Table 2 and Figure 1, illustrates that serum NLRP3 levels significantly escalated with the progression of BC stages in women. However, no statistically significant differences were observed in mean age (between stages II and III, II and IV, and III and IV), body mass index (across all stages), TC (between stages III and IV), HDL-C (between stages III and IV), LDL-C (between stages II and III), or NLRP3

Table 1. Demographic and Clinical Data of Breast Cancer Patients' healthy Controls

parameters	Patients group (mean ± SD)	Controls group (mean ± SD)	P- value
Number	90	90	-------
Age (years)	48.788 ± 6.147	49.866 ± 7.017	p < 0.2745 (NS)
BMI (kg/m^2)	28.225± 3.467	28.612 ± 5.690	p < 0.5823 (NS)
TG (mg/dL)	211.054 ± 27.543	194.054 ± 31.142	p < 0.0001
TC (mg/dL)	230.308 ± 27.286	211.269 ± 32.322	p < 0.0001
HDL-C (mg/dL)	35.570 ± 5.291	49.527 ± 5.302	p < 0.0001
LDL-C (mg/dL)	152.527 ± 25.603	95.930 ± 29.567	p < 0.0001
VLDL-C (mg/dL)	42.210 ± 5.508	38.810 ± 6.228	p < 0.0001
NLRP3 (ng/mL)	6.149 ± 0.643	2.666 ± 1.200	p < 0.0001

Data displayed as mean ± SD, were SD denotes: Standard Deviation, NS: non-significant, BMI: Body Mass Index, TG: Triglyceride, TC: Total Cholesterol, HDL-C: High Density Lipoprotein-Cholesterol, LDL-C: Low density Lipoprotein-Cholesterol, VLDL-C: Very Low Density Lipoprotein-Cholesterol, NLRP3. Nod–Like Receptor Protein 3.

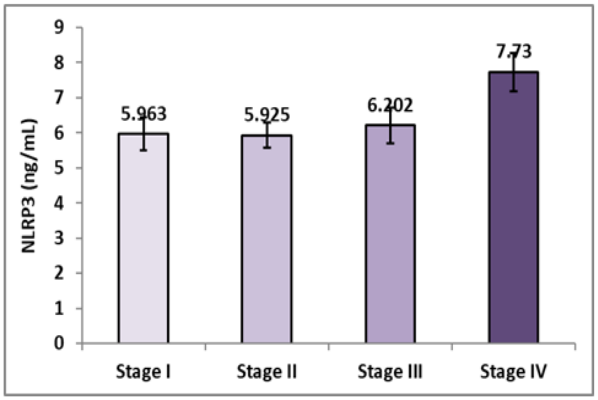


Figure 1. Comparison of Serum NLRP3 Levels in Patients' stages

protein (between stages I and II), apart from these, statistically significant differences were found between the stages. A significant difference was observed in TG between all stages. Table 3 shows the correlation among the measured parameters and variables. The results of this study indicated a positive correlation between NLRP3 and all the parameters examined, but it was not statistically significant except for age ($r = 0.22$ $p < 0.05$). Receiver operating characteristics (ROC) analysis was conducted to evaluate the efficacy of the measured NLRP3 in identifying BC patients by assessing its diagnostic sensitivity and specificity. Figure 2 illustrates the plotted ROC curves. Table 4 presents the coordinates of the ROC results along with the concentration cutoff that yields optimal sensitivities and specificities. The results indicates that NLRP3 levels exceeding the projected cut-off values suggest that subjects may have BC, with sensitivities of 93.3% and specificities of 93.3%, $p < 0.0001$ at a cut-off value of 5.380 ng/mL.

Data displayed as mean ± SD, were SD denotes: Standard Deviation, NS: non-significant, BMI: Body Mass Index, TG: Triglyceride, TC: Total Cholesterol, HDL-C: High Density Lipoprotein-Cholesterol, LDL-C: Low density Lipoprotein-Cholesterol, VLDL-C: Very Low Density Lipoprotein-Cholesterol, NLRP3. Nod–Like Receptor Protein 3.

Discussion

Breast cancer is a highly malignant tumor that poses a serious threat to women's health. According to global cancer statistics from 2020 [12], breast cancer has officially surpassed lung cancer as the most common type of cancer, exhibiting the highest incidence and mortality rates among cancers affecting women worldwide. Survival rates and prognostic factors varied across age groups, with older women having the lowest survival rates. Therefore, breast cancer treatment plans should be tailored to the age of the women diagnosed to promote better care and increase the chances of recovery [13]. NLRP3, the most studied type of inflammasome, consists of the NLRP3 sensor, the ASC transforming protein, and the caspase-1 effector protein. The NLRP3 inflammasome is widely present in immune cells, including granulocytes, dendritic cells, macrophages, epithelial cells and osteoblast [14]. Inflammasomes are multiprotein complexes that activate caspase-1, which leads to maturation of the proinflammatory cytokines interleukin 1β (IL-1β) and IL-18 and the induction of pyroptosis. Members of the Nod-like receptor (NLR) family, including NLRP1, NLRP3 and NLRC4, and the cytosolic receptor AIM2 are critical components of inflammasomes and link microbial and endogenous

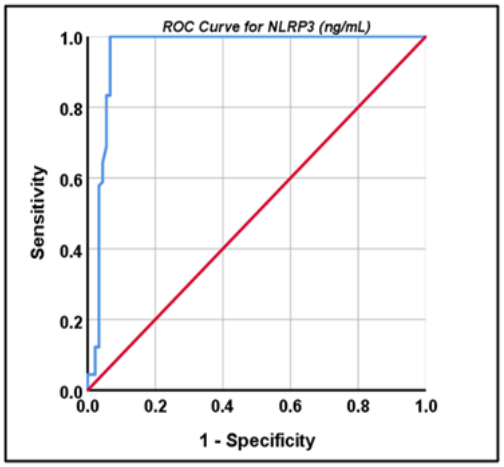


Figure 2. Receiver Operating Characteristic Curve for Breast Cancer Patients vs. Healthy Control

Table 2. Comparison of Variables between the Stages of Breast Cancer Patients

Variables	Patients Stages (No.= 90)				
	Stage I	Stage II	Stage III	Stage IV	p-value
Number (n%)	23 (25.555%)	27 (40%)	24 (26.666%)	16 (7.777%)	---------
Age (years)	44.043 ± 5.858	50.027 ± 5.848	50.875 ± 5.127	50.586 ± 4.059	a) 0.001 b) 0.001 c) 0.002 d) 0.351 e) 0 .687 h) 0.397
BMI (kg/m^2)	27.510 ± 3.167	28.594 ± 4.236	28.375 ± 2.719	28.154 ± 2.364	a) 0.251 b) 0.235 c) 0.853 d) 0.879 e) 0 .582 h) 0.533
TG (mg/dL)	207.718 ±27.964	198.35 ±19.206	223.286 ± 26.758	243.±26.745	a) 0.001 b) 0.001 c) 0.001 d) 0.001 e) 0.001 h) 0.001
TC (mg/dL)	225.321 ± 30.969	227.299 ± 27.627	237.180 ± 25.862	238.615±29.583	a) 0.05 b) 0.001 c) 0.001 d) 0.001 e) 0.001 h) 0.625
HDL-C (mg/dL)	35.572 ± 4.914	33.723 ± 4.736	38.927 ± 4.980	33.547 ± 5.525	a) 0.001 b) 0.01 c) 0.01 d) 0.001 e) 0.254 h) 0.001
LDL-C (mg/dL)	148.205 ± 28.118	153.828 ± 25.498	153.594 ± 26.590	156.382±34.759	a) 0.001 b) 0.001 c) 0.001 d) 0.489 e) 0.001 h) 0.01
VLDL-C (mg/dL)	41.543 ± 5.592	39.747 ± 3.841	44.657 ± 5.351	48.685 ± 5.349	a) 0.001 b) 0.01 c) 0.001 d) 0.001 e) 0.0001 h) 0.0001
NLRP3 (ng/mL)	5.963 ± 0.461	5.925 ± 0.360	6.202 ± 0.497	7.730±0.545	a) 0.562 b) 0.001 c) 0.0001 d) 0.001 e) 0.0001 h) 0.001

Data displayed as mean ± SD, were SD denotes: Standard Deviation, NS: non-significant, BMI: Body Mass Index, TG: Triglyceride, TC: Total Cholesterol, HDL-C: High Density Lipoprotein-Cholesterol, LDL-C: Low density Lipoprotein-Cholesterol, VLDL-C: Very Low Density Lipoprotein-Cholesterol, NLRP3. Nod–Like Receptor Protein 3.Significant differences between stages; a) Stage I vs. Stage II, b) Stage I vs. Stage III, c) Stage I vs. Stage IV, d) Stage II vs. Stage III, e) Stage II vs. Stage IV, h) Stage III vs. Stage IV.

danger signals to the activation of caspase-1 [15]. The activation of the NLRP3 inflammasome requires two steps. In the first step (initiation), microbial or endogenous molecules such as TLR ligands activate NF-κB, which in turn induces the transcription and production of pro-IL-1β, pro-IL-18, and NLRP3. In the second step (activation), the NLRP3 inflammasome recognizes various PAMPs and DAMPs from diverse stimuli, such as fungi [16]. Although the inflammasomes mainly exert their beneficial roles in the innate immune defense, aberrant activation of the inflammasomes and concurrent overexpression/overproduction of their effector molecules have been observed in several types of human malignancies [17]. In this context, several data suggested a role of NLRP3 activation in breast cancer development [18]. The NLRP3 inflammasome is a multiple-protein complex comprising NLRP3, apoptosis-associated speck-like protein containing a caspase recruitment domain (ASC), and caspase 1. The activation of this complex, in turn, activates caspase 1, which cleaves pro-IL-1β and pro-IL-18, generating the mature forms of these inflammatory cytokines, IL-1β and IL-18. There is considerable evidence suggesting the role of NLRP3 in tumor pathogenesis,

Table 3. Correlation between NLRP3 and Studied Parameters in Breast Cancer Patients

Parameters	r	p-value	Parameters	r	p-value
Age (years)	0.220*	0.037	HDL-C (mg/dL)	0.007	0.949
BMI (kg/m^2)	0.009	0.932	LDL-C (mg/dL)	0.151	0.156
TG (mg/dL)	0.201	0.058	VLDL-C (mg/dL)	0.201	0.058
TC (mg/dL)	0.183	0.084			

*. Correlation is significant at the 0.05 level (2-tailed). r: Pearson correlation coefficient.

Table 4. Receiver Operating Characteristic-area under Curve Analysis of the Diagnosis for Breast Cancer Patients vs. Healthy Control

Variables	NLRP3(ng/mL)
Area Under Curve (AUC)	0.959
Cut-off value	5.38
P-value	p < 0.0001
Specificity	93.30%
Sensitivity	93.30%
Confidence interval 95%	0.922-0.995

although results are often controversial. Some studies have shown that NLRP3 accelerates tumor progression by inducing epithelial-mesenchymal transition (EMT) and angiogenesis [19]. Overall, this research provides insight into the function and regulatory mechanisms of NLRP3 in BC, and may offers important clues for understanding the dynamic changes of TME in BC, furnishing new references as well as potential targets for the prevention, diagnosis, and treatment of BC [20]. Inflammasomes are intracellular multiprotein complexes in the cytoplasm that regulate inflammation activation in the innate immune system in response to pathogens and to host self-derived molecules. Recent advances greatly improved our understanding of the activation of nucleotide-binding oligomerization domain-like receptor (NLR) family pyrin domain containing 3 (NLRP3) inflammasomes at the molecular level [21]. The NLR family pyrin domain containing 3 (NLRP3) inflammasome, composed by NLRP3 oligomers and apoptosis-associated speck-like (ASC) adapter protein, is a key innate immune pathway through triggering the activation of caspase-1, leading to the processing of interleukin-1 beta (IL-1β) and IL-18, as well as inducing of pyroptosis and malignant transformation [19]. The protein NLRP3 has been shown to interact directly with PTEN, a lipid-protein phosphatase known for its tumor-suppressive properties, removing phosphate. This interaction enhances the interaction of NLRP3 with ASCs and the formation and activation of inflammasomes. Myeloid PTEN is also used to assess cellular response to chemotherapy by enhancing NLRP3-dependent anti-tumor immunity [22]. Since NLRP3 activation causes the death of inflammatory immune cells and the release of pro-inflammatory cytokines, direct activation of intratumoral inflammasomes may be a crucial mechanism for stimulating anticancer immunity [23].

In conclusion, The elevated level of NLRP3 in BC women was identified as the most significant predictor of prognosis and a valuable instrument for prognostic evaluation and potentially indicating its involvement in disease pathogenesis. It can produce an inflammatory microenvironment by serving as a central interface for inflammatory cytokines. Furthermore, elevated NLRP3 levels are significantly linked with reduced overall patient survival. NLRP3 may serve as a potential biomarker and immunotherapeutic target in patients with breast cancer.

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Declaration of interest

The authors declare no conflict of interest.

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