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Primary seminal vesicle adenocarcinoma: clinical and pathologic features

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ABSTRACT

Primary seminal vesicle adenocarcinoma (PSVA) is an extremely rare neoplasm with an unknown pathogenesis. Prompt diagnosis remains challenging, as most patients present with non-specific genitourinary symptoms, including hematuria, hematospermia, and weak urinary stream. We present a case of PSVA in a 51-year-old man who presented with hematuria and intermittent hematospermia. Imaging revealed a large, heterogeneous mass expanding the right seminal vesicle; subsequent histopathologic examination following robotic excision of the mass confirmed the diagnosis of PSVA. Approximately 20 months postoperatively, the patient developed a recurrence in the right ejaculatory duct. Given that most cases of PSVA have a poor prognosis due to delayed diagnosis, this case report aims to highlight key clinical and pathologic diagnostic clues to facilitate an earlier intervention.

Keywords: Seminal vesicle, adenocarcinoma, genitourinary, neoplasm, urology, pathology.

Type of Article: CASE REPORT

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Introduction

In 1925, the first case of PSVA was reported by Oliver Lyons in France [1]; since then, only approximately 100 cases have been reported, highlighting the rarity of this neoplasm [2]. While the average age at diagnosis is 63 years, reported cases have involved patients ranging from 19 to 90 years of age [3]. Currently, associated risk factors for PSVA remain unknown. This entity is usually asymptomatic in its early stages, however, it can present with non-specific lower urinary tract symptoms such as hematuria, nocturia, and hematospermia. Due to this lack of symptoms, most cases present as locally advanced disease with direct invasion into neighboring organs, such as the prostate gland, or with distant metastasis at the time of diagnosis [4].

Secondary involvement of the seminal vesicles by metastatic disease, predominantly prostatic adenocarcinoma, is significantly more common than PSVA. Imaging studies, such as computed tomography (CT) scans or magnetic resonance imaging (MRI), can help characterize the mass and identify possible distant metastases [5], although, histopathologic examination is essential for making a definitive diagnosis as well as excluding metastatic disease from adjacent organs like the prostate, bladder, and rectum. The prognosis is generally poor once the diagnosis is made and approximately 95% of patients die within 3 years of diagnosis [6].

Case Presentation

A 51-year-old man presented to the emergency department with a two-month history of hematuria, weakness, and mild lower urinary tract symptoms, including increased urinary frequency, weak urinary stream, intermittent hematospermia, and mild perineal/suprapubic discomfort. His vital signs and physical examination were unremarkable. A point-of-care ultrasound (POCUS) scan noted a mass posterior to the bladder. Subsequent computed tomography (CT) scan of the abdomen and pelvis revealed an extensive heterogeneous tubular mass within the right hemipelvis measuring 16.7 cm in greatest dimension. The mass was distended with fluid, exhibited heterogeneous internal components, and appeared to originate from the right seminal vesicle (Figure 1). The differential diagnosis at that time included infection, inflammation, or an underlying malignancy of the right seminal vesicle. The patient was referred to the Departments of Urology and Oncology for further management.

A CT-guided biopsy was performed, revealing a spindle cell and epithelial proliferation (Figure 2). On immunohistochemistry (IHC), the spindle cells

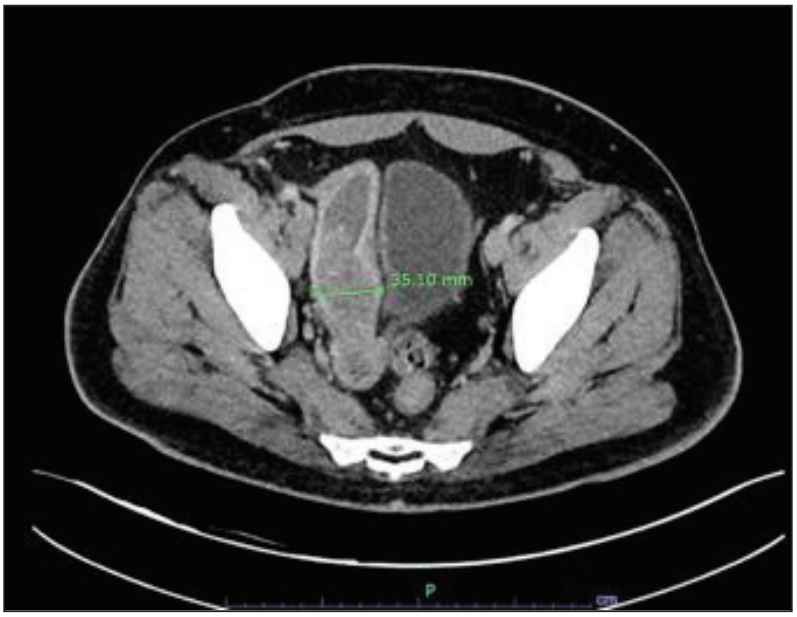


Figure 1. *CT of the abdomen and pelvis showing a large heterogeneous tubular mass within the right hemipelvis measuring up to 16.7 cm in the region of the right seminal vesicle.*

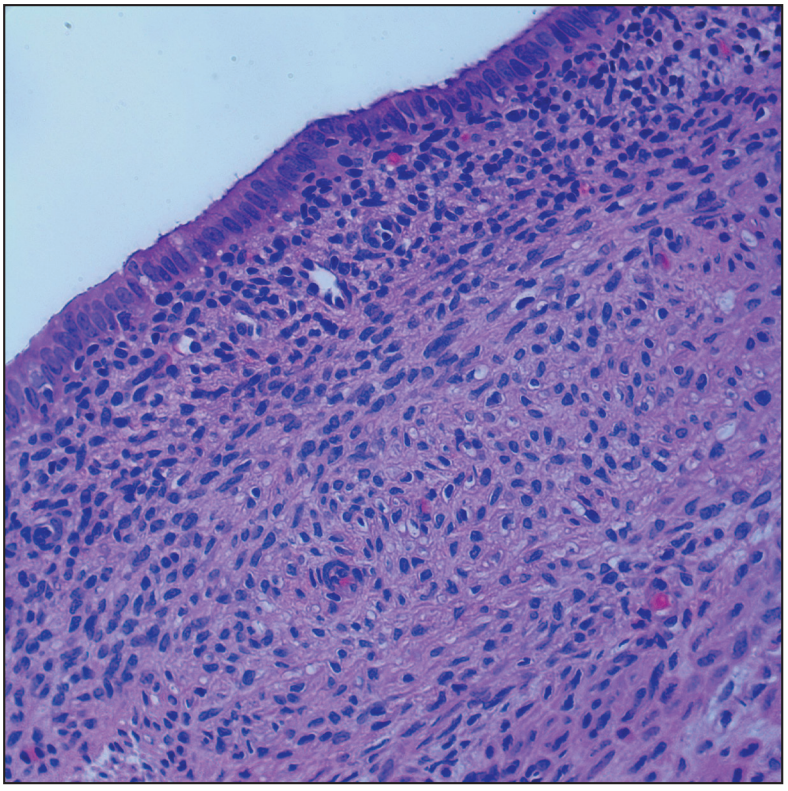


Figure 2. *Biopsy of the seminal vesicle mass showing an epithelial proliferation composed of cuboidal to low columnar cells without significant atypia associated with a stromal spindle cell proliferation with focal condensation around the epithelium. Mitotic activity or necrosis were absent (H&E, 20 and 40x objectives, respectively).*

were positive for vimentin, estrogen receptors (ER), progesterone receptors (PR), desmin, and actin-1A4 (SMA), while negative for CD34 (Figure 3). Based on these findings and the histologic features, a diagnosis of a mixed epithelial and stromal tumor (MEST) was favored.

The patient subsequently underwent surgical excision of the right seminal vesicle mass. Gross examination

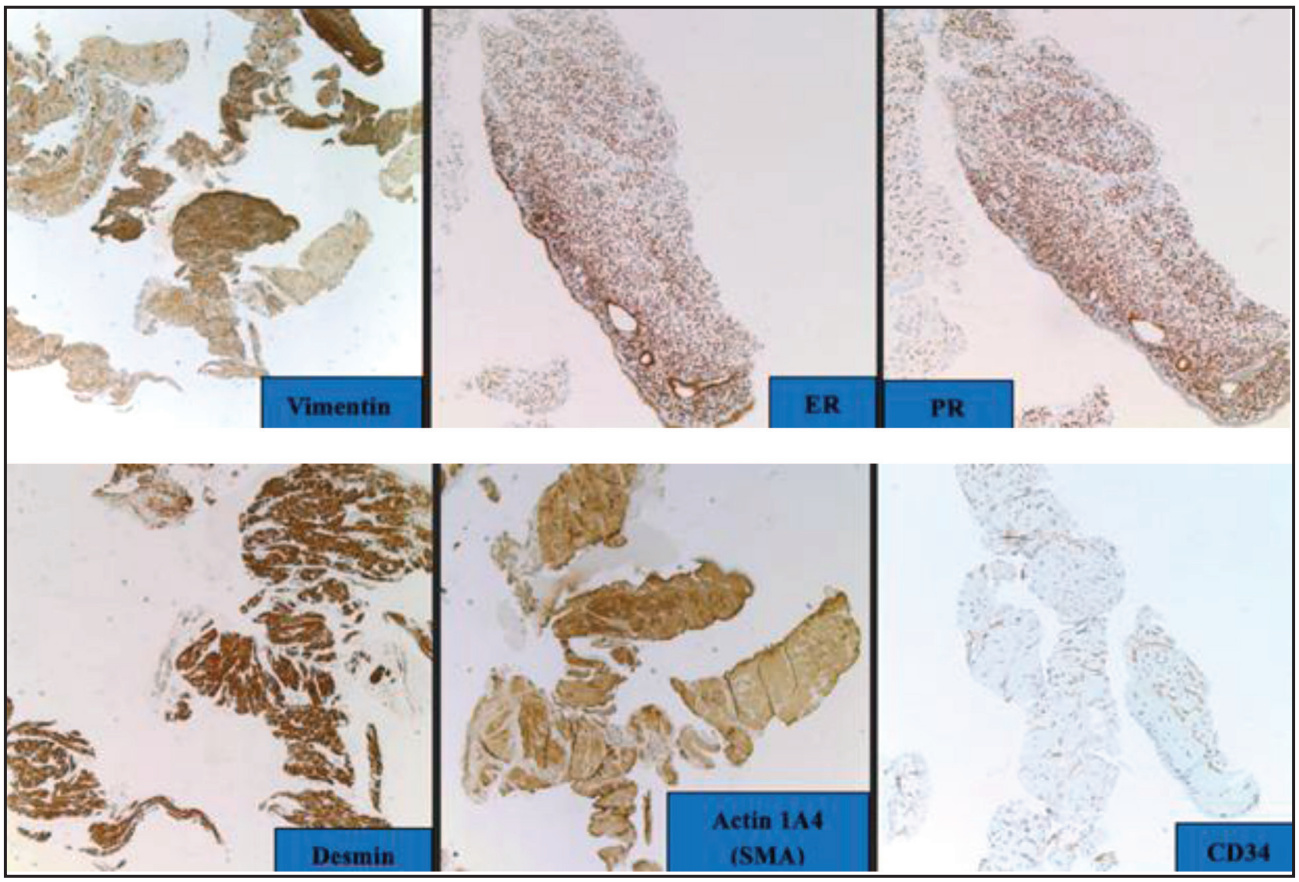


Figure 3. *The spindle cells on the biopsy showed positive staining for vimentin, ER, PR, desmin, and actin-1A4 (SMA) and negative for CD34.*

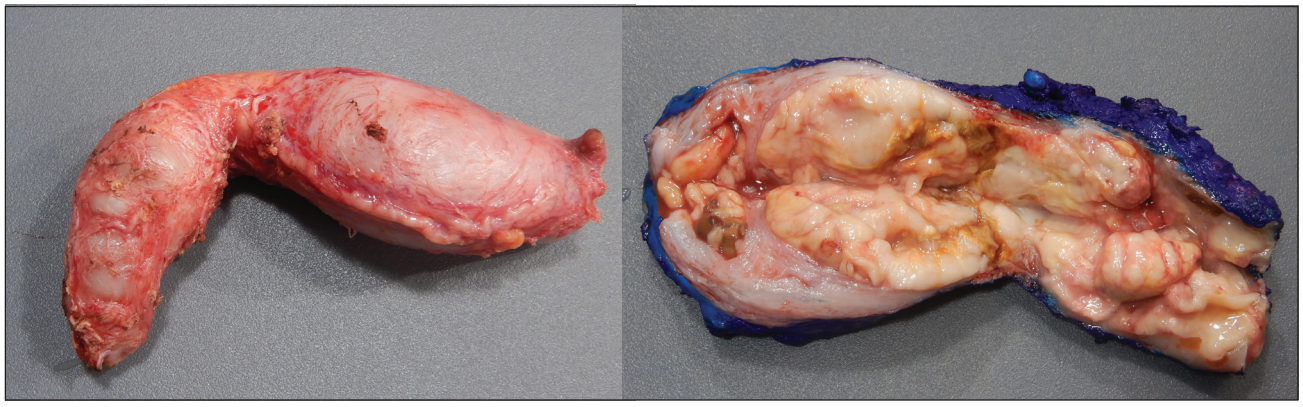


Figure 4. *Gross examination of the seminal vesicle mass revealed an elongated tan-pink mass measuring 14 cm in greatest dimension.*

revealed an elongated, tan-pink, soft tissue mass measuring 14 cm in greatest dimension. Sectioning revealed a lobulated, rubbery cut surface with areas of necrosis (Figure 4).

On histopathology, the tumor showed solid areas with focal gland formation associated with normal seminal vesicle epithelium (Figure 5).

On immunohistochemistry, the tumor cells were positive for PAX-8, CK7, and CEA (focal), while negative for PSA, PSAP, p63, GATA3, androgen, and CK20 (Figure 6). NKX-3 was also negative (not pictured).

In conjunction with the histologic features, this immunoprofile supported the diagnosis of PSVA. The patient completed three cycles of cisplatin and paclitaxel. Approximately 1.5 years following initial surgical excision, a surveillance MRI showed a 2.5 cm mass in the right ejaculatory duct, suggestive of recurrence. A radical prostatectomy was performed, and microscopic examination revealed an adenocarcinoma with similar morphology (Figure 7) and identical immunoprofile as the PSVA previously diagnosed (Figure 8). The recurrence did not show invasion into the prostatic stroma or the bladder

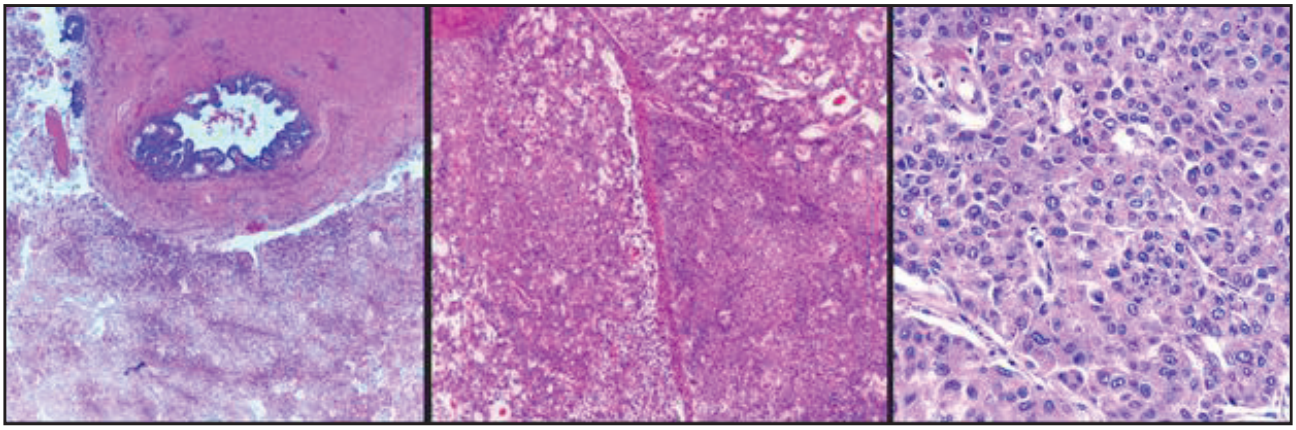


Figure 5. *Microscopic examination revealed malignant cells forming glands along with normal seminal vesicle tissue. A solid component was also identified within the tumor (H&E, 5x, 20x, and 40x objectives, respectively).*

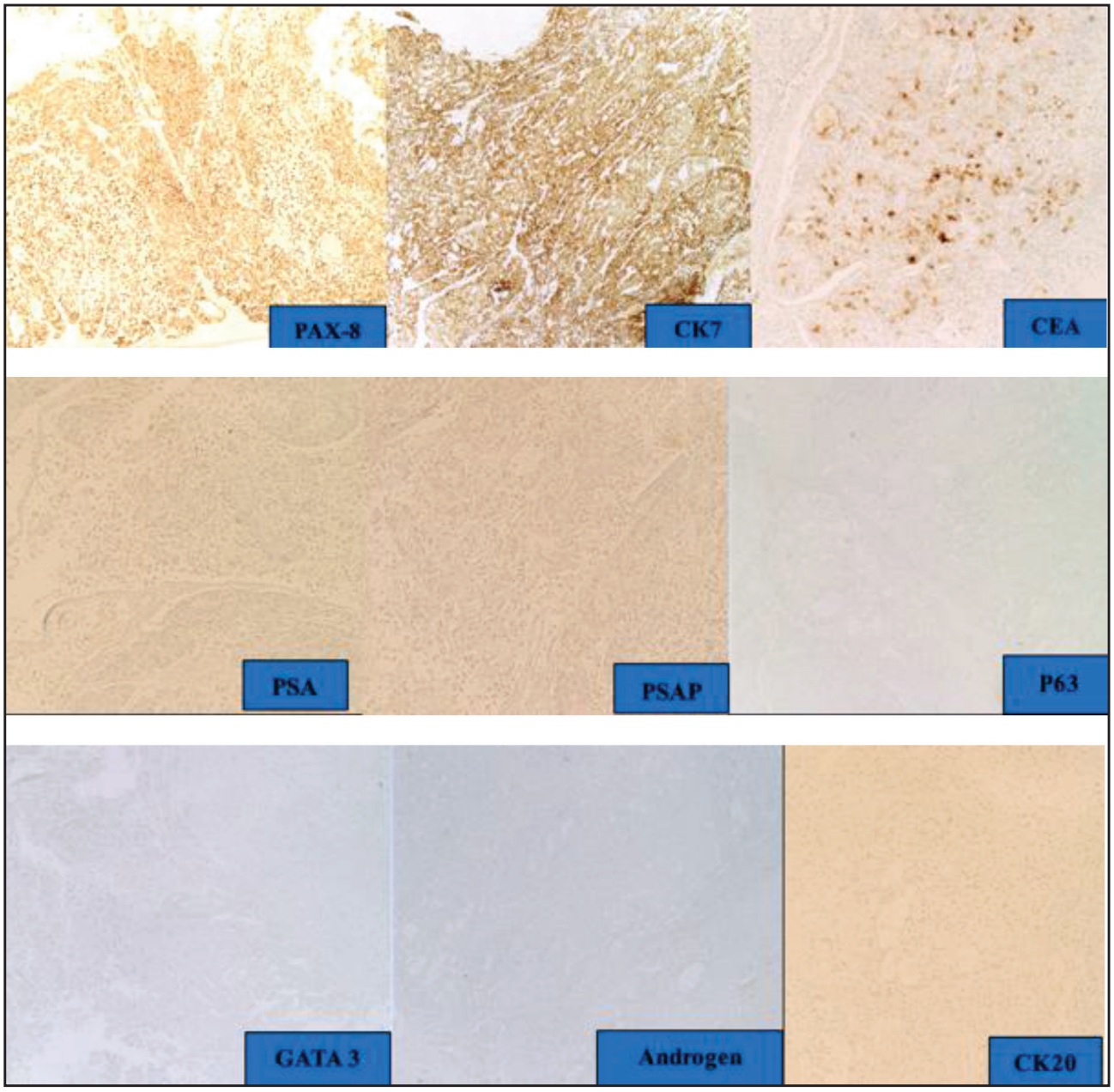


Figure 6. *The tumor cells were positive for PAX-8, CK7, and CEA (focal), while negative for PSA, PSAP, p63, GATA3, androgen, and CK20.*

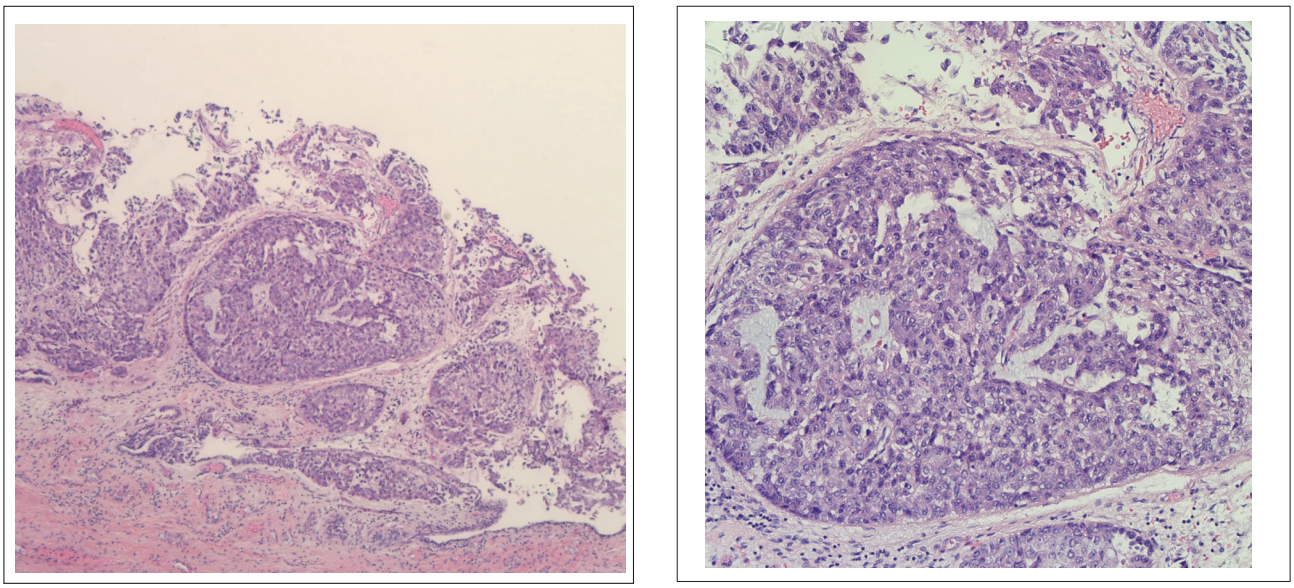


Figure 7. *Microscopic examination of the radical prostatectomy revealed an adenocarcinoma with the same morphology as the one previously diagnosed in the right seminal vesicle resection (H&E, 5x and 20x objectives, respectively).*

neck, and perineural invasion was not identified. Although the patient currently appears to be asymptomatic, the multidisciplinary tumor board recommended radiotherapy as a potential treatment for the recurrence. However, further treatment has not yet commenced as the patient has missed subsequent oncology appointments.

Discussion

In the present case, the biopsy performed prior to the seminal vesicle excision showed only a spindle and epithelial cell proliferation, suggestive of a MEST. This entity is characterized by a benign epithelial component and a stromal component that can range from benign to malignant. Given that malignant cells were absent in this initial biopsy, the tissue sampled likely represents an area of reactive stromal changes adjacent to the actual PSVA.

Grossly, PSVA may present as a solid mass, as observed in our case, or as a cystic lesion. Microscopically, most documented cases are categorized as papillary adenocarcinomas, although they may show nested, solid, or cord-like growth patterns in poorly-differentiated tumors [7]. In our case, the solid pattern was identified.

Differentiating PSVA from metastatic involvement of the seminal vesicles is one of the key tasks for pathologists when evaluating a seminal vesicle mass. While there is no IHC stain uniquely specific for PSVA, a panel of markers is instrumental in narrowing the diagnosis. To exclude prostatic adenocarcinoma, prostate-specific (PSA), and prostate-specific acid phosphatase (PSAP) can be used as most prostatic carcinomas express these markers. Meanwhile, the use of cytokeratin 7 (CK7) and cytokeratin 20 (CK20) in tandem can play an essential role in differentiating primary seminal vesicle adenocarcinoma from metastases [8]. Seminal vesicle adenocarcinoma is positive for CK7 and negative for CK20, while colorectal adenocarcinoma demonstrates a reverse pattern of staining. In contrast, urothelial carcinoma of the bladder is positive for both CK7 and CK20. Cancer antigen-125 (CA125), which is positive in PSVA, can also help differentiate PSVA from prostatic, bladder, and colorectal adenocarcinomas since those tumors are usually negative [9]. In a case of PSVA published by Ohmori et al., the tumor was a CA125-producing adenocarcinoma, and the patient presented high serum levels of this marker [10]. Bladder adenocarcinomas and mesotheliomas may also enter into the differential diagnosis. Negative immunoreactivity with GATA-3 and CK20 would exclude bladder adenocarcinoma, while negative staining with WT1 would help exclude mesothelioma.

Currently, there is limited consensus about patient treatment, management, and follow-up. Standard treatment guidelines are not currently available. Once the diagnosis is confirmed microscopically, patients should be managed by a multidisciplinary team [11]. Most cases have been managed with some form of combination of radical surgery, radiation, chemotherapy, and androgen deprivation therapy [11]. In our case, the patient was treated with a combination of surgery and chemotherapy.

Given the rarity of PSVA, every documented case serves as a vital contribution to the limited body of clinical evidence. Our case reinforces the diagnostic necessity of a broad IHC panel, specifically the CK7+/CK20-/CA125+ signature, to reliably distinguish this malignancy from more common entities. Our findings provide clinicians and pathologists with a refined diagnostic framework and

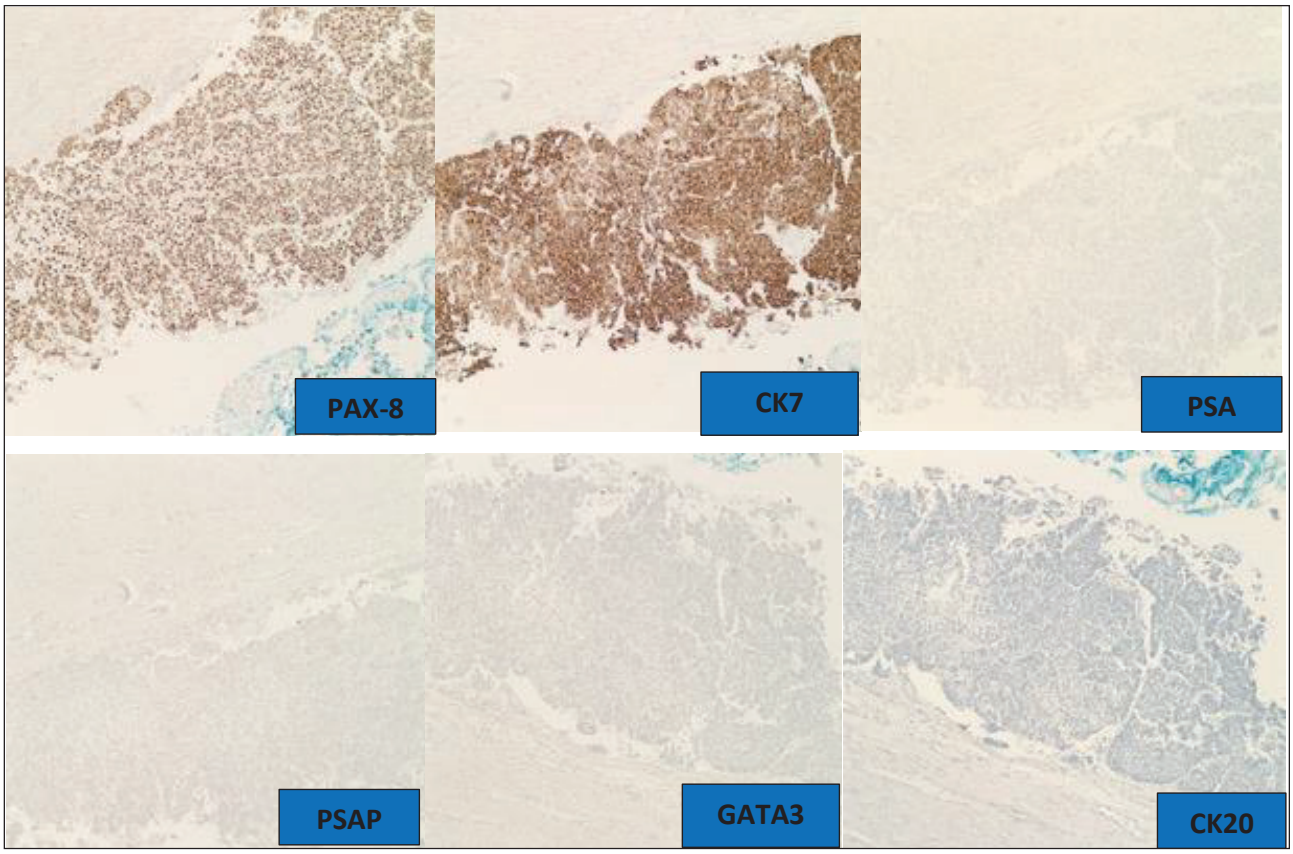


Figure 8. *The tumor cells in the recurrence had the same immunoprofile as the previously diagnosed PSVA.*

support the continued exploration of aggressive, multidisciplinary interventions for PSVA.

Conclusion

Seminal vesicle adenocarcinoma is an extremely rare malignancy; however, it must be considered in the differential diagnosis of pelvic genitourinary tumors in men. Most cases are diagnosed at an advanced stage of the disease because early clinical signs and symptoms are usually absent or nonspecific. The inclusion of seminal vesicle adenocarcinoma in the differential diagnosis of non-specific lower urinary tract symptoms can ensure an earlier diagnosis. Secondary involvement of the seminal vesicles by adenocarcinomas arising in adjacent organs, such as the prostate and colon, occurs far more commonly and, therefore, metastatic disease should be considered in the differential diagnosis.

For challenging cases, immunohistochemistry can assist in differentiating metastatic tumors from PSVA. Both diagnosis and differentiation of PSVA from colorectal, bladder, and prostate carcinomas can be greatly aided by immunohistochemical investigation. Immunohistochemical staining with CA-125 can help in the distinction of PSVA from prostatic, bladder, and rectal tumors, since these carcinomas are usually negative. Measurement of serological CA-125 in the clinical evaluation of men presenting with pelvic masses presumed to have a genitourinary origin is a possible recommendation, considering production of CA-125 by PSVA has been described in the literature.

Due to the delayed diagnosis of seminal vesicle adenocarcinoma, the prognosis is poor in most cases. Most cases are treated with radical surgery. However, the complex anatomy of the area, coupled with the frequent delay in diagnosis, increases the chance of residual disease, as well as the need for adjuvant chemotherapy/radiation.

What is new?

PSVA is a rare malignancy that typically presents with non-specific genitourinary symptoms, often resulting in most cases being diagnosed at an advanced local stage. The underlying pathogenesis remains poorly understood. This case expands the limited literature by highlighting the clinical presentation and diagnostic challenges associated with this entity. It further proposes a practical diagnostic framework to aid pathologists in the accurate identification of PSVA.

List of Abbreviations

CA-125	Cancer antigen 125
CEA	Carcinoembryonic antigen
CT	Computed tomography
ER	Estrogen receptor
MEST	Mixed epithelial and stromal tumor
MRI	Magnetic resonance imaging

PR	Progesterone receptor
PSA	Prostate-specific antigen
PSAP	Prostatic acid phosphatase
PSVA	Primary seminal vesicle adenocarcinoma
SMA	Smooth muscle actin

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Ethical approval

The Institutional Review Board at Mount Sinai Medical Center granted approval and waived patient consent. For more information, please contact Juan Sabater, MD, Institutional Official for Research (email: Juan.Sabater@msmc.com /telephone number: 305-674-2790).

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Summary of the case

Patient (gender, age)	51 years old, man
Final diagnosis	Primary seminal vesicle adenocarcinoma
Symptoms	Hematuria, weakness, increased urinary frequency, weak urinary stream, intermittent hematospermia, and mild perineal/suprapubic discomfort
Medications	Losartan, Amlodipine, Hydrochlorothiazide and Glipizide
Clinical procedure	Robotic-assisted right seminal vesicle mass excision
Specialty	Pathology and Urologic Oncology