The role of conservative treatment in esophageal perforation caused by the ingestion of a blister pack: A case report and literature review

Perforación esofágica debida a la ingestión de un blíster, el papel del tratamiento conservador: reporte de un caso y revisión de la literatura al respecto

Gastrointestinal (GI) tract perforation is a complication found in only 1% of cases of foreign body ingestion.[1–4] Medication blister packs are a rare cause of GI tract perforation, mainly affecting the esophagus and ileum.[1,2,5] Currently, there is no consensus on diagnosing and treating esophageal perforations. Surgical management is the most accepted method, although less invasive therapies have produced good results in some instances.[6]

A 73-year-old man had a past medical history of prostate cancer with metastasis to the right acetabulum, treated with radiotherapy and goserelin; diabetes controlled with metformin; and recurrent episodes of anxiety. He came to the emergency service due to dysphagia to solids and intense neck pain that presented 8 days after having taken, in a crisis of anxiety, several tablets of paracetamol/tramadol, on a single occasion. Upon admission, his vital signs were BP 114/78 mmHg, HR 80 bpm, RR 20 bpm, temperature 36.2 °C, and oxygen saturation 94%. The patient had no signs of bleeding, hemodynamic instability, or systemic inflammatory response and presented only with crackling at the level of the muscular triangle. Laboratory work-up reported hemoglobin 13.4 g/dL, leukocytes 7.14 thousand/mm^3, absolute neutrophils 5.2 thousand/mm^3, platelets 259 thousand/mm^3, PT 15 s, aPTT 29.2 s, and INR 1.35.

A computed tomography scan was ordered and revealed retropharyngeal air and a foreign body in the cervical esophagus (Fig. 1A). The patient underwent esophagogastroduodenoscopy with an Olympus GIF HQ190 gastroscope. A 3 mm perforation was found in the hypopharynx (Fig. 1B) and a tablet still inside its blister pack was detected 2 cm distal to the cricopharyngeal muscle (Fig. 1C). The blister pack was removed with foreign body forceps. A second perforation, measuring 25 × 5 mm, was observed at the level of the cervical esophagus. A nasogastric tube was placed, and a prophylactic antibiotic regimen with ceftriaxone and metronidazole was started.

The patient's post-procedural progression was favorable, with improvement in pain and no signs of a systemic inflammatory response. At 48 h from treatment, enteral nutrition was started. Five days later, the patient was discharged from the hospital, with a computed tomography scan showing no signs of a lesion (Fig. 1D). The management video is available in the annex (Video 1 in Supplementary material).

Three weeks later, a control esophagogastroduodenoscopy revealed no signs of a lesion, and so the patient was prescribed a bland diet. At the one-year follow-up, he is asymptomatic, with no sequelae.

Perforation of the esophagus is rare.[3] It is a medical emergency, with a 15–30% mortality rate.[7] The intrathoracic region of the esophagus is the most affected (55%), followed by the cervical (25%) and abdominal (20%) areas.[3,6]

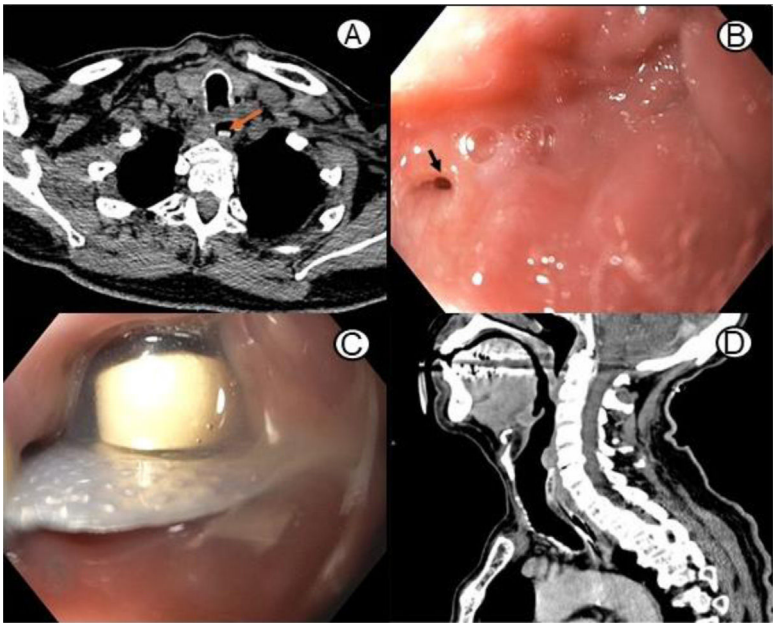

Figure 1 Tomographic and endoscopic findings of a foreign body impacted in the cervical region of the esophagus. A) Axial CT view showing a foreign body (arrow) in the esophageal lumen. B) EGD showing a 3 mm perforation (arrow) on the left side of the hypopharynx. C) EGD showing the tablet and its blister pack in the cervical esophagus. D) Control CT at 5 days after the endoscopic examination, showing no signs of esophageal perforation.

Table 1 Cases of esophageal perforation related to the ingestion of medication blister packs reported in the literature.

Author	Progression interval at the time of the initial evaluation	Initial clinical data	Diagnostic method	Lesion size	Extraction and management	Follow-up
Gupta et al.[3]	2 weeks	Chest pain, dyspnea, and fever	Endoscopy	NR	Endoscopic extraction	Contrast-enhanced control study, with no signs of lesion
					Conservative	Death due to cardiac arrythmia during hospitalization
Campos Franco et al.[5]	24 h	Dysphagia, dyspnea, and fever	Esophagoscopy and rigid hypopharyngoscopy	2 cm	Extraction through esophagoscopy and rigid hypopharyngoscopy Conservative	No signs of lesion after 3 weeks
Purnak et al.[11]	NR	Emesis Crackling in the neck	Endoscopy	NR	Endoscopic extraction Sclerotherapy	NR
Coulier et al.[4]	NR	Chest pain	MDCT	NR	Palliative	Patient died
Horton and Clayton[1]	5 days	Odynophagia, dysphagia, and fever	Endoscopy	NR	Endoscopic extraction Conservative	No signs of lesion in control studies
Current	8 days	Dysphagia and cervicalgia	Endoscopy	3 mm	Endoscopic extraction Conservative	No signs of lesion after 3 weeks

MDCT: multidetector computed tomography; NR: not reported.

There is no consensus on the treatment of esophageal perforation. Variables, such as the size and location of the lesion, progression time, and the presence of contamination, should be considered. To choose the optimum management, the patient's condition must be considered.[6]

The Cameron criteria (well-circumscribed intramural or transmural perforation, a sealed lesion, and no signs of sepsis, distal obstruction, or malignant esophageal disease outside of the abdominal esophagus) aid in selecting the patients that can be managed conservatively. The patient that does not meet those criteria should be treated surgically.[7] The European Society of Gastrointestinal Endoscopy suggests that conservative treatment include broad-spectrum antibiotic therapy, symptom control with analgesics and antacids, fasting, nasogastric aspiration, and strict surveillance.[8] There is a lower risk for contamination of the mediastinum in the cervical esophagus. Thus, conservative treatment in that region offers better results.[8] Our patient met all the Cameron criteria, and the perforation was contained in the cervical esophagus, resulting in our opting for conservative treatment.

The period from perforation to its diagnosis is the main prognostic factor for survival in these patients.[6,7] In a meta-analysis, Biancari et al.[9] reported a 7.4% mortality rate in the patients whose treatment started before the first 24 h from the time of the perforation and rose to 20.3% in the patients whose treatment began after the first 24 h.[9] Most of the patients with esophageal perforation due to blister pack ingestion were diagnosed and treated 24 h after said ingestion. All the literature regarding patient follow-up shows that no signs of lesions were found in the control studies, and two of those patients died. In the case of our patient, no lesion was detected in the control endoscopy at 3 weeks and his progress at one year was favorable, with no sequelae.

In contrast to our case, in 2023, Yu et al.[10] reported on a patient, who after nine days from having ingested a blister pack, was managed surgically through laparotomy, after evidence of an uncontained lesion in the esophageal wall. Despite said management, the patient needed mechanical ventilation in the postoperative period and presented with bilateral pleural effusion; he developed multiorgan failure

and died on postoperative day 15.[10] By comparison, our case underlines the importance of having different factors for adequate management selection, such as the time from the perforation to its diagnosis and the clinical conditions of the patient. In addition, even if the selection of the therapeutic method is satisfactory, different associated factors can result in considerable differences in therapeutic success.

Foreign body ingestion is a common problem in the advanced-age population, and due to polypharmacy, the incidence of blister pack ingestion is expected to increase.[4] In 2015, Yao et al.[2] described 17 cases of GI tract perforation related to medication blister packs, 14 located in the ileum and 3 in the esophagus. Since then, according to our research, 2 more cases of esophageal perforation due to medication blister pack ingestion have been reported (Table 1).[1,3–5,11]

Blister pack ingestion is a rare but increasing cause of esophageal perforation. Early diagnosis and treatment are vital for preventing fatal outcomes. Conservative management can be an acceptable option with good results and prognosis in a select group of patients.

Author contributions

JPPM: research, supervision, writing of the original draft, and review and editing of the writing; MERS: validation and visualization; AYOC: research, supervision, writing of the original draft; AIHG: validation and visualization; EFMB: review and editing of the writing. All the authors read and approved the final manuscript.

Ethical considerations

The authors declare that this article contains no information that could identify the patient. Informed consent was obtained from the patient at the time of receiving medical attention, undergoing treatments. This report complies with the current bioethical research regulations, did not require the authorization of the Bioethics Committee because the integrity, health of the patient were not compromised.

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Conflict of interest

The authors declare that there is no conflict of interest.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.rgmxen.2024.11.001

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Acute small bowel obstruction caused by a hemobezoar in a peptic ulcer case

Hemobezoar causa obstrucción aguda de intestino delgado en un caso de úlcera péptica

The formation of bezoars from indigestible materials can cause intestinal obstruction.[1] A hemobezoar is an accumulation of blood residue in the intestinal lumen and is a rare complication of upper gastrointestinal bleeding. The majority of cases occur after bariatric procedures, in particular, in Roux-en-Y gastric bypass (RYGB) surgery. Hemobezoars can cause acute small bowel obstruction.[2–5]

A 92-year-old woman had a past medical history of chronic NSAID use. She arrived at the emergency department presenting with hematemesis, lethargy, dehydration, abdominal distension, nausea, and vomiting. The initial laboratory work-up showed Hb of 5.6 g/dl, creatinine of 1.2 mg/dl, and BUN of 38 mg/dl. She underwent transfusion with three units of packed red blood cells and first received an 80 mg intravenous (IV) bolus dose of omeprazole, followed by 40 mg IV twice a day.

Upper endoscopy revealed a Forrest IIB duodenal ulcer (Fig. 1), which due to the complexity associated with its anatomic location, was treated only with removal of the attached clot and endoscopic adrenaline injection. Erosive gastritis and Los Angeles grade C esophagitis were also identified. Twenty-four hours later, the patient developed acute oral feeding intolerance, with a tendency toward low blood pressure. She showed neurologic signs of lethargy and presented with skin and mucous membrane dehydration, associated with a serum sodium level of 149 mmol/l, oliguria with creatinine of 1.2 mg/ dl, and BUN of 38 mg/dl. She also presented with abdominal distension, epigastric pain, and multiple episodes of nausea and vomiting. Given that her hemoglobin level did not drop, and her clinical status did not worsen, suspected rebleeding was ruled out.

Additional evaluation was carried out by consulting with the internal medicine service. An abdominal computed tomography (CT) scan with oral contrast was ordered that revealed the presence of a hemobezoar in the second and third part of the duodenum, causing acute small bowel obstruction (Fig. 2).

The primary therapeutic approach consisted of hydration with crystalloid solutions, nasogastric tube placement, and intestinal lavage with a carbonated soda, with no success. The general surgery team suggested a surgical approach, but the patient and her relatives rejected it, and so conservative treatment through the placement of a nasojejunal feeding tube for enteral nutrition and the prevention of major obstruction was employed. Clinical improvement was achieved in two days, with the patient becoming hemodynamically stable and adequately tolerating the enteral nutrition tube. There were no signs of rebleeding, and so we decided to discharge the patient.

An abdominal CT scan with oral contrast was programmed for one month after her discharge and it showed that the duodenal obstruction had resolved (Fig. 3). The feeding tube was removed, and the patient remained under surveillance.

Our case is an example of successful conservative treatment of acute small bowel obstruction related to upper gastrointestinal bleeding, which can serve as a guide for physicians faced with similar cases. The main limitation of the present study is the fact that it describes only one case report, thus limiting its generalization.

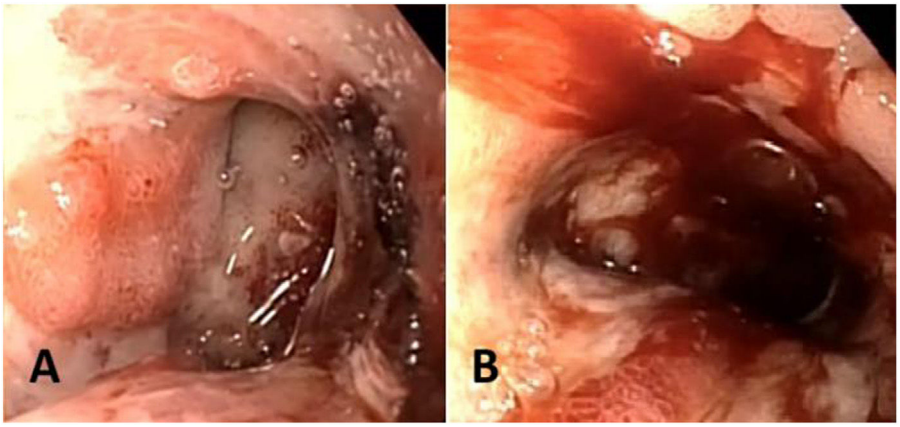

Figure 1 A) Forrest IIB duodenal ulcer. B) Bleeding after clot removal.