Case Report

Gangrenous Colitis in a Patient with Polypharmacy: A Case Study

Hassan Malik*, Sivananthan Suntharalingam, Muhammad Majid Ali

Hervey Bay Hospital, Queensland, Australia

Email address:

hazymalik@yahoo.com (H. Malik)

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Abstract: A 20-year-old female, otherwise fit and well, was admitted to intensive care unit intubated with polypharmacy secondary to drug over dose. She developed a seizure episode and a rise in serum lactate within 24 hours of admission, followed by abdominal distension, with metabolic acidosis and a further elevation of serum lactate levels. CT abdomen revealed gangrenous colitis, which was confirmed on laparotomy. The patient underwent subtotal colectomy with end ileostomy, which was further complicated a week later by necrosis of the stoma due to knotting of the small bowel around the ileostomy loop. She underwent another laparotomy, for fixation of internal herniation of small bowel loops as well re-fashioning of the end ileostomy, and had an uneventful recovery. Final pathology of the colon showed ischaemic colitis with mural necrosis, peritonitis and viable resection margins.

Keywords: Young Female, Gangrenous Colitis, Ischaemic Colitis(IC), Polypharmacy, CT, Laparotomy, Ileostomy

1. Introduction

Ischaemic colitis (IC), first described by Boley et al, is the most common form of ischaemic injury to the gastrointestinal tract representing more than half of the cases with gastrointestinal ischaemia [3, 4]

Although the colon has a generous overlapping blood supply, any interruption in blood flow produces ischaemia. Ischaemic colitis due to miscellaneous drugs has been reported in the literature with a spectrum of mild to severe disease. Most of the patients with ischaemic colitis are middle aged to elderly with peripheral vascular disease, diabetes etc, but it has been reported in young individuals with no medical co-morbidities. [1] Clinically, it manifests from transient self-limited ischemia involving the mucosa and submucosa, carrying a good prognosis, to acute fulminant ischemia with transmural infarction, which may progress to necrosis and death. The majority of cases with ischaemic colitis can be managed with non-operative treatment, which involves hydration with intravenous fluids, antibiotics, clear liquid diet, maintaining an adequate urine output and observation. A few cases, can progress to fulminant necrosis and gangrene, warranting operative intervention with bowel resection. In younger patients, who are mostly women, there has been an

association between ischaemic colitis and the use of other drugs. [2] We report a case of gangrenous colitis in a young 20-year-old female with polypharmacy secondary to drug overdose. The aim of this publication is to maintain a high degree of suspicion, even in younger individuals, who can potentially develop ischaemia of the colon.

2. Case Report

A 20-year-old female with a history of depression, and previous history of polypharmacy overdose, was admitted to the ICU with another polypharmacy drug overdose. As per the family, the patient had overdosed on her mother's medications at 2200. She was brought to the Emergency department at 2300 with a blood pressure of 130/80, pulse 120/minute (regular), oxygen saturation 95% on 2 Litres of oxygen, respiratory rate 8, blood sugar 6.9mmol/L, serum lactate of 2.4mmol/L with a GCS 14/15, which dropped to 3/15 in next 15 minutes. She was intubated, ventilated, and transferred to ICU with no vasopressor support at 0400. Charcoal was administered via nasogastric tube on admission for decontamination of the gut and to prevent drug absorption.

The history from the parents and the ambulance staff

revealed that the patient had 200 tablets comprising of Venlafaxine 150mg x 30 tablets, Paracetamol with codeine 500mg /30mgx 30 tablets, Quetiapine 25mg x 60 tablets, Escitalopram 20mg x 30 tablets, Amiloride with hydrochlorothiazide 5mg/50mg x 30 tablets, Tramadol 100mg x 30 tablets.

The patient suffered a brief seizure episode at 0615, lasting for 2 minutes, with a blood pressure of 35/15mm Hg, pulse 140/minute and was administered midazolam, as well commenced nor-adrenaline 10mcgm/Hr. Post seizure activity, the patient was in acidosis, with a lactate of 11.2mmol/L, and a 7.03 pH. Subsequently, the lactate was cleared to 6.1 at 0900, 3.9 at 1600, and 3.4 at 1800. At 2000, abdominal distension was found on the examination, and progressive acidosis with a lactate of 4.5mmol/L, followed by a repeat lactate of 5.2 at 2200, and 6.1 at 0015. Surgical consultation was sought by the ICU for acidosis and abdominal distension with tenderness. CT abdomen with contrast at 0150 revealed frank colonic infarction with air in the portal system of the liver [Figure 1]

Figure 1. Abdominal CT- frank colonic infarction with air in the portal venous system of the liver.

The patient underwent midline laparotomy, which showed gangrenous colon from the caecum to proximal sigmoid colon, with a viable small bowel and distal sigmoid colon [Figure 2] Subtotal colectomy and end ileostomy was performed by closing the distal stump with a linear stapler. The patient was transferred to ICU post op. Successful serum lactate clearance was achieved within 12 hours.

Figure 2. Gangrenous colon from the caecum to proximal sigmoid colon, with a viable small bowel and distal sigmoid colon.

Figure 3. Resected colon specimens.

The subtotal colectomy removed 730mm of colon which had a maximal diameter of 68mm [Figure 3]. When opened in the Pathology Department, the colonic lumen contained a large volume of black material compatible with charcoal and occasional white bead-like structures. The charcoal coated the mucosal surface. The colonic wall was thinnest (1mm) in the caecum, but perforation was not identified. Microscopic examination showed ischaemic colitis. This was more severe in the caecum where there was transmural necrosis [Figure 4]. It became less severe distally where the mucosa was necrotic but the underlying wall was a viable [Figure 5]. The surgical resection margins were viable. The blood vessels within the specimen did not show evidence of vasculitis, thromboembolism or atherosclerosis.

Figure 4. Proximal colon showing transmural necrosis. The mucosa (upper) is unrecognisable and most cellular detail is lost (H&E, x100).

Figure 5. Distal colon showing viable muscularis propria (lower) although the mucosa (upper) still shows necrosis. The black material on the surface is charcoal (H&E x40).

She had uneventful recovery, until post operative day 8 when the stoma showed full thickness necrosis. CT-angiogram showed no evidence of major vascular occlusion. The patient underwent an ileoscopy, which showed only distal 3cm of mucosal necrosis, requiring a re-laparotomy, which revealed knotting of the small bowel loops around the stoma bowel loop. The stoma was re-fashioned to healthy tissue in the Right lower quadrant.

The patient was discharged in a stable condition, 15 days post drug overdose, with psychiatry follow up. Laboratory screening for thrombophilia, vasculitis and lupus was negative.

3. Discussion

Boley *et al* [3] described ischemic colitis in the 1960s, but Marston *et al* [5] coined the term ischaemic colitis. Although ischaemic colitis may develop spontaneously, a broad spectrum of multifactorial conditions is often present. Typically, the patient is elderly and has a consistent past history compatible with arteriosclerosis, low flow states of any nature, and congestive heart failure

However, ischaemic colitis has also been reported in younger patients without premorbid conditions, but has been noted in patients with vasculitis, cocaine abuse, marathon running, coagulopathies, and drugs such as digitalis, nasal decongestants, oral contraceptives, and some NSAIDs, and rarely as a result of barium enema and colonoscopy [6, 7]

The differential diagnosis of ischaemic colitis includes arterial occlusion, caused by emboli or thrombus, venous thrombosis, and non-occlusive ischaemia caused by systemic hypotension or, as is strongly suspected in this case, drug-induced vasospasm. A diagnosis of drug induced vasospasm is a diagnosis of exclusion, made when all other etiologies of ischaemia have been ruled out.

In our case report, an arterial occlusion is unlikely, because there was no palpable thrombus and there was absence of ischaemic demarcation to suggest a major vessel distribution. There was no history of cardiac arrhythmias, such as atrial fibrillation, to predispose her to emboli and she was a non-smoker and had no other cardiac risk factors such as

atherosclerosis, neither pathology specimen nor hyperlipidemia. There were no clinical features to suggest the presence of a systemic vasculitis.

Although venous thrombosis could account for ischaemia, our patient had no signs of venous congestion or stasis at the time of surgery. The patient had no hypercoagulable disorder and on examination the colon was not oedematous and no clot was identified in the vasculature.

Global ischaemia secondary to systemic hypotension can result in generalized colonic hypoperfusion and vaso-constriction which would have resulted in a watershed-type distribution of gangrene. In contrast, the distribution was fairly widespread, from the caecum to proximal sigmoid colon.

The diagnosis of ischaemic colitis in a young person should always provoke consideration of drug involvement.[8] Gut infarction due to the use of oral contraceptive use has been widely reported in the literature, but our patient did not report an use of oral contraceptive drugs. [9]

Diuretics have been reported to cause hypotension and contribute to non-occlusive mesenteric ischaemia due to volume depletion. [10] Colonic perfusion is also affected by the functional motor activity of the colon and by patient straining from constipation. [11] Of note, chronic constipation has been found to be strongly associated with ischaemic colitis. [12] The use of charcoal can also lead to transient colonic obstruction, increased in intra-luminal pressure, bacterial translocation followed by ischaemia. Our patient, continued to pass flatus from the day of admission till the night of colonic infarction.

There has been a suggestion from the Queensland Poisons Information Centre that SSRI's can lead to colonic paralysis, moderate to significant distension followed by arteriolar ischaemia leading to gangrenous colitis.

4. Conclusion

Ischaemic colitis, although rare in the younger population, should be considered in the differential diagnosis of intensive care patients with progressive acidosis, refractory to treatment. A high index of suspicion should be kept in patients with polypharmacy, as a multitude of drugs are associated with colonic hypoperfusion leading to ischaemia and necrosis. Serum lactate is a marker of hypoperfusion, but can be used as an adjunct to diagnose gut ischaemia in patients with abdominal signs of peritonism. Although most patients improve within several days with supportive care, others will require laparotomy with bowel resection.

CT with contrast is gold standard to define and exclude colonic ischaemia, although the CT findings could be a late sequel of ischaemic colitis where frank infarction has ensued.

Author's Contributions

HM initiated the case series analysed and interpreted the patient data as well writing the manuscript. MMA performed the literature search, and analysed patient data. SS was the

consultant surgeon in charge and a major contributor in drafting the manuscript. All authors read and approved the final manuscript.

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Consent

A formal consent was obtained from the patient and family for this publication.

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