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Neutrophils –to- Lymphocytes, Platelets –to- Lymphocyte and Neutrophil-to-Eosinophil Ratios as Predictors of Breast Cancer Disease: Correlation with Superoxide Dismutase Activities

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Abstract

The histopathological classification of breast carcinoma (BC) is based on the diversity of the morphological features of the tumors.70%–80% of BC patients belongs to invasive ductal carcinomas (IDCs) or invasive lobular carcinoma (ILC). Luminal A, Luminal B, HER2 +, and Triple Negative are molecular subtypes. NLR, PLR, and NER are inflammatory ratios. Superoxide dismutase (SOD) is antioxidant. The aim: is to study the role of latter ratios and enzyme activity in BC patients. Patients and methods: blood sample were withdrawn from BC patients (n=90) and from the healthy control (n=15). Complete blood picture was done and ratios were calculated. Also, SOD activity was assayed. Results: The mean of NLR and PLR were significantly increased in the blood of the BC patients when compared with those of healthy control (P=0.001). At cutoff value >1.59 and >102.59, ROC curve had the ability to differentiate the patients from healthy control (AUC=0.87 and 0.9, P<0.0001). The mean of NER and SOD activity were significantly decreased when compared with those of healthy control (P<0.0001). The Roc curve at cutoff value of ≤69.7 and ≤ 167.4 can significantly differentiate BC patients from the healthy control with AUC values of 0.84 and 1.0, respectively. The molecular subtypes also showed the same patterns of the later classifications. Conclusion: The increment in NLR, PLR and the decrement in both SOD activity and NER confirm the inflammatory picture of BC, and can be used for its assessment.

Keywords: BC, PLR, NLR, NER, SOD

Introduction

Breast cancer (BC) is a metastatic cancer (Stewart, et al., 2014). Early diagnosis of such disease can lead to a good prognosis together with a high survival rate (Majeed, et al., 2014). Gene mutations can increase the development of BC (Siegel, et al., 2017). The frequency of BC occurring among women is 100 times higher than that in men (Early Breast cancer Trialists' Collaborative group (EBCTCG),

2005, DeSantis et al., 2019).

Metastasis is the main cause of death. The rates of metastasis and mortality in breast cancer patients have decreased as a result of implementation of systemic adjuvant therapy after early screening by mammographic use (Hellman, et al., 2000, Vrijland, et al., 2018).
Histologically, breast cancer is classified as invasive carcinoma when it grows into the stroma, originating from the lobules that supply milk to the ducts or from the inner epithelial layer of the ducts (Rosai, et al., 2011). The tumor called in situ carcinoma if it only impacts the breast epithelium. The most common types of invasive breast accounting (55%) of cases is termed invasive ductal carcinoma (Eheman, et al., 2009). The second major followed by the invasive mammary carcinoma is Invasive lobular carcinoma (ILC) (5%-15%). It usually affects older aged women (Lakhani, et al., 2012). The intralobular proliferation of small, loosely cohesive cells that arise in the tubular ductal lobular unit (TDLU) both with or without interaction of terminal ducts is known as ductal carcinoma in situ (DCIS) or lobular carcinoma in situ (LCIS) (Fattaneh, et al., 2003, Lakhani, et al., 2012).

There are at least 4 main subtypes of BC which have a considerable impact on prognosis (*Sørlie, et al., 2001, Makki, et al., 2015*). A bout 50% of invasive breast carcinoma is Luminal A subtype. It is either HER2 negative or ER/PR positive. It present with low grade and ER positive. Luminal B subtype is 20% of the invasive BC. This subtype shows variable (positive or negative) HER2/neu expression together with positive ER/PR. Ki-67, which measures the proliferation index rate, and histological grades that are relatively higher than those of luminal A. Compared with luminal A, its prognosis is worse. Her-2 overexpression subtype is only equivalent to15% of all BC patients. While HER2/neu typically has strong positive patterns, the PR/ER pattern is typically negative. Their gene signature usually show higher HER2 expression but with lower ER expression (Rosai, et al., 2011). In the basal like subtype, ER/ PR and Her-2 are negative (triple negative) (Shawarby, et al., 2013). It is classified by a TP53 mutation and elevated Ki_67 index expression. It has no response to endocrine therapy (Schnitt, 2010, Correa, et al., 2009, Yuanping, et al.2020).

Oxidative stress is due to imbalance in the ratio between free radicals (oxidants) and antioxidants scavengers. Imbalance has been implicated in the BC pathogenesis (Himmetoglu, et al., 2009). Antioxidants can be divided into two systems: enzymatic and non-enzymatic. The enzymatic system including superoxide dismutase (SOD). They are produced by the organism itself under physiological conditions. It is a defender against superoxide anion, while the enzyme catalase acts on H_2O_2 (Fu, et al., 2018). The produced free radicals during aerobic metabolism can damage almost all kind of molecules in living cells (lipids, proteins or nucleic acids), but in this regards SOD blocks the initiation of free radical chain reactions (Rao, et al.,1996, Kumar, et al., 2015).

Substantial evidence importantly showed that inflammation based scores, such as the systemic immune-inflammation index; NLR, PLR and NER, which were useful indicators for predicting the prognosis of various solid cancers (Fu, et al., 2014, Lusho, et al.2021) and BC. They were the most frequently applied indicators and therefore they will be investigated for their value in predicting prognosis of BC patients.

Many others recognized elevation of peripheral NLR, PLR and NER as prognostic factors (Krenn-Pilko, et al., 2014, Cho, et al., 2018, Sahin, et al., 2024). Other revealed some patients with elevated PLR, but usually with better survival outcomes (Gündüz, et al., 2015). Therefore, this study was conducted to evaluate the role of NLR, PLR and NER as well as SOD activity in assessing the presence of BC or not; if any. Their inter-relationship between each other in such disease will also be evaluated.

Patients and methods:

Patients' selection

105 participants were recruited in the present study. They include 90 female with BC and 15 of healthy controls with matched age 26 to 86 years. They were enrolled from the oncology center Mansoura, University, Dakahlia, Egypt. None of the patients had received any BC treatment before blood sampling.

Blood sampling

Five milliliters of venous blood were obtained of which 3 ml were left to clot, centrifuged and the serum fraction was separated, they either freshly used or stored at−80 °C until used for SOD assay. The other 2ml of whole blood were poured onto EDTA tube for the hematological assays.

Methods

Hematological parameters:

Complete blood counts including white blood cells (WBC), platelet (PLT) neutrophils count, lymphocyte count, monocyte count, differential parameters of the blood were also analyzed using (ALINITY H automated Hematology analyzer, US).

Neutrophils to Lymphocyte Ratio (NLR)

NLR was estimated by dividing the count of neutrophils on that of lymphocyte.

Platelets to Lymphocyte Ratio (PLR)

PLR was evaluated by dividing the platelets count on that of lymphocytes.

Neutrophil to eosinophil Ratio (NER)

NER was calculated via dividing the neutrophils on that of eosinophils.

Measurement of superoxide dismutase (SOD):

Superoxide dismutase activity in serum was determined by the standard method (18). The method depends on the ability of the enzyme to inhibit the phenazine methosulphate –mediated reduction of nitro blue tetrazolium dye. The reduction in the optical density of this dye was measured at 560nm, using colorimeter (**Humalyzer3000**, **Germany**).

Statistical analyses:

All statistical analyses Medcalc software (version 14.8.1.; medcalc software Bvba, Ostend, Belgium) was used. IBM SPSS (version 29) was also used. Continuous variables were expressed as mean± standard deviation (SD). Comparison of markers was analyzed using a two terminal P values. Person's correlation coefficient was used in establishing correlation among parameters. ROC curve was done to determine the cutoff point, AUC, sensitivity, specificity, PPV and NPV of presence of breast tumer. A P value < 0.05 was considered statistically significant.

Results

The demographic distribution of prognostic parameters in BC patients

The demographic, clinicopathological and biomarker parameters were acquired from patients' medical records and displayed (**Table 1**).The number and percent of each metric in relation to BC patients are represented by various attributes which are listed in the table 1.the molecular subtypes luminal A (HR+/Her-2 -) was 61.1%, Luminal B (HR+/Her-2 +) was 24.4%, enriched Her-2 (HR-/ Her-2 +) was 10%, triple negative (TNBC) was 4.4%, lymph node involvement was 22.2%, no lymph node involvement was 77.8%, Ki-67 proliferation <30% was 41.1%, Ki-67 proliferation >30% was >58.9%. Patients were (26-86) years old.

Table 1: Comparison of the demographic and clinical characteristic of the patients with breast cancer:

Parameters	**BC PATIENTS (N=90)**		**Healthy control (N=15)**	
	N	%	N	%
Molecular subtypes				
Luminal A (HR+/Her-2-)	55	61.1%	-	-
Luminal B (HR+/Her-2+)	22	24.4%	-	-
Her-2 +ve (HR-/Her-2+)	9	10%	-	-
TNBC(HR-/Her-2-)	4	4.4%	-	-
Lymph node involvement				
Yes	20	22.2%	-	-
No	70	77.8%	-	-
Tumer subtype				
Ductal carcinoma	81	90%	-	-
Lobular carcinoma	9	10%	-	-
Ki-67 proliferation				
<30%	37	41.1%		
>30%	53	58.9%		
Age range years (Min – max)	(26 - 86)		(26 – 58)	

BC: breast cancer, N: patient numbers, pre: premenopausal, post: postmenopausal, G: grade, HR: hormonal receptor (Estrogen, progesterone), Her-2: Human epidermal growth factor receptor-2, Min: minimum, Max: maximum.

A- Comparison of all studied of the parameters according to the pathological microscopic subtypes as well as healthy control

White blood cells (WBCs):

The mean count of WBCs in blood of healthy control was 7.1±0.56 K/uL, n=15 and this value in that of patients with in situ ductal carcinoma was 8.1±1.9 K/uL. (n=20, P value 0.05, **Table2**). Also, the mean of WBCs counts in invasive breast ductal carcinoma was 8.2± 1.9 K/uL, (n=28, in metastatic ductal carcinoma was 9.1±2.1 K/uL, (n=33), in *in situ* lobular carcinoma was 8.5±2.2 K/uL (n=33), and in metastatic lobular carcinoma was 8.1±0.8 K/uL, (n=7, **Table2**, **3**).
The differences between all of these mean values and that of the control were statistically significant (P value < 0.05).

Platelets

The mean count of platelets in healthy control was 220.1±25.7 K/uL and this value in patients with in situ ductal carcinoma was 274.1±62.2 K/uL.(n=20, **Table2**). Also, the mean count of platelets in invasive ductal carcinoma was 249.7± 55.5 K/uL (n=28), in metastatic ductal carcinoma was 273.9±65.7 K/uL, (n=33), in *in situ* lobular carcinoma was 287.3±75.3 K/uL, (n=2) and in metastatic lobular carcinoma was 256.2±57.3 K/uL, (n=7). The differences between mean values and that of control were statistically significant (**Tables 2**, and **3**).

Lymphocytes

The mean count of Lymphocytes in the blood of the healthy control was 2.6±0.14 K/uL, n=15 and this value in patients with in situ ductal carcinoma was 2.1±0.3 K/uL, (n=20), **(Table 2)**. Also, the mean of this parameters in invasive ductal carcinoma was 2.1± 0.4 K/uL, (n=28), in metastatic ductal carcinoma was 2.1±0.36 K/uL, (n=33), in *in situ* lobular carcinoma was 2.1±0.19 K/uL, (n=2), in metastatic lobular carcinoma was 2.0±0.46 K/uL, (n=7). The differences between these mean values and that of control were statistically significant (**Table 2**, **3**).

Neutrophils

The mean count of neutrophillic leucocytes of the healthy control was 4.2±0.62, n=15 and this value in the blood of patients with in situ ductal carcinoma was 5.4±1.4 K/uL, (n=20), (**Table2).** Also, the mean count of neutrophils in invasive ductal carcinoma was 5.1±1.4 K/uL, (n=28), in metastatic ductal carcinoma was 5.9±1.7 K/uL, (n=33), in *in situ* lobular carcinoma was 5.6±1.9 K/uL, (n=2), in metastatic lobular carcinoma was 4.7±0.6 K/uL, (n=7). The mean differences and that of control showed statistically significant differences (**Table2**, **3**).

Eosinophils

The mean count of eosinophils in the blood of the healthy control was 0.05±0.01 x10 ^{9}/L, (n=15) and this value in the blood of the patients with in situ ductal carcinoma was 0.14±0.11 x10 ^{9}/L, (n=20), (P value 0.003). The mean count of eosinophils in invasive ductal carcinoma was 0.17±0.16 x10 ^{9}/L, (n=28), in the blood of metastatic ductal BC patients was 0.16±0.13 x10 ^{9}/L, (n=33), in *in situ* lobular carcinoma was 0.18±0.03 x10 ^{9}/L, (n=2), in metastatic lobular carcinoma was 0.29±0.24 x10 ^{9}/L, (n=7). The differences between these mean values and that of control were significant (**Table2**, **3**).

Neutrophils to Lymphocyte Ratio (NLR)

The mean NLR of the healthy control was 1.4±0.27, (n=15) and this value in the blood of patients with *in situ* ductal carcinoma was 2.1±0.8, (n=20). Also, the mean of NLR ratio of BC patients with invasive ductal carcinoma was 2.5±0.9, (n=28), in metastatic ductal carcinoma patients was 3.0±1.5, (n=33), in *in situ* lobular carcinoma patients was 2.7±0.68, (n=2) and patients in metastatic lobular carcinoma was 2.4±0.65, (n=7). These mean values were significantly higher than that of the control value (P<0.0001, **Table2, 3**)

Platelets to Lymphocyte Ratio (PLR)

The mean PLR ratio of the healthy control was 84.0±11.3, (n=15) and this value in the blood of patients with *in situ* ductal carcinoma was 135.3±38.4, (n=20). Also, the mean of PLR in BC patients with invasive ductal carcinoma was

119.7±33.4, (n=28), in metastatic ductal carcinoma patients was 138.4±50.3, (n=33), in *in situ* lobular carcinoma was 136.2±23.7, (n=2) and patients in metastatic lobular carcinoma was 133.3±43.1, (n=7). These mean values were significantly higher than that of the control value (P<0.0001, **Table2**, **3**)

Neutrophils to Eosinophil Ratio (NER)

The mean of NER ratio of healthy control was 90.3±16.9, (n=15) and this value in the blood of in situ ductal carcinoma patients was 54.1±33.7, (n=20). Also, the mean of NER ratio in invasive ductal carcinoma patients was 45.7 ±28.6, (n=28), in metastatic ductal carcinoma patients was 59.2±44.2, (n=33), in *in situ* lobular carcinoma patients was 32.9±15.9, (n=2) and in metastatic lobular carcinoma patients was 24.5±15.9, (n=7). These mean values were significantly lower than that of the control values (P < 0.05, **Table 2**, **3**)

Superoxide dismutase activities (SOD) in BC patients versos that of the healthy control

The mean activities of SOD in sera of the healthy control was 204.9±16.5 U/ml, (n=15) and this value in *in situ* ductal carcinoma patients was 151.9±10.5 U/ml, (n=20, P<0.0001, **Table2**). Also, the mean of this enzyme activities in sera of invasive ductal carcinoma patients was 108.0 ±20.1 U/ml, (n=28), in metastatic ductal carcinoma patients was 81.5±10.7 U/ml, (n=33), in *in situ* lobular carcinoma was 141.2±1.8 U/ml, (n=2) and patients. In metastatic lobular carcinoma was 84.1 ±8.9 U/ml, (n=7). The differences between these means values were statistically differently if compared with the control mean (**Table 2**, **3**)

B- Comparison of all studied parameters according to molecular pathological subtype's data of the BC patients as well as those of the healthy control

In general NLR were elevated in different molecular pathological subtypes of BC patients (P value 0.001) when compared with that of the healthy control 1.4± 0.27, (n=15). In luminal A (HR+/Her-2 -) BC patients, the NLR was 2.5±1.1, (n=55). In those with luminal B (HR+/ HER-2 +) it was 2.5±1.5, (n=22). And in enriched her-2 BC it was 2.0±0.9, (n=9). These mean values were highly significantly increased when compared with the control values. The mean ±SD of NLR in triple negative BC (TNBC) patients was 1.7±0.2, (n=4). This mean value was significantly increased when compared with control (P value 0.04, **Table4**).

Also, PLR ratio showed elevations in the different molecular pathological subtypes of BC patients when compared with healthy control .The mean± SD of PLR ratio of healthy control was 84.0± 11.3,(n=15). That of luminal A (HR+/Her-2 -) BC patients was 130.2±37.9, (n=55). It showed highly significant (P value 0.001, **Table4**) when compared with that of healthy control. The mean ±SD of PLR ratio in Luminal B (HR+/ HER-2 +) BC patients was 141.9±57.3, (n=22). Such difference was significantly higher than that of the control (P value 0.001, **Table4**). The mean ±SD of PLR in enriched her-2 BC patients was 121.2±25.5, (n=9). It showed high significant differences when compared with the control values (P value 0.008, **Table4**). The mean ±SD of PLR in triple negative BC (TNBC) patients was 114.4±10.1, (n=4). This ratios showed a highly significantly differences (P<0.0001, **Table4**) when compared with that of healthy control.

NER ratio showed decreased values in different molecular pathological subtypes of BC patients when compared with healthy control. The mean± SD of NER ratio of healthy control was 90.3± 16.9, (n=15). That of luminal A (HR+/Her-2 -) BC patients was 45.3±33.3, (n=55). It showed high significant (P<0.0001, **Table4**) when compared with that of the healthy control. The mean ±SD of NER ratio in Luminal B (HR+/ HER-2 +) BC patients was 57.9±38.8, (n=22). Such differences were significantly higher than that of the control (P value 0.005, **Table4**). The mean ±SD of NER in enriched her-2 BC patients was 60.5±40.1, (n=9). It showed high significant differences when compared with the control values (P value 0.02, **Table4**). The mean ±SD of NER in triple negative BC (TNBC) patients was 39.8±11.7, (n=4). It showed high significant differences when compared with the control values (P<0.0001, **Table4**).

SOD enzyme activity showed decreased values in different molecular pathological subtypes of BC patients when compared with the other healthy control .The mean± SD of healthy control SOD enzyme was 204.9± 16.5 (U/ml),(n=15). That of luminal A (HR+/Her-2 -) BC patients was 110.3±29.8 (u/ml), (n=55). It showed high significant (P<0.0001, **Table4**)

when compared with that of the healthy control. The mean ±SD of SOD enzyme in Luminal B (HR+/ HER-2 +) BC patients was 106.2±29.9 (U/ml), (n=22). Such differences was significantly higher than that of the control (P<0.0001, **Table4**). The mean ±SD of SOD enzyme in enriched her-2 BC patients was 107.2±35.5 (U/ml), (n=9). It showed high significant differences when compared with the control values (P<0.0001, **Table4**). The mean ±SD of SOD enzyme in triple negative BC (TNBC) patients was 63.4±3.3 (U/ml), (n=4). This ratios showed a highly significant differences (P<0.0001, **Table4**) when compared with that of the control.

In general, we observed that NLR and PLR elevated in all studied BC patients, but NER and SOD decreased in all studied BC patients (**Figure1**).

Table 2: comparison of studied parameters in ductal carcinoma patients groups as well as healthy control

Parameters	Ductal carcinoma N=81			Healthy control N=15
	Insitu N=20	Invasive N=28	Metastatic N=33	
WBCs K/uL Mean±SD P	8.1±1.9 0.05	8.2±1.9 0.04	9.1±2.1 0.001	7.1±0.56
Platelets K/uL Mean±SD P	274.1±62.2 0.003	249.7±55.5 0.05	273.9±65.7 0.003	220.1±25.7
Neutrophils K/ul Mean±SD P	5.4±1.4 0.003	5.1±1.4 0.01	5.9±1.7 0.001	4.2±0.62
Lymphocytes K/uL Mean±SD P	2.1±0.3 <0.0001	2.1±0.4 <0.0001	2.1±0.36 <0.0001	2.6±0.14
Eosinophils $x10^3$/L Mean±SD P	0.14±0.11 0.003	0.17±0.16 0.004	0.16±0.13 0.001	0.05±0.01
NLR Mean±SD P	2.1±0.8 <0.0001	2.5±0.9 <0.0001	3.0±1.5 <0.0001	1.4±0.27
PLR Mean±SD P	135.3±38.4 <0.0001	119.7±33.4 <0.0001	138.4±50.3 <0.0001	84.0±11.3
NER Mean±SD P	54.1±33.7 <0.0001	45.7±28.6 <0.0001	59.2±44.2 0.01	90.3±16.9
SOD Mean±SD P	151.9±10.5 <0.0001	108.0±20.1 <0.0001	81.5±10.7 <0.0001	204.9±16.5

N: patients number, **SD**: stander deviation, **P**: probability, **P** <0.05: significant p value, **P** <0.01: Highly significant P- value, **P** =0.001 or less: very highly significant P-value, **WBCs**: white blood cells, **NLR**: Neutrophils to lymphocyte ratio, **PLR**: platelets to lymphocyte ratio, **NER**: Neutrophils to Eosinophils, **SOD**: superoxide dismutase enzyme activity.

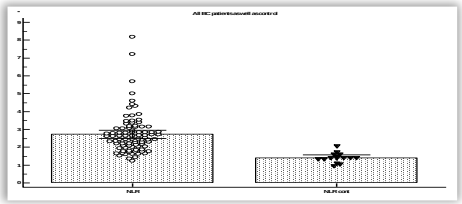

Fig a: representation of NER, PLR, and SOD values in all BC

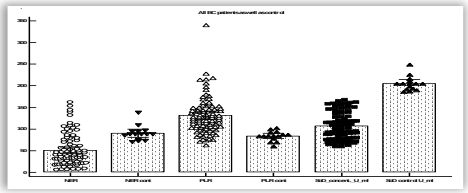

Fig b: representation of NLR ratios as Well as its mean values in all BC patients

Figure 1: comparison of studied parameters in all studied BC patients as well as healthy control

Table 3: comparison of studied parameters in lobular carcinoma patients groups compared to healthy control

Parameters	Lobular carcinoma N=9		Healthy control N=15
	Insitu N=2	Metastatic N=7	
WBCs K/uL Mean±SD P	8.5±2.2 0.03	8.1±0.81 0.002	7.1±0.56
Platelets K/uL Mean±SD P	287.3±75.3 0.01	256.2±57.3 0.05	220.1±25.7
Neutrophils K/ul Mean±SD P	5.6±1.9 0.026	4.7±0.55 0.07	4.2±0.62
Lymphocytes K/uL Mean±SD P	2.1±0.19 <0.0001	2.0±0.46 <0.0001	2.6±0.14
Eosinophils $x10^3$/L Mean±SD P	0.18±0.03 <0.0001	0.29±0.24 0.001	0.05±0.01
NLR Mean±SD P	2.7±0.68 <0.0001	2.4±0.65 <0.0001	1.4±0.27
PLR Mean±SD P	136.2±23.7 <0.0001	133.3±43.1 <0.0001	84.0±11.3
NER Mean±SD P	32.9±15.9 <0.0001	24.5±15.9 <0.0001	90.3±16.9
SOD Mean±SD P	141.2±1.8 <0.0001	84.1±8.9 <0.0001	204.9±16.5

N: patients number, **SD**: stander deviation, **P**: probability, **P**<0.05: significant p value, **P**<0.01: Highly significant P value, **P**=0.001 or less: very highly significant p value, **WBCs**: white blood cells, **NLR**: Neutrophils to lymphocyte ratio, **PLR**: platelets to lymphocyte ratio,

NER: Neutrophils to Eosinophils**, SOD**: superoxide dismutase enzyme activity.

Table 4: comparison of all studied parameters according to their molecular pathological subtypes in BC patients compared to in healthy control

Molecular Subtypes parameters	**Luminal A N=55**	**Luminal B N=22**	**Her-2 enriched N=9**	**TNBC N=4**	**Healthy control N=15**
NLR					
Mean±SD	2.5±1.1	2.5±1.5	2.0±0.9	1.7±0.2	1.4±0.27
P	0.001	0.001	0.001	0.04	
PLR					
Mean±SD	130.2±37.9	141.9±57.3	121.2±25.5	114.4±10.1	84.0±11.3
P	0.001	0.001	0.008	<0.0001	
NER					
Mean±SD	45.3±33.3	57.9±38.8	60.5±40.1	39.8±11.7	90.3±16.9
P	<0.0001	0.005	0.02	<0.0001	
SOD					
Mean±SD	110.3±29.8	106.2±29.9	107.2±35.5	63.4±3.3	204.9±16.5
P	<0.0001	<0.0001	<0.0001	<0.0001	

N: patients number, **SD**: stander deviation, **P**: probability, **P**<0.05: significant p value, **P**<0.01: Highly significant P value, **P**=0.001 or less: very highly significant p value, **NLR**: Neutrophils to lymphocyte ratio, **PLR**: platelets to lymphocyte ratio, **NER**: Neutrophils to Eosinophils**, SOD**: superoxide dismutase enzyme activity

Receiver operating characteristic (ROC) curve analysis of all the studied parameters

When ROC curve was used to differentiate between BC patients and control values the AUC values were represented in table 4
This table shows the cut off values, AUC, specificity (SP), sensitivity (Sn), predictive values of Positive (+PV), and negative predictive values (-PV) of NLR, PLR, NER and superoxide dismutase (SOD activities were highly significantly differences when all BC patients values were compared with those of the control group (P<0.0001, **Table4**)

Table 5: Roc curve analysis of NLR, PLR, NER and SOD activities of all the studied groups

parameters	**cutoff**	**AUC**	**SP**	**Sn**	**+PV**	**-PV**	**P**
NLR	>1.5911	0.95	86.67	95.56	97.7	76.5	<0.0001
PLR	>102.59	0.9	100	77.78	100	41.7	<0.0001
NER	≤69.703	0.87	100	74.44	100	39.5	<0.0001
SOD	≤167.4	1.0	100	100	100	100	<0.0001

AUC: Area under the curve, **SP**: specificity, **Sn**: sensitivity, **+PV**: positive predictive value, -**PV**: negative predictive value, **P**: probability, **P**<0.05: is significant, **P**<0.01: is highly significant, **P**<0.001 is very highly significant.

Table 6: Pearson correlation coefficients between all the studied parameters

		PLR			NER			SOD		
parameters	n	r	P	CI 95%	R	P	CI 95%	r	P	CI 95%
NLR										
Luminal A	55	0.4	0.002	0.16-0.61	0.1	0.4	-0.16-0.36	0.08	0.5	-0.18-0.34
Luminal B	22	0.76	0.001	0.49-0.89	0.09	0.6	-0.33-0.5	-0.25	0.27	-0.16-0.19
Her-2 +ve	9	0.7	0.03	0.07-0.93	0.37	0.33	-0.38-0.83	-0.24	0.26	-0.61-0.19
Triple -ve	4	0.65	0.34	-0.83-0.9	0.7	0.21	-0.71-0.9	0.4	0.56	-0.9-0.98
PLR										
Luminal A	55	-	-	-	-0.04	0.7	-0.03-0.22	0.05	0.6	-0.21-0.31
Luminal B	22	-	-	-	0.14	0.54	-0.29-0.53	-0.15	0.4	-0.54-0.29
Her-2 +ve	9	-	-	-	0.51	0.16	-0.23-0.87	0.24	0.5	-0.78-0.5
Triple -ve	4	-	-	-	0.28	0.71	-0.93-0.97	0.07	0.9	-0.95-0.97
NER										
Luminal A	55	-0.04	0.7	-0.03-0.22	-	-	-	0.04	0.9	-0.26-0.3
Luminal B	22	0.14	0.54	-0.29-0.53	-	-	-	0.17	0.43	-0.26-0.5
Her-2 +ve	9	0.51	0.16	-0.23-0.87	-	-	-	-0.59	0.09	-0.9-0.11
Triple -ve	4	0.28	0.71	-0.93-0.97	-	-	-	-0.71	0.26	-0.9-0.76

N: number, **r**: Pearson correlation coefficient, **P**: probability, **P**<0.05: is significant, **P**<0.01: is highly significant, **P**<0.001 is very highly significant, **CI95%:** confidence interval.

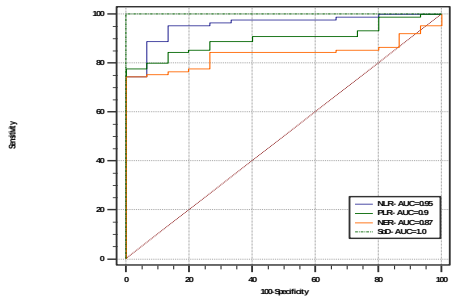

Figure 2: Roc curve analysis of all parameters in the studied groups

Correlation coefficients between the studied parameters in BC patients and in the control group

In Luminal A (HR+/ Her-2 -) BC patients (n=55), in luminal B (HR+/HER-2 +) (n=22), and in enriched HER-2 (HR-/HER-2+) (n=9), NLR and PLR were positively correlated with each other (r =0.4, P value 0.002, r =0.76, P value 0.001, and r =0.7, P value 0.03, respictivily **Table 6**).

There was no observed correlation between SOD activities and NLR, PLR, and NER inflammatory markers in any of the pathologies of BC patients (**Table 6**)

(Teramukai, et al., 2009, Balkwill et al., 2012). Thus the difference in PLR among BC patients and control value in current study may lead one to suggest that platelets help the adhesion of tumer cells with other cell, and thus tumer growth.

Lymphocytes target tumor growth of cell proliferation and metastasis, even though; they play a critical role in cancer immune surveillance (Mattes et al., 2003). By their cytotoxic effects, CD8+ T-cells can modify tumor growth in association with CD4+ T-cells. Further to these cytotoxic effects, the tumor cells undergo apoptosis, which reduces or prevent tumor growth (Asahi et al., 1996, Quail and Joyce, 2013).

Eosinophils are a subset of granulocytes which are generally involved in parasitic infections as in allergic reactions (Alexandrakis , et al., 2002, *Arnold, et al., 2020*).Recent studies showed that tumor-infiltrating eosinophils are not only able to secrete chemokines that attract CD8+ T cells into the tumor. They help M1 macrophage polarization with consequent promotion of the inflammation and phagocytosis of the tumer cells (Ownby, et al., 1983). Moreover, eosinophils express both the major histocompatibility complexes I and II (MHC I and II) on their cell surface. Via these expressions, they can act as antigen-presenting cells. They also express costimulatory molecules, including CD86, CD40, CD40 L and CD28, Thus, they can directly stimulate T cells (Ownby, et al., 1983, Hiraoka, et al., 2006, *Ghaffar, et al., 2023*).

In this regarded (Reichman, et al., 2016) showed that, Th2 cells are responsible for the inhibition of metastases of melanoma in mice; possibly through eosinophilic recruitment into the tumor (Carretero, et al., 2015). Eosinophils also promote tumor metastasis via their

Discussion

Several scoring systems based on inflammatory molecules have been investigated as prognostic indicators in a wide range of malignant tumors (Balkwill et al., 2014). Further, tumor progression requires its interaction with inflammatory response molecules in the tumor microenvironment (Mahadik et al., 1996, Kim et al., 2015).

Neutrophils inhibit the cytolytic interaction of activated T-cells, natural killer cells, and lymphocytes. (Wang, et al., 2013). Moreover, neutrophils attached to tumors encourage extracellular matrix remodeling, which cause the release of basic fibroblast growth factor, endothelial cell migration, and the isolation of tumor cells from their primary mass. All of these events ultimately result in enhancement of tumer angiogenesis growth, and the progress of its metastasis (Hashimoto, et al., 2005, Takeuchi , et al., 2016). In this regard, the increment in Neutrophils/lymphocyte ratio not only reflects inhibit cytolytic interaction of activated T- cell, natural killer cells, and lymphocytes but also modulate ECM modulation and finally tumer growth and metastasis.

Interleukin (IL)-1, IL-3, and IL-6 are inflammatory intermediates that are released by the tumor or its microenvironment and can cause platelets to accumulate after stimulating megakaryocytes (Yamanaka et al., 2007, Galdiero et al., 2013). Furthermore, platelets have the ability to release higher amounts of platelet-derived growth factor, platelet factor 4, and vascular endothelial growth factor. All of these have the ability to stimulate the growth and adhesion of tumor cells to other cells, finally resulting in tumor growth and metastasis

protumorigenic manner; this promotion is metalloproteinase 9 through the secretion of matrix. This in turn promotes angiogenesis and tissue healing via VEGF, FGF and PDGF as well as polarization of macrophage to its M2 phenotype via IL-4/IL-13 productions (Ownby, et al., 1983, Carretero, et al., 2015).

In this study, elevation of WBCs, neutrophils, platelets in breast cancer patients ($P< 0.05$) than those of the healthy control were observed. Further, the numbers of lymphocytes were decreased in breast cancer patients. Therefore, In NLR and PLR in such disease patients will be elevated than in those of the healthy control ($P<0.001$). Further, NLR and PLR were also elevated in patients with breast cancer having positivity for hormonal receptor. On the other hand, NER was decreased in patients having breast cancer and in the blood of those having hormonal receptors when compared with those of the controls (**Table 2, 3, 4**).

We examined the relationships among breast cancer molecular subtypes and the NLR, PLR and NER as indicators of inflammation. Several studies have shown that, compared with the luminal A, luminal B subtype, the triple-negative and HER2-enriched breast cancer subtypes are more immunogenic and associated with poorer prognosis. A neutrophillic host response to tumor has also been associated with poor prognosis because it can inhibit the immune system by suppressing the cytotoxic activity of T cells. According to Noh et al., (2013), we hypothesized that the degree of inflammatory response, as indicated by NLR, PLR, and NER may be different among breast cancer molecular subtypes. We observed that, elevating of NLR, PLR, and decreasing of NER ratios in all BC subtypes with high significant ($P= 0.04$) recorded and significant correlation ($P=0.03$) was detected between elevation of NLR and PLR ratios in all BC molecular subtypes except TNBC. with high significant $P < 0.03$. However, no significant correlation was found between NER and other studied inflammatory factors.

Consequently, combined index of the NLR and PLR could be used as a potential and novel indicator of malignant as well as markers of prognosis in various tumors, including breast. As reported by other authors that, the PLR shows the variation in both platelets and lymphocytes, they comprehensively indicate an immune status change during the disease period, including that of the breast (Mattes, et al., 2003, Coradi, et al., 2024).

Several studies have revealed that mechanisms of antioxidant resistance are significantly compromised in patients with malignant breast tumors. Therefore, reductions in SOD activity make cells to produce free radicals. The imbalance between free radical generation and mechanisms of defense that scavenge these free radicals play substantial role which lead to tumor development (Jain, et al., 2014, Guidotti, et al., 2015). Thus, the overexpression of SOD might be serving as compensatory mechanism to protect cells from oxidative stress (Al-Ani A, 2021). Unfortunately, SOD activity was decreased in the blood of BC patients than its value of the healthy control. Such decrease was optimum in breast cancer patients having positively in hormonal receptor elevation with breast cancer hormonal receptor compared with in that of the healthy control.

Conclusion

The systemic inflammatory markers PLR, NLR and NER may be used for predicting the severity of BC and disease prognostic, as well as the reduction of SOD enzyme which may appear due to the oxidative stress imbalance.

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الملخص العربي

عنوان البحث: نسبة النيتروفيل الى الليمفوسيت ، الصفائح الى الليمفوسيت والنيتروفيل الى الازينوفيل كمتنبأت لمرض سرطان الثدى : مدى الارتباط بنشاط انزيم السوبر اكسيد ديثميوتيز

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يعتمد التقسيم الهيستوباثولوجى لسرطان الثدى على العديد من التغيرات المورفولوجية للورم . لقد وجد ان ٧٠-٨٠% من المرضى ينتمون الى سرطان الاقنية الغازية وكذلك الى سرطان الفصوص الغازية والنوع الجزيئى ينتمى الى اللمعية أ واللمعية ب و الغنى ب هير ٢ و الثلاثى السلبى لمستقبلات الهرمونات.
نسبة النيتروفيل الى نسبة الليمفوسيت و نسبة النيتروفيل الى الايزينوفيل ونسبه الصفائح الى الليمفوسيت هى نسب تدل على الالتهاب كما ان انزيم السوبر اكسيد ديثميوتيز من مضادات الاكسدة والهدف هو دراسة هذه النسب ونشاط هذا الانزيم فى سرطان الثدى . الطرق والمرضى : اخذت عينات من ٩٠ مريض و ١٥ من الاصحاء وتم عمل صورة دم نوعى للخلايا وحساب النسب المختلفة وكذلك تقدير نشاط السوبر اكسيد ديثميوتيز . النتائج : ازدادت نسبه النيتروفيل الى الليمفوسيت ونسبخ الصفائح الى الليمفوسيت بينما قلت نسبة النيتروفيل الى االيزينوفيل وكذا قلت نسبة انزيم السوبر اكيد ديثميوتيز فى دماء مرضى سرطان الثدى مقارنة بالاصحاء ز ولقد استخدم منحنى خصائص تشغيل الاستقبالى) للتفرقة بين الاصحاء والمرضى حيث وجد ان هذا المنحنى يفرق بين الاصحاء والمرضى ولقد وجد ان هذا المنحنى يفرق بين مجموعة المرضى والاصحاء فعند خط فاصل اكبر من ١,٥٩ واكبر من ١٠٢,٥٩ وبمساحة تحت المنحنى ٠,٨٧و ٠,٩ استطاعت نسبة النيروفيل الى الليمفوسيت ونسبة الصفائح الى الليمفوسيت التفرق بين المرضى والاصحاء وكذلك استطاعت نسبة النيتروفيل الى الايزينوفيل ونسبة السوبر اوكسيد ديسميوتيز التفرقة بين المرضى والاصحاء عند خط فاصل اكبر من او يساوى ٦٩,٧و ١٦٧,٤ بقيمة تحت المنحنى ٠,٨٤و ١,٠ . الخلاصة:هذه النسب اثبتت ان الطبيعة الالتهابية لسرطان الثدى وان النقص فى نشاط انزيم السوبراكسيد ديثميوتيزوكذلك نسبة النيتروفيل الى نسبة الليمفوسيت و نسبة النيتروفيل الى الايزينوفيل ونسبه الصفائح الى الليمفوسيت لهم دور فى احداث الورم وكذلك تم التفرقة بين مجموهة المرضى والاصحاء.