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The Impact of the Percent of Residual Prostate-Specific Antigen on Metastasis-Free Survival in Patients with Persistent Prostate-Specific Antigen after Radical Prostatectomy

Dan Bee Lee[1], Jae Yeon Kim[1], Won Hoon Song[1], Jong Kil Nam[1], Hyun Jung Lee[2], Tae Un Kim[3], Sung-Woo Park[1,4]

[1]Department of Urology, Pusan National University Yangsan Hospital, [2]Department of Pathology, Pusan National University Yangsan Hospital, [3]Department of Radiology, Pusan National University Yangsan Hospital, [4]Research Institute for Convergence of Biomedical Science and Technology, Pusan National University Yangsan Hospital, Yangsan, Korea

Purpose: Persistent levels of prostate-specific antigen (PSA) is a poor prognostic factor for recurrence after radical prostatectomy (RP). We investigated the impact of the percentage of residual PSA (%rPSA) [(post-/preoperative PSA)×100], representing a biochemical residual tumor, and the first postoperative PSA (fPSA) level on metastasis-free survival (MFS) in men with persistent levels of PSA after RP.

Materials and Methods: We retrospectively identified male patients within a single tertiary referral hospital database who harbored persistent (≥0.1 ng/mL) *vs*. undetectable (<0.1 ng/mL) PSA levels 4 to 8 weeks after RP. Kaplan–Meier analyses and Cox regression models were used to test the effect of persistent PSA levels, the fPSA level, and %rPSA on MFS.

Results: Of 1,205 patients, 178 patients with persistent PSA levels were enrolled. Seven-year MFS rates were 60.5% *vs*. 84.3% (p<0.001) for patients with a %rPSA ≥6% and <6%, respectively. Multivariable Cox regression models of the overall cohort revealed that persistent PSA levels (hazard ratio [HR], 3.94; p=0.010), extracapsular extension (HR, 4.17; 95% confidence interval [CI], 1.06–16.41; p=0.041), and pathological Gleason grade group (pGGG) (HR, 3.69; 95% CI, 1.32–10.27; p=0.013) were independent predictors of metastasis. Multivariable Cox regression models in men with persistent PSA levels revealed that the %rPSA (HR, 8.92; 95% CI, 1.74–45.71; p=0.009) and pGGG 4–5 (HR, 4.13; 95% CI, 1.22–13.96; p=0.022) were independent predictors of distant metastasis, but not the fPSA level after surgery.

Conclusions: Persistent levels of PSA were associated with worse MFS after RP. In men with persistent PSA levels after RP, the %rPSA is a valuable predictor of MFS unlike the fPSA level.

Keywords: Metastasis; Prostate cancer; Prostate-specific antigen; Prostatectomy; Survival

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Correspondence to: Sung-Woo Park https://orcid.org/0000-0002-9895-3461
Department of Urology, Pusan National University Yangsan Hospital, 20 Geumo-ro, Mulgeum-eup, Yangsan 50612, Korea.
Tel: +82-55-360-2672/2134, **Fax:** +82-55-360-2164, **E-mail:** psw@pusan.ac.kr

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INTRODUCTION

Radical prostatectomy (RP) can provide good long-term oncological outcomes in patients with localized and locally advanced prostate cancer (PCa) [1,2]. RP aims to fully remove the tumor burden, and the goal following surgery is for levels of prostate-specific antigen (PSA) to be undetectable [3]. Undetectable levels of PSA (<0.1 ng/mL) after RP means there is no biochemically active tumor burden. Conversely, persistent levels of PSA (≥0.1 ng/mL) mean that there is a residual biochemically active tumor burden, regardless of imaging findings [4]. Therefore, persistent PSA is a poor prognostic factor for recurrence after RP [4-11].

Several investigators have reported the impact of persistent PSA levels after RP on oncological outcomes [4,6-11]. However, most studies have focused on persistent PSA levels (≥0.1 ng/mL) only and not on the accurate level of PSA. Within 15 years, approximately 75% and 47% of patients with persistent PSA levels after RP develop biochemical recurrence (BCR) and metastasis, respectively [7,11]. These patients have heterogeneous progression patterns. Therefore, we should stratify the risk of progression in this population to determine the appropriate salvage treatment.

In men with persistent PSA levels after RP, presence of a measurable residual tumor on conventional imaging, such as computed tomography/magnetic resonance imaging, and bone scan, is uncommon. Thus, we need another test to measure the residual tumor burden in addition to imaging tests. Theoretically, levels of PSA after RP may indicate residual tumor burden because PCa cells express PSA [3,12]. Therefore, measurement of postoperative PSA levels may provide more information regarding PCa tissue burden.

Here, we describe the relationship between persistent levels of PSA at 4 to 8 weeks after RP and oncological outcomes within a single tertiary referral hospital database. To stratify the risk of progression in men with persistent PSA levels, we investigated the impact of the first postoperative PSA (fPSA) level and the percentage of residual PSA (%rPSA) [(postoperative PSA/preoperative PSA)×100] on the metastasis-free survival (MFS) rate.

MATERIALS AND METHODS

1. Ethics statement

This retrospective, single-center study was approved by Pusan National University Yangsan Hospital Institutional Review Board (approval number: 05-2019-174). The requirement for informed consent was waived by the board as all data was being analyzed retrospectively, after de-identification.

2. Study population

A total of 1,205 patients who underwent RP (December 2008–December 2020) from a single tertiary referral hospital (Pusan National University Yangsan Hospital, Yangsan, Korea) were identified from its database. Patients were stratified according to either persistent PSA levels (≥0.1 ng/mL) *vs.* undetectable PSA levels (<0.1 ng/mL) at 4 to 8 weeks after RP. RP was performed using an open retropubic, pure laparoscopic, or robot-assisted approach. All RP specimens were evaluated in a whole-mount fashion by a dedicated uropathologist. Exclusion criteria consisted of unknown PSA level at 4 to 8 weeks after RP, neo-/adjuvant androgen deprivation treatment, and/or adjuvant radiation.

3. Outcomes

We collected serum PSA level before prostate biopsy (within 1 mo) as a preoperative PSA. After biopsy, RP were performed within 3 months in most patients. PSA levels were first assessed 4 to 8 weeks after surgery, and then, every 3 months until 2 years. After 2 years, PSA was measured every 6 months. Imaging tests were performed every year until 5 years or when PSA levels increased.

Distant metastasis was diagnosed by positive imaging for persistent PSA levels or BCR (two consecutive PSA values of at least 0.2 ng/mL after surgery). Imaging procedures consisted of bone scan, computed tomography, and/or abdominal magnetic resonance imaging. MFS was calculated as the time from RP to metastasis or the last follow-up.

4. Covariates

Covariates included age, preoperative PSA level, pathologic Gleason grade group (pGGG), pathologic tumor stage, surgical margin status, pathologic lymph node status, and early salvage radiation (eSRT). eSRT was defined as radiation delivered within 3 months of

BCR. The decision to undergo salvage radiation was at the discretion of the urologist based on the PSA level and/or positive imaging findings.

The salvage radiation field included a prostatic bed and the entire pelvis. The median radiation dose was 72 Gy, with a median number of 36 fractions and 2 Gy per fraction. Among the patients with persistent PSA levels, 72 patients (40.4%) underwent salvage radiation. Thirty-five (48.6%) and 37 (51.4%) patients underwent delayed (≥3 mo) or earlier (<3 mo) radiation after BCR, respectively. Information about the receipt and duration of androgen deprivation treatment was not available for all patients.

5. Statistical analyses

Descriptive statistics were reported using frequencies and proportions for categorical variables and medians and interquartile ranges for continuous variables. Chi-square and Student's t-tests were used to evaluate the statistical significance of differences in categorical and continuous variables, respectively.

Kaplan–Meier analyses depicted MFS according to the fPSA level or %rPSA. The first set of multivariable Cox regression models was fitted to test the relationship between PSA persistence and MFS in the entire study cohort. Subsequently, multivariable Cox regression models were repeated in the subgroup of patients with persistent PSA levels. Specifically, the second set tested the relationship between the fPSA level and MFS, and the third set tested the relationship between %rPSA and MFS. All multivariable Cox models were adjusted for age, preoperative PSA level, surgical margin status, pathological stage, and pGGG. Multivariate Cox models in the subgroup were adjusted for eSRT.

All statistical analyses were performed using SPSS (version 26.0; IBM Corp., Armonk, NY, USA). A two-sided statistical significance was defined as a p-value <0.05.

RESULTS

1. Descriptive statistics

Of 1,205 identified patients, 14.9% (n=180) and 85.1% (n=1,025) harbored persistent or undetectable PSA levels, respectively. After exclusion, 1,133 patients, including 178 patients with persistent PSA levels, were enrolled. Approximately 15% (n=27) of patients with persistent PSA levels had an undetectable PSA level in subsequent PSA testing without any salvage treatment (Fig. 1).

Patients with persistent PSA levels had a higher proportion of pGGG 4–5 (59.0% *vs.* 17.2%, p<0.001), and more frequently harbored positive surgical margins (52.8% *vs.* 21.6%, p<0.001), seminal vesicle invasion (52.2% *vs.* 8.2%, p<0.001), and lymph node invasion (pN1: 12.4% *vs.* 0.7%, p<0.001) compared to patients with undetectable PSA levels (Table 1). Moreover, patients with persistent PSA levels more frequently received salvage radiation (40.4% *vs.* 6.8%, p<0.001). The median follow-up was 32 months and 36 months for patients with undetectable and persistent PSA levels, respectively. During follow-up, 13.1% (n=134), 0.7% (n=7), and 2.1% (n=22) of patients with undetectable PSA levels after surgery developed BCR, local recurrence, and distant metastasis, respectively. However, 84.8% (n=151), 11.2% (n=20), and 11.2% (n=20) of patients with persistent PSA levels developed BCR, local recurrence, and distant metasta-

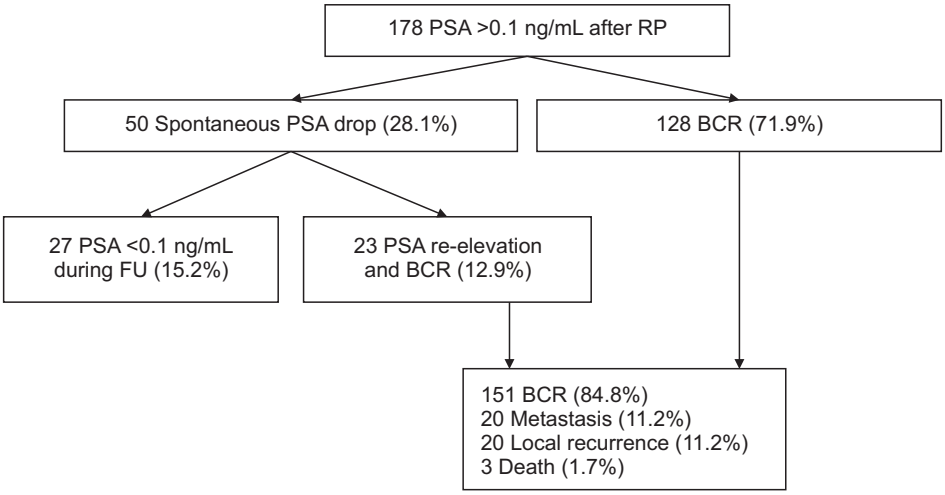

Fig. 1. Follow-up (FU) results in patients with persistent prostate-specific antigen (PSA) after radical prostatectomy. Although 27 patients (15.2%) had persistently high PSA level (>0.1 ng/mL) after radical prostatectomy, they did not experience biochemical recurrence (BCR) during FU. Among the patients with persistent PSA level (>0.1 ng/mL) after radical prostatectomy, distant metastasis and local recurrence was diagnosed in 20 patients (11.2%) and 20 patients (11.2%), respectively. FU: 12 mo-143 mo.

Table 1. Descriptive characteristics of patients treated with radical prostatectomy, stratified according to postoperative PSA (persistent PSA *vs.* undetectable PSA) (n=1,133)

Variable	Undetectable PSA (n=955)	Persistent PSA (n=178)	p-value
Age, y	67 (62–72)	69 (64–73)	0.078
Preoperative PSA, ng/mL	7.3 (5.2–11.0)	17.3 (9.0–36.1)	<0.001
Prostate volume, mL	33.5 (26.6–43.3)	34.8 (27.0–44.0)	0.228
Tumor volume, %	13 (6–22)	35 (18–56)	<0.001
Surgical margin status, positive	206 (21.6)	94 (52.8)	<0.001
Pathological T stage			
pT2	648 (67.9)	33 (18.5)	<0.001
pT3a	229 (24.0)	48 (27.0)	<0.001
pT3b	78 (8.2)	93 (52.2)	<0.001
pT4	0 (0.0)	4 (2.2)	<0.001
Pathological lymph node status			<0.001
pN0	99 (10.4)	54 (30.3)	
pNx	849 (88.9)	102 (57.3)	
pN1	7 (0.7)	22 (12.4)	
Salvage radiation, yes	65 (6.8)	72 (40.4)	<0.001
Salvage androgen deprivation, yes	101 (10.6)	131 (73.6)	<0.001
Pathological Gleason grade group			<0.001
1–3	791 (82.8)	73 (41.0)	
4–5	164 (17.2)	105 (59.0)	
First PSA after surgery, ng/mL	0.01 (0.00–0.02)	0.40 (0.17–1.52)	<0.001
Percent of residual PSA, %	0 (0–0)	3 (1–7)	<0.001
Follow-up duration, mo	32 (11–59)	36 (10–56)	0.832

Values are presented as median (interquartile range) or number (%).
PSA: prostate-specific antigen.

Table 2. Cox regression analysis models predicting metastasis in the entire study cohort (n=1,133)

Variable	Univariate			Multivariable		
	HR	95% CI	p-value	HR	95% CI	p-value
Persistent prostate-specific antigen	16.71	7.01–39.56	<0.001	3.94	1.40–11.11	0.010
Age, y	1.02	0.96–1.09	0.499	1.01	0.95–1.08	0.727
Preoperative prostate-specific antigen, ng/mL	1.02	1.01–1.04	<0.001	0.99	0.98–1.01	0.316
Positive surgical margin	4.28	1.96–9.38	<0.001	1.07	0.43–2.64	0.884
Extracapsular extension	17.52	5.25–58.39	<0.001	4.17	1.06–16.41	0.041
Seminal vesicle invasion	12.08	5.29–27.62	<0.001	1.76	0.66–4.70	0.261
Pathological lymph node metastasis	12.56	5.48–28.79	<0.001	2.08	0.81–5.35	0.129
Pathological Gleason grade group 4–5 (*vs.* 1–3)	14.65	5.90–36.38	<0.001	3.69	1.32–10.27	0.013

HR: hazard ratio, CI: confidence interval.

sis, respectively.

2. Effect of prostate-specific antigen persistence on metastasis free survival

Metastasis developed in 20 (11.2%) and 7 (0.7%) patients with persistent and undetectable PSA levels, respectively (p<0.001). Seven years after RP, the MFS rate was 77.5% *vs.* 98.5% (p<0.001) in patients with persistent *vs.* undetectable PSA levels, respectively.

Multivariable Cox regression models performed on the overall cohort tested the relationship between PSA persistence after RP and metastasis (Table 2). Persistent levels of PSA were independent predictors of metastasis (hazard ratio [HR], 3.94; 95% confidence

Table 3. Cox regression analysis models predicting metastasis in men with persistent prostate-specific antigen (n=178)

Variable	Univariate			Multivariable (Model I)			Multivariable (Model II)		
	HR	95% CI	p-value	HR	95% CI	p-value	HR	95% CI	p-value
Percent of residual prostate-specific antigen	5.61	1.43–21.99	0.013	8.92	1.74–45.71	0.009	-	-	-
First prostate-specific antigen after surgery	1.03	0.99–1.06	0.135	-	-	-	1.02	0.98–1.06	0.330
Age, y	0.98	0.91–1.05	0.513	-	-	-	-	-	-
Positive surgical margin	1.28	0.51–3.23	0.605	-	-	-	-	-	-
Extracapsular extension	2.78	0.64–11.98	0.171	-	-	-			
Seminal vesicle invasion	2.21	0.80–6.10	0.125	-	-	-	-	-	-
Pathological lymph node metastasis	2.57	1.02–6.50	0.046	2.10	0.79–5.58	0.138	2.12	0.78–5.76	0.141
Pathological Gleason grade group 4–5 (*vs.* 1–3)	3.47	1.16–10.44	0.027	4.13	1.22–13.96	0.022	3.40	1.08–10.69	0.036
Early salvage radiation	0.51	0.15–1.76	0.284	0.36	0.10–1.27	0.110	0.34	0.09–1.19	0.091

HR: hazard ratio, CI: confidence interval, -: not available.

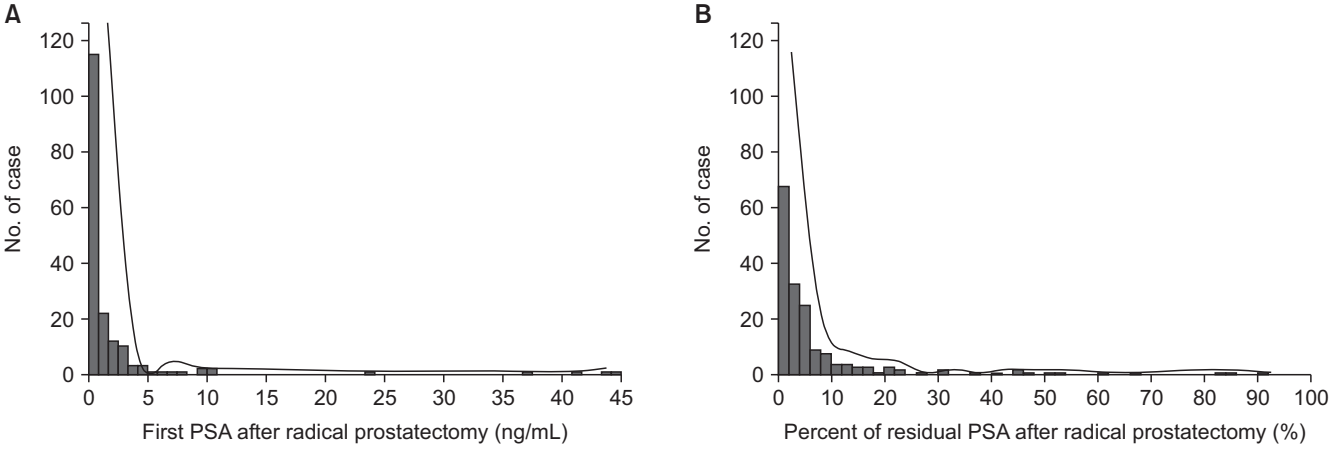

Fig. 2. Distribution of the first prostate-specific antigen (PSA) and the percent of residual PSA in men with persistent PSA after radical prostatectomy. (A) First PSA after radical prostatectomy. It was distributed homogeneously with low PSA levels (<1.0 ng/mL). (B) Percentage of residual PSA after radical prostatectomy. Approximately 30% of patients with persistent PSA levels had a percentage of residual PSA levels higher than >6%. It could be an ideal cut-off.

interval [CI], 1.40–11.11; p=0.010) after adjustment for all covariates. In addition, extracapsular extension (HR, 4.17; 95% CI, 1.06–16.41; p=0.041) and pGGG (HR, 3.69; 95% CI, 1.32–10.27; p=0.013) were independent predictors of MFS.

3. Effect of the percentage of residual prostate-specific antigen and first prostate-specific antigen level after radical prostatectomy on metastasis frees survival

In subgroup analyses focusing exclusively on patients with PSA persistence, pathological lymph node metastasis (HR, 2.57; 95% CI, 1.02–6.50; p=0.046), pGGG 4–5 (HR, 3.47; 95% CI, 1.16–10.44; p=0.027), and %rPSA (HR, 5.61; 95% CI, 1.43–21.99; p=0.013) were associated with a higher risk of metastasis in univariate logistic regression analysis (Table 3). In two multivariable regression analysis models, %rPSA (HR, 8.92; 95% CI, 1.74–45.71; p=0.009) and pGGG 4–5 (HR, 4.13; 95% CI, 1.22–13.96; p=0.022) were independent predictors of distant metastasis (Table 3). However, the fPSA level after surgery was not a predictor of distant metastasis.

Fig. 2A shows the distribution of fPSA in men with persistent PSA. Because it was distributed homogeneously with low PSA level (<1.0 ng/mL), the ideal cut-off could not be identifiable. Fig. 2B shows the distribution of %rPSA in patients with persistent levels of PSA after RP. Approximately 30% of patients with persistent PSA levels had a percentage of residual PSA levels higher than >6%. %rPSA ≥6% is considered the most ideal cut-off based on this distribution. Patients with a %rPSA ≥6% were younger (p=0.021) and had a

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Table 4. Descriptive characteristics of patients treated with radical prostatectomy, stratified according to postoperative PSA (persistent PSA *vs.* undetectable PSA) (n=178)

Variable	%rPSA		p-value
	<6% (n=126)	≥6% (n=52)	
Age, y	70 (65–74)	65 (62–71)	0.021
Preoperative PSA, ng/mL	19.4 (10.6–37.1)	14.3 (7.5–34.5)	0.598
Prostate volume, mL	35.0 (26.7–44.0)	33.6 (27.3–47.0)	0.651
Tumor volume, %	35 (15–58)	34 (20–50)	0.999
Surgical margin status, positive	71 (56.3)	23 (44.2)	0.186
Pathological T stage			
pT2	26 (20.6)	7 (13.5)	0.297
pT3a	35 (27.8)	13 (25.0)	0.430
pT3b	64 (50.8)	29 (55.8)	0.250
pT4	1 (0.8)	3 (5.8)	
Pathological lymph node status			<0.001
pN0	37 (29.4)	17 (32.7)	
pNx	79 (62.7)	23 (44.2)	
pN1	10 (7.9)	12 (23.1)	
Salvage radiation, yes	49 (38.9)	23 (44.2)	0.508
Salvage androgen deprivation, yes	90 (71.4)	41 (78.8)	0.354
Gleason grade group			0.094
1–3	57 (45.2)	16 (30.8)	
4–5	69 (54.8)	36 (69.2)	
First PSA after surgery, ng/mL	0.25 (0.14–0.48)	2.41 (1.06–4.79)	<0.001
Percent of residual PSA, %	1.5 (0.8–3.4)	14.9 (8.8–31.1)	<0.001
Follow-up duration, mo	37 (12–58)	36 (7–53)	0.311

Values are presented as median (interquartile range) or number (%).
PSA: prostate-specific antigen, %rPSA: percent residual PSA.

higher proportion of pathological lymph node metastasis (p<0.001) compared to patients with a %rPSA <6% (Table 4). Otherwise, there was no difference between the two groups. In men with persistent levels of PSA, the 7-year MFS rates were 60.5% *vs.* 84.3% for patients with a %rPSA ≥6% *vs.* <6% (log-ranked test, p<0.001) (Fig. 3).

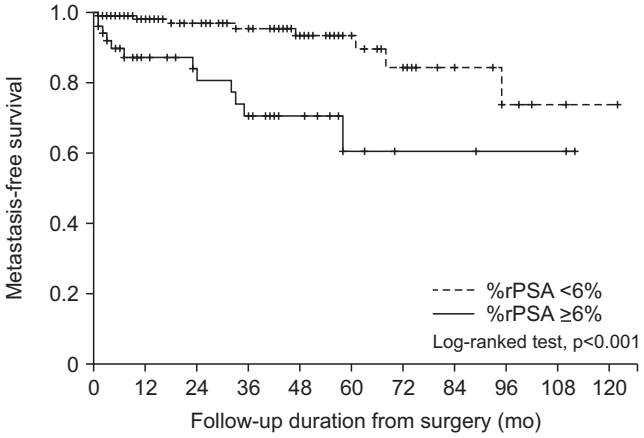

Fig. 3. Metastasis-free survival according to the percent of residual prostate-specific antigen (PSA).

DISCUSSION

Levels of PSA represent the cornerstone of the follow-up for patients who have undergone RP. In particular, early PSA values after RP could help predict worse oncological outcomes. Previous studies have shown poor oncological outcomes based on the presence of PSA persistence measured 4 to 8 weeks after RP [5-11]. However, most of these studies evaluated PSA persistence itself, with no analysis based on PSA values. If early PSA values after RP indicate the burden of residual tumor, the prognosis will vary according to its value. Moreover, it would be helpful if we could measure the burden of residual tumor to determine if salvage treatment should be administered, such as salvage radiation.

PSA is secreted by epithelial cells of the prostate gland and PCa tissues [3,12]. The half-life of PSA is 3.15 days and should reach an undetectable level within 4 weeks in patients who have undergone complete pathological resection [12]. As a result, persistently detectable PSA levels 4 to 8 weeks after RP indicate either residual PCa, residual benign tissue, recurrence in the prostatic bed, distant micro-metastasis, or a combination of these [4].

To the best of our knowledge, this is the first study to investigate the impact of %rPSA after RP on oncological outcomes. In the present study, fPSA levels and %rPSA were evaluated as indicators of residual tumors. Unfortunately, a simple fPSA level was not a predictor of distant metastasis. However, %rPSA was well established in this study as an independent predictor of MFS in patients with persistent PSA levels after RP. In particular, the MFS rate differed between patients with a %rPSA ≥6% and those with a %rPSA <6% groups (log-rank test, p<0.001).

The fPSA level has already been evaluated as a predictor of cancer-specific mortality [13]. Although the fPSA level was a predictor of cancer-specific mortality in the overall cohort with RP, it was not observed in men with persistent PSA levels after RP [13]. Therefore, the fPSA level has limited usefulness. We need to clarify the relationship between residual tumor cells in aggressive and benign cancers. The %rPSA reflects these characteristics. Therefore, it could be an ideal indicator for predicting metastatic progression.

A recent meta-analysis reported that approximately 12% of patients treated with RP experienced PSA persistence 4 to 8 weeks after RP [4]. A recent German study reported that 8.8% of patients had persistent PSA levels 6 weeks after RP [11]. The incidence of PSA persistence in our study was 14.9%. The reason for the increased incidence of these populations in our study is that our data included more aggressive cancers. The incidence of pGGG 4–5 in our study was 17.2% *vs.* 59.0% in patients with undetectable *vs.* persistent PSA levels after RP. However, those in the German data were 3.3% and 21.6%, respectively [11]. The incidence of pT3b in our study was 8.2% *vs.* 52.2% in patients with undetectable *vs.* persistent PSA levels after RP. However, the incidences in the German data were 8.1% *vs.* 45.2%, respectively [11]. In addition, we measured the PSA levels at 4 to 6 weeks after RP in most patients. In previous studies, PSA levels were measured 4 to 8 weeks after RP. Measuring PSA levels at an earlier time point may result in an insufficient reduction in the PSA level. Conversely, Bianchi et al [8] reported that the incidence of persistent PSA levels after RP was 26%. The reason for the high incidence of persistent PSA levels in that study was due to the high proportion of lymph node metastasis. In an earlier French study, 34.6% of their cohort showed persistently elevated PSA levels after RP [6].

Previous studies have demonstrated that the prognosis of men with PSA persistence is not invariably poor. Recently, long-term follow-up data of 11,604 patients who underwent RP were reported [11]. Fifteen years after RP, the MFS rate, overall survival, and cancer-specific survival were 53.0% *vs.* 93.2% (p<0.001), 64.7% *vs.* 81.2% (p<0.001), and 75.5% *vs.* 96.2% (p<0.001) for persistent *vs.* undetectable PSA levels, respectively [11]. In multivariable Cox regression models, a persistent level of PSA was an independent predictor of metastasis (HR, 3.59; p<0.001), death (HR, 1.86; p<0.001), and cancer-specific death (HR, 3.15; p<0.001) [11]. In a recent meta-analysis, PSA persistence was associated with BCR (HR, 4.44; 95% CI, 2.84–6.93), disease recurrence (HR, 3.43; 95% CI, 1.62–7.25), and cancer-specific mortality (HR, 2.32; 95% CI, 1.83–2.95) [4]. Our data showed concordant results that persistent PSA levels after RP was associated with MFS (HR, 3.94; p=0.010).

Additional treatment should be considered for patients with persistent levels of PSA after RP to control the residual tumor [8,11,13,14]. To select appropriate candidates for adjuvant or salvage treatment, additional risk factors must be considered, such as pT3b, pGGG 3–5, positive surgical margin, or pN1 disease [8,11,13,14]. Preisser et al [11] demonstrated that salvage radiation was associated with improved overall survival (HR, 0.37; p=0.02) and cancer-specific survival (HR, 0.12; p<0.01). Most studies support the benefit of salvage radiation in patients with persistent levels of PSA after RP [8,13,14]. Fossati et al [14] developed an accurate risk stratification tool to facilitate the individualized recommendation for eSRT based on PCa characteristics. Among patients with persistent levels of PSA after RP, eSRT administration provided better MFS only in patients with a Gleason score ≤7. Patients with a Gleason score ≥8 did not appear to benefit from eSRT, as these patients had a relatively constant high rate of metastases regardless of the PSA level when they received radiation.

The current study has several limitations. First, we included retrospective data, excluding patients from the analysis if complete information was not available. This may have created a selection bias. Second, the median follow-up duration was relatively short. Third, the proportion of patients with pelvic lymph node dissection was low in our institution. Finally, we used only computed tomography/magnetic resonance imaging and bone scan to measure the residual tumor after RP. Prostate-specific membrane antigen positron emission tomography is the most recommended test for patients with low PSA levels in these days [15].

CONCLUSIONS

Persistent levels of PSA were associated with worse MFS after RP. However, the fPSA level after RP was not a predictor of distant metastasis in patients with persistent levels of PSA. To our knowledge, this is the first study to demonstrate that %rPSA is an independent predictor of MFS in patients with persistent levels of PSA after RP. Further, in men with persistent levels of PSA after RP, biochemical residual tumors can be measured by %rPSA. Therefore, we hypothesize that %rPSA may improve the currently available prognostic models to help physicians decide whether to use a salvage treatments, such as radiation therapy, after RP for patients with PCa and scheduling follow-up patient counseling.

Conflict of Interest

The authors have nothing to disclose.

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None.

Author Contribution

Conceptualization: DBL, SWP. Data curation: DBL, JYK, WHS, JKN, HJL, TUK, SWP. Formal analysis: DBL, JYK, SWP. Funding acquisition: SWP. Investigation: DBL, JYK, SWP. Methodology: DBL, SWP. Project administration: TUK, SWP. Resources: SWP. Software: SWP. Supervision: SWP. Validation: SWP. Visualization: SWP. Writing – original draft: DBL, SWP. Writing – review & editing: DBL, TUK, SWP.

Data Sharing Statement

The data analyzed for this study have been deposited in HARVARD Dataverse and are available at https://doi.org/10.7910/DVN/9EJPB8.

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