Intramural rupture of the esophagus: clinical presentation and outcome

C. Ortiz Moyano, B. J. Gómez Rodríguez, F. Pellicer Bautista and J. M. Herrerías Gutiérrez

Division of Gastrointestinal Endoscopy. Department of Gastroenterology and Hepatology. Hospital Universitario Virgen Macarena. Seville, Spain

A 59-year-old man with a history of chronic obstructive pulmonary disease and alcoholic habit was admitted in our emergency room because of an episode of sudden dysphagia and vomiting with a small quantity of fresh blood, followed by further “coffee ground” vomiting, after ingestion of a piece of red meat. During the exploration the patient showed no
fever, was hemodynamically stable, with no signs of subcutaneous emphysema, but complained of substernal pain. A chest radiograph revealed neither evidence of perforation nor mediastinic air, reason why an upper GI endoscopy was performed, which demonstrated the presence of a deep mucosal laceration about 10 centimeters in length located from mid to distal esophagus and partially affecting the muscular layer, with no active bleeding at the time of exploration and it seemed it had been caused by the ejection of a foreign body during vomiting (Figs. 1A and 1B).

A thorax and abdominal CT with water-soluble contrast was then performed, which demonstrated no air or contrast leaks into the mediastinum or peritoneum. An intravenous antibiotic treatment with piperacillin/tazobactam was initiated, combined with intravenous proton pump inhibitors, while the patient remained fasted. The outcome was satisfactory, and no signs of perforation developed. On day 6 the upper GI endoscopy was repeated, and revealed the same laceration in a phase of epithelization with fibrin remnants; the patient started to progressively tolerate a soft oral diet (Fig. 1C). On day 35 a follow-up upper GI endoscopy was performed, which showed a normal esophagus (Fig. 1D).

Intramural rupture of the esophagus (IRE) is the least frequent acute esophageal trauma; it is defined as a laceration deeper than those seen in the Mallory-Weiss syndrome, but not extending through the muscular layer. The leading mechanism is habitually vomiting, coughing or a fast increase in intra-abdominal pressure. Nevertheless, it has also been related to coagulation disorders, sclerotherapy for esophageal varices, endoscopic instrumentation, presence of foreign bodies, and in healthy patients (1). However, deeper injuries affecting the muscular layer can cause an esophageal perforation or Boerhaave’s syndrome (2).

The most frequent clinical presentation is acute substernal or epigastric pain, which may be very severe, leading to rule out cardiovascular emergencies. Other symptoms include dysphagia, odynophagia and hematemesis, usually in amounts smaller than those of Boerhaave’s and Mallory-Weiss syndrome (3).

The diagnosis is made by upper GI endoscopy – IREs are usually long, deep, and proximal to the esophagogastric union in 20-30% of all cases (4). In patients where there is concern regarding a possible perforation thoracic and abdominal CT scans are mandatory to delineate the need for early surgery. Most patients respond to conservative management consisting of intravenous fluid supply; in the absence of clinical and radiologic signs of perforation, surgery is not required and no long-term esophageal sequelae develop in most patients (5).

REFERENCES