



EP11.29: Continuous Tumor Cell PD-L1 Expression Is Associated With Inferior Overall Survival in a Real-World Lung Cancer Cohort

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INTRODUCTION

Programmed death-ligand 1 (PD-L1) is widely used to guide immunotherapy selection in thoracic oncology, yet its independent prognostic relevance in routine practice remains uncertain. We evaluated whether continuous tumor-cell PD-L1 expression was associated with overall survival (OS) in a large single-center, record-level lung cancer cohort.

METHODS

STUDY DESIGN AND COHORT

Retrospective single-center analysis of 3,545 pathology-linked lung-cancer records with tumor-cell PD-L1 testing. We excluded 101 records lacking numeric PD-L1 (n=57), calculable OS (n=41), or positive OS (n=3), leaving 3,444 records and 903 deaths.

EXPOSURE AND OUTCOME

Tumor-cell PD-L1 was modeled per 10-percentage-point increase and as <1%, 1-49%, and ≥50%. OS was measured from the recorded examination date to death or last follow-up.

STATISTICAL ANALYSIS

Kaplan-Meier estimates and a global log-rank test compared categories. Multivariable Cox regression adjusted for age, sex, stage, histology, and documented immunotherapy, targeted therapy, and chemotherapy; missing stage/histology were retained as categories. The PD-L1 × immunotherapy-record interaction was exploratory because treatment timing was unavailable.

REFERENCES, FUNDING & AI USE DISCLOSURE

REFERENCES

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AI USE DISCLOSURE

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DISCUSSION AND CONCLUSION

DISCUSSION

Prior prognostic evidence in lung cancer, particularly NSCLC, is heterogeneous. PD-L1 was associated with poorer OS in 1,070 resected NSCLC cases, but weakened after postoperative-therapy adjustment¹; it was not independently prognostic in 982 specimens from three adjuvant NSCLC trials². A 50-study lung-cancer meta-analysis linked higher PD-L1 expression with worse OS, especially in Asian and resected/earlier-stage cohorts³. Our record-level cohort shows a similar adverse association, but comparisons are limited by missing histology and differences in assay, stage, treatment timing, and residual confounding. Agreement between categorical and continuous analyses reduces cutoff dependence but requires external confirmation.

KEY FINDING

Higher tumor-cell PD-L1 was associated with poorer OS in both categorical and continuous analyses.

CONCLUSION

Continuous tumor-cell PD-L1 warrants further evaluation as a prognostic marker. This association is neither a causal treatment-effect estimate nor evidence for stand-alone treatment selection. Validation requires standardized assays, time-resolved treatment data, and complete covariates.

LIMITATIONS

Limitations include the retrospective single-center design, missing stage/histology, heterogeneous assays, unavailable treatment timing, residual confounding, and unverified unique-patient N. The PD-L1 proportional-hazards diagnostic was borderline (P=0.0506); HRs summarize model-based associations rather than constant effects over follow-up.

TABLE 1. COHORT PROFILE

Characteristic	Value
Analyzed records	3,444
Deaths	903 (26.2%)
Age, median (IQR), years	61 (53-67)
Male sex	1,937/3,443 (56.3%)
Documented immunotherapy	244/3,444 (7.1%)
Median follow-up, months	89.3
PD-L1 <1%	2,130 (61.8%)
PD-L1 1-49%	907 (26.3%)
PD-L1 ≥50%	407 (11.8%)

Values are n (%) unless otherwise stated. Denominators vary where sex data were unavailable; follow-up was estimated by reverse Kaplan-Meier.

RESULTS

Among 3,444 eligible records (903 deaths), unadjusted OS differed across PD-L1 categories (global log-rank P=3.2 × 10⁻³⁷), with lower 36-month Kaplan-Meier estimates at higher PD-L1: 86.9%, 75.7%, and 66.6% for <1%, 1-49%, and ≥50%. Each 10-percentage-point increase in tumor-cell PD-L1 was associated with an 8.8% higher adjusted hazard of death (HR 1.088, 95% CI 1.064-1.113; P=2.8 × 10⁻¹³). The exploratory interaction comparing records with versus without a documented immunotherav record was not statistically significant (ratio of HRs 0.923, 95% CI 0.849-1.003; P=0.060).

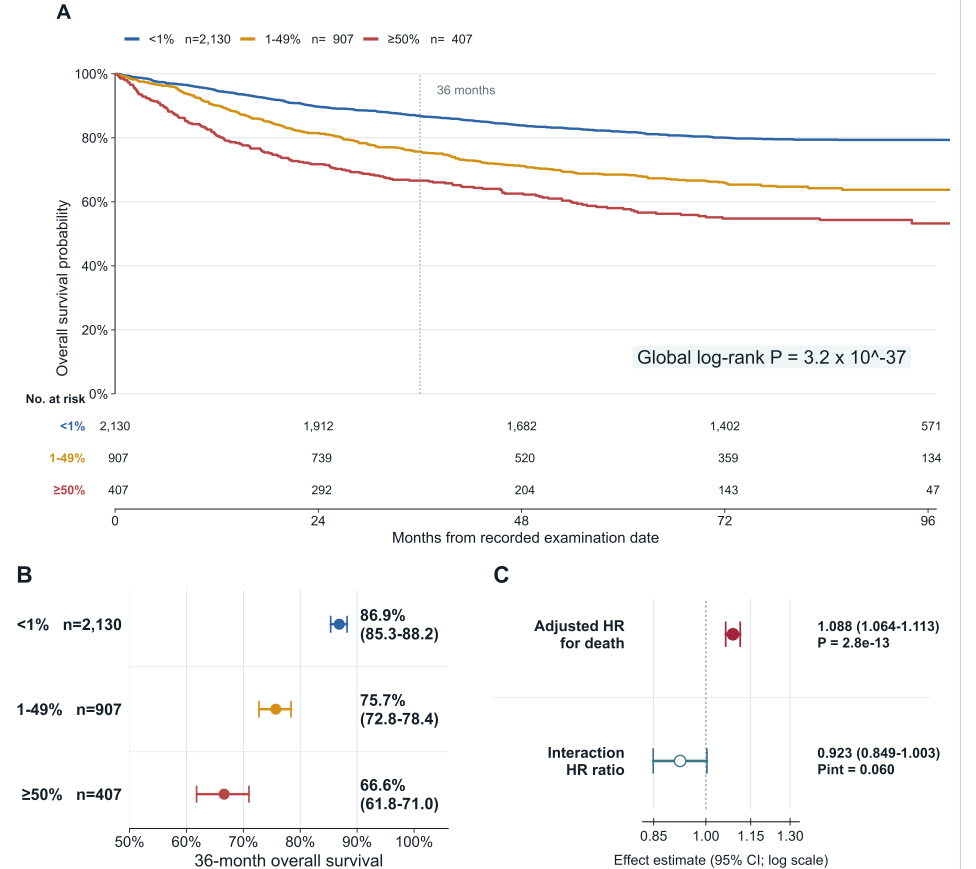


Figure 1. A, Unadjusted Kaplan-Meier OS by PD-L1 category with records at risk; the global log-rank test compares all categories; censor marks omitted. B, 36-month OS (95% CI) from the same curves; not an independent analysis. C, Adjusted HR for death per 10-percentage-point PD-L1 increase (primary Cox model) and exploratory ratio of PD-L1 HRs in records with versus without a documented immunotherapy record (separate interaction Cox model). Cox models adjusted for age, sex, stage, histology, and documented systemic therapies; missing stage/histology were categories. Counts denote records; unique-patient N was unverified. The interaction CI included 1.0 (P=0.060); unavailable treatment timing precludes treatment-effect or predictive-biomarker interpretation.