Case reports

Anaesthetic and surgical management of gastric perforation secondary to a diving incident: a case report

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Keywords
Hypoxia; Trauma; Surgery; Anaesthesia; Treatment; Ascent

Abstract

(Ben Ayad I, Damman C, vander Essen L, Majerus B. Anaesthetic and surgical management of gastric perforation secondary to a diving incident: a case report. Diving and Hyperbaric Medicine. 2024 20 December;54(4):350–353. doi: 10.28920/dhm54.4.350-353. PMID: 39675744.)

Gastric perforation secondary to barotrauma is a rare surgical condition which may manifest as an acute abdomen and potentially lead to complications such as pneumoperitoneum. A 50-year-old, healthy, experienced diving instructor was transported to our emergency department for an acute abdomen and severe dyspnoea after a diving incident. Clinical suspicion combined with computed tomography scanning lead to the diagnosis of linear rupture of the stomach. Exsufflation of the abdominal cavity was performed in the emergency department and then the patient was sent to the operating room for emergency laparoscopic gastric repair. Post-operative management was focused on decompressing the stomach with a nasogastric tube and abdominal radiography with barium ingestion was performed to confirm the absence of leakage. The patient was discharged at postoperative day four. We found 16 similar cases in the published literature. Gastric perforation secondary to a diving accident is rare but requires rapid diagnosis and surgical treatment.

Introduction

Gastric perforation secondary to a diving accident is a rare event[1] that should be suspected in any patient who presents with abdominal pain after rapid ascent. The expansion of air in the stomach during ascent usually causes abdominal discomfort or pain, but in extreme cases, it can be responsible for a gastric or intestinal rupture with the development of a huge pneumoperitoneum. In most of the cases found in the literature[1,2] the perforation is secondary to a rapid ascent or dysfunction in the diving equipment, and occurs at the level of the lesser gastric curvature. Prompt diagnosis and surgery are needed to avoid serious complications such as peritonitis and sepsis.[3] In this article, we report a case of gastric rupture following barotrauma and the specifics of our anaesthetic and surgical management.

Case report

The patient consented to deidentified publication of his case details and images.

A healthy 50-year-old man, weighing 96 kg, was admitted to the emergency room after a diving accident with abdominal pain and severe dyspnoea. The patient was an experienced diving instructor, having over 600 dives to his credit. The medical history consisted of high blood pressure treated with perindopril and amlodipine. He had a meal and a glass of champagne about six hours before the dive and nothing but clear water afterward. At 40 m depth he felt dizzy and panicked, deciding to begin a rapid ascent towards the surface, while exhaling. He was conscious for the first 20 m of ascent, then passed out after experiencing sudden abdominal pain. His team pulled him out of the water and he spontaneously regained consciousness about two minutes later. At this point, he complained of severe abdominal pain and dyspnoea.

Emergency services were immediately contacted, and when the ambulance arrived 100% oxygen first aid was provided based on an initial diagnosis of decompression sickness. The patient was first taken to a recompression centre, where doctors determined he did not require recompression therapy and referred him to our hospital for treatment of the suspected gastric rupture. Thoracic and abdominal computed tomography (CT) (Figures 1 and 2) showed severe pneumoperitoneum with suspected gastric perforation. The images also revealed a significant elevation of the diaphragm

Figure 1
Computed tomography scan (axial view) showing severe pneumoperitoneum and gastric perforation at the lesser curvature of the stomach

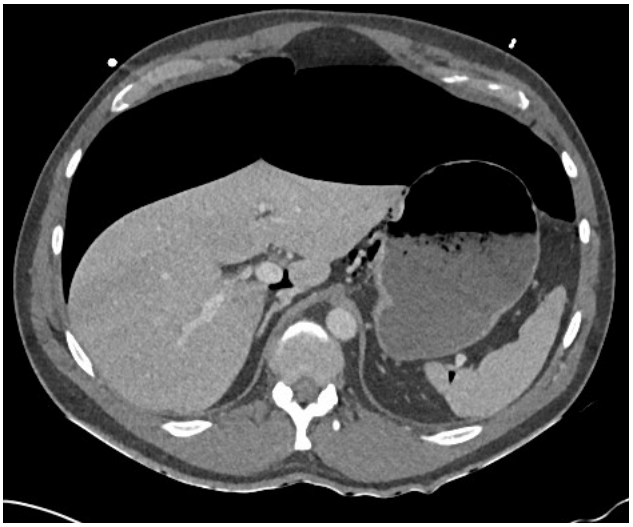
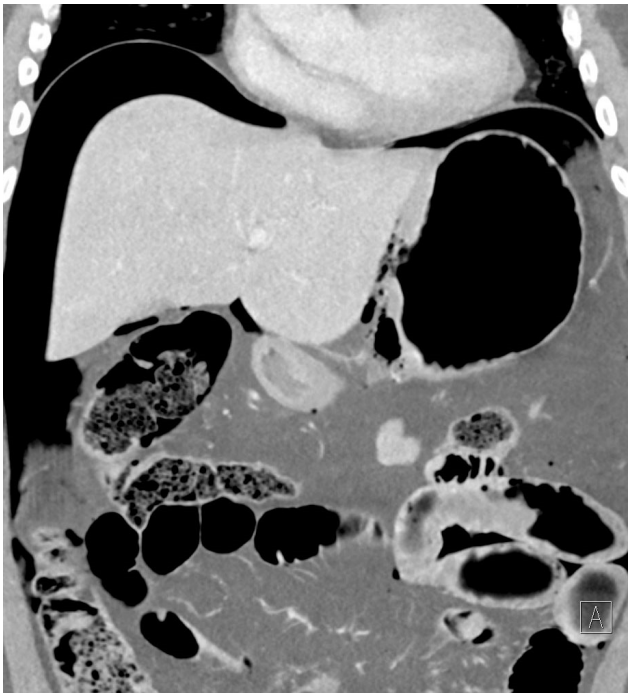


Figure 2
Computed tomography scan (coronal view) showing severe pneumoperitoneum and gastric perforation at the lesser curvature of the stomach



limiting lung expansion. The patient's respiratory condition worsened, with symptoms of respiratory failure indicated by mild hypoxaemia and tachypnoea, but he remained stable haemodynamically. After a discussion between surgeons, anaesthesiologists, and emergency physicians, it was decided to perform a bedside exsufflation of the abdomen under local anaesthesia. The patient was also administered 500 ml of intravenous NaCl. He then rapidly improved clinically and his respiratory parameters were better as oxygen supplementation was no longer required. However, abdominal distension and discomfort were still present. A gastric tube was placed, and prophylactic intravenous antibiotics were administrated. The patient was then sent to the operating room.

ANAESTHETIC CONSIDERATIONS

Laparoscopic exploration was planned to be performed under general anaesthesia with orotracheal intubation. Due to the large volume of gastric contents on the scanner and to protect the airways, we decided to perform a rapid sequence induction without ventilation after three minutes of pre-oxygenation with 100% oxygen. For the induction we used sufentanil, ketamine, lidocaine, and propofol, along with a muscle relaxant (rocuronium). For haemodynamic support, the patient was preloaded with 500 ml of IV crystalloid fluid. Our main concern for induction was the inferior vena cava, which was invisible on the CT scan, being compressed by the pneumoperitoneum. The consequent decrease in preload and thus the decrease in cardiac output posed a risk of cardiac arrest during induction due to the additional hypotensive effect of the drugs used. Despite the risk being at least partially eliminated[4] by the exsufflation performed preoperatively, we preferred to consider the patient at risk and took precautions to minimise the reduction of preload during induction. Additionally, divers are often relatively dehydrated, which prompted us to administer fluids to the patient prior to induction. In addition, surgeons were ready to perform an urgent laparotomy during induction if necessary, to relieve pressure on the inferior vena cava. Fortunately, no complication was reported with the induction of general anesthesia. Blood gas analysis was performed shortly thereafter and showed only respiratory acidosis, possibly secondary to the restrictive syndrome. Since several studies[5,6] have failed to demonstrate the effectiveness of cricoid pressure in reducing complications such as gastric regurgitation and aspiration, it has become standard practice at our institution not to use cricoid pressure. We chose rocuronium at a dose of 1.2 mg / kg over succinylcholine because it has a longer duration of action (lasting approximately 30 minutes) and can be re-administered as necessary to maintain muscle relaxation throughout the procedure. Additionally, rocuronium was chosen for its benefit of having an antidote available to reverse paralysis.

SURGICAL CONSIDERATIONS

Laparoscopic exploration was performed. A 1 cm supraumbilical incision was made for the insertion of the first optical trocar. Carbon dioxide was insufflated into the peritoneal cavity, followed by the insertion of the camera. We initially insufflated CO_2 at a pressure of 9 mmHg (instead of 12 mmHg) with a low flow rate. Once approved with our anaesthesia team, we proceeded to increase the pressure to 12 mmHg. There were omental adhesions to the peri-and infra-umbilical part of the anterior wall of the abdomen and the suspensory ligament of the liver. Complementary

Figure 3
Laparoscopic view of the haematoma at the lesser gastric curvature with contained perforation

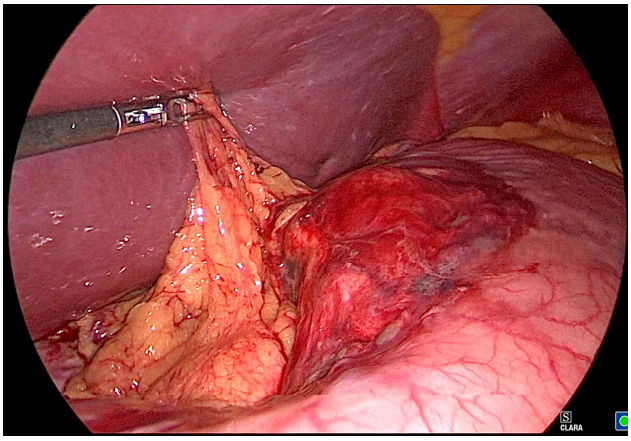
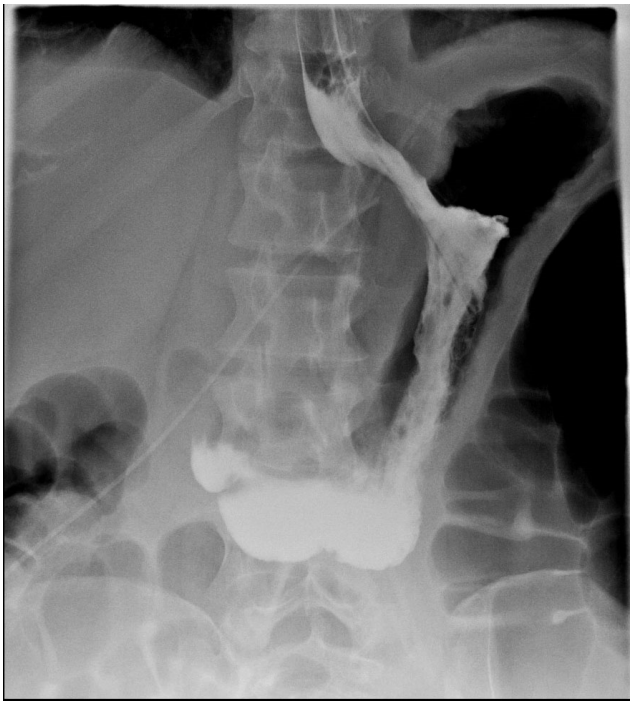


Figure 4
Radiographic examination with barium ingestion a day two after surgery confirming the absence of leakage



trocars were placed in the two hypochondria and then in the two flanks. The exposure of the stomach was made without difficulty. There was a haematoma (Figure 3) at the level of the lesser gastric curvature with suspicion of a contained perforation. Dissection along the medial border of the stomach confirmed the presence of a 6 cm long perforation, the exposure of which was improved by releasing its posterior border. A primary closure was then performed with a V-LOC 2-0 absorbable suture. Injection via a gastric tube of 700 ml of methylene blue-stained saline solution confirmed gastric wall integrity. The abdominal cavity was checked and widely washed, including the omental bursa and pelvic cavity. Two drains were positioned along the stomach. The patient was extubated at the end of the surgery and kept under close observation. A radiographic examination of the upper gastrointestinal tract with ingestion of gastrografin (barium) was performed two days after surgery to confirm the absence of leakage (Figure 4). Nasogastric aspiration was then discontinued and the patient was allowed to eat. He was discharged safely on the fourth day without any symptoms except for mild pain at the incision sites. His recovery progressed smoothly, and he achieved a full recovery by his six-week follow-up.

Discussion

Gastric rupture secondary to barotrauma during a diving accident is a rare event but should be suspected based on history. Quick diagnosis is required as potentially lethal complications such as peritonitis and sepsis are to be considered. Specific pathophysiological considerations must be understood (e.g., pneumoperitoneum) as they can be challenges for the induction of general anesthesia and the surgical procedure.

Our PubMed search using the terms 'diving' OR 'barotrauma' AND 'gastric' OR 'stomach' AND 'perforation' OR 'rupture' revealed 16 case reports. In virtually all the reported cases, gastric perforation typically occurs at the lesser curvature where the stomach is relatively fixed to adjacent structures and presents a single muscular layer and fewer mucosal folds, making it less elastic than the rest of the stomach. Rapid ascents lead to increases in airspace volumes, potentially resulting in barotrauma and decompression sickness,[7] which is why they are consistently discouraged. The pneumoperitoneum secondary to a gastric rupture will lead to a compression of other intra-abdominal structures, including the inferior vena cava, and push the diaphragm upwards, potentially causing an acute respiratory insufficiency. At sea level, the atmospheric pressure is 101 kPa, and each metre of descent increases the hydrostatic pressure by approximately 10 kPa.[8] The absolute pressure is the sum of the atmospheric pressure + the hydrostatic pressure, meaning that at 40 metres it is about 500 kPa, and at 20 metres around 300 kPa. We can then observe that the pressure increases by 100% between the surface and 10 metres and by only 20% between 30 and 40 metres. According to Boyle's law (pressure x volume = constant) a rapid ascent from 40 metres would result in up to a 5-fold increase in gas volume in the stomach (depending on its distensibility). Since our patient did not drink any soda beverage before the dive and had a presumed empty stomach (his last meal was taken six hours before the dive), the most likely origin of the gas in his stomach is aerophagia, either caused by panic or by reflex during the dive or ascent.

Under normal conditions, the oesophagus functions as a pressure release valve, opening when stomach pressure exceeds a certain threshold to prevent excessive buildup. In this case, however, two scenarios are possible: a pre-

existing weakness in the gastric wall could have led to rupture at a lower pressure than the oesophageal opening threshold, or the pressure required to open the oesophagus was unusually high, allowing the stomach to rupture without venting. Potential contributing factors to the gastric wall's vulnerability, prior to the dive, include chronic gastritis, peptic ulcers, or subclinical tears that compromised the stomach lining. Furthermore, previous reports have identified fundoplication, a surgical procedure to treat gastroesophageal reflux disease, as a risk factor for gastric rupture among divers.[9]

Stomach rupture has occurred after ascent from varying diving depths, with documented cases reported as shallow as 27 metres.[10] The majority of documented cases involving gastric rupture from barotrauma are typically treated through operative management.[11,12] However, there are a few instances where non-operative approaches have been successfully employed for small, localised perforations in otherwise healthy, minimally symptomatic patients.[13,14] Rapid ascents from moderate depths that are likely to cause gastric barotrauma may also result in pulmonary barotrauma and arterial gas embolism and decompression sickness.[7] It has been suggested that if arterial gas embolism or decompression sickness is present or suspected, the patient should be admitted to a recompression centre and hyperbaric oxygen treatment should be administered prior to surgery.[10]

Conclusions

Gastric rupture secondary to a diving accident is rare but requires rapid diagnosis and surgical treatment. Understanding this rare complication and its underlying pathophysiology is important because treatment should be administered emergently. Our case report differs by emphasising our use of pre-induction bedside abdominal decompression, which has not been prominently featured in prior reports. This preventive strategy before induction of anaesthesia aimed to reduce intra-abdominal pressure, thereby mitigating the risk of difficulty with ventilation, induction-related hypotension and cardiovascular collapse.

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Conflicts of interest and funding: nil

Submitted: 21 April 2024
Accepted after revision: 24 August 2024