EJCRIM
European Journal
of Case Reports
in Internal Medicine

ISCHAEMIC GASTROPATHY: AN UNDER-RECOGNISED CAUSE OF ABDOMINAL PAIN

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Received: 24/01/2024 **Accepted:** 02/02/2024 **Published:** 28/02/2024

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Signed consent was obtained from the patient.

How to cite this article: Touza M, Emehinola O, Ali R, Jamil A, Hussain M, Atiyat R, Miller R. Ischaemic gastropathy: an under-recognised cause of abdominal pain. *EJCRIM* 2024;**11**:doi:10.12890/2024_004322.

ABSTRACT

Ischaemic gastropathy is an under-recognised phenomenon with a particularly poor prognosis, where early diagnosis is crucial for successful medical intervention and the prevention of life-threatening complications. We present a case involving a 42-year-old female with no history of vascular insufficiency who developed ischaemic gastropathy following a prolonged stay in the intensive care unit, from septic shock secondary to *Escherichia coli* bacteraemia due to complicated acute appendicitis. This case underscores the importance of the physician's awareness regarding this rare entity and the necessity to consider it in the differential diagnosis of abdominal pain and haematemesis.

Prompt diagnosis and treatment may significantly improve survival outcomes in this less-documented pathology, especially in the younger adult population.

KEYWORDS

Ischaemic gastropathy, gastric hypoperfusion, gastric ischaemia, appendectomy

LEARNING POINTS

- Awareness needs to be increased regarding the consideration of ischaemic gastropathy as a differential diagnosis.
- A patient without a history of vascular compromise could have a diagnosis of ischaemic gastropathy.
- This is possibly the first noted case of ischaemic gastropathy occurring after an appendectomy, which is complicated by gram-negative bacteraemia and haemodynamic instability.

INTRODUCTION

Gastric ischaemia, a seldom-seen cause of gastropathy, is considered an uncommon entity due to the stomach's robust collateral blood supply. It is infrequently documented in the literature, particularly among the younger adult population, leading to clinical and histopathological under-recognition of this condition[1]. While a few cases of ischaemic gastropathy have been reported in the elderly population with risk factors for ischaemic events, our knowledge is limited. This case presents a unique instance of a 42-year-old female developing ischaemic gastropathy in the absence of significant vascular abnormalities, with critical illness requiring prolonged pressors due to Gram-negative bacteraemia and major surgery identified as probable risk factors for gastric hypoperfusion. To our knowledge, this is the first reported case of ischaemic gastropathy following

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an appendectomy. This case underscores the significance of early diagnosis through endoscopy and an intervention plan to enhance survival outcomes in such patients.

CASE DESCRIPTION

A 42-year-old female with no past medical history presented to the Emergency Department with complaints of fever, worsening generalised abdominal pain, nausea, non-bloody, non-bilious vomiting, being unable to tolerate diet and having diarrhoea for the previous 5 days. On evaluation, she was febrile with a max temperature of 38.3 °C, heart rate was 112 bpm, respiratory rate 36, blood pressure 99/42 mmHg and saturating 97% on room air. On physical examination, she appeared ill and in moderate respiratory distress due to pain. Her abdominal examination revealed generalised tenderness, peritoneal signs on the right lower quadrant and hypoactive bowel sounds. A cardiac examination showed tachycardia with normal S1 and S2. Respiratory examination was positive for tachypnoea with no adventitious lung sounds heard. Her admission laboratory tests were significant for leukocytosis and hypokalaemia; the remainder of her test results are shown in *Table 1*. Abdominal X-ray showed a non-obstructive bowel gas pattern, and a chest X-ray was unremarkable. A computed tomography (CT) scan of the abdomen and pelvis with oral contrast showed extensive inflammatory changes in the right lower quadrant adjacent to the cecum that ascends to the level of Morison's pouch, along with contained extraluminal air suspicious of ruptured appendicitis. The patient was intubated due to acute respiratory distress and started on broad-spectrum antibiotics, fluids, and vasopressor support for hypotension. She was taken to the operating room, and an open appendectomy was performed. She was found to have gangrenous necrotic acute appendicitis with perforation. Blood cultures grew Gram-negative rods (4/4 bottles) within 24 hours, and identification came back as pan-sensitive *Escherichia coli*. During the course of her hospital stay, she was treated for septic shock secondary to *E. coli* bacteraemia and renal failure secondary to septic shock, which required a brief period of continuous renal replacement therapy. She

Laboratory parameters	Values	Reference range
Sodium	129	136–145 mmol/l
Potassium	2.8	3.5–5.3 mmol/l
Chloride	97	98–110 mmol/l
Blood urea nitrogen (BUN)	40	6–24 mg/dl
Creatinine	2.4	0.6–1.2 mg/dl
Aspartate transaminase (AST)	24	10–36 U/l
Alanine transaminase (ALT)	18	9–46 U/l
White blood cell (WBC)	3.7	4.4–11 × 10^3/ul
Haemoglobin	14.4	13.5–17.3 g/dl
Platelets	133	150–450 × 10*3/µl

Table 1. Laboratory test results.

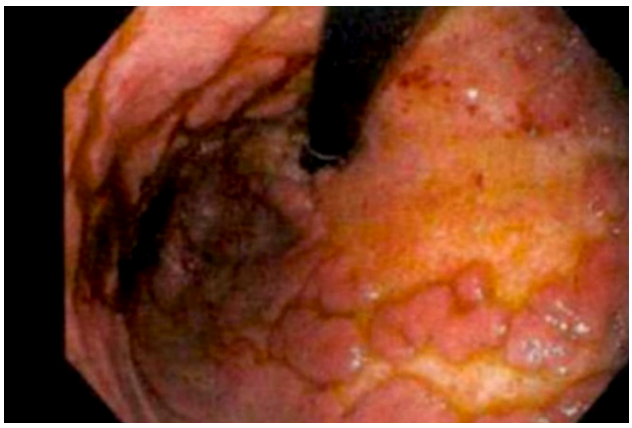

Figure 1. Retroflexion view showing ulcerated mucosa with giant ulcers containing exudates surrounded by pale, friable and nodular mucosa.

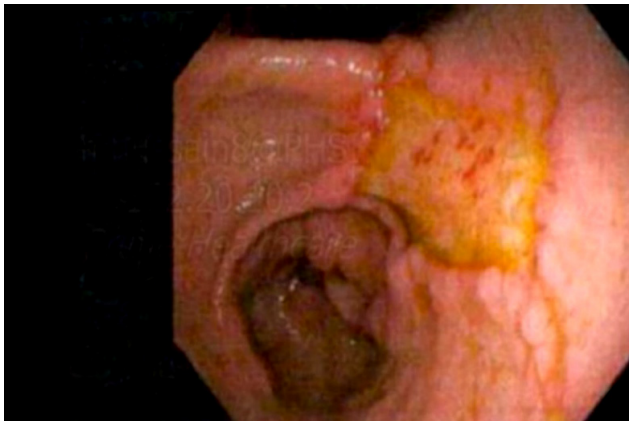

Figure 2. Anteflexion view demonstrates a giant ulcer with exudates surrounded by oedematous and nodular mucosa.

was persistently on vasopressors during the first week of her hospital course and tapered down completely by the end of the second week. She was successfully extubated after two weeks and started to make urine with improved renal function. She was initially started on a clear liquid diet and then advanced to a regular diet as tolerated. However, she continued to complain of moderate abdominal pain with judicious use of paracetamol and morphine for pain relief. On day 17, the patient started having episodes of haematemesis with blood clots, and a drop in haemoglobin from 14.4 to 8 g/dl. She was started with intravenous pantoprazole, and the gastroenterology department was consulted. On medication review, she was not given any non-steroid anti-inflammatory drugs (NSAIDs) and the patient also denied taking any NSAIDs at home. An oesophagogastroduodenoscopy (as shown in *Fig. 1 and 2*) showed ulcerated mucosa with giant ulcers in the gastric antrum and proximal corpus with exudates surrounded by the pale, friable, and nodular mucosa. Histopathology showed acute inflammation with mucosal ulceration and immunohistochemical staining negative for *Helicobacter pylori*. Vascular duplex ultrasound of abdomen and pelvis showed patent mesenteric arteries. Her complex septic shock course led to ventilator-dependent respiratory failure due to intra-abdominal infection. Coupled with endoscopic findings, negative biopsies ruling out other causes and the absence of risk factors for peptic

ulcer disease such as NSAIDs or *H. pylori* infection, these manifestations were indicative of ischaemic gastropathy. The patient was managed conservatively with intravenous pantoprazole 40 mg twice daily, and sucralfate. She did not have further episodes of upper gastrointestinal bleeding and her haemoglobin remained stable over the next few days. She was discharged with close outpatient follow-up with gastroenterology.

DISCUSSION

The term 'gastropathy' refers to epithelial or endothelial damage without inflammation, whereas 'gastritis' refers to conditions characterised by evidence of inflammation. Ischaemic gastropathy can manifest with structural features such as mucosal congestion, erosive/ulcerative lesions with or without haemorrhage, and overt mucosal necrosis. Functional impairments may also be present, including gastroparesis and gastric dysrhythmias[2]. The extensive vascular network of the stomach contributes to a low incidence of isolated gastric ischaemia. Despite being less frequently documented in the literature, it is not entirely uncommon. In our specific case, the patient had no history of vascular abnormality but developed ischaemic gastropathy following a prolonged intensive care unit stay due to septic shock secondary to complicated acute appendicitis.

Acute haemorrhagic erosive gastropathy can be caused by medications such as NSAIDs, alcohol, stress and portal hypertension, or may result from a decrease in blood flow[3]. An uncommon cause of erosive gastropathy is ischaemia, a condition that can be triggered by factors such as hypovolaemia, sepsis, trauma and mucosal prolapse. Similar concepts apply as in non-occlusive mesenteric ischaemia. Furthermore, damage to the central nervous system can lead to ulcerative lesions and inflammation in the stomach and duodenum, referred to as Cushing's ulcers. The damage inflicted on the gastric mucosal wall allows acid and other substances to penetrate the lamina propria, causing injury to the vasculature and resulting in ischaemia. Oxygen-free radicals produced by infiltrating neutrophils exacerbate mucosal injury during reperfusion following ischaemia. This pathogenesis disrupts the protective barrier, including the epithelium, bicarbonates and secreted mucins[4]. Cocaine use is another mechanism that could trigger ischaemic gastropathy due to vasoconstriction and subsequent ischaemia[5].

Erosive gastropathy typically presents as asymptomatic, although patients may exhibit symptoms such as anorexia, gastric pain, haematemesis (coffee ground), emesis, bloody aspirate and melaena. It is rare for patients to experience haemodynamic instability due to bleeding, since gastritis is generally superficial[3]. In the case of Cushing's ulcers, lesions tend to appear near the gastroesophageal junction in the fundus and the body of the stomach. Erosions caused by alcohol or NSAID use are usually smaller and may reverse more rapidly compared to lesions in ischaemic gastropathy. Shock-induced haemodynamic instability is more likely to result in ulcerations, typically located in the corpus and fundus, with a shallow diameter of 0.5 cm to 2 cm. More recent ulcers appear necrotic, while older ones demonstrate granulation tissue and inflammation with epithelial cell regeneration[6-9].

Ischaemic gastropathy, although rare, carries a poor prognosis and necessitates early diagnosis and intervention for appropriate management. The diagnostic workup for gastric ischaemia includes endoscopy and the use of imaging modalities such as CT scan or CT angiography, as well as selective mesenteric angiography. In our case, the diagnosis was established through endoscopy, revealing severe inflammation with oedema, erosion, friability, granularity, nodularity, and deep ulceration throughout the entire stomach.

The management of gastropathy is contingent on addressing the underlying cause. During the acute phase, the primary objectives involve eliminating the causative agent, reversing the initial vascular compromise, and implementing fluid resuscitation. To prevent and alleviate gastric distension, measures include air and fluid aspiration with a nasogastric tube placement. Intravenous proton pump inhibitors are utilised to reduce acidity, and in cases of sepsis or gastric pneumatosis, broad-spectrum antibiotics are administered. However, chronic ischaemia requires corrective surgical intervention[1]. In our case, symptomatic management led to a gradual improvement in symptoms.

CONCLUSION

Ischaemic gastropathy is an exceptionally rare phenomenon, particularly among patients lacking any risk factors for ischaemic events, given the rich collateral blood supply of the stomach. The potential for a missed diagnosis of this condition underscores the importance of considering it in the differential diagnosis of abdominal pain and haematemesis in a hospitalised patient, as failure to recognise it can result in heightened morbidity and mortality[1]. Therefore, we emphasise that, despite its rarity, clinicians should include ischaemic gastropathy in their considerations when evaluating patients with prolonged hospital courses complicated with shock, developing abdominal pain and haematemesis. Early diagnosis and the implementation of aggressive management are imperative for enhancing outcomes in such cases.

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