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Gastric emphysema secondary to laparoscopic gastric band erosion



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ABSTRACT

INTRODUCTION: Gastric band erosion is a known complication of adjustable gastric band surgery. There are no previous reports of gastric band erosion associated with gastric emphysema (GE) or emphysematous gastritis (EG), a rare condition with a mortality rate exceeding 50%.

PRESENTATION OF CASE: We report the first known case of GE found in a 58-year-old lady presenting with acute onset epigastric abdominal pain and haematemesis in the setting of a chronically eroded gastric band. GE was visualised in the anterior gastric wall of the stomach without evidence of EG. Endoscopic and surgical examination of the stomach was undertaken along with band removal followed by defect repair.

DISCUSSION: GE can result from obstructive, traumatic and pulmonary causes. EG is a rare and often lethal form of GE resulting from bacterial invasion of the gastric wall through a mucosal defect leading to sepsis and gastric necrosis. Early reports documented early definitive operative debridement of necrotic gastric wall of patients with EG while recent reports have demonstrated a feasible non-operative approach among highly selected patients with no evidence of gastric necrosis. There are no previous reports on the treatment of patients with gastric band erosion and suspected EG.

CONCLUSION: Patients presenting acutely with symptomatic gastric band erosion, radiological evidence of GE with evidence of leucocytosis, peritonism or sepsis may develop EG. A high index of suspicion, low threshold for operative exploration and optimal management with antimicrobial therapy and close supportive care are necessary to ensure the best survival outcomes for these patients.

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1. Introduction

Gastric-banding is a well-known restrictive bariatric surgery technique that has established benefits of weight loss and reduction in metabolic and cardiovascular disease burden.¹ It has relatively fewer serious complications compared to other techniques such as Roux-en-Y gastric bypass. Band specific complications including band slippage, band erosion, pouch or oesophageal dilatation, port-site malfunction or device leakage.² We report on the first case of gastric emphysema (GE) from gastric band erosion to highlight the importance of distinguishing between the multiple causes for this rare finding in order to provide the most appropriate treatment.

2. Case presentation

A 58-year-old lady presented to the emergency department with a one day history of progressive constant, non-radiating epigastric pain and sudden onset haematemesis. She was brought in by ambulance for urgent review after four episodes of bright

haematemesis with clots and associated dizziness. She reported a recent history of reflux symptoms but denied any previous episodes of haematemesis, malaena or rectal bleeding. Her bowels had opened twice that day with soft, brown stools and was actively passing flatus.

Past medical history included laparoscopic adjustable gastric band insertion in 2000, hysterectomy, diverticular disease, moderate obesity, insulin-dependent diabetes mellitus, asthma, hypertension and hypercholesterolaemia. Her gastric band port had been dislodged into her pelvis five years earlier. There was no previous history of gastric band slippage or erosion. She denied any gastroscopies and colonoscopies within the past five years. Her regular medications included her insulin regimen, rosuvastatin and irbesartan.

On clinical examination she appeared generally well with mild pallor. She had mild tachycardia (115 beats/minute) with a normal blood pressure (systolic 150 mmHg). She remained afebrile during her emergency department admission. Abdominal examination revealed an upper midline incisional scar. She had epigastric tenderness with guarding. Her abdomen was otherwise soft with a negative Murphy's sign and no palpable masses. The gastric band port was not palpable.

Laboratory findings revealed leucocytosis (14.8×10^9 cells/L) with neutrophilia (10.2×10^9 cells/L). A mildly raised lactate

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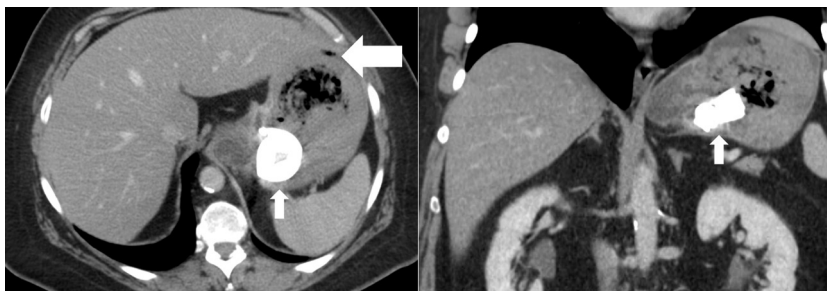


Fig. 1. Gastric band erosion in posterior gastric wall (small arrows) with gastric emphysema (large arrow)

level was seen on venous blood gas analysis (2.8 mmol/L) with normal pH (7.38) and base excess (−2.1 mmol/L). Her biochemistry was unremarkable aside from hyperglycaemia (27.1 mmol/L).

An abdominal and pelvic computed tomography (CT) scan with intravenous contrast was performed on the suspicion of a perforated gastric or duodenal ulcer. The gastric band was malpositioned with each end pointing towards the 3 and 9 o'clock positions on the coronal view. It also appeared to be in the posterior wall of the gastric fundus with marked wall thickening (Fig. 1) and gastric emphysema (Fig. 2). There was prominent debris within the lumen distending the duodenal cap and pylorus. The gastric band port remained attached to the band lying in the upper right pelvis. There was no evidence of free intraperitoneal gas, fluid or pneumomediastinum.

The patient proceeded to laparoscopy on a provisional diagnosis of gastric ischaemia secondary to gastric prolapse due to band slippage. She was given preoperative doses of ceftriaxone 1 g, metronidazole 500 mg and pantoprazole 40 mg. Initial laparoscopic examination revealed dense adhesions involving the lesser curve of the stomach but no evidence of the band. An estimated 800–1000 ml of clotted blood was found within the stomach on gastroscopy. Adequate views were difficult to obtain due to the significant haematoma. A laparoscopic anterior gastrotomy was performed for further exploration but was impaired by significant inflammation and dense clot formation within the stomach. A midline laparotomy was performed to improve visualisation. A chronically eroded band was found after clot evacuation. The band appeared to be 80% intraluminal with no evidence of active bleeding or gastric necrosis. The posterior gastric wall defect and anterior gastrotomy were repaired with 3-0 polydioxanone sutures after band and port removal. A drain was placed alongside the lesser curve and abdominal wall closed primarily.

The patient was diagnosed with gastric band erosion and associated GE without evidence of EG. She was managed in the intensive care unit for eight days, extubated on postoperative day one and commenced on sips of water progressing to clear fluids. There was no evidence of a leak on injection of methylene blue dye via nasogastric tube or an oral gastrograffin contrast abdominal CT scan on day four. Parenteral feeding was commenced on day four due to her inability to tolerate enteral feeding and continued until she was able to tolerate a full oral diet. Her nasogastric tube was removed on day six and abdominal drain on day seven. Intravenous antibiotics and oral pantoprazole were continued until discharge. She was instructed to continue oral pantoprazole 40 mg twice daily until her next outpatient review.

Our patient developed several minor complications. These included a small inferior wound separation treated by negative pressure dressing, atrial fibrillation that resolved with an amiodarone infusion and left subphrenic collection that did not require drainage (Fig. 3). There were no major complications.

3. Discussion

We have described, to our knowledge, the first reported case of GE secondary to gastric erosion from a laparoscopic gastric band. Our patient was a middle-aged lady with multiple metabolic comorbidities that responded well to operative repair of her band erosion. She did not require debridement and was discharged after a short postoperative recovery. This patient had a prosthetic-related cause of their pneumatosis as opposed to an infectious mechanism seen in EG, a condition which often has a similar clinical presentation but may be managed non-operatively with antibiotic and supportive therapy.³ Nonetheless, we recognise the importance of operative management in cases of gastric band erosion with radiological evidence of GE to exclude EG. Gastric band erosion is a rare major complication of laparoscopic adjustable gastric band surgery with incidence rates ranging from 0.2 to 0.5%.^{4,5} Proposed mechanisms include iatrogenic injuries at the time of band implantation leading to micro-abscess formation and progressively to gastric wall perforation.^{6,7} Other theories include chronic gastric wall ischaemia and foreign body reactions especially in chronic band erosion.⁵ Most erosions, including our patient, are likely multifactorial events that involve a combination of these events.

GE is a radiological sign of air or gas within the stomach wall. It is a rare condition that may result from self-limiting to life-threatening causes such as EG. These can be categorised into obstructive, traumatic and pulmonary mechanisms producing a mucosal tear, decreased mucosal integrity or a ruptured pulmonary bullae leading to emphysema. Multiple reports have since described this finding in newborns to adults and from infectious EG⁸ to non-infectious causes such as instrumentation,⁹ orthotic devices,¹⁰ gastric outlet obstruction,¹¹ ingested toxins,¹² pulmonary disease,¹³ pancreatitis,¹⁴ atypical hernias,¹⁵ gastric volvulus or small bowel obstruction.¹⁶ Distinguishing between EG and other more benign causes of GE is important to ensure the patient receives the most appropriate management.

EG is a condition characterised by severe gastric wall inflammation secondary to bacterial invasion, systemic toxicity and radiologic findings of GE. It has a significantly higher overall mortality rate up to 61% compared to other benign causes of GE.^{17,18} Most bacterial pathogens have been implicated with non-haemolytic *Streptococci*, *Escherichia coli*, *Enterobacter* species, *Pseudomonas aeruginosa* and *Clostridium perfringens* the most commonly isolated organisms on gastric biopsy or blood culture.¹⁹ It is distinguished from GE based on clinical and laboratory evidence of bacterial infection. Patients typically present with evidence of vomiting, peritonism, sepsis and occasionally haematemesis along with leucocytosis. Those with risk factors such as diabetes, COPD, renal failure, leukaemia, lymphoma, steroids, immunosuppressant therapy, alcohol abuse, ingestion of corrosive substances, malignancy or post-abdominal surgery are at higher risk of developing EG.²⁰

Early reports advocated aggressive surgical debridement for EG with variable outcomes, notably among unwell patients with

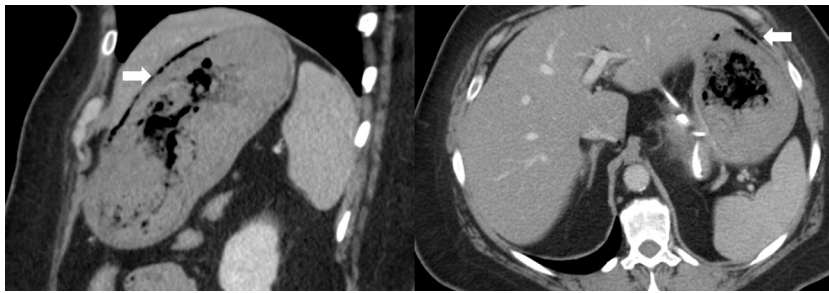


Fig. 2. Anterior wall gastric emphysema (small arrows)

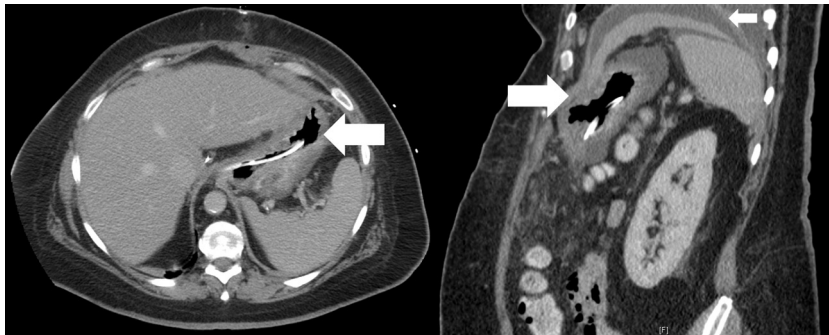


Fig. 3. Complete resolution of gastric emphysema (large arrows) and simple left subphrenic collection (small arrow)

multiple chronic medical conditions.¹⁷ More recently patients with EG have been treated successfully using a non-operative approach of antibiotic, supportive therapy with endoscopic surveillance for gastric necrosis.^{3,21} One approach involved initial stabilisation of the septic EG patient with fluid resuscitation and empirical gram-negative and anaerobic antibiotic cover.¹⁷ Operative management (subtotal or total gastrectomy) is appropriate in patients that have large areas of gastric necrosis, peritonism, gastric infarction or failed optimal medical management. Patients with small areas of necrosis and minimal co-morbidities may be successfully managed with medical therapy alone.

Our case highlights the importance of maintaining a high index of suspicion for EG from band erosion among laparoscopic gastric band patients presenting acutely unwell with upper abdominal pain as prompt surgical intervention among those with multiple comorbidities may improve their survival. Although our patient did not develop EG, her tachycardia and leucocytosis were concerning enough to warrant an endoscopic and subsequent laparoscopic examination of her stomach to exclude necrosis. Recognising the potential for EG among patients with band erosion is important to ensure they receive timely operative management and adequate supportive care and aggressive antimicrobial therapy in a high-dependency unit.

4. Conclusion

Emphysematous gastritis is a devastating condition with considerable mortality. Gastric band erosion can produce signs of gastric emphysema and may predispose patients to EG. Early endoscopic and surgical examination of gastric integrity was valuable in excluding EG in our patient. Those with evidence of necrotic EG require surgical debridement along with antimicrobial therapy and supportive care in a high dependency unit to ensure optimal survival.

Conflict of interest statement

We have no conflicts of interest to declare.

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All costs related to this research paper were covered by the authors.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Authors' contributions

Michael Su: study design, data acquisition, drafting and critical revision of the manuscript. William Munro: study design, drafting and critical revision of the manuscript.

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