Clinical Case Report

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An uncommon cause of gastric outlet obstruction

A case report

Manjusha Bandireddy, MBBS[a,*], Noemi Baffy, MD[b]

Abstract

Rationale: Metastatic involvement of the gastrointestinal tract is an uncommon scenario encountered in the clinical practice. Our case represents a gastric outlet obstruction (GOO) as a consequence of distant Transitional cell carcinoma (TCC) metastasis without any lymph node involvement in association with inflammatory stranding leading to extrinsic duodenal obstruction.

Patient concerns: We report an unusual case of a 73-year-old male presented with a five-week history of nausea, vomiting and abdominal pain due to the metastatic extension from TCC that had been considered in remission.

Diagnoses: Computed tomography (CT) of the abdomen and pelvis revealed new circumferential thickening and inflammatory stranding involving the ascending colon extending to the hepatic flexure. Based on the imaging findings, colonoscopy was pursued which demonstrated a mass at the hepatic flexure and biopsies obtained confirmed invasive transitional cell cancer.

Intervention: Patient underwent a Wall Flex (22 mm × 120 mm) metal stent to help alleviate the gastric outlet obstruction. Chemotherapy was planned by oncology.

Lessons: Our case highlights the importance of ruling out distant metastases in the evaluation of new gastrointestinal tract pathology, for instance, Gastric Outlet Obstruction in our patient; with a prior history of TCC without any lymph node involvement under remission.

Abbreviations: CT = computed tomography, GOO = gastric outlet obstruction, MRI = magnetic resonance imaging, NMIBC = non-muscle-invasive bladder cancer, TCC = transitional cell carcinoma.

Keywords: angiolymphatic invasion, duodenal stenosis, gastric outlet obstruction (GOO), metastasis, transitional cell carcinoma (TCC)

1. Introduction

Gastric outlet obstruction (GOO) can be a complication of peptic ulcer disease, gastric polyps, malignancy or gallstone obstruction.[1] The most common malignancy to cause GOO is primary gastric adenocarcinoma and it is followed by carcinoma of the pancreas and gallbladder resulting in extrinsic malignant obstruction of the duodenum from local invasion.[2] A variety of neoplastic, inflammatory, or vascular masses extrinsic to the bowel may cause small bowel obstruction. These masses compress the bowel lumen and distort the lumen by a desmoplastic reaction involving the mesentery and peritoneal surfaces of the bowel.[2]

Transitional cell carcinoma (TCC) is the most common malignancy of the urinary tract and is the second most common cause of death among genitourinary tumors.[3] Approximately 90% of all bladder cancers are TCCs.[4] Bladder cancer typically spreads first to regional nodes, then disseminates with about equal frequency to the lungs, liver, and bone. Late in the course, subcutaneous and brain metastases are common.[5] Our case demonstrates a novel presentation of gastric outlet obstruction due to duodenal wall circumferential thickening with adjacent inflammatory stranding from transitional cell carcinoma metastasis.

2. Case presentation

A 73-year-old male presents with a 5-week history of nausea, vomiting, and abdominal pain. Past medical history is significant for stage IV transitional cell cancer with angiolymphatic invasion treated with neo-adjuvant chemotherapy and radiation followed by radical cystectomy with ileal conduit formation and bilateral pelvic lymphadenectomy. He had also undergone a diverting sigmoid colostomy for rectal outlet obstructive symptoms from metastases to the rectum. Subsequent surveillance imaging and colonoscopies were negative for any recurrence as recent as nine months prior to the current symptoms.

For the abdominal pain, patient was evaluated with CT of the abdomen and pelvis which revealed new circumferential thickening and inflammatory stranding involving the ascending colon extending to the hepatic flexure as well as the descending/transverse duodenal junction with adjacent inflammatory

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Ethical approval was waived because we did not modify our clinical care to the patient due to any research study.

Informed consent was obtained from patient's spouse for publication of the case details.

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[a] *Jawaharlal Nehru Medical College, Belgaum, India,* [b] *Department of Gastroenterology, Mayo Clinic, Arizona.*

[*] *Correspondence: Manjusha Bandireddy, Jawaharlal Nehru Medical College, Belgaum, India (e-mail: drmanjub91@gmail.com).*

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stranding (Figs. 1 and 2). Diffuse intra- and extra-hepatic biliary ductal and main pancreatic duct dilatation (double duct sign) was noted as well. Transaminases and bilirubin were initially normal.

Magnetic resonance imaging (MRI) of the abdomen was pursued to further investigate the double duct sign, which did not reveal choledocholithiasis or pancreaticobiliary malignancy. The dilated biliary tree was suspected to be secondary to thickened descending duodenum. This thickening was abutting the hepatic flexure. Additional findings included hydroureteronephrosis on the right with a focally enhancing soft tissue mass (Fig. 3).

Upper endoscopy for evaluation of gastric outlet obstruction demonstrated duodenal stenosis with significant edema and congestion without an obvious ulcer (Fig. 4). The stenosis could be traversed only with an ultrathin endoscope but biopsies were nondiagnostic. Based on the MR imaging findings, endoscopic evaluation of the colon was pursued in hope of getting a better yield for diagnosis. Colonoscopy demonstrated a mass at the hepatic flexure (Fig. 5). Biopsies confirmed invasive transitional cell cancer. Patient underwent a Wall Flex (22 mm × 120 mm) metal stent to help alleviate the gastric outlet obstruction (Fig. 6). Chemotherapy was planned by oncology. Patient had progressively deteriorated and expired one month later after establishing an agreement on comfort care only.

3. Discussion

TCC is the fifth most common cancer diagnosis in the United States and is strikingly related to cigarette smoking.[5] Transitional cell epithelium lines the urinary tract from the renal pelvis to the ureter, urinary bladder, and the proximal two-thirds of the urethra. Cancers can occur at any point: 90% of malignancies develop in the bladder, 8% in the renal pelvis, and 2% in the ureter or urethra.[6] Gross, painless hematuria is the primary symptom in 85% of patients with a newly diagnosed bladder tumor, and microscopic hematuria occurs in virtually all patients. At initial presentation, approximately 75% of urothelial tumors are superficial, localized to the bladder.[6] Only about 20% of

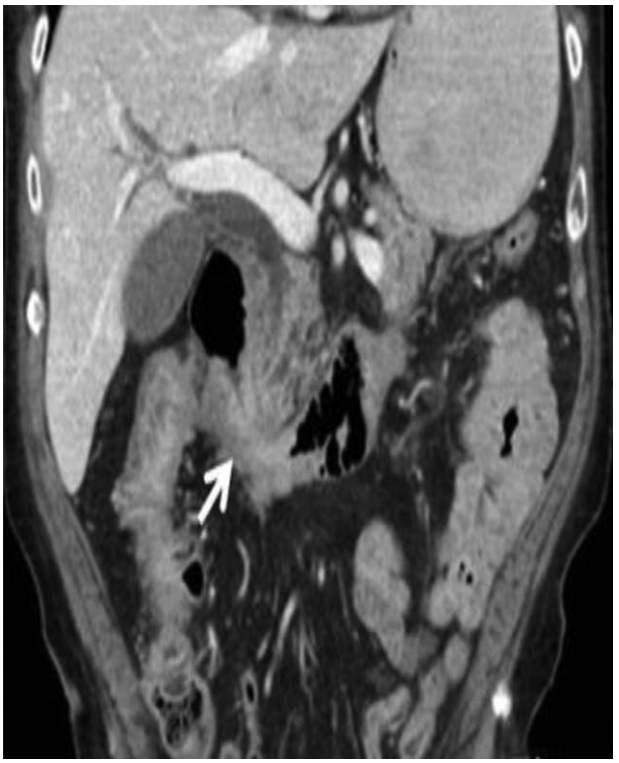

Figure 1. Arrow pointing to duodenal wall thickening and inflammatory stranding.

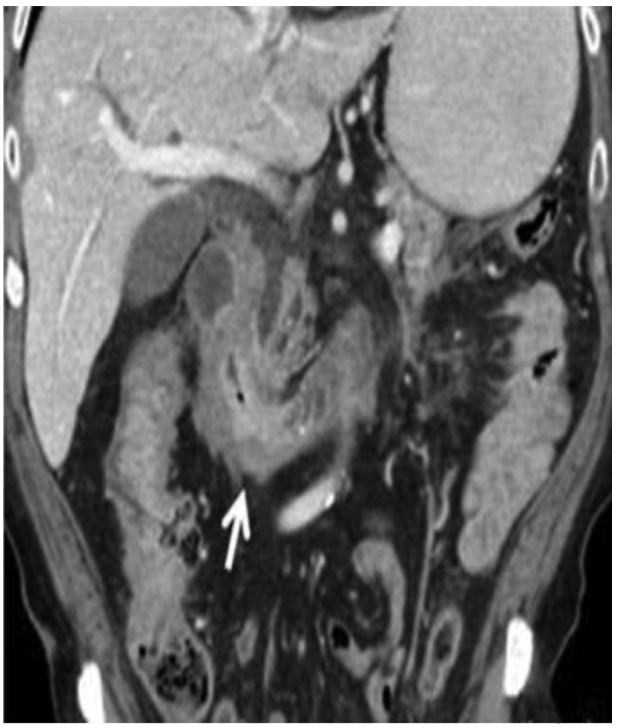

Figure 2. Arrow pointing to duodenal wall thickening and inflammatory stranding.

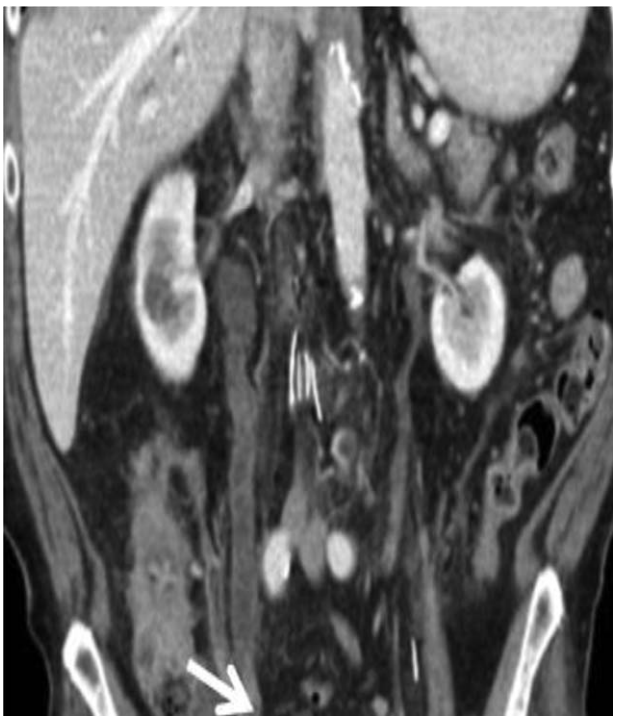

Figure 3. Arrow pointing to focally enhancing soft tissue by right ureter.

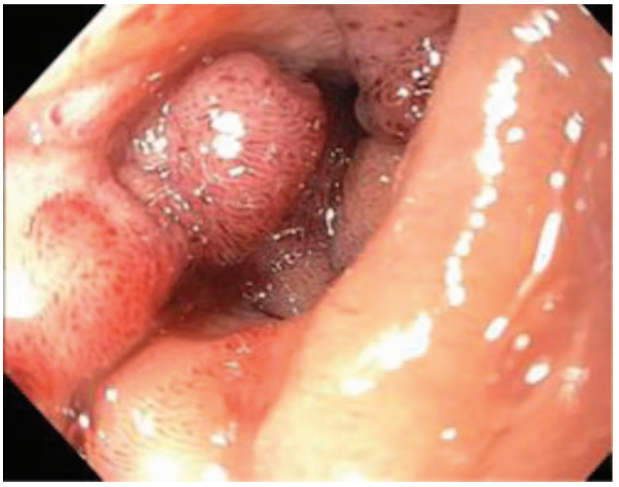

Figure 4. Proximal aspect of the duodenal stenosis.

patients present with disease that invades into the muscle wall. Fewer than 5% of patients present with locally advanced (i e, clinically extravesical) disease, and another 5% or so present with clinically apparent metastatic disease.[5] There is a high progression rate from non-muscle-invasive bladder cancer (NMIBC) to muscle-invasive bladder cancer, approaching 70%, with a high subsequent metastatic rate despite treatment.[4] A hallmark of urothelial cancer is the high recurrence rate, which approaches 80% for high-malignant potential NMIBC. The two primary theories for recurrent tumor formation are field change effects within the bladder and tumor implantation. Tumor implantation during a transurethral resection of the bladder tumor has been suggested as a possible cause for recurrent tumor formation.[4] In addition, up to 50% of patients will develop metastases following radical cystectomy for clinically localized disease.[8] The key phenotypic change that occurs in urothelial cancer that is destined to metastasize is the ability to invade the angiolymphatic system, which is seen in approximately 25% of invasive urothelial carcinoma. Angiolymphatic invasion is a poor prognostic sign with a 40% risk of nodal disease and is an independent predictor of overall and cancer-specific survival.[4]

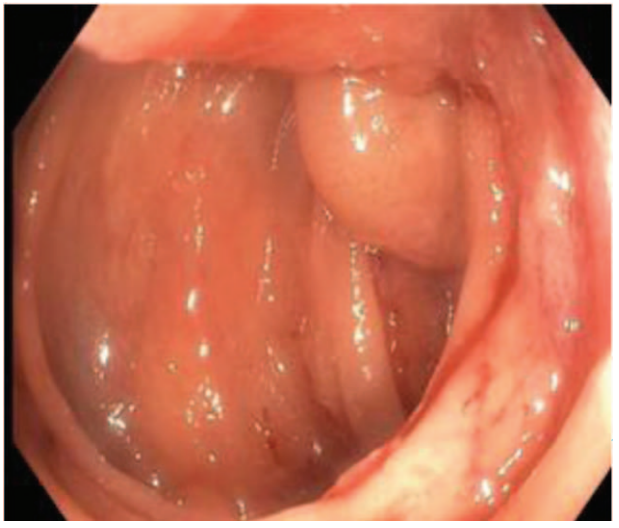

Figure 5. Fungating mass visualized at the hepatic flexure.

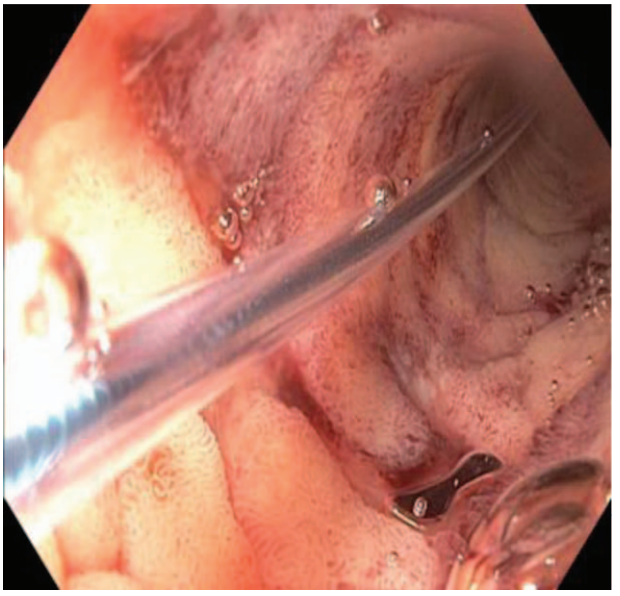

Figure 6. Duodenal stenosis during balloon dilation.

Gastric outlet obstruction can result from functional impairment of antral motility caused by the effects of acute inflammation and edema or from mechanical obstruction caused by scarring near the gastroduodenal junction.[7] Pyloric obstruction occurs when the outlet of the stomach narrows to the point of serious interference with gastric emptying.[9] Gastric distention can be due to a number of causes that can be divided into physiologic and metabolic or obstructive.[10] If causes of gastric dilatation and poor motility, such as diabetes, narcotic drugs, and others are not evident, obstructive causes such as neoplasms must be excluded.[10] With the advent of potent antisecretory therapy with proton pump inhibitors and the eradication of H. pylori, obstruction now is less commonly caused by peptic ulcers than by gastric neoplasms or extrinsic duodenal obstruction by intra-abdominal neoplasms.[11] Infrequent causes include Crohn's disease, sarcoidosis, tuberculosis, syphilis (granulomatous diseases) and pancreatitis or pancreatic cancer.[12] The symptoms of gastric outlet obstruction tend to be insidious and can manifest as gastroesophageal reflux, early satiety, weight loss, abdominal pain, vomiting, and signs may include abdominal distention and a succussion splash.[7,13] Small bowel cancer is a rare malignancy representing approximately 2% of gastrointestinal neoplasms.[5] Metastatic tumor can involve the small bowel via intraperitoneal spread (the most common route of spread), hematogenous dissemination, or lymphatic channels. Hematogenous spread to the small bowel is usually from malignant melanoma or breast or lung cancer.[2] Carcinoma of the cervix, endometrium, and ovary often affects the distal small bowel by intraperitoneal seeding or direct invasion; the colon may also be involved.[14] Extrinsic lesions resulting in small bowel obstruction include peritoneal adhesions, hernias and extrinsic tumors in mesentery or retroperitoneum.[14] Carcinomatosis is frequently multifocal, occurring at dependent sites in the peritoneal cavity in which ascitic fluid accumulates (including the mesenteric border of the distal ileum, the medial base of the cecum, sigmoid mesentery, pararectal fossae, and rectovesical or rectouterine space).[2]

Gastric outlet obstruction may be diagnosed as an enlarged stomach seen on a plain abdominal x-ray or as marked dilation of the stomach with a collapsed duodenum on CT.[7,10] Malignant

gastric outlet obstruction can be relieved with endoscopically placed expandable stents in patients with inoperable malignancy.[6]

4. Conclusions

Although cancer predominately spreads through definite routes, metastatic disease can invariably present in diverse ways. Our case creates awareness in clinicians and radiologists to consider atypical presentations of distant metastases in the differential diagnosis of a recent obscured pathogenic process with an indistinct etiology in a patient with previous cancer considered in remission. Another interesting fact illustrated in our case is distant metastases of TCC in the absence of any regional lymphadenopathy. A comprehensive survey of the literature showed only six cases reported with transitional cell cancer metastases to the duodenum with two of them presenting as duodenal obstruction.

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