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Bladder Metastasis from Gastric Signet-Ring Cell Adenocarcinoma: A Report of Two Cases

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Abstract

Although primary bladder cancer is the second most common urological cancer after prostate cancer, bladder metastases are rare, and those originating from the stomach are exceptional. Secondary bladder metastases of signet ring cell carcinoma (SRCC) are an exceptional occurrence, presenting considerable diagnostic and therapeutic challenges. The aim is to present two rare cases of secondary bladder tumors arising from primary gastric tumors in order to better characterize their clinicopathological features and diagnosis. We report two cases of patients aged 58 and 21, respectively, presenting with macroscopic hematuria and acute obstructive renal failure, with abnormalities suspected during cystoscopy. Bladder biopsy, accompanied by histopathological analysis, revealed a poorly differentiated signet ring cell adenocarcinoma, linked to the previously identified gastric lesion. The patients were referred for chemotherapy, and one died six months after diagnosis. Bladder metastasis from gastric signet-ring cell carcinoma is extremely rare, but it should be considered when patients with gastric signet-ring cell carcinoma develop urinary symptoms.

Keywords

Bladder Metastasis, Gastric Adenocarcinoma, Signet Ring Cells, SRCC

1. Introduction

Bladder cancer is the ninth most common cancer worldwide, with approximately 614,000 new cases and 220,000 deaths estimated in 2022 [1]. In France, the number of new cases of invasive bladder cancer was estimated at 13,074 in 2018, with 5335 deaths [2]. Bladder metastases are rare: in the largest published series of sec-

ondary bladder neoplasms, they represented 2.3% of all bladder neoplasms examined, and the stomach was the primary site in 4.3% of them [3]. Among these, gastric signet-ring cell carcinomas (SRCCs) represent an exceptional presentation, posing major diagnostic and therapeutic challenges. These tumors are characterized histologically by intracytoplasmic mucin displacing the nucleus to the periphery, giving them the characteristic "signet ring" appearance [4]. It is important to distinguish this form from signet ring cell primary bladder adenocarcinoma, which is itself extremely rare. Given the rarity of bladder metastasis from gastric SRCC, we report two cases to better characterize their anatomoclinical, radiological, and therapeutic features.

2. Case Presentations

2.1. Case 1

This is a 58-year-old hypertensive patient with a history of smoking (60 pack-years) and abstinence from alcohol. He was hospitalized in nephrology for malaise, difficulty swallowing, and abdominal pain, with a decline in his general condition that had been progressing for 3 months (WHO score 2). Urological examination revealed paroxysmal, non-clotting macroscopic hematuria. Digital rectal examination showed a small, firm prostate with an infiltrated bladder base. Laboratory tests revealed acute kidney injury (KDIGO 1) with a serum creatinine level of 100 µmol/L.

Uro-CT scan revealed moderate bilateral ureterohydronephrosis proximal to bladder wall thickening (**Figure 1**).



Figure 1. Bladder wall thickening.

Gastroscopy revealed a suspicious ulcerated subcardial lesion. Multiple biopsies confirmed an invasive gastric adenocarcinoma with poorly differentiated signet ring cells. We noted an absence of HER2 expression and positive PD-L1 expression (20%).

Cystoscopy had shown diffuse inflammatory lesions predominating at the level of the bladder dome without any detectable polyps and non-visualized ureteral orifices (**Figure 2**). There was no urinary drainage.

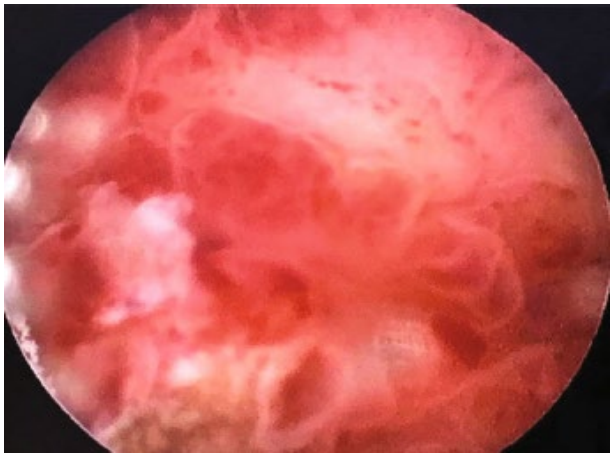


Figure 2. Macroscopic appearance of an inflammatory bladder lesion.

Bladder biopsy by transurethral resection of the bladder with image optimization technique (HEXVIX) and histopathological examination concluded that the bladder mucosa was invaded by signet ring cell adenocarcinoma, the mode of infiltration of which appeared to be secondary, related to the known gastric lesion, rather than primary (**Figure 3**). Furthermore, the presence of intracellular mucus, the negative GATA3 test (**Figure 4**), and the absence of carcinoma *in situ* (CIS) were compatible with, but did not by themselves establish, a non-urothelial origin.

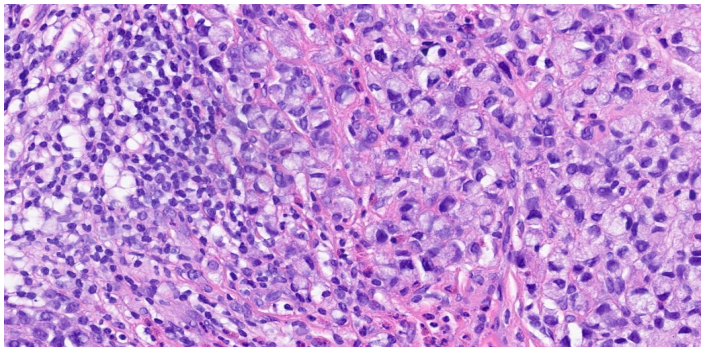


Figure 3. The bladder mucosa is infiltrated by tumor cells with large, hyperchromatic nuclei pushed to the periphery by abundant cytoplasm, giving them a "signet ring" appearance.

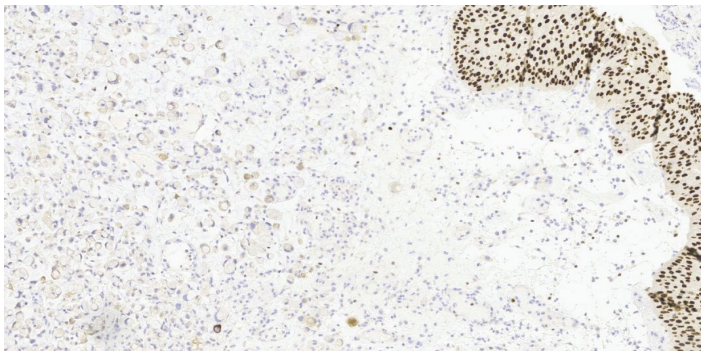


Figure 4. Immunohistological examination of GATA3, absence of nuclear staining.

The PET scan revealed diffuse thickening of the gastric walls and the lower third of the esophagus, moderately hypermetabolic (SUVmax 4.3 in the gastric walls and SUVmax 5.7 at the esophagogastric junction). There was a small amount of fluid with low-metabolic abdominopelvic peritoneal thickening. No other suspicious hypermetabolic foci were identified. Given the peritoneal thickening with fluid, the infiltrated bladder base on rectal examination, and the absence of other hypermetabolic foci, peritoneal (transcoelomic) dissemination with secondary invasion of the bladder wall is the most plausible route of bladder involvement in this patient; hematogenous or lymphatic spread cannot be formally excluded.

Following a multidisciplinary tumor board meeting, initial chemotherapy was decided upon. The patient was referred to oncology. The patient passed away after 11 months of chemotherapy. The protocol, however, is not known.

2.2. Case 2

A 21-year-old patient with no significant past medical history presented with bloody diarrhea, a decline in general condition (WHO 3), and acute obstructive renal failure with a serum creatinine level of 141 µmol/L.

A thoraco-abdomino-pelvic CT scan revealed gastric wall thickening, a peritoneal carcinomatosis nodule, moderate ascites, bilateral dilation of the renal pelvis and calyces, and a cluster of bilateral lateral aortic lymphadenopathy (**Figure 5**). The peritoneal carcinomatosis nodule with ascites makes peritoneal (transcoelomic) dissemination the most likely route of bladder involvement; the lateral aortic lymphadenopathy indicates retroperitoneal nodal disease but does not by itself demonstrate a lymphatic route to the bladder, and hematogenous spread cannot be assessed from the available data.

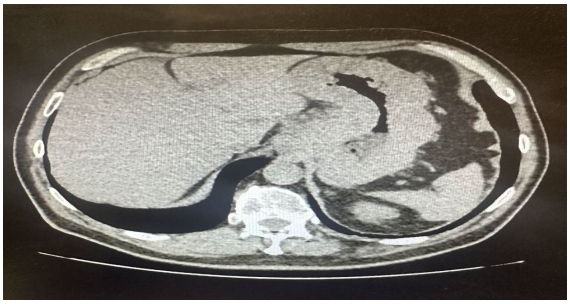


Figure 5. Gastric wall thickening.

Gastroscopy revealed an edematous and difficult-to-pass pylorus. Biopsies confirmed a poorly differentiated signet-ring cell gastric carcinoma (**Table 1**).

Table 1. Immunohistochemical results of the bladder and gastric specimens in both cases.

Marker	Case 1—bladder	Case 1—stomach	Case 2—bladder	Case 2—stomach
GATA3	Negative	Not done	Negative	Positive
CK20	Negative	Not done	Not done	Not done

Continued

CK7	Positive	Positive	Positive	Positive
p63	Negative	Negative	Negative	Negative
Uroplakin III	Not done	Not done	Not done	Not done
AE1/AE2	Positive	Positive	Positive	Positive
HER2	Not done	Negative (score 0)	Not done	Negative (score 0)
PD-L1 (CPS)	Not done	Positive (20%)	Not done	Positive (30%)
MMR/MSI	Not done	Negative	Not done	Negative

Cystoscopy performed during upper urinary tract drainage (bilateral ureteral stents) showed inflammatory lesions in the trigone, and biopsies were taken during the same surgical procedure. The results were consistent with signet-ring cell adenocarcinoma secondary to the gastric lesion.

Serum creatinine levels normalized postoperatively, and after multidisciplinary team meeting, the patient was referred to oncology for chemotherapy. The patient died six months after diagnosis from complications of the disease.

3. Discussion

Bladder metastases from gastric tumors are rare [4]; secondary neoplasms account for 1.6% - 3.0% of solid malignancies in genitourinary surgical specimens [5]. In cases of bladder metastasis from a distant primary cancer, the stomach is among the most frequently reported primary sites, together with skin (melanoma), lung, and breast [5]. Several routes of spread to the bladder have been proposed for gastric adenocarcinoma: transcoelomic (peritoneal) seeding with implantation of tumor cells on pelvic structures, lymphatic dissemination [6], and hematogenous dissemination [7]. These mechanisms are not mutually exclusive and are rarely demonstrable in an individual patient. In our patients, the peritoneal findings (peritoneal thickening with fluid in case 1; a peritoneal carcinomatosis nodule with ascites in case 2) make peritoneal dissemination with secondary invasion of the bladder wall the most plausible route. The lateral aortic lymphadenopathy in case 2 does not by itself establish a lymphatic route, and a hematogenous route cannot be proven or excluded in either patient. Gastrointestinal origin should be suspected when a bladder biopsy shows signet-ring cell adenocarcinoma; the stomach, colon, and breast are the main primary sites to exclude [8].

In the series by Ganem *et al.* (1994) [9], secondary bladder tumors represented 0.17% of cystectomy specimens, with a predominance of gastrointestinal origins. In a review of 18 reported cases of bladder metastasis from a primary gastric tumor, hematuria was the only consistent urinary symptom (12/18 patients), and the bladder metastasis could be synchronous or metachronous, with a mean interval of six years after the gastric primary [10]. The presentation is often misleading. The most frequent symptoms are macroscopic hematuria, bladder irritation, and sometimes obstructive symptoms. In some reported cases, bladder metastasis

revealed the primary tumor. In our patients, hematuria and acute obstructive renal failure were the presenting urological signs. The lesion may be single or multiple and may mimic a primary urothelial tumor [10].

The diagnosis is based on biopsy and histopathological analysis. The histological appearance shows sheets of signet ring cells with abundant intracytoplasmic mucin (PAS+, Alcian blue+) (**Figure 6**).

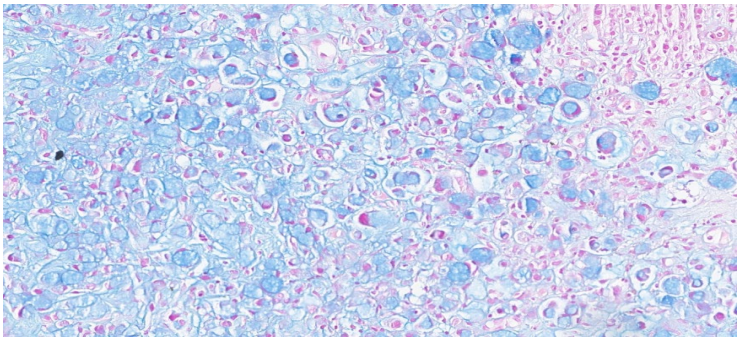


Figure 6. Alcian blue positivity.

Immunohistochemistry is essential for identifying the primary tumor. For tumors of gastric origin, positivity is observed for the markers CDX2, CK20, MUC5AC, and MUC6. For primary bladder tumors, the markers CK7, GATA3, and p63 are positive, and the marker CDX2 is negative [11]. No single marker is conclusive, since CK7 and CK20 may be variably expressed in both settings, and the assignment of origin rests on the panel as a whole in its clinical context. In our patients, the diagnosis of bladder metastasis from a gastric primary is based on the combination of the known gastric SRCC, the histology (signet-ring cells with intracytoplasmic mucin), and the immunohistochemical profile summarized in **Table 1**; negative GATA3 staining and the absence of CIS are supportive but not sufficient on their own.

It is important to distinguish this form from signet ring cell primary bladder adenocarcinoma, which is itself exceptional (<0.5% of bladder tumors), notably described by Grignon *et al.* in 1991 [12]. Several factors can help differentiate between the two: first, the clinical history, which generally guides the diagnosis, as was the case with our patients. The cystoscopic appearance can also be useful. Indeed, unlike primary bladder cancer, secondary tumors are usually solitary and most often located in the trigone or bladder neck; in our patients, the lesions predominated at the bladder dome (case 1, diffuse inflammatory lesions) and in the trigone (case 2). Histologically, however, signet ring cell bladder adenocarcinomas are indistinguishable from their counterparts in other organs.

There is no established therapeutic consensus specific to bladder metastasis from gastric SRCC. Systemic treatment is determined by the metastatic gastric primary, whereas urinary obstruction and hematuria require separate local management. Systemic treatment. In unresectable or metastatic gastric adenocarcinoma, first-line treatment is a fluoropyrimidine-platinum doublet (FOLFOX or CAPOX), se-

lected according to biomarkers [13] [14]. Trastuzumab is added in HER2-positive disease [15]; an anti-PD-1 antibody (nivolumab with chemotherapy) improved survival in untreated advanced gastric, gastroesophageal junction, and esophageal adenocarcinoma, with the greatest benefit at higher PD-L1 CPS [16]; and zolbetuximab plus mFOLFOX6 improved outcomes in CLDN18.2-positive, HER2-negative disease [17]. HER2, PD-L1 CPS, MSI/MMR, and CLDN18.2 status should therefore be documented at diagnosis [18]. These data come from trials in gastric and gastroesophageal junction adenocarcinoma and not from patients with bladder metastasis; the prognostic impact of signet-ring histology and its sensitivity to chemotherapy remain debated [19]. Local management. Obstructive uropathy from bladder involvement is treated independently of systemic therapy by urinary decompression, using retrograde ureteral stenting or percutaneous nephrostomy depending on feasibility (the ureteral orifices may not be identifiable when the bladder base is infiltrated), expected survival, and quality of life [20]. Transurethral resection serves to establish the diagnosis and may help control hematuria. (To complete: local procedures actually performed in each patient.) The prognosis is poor: in a recent single-centre series of secondary bladder neoplasms, the two patients with a gastric primary had a median survival of 66 days [21], and malignant ureteral obstruction from advanced malignancy carries a median survival of 3 to 7 months [22]. After multidisciplinary discussion, the patients were referred to oncology for chemotherapy and died 11 and 6 months after diagnosis.

4. Conclusion

Bladder metastases from signet ring cell adenocarcinomas are a rare and difficult-to-diagnose entity, requiring rigorous correlation of clinical, endoscopic, radiological, and histopathological data. Immunohistochemistry is the key tool for determining the primary origin and guiding treatment. The prognosis remains poor, and management must be part of a multidisciplinary oncological approach centered on the primary tumor, with decompression of the urinary tract when needed.

Ethics and Publication Statements

Both patients died before the preparation of this manuscript. Despite several attempts to reach their next of kin by telephone, no contact could be established, and consent for publication could therefore not be obtained. All information that could identify the patients has been removed from the text, and none of the images (CT, cystoscopy, histology) contains any element allowing their identification.

Author Contributions

Djiby Dieng, Mohamed Ben Jemaa, and Emmanuel Van Glabeke participated in patient care and the writing of this article. Abdelghani Benaida also participated in patient care. All authors contributed to this work and declare that they have read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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