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Clinical Predictors of Progression-free Survival in Patients with Metastatic Castration-resistant Prostate Cancer Treated with Lu-177 PSMA: A Real-world Study

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ABSTRACT

Objective: Accurate pretreatment risk assessment remains an important challenge for patients with metastatic castration-resistant prostate cancer (mCRPC) undergoing lutetium- 177-labeled prostate-specific membrane antigen (Lu-177 PSMA) radioligand therapy, highlighting the need for reliable prognostic markers.

Material and Methods: We retrospectively analyzed 52 consecutive patients with mCRPC who underwent Lu-177 PSMA radioligand therapy at a single tertiary referral center. Baseline demographic, laboratory, and imaging characteristics were recorded before treatment initiation. Progression-free survival (PFS) and overall survival (OS) were estimated using the Kaplan-Meier method, and baseline factors associated with PFS were examined using Cox proportional hazards regression models.

Results: After a median follow-up of 14.0 months, the median PFS and OS were 8.34 and 16.99 months, respectively. Disease progression occurred in 48 patients (92.3%), while 42 (80.8%) died during follow-up. Elevated baseline prostate-specific antigen (PSA) levels and the presence of visceral metastases were associated with significantly shorter PFS. In multivariable analysis, baseline PSA [adjusted hazard ratio (HR): 1.446; 95% confidence interval (CI): 1.204-1.738; p<0.001] and visceral metastasis (adjusted HR: 2.441; 95% CI: 1.049-5.682; p=0.038) remained independently associated with disease progression. Baseline maximum standardized uptake value (SUV_{max}) and mean SUV did not demonstrate significant prognostic value.

Conclusion: Baseline PSA and visceral metastasis were independently associated with shorter PFS in patients with mCRPC treated with Lu-177 PSMA, whereas conventional semiquantitative PSMA positron emission tomography/computed tomography parameters were not. These findings suggest that routinely available baseline clinical characteristics may help identify patients at increased risk of early progression prior to radioligand therapy.

Keywords: Metastatic castration-resistant prostate cancer; Lu-177 PSMA; prostate-specific antigen; progression-free survival; visceral metastasis

INTRODUCTION

Metastatic castration-resistant prostate cancer (mCRPC) represents the terminal stage of prostate cancer progression and continues to impose a substantial clinical burden despite remarkable advances in systemic therapy. The introduction of androgen receptor signaling inhibitors, taxane-based chemotherapy, PARP inhibitors in selected patients, and prostate-specific membrane antigen (PSMA)-targeted radioligand therapy has significantly prolonged survival. Nevertheless, disease progression remains inevitable for most patients, highlighting the importance of reliable prognostic markers that may facilitate individualized treatment planning and optimize therapeutic sequencing.[1,2]

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Among recently introduced treatment strategies, lutetium-177-labeled prostate-specific membrane antigen (Lu-177 PSMA) radioligand therapy has become an established option for patients with PSMA-positive mCRPC. Its clinical benefit was demonstrated in the VISION and TheraP trials, which showed improvements in progression-free survival (PFS), overall survival (OS), and patient-reported outcomes compared with standard therapeutic approaches. Consequently, Lu-177 PSMA has been incorporated into contemporary international treatment guidelines for appropriately selected patients with advanced disease.[3-6]

Although many patients derive meaningful benefit from Lu-177 PSMA therapy, treatment outcomes remain heterogeneous. Some individuals achieve durable disease control, whereas others experience rapid progression despite adequate PSMA uptake on baseline positron emission tomography/computed tomography (PET/CT) imaging. This variability has increased interest in identifying pretreatment factors that may predict clinical outcomes and support risk stratification before therapy initiation.[7-9]

Previous studies have identified several baseline clinical characteristics, including serum prostate-specific antigen (PSA), disease burden, visceral metastatic involvement, and performance status, as potential prognostic indicators in patients undergoing Lu-177 PSMA therapy. However, the reported prognostic impact of these variables has not been entirely consistent, likely reflecting differences in patient characteristics, treatment sequences, and study methodology across published cohorts.[9-11]

Baseline PSMA PET/CT is indispensable for confirming sufficient PSMA expression and determining eligibility for radioligand therapy. In addition to its role in patient selection, semiquantitative imaging parameters such as maximum standardized uptake value (SUV_{max}) and mean SUV (SUV_{mean}) have been investigated as possible prognostic biomarkers. Nevertheless, existing evidence remains conflicting, and it is uncertain whether these PET-derived parameters provide prognostic information beyond that offered by routinely available clinical variables.[12-14]

Real-world investigations provide valuable complementary evidence to randomized clinical trials by reflecting the diversity of patients encountered in routine oncology practice. Therefore, the present study aimed to evaluate baseline clinical and imaging factors associated with PFS in patients with mCRPC treated with Lu-177 PSMA in a real-world setting, with particular emphasis on comparing the prognostic contributions of conventional clinical variables and semiquantitative PSMA PET/CT parameters.

MATERIAL AND METHODS

Study Design and Patient Population

This retrospective observational study was conducted at a single tertiary referral center and included consecutive patients with mCRPC who underwent Lu-177 PSMA radioligand therapy at Pamukkale University between May 2018 and April 2025. Survival follow-up was updated through May 2026. Patients were eligible if they had histologically confirmed prostate adenocarcinoma, metastatic castration-resistant disease, a baseline PSMA PET/CT examination performed before treatment initiation, and complete clinical follow-up data. Individuals lacking pretreatment imaging or essential clinical information were excluded from the analysis.

The study protocol was approved by the Pamukkale University Non-Interventional Clinical Research Ethics Committee (approval number: E.895531, date: 01.07.2026) and carried out in accordance with the ethical principles of the Declaration of Helsinki. The requirement for written informed consent was waived due to the retrospective design of the study.

Clinical information was retrieved from electronic medical records, including demographic characteristics, Gleason score, serum PSA level at baseline, interval from initial diagnosis to Lu-177 PSMA therapy, previous systemic treatments, metastatic disease sites, and the total number of treatment cycles administered.

PSMA PET/CT Assessment

Pretreatment PSMA PET/CT examinations obtained before the first Lu-177 PSMA administration were reviewed. The semiquantitative imaging variables analyzed were the SUV_{max} and SUV_{mean}, both recorded on the baseline scan. SUV_{max} and SUVmean were measured in the PSMA-avid lesion with the highest tracer uptake on the baseline PET/CT examination, according to the institutional imaging protocol.

Follow-up and Study Endpoints

Patients were monitored from the initiation of Lu-177 PSMA therapy until radiological or clinical progression, death, or the last available follow-up. PFS was defined as the interval between the first treatment cycle and documented disease progression or death from any cause, whichever occurred first. OS was calculated from the first administration of Lu-177 PSMA to death from any cause or to the date of the last follow-up for surviving patients. Median follow-up was calculated from the date of the first Lu-177 PSMA administration to the date of the last available follow-up or death.

Statistical Analysis

Continuous variables are presented as medians with interquartile ranges (IQRs), whereas categorical variables are reported as frequencies and percentages. Survival distributions were estimated using the Kaplan-Meier method and compared with the log-rank test. Potential prognostic factors associated with PFS were initially assessed using univariable Cox proportional hazards regression. Variables considered clinically relevant, together with those demonstrating a p-value below 0.10 in univariable analyses, were subsequently entered into a multivariable Cox regression model. Hazard ratios (HRs) and corresponding 95% confidence intervals (CIs) were calculated, and statistical significance was defined as a two-sided p-value below 0.05. All statistical analyses were performed using IBM SPSS Statistics, version 26.0 (IBM Corp., Armonk, NY, USA). Baseline PSA was entered into the Cox proportional hazards regression model as a continuous variable. Variables included in the multivariable model were selected based on clinical relevance and on a univariable p-value <0.10.

RESULTS

Patient Characteristics

A total of 52 patients with mCRPC who were treated with Lu-177 PSMA therapy were included. The median age was 64.5 years (IQR: 58.8-71.3), and the median interval from initial diagnosis to Lu-177 therapy was 37.0 months (IQR: 20.3-77.3). The median baseline PSA level was 78.07 ng/mL (IQR: 21.43-226.50), and patients received a median of 3 Lu-177 cycles (IQR: 2-6). Most patients had Gleason scores of 9-10 (63.5%). Bone metastases were present in all patients, whereas lymph node and visceral metastases were observed in 55.8% and 23.1% of patients, respectively. The median baseline SUV_{max} and SUV_{mean} values were 19.46 (IQR: 13.39-36.67) and 8.25 (IQR: 5.98-14.04), respectively (Table 1).

TABLE 1: Baseline demographic, clinical, treatment, and PSMA PET/CT characteristics of patients (n=52).

Variable	Overall (n=52)
Age, years, median (IQR)	64.5 (58.8-71.3)
Time from initial diagnosis to Lu-177 therapy, months, median (IQR)	37.0 (20.3-77.3)
Baseline PSA before Lu-177 (ng/mL), median (IQR)	78.07 (21.43-226.50)
Gleason score	
≤7	11 (21.2%)
8	8 (15.4%)
9-10	33 (63.5%)
Number of Lu-177 cycles, median (IQR)	3 (2-6)
Treatment history before Lu-177 PSMA therapy	
ADT only	5 (9.6%)
ADT + taxane chemotherapy	25 (48.1%)
ADT + radiotherapy	4 (7.7%)
ADT + taxane chemotherapy + radiotherapy	17 (32.7%)
Taxane chemotherapy only	1 (1.9%)
Metastatic sites before Lu-177	
Bone metastasis	52 (100%)
Lymph node metastasis	29 (55.8%)
Visceral metastasis	12 (23.1%)
Baseline PSMA PET/CT parameters	
SUV_{max}, median (IQR)	19.46 (13.39-36.67)
SUV_{mean}, median (IQR)	8.25 (5.98-14.04)

ADT: Androgen deprivation therapy; IQR: Interquartile range; PET/CT: Positron emission tomography/computed tomography; PSA: Prostate-specific antigen; PSMA: Prostate-specific membrane antigen; SUV: Standardized uptake value; Lu: Lutetium.

Survival Outcomes

After a median follow-up of 14.0 months, 48 patients (92.3%) experienced disease progression. The median PFS was 8.34 months, whereas the median OS was 16.99 months. During follow-up, 42 patients (80.8%) died (Table 2).

TABLE 2: Survival outcomes of patients treated with Lu-177 PSMA (n=52).

Outcome	Value
Median follow-up, months	14.0
Median PFS, months	8.34
PFS events, n (%)	48 (92.3)
Median OS, months	16.99
Deaths, n (%)	42 (80.8)

Median follow-up was calculated from the initiation of Lu-177 PSMA therapy to the last follow-up or death.
OS: Overall survival; PFS: Progression-free survival; PSMA: Prostate-specific membrane antigen; Lu: Lutetium.

Prognostic Analyses

Patients with baseline PSA values above the cohort median (>78.07 ng/mL) had significantly shorter PFS than patients with lower PSA values (median PFS: 4.96 vs. 14.29 months; log-rank p<0.001) (Figure 1). Likewise, patients with visceral metastases had significantly shorter PFS compared with those without visceral involvement (median PFS: 4.17 vs. 11.43 months; log-rank p<0.001) (Figure 2).

In contrast, no significant differences in PFS were observed based on baseline SUV_{max} (8.34 vs. 7.82 months; log-rank p=0.931) or baseline SUV_{mean} (7.72 vs. 8.61 months; log-rank p=0.612) (Table 3).

In the univariable Cox regression analysis, baseline PSA (HR: 1.524; 95% CI: 1.272-1.825; p<0.001) and visceral metastasis

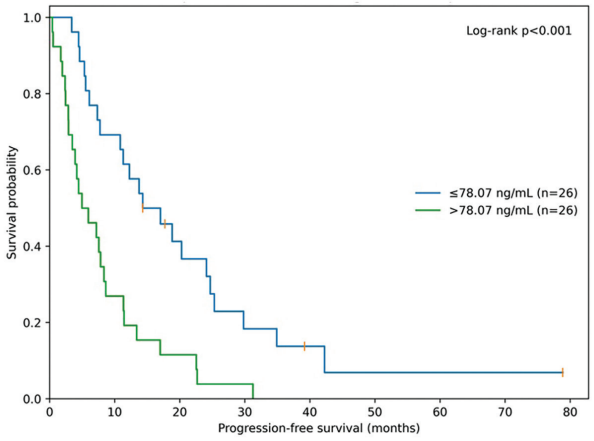

FIGURE 1: Kaplan-Meier curve for PFS according to baseline PSA.

PSA: Prostate-specific antigen; PFS: Progression-free survival

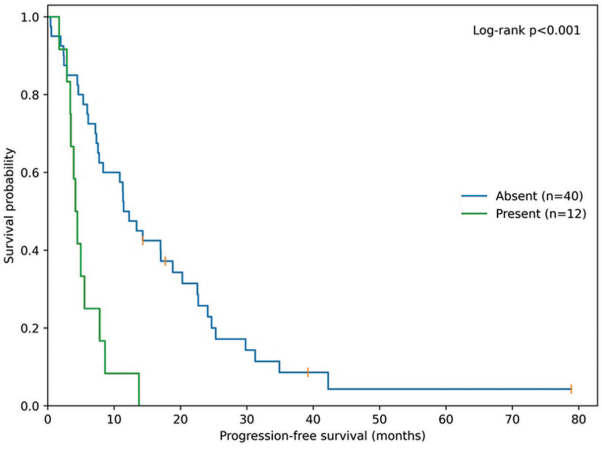

FIGURE 2: Kaplan-Meier curve for PFS according to visceral metastasis.

PFS: Progression-free survival

TABLE 3: Kaplan-Meier and log-rank comparisons for PFS.

Variable	Group	Median PFS (months)	Log-rank p-value
Baseline PSA	≤78.07 ng/mL	14.29	**<0.001**
	>78.07 ng/mL	4.96	
Visceral metastasis	Absent	11.43	**<0.001**
	Present	4.17	
SUV_{max}	≤19.46	8.34	0.931
	>19.46	7.82	
SUV_{mean}	≤8.25	7.72	0.612
	>8.25	8.61	

Bold values indicate statistically significant results (p<0.05).
PSA: Prostate-specific antigen; PFS: Progression-free survival; SUV: Standardized uptake value.

(HR: 3.815; 95% CI: 1.827-7.966; p<0.001) were significantly associated with shorter PFS. Age, time from diagnosis to Lu-177 therapy, Gleason score, lymph node metastasis, SUV_{max}, and SUV_{mean} were not significantly associated with PFS (Table 4).

In the multivariable model, baseline PSA remained independently associated with shorter PFS (adjusted HR: 1.446; 95% CI: 1.204-1.738; p<0.001). Visceral metastasis also remained independently associated with inferior PFS (adjusted HR: 2.441; 95% CI: 1.049-5.682; p=0.038). Neither age nor baseline SUV_{max} retained statistical significance (Table 5).

TABLE 4: Univariable Cox regression analysis for progression-free survival.

Variable	n	HR	95% CI	p-value
Age (per 1-year increase)	52	1.025	0.990-1.062	0.165
Time from initial diagnosis to Lu-177 therapy (per 1-month increase)	50	0.999	0.992-1.006	0.748
Baseline PSA (per doubling, log2-transformed)	52	1.524	1.272-1.825	**<0.001**
Gleason score ≥9 (vs. ≤8)	52	1.514	0.832-2.756	0.175
Visceral metastasis (yes vs. no)	52	3.815	1.827-7.966	**<0.001**
Lymph node metastasis (yes vs. no)	52	1.353	0.759-2.414	0.305
SUV_{max} (per 1-unit increase)	52	0.999	0.987-1.010	0.832
SUV_{mean} (per 1-unit increase)	52	0.989	0.952-1.028	0.583

Bold values indicate statistically significant results (p<0.05). Baseline PSA was log2-transformed; therefore, the HR represents the change in progression risk for each doubling of PSA.
CI: Confidence interval; HR: Hazard ratio; PSA: Prostate-specific antigen; SUV: Standardized uptake value; Lu: Lutetium.

TABLE 5: Multivariable Cox regression analysis for progression-free survival.

Variable	Adjusted HR	95% CI	p-value
Age (per 1-year increase)	1.033	0.997-1.070	0.072
Baseline PSA (per doubling, log2-transformed)	1.446	1.204-1.738	**<0.001**
Visceral metastasis (yes vs. no)	2.441	1.049-5.682	**0.038**
SUV_{max} (per 1-unit increase)	0.990	0.977-1.004	0.164

Bold values indicate statistically significant results (p<0.05).
CI: Confidence interval; HR: Hazard ratio; PSA: Prostate-specific antigen; SUV: Standardized uptake value.

DISCUSSION

The present study explored baseline predictors of PFS in a real-world cohort of patients with mCRPC treated with Lu-177 PSMA radioligand therapy. Two principal observations emerged from our analysis. First, elevated pretreatment PSA levels and visceral metastatic disease were consistently associated with an increased risk of progression. Second, these associations remained significant after multivariable adjustment, whereas conventional semiquantitative PSMA PET/CT parameters, including SUV_{max} and SUV_{mean}, did not provide independent prognostic information.

The survival outcomes observed in this cohort were comparable with those reported in major prospective trials and real-world studies evaluating Lu-177 PSMA therapy. Median PFS and OS were similar to the ranges described in the VISION trial and subsequent observational series, supporting the representativeness of our patient population.[3,4,9,15] Although randomized clinical trials have established the efficacy of Lu-177 PSMA, observational studies remain essential because they reflect the broader spectrum of patients encountered in routine oncology practice. Consequently, real-world evidence plays an important role in evaluating prognostic factors that may influence treatment outcomes outside the controlled setting of clinical trials.

Among all baseline variables evaluated, serum PSA demonstrated the strongest association with disease progression. Patients with higher pre-treatment PSA concentrations had significantly shorter PFS, and this relationship persisted after adjustment for potential confounding variables. Elevated PSA is widely regarded as a surrogate marker of tumor burden and biological aggressiveness, which may partly explain the observed association. Previous investigations have similarly identified pretreatment PSA as an adverse prognostic factor in patients receiving Lu-177 PSMA, although the magnitude of its impact has varied among published cohorts because of differences in study design, patient selection, and therapeutic sequencing.[7,9-11] Our findings support the potential clinical usefulness of baseline PSA as a simple and readily available biomarker that may assist pretreatment risk assessment.

Our results also extend previous observations regarding PSA-based prognostication. Whereas Soydal et al. demonstrated that PSA response after treatment was associated with improved clinical outcomes, the present study focused exclusively on pretreatment characteristics. The independent association between baseline PSA and PFS suggests that serum PSA may provide prognostic information before therapy is initiated, complementing its established role as a marker of treatment response.[16]

Visceral metastasis was also independently associated with shorter PFS. Patients with visceral involvement experienced substantially shorter PFS than those without visceral disease. This finding is consistent with the recognized adverse biological behavior of visceral metastatic spread in mCRPC and agrees with previous reports identifying visceral involvement as an indicator of poor prognosis in patients undergoing Lu-177 PSMA therapy.[7,8,11] The persistence of this association after multivariable adjustment suggests its potential clinical relevance during pretreatment evaluation.

Unlike the clinical variables evaluated in this study, neither baseline SUV_{max} nor SUV_{mean} was independently associated with PFS. Although PSMA PET/CT is indispensable for confirming adequate PSMA expression and selecting candidates for radioligand therapy, the prognostic relevance of conventional semiquantitative uptake parameters remains uncertain. Previous studies have produced inconsistent findings, with some reporting a relationship between higher tracer uptake and favorable treatment outcomes, whereas others failed to demonstrate an independent association after adjustment for established clinical variables.[10,12,13] Differences in patient selection, imaging protocols, lesion measurement strategies, treatment timing, and study endpoints likely contribute to these conflicting observations. In this cohort, conventional SUV-based measurements alone were not significantly associated with PFS.

Several aspects of the present study strengthen its clinical relevance. All patients were treated at a single tertiary referral center following a relatively consistent therapeutic approach, and baseline PSMA PET/CT scans were available for every patient before treatment initiation. In addition, the concordance between the Kaplan-Meier survival analyses and the Cox regression models supports the robustness of the observed associations for baseline PSA and visceral metastatic disease.

Study Limitations

The findings of this study should, however, be interpreted in light of several limitations. The single-center setting may limit generalizability, and residual confounding by unmeasured clinical factors cannot be excluded. No internal or external validation was performed; therefore, the multivariable findings should be considered exploratory. The retrospective design introduces the possibility of selection bias, and the relatively limited sample size may have reduced the statistical power to detect weaker associations. Although multivariable analysis was performed, the number of available events limited the complexity of the regression model and precluded inclusion of a larger set of covariates. Furthermore, only conventional semiquantitative PET parameters (SUV_{max} and

SUV_{mean}) were evaluated. Because SUV_{max} and SUV_{mean} were derived from a single target lesion, they may not reflect whole-body tumor burden. More comprehensive volumetric biomarkers, including PSMA-derived tumor volume and total lesion PSMA, were not available; they may provide additional prognostic information. Treatment-related variables, such as PSA response during therapy and serial imaging assessments, were intentionally excluded because the primary objective was to identify prognostic factors available before the initiation of Lu-177 PSMA radioligand therapy. In addition, heterogeneity in post-progression treatments may have influenced OS. Furthermore, SUV measurements may be affected by differences in image acquisition and reconstruction protocols, which may limit the generalizability of PET-derived parameters.

CONCLUSION

Pre-treatment PSA level and the presence of visceral metastases were independently associated with shorter PFS in patients with mCRPC receiving Lu-177 PSMA radioligand therapy. By contrast, baseline SUV_{max} and SUV_{mean} were not significantly associated with PFS. These exploratory findings suggest that simple clinical variables available before treatment initiation may contribute to risk stratification. Prospective multicenter studies incorporating larger patient populations and advanced PSMA PET-derived volumetric biomarkers are warranted to further validate these observations.

Ethics

Ethics Committee Approval: The study protocol was approved by the Pamukkale University Non-Interventional Clinical Research Ethics Committee (approval number: E.895531, date: 01.07.2026)

Informed Consent: The requirement for written informed consent was waived due to the retrospective design of the study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: T.G.K., B.Y.T., G.G.D., Concept: T.G.K., T.D., B.Y.T., A.G.D., A.Y., Design: A.G.D., A.Y., T.Ş., G.G.D., Data Collection or Processing: T.G.K., S.T., T.D., M.Ö., T.Ş., Analysis or Interpretation: T.G.K., S.T., T.D., M.Ö., A.G.D., A.Y., T.Ş., Literature Search: T.G.K., S.T., M.Ö., B.Y.T., G.G.D., Writing: T.G.K., G.G.D.

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