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Prognostic Impact of Seminal Vesicle Mucosal Invasion in pT3b Prostate Cancer Following Radical Prostatectomy

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Purpose: The extent of seminal vesicle invasion (SVI) in prostate cancer can be classified into muscle layer invasion (SVI-muscle) and mucosal layer invasion (SVI-mucosa). This study aimed to evaluate the prognostic significance of the extent of SVI after radical prostatectomy.

Materials and Methods: SVI-mucosa data were prospectively collected since 2014. Among 1,659 radical prostatectomy specimens from 2014 to 2023, 259 cases (15.6%) with extraprostatic SVI were enrolled. A total of 252 cases with available follow-up data were included in the final analysis.

Results: SVI-mucosa was identified in 63 cases (25.0%), and SVI-muscle was identified in all 252 cases. Extracapsular extension was present in nearly all SVI cases (99.6%). The mean tumor volume percentage in final specimens was significantly higher in patients with SVI-mucosa (50%) compared to those without it (35%) ($p<0.001$). A high Gleason score (≥8) was more common in men with SVI-mucosa ($p=0.021$). Only 10 (5.3%) and 4 patients (6.3%) with and without SVI-mucosa, respectively, received adjuvant therapy ($p=0.544$). Biochemical recurrence-free survival did not significantly differ between men with SVI-mucosa and those with SVI-muscle alone (log-rank test, $p=0.309$). The 5-year metastasis-free survival and prostate cancer-specific survival rates were 86.0% vs. 91.6% ($p=0.654$) and 99.5% vs. 100% ($p=0.865$) in patients with and without SVI-mucosa, respectively.

Conclusion: The prognosis of patients with SVI is not uniformly poor. SVI-mucosa was associated with more aggressive pathological features. In most cases, SVI-muscle appears to develop first following extracapsular extension, with subsequent progression to SVI-mucosa. However, the presence of SVI-mucosa, an advanced status of SVI, did not significantly impact biochemical recurrence and metastasis-free survival rates.

Key Words: Prostate neoplasm, Radical prostatectomy, Seminal vesicle, Survival

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- **Conflicts of Interest:** The authors have nothing to disclose.

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INTRODUCTION

Prostate cancer continues to be one of the most common malignancies affecting men worldwide, with its prognosis largely determined by pathological findings following radical prostatectomy [1]. Among the various prognostic factors, seminal vesicle invasion (SVI) is widely recognized as an indicator of advanced disease and is associated with poorer oncological outcomes [2]. The reported frequency of SVI in radical prostatectomy specimens ranges from 4.5% to 17.9% and is associated with significantly higher mortality compared to pT3a disease [3-5]. However, SVI is not a uniform pathological entity; it includes different patterns and depths of invasion, such as mucosal and muscular involvement, which may carry distinct prognostic implications [4,6-9].

Seminal vesicle mucosal invasion (SVI-mucosa) represents a potential subset of SVI that warrants further investigation [6,10]. Based on current observations and limited evidence, SVI-mucosa is hypothesized to signify a more advanced stage of prostate cancer progression and may be associated with more aggressive tumor behavior [6,10]. While muscular invasion might reflect earlier local extension, SVI-mucosa could plausibly indicate deeper infiltration and a later stage of disease, potentially correlating with worse prognosis [4,6,11]. However, these assumptions remain speculative and require rigorous investigation to confirm their validity.

Despite its potential clinical relevance, the prognostic significance of SVI-mucosa has not been thoroughly investigated. Current staging and risk stratification models often overlook the differentiation between mucosal and muscular invasion, potentially leading to gaps in predicting patient outcomes. Addressing this gap, the present study seeks to explore the impact of SVI-mucosa on oncological outcomes, including disease progression and survival. By characterizing the unique prognostic role of SVI-mucosa, this research aims to contribute to a more nuanced understanding of SVI and enhance clinical decision-making processes for patients undergoing radical prostatectomy. Ultimately, these findings could help refine risk stratification, optimize therapeutic strategies, and improve personalized care for individuals with prostate cancer.

MATERIALS AND METHODS

This prospective cohort study was approved by the Institutional Review Board of the Pusan National University Yangsan Hospital (IRB No. 05-2016-007). After introducing the new parameter, SVI-mucosa, we established a radical prostatectomy database, prospectively collecting data from July 2014 to December 2023. Among 1,659 patients, 259 (15.6%) were identified as having SVI. After excluding cases with unavailable follow-up data, a total of 252 patients were included in the final analysis. Patients who underwent neoadjuvant/adjuvant hormonal or radiation therapy were excluded from the biochemical recurrence analysis. Open, pure laparoscopic, and robot-assisted laparoscopic radical prostatectomy were performed in 7 (2.8%), 14 (5.6%), and 231 men (91.7%), respectively. Standard pelvic lymph node dissection including external, internal, and obturator lymph node, was done in 214 men (84.9%) at radical prostatectomy.

Biochemical recurrence was defined as a single serum prostate-specific antigen (PSA) level of 0.2 ng/ml or greater [12]. Their PSA levels were checked every 3 months after surgery, and every 6 months thereafter. Clinical and pathological stages were determined according to the 2002 TNM classification [13]. Radical prostatectomy specimen samples were step-sectioned at a 4-mm thickness in a plane perpendicular to the urethra using a standardized processing protocol [14]. The apical and basal parts of the prostate were separately sectioned and examined in parallel slices. Tumor percentage was calculated by estimating number of blocks and tumor proportion of each slide, measuring 2 dimensions of tumor foci. The blocks containing the apical part and bladder base part regarded as 1/2 block. All specimens were evaluated by one experienced uropathologist (HJL).

SVI was defined as tumor invading the muscular wall or mucosa of the extraprostatic seminal vesicles. In all cases, the entire seminal vesicle was embedded and submitted by serial sectioning [8]. The extent of SVI was classified into SVI-muscle and SVI-mucosa. The percentage of tumor volume and the laterality of the involved prostate lobe were also evaluated.

Differences between subgroups (SVI-mucosa [–] vs. SVI-mucosa [+]) in age, preoperative PSA, pathologic Gleason sum, positive surgical margin, extracapsular extension,

lymph node metastasis, and percent of tumor volume were compared using the t-test for continuous variables and the chi-square test for categorical variables. Age at radical prostatectomy, preoperative PSA, and percentage of tumor volume were examined as continuous variables. Pathological Gleason scores (6–7, and ≥8), positive surgical margin, extracapsular extension, lymph node metastasis, and bi-SVI were examined as categorical variables. In men with pT3b tumors, we compared their biochemical recurrence-free survival rates according to SVI-mucosa using Kaplan-Meier estimates. Cox proportional hazard regression models for metastasis-free survival were used to determine the hazard ratio of additional pathologic and clinical features. All tests were 2-sided, with a $p<0.05$ considered statistically significant. SPSS ver. 17.0 (SPSS Inc., Chicago, IL, USA) was used for the statistical analyses.

RESULTS

Among pT3b patients, SVI-mucosa and SVI-muscle were involved in 63 (25.0%) and 252 (100%), respectively. Table 1 shows the demographic and pathological characteristics according to the SVI-mucosa, and comparisons of characteristics between subgroups (SVI-mucosa [+] vs. SVI-mucosa [–]). The mean follow-up duration was 46 months and the mean age for those with SVI was 69 years. In the entire cohort, lymph node metastasis, extracapsular extension, and positive surgical margin were found in 30 (11.9%), 251 (99.6%), and 129 (51.2%), respectively. Pathological Gleason score (p=0.021) and the percent of tumor volume (p<0.001) were significantly different between subgroups SVI-mucosa (+) and SVI-mucosa (–). The SVI-mucosa (+) subgroup had more aggressive tumor characteristics than did the SVI-mucosa (–) subgroup. However, no difference was observed in age, preoperative PSA, or extracapsular extension rate between subgroups.

In overall patients with SVI, the actuarial biochemical recurrence-free survival rates were 39.6% at 1 year, 18.9% at 3 years, and 12.6% at 5 years. Despite the high biochemical recurrence rate, metastasis-free survival remained relatively favorable, with rates of 94.8% at 1 year, 91.8% at 3 years, and 90.3% at 5 years. Moreover, the 5-year prostate cancer-specific survival rate was 99.6%. Fig. 1 shows the oncological outcomes after radical prostatectomy according to SVI-mucosa. The probabilities of biochemical recurrence-free survivals did not differ significantly between subgroups (SVI-mucosa [+] vs. SVI-mucosa [–] by log-rank test, p=0.309). Similarly, distant metastasis-free survivals and prostate cancer-specific survivals showed no significant differences between subgroups (SVI-mucosa [+] vs. SVI-mucosa [–] by log-rank test, p=0.654 and p=0.865, respectively).

Table 2 shows the predictive factors for biochemical

Table 1. Baseline characteristics according to the invasion of seminal vesicle mucosa layer in patients with seminal vesicle invasion (n=252)

Characteristic	SVI-mucosa (–) (n=189)	SVI-mucosa (+) (n=63)	p-value
Age (yr)	69 (65–74)	68 (65–71)	0.226
Surgery type			0.930
Open	5 (2.6)	2 (3.2)	
Laparoscopic	11 (5.8)	3 (4.8)	
Robotic	173 (91.5)	58 (92.1)	
Prostate volume (g)	34 (28–41)	37 (33–52)	0.004
PSA (ng/mL)	13.9 (8.2–25.7)	17.9 (10.4–32.7)	0.152
Positive surgical margin	84 (44.4)	45 (71.4)	<0.001
Extracapsular extension	188 (99.5)	63 (100)	0.589
Pathological GS			0.021
6–7	55 (29.1)	10 (15.9)	
8–10	134 (70.9)	53 (84.1)	
LNM status			0.126
pN0	138 (73.0)	46 (73.0)	
pN1	18 (9.5)	12 (15.8)	
pNx	37 (17.5)	5 (11.1)	
Tumor volume (%)	35 (22–50)	50 (34–80)	<0.001

Values are presented as median (interquartile range) or number (%).
SVI-mucosa, seminal vesicle mucosa invasion; PSA, prostate-specific antigen; GS, Gleason score; LNM, lymph node metastasis.

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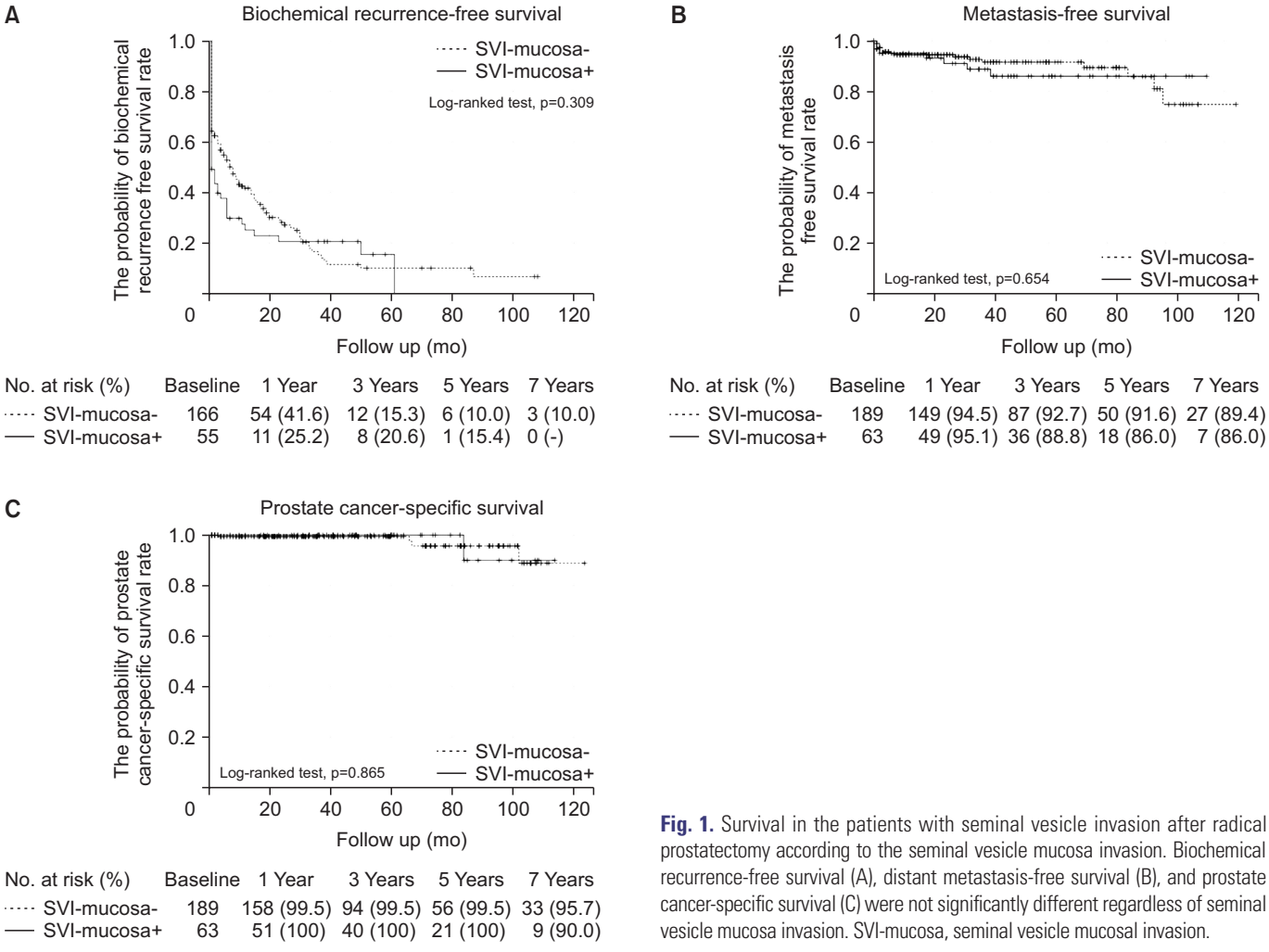


Fig. 1. Survival in the patients with seminal vesicle invasion after radical prostatectomy according to the seminal vesicle mucosa invasion. Biochemical recurrence-free survival (A), distant metastasis-free survival (B), and prostate cancer-specific survival (C) were not significantly different regardless of seminal vesicle mucosa invasion. SVI-mucosa, seminal vesicle mucosal invasion.

Table 2. Uni-/multivariable analysis for predictors of biochemical recurrence- and metastasis-free survival after radical prostatectomy

Variable	Univariate analysis			Multivariable analysis		
	HR	95% CI	p-value	HR	95% CI	p-value
Biochemical recurrence-free survival (n=221)						
SVI-mucosa	1.17	0.83–1.66	0.373	0.87	0.60–1.26	0.460
Age	1.00	0.98–1.03	0.922	1.01	0.99–1.04	0.461
PSA	1.02	1.01–1.03	<0.001	1.02	1.01–1.02	<0.001
PSM	1.35	0.99–1.82	0.055	1.34	0.96–1.89	0.086
Pathological GS (6–7 vs 8–10)	2.49	1.68–3.70	<0.001	2.45	1.61–3.71	<0.001
LNM	2.59	1.61–4.17	<0.001	1.96	1.19–3.22	<0.001
Metastasis-free survival (n=252)						
SVI-mucosa	1.13	0.51–2.51	0.758	1.06	0.46–0.24	0.893
Age	0.97	0.92–1.02	0.216	0.98	0.92–1.03	0.399
PSA	1.01	1.00–1.02	0.038	1.01	0.99–1.02	0.228
PSM	1.16	0.59–2.30	0.664	0.63	0.28–1.42	0.264
Pathological GS (6–7 vs 8–10)	7.75	2.33–25.77	0.001	5.68	1.64–19.65	0.006
LNM	5.65	2.85–11.20	<0.001	4.84	2.24–10.49	<0.001
Adjuvant RTx	0.60	0.14–2.50	0.480	0.90	0.21–3.90	0.892

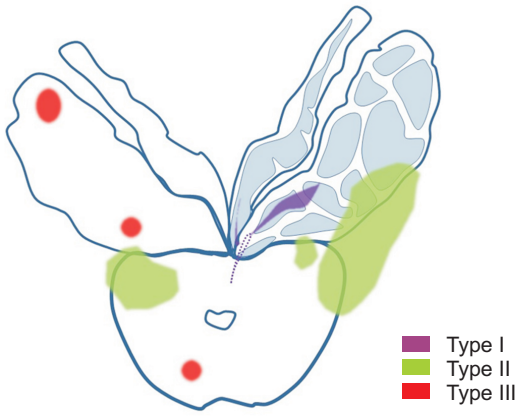
HR, hazard ratio; CI, confidence interval; SVI-mucosa, seminal vesicle mucosal invasion; PSA, prostate-specific antigen; PSM, positive surgical margin; GS, Gleason score; LNM, lymph node metastasis; RTx, radiation therapy.

recurrence-free survival and metastasis-free survival in men with SVI. Preoperative PSA (hazard ratio [HR], 1.02; 95% confidence interval [CI], 1.01–1.02; p<0.001), pathological Gleason score (HR, 2.45; 95% CI, 1.61–3.71; p<0.001), and lymph node metastasis (HR, 1.96; 95% CI, 1.61–3.71; p< 0.001) were independent predictive factors of biochemical recurrence following radical prostatectomy. In addition, pathological Gleason score (HR, 5.68; 95% CI, 1.64–19.65; p=0.006) and lymph node metastasis (HR, 4.84; 95% CI, 2.24–10.49; p<0.001) were independent predictors of distant metastasis following radical prostatectomy.

Fig. 2 illustrates the patterns of prostate cancer spread in patients with SVI and estimates the proportion of each SVI subtype. Tumor invasion typically progresses from extracapsular extension into the seminal vesicle muscle layer, followed by further infiltration into the mucosal layer.

DISCUSSION

Given the diverse progression pathways and prognoses of SVI by prostate cancer, further categorization is necessary



Ohori classification [9]	Type I	Type II		Type III
ECE	+	+	+	-
SVI-muscle	-	+	+	+
SVI-mucosa	+	-	+	-/+
No patients (%)	0	188 (74.6)	63 (25.0)	1 (0.3)

Fig. 2. The proportion of the patterns of seminal vesicle invasion according to Ohori et al. [9] classification. Most tumors invade the muscular layer of the seminal vesicles through extracapsular extension, followed by infiltration into the mucosal layer. Rarely, distant metastasis occurs without consecutive lesions. However, direct progression through the ejaculatory duct alone was not observed. ECE, extracapsular extension; SVI-muscle, seminal vesicle muscular invasion; SVI-mucosa, seminal vesicle mucosal invasion.

[2,4,15]. In this study, we classified SVI into SVI-mucosa and SVI-muscle to assess its extent. Notably, all cases exhibited SVI-muscle, and all but one had extracapsular extension, suggesting that prostate cancer typically invades the seminal vesicle muscle layer following extracapsular extension. The requirement for SVI to be accompanied by SVI-muscle is consistent with what Epstein et al. [8] reported. In contrast, SVI-mucosa was present in only a quarter of cases, suggesting it may represent a later stage of disease progression. In pT3b patients, SVI-mucosa was associated with a larger tumor burden and higher pathological Gleason scores compared to those without it. Despite these pathological differences, there was no significant impact of SVI-mucosa on biochemical recurrence-free survival, metastasis-free survival, or prostate cancer-specific mortality in our results.

The reported frequency of SVI varies widely, likely due to differences in patient selection for radical prostatectomy and inconsistencies in the definition of SVI. SVI is commonly defined as extraprostatic invasion of the seminal vesicles, requiring thorough examination of the entire seminal vesicle in the radical prostatectomy specimen [8,11]. Intraprostatic SVI was not associated with poor biochemical recurrence-free survival rate (HR, 1.1; 95% CI, 0.5–2.4; p=0.76), whereas extraprostatic SVI was associated with worse outcomes (HR, 1.7; 95% CI, 1.1–2.6; p=0.015) [6]. In addition, surgical artifacts may occasionally occur, potentially impacting the accuracy of diagnosis.

Ohori et al. [9] were the first to categorize the route of SVI, identifying 3 types: type 1, where invasion occurs along the ejaculatory duct; type 2, involving direct extension into the seminal vesicle from the prostate or through surrounding connective tissue; and type 3, which refers to discontinuous metastases to the seminal vesicle. Among those, type II invasion is the most common route, occurring in 61% of cases, and was characterized by tumor spread through the prostate capsule [9]. Subsequent studies by Epstein et al. [8], Billis et al. [7], and Galosi et al. [4] similarly found that transmission through the prostate capsule was the predominant route, observed in 88%, 89%, and 60.7% of SVI cases, respectively. Other pathways, such as spread via the ejaculatory ducts or discontinuous metastasis, were less frequent [4,7-9]. In addition, Miyai et al. [10] suggested that SVI-mucosa appears to result from direct invasion of the seminal vesicle muscle

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layer rather than spread through the ejaculatory duct system. These findings emphasize the importance of assessing the depth of invasion rather than the pathway of tumor spread. Given the limitations of pathological examination and the need for greater reproducibility, SVI-mucosa may be a more valuable prognostic parameter than the SVI route [10].

The association between SVI-mucosa and prognosis after radical prostatectomy is inconsistent [6,10]. Kristiansen et al. [6] identified SVI-mucosa in 49 of 60 patients (81.7%) with SVI, and it was associated with poor biochemical recurrence-free survival (HR, 4.2; 95% CI, 1.2–14.2; p=0.021) [6]. However, SVI route, surgical margin status, and local Gleason score were not associated with prognosis. However, Miyai et al. [10] identified SVI-mucosa in 18 of 109 cases (17%) of SVI, and it was not associated with biochemical recurrence-free survival. Similarly, our data shows that biochemical recurrence-free survival was not different in 63 of 252 SVIs (25%) with SVI-mucosa compared to those without it. While early postoperative progression was observed, it did not reach statistical significance. Predictors of biochemical recurrence included preoperative PSA, Gleason score, and lymph node metastasis, with the latter two also predicting distant metastasis in this study. Although this study, as well as almost all others, found that the SVI-mucosa was associated with adverse pathological features, oncological outcomes after radical prostatectomy were not different [6,10]. This may be due to the progression of SVI-mucosa being confined to the inner space of the seminal vesicle, a condition that may not be favorable condition for distant metastasis. However, this theory needs to be confirmed in larger studies.

Various efforts have been made to classify SVI, incorporating factors such as extracapsular extension and bilateral SVI in addition to SVI routes [15-19]. Mikel Hubanks et al. [20] reported significantly difference in systemic progression rate (HR, 1.56; p=0.006) and prostate cancer-specific mortality (HR, 1.71; p=0.01) between SVI with extracapsular extension and those without it. However, extracapsular extension is a common accompanying pathologic feature of SVI cases, with a frequency of 61%–89% [4,5,7,20]. In our study, initial diagnoses identified extracapsular extension in 230 out of 252 cases (91.3%), however, a repeated meticulous pathological review confirmed extracapsular extension in all but 1 case (99.6%). This suggests that SVI is almost always accompanied by extracapsular extension, if focal extracapsular extension is also considered. Epstein et al. [8] emphasized that the presence of SVI inherently indicates extra-prostatic extension, as the cancer is no longer confined to the prostate, regardless of whether it spreads through the ejaculatory duct.

Bilateral SVI has recently been recognized as a predictor of poorer postoperative outcomes compared to unilateral SVI [5,16,21-23]. In our previous study, bilateral SVI was identified in 37.6% (35 of 93) of pT3b patients and, along with preoperative PSA levels and lymph node metastasis, were independent predictors of biochemical recurrence (HR, 1.75; p=0.047) [5]. Similarly, other studies have reported that patients with bilateral SVI tend to have higher preoperative PSA levels, more advanced clinical T stages, and a greater incidence of adverse pathological features, including extracapsular extension, lymph node metastasis, and positive surgical margins, all of which contribute to worse oncological outcomes [16,21,23]. While most studies have suggested that bilateral SVI is an independent prognostic factor, conflicting finding has also been reported [5,21,22]. Bilateral SVI and SVI-mucosa are associated with larger tumor volumes, while tumor volume in the radical prostatectomy specimen is also associated with oncological outcomes [6,16,24]. Therefore, identifying which of these parameters serves as an independent prognostic factor is essential for improving risk stratification and treatment decisions.

This study has certain limitations and potential biases. The variability in the method and timing of adjuvant radiation complicates the assessment of treatment effects; however, this bias may be minimal, as the proportion of patients receiving radiation was similar between groups (5% vs. 6%). Additionally, the extent of SVI-mucosa and the Gleason score at the invasion site were not evaluated, which may have prognostic significance. Lastly, vas deferens invasion may occasionally coexist with SVI. Although it may have prognostic significance, its impact was not separately analyzed in this study. Despite these limitations, relatively large sample size and extended follow-up period strengthen its findings. Identifying and evaluating novel pathological and prognostic factors remain crucial and should be approached with caution in future research.

CONCLUSIONS

The prognosis for prostate cancer with SVI is not uniform. Mucosal invasion within the seminal vesicles may serve as a pathological indicator of disease progression. In this study, most prostate cancers first invaded the muscular layer of the seminal vesicle via prostate capsule before reaching the seminal vesicle mucosa. However, the invasion of seminal vesicle mucosal layer did not significantly affect the prognosis. Therefore, further studies should explore how to utilize this mechanism of SVI to subclassify pT3b prostate cancer.

NOTES

- **Author Contribution:** Conceptualization: SWP, HJL; Data curation: WHS, SSL, JKN; Formal analysis: SWP, HJL; Funding acquisition: SWP; Methodology: SWP, HJL; Project administration: SWP; Visualization: SWP, HJL; Writing - original draft: SWP, HJL; Writing - review & editing: SWP, HJL
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