Hyperamylasaemia and ischaemic colitis

F. Kum a,*, A. Gulati b, A. Hussain a,c

a Department of General Surgery, Princess Royal University Hospital, Farnborough Common BR6 8ND, United Kingdom
b Department of General Surgery, Norfolk & Norwich University Hospital, Norwich NR4 7UY, United Kingdom
c King’s College London Medical School, London SE1, United Kingdom

ABSTRACT

INTRODUCTION: Ischaemic colitis is a differential diagnosis to be considered in patients who have a high cardiovascular risk. Presentation of severe ischaemia is usually that of an acute abdomen with passage of fresh blood per rectum, and hyperamylasaemia.

PRESENTATION OF CASE: A 66-year-old gentleman was admitted to A&E with a short history of central abdominal pain, nausea, vomiting and fresh bleeding per rectum. A diagnosis of ischaemic colitis was made by the computed tomography (CT) scan findings of colonic thickening and pneumatosis, in addition to colonoscopy demonstrating sloughy mucosa and ulceration. Symptoms did not resolve with conservative management, therefore laparotomy + Hartmann’s procedure was performed. Histology showed extensive areas of both partial and full thickness ischaemia with stricture.

DISCUSSION: Amylase is an indicator of intra-abdominal inflammatory processes. Hyperamylasaemia (normal <100 U/L) is most frequently associated with pancreatitis; however, causation is not exclusive and other differentials including bowel ischaemia must be considered, although amylase is not a specific marker for ischaemic colitis. It is important to distinguish between ischaemic and ulcerative colitis.

CONCLUSION: Intestinal ischaemia is a serious acute abdominal pathology that is associated with hyperamylasaemia, and frequently requires prompt surgical intervention to prevent subsequent mortality.

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1. Introduction

Ischaemic colitis is a differential diagnosis to be considered in patients who have a high cardiovascular risk. Presentation of severe ischaemia is usually that of an acute abdomen with passage of fresh blood per rectum, and hyperamylasaemia also features. Incidence is estimated as 7.2 per 100,000 person-years, with increasing frequency with age and in those with co-existing cardiovascular risk factors. Low-grade chronic ischaemia may be asymptomatic and spontaneously resolve; however, as in the case presented here, it is important to recognise early symptoms for prompt management, which may include surgical resection of the ischaemic bowel and stoma formation.

2. Case presentation

A 66-year-old gentleman was brought to A&E as a blue light call with an immediate preceding history of sudden onset severe central and lower abdominal pain, associated with nausea and vomiting. He also reported several episodes of fresh bleeding per rectum (PR) on the same day of admission, prior to which he reported a 2-week history of intermittent constipation and loss of appetite. He reported no recent fevers or other associated symptoms or illnesses.

The patient had a known extensive vasculopathic history comprised of hypertension, ischaemic heart disease, a previous stroke, atrial fibrillation and an abdominal aortic aneurysm (AAA) of 4.3 cm. He had undergone a colonic polypectomy 2 years ago, with otherwise normal colonoscopy and there was no family history of bowel cancers. Drug history included Acenocoumarin (Sintrom), a new coumarin anticoagulant.

At paramedics’ arrival he was hypotensive at 68/38 mmHg and tachycardic at 140 bpm, but responded to initial fluid resuscitation, BP 89/52 mmHg, pulse 120 bpm in A&E. Abdominal examination elicited lower abdominal tenderness and a palpable AAA. Rectal examination showed fresh blood.

Amylase (3776 U/L), lactate dehydrogenase (492 IU/L), urea (11.8 mmol/l) and creatinine (149 μmol/l) were all raised. Arterial blood gas (ABG) analysis revealed a metabolic acidosis of pH 7.33 and a raised lactate of 3.49 mmol/l with an accompanying base excess of -5.3 mmol/l. His Modified Glasgow Score for pancreatitis was 1, indicating a low suspicion for this diagnosis.

Conservative management was started, consisting of intravenous fluid resuscitation, antibiotics, nil-by-mouth, nasogastric-tube insertion, urinary catheterization and close observation. Chest
Fig. 1. Chest X-ray showing mild bi-basal shadowing, but no evidence of bowel perforation.

X-ray did not show any sign of perforation (Fig. 1). An urgent CT scan reported circumferential bowel wall thickening in the descending and sigmoid colon and pneumatosis, indicative of possible transmural infarction (Fig. 2). Flexible sigmoidoscopy and subsequent colonoscopy (Fig. 3) found ischaemic colitis of the sigmoid colon characterised by sloughy mucosa and ulceration.

There was negligible clinical improvement with conservative management; therefore, on day 3 of admission, it was decided to proceed with laparotomy + Hartmann’s procedure. Pre-operatively, haemoglobin was 10.2 g/dl, platelets 162 × 10⁹/l and INR 2.0, therefore 2 units of fresh frozen plasma were given.

Macroscopically, the excised colonic sample was 360 mm x 35 mm, with areas of bowel wall measuring up to 8 mm thick and ischaemia extending to one margin of the sample. Microscopically, histology showed extensive partial thickness infarction characterised by mucosal ischaemia and submucosal oedema. There was also a focal area of full thickness ischaemia. A stricture was noted on histology, but no malignancy was present and the lymph nodes identified were reactive.

Intensive therapy unit (ITU) care was required in the immediate post-operative period and the patient made an uneventful recovery, albeit a minor wound infection around the 7th post-operative

Fig. 2. Axial CT scan slices. CT scan showed circumferential bowel wall thickening in the descending and sigmoid colon and pneumatosis. No evidence of AAA leakage, infrarenal aneurysm measured 4.3 cm.

Fig. 3. Colonoscopy revealed findings consistent with moderately active ischaemic colitis of the sigmoid colon: (a) descending colon, (b) upper sigmoid, (c and d) sigmoid colon.
day, which was successfully treated. The patient was discharged home and has made good progress since. By 6 months post-operatively he had gained 1 stone in weight and has since had a successful reversal of the colostomy.

3. Discussion

This case illustrates the importance of full assessment and investigation in the work-up of a patient with an acute abdomen. Efficient management of such cases is essential for prevention of local intra-abdominal and subsequent systemic complications.

Amylase is an acute phase indicator, often of intra-abdominal inflammatory processes, one of which being bowel ischaemia. Hyperamylasaemia (normal <100U/l) is most frequently associated with pancreatitis, however causation is not exclusive. Other differentials should be considered including parotid or salivary gland inflammation or recent surgery, sialadenitis, sarcoidosis, connective tissue diseases such as systemic lupus erythematosus and Sjögren’s syndrome, anorexia and bulimia, diabetic ketoacidosis, porphyria, pregnancy, chronic renal failure, burns and splenic injury.

Amongst the plethora of differential diagnoses of the acute abdomen, a high index of suspicion for acute gut ischaemia should be considered, particularly for the patient presented here, who was of older age, had an extensive vasculopathic history, and a raised lactate.

Although amylase levels, along with various other biochemical markers, including lactate dehydrogenase, creatine phosphokinase and leucocytes have been investigated in acute bowel ischaemia, none has been found to be sufficiently specific to diagnose ischaemic colitis. Furthermore, an elevation of the aforementioned markers is uncommon in mild ischaemia and they are only increased in more advanced stages of the disease process. There is a general lack of evidence regarding the use of amylase as a prognostic indicator in the conservative management of ischaemic colitis, and as such, this is a potential area for future research.

Despite the greater availability of CT scanning, colonoscopy remains the gold standard for diagnosis of colonic ischaemia. Suggestive findings are seen in 75% of patients and colonoscopy has a better sensitivity when compared with CT scans. Furthermore, colonoscopy enables direct visualisation and biopsy of lesions. Barium enema studies are becoming obsolete in the work-up of many patients as residue obscures vision and interferes with later angiography.

Ischaemic colitis occurs as a spectrum of severities, and patients with chronic gut ischaemia may be asymptomatic with only mild, transient ischaemia to the submucosal level. However, in the acute setting with associated abdominal pain and haemodynamic instability, it is a serious condition resulting in 20% requiring surgical intervention. Risk factors for developing ischaemic bowel include hypertension, diabetes mellitus, atrial fibrillation, coronary artery disease, peripheral vascular disease and renal failure, particularly those requiring haemodialysis. Splicenic flexure is reported as the most common site for ischaemic colitis, a contributory factor to this being the congenital absence of the marginal artery of Drummond in 5% of the population.

Indications for early surgery include signs of haemodynamic instability, sepsis, peritonitis, perforation and pneumoperitoneum, or gangrene of the ischaemic segment. Surgery must also be considered if symptoms are unresolving and refractory to conservative management. Stoma formation is preferred following resection for ischaemia and reversal can be considered at a later date.

An estimated 80–85% of patients with gut ischaemia recover with conservative management and improvements are seen within a few days. Long-term effects of ischaemic colitis following conservative management may include ischaemic strictureing and fibrosis, which are an indication for later surgical intervention.

It is important to distinguish between ischaemic colitis and exacerbations of ulcerative colitis as both clinical presentations are very similar. Ischaemic colitis may supersede underlying ulcerative colitis. The differentiating factor may be that of a raised lactate and raised amylase in ischaemic pathologies with a less marked inflammatory white cell and neutrophilic response than in ulcerative colitis. At colonoscopy, a tendency towards diagnosis of ischaemic rather than inflammatory colitis is that of segmental well demarcated involvement with rectal sparing, and a more rapid resolution of transient ischaemic symptoms in usually less than seven days. Furthermore, cases of chronic gut ischaemia may be misdiagnosed as inflammatory bowel disease, thus subjecting patients to the undesirable effects of long-term steroid and immunosuppressive therapies. Occurrence of colonic ischaemia in patients with irritable bowel syndrome is estimated as being 3.4 times more than those without.

4. Conclusion

Intestinal ischaemia is a serious acute abdominal pathology that is associated with hyperamylasaemia, and requires prompt diagnosis and management to prevent complications and mortality. Early diagnosis and active monitoring of patients is crucial to formulate an appropriate management plan, yet there is a need for surgical intervention in a significant proportion of patients. Ultimately, long-term prevention is advisable by optimisation of modifiable cardiovascular risk factors in those who are at high risk of ischaemia.

Conflict of interest
All authors confirm that there is no conflict of any kind in relation to this manuscript. There are no financial ties to any companies.

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Ethical approval
Informed written consent for anonymous case report publication and use of images was obtained from the patient. No identifying details are included.

Author contributions
Francesca Kum led the write-up of the case presentation, obtained patient consent and contributed to literature review. Arun Gulati contributed to review of literature, writing and reviewing of the case report. Abdulzahra Hussain selected the case for submission, was involved in the critique of published literature and review of report writing.

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