EDITORIAL

5839  Orthopedic manifestations of Li-Fraumeni syndrome: Prevention and treatment of a polymorphic spectrum of malignancies  
Cenci G, Pace V

5845  Confocal laser endomicroscopy as a new diagnostic tool for poorly differentiated gastric adenocarcinoma  
Evola G, Vacante M, Evola FR

5850  Proteomics for early prenatal screening of gestational diabetes mellitus  
Wu L, Wang XP, Zhu YX, Tan YP, Li CM

5854  Paired box proteins as diagnostic biomarkers for endocervical adenocarcinoma  
Zhou JH, Zhang XN

5859  Endoscopic ultrasound-guided biliary drainage using electrocautery-enhanced lumen-apposing metal stent for malignant biliary obstruction: A promising procedure  
Wu SZ

5863  Cardiac implications in myasthenia gravis  
Elmati PR, Jagirdhar GSK, Sarani S

ORIGINAL ARTICLE

Case Control Study

5868  Multivariate analysis of oral mucosal ulcers during orthodontic treatment  
Chang J, Li X

5877  Impact of web-based positive psychological intervention on emotions, psychological capital, and quality of life in gastric cancer patients on chemotherapy  
Xin YY, Zhao D

Retrospective Cohort Study

5885  Risk factors and clinical significance of posterior slip of the proximal vertebral body after lower lumbar fusion  

Retrospective Study

5893  Predictive value of diaphragm ultrasound for mechanical ventilation outcome in patients with acute exacerbation of chronic obstructive pulmonary disease  
Qu LL, Zhao WP, Li JP, Zhang W
## Contents

**World Journal of Clinical Cases**

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<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>5901</td>
<td>Influence of perinatal factors on full-term low-birth-weight infants and construction of a predictive model</td>
<td>Xu L, Sheng XJ, Gu LP, Yang ZM, Feng ZT, Gu DF, Gao L</td>
</tr>
<tr>
<td>5908</td>
<td>Magnetic resonance imaging-based radiomics model for preoperative assessment of risk stratification in endometrial cancer</td>
<td>Wei ZY, Zhang Z, Zhao DL, Zhao WM, Meng YG</td>
</tr>
<tr>
<td>5922</td>
<td>Application of real-time shear wave elastography to Achilles tendon hardness evaluation in older adults</td>
<td>He X, Wei X, Hou J, Tan W, Luo P</td>
</tr>
<tr>
<td>5930</td>
<td>Study of the intensive care unit activity scale in the early rehabilitation of patients after direct cardiac surgery</td>
<td>Wang L, Lu JY, Ma XX, Ma LO</td>
</tr>
<tr>
<td>5937</td>
<td>Modifiable factors mediating the effects of educational attainment on gestational diabetes mellitus: A two-step Mendelian randomization study</td>
<td>Ma MY, Zhao YS</td>
</tr>
<tr>
<td>5946</td>
<td>Periorbital purpura can be the only initial symptom of primary light chain amyloidosis: A case report</td>
<td>Wang XF, Li T, Yang M, Huang Y</td>
</tr>
<tr>
<td>5960</td>
<td>Stage IV non-small cell lung cancer with multiple metastases to the small intestine leading to intussusception: A case report</td>
<td>Niu QG, Huang MH, Kong WQ, Yu Y</td>
</tr>
<tr>
<td>5974</td>
<td>Organizing pneumonia secondary to pulmonary tuberculosis: A case report</td>
<td>Liu M, Dong XY, Ding ZX, Wang QH, Li DH</td>
</tr>
<tr>
<td>5983</td>
<td>Sclerosing epithelioid fibrosarcoma of the pancreas: A case report</td>
<td>Sun MQ, Guo LN, You Y, Qiu YY, He XD, Han XL</td>
</tr>
</tbody>
</table>
LETTER TO THE EDITOR

5998 Fragile hearts: Unveiling the crucial layers of frailty in elderly patients undergoing percutaneous coronary interventions
   Mitsis A, Myrianthefs M

6001 T lymphocyte proportion in Alzheimer’s disease prognosis
   Willman M, Patel G, Lucke-Wold B
ABOUT COVER
Peer Reviewer of World Journal of Clinical Cases, Ralph Victor Yap, MD, RN, Assistant Professor, Surgeon, Department of Surgery, Cebu Doctors' University Hospital, Cebu 6000, Philippines. rvyapmd@gmail.com

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Concurrent occurrence of adenocarcinoma and urothelial carcinoma of the prostate gland: A case report

Jhe Yuan Hsu, Yi Sheng Lin, Li Hua Huang, Tang Yi Tsao, Chao Yu Hsu, Yen Chuan Ou, Min Che Tung

Abstract

BACKGROUND
Adenocarcinoma is the most common subtype of prostate cancer. Prostatic urothelial carcinoma (UC) typically originates from the prostatic urethra. The concurrent occurrence of adenocarcinoma and UC of the prostate gland is uncommon.

CASE SUMMARY
We present the case of an 82-year-old male patient with simultaneous adenocarcinoma and UC of the prostate gland. The patient underwent a transrectal ultrasound-guided biopsy, and the pathology test revealed UC. Subsequently, transurethral laser prostatectomy was performed, and the pathology test indicated adenocarcinoma of the prostate with a Gleason score of 3 + 4 and high-grade UC. Therefore, the patient was treated with androgen deprivation therapy, systemic chemotherapy, and immunotherapy. Magnetic resonance imaging performed during follow-up revealed a prostate tumor classified as cT2cN1M0, stage IVA. Therefore, the patient underwent robotic-assisted radical prostatectomy and bilateral pelvic lymph node dissection. The final pathology test of the prostate gland revealed acinar-type adenocarcinoma, Gleason pattern 4 + 3, pT2N0M0, and high-grade UC. The patient regularly presented to the clinic for postoperative follow-up evaluations. He did not experience any urinary discomfort.

CONCLUSION
According to our literature review, this is the first reported case of coexisting adenocarcinoma and UC of the prostate gland.
Key Words: Adenocarcinoma; Urothelial carcinoma; Prostate; Coexist; Case report

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Core Tip: This report of synchronous adenocarcinoma and urothelial carcinoma (UC) of the prostate gland describes the unique prostate cancer manifestations in a male patient as well as the clinical journey from his initial symptoms of urinary retention and gross hematuria to the final treatment comprising robotic-assisted radical prostatectomy and bilateral pelvic lymph nodes dissection. This rare co-occurrence of two distinct cancer subtypes of the prostate gland without a history of UC of the urinary bladder and evident recurrence after treatment emphasizes the need for heightened diagnostic awareness and suggests novel oncogenic pathways and genetic predispositions.

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INTRODUCTION
Prostate cancer is the second most common cancer among men worldwide, and its incidence increases with age. The predominant subtype of carcinoma of the prostate is adenocarcinoma. Adenocarcinoma is graded using the Gleason scoring system. Prostate urothelial carcinoma (UC) is a subtype that usually develops from the urothelium lining the prostatic urethra and the proximal sections of the prostatic ducts [1]. As far as we are aware, this is the first documented report of adenocarcinoma and UC co-occurring in the prostate gland.

CASE PRESENTATION

Chief complaints
An 82-year-old male patient presented to the urology clinic with a 5-day history of acute urinary retention and gross hematuria.

History of present illness
The patient experienced gross hematuria and intermittent acute urinary retention for 5 days before presentation.

History of past illness
The patient was initially evaluated at another hospital because he experienced acute urinary retention and gross hematuria. A digital rectal examination (DRE) revealed firm prostate nodules and an increased prostate-specific antigen (PSA) level of 53 ng/mL. In October 2020, the patient underwent a transrectal ultrasound-guided biopsy that revealed UC. Subsequently, he underwent transurethral laser prostatectomy. The pathology test indicated prostatic adenocarcinoma with a Gleason score of 3 + 4 and high-grade UC. Cystoscopy revealed no papillary tumors in the prostatic urethra or urinary bladder. No evidence of bone metastasis was observed during a bone scan. Therefore, the patient was treated with androgen deprivation therapy (ADT) with leuprorelin and bicalutamide in December 2020. In January and February 2021, the patient was treated with systemic chemotherapy with gemcitabine and cisplatin. In February 2021, the patient was treated with immunotherapy with nivolumab at another hospital (Figure 1).

Personal and family history
The patient had a history of hypertension and arrhythmia that were controlled with medication. He did not have a family history of malignant tumors.

Physical examination
During the physical examination, the external genitalia appeared normal. The DRE revealed a firm prostate with an estimated volume of 25 cm³.

Laboratory examinations
The preoperative blood test revealed a white blood cell count of $8.2 \times 10^3/\mu L$, hemoglobin level of 12.3 g/dL, platelet count of $263 \times 10^3/\mu L$, glutamic oxaloacetic transaminase level of 21 IU/L, blood urea nitrogen level of 21 mg/dL, creatinine level of 1.01 mg/dL, estimated glomerular filtration rate of 75, and PSA level of 8.877 ng/dL. The urinalysis results were within normal ranges.
Imaging examinations

Ultrasonography was performed to estimate the volume of the prostate gland (25 cm³). The postvoid volume was 29 mL. Magnetic resonance imaging was performed during follow-up and revealed a prostate tumor classified as cT2cN1M0, stage IVA (Figure 2).

MULTIDISCIPLINARY EXPERT CONSULTATION

A technetium-99 m methylene diphosphonate whole-body bone scan revealed no evidence of bone metastasis (Figure 3). An 18F-fluorodeoxyglucose positron emission tomography examination revealed no evidence of metastasis (Figure 4). Cystoscopy revealed no papillary tumors in the urinary bladder or urethra.

FINAL DIAGNOSIS

The final diagnosis was adenocarcinoma and high-grade UC of the prostate classified as cT2cN1M0, stage IVA.

TREATMENT

The patient presented to our hospital for a second opinion regarding the diagnosis. Radical cystoprostatectomy was suggested; however, the patient refused this procedure. Therefore, he underwent robotic-assisted radical prostatectomy (RaRP) and bilateral pelvic lymph node dissection (BPLND) on May 11, 2021 (Figure 5).

OUTCOME AND FOLLOW-UP

The results of the final pathology test of the prostate gland indicated acinar-type adenocarcinoma, gleason pattern 4 + 3 (grade group 3), pT2N0M0, and high-grade UC (Figure 6). The surgical margins were clear, and extraprostatic extension was not observed. The immunohistochemical staining results were as follows: Negative CK5; Positive α-methylacyl coenzyme A racemase; Positive synaptophysin (20%); and Positive GATA binding protein 3 (GATA3). These findings indicated concurrent adenocarcinoma and UC of the prostate gland. The patient was discharged 6 days postoperatively. He experienced good recovery and did not report any major complications. Outpatient evaluations comprising PSA monitoring and cystoscopy were performed every 3 months. The PSA levels remained less than 0.008 ng/dL. Urinary discomfort and signs of recurrence were not observed during the most recent clinical evaluation. The patient expressed that he was highly satisfied with the surgical outcome and that his quality of life had significantly improved with treatment.

DISCUSSION

To our knowledge, this is the first reported case of simultaneous adenocarcinoma and UC of the prostate gland in Taiwan. Typically, prostate cancer manifests as adenocarcinoma. Although there have been case reports of adenocar-
Figure 2 Image of the pelvis obtained during follow-up with magnetic resonance imaging. A: A 2.4-cm lesion with low T2 signal is observed in the posterolateral portion of the left transitional zone; B: Apparent diffusion coefficient of the lesion with low signal; C: Increased diffusion tensor imaging signals are observed.

Figure 3 Technetium-99m methylene diphosphonate whole-body bone scan. Focal areas of increased radioactivity uptake are noted in the lumbar spine at levels L1/2, L3/4, and L4/5, as well as in the left elbow and bilateral acromioclavicular joints. These findings suggest a benign etiology.

carcinoma and tubulovillous adenoma of the urinary bladder, UC predominantly occurs in the urinary bladder or ureter[2, 3]. One of the primary objectives of this study was to stimulate further research into the mechanisms underlying the coexistence of these conditions.

According to the World Health Organization GLOBOCAN database, prostate cancer is a significant medical concern because of its prevalence, impact on the quality of life, and mortality. Risk factors include age, ethnicity, genetics, diet, hormones, obesity, and others[4-8]. Patients are often asymptomatic initially. Bone pain is a common clinical presentation among patients with metastatic prostate cancer at the time of diagnosis because the bones are the predominant sites of dissemination[9].

Prostate cancer is typically suspected when increased PSA levels or abnormal DRE findings are observed. There is no single threshold for abnormal PSA levels. However, age-specific reference PSA levels, which increase faster in older men,
Figure 4 Images obtained during an 18F-fluorodeoxyglucose positron emission tomography evaluation of the region comprising the head to the upper thigh. From left to right: Ill-defined fluorodeoxyglucose (FDG) avidity is observed in the prostate; No FDG-avid regional or distant lymph nodes are apparent; Ill-defined FDG avidity is noted in the right buccal region after the dental prosthesis; and Ill-defined FDG avidity in the right T1, T4, T8, T12, and left upper ilia with sclerotic changes, suggesting a benign etiology.

Figure 5 Gross examination of the prostate tumor after robotic-assisted radical prostatectomy. 4 cm × 3 cm × 4 cm prostate gland with a volume of 24.96 mL and weight of 29 g; The 19 cm × 1.3 cm × 0.7 cm adenocarcinoma is located in the left anterior lateral lobe and, specifically, in the middle area transitioning to the peripheral zone. The 1 cm × 0.6 cm × 0.6 cm carcinoma of the urethra is observed in the right posterior lobe, middle area, and near the periurethra.

and the velocity of increase in the PSA level, which often has a cutoff of more than 0.75 ng/dL within 1 year, are considered significant findings[10].

A definitive diagnosis requires the evaluation of tissue samples obtained during a transrectal or transperineal biopsy [11]. Most malignant prostatic neoplasms are carcinomas that originate from and are differentiated from epithelial tissue. Adenocarcinoma, which accounts for more than 95% of all cases, is characterized by its glandular structure, absence of basal cells, and distinct nuclear characteristics of the glandular epithelial cells[12]. Compared to benign glands, malignant glands present more frequently with intraluminal crystalloids, amorphous secretions, or blue-tinged mucin. Adenocarcinomas are mostly acinar; the ductal types are less common[13]. UC, which is another less common subtype, typically occurs concurrently with bladder carcinoma; however, it can arise as the primary disease. Immunohistochemical findings that differentiate UC from adenocarcinoma of the prostate include thrombomodulin, GATA3, p63, and high-molecular-weight cytokeratins[14].
The staging and evaluation of adenocarcinoma of the prostate as well as its treatment strategies are based on its risk stratification determined by the DRE results, serum PSA levels, pathology results of the prostate biopsy sample, and imaging results. Treatment options for very low-risk disease include active surveillance and, if necessary, definitive local therapy such as radiation therapy (RT) and radical prostatectomy[15]. Definitive treatment options for patients with low-risk prostate cancer and a life expectancy of more than 10 years include RT, radical prostatectomy, and active surveillance[16]. Intermediate-risk disease can be treated with RT and radical prostatectomy. Clinically localized high-risk prostate cancer is typically managed with external beam RT combined with brachytherapy, ADT, or radical prostatectomy[15]. Treatments for locally advanced and very high-risk prostate cancer include external beam RT with or without brachytherapy, long-term ADT, and radical prostatectomy[17]. The outcomes of these treatments depend on the disease stage and patient compliance.

A review of the literature indicated that most patients with UC of the prostate had a history of UC of the urinary bladder[18]. Additionally, concurrent UC of the urinary bladder and prostate was diagnosed incidentally after cystoprostatectomy[19,20]. In this case, UC was not observed in the urinary bladder. However, the coexistence of UC and adenocarcinoma of the prostate was incidentally discovered after the prostate biopsy. According to previous research of the different immunohistochemical findings that distinguish prostate carcinoma from UC, the sensitivities of prostatic markers of prostate adenocarcinoma were as follows: 100% for PSA; 83.8% for prostate-specific membrane antigen; 91.9% for prostate acid phosphatase; 93.7% for PS101s; 88.3% for NKX 3.1; and 66.7% for α-methylacyl coenzyme A racemase. In contrast, the sensitivities of urothelial markers of UC were 75.4% for CK34βE12, 73.9% for p63, 45.7% for thrombomodulin, 22.5% for S100P, and 84.8% for GATA3[21]. The immunohistochemical staining results of the prostate samples from our patient were positive for both α-methylacyl coenzyme A racemase and GATA3; therefore, the coexistence of adenocarcinoma and UC was strongly suggested.

We proposed three hypotheses regarding the potential mechanisms of synchronous adenocarcinoma and UC of the prostate. First, metaplastic transformation may result in this phenomenon. Metaplastic transformation involves the conversion of one differentiated cell type to another. In this context, some glandular cells within the prostate may have undergone metaplastic transformation to urothelial cells, potentially driven by chronic inflammation or other local environmental factors[22]. Second, the presence of multipotent stem cells within the prostate could explain the coexistence of adenocarcinoma and UC. Multipotent stem cells can differentiate into various cell types, including glandular and urothelial cells. This pluripotency may lead to the simultaneous emergence of adenocarcinoma and UC in the same tissue[23,24]. Third, the intraluminal spread of UC cells from an occult site within the urinary tract to the prostate could cause this uncommon disease occurrence. Intraluminal spread can occur without a documented history of primary UC, particularly if the primary site is very small or has regressed[25]. Although these hypotheses provide various potential explanations for the simultaneous presence of adenocarcinoma and UC of the prostate, they have not been confirmed.

Our patient initially underwent ADT for adenocarcinoma of the prostate because he preferred nonsurgical intervention. Consequently, leuprorelin and bicalutamide were administered for several months. Although the PSA level of...
our patient decreased to 8.78 ng/mL after 4 months of treatment, he sought a second opinion at our hospital. Because the tumor was restricted to the prostate stroma without involvement of the prostatic urethra, as confirmed by cystoscopy and magnetic resonance imaging, and because the patient’s Eastern Cooperative Oncology Group performance status was good, radical cystoprostatectomy was recommended. However, after a thorough discussion and explanation, the patient chose RaRP and BPLND as bladder-sparing surgery. Additionally, four cycles of systemic chemotherapy with gemcitabine and cisplatin for UC of the prostate were initially administered at another hospital by a physician with clinical experience with UC of the prostate with stromal invasion. Subsequently, second-line systemic immunotherapy with Nivolumab was administered.

Despite the absence of a history of UC in the urinary bladder, cystoscopy revealed no recurrence following ADT, chemotherapy, and immunotherapy. Two years after RaRP and BPLND, the patient was recurrence-free. No reports of synchronous adenocarcinoma and UC of the prostate gland were found during our literature review.

CONCLUSION

To the best of our knowledge, this is the first reported case of synchronous adenocarcinoma and UC of the prostate gland. Although a correlation between adenocarcinoma and UC of the prostate gland has not been proven, this case highlights the potential for the coexistence of these two cancer subtypes in the prostate gland and suggests the need for further genetic studies and case reports to improve the understanding of the causes and mechanisms of their coexistence.

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FOOTNOTES

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