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Clinical Outcomes and Prognostic Significance of PSA90 in Patients With Prostate Cancer and Initial PSA >1,000 ng/ml at Diagnosis

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Abstract

Background/Aim: Prostate cancer (PCa) patients with an initial prostate-specific antigen (PSA) level >1,000 ng/ml at diagnosis represent a rare and high-risk subgroup for which prognostic factors remain poorly defined. Therefore, we investigated the clinical features of this population and identified predictors of overall survival (OS).

Patients and Methods: The present study retrospectively analyzed 35 patients diagnosed with PCa and initial PSA >1,000 ng/ml at our institution between August 2013 and December 2024. Clinical characteristics, treatment courses, PSA responses, and survival outcomes were collected. A nadir PSA level ≤0.2 ng/ml and a 90% decline in PSA from baseline (PSA90) were evaluated. OS was estimated using the Kaplan-Meier method, and the impact of clinical variables on OS was assessed using univariate and multivariate Cox proportional hazards regression models.

Results: Median age was 74 years, and median initial PSA was 3,027 ng/ml. The median time to castration-resistant prostate cancer was 18.25 months, and median OS was 55.0 months. Univariate analyses revealed that PSA90 achievement correlated with longer OS, whereas age ≥75 years, an Eastern Cooperative Oncology Group performance status ≥1, elevated lactate dehydrogenase, and elevated C-reactive protein correlated with shorter OS. Multivariate analyses identified PSA90 achievement as the only independent predictor of prolonged OS (hazard ratio=0.13).

Conclusion: These results suggest the potential of PSA90 as a simple and valuable early biomarker for prognostic stratification in patients with PCa and initial PSA >1,000 ng/ml.

Keywords: Prostate cancer, PSA, prognosis, overall survival, biomarker.

Introduction

Prostate-specific antigen (PSA) is a glycoprotein secreted by prostatic epithelial cells and serves as a widely used biomarker for the detection, monitoring, and management of prostate cancer (PCa) (1). While the upper limit of normal PSA levels is generally considered to be ≤4.0 ng/ml, markedly elevated values are often associated with advanced or

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metastatic disease (2). PSA remains a cornerstone in the diagnosis of PCa and is routinely used to evaluate treatment responses and disease progression at all clinical stages (3).

Patients presenting with initial PSA >1,000 ng/ml are uncommon but often have widespread metastases and an aggressive tumor biology (4). Despite their clinical importance, these high-risk patients are frequently excluded from clinical trials, and data on their prognosis and treatment outcomes remain limited (5). Although the introduction of androgen receptor signaling inhibitors (ARSIs), such as abiraterone acetate and enzalutamide, has transformed the therapeutic landscape, prognostic studies specifically targeting this extreme PSA subgroup in the ARSI era remain limited.

PSA kinetics, including the PSA nadir and percentage decline from baseline, have been investigated as surrogate markers of treatment efficacy and survival in metastatic PCa (6, 7). The achievement of a PSA nadir ≤0.2 ng/ml or a ≥90% PSA decline from baseline (PSA90) has been associated with improved survival in general metastatic populations (8, 9). However, it remains unclear whether these surrogate endpoints retain their predictive value in patients with markedly elevated PSA levels.

Therefore, the present study investigated the clinical characteristics of patients with newly diagnosed PCa and initial PSA >1,000 ng/ml and identified prognostic factors for overall survival (OS). Specifically, we evaluated the prognostic relevance of achieving a PSA nadir ≤0.2 ng/ml and PSA90 in this understudied, high-risk cohort.

Patients and Methods

The present study was conducted in accordance with the guidelines of the Declaration of Helsinki and was approved by the Research Ethics Committee at our institution (No. 21-088). The need to obtain informed consent from patients was waived because of its retrospective design; however, an opportunity to opt out from this study was provided through the website of each institution.

Study design and population. The present study included 35 consecutive Japanese patients who were newly diagnosed with PCa and had an initial serum PSA level >1,000 ng/ml at our institution between August 2013 and December 2024. All patients were diagnosed with adenocarcinoma of the prostate based on histopathological findings from transrectal ultrasound-guided biopsy.

Treatment. The selection of first-line androgen deprivation therapy (ADT)-based hormonal therapy was generally made at the discretion of the attending physician without strictly determined criteria. ADT consisted of luteinizing hormone-releasing hormone (LHRH) agonists, such as leuprorelin or goserelin acetate, or the LHRH antagonist degarelix. Surgical castration was not performed on any patient. As a rule, bicalutamide, abiraterone acetate, apalutamide, enzalutamide, and darolutamide plus docetaxel were administered according to the standard dosing schedule, as described in previous studies (10-14). Treatment with any agent was continued until the occurrence of disease progression or intolerable adverse events (AEs). In cases of treatment-related AEs, dose modifications to agents were permitted.

Evaluation. The clinicopathological data of each patient assessed in the present study was obtained from electronic medical records. Prior to the initiation of first-line therapy, laboratory data, including PSA, alkaline phosphatase, lactate dehydrogenase (LDH), hemoglobin (Hb), and C-reactive protein (CRP), were measured with standard clinical testing methods, and all patients underwent computed tomography of the chest, abdomen, and/or pelvis as well as bone scintigraphy to evaluate the baseline extension of PCa. After the initiation of first-line therapy, measurements of PSA in addition to bone marrow, renal, and liver functions were performed every 4-6 weeks, while the intervals of radiological examinations were selected based on the discretion of each physician. A nadir PSA level ≤0.2 ng/ml and PSA90 were evaluated as potential surrogate markers of treatment responses and survival. Disease progression after the introduction of first-line therapy was assessed based on the Prostate Cancer Working Group 3 criteria (15).

Table I. *Characteristics of the 35 patients diagnosed with prostate cancer and initial PSA >1,000 ng/ml.*

Variables	n (%)
Age, years	74 (60-86)
Initial PSA*, ng/ml	3,027 (1,064-15,137)
ECOG PS	
0	10 (28.6)
1	19 (54.3)
2	4 (11.4)
3	2 (5.7)
Gleason score	
8	10 (28.6)
9	21 (60.0)
10	4 (11.4)
Metastatic sites	
Lung	8 (22.9)
Bone	33 (94.3)
Lymph node	29 (82.9)
ALP*, IU/l	268 (66-5,029)
LDH*, IU/l	222 (122-1,046)
Hb*, g/dl	12.7 (5.3-15.9)
CRP*, mg/dl	0.53 (0.01-20.6)

*Data presented as median (range). PSA, Prostate-specific antigen; ECOG PS, Eastern Cooperative Oncology Group performance status; ALP, alkaline phosphatase; LDH, lactate dehydrogenase; Hb, hemoglobin; CRP, C-reactive protein.

Statistical analysis. All statistical analyses were conducted using EZR software (Saitama Medical Center, Jichi Medical University, ver. 1.68) and *p*-values <0.05 were considered statistically significant. OS was defined as the time from the initiation of treatment for PCa to death. OS rates were calculated using the Kaplan-Meier method, and the log-rank test was conducted to analyze the significance of differences. The prognostic impact of each variable was assessed by uni- and multi-variate Cox proportional hazards regression models. The optimal cut-off values for variables to predict OS were selected using receiver operating characteristic curves.

Results

Clinicopathological characteristics are shown in Table I. All patients were diagnosed with stage IV disease at presentation. Median age was 74 years (range=60-86), and the median initial PSA level was 3,027 ng/ml (range=1,064-15,137). The Eastern Cooperative Oncology Group Performance status (ECOG PS) was ≥1 in 25 patients (71.4%), and all patients had a Gleason score ≥8. Pulmonary metastases were observed in 8 patients (22.9%), whereas no liver metastases were detected.

Table II. *First-line therapy and clinical outcomes of the 35 prostate cancer patients with initial PSA >1,000 ng/ml.*

Variables	n (%)
First-line therapy	
ADT only	3 (8.6)
Bicalutamide	14 (40.0)
Abiraterone acetate	10 (28.6)
Apalutamide	4 (11.4)
Enzalutamide	2 (5.7)
Darolutamide plus docetaxel	2 (5.7)
PSA nadir*, ng/ml	2.03 (0.001-505)
PSA nadir ≤0.2 ng/ml	9 (25.7)
PSA90	27 (77.1)
Progression to CRPC	25 (71.4)
All-cause mortality	14 (40.0)

*Data presented as median (range). ADT, Androgen deprivation therapy; PSA, prostate-specific antigen; PSA90, a 90% decline in PSA from baseline; CRPC, castration-resistant prostate cancer.

Table II summarizes the first-line therapies and clinical outcomes of the 35 patients. Three patients (8.6%) received ADT alone, while 14 (40.0%) received ADT in combination with bicalutamide. The remaining patients received ADT combined with ARSIs, including abiraterone acetate in 10 (28.6%), apalutamide in 4 (11.4%), enzalutamide in 2 (5.7%), and darolutamide plus docetaxel in 2 (5.7%) patients. During the observation period (median=31.0 months, range=2.18-138.3), PSA90 was achieved in 27 patients (77.1%), and a nadir PSA level of ≤0.2 ng/ml was observed in 9 (25.7%) patients. Twenty-five patients (71.4%) progressed to castration-resistant PCa, and 14 (40%) died from any cause during the follow-up. The Kaplan-Meier curve (Figure 1) shows that median OS was 55.0 months (95% CI=29.5-NR).

As shown in Table III, univariate analyses revealed that older age (≥75 years), poor ECOG performance status (≥1), elevated LDH, and low Hb were associated with shorter OS, while PSA90 achievement was associated with longer OS. Among these significant variables, only PSA90

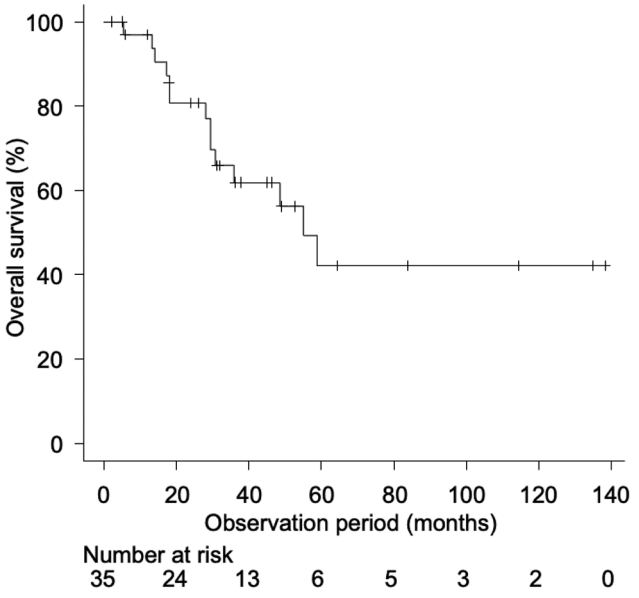

Figure 1. *Overall survival of 35 patients with prostate cancer and initial prostate-specific antigen level >1,000 ng/ml.*

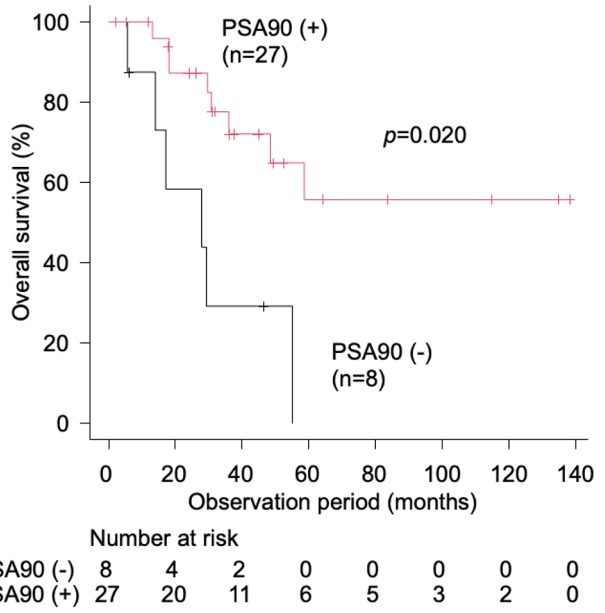

Figure 2. *Overall survival stratified by the achievement of a ≥90% prostate-specific antigen (PSA) decline from baseline (PSA90) in 35 patients with prostate cancer and initial PSA level >1,000 ng/ml.*

achievement was identified as an independent predictor of OS in the multivariate analysis. A Kaplan-Meier curve according to the presence of PSA90 achievement is shown in Figure 2. Median OS was not reached in the PSA90(+) group, while it was 28.0 months (95%CI=5.54-NR) in the PSA90(-) group (p=0.0020).

Discussion

This retrospective study focused on a rare and high-risk subgroup of patients with PCa presenting with initial PSA >1,000 ng/ml. The results obtained showed that PSA90 achievement was associated with prolonged OS and was identified as an independent prognostic factor. In contrast, patients achieving a PSA nadir ≤0.2 ng/ml did not have a significant survival benefit in this cohort.

Although PSA is widely used to monitor treatment responses and disease progression in clinical practice, few studies have examined the prognostic value of PSA kinetics specifically in patients with markedly high initial PSA levels (4). In the present study, 77.1% of patients achieved PSA90 and significantly longer OS, compared to patients who did not achieve PSA90. This result is consistent with previous studies showing the utility of a PSA decline as a surrogate marker for survival in metastatic PCa (6-9). Moreover, Nagata *et al*. demonstrated that the initial-to-nadir PSA ratio during primary combined androgen blockade was an independent predictor of both PSA response and survival in patients subsequently treated with first-line enzalutamide for metastatic castration-resistant prostate cancer (16). These findings collectively support the importance of PSA kinetics, including PSA90 and the initial-to-nadir PSA ratio, as prognostic indicators across different disease settings.

On the other hand, a PSA nadir ≤0.2 ng/ml was not associated with OS. One possible explanation is that only 25.7% of patients achieved this level of suppression, which may have limited the statistical power. Furthermore, several patients who failed to reach a nadir ≤0.2 ng/ml still had long-term survival, suggesting that PSA90 represents a more attainable and clinically meaningful endpoint for prognostic stratification in this population. In support of this result, Suzuki *et al*. recently reported that a PSA

Table III. *Cox regression analysis of overall survival in prostate cancer patients with initial PSA >1,000 ng/ml (N=35).*

	Univariate analysis		Multivariate analysis	
Variables	HR (95% CI)	*p*-Value	HR (95% CI)	*p*-Value
Age (≥75 *vs.* <75 years)	3.80 (1.18-12.3)	0.025	2.95 (0.77-11.3)	0.11
ECOG PS (≥1 *vs.*0)	6.24 (1.77-22.1)	0.005	2.61 (0.57-11.8)	0.22
Gleason score (≥9 *vs.*8)	1.94 (0.54-7.00)	0.310		
Visceral metastasis (Yes *vs.* No)	0.67 (0.18-2.40)	0.530		
ALP (≥270 vs.<270)	2.72 (0.85-8.71)	0.091		
LDH (≥226 vs.<226)	5.01 (1.39-18.1)	0.014	1.67 (0.35-7.84)	0.52
Hb (≥12.3 vs.<12.3)	1.55 (0.48-4.99)	0.460		
CRP (≥1.10 vs.<1.10)	4.05 (1.38-11.9)	0.011	3.97 (0.86-18.3)	0.078
Treatment with ARSI (Yes *vs.* No)	1.16 (0.40-3.38)	0.790		
PSA nadir ≤0.2 ng/ml (Yes *vs.* No)	0.36 (0.079-1.60)	0.180		
PSA90 (Yes *vs.* No)	0.21 (0.068-0.62)	0.005	0.13 (0.027-0.63)	0.010

HR, Hazard ratio; CI, confidence interval; ECOG PS, Eastern Cooperative Oncology Group performance status; ALP, alkaline phosphatase; LDH, lactate dehydrogenase; Hb, hemoglobin; CRP, C-reactive protein; ARSI, androgen receptor signaling inhibitor; PSA, prostate-specific antigen; PSA90, a 90% decline in PSA from baseline.

decline to ≤0.2 ng/ml by 12 weeks was associated with improved outcomes in a large real-world cohort of metastatic hormone-sensitive PCa patients but also noted that individuals with very high baseline PSA (≥200 ng/ml) were less likely to achieve this target (17). This underscores the limitation of relying solely on PSA ≤0.2 ng/ml as a universal prognostic marker in patients with extremely elevated PSA levels.

The present results complement the findings reported by Kan *et al*., who analyzed 90 Taiwanese patients with initial PSA >1,000 ng/ml and found that PSA responses were associated with survival; however, their cohort did not include patients treated with ARSIs (4). In contrast, the present study included ARSI-based treatments in more than 50% of patients, reflecting a more contemporary treatment landscape. Despite the widespread use of ARSIs, their administration did not emerge as an independent predictor of OS in our cohort. One possible explanation is the heterogeneity in ARSI use and timing, which may have diluted individual prognostic impacts. Additionally, our small sample size may have limited our ability to detect significant differences based on the treatment modality alone.

Several other clinical factors, such as older age, poor ECOG PS, elevated LDH, and elevated CRP, were associated with shorter OS in univariate analyses, which is consistent with previous findings on metastatic PCa (18, 19). However, these variables did not remain significant in multivariate analyses, suggesting that early PSA kinetics provide a more robust prognostic signal in this unique population.

Study limitations. It was a single-center, retrospective analysis with a limited sample size, which may have introduced a selection bias. Furthermore, treatment decisions were not standardized, and ARSI use was selected by physician preferences and patient characteristics, leading to potential confounders. In addition, due to the long study period (2013-2024), the evolution of treatment strategies and imaging modalities may have affected clinical outcomes.

Conclusion

Achievement of PSA90 was associated with prolonged OS and emerged as the only independent prognostic biomarker for OS in PCa patients with an initial PSA >1,000 ng/ml. These results highlight the potential of PSA90 as a simple, early, and clinically meaningful marker for prognostic stratification in this high-risk patient population.

Conflicts of Interest

The Authors declare that they have no conflicts of interest.

Authors' Contributions

Research conception and design: Ryo Sato. Data acquisition: Ryo Sato, Yukihiro Yoshimi, Tetsuharu Nishio, and Yu Matsunaga. Statistical analysis: Ryo Sato. Data analysis and interpretation: Ryo Sato, Yukihiro Yoshimi, Tetsuharu Nishio, and Yu Matsunaga. Drafting of the manuscript: Ryo Sato. Critical revision of the manuscript: Rikiya Matsumoto. Supervision: Rikiya Matsumoto.

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Artificial Intelligence (AI) Disclosure

No artificial intelligence (AI) tools, including large language models or machine learning software, were used in the preparation, analysis, or presentation of this manuscript.

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