

Prognosis of Prostate Cancer With Mucinous Components: A Propensity Score-Matched Study

Jin Hyeok Choi¹, Hyunho Han¹, Jongsoo Lee¹, Won Sik Jang¹, Won Sik Ham¹, Nam Hoon Cho², Young Deuk Choi¹, Ji Eun Heo¹

¹Department of Urology, Yonsei University College of Medicine, Seoul, Korea

²Department of Pathology, Yonsei University College of Medicine, Seoul, Korea

Received June 11, 2025
Revised September 23, 2025
Accepted October 10, 2025

Corresponding author:

Ji Eun Heo
Department of Urology, Yonsei University College of Medicine, 50-1, Yonsei-ro, Seodaemun-gu, Seoul 03722, Korea
Email: HE0JI87@yuhs.ac
<https://orcid.org/0000-0002-4184-8468>

Purpose: Acinar adenocarcinoma with mucinous components is a rare histologic variant of prostate cancer (PC) that was previously reported to exhibit more aggressive behavior than typical PC. However, recent studies have suggested that PC with mucinous components may not be more aggressive and could even have a more favorable prognosis. Therefore, this study investigated the clinical outcomes of PC with mucinous components.

Materials and Methods: We reviewed 7,983 patients with PC who underwent radical prostatectomy between 2006 and 2019. Propensity score matching and Kaplan-Meier analyses were performed to compare outcomes between patients with typical PC (group 1) and those with PC containing mucinous components (group 2). Matching variables included age, initial prostate-specific antigen level, clinical stage, and pathological Gleason score (GS). Biochemical recurrence-free survival (BCRFS) and cancer-specific survival (CSS) were analyzed using Cox regression analysis to identify survival predictors.

Results: Sixty-one patients (0.76%) had PC with mucinous components. No significant differences were observed in matched variables between the 2 groups. Pathological stage, lymph node invasion (LNI), and positive surgical margin rates were also comparable. At a median follow-up of 53 (interquartile range, 24–80) months, biochemical recurrence occurred in 29 patients in group 1 and 24 in group 2 ($p=0.361$). Two patients in group 1 and 3 in group 2 died from PC ($p>0.999$). BCRFS and CSS did not differ significantly between the 2 groups ($p=0.676$ and $p=0.458$, respectively). High GS (≥ 8) ($p=0.007$) and pT3b stage ($p=0.035$) were independent risk factors for BCRFS, while LNI ($p=0.001$) predicted CSS. The presence of mucinous components was not a significant predictor of either BCRFS or CSS ($p=0.127$ and $p=0.561$, respectively).

Conclusion: PC with mucinous components demonstrated clinical outcomes comparable to those of typical PC and was not an independent prognostic factor for survival. PC with mucinous components may not be as aggressive as previously believed.

Key Words: Mucinous adenocarcinoma, Prostatic neoplasms, Survival

- **Grant/Fund Support:** This study was supported by a faculty research grant from the Yonsei University College of Medicine (6-2021-0097).
- **Research Ethics:** This retrospective study was reviewed and approved by the Institutional Review Board (IRB) of the Yonsei University Severance Hospital (IRB number: 4-2020-1279).
- **Conflicts of Interest:** The authors have nothing to disclose.

INTRODUCTION

Mucinous adenocarcinoma refers to an unusual histological pattern of prostatic acinar adenocarcinoma in which more than 25% mucin components are present, whereas cases with less than 25% mucin components are defined as adenocarcinomas with mucinous features [1,2]. While mucinous adenocarcinoma accounts for only 0.2%–0.4% of all prostate cancers (PCs) [1,3-7], it is known to exhibit aggressive behavior and have a poor prognosis compared to typical PC [8-10]. In 2005, the International Society of Urological Pathology (ISUP) suggested that mucinous adenocarcinoma should be classified as Gleason pattern 4, and that these cases should have a Gleason score (GS) of 7 or higher [11].

However, recent studies have shown that mucinous adenocarcinomas do not exhibit more aggressive behavior or poorer prognosis compared with typical PC [1,2,12,13]. In 2014, the ISUP recommended that the grading of mucinous carcinoma of the prostate should be based on its underlying growth pattern, rather than grading them all as Gleason pattern 4 by default [14]. Additionally, the 25% threshold used to distinguish mucinous adenocarcinomas from adenocarcinomas with mucinous features remains a subject of debate. This cutoff was arbitrarily established in 1985 based on the criteria for mucinous carcinomas of the breast or digestive tract [8]. Recent reports have indicated no significant differences in oncological outcomes between tumors with $\geq 25\%$ mucinous components and those with $< 25\%$ mucinous features [2,12].

In South Korea, only a few case reports have documented this rare pattern of PC [15-18]. In this study, we aimed to evaluate the incidence, clinicopathological characteristics, and oncological outcomes in 61 patients with mucinous adenocarcinoma treated at our tertiary center. Our study represents the largest reported cohort of patients with mucinous adenocarcinoma from South Korea.

MATERIALS AND METHODS

This retrospective study was reviewed and approved by the Institutional Review Board (IRB) of the Yonsei University Severance Hospital (IRB number: 4-2020-1279).

The requirement for informed consent was waived because of the retrospective nature of the study and the use of anonymous clinical data. We reviewed the data of 7,983 patients who underwent radical prostatectomy (RP) for PC at our institution between January 2006 and December 2019. Patients who received neoadjuvant therapy, were not diagnosed with acinar adenocarcinoma, had patterns or subtypes other than mucinous components, or had incomplete clinicopathological or follow-up data were excluded.

Mucinous adenocarcinoma was defined as prostatic acinar adenocarcinoma with more than 25% mucinous component (n=61, group 2). For comparison with typical PC, defined as prostatic acinar adenocarcinoma without any patterns or subtypes (group 1), nearest-neighbor 1:1 propensity score matching (PSM) was performed to balance the potential confounding variables and minimize selection bias. Propensity scores were calculated based on preoperative covariates, including age, initial prostate-specific antigen (PSA), clinical stage, and pathological GS, using multivariate logistic regression analysis. The balance between the 2 groups was assessed using absolute standardized differences before and after matching. After matching, the balance of baseline covariates was evaluated using absolute standardized mean differences (SMDs).

Biochemical recurrence (BCR) was defined as detectable PSA after RP or any 2 consecutive increases of ≥ 0.2 ng/mL in PSA levels with undetectable PSA after RP [19]. Biochemical recurrence-free survival (BCRFS) was defined as the time from RP to BCR. Data on mortality and cause of death were collected from medical records in the Cancer Registry Center database at our institution.

Continuous variables are expressed as medians (interquartile ranges), whereas categorical variables are reported as the number of occurrences and frequency. The Student t-test was used to compare continuous variables, whereas the chi-square test was used to compare categorical variables. We analyzed BCRFS and cancer-specific survival (CSS). Kaplan-Meier survival analysis was used to estimate survival curves. Cox regression analysis was used to evaluate the predictors of survival. The level of significance was set at 0.05. All statistical analyses were performed using IBM SPSS Statistics ver. 26.0 (IBM Co., USA).

RESULTS

A total of 61 patients with typical PC and 61 patients with mucinous PC were included in the analysis after PSM. The clinicopathological characteristics of the 2 groups after matching are summarized in Table 1, which includes p-values and SMD for balance assessment. After matching, the median patient age (67 years vs. 66 years, $p=0.522$) and median serum PSA level (10.17 ng/mL vs. 8.41 ng/mL, $p=0.064$) were comparable between the 2 groups. Furthermore, no significant differences were found in the clinical T stage ($p=0.580$), clinical N stage, ($p>0.999$), clinical M stage ($p>$

0.999) or pathological GS ($p=0.090$), confirming that the cohorts were successfully matched. The 2 matched groups did not differ in other variables such as biopsy GS, pathological T, lymph node invasion (LNI), and positive surgical margin. However, while age, clinical T stage, clinical N stage, clinical M stage, and pathological GS were well-matched with an $SMD<0.1$, the PSA level showed an imbalance with an SMD of 0.458 (Table 1).

Comparison of outcomes indicated no significant differences between the matched groups in terms of BCR ($p=0.361$), all-cause mortality ($p=0.283$), or cancer-specific mortality ($p>0.999$) (Table 1). In the Kaplan-Meier survival

Table 1. Clinicopathological characteristics and outcome of patients

Characteristic	Typical PC (n=61)	Mucinous PC (n=61)	p-value	SMD
Age (yr)	66 (60–69)	67 (60–71)	0.522	0.054
PSA (ng/mL)	8.41 (5.26–14.70)	10.17 (6.00–20.66)	0.064	0.458
Biopsy Gleason score			0.710	-
6	19 (31.1)	5 (8.2)		
7 (3+4)	11 (18.0)	17 (27.9)		
7 (4+3)	7 (11.5)	7 (11.5)		
8	16 (26.2)	14 (23.0)		
9	8 (13.1)	6 (9.4)		
Unknown	0 (0)	2 (3.3)		
cT stage			0.580	0.023
T1	14 (23.0)	12 (19.7)		
T2	25 (41.0)	33 (54.1)		
T3a	4 (23.0)	10 (16.4)		
T3b	7 (11.5)	4 (6.6)		
T4	1 (1.6)	2 (3.3)		
cN stage			>0.999	0.062
N0	57 (93.4)	56 (91.8)		
N1	4 (6.6)	5 (8.2)		
cM stage			>0.999	0.082
M0	58 (95.1)	59 (96.7)		
M1	3 (4.9)	2 (3.3)		
Pathological Gleason score			0.090	0
6	7 (11.5)	2 (3.3)		
7 (3+4)	17 (27.9)	19 (31.1)		
7 (4+3)	17 (27.9)	20 (32.8)		
8	7 (11.5)	14 (23.0)		
9	13 (21.3)	6 (9.8)		
pT stage			0.418	-
T2	33 (54.1)	25 (41.0)		
T3a	20 (32.8)	23 (37.7)		
T3b	7 (11.5)	10 (16.4)		
T4	1 (1.6)	3 (4.9)		
Lymph node invasion	3 (4.9)	4 (6.6)	>0.999	-
Positive surgical margin	20 (32.8)	26 (42.6)	0.262	-
Biochemical recurrence	29 (47.5)	24 (39.3)	0.361	-
All-cause mortality	6 (9.8)	10 (16.4)	0.283	-
Cancer-specific mortality	2 (3.3)	3 (4.9)	>0.999	-

Values are presented as median (interquartile range) or number (%).

PC, prostate cancer; SMD, standardized mean difference; PSA, prostate-specific antigen; cT, clinical tumor; cN, clinical node; cM, clinical metastasis; pT, pathologic tumor.

analysis, no statistically significant differences were observed in BCRFS ($p=0.676$), or CSS ($p=0.458$) between the 2 groups (Figs. 1 and 2).

Cox regression analysis was performed to evaluate BCR (Table 2). In multivariable analysis, pathological GS was a statistically significant predictor of BCR, with GS 8 (adjusted hazard ratio [AHR], 18.256; 95% confidence interval [CI], 2.177–153.113; $p=0.007$) and GS 9 (AHR, 8.912; 95% CI, 1.121–70.843; $p=0.039$) being significant predictors. Pathological T3b stage (AHR, 2.659; 95% CI, 1.070–6.604; $p=0.035$) was also a significant predictor of BCR. Mucinous PC did not

have a significant effect on the risk of recurrence (AHR, 0.620; 95% CI, 0.336–1.145; $p=0.127$).

In the Cox regression analysis for CSS, multivariable analysis showed that only LNI was a significant independent predictor (AHR, 20.460; 95% CI, 3.395–123.319; $p=0.001$). Mucinous PC was not a significant factor for CSS (AHR, 1.707; 95% CI, 0.281–10.373; $p=0.561$) (Table 3).

DISCUSSION

In this study, we evaluated the prognostic outcomes of

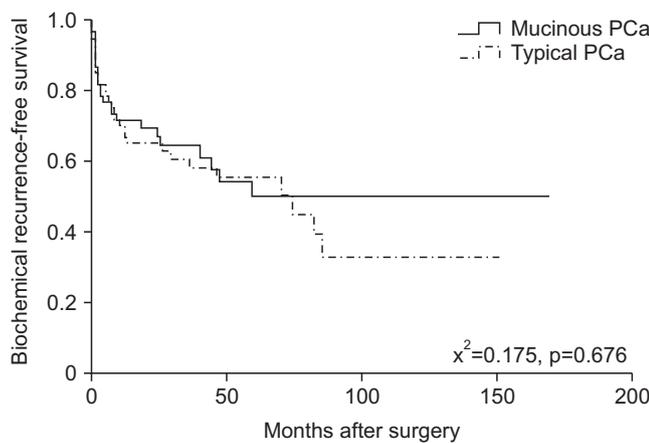


Fig. 1. Kaplan-Meier plots of biochemical recurrence-free survival. PCa, prostate cancer.

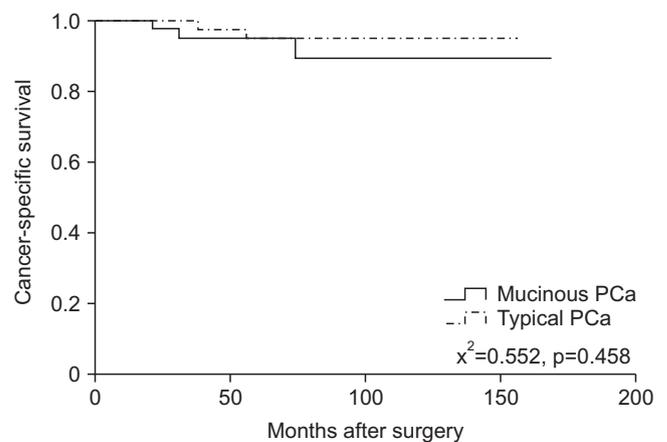


Fig. 2. Kaplan-Meier plots of cancer-specific survival. PCa, prostate cancer.

Table 2. Cox regression analysis of biochemical recurrence

Variable	Univariable model			Multivariable model		
	HR	(95% CI)	p-value	AHR	(95% CI)	p-value
Age	1.015	(0.983–1.047)	0.367	-	-	-
PSA	1.020	(1.011–1.028)	<0.001	1.006	(0.994–1.019)	0.337
Group						
Typical	-	Reference	-	-	Reference	-
Mucinous	0.892	(0.519–1.535)	0.681	0.620	(0.336–1.145)	0.127
Pathological GS						
6	-	Reference	-	-	Reference	0.012
7 (3+4)	5.479	(0.721–41.647)	0.100	5.858	(0.752–45.631)	0.091
7 (4+3)	4.319	(0.559–33.404)	0.161	5.187	(0.653–41.224)	0.120
8	17.354	(2.228–135.173)	0.006	18.256	(2.177–153.113)	0.007
9	11.113	(1.419–87.009)	0.022	8.912	(1.121–70.843)	0.039
pT stage						
T2	-	Reference	-	-	Reference	0.203
T3a	1.700	(0.904–3.196)	0.099	1.703	(0.835–3.471)	0.143
T3b	3.370	(1.576–7.205)	0.002	2.659	(1.070–6.604)	0.035
T4	5.239	(1.531–17.927)	0.008	1.839	(0.337–10.031)	0.482
LN invasion	4.817	(2.001–11.595)	<0.001	2.329	(0.774–7.009)	0.133
PSM	1.903	(1.091–3.321)	0.023	1.189	(0.603–2.345)	0.617

HR, hazard ratio; CI, confidence interval; AHR, adjusted HR; PSA, prostate-specific antigen; GS, Gleason score; pT, pathologic tumor; LN, lymph node; PSM, propensity score matching.

Table 3. Cox regression analysis of cancer-specific mortality

Variable	Univariable model			Multivariable model		
	HR	(95% CI)	p-value	AHR	(95% CI)	p-value
Age	1.024	(0.919–1.141)	0.666	-	-	-
PSA	1.017	(0.998–1.037)	0.074	-	-	-
Group						
Typical	-	Reference	-	-	Reference	-
Mucinous	1.947	(0.325–11.674)	0.466	1.707	(0.281–10.373)	0.561
Pathological GS						
6	-	Reference	0.605	-	-	-
7 (3+4)	8,864.789	(0–)	0.956	-	-	-
7 (4+3)	1.001	(0–)	>0.999	-	-	-
8	22,467.304	(0–)	0.952	-	-	-
9	55,968.779	(0–)	0.947	-	-	-
pT stage						
T2	-	Reference	0.834	-	-	-
T3a	98,712.095	(0–)	0.946	-	-	-
T3b	230,564.821	(0–)	0.942	-	-	-
T4	0.999	(0–)	>0.999	-	-	-
LN invasion	21.102	(3.518–126.581)	0.001	20.460	(3.395–123.319)	0.001
PSM	2.164	(0.360–13.003)	0.399	-	-	-

HR, hazard ratio; CI, confidence interval; AHR, adjusted HR; PSA, prostate-specific antigen; GS, Gleason score; pT, pathologic tumor; LN, lymph node; PSM, propensity score matching.

mucinous PC compared to those of typical PC and found no statistically significant differences between these histological subtypes in terms of BCRFS and CSS. A high GS of 8 and 9 and pathologic T3b stage, but not mucinous PC, emerged as significant independent predictors of BCR. Similarly, only LNI was identified as an independent prognostic determinant for CSS.

However, while the PSM in our study successfully balanced most baseline characteristics, a significant imbalance in the initial PSA level, with a SMD of 0.458, remained after matching. To control for the potential confounding bias arising from this PSA imbalance, we utilized a multivariable Cox regression model as the core analytical tool. This analysis statistically adjusts for the effects of powerful prognostic factors such as pathological GS and stage, in addition to PSA. The finding that mucinous histology was not significant even after this rigorous adjustment provides strong evidence that our main conclusion is not an artifact of the observed difference in PSA.

Historically, mucinous PC has been considered to exhibit a more aggressive biological phenotype and greater propensity for bone metastasis than typical PC. Early case series, such as the 1985 study by Epstein and Lieberman, reported aggressive behavior in 6 cases [8], and a study from 1999

involving 88 cases also identified an unfavorable prognosis for mucinous carcinoma with signet ring cells [10]. Furthermore, a study based on data from 1964 to 1990 found that mucinous adenocarcinoma was associated with worse clinical outcomes [14]. These early findings led the ISUP in 2005 to recommend classifying all mucinous adenocarcinomas as Gleason pattern 4, thereby assigning them a GS of 7 or higher [11].

However, research from the mid-2000s onward challenged this traditional view. Two small cohort studies (n=12 and n=14) reported no significant prognostic differences between patients with mucinous PC and those with typical PC [9,12]. Furthermore, a study by Osunkoya et al. [13] involving 47 patients with mucinous PC revealed a BCRFS of 97.2%, which was notably higher than the predicted 85.4% rate for typical PC. Studies by Carolin et al. [6,7] indicated no significant difference in OS and cancer-specific mortality (CSM) between mucinous PC and typical PC. This supports the conclusion that mucinous PC is not more aggressive and may be less aggressive than typical PC [13]. Consequently, the 2014 ISUP consensus recommended that the grading of mucinous carcinoma should be based on its underlying architectural pattern, with the mucinous component no longer dictating the grade [14].

Regarding BCRFS, conventional wisdom parallels the historical view of the overall prognosis of mucinous PC, and recent evidence also refutes this notion. In the pre-PSA era, mucinous PC was considered to have a tendency for aggressive behavior and widespread metastasis [8]. PSA elevation rate in patients with mucinous PC undergoing endocrine therapy was 77.8%, similar to that in patients with high-grade adenocarcinoma [10]. Otherwise, Lane et al. [12], who found no differences in BCRFS after matching mucinous PC and adenocarcinoma with focal mucin (AFM). Similarly, in a large analysis of 143 cases, although mucinous PC is often high grade, its BCR rate was not significantly different from that of typical PC with a similar grade (12.5% vs. 17%, $p=0.15$) [2]. Consistent with recent studies, our study found no significant difference in BCRFS between mucinous and typical PC. Interestingly, patients with mucinous PC demonstrated a paradoxically higher BCRFS of 97.2% following RP, compared to 85.4% in patients with typical PC.

We observed no statistically significant difference in CSS between the mucinous PC and typical PC. In the aforementioned study analyzing the 88 patients, mucinous carcinoma with signet ring cells showed a very poor prognosis, with a 3-year survival rate of 16.7% [10]. In contrast, Lane et al. [12] reported 5-year OS rates of 100% for both mucinous PC and AFM patients, compared to 96.6% for typical PC patients, suggesting a favorable prognosis. In a large population-based analysis of SEER data, 360 patients with mucinous carcinoma were included. Similar to our study, outcomes were compared with those of typical PC after PSM, and no significant difference in CSS was observed ($p=0.23$) [1].

Various gene expression is related with mucinous adenocarcinoma. MUC1, MUC2, MUC4, MUC13, and MUC16 influence the expression of mucinous adenocarcinoma in various organ such as breast, prostate, lung, pancreas [20]. MUC1 expression in PC was associated with an elevated risk of recurrence ($p=0.003$) [21]. In the case of MUC2, 25 of 25 cases (100%) of mucinous PC were positive for MUC2 expression, whereas only 6 of 25 cases (24%) of typical PC were MUC2 positive [22]. MUC2 may contribute to the relatively slow growth of tumors, which interferes with their ability to spread. Moreover, in a multi-institutional study that analyzed 92 cases of PC with mucinous features, 79 (86%) were positive for PTEN expression [23]. Loss of PTEN

expression is known to occur in aggressive, high-grade PC [22]. These mucinous PCs may be less aggressive than typical PC, even with high GS, as 71 (77.2%) cases had GS 7 [4+3] or higher). Additionally, TMPRSS2-ERG fusion gene has been detected in 25 of 51 cases (47%) of mucinous PC, a frequency similar to that observed in patients with typical PC (40%–70%) [24]. These findings suggest that mucinous PC is clonally related to typical PC. Taken together, gene expression in mucinous PC is sometimes similar to that of typical PC, and even when differences are observed, they are not necessarily associated with aggressive prognosis.

The present study is limited by its small sample size, retrospective design, and single-center nature. Although PSM was employed to overcome heterogeneity, the potential for residual confounding and information bias inherent to retrospective studies remains. Specifically, a key limitation of this study is that PSM failed to fully balance PSA levels; therefore, the study's conclusions rely heavily on the statistical adjustment provided by the multivariable Cox model.

Furthermore, the analysis for CSS was limited by the small number of events, with only 5 in total. This resulted in insufficient statistical power, which may have prevented the detection of a clinically meaningful, moderate-sized difference. Therefore, the hazard ratio estimates for CSS should be interpreted with caution.

Despite these limitations, this study represents the largest cohort of studies on mucinous PC in South Korea. Additionally, as the study was conducted on patients who underwent RP, it allowed for a more comprehensive histological evaluation of the mucinous component than was possible with biopsy specimens.

CONCLUSIONS

PC with mucinous components showed similar outcomes, including BCRFS and CSS, compared to typical PC. The type of PC was not a significant predictor for either BCRFS or CSS. Therefore, PC with mucinous components may not be as aggressive as previously thought. However, further study is needed to clarify these findings, given the controversial nature of existing findings.

NOTES

• **Author Contribution:** Conceptualization: JHC, JEH; Data curation: WSJ, WSH, YDC; Formal analysis: JHC, JL, HH, JEH; Funding acquisition: JEH; Methodology: JHC, JEH; Project administration: NHC, YDC; Visualization: JEH; Writing - original draft: JHC; Writing - review & editing: JHC, JEH.

• ORCID

Jin Hyeok Choi: <https://orcid.org/0009-0008-2899-7240>

Hyunho Han: <https://orcid.org/0000-0002-6268-0860>

Jongsoo Lee: <https://orcid.org/0000-0002-9984-1138>

Won Sik Jang: <https://orcid.org/0000-0002-9082-0381>

Won Sik Ham: <https://orcid.org/0000-0003-2246-8838>

Nam Hoon Cho: <https://orcid.org/0000-0002-0045-6441>

Young Deuk Choi: <http://orcid.org/0000-0002-8545-5797>

Ji Eun Heo: <https://orcid.org/0000-0002-4184-8468>

REFERENCES

- Zhao F, Yu X, Xu M, Ye S, Zang S, Zhong W, et al. Mucinous prostate cancer shows similar prognosis to typical prostate acinar carcinoma: a large population-based and propensity score-matched study. *Front Oncol* 2019;9:1467.
- Samaratunga H, Delahunt B, Srigley JR, Yaxley J, Johannsen S, Coughlin G, et al. Mucinous adenocarcinoma of prostate and prostatic adenocarcinoma with mucinous components: a clinicopathological analysis of 143 cases. *Histopathology* 2017;71:641-7.
- Netto GJ, Amin MB, Berney DM, Comperat EM, Gill AJ, Hartmann A, et al. The 2022 World Health Organization classification of tumors of the urinary system and male genital organs-Part B: prostate and urinary tract tumors. *Eur Urol* 2022;82:469-82.
- Zhou J, Ding J, Qi J. Comparison of typical prostate adenocarcinoma and rare histological variant prostate cancer showed different characteristics and prognosis: a surveillance, epidemiology, and end results database analysis. *Eur Urol* 2022;82:152-5.
- Chandler B, Sohrab A, Akshay S, Deepansh D, Jacob K, Alex B, et al. Rare histological variants of prostate adenocarcinoma: a national cancer database analysis. *J Urol* 2020;204:260-6.
- Carolin S, Mario de A, Letizia M, Francesco Di B, Natali R, Jordan A, et al. Life expectancy in rare histological prostate cancer subtypes. *Int J Cancer* 2025;156:2311-9.
- Siech C, de Angelis M, Jannello LM, Di Bello F, Rodriguez Peñaranda N, Goyal JA, et al. Rare histological prostate cancer subtypes: cancer-specific and other-cause mortality. *Prostate Cancer Prostatic Dis* 2025;28:748-54.
- Epstein JI, Lieberman PH. Mucinous adenocarcinoma of the prostate gland. *Am J Surg Pathol* 1985;9:299-308.
- Ro JY, Grignon DJ, Ayala AG, Fernandez PL, Ordonez NG, Wishnow KI. Mucinous adenocarcinoma of the prostate: histochemical and immunohistochemical studies. *Hum Pathol* 1990;21:593-600.
- Saito S, Iwaki H. Mucin-producing carcinoma of the prostate: review of 88 cases. *Urology* 1999;54:141-4.
- Epstein JI, Allsbrook WC Jr, Amin MB, Egevad LL, Committee IG. The 2005 International Society of Urological Pathology (ISUP) Consensus Conference on Gleason grading of prostatic carcinoma. *Am J Surg Pathol* 2005;29:1228-42.
- Lane BR, Magi-Galluzzi C, Reuther AM, Levin HS, Zhou M, Klein EA. Mucinous adenocarcinoma of the prostate does not confer poor prognosis. *Urology* 2006;68:825-30.
- Osunkoya AO, Nielsen ME, Epstein JI. Prognosis of mucinous adenocarcinoma of the prostate treated by radical prostatectomy: a study of 47 cases. *Am J Surg Pathol* 2008;32:468-72.
- Humphrey PA, Moch H, Cubilla AL, Ulbright TM, Reuter VE. The 2016 WHO Classification of Tumours of the urinary system and male genital organs-Part B: prostate and bladder tumours. *Eur Urol* 2016;70:106-19.
- Lee HI, Kim DG, Seo YJ, Kim JR, Lee KS. Mucinous adenocarcinoma of prostate. *Korean J Urol* 2003;1187-9.
- Son HJ, Jeong JS, Moon WS, Kang MJ. Mucinous adenocarcinoma of the prostate: a case report. *J Pathol Transl Med* 2003;37:221-3.
- Song JW, Kang DG, Jung TY, Shon JH, Jeong HH, Choi NG. A case of epididymal metastasis from mucinous adenocarcinoma of the prostate. *Korean J Urol* 1998;39:819-22.
- Song YM, Jung JC, Kim SW, Yeum KY, Choi SI, Park RJ. A case of mucinous adenocarcinoma of the prostate gland. *Korean J Urol* 1991;32:688-90.
- Cookson MS, Aus G, Burnett AL, Canby-Hagino ED, D'Amico AV, Dmochowski RR, et al. Variation in the definition of biochemical recurrence in patients treated for localized prostate cancer: the American Urological Association Prostate Guidelines for Localized Prostate Cancer Update Panel report and recommendations for a standard in the reporting of surgical outcomes. *J Urol* 2007;177:540-5.
- Kufe DW. Mucins in cancer: function, prognosis and therapy. *Nat Rev Cancer* 2009;9:874-85.
- Lapointe J, Li C, Higgins JP, van de Rijn M, Bair E, Montgomery K, et al. Gene expression profiling identifies clinically relevant subtypes of prostate cancer. *Proc Natl Acad Sci U S A* 2004;101:811-6.
- Osunkoya AO. Mucinous and secondary tumors of the

- prostate. *Mod Pathol* 2018;31(Suppl 1):S80-95.
23. Bertsch EC, Magi-Galluzzi C, Cheng L, Osunkoya AO. PTEN expression in mucinous prostatic adenocarcinoma, prostatic adenocarcinoma with mucinous features, and adjacent conventional prostatic adenocarcinoma: a multi-institutional study of 92 cases. *Appl Immunohistochem Mol Morphol* 2018;26:225-30.
24. Johnson H, Zhou M, Osunkoya AO. ERG expression in mucinous prostatic adenocarcinoma and prostatic adenocarcinoma with mucinous features: comparison with conventional prostatic adenocarcinoma. *Hum Pathol* 2013;44:2241-6.