Prognostic Role of ^{68}Ga-PSMA11 PET–Based Response in Patients with Prostate Cancer Undergoing Taxane-Based Chemotherapy

Lukas Lunger*[,1], Maythinee Chantadisai*[,2,3], Amir Karimzadeh[2], Isabel Rauscher[2], Calogero D'Alessandria[2], Benedikt Feuerecker[2], Thomas Langbein[2], Robert Tauber[1], Stefan Schiele[1], Wolfgang Weber[2], and Matthias Eiber[2]

[1]*Department of Urology, Rechts der Isar Medical Center, Technical University of Munich, Munich, Germany;* [2]*Department of Nuclear Medicine, Rechts der Isar Medical Center, Technical University of Munich, Munich, Germany; and* [3]*Division of Nuclear Medicine, Department of Radiology, Faculty of Medicine, Chulalongkorn University, King Chulalongkorn Memorial Hospital, The Thai Red Cross Society, Bangkok, Thailand*

This study was performed to assess the prognostic utility of conventional biochemical and imaging response criteria and ^{68}Ga-PSMA11 PET–adapted or –specific systems regarding overall survival (OS) in men with metastatic hormone-sensitive and castration-resistant prostate cancer (PC) treated with taxane-based chemotherapy. **Methods:** A total of 103 patients (metastatic hormone-sensitive PC, $n = 57$; castration-resistant PC, $n = 46$) underwent taxane-based chemotherapy. All patients had a minimum of 2 prostate-specific membrane antigen (PSMA) PET scans (at baseline and up to 3 mo after treatment). PSMA PET response was assessed by RECIST 1.1, adapted Prostate Cancer Working Group Criteria 3 (using PSMA PET instead of bone scan), aPERCIST, and PSMA PET progression (PPP) criteria. Response by each criterion was stratified by either progressive disease (PD) or non-PD. For aPERCIST, stratification by PD, stable disease (SD), and partial/complete remission (PR/CR) was performed. Biochemical response was determined by a prostate-specific antigen decrease of at least 50%. Subgroup analyses were performed by castration status. Univariable Cox proportional hazards regression analyses including Harrell's concordance indices were calculated to investigate the association of PD by response criteria and OS. Kaplan–Meier tests including log-rank statistics were calculated for survival analyses. **Results:** Twenty-six (25%) patients had unmeasurable disease by RECIST 1.1. PD by any response criterion was associated with an at least 2.5-fold increased risk of death and was highest for PD versus CR/PR by aPERCIST (hazard ratio, 11.4) on univariable regression. Stratified by castration status, a similar pattern was observed. PD by any criterion as associated with significantly shortened OS across overall and subgroup analyses. PR/CR by aPERCIST identified patients with lower risk of death and longer OS compared with patients with PD or SD. **Conclusion:** PSMA PET–based response criteria (PPP, aPERCIST, adapted Prostate Cancer Working Group Criteria 3) have high prognostic utility in men with metastatic PC undergoing taxane-based chemotherapy. PPP is simple to use, identified most patients with PD, and showed best prognostic utility regarding OS. PR/CR by aPERCIST identifies a subgroup of responders (PR/CR) showing better outcomes than patients with PD or SD. Future studies are warranted to amend the current paradigm relying on mere differentiation of PD versus non-PD in metastatic PC and to identify true treatment responders by imaging criteria.

Key Words: metastatic prostate cancer; taxane-based chemotherapy; ^{68}Ga-PSMA11 PET/CT; treatment monitoring; survival

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Prostate cancer (PC) is the most common malignant tumor in men and the second most common cause of cancer-associated mortality (*1*). During the initial hormone-sensitive stage, metastatic PC (mHSPC) typically responds well to androgen deprivation. Nevertheless, most patients will eventually progress despite androgen deprivation, and metastatic castration-resistant PC (mCRPC) develops. This final stage of the disease is associated with poor prognosis and a significantly decreased overall survival (OS) (*2,3*).

Despite the development of novel treatment strategies in both mHSPC and mCRPC, taxane-based chemotherapies remain a standard of care in metastatic PC treatment. Conventional assessment of treatment response in metastatic PC traditionally relies on radiographic criteria including CT and bone scans as proposed by the Prostate Cancer Working Group Criteria 3 (PCWG3) guidelines (*4*). The PCWG3 imaging framework only allows the stratification of progressive disease (PD) versus nonprogression (non-PD) and lacks identifying patients as responders by imaging criteria. The introduction of prostate-specific membrane antigen (PSMA) PET improved the detection of PC metastases compared with conventional imaging (*5*). Current guidelines recommend a PSMA PET in patients with rising or persistently elevated prostate-specific antigen (PSA) after radical treatment (*6,7*).

Whole-body PET imaging has evolved as a reliable tool for assessing response in metastatic disease from various tumor entities (*8,9*). Different frameworks exist for various tumor entities that use either cross-sectional imaging (e.g., RECIST 1.1) or have been introduced for FDG PET (e.g., PERCIST). Most recently, Fanti et al. (*10*) proposed the PSMA PET progression (PPP) criteria for potential use in metastatic PC. However, the use of PSMA PET imaging for assessing response in patients with metastatic PC undergoing systemic treatment is less explored (*11–14*). Despite the introduction of novel frameworks for the application of PSMA PET in metastatic PC, data are limited regarding the prognostic utility of such criteria in comparison to traditional frameworks for the monitoring of treatment response.

The aim of this retrospective analysis was to investigate the comparative prognostic utility of traditional treatment response criteria

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For correspondence or reprints, contact Lukas Lunger (lukas.lunger@tum.de).
*Contributed equally to this work.
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with PSMA PET criteria regarding OS in patients with metastatic PC (mHSPC and mCRPC) undergoing taxane-based chemotherapy. Traditional criteria comprised nonimaging PSA-based response and conventional anatomy–based RECIST 1.1 (*15*). Specifically, PSMA PET criteria included ^{68}Ga-PSMA11 PET–adapted PERCIST (*16*), adapted PCWG3 (*4*), and PPP criteria (*10*). In addition, we aimed to assess whether the definition of response used in PERCIST would further add prognostic information in the group of patients with response.

MATERIALS AND METHODS

Patients

Patients with either mCRPC or mHSPC undergoing taxane-based chemotherapy and ^{68}Ga-PSMA11 PET before and after treatment between January 2014 and December 2018 at the Technical University Munich were included. The term PSMA PET is used throughout the remaining article and refers to the use of ^{68}Ga-PSMA11 PET/CT in the setting of this retrospective analysis.

We included only patients with pairs of PSMA PET that were performed within a maximum interval of 3 mo before initiation of chemotherapy and up to 3 mo after completion of treatment. Patients without follow-up information and survival data were excluded. Patients with mCRPC underwent up to 1 additional interim PSMA PET after 3 mo from initiation of treatment because of the high risk of progression in these patients. Clinical characteristics and serum PSA level were collected both at baseline and at the posttreatment visit.

All reported investigations were conducted in accordance with the Declaration of Helsinki and with national regulations (*17*). The retrospective study was approved by the Ethics Committee of the Technical University Munich (permit 5665/13), and a waiver of consent was granted. The administration of PSMA11 complied with The German Medicinal Products Act (AMG §13 2b) and the responsible regulatory body (Government of Oberbayern).

Response Assessment

Biochemical response was defined as a PSA decrease of at least 50% after chemotherapy. PET-based response was evaluated as follows: all patients underwent PSMA PET from the skull base to the midthigh using a previously described protocol (*11*). In brief, PET scans were acquired in 3D mode, combined with an intravenous and oral contrast-enhanced CT scan. Images were reviewed by an experienced, board-certified nuclear medicine physician using the Prostate Cancer Molecular Imaging Standardized Evaluation criteria for lesion assessment. Any focal uptake higher than that of the surrounding background and not associated with physiologic uptake was considered suspicious for malignancy (*18*). The posttreatment response was subsequently assessed in accordance with RECIST 1.1 (*15*), PPP (*10*), adapted PERCIST (*16*), and adapted PCWG3 (*4*) criteria as described below.

RECIST 1.1. The revised RECIST 1.1 criteria are widely used for response assessment in solid tumors (*19*). The 2 morphologically largest lesions per organ system were selected in CT as target lesions with a maximum of 5 lesions in total. For targeted tumor lesions, the longest diameter with a minimum size of 10 mm by CT scan was measured. For targeted pathologic nodes, the lesions must meet the criterion of short axis greater than 15 mm by CT images. Finally, a sum of the diameters of all target lesions is evaluated, and the results categorize patients into complete response (CR), partial response (PR), stable disease (SD), and progressive disease (PD). Patients who had only nontarget lesions in the prechemotherapy PET scan, without clear progression or disappearance in postchemotherapy PET, were classified as unmeasurable.

PPP criteria. The PPP criteria were recently proposed by Fanti et al. (*10*). Progression using PSMA PET was defined as follows: appearance of 2 or more new PSMA-positive distant lesions, appearance of 1 new PSMA-positive lesion plus consistent clinical or laboratory data, and an increase in size or PSMA uptake of 1 or more existing lesions of at least 30% plus consistent clinical or laboratory data, together with the confirmation by biopsy or correlative imaging within 3 mo. Clinical and laboratory data included changes in Eastern Cooperative Oncology Group score, the record of any improving or worsening of bone pain, or changes in PSA level before and after treatment. For the last criterion, SUV_{max} was used to evaluate changes in PSMA uptake and lesion size was measured according to the RECIST 1.1 protocol.

Adapted PERCIST. PERCIST 1.0 criteria (*16*) were adapted to the use of PSMA PET as follows: 5 organ systems (prostate or prostate bed, lymph nodes, bone, liver, and other visceral metastatic sites) were recorded per patient. For each organ system, up to 2 lesions with the highest PSMA PET uptake identified on maximum intensity projection PET images were selected on the prechemotherapy PET scan (PET1). To measure the SUV_{peak}, a circular 1.2-cm-diameter volume region of interest was placed over the transaxial slice with maximum ^{68}Ga-PSMA11 PSMA uptake. The posttherapeutic PET (PET2) was compared with the prechemotherapy PET scan (PET1) and interpreted as follows: the absence of any PSMA uptake on PET2 in all target lesions seen on PET1 was considered CR; a decrease in the SUV_{peak} sum of at least 30% (minimum decrease in SUV_{peak} of 0.8) was considered PR; the appearance of a new PET positive lesion on PET2 or an increase in SUV_{peak} sum of at least 30% (minimum increase in SUV_{peak} of 0.8) was considered PD; and an intermediate change in summed SUV_{peak} between −30% and +30% without the appearance of new target lesions was considered SD.

Adapted PCWG3 criteria. PCWG3 criteria (*4*) were adapted to the use of PSMA PET as follows: application of RECIST 1.1 for soft-tissue lesions remained unchanged, and information from PSMA PET was used for assessment of bone lesions instead of a bone scan. Patients who exhibited progression according to RECIST 1.1 or had at least 2 new bone lesions on PET2 were classified as PD. Other conditions were defined as non-PD. Given the high specificity of PSMA PET, no additional confirmation of new bone lesions was demanded.

Statistical Analysis

All values are reported as average (SD) or median (interquartile range [IQR]) for continuous variables and as number and percentage for categoric variables. All statistical tests were conducted for the overall collective and after stratification by castration status. Kaplan–Meier tests including log-rank statistics were calculated for survival analyses. OS was defined as the time from initiation of chemotherapy until death from any causes. Patients who were alive or lost to follow-up were censored at the last date they were known to be alive.

The association between biochemical response and PET-based criteria (aPCWG3, aPERCIST, and PPP) with OS was evaluated using univariate Cox regression analyses and reported as hazard ratio (HR) and 95% CI. To evaluate the goodness of fit of performed Cox regression analyses, the Harrell's concordance index (C-index) was calculated. Ties were included in the calculation of the C-index. $P < 0.05$ was considered statistically significant. All statistical analyses were performed using SPSS version 26 (SPSS).

RESULTS

Patients and Disease Characteristics

A total of 103 patients (mHSPC, $n = 57$; mCRPC, $n = 46$) were included. Clinical characteristics are outlined in Table 1. Most patients had bone metastasis (M1b; $n = 80$ of 103, 78%) and extrapelvic lymph node metastasis (M1a; $n = 72$ of 103, 70%).

Median duration from prechemotherapy PSMA PET to initial chemotherapy was 27 d (IQR, 14–49), whereas the median duration from last chemotherapy application to posttreatment PET was 27 d (IQR, 18–39).

TABLE 1
Clinical Characteristics

Characteristic	Data
Median age (y)	71 (range, 43–85)
Median pre-CTX PSA level (ng/mL)	
mHSPC	15 (IQR, 2–39)
mCRPC	45 (IQR, 10–155)
Gleason score (*n*)	
≤7	31 (30%)
≥8	67 (65%)
NA	5 (5%)
ECOG performance status (*n*)	
0	53 (52%)
1	26 (25%)
2	1 (1%)
NA	23 (22%)
Castration status (*n*)	
mHSPC	57 (55%)
mCRPC	46 (45%)
Pre-CTX miTNM staging (*n*)	
No distant metastasis (M0)	6 (6%)
Extrapelvic node metastasis (M1a)	72 (70%)
Bone metastasis (M1b)	80 (78%)
Visceral metastasis (M1c)	16 (16%)
Pattern of metastatic spread (*n*)	
LN only	21 (21%)
Bone only	9 (9%)
Visceral only	1 (1%)
LN and bone	54 (52%)
LN and visceral organs	1 (1%)
LN, bone, and visceral organs	14 (14%)
LN, bone, and others (subcutaneous, skin metastasis)	2 (2%)
Bone and others (penis)	1 (1%)
Local treatment for PC (*n*)	
Prostatectomy ± lymphadenectomy	58 (56%)
Primary EBRT	13 (13%)
Type of chemotherapy (*n*)	
Docetaxel	95 (92%)
Cabazitaxel	7 (7%)
Docetaxel and cabazitaxel	1 (1%)
Reduction in serum PSA ≥ 50% (*n*)	61 (59%)

CTX = chemotherapy; ECOG = Eastern Cooperative Oncology Group; EBRT = external beam radiotherapy; NA = not available; LN = lymph nodes.

Biochemical Response

Overall, 61 patients (59%) had a PSA decrease of at least 50% after taxane-based chemotherapy. Stratified by castration status, 45 (79%) mHSPC patients and 16 (35%) mCRPC patients had a PSA decrease of at least 50%.

For mHSPC, median pre- and postchemotherapy PSA values were 14.9 (IQR, 1.9–39.3) and 0.6 ng/mL (IQR, 0.04–3.5), respectively. For mCRPC, median pre- and postchemotherapy PSA values were 45 (IQR, 9.5–154.5) and 34 ng/mL (IQR 6.7–153.8), respectively.

Imaging-Based Response

By RECIST 1.1 criteria, 26 (25% of all patients) had unmeasurable disease. Of those, 5 patients had isolated bone metastases without soft-tissue component, 6 patients had nontarget lymph nodes with or without nontarget prostatic bed lesions, and 15 patients had coexisting nontarget nodes and bone lesions. Survival and further response analyses for RECIST 1.1 were therefore omitted. An overview of response rates by PET response criteria is given in Table 2.

Overall, by adapted PERCIST criteria, PD was observed in 33 (32%) patients. For mHSPC and mCRPC, PD was observed in 8 (14%) and 25 (54%) patients, respectively. By aPCWG3 criteria, PD was observed in 34 (33%) patients. For mHSPC and mCRPC, PD was observed in 9 (16%) and 25 (54%) patients, respectively. By PPP criteria, PD was observed in 41 (40%) patients. Stratified by castration status, PD was observed in 11 (19%) mHSPC patients and 30 (65%) mCRPC patients.

Survival Analyses by Response Criteria

Median OS of all patients was 50 mo (95% CI, 31–69). Median OS for patients with mCRPC was 18 mo (95% CI, 15–21 mo) and was not reached for patients with mHSPC.

Kaplan–Meier analyses by biochemical response and imaging-based response criteria are shown in Figures 1A–1D. Progression by any investigated criterion was associated with a significantly shorter OS compared with response (median survival ranging from 14 to 17 mo [PD] vs. median survival ranging from 57 mo – not reached. [no-PD]). Additional survival analyses for stratification by castration status are shown as a cumulative illustration in Supplemental Figure 1 (supplemental materials are available at http://jnm.snmjournals.org).

TABLE 2
Overall Response Rates by Imaging Response Criteria

Criteria	Response	*N* (%)
RECIST 1.1	n/m	26 (25)
	PD	20 (19)
	SD	38 (37)
	PR	15 (15)
	CR	4 (4)
aPERCIST	PD	33 (32)
	SD	18 (18)
	PR	45 (44)
	CR	7 (7)
aPCWG3	PD	34 (33)
	No-PD	69 (67)
PPP	PD	41 (40)
	No PD	62 (60)

n/m = unmeasurable disease.

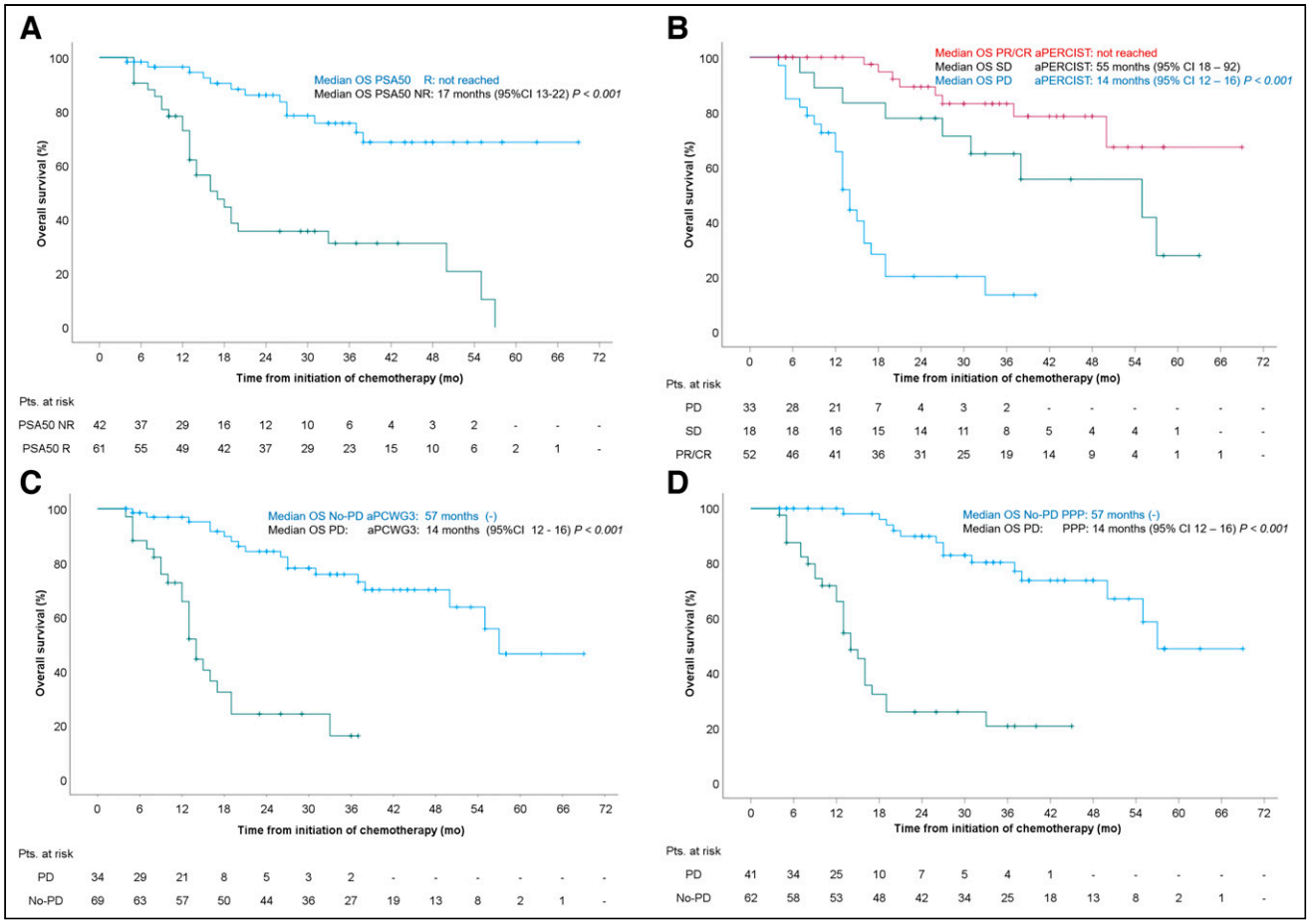

FIGURE 1. Kaplan–Meier estimates of OS by biochemical response (A), adapted PERCIST (B), adapted PCWG3 (C), and PPP (D).

Prognostic Value of Response Criteria

Cox regression analyses investigating the associations of PET-based response criteria and biochemical response with OS are given in Table 3. Of note, on univariable analysis, PD (independent of castration status) as defined by any of the investigated PET criteria in this study was associated with a significantly increased risk of death (HR range, 4.1–8.1; 95% CI, 2.5–16.7). C-index analyses revealed the strongest prognostic values for PPP (HR, 0.77; 95% CI, 0.72–0.81) and aPERCIST (HR, 0.75; 95% CI, 0.69–0.80). Details are presented in Table 3.

Stratified by castration status, for mHSPC, aPCWG3 and PPP criteria showed a strong prognostic value with a C-index > 0.73). For mCRPC status, aPERCIST and PPP criteria C-indices were highest (>0.69; see supplemental Tables 1 and 2 for more detailed results).

DISCUSSION

The objective and reliable evaluation of response to systemic treatment is critical to both clinical research and practice. To date, a variety

TABLE 3
Univariable Cox Regression Analyses for Association of Response Criteria with OS, All Patients (mCRPC and mHSPC) Included

Criteria	Response	*N**	HR	95% CI	*P*	C-index
PSA50	PD vs. no-PD	42 vs. 61	4.8	2.5–9.3	<0.001	0.695 (0.629–0.761)
aPERCIST	PD vs. no-PD	33 vs. 70	8.1	4.1–16.2	<0.001	0.746 (0.690–0.802)
	PD vs. SD	33 vs. 18	4.1	1.7–10.1	<0.001	
	PD vs. PR/CR	33 vs. 52	11.4	4.7–27.1	<0.001	
aPCWG3	PD vs. no-PD	34 vs. 69	7.1	3.5–14.2	<0.001	0.729 (0.670–0.788)
PPP	PD vs. no-PD	41 vs. 62	8.1	4.0–16.7	<0.001	0.765 (0.721–0.808)

n/m = unmeasurable disease; PSA50 = dichotomous, biochemical response with PSA reduction ≥ 50%; *Number of patients.

of frameworks exists to determine response to PC treatment, but data on their prognostic value and use in routine clinical practice are limited. Here, we present a considerably large and evenly balanced cohort including both mCRPC and mHSPC patients to retrospectively compare the prognostic utility of response to taxane-based chemotherapy by serum PSA decrease, RECIST 1.1, aPERCIST, aPCWG3, and PPP criteria regarding OS. Within the used imaging biomarkers, aPERCIST offers the possibility not only to detect PD versus non-PD but also to identify PR/CR.

Overall, PD (independent of castration status) as defined by traditional and PSMA PET frameworks was associated with an at least 2.5-fold increased risk of death by univariable analyses in this study. PD by aPERCIST was associated with the highest risk of death (HR, 11.4; 95% CI, 4.7–27.1) compared with patients with PR/CR ($P < 0.001$). To compare the prognostic utility of reported HRs, C-index analyses were performed. Based on its C-index, PPP was the framework with the highest prognostic value compared with the other investigated criteria; however, it was not statistically different. Overall, Kaplan–Meier curve analyses revealed that PD by any investigated criterion was associated with significantly reduced OS. Of interest, across all criteria, only in patients classified as having PR/CR by aPERCIST was median OS not reached and significantly shorter in patients with PD and SD (14 [95% CI, 12–16] and 55 mo [95% CI, 18–92], respectively; $P < 0.001$). The same pattern was observed after stratification by castration status (Supplemental Figure 1).

A recent review investigated the role of PET-based imaging for response to systemic treatment in metastasized PC (*14*). The results suggest that the volumetric extent of metastatic burden or the total lesion PSMA estimated by PSMA PET may have prognostic value in patients undergoing taxane-based chemotherapy (*14*). However, the evidence backing this conclusion is sparse and relies on a limited number of studies with small sample sizes. Moreover, most studies investigate different endpoints and lack standardized definitions of response and comparable follow-up. Similarly, 2 works by Has Simsek et al. (*12*) and Shagera et al. (*13*) most recently aimed to investigate the prognostic role of total metastatic burden. Of interest, both studies found that PD by PSMA total tumor volume was associated with a significantly shortened OS. Yet, the significance of presented results appears limited by the overall small sample size, the lack of posttreatment PET scans, and the combination of both mCRPC and mHSPC patients in 1 cohort. In contrast, we focused on the use of different diagnostic frameworks for response assessment. Here, we assessed response by comparing a limited number of lesions across 2 different timepoints and assessing the presence of new lesions in follow-up scans. Of note, the investigated methods in our study did not require the determination of the whole-body tumor volume. However, our data clearly indicate that PD, as defined in PPP, PCWG3, and aPERCIST, was associated with shorter OS. Yet, future prospective studies are warranted to validate and determine the prognostic benefit of one framework over the other, including a definition of reliable cutoffs for the measurement of whole-body metastatic tumor volume.

Comparing the investigated frameworks provided insights, and in contrast to previous studies (*11*), our analysis is based on long-term follow-up, allowing a comparison of results from different frameworks with OS as a clinically most meaningful outcome parameter. First, our results showed that PPP and aPERCIST had the highest comparative prognostic value. However, whereas response assessment using PPP is much more feasible in routine practice given its simple application, aPERCIST provides the potential not only to discriminate between PD and non-PD but also to identify responders (CR/PR) within this group with substantially better outcome compared with SD. This finding highlights an important aspect of aPERCIST worth investigation in further prospective studies. To date, the use of software solutions (semi-)automatically detecting, quantifying, and following tumor lesions over time is required to facilitate its adoption. Such tools are currently under development by various vendors but are not yet fully implemented in standard software solutions. Thus, the application of aPERCIST criteria remains currently limited to scientific investigations until automated analyses are routinely available.

Conversely, response by aPCWG3 as a straightforward adoption of PCWG3 for the use with PSMA PET did not outperform aPERCIST or PPP in terms of prognostic utility in this study. In addition, despite the incorporation of similar criteria compared with PPP for bone assessment, the application remains time consuming and is hampered by the manual quantitative measurement of soft-tissue metastases. Similar to previous investigations, response by traditional RECIST 1.1 is not useful for metastasized PC (*20*). In our cohort, 25% of included patients only had unmeasurable disease, confirming a major limitation of this framework for response assessment in metastatic PC. Furthermore, typical for metastasized PC, 78% of patients had bone metastases in this study. However, these lesions cannot be assessed quantitatively within this framework in the absence of extraosseous soft-tissue extension and osteolytic lesions. Progression of sclerotic bone metastases in RECIST 1.1 can only be determined in the case of "unequivocal progression" of nontarget lesions, which is prone to subjective interpretation. These observations led to its combination with bone scintigraphy and their combined use within the PCWG framework (*4*). Taken together, our results suggest that PPP seems most useful for determination of treatment response of metastasized PC in routine clinical practice, given its easy adoption and its comparable prognostic utility to aPCWG3 or aPERCIST.

Here, we also analyzed the utility of traditional response by serum PSA reduction as a prognostic biomarker. Although serum PSA measurement effectively stratified patients in PD and non-PD, its C-index was lower compared with the other imaging-based response criteria. In addition, it is known that its sole use is hampered by the known inter- and intratumor heterogeneity associated especially with mCRPC (*21*), as well as its inability to detect clinically relevant complications (e.g., fractures and embolism) compared with imaging-based response assessment.

Several limitations of this study are noteworthy including the small cohort size and the retrospective study design, associated with selection and misclassification bias. Additionally, the current study reflects a single center experience, and all imaging data sets were reviewed by a single physician. One particularly relevant limitation concerns the study population: here, both patients with mCRPC and mHSPC were included for survival analyses. The inclusion of mHSPC patients might have introduced substantial bias of obtained univariable regression results because of the small study population and the low rate of PD events compared with mCRPC patients alone. Nevertheless, all results followed a similar trend even after stratification by castration status and expand the available knowledge on the prognostic role of PSMA PET–based response criteria regarding OS in a large cohort of patients with metastatic PC.

CONCLUSION

The PSMA PET–based response criteria PPP, aPERCIST, and aPCWG3 are reliable and prognostic tools for the assessment of treatment response after taxane-based chemotherapy in both mHSPC and

mCRPC patients. PPP appears most useful for determination of treatment response of metastasized PC in routine clinical practice. PPP is easy to adopt and implement in clinical routine, and its prognostic utility was similar to aPCWG3 or aPERCIST in this study while lacking the need of (semi)-automated software applications. In contrast, aPERCIST offers the possibility to also identify a subgroup of responders (PR/CR) showing reduced risk of death and associated with a significantly longer OS compared with patients with PD and SD. Its further prospective investigation is warranted to potentially expand the current paradigm assessing only PD versus non-PD to the identification of true response by an imaging biomarker for response in metastasized PC.

DISCLOSURE

Matthias Eiber reports fees from Blue Earth Diagnostics Ltd. (consultant, research funding), Novartis/AAA (consultant), Telix (consultant), Bayer (consultant, research funding), RayzeBio (consultant), Point Biopharma (consultant), Janssen Pharmaceuticals (consultant, speakers bureau), Parexel (image review), and Bioclinica (image review) outside the submitted work and a patent application for rhPSMA. No other potential conflict of interest relevant to this article was reported.

KEY POINTS

QUESTION: What is the comparative prognostic utility of traditional treatment response criteria with PSMA PET criteria for OS in metastatic PC patients undergoing taxane based chemotherapy?

PERTINENT FINDINGS: This retrospective cohort study on (n = 103) metastatic PC patients showed that PD by any response criterion was associated with an at least 2.5-fold increased risk of death and was highest for PD versus CR/PR by aPERCIST. PPP as an easy to determine parameter showed best prognostic utility regarding OS.

IMPLICATIONS FOR PATIENT CARE: PSMA PET–based response criteria have high prognostic utility in men with metastatic PC undergoing taxane-based chemotherapy and may help to identify patients at high risk for reduced OS.

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