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Radiographic progression at castration-resistant transition in metastatic castration-sensitive prostate cancer: prognostic impact and clinical features

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Abstract

Background

Radiographic progression (rPD) at castration-resistant prostate cancer (CRPC) diagnosis remains poorly characterized in real-world settings. We assessed its prognostic impact and the clinical features of patients with rPD in real-world de novo metastatic castration-sensitive prostate cancer (mCSPC).

Methods

We retrospectively analyzed 330 patients with de novo mCSPC who progressed to CRPC from two multi-institutional cohorts: a conventional androgen deprivation therapy or combined androgen blockade (ADT/CAB) cohort (*n* = 272) and an upfront intensification cohort (*n* = 58). At CRPC diagnosis, patients were categorized as having prostate-specific antigen (PSA)-only progression or PSA + rPD. Post-CRPC overall survival (OS) was evaluated using Kaplan–Meier and Cox proportional hazards models, while rPD-associated characteristics were examined using logistic regression.

Results

Across both cohorts, patients with PSA + rPD had significantly shorter post-CRPC OS than those with PSA-only progression (ADT/CAB: 21 vs. 44 months, $P < 0.001$; upfront: 14 vs. 24 months, $P = 0.008$). Univariate Cox regression confirmed the adverse prognostic impact of rPD (hazard ratios 2.25 [95% CI 1.53–3.29] and 2.49 [95% CI 1.22–5.07]). Lower baseline PSA was significantly associated with rPD in the upfront cohort (odds ratio [OR] 0.30, 95% CI 0.12–0.75, $P = 0.010$), with a non-significant trend in the ADT/CAB cohort (OR 0.71, 95% CI 0.47–1.06, $P = 0.092$).

Conclusion

Radiographic progression at CRPC transition predicts poor post-CRPC survival in real-world mCSPC patients, regardless of initial treatment. Lower baseline PSA may identify patients at increased risk of rPD, supporting careful radiographic monitoring at CRPC transition.

Introduction

The therapeutic landscape for metastatic castration-sensitive prostate cancer (mCSPC) has undergone substantial evolution over the past decade. The conventional strategy of androgen deprivation therapy (ADT) monotherapy or combined androgen blockade (CAB) has been largely supplanted by upfront intensification approaches, including androgen receptor signaling inhibitors (ARSIs) and docetaxel [1]. Although these strategies have improved overall survival (OS), a subset of patients continues to exhibit early progression and poor outcomes.

Prostate-specific antigen (PSA) remains a sensitive biomarker for monitoring disease activity and serves as the primary basis for clinical decision-making. Concurrently, the clinical relevance of radiographic progression (rPD) has increased. Recent prospective trials have adopted radiographic progression-free survival as a surrogate endpoint for OS [2], and current definitions of CRPC progression incorporate both PSA elevation and radiographic evidence of disease worsening [3]. However, in real-world practice, implementing standardized and frequent radiographic assessments comparable to those in clinical trials remains challenging [4]. Consequently, evidence regarding the prognostic impact of rPD at CRPC diagnosis in real-world settings is limited, particularly in the era of upfront intensification therapies [5].

This study aimed to evaluate the prognostic significance of rPD at CRPC diagnosis in patients with de novo mCSPC. Two independent cohorts—those treated with conventional ADT/CAB and those receiving upfront therapy—were analyzed to determine whether progression patterns consistently influence post-CRPC survival across treatment eras.

Patients and Methods

Ethical considerations

This study adhered to the Declaration of Helsinki and received approval from the Institutional Review Board of Kyoto University Hospital and participating institutions (Approval No. R1720-4). Informed consent was waived due to the retrospective design; instead, an opt-out approach allowed patients to withdraw their data.

Study design and patients

This multi-institutional retrospective study included two independent cohorts of patients with de novo mCSPC.

1. ADT/CAB cohort: patients diagnosed between 2014 and 2021 at Kyoto University Hospital and 15 affiliated institutions who initiated ADT monotherapy or CAB with bicalutamide [6].
2. Upfront cohort: patients diagnosed between 2016 and 2024 at Kyoto University Hospital and two affiliated institutions who initiated ADT combined with upfront therapy using ARSIs and/or docetaxel.

Eligible patients were those who subsequently progressed to CRPC and underwent radiographic evaluation at CRPC diagnosis.

Definition of CRPC and classification based on progression patterns

CRPC was defined by the treating physician as follows: (1) serum testosterone levels <50 ng/dL with a PSA increase ≥25% and ≥2 ng/mL above the nadir, confirmed by a second value ≥3 weeks later, or (2) radiographic evidence of disease progression, defined as worsening of existing lesions or emergence of new lesions [4]. Radiographic evaluation relied on conventional imaging, including computed tomography and/or bone scintigraphy, performed in routine clinical practice near the time of CRPC diagnosis. Treatment selection after CRPC progression was determined by the treating physician.

Patients meeting PSA criteria for CRPC were classified into two groups according to the presence of concurrent rPD: the PSA-only group (no rPD at CRPC diagnosis) and the PSA + rPD group (concurrent rPD at CRPC diagnosis). Patients with rPD without PSA elevation were excluded because of their distinct clinical characteristics and small sample size. Imaging was not routinely conducted at fixed intervals independent of PSA progression; therefore, rPD-only cases were considered highly susceptible to ascertainment bias inherent to retrospective analyses. Treatment selection after CRPC progression remained at physician discretion.

Study outcomes

The primary objective was to assess the prognostic impact of rPD at CRPC diagnosis in both cohorts. The primary endpoint was OS after CRPC diagnosis, defined as the interval from CRPC diagnosis to death from any cause. Post-CRPC OS was compared between the PSA-only and PSA + rPD groups. Forest plots of hazard ratios were generated to further evaluate how rPD affects post-CRPC OS across the main and prespecified subgroups in each cohort. Moreover, OS-associated prognostic factors were identified. A secondary analysis evaluated clinical factors associated with the presence of rPD at CRPC progression.

Statistical analysis

Categorical variables were compared using the chi-square or Fisher exact test, whereas continuous variables were analyzed using the Mann–Whitney U test. Survival curves were estimated using the Kaplan–Meier method and compared with the log-rank test. Forest plots were generated to visualize hazard ratios (HRs) and 95% confidence intervals (CIs) derived from univariate Cox proportional hazards regression, applied both to the overall population and prespecified subgroups in each cohort. Univariable and multivariable Cox proportional hazards models were used to identify independent prognostic factors for OS after CRPC. In the upfront cohort, only variables with statistical significance in univariable analysis (P <0.05) were included in the multivariable model due to the limited number of events (n = 32). Logistic regression analysis identified factors associated with rPD at CRPC progression; the same variable selection strategy was applied in the upfront cohort because of the limited number of rPD events (n = 22). All analyses were conducted using R software (version 4.4.1; R Foundation for Statistical Computing, Vienna, Austria) [7]. A two-sided P-value <0.05 was considered statistically significant.

Results

Patient selection and group classification

The study flow and patient selection are summarized in the CONSORT diagram (Figure 1). In the ADT/CAB cohort, 521 patients with de novo mCSPC initiated ADT as monotherapy or CAB with bicalutamide. Among them, 347 progressed to CRPC with a median follow-up of 12 months (IQR: 6–19). After excluding 64 patients without radiographic evaluation at CRPC diagnosis, 283 were classified according to PSA elevation and/or rPD. Ultimately, 272 patients were included: 192 in the PSA-only group and 80 in the PSA + rPD group. Eleven patients with rPD without PSA elevation were excluded.

In the upfront cohort, 230 patients with de novo mCSPC received ADT plus upfront ARSI and/or docetaxel. Of these, 74 progressed to CRPC with a median follow-up of 11 months (IQR: 6–19). After excluding 11 patients without radiographic assessment at CRPC diagnosis, 63 patients were stratified. A total of 58 patients were analyzed: 36 in the PSA-only group and 22 in the PSA + rPD group, while five patients with rPD without PSA elevation were excluded.

Patient characteristics in the ADT/CAB and upfront cohorts

Baseline and clinical characteristics are summarized in **Table 1**. In the ADT/CAB cohort, no significant differences were observed between groups. However, the PSA + rPD group showed trends toward lower baseline PSA levels (median: 216 vs. 346 ng/mL), a lower proportion of high-volume disease (50% vs. 62%), and shorter time to CRPC (median: 9 vs. 11 months).

In the upfront cohort, treatment included ARSI plus ADT in 47 patients (81%), docetaxel plus ADT in nine (16%), and triplet therapy with ARSI and docetaxel in two (3%). Compared with the PSA-only group, the PSA + rPD group had lower baseline PSA levels (median: 137 vs. 653 ng/mL, P = 0.001) and older age at mCSPC diagnosis (median: 75 vs. 71 years, P = 0.046).

PSA nadir responses differed between cohorts. In the ADT/CAB cohort, a PSA nadir <0.2 ng/mL was less frequent in the PSA + rPD group (18% vs. 28%). In contrast, in the upfront cohort, the PSA + rPD group had a higher proportion of deep responders (46% vs. 19%, P = 0.043).

Treatment modalities after progression to CRPC

Subsequent treatments after CRPC progression are shown in **Supplementary Figure S1**. In the ADT/CAB cohort, first-line CRPC treatments included ARSIs in 53% (145/272) and taxanes in 11% (31/272), with taxanes more frequently used in the PSA + rPD group than in the PSA-only group (20% [16/80] vs. 8% [15/192]). In the upfront cohort, ARSIs were administered in 24% (14/58) and taxanes in 57% (33/58) of patients.

Impact of radiographic progression on post-CRPC OS in both cohorts

The median follow-up from CRPC diagnosis was 21 months (IQR: 9–32) in the ADT/CAB cohort and 14 months (IQR: 11–23) in the upfront cohort. Kaplan–Meier analysis demonstrated significantly shorter post-CRPC OS in the PSA + rPD group than in the PSA-only group in both cohorts (**Figure 2**). In the ADT/CAB cohort, median OS was 21 months in the PSA + rPD group versus 44 months in the PSA-only group (P <0.001). Similarly, in the upfront cohort, the PSA + rPD group had a significantly shorter median post-CRPC OS (14 vs. 24 months; P = 0.008). Univariate Cox regression confirmed the adverse prognostic impact of rPD at CRPC, with HRs of 2.25 (95% CI: 1.53–3.29) and 2.49 (95% CI: 1.22–5.07) in the ADT/CAB and upfront cohorts, respectively. Subgroup analyses showed a consistent trend toward higher HRs in the PSA + rPD group across all prespecified subgroups, including performance status, baseline PSA, Gleason grade group, bone and visceral metastases, PSA nadir <0.2 ng/mL, and time to CRPC (**Figure 3**).

Supplementary analysis of prognostic factors for post-CRPC OS

Multivariable Cox regression identified radiographic progression at CRPC and shorter time to CRPC (<12 months) as independent predictors of worse post-CRPC OS in both cohorts (**Supplementary Table S1**). For rPD, HRs were 1.86 (95% CI: 1.22–2.83, P = 0.004) in the ADT/CAB cohort and 2.64 (95% CI: 1.16–5.50, P = 0.010) in the upfront cohort. Similarly, a time to CRPC < 12 months was significantly associated with poorer survival, with HRs of 1.70 (95% CI: 1.06–2.74, P = 0.028) and 2.31 (95% CI: 1.08–4.97, P = 0.032) in the ADT/CAB and upfront cohorts, respectively. Furthermore, visceral metastasis and low performance status at the baseline were significantly associated with poorer survival in the ADT/CAB and upfront cohorts (HR 1.65 and 3.79; 95 % CI: 1.05–2.59 and 1.46–9.83; P = 0.030 and 0.006), respectively.

Clinical characteristics associated with radiographic progression at CRPC conversion

Logistic regression analysis evaluated clinical factors associated with rPD at CRPC with PSA progression (**Table 2**). In the ADT/CAB cohort, lower baseline PSA was associated with rPD in univariable analysis (OR: 0.71, 95% CI: 0.51–0.998, P = 0.049) but not in multivariable analysis (OR: 0.71, 95% CI: 0.47–1.06, P = 0.092). In the upfront cohort, lower baseline PSA was significantly associated with rPD in both univariable (OR: 0.26, 95% CI: 0.11–0.63, P = 0.003) and multivariable analyses (OR: 0.30, 95% CI: 0.12–0.75, P = 0.010).

Discussion

This analysis identified rPD at CRPC diagnosis as a strong predictor of poor post-CRPC survival in real-world patients with mCSPC, regardless of treatment with conventional ADT/CAB or upfront intensification. Moreover, we observed that a lower baseline PSA level at mCSPC diagnosis tended to be associated with rPD at progression, a trend more evident in the upfront cohort.

The association between rPD at CRPC transition and shorter survival was consistent across cohorts, aligning with prospective trials demonstrating that radiographic progression correlates with adverse outcomes [2]. These findings confirm the relevance of this relationship in real-world settings and emphasize the importance of radiographic assessment at CRPC transition. Furthermore, subgroup analyses yielded a consistent trend toward an adverse prognostic impact of rPD across all prespecified subgroups, thereby reinforcing the robustness of this association across diverse patient profiles. Treatment strategies have shifted beyond reliance on PSA kinetics, with radiographic progression increasingly influencing therapeutic decisions [8]. Patients with PSA-only progression exhibited more favorable post-CRPC survival than those with concurrent rPD, whereas the poorer outcomes in the PSA + rPD group suggest that rPD identifies a high-risk subgroup requiring earlier and more intensive intervention. Despite taxane-based therapy being administered to more than half of patients with rPD in both cohorts after CRPC progression, survival outcomes remained poor. This suggests that rPD at CRPC diagnosis reflects biologically aggressive disease insufficiently addressed by current therapies. Beyond rPD, a shorter time to CRPC (< 12 months) was also identified as an independent predictor of poor post-CRPC survival in both cohorts, consistent with real-world retrospective analyses reporting that shorter time to CRPC is consistently associated with adverse survival outcomes [9]. However, the majority of these studies were conducted in the era of conventional ADT or CAB, and data in the context of upfront treatment intensification remain limited. Our findings suggest that the prognostic value of PSA-defined early CRPC transition persists even in the contemporary treatment landscape. For this high-risk population, proactive strategies may be warranted. Early comprehensive genomic profiling may identify actionable alterations [10], and PSMA-targeted therapies represent a promising option [11]. Although the optimal timing for these approaches remains unclear, these findings support earlier intervention in patients with rPD at CRPC conversion.

Lower baseline PSA at mCSPC diagnosis was independently associated with increased risk of rPD at CRPC progression. Tumors with low PSA production may harbor androgen-indifferent or poorly differentiated clones early in the disease course [12]. In aggressive variant prostate cancer, low PSA levels may occur despite substantial tumor burden, indicating that PSA may not adequately reflect disease activity in certain biologically aggressive subtypes [13]. Reduced PSA secretion may therefore indicate androgen-independent cell populations less responsive to hormonal therapy and more prone to radiographic progression. This interpretation is supported by the higher prevalence of grade group 5 tumors in the rPD group within the ADT/CAB cohort. High-grade disease has been associated with reduced dependence on androgen receptor signaling and increased lineage plasticity [12]. However, low baseline PSA does not uniformly indicate aggressive biology; it may also reflect low tumor burden and indolent disease [14]. Because this analysis included only patients who developed CRPC, slower-progressing cases were likely underrepresented. Accordingly, the association between low baseline PSA and rPD should be interpreted in the context of CRPC transition, and such patients warrant careful radiographic surveillance irrespective of PSA trajectory.

A discrepancy between cohorts was observed regarding PSA nadir. In the ADT/CAB cohort, a PSA nadir <0.2 ng/mL was less frequent in the rPD group, whereas in the upfront cohort, deep PSA responses were more common among patients who later developed rPD. This contrast indicates that the relationship between PSA suppression and radiographic progression varies by treatment context. Similar findings have been reported in post hoc analyses of prospective mCSPC trials, where discordant radiographic progression without PSA elevation occurred more frequently with ARSI-based regimens [15].

Although PSA generally correlates with disease activity and often precedes radiographic progression [3], some patients develop imaging progression despite sustained PSA suppression. This discordance may reflect tumor evolution toward androgen-indifferent phenotypes, including treatment-emergent neuroendocrine [16] or double-negative subtypes [17], although these were not directly evaluated. Consistent with prior real-world reports, rPD-only cases were uncommon, likely due to the absence of standardized imaging schedules in routine practice [3]. Although a PSA nadir <0.2 ng/mL is considered a favorable prognostic marker [18], these findings indicate that deep PSA suppression does not exclude early radiographic progression, particularly with upfront intensification, thereby supporting the need for regular imaging surveillance.

This study has several limitations. First, the retrospective design introduces potential selection bias and unmeasured confounding, and treatment and follow-up strategies were not standardized across institutions. Patients with rPD-only progression at CRPC diagnosis were excluded, which may have affected representativeness; the clinical impact of purely radiographic progression requires further study. Second, imaging was performed according to routine clinical practice rather than a predefined protocol. A substantial proportion of patients were excluded due to absence of imaging at CRPC diagnosis (ADT/CAB: 64/347 [18%]; upfront: 11/74 [15%]), which may have introduced bias. Furthermore, even among those imaged, the decision to perform radiographic evaluation was likely influenced by PSA dynamics, potentially biasing rPD detection. Third, detailed characterization of radiographic progression patterns was not feasible; specifically, differentiation between new lesions and progression of existing lesions was not possible, although these may have distinct biological and prognostic implications [15]. PSA doubling time at CRPC diagnosis was also unavailable, precluding its evaluation as a prognostic factor [19]. Finally, post-CRPC treatment selection was at physician discretion, resulting in heterogeneous management that may have influenced survival outcomes.

In conclusion, rPD at CRPC transition is a consistent predictor of poor survival regardless of initial treatment strategy in real-world settings. Lower baseline PSA may identify patients at increased risk of radiographic progression. However, findings from the upfront cohort remain exploratory due to the limited sample size and require validation in larger prospective studies. Vigilant imaging surveillance at CRPC conversion is warranted, including in patients achieving deep PSA responses.

Declarations

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Conflict of interests

The authors have no conflicts of interest to disclose.

Data availability statement

The datasets are available from the corresponding author on reasonable request.

Ethical approval

This study adhered to the Declaration of Helsinki and received approval from the Institutional Review Board of Kyoto University Hospital and participating institutions (Approval No. R1720-4).

Informed consent

Informed consent was waived due to the retrospective design; instead, an opt-out approach allowed patients to withdraw their data.

Author contributions

T.F., T.S., T.G and T.K. contributed to the conception and design of this study. T.F., T.S., T.K., T.T., H.F., M.K., H.K., N.S., K.Y., Y.N., R.K, Y.M., N.M., T.M., Y.M., Y.K., K.M. and S.A. contributed to the acquisition of data. T.F. contributed to the drafting of the manuscript and the analysis and interpretation of data. T.S., K.M. and T.G. contributed to the revision of the manuscript. All authors contributed to the final approval of the manuscript.

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Tables

Table 1. Clinical characteristics of the ADT/CAB and upfront cohorts, comparing the PSA-only and PSA + rPD groups

	ADT/CAB cohort				Upfront cohort		
	Total	PSA-only	PSA + rPD	*P*-value	Total	PSA-only	PSA + rP
	n = 272	*n* = 192	*n* = 80		*n* = 58	*n* = 36	*n* = 22
Age, years	74 (68–79)	74 (69–79)	74 (68–80)	0.66	71 (66–75)	71 (65–73)	75 (68–7
ECOG-PS ≥ 2 at baseline	29 (11)	20 (11)	9 (11)	0.83	7 (12)	2 (6)	5 (23)
Missing	1 (0.4)	1 (0.5)	0 (0)		0 (0)	0 (0)	0 (0)
Baseline PSA, ng/mL	284 (90–941)	346 (98–992)	216 (75–695)	0.062	416 (145–1567)	653 (363–2229)	137 (40–
ISUP grade group 5	150 (58)	99 (54)	51 (66)	0.10	49 (85)	33 (92)	16 (73)
Missing	11 (4)	9 (5)	2 (3)		1 (2)	0 (0)	1 (5)
EOD score ≥ 2	167 (61)	122 (64)	45 (56)	0.28	52 (90)	34 (94)	18 (82)
Visceral metastasis	59 (22)	39 (20)	20 (25)	0.42	9 (16)	5 (14)	4 (18)
CHAARTED volume criteria				0.079			
High-volume	159 (58)	119 (62)	40 (50)		54 (93)	33 (92)	21 (96)
Low-volume	113 (42)	73 (38)	40 (50)		4 (7)	3 (8)	1 (5)
LATITUDE risk criteria				1.00			
High-risk	195 (72)	137 (71)	58 (73)		55 (95)	35 (97)	20 (91)
Low-risk	71 (26)	50 (26)	21 (26)		2 (3)	1 (3)	1 (5)
Missing	6 (2)	5 (3)	1 (1)		1 (2)	0 (0)	1 (5)
Upfront therapy regimens				-			
ARSI doublet	-	-	-		47 (81)	29 (81)	18 (82)
Docetaxel doublet	-	-	-		9 (16)	6 (17)	3 (14)
Triplet therapy	-	-	-		2 (3)	1 (3)	1 (5)
PSA nadir < 0.2 ng/mL before CRPC	67 (25)	53 (28)	14 (18)	0.090	17 (29)	7 (19)	10 (46)
Time to CRPC, months	11 (10–12)	11 (10–13)	9 (7–10)	0.077	11 (10–13)	11 (10–18)	10 (5–18

*Data are presented as *n* (%) or median (interquatile range)

Abbreviations: ADT/CAB, androgen deprivation therapy/combined androgen blockade; ARSI, androgen receptor signaling inhibitor; CRPC, castration-resistan cancer; ECOG-PS, eastern cooperative oncology group-performance status; EOD, extent of disease; ISUP, international society of urological pathology; mCSP hormone sensitive prostate cancer; PSA, prostate-specific antigen; rPD, radiographical progression disease

Table 2. Logistic regression analyses of factors associated with radiographic progression at CRPC diagnosis in the ADT/CAB and upfront cohorts

	ADT/CAB cohort				Upfront cohort		
	Univariate analysis		Multivariable analysis		Univariate analysis		Multivariable ana
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)
Age (per year)	0.99 (0.96–1.03)	0.53	0.99 (0.96–1.03)	0.65	1.06 (0.99–1.13)	0.11	
ECOG-PS ≥ 2 at baseline	1.08 (0.47–2.50)	0.85	1.18 (0.48–2.92)	0.72	5.00 (0.88–28.5)	0.070	
Log_{10} baseline PSA (ng/mL)	0.71 (0.51–0.998)	0.049	0.71 (0.47–1.06)	0.092	0.26 (0.11–0.63)	0.003	0.30 (0.12–0.75)
ISUP grade group 5	1.60 (0.93–2.78)	0.093	1.77 (0.99–3.17)	0.054	0.29 (0.06–1.37)	0.12	
EOD score ≥ 2 at baseline	0.74 (0.43–1.25)	0.26	0.70 (0.38–1.29)	0.25	0.27 (0.04–1.59)	0.15	
Visceral metastatis at baseline	1.31 (0.71–2.42)	0.39	1.54 (0.80–2.99)	0.20	1.38 (0.33–5.80)	0.66	
PSA nadir ≥ 0.2 ng/mL before CRPC	1.80 (0.93–3.47)	0.081	1.62 (0.75–3.50)	0.22	0.29 (0.09–0.94)	0.039	0.50 (0.14–1.84)
Time to CRPC < 12 months	1.78 (1.04–3.06)	0.036	1.56 (0.83–2.94)	0.17	1.29 (0.44–3.78)	0.64	

Note: The multivariable model included all variables from univariable analysis. In the upfront cohort, only variables with *P* <0.05 in univariable analysis were i due to the limited number of rPD events (ADT/CAB cohort: *n* = 80; upfront cohort: *n* = 22).

Abbreviations: ADT/CAB, androgen deprivation therapy/combined androgen blockade; CI, confidence interval; CRPC, castration-resistant prostate cancer; EC eastern cooperative oncology group-performance status; EOD, extent of disease; ISUP, international society of urological pathology; OR, odds ratio; PSA, pros specific antigen

Figures

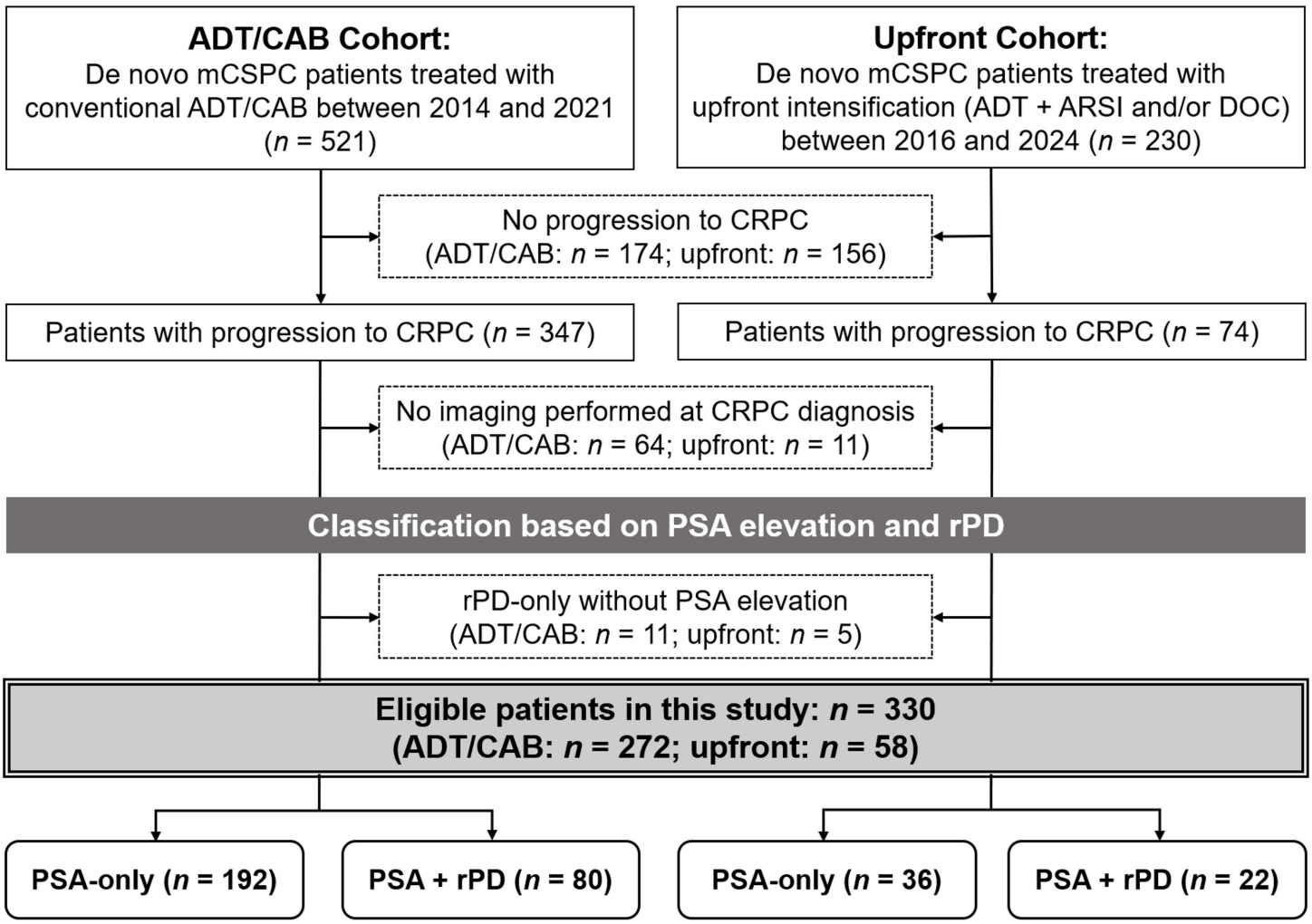


Figure 1

CONSORT diagram of the study. Abbreviations: ADT, androgen deprivation therapy; ARSI, androgen receptor signaling inhibitor; CAB, combined androgen blockade; CRPC, castration-resistant prostate cancer; DOC, docetaxel; mCSPC, metastatic castration-sensitive prostate cancer; PSA, prostate-specific antigen; rPD, radiographic progression disease.

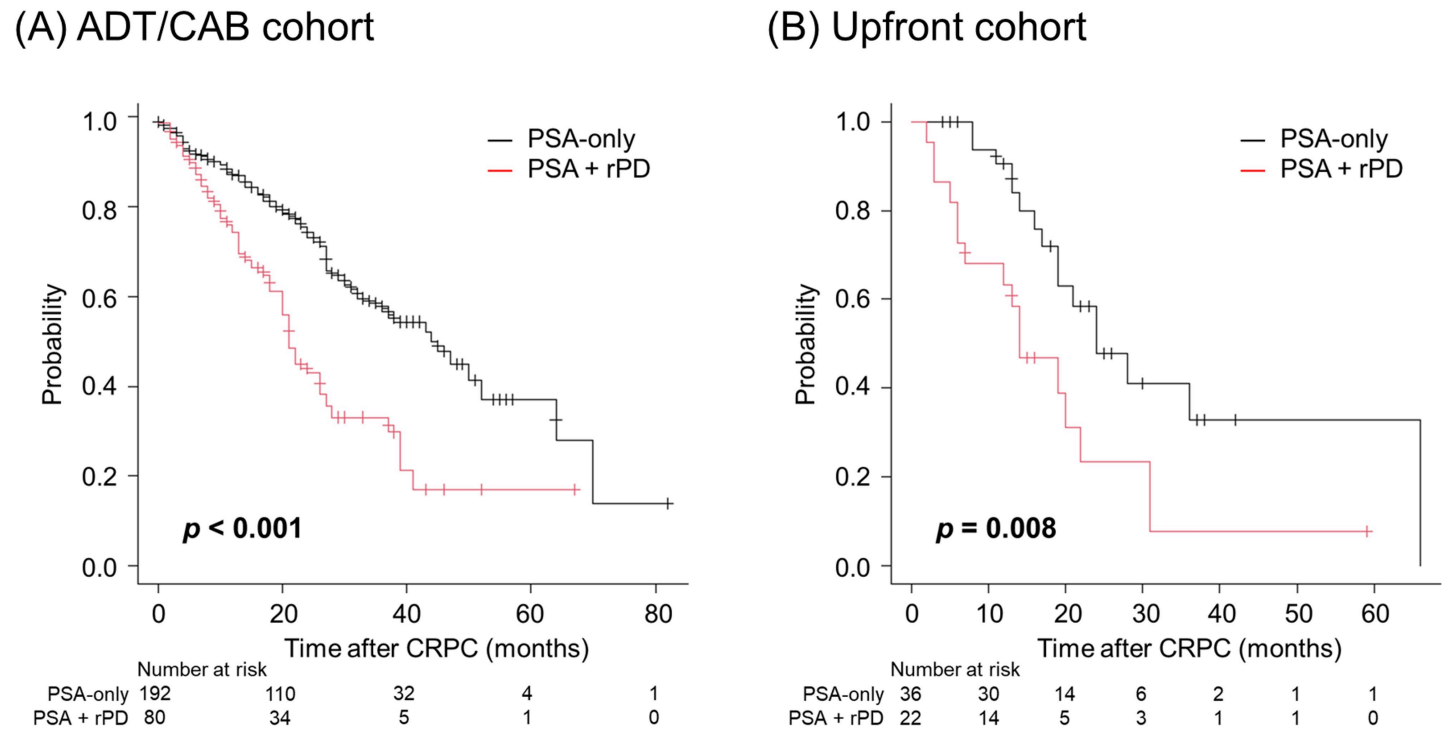


Figure 2

Kaplan–Meier analysis of post-CRPC overall survival in the PSA-only and PSA + rPD groups in the (A) ADT/CAB and (B) upfront cohorts. Abbreviations: ADT/CAB, androgen deprivation therapy/combined androgen blockade; CRPC, castration-resistant prostate cancer; PSA, prostate-specific antigen; rPD, radiographic progression disease.

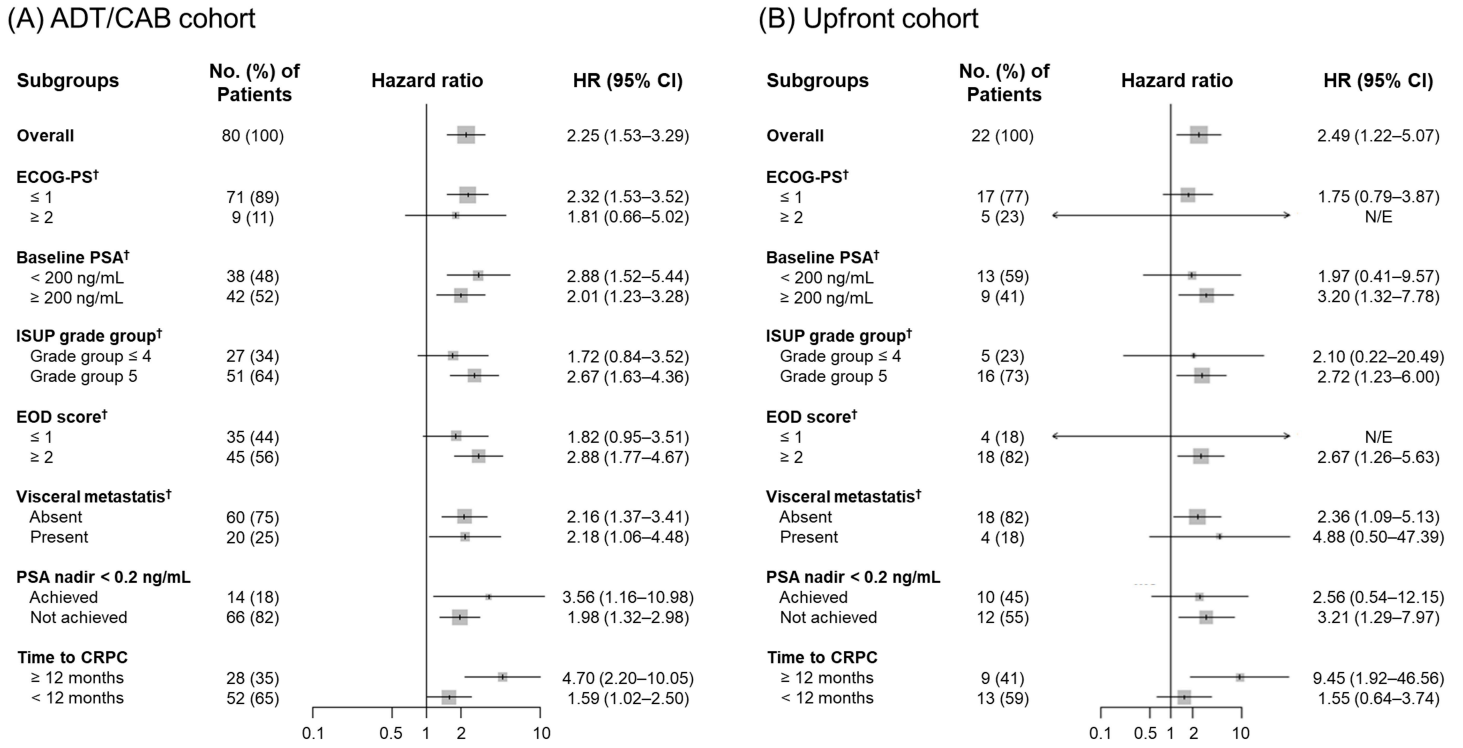


Figure 3

Forest plots of hazard ratios to assess the impact of rPD at CRPC on post-CRPC overall survival, calculated using univariate Cox proportional hazards regression in (A) ADT/CAB and (B) upfront cohorts for the main and subgroups analyses. Abbreviations: ADT/CAB, androgen deprivation therapy/combined androgen blockade; CRPC, castration-resistant prostate cancer; ECOG-PS, eastern cooperative oncology group-performance status; EOD, extent of disease; ISUP, international society of urological pathology; N/E, not estimable; PSA, prostate-specific antigen; rPD, radiographic progression disease. †Characteristics at the metastatic castration-sensitive prostate cancer diagnosis baseline.

Supplementary Files

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- supplementaryfigureS1.jpg
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