

Metastatic Sigmoid Adenocarcinoma at the Right Ureter

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Abstract

Colorectal cancer and ureteral obstruction are both relatively common clinical entities. Although advancing carcinomas from neighboring organs can infiltrate and eventually block the ureter, distant ureteral metastasis from colon adenocarcinoma is a very rare condition. We present a case of a 68-year-old Caucasian female, with a right ureteral metastasis and a history of sigmoid cancer. The patient was referred to our department with asymptomatic moderate right hydronephrosis and hydroureter. She underwent right ureteroscopy, which revealed the abnormal tissue, and right nephroureterectomy. Histology showed infiltration of all layers of the ureteral wall, including the mucosa, by neoplastic cells with morphological and immunohistochemical features of colon adenocarcinoma. The patient was referred to the oncological department for oncological reevaluation.

Keywords: Colorectal cancer; Ureteral metastasis; Ureteral obstruction; Nephroureterectomy

Introduction

The obstruction of the ureter by direct invasion of retroperitoneal or pelvic tumors is relatively frequent. On the other hand, the development of a distant ureteral metastasis is an infrequent event, with only approximately 400 cases reported, from various primary tumors [1]. Distant ureteral metastases may occur via lymphatic and/or blood vessels [2, 3]. The criteria for establishing true ureteral metastasis from any cancer type, have been defined and consist of the following features: a clear distance between the primary tumor and metastasis, an identi-

cal cellularity of the two tumors, and the presence of malignant cells in the vascular and lymphatic system of the ureteral wall [2, 3]. Primary tumors have been described as these of the breast, colon, rectum, stomach, lung, cervix, prostate, pancreas, and lymphoma [3–5]. We present a case of distant ureteral metastasis from sigmoid adenocarcinoma, which is considered to be very rare [1–6].

Case Report

A 66-year-old Caucasian female with no medical history presented to the emergency department with a clinical presentation of acute abdomen, due to sigmoid colon perforation. She was admitted to the hospital and underwent urgent Hartmann's sigmoidectomy, with the formation of an end colostomy. The histological examination revealed sigmoid colon adenocarcinoma (grade II, stage pT4aN2b), with tumor infiltration in 13 of the 20 resected lymph nodes, and R0 surgical margins. The microsatellite instability and mismatch repair genes mutation tests, as well as the Kirsten rat sarcoma virus oncogene homologue (*KRAS*), neuroblastoma rat sarcoma virus oncogene homologue (*NRAS*) and v-raf murine sarcoma viral oncogene homolog B1 (*BRAF*) gene mutation tests proved negative for the aforementioned adenocarcinoma. Magnetic resonance imaging (MRI) examination of the abdomen and computed tomography (CT) examination of the chest, after the surgery, did not confirm metastatic lesions. Biomarkers carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA19-9) levels were not elevated at first diagnosis or during routine biochemical follow-up.

The patient was then referred to the oncological department and received adjuvant chemotherapy with folinic acid, 5-fluorouracil, oxaliplatin (FOLFOX) regimen, which was completed 6 months after the emergency sigmoidectomy. At the same time, she was subjected to follow-up examinations with abdomen MRI and chest CT.

During subsequent follow-up, an abnormal pelvic mass at the position of the left ovary was identified. Eleven months after the first surgery, the patient underwent resection of this pelvic mass, along with total hysterectomy with a bilateral salpingo-oophorectomy. The pathological examination confirmed left ovary metastasis of the primary sigmoid adenocarcinoma, with an R0 resection, and negative *KRAS*, *NRAS* and *BRAF* gene mutation tests. 18F-fluorodeoxyglucose positron emission tomography-computed tomography ((18F)-FDG PET-CT) performed 2 months after the second surgery (pelvic

Manuscript submitted January 28, 2026, accepted May 4, 2026
Published online May 22, 2026

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doi: <https://doi.org/10.14740/jcs1026>

Journal of Current Surgery
1927-1298 (print), 1927-1301 (online)

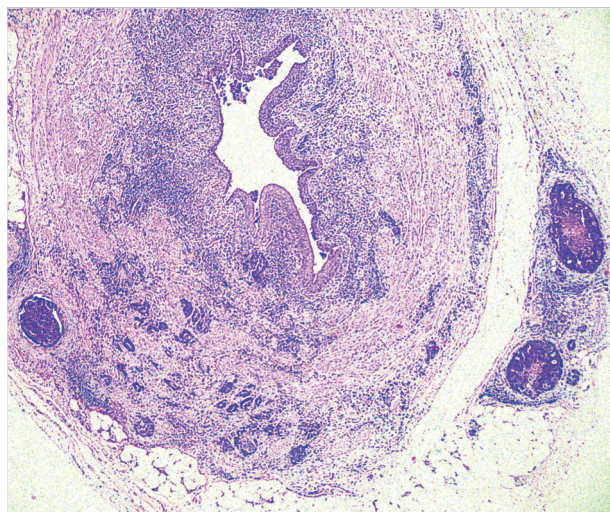


Figure 1. Neoplastic aggregates in perirenal and periureteral fat with concurrent infiltration of all ureteral layers and papillary intraluminal morphology (× 200).

mass resection) showed abnormal para-aortic and iliac lymph nodes. Consequently, following oncological evaluation, the patient was introduced to first-line chemotherapy/immunotherapy with folinic acid, 5-fluorouracil, irinotecan (FOLFIRI)/panitumumab regimen, which was completed 19 months after the first surgery (sigmoidectomy). At that time, there was radiographically complete response of the retroperitoneal lymphadenopathy.

The patient remained free of recurrent disease for the following 5 months. During follow-up, CT of the abdomen detected intraluminal lesions of the middle and lower third of the right ureter, which manifested as right moderate hydronephrosis and hydroureter. The patient had no symptoms and normal renal function. Right ureteroscopy confirmed the presence of papillary lesions in the middle and lower third of the right ureter, totally obstructing it, while urine cytology was negative for malignant cells. Twenty-six months after the resection of the primary tumor (urgent sigmoidectomy due to perforation), following discussion in the hospital multidisciplinary tumor board, the patient was subjected to right nephroureterectomy. No post-nephroureterectomy complications were observed, and the renal function remained normal. The patient recovered and was discharged without any unexpected events.

The histology identified solid and cribriform neoplastic aggregates with focal comedo necrosis involving the perirenal and periureteral fat, with concurrent infiltration of the serosa/adventitia, muscular layer, and mucosa of the ureter. In the latter, the neoplasm showed a papillary morphology consistent with ureteroscopy findings (Figs. 1, 2).

The immunohistochemistry profile of the neoplasm showed caudal-type homeobox 2 (CDX-2) (+), special adenine-thymine-rich sequence-binding protein 2 (SATB2) (+), cytokeratin 20 (CK20) (+, focal), cytokeratin 7 (CK7) (–), guanine-adenine-thymine-adenine-binding protein 3 (GATA3) (–), consistent with an adenocarcinoma of colon origin according to clinical history (Figs. 3–5).

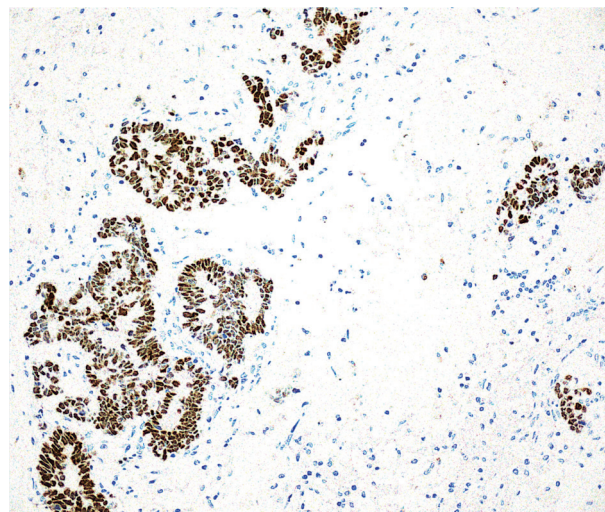


Figure 3. Immunohistochemistry profiling, CDX-2 positive (× 200). CDX2: caudal-type homeobox 2.

Following these findings, the patient was referred to the oncological department for oncological reevaluation (Table 1).

Discussion

A considerable number of colorectal cancer patients develop distant metastases [7]. Distant ureteral metastasis from primary colon adenocarcinoma is very rare and was first reported in 1936 [2]. Hu et al conducted a systematic review of ureteral involvement by metastatic malignant disease, which identified 265 cases with various cancer metastasis to the ureter. The most common sites of primary tumor included the prostate, bladder, breast, intestines, and lymphoma. Only 35 of the 265 cases involved ureteral metastasis from a small or large intestine primary cancer. In this analysis, metastatic ureteral cancer from an intestinal pri-

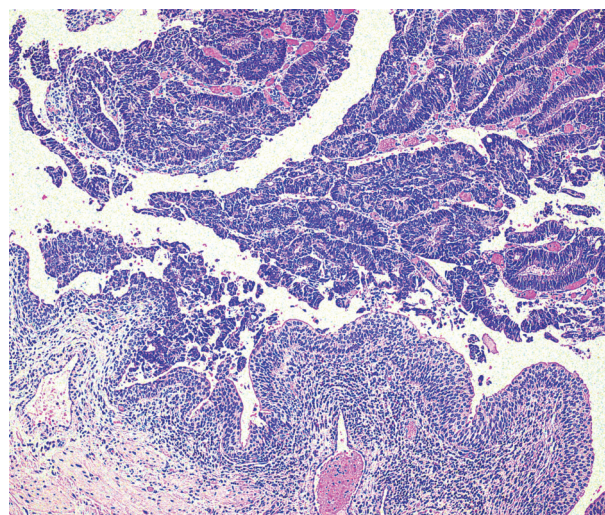


Figure 2. Solid and cribriform neoplastic aggregates focally with comedo necrosis (× 100).

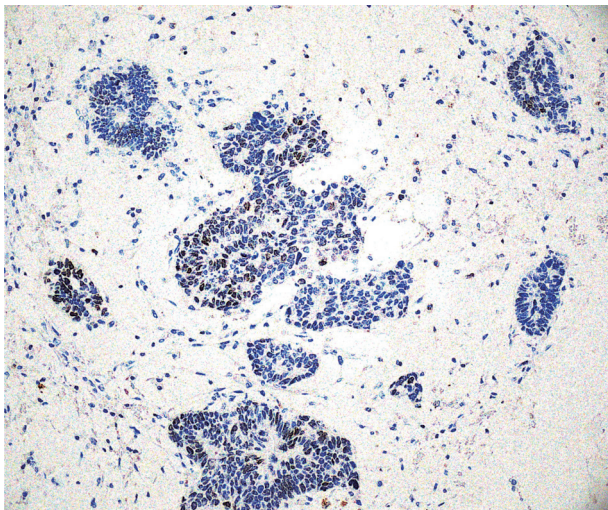


Figure 4. Immunohistochemistry profiling, SATB2 positive (× 200). SATB2: special adenine-thymine-rich sequence-binding protein 2.

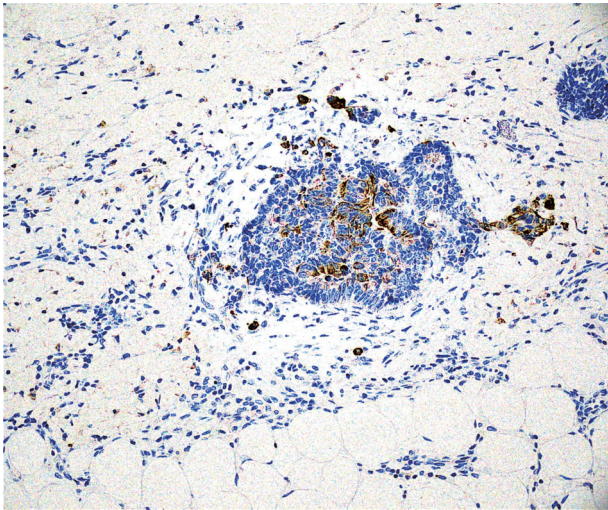


Figure 5. Immunohistochemistry profiling, CK20 positive (focal, × 200). CK20: cytokeratin 20.

mary was diagnosed at an average of 24 months after diagnosis of the primary lesion, which is consistent with our case [6].

There are three different types of metastatic cancer involvement of the ureter [4, 5, 8, 9]: 1) Type I—periureteral adventitial layer infiltration by the malignant cells, the most common one of the three types, usually resulting in compression of the ureter; 2) Type II—partial involvement of the layers of the ureteral wall, with presence of tumor cells in the muscular layer and/or transmural lymphatic/vascular involvement; 3) Type III— involvement of ureteral mucosa, with or without muscular layer, often presenting as submucosal nodules and representing the most uncommon.

Type I and type II result in ureteral stricture formation and

may even cause obstruction of the ureter with or without associated mass. Type III may manifest as intraluminal lesions seen radiographically, such as in our case [9].

According to publish data, as in our patient, the metastatic involvement of ureter is usually asymptomatic. Surgeons should receive a high index of suspicion of ureteral metastasis when hydronephrosis is identified during follow-up [10, 11].

Metastatic colon adenocarcinoma at the ureter has an aggressive progression and a poor prognosis, as depicted by the unfavorable long-term survival evidence of the few reported cases. Distinguishing primary carcinoma of the urinary tract from metastatic adenocarcinoma is essential for excellent patient care and therapy schedule. There are no standardized

Table 1. Patient Timeline

Months	Findings
0	Urgent Hartmann’s sigmoidectomy, due to perforation of the underlying sigmoid colon adenocarcinoma (grade II, stage pT4aN2b), R0 resection
1	Staging: abdomen MRI and chest CT did not confirm metastatic lesions
1–6	Adjuvant chemotherapy with FOLFOX regimen
6	Follow-up abdomen MRI and chest CT, without evidence of recurrence or progression
11	Follow-up abdomen CT revealed abnormal lesion at the left ovary
11	Total hysterectomy with a bilateral salpingo-oophorectomy, which confirmed left ovary metastasis of the primary sigmoid adenocarcinoma, R0 resection
13	Examination with 18-FDG PET-CT, which showed abnormal para-aortic and iliac lymph nodes
13–19	First-line chemotherapy/immunotherapy with FOLFIRI/panitumumab regimen
19	Follow-up abdomen and chest CT, without evidence of recurrence or progression, and with radiographically complete response of the retroperitoneal lymphadenopathy
24	Follow-up abdomen and chest CT detected intraluminal lesions of the middle and lower third of the right ureter
25	Diagnostic right ureteroscopy confirmed the presence of papillary lesions totally obstructing the ureter
26	Right nephroureterectomy, with histopathological examination confirming metastatic adenocarcinoma of colon origin, R0 resection

CT: computed tomography; FOLFOX: folinic acid, 5-fluorouracil, oxaliplatin; MRI: magnetic resonance imaging; 18F-FDG PET-CT: 18F-fluorodeoxyglucose positron emission tomography–computed tomography; FOLFIRI: folinic acid, 5-fluorouracil, irinotecan.

treatment protocols, since it constitutes a very rare condition, but chemotherapy in combination with surgical treatment forms a reasonable option [3–6, 12]. There is evidence indicating that patients who underwent surgery had better outcomes [6], which demonstrates that ureteral colon cancer metastasectomy follows the evidence of liver and lung metastasectomy from colon cancer, which improves prognosis.

Conclusions

Metastatic colon adenocarcinoma at the ureter is rarely encountered in clinical practice. However, it should be considered in the differential diagnosis, once there is clinical or radiographic evidence of ureteral obstruction in a patient, who has a history of colon cancer, regardless of the stage and previous therapy.

Acknowledgments

None to declare.

Financial Disclosure

No funding was received in regard to the production of this paper.

Conflict of Interest

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

Informed Consent

Informed consent was obtained from the patient for publication of this case report.

Author Contributions

Dr. Nikolaos Gianniris: conceptualization, literature review, data collection, data analysis, original manuscript drafting, visualization. Dr. Anastasia Therapontos: literature review, data collection, data analysis. Dr. Triantafyllos Papadopoulos: literature review, data collection, data analysis. Dr. Charalampos Kypraios: literature review, data collection, data analysis. Dr. Ioannis Xoxakos: literature review, data collection, data analysis. Dr. Charikleia Kouvidou: literature review, data collection, data analysis, manuscript review and editing. Dr. Ioannis Efthimiou: literature review, data collection, data analysis, manuscript review and editing.

Data Availability

All relevant data supporting the findings of this case report are

included in the manuscript.

Abbreviations

KRAS: Kirsten rat sarcoma virus oncogene homologue; NRAS: neuroblastoma rat sarcoma virus oncogene homologue; BRAF: v-raf murine sarcoma viral oncogene homolog B1; CT: computed tomography; CEA: carcinoembryonic antigen; CA19-9: carbohydrate antigen 19-9; FOLFOX: folinic acid, 5-fluorouracil, oxaliplatin; MRI: magnetic resonance imaging; 18F-FDG PET-CT: 18F-fluorodeoxyglucose positron emission tomography–computed tomography; FOLFIRI: folinic acid, 5-fluorouracil, irinotecan; CDX-2: caudal-type homeobox 2; SATB2: special adenine-thymine-rich sequence-binding protein 2; CK20: cytokeratin 20; CK7: cytokeratin 7; GATA3: guanine-adenine-thymine-adenine-binding protein 3

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