Acute Ischaemic Colitis; A Case Report

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ABSTRACT

Acute ischaemic colitis (AIC) is being increasingly recognised as an uncommon cause of abdominal pain associated with fresh bleeding per rectum. It is paramount to maintain a high index of suspicion and adopt appropriate management strategies to avoid complications and inappropriate interventions. In this paper, we describe a case of AIC and review literature pertinent to the management of this condition.

Keywords: *Ischaemic colitis, acute abdomen, management*

INTRODUCTION

Acute ischaemic colitis (AIC) is being increasingly recognised as an important cause of acute abdominal pain associated with passage of fresh blood per rectum. Local hypoperfusion and reperfusion injury are both thought to contribute to the disease process, which manifests with a wide spectrum of injury including reversible subepithelial haemorrhage and oedema, transient colitis, chronic colitis, stricture, gangrene leading perforation, peritonitis and septic complciations.[1] This condition is associated with significant morbidity and mortality, both in short and long-terms that mandates prompt diagnosis and treatment.[2] AIC can remain unrecognised, if a high index of suspicion is not maintained and appropriate investigations not undertaken.[3] Here, we describe the management of a patient with AIC involving the splenic flexure of colon and provide an up to date review of literature pertinent to the condition.

CASE REPORT

A 60-year-old female was admitted via accident and emergency department with sudden onset of a colicky lower abdominal pain one hour following meal, which was associated with nausea, vomiting and passage of large amount fresh blood per rectum. There was no recent change in her bowel habit. Her body weight was unchanged despite some decrease of appetite. She had suffered from two episodes of myocardial infarction previously, which were managed with coronary angioplasties. However, she was not on any antiplatelet or anticoagulation therapy. She denied any anginal symptoms. Her exercise tolerance was restricted to 200 yards due dyspnoea secondary to chronic obstructive pulmonary disease.

On admission, she had a blood pressure of 130/75 mm Hg, heart rate 72/min, respiratory rate 16/min, oxygen saturations of 98% on air, and a temperature of 37.1°C. Abdominal examination revealed mild central abdominal

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tenderness with no signs of peritonism. The fingerstall was blood-stained on digital rectal examination. Proctoscopy and rigid sigmoidoscopy showed presence of fresh blood with no obvious source of bleeding.

Routine investigations showed normal haemoglobin (14.3 gm/dL), neutrophilic leucocytosis ($12.1x10^9$/L), a normal platelet count, serum amylase, coagulation profile, renal, liver function tests, arterial blood gas, blood lactate and electrocardiogram. A stool culture showed no growth of pathogenic organisms. Chest x-ray showed hyperinflated lungs consistent with chronic obstructive pulmonary disease. Plain abdominal x-ray showed gas in the splenic flexure of colon (Figure 1).

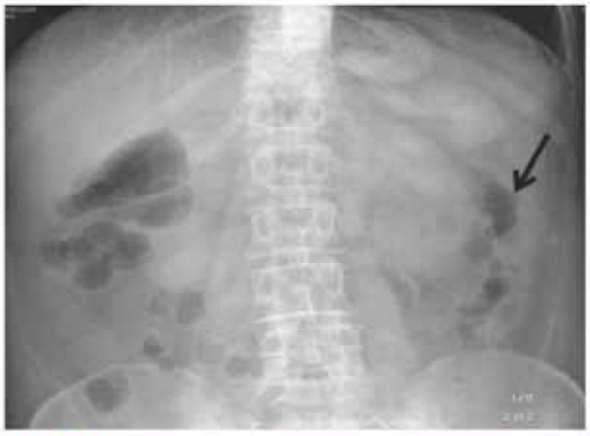

Figure 1. Plain abdominal x-ray showing gas in the splenic flexure of colon, which was site of AIC (arrow)

Computerised tomographic (CT) scan of abdomen showed an area of mural thickening and inflammatory stranding in the region of the splenic flexure with surrounding peri-colic inflammation. Calcified areas were seen within the aorta and coronary arteries. A moderate amount of atheromatous change was seen in the abdominal aorta and at the origin of the coeliac and superior mesenteric arteries (Figure 2).

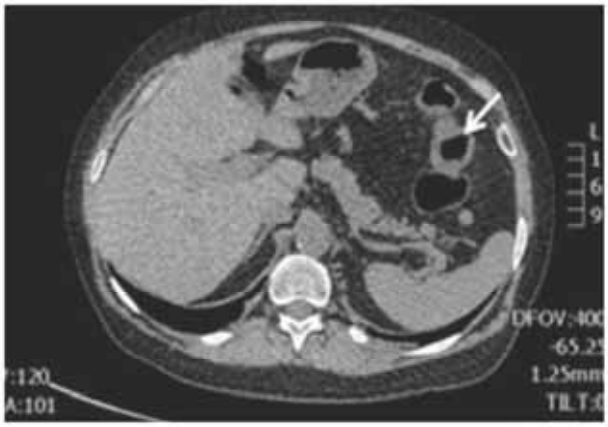

Figure 2. Computerised tomographic scan of abdomen showing features of ischaemic clitis involving the splenic flexure (arrow)

Based on the clinical and CT appearances, diagnosis of an AIC was made. Blood was grouped and saved. Vital signs were monitored 2 hourly and a meticulous record of the volume and episodes of rectal bleeding were maintained. She was kept nil-by-mouth initially and intravenous fluid replacement was continued. The rectal bleeding and abdominal pain gradually resolved in response to conservative management and she was discharged home 1 week later. A colonoscopy was performed on an outpatient basis after 4 weeks, which showed normal-looking colonic mucosa with no obvious source of bleeding.

DISCUSSION

The term "ischaemic colitis" was coined by Adrian Marston in 1966, a condition in which inflammation and injury of the large intestine results from inadequate blood supply.[4] Although uncommon in general population, AIC is being increasingly recognised in the elderly population as the most common cause of intestinal ischaemia, associated with abdominal pain and seen on approximately 1 in 100 endoscopies.[5] Men and women are affected equally; however AIC is a disease of the elderly, with more than 90% of cases occurring in people over the age of 60.[6] In a study involving 313 cases of biopsy proven or compatible AIC the patterns and frequencies of involvement were: right colon, 25.2%; transverse colon, 10.2%; left colon, 32.6%; distal colon, 24.6%; and pancolon, 7.3%.[7]

Acute ischaemia may be preceded by features of chronic intestinal ischaemia such as post-prandial abdominal pain and unexplained weight loss. The explanation for the postprandial onset of symptoms and the involvement of the splenic flexure in our case has anatomical and pathological bases. The intestinal circulation receives 10-20% of the resting and approximately 35% of the post-prandial cardiac output. This post-prandial increase in circulation occurs principally due to the increased metabolic requirements of the intestinal mucosa and begins 30-60 minutes after the onset of food intake and persists for 2-6 hours after the meal, explaining the temporal relationship that exists between eating and the onset of abdominal pain chronic mesenteric ischaemia. If blood flow to the colon drops by more than about 50%, ischemia develops. The arteries feeding the colon are very sensitive to vasoconstrictors; presumably this is an evolutionary adaptation to shunt blood away from the bowel and to the heart and brain in times of stress. As a result, during periods of low blood pressure, the arteries feeding the colon clamp down vigorously resulting in non-occlusive ischemic colitis.[8]

Following acute ischaemia of the colon, the initial hyperactive phase, in the majority of patients, is

characterised by passage of fresh blood and abdominal pain, which resolves in the majority of them with conservative treatment. If the ischaemia continues, a paralytic phase follows. The abdominal pain becomes more widespread and the abdominal tenderness and distension increases. Finally, a shock phase can develop as fluids start to leak through the damaged colon lining. This can result in shock and metabolic acidosis with dehydration, hypotension, tachycardia, and confusion. Patients who progress to this phase are often critically ill and require intensive care.[9]

Neutrophilic leucocytosis is present in about 25% of patients and metabolic acidosis in 33% of patients. Plain X-rays are often normal or show non-specific findings. In a series of 73 patients, plain abdominal radiography showed colonic distension in 53% or a pneumoperitoneum in 3% of cases.[10] CT scans are often used in the evaluation of abdominal pain and rectal bleeding, and may suggest the diagnosis of ischemic colitis, pick up complications, or suggest an alternate diagnosis. Mucosal oedema and pericolic stranding with perfused bowel wall in a contrast-enhanced CT scan are the typical findings of ischaemic colitis. A full-thickness infarction is diagnosed if the segment of colon shows no evidence of enhancement on the CT scan, which mandates surgical resection of the ischaemic colon.[11]

Endoscopic evaluation, via colonoscopy or flexible sigmoidoscopy, is the gold standard of diagnosis. Ischemic colitis has a distinctive endoscopic appearance; endoscopy can also facilitate alternate diagnoses such as infection or inflammatory bowel disease. In our case colonoscopy was performed 4 weeks after the acute episode of ischaemia, when the mucosal regeneration has already taken place, thereby showing a normal appearance. In patients who continue to bleed, either a mesenteric angiogram or a CT angiogram shows the site of bleeding as evidenced by luminal extravasation of contrast at the site of bleeding.[12]

Prevention of AIC is similar to the prophylactic measures taken to minimise the progression of atherosclerosis, such as control of diabetes, hypertension, hyperlipidaemia, cessation of smoking, regular exercises, weight reduction, and healthy dietary habits. In critically ill patients, prevention of hypotension and correction of hypercoagulable states is paramount to prevent AIC.

Patients with mild to moderate ischaemic colitis are successfully treated with intravenous fluids, analgesics and rest to the bowel, that is, nil orally, until symptoms resolve. The use of antibiotics in severe AIC is associated with improved survival and reduces morbidity. However, a prolonged course carries the risk of Clostridium difficile colitis, which can prove fatal. Patients should be monitored closely for worsening of symptoms and physical signs such as leucocytosis, pyrexia, increased bleeding and peritonitis, which are indications for surgical interventions.[13] Those with severe ischaemia with full-thickness infarction of the bowel wall develop complications such as sepsis, severe bleeding, intestinal gangrene with perforation and peritonitis requires more aggressive interventions such as surgery and intensive care.[14]

Surgery usually entails laparotomy and resection of the ischaemic segment of colon with an end colostomy and a mucous fistula formation. Primary anastomosis should be avoided as the risk of anastomotic dehiscence remains high. A low threshold should be maintained for second-look laparotomy. Most patients make a full recovery. Occasionally after severe ischaemia, patients develop a stricture leading to intestinal obstruction, which may require resection or endoscopic dilatation and deployment of self-expanding stents. 20% of severe AIC leads to chronic ischaemic colitis, which presents with recurrent infections, bloody diarrhoea, weight loss and chronic abdominal pain, This often requires resection of the chronically diseased portion of the colon.[15]

In conclusion, AIC is being increasingly recognised as an important cause of abdominal pain and bleeding per rectum in elderly population. A contrast-enhanced CT scan of the abdomen and colonoscopy are helpful in establishing the diagnosis. Conservative treatment is effective in majority of patients. However, for severe cases of AIC, management in critical care units and a low threshold for early surgical intervention is mandatory.

REFERENCES

1. Feuerstadt P, Brandt LJ. Colon ischemia: recent insights and advances. Curr Gastroenterol Rep. 2010; 12(5):383-90.
2. Acosta S, Ogren M, Sternby NH, et al. Fatal colonic ischemia: A population-based study. Scand J Gastroenterol. 2006; 41,11:1312-9.
3. Barnert J, Messmann H. Diagnosis and management of lower gastrointestinal bleeding. Nat Rev Gastroenterol Hepatol. 2009; 6,11:637-46.
4. Marston A, Pheils MT, Thomas ML, Morson BC. Ischaemic colitis. Gut. 1966; 7,1:1-15.
5. Green BT, Tendler DA. Ischemic colitis: a clinical review. South Med J. 2005; 98,2:217-22.
6. Leardi S, DeVita F, Felici S, et al. Acute ischaemic colitis: outcome in elderly patients. Chir Ital. 2006; 58,3:309-13.
7. Brandt LJ, Feuerstadt P, Blaszka MC. Anatomic patterns, patient characteristics, and clinical outcomes in ischemic colitis: a study of 313 cases supported by histology. Am J Gastroenterol. 2010; 105,10:2245-52; quiz 2253.
8. Kolkman JJ, Mensink PB. Non-occlusive mesenteric ischaemia: a common disorder in gastroenterology and intensive care. Best Pract Res Clin Gastroenterol. 2003; 17,3:457-73.
9. Boley SJ, Brandt LJ, Sammartano RJ. History of mesenteric ischemia. The evolution of a diagnosis and management. Surg Clin North Am. 1997; 77,2:275-88.
10. Abreu MT, Harpaz N. Diagnosis of colitis: making the initial diagnosis. Clin Gastroenterol Hepatol. 2007; 5,3:295-301.
11. McSweeney SE, O'Donoghue PM, Jhaveri K. Current and emerging techniques in gastrointestinal imaging. J Postgrad Med. 2010; 56,2:109-16.
12. Montoro MAH, Santolaria SP. Diagnostic approach to ischemic colitis. Gastroenterol Hepatol. 2006; 29,10:636-46.
13. Renner P, Kienle K, Dahlke MH, et al. Intestinal ischemia: current treatment concepts. Langenbecks Arch Surg. 2011; 396,1:3-11.
14. Elder K, Lashner BA, Al-Solaiman F. Clinical approach to colonic ischemia. Cleve Clin J Med. 2009; 76,7:401-9.
15. Beck DE, deAguilar-Nascimento JE. Surgical management and outcome in acute ischemic colitis. Ochsner J. 2011; 11,3:282-5.