Original paper

Impact of age and histological type on programmed death-ligand 1 expression in non-small cell lung cancer – a single-center analysis of 1,606 cases

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Programmed death-ligand 1 (PD-L1) guides immune checkpoint inhibitor use in non-small cell lung cancer (NSCLC), yet its variation by age and histology remains uncertain. We retrospectively evaluated 1,606 consecutive NSCLC cases (2017–2022) with PD-L1 immunohistochemistry (IHC) on formalin-fixed, paraffin-embedded samples. Patients were grouped by age (< 60, 60–79, ≥ 80 years) and tumor proportion score (TPS), categorized as < 1%, 1–49%, or ≥ 50%. Associations were tested using the χ^2 test, and independent predictors were identified using multinomial logistic regression.

High PD-L1 expression (TPS ≥ 50%) occurred in 33.6% of patients, intermediate (1–49%) in 23.6%, and negative (< 1%) in 42.7%. Programmed death-ligand 1 expression ≥ 1% was most frequent in squamous cell carcinoma (63.0%), followed by adenocarcinoma (55.0%), and was least common in large cell carcinoma (36.0%; $p = 0.002$). Overall proportions of PD-L1 ≥ 1% did not differ significantly by age. However, patients aged ≥ 80 had nearly twice the likelihood of high expression compared to those < 60 (relative risk ratio, 1.92; 95% CI: 1.11–3.34; $p = 0.02$), independent of the histotype.

Programmed death-ligand 1 expression in NSCLC shows distinct histotype-related patterns and a modest, age-related trend toward higher values in the oldest group. These data support routine PD-L1 assessment and suggest that advanced age alone should not preclude consideration of immunotherapy. Findings may inform trial design and real-world treatment decision-making.

Key words: PD-L1, non-small cell lung cancer, expression, age, histological type.

Introduction

The efficacy of immune checkpoint inhibitors (ICIs) in treating non-small cell lung cancer (NSCLC) has been demonstrated in multiple clinical trials [1]. First-line treatment strategies for NSCLC include the administration of programmed cell death protein 1 (PD-1) or programmed death-ligand 1 (PD-L1) inhibitors, either as monotherapy or in combination with platinum-based chemotherapy. Programmed death-ligand 1 expression on tumor cells remains the most extensively studied biomarker for ICIs.

Determining the level of PD-L1 expression on the tumor cell surface influences diagnosis, treatment selection, and prognosis. Patients with PD-L1 expression above 50% derive the most significant benefit in terms of progression-free survival (PFS) and overall survival (OS), although clinical benefit has also been observed in patients lacking PD-L1 expression [2]. These results highlight the complexity and multidirectional nature of the tumor microenvironment, suggesting that additional clinical and biological factors influence the efficacy of immunotherapy.

In routine pathological diagnostics, PD-L1 expression is assessed by IHC on formalin-fixed, paraffin-embedded tissue samples, most commonly using antibody clones 22C3 (Agilent Technologies/Dako), SP263 (Ventana), or 28-8 (Agilent Technologies/Dako) [3]. The reliability of testing depends on adherence to optimal tissue handling protocols, including minimal cold ischemia time, proper fixation, and controlled temperature and storage conditions [4]. Interpretation can be challenging due to tumor-specific factors, sampling limitations, and high intratumoral heterogeneity of PD-L1 expression over time. Moreover, IHC detects only the membranous PD-L1 protein and excludes soluble forms, which are present in exosomes or in the free form in the circulation [2]. For these reasons, sections may be excluded from evaluation if the minimum number of viable cancer cells is below the recommended threshold, typically ≥ 100 tumor cells [3, 4]. Standardized scoring criteria exist for each antibody clone: 22C3 and 28-8, based on the tumor proportion score (TPS), and SP263, based on the percentage of tumor cells or immune cells.

Global, multicenter observational studies have shown that the prevalence of tumor PD-L1 expression in NSCLC is generally consistent across geographic regions, sampling methods, and whether tissue is obtained from primary or metastatic lesions [4, 5]. Demographic variables such as age and gender, as well as histological subtype (squamous vs. non-squamous), have not demonstrated a significant impact on PD-L1 expression in these large cohorts, supporting its robustness as a predictive biomarker [4]. However, other investigations have reported notable variability in PD-L1 levels and highlighted challenges in assay reproducibility [6]. The specific influence of patient age on PD-L1 expression in NSCLC remains insufficiently explored and inconclusive. Immunosenescence – marked by thymic involution, expansion of terminally differentiated T-cells, and chronic low-grade inflammation – reshapes the tumor-immune interface in older individuals, potentially affecting PD-L1 regulation without consistently lowering its expression in tumor cells [7]. While PD-L1 levels may remain similar between younger and older patients, the functional decline of effector T-cells and increased expression of inhibitory receptors, such as PD-1, can modify the clinical response to PD-1/PD-L1 blockade [8]. These findings suggest that in elderly patients, the prognostic and predictive significance of PD-L1 should be interpreted within the broader framework of age-related immune remodeling [9].

This study aimed to analyze PD-L1 expression in a large cohort of Caucasian patients, evaluating clinical factors that potentially influence expression levels, including histological subtype and patient age. All analyses were conducted within a single center to minimize variability related to testing procedures. Furthermore, the impact of age was assessed using the expanded categorization framework recommended by the World Health Organization (WHO) [3].

Material and methods

A retrospective analysis of PD-L1 expression was conducted in NSCLC patients diagnosed and treated at the Maria Skłodowska-Curie National Research Institute of Oncology, Warsaw, between 2017 and 2022. All datasets were either anonymized before analysis, or no information that could enable patient identification was accessible to the investigators. Patients were categorized by age according to the WHO recommendations into adult (≤ 59 years), older adult (60–79 years), and oldest adult (≥ 80 years) groups. Programmed death-ligand 1 expression was assessed using the qualitative immunohistochemical assay PD-L1 IHC 22C3 pharmDx (Agilent Technologies/Dako) on the Dako Omnis platform, employing the monoclonal mouse anti-PD-L1 antibody, clone 22C3, according to the manufacturer's instructions. Formalin-fixed, paraffin-embedded tumor samples were used, with fixation in 10% neutral buffered formalin for 6–48 hours to ensure optimal preservation of antigenicity. A minimum of 100 viable tumor cells was required for a valid assessment. Programmed death-ligand 1 expression was quantified using the TPS, defined as the percentage of viable tumor cells exhibiting partial or complete membranous staining of any intensity. Tumor proportion score values were categorized as follows: < 1% (PD-L1 negative), 1–49% (low to intermediate expression), and ≥ 50% (high expression, indicative of eligibility for pembrolizumab monotherapy in the first-line setting) (Table I). The staining interpretation was performed by board-certified pathologists experienced in PD-L1 evaluation. Internal positive controls (e.g., PD-L1 positive immune cells) were used to confirm the technical adequacy of staining. Both internal and external quality control measures were implemented throughout the IHC process to ensure accuracy and reproducibility. Descriptive statistical analysis was performed, including χ^2 tests to compare PD-L1 expression categories between groups, and multinomial regression analysis to calculate relative risk ratios (RRR).

Table I. Characteristics of the study sample and programmed death-ligand 1 expression (≥ 1%) in the subgroups

Parameters	No.	Percentage	PD-L1 expression ≥ 1%		*p*-value
			Proportion	95% CI	
Sex			0.696		
Male	909	59.60	0.57		[0.54–0.60]
Female	615	40.40	0.58		[0.54–0.62]
Age group (years)			0.096, test for trend 0.0446		
Adult (< 60)	254	16.70	0.54		[0.47–0.60]
Older adult (60–79)	1,180	77.40	0.57		[0.55–0.60]
Oldest adult (80+)	90	5.90	0.67		[0.56–0.76]
Histopathology			0.002		
AD	788	51.70	0.55		[0.51–0.58]
ADSQ	9	0.60	0.67		[0.33–0.89]
LC	50	3.30	0.36		[0.24–0.50]
NOS	40	2.60	0.55		[0.40–0.70]
SQ	544	35.70	0.63		[0.59–0.67]
Other	93	6.10	0.59		[0.49–0.69]
Total	1,524	100.00	0.57		[0.55–0.60]

AD – adenocarcinoma, ADSQ – adenosquamous carcinoma, LC – large cell carcinoma, NOS – not-otherwise specified, SQ – squamous carcinoma

Results

Data from 1,710 patients were extracted, of whom 1,606 who had PD-L1 expression tested with 22C3 antibodies were analyzed. In 5% of the cases, non-diagnostic material was obtained due to insufficient cellularity of the specimens (< 100), and these were excluded from further analysis. The median age of patients was 67 years; 16.7% of patients were < 60, while 5.9% were 80 years old or older. The prevalence of squamous-cell carcinoma (SQ) in the entire population was 35.7%, adenocarcinoma 51.7%, NSCLC, not-otherwise specified 2.6%, and other types 10.1% (Figure 1A). Programmed death-ligand 1 expression ≥ 50% was found in 33.6% of patients. In comparison, 1–49% and < 1% in 23.6% and 42.7% of patients, respectively (Figure 2A). Programmed death-ligand 1 expression ≥ 1% was observed more frequently in patients with SQ (63.0% vs. 54.1%; $p = 0.001$), and was the least frequent in patients with large cell carcinoma (36%) (Table I) (Figure 1B). Gender had no impact on the prevalence of PD-L1 expression in the entire population. Additionally, there was an increasing trend in PD-L1 expression with increasing age (Figure 2B). Further analysis using multinomial regression was performed to investigate the impact of histology and age group on PD-L1 expression, considering the level of expression. While squamous vs. non-squamous histology was the most importantly predicting the lower level expression (1–49%), RRR 1.69 (95% CI: 1.29–2.20), the expression of 50% or more was indicated by the oldest age group, even adjusting for the histology, RRR 1.92 (95% CI: 1.11–3.34) for age 80+ as compared to < 60 years (Figure 3, Table II). The summary of PD-L1 expression profiles with different patterns was presented in Figures 4 and 5.

Discussion

The present study includes one of the largest single-center cohorts of Caucasian patients with lung cancer in whom PD-L1 expression was assessed. According to our results, the PD-L1 expression profile showed the following: 34% TPS ≥ 50%, 57% TPS ≥ 1%, and 43% TPS < 1%. Previous investigations have typically been conducted on smaller cohorts or have pooled data from multiple centers and ethnically diverse populations. Our findings are consistent with those reported in other European series. In the EXPRESS study, a multicenter retrospective analysis of 2,368 patients with advanced NSCLC, 22% had a PD-L1 TPS of ≥ 50%, 52% had a TPS of ≥ 1%, and 48% had a TPS of < 1% [4]. Programmed death-ligand 1 expression rates in this study were comparable across geographic regions, biopsy methods, and whether tissue was obtained from primary or metastatic sites. Similarly, the Australian Real-World study, which analyzed 6,690 PD-L1 tests in NSCLC, reported TPS ≥ 50% in 30% of cases, TPS ≥ 1% in 62%, and TPS < 1% in 38%, with a slightly higher expression in females and tumors of more advanced stages [10]. A recent meta-analysis, encompassing

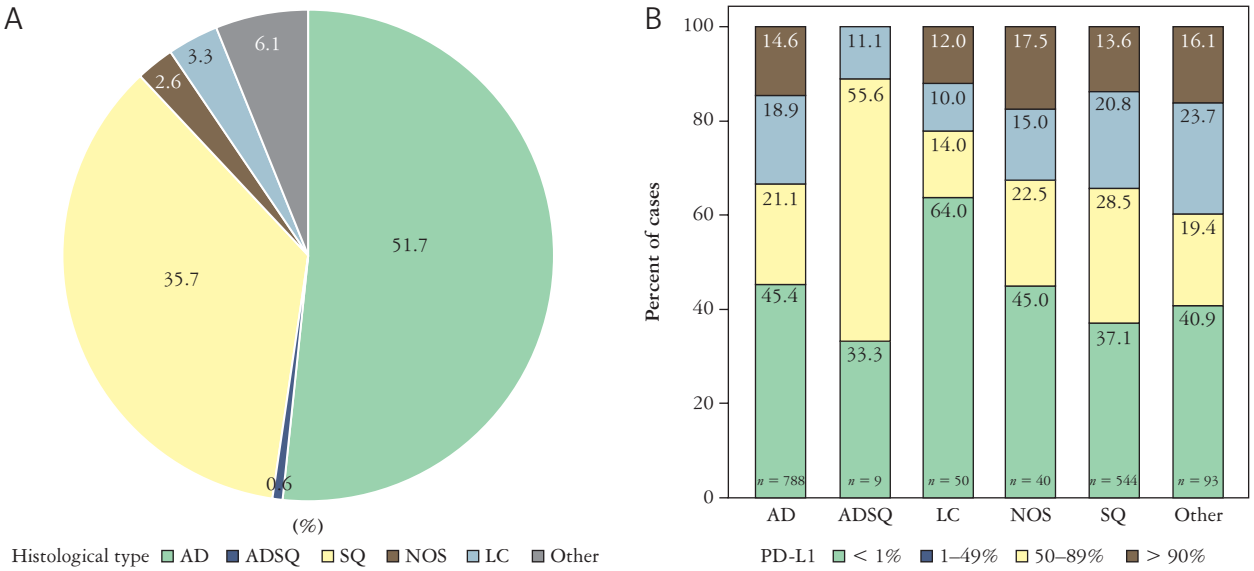

Figure 1. A) Distribution of non-small cell lung cancer histology types. **B)** Programmed death-ligand 1 expression level by non-small cell lung cancer histology types

AD – adenocarcinoma, ADSQ – adenosquamous carcinoma, LC – large cell carcinoma, NOS – not-otherwise specified, PD-L1 – programmed death-ligand 1, SQ – squamous carcinoma

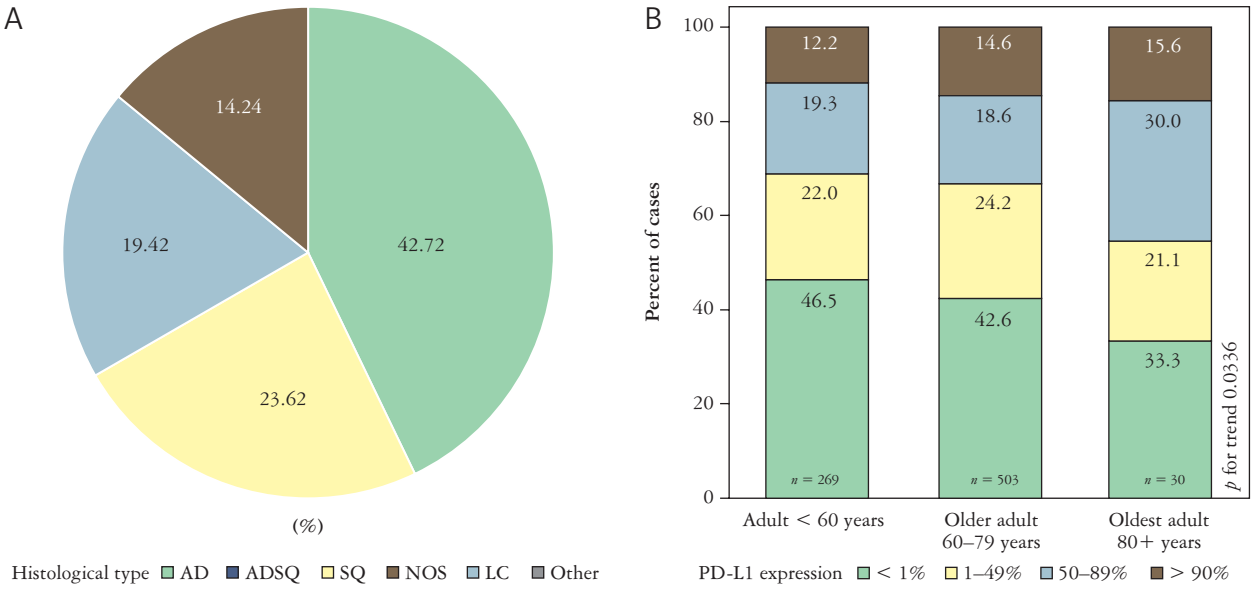

Figure 2. A) Distribution of cases by the programmed death-ligand 1 (PD-L1) expression level. **B)** PD-L1 expression level by age group

PD-L1 – programmed death-ligand 1

78 NSCLC cohorts, found that on average 27% of patients had PD-L1 TPS ≥ 50%, 58% had TPS ≥ 1%, and 42% had TPS < 1% [6]. This synthesis of global data supports the consistency of PD-L1 prevalence and reinforces its role as a robust predictive biomarker across diverse clinical and demographic subgroups.

Recent evidence suggests that very high PD-L1 expression (TPS ≥ 90%) may identify a subgroup of patients who derive a particularly pronounced benefit from ICIs. The biological rationale is that significantly elevated PD-L1 expression may reflect a tumor microenvironment with high interferon-γ-driven immune activation and abundant infiltrating cytotoxic T lymphocytes, creating a setting in which PD-1/PD-L1 blockade rapidly restores anti-tumor immunity [11]. In our study, 14% of patients presented PD-L1 TPS ≥ 90%, which constitutes 41% of all TPS ≥ 50% NSCLC. These findings are consistent with the largest published series, in which Ricciuti et al. reported a 32.2% prevalence of very high PD-L1 expression (TPS ≥ 90%) within the PD-L1 ≥ 50% subgroup, and Barrichello et al. observed PD-L1

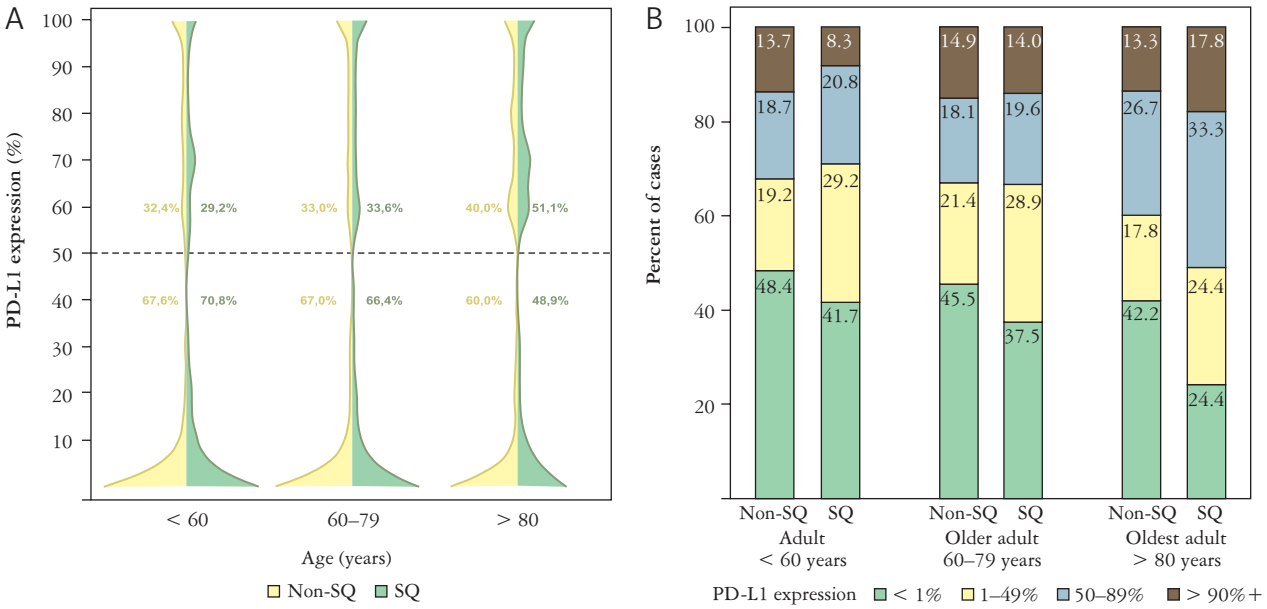

Figure 3. **A**) Programmed death-ligand 1 (PD-L1) expression distribution by age group and histology type. Percentages with PD-L1 expression < 50% and ≥ 50% are provided for each subgroup. **B**) PD-L1 expression distribution by age group and histology type including 4-tier PD-L1 expression cut-off levels

PD-L1 – programmed death-ligand 1, SQ – squamous carcinoma

Table II. Age and histology as predictors of the programmed death-ligand 1 expression level

PD-L1 level	Factor	RRR (95% CI)	*p*-value
1–49%			
	Histopathology		
	Non-squamous	Ref.	
	Squamous	1.69 (1.29–2.20)	< 0.0001
	Age group		
	Adult (< 60)	Ref.	
	Older-adult (60–79)	1.15 (0.81–1.63)	0.449
	Oldest-adult (80+)	1.19 (0.61–2.31)	0.601
≥ 50%			
	Histopathology		
	Non-squamous	Ref.	
	Squamous	1.25 (0.98–1.60)	0.073
	Age group		
	Adult (<60)	Ref.	
	Older adult (60–79)	1.13 (0.82–1.54)	0.444
	Oldest adult (80+)	1.92 (1.11–3.34)	0.02

PD-L1 – programmed death-ligand 1, RRR – relative risk ratios

TPS ≥ 90% in 41.2% and 38.7% of patients aged < 80 and ≥ 80 years, respectively [11, 12]. From a clinical perspective, in the Phase 3 EMPOWER-Lung 1 trial, patients with TPS ≥ 90% treated with cemiplimab monotherapy achieved higher objective response rates, longer progression-free survival, and markedly prolonged OS compared with those with TPS 50–89%. At the same time, no such differential effect was observed with chemotherapy [13]. Real-world and pooled analyses have confirmed this pattern for pembrolizumab, with TPS ≥ 90% predicting higher response rates and more durable remissions [14]. Nevertheless, TPS ≥ 90% is not currently recognized as a separate treatment threshold in interna-

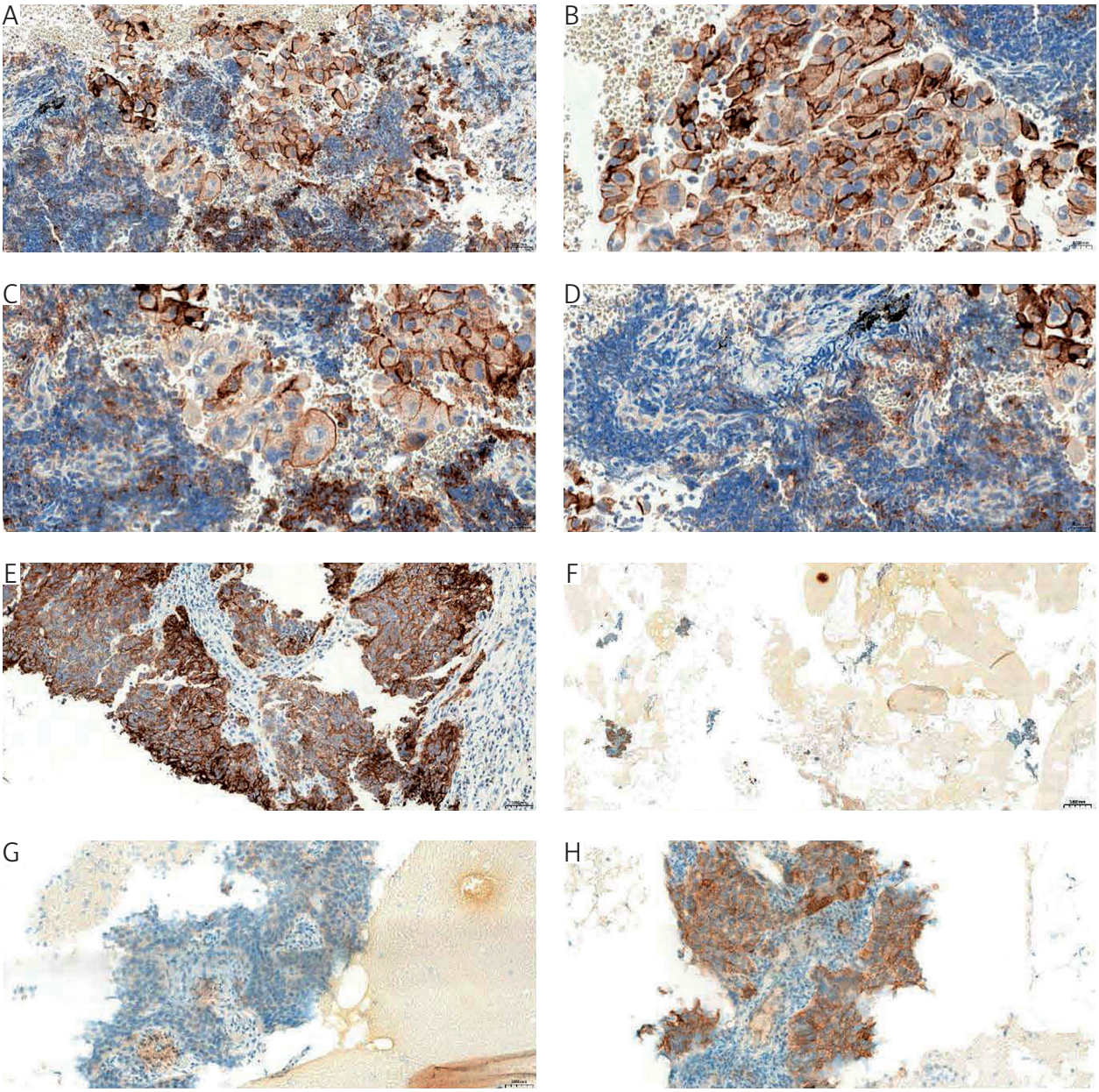

Figure 4. Programmed death-ligand 1 (PD-L1) no or low-expression cases. A) Adenocarcinoma with negative result, the weak brown coloration is located mainly within necrosis (200× magnification). B) Some AD cells express cytoplasmic reaction, which should not be counted as positive (400×). C, D) Non-squamous carcinoma with low-positive expression (2%) (200× and 400× magnification). E, F) Squamous carcinoma with 10% expression, in background the scattered macrophages can also present PD-L1 expression, which may lead to overestimation (200× and 400× magnification). **G, H**) Most of the material presents as negative (200×), but there is a single field with ≥ 100 PD-L1 positive cells (200×) – in such cases the pathologist can overestimate the immunohistochemistry expression level

tional guidelines. Clinical recognition of this subset, however, may improve patient selection for monotherapy and allow for tailored counseling regarding the likelihood of long-term disease control. Notably, some reports suggest an increased incidence of severe immune-related adverse events in this subgroup, underscoring the need for vigilant toxicity monitoring [15].

Current evidence does not support a consistent relationship between patient age and prevalence of PD-L1 expression in NSCLC. Large-scale diagnostic datasets, such as the Spanish reference-center cohort, report stable distributions of TPS (< 1%, 1–49%, ≥ 50%) across all age strata, including patients aged 75 years or older [16]. Comparable findings emerge from trial-screening cohorts in KEYNOTE-001, –010, and –024, where the proportion of PD-L1-high tumors remained similar irrespective of age, and from other real-world analyses in diverse healthcare settings [17, 18]. These observations underscore that chrono-

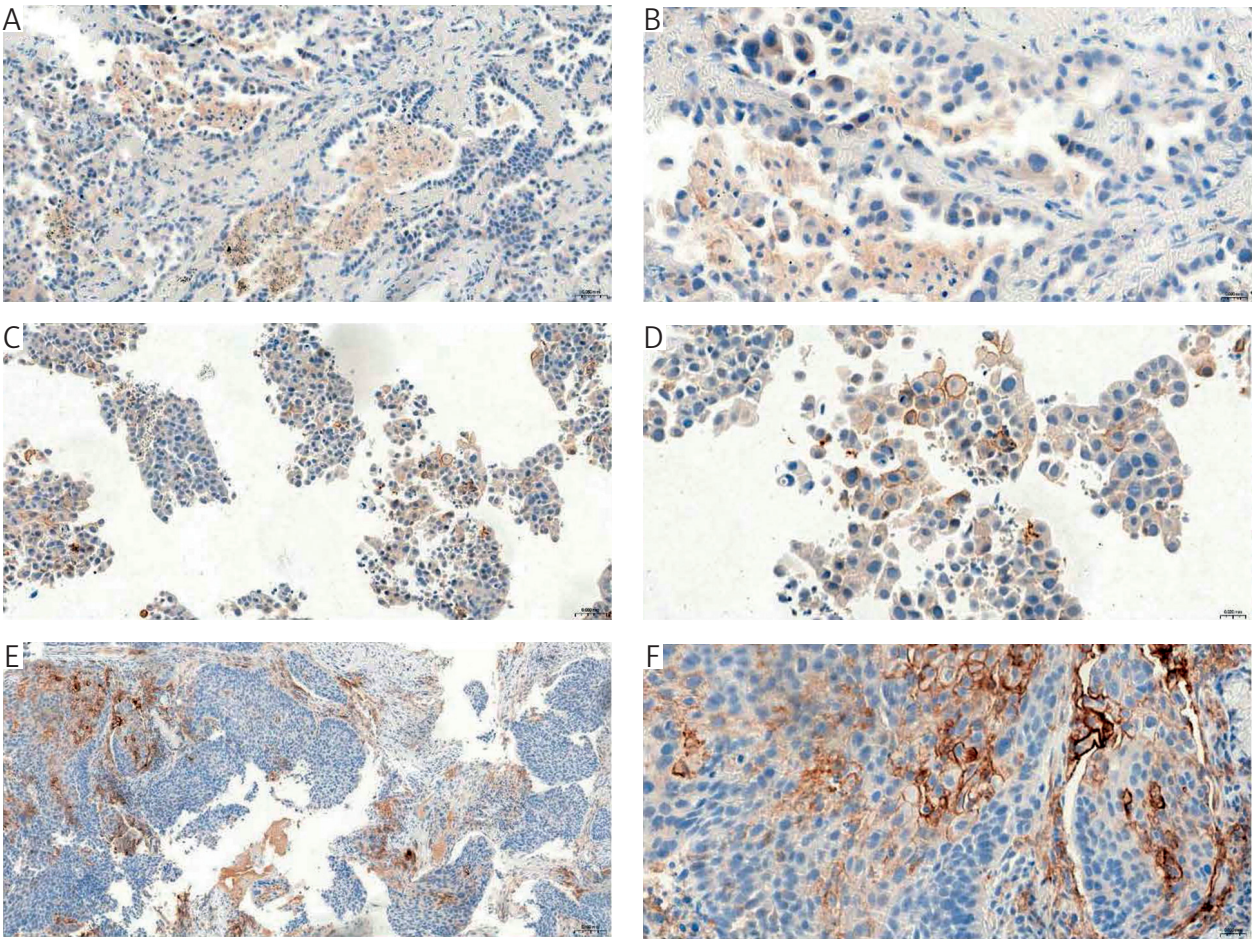

Figure 5. The PD-L1 no or low-expression cases. **A**) Adenocarcinoma with negative result, the weak brown coloration is located mainly within necrosis (200× magnification) **B**) Some adenocarcinoma cells express cytoplasmic reaction, which should not be counted as positive (400×). **C, D**) Non-squamous carcinoma with low-positive expression (2%) (200× and 400× magnification). **E, F**) Squamous carcinoma with 10% expression, in background the scattered macrophages can also present PD-L1 expression, which may lead to overestimation (200× and 400× magnification)

logical age alone is not a driver of PD-L1 expression patterns, and that histopathological features – such as squamous histology and poor differentiation – are stronger correlates of PD-L1 overexpression [19].

In our study, the oldest adult group (≥ 80 years) demonstrated a statistically significant prevalence of PD-L1 TPS ≥ 50%, even after adjustment for histology, indicating that these patients should not be excluded from consideration for immunotherapy and may represent an increasingly crucial treatment-eligible population in aging societies. Latest meta-analyses and pooled trial data show that older adults (≥ 60–79 years) and patients aged ≥ 75 years achieve similar efficacy and durability of response to PD-1/PD-L1 inhibitor monotherapy compared with younger adults, with no excess in treatment-related toxicity and a safety profile generally superior to chemotherapy [20, 21]. In the oldest adult category (≥ 80 years), data remain limited, but the retrospective multicenter series report acceptable tolerability (grade ≥ 3 immune-related adverse events ~10–15%) and clinical benefit in a subset of patients, reinforcing the importance of careful functional and geriatric assessment rather than age-based exclusion [22, 23]. Overall, these findings suggest that age should not be used as a biomarker for PD-L1 testing or immunotherapy eligibility, and instead support a nuanced, biology- and function-driven approach to treatment selection in older NSCLC populations.

Marked differences in PD-L1 expression have been documented across histological subtypes of NSCLC. Several large series and single-center studies report that squamous cell carcinomas are more likely to exhibit high PD-L1 expression. In contrast, non-squamous tumors, particularly adenocarcinomas, more often display low (< 1% TPS) or absent staining [16]. These trends have been reproduced in independent cohorts, although other analyses have not confirmed statistically significant differences [21, 24, 25]. At the high-expression threshold (TPS ≥ 50%), results remain inconsistent: some studies demonstrate a higher prevalence in squamous histology, while

others show equivalent or even lower rates compared to non-squamous tumors [16, 26, 27]. Such discrepancies likely arise from a combination of biological heterogeneity, variability in PD-L1 IHC assays and interpretation criteria, and differences in the cut-off points applied for classification [6, 28]. In our study, the likelihood of intermediate PD-L1 expression (1–49%) was significantly higher in squamous histology than in non-squamous histology, with an RRR of 1.69 (95% CI: 1.29–2.20).

Assessment accuracy is further influenced by intratumoral heterogeneity and the type of tissue specimens. Comparative evaluations of paired small biopsies and surgical resections have revealed discordance rates exceeding 30%, reflecting the propensity of limited samples to both under- and overestimate PD-L1 status [29, 30]. A meta-analysis confirmed that PD-L1 detection rates are significantly lower in small biopsies compared with excised specimens, underscoring the superior reliability of surgical material for guiding immunotherapy decisions [31]. The recent Bayesian meta-analysis by Ngo *et al*. showed a comprehensive explanation for these discrepancies, demonstrating that differences in PD-L1 prevalence are driven not only by biological heterogeneity but also by methodological and technical variability in testing practices [6]. The observed variation in PD-L1 expression between studies is significantly influenced by pre-analytical factors (i.e., the longer the storage, the lower the detectability of PD-L1). Moreover, inter-laboratory variability remained a significant contributor to inconsistent results; however, the inclusion of data from multiple laboratories in a single study can help mitigate this effect through statistical normalization. While tumor characteristics, including histologic subtype, stage, and driver mutation status, do contribute to the underlying expression of PD-L1, they do not fully explain the wide range of reported prevalence rates. For example, the pooled prevalence of PD-L1 expression ≥ 1% using the 22C3 clone was estimated at 58.3% (95% CI: 49.8–66.1%), and ≥ 50% at 27.0% (95% CI: 21.2–33.1%), despite being measured in broadly comparable NSCLC populations [6]. A key strength of our study lies in its large sample size and single-center design, ensuring that all specimens underwent a uniform, standardized pre-analytical workflow, including tissue handling, fixation, and staining protocols, under the same laboratory conditions. This methodological consistency minimizes inter-laboratory variability, limits the influence of technical artifacts, and enhances the reliability and comparability of PD-L1 assessment across the study cohort.

The possibility that pathologists' knowledge of clinical context, including treatment access and eligibility criteria, may influence interpretation has been an area of increasing concern regarding both ethics and methodology, although direct evidence remains limited. To date, there has been no definitive study demonstrating that pathologists are prone to overestimating PD-L1 expression to facilitate patient access to immunotherapy. Nevertheless, indirect evidence from pathology and cognitive science literature supports the plausibility of such a phenomenon. Known as contextual bias or expectation bias, this form of cognitive distortion occurs when a diagnostician's knowledge of external factors – such as the patient's clinical status, therapeutic options, or anticipated benefit – unconsciously influences their interpretation of morphologic or immunohistochemical findings. In clinical practice, PD-L1 expression is often assessed by IHC using thresholds (e.g., TPS ≥ 1% or ≥ 50%) that determine access to costly therapies such as pembrolizumab. Several studies, including international surveys, have revealed interobserver and inter-laboratory variability in PD-L1 scoring, despite the use of standardized assays such as 22C3 or SP263 [32, 33]. While such variability is frequently attributed to technical factors (e.g., fixation time, platform differences), the role of human interpretation – particularly at low-expression thresholds – cannot be discounted. A study by Gosney *et al*. found that pathologists vary in their thresholds for scoring PD-L1 positivity and that awareness of therapeutic implications may subconsciously influence the assignment of borderline scores [33]. Similarly, interobserver variation studies suggest that even trained pathologists may produce divergent results on the same specimen, especially when TPS scores fall near clinically significant cut-offs [34, 35]. The current consensus among professional societies is unequivocal: interpretations should remain blinded to clinical context whenever possible, especially in research or trial settings (IASLC, 2021) [3]. In routine practice, while complete blinding may not be feasible, adherence to validated scoring protocols, interobserver calibration, and participation in external quality assurance programs are strongly recommended to mitigate such biases.

In this context, the rapid development of AI-based PD-L1 assessment tools in NSCLC has generated considerable interest as a means of reducing inter- and intra-observer variability. AI-assisted platforms, such as Lunit SCOPE PD-L1, PathAI's AIM-PD-L1, Indica Labs' Lung PD-L1 AI, and Roche's uPath PD-L1 (SP263), have demonstrated promising results in improving interobserver agreement and standardizing TPS evaluation [36–38]. For example, the use of Lunit SCOPE PD-L1 increased diagnostic concordance among pathologists by 81.4–90.2% ($p < 0.001$), and identified additional cases with potential eligibility for immunotherapy in the PD-L1 negative subgroup [39]. However, these AI algorithms currently remain research-use only, and none of the above tools has received FDA approval for routine clinical application to date.

While PD-L1 expression remains the principal biomarker for selecting patients with NSCLC for immune checkpoint blockade, recent advances have expanded therapeutic options even for those with PD-L1 negative tumors (TPS < 1%). In the pivotal KEYNOTE-407 and KEYNOTE-189 trials, the combination of pembrolizumab with platinum-based chemotherapy significantly improved OS and PFS across all PD-L1 subgroups, including those with no detectable PD-L1 expression. However, the benefit of treatment was less in this subgroup [40, 41]. Similarly, the CheckMate 9LA study demonstrated that first-line therapy with nivolumab plus ipilimumab, followed by two cycles of chemotherapy, improved survival outcomes in both PD-L1 positive and PD-L1 negative patients [42]. Moreover, novel checkpoint inhibitors targeting LAG-3 (e.g., relatlimab, as studied in melanoma and under investigation in NSCLC and TIGIT (e.g., tiragolumab) show early signs of efficacy in PD-L1 low or negative tumors, potentially broadening future treatment indications [43, 44]. These developments collectively challenge the binary paradigm of PD-L1 positive vs. negative and emphasize that therapeutic decisions should not rely solely on threshold-based eligibility. In our study, 47% of NSCLCs were classified as PD-L1 negative, which represents a substantial proportion of patients who may still be eligible for emerging treatment strategies beyond PD-1/PD-L1 monotherapy.

Conclusions

Thus, a well-conducted, standardized PD-L1 evaluation ensures appropriate patient stratification and access to the most effective therapeutic options, including monotherapy, chemoimmunotherapy, or novel combination strategies. In this context, pathologists play a pivotal role as gatekeepers of precision oncology, and ensuring quality, objectivity, and consistency in PD-L1 testing is crucial for delivering optimal care.

Disclosures

1. Institutional review board statement: The study was conducted in accordance with the principles of the Declaration of Helsinki, and strict measures were taken to ensure the protection of patient confidentiality and data privacy.
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

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