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CASE REPORT

Small-bowel Metastasis from Gastric Sarcomatoid Carcinoma: A Case Report

Yu-Jen Chen[1,2], Hao Yen[3], Si-Yuan Wu[1]

[1]*Division of General Surgery, Department of Surgery, Tri-Service General Hospital, National Defense Medical Center,*
[2]*Division of Pediatric Surgery, Department of Surgery, Tri-Service General Hospital, National Defense Medical Center,*
[3]*Department of Pathology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan*

Sarcomatoid carcinoma, distinguished by its histological presentation of undifferentiated, spindle-shaped cells, is a rare variant of gastric cancer when contrasted with the more typical adenocarcinoma. Gastric cancer rarely metastasizes to the small bowel; these cancers typically arise from breast, lung, or melanoma origins. Herein, we present the case of a 70-year-old male who experienced melena and significant weight loss over 3 months. An esophagogastroduodenoscopy revealed an extensive ulcerative lesion in the lower curvature of the high body of the stomach, which is consistent with the results of the computed tomography scans. Surgery involving total gastrectomy and resection of multiple segments of small-bowel tumors was conducted. Pathological examination confirmed the presence of sarcomatoid carcinoma with poorly differentiated adenocarcinoma, along with metastases to the small intestine. This report highlights the aggressiveness of gastric sarcomatoid carcinoma, as well as the potential for the small bowel to be a potential metastasis site.

Key words: Gastric cancer, sarcomatoid carcinoma, small-bowel metastasis

INTRODUCTION

Gastric cancer is a disease with significant morbidity.[1] Adenocarcinoma is a predominant histological subtype of gastric cancer, while sarcomatoid carcinomas are particularly rare. Sarcomatoid carcinoma is usually detected in the esophagus, but may rarely be detected in the stomach.[2]

Malignant tumors originating in the small bowel comprise <2% of all cancers affecting the digestive tract and are infrequently secondary.[3] When these cancers are secondary, primary cancers of the breast, lung, and melanoma are frequently identified.[4] However, instances of small-bowel metastasis stemming from gastric cancer are rarely documented in the literature.

Here, we present the case of small-bowel metastasis arising from gastric sarcomatoid carcinoma.

CASE REPORT

A 70-year-old Taiwanese male with no known underlying diseases experienced melena and significant weight loss for over 3 months. Physical examination showed low-normal blood pressure without significant tachycardia; pale conjunctiva; no jaundice; and no palpable masses at the neck, axilla, supraclavicular fossa, inguinal region, and abdomen. Laboratory tests showed severe anemia (hemoglobin of 5.0 g/dL) and leukocytosis (white blood cell counts of 21,910/μL). The tumor markers were within the accepted limits (carcinoembryonic antigen [CEA], 1.45 ng/ml; carbohydrate antigen [CA 19-9], 25.44 U/ml; and squamous cell carcinoma antigen [SCC], 0.50 ng/ml). A computed tomography (CT) scan revealed wall thickening in the high body of the stomach, along with splenic invasion [Figure 1].

An esophagogastroduodenoscopy revealed a big, bleeding, ulcerative lesion in the lesser curvature of the high body of the stomach [Figure 2]. Histopathological examination of the endoscopic biopsy showed poorly differentiated carcinoma.

Due to signs of active bleeding despite endoscopic intervention, the patient underwent an exploratory laparotomy with total gastrectomy and D2 lymph node dissection shortly after receiving a blood transfusion. During the surgery, two bulky

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Corresponding Author: Dr. Si-Yuan Wu, Division of General Surgery, Department of Surgery, Tri-Service General Hospital, National Defense Medical Center, No. 325, Sec. 2, Chenggong Rd., Neihu Dist., Taipei 11490, Taiwan. Tel: +886-2-8792-7191; Fax: +886-2-8792-7372. E-mail: fiftyonedollars@gmail.com

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tumors were identified, a 7 cm Bormann type 3 tumor at the lesser curvature and another 3.5 cm Bormann type 3 tumor at the greater curvature [Figure 3] with direct invasion of the spleen. *En bloc* splenectomy was performed to ensure adequate surgical margin. Besides, six 3–4 cm submucosal ulcerative tumors with blood clots in the lumen were detected throughout the jejunum and ileum. One of which had intussusception, and the intussuscepted jejunal segment was resected following manual reduction [Figure 4].

The pathology report revealed a sarcomatoid carcinoma with components of poorly differentiated adenocarcinoma. Nodal metastases from the dissected regional lymph nodes were not observed (0 out of 15 nodes examined). The surgical margins were free. Microscopic examination revealed polygonal or spindle-shaped tumor cells which have rhabdoid, epithelioid morphology, pleomorphic nuclei, and prominent nucleoli [Figure 5] involving the gastric antrum and lower body (greatest dimension: 7.0 cm × 4.2 cm). These cells infiltrated the subserosa layer and extended beyond the spleen, involving the extrasplenic fat and splenic capsule. Perineural invasion was observed. In addition, the morphology of the small-bowel tumors was identical to the gastric tumor. The final stage was T4bN0M1, Stage IV.

Immunohistochemically, the carcinoma components exhibited a variable positive reaction to cytokeratin CK7 [Figure 6]. In contrast, the tumor cells showed paranuclear expression of vimentin [Figure 7] and were negative for CK20 [Figure 8]. Both gastric and intestinal tumors were positive for CK7 and negative for CK20.

Following surgery, the patient resumed oral feeding within 1 week without signs of bowel obstruction or anastomotic leaks. Following a steady recovery, he was discharged after 36 days in stable condition. However, pulmonary and brain metastasis was identified 2 weeks later, owing to increasing shortness of breath and right hemiparesis. Chemotherapy was administrated. However, it was complicated by hemorrhage of a brain tumor; the patient refused to undergo aggressive treatment and passed away 8 weeks after surgery.

DISCUSSION

Gastric cancer is ranked as the fifth most common cancer

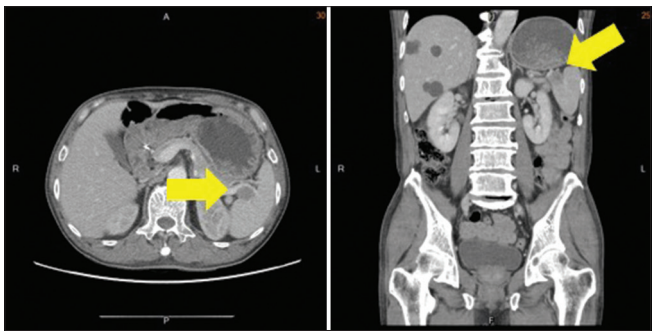

Figure 1: Abdominal computed tomography revealed wall thickening in the high body of the stomach with splenic invasion (yellow arrows)

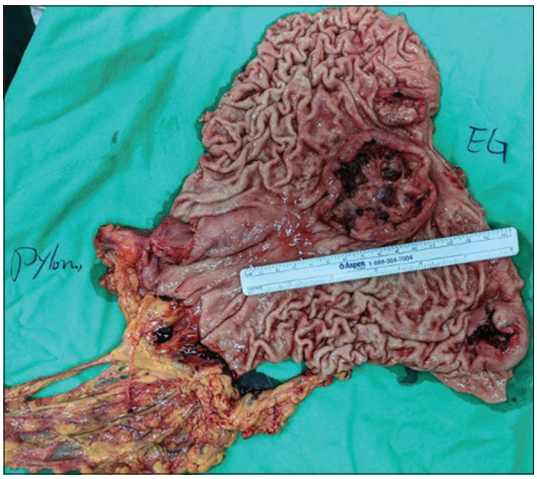

Figure 3: Two bulky tumors were identified: A 7 cm Bormann type 3 tumor at the lesser curvature and another 3.5 cm Bormann type 3 tumor at the greater curvature

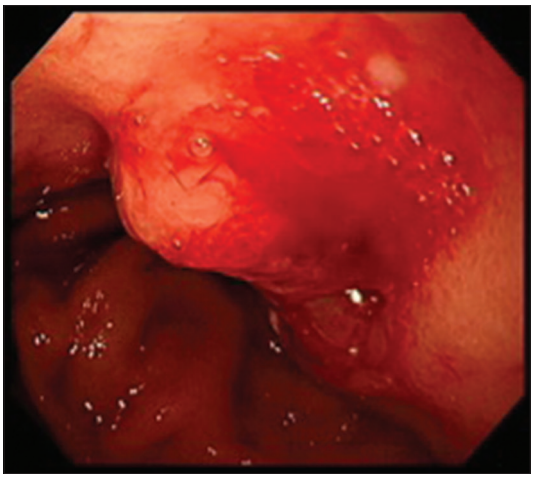

Figure 2: Esophagogastroduodenoscopy revealed an extensive, bleeding, ulcerative lesion in the lesser curvature of the high body of the stomach

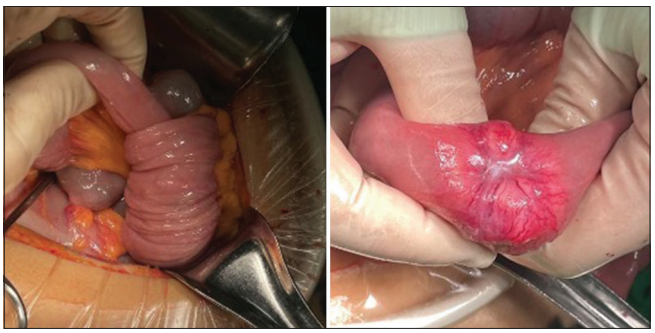

Figure 4: The intussuscepted jejunal segment was resected following manual reduction

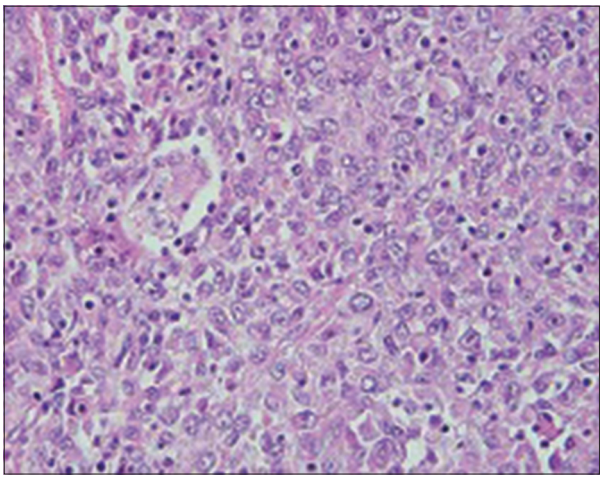

Figure 5: The tumor cells have epithelioid, rhabdoid cytoplasm, pleomorphic nuclei with a high N/C ratio, as well as prominent nucleoli (H and E, ×400)

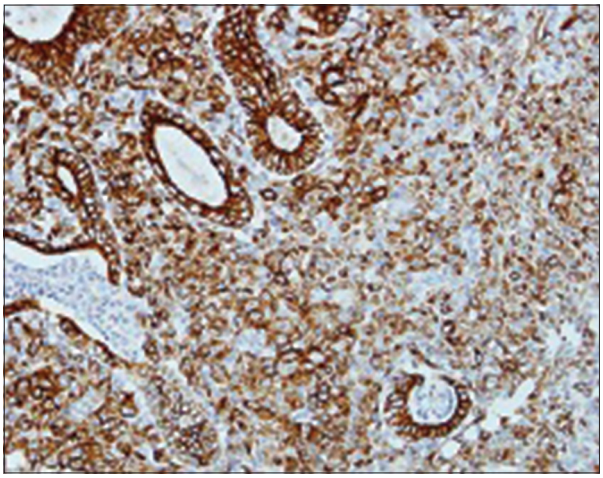

Figure 6: The tumor cells show variable staining of CK7. Decreased expression of cytokeratins indicates the mesenchymal feature in the tumor (immunohistochemical stain, CK7, ×200)

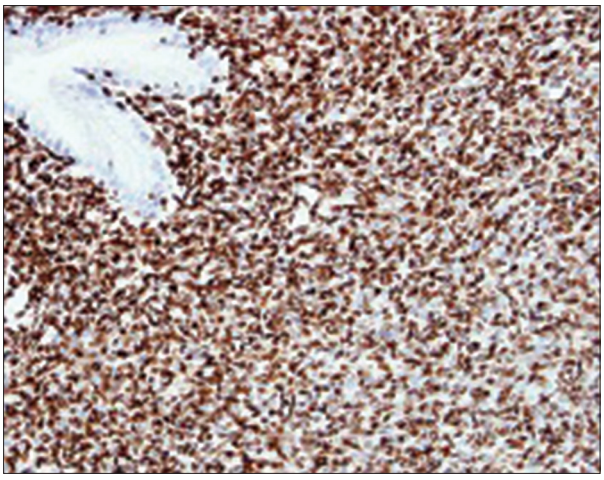

Figure 7: The tumor cells show paranuclear expression of Vimentin, indicating the sarcomatoid feature of the tumor (immunohistochemical stain, vimentin, ×200)

globally and is the fourth leading cause of cancer-related deaths.[1] Sarcomatoid carcinoma, consisting of both epithelial and sarcomatous components, is usually detected in the esophagus but rarely seen in the stomach. Onset typically occurs in the sixties, with men more likely to be affected. The malignancy can arise from any region within the stomach,[5] and associated symptoms include bleeding, obstruction, or pain, depending on the size and location of the tumor.[5]

Endoscopic examination stands as the gold standard for diagnosis, while contrast-enhanced CT scans are important in disease staging. However, distinguishing between carcinosarcomas and adenocarcinomas based on preoperative clinical manifestation, endoscopic biopsy, or cross-sectional radiological examination alone is challenging. The key challenge in interpreting preoperative biopsies lies in the risk of misdiagnosis owing to sampling either the epithelial or sarcomatous component of the tumor. Therefore, multiple biopsies and immunohistochemical staining may be required to achieve a conclusive diagnosis.[6] Specific markers such as cytokeratin, CEA, epithelial membrane antigen, chromogranin A, CD56, and synaptophysin may aid in the identification of the carcinoma components, whereas vimentin, desmin, and actin demonstrate affinity for the sarcomatous elements.[7]

Radical surgery is required for the cure, while palliative surgery, aimed at restoring intestinal continuity or cytoreduction, is indicated if confirmed distant metastasis or extensive involvement of esophagus, duodenum, pancreas, or celiac trunk.[8] The extent of gastrectomy depends on the tumor location and involvement of the surrounding visceral. Total gastrectomy is often required to achieve adequate surgical margin given the bulky nature of the tumor. Depending on the invasion of the surrounding structures, splenectomy or partial pancreatectomy may be required.[5]

In our case, surgery was indicated for uncontrolled gastrointestinal bleeding. Because the intraoperative diagnosis was adenocarcinoma of the stomach, the mass was located high in the gastric body, and the tumors were sT4aN2M0 and Stage IIIB, the surgery had a curative intent. However, the pathological examination revealed synchronous malignant neoplasms of the small bowel instead of metastases at initial and small-bowel metastases was rare. Radical resection of small-bowel tumors was performed because of recent active bleeding, intussusception, and the possibility of concomitant small-bowel tumors that could not be excluded by intraoperative frozen sections. Palliative surgery would have been performed if the patient had distant metastases.

Metastatic spread of gastric cancer poses a significant threat, with the liver, peritoneum, and lungs being the most common sites.[9] Small-bowel metastases, in contrast, are exceptionally rare.[10] The small bowel is an unusual location for metastases, with primary tumors typically originating from sites such as the

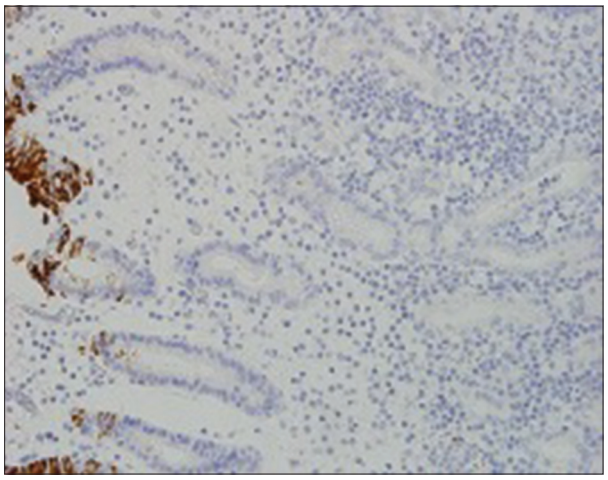

Figure 8: Tumor cells show negativity of CK20 (immunohistochemical stain, CK20, ×200)

breast, lung, melanoma, and, rarely, the stomach.[4] Symptoms of small-bowel metastases are generally nonspecific, often manifesting as obstructive symptoms such as abdominal distension, vomiting, or bleeding symptoms including melena and anemia. Some patients may present with perforation or a palpable mass.[10] For the asymptomatic forms, preoperative detection may be difficult, particularly when the metastatic lesions are small and not detectable on conventional CT scans. Some studies advocate using 18F-fluorodeoxyglucose positron emission tomography-CT scans to aid in their detection.[4]

Metastasis of gastric cancer to the small intestine remains a rare phenomenon, with most cases involving direct invasion or dissemination; lymphatic or hematogenous metastases are particularly uncommon.[10] In our case, the presence of tumor cells near the lymphovascular area in the small intestine specimen [Figure 9], coupled with the absence of regional lymph nodal metastases, peritoneal dissemination, and a bowel tumor arising from the submucosal layer, suggests hematogenous spread as the most likely mechanism of metastasis.

CONCLUSION

Gastric sarcomatoid carcinoma is rare and aggressive, with the small bowel being a potential site of metastasis. When encountering bulky tumors with poorly differentiated gastric adenocarcinomas on endoscopic biopsy, clinicians should be aware of the rare subtype of gastric cancer and include it in the differential diagnosis, which renders immunohistochemical panel. Such practice may inform the optimal extent of surgery and adjuvant therapy.

Ethical statement

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and its amendments. The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

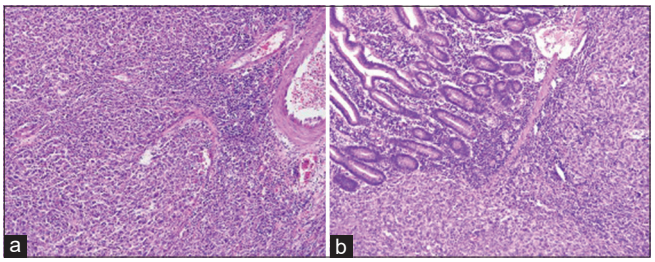

Figure 9: The small intestine shows involvement from vascular area (a) to lamina propria (b). The abrupt transition between the tumor cells and intestinal mucosa indicated the metastatic lesion in the small intestine (H and E, ×200)

Acknowledgments

We would like to thank the patient for allowing us to share his details.

Data availability statement

The data supporting this study's findings are available from the corresponding author, SY W, upon reasonable request.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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