

SCIENTIFIC LETTERS

Gastric perforation by intragastric balloon in patient with Nissen fundoplication[☆]



Perforación gástrica por balón intragástrico en paciente con funduplicatura de Nissen

Obesity is a pandemic affecting the western world, with very high associated morbidity and mortality rates. The intragastric balloon is a device used in the temporary treatment of morbid obesity as a bridge to its definitive treatment. It is placed by endoscopy and remains free inside the stomach.¹ Together with changes in dietary habits, this treatment achieves good results for weight loss. However, this therapy is not risk-free and can have complications, some of which are potentially serious. Consequently, even though the rate of complications is low, patients, particularly those at high risk, should be closely monitored so that severe complications can be detected early.^{1,2} We describe the case of a patient with previous gastric surgery who presented with a gastric perforation following the placement of an intragastric balloon.

A 67-year-old morbidly obese man (body mass index 45) underwent surgery for a hiatal hernia (Nissen fundoplication) 15 years ago. Fifteen days after the insertion of an intragastric balloon (Apollo or Medsil Orbera balloon filled with 600 ml physiological saline solution and methylene blue), he came to the Emergency Department of our hospital with signs and symptoms of onset 48 h previously of severe pain in the upper abdomen, oral intolerance with nausea and vomiting and a poor general state of health. The physical examination revealed hypotension (80/50 mmHg), tachycardia (124 bpm) and board-like abdominal rigidity. Notable in the laboratory test results were leukocytosis of 19,170 with left shift (87% neutrophils) and prothrombin activity of 76%. An X-ray of the abdomen showed significant gastric dilatation and pneumoperitoneum (Fig. 1a and b). A computed tomography (CT scan) of the abdomen confirmed the findings (Fig. 1c). An emergency midline laparotomy was performed after previously revealing extensive necrosis of the lesser curvature of the stomach and subcardial perforation. A gastrectomy involving the affected zone was performed after extraction of the gastric balloon, which was

found impacted at the pylorus (Fig. 1d). The patient's post-operative progress was poor, and he presented with signs and symptoms of sepsis which required antibiotic treatment and vasoactive agents, respiratory distress and an infection of the wound. The patient gradually improved, and on day 20 post-surgery showed signs and symptoms of evisceration which required another operation. During surgery, it was discovered that the gastric remnant showed an area of subcardial ischaemia. After releasing the stomach, the poor quality of the gastric remnant was observed, and as a result a total gastrectomy and Roux-en-Y oesophagojejunostomy were performed. The patient made good progress following the second surgery, on day 15 presenting with a residual abscess which was treated by image-guided drainage. He was discharged 50 days after admission. Currently, at 3 years post-surgery, he has a good quality of life and is followed up by the Nutrition Unit for monitoring of his nutritional status post-gastrectomy.

Endoscopic therapy can be an effective alternative to pharmacological treatment for class I and II obesity, since it is effective in the short term, reducing body weight and improving the comorbidities associated with obesity.^{2–4} It is based on the gastric space-occupying effects which increase feelings of fullness. The process is well-tolerated, and most of the complications are mild and well tolerated, particularly nausea and vomiting which occur in 20–25% of cases, and abdominal pain, which occurs in 15–20%.⁵ However, cases of mortality (0.05%) and severe complications, such as gastric perforations (0.1%), have been described.³

At present, previous gastric surgery is considered a contraindication for intragastric balloon placement, although absolute contraindication is reserved strictly for partial gastrectomy cases. However, in patients with previous abdominal surgery, monitoring must be strict to avoid severe complications. Therefore, as is seen in our case, gastric surgery, and particularly anti-reflux surgery, are significant risk factors since they make evaluating the stomach difficult, because of the same anti-reflux mechanism, in case of impaction of the balloon at the pylorus. In these cases, if the obstruction is not diagnosed early, and in view of the fact that vomiting is not possible, progressive gastric dilatation occurs, which can result in gastric necrosis and perforation. In our case, the patient was not warned of the risk and did not consult a doctor in spite of the discomfort, finally coming to the Emergency Department in a state of septic shock. It should be noted that abdominal pain, nausea and vomiting are common adverse effects in the first few days following balloon placement but are uncommon after 15 days.

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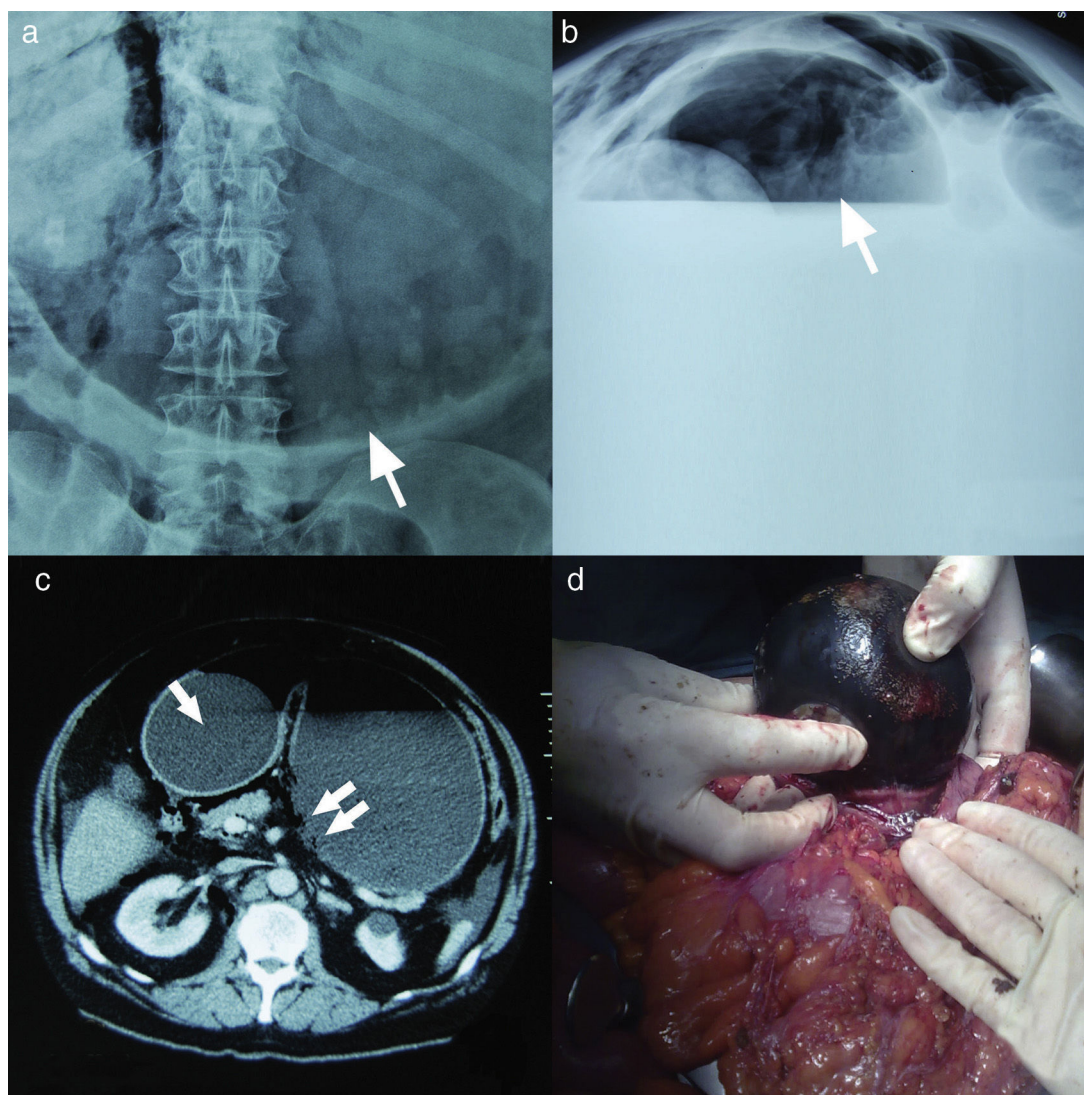


Figure 1 (a and b) Plain X-ray of the abdomen, anteroposterior view (a) and in decubitus with horizontal beam (b): the massive gastric dilatation is observed (arrow); (c) computed tomography (CT scan): note balloon in antral region (arrow) and massive gastric dilatation with perforation in the posterior wall (double arrow); (d) extraction of the intragastric balloon.

In cases of gastric perforation, the treatment should be as conservative as possible given that this is a benign disease. Currently, in cases diagnosed early, laparoscopy can be performed and the orifice closed, obtaining good results.¹ However, dramatic cases such as the one presented here, which involve subcardial gastric necrosis, can even require a total gastrectomy. Endoscopic treatment would have had to be ruled out for the patient described in this article because of his surgical history and body mass index, which, at 45 kg/m² (morbid obesity) means that immediate surgery would be advised.

In conclusion, we can confirm that intragastric balloon placement is not free from complications, and accordingly, it is essential that patients be monitored closely and that they are warned of the risks and the importance of attending for follow-up examinations in case of the onset of clinical signs and symptoms suggestive of complications. This is particularly the case in patients who have previously undergone anti-reflux surgery.

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Weil's disease and acute multifactorial hepatitis. About a case[☆]



Enfermedad de Weil y hepatitis aguda multifactorial. A propósito de un caso

Leptospirosis is a zoonosis characterised by outbreaks in South East Asia and Central and South America. In Europe, leptospirosis has been reported in Denmark, Greece, Portugal, France, Germany and the Netherlands. Incidence rates range from 10 to 100 cases per 100,000 people in endemic regions.¹ In Spain, the incidence was 0.86 cases per million inhabitants/year according to a study conducted between 2009 and 2012 by Domingo et al. The autonomous communities with the highest number of cases were the Canary Islands and the Basque Country, with no cases recorded in Aragon, La Rioja or Melilla.²

Factors contributing to higher levels of prevalence are local agricultural practices, close proximity to mammalian reservoirs (especially rodents, but also horses, sheep, goats, pigs and dogs), poor sanitation and high rainfall. The severest form of leptospirosis, known as Weil's disease, has mortality rates of up to 50%. Its non-specific clinical presentation makes it difficult to diagnose and distinguish from other infections and conditions involving acute liver failure and laboratory tests are therefore essential. According to a 2012 Cochrane review, the use of antibiotics seems to decrease the duration of symptoms by 2–4 days in mild forms, although their benefit in severe forms or Weil's disease is uncertain.³

The case considered in this article is unusual given the geographical area in which it occurred (the only case of leptospirosis reported since 1992 in La Rioja), the fact that it did not occur in summer, and the dominance of hepatic cytolysis in this patient. The patient was a 38-year-old Spanish man, who was a chef, with no medical or surgical history of interest. He occasionally smoked cocaine (at the weekend) with his last use one week prior to admission to hospital. He also drank 2 beers every weekend. He said he had not travelled abroad or taken any antibiotics or other

herbal products recently. There was no evidence of contact with farm animals, sewage or rice fields.

He had a fever (39–40 °C), myalgia, vomiting, epigastric pain and jaundice, which started 7 days prior to admission. He had taken paracetamol 1 g and ibuprofen 600 mg 3 times a day from the onset of symptoms. His vital signs upon arrival at the emergency department were: temperature 38 °C, pulse 110 beats per minute and blood pressure 91/55 mm Hg. The patient was fully oriented. A physical examination revealed conjunctival jaundice and tenderness in the epigastric region upon palpation. Initial laboratory test results were as follows: urea 85 mg/dL, creatinine 3.94 mg/dL, AST 6788 U/L, ALT 3135 U/L and direct bilirubin 2.3 mg/dL, creatine kinase 3244 U/L, leukopenia (2300) and platelets 116,000. The peripheral blood film showed no evidence of schistocytes. The chest and abdominal X-rays, electrocardiogram, abdominal ultrasound and Doppler ultrasound of the liver were normal. Antibiotic treatment with ceftriaxone and piperacillin/tazobactam was commenced in the emergency department and piperacillin/tazobactam was continued throughout the patient's hospital stay. The patient was transferred to the intensive care unit. Serological tests were ordered for hepatotropic viruses, which were all negative (hepatitis A, B, C, D and E). Likewise, serological tests for cytomegalovirus, herpes virus and Epstein Bar virus and blood cultures were all negative. Paracetamol levels were normal and ethanol levels were below 10 mg/dL. Urine toxicology screening was also negative. Twenty-four hours after admission, the patient's renal failure, thrombocytopenia, anaemia and hyperbilirubinemia worsened, requiring a plasma transfusion (1000 ml), 2 units of packed red blood cells and one unit of platelet concentrate. After observing this deterioration, a serological test for leptospirosis was ordered.

The patient had an intermittent fever, bleeding from the mouth and atrial fibrillation, which was reverted with amiodarone. His level of consciousness gradually decreased, with neurological signs of hepatic encephalopathy (asterixis, myoclonia, seizures). He also has hypotension and signs of coagulopathy (petechiae and bruising). A brain CT scan showed no acute lesions. Renal replacement therapy and inotropic medication were started that day with no signs of response, resulting in the patient's death. The results of tests for antibodies to *Leptospira interrogans* were positive, with titres of 1:1280; however, the serotype was not identified. A liver biopsy was not possible but an autopsy was ordered; the results of this are not yet available.

Weil's disease is the severest form of leptospirosis. Symptoms include fever, jaundice, renal failure, hepatic necrosis,

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