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Research Article

Clinical implication of peri-seminal vesicle soft-tissue invasion in patients with pT3b prostate cancer

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ABSTRACT

Background: Prognosis of patients with prostate cancer seminal vesicle invasion (SVI) varies considerably, suggesting that SVI may be a heterogeneous pathological entity. This study aimed to perform a detailed histopathological analysis of seminal vesicle (SV) specimens in prostate cancer to better characterize SVI and evaluate its clinical significance.
Materials and methods: We retrospectively reviewed a database of robotic prostatectomies performed between July 2020 and December 2024. Since July 2020, a refined histopathological protocol has been employed in which the prostate and SVs are axially sectioned and separated, and the SVs and vas deferens are sectioned along their natural anatomical axis. This approach allowed a detailed assessment of SV stromal invasion, peri-SV soft-tissue involvement, and surgical margin status.
Results: We identified 73 patients with pT3b prostate cancer. Unilateral SVI was present in 40 patients (54.8%) and bilateral SVI in 33 patients (45.2%). Notably, peri-SV soft-tissue invasion was observed in 54 patients (74.0%), with it being unilateral in 28 (38.4%) and bilateral in 26 (35.6%). Positive surgical margins in the peri-SV soft-tissue were found in five patients (9.3%). Among 58 patients who underwent pelvic lymph node dissection without preoperative androgen deprivation therapy, 25 (43.1%) had lymph node metastases. On multivariate analysis, lymph node metastasis was significantly associated with lymphovascular invasion only, but not with peri-SV soft-tissue invasion.
Conclusion: In pT3b patients, peri-SV soft-tissue invasion and lymph node metastasis are common. These findings suggest that a more radical surgical approach is warranted in patients with suspected SVI.

1. Introduction

Prostate cancer (PCa) with seminal vesicle invasion (SVI) is considered a high-risk feature according to the National Comprehensive Cancer Network (NCCN) guidelines and is recognized as a poor prognostic factor.[1] However, the reported 5-year biochemical recurrence (BCR)-free survival rates of patients with SVI vary widely across studies, ranging from 5% to 60%.[2,3] This variability suggests that SVI may not be a uniform pathological entity and can potentially be subcategorized.

Previous studies have attempted to identify the different mechanisms of cancer spread in seminal vesicles (SVs) and stratify patients with SVI. Ohori et al suggested three types of SVI. They defined type I SVI as direct spread along the lumen of the ejaculatory duct and type III SVI as isolated tumor foci in the SVs without involvement of the ejaculatory duct complex or the presence of an adjacent extraprostatic tumor. They found that tumors exhibiting type I invasion were associated with worse prognoses than those exhibiting type III invasion.[4] In contrast, Galosi et al reported that patients with type III invasion experienced a shorter time to BCR than those with other patterns.[5]

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Debras et al and Fukunaga et al focused on the extent of SVI,[6,7] and both studies concluded that the extent of SVI was an independent prognostic factor for BCR, despite using different definitions. More recently, bilateral SVI has been found to be strongly associated with adverse histopathological features of radical prostatectomy and poor prognosis,[8,9] although this remains controversial.[10]

Despite substantial efforts to investigate the prognostic value of the SVI pattern and extent, no consensus has been reached regarding the subclassification of T3b PCa. Notably, there is no standardized method for sampling or analyzing SVI. According to the 2011 International Society of Urological Pathology (ISUP) consensus, 38% of respondents reported performing complete SV sampling, 52% conducted selective sampling, and 10% adapted their approach based on individual cases. This inconsistency underscores the need for a standardized approach for evaluating SVI.[11]

We implemented a novel pathological analysis method to precisely assess SVI. In this approach, the prostate and SV are axially divided to distinguish extraprostatic extension (EPE) and peri-SV soft-tissue invasion as separate pathological features. The SVs and vas deferens were thoroughly examined by sectioning along their natural anatomical axes, which enabled a detailed assessment of the surrounding tissues. This approach allowed a detailed assessment of SV stromal invasion, peri-SV soft-tissue involvement, and surgical margin status. Here, we present our early experience with this approach to better characterize the extent and pattern of SVI and evaluate its clinical significance.

2. Materials and methods

2.1. Study population and design

All studies were conducted in accordance with relevant guidelines and regulations. The requirement for written informed consent was waived due to the retrospective design of the study (Gangnam Severance Hospital Institutional Review Board, IRB No. 3-2025-0055, Approval date: April 21st, 2025). This study was approved by the Gangnam Severance Hospital Institutional Review Board after reviewing the study protocol and was conducted in accordance with the Declaration of Helsinki. We retrospectively reviewed the database of 351 robot-assisted radical prostatectomy (RARP) procedures performed at Gangnam Severance Hospital, a tertiary hospital, between July 2020 and December 2024. All the RARP procedures were performed by a single surgeon, in a conventional manner, using the da Vinci Xi surgical system. For patients classified as unfavorable intermediate-, high-, and very high-risk according to the NCCN risk group, an extended pelvic lymph node dissection (PLND) was performed. After excluding 276 patients who had pT3a or lower disease, one patient who had undergone salvage RARP following definitive radiotherapy, and one patient with oligometastatic disease, a total of 73 patients with pT3b PCa were included in this analysis. A detailed patient selection flowchart is provided in the Supplementary Fig. 1.

2.2. Preoperative clinical and pathological data collection

Basic clinical information including sex and age at the time of surgery was collected. Clinical data such as initial prostate-specific antigen (PSA) levels, preoperative androgen deprivation therapy (ADT) status, clinical TNM stage, biopsy Gleason scores, and NCCN risk group classification were reviewed. Biopsy Gleason scores were converted to the corresponding ISUP Gleason grade groups (GGGs). Preoperative imaging, including prostate magnetic resonance imaging, abdomen–pelvis and chest computed tomography, and whole-body bone scans, were also reviewed to determine the clinical TNM staging.

2.3. Refined histopathological analysis and margin definition

A single uropathologist evaluated all specimens and reported the findings in accordance with the protocol of the College of American Pathologists. This method is illustrated in Fig. 1. Prostate specimens were fixed in formalin for at least 24 h. After fixation, the base of the prostate and the SV were cut axially and separated. The apex and base of the prostate were then vertically sectioned at 2–3 mm intervals, and the remaining prostate was serially sectioned perpendicular to the posterior surface from apex to base at approximately 3 mm intervals. All the sections were embedded in paraffin and prepared as slides. EPE was defined as tumor infiltration beyond the prostate capsule into the surrounding soft tissue of a prostate specimen.

For the SV analysis, the vas deferens and SV were cut along the midline sagittal plane to separate the left and right SVs, followed by up to four coronal cuts aligned with the SV axis. This method allows a thorough inspection of the SV and peri-SV soft tissues because the cutting plane entirely encompasses the SV, ductal structures, and surrounding adipose tissue. Peri-SV soft-tissue involvement was defined as tumor infiltration into the soft tissue surrounding the SV in the specimen. Pathological evaluation included SV stromal involvement, peri-SV soft-tissue involvement, vas deferens invasion (VDI), and surgical margin status of the vas deferens and peri-SV soft tissue. The gross and microscopic appearances of the representative cases are shown in Fig. 2. For lymph nodes (LNs), the total number retrieved, number of involved LNs, and maximal diameter of LN metastases were recorded.

2.4. Postoperative data collection and statistical analysis

The postoperative variables included pathological GGG level, tumor volume, pathological TNM stage, EPE, lymphovascular invasion (LVI), perineural invasion, prostatic intraepithelial neoplasia, SVI, VDI, peri-SV soft-tissue tumor infiltration, surgical margin status, and LN metastasis. Postoperative PSA nadir values were also recorded. Associations between clinical and pathological outcomes were assessed using the Fisher's exact test as well as univariate and multivariate logistic regression analyses. Statistical significance was defined as $P < 0.05$. Statistical analyses were performed using SAS System for Windows, version 9.4 (SAS Institute Inc., Cary, NC, USA).

3. Results

3.1. Preoperative clinical and pathological features

In total, 73 patients who underwent RARP and were diagnosed with pT3b PCa were identified. The mean age was 67.8 years (range: 54–78 years), with an initial PSA level of 23.31 ng/mL (range: 3.60–127.0 ng/mL) (Table 1). Preoperatively, 35 patients (47.9%) were staged as cT3b, whereas 38 (52.0%) had cT3a or lower. Regarding the prostate biopsy specimens, 64 patients (68.5%) had GGG of 4 or 5. According to the NCCN risk group classification, 64 patients (87.7%) were classified as high risk or above. Seven patients (9.6%) received preoperative ADT, and the remaining 66 (90.4%) were treatment-naïve.

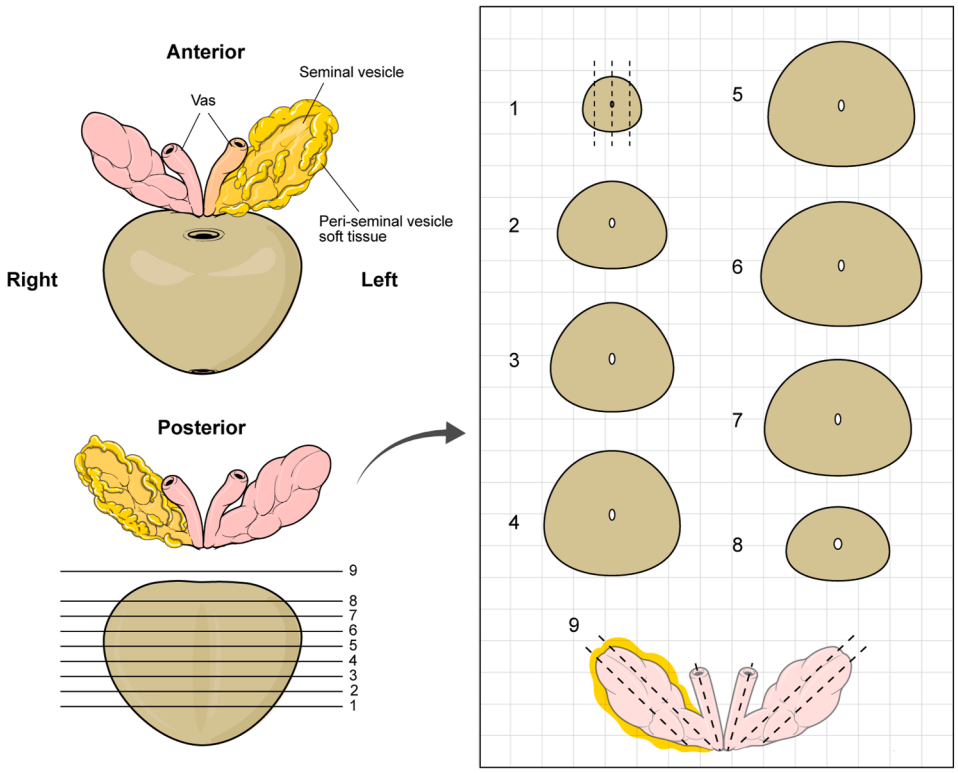

Fig. 1. Schematic display of our refined pathological analysis method for the prostate and seminal vesicle (SV) evaluation. The anterior and posterior views are shown. An illustration of the peri-SV tissue is provided for the left SV. The prostate was axially and serially sectioned at 3 mm intervals (nos. 1–8), with the total number of slices depending on the size of the prostate. Eight transverse slices are presented as illustrative examples. An additional axial section (no. 9) was made at the base of the prostate to separate it from the vas deferens and SVs. Standard pathological examination was performed to assess intraprostatic cancer and extraprostatic extension. For the SV analysis, the vas deferens and SV were sectioned longitudinally along their axes. Cancer infiltration into the vas deferens, SV, and surrounding peri-SV tissues was evaluated. SV: seminal vesicle.

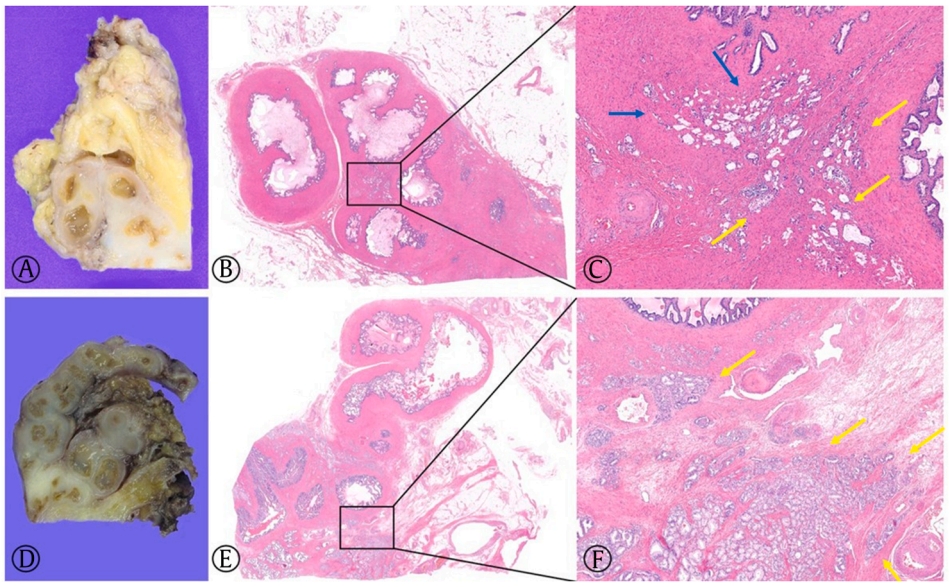

Fig. 2. Identifying peri-seminal vesicle tumor involvement using coronal sectioning along the seminal vesicle axis. Panels A, B, and C are from the same patient, whereas panels D, E, and F are from a different patient. A and D show gross coronal sections aligned with the SV axis, including the SVs, their ducts, and surrounding peri-SV tissues, thereby preserving the continuity of the adjacent structures. B, C, E, and F show the H&E-stained sections. Upon magnification of B and E, panels C and F, respectively, revealed peri-SV soft-tissue tumor involvement outside the SV muscularis, demarcated with yellow arrows. In panel C, blue arrows indicate intra-SV muscularis invasion of prostate cancer. H&E: hematoxylin and eosin; SV, seminal vesicle.

Table 1
Summary of preoperative clinical and pathological features in the entire cohort.

	N (%)
Total number of patients	73 (100)
Age at surgery (years)	67.8 (54–78)[a]
<65	21 (28.8)
≥65	52 (71.2)
Initial PSA (ng/mL)	23.31 (3.60–127.00)[a]
<20	45 (61.6)
≥20	28 (38.4)
Preoperative ADT	7 (9.6)
Clinical T stage	
cT1	1 (1.4)
cT2	14 (19.2)
cT3a	23 (31.5)
cT3b	35 (47.9)
Clinical N stage	
cN0	69 (94.5)
cN1	4 (5.5)
Biopsy gleason grade group	
Grade group 1	3 (4.1)
Grade group 2	9 (12.3)
Grade group 3	11 (15.1)
Grade group 4	21 (28.8)
Grade group 5	29 (39.7)
NCCN risk groups	
Favorable intermediate risk group	7 (9.6)
Unfavorable intermediate risk group	2 (2.7)
High-risk group	25 (34.2)
Very high-risk group	35 (47.9)
Regional risk group	4 (5.5)

ADT, androgen deprivation therapy; NCCN, National Comprehensive Cancer Network; PSA, prostate-specific antigen.
[a] Data presented as mean (range).

3.2. Pathological analysis for SVs and their surrounding tissues

Radical prostatectomy specimens revealed that 40 patients (54.8%) had GGG 4 or 5, and EPE was observed in 60 patients (82.2%). A total of 32 patients (43.8%) had positive surgical margins (PSMs) (Table 2).

Regarding SVs and their surrounding tissues, unilateral SVI was present in 40 patients (54.8%) and bilateral SVI in 33 patients (45.2%). Notably, peri-SV soft-tissue invasion was observed in 54 patients (74.0%), with unilateral involvement in 28 (38.4%) and bilateral involvement in 26 (35.6%). PSMs in the peri-SV soft tissue were found in five patients (9.3%). VDI was present in 25 patients (34.2%), with 11 cases (15.0%) unilateral and 14 cases (19.2%) bilateral.

3.3. Pathologic outcomes for PLND

Of the 73 patients, extended PLND was performed in 65 patients. After excluding seven patients who had received preoperative ADT, 58 were included in the analysis for relationship between clinicopathological features and lymph node involvement (Supplementary Fig. 1). LN metastasis was identified in 25 (43.1%) patients (Table 3). Among these, three patients (12.0%) had preoperative clinical N1 staging, while the remaining 22 (88.0%) had no evidence of LN metastasis on preoperative imaging. The maximum diameter of LN metastases was less than 10 mm in 20 patients (80.0%) and less than 5 mm in 12 patients (60.0%). We performed a univariate logistic regression analysis and found out that GGG 4 or 5, LVI, and VDI were significantly associated with LN metastasis ($P = 0.011$, $P < 0.001$, and $P = 0.024$, respectively). Peri-SV soft-tissue invasion was also significantly associated with LN metastasis ($P = 0.043$) (Table 3). However, a multivariate logistic regression analysis revealed that only LVI was significantly associated with LN metastasis (odd ratio = 44.30, 95% confidence interval: 3.79–517.76; $P = 0.003$) (Table 4).

Table 2
Summary of pathological features of radical prostatectomy in the entire cohort.

	N (%)
Total number of patients	73 (100)
Pathologic gleason grade group	
Grade group 2	17 (23.3)
Grade group 3	16 (21.9)
Grade group 4	18 (24.7)
Grade group 5	22 (30.1)
Tumor volume (mL)	7.1 (0.49-52.5)[a]
<5	39 (53.4)
≥5	34 (46.6)
Extracapsular extension	60 (82.2)
Seminal vesicle invasion	73 (100.0)
Unilateral	40 (54.8)
Bilateral	33 (45.2)
Peri-seminal vesicle soft-tissue invasion	54 (74.0)
Unilateral	28 (38.4)
Bilateral	26 (35.6)
Vas deferens invasion	25 (34.2)
Unilateral	11 (15.0)
Bilateral	14 (19.2)
Lymphadenectomy	65 (89.0)
No. of lymph node(s) harvested	15.46 (3–34)[a]
<10	7 (10.8)
≥10, <20	43 (66.2)
≥20	15 (23.0)
No. of lymph node(s) involved	1.06 (0–16)[a]
0	39 (60.0)
1	11 (16.9)
2	8 (12.3)
≥3	7 (10.8)
Maximum diameter of involved lymph node	7.37 (0.5–21.4)
<5 mm	12 (46.2)
≥5, <10 mm	7 (26.9)
≥10, <20 mm	5 (19.2)
≥20 mm	2 (7.7)
Lymphovascular invasion	45 (61.6)
Perineural invasion	72 (98.6)
Prostatic intraepithelial neoplasia	37 (50.7)
Positive surgical margin	32 (43.8)
Location of positive surgical margin	
Apex	14 (19.2)
Base	18 (24.7)
Circumferential	7 (9.6)
Peri-seminal vesicle soft tissue	5 (6.8)
Postoperative PSA nadir	
<0.1 ng/mL	52 (71.2)[b]
≥0.1, <0.2 ng/mL	5 (6.8)
≥0.2, <1 ng/mL	10 (13.7)
≥1 ng/mL	6 (8.3)

PSA, prostate-specific antigen.
[a] Data presented as mean (range).
[b] Four patients who underwent preoperative androgen deprivation therapy were included.

4. Discussion

In this study, we introduced a novel method for the pathological analysis of SVs, peri-SV tissues, and the vas deferens in radical prostatectomy specimens. Although the conventional analysis of prostatectomy specimens typically reports only the presence or absence of SVI, we assessed and reported both SVI and peri-SV tissue invasion (Fig. 2). Peri-SV tissue invasion has traditionally been either unreported or considered a form of EPE to avoid ambiguity. However, in our study, we identified and described peri-SV tissue invasion as a distinct entity with the aim of investigating its clinical significance. Kristiansen et al reported that 50 of 60 patients (83.3%) had tumor invasion of the connective tissue surrounding the SVs.[12] This aligns closely with our findings, in which 54 of 73 patients (74.0%) exhibited peri-SV tissue invasion. Despite our efforts to perform an extended resection including as much peri-SV fat tissue as possible in patients suspected of having T3b

Table 3
Univariate analysis for relationship between clinicopathological features and lymph node involvement.

Features	Lymph node involvement (N = 58) (%)		*P* value[a]
	Absence (N = 33)	Presence (N = 25)	
Age >65 years	24 (72.7)	18 (72.0)	0.951
PSA >20 ng/mL	9 (27.3)	13 (52.0)	0.055
Pathologic gleason grade group ≥4	14 (42.4)	19 (76.0)	0.011
Tumor volume >5 mL	14 (42.4)	16 (64.0)	0.103
Extracapsular extension	29 (87.9)	21 (84.0)	0.671
Perineural invasion	32 (97.0)	25 (100.0)	0.380
Prostatic intraepithelial neoplasm	16 (48.5)	12 (48.0)	0.971
Lymphovascular invasion	13 (39.4)	24 (96.0)	<0.001
Bilateral seminal vesicle invasion	11 (33.3)	14 (56.0)	1
Peri-seminal vesicle soft-tissue invasion			0.043
None	7 (21.2)	6 (24.0)	
Unilateral	18 (54.5)	6 (24.0)	
Bilateral	8 (24.2)	13 (52.0)	
Vas deferens invasion			0.024
None	26 (78.8)	11 (44.0)	
Unilateral	3 (9.1)	6 (24.0)	
Bilateral	4 (12.1)	8 (32.0)	
Positive surgical margin	13 (39.4)	15 (60.0)	0.120

PSA, prostate-specific antigen.
[a] By Fisher's exact test.

Table 4
Multivariate logistic regression analysis for relationship between clinicopathological features and lymph node involvement (N = 58).

	Odds ratio	95 % confidence intervals	*P* value
Pathologic gleason grade group (≥4 vs. < 4)	4.94	0.84–29.02	0.077
Lymphovascular invasion (yes vs. no)	44.30	3.79–517.76	0.003
Peri-seminal vesicle soft-tissue invasion			
Unilateral vs. none	0.12	0.01–1.31	0.082
Bilateral vs. none	0.19	0.01–1.75	0.117
Vas deferens invasion			
Unilateral vs. none	4.93	0.41–58.87	0.207
Bilateral vs. none	7.80	0.64–94.79	0.107
PSA (≥20 vs. <20 ng/mL)	4.86	0.85–27.79	0.076

PSA, prostate-specific antigen; SV, seminal vesicle.

PCa, PSMs in the peri-SV tissues were identified in five patients (9.3%). This finding suggests the presence of residual cancer cells in the tumor bed. These findings underscore the potential need for wider meticulous resection that includes peri-SV tissue during SV excision in patients with T3b PCa to improve local cancer control.

Attempts have been made to seek a deeper understanding of cancer-spreading mechanisms within SVs. Ohori et al defined type III SVI as isolated tumor foci in SVs without the involvement of the ejaculatory duct complex or adjacent extracapsular tumors. Among the 64 patients with SVI, eight (12.5%) were classified as type III, suggesting that 87.5% had prostatic base involvement adjacent to the SV.[4] Koh et al found that both the presence and quantity of cancer in prostate-based biopsy samples correlated with postoperative SVI.[13] These findings suggest that most SVI originate from the base of the prostate and extend into the muscularis SV. Several studies have attempted to subclassify pT3b patients with PCa into risk groups. Suh et al analyzed 770 patients with pT3b PCa and found that bilateral SVI was an independent risk factor for BCR (hazard ratio, 1.197; $P = 0.049$), clinical progression ($P = 0.022$), and cancer-specific survival ($P = 0.038$) in a covariate-adjusted Cox regression analysis.[14] Numbere et al reported that bilateral SVI was associated with worse pathological features and poorer prognosis.[8] Rehman et al analyzed 69 patients and found that the BCR rate was 33.9% in those with combined EPE and SVI, compared with 12.5% in those with SVI alone (relative risk = 2.71). They further subdivided pT3 PCa into three groups: EPE alone (pT3a), SVI alone (pT3b), and a combination of EPE and SVI (pT3c).[15] Similarly, Fukunaga et al identified the extent of SVI as an independent risk factor for BCR in multivariate analysis.[7]

Given the close anatomical relationship between the vas deferens and seminal vesicles, the presence of VDI was also inspected in previous studies regarding pT3b patients. Jang et al retrospectively reviewed 350 patients with pT3b PCa and found that 87 (24.9%) had VDI, whereas the remaining 263 (75.1%) did not. They identified VDI as an independent risk factor for BCR.[16] Among the 73 patients with pT3b PCa included in the analysis, VDI was observed in 25 (34.2%). Of the 58 patients included in the analysis of LN involvement, 21 (36.2%) had VDI and LN metastasis was identified in 14 (66.7%). These findings suggest that a thorough pathological evaluation of peri-SV tissues, including the vas deferens, may warrant further stratification of the risk groups within the current pT3b classification.

In our study, pathologically proven LN metastasis was identified in approximately 40% of the cases. Among these, the maximum diameter of the LN metastases was less than 10 mm in 73% of cases and less than 5 mm in 46% of cases. Similarly, Falkenbach et al reviewed 2,705 patients and reported that the median LN metastasis size was 4.5 mm;of the 7,510 LN metastases, 1,966 (26%) were micrometastases (≤2 mm).[17] These findings reaffirm that pT3b patients have a higher likelihood of concurrent LN metastasis, which are usually difficult to diagnose using conventional imaging due to their small size and micrometastasis.[18] In fact, a minimum of 4.9 mm for a lymph node is required for a detection rate of 90% by prostate-specific membrane antigen positron emission tomography/computed tomography.[19] Therefore, the decision to perform lymphadenectomy should not be based solely on imaging but should also incorporate risk stratification, nomograms, and clinical judgment. Meanwhile, Pessoa et al reviewed 2,043 patients who underwent radical prostatectomy and identified with SVI, and found out that the presence of two or more metastatic pelvic LNs and a 20% LN density cut off was independently associated with worse metastasis-free survival, cancer-specific survival, and overall survival.[20]

From an anatomical perspective, the clinical relevance of the peri-SV region in patients with PCa may be explained by lymphatic drainage pathways of the prostate. Lymphatic drainage from the superior part of the prostate flows to the medial border of the SVs and above the insertion of the ureters and then passes laterally over the umbilical artery to terminate in the middle prevenous nodes. Additional lymphatics originate in front of or behind the SVs. The inferior portion of the prostate drains along the posterior surface of the gland to the base, follows the prostatic branches of the middle hemorrhoidal artery, and terminates at the hypogastric nodes. For the posterior prostate, the drainage travels along the rectovesical fascia and terminates at the parasacral nodes at level S2 near the sacral promontory.[21] In addition, recent studies have suggested that periprostatic adipose tissue may be associated with the aggressiveness of PCa.[22–25] In this context, we initially hypothesized that peri-SV soft-tissue invasion would be strongly

associated with LN metastasis. Indeed, univariate analysis demonstrated a significant association between peri-SV soft-tissue invasion and LN metastasis (Table 3). However, in multivariate analysis, peri-SV invasion did not remain an independent predictor of LN metastasis. Nevertheless, in patients with suspected SVI, a more aggressive surgical approach that includes wide resection of the seminal vesicle along with the adjacent peri-SV soft tissue may be warranted to ensure optimal locoregional control.

This study has several limitations. First, the small sample size may have affected the generalizability of our findings. Additionally, most patients had treatment-naïve PCa; therefore, the outcomes may differ among patients undergoing neoadjuvant therapies. Further research is needed to determine whether peri-SV tissue positivity is correlated with the long-term prognosis of patients with PCa. However, a key strength of our study is the consistency of the data, as all surgeries were performed by a single surgeon and all pathology reviews were conducted by a single pathologist. Similar studies in this field often involve relatively small patient populations. Although the cohort is relatively large compared with other pT3b studies, the data are very recent, and therefore we plan to continue follow-up and provide data on BCR and other survival analyses in future studies. Further validation using larger multicenter studies is necessary to confirm our findings. In summary, our standardized method for assessing seminal vesicle invasion should be universally adopted to ensure consistent pathological results and to generate high-quality, clinically valuable data for future studies.

5. Conclusion

In pT3b patients, peri-SV soft-tissue invasion and lymph node metastasis are common. These findings suggest that a more radical surgical approach is warranted in patients with suspected SVI. The histopathological approach to SV specimens proposed in this study appears to improve the delineation of the SVI pattern and extent. Further large-scale, long-term studies are necessary to validate the utility of this novel methodological approach and analyze the impact of peri-SV soft-tissue invasion on oncological outcomes.

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Presentation

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Conflicts of interest

All authors have no conflict of interest to declare.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.prnil.2025.06.005.

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