Ischemic Necrosis of the Gastric Remnant without Splenic Infarction Following Subtotal Gastrectomy

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Gastric remnant necrosis after a subtotal gastrectomy is an extremely uncommon complication due to the rich vascular supply of the stomach. Despite its rareness, it must be carefully addressed considering the significant mortality rate associated with this condition. Patients vulnerable to ischemic vascular disease in particular need closer attention and should be treated more cautiously. When gastric remnant necrosis is suspected, an urgent endoscopic examination must be performed. We report a case of gastric remnant necrosis following a subtotal gastrectomy and discuss possible risk factors associated with this complication.

INTRODUCTION

The stomach, with five major, six minor, and several collateral arterial sources¹ has a rich vascular supply through a complex and extensive collateral plexus. It has been demonstrated in animal models that ligation of up to 95% of the arterial supply to the stomach did not negatively affect the gastric mucosa.²

Gastric remnant necrosis was first reported in literature in 1953,³ and a subsequent study has reported that only 3 of 12 patients survived.⁴ Only 7 of 11 patients reported in Japan between 1991 and 2011 have survived, which, while slightly better than historic rates, is still a significantly high mortality rate.⁵ It is not only one of the most uncommon complications, but also one of the most fatal. In this case report, we describe a patient who developed gastric remnant necrosis after a subtotal gastrectomy.

CASE REPORT

A 56-year-old man presented with abdominal pain for 3 months. His past history was unremarkable except for iron-deficiency anemia. He was a heavy smoker with a 30-pack-year history and had been drinking more than 20 alcoholic drinks daily for 30 years. Initial laboratory tests showed a serum hemoglobin level of 8.0 g/dL and serum carcinoembryonic antigen level of 92.04 ng/mL. Endoscopic examination revealed a 2.5×2.5 cm-sized irregular deep ulcer with an elevated thick mucosal fold at the lesser curvature of the antrum with an invasion into the pyloric ring (Fig. 1). Biopsy showed a moderately differentiated adenocarcinoma. Abdominal computed tomography (CT) demonstrated focal circumferential wall thickening of the prepyloric antrum (T3) with multiple enlarged perigastric lymph nodes (N2) (Fig. 2) without any evidence of metastasis. He underwent laparoscopic exploration and an open radical subtotal gastrectomy with Roux-en-Y gastrojejunostomy (Billroth I). Postoperative
Histopathological findings revealed moderately differentiated adenocarcinoma with a TNM score of pT3N2M0.

On postoperative day 1, he developed a high fever and abdominal pain. Laboratory data showed an increased C-reactive protein level of 8.5 mg/dL and a white blood cell count of 20,450/μL. Because serial laboratory data showed a steep increase in C-reactive protein level up to 31.7 mg/dL with sustained leukocytosis and high fever, abdominal CT and endoscopy were performed. Abdominal CT demonstrated severe edematous wall thickening with poor mucosal enhancement of the remnant stomach, suspected splenic infarction, and fluid collection with air-fluid levels observed in the left subphrenic space (Fig. 3). Endoscopic examination revealed a dark blue colored mucosal edema noted in almost the entire remnant stomach (Fig. 4). Therefore, an emergency exploratory laparotomy was performed.

Laparotomy revealed a necrotic gastric remnant; however, the spleen was not significantly dark in color, and its shape
was intact. Although decreased blood supply was suspected, there was no overt necrosis. The splenic artery was intact, the short gastric vessels were well preserved, and there were no other obvious causes of splenic infarction. Therefore, we decided to preserve the spleen. After almost the entire necrotic gastric remnant had been removed, the Roux-en-Y esophagojejunal pouch anastomosis was revised with a drainage catheter placed in the left subphrenic space (Fig. 5). Histopathological findings included transmural infarction with purulent inflammation.

Following a day’s stay in the intensive care unit, oral intake was initiated 2 days after reoperation. Ultrasonographic evaluation of the spleen performed on post-operative day 6 revealed that splenic infarction had incrementally improved compared to images noted on the abdominal CT performed just prior to reoperation. He was discharged in a stable condition 20 days later. He has been doing well to date, with no evidence of recurrence on CT and endoscopic exams.
DISCUSSION

Gastric remnant necrosis after a subtotal gastrectomy is very rare because of the rich blood supply to the stomach, but is potentially fatal, with mortality rates reaching up to 70%. Only a small number of cases have been reported in literature, and several of these cases have occurred secondary to splenic infarction. However, in this case, the spleen was intact, and there were no other obvious causes to account for splenic infarction, at least at the time of reoperation. Nevertheless, as gastric remnant necrosis was obvious, we concluded that diminished blood supply to the remnant stomach had precipitated this condition.

Although a CT scan and ultrasonography did not reveal these findings, the possibility of ischemia caused by intra-arterial occlusion or embolism cannot be excluded. The patient was a heavy smoker with a 30-pack year history and a heavy drinker of more than 20 alcoholic drinks daily for 30 years—both being known risk factors for ischemic vascular disease. Furthermore, he related a positive history of iron-deficiency anemia with a serum hemoglobin level of 8.0 g/dL, and a relationship between anemia and ischemic vascular disease has also been widely described. Although this has not yet been well studied, a few published case reports have described arteriosclerosis-related splenic infarction. Additionally, a case report has described a patient with arteriosclerotic vascular disease-related gastric ischemia.

While there was no obvious splenic infarction demonstrated in our case, this does not mean that sufficient blood flow had been maintained to the organ. Furthermore, the possibility of reduced blood flow to the gastric remnant cannot be ignored. Potential risk factors mentioned above might cause chronic progressive atherosclerosis, which could lead to gastric remnant necrosis.

Clinical features of gastric remnant necrosis are summarized in Table 1. Patients were predominantly men (80%) with a median age of 60 years. Billroth I reconstruction was performed in 57% of patients. The median number of days after operation when gastric remnant necrosis was confirmed was 7. Of 24 patients, 13 (54%) were treated with a total gastrectomy and 9 (69%) of them survived. However, 4 of 5 (80%) patients treated with jejunostomy and 4 of 6 (67%) patients treated conservatively died. Eventually, we found that nearly

**Table 1. Clinical Features of Patients Showing Gastric Remnant Necrosis Following a Subtotal Gastrectomy**

| Characteristic                             | Total (n=36) | Survived (n=13) | Died (n=23) |
|-------------------------------------------|--------------|-----------------|-------------|
| Age (yr)                                  | 60 (28–78)   | 60 (32–78)      | 60 (28–76)  |
| Sex                                       |              |                 |             |
| Men                                       | 28 (80.0)    | 11 (39.3)       | 17 (60.7)   |
| Women                                     | 7 (20.0)     | 2 (28.6)        | 5 (71.4)    |
| Type of reconstruction surgery            |              |                 |             |
| Billroth I                                | 20 (57.1)    | 7 (35.0)        | 13 (65.0)   |
| Billroth II                               | 13 (37.1)    | 4 (30.8)        | 9 (69.2)    |
| Roux-en-Y                                 | 2 (5.7)      | 2 (100)         | 0 (0)       |
| Duration of postoperative treatment (day) | 7 (1.5–26)   | 8 (1.5–26)      | 7 (1.5–26)  |
| Treatment modality                        |              |                 |             |
| Total gastrectomy                         | 13 (54.2)    | 9 (69.2)        | 4 (30.8)    |
| Jejunostomy                               | 5 (20.8)     | 1 (20.0)        | 4 (80.0)    |
| Conservative                              | 6 (25.0)     | 2 (33.3)        | 4 (66.7)    |

Values are presented as median (range) or number (%).
Adapted from Hajime et al., Fujiwara et al., and Isabella et al.15
64% of patients died.

Abdominal CT scanning, ultrasonography, or endoscopy could be used to diagnose gastric remnant necrosis. In light of our case, we can propose that endoscopic examination may be more helpful for confirming gastric remnant necrosis by directly visualizing necrotic mucosal changes. Typically, these changes manifest as a discolored necrotic mucosa with large and confluent ulcerations. Patients showing a high index of suspicion for rapidly progressive gastric remnant necrosis require urgent endoscopic examination.

In conclusion, even allowing for its rareness, gastric remnant necrosis must be carefully addressed considering the significant mortality rate associated with it. Patients vulnerable to ischemic vascular disease in particular need closer attention and cautious treatment. Routine postoperative endoscopy is warranted in such patients, and those with a high index of suspicion for gastric remnant necrosis require urgent endoscopic examination.

Conflicts of Interest

The authors have no financial conflicts of interest.

REFERENCES

1. Brown JR, Derr JW. Arterial blood supply of human stomach. AMA Arch Surg 1952;64:616-621.
2. Jacobson ED. The circulation of the stomach. Gastroenterology 1965;48:85-109.
3. Rutter AG. Ischaemic necrosis of the stomach following subtotal gastrectomy. Lancet 1953;2:625:1021-1022.
4. Jackson PP. Ischemic necrosis of the proximal gastric remnant following subtotal gastrectomy. Ann Surg 1959;150:1071-1074.
5. Hajime I, Akihito E, Hiroharu N, Masataka H, Hiroki M, Junzo Y. Gastric remnant necrosis following splenic infarction after distal gastrectomy in a gastric cancer patient. Int J Surg Case Rep 2013;4:583-586.
6. Fujiwara H, Ishikawa Y, Iwanaga Y, et al. A case of necrosis of the gastric remnant after partial gastrectomy. Jpn J Gastroenterol Surg 1995;28:699-703.
7. Yazici P, Kaya C, Isil G, Bozkurt E, Mihmanli M. Splenic infarction - a rare cause of acute abdominal pain following gastric surgery: a case series. Int J Surg Case Rep 2015;10:88-90.
8. Mukamal KJ. The effects of smoking and drinking on cardiovascular disease and risk factors. Alcohol Res Health 2006;29:199-202.
9. Zeidman A, Fradin Z, Blecher A, Oster H3, Avrahami Y, Mittelman M. Anemia as a risk factor for ischemic heart disease. Isr Med Assoc J 2004;6:16-18.
10. Bellotto F, Fagioli S, Pavei A, et al. Anemia and ischemia: myocardial injury in patients with gastrointestinal bleeding. Am J Med 2005;118:548-551.
11. Genc V, Cetinkaya OA, Kayiloglu I, Karaca AS, Cipe G, Unal AE. Splenic infarction as a complication of celiac artery thromboembolism: an unusual cause of abdominal pain. J Korean Surg Soc 2011;81:360-362.
12. Frippiat F, Donckier J, Vandenvossche P, Stoffel M, Boland B, Lambert M. Splenic infarction: report of three cases of atherosclerotic embolization originating in the aorta and retrospective study of 64 cases. Acta Clin Belg 1996;51:395-402.
13. Becker S, Bonderup OK, Fonset TO. Ischaemic gastric ulceration with endoscopic healing after revascularization. Eur J Gastroenterol Hepatol 2006;18:451-454.
14. Casey KM, Quigley TM, Kozarek RA, Raker EJ. Lethal nature of ischemic gastropathy. Am J Surg 1993;165:646-649.
15. Isabella V, Marotta E, Bianchi F. Ischemic necrosis of proximal gastric remnant following subtotal gastrectomy with splenectomy. J Surg Oncol 1984;25:124-132.
16. Tanj SJ, Daram SR, Wu R, Bhaijee F. Pathogenesis, diagnosis, and management of gastric ischemia. Clin Gastroenterol Hepatol 2014;12:246-252.e1.