

Programmed Death Ligand 1 Receptor Protein Expression in a Group of Triple-Negative Breast Carcinoma Patients and its Correlation with the Clinicopathological Parameters

Heba A. Hameed*¹ , Kifah H. Abdul Ghafour¹ 

¹Department of Pathology and Forensic Medicine, College of Medicine, University of Baghdad, Baghdad, Iraq.



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Abstract:

Background: Triple-negative breast carcinoma has an aggressive nature, poor prognosis, with high recurrence rates and metastasis. It has a poor response to targeted therapies, leaving a restricted number of efficient treatments, including chemotherapy and radiotherapy. Some types of triple-negative breast carcinoma are considered immunogenic types, which mean that these tumors may be susceptible targets to a new line of treatment known as immunotherapy. Several biomarkers have been discovered to determine patients who could be eligible candidates to receive immunotherapies. One of these biomarkers is the programmed death receptor 1 (PD1)/ programmed death ligand 1 (PD-L1) overexpression.

Objectives: To assess the expression status of the PD-L1 protein in a group of triple-negative breast carcinoma patients, and its correlation with the clinicopathological parameters.

Methods: The study was conducted from October 2024 to April 2025. A cross-sectional study involved 53 patients, who were diagnosed as triple-negative invasive ductal breast carcinoma. The formalin-fixed paraffin-embedded blocks were retrieved from the archives department of the histopathological laboratories in the Medical City Complex hospitals, Baghdad, Iraq. The clinical data of these patients were reviewed, and the blocks were sectioned, prepared, and stained with monoclonal anti-PD-L1 antibody.

Results: The majority of the cases (45 patients, 84.9%) had negative expression of PD-L1 protein, while eight patients (15.1%) had positive expression of PD-L1 protein. A non-significant correlation was found between the PD-L1 protein expression and the clinicopathological parameters.

Conclusion: The vast majority of the studied cases had a negative expression of PD-L1 protein. It seemed that the correlation between PD-L1 expression status and the studied clinicopathological parameters was not significant.

Key words: Breast carcinoma; Immunotherapies; Immune checkpoint inhibitors; PD-L1 protein; Triple negative breast carcinoma.

Introduction

Breast carcinoma is considered the most common malignancy affecting women around the world, and the second most common cause of carcinoma-related death in women (1). Globally, according to the WHO registration in 2022, there were 2.3 million women diagnosed with breast carcinoma and 670,000 deaths (2). According to the Iraqi cancer registry in 2023, breast carcinoma is considered to be the most common carcinoma in Iraqi females, representing (34.8%) of all carcinoma cases diagnosed among females in Iraq; it is also the most common cause of carcinoma-related death in Iraqi women (3). Depending on the expression status of the hormonal receptors (estrogen and progesterone) and human epidermal growth factor receptor 2 (HER2), the molecular classification of breast carcinoma divides breast carcinoma into four subtypes: Luminal A, Luminal B, HER2-enriched, and triple negative breast carcinoma (TNBC).

TNBC is distinguished by the absence of estrogen, progesterone, and HER2 receptors expression (4), distant metastasis rates (5), associated with limited responsiveness to targeted anti-hormonal and anti-HER2 therapies, leaving a small list of effective treatments, including chemotherapy and radiotherapy (6). It represents 15%-20% of all breast carcinomas (7).

Some TNBCs exhibit elevated levels of tumor-infiltrating lymphocytes, making them immunogenic and potentially responsive to immunotherapies (5). Immunotherapies have emerged to be beneficial in this type of carcinoma; they work by modulating the immune response to tumor cells, either by activating anti-tumor activity of immune cells or removing the suppression imposed on immune checkpoint regulators. A mechanism developed by the tumor cells to evade destruction by the immune cells (8). Several biomarkers have been discovered to predict response to immunotherapies in solid tumors, one of which is the programmed death ligand 1 (PD-L1) overexpression (9). PD-L1 is also known as B7-H1 or

*Corresponding author:
heba.adel2305m@comed.uobaghdad.edu.iq

author:

CD274. It is a transmembrane protein that binds to its associated molecule, which is the programmed death receptor 1 (PD1). Normally, the PD1/PD-L1 complex is considered to be a part of the immune system. It serves as one of the immune checkpoint regulators. PD1 is expressed on the surface of activated T and B cells, and dendritic cells, while PD-L1 is expressed on epithelial cells, T-cells, B-cells, antigen-presenting cells, and monocytes. When these two molecules bind together, it leads to downregulation of the activity of T-cells, repressing their cytotoxic effect, preventing the immune system and its effector cells from attacking self-antigens and body tissues. This is a part of immunotolerance (10).

As one of the immune system functions is attacking the abnormal cells (like tumor cells), some tumors develop the ability to express PD-L1 on their cell surfaces, taking advantage of the role of PD-L1 in suppressing T-cell functions by binding with the PD1 receptor (11). This is one of the mechanisms tumors use to evade immune system detection and destruction (12). To overcome this mechanism, a new line of treatment has been developed to block the interaction between PD1 and PD-L1, to release the T-cells from their state of suppression, and restore their anti-tumor activity. These medications are known as immune checkpoint inhibitors (ICIs) (12). The prevalence of PD-L1 positivity varies between 17%-59% in TNBC (13).

Therefore, the aim of this study is to assess the expression status of PD-L1 in a group of TNBC patients, and its correlation with the clinicopathological parameters.

Patients, Materials and Methods

Patients Selection: A cross-sectional study was conducted from October 2024 to April 2025. Fifty-three patients were included in the study; 25 of them had diagnostic Tru-Cut biopsies, and 28 had mastectomy samples. The clinical data of these patients were reviewed from the archived files in the histopathological laboratories of the Medical City complex hospitals. All of the involved patients were diagnosed with triple-negative invasive ductal breast carcinoma. Any patient who had received chemotherapy or radiotherapy was excluded from the study. Male patients were also excluded. The formalin-fixed, paraffin-embedded blocks were retrieved. A section of four-micron thickness was taken from each block and mounted on chargeable slides. These sections were prepared and stained with a monoclonal anti-PD-L1 antibody.

Positive and Negative Controls: The PD-L immunohistochemistry (IHC) 22C3 pharmDx control slides. These slides include NCI-H226 and MCF-7, which represent sections of two pelleted formalin-fixed, paraffin-embedded cell lines: **NCI-H226**, which reveals moderate PD-L1 expression, and **MCF-7**, which serves as the negative control with no PD-L1 expression.

Staining Method: After sectioning the tissue into four-micron-thick sections and mounting them on

chargeable slides, the de-paraffinization process was performed. Following this, the slides were put in the retrieval solution (low pH). Then the hydrogen peroxide was added to block the endogenous peroxidase activity. Following this, the primary antibody (monoclonal mouse anti-PD-L1 antibody clone 22C3, Agilent technologies, Dako, Denmark) was added and left for 60 minutes. Next, the mouse linker was added and left for 30 minutes, after which the secondary antibody (HRP) was added and left for 30 minutes. The DAB substrate chromogen was then added for 10 minutes. The hematoxylin counterstain was added to the background for only one minute. Finally, the dehydration process was performed. Then, the slides were cleared and covered with a cover slip.

Assessment of PD-L1 expression: The programmed death ligand 1 protein has a membranous expression pattern (partial or complete) of any intensity expressed by tumor cells, while immune cells have a cytoplasmic or membranous expression (14). The combined positive scoring system was employed which was calculated by summing up the PD-L1 positive tumor cells + PD-L1 positive immune cells / total number of viable tumor cells \times 100, with 10% counted as a cut-off value (it was considered a positive result when the PD-L1 positivity was \geq 10%) (14).

Statistical analysis: The statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) version 20.0 for windows, SPSS, Chicago, IL, USA. The data were presented using the descriptive statistical measures including mean, standard deviation, frequency, percentage, and range (minimum and maximum). The chi-square test was used to assess the associations between variables. The student's T-test was used to identify the significance of difference between means. A P-value of $<$ 0.05 was considered to be significant.

Results

The mean age of the studied cases was 50.6 ± 12.77 years, with a range of 24-81 years. Just under one half (47.2%) of the patients were between 35-54 years of age. Just over a half of the patients (50.9%) were in grade II category. The pathological T and N stages were applied for mastectomy samples. More than a half of the patients were in the T2 category (57.1%), while the N0 stage and Nx (no information was available about the regional lymph node status) were (25%) of the patients each. As for the family history of breast carcinoma or other tumors, 10 patients had no available data, 32 out of 43 patients had no family history of breast carcinoma, while 36 out of 43 patients had no family history of other tumors. The majority of the studied cases were postmenopausal. The age of menopause was determined from the patients' clinical reports. The majority of the patients had no lymphovascular invasion, nor perineural invasion, **Table 1**.

Table 1: Clinical characteristics of the study group

Variables	Statistics	Results
Age (in years)	Mean± SD	50.6±12.77
	Median	51
	Range	24-81
Age group (age at time of diagnosis) No. (%)	<35	6 (11.3%)
	35-54	25 (47.2%)
	55-74	21 (39.6%)
	≥75	1 (1.9%)
Tumor size (in cm)	Mean± SD	5.6±4.04
	Median	4.5
	range	1-16
Tumor Variables	Categories	No. (%)
Grade	GI	6 (11.3%)
	GII	27 (50.9%)
	GIII	20 (37.7%)
pT stage	T1	3 (10.7%)
	T2	16 (57.1%)
	T3	6 (21.4%)
	T4	3 (10.7%)
pN stage	N0	7 (25.0%)
	N1	5 (17.9%)
	N2	3 (10.7%)
	N3	6 (21.4%)
	Nx	7 (25.0%)
Family history of breast carcinoma	present	11 (25.6%)
	absent	32 (74.4%)
Family history of other tumors	present	7 (16.3%)
	absent	36 (83.7%)
Menopausal status	pre-menopause	23 (43.4%)
	post-menopause	30 (56.6%)
Lymphovascular invasion	Present	19 (35.8%)
	Absent	34 (64.2%)
Perineural invasion	Present	4 (7.5%)
	Absent	49 (92.5%)

The current study revealed that 45 out of 53 of the studied cases (84.9%) were with negative expression of the PD-L1 protein, while only 8 out of 53 (15.1%) showed positive PD-L1 expression. Of these positive results, four patients were Tru-Cut biopsies, and the

remaining four were mastectomy samples. A non-significant correlation was found between PD-L1 expression status and the studied clinicopathological parameters, Tables 2 and 3.

Table 2: The correlation of PD-L1 expression status with age and tumor size

Variables	PD-L1 status								P-value
	Positive (No= 8)				Negative (No= 45)				
	Mean	SD*	Min.§	Max.¶	Mean	SD	Min.	Max.	
Age (years)	56.5	10.77	41.0	73.0	49.6	12.92	24.0	81.0	0.160
Tumor size (cm)	6.7	6.29	2.50	16.0	5.5	3.71	1.0	16.0	0.591

*: Standard deviation, §: Minimum, ¶: Maximum. Student's T test, showed insignificant correlation between PD-L1 expression and age and tumor size

Table 3: Distribution of PD-L1 expression according to clinico pathological parameters

Variables	Categories	PD-L1 status		P-value
		PD-L1 positive (No=8)	PD-L1 negative (No=45)	
Grade	GI	0 (0%)	6 (13.3%)	0.281
	GII	6 (75.0%)	21 (46.7%)	
	GIII	2 (25.0%)	18 (40.0%)	
pT stage	T1	0 (0%)	3 (12.5%)	0.321
	T2	4 (100.0%)	12 (50.0%)	
	T3	0 (0%)	6 (25.0%)	
	T4	0 (0%)	3 (12.5%)	
pN stage	N0	1 (25.0%)	6 (25.0%)	0.068
	N1	0 (0%)	5 (20.8%)	
	N2	2 (50.0%)	1 (4.2%)	
	N3	1 (25.0%)	5 (20.8%)	
	Nx	0 (0%)	7 (29.2%)	
Family history of breast carcinoma	present	1 (12.5%)	10 (28.6%)	0.656
	absent	7 (87.5%)	25 (71.4%)	
Family history of other tumors	present	2 (25.0%)	5 (14.3%)	0.579
	absent	6 (75.0%)	30 (85.7%)	
Menopausal status	Pre-menopause	2 (25.0%)	23 (51.1%)	0.256
	Post-menopause	6 (75.0%)	22 (48.9%)	
Lymphovascular invasion	Present	2 (25.0%)	17 (37.8%)	

<i>Continue</i>	Absent	6 (75.0%)	28 (62.2%)	0.696
Perineural invasion	Present	1 (12.5%)	3 (6.7%)	0.491
	Absent	7 (87.5%)	42 (93.3%)	

Chi-square test, showed non-significant associations between the PD-L1 expression the clinicopathological parameters

The expression status of the PD-L1 protein is shown in Figure 1.

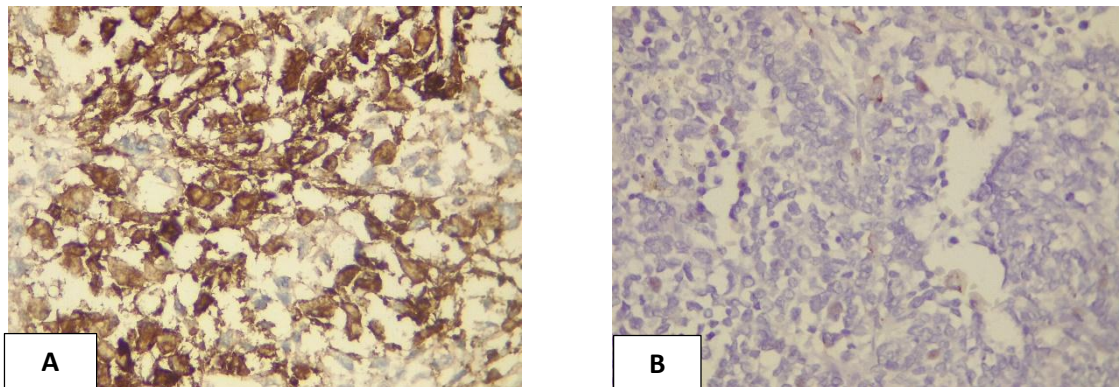


Figure 1: Microphotograph showing the PD-L1 expression status, 40×. A: microphotograph showing the membranous expression pattern of PD-L1 protein. B: microphotograph showing the negative expression of PD-L1 protein

Discussion

The results of the current study are close to the results from other studies, as the mean age reported by Troschel *et al.* was 52 ± 8 years, with age ranging from 34-63 years (15). Schmidt *et al.* reported that the mean age was 55 years with a range of 29-91 years (16). While Lee *et al.* reported that the median age was 50 years with a range of 30-83 years (17). Rammal *et al.* reported a mean age of 64 years with a range between 27 and 100 years (18).

Ben Hammouda *et al.* reported a mean tumor size of 3.55 cm with a range between (0.2-12) cm (19), while Rammal *et al.* reported a mean tumor size of 2.2 cm with a range of (0.5-12) cm (18). Troschel *et al.* reported that the majority of patients were in the grade II category (89.4%), which was in accordance with the result of the current study (15). Hu *et al.* reported that the majority of patients in their study were in the grade III category (75%) (20). Hermansyah *et al.* (2022) also reported that (77.5%) of their cases were in grade III category (21).

Hermansyah *et al.*, Özcan *et al.*, and Ren *et al.* reported that the majority of patients were in the T2 stage, with percentages of 67.5%, 43%, and 49.7%, respectively (21-23). Rammal *et al.*, and Ren *et al.*, reported that the N0 stage was the most frequently reported, with percentages of 83% and 56.1%, respectively (18, 23). Troschel *et al.*, reported that 57.9% of the patients in their study were postmenopausal, and this was in agreement with the result in the current study (15). Lee *et al.* reported that 61.1% of their cases were premenopausal (17). With regard to family history of breast carcinoma or other tumors, Li *et al.*, reported that the majority of their cases had a family history of breast carcinoma (24). On the other hand, the Ren *et al.* and Horimoto *et al.* reported that their cases had no family history of other tumors (23, 25). Rammal *et al.* reported that 82% of their cases had no lymphovascular invasion (18). Troschel *et al.* showed that 57.9% had no

lymphovascular invasion (15). Ilieva *et al.* reported that lymphovascular invasion was observed in 14.8% of their cases, while perineural invasion was observed in 8% of the cases (26).

Schmidt *et al.* reported that (77.9%) of their cases were negative for PD-L1 protein, and (20.3%) were positive for the same protein (16). Lee *et al.* compared the PD-L1 expression in TNBC using three different assays of PD-L1 at different cut-offs, and reported that the proportion of patients with positive PD-L1 at the cut-off $\geq 10\%$ were 6.35%, while 93.65% were negative (17). Hermansyah *et al.* found that 45% of their cases were positive for PD-L1 and 55% were negative for the same protein (21).

Schmidt *et al.* reported that there was no significant correlation between PD-L1 expression status and the clinicopathological parameters, which is in agreement with the current study (16). Ilieva *et al.* also reported that PD-L1 expression was non-significantly correlated with age, pathological T stage, grade, and lymph node metastasis. However, it was significantly correlated with other variables, which were not included in the current study (26). Mendivelso-González *et al.* reported that no statistically significant correlation was found between PD-L1 expression and the clinicopathological parameters including the lymphovascular invasion and the perineural invasion except for the tumor grade (27). The reasons for the differences between the current work and other studies can be attributed to the small sample size, the sample type that almost half of the included cases in the current study were Tru-Cut biopsies that may affect the accuracy of the PD-L1 assessment. The staining procedure could be another reason.

Limitations

This study was limited by the small sample size due to the retrieval of some paraffin blocks by the patients

themselves. The poor preparation of some paraffin blocks resulted in poor results, causing the exclusion of these cases; the missing data of some patients was another reason. Nearly half of the studied population underwent Tru-Cut biopsies that may affect the accuracy of the PD-L1 assessment.

Conclusion

The vast majority of the studied cases had a negative expression of PD-L1 protein. It seems that the correlation between PD-L1 expression status and the studied clinicopathological parameters was not significant.

Authors' declaration:

The authors confirm that all the figures and tables in the manuscript are original work and belong to the current study. The authors signed on ethical consideration's Approval-Ethical Clearance: the study received ethical approval by the research ethical committee of the department of pathology and forensic medicine (College of Medicine/ University of Baghdad) according to the code 133B (15th of October- 2024).

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Data Availability: Upon reasonable request, the corresponding author will make the data sets generated and/or analyzed during the current work available.

Authors' contribution

Study conception and design: (Kifah H. Abdu Ghafour, Heba A. Hameed). Literature search: (Heba A. Hameed). Data acquisition: (Heba A. Hameed). Data analysis and interpretation: (Heba A. Hameed, Kifah H. Abdul Ghafour). Manuscript preparation: (Heba A. Hameed). Manuscript editing and review: (Kifah H. Abdul Ghafour, Heba A. Hameed).

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التعبير عن بروتين مستقبل موت الخلية المبرمج 1 في عينة من المرضى المصابين بسرطان الثدي ثلاثي السلبية وعلاقته الارتباطية بالعوامل السريرية والمرضية

هبه عادل حميد¹، كفاح حمدان عبد الغفور¹

¹ فرع علم الامراض و الطب العدلي، كلية الطب،/ جامعة بغداد،/ بغداد،/ العراق.

الخلاصة:

خلفية البحث: سرطان الثدي ثلاثي السلبية له طبيعة عدوانية ويمثل 15-20% من بين جميع انواع سرطان الثدي، ويتميز بافتقار الخلايا السرطانية الى التعبير عن المستقبلات الهرمونية ومستقبل عامل النمو البشري الثاني (HER2). هذا النوع من سرطان الثدي لديه استجابة ضعيفة للعلاجات المستهدفة للمستقبلات الهرمونية والمستهدفة لمستقبل (HER2) مما يترك عددا محدودا من العلاجات الفعالة بما في ذلك العلاج الكيميائي والعلاج الاشعاعي. تعتبر بعض انواع سرطان الثدي ثلاثي السلبية انواعا مناعية وتتميز بزيادة في عدد الخلايا للمفاوية المتسللة للورم. هذا يعني ان هذه الاورام قد تكون اهدافا جيدة لنوع جديد من العلاجات المعروفة باسم العلاجات المناعية التي تعمل عن طريق تعديل استجابة الجسم المناعية للخلايا السرطانية. هناك العديد من المؤشرات الحيوية التي تم اكتشافها لتحديد المرضى الذين من الممكن ان يكونوا مرشحين مؤهلين لتلقي هذا النوع من العلاجات ومن ضمنها فرط التعبير عن بروتين مستقبل موت الخلية المبرمج 1.

الاهداف: تقييم الحالة التعبيرية لبروتين مستقبل موت الخلية المبرمج وعلاقته الارتباطية بالعوامل السريرية والمرضية في عينة من المرضى المصابين بسرطان الثدي ثلاثي السلبية.

المنهجية: شملت هذه الدراسة المقطعية (53) حالة مشخصين بسرطان الثدي ثلاثي السلبية. تم استرداد الكتل النسيجية المثبتة بالفورمالين والمضمنة في البارافين من قسم الارشيف في المختبرات النسيجية المرضية التابعة لمستشفيات دائرة مدينة الطب. تمت مراجعة البيانات السريرية الخاصة بهذه الحالات. تم اخذ مقاطع بسلك (4) ميكرون من كل كتلة بارافينية وصبغها بالاجسام المضادة لمستقبل موت الخلية المبرمج 1 من شركة دايكو.

النتائج: كشفت الدراسة الحالية ان غالبية الحالات، (45) حالة بنسبة مئوية (84.9%) لديها تعبير سلبي لمستقبل موت الخلية المبرمج 1، في حين ان (8) حالات (15.1%) فقط اظهرت تعبير ايجابيا (فرط التعبير) لمستقبل موت الخلية المبرمج 1.

الاستنتاجات: اظهرت الغالبية العظمى من الحالات التي درسناها عدم وجود تعبير لبروتين مستقبل موت الخلية المبرمج 1. ويبدو أن العلاقة بين حالة التعبير عن مستقبل موت الخلية المبرمج 1 والمعايير السريرية المرضية المدروسة لم تكن ذات دلالة إحصائية.

مفتاح الكلمات: سرطان الثدي؛ العلاجات المناعية؛ مثبطات نقاط التفتيش المناعية؛ بروتين مستقبل موت الخلية المبرمج 1 (PD-L1)؛ سرطان الثدي ثلاثي السلبية.