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c-MYC Levels and Metabolic Parameters in Triple-Positive and Triple-Negative Breast Cancer

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Abstract

Background: Breast cancer is a complex, heterogeneous disease and the most common malignancy among women. It is classified based on the presence of estrogen receptors (ER), progesterone receptors (PR), and HER2. Rapid proliferation of cancer cells increases their energy demands leading to enhanced glycolysis and lactate accumulation. Lactate dehydrogenase (LDH) is pivotal in this process, particularly in tumors with anaerobic metabolism. The transcription factor c-Myc (cellular myelocytomatosis oncogene) promotes aerobic glycolysis by increasing glucose uptake and lactate production are a hallmark of the Warburg effect.

Objectives: This study was aimed to evaluate c-Myc expression levels in patients with triple-positive breast cancer (TPBC) and triple-negative breast cancer (TNBC), comparing them to healthy control.

Patients and Methods: The study included 80 women (ages 35–66): 20 with TNBC, 20 with TPBC, and 40 individual healthy as a control, matched for age, sex, and Body Mass Index (BMI). Serum levels of c-Myc, CA 27-29 was measured using an ELISA sandwich technique. Additionally, lactate dehydrogenase (LDH), glycated hemoglobin (HbA1c), liver enzymes (ALT and AST), and lipid profiles were assessed using spectrophotometric techniques.

Results: c-Myc levels were significantly higher in breast cancer patients (4.71 ± 3.75 ng/ml) compared to controls (0.81 ± 0.44 ng/ml, $p = 0.001$). LDH and CA 27-29 levels were significantly elevated ($p = 0.001$). Metabolic parameters, including HbA1c%, ALT, AST, and lipid profiles (except HDL), showed significant changes, with reduced HDL levels in cancer patients. Notably, TNBC patients exhibited higher c-MYC and HbA1c levels compared to TPBC patients.

Conclusion: Elevated c-Myc levels are associated with metabolic reprogramming in breast cancer and may serve as a potential therapeutic target. The higher c-Myc expression in TNBC correlates with its more aggressive nature, suggesting c-Myc's role in tumor progression.

Keywords: Breast cancer, c-Myc, Lactate dehydrogenase, Triple-negative breast cancers, Triple-positive breast cancer.

Introduction

Breast cancer (BCa) is the most commonly diagnosed cancer in women (1), and has been the leading cause of death among women in Iraq for the past three decades (2). Risk factors include age, gender, genetics, lifestyle, and environmental toxins (3). BCa is categorized into four molecular subtypes based on the status of progesterone receptor (PR), estrogen receptor (ER), and human epidermal growth factor receptor 2 (HER2) (4). Breast cancer subtypes can be classified by immunohistochemistry as follows: luminal A (ER+ and/or PR+ and HER2−), luminal B (ER+ and/or

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PR+ and/or HER2+), HER2- enriched (ER−, PR−, HER2+), and triple-negative (ER−, PR−, HER2−) (5). Triple-negative breast cancer (TNBC) is characterized by the absence of ER, PR, and HER2 expression, which occurs in 1 in 5 cases of women (6). TNBC tumors are more aggressive, have a poor prognosis, high recurrence rates, and low survival, with metastasis commonly affecting the brain and visceral organs (7). In contrast, TPBC is a distinct subtype within the HER2-positive luminal B category, characterized by the presence of all three hormone receptors (4).

c-Myc (cellular myelocytomatosis oncogene), the first member of the Myc family in mammalian cells (along with N-Myc and L-Myc), is a 62 kDa protein comprising 439 amino acids (8). It plays a crucial role in tumor metabolic reprogramming by regulating glucose and glutamine uptake, metabolism, and the invasive properties of cancer cells (9). c-Myc activation is associated with several key cancer hallmarks, including uncontrolled proliferation, metastasis, immune evasion, genomic instability, and metabolic reprogramming (10). Additionally, c-Myc regulates cell growth, division, metabolism, and apoptosis. Despite its central role in cancer, no approved clinical inhibitors of c-Myc exist, presenting a significant challenge for cancer therapy (11).

Lactate dehydrogenases (LDH, EC 1.1.127) are essential enzymes in anaerobic metabolism, catalyzing the reversible conversion of pyruvate to lactate during the final step of glycolysis, with NADH as the coenzyme (12). Lactate enhances tumor invasiveness by promoting key steps in metastasis: (I) angiogenesis, (II) immune evasion, and (III) extracellular matrix degradation and cell migration (13).

CA 27.29 is a carbohydrate-containing protein antigen, or Breast Carcinoma-associated antigen, released into the bloodstream by breast cancer cells (14).

This study aimed to evaluate the expression levels of the c-Myc gene and several other metabolic markers in patients with triple-positive breast cancer (TPBC) and triple-negative breast cancer (TNBC), and to compare these levels with those of healthy controls.

Patients and Methods

Study population and data collection: Blood samples were collected from patients with breast cancer at the Tumors Teaching Centre of the Medical City of Baghdad and the Oncology Unit at Al-Yarmook Hospital between August and November 2023. The study involved 80 participants, including 20 patients with triple-negative breast cancer (TNBC) and 20 with triple-positive breast cancer (TPBC), all of whom were undergoing hormone therapy, radiation, and chemotherapy. Additionally, 40 women without breast cancer served as controls. The majority of cancer patients were in stages II and III. The participants, aged 35 to 66, were matched for age and body mass index (BMI), and none had diabetes, polycystic ovary syndrome (PCOS), or other hormonally-related conditions affecting glucose metabolism. Each patient underwent a metastatic biopsy to confirm diagnosis and evaluate receptor status (HER2, PR, ER). Anthropometric and biochemical data were collected via questionnaires

Blood collection and laboratory analysis: Ten milliliters of venous blood were collected from each participant (patients and controls) using a disposable syringe. After 3 ml was extracted for HbA1c analysis, serum was separated into a gel tube, centrifuged at 3000 RPM for 10 minutes at room temperature, and stored in Eppendorf tubes at -20°C. Serum c-MYC concentration was measured using an ELISA sandwich technique by (ELK3329 Human Myc BP). The process involved adding samples or standards to pre-coated microtiter plate wells, followed by the addition of a biotin-conjugated antibody specific to human MYCBP and enzyme-

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linked avidin. After the TMB substrate was added, a color change occurred in the wells containing human MYCBP, which was measured spectrophotometrically at 450 nm. The c-MYC concentration was determined based on the color intensity. Human total serum CA 27-29 was measured using a sandwich enzyme-linked immunosorbent assay (ELISA) method (Human Cancer Antigen (27-29). The micro Elisa strip plate, pre-coated with a CA 27-29-specific antibody, was incubated with samples or standards, followed by a horseradish peroxidase (HRP)-conjugated antibody. After adding the TMB substrate, a color change occurred in wells containing CA 27-29, which turned yellow upon addition of the stop solution. Optical density (OD) was measured at 450 nm, and CA 27-29 concentration was determined by comparing the OD values to a reference curve. Lipid profile, ALT, AST, and LDH were analyzed using Siemens Healthcare equipment, and HbA1c was measured by Siemens Healthineers. Body mass index (BMI) was calculated as weight (kg) / height² (m).

Statistical analysis

The correlation coefficient (r) between parameters, the T-test, and the differences between three independent variables were assessed using analysis of variance (ANOVA). Statistical significance was defined as $p < 0.05$, with $p > 0.05$ indicating no significant difference. Data were analyzed using SPSS version 26, and results are presented as mean ± SE.

Results

The demographic characteristics of patients and controls: The study involved 80 participants, including 40 patients with breast cancer and 40 control patients without breast cancer. All participants were women, aged 35 to 66 years, and were matched for age and body mass index (BMI), as detailed in Table 1.

Table 1. The demographic characteristics of patients and control.

Parameter	Control	Patients
Number	40	40
Age (year)	49.45±6.79	50.60±9.86
BMI (kg.m^2)	27.24±2.31	28.51±4.40
Type of tumor		
Triple -ve		20 (50%)
Triple +ve		20 (50%)
Family history		24 (60%)

The breast cancer patients were classified into two molecular subtypes: triple-positive breast cancer (TPBC) and triple-negative breast cancer (TNBC), with 20 patients assigned to each subtype. Notably, 60% of the breast cancer patients had a family history of the disease. These molecular classifications of breast cancer play a crucial role in treatment decisions and patient assessment.

Comparison of biochemical markers between patients and healthy controls: Table 2 presents various tests and examinations used to monitor disease progression and treatment response. Notably, the CA 27-29 and c-Myc markers are critical for assessing patient health. Additionally, variables such as HbA1c, LDH, ALT, AST, and lipid profiles were evaluated, with patients showing significantly higher values compared to controls.

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Table 2. Comparison of biochemical markers between patients and healthy controls.

Marker	Control	Patients	p-value
CA27-29(U/mL)	0.986±0.294	1.752±0.573	0.0001
C-MYC (ng/ml)	0.81±0.44	4.71±3.75	0.0001
HbA1c %	4.36±0.64	5.27±0.70	0.0001
LDH (IU/L)	84.38±19.68	300.86±169.69	0.0001
ALT (IU/L)	14.60±6.72	21.64±8.00	0.0001
AST (IU/L)	14.35±4.09	20.87±7.17	0.0001
TC (mg/dL)	161.83±13.73	174.51±24.27	0.0001
TGs (md/dL)	103.58±19.45	122.24±31.70	0.0001
HDL-C (mg/dL)	53.85± 6.09	50.51± 8.51	0.015
VLDL-C (mg/dL)	20.72± 3.89	24.45± 6.34	0.0001
LDL-C (mg/dL)	87.26± 15.56	99.55± 25.24	0.001

The results revealed significant differences ($p < 0.05$) in several biomarkers between breast cancer patients (TPBC, TNBC) and the control group. Specifically, CA 27-29 levels were higher in the patient groups with mean values of (1.752±0.573 and 0.986±0.294). Similarly, c-Myc levels were significantly elevated in breast cancer patients with mean values of (4.71±3.75 ng/ml) compared to controls (0.81±0.44 ng/ml). These findings highlight the relationship between breast cancer and metabolic/functional changes, offering insights that could enhance treatment strategies and deepen understanding of the disease's biological processes.

Additional metabolic markers, such as cumulative sugar analysis (%HbA1c), lactate dehydrogenase (LDH), ALT, and AST levels, were also significantly altered in breast cancer patients. HbA1c levels were higher in patients without diabetes (5.27±0.70) compared to controls (4.36±0.64), reflecting elevated blood glucose. LDH levels were significantly higher in breast cancer patients (300.86±169.69) than in controls (84.38±19.68). ALT and AST activities were elevated in patients undergoing chemotherapy, radiation, or hormonal therapy. Furthermore, lipid profiles showed a significant decrease in high-density lipoprotein (HDL) levels and an increase in cholesterol, triglycerides, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) levels in breast cancer patients. Notably, none of the patients in the study had obesity.

Biomarkers in breast cancer patients, categorized by tumor type: Table 3 presents the relationship between various biomarkers (CA 27-29, C-Myc, HbA1c, LDH, AST, ALT, and the lipid profile) in the two disease groups. This provides valuable insights into the aggressiveness and characteristics of the molecular subtypes of breast cancer, which can inform treatment decisions and enhance the understanding of this complex condition.

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Table 3. Biomarkers in breast cancer patients according to the type of tumor.

Parameter	Triple-ve	Triple+ve	p-value
CA27-29(U/mL)	1.90±0.61 ^	1.79±0.60 ^	0.132
C-MYC(ng/ml)	6.25±4.98 #^	3.66±2.19 ^	0.029
HbA1c %	5.46±0.57 #^	4.91±0.68 *^	0.048
LDH (IU/L)	323.95±172.49 ^	269.55±123.34 ^	0.096
ALT (IU/L)	21.55±7.04 ^	18.72±7.02	0.238
AST (IU/L)	23.35±6.69 ^	18.81±6.40	0.053
TC (mg/dL)	178.85±17.89 ^	174.40±22.96	0.260
TGs (md/dL)	133.70±28.99 ^	118.80±30.88	0.288
HDL-C (mg/dL)	46.55±7.07 ^	52.25±9.42	0.108
VLDL-C (mg/dL)	26.74±5.80 ^	23.76±6.18	0.288
LDL-C (mg/dL)	105.56±18.31 ^	98.39±25.73	0.322
* significant corresponding to triple -ve, # significant corresponding to triple +ve, , ^ significant corresponding to control.			

When studying the relationship between the two tumor groups concerning the biomarkers CA27.29 and c-Myc, no statistically significant difference was observed for the CA27.29 biomarker ($p > 0.05$). However, a significant difference was identified between the two groups with respect to c-Myc ($p < 0.05$).

This study compared HbA1c levels between two subgroups of breast cancer patients: triple-negative breast cancer (TNBC) and triple-positive breast cancer (TPBC). The results revealed HbA1c levels of 5.46±0.57 in TNBC and 4.91±0.68 in TPBC, with a statistically significant difference between the two groups ($p < 0.05$). However, no statistically significant differences were observed in lactate dehydrogenase (LDH) levels, lipid profiles, or liver function between the TNBC and TPBC groups. These findings suggest that these biomarkers are not strongly associated with tumor subtype, whether triple-positive or triple-negative.

c-Myc levels in breast cancer patient groups: Figure 1 illustrates the levels of c-Myc in breast cancer patient groups with the two subtypes examined in this study, as well as in the healthy control group.

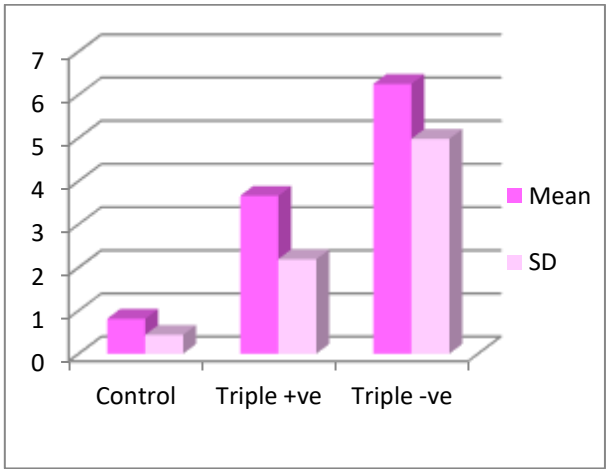

Figure 1. c-Myc level in breast cancer patients according to the type of tumor.

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Correlation of c-Myc with other biomarkers in breast cancer patients: The Correlation coefficient of c-Myc levels in ng/ml with (CA27-29, Age, BMI, HbA1c, LDH, ALT, AST, TC, TGs, HDL-C, VLDL-C,and LDL-C) as shown in Table 4.

Table 4. Correlation of c-Myc with other biomarkers in breast cancer patients.

Parameters	c-MYC					
	Breast cancer patients		Triple -ve breast cancer patients		Triple +ve breast cancer patients	
	r	p	r	p	r	p
CA27-29(U/mL)	-0.242*	0.031	-0.124	0.603	-0.379	0.100
Age (Years)	-0.306*	0.006	0.473*	0.035	-0.263	0.263
BMI(kg.m2)	-0.136	0.230	-0.145	0.541	-0.095	0.690
HbA1c%	0.101	0.374	-0.105	0.661	0.615*	0.004
LDH(IU/L)	0.091	0.420	0.209	0.377	0.267	0.255
ALT(IU/L)	0.279*	0.012	0.069	0.772	0.557*	0.011
AST(IU/L)	0.155	0.171	0.203	0.391	0.413	0.070
TC(mg/dL)	-0.023	0.842	0.201	0.396	0.292	0.212
TGs(md/dL)	-0.032	0.777	0.246	0.295	0.317	0.173
HDL-C(mg/dL)	0.011	0.923	-0.149	0.530	-0.181	0.444
VLDL-C(mg/dL)	-0.032	0.777	0.246	0.295	0.317	0.173
LDL-C(mg/dL)	-0.017	0.878	0.176	0.459	0.251	0.286

Discussion

The molecular classifications of breast cancer used in this study, with 20 patients assigned to the TNBC subtype and 20 patients assigned to the TPBC subtype, are outlined as noted by Saroglu et al. This classification aids in understanding tumor behavior and guiding treatment decisions (15). All groups were matched for age and BMI. Sixty percent of patients had a family history of the disease, in contrast to the study by Rihab Ibrahim Ahmed, where only 11% had a first- or second-degree relative with breast cancer (2).

In agreement with the study of Pekarek et al. (16), who reported an association between elevated CA 27-29 levels and tumor burden, metastatic spread, and potential disease progression, suggesting it could be an early marker of treatment failure or recurrence (16). In contrast, Kaur et al. noted that CA 27-29 levels increase progressively with advancing cancer stages, helping to stage new cases but not those with existing disease (17).

c-Myc expression was elevated in cancer patients compared to healthy individuals, consistent with findings by Gao et al., who emphasized a strong association between tumor growth and c-Myc expression (18). Similarly, Al-Hassany et al. reported c-Myc expression in various malignant tumors, highlighting its role in regulating cell proliferation and metabolism (19).

When blood markers, including %HbA1c and lactate dehydrogenase (LDH), were measured in breast cancer patients and compared to healthy controls, significant differences ($p \leq 0.05$) were observed in HbA1c levels. Even without diabetes, breast cancer

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patients exhibited higher blood glucose levels, which supports findings by Yoo et al. linking increased HbA1c levels, even within the nondiabetic range, to higher cancer-related mortality (20). Elevated LDH levels in patients align with Al-Daam et al.'s findings, which associate higher LDH levels with advanced cancer, tissue damage, and disease severity (21). Similarly, Barrak et al. highlighted high LDH levels as indicators of poor prognosis and chemotherapy resistance (22). Additionally, elevated ALT, AST, cholesterol, triglycerides, LDL, and VLDL levels were observed, while HDL levels were significantly lower. These findings are consistent with Ahmad et al., who reported significantly higher triglycerides (TG), LDL, ALT, AST, and cholesterol levels in breast cancer patients, with lower HDL levels (23). Notably, none of the patients were obese. However, in contrast, Hamid Ali et al. suggested that liver function tests may not serve as effective biochemical markers for monitoring breast cancer during treatment, as no significant differences were found between patient and control groups (24).This research underscores the importance of continuous patient assessment, regardless of breast cancer subtype, and emphasizes the need for using CA 27-29 within a broader set of tests to obtain a comprehensive understanding of the patient's condition. However, no studies have confirmed a direct association between CA 27-29 levels and molecular subtypes of breast cancer, nor can the subtype be determined based on this marker alone. Our analysis revealed elevated c-Myc levels in patients with triple-negative breast cancer (TNBC) compared to those with triple-positive breast cancer (TPBC). This is consistent with findings by Xiao-Ning Yuan et al., who noted that TNBC exhibits higher c-Myc levels, an oncogene transcription factor linked to aggressive cancer progression and metastasis (25). In contrast, Elena A. Dukhanina et al. reported that TPBC presents more aggressive clinical characteristics, with a lower overall survival rate compared to TNBC five years after treatment (26). Additionally, increased HbA1c levels were observed in TNBC patients compared to those with TPBC, which is more responsive to hormonal therapy. Although research on the impact of HbA1c across molecular subtypes is limited, studies, including one by Nehad M. Ayoub et al., suggest that TNBC is more aggressive and linked to poorer clinical outcomes (27). Their research also advocates for screening breast cancer patients for glycemic status at diagnosis and exploring therapies to manage hyperglycemia, potentially improving prognosis and clinical outcomes (27).

The findings suggest that LDH may not be a reliable biomarker for distinguishing between breast cancer subtypes, as it is not specific to any particular tumor type, a point also supported by Vladimir Jurišić et al. (28). Additionally, our study found no statistically significant differences between the two disease groups regarding ALT, AST, and lipid profile. This contrasts with Ameer Jawad Hadi et al.'s research, which indicated that estrogen may induce hyperlipidemia by altering lipid metabolism, increasing triglycerides (TG) and very-low-density lipoprotein (VLDL) (29).

Table 4 shows that c-Myc may be associated with factors such as age, liver function, and glucose levels, particularly in patients with triple-positive breast cancer. These findings suggest that c-Myc could play a role in multiple biological pathways influencing disease progression.

Conclusions

The study highlights the significant role of c-Myc in breast cancer pathogenesis, suggesting its potential as a key biomarker for distinguishing subtypes. c-Myc may also serve as a valuable molecular target for understanding tumor aggressiveness and developing tailored therapeutic strategies. Additionally, it was recommended for studying c-

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Myc in other cancer types that share similar cellular pathways could help to determine them.

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Ethical clearance: Ethical approval for this study was obtained from the Consultancy of the Scientific Board in the Department of Chemistry at the College of Science for Women, University of Baghdad, under reference number 4846/22, on August 31, 2023. In addition, there was a verbal consent form obtained from each participant enrolled in the study.

Conflict of interest: None.

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مستويات السي-مايسين والمعايير الايضية في سرطان الثدي الثلاثي الايجابي والثلاثي السلبي

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الملخص

الخلفية: سرطان الثدي مرض معقد ومتنوع وهو اكثر انواع الاورام الخبيثة شيوعا بين النساء. يتم تصنيفه على وجود مستقبلات هرمون الاستروجين, ومستقبلات البروجستيرون, ومستقبل عامل نمو البشرة. يؤدي الانتشار السريع للخلايا السرطانية الى زيادة احتياجها من الطاقة, مما يؤدي الى زيادة تحلل الجلوكوز وتراكم اللاكتات. يعتبر لاكتات ديهيدرزجينيز دورا محوريا في هذه العملية, وخاصة في الاورام ذات التمثيل الغذائي الهوائي. يعزز عامل النسخ سي-مايسين تحلل الكلوكوز الهوائي عن طريق زيادة امتصاص الكلوكوز وانتاج اللاكتات, وهي الصفة المميزة لتاثير واربورغ.

الأهداف: هدفت هذه الدراسة الى تقييم مستويات التعبير عن جين السي-مايسين لدى مريضات سرطان الثدي الثلاثي الايجابي وسرطان الثدي الثلاثي السلبي, ومقارنتهن بالمجموعة الضابطة الصحية.

المرضى والطرق: شملت الدراسة ٨٠ امراه (تتراوح اعمارهن بين ٣٥ و٦٦ عاما): ٢٠ مصابة بسرطان الثدي الثلاثي الايجابي, و٢٠ مصابة بسرطان الثدي الثلاثي السلبي, و٤٠ امراه سليمة كمجموعة ضابطة متطابقة من حيث العمر والجنس ومؤشر كتلة الجسم. تم قياس مستويات السي-مايسين وجين الورم ٢٧-٢٩ باستخدام تقنية الشطيرة (الاليزا). اما مستويات لاكتات الهيدروجين, السكر التراكمي, انزيمات الكبد, ومستويات الدهون فقد تم تقييمها بتقنيات القياس الطيفي الضوئي باستخدام معدات شركة سيمنز.

النتائج: كانت مستويات السكر اعلى بشكل ملحوظ لدى مريضات سرطان الثدي (٤,٧١+٣,٧٥ نانوجرام\مل), مقارنة بالضوابط (٠,٨١+٠,٤٤ نانوجرام\مل) ص=٠,٠٠٠١. كما ارتفعت مستويات لاكتات ديهيدروجيبيز وجين الورم ٢٧-٢٩ بشكل ملحوظ (ص=٠,٠٠٠١). اظهرت المعايير الايضية, بما في ذلك الهيموكلوبين السكري, وظائف الكبد, وملف الدهون (بأستثناء البروتين الدهني عالي الكثافة), تغييرات كبيرة مع انخفاض مستوى البروتين الدهني عالي الكثافة لدى مريضات السرطان. ومن الجدير بالذكر ان مريضات سرطان الثدي الثلاثي السلبي اظهرن مستويات سي-مايسين والهيموكلوبين السكري أعلى مقارنة بمريضات سرطان الثدي الثلاثي الايجابي.

الاستنتاج: ترتبط مستويات السي-مايسين المرتفعة بأعادة برمجة التمثيل الغذائي في سرطان الثدي وقد تعمل كهدف علاجي محتمل. يرتبط التعبير الاعلى عن السي-مايسين في سرطان الثدي الثلاثي السلبي بطبيعته الاكثر عدوانية, مما يشير الى دوره في تطور الورم.

الكلمات المفتاحية: سرطان الثدي, سي-مايسين, لاكتات الهيدروجين, بسرطان الثدي الثلاثي الايجابي, سرطان الثدي الثلاثي السلبي.

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