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RESEARCH ARTICLE

ALKALINE PHOSPHATASE-TO-ALBUMIN RATIO (APAR) AS A PROGNOSTIC BIOMARKER IN METASTATIC HORMONE-SENSITIVE PROSTATE CANCER

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ABSTRACT

Purpose: Metastatic hormone-sensitive prostate cancer requires accessible biomarkers for better prognostication. This study investigated the prognostic value of the alkaline phosphatase-to-albumin ratio (APAR) in metastatic hormone-sensitive prostate cancer survival risk stratification.
Methods: We retrospectively analyzed 128 metastatic hormone-sensitive prostate cancer patients treated at a single institution between January 2015 and January 2025. A cut-off of 2.92 for the alkaline phosphatase-to-albumin ratio was determined via receiver operating characteristic analysis. Overall survival was assessed using Kaplan–Meier methods and multivariable Cox regression adjusting for standard prognostic factors, including tumor volume and Gleason score.
Results: Patients with an alkaline phosphatase-to-albumin ratio of 2.92 or higher had significantly shorter overall survival compared to those with a ratio lower than 2.92 (log-rank $p<0.001$). In multivariable analysis, an alkaline phosphatase-to-albumin ratio of 2.92 or higher remained an independent predictor of poor overall survival (Hazard Ratio 3.26, 95% Confidence Interval 1.76–5.97; $p<0.001$), alongside Eastern Cooperative Oncology Group performance status (ECOG PS).
Conclusion: The alkaline phosphatase-to-albumin ratio is an inexpensive, accessible marker that independently predicts survival in metastatic hormone-sensitive prostate cancer. APAR should be interpreted as a supportive, complementary marker alongside established clinical parameters such as ECOG PS, Gleason grade, and metastatic burden rather than as a standalone determinant. It holds promise for early risk stratification, though prospective validation is warranted

Keywords: Alkaline phosphatase-to-albumin ratio (APAR), Metastatic hormone-sensitive prostate cancer (mHSPC), Prognostic biomarker, Risk stratification

INTRODUCTION

Prostate cancer is among the most frequently diagnosed cancers in men and represents a significant cause of cancer-related mortality worldwide (1). Within the disease spectrum, metastatic hormone sensitive prostate cancer (mHSPC) marks a clinically pivotal stage, as it often sets the trajectory for subsequent treatment response

and survival (2). Although substantial therapeutic advances have been achieved with the addition of docetaxel and novel androgen receptor targeted agents to androgen deprivation therapy (ADT), patient outcomes in mHSPC remain heterogeneous (3,4). As a result, there is a growing demand for reliable, accessible, and cost effective biomarkers that can help identify patients at higher risk of poor outcomes and guide early treatment intensification strategies (5,6).

Currently used prognostic indicators in mHSPC such as Gleason score, prostate specific antigen (PSA) levels, extent of disease, and metastatic burden primarily reflect tumor characteristics. However, they often overlook host related factors such as nutritional status, systemic inflammation, and metabolic reserve, all of which are increasingly recognized as contributors to cancer progression and treatment response (7,8). In this context, serum based composite markers are gaining attention for their potential to integrate tumor biology and host physiology into a single metric.

The alkaline phosphatase to albumin ratio (APAR) is one such emerging biomarker. It combines two routinely available laboratory values: serum albumin, a marker of nutritional and inflammatory status, and alkaline phosphatase (ALP), which reflects bone turnover and metastatic activity, particularly in patients with skeletal involvement (9,10) A higher APAR may indicate a state of both high tumor burden and poor nutritional reserve, both of which are associated with adverse outcomes. While APAR has shown prognostic relevance in several solid tumors including hepatocellular carcinoma, and pancreatic ductal adenocarcinoma, its clinical utility in prostate cancer, particularly in the mHSPC setting, remains unclear (11,12).

In this study, we aimed to investigate the prognostic significance of APAR in patients with mHSPC and to evaluate whether it can serve as a simple, accessible marker to stratify patients by survival risk prior to treatment initiation.

MATERIALS AND METHODS

This retrospective study included 128 patients diagnosed with metastatic hormone-sensitive prostate cancer (mHSPC) who were treated at our institution between January 2015 and January 2025. Inclusion criteria were: histologically confirmed prostate adenocarcinoma, radiologically confirmed distant metastases at diagnosis or during follow-up prior to castration resistance, initiation of systemic therapy (ADT alone or in combination), and availability of baseline serum albumin and ALP values within 30 days before treatment start. Patients were excluded if they had prior castration-resistant prostate cancer, missing key laboratory or survival data, or a follow-up of less than one month. All patients had radiologically confirmed distant metastases and received androgen deprivation therapy (ADT) either alone or in combination with other systemic agents (e.g., docetaxel, novel hormonal therapies).

Basic clinical data (age, ECOG PS, Gleason score, PSA levels, and metastatic burden) were collected from electronic medical records. Laboratory parameters, particularly serum albumin and alkaline phosphatase (ALP) levels, were recorded prior to the initiation of systemic therapy. The alkaline phosphatase to albumin ratio (APAR) was calculated by dividing the ALP (U/L) value by the albumin (g/L) value.

Patients with missing key data or follow-up information were excluded. The primary outcome was overall survival (OS), defined as the time from initiation of systemic therapy to death from any cause or last follow-up. The study was conducted in accordance with the Declaration of Helsinki, and institutional ethics approval was obtained. Ethical approval obtained from the Dokuz Eylül University, Non-Interventional Research Ethics Committee (Date: 19.02.2025, Decision No: 2025/06-14)

Descriptive statistics were used to summarise the characteristics of the patients. Continuous variables were presented as the median (interquartile range) or the mean (± standard deviation), depending on the distribution. Categorical variables were summarised as counts and percentages. To determine the optimal cut-off value for APAR in predicting overall survival, receiver operating characteristic (ROC) curve analysis was used to select the threshold corresponding to the highest Youden index. Patients were then divided into low and high APAR groups. Additionally, ROC analysis was performed to determine the optimal cut-off value for PSA.

Survival probabilities were estimated using the Kaplan–Meier method and differences between groups were assessed using the log-rank test. Cox proportional hazards regression analysis was employed to evaluate the prognostic value of APAR in both univariate and multivariate models. Variables with a p-value of less than 0.10 in the univariate analysis were included in the multivariate model.

Collinearity was evaluated using variance inflation factors (VIF). CHAARTED and LATITUDE were highly correlated (VIF = 4.1); accordingly, CHAARTED was retained and LATITUDE was omitted from the multivariable models. We performed the same check for APAR and the metastatic pattern (M1a/M1b/M1c entered as indicator variables); VIFs were ~1.0, indicating no material collinearity, so these variables were kept in the model. Hazard ratios (HR) and 95% confidence intervals (CI) were reported. All statistical analyses were performed using SPSS version 30.0 (IBM Corp., Armonk, NY), with a p-value of less than 0.05 being considered statistically significant.

A formal a priori sample size calculation was not performed given the retrospective design. With 128 patients and an estimated 48 events, post-hoc power analysis (Schoenfeld method, two-sided α=0.05) indicated that the study had >98% power to detect the observed hazard ratio of 3.26. However, the study would have been underpowered (~66%) to detect a more modest effect size of HR=2.0, and approximately 88% power for HR=2.5. These findings should be interpreted with this limitation in mind, and prospective studies with larger sample sizes are warranted to define the minimum clinically meaningful effect.

RESULTS

A total of 128 patients were included in the study. Based on ROC curve analysis, a cut-off value of 2.92 was identified, and patients were grouped accordingly: 54 patients had values above this threshold, and 74 patients had values below. Age distribution (<70 vs ≥70 years) and ECOG PS were similar between groups (p=0.36 and p=0.12, respectively). Baseline PSA was higher in the APAR ≥2.92 group (559±1334 vs 132±208 ng/mL; p<0.001). International Society of Urological Pathology (ISUP) grade groups (p=0.73), prior primary surgery (p=0.27), and first-line treatment composition (p=0.12) did not differ meaningfully. Patients with APAR ≥2.92 more often had high-volume disease by CHAARTED (72.2% vs 45.0%; p=0.002) and high-risk disease by LATITUDE (74.1% vs 52.7%; p=0.014), and the distribution of metastatic sites also differed (M1a/M1b/M1c; p=0.006) (Table 1).

The median overall survival (OS) in our cohort was 50 months (95% CI, 44–56). The median OS was significantly shorter in patients with APAR ≥2.92: median OS 33 months (95% CI, 19–47) versus 66 months (95% CI, 36–96) in those with APAR <2.92 (p<0.001) (Figure 1).

In univariable Cox models, younger age (<70 vs ≥70; HR 2.06, 95% CI 1.20–3.50; p=0.008), ECOG PS ≥1 (vs 0; HR 3.83, 95% CI 2.22–6.62; p<0.001), higher grade (ISUP 4–5 vs 1–3; HR 3.34, 95% CI 1.32–8.44; p=0.011), high-volume disease (CHAARTED; HR 1.90, 95% CI 1.10–3.30; p=0.022), high-risk disease (LATITUDE; HR 1.84, 95% CI 1.05–3.24; p=0.033), and metastatic pattern (vs M1a: M1b HR 3.01, 95% CI 1.06–8.55; p=0.042; M1c HR 3.99, 95% CI 1.36–11.76; p=0.012) were associated with poorer OS; log-transformed PSA was not (p=0.138).

In the multivariable model, APAR remained an independent predictor of OS alongside performance status. Higher APAR (≥2.92) was associated with inferior survival (HR: 3.26, 95% CI 1.76–5.97; p<0.001), and ECOG PS ≥1 predicted worse outcomes (HR 2.63, 95% CI 1.38–5.01; p=0.003). Age, grade group, CHAARTED/LATITUDE classifications, metastatic site, and first-line treatment were not independently associated with OS after adjustment (all p>0.05).

Overall, an APAR threshold of 2.92 distinguished prognosis: Patients with APAR ≥2.92 experienced significantly shorter OS, and APAR functioned as an independent prognostic factor in mHSPC alongside ECOG PS. These findings suggest that APAR may serve as an independent prognostic marker in mHSPC patients (Table 2).

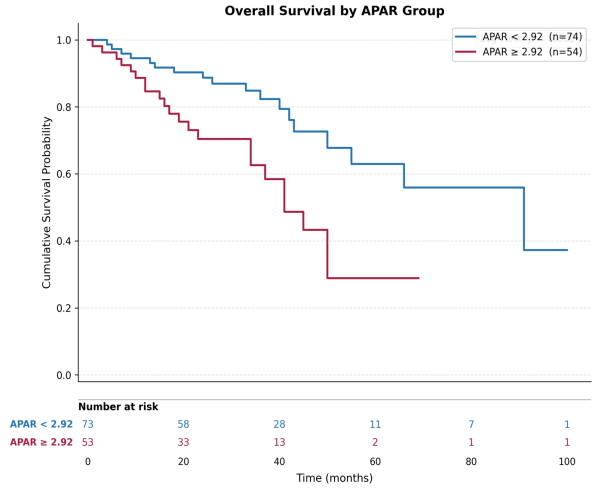


Figure 1. Overall survival according to APAR

Table 1. Patient characteristics (*p ≤0.05)

	All cohort n=128, %	≥2.92 n=54, %	<2.92 n=74, %	p-value*
Age, n (%)				
<70	63 (49.2)	24 (44.4)	39 (52.7)	0.36
≥70	65 (50.8)	30 (55.6)	35 (47.3)	
ECOG PS, n (%)				
0	84 (65.6)	30 (55.6)	54 (73)	0.12
≥1	44 (34.4)	24 (44.4)	20 (27)	
PSA (mean±SD)	404±1089	559±1334	132±208	<0.001
ISUP grade group, n (%)				
1	2 (2.6)	1 (1.9)	1 (1.4)	0.73
2	8 (6.2)	3 (5.6)	5 (6.8)	
3	15 (11.7)	4 (7.4)	11 (14.9)	
4	14 (10.9)	7 (13)	7 (9.5)	
5	89 (69.5)	39 (72.2)	50 (67.6)	
Primary surgery, n (%)				
Yes	87(68)	37 (68.5)	50(67.6)	0.27
No	41(32)	17 (31.5)	24(32.4)	
CHAARTED, n (%)				
Low volume	56 (43.8)	15 (27.8)	41 (55)	0.002
High volume	72 (56.2)	39 (72.2)	33 (45)	
LATITUDE , n (%)				
Low risk	49 (38.3)	14 (25.9)	35 (47.3)	0.014
High risk	79 (61.7)	40 (74.1)	39 (52.7)	
Metastasis site, n (%)				
M1a	20 (15.6)	2 (3.7)	18 (24.3)	0.006
M1b	72 (56.2)	34 (63)	38 (51.4)	
M1c	36 (28.1)	18 (33.3)	18 (24.3)	
First line treatment, n (%)				
ADT monotherapy	28 (21.9)	11 (20.4)	17 (23)	0.12
Docetaxel+ADT	49 (38.3)	19 (35.2)	30 (40.5)	
Enzalutamide+ADT	20 (15.6)	7 (13)	13 (17.6)	
Abiraterone+ADT	19 (14.8)	13 (24.1)	6 (8.1)	
Chemohormonal therapy (Triplet)	12 (9.4)	4 (7.4)	8 (10.8)	

DISCUSSION

This study demonstrates the prognostic utility of the alkaline phosphatase-to-albumin ratio (APAR) in patients with metastatic hormone-sensitive prostate cancer (mHSPC). Patients with higher APAR values had significantly shorter overall survival, supporting APAR as a practical and accessible tool for early survival risk stratification. Although the CHAARTED/LATITUDE criteria and TNM M1a/b/c subgroups are commonly used to assess metastatic disease volume and have shown prognostic value in previous studies, they were not independently significant in our multivariate analysis (13–15).

Power may have been limited by category imbalance, and prognostic separation can be blurred by imaging heterogeneity and stage migration. This is particularly true when Prostate-specific membrane antigen (PSMA) PET coexists with conventional staging (16). Moreover, early treatment intensification (ARPI ± docetaxel) likely narrows outcome differences between high and low burden groups. It is also plausible that biology weighted laboratory information captured by APAR, reflecting both tumor load through ALP and host status through albumin, overlaps with or mediates the signal carried by purely anatomic schemas, reducing their incremental effect once APAR and clinical covariates are included.

Finally, some degree of overadjustment and residual confounding cannot be excluded.

Table 2. Univariate and multivariate analysis for overall survival (*p ≤0.05)

	Univariate analysis HR (95%CI)	**p- value***	**Multivariate analysis HR (95%CI)**	**p- value***
Age (<70 vs ≥70)	2.06 (1.2-3.5)	0.008	1.06 (0.56-2.07)	0.86
ECOG PS (0 vs ≥1)	3.83 (2.22-6.62)	<0.001	2.63 (1.38-5.01)	0.003
PSA (logPSA)	1.13 (0.96-1.32)	0.138		
Grade groups (1-3 vs 4-5)	3.34 (1.32-8.44)	0.011	2.16 (0.8-5.82)	0.13
CHAARTED (Low vs high)	1.9 (1.1-3.3)	0.022	0.99 (0.51-1.92)	0.98
LATITUDE (Low vs high risk)	1.84 (1.05-3.24)	0.033		
Metastasis site		0.042		0.48
M1a	Reference	-	-	-
M1b vs M1a	3.01 (1.06-8.55)	0.038	1.1 (0.33-3.59)	0.88
M1c vs M1a	3.99 (1.36-11.76)	0.012	1.62 (0.48-5.5)	0.44
First-line treatment		0.32		0.58
ADT monotherapy	Reference	-	-	-
Docetaxel+ADT	0.61 (0.33-1.12)	0.11	0.65 (0.34-1.25)	0.2
ARPIs+ADT	0.97 (0.48-1.95)	0.93	0.97 (0.47-2.0)	0.94
Chemohormonal therapy (Triplet)	1.43 (0.33-6.28)	0.64	1.1 (0.24-5.06)	0.91
APAR (<2.92 vs ≥2.92)	3.75 (2.16-6.51)	<0.001	3.26 (1.76-5.97)	<0.001

Taken together, these points suggest that in today's mHSPC practice, biology informed metrics such as APAR may complement and in some settings outperform traditional burden based classifications, a hypothesis that merits prospective validation and sensitivity analyses across imaging modalities and treatment eras.

The prognostic value of APAR likely stems from its ability to reflect both tumor burden and host physiological reserve simultaneously. Albumin serves as a marker of nutritional and inflammatory status, and low serum albumin levels have been associated with worse outcomes across multiple cancer types. Tang et al. found that survival was shorter as albumin levels decreased in many cancers, including prostate cancer (17). Similarly, low albumin levels in the EPIC cohort were found to be associated with mortality in prostate cancer (18). Alkaline phosphatase is often elevated when cancer spreads to the bones. Li et al. demonstrated a correlation between high ALP levels and low OS in prostate cancer in their meta-analysis. Ertürk et al. found a correlation between high ALP values and metastasis rates. (19). Taken together, the combination of ALP and albumin in a single ratio integrates tumor activity and host status into a composite metric with meaningful prognostic signal.

In our study, patients with high APAR values were more likely to have aggressive disease features such as higher PSA levels, more poorly differentiated tumors, and greater metastatic burden. Interestingly, even when we took these factors into account, APAR still remained a strong and independent predictor of overall survival. Wang et al. determined that a threshold value of 2.167 could serve as a potential target for reducing cancer-related deaths (20). Similarly, in our study, we determined the APAR threshold to be 2.92. Prospective studies involving more patients are needed to determine the optimal threshold.

Clinically, APAR has a number of advantages. It is low-cost, easy to calculate, and already included in routine blood tests. These qualities make it a practical and accessible tool that could be used in everyday oncology practice. Despite its ease of use, the only study in prostate cancer patients investigated its relationship with recurrence in patients who underwent radical prostatectomy and found it to be significant (21). As treatment options for mHSPC

continue to expand and become more personalized, having a straightforward method like APAR to help guide early treatment decisions could be very valuable. In practice, APAR may support risk-adapted management: patients with elevated APAR could be considered for early treatment intensification (where clinically appropriate) and scheduled for closer surveillance with more frequent clinical evaluations and laboratory/imaging assessments, whereas those with lower APAR might follow standard monitoring intervals. These applications warrant prospective validation.

The absence of a significant PSA effect in the multivariable model warrants comment. Log-transformed PSA did not reach the pre-specified entry threshold in univariable analysis (p=0.138) and was therefore excluded from the multivariable model. This likely reflects the known heterogeneity of PSA as a prognostic marker in the mHSPC setting, where PSA kinetics and nadir rather than absolute baseline values may be more informative (22,23). Future prospective studies with larger, diverse cohorts should assess whether baseline PSA adds incremental value beyond APAR and ECOG PS, and whether combining APAR with other inflammatory or nutritional indices, such as NLR or PNI, offers additive prognostic information. Importantly, APAR should be regarded as a complementary tool that augments, rather than replaces, established risk stratification parameters including ECOG PS, Gleason grade, and metastatic burden.

Limitations

This was a single center and retrospective study with a modest sample. From 2015 to 2025, standards of care and imaging evolved, especially wider ARPI use and the adoption of PSMA PET, which may cause period effects and stage migration. Because treatment intensification was not randomized, confounding by indication remains possible. APAR was computed from total ALP and albumin; bone specific ALP was unavailable and albumin can vary with acute phase and nutritional status. The 2.92 cutoff was derived internally, so optimism and information loss from dichotomization are possible; validation on the continuous scale is warranted. Multiple imputation addressed missing data, yet the mechanism of missingness may still influence estimates. There was no blinded central imaging review, and Response Evaluation Criteria in Solid Tumors (RECIST) 1.1 has limitations for bone predominant disease. Cox model assumptions and potential collinearity between CHAARTED and LATITUDE also apply, and external generalizability is limited.

CONCLUSION

Our findings suggest that APAR deserves further attention as a prognostic tool. It reflects both the biology of the tumor and the host environment, and its simplicity makes it especially appealing. Future prospective studies could help clarify how best to use APAR in clinical decision-making, but even now it offers a promising way to improve how we assess and manage patients with metastatic prostate cancer.

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Conflict of interest: None.
Ethical approval: The study was conducted after obtaining approval from the Dokuz Eylul University, Non-Interventional Research Ethics Committee (Date: 19.02.2025, Decision No: 2025/06-14)
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