

Giant Benign Gastric Ulcer in an Elderly Subject: A Rare Entity

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Abstract

Case Report

A 60-year-old man in reasonably good overall health presented with hematemesis and melena, along with chronic epigastric pain suggestive of an ulcer, without vomiting or fever. Clinical examination revealed epigastric tenderness, and laboratory tests showed anemia. Abdominal CT scan revealed thickening of the gastric wall, and esophagogastroduodenal endoscopy identified a giant gastric ulcer measuring 50 mm in diameter at the antral level. The initial series of multiple and deep biopsies did not indicate any signs of malignancy. The patient was treated with a double dose of proton pump inhibitors (PPIs), and an endoscopic follow-up after six weeks showed no clinical or endoscopic improvement. The second series of biopsies after treatment was also negative. Partial gastrectomy was performed, and histopathological examination of the resected specimens confirmed a benign gastric ulcer measuring 50 mm at the antre level.

Keywords: Giant Ulcer, Esophagogastroduodenal Endoscopy, Gastrectomy.

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INTRODUCTION

Gastro-duodenal ulcer (GDU) is a relatively common condition that is easily treated with lifestyle modifications and proton pump inhibitors (PPIs). Clinicians are generally aware of known risk factors, including *Helicobacter pylori* infection, non-steroidal anti-inflammatory drugs (NSAIDs) use, and lifestyle factors such as stress and smoking [1].

A giant gastric ulcer is an ulcer with a diameter of more than 2-3 cm, which can lead to complications such as perforation, bleeding, obstruction, and malignant transformation. This type of ulcer was once a common presentation of GDU before the advent of PPIs in the 1980s and the treatment of *Helicobacter pylori* infection in the 1990s.

It is usually located in the lower curvature of the stomach but can be found in any part of the stomach. It is more likely to be malignant, especially in elderly individuals, and is highly prone to perforation due to its size [2]. We report this case of a giant benign gastric ulcer in an elderly subject with characteristics suggesting malignancy during initial visual, histological, and radiological inspection [3].

CASE REPORT

A 60-year-old man was referred to our facility for several episodes of moderate hematemesis and melena over the past three weeks, accompanied by ulcer-like epigastric pain. The patient was a chronic smoker with no history of taking non-steroidal anti-inflammatory drugs (NSAIDs) or other significant comorbidities.

On clinical examination, the patient was stable in terms of hemodynamics and respiration, afebrile, and in reasonably good overall health, with tenderness in the epigastric region upon abdominal palpation.

Upon admission, his laboratory tests showed hypochromic microcytic anemia with a hemoglobin level of 7.4 g/dL and low ferritin levels. The white blood cell count was 4570/mm³, and platelet count and coagulation factors were normal. Biochemical tests revealed elevated serum blood urea nitrogen (95.1 mg/dL) and elevated serum creatinine (2.37 mg/dL). The patient was admitted and immediately started on high-dose PPI therapy after adequate resuscitation.

Abdominal X-ray did not show free intraperitoneal air. Abdominal CT scan revealed

thickening of the gastric wall without neighboring lymphadenopathy (Fig. 1). Esophagogastroduodenal endoscopy revealed an active giant gastric ulcer, measuring 50 mm in diameter, at the antral level, with multiple elevated white lesions at the base of the ulcer, presenting a budding appearance, and food debris within the ulcer (Fig. 2). A diagnosis of a giant gastric ulcer or gastric cancer was suggested. Two series of

multiple and deep biopsies were performed, and the histopathological examination of the resected specimens confirmed a benign gastric ulcer with no signs of malignancy, and *Helicobacter pylori* was not detected. A partial gastrectomy of the antrum was performed. The patient remained asymptomatic at the first follow-up, one and a half years later.

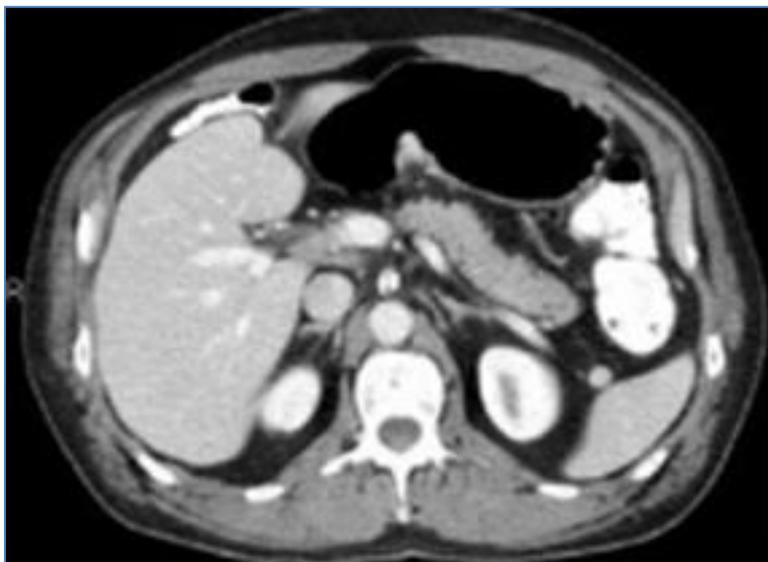


Figure 1: Axial CT scan section showing a localized thickening of the gastric wall.

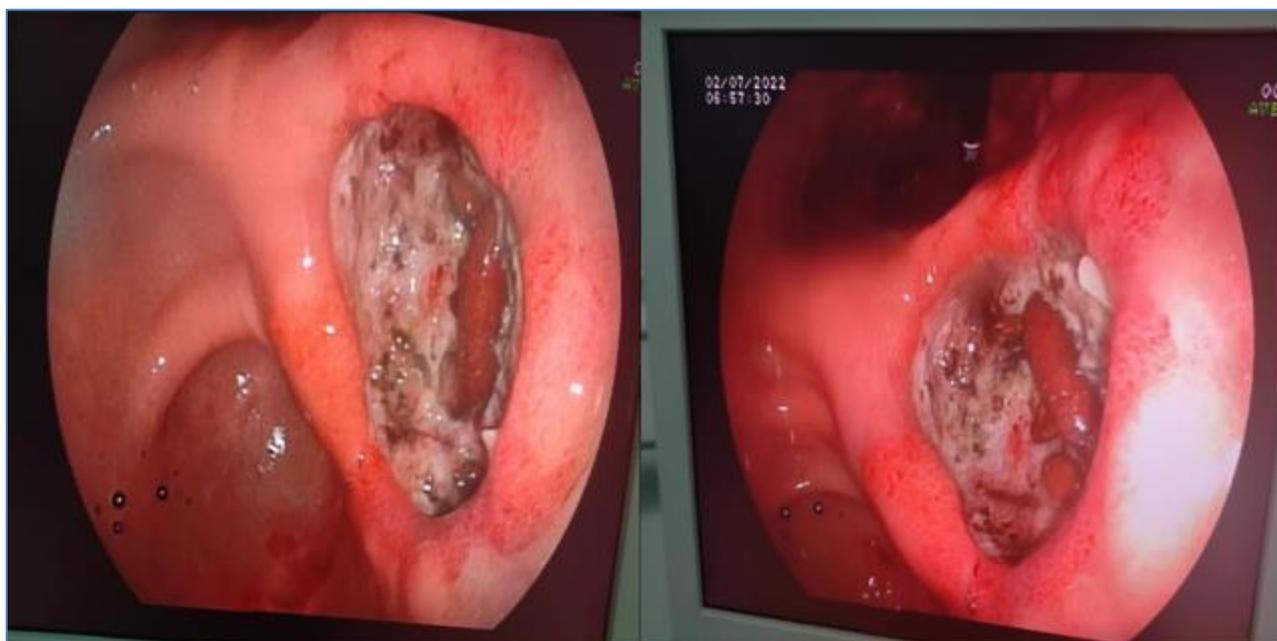


Figure 2: Esophagogastroduodenal endoscopy showing an active gastric ulcer, measuring 50 mm in diameter, at the antral level, and the presence of multiple elevated white lesions at the base of the ulcer with a budding appearance of the edges and food debris within the ulcer.

DISCUSSION

GDU remains a common medical issue worldwide, with a lifetime prevalence ranging from approximately 11% to 20% despite the widespread use of proton pump inhibitors (PPIs) [4]. The

etiopathogenesis of GDU is heterogeneous, and the most significant factors in ulcer causality are the use of NSAIDs and *Helicobacter pylori* infection. The development of gastric ulcers is mainly associated with a weakened defense capacity of the gastric mucosa and normal or decreased levels of gastric acid secretion,

while duodenal ulcers are more often linked to hypersecretion of gastric acid [5]. Giant gastric ulcers are arbitrarily defined as ulcers larger than 2 cm in diameter [6]. This situation remains an enigma in clinical practice. Such cases should be discussed during your oncology meeting. Poor healing seems to be more frequent in the elderly, those with significant comorbidities, and those on certain medications, such as NSAIDs, potassium chloride, bisphosphonates, doxycycline, or nicorandil. Chronic gastric ulcerations have also been reported following invasive adenocarcinoma, lymphoma, stromal tumor, cytomegalovirus (CMV) infection, and lymphoma [7]. The vast majority of giant ulcers occur in the body of the stomach compared to those that develop in the pre-pyloric region or in the duodenum [8]. Data regarding the pathophysiology of giant peptic ulcers are scarce, and various explanations have been proposed for why some subsets of patients develop these giant ulcers. These factors include genetic predisposition, dietary or environmental factors, microbial influence, variations in the immune response, or a combination of all these factors, in addition to *H. pylori* and NSAID use [9].

They are associated with a statistically significantly higher need for emergency surgery, overall higher mortality, and a tendency towards higher operative mortality than those associated with gastric ulcers measuring less than 3 cm in diameter [10]. The most serious complications of peptic ulcers include bleeding, perforation, obstruction, and malignant transformation. The majority of GDU cases present with bleeding, manifesting as melena, hematochezia, hematemesis, or a combination of these. Complete endoscopic hemostasis is much more difficult to achieve in actively bleeding giant duodenal ulcers than giant gastric ulcers [11]. Perforation occurs in approximately 2 to 10% of ulcers. It usually involves the anterior wall of the duodenum (60 percent), although it can also occur in antral gastric ulcers (20 percent) [12]. An article reported the outcomes of 129 "giant gastric ulcers" (defined in this study as ulcers large enough to occupy at least one stomach wall) [7]. Compared to patients with smaller gastric ulcers (likely occupying less than one gastric wall), patients with giant gastric ulcers were significantly older ($p < 0.05$), had more aggressive disease with a higher risk of bleeding, and more frequently presented with anorexia and weight loss. In this study, the most common location for a giant gastric ulcer was the gastric body. The healing rate at 12 weeks was 88% in patients under PPI therapy. Only two of the identified giant gastric ulcers were eventually found to be malignant. In the context of the patient's hemodynamic stability, medical management is preferable. Available clinical data suggest that PPI therapy should be the first-line medical management [13]. Although there is an increased rate of malignancy compared to regular gastric ulcers (approximately 7% versus 2%), current recommendations advocate for endoscopic evaluation

with multiple biopsies and brush cytology for assessment rather than resection [14, 15]. Surgery is not indicated in the initial management and is reserved only for emergency situations or cases of proven malignancy by biopsy.

As I have seen several negative cancers correctly identified by endoscopy and biopsy but confirmed by laparoscopy with gastric wedge biopsy, although a gastric wedge biopsy definitively excludes cancer, there is a risk of ulcer complications such as bleeding (12 to 44%) and perforation (1 to 2%) [16]. For this reason, a portion of benign giant ulcers may eventually be treated with partial gastrectomy; I believe this would be the best course of action for this patient. However, perioperative mortality can be high with emergency surgery in an elderly patient [17- 19]. Our patient underwent partial gastrectomy with a straightforward post-operative course and good progress after one year of follow-up.

CONCLUSION

Despite advancements in the management of GDU with the use of PPIs and endoscopic techniques, giant gastric ulcers are still associated with high rates of morbidity and mortality. Therefore, surgical evaluation of a patient with giant GDU should be an integral part of patient care. Discontinuation of NSAIDs and eradication of *H. pylori* are recommended as part of the treatment protocol. Giant ulcers require radical surgery rather than routine omental patch closure or ulcer excision.

REFERENCES

1. Malouf, J., Alam, S., Kanj, H., Mufarrij, A., & Der Kaloustian, V. M. (1985). Hypergonadotropic hypogonadism with congestive cardiomyopathy: An autosomal-recessive disorder?. *American Journal of Medical Genetics*, 20(3), 483-489.
2. Strange, S. L. (1959). Giant innocent gastric ulcer. *British Medical Journal*, 1(5120), 476.
3. Cohn, J. r. I., & Sartin, J. (1958). Giant gastric ulcers. *Annals of Surgery*, 147(5), 749.
4. Gustavsson, S., Kelly, K. A., Hench, V. S., & Melton III, L. J. (1987). Giant gastric and duodenal ulcers: a population-based study with a comparison to nongiant ulcers. *World Journal of Surgery*, 11(3), 333-338.
5. Salas, M., Ward, A., & Caro, J. (2002). Are proton pump inhibitors the first choice for acute treatment of gastric ulcers? A meta analysis of randomized clinical trials. *BMC gastroenterology*, 2, 1-7.
6. Kennedy, J. S., Hanly, E., Marohn, M. R., Arciero, C., & Mittendorf, E. A. (2004). Management of giant gastric ulcers: case report and review of the literature. *Current surgery*, 61(2), 220-223.
7. Raju, G. S, et al. Ulcère gastrique géant: son histoire naturelle et son issue à l'ère H2RA. *AmJGastro*1999;94:34783486.

8. SHOULDERS, H. H., & LISCHER, C. E. (1953). Surgical treatment of giant-sized benign penetrating ulcers of the stomach. *AMA Archives of Surgery*, 67(3), 451-461.
9. McPherson, E., Turner, L., Zador, I., Reynolds, K., Macgregor, D., & Giampietro, P. F. (2009). Ovarian failure and dilated cardiomyopathy due to a novel lamin mutation. *American Journal of Medical Genetics Part A*, 149(4), 567-572.
10. Narahara, K., Kamada, M., Takahashi, Y., Tsuji, K., Yokoyama, Y., Ninomiya, S., & Seino, Y. (1992). Case of ovarian dysgenesis and dilated cardiomyopathy supports existence of Malouf syndrome. *American journal of medical genetics*, 44(3), 369-373.
11. McPherson, E., Turner, L., Zador, I., Reynolds, K., Macgregor, D., & Giampietro, P. F. (2009). Ovarian failure and dilated cardiomyopathy due to a novel lamin mutation. *American Journal of Medical Genetics Part A*, 149(4), 567-572.
12. Nguyen, D., Leistriz, D. F., Turner, L., et al. (2007). Expression du collagène dans les fibroblastes avec une nouvelle mutation LMNA. *Bio chem Bio phys Res Commun*. 352(3), 603–608.
13. Chen, L., Lee, L., Kudlow, B. A., Dos Santos, H. G., Sletvold, O., Shafeghati, Y., ... & Oshima, J. (2003). LMNA mutations in atypical Werner's syndrome. *The Lancet*, 362(9382), 440-445.
14. Gersak, K., Strgulc, M., Gorjup, V., Dolenc-Strazar, Z., Jurcic, V., Penny, D. J., & Fan, Y. (2013). Dilated cardiomyopathy and ovarian dysgenesis in a patient with Malouf syndrome: A case report. *Molecular Medicine Reports*, 8(5), 1311-1314.
15. Gursoy, A., Sahin, M., Ertugrul, D. T., Berberoglu, Z., Sezgin, A., Tutuncu, N. B., & Demirag, N. G. (2006). Familial dilated cardiomyopathy hypergonadotrophic hypogonadism associated with thyroid hemiagenesis. *American Journal of Medical Genetics Part A*, 140(8), 895-896.
16. Chua, C. L., Jeyaraj, P. R., & Low, C. H. (1992). Relative risks of complications in giant and nongiant gastric ulcers. *The American journal of surgery*, 164(2), 94-98.