



Primary Signet-Ring Cell Adenocarcinoma of the Bladder Presenting with Severe Obstructive Uropathy and Mimicking Colorectal Carcinoma: A Rare Case Report

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Abstract

Primary adenocarcinoma of the bladder is an extremely rare form of urinary malignancy, accounting for less than 2% of all bladder cancers. Unlike urothelial carcinoma, adenocarcinomas commonly originate from the glandular cells lining the bladder, which is frequently associated with cystitis glandularis, demonstrating a large variety of growth patterns (enteric, mucinous, clear cell, and signet ring). Signet ring cell carcinoma of bladder, which is rarely reported, is always considered as the most aggressive urinary malignancy with the shortest 5 year survive rate. Here, we report a 48-year-old man who presented to our emergency room with one month of gross hematuria and severe obstructive renal failure. Computed tomography demonstrated a mass-like thickening at the posterior bladder wall with bilateral obstructive hydroureteronephrosis, as well as iliac and inguinal lymphadenopathy. A cystoscopy and transurethral bladder tumor resection was performed that detected a 6 cm mass at posterior bladder wall tumor involving the trigon. Pathology examination reveals signet-ring carcinoma diffuse invading the muscularis propria. A scant fragment of adenomatous mucosa was noted. Immunohistochemistry stains showed that the tumor cells were positive to CDX2 and CK20, and that they were CK7, GATA3, NKX3.1, and TTF-1/Napsin-A. β -catenin showed a membranous, not nuclear/cytoplasmic, staining pattern. These findings support a diagnosis of signet-ring carcinoma of bladder, although a more definitive diagnosis is pending for colonic endoscopic examination and further imaging study.

Introduction

Adenocarcinoma in the bladder is extremely rare that is commonly divided as primary or secondary malignancy. Primary adenocarcinoma of the bladder, originated from bladder glandular epithelium, only accounts for a very small minority of

bladder malignancies. Based on morphologic features, it can be classified as enteric, mucinous, signet-ring, and mixed subtypes [1]. The signet-ring variant is less common with most aggressive behavior and shortest 5 year survive rate. A 2024 systematic review pooling 77 cases reported a median age of 64 years and a five-year survival of only 9.4% [2].



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Since primary bladder adenocarcinomas normally demonstrate morphological and phenotypic features that are overlapping with adenocarcinoma arising from some other organs (such as gastrointestinal, lung, gynecologic, ect), a final diagnosis should be made without extensive studies to exclude possible metastatic carcinoma (i.e. secondary malignancy) [1,3].

In this case, we reported a signet-ring carcinoma detected in the Transurethral Bladder Tumor Resection (TUBR) specimen collected from a relatively young male patient. Although the morphologic and phenotype features raising the concern for colorectal origin, the findings of scant adenomatous fragment and β -catenin membranous stain pattern, support the diagnosis of bladder primary. Molecular studies have been requested for further investigation and treatment options.

Case

A 48-year-old man presented to our hospital with approximately one month of intermittent gross hematuria that progressed to the passage of clots, with acute worsening and dysuria. His medical history included hypertension, non-insulin-dependent diabetes mellitus, hyperlipidemia, heart failure with preserved ejection fraction, benign prostatic hyperplasia, chronic kidney disease stage 3, and bilateral hydronephrosis. He was a heavy smoker with smoking history of 43.5 pack-years. No documented personal history of malignancy was seen.

On admission, he was found to have severe obstructive renal failure (Table 1). Computed Tomography (CT) (without contrast due to renal impairment) demonstrated severe bilateral hydro-ureteronephrosis, a posterior bladder wall mass extending into the trigon that was radiographically indistinguishable from the prostate, and bulky iliac and inguinal lymphadenopathy (external iliac node 3.2×2.8 cm; iliac node 3.5×1.9 cm). No bowel wall thickening or hepatic lesion was identified.

After bilateral percutaneous nephrostomy tubes placement, a substantial recovery of renal function was achieved. Seven weeks later, cystoscopy with Transurethral Bladder Tumor Resection (TUBR) identified a 6 cm mass on the posterior bladder wall and trigone region that invades / encases the prostatic urethra.

Histological examination of resected bladder mass revealed a poorly differentiated adenocarcinoma with diffuse signet-ring cell morphology that invaded into the muscularis propria (Figure 1). No conventional urothelial carcinoma, carcinoma in situ was identified. One tissue fragment with adenomatous features was also noted.

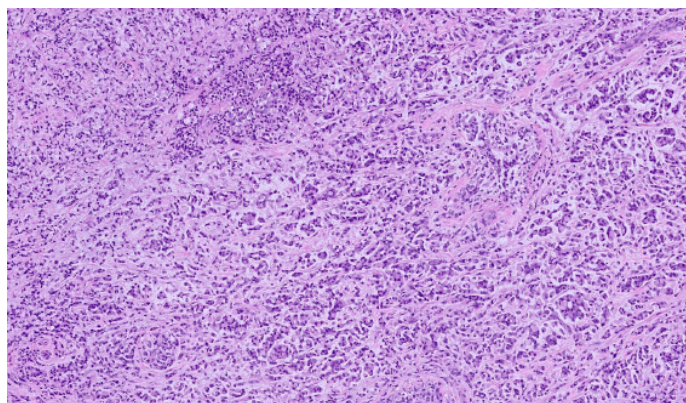


Figure 1: Photomicrograph of the transurethral bladder resection specimen. Histology examination showed poorly differentiated adenocarcinoma with signet-ring features. (H&E, magnification ×100.)

Immunohistochemical studies were performed. The tumor cells were strong positive for CDX2 and CK20 (Fig 2A, 2B). β -catenin showed a membranous (not nuclear) staining pattern (Figure 2C). These cells were negative for CK7, GATA3, NKX3.1, and TTF-1/Napsin-A (Figure 3).

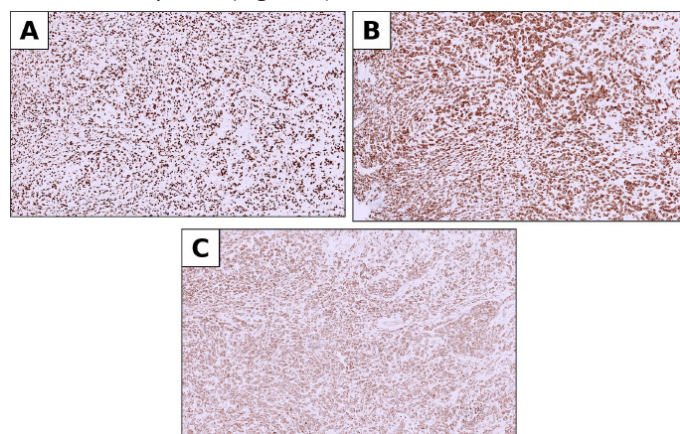


Figure 2: Immunohistochemical stains showed diffuse positivity for CDX2 and CK20 (A,B). β -catenin (C) demonstrated a membranous staining pattern without nuclear reactivity (magnification×100.).

Given the extent of local invasion and the radiographic lymphadenopathy, the tumor was considered at least clinical stage III (cN2–N3), pending complete staging. Positron emission tomography/CT was planned in view of the incomplete (noncontrast) initial study and the degree of renal dysfunction. The patient was evaluated by medical oncology, with definitive management planned following completion of staging and multidisciplinary tumor board review. Next-generation Sequencing (NGS), HER2 immunohistochemistry, and microsatellite instability/mismatch repair (MSI/MMR) testing were requested but had not resulted at the time of this report.

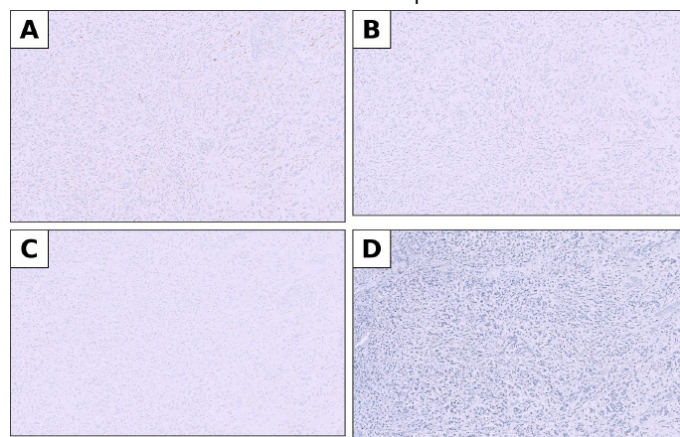


Figure 3: Immunohistochemical stains showed that the tumor cells were negative for CK7 (A), GATA3 (B), NKX3.1 (C), and TTF-1/Napsin-A (D) (magnification×100.).

Table 1: Selected laboratory and renal function values at presentation.

Parameter	Value
Serum creatinine (presentation / peak / baseline)	13.6 / 15.29 / ~1.01–1.29 mg/dL
Estimated GFR	4 mL/min/1.73m ²
Blood urea nitrogen	97 mg/dL
Hemoglobin	9.9 g/dL

Table 2: Immunohistochemical profile of the transurethral resection specimen.

Marker	Result
CDX2 / CK20	Positive
CK7 / GATA3 / NKX3.1	Negative
TTF-1 / Napsin-A	Negative
β-catenin	Membranous only; nuclear negative
CD31 / D2-40	Suspicious for lymphovascular invasion

Discussion

Primary bladder adenocarcinoma is traditionally divided into urachal and non-urachal disease. The signet-ring subtype, although uncommon, carries the worst prognosis, with a pooled five-year survival of 9.4% in the recent report. A significantly worse outcome is seen for these cases with pure signet ring morphology compared to the mixed morphology subtype. The same poor outcome is also seen in the non-urachal adenocarcinoma when compared with the urachal originated counterpart [2,4]. Additionally, our patient was diagnosed at 48 years old, a much younger age than the median ages reported in both a classic 12-case series (58 years) [4] and the 2024 pooled review (64 years) [2].

The central diagnostic problem in this case was that CDX2 and CK20 positivity is classically associated with colorectal origin, and metastatic colorectal signet-ring adenocarcinoma is a well-recognized mimic of primary bladder adenocarcinoma. Morphologic and phenotypic features overlap substantially between the two, and there is no definitive marker to separate these two [3]. There were several features nonetheless favored a primary bladder origin in this case. First, CT did not identify a colorectal mass or colonic wall thickening. Second, the tumor was found on posterior wall and trigon, a location unfavorable for urachal adenocarcinoma. Additionally, a small tissue fragment with adenomatous change was found within the tumoral area, suggesting the bladder primary. Finally, β-catenin immunohistochemical stain showed a membranous staining pattern, which is seen in bladder carcinoma [3]. Nevertheless, a colonic endoscopy and imaging study are highly recommended for the confirmative diagnosis.

Clinically, this case was notable for the severity of its presentation. Trigonal obstruction produced bilateral hydronephrosis and severe obstructive acute kidney injury, with a creatinine of 15.29 mg/dL, before any tissue diagnosis had been established. This is consistent with the aggressive local behavior reported for pure and diffuse signet-ring histology [2,4]. NGS, HER2, and MSI/MMR testing were requested but unresulted at the time of this report; molecular characterization of bladder and urachal adenocarcinoma is an area of growing interest that may eventually inform both diagnosis and therapy in these uncommon tumors [6].

Conclusion

The diagnosis of primary signet ring carcinoma of bladder is challenging due to its overlaying features with colorectal carcinoma. The work up includes histology examination of the

presence of adenomatous component, immunohistochemical stain of β-catenin, endoscopic evaluation of tumor location, and additional studies to exclude other primary malignancies. Accurate diagnosis is crucial to distinguish primary bladder adenocarcinoma from secondary spread, as the treatment approaches are vastly different.

Author declarations

Author contributions

Asad Mehmood and Hassan Syed contributed equally to this work and are designated co-first authors. Asad Mehmood and Hassan Syed: Involved in literature review, data collection, and manuscript drafting and revision. Haoliang Xu: Involved in pathological diagnosis, image acquisition, intellectual supervision, and critical revision of the manuscript. Asad Mehmood: Corresponding author; responsible for manuscript submission and correspondence. All authors read and approved the final manuscript.

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Conflicts of interest

All authors declare that they have no potential conflict of interest.

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Consent

Written informed consent for publication was obtained from the patient. [Confirm status before submission.

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