CASE REPORTS

Avulsion of short gastric arteries caused by vomiting

N Hayes, P D Waterworth, S M Griffin

Abstract
A case is presented describing a new, potentially life threatening complication of vomiting after a 21 year old man presented in shock with a haemoperitoneum caused by violent, self induced emesis. (Gut 1994; 35: 1137–1138)

Forceful or prolonged retching, from whatever cause, can lead to life threatening oesophagogastric complications. The commonest, a Mallory-Weiss tear, often presenting as an upper gastrointestinal haemorrhage, arises from a breach of the mucosa around the oesophagogastric junction. Occasionally there is a full thickness perforation, usually of the left wall of the oesophagus several centimetres above the cardia, which was first described by Boerhaave in the eighteenth century. We describe a hitherto unreported serious local complication of vomiting occurring after self induced emesis.

Case report
A 21 year old man was referred as an emergency by his general practitioner with acute abdominal pain. The preceding evening he had consumed eight pints of lager followed by a take away Chinese meal. At 7 am on the day of admission, he awoke feeling uncomfortable and bloated, so he forced himself to vomit by putting his fingers down his throat. The resulting retching was surprisingly violent, and epigastric pain developed shortly afterwards. When he later visited his doctor, the pain had radiated to both shoulders and he was referred to casualty. On admission, the patient recounted the above history and was clear that he had suffered no recent external trauma. Examination showed the patient to be afebrile, but pale and distressed with a pulse rate of 112 bpm and blood pressure 75/35 mm Hg. His hypotension responded to an intravenous 'fluid challenge' with 1000 ml colloid solution. There was tenderness in both the epigastrium and the right iliac fossa. He had a white cell count of 20.4×10⁹/l, haemoglobin of 121 g/l, and haematocrit of 36.1%. Serum urea, amylase, and electrolyte tests were normal. Although subphrenic gas was absent on an erect chest film, a diagnosis of perforated viscus was made.

At laparotomy, a haemoperitoneum of about 1.5 litres was discovered. There was a short gastroepiploic ligament which, with the greater omentum, was torn at its reflection with the stomach, effectively mobilising the stomach along the greater curvature (Figure). Four short gastric arteries and branches of the gastroepiploic artery had been torn, these vessels were in the main sealed by a fresh clot. After controlling the damaged vessels with polyglactin ligatures, inspection showed no damage to the stomach muscle and as no blood was seen in aspirates from the nasogastric tube, endoscopy was not performed. The abdomen was closed leaving a drain at the splenic hilum. The patient made an uneventful recovery.

Discussion
Injury to the upper gastrointestinal tract from external violence can take a number of forms, but recognised lesions caused by vomiting are mainly partial or full thickness mural lacerations. In this case, there was no clinical
evidence of injury to any aspect of the oesophageal or gastric wall and yet a potentially life threatening laceration of the gastroplenic omentum resulted. We believe that the mechanism of injury may have been either an intussusception or partial volvulus of the greater curve of stomach caused by violent retching, with movement hampered by a short gastroplenic ligament that subsequently tore.

Although the case described is unusual, there are previously reported cases of spontaneous haemoperitoneum. We believe that if a patient presents with a history of forceful retching followed by abdominal pain and clinical signs of haemorrhage, the possibility of a tear in the gastroplenic omentum with intraperitoneal haemorrhage should be considered. There seems therefore to be three serious gastro-oesophageal complications of vomiting: (a) Mallory-Weiss tear, (b) Boerhaave’s syndrome, and (c) avulsion of the short gastric vessels.

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