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Article

Galactin-3 and Galactin-16: Proposed Novel Tumor Markers for Breast Cancer Diagnosis and Evaluation of Chemotherapy Efficacy

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Abstract

Background: Breast cancer is a multifactorial disease with major lifestyle behaviors and genetic components, any factor that increases the likelihood of developing breast cancer is considered a risk factor. These factors may be a disease, a habit, or a substance. These multiple factors combine or come together to account for most cancers, including breast cancer[60]. Therefore, the risk factors could be classified menially to unchangeable and changeable. Subjects and Methods: Over a period of seven months (from the beginning of October 2023 to the end of April 2024), 45 women were enrolled in the current study. The participants in the current study were divided into three groups based on their health status (women with breast tumors and healthy women), and depending on the type of tumor, the patients were divided into: women with malignant breast tumors and women with benign breast tumors. The first patients group consisted of 15 females with malignant breast tumors who participated in the work before starting chemotherapy and were followed up during receiving 3 consecutive doses of chemotherapy. The second patients group consisted of 15 females with benign tumors (enrolled as a pathological control group), while the healthy control group consisted of 15 females, their age ranged from 22 to 45 years. Sandwich enzyme linked immune sorbent assay (Sandwich ELISA) method was applied to evaluate C-Type Lectin Domain Family 8, member A (CLEC8A)and P-Selectin concentrations in the sera samples of the study individuals. Results: The present work aims to study the effect of body massindex (BMI) on the occurrence of carcinogenesis. The result of the present work shows that there are no statistically

significant differences in BMI in patients with different breast tumors when comparing the two groups together (p=0.090), as well as when comparing the group of patients with malignant tumors with the healthy control group (p=0.081), or when comparing the group of females with benign breast tumors with the healthy control group, the difference (p=0.957). Galectin-3 levels were measured in the sera samples of the study groups, and the results showed a significant elevation (p=0.000) in Galectin-3 in the malignant tumor group compared to the benign tumor as well as the health control group (p=0.000).On the other hand, the results of the present study did not record variations in Galectin-3 concentration in benign breast tumors and healthy groups (p=0.690). The results showed a clear decrease in Galectin-3 concentrations in the cancer patients' group after receiving consecutively three doses of chemotherapy compared to their levels in the pre-treatment period. The results of the study indicate a decrease in Galectin-3 levels after receiving the last doses of the course determined by the specialist physician compared to the levels of this lectin before starting chemotherapy, although the change was not statistically significant. In addition, the levels of Galectin-3 after receiving the last doses of treatment in the course designed by the specialist physician were not within the levels in the control groups, whether the disease or the healthy. The results showed significant statistical differences when comparing galactin-16 levels in the group of patients with malignant breast tumors compared to the group of patients with benign breast tumors (p=0.000) or healthy women in the control group (p=0.000). On the other hand, the results of the current study did not show statistical differences in galactin-16 concentration when comparing benign breast tumor patients with healthy women (p=0.467). Galectin-16 levels were monitored during three consecutive chemotherapy doses. The study showed a clear decrease in Galectin-16 concentration after receiving the first dose of chemotherapy, and its level quickly rose to double its pre-treatment level after receiving the second dose of chemotherapy. After receiving the third dose, Galectin-16 levels decreased again to their levels before receiving the first dose of chemotherapy. The study proved that there were no significant differences when comparing Galectin-16 levels before receiving treatment and after the last dose of the treatment course prescribed by the specialist physician. According to the data, a positive correlation ($r=0.938$ at $p<0.001$) was observed when testing the relationship between galactin-3 and galactin-16 in breast cancer patients. The relationships between galactin-3 and galactin-16 were positive but not significant in the groups of benign breast tumors and healthy individuals. The calculation of sensitivity and specificity is used for assessing the efficiency of the tested parameters to suggest them as diagnostic markers. The diagnostic efficiency of the included criteria in this work were evaluated by applying the receiver operating characteristic (ROC). The results showed the area under the curve and the cut-off value for the criteria evaluated in the current study. The individual efficiency (sensitivity) of the criteria evaluated in the current study for distinguishing between cancerous and

benign breast tumors reached the highest level (100%), while the lowest specificity (63%) was noted for Galectin-3. The combined sensitivity of Galectin-3 and Galectin-16 were examined in the breast tumor, these criteria showed a similar maximum sensitivity for each parameters (100%). When calculating the value of the combined specificity, the study indicated that the percentage of specificity reached 93% when evaluating both lectins together. Conclusion: the results of the work indicate the possibility of employing these criteria in evaluating the response of cancer patients to chemotherapy. Therefore, the current study provides excellent tools for diagnosing breast cancer and following up the recovery phase, which helps the specialist physician in designing the treatment strategy provided and evaluating the level of recovery.

Keywords: Galectin-3, Galectin-16, Breast Cancer, Chemotherapy

Introduction

Breast cancer is one of the leading causes of death in the world and is the most common coming women among all cancers, as result of several internal and external factors, breast cancer may occur and develop[1]. It is an advanced, multi-stage malignant tumor that affects women, and a small percentage of men breast cancer can occur because of changes in breast cells[2].Usually, breast cancer begins in the cells that make up the ducts that carry milk from the glands to the nipple, which are tubes. Cancer that begins in this area is called ductal carcinoma, a subtype of breast cancer. When a cancer occurs in the milk-producing glands, it is called lobular carcinoma[3]. There are also fewer common types that occur in breast cancer, such as Paget's disease, triple-negative breast cancer, inflammatory breast cancer, soft tissue sarcoma, and lymphoma[4]. It has been proven that 20 to 30% of breast cancer is due to modifiable factors, while 5 to 10% is due to genetic mutations and family history[5]. Breast cancer can develop in women who suffer from risk factors that increase the likelihood of infection, and women are more likely than men to develop breast cancer when breast cells are exposed to estrogen and progesterone[6],these factors help breast cancer grow. Breast cancer is a complicated disease by different biological and histological characteristics such as metastasis and invasiveness proto-oncogene activation also plays a role in tumor progression and formation[7-9]. Galectins constitute a remarkable family of lectins. There are several galectins that differ in structure, function, size, and tissue distribution in humans. Galectins directly kill bacteria, and therefore play a role in the adaptive immune response. Their levels increase with inflammation and infection[10].Galectins are the first line of defense against infection by coordinating with immune cells. Galectins can also be linked to glycoproteins to form a glycan network on the cell surface. This network has been shown to increase phagocytosis by immune cells when applied to bacteria[11]. Furthermore, the only lectin family is the galectins, which have little galectin-binding function inside the cell and can be found in the cytoplasm and nucleus[12]. Galectin-3 is a

protein belonging to the lectin family that binds β-galactosidase has many regulatory activities with multiple functions in addition to cellular physiological functions such as apoptosis, proliferation, and tissue repair. Thirty-five KDa is the molecular weight of galectin-3 and is encoded by LGAIS3 gene which located on chromosome 14[13]. Galectin-3 is the only member of the galectin family with a hybrid structure and is also the only member that can form a pentamers. Galectin-3 contains an amino-terminal domain containing a phosphorylation site that regulates its nuclear localization and reduces its ligand affinity at serine 6[14].The hybrid structure of galectin-3 also features a non-lectin amino-terminal domain that promotes the formation of capsomeres as well as a carbohydrate recognition domain, allowing pentamers formation. It releases a collagen-like sequence in the region between these two domains, which consists of about 100 amino acids and contains an H domain and can be cleave by collagenase [15]. Galectin-3 is found primarily in the cytoplasm, but is occasionally found in the nucleus, the intracellular environment, and on the cell surface[16].Galectin-3 is contributed to several pathological and physiological mechanisms[17]. Galectin-16 is one of the lectins discovered by Then, *et al.,* in placental tissue[18]. The genes for this galectin are located within a cluster of four galectin genes on chromosome 19 that encode the human protein[19].The original galectin-16 has not been studied at the protein level, but the recombinant protein has been produced and synthesized. Structure of this protein the structure consists of 142 amino acids and has a typical galectin structure, a beta-encoded CRD with two sheets containing five beta strands on the neutral side (F1-F5) and six beta strands on the concave side (S1-S6)[20]. Also, any disruption of the regulation of galectin-16 in the incubator will result in a large amount of this preeclampsia and thus be lethal to all fetuses. Galectin-16 also adopts a distinctive monomeric pattern, in contrast to typical galectins that usually crystallize in dimmers [21].

Materials and methods

The cohort study was applied in the present work with the aim of finding new tumor diagnostic markers for breast cancers and distinguishing them from non-cancerous breast tumors (benign breast tumors), first by evaluating the levels of lectins that have not been studied previously in breast tumors, as well as trying to obtain biochemical tools to evaluate the efficiency of chemotherapy in suppressing breast cancers.

The Study Population

Over a period of seven months (from the beginning of October 2023 to the end of April 2024), 45 women were enrolled in the current study. The participants in the current study were divided into three groups based on their health status (women with breast tumors and healthy women), and depending on the type of tumor, the patients were divided into: women with malignant breast tumors and women with benign breast tumors. The first patients group consisted of 15

females with malignant breast tumors who participated in the work before starting chemotherapy and were followed up during receiving 3 consecutive doses of chemotherapy. Their ages ranged between 32 and 61 years, their weight ranged between 47 and 111 Kg, while their length ranged between 137 and 160 cm. The second patients group consisted of 15 females with benign tumors (enrolled as a pathological control group), whose ages ranged from 15 to 52 years, their weight was ranged between 36 and 100 Kg, while their length ranged between 150 and 170 cm. while the healthy control group consisted of 15 females, their age ranged from 22 to 45 years.

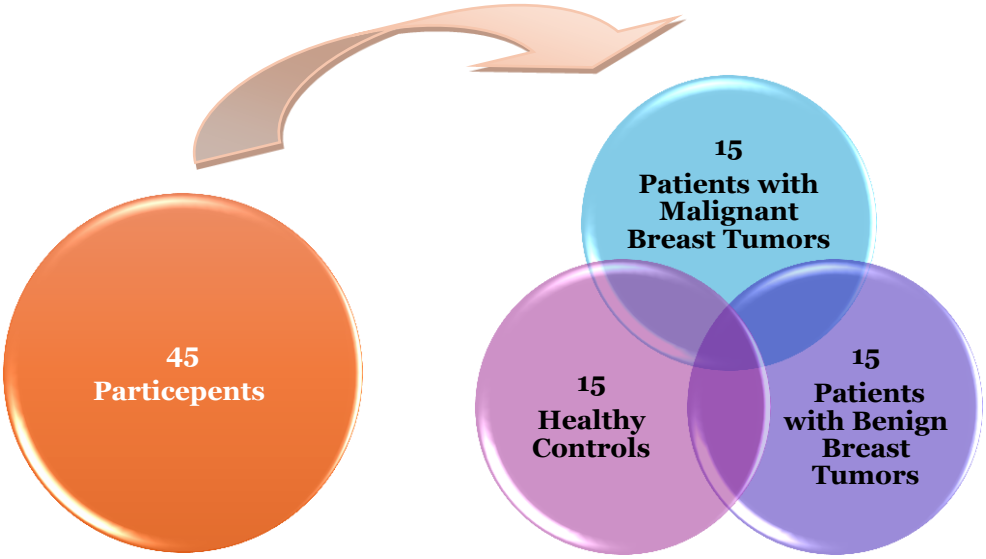

Figure 1: Distribution of the Study Individuals

Seven of patients with malignant tumor had a tumor location in the right breast and eight of them had left breast tumor location. The stages of the patients were divided between the second and the fourth, where they were divided as follows: 9 females were in the second (П) stage of cancer, 5 of them were in the stage third (Ш) and finally only one case of them was in the fourth stage (IV). All patients with malignant tumors except two were married and had 2-7 children.

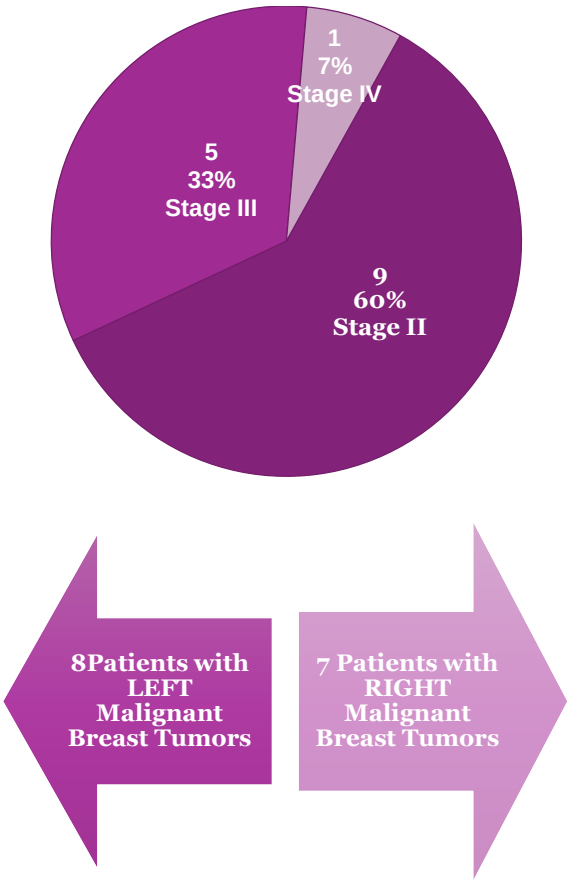

Figure 2: Distribution of Malignant Breast Tumors According to the Stage of Disease and Location of Tumor

Eight of the 15 patients with benign breast tumors had their tumors in the right breast and the remaining number had their tumors in the left breast. With the exception of only five cases, the patients with benign breast tumors were married and had 1-8 healthy births. In addition, all patients with benign breast tumors were not take contraceptives during the appearance of the tumor. Finally, some patients in this group had undergone cesarean section only as a surgical intervention before the occurrence. According to the questionnaire prepared based on the opinion of specialist doctors, which included complete information about age, place of residence, profession, period of onset of symptoms and medical history, the cases were collected. The samples of patients with malignant breast tumors were collected from the National Oncology and Hematology Hospital before receiving chemotherapy and were followed up during the chemotherapy period. The patients with cancerous breast tumors had undergone surgical treatment 3-5 weeks before receiving the first dose of chemotherapy. Samples of patients with benign breast tumors were collected from the Breast Cancer Early Detection Unit in Al-Sadr Medical City, while samples of the healthy control group were collected from the study community environment, such as postgraduate students and their relatives, as well as workers in the centers and hospitals from which the infected samples were collected. The current study

required exclusion the following female cases: All participants (patients with breast tumors or healthy controls) who had suffered chronic diseases, *i.e.;* liver, renal, cardiovascular diseases, diabetes, hypertension and morbid obesity. Breast tumor patients who did have any types of tumors before diagnosis breast tumor. Patients whose disease symptoms coincided with taking oral or intravenous contraceptives or who took oral contraceptives for 3 consecutive years before the onset of symptoms. Cases who underwent surgery within 5 years of onset of symptoms, pregnant and smokers.

Assessment of Galectin-3 and Galectin-16

Sandwich enzyme linked immune sorbent assay (Sandwich ELISA) method was applied to evaluate Galectin-3, and Galectin-16 concentrations in the sera samples of the study individuals.

The Statistical Analysis of the Data

The outcomes of the present study were analyzed through the statistical package for the social sciences (SPSS) version 26 software application statistical analysis system and excel (statistical package). The variables were illustrated by mean ± S.D, minimum, maximum, frequencies, and percentages. Graphics are presented using pie and bar charts. Inferential data analysis includes analysis of variance (ANOVA) test was applied to assess differences between the levels of the studied parameters. Pearson's correlation was applied to determine the relation among the parameters of the present study. The probability of deflection than controls are considered statistically significant if *p*-value is below 0.05. Receiver operating characteristic (ROC) curve was applied to present the sensitivity of the evaluated parameters. Combined sensitivity and specificity percentages were calculated according to biomedical statistical.

Results and Discussion

Topic of Age

According to the results of the analysis of variance (ANOVA) test, there were no statistical differences in age when comparing the two groups with breast tumors (p=0.054), and in the same manner when comparing the group with malignant breast tumors and healthy individuals (p=0.270). However, the results were contrary to what was previously mentioned when comparing the ages in the group of individuals with benign breast tumors with their counterparts in the group of healthy individuals, as the study showed the presence of significant differences (p=0.003) in age when comparing these two groups (**Table 1**).

Table 1: Age of the Study Subgroups

Study Groups (n)	Age (Year) Mean ± S.D.	Min.-Max. Range	*p-value*
Malignant Tumors 15	44.333±8.673	32.00-61.00 29.00	0.054 for MT *vs* BT
Benign Tumors 15	35.466±17.747	15.00-82.00 67.00	0.003 for MT *vs* C
Controls 15	30.466±7.698	22.00-45.00 23.00	0.270for BT *vs* C

The mean difference is significant at the 0.05 level. MT: Malignant Tumors, BT: Benign Tumors, and C: Controls

Based on information from the information form used, the study did not find the effect of age on the incidence of cancer, where the injury included women in the third decade until the seventh decade, moreover; the study found that the genetic factor contributes significantly to the incidence of cancer at an early age where most of the patients had a family history of cancer.

With increasing age, the risk of breast cancer increases[22], and about 80% of new breast cancer cases are in women over the age of 50[23].Previous studies have shown molecular changes in age-specific patterns. Young breast cancer patients have a worse prognosis than older patients, and higher frequencies of aggressive clinical pathological feature[24].

Topic of Body Mass Index

The present work aims to study the effect of body massindex (BMI) on the occurrence of carcinogenesis. The result of the present work shows that there are no statistically significant differences in BMI in patients with different breast tumors when comparing the two groups together (p=0.090), as well as when comparing the group of patients with malignant tumors with the healthy control group (p=0.081), or when comparing the group of females with benign breast tumors with the healthy control group, the difference (p=0.957), as illustrated in **Table 2**.

In general, as it was observed that there was no significant increase in the body mass index in the three groups, this indicates that there is no relationship between the BMI of patients with malignant breast tumors and the carcinogenicity of the cases recorded in the current study. The lowest BMI (17.800 Kg/m^2) was recorded for case in the benign tumor group, on the other hand; the highest value of BMI (47.420 Kg/m^2) was for a patient in cancerous breast tumor group. Obesity is widely recognized as a poor prognostic factor for breast cancer, despite being shown as a risk factor[25]. The link between breast cancer risk and body mass index (BMI) has gotten increasing attention in recent years, although the results are still debatable[26], overweight, and obesity have risks and a strong relationship with breast cancer[27].

Table 2: Body Mass Index (Kg/m^2) of the Study Groups

Study Groups (n)	BMI (Kg /m^2) Mean ± S.D.	Min.-Max. Range	*p-value*
Malignant Tumors 15	31.572±7.013	24.320-47.420 23.090	0.090 for MT *vs* BT
Benign Tumors 15	25.921±8.916	24.160-43.630 19.470	0.081 for MT *vs* C
Controls 15	25.744±4.728	17.800-38.100 20.300	0.957 for BT *vs* C

The mean difference is significant at the 0.05 level. MT: Malignant Tumors, BT: Benign Tumors, and C: Controls

Estimation of Galectin-3 Levels in the Study Groups

Galectin-3 levels were measured in the sera samples of the study groups, and the results showed a significant elevation (p=0.000) in Galectin-3 in the malignant tumor group compared to the benign tumor as well as the health control group (p=0.000).On the other hand, the results of the present study did not record variations in Galectin-3 concentration in benign breast tumors and healthy groups (p=0.690); as demonstrated in **Table 3.**

Table 3: Levels of Galectin-3 (ng/L) in the Sera Samples of the Study Groups

Study Groups (n)	Galectin-3 (ng/L) Mean ± S.D.	Min.-Max. Range	*p-value*
Malignant Tumors 15	8.601±1.076	7.060-10.270 3.210	0.000 for MT *vs* BT
Benign Tumors 15	6.526±1.575	5.700-7.900 2.200	0.000 for MT *vs* C
Controls 15	6.718±1.883	3.370-8.760 5.390	0.690 for BT *vs* C

The mean difference is significant at the 0.05 level. MT: Malignant Tumors, BT: Benign Tumors, and C: Controls

The study showed that the highest levels of Galectin-3 (10.27 ng/L) were in an obese female patient (BMI = 38.947) with breast cancer at the age of 31 years, while the lowest levels of this protein (3.37 ng/L) were observed in a sample of a healthy female from the non-obese control group (BMI = 26.839) at the age of 40 years (**Table 2)**. **Figure 3** shows a clear decrease in Galectin-3 concentrations in the cancer patients' group after receiving consecutively three doses of chemotherapy compared to their levels in the pre-treatment period.

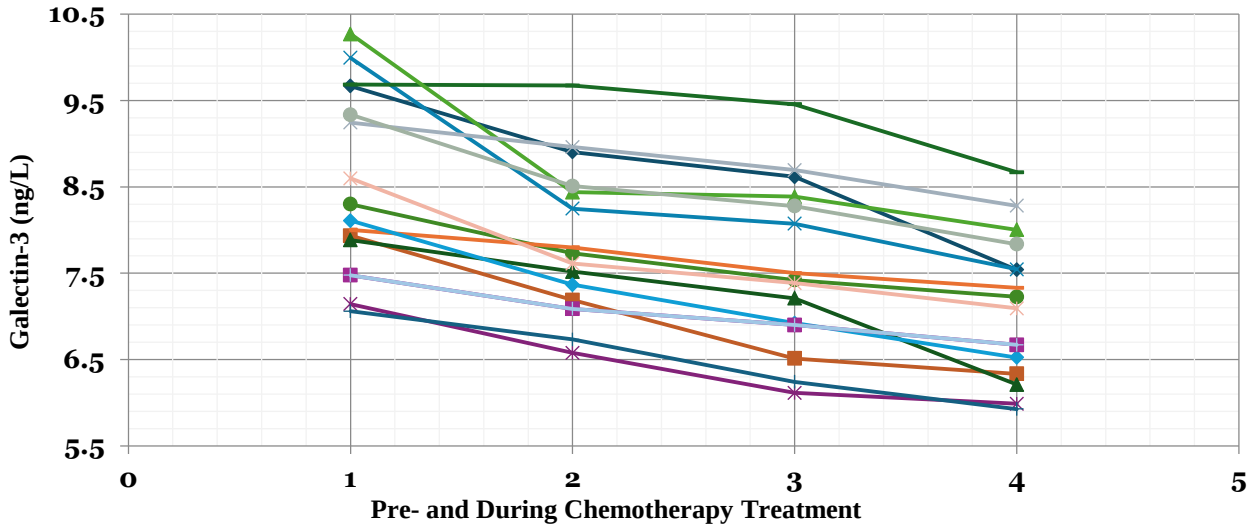

Figure 3: Follow-up of Galectin-3 Levels during Consecutive Chemotherapy

The results of the study indicate a decrease in Galectin-3 levels after receiving the last doses of the course determined by the specialist physician compared to the levels of this lectin before starting chemotherapy, although the change was not statistically significant **(Figure 4)**. In addition, the levels of Galectin-3 after receiving the last doses of treatment in the course designed by the specialist physician were not within the levels in the control groups, whether the disease or the healthy.

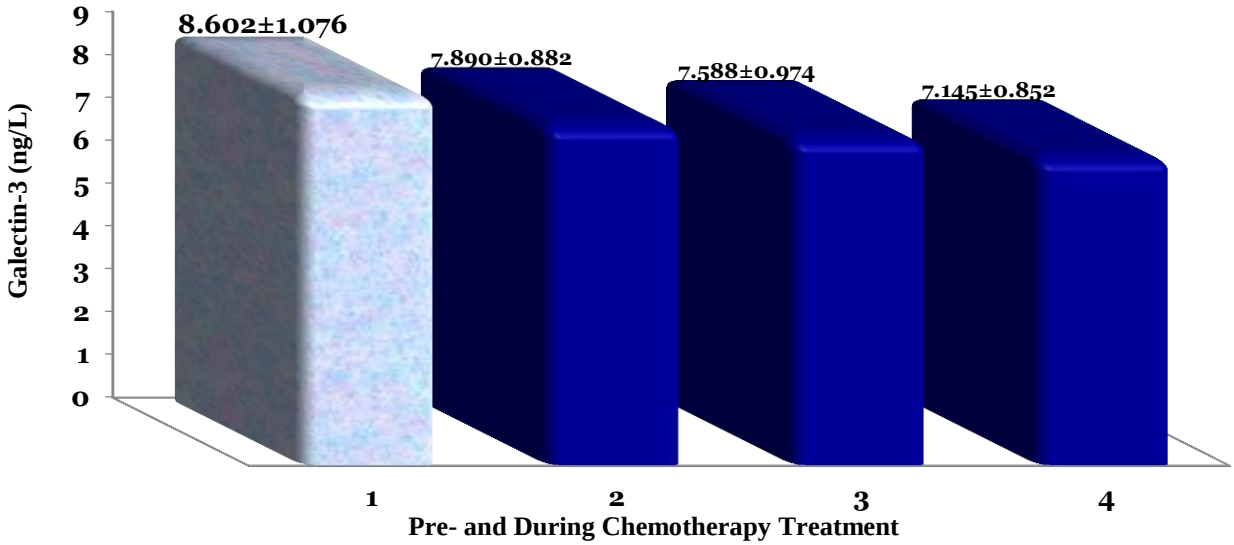

Figure 4: Comparison Levels of Galectin-3 in Cancerous Patients at Pre and Post Chemotherapy Treatment

Galactin-3 was estimated in a previous study in 2022 in type 2 diabetic patients undergoing therapeutic catheterization and healthy individuals, where there were highly statistically significant differences when comparing males and females in both groups, i.e., there was an increase in galactin-3 levels when

comparing patients of both sexes with healthy individuals[28]. While another study showed that a significant increase in Galectin-3 concentration was also recorded when comparing males and females who underwent elective single and multiple catheter interventions as a subgroup with their counterparts from a qualitative control group of healthy individuals[29]. Galectin-3 has been shown to inhibit nitric oxide-induced apoptosis in human breast cancer cells. Treatment of Galectin-3 deficient breast cancer cells with the apoptosis-inducing agent arsenic oxide was found to increase its apoptotic effects compared to Galectin-3 positive breast cancer cells, suggesting an association between chemotherapy resistance and Galectin-3 expression. Galectin-3 can also disrupt N-cadherin intercellular junctions, providing a mechanism to promote metastasis and cancer cell motility [30]. The results of this study are consistent with conducted in 2024, which showed that the level of Galectin was significantly higher in breast cancer patients compared to healthy women. There were significant differences at the level of ≤ 0.0004, which indicates an increase in the concentration of Galectin-3 in women with breast cancer. These high levels of Galectin-3 in women with breast cancer indicate the possibility of using Galectin-3 as biomarkers for breast cancer, as these biomarkers become essential tools in the early detection of breast cancer or as an indicator of disease progression [31, 32]. Also, Galectin-3 levels depend on stimulants and cell type. and Breast cancer cells are resistant to chemotherapy when the concentration of Galectin-3 protein increases in these cells[33].

Estimation of Galectin-16 Levels in the Study Groups

Galactin-16 levels were evaluated in serum samples of women with different breast tumors and healthy women. The results showed significant statistical differences when comparing galactin-16 levels in the group of patients with malignant breast tumors compared to the group of patients with benign breast tumors (p=0.000) or healthy women in the control group (p=0.000). On the other hand, the results of the current study did not show statistical differences in galactin-16 concentration when comparing benign breast tumor patients with healthy women (p=0.467); as shown in **Table 4.** The highest levels of Galectin-16 (21.80 pg/mL) were recorded in a woman in her early forties (32 years) with stage III breast cancer, while the study showed that the lowest concentration of Galectin-16 (9.30 pg/mL) was in a sample of a woman in her forties (33 years) among the healthy control group.

Table 4: Levels of Galectin-16 (pg/mL) in the Sera Samples of the Study Groups

Study Groups (n)	Galectin-16 (pg/mL) Mean ± S.D.	Min.-Max. Range	*p-value*
Malignant Tumors 15	19.189±1.077	17.590-21.800 4.210	0.000 for MT *vs* BT
Benign Tumors 15	15.416±1.958	11.000-18.000 7.000	0.000 for MT *vs* C
Controls 15	14.909±2.398	9.300-18.000 8.700	0.467 for BT *vs* C

The mean difference is significant at the 0.05 level. MT: Malignant Tumors, BT: Benign Tumors, and C: Controls

Galectin-16 levels were monitored during three consecutive chemotherapy doses. The study showed a clear decrease in Galectin-16 concentration after receiving the first dose of chemotherapy (**Figure 5**), and its level quickly rose to double its pre-treatment level after receiving the second dose of chemotherapy. After receiving the third dose, Galectin-16 levels decreased again to their levels before receiving the first dose of chemotherapy (**Figure 6**). The study proved that there were no significant differences when comparing Galectin-16 levels before receiving treatment and after the last dose of the treatment course prescribed by the specialist physician.

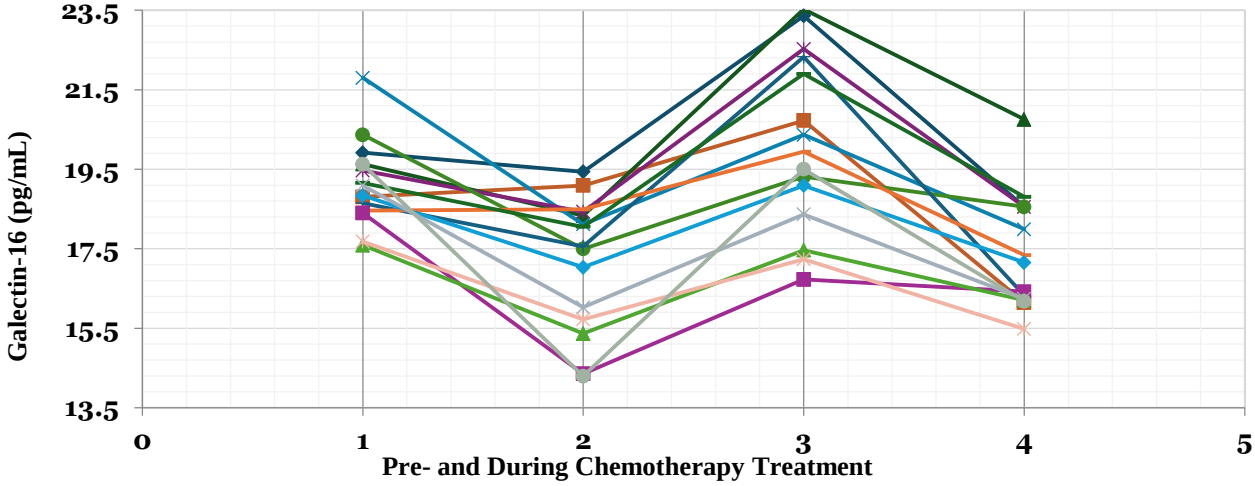

Figure 5: Follow-up of Galectin-16 Levels during Consecutive Chemotherapy

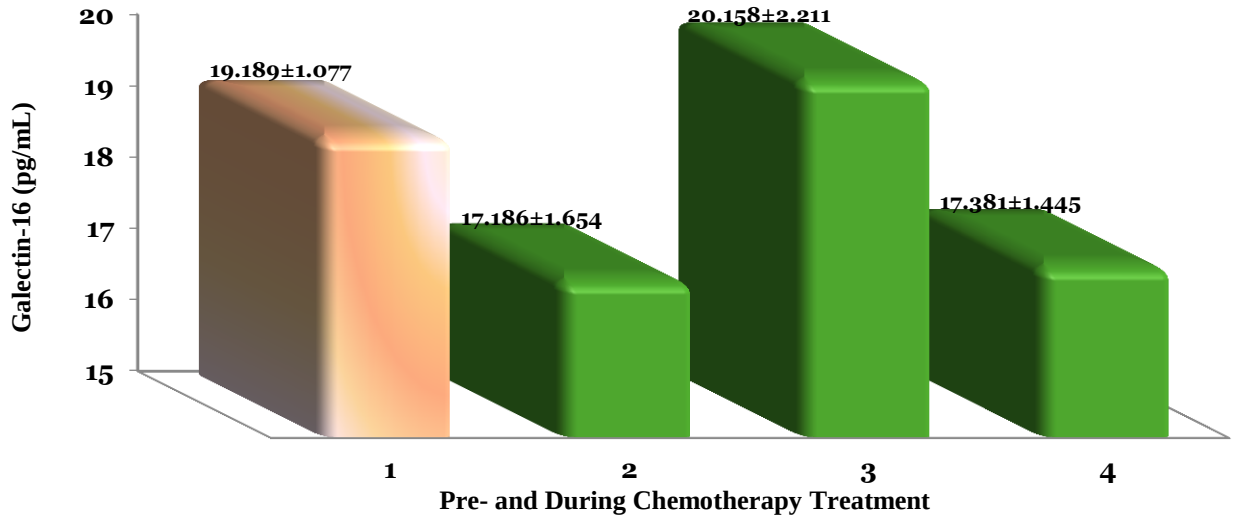

Figure 6: Comparison Levels of Galectin-16 in Cancerous Patients at Pre and Post Chemotherapy Treatment

Galectin-16 is a placental Galectin that binds through protein-protein interactions to c-Rel, a subunit of NF-kB, which plays a central role in many types of cancer, including breast cancer. These protein-protein interactions control many cellular processes involved in cancer development, such as expression of oncogenes and cell transformation[34]. The biological properties of Galectin-16 are linked to functions involved in cancer development at several points. Since Galectin-16 is a subset of placental organisms, these organisms may be involved in cancer. Galectin-16 is the most abundant member of the Galectins[35]. Chronological transcription of Galectin-16 has been found in breast cancer, where *LGALSB* was equal to 40% of Galectin-16 transcripts[36]. Galectin-16 levelswereestimated in a previous non-cancerousstudyof patients withthyroid disorders. This study showed thatthis protein was not synthesized in patients with hypothyroidism. On the other hand, the studydid not record any significant differences between the two groups of patients with hyperthyroidism and the control group[37]. Whilethere is another study that estimated the levels of Galectin-16 in patients with poly cystic ovary syndrome (PCOS). The results of this study were showed that the levels of Galectin-16 in patients with PCOS were approximated to the levels of the control group. This indicates that the production of Galectin-16 is not affected by changes associated with excessive production of ovarian hormones[48]. The present study suggests the possibility of using Galectin-16 as a diagnostic tool to distinguish between malignant and benign breast tumors.

Study the Relationship between Galectin-3and Galectin-16in the Breast Tumors and Healthy Control Groups

According to the data in **Figure 7A**, a positive correlation ($r=0.938$ at $p<0.001$) was observed when testing the relationship between galactin-3 and

galactin-16 in breast cancer patients. The relationships between galactin-3 and galactin-16 were positive but not significant in the groups of benign breast tumors (**Figure 7B**) and healthy individuals (**Figure 7C**).

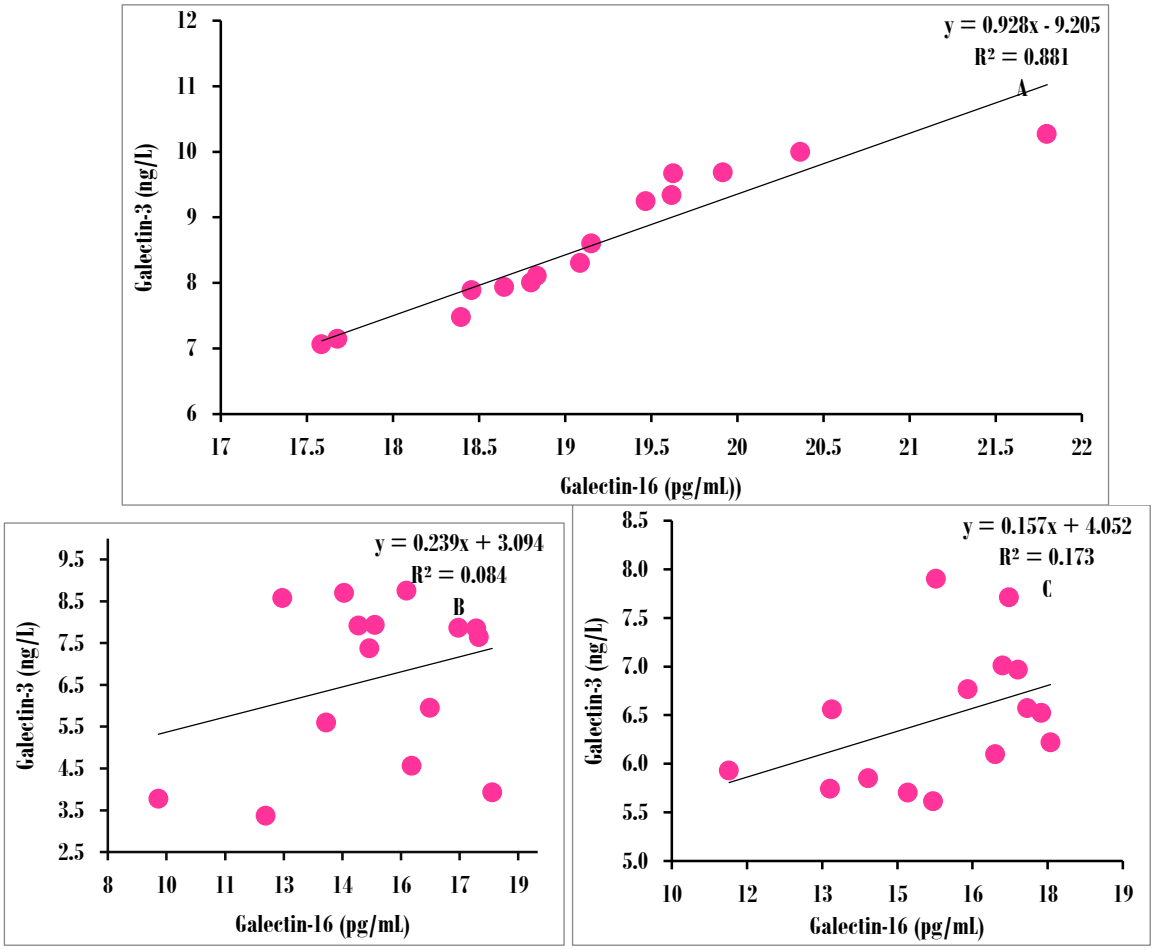

Figure 7: Relationship between Galectin-3 and Galectin-16 in (A) Cancer Tumors, (B) Benign Tumors, and (C) Healthy Control

Sensitivity and Specificity of the Evaluated Parameters

Sensitivity is known as the true positive rate or the probability of detection, it measures the proportion of positives that are correctly identified. Specificity is known as the true negative rate; it measures the proportion of negatives that are correctly identified. The calculation of sensitivity and specificity is used for assessing the efficiency of the tested parameters to suggest them as diagnostic markers. The diagnostic efficiency of the included criteria in this work were evaluated by applying the receiver operating characteristic (ROC) as demonstrated in **Figurers 8,9 for** Galectin-3 and Galectin-16; respectively. **Table 5** shows the area under the curve and the cut-off value for the criteria evaluated in the current study. The individual efficiency (sensitivity) of the criteria evaluated in the current study for distinguishing between cancerous and benign breast tumors reached the highest level (100%), while the lowest specificity (63%) was noted for Galectin-3.

Table 5: Receiver Operating Characteristic Analysis of CLEC8A and P-Selectin as Prognostic Tumor Markers for Breast Cancer

Criteria	AUC	SE	p-value	Cutoff value	Sensitivity%	Specificity%	CI (95%)
Galectin-3	0.883	0.049	0.000	7.036	100	63	0.787-0.979
Galectin-16	0.991	0.009	0.000	17.541	100	93	0.973-1.000

AUC: Area Under Curve, SE: Standard Error

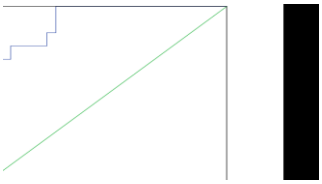

Figure 8: Receiver Operating Characteristic Curve of Galectin-3

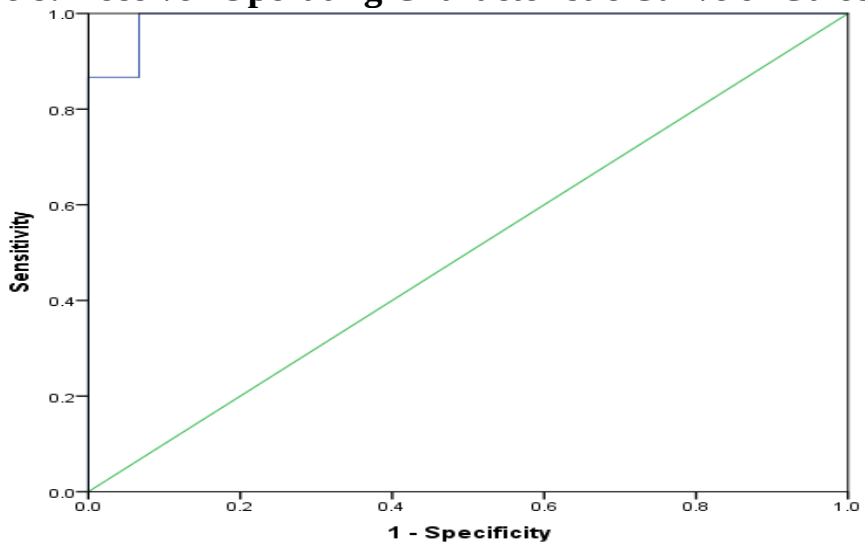

Figure 9: Receiver Operating Characteristic Curve of Galectin-16

The combined sensitivity of Galectin-3 and Galectin-16 were examined in the breast tumor, these criteria showed a similar maximum sensitivity for each parameters (100%).

Table 6: Combined Sensitivity of the Evaluated Parameters

Criteria	Galactin-3	Galactin-16
Galactin-3	100	100
Galactin-16		100

The absolute sensitivity of the criteria evaluated in the current work provides them as tools to differentiate between malignant and benign breast tumors. Moreover, the results of the work indicate the possibility of employing these criteria in evaluating the response of cancer patients to chemotherapy. Therefore, the current study provides second excellent tools for diagnosing breast cancer and following up the recovery phase, which helps the specialist physician in designing the treatment strategy provided and evaluating the level of recovery.

When calculating the value of the combined specificity, while the study indicated that the percentage of specificity reached 93% when evaluating both lectins together, as shown in **Table 7.**

Table 7: Combined Specificity of the Evaluated Parameters

Criteria	Galactin-3	Galactin-16
Galactin-3	63	93
Galactin-16		93

Referensers

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