

*Case Report*

## **Postmortem diagnosis of massive gastrointestinal bleeding**

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**Giant peptic ulcers have a poor prognosis and are associated with more frequent bleeding, together with very high morbidity. The prognosis worsens if it also spreads to the duodenum, with probable involvement of adjacent organs such as the pancreas. The most serious complication of these is perforation, this being the first manifestation is up to a third cases. A 62-year-old male patient with a history of high blood pressure, progressive cognitive disorder, and complications related to alcohol abuse and smoking. The patient came to the emergency room complaining of frequent stools mixed with blood. Ten hours after admission, he experienced massive hematemesis, followed by cardiorespiratory arrest and death. An autopsy was requested. In the pylorus and the first portion of the duodenum, an ulcerated lesion measuring 6.5 × 5 cm was identified, with smooth and raised edges, a fibrinoid bottom and a blackish appearance. During the histological study, an abrupt transition between the mucosa and the ulcerated area was observed, with involvement of the pancreas, the ampulla of Vater and adjacent tissues. Signs of hypersecretory hypertrophic gastropathy were found, with no signs of malignancy or associated *Helicobacter pylori*. Complicated giant peptic ulcers represent a medical emergency associated with increased morbidity, mortality, and costs. The variability of the associated symptoms makes it difficult in certain cases to identify the risk of massive bleeding, which eventually manifests as significant hematemesis due to vascular involvement. Complications, such as perforation and penetration, are important mortality risks, which make up a wide spectrum of signs and symptoms that precede a fatal outcome, which correspond to the autopsy findings of the case presented here.**

**Key word:** Autopsy, peptic ulcer, hemorrhage.

### **INTRODUCTION**

Peptic ulcer continues to be the most frequent cause of dark bloody stools. It constitutes between 37 and 50% of the variants, and bleeding is more frequent when the duodenal mucosa is affected (Dunne et al., 2019). The

magnitude of the bleeding and the unfavorable prognosis occur when the ulcer is greater than 2 cm (that is, giant) (Ooka et al., 2019).

A giant ulcer is also associated with very high morbidity

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**Figure 1A.** Macroscopic appearance of the lesion, fresh injury.

and the prognosis becomes worse if it spreads to the duodenum, due to the high risk of involvement of adjacent organs such as the pancreas. Perforation is the most serious complication and occurs in 1-6% of patients with an ulcer; in up to one-third of cases, this is its first manifestation (Das and Wong, 2004).

### CASE REPORT

A 62-year-old male patient with a history of high blood pressure, a progressive cognitive disorder, and complications related to alcohol abuse and smoking presented to the emergency room complaining of frequent bowel movements mixed with blood previous 2 days. He reported increasing asthenia accompanied by epigastric pain and lack of appetite, which had evolved over the previous 2 weeks. Initial assessment revealed hypotension without tachycardia, icteric skin, and yellow mucosa, with marked ascitic semiology. Ten hours after admission, the patient experienced massive hematemesis, followed by cardiorespiratory arrest, and death. An autopsy was requested. During the postmortem, the esophagus did not show external alterations and its mucosa did not present specific findings. At the opening of the stomach, multiple blood clots were observed that weighed 1,300 g in total. In the pylorus and the first portion of the duodenum, an ulcerated lesion measuring 6.5 × 5 cm, with smooth and

raised edges and a fibrinoid and blackish-looking background was observed (Figure 1). The mucosa of the stomach body showed prominent folds without other alterations (Figure 2). The pancreas, with the exception of the subduodenal area, was of normal coloration; its parenchyma was firm and showed normal architecture. The other organs had no relevant findings.

During histopathology examination, in serial sections of the ulcerated lesion stained with hematoxylin and eosin, an abrupt transition was observed between the mucosa and the ulcerated area. At the bottom of the ulceration, a superficial layer of fibrinopurulent exudate, granulated tissue, and fibrous tissue were identified, and were arranged in depth in the order mentioned above.

Pancreatic tissue was observed in the deep margin of the lesion, confirming involvement of the head of the pancreas, a blister in the ampulla of Vater and adjacent tissues (Figure 3). Histological signs of hypersecretory hypertrophic gastropathy were identified, but *Helicobacter pylori* and associated malignancy were not observed (Figures 4 and 5).

### DISCUSSION

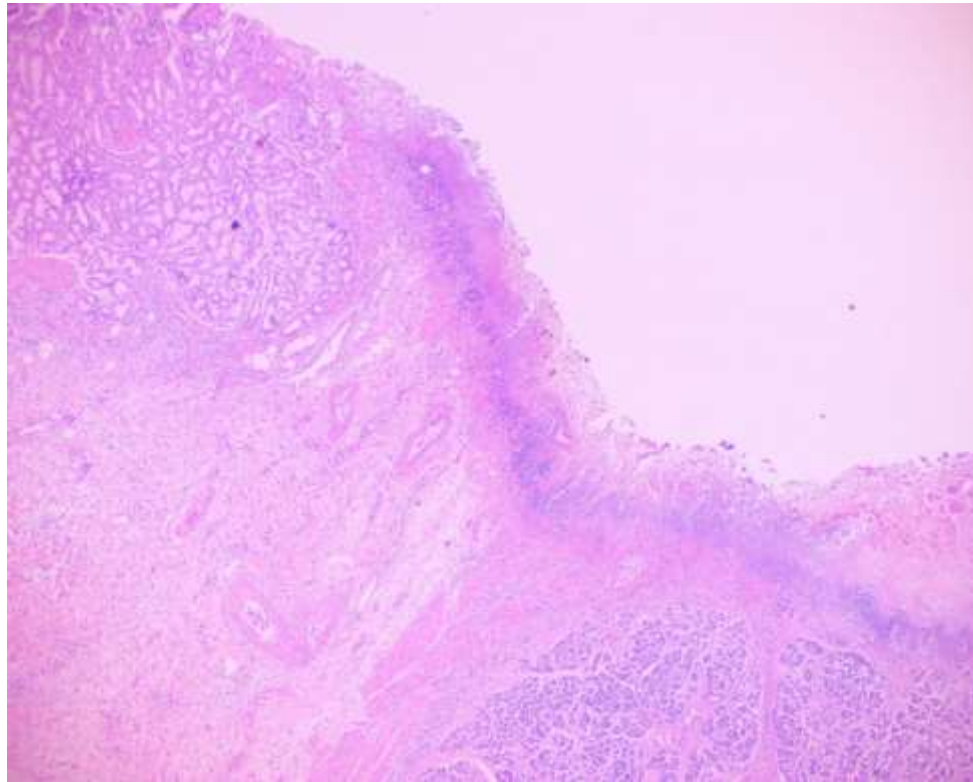
Giant gastroduodenal ulcers are those whose longitudinal diameter exceeds 2 cm<sup>2</sup>. The ulcerated lesion described herein had a maximum diameter of 6.5 cm, which definitely qualifies it as such and confers its particular



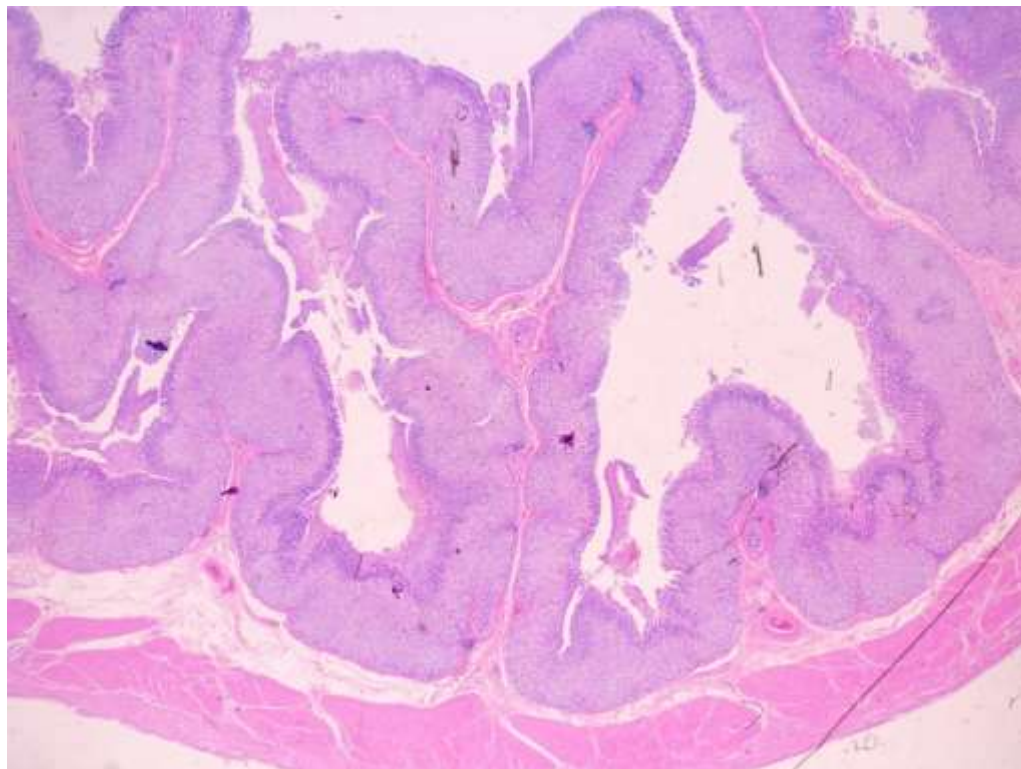
**Figure 1B.** Macroscopic appearance of the lesion, after formalin fixation.



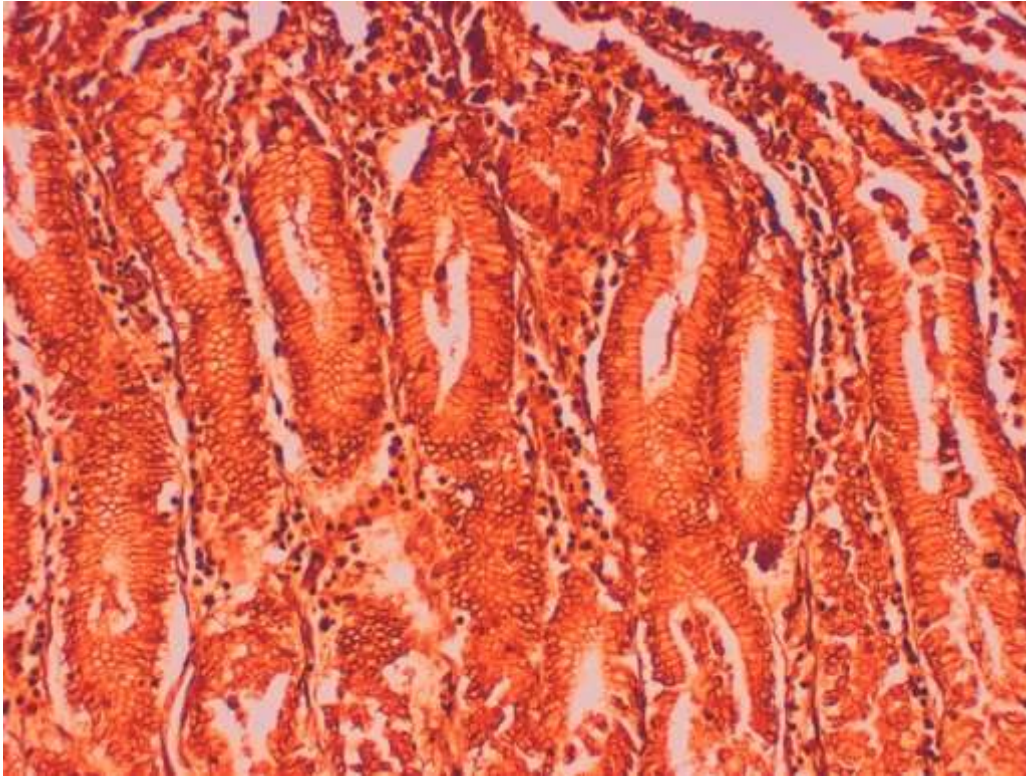
**Figure 2.** Gastric mucosa with prominent and thickened folds, with a nodular aspect to the surface.



**Figure 3.** Interruption of the mucosa by a superficial layer of fibrino-purulent exudate, showing involvement of the pancreatic tissue.



**Figure 4.** Mucosa of the gastric body with hyperplasia of superficial cells and glandular component.



**Figure 5.** Whartin staining without *Helicobacter pylori* findings.

characteristics.

According to the literature, 40% of upper gastrointestinal bleeding comes from peptic ulcers as in the present case; 5% comes from esophageal varices and 10% from esophagitis (Brunicardi et al., 2006) the latter two causes were ruled out in the postmortem in the present case. It is important to note that in 24% of gastrointestinal bleeding the cause is not evident, as in this case, where the clinical autopsy sheds light on the origin (Brunicardi et al., 2006). In autopsy studies carried out in cases of massive upper gastrointestinal bleeding, in 30% of cases the source of bleeding was not identified until the postmortem study, (Berkowitz, 1963) with the most commonly identified cause being esophageal varices due to liver cirrhosis.6 This cause was considered in our patient due to his previous history and clinical picture, but excluded by autopsy.

Rare presentations of involvement in the ampulla of Vater were also published, sharing clinical jaundice for cholestasis that the patient also presented. In other reports, even simulating signs and symptoms produced by tumors lodged in the blister (Maldonado et al., 2017; Diéguez-Castillo et al., 2018). The factors most commonly associated with peptic ulcers are *H. pylori* infection and a history of the use of non-steroidal anti-inflammatory drugs, (Brunicardi et al., 2006) which were ruled out in our patient. Other syndromes, such as the Zollinger Ellison syndrome, were also excluded as no

increase in gastrin levels was found, and there was no evidence of gastrinoma. In studies carried out in similar patients with hypersecretory hypertrophic gastropathy, frequent presentations of gastroduodenal ulcers and associated increased risk were found compared to patients with normal gastric mucosa (Fenoglio-Preiser et al., 2008). Hypersecretory hypertrophic gastropathy represents a more common entity than is considered and its role is significant enough to consider it as an etiological factor in peptic ulcer disease. In addition, smoking and alcohol abuse are currently considered as independent risk factors for complications of bleeding and perforation (Andersen et al., 2000).

As mentioned above, in 24% of gastrointestinal bleeds, the origin is not identified. In the case presented, the differential diagnoses considered clinically were esophageal varices, gastrointestinal ulcer or some tumor pathology lodged in the duodenum that caused the jaundice that the patient presented.

Definitively, the autopsy findings showed that the extent of the giant ulcer was so wide that it involved the pancreas and ampulla of Vater, resulting in jaundice. In addition, the report of diagnosed secretory hypertrophic gastropathy recalls the importance of considering this condition in patients with associated risk factors such as smoking and alcoholism, since it increases the possibility of a fatal hemorrhagic outcome in these patients. The report of this case aims to expose the consideration of

the origin of massive bleeding, in gastroduodenal ulcers of wide diameter, called giants. The clinical picture on many occasions offers confusion as other symptoms such as jaundice are added and due to the explosiveness and rapid fatal evolution of the picture, it is difficult to apply various diagnostic tools, reaching the final diagnosis through the valuable clinical autopsy.

A complicated peptic ulcer represents a medical emergency associated with increased morbidity, mortality, and costs. However, its early identification and timely treatment improve the patient's prognosis and optimizes health resources.

In conclusion, the chronic nature of gastrointestinal bleeding adds to the variability of the symptoms, which makes it difficult in certain cases to identify the risk of massive hemorrhage, which eventually manifests as significant hematemesis due to vascular involvement.

Therefore, complications, such as perforation and penetration, are significant mortality risks. In turn, they configure a wide spectrum of signs and symptoms that precede a fatal outcome, which correspond to the autopsy findings of the case presented here.

## CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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