Blocked by a Ring: A Case of Gastric Linitis Plastica Presenting as Large Bowel Obstruction Secondary to Rectal Stenosis

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ABSTRACT

Metastatic gut lesions from primary gastric carcinoma occur via hematogenous, lymphatic, or peritoneal seeding. We report an unusual case of large bowel obstruction secondary to rectal stenosis due to metastatic signet ring cell gastric cancer. A 61-year-old woman with a history of 8 weeks’ duration of alternation in bowel movements presented with symptoms of bowel obstruction. Computed tomography revealed rectal wall thickening, and sigmoidoscopy demonstrated edematous and fibrotic rectal mucosa. Superficial biopsies were negative for malignant disease. Because of worsening of obstructive symptoms, an emergent surgical diversion was performed. Surgical biopsies were consistent with poorly differentiated adenocarcinoma. Gastroscopy established diagnosis of gastric adenocarcinoma with signet ring type cells. Rectal stenosis on examination and demonstration of rectal wall thickening on imaging should raise suspicion for Schnitzler’s metastasis, and an upper endoscopy should be performed.

INTRODUCTION

Gastric cancer remains the second leading cause of cancer-related deaths and the fourth most commonly diagnosed cancer globally.1 Primary gastric carcinoma arises from the gastric mucosa, and distant metastasis may occur via the venous, lymphatic system and/or peritoneal seeding.2 Rectal stenosis, an atypical presentation of advanced gastric carcinoma, may be associated with peritoneal dissemination or less commonly as a result of spread of the poorly differentiated signet ring cell adenocarcinoma within the rectal submucosa via the lymphatic system (Schnitzler’s metastasis).2,3 The stomach, colon, and breast are sites of origin of signet ring cell carcinoma (SRCC) in the majority (90%) of cases. This particular subtype of adenocarcinoma has the propensity to metastasize early in the clinical course.4 Aggressive gastric malignancies such as SRCC often cause gastric outlet obstruction. However, large bowel obstruction as the first manifestation is unusual.5

CASE REPORT

A 61-year-old woman with a history of 8 weeks’ duration of multiple emergency department visits for refractory nausea, abdominal pain, and alternation in bowel movements was admitted to the hospital with symptoms consistent with bowel obstruction. Her medical history was significant for hypertension and irritable bowel syndrome. On physical examination, her abdomen was distended with generalized tenderness, but no guarding was noted. On digital rectal examination, tenderness and hard, concentric stenosis was detected. The results of blood tests and stool studies (including stool culture and fecal calprotectin) were all normal. The carcinoembryonic antigen level was mildly elevated at 9.7 ng/mL. Abdominopelvic computed tomography (CT) and pelvic magnetic resonance imaging findings revealed circumferential rectal wall thickening (Figures 1 and 2). Flexible sigmoidoscopy demonstrated markedly edematous and fibrotic rectal mucosa, and superficial biopsies were histologically negative for malignant disease (Figure 3).
The patient was referred to an outside hospital for an endoscopic ultrasound (EUS) of the rectum; however, because of progressive clinical deterioration and worsening obstructive symptoms, a decision was made to perform an emergent surgical diversion. The patient was taken to the operating room and underwent a flexible sigmoidoscopy with biopsy, laparotomy with end colostomy creation, and subcutaneous implantation of the rectal stump. During the procedure, the surgeon noted some nodularity and inflammation of the rectum and the peritoneum over the bladder. Some friable tissue was also seen around the left ovary. Hence, biopsies of the periovarian tissue and bladder peritoneum were obtained. The biopsies were consistent with poorly differentiated adenocarcinoma of intestinal origin, but the endoscopic biopsy of the rectum remained negative. Abdominal/pelvic magnetic resonance imaging was reviewed, and findings were concerning for linitis plastica and primary gastric cancer. Gastroenterology was consulted, and esophagogastroduodenoscopy was performed. Gastric ulcers noted on the esophagogastroduodenoscopy were biopsied and found to be invasive adenocarcinoma with signet ring–type cells.

The patient had rapid progression of her disease process, and repeat imaging 6 to 8 weeks later demonstrated extensive lymphadenopathy in the cervical, mediastinal, axillary, and retroperitoneal regions. She was evaluated by medical oncology and is currently receiving FOLFOX (leucovorin, 5-fluorouracil, and oxaliplatin) as a palliative chemotherapy regimen.

**DISCUSSION**

Metastatic gut lesions from gastric cancer are uncommon, although when they occur are predominantly associated with the diffuse-type SRCC and peritoneal spread. It can occur because of direct invasion via venous or lymphatic vessels or by peritoneal dissemination, although in a few cases, more than 1 route of spread has been implicated. The classic example of lymphatic spread is left supraclavicular lymphadenopathy (also known as Virchow’s node), which was also seen in our patient later in her clinical course. Similarly, Krukenberg tumor of the ovaries and Plummer’s rectal shelf (tumor implants in the pouch of Douglas) are characteristic examples of metastatic peritoneal seeding. However, scarce data exist on involvement of the rectum secondary to gastric adenocarcinoma. Schnitzler’s metastasis to the rectum is a rare phenomenon and leads to rectal stenosis and symptoms of bowel obstruction. It can occur via hematogenous or lymphangitic dissipation of poorly differentiated signet ring cells within the submucosa of the rectum. Our patient’s clinical picture was highly suggestive of Schnitzler’s metastasis, although the endoscopic biopsies of the rectum showed no evidence of tumor cells. Colonoscopy-guided biopsy has a high false-negative rate of more than 50% because of the peculiar histological pattern of metastatic SRCC. Possible explanations include mucosal sparing and involvement of the submucosa and also due to marked fibrotic response within the bowel wall and sparseness of tumor cells in SRCC. Olano et al and Rausei et al described similar cases of Schnitzler’s metastasis with negative initial colonoscopic biopsies, and the diagnosis was ultimately made after deep, surgical biopsies showed evidence of malignant cells.

CT is a reasonable, initial approach to detect intestinal metastasis from gastric adenocarcinoma. Moreover, it can also help to detect ascites, peritoneal and omental implants, lymphadenopathy, and metastases elsewhere. Tumor infiltration of the bowel usually has target-like appearance because of concentric bowel thickening. An important drawback to consider when...
using CT in this clinical setting is that it is unable to discern the individual layers of the bowel wall because of its limited resolution. EUS is the preferred imaging modality for gastric linitis and associated rectal metastasis because it can provide a circumferential view of the individual layers of the rectum and can detect intramural tumor infiltration even if the overlying mucosa appears normal.

Decompressive treatments including diversion colostomy and colonic stenting have been used for the prevention and management of bowel obstruction from metastatic colon stenosis. Chemotherapy is given to all patients irrespective of the stage at presentation.

This case is one of the handful existing cases describing synchronous Schnitzler’s metastasis associated with primary gastric cancer. Abrupt changes in bowel habits such as alternation in bowel movements and associated rectal stenosis on examination and demonstration of circumferential rectal wall thickening on imaging along with tumor-free rectal mucosa should raise suspicion for Schnitzler’s metastasis. Prompt surveillance testing including further imaging (such as EUS) and endoscopic evaluation should be performed. Despite negative superficial biopsies, if EUS shows thickening of the rectal submucosa and muscularis propria, deep, surgical biopsies are warranted to rule out metastatic disease.

DISCLOSURES

Author contributions: T. Tariq was involved in drafting the initial manuscript and is the article guarantor. A. Turk reviewed the manuscript. M. Reaume, A. Muddasani, and M. Parmar reviewed and edited the manuscript.

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