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CASE REPORT | SMALL BOWEL

Mallory-Weiss Tear in the Duodenum

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Abstract

Mallory-Weiss tears are mucosal lacerations caused by forceful retching and are typically located at the gastroesophageal junction. Mallory-Weiss tears have not been described in the duodenum. We report of a Mallory-Weiss tear in the descending duodenum of a 57-year-old man who presented with hematemesis preceded by forceful retching. We discuss the pathophysiology of a duodenal injury in comparison to typical tears occurring at the gastroesophageal junction.

Introduction

Mallory-Weiss syndrome was first described by G. K. Mallory and S. Weiss as gastrointestinal (GI) bleeding caused by mucosal lacerations of the cardioesophageal junction.[1] These tears are commonly caused by vomiting or forceful retching, and have been reported to be caused by upper endoscopy.[2] Mallory-Weiss syndrome is responsible for approximately 3–11% of upper GI bleeding.[2–4] The lacerations are typically longitudinal along the axis of the esophagus with extensions into the mucosa and submucosa.[5] Although they are generally associated with the cardioesophageal junction, tears have been reported in the lower esophagus and the curvatures and body of the stomach.[6]

Case Report

A 57-year-old man presented with a 2-day history of emesis of food contents followed by hemetemesis, which started shortly after eating. He reported 25–30 episodes of hematemesis and 5 episodes of melena in the 24 hours before presentation. He denied abdominal pain, trauma, or ingestion of any sharp or caustic substances. The patient had a past medical history significant for peptic ulcer disease, for which he had surgery in Mexico 5 years prior to his visit. On physical examination, his vital signs were normal, and he had a benign abdominal exam. Melena was found in the rectal vault.

Laboratory data showed white blood cell count 18.3 K/uL, hemoglobin 14.3 g/dL, hematocrit 39.1%, and mean corpuscular volume 90 fL. Within 12 hours, his hemoglobin dropped to 10.7 g/dL. An esophagogastroduodenoscopy (EGD) was urgently performed. The esophagus and stomach appeared normal, but upon entry into the duodenum, a 1.5-cm linear tear was found in the descending duodenum with hematin seen at both ends of the tear (Figure 1). A small visible blood vessel was seen at the distal end of the tear with spontaneous bleeding that occurred during the procedure. A 7 Fr gold probe was used to perform cautery after injection with 1:10,000 of epinephrine, followed by placement of 2 hemoclips to achieve hemostasis. After the procedure, the patient had no further evidence of bleeding.

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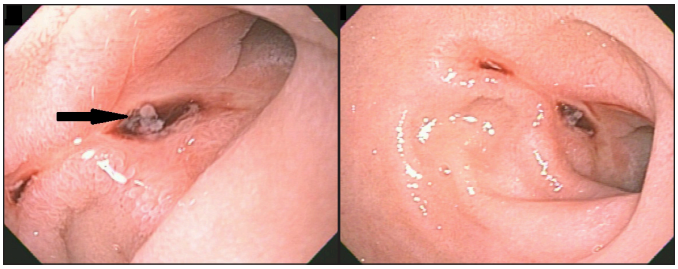

Figure 1. Endoscopic images of a 1.5-cm mucosal laceration in the descending portion of the duodenum. A visible blood vessel is also seen (arrow).

Discussion

Mallory-Weiss tears were first described in 1929 and are classically known to occur at the cardioesophageal junction. However, subsequent literature has suggested that these tears can occur in other areas of the esophagus and stomach. The precise mechanism is not completely understood, but theoretically they occur from an increased transmural pressure gradient between the thoracic and gastric areas during retching or vomiting.[6] The rise in intraluminal pressure at the cardioesophageal junction leads to barotrauma of the adjacent mucosa.

A duodenal Mallory-Weiss tear has not been described in the medical literature. Most cases of duodenal perforation are believed to be due to closed-loop injuries in gas-filled bowel.[7] Closed-loop injuries occur when 2 ends of a bowel wall loop are occluded. Subsequent perforation can occur after a direct blow or force is applied over the abdomen.[8]

During vomiting, contractions of the longitudinal fibers of the stomach begin at the gastroesophageal junction and move downwards towards the pylorus, at which point a firm contracture of the pyloric end of the stomach is created.[9] The closed pylorus could be sufficient to represent the proximal end of a closed loop, and vomiting could create an increased intra-abdominal pressure necessary to complete distal end of closed loop. Iqbal et al demonstrated that the 140 mm Hg of intra-abdominal pressure needed to create perforation in the small bowel of humans could be generated through the act of severe vomiting and retching.[10,11]

In our case, we suspect that the repeated rises in intra-abdominal pressure due to recurrent episodes of retching and vomiting generated the necessary force to cause mucosal tearing in a closed loop of bowel, and that adhesions in the luminal wall facilitated a duodenal tear rather than a gastroesophageal junction injury.

Disclosures

Author contributions: JS Chouhan wrote and edited the article, and is the author guarantor. YA Ghouri wrote and edited the article. KA Jelinek edited and approved the article.

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