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CASE REPORT | STOMACH

Gastric Outlet Obstruction Secondary to Metastatic Breast Carcinoma 25 Years After Initial Diagnosis: A Mimic of Primary Gastric Carcinoma

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ABSTRACT

Although breast cancer is the most common form of cancer in women, metastasis to the stomach is incredibly rare. Gastric metastasis of breast cancer has been described in the literature, with an estimated median of 6 years from the initial diagnosis of primary breast cancer to metastasis and multiple instances greater than 10 years. In this case, a patient presented with gastric outlet obstruction in the setting of breast cancer metastasis to the pylorus 25 years after the original diagnosis.

INTRODUCTION

Breast cancer is the most common cancer in women with a yearly incidence rate of 43.1 per 100,000 women.[1] Although bones, the lungs, the liver, and the brain are the most common sites of metastasis, gastric metastasis is exceedingly rare, occurring in only 0.3% of primary breast cancer cases.[2–4] Often, patients present with symptoms of abdominal pain, dyspepsia, and fullness.[3] This case of breast carcinoma

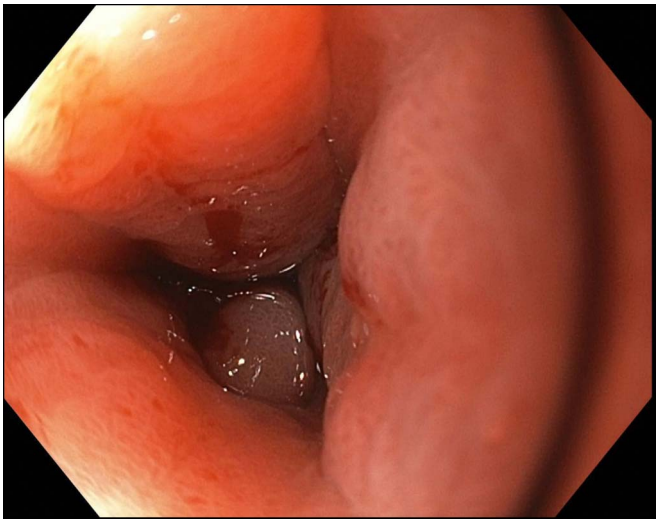

Figure 1. Endoscopic visualization of the gastric outlet obstruction at the level of the antrum.

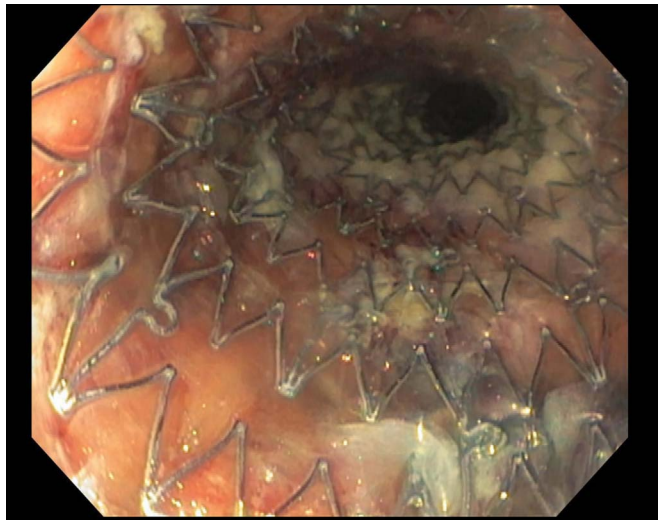

Figure 2. Status after placement of gastric stent for the treatment of gastric outlet obstruction.

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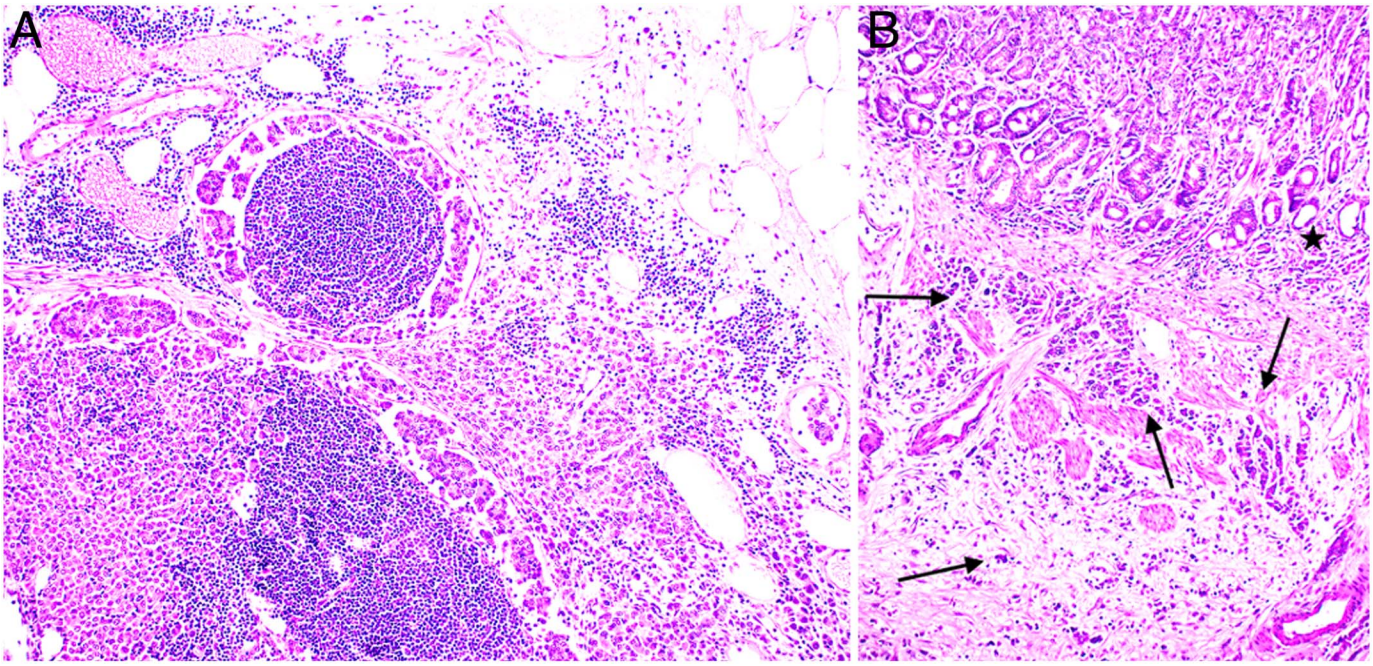

Figure 3. (A) Breast carcinoma located in the subcapsular space of the lymph node. (B) Poorly cohesive breast carcinoma in the submucosa, splitting the muscularis mucosa (arrows) and very focally in the mucosa (star) (A and B, hematoxylin and eosin 10× magnification).

metastatic to the stomach occurred 25 years after the initial diagnosis—a length of time not documented in the literature.

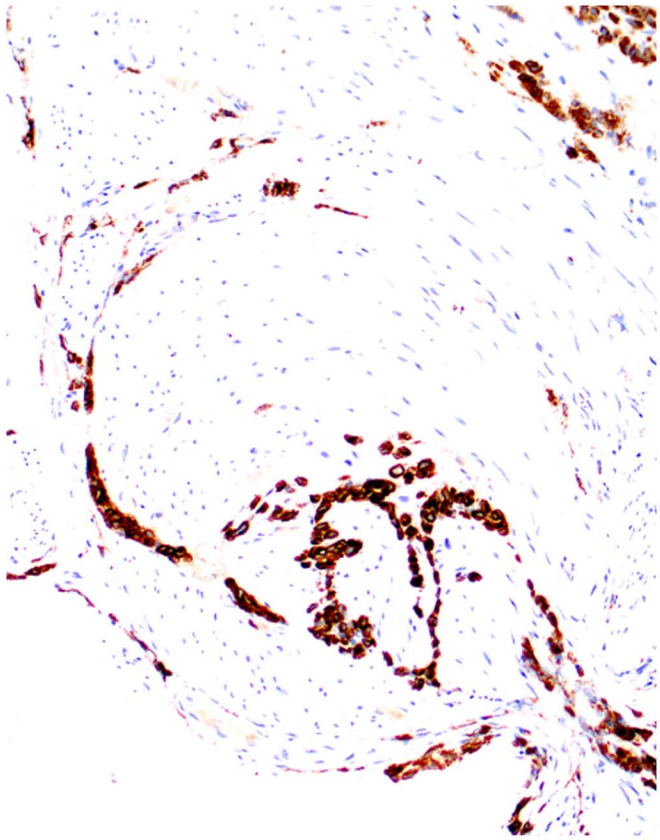

Figure 4. Carcinoma in muscularis propria positive for keratin CAM 5.2 (hematoxylin and eosin 20× magnification).

CASE REPORT

A 71-year-old woman with a history of breast ductal adenocarcinoma diagnosed 25 years earlier was considered in remission after a radical right mastectomy followed by adjuvant tamoxifen chemotherapy. She presented with recent weight loss and unrelenting nausea and vomiting. Cross-sectional imaging revealed a gastric mass. Initial esophagogastroduodenoscopy revealed gastric outlet obstruction with circumferential thickening at the antrum without a focal lesion identified (Figure 1). A subsequent esophagogastroduodenoscopy with endoscopic ultrasound demonstrated thickened layers of the antrum and pyloric channel without hypoechoic lesions or evidence of perigastric lymphadenopathy. An endoscopic mucosal resection specimen showed only a few atypical cells within the muscularis propria, and ancillary immunohistochemical staining did not establish a definitive diagnosis. Thus, a surgical specimen was necessary to make the diagnosis.

Owing to obstructive symptoms, a pyloric stent (Figure 2) was placed as a bridge to subtotal gastrectomy with Billroth II gastrojejunal reconstruction. Histologic evaluation of the gastrectomy specimen revealed poorly cohesive carcinoma involving the deeper layers of the stomach and regional lymph nodes with focal involvement in mucosal layers (Figure 3). The malignant cells were positive for keratin CAM5.1, confirming a carcinoma (Figure 4). Surprisingly, the malignant cells were also positive for GATA3, mammaglobin, estrogen receptor, progesterone receptor, and E-cadherin, an immunoprofile supporting a ductal breast adenocarcinoma (Figure 5). HER2, BRST2, PAX8, and CDX2 were all negative. The negative PAX8 excluded a

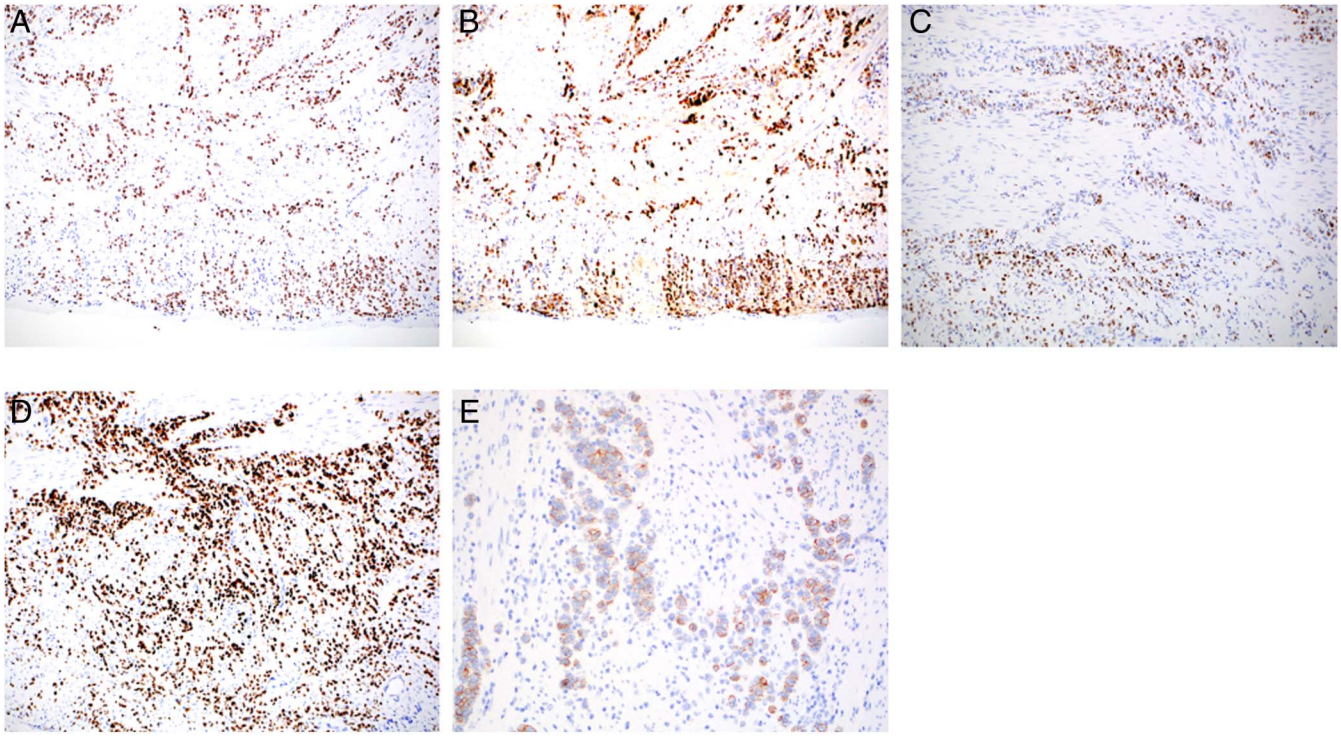

Figure 5. The slides and respective stains reveal positivity for all or nearly all malignant cells with (A) GATA3 positive, (B) mammaglobin positive, (C) estrogen receptor positive, (D) progesterone receptor positive, and (E) E-cadherin positive, confirming metastatic breast ductal adenocarcinoma (all 10× magnification except E-cadherin at 20×).

gynecologic primary malignancy, and the negative CDX2 excluded a primary carcinoma with an intestinal phenotype, including some gastric carcinomas. The histopathology with this immunoprofile supported a diagnosis of a poorly cohesive breast ductal adenocarcinoma. Comparatively, the histopathology from the patient's breast ductal adenocarcinoma 25 years earlier was described in the originating pathology report identically to that seen in the gastrectomy specimen. Furthermore, a complete workup did not reveal a recurrent or second breast primary carcinoma, supporting the diagnosis of chronologically late metastatic breast cancer to the stomach. Given the advanced presentation, the patient pursued palliative chemotherapy.

DISCUSSION

One of the challenges in the evaluation of metastatic carcinoma to the stomach is obtaining an adequate biopsy of diagnostic quality. Given that the metastases may occur in the submucosal layers or beyond, a surgical specimen is often required for optimal diagnostic quality.[3] This is especially true when a malignant proliferation is extramucosal. In this case, the patient's endoscopic mucosal resection specimen was devoid of malignant cells within the mucosa or submucosa. Because of this, a surgical specimen including all gastric layers in a wider span was necessary to make the diagnosis. In most cases, ancillary immunohistochemical studies are necessary to secure an accurate diagnosis.[5] A particularly unique feature of this case is the incredibly long time to recurrence and distant metastasis after diagnosis of the primary cancer of 25 years. Previous studies have indicated a median time from initial diagnosis to recurrence of 6 years.[6,7] A literature review revealed our case to be the longest time from diagnosis of primary breast cancer to distant metastasis to the stomach documented, with the next longest being 13 years.[7] Because breast cancer has been estimated to be the most common source of gastric metastasis, accounting for up to 37% of cases, followed by melanoma (25%) and ovarian cancer (14%), clinicians and pathologists must have a high level of clinical suspicion.[8] Thus, this case illustrates the importance of a thorough knowledge of the patient's history of even remote diseases.

DISCLOSURES

Author contributions: W. Abel wrote the manuscript and obtained informed consent to publish. B. Ganjineh and S. Reddy wrote and edited the manuscript. V. Gohil and P. Yeaton reviewed and edited the manuscript. D. Grider wrote and edited the manuscript, conducted the pathology review, and is the article guarantor.

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