Asian Journal of Case Reports in Surgery

11(3): 27-32, 2021; Article no.AJCRS.73364

Acute Massive Lower Gastrointestinal Bleeding Secondary to Obstructive Colitis Proximal to Obstructing Cancer of the Sigmoid Colon

Sze Li Siow[1,2*], Myo Nyunt[2] and Hans Alexander Mahendran[3]

[1]Department of General Surgery, Sarawak General Hospital, Jalan Hospital, 93586, Kuching, Sarawak, Malaysia.
[2]Department of Surgery, Faculty of Medicine and Health Sciences, University Malaysia Sarawak, Kota Samarahan, Sarawak, Malaysia.
[3]Department of General Surgery, Sultanah Aminah Hospital, Johor Bahru, Johor, Malaysia.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Editor(s):
(1) Dr. Ashish Anand, G.V. (Sonny) Montgomery VA Medical Center, USA.
Reviewers:
(1) Mokrane Ahlem, Saad Dahlab University of Blida, Algeria.
(2) Gerald Tumusiime, Uganda Christian University, Uganda.
Complete Peer review History: https://www.sdiarticle4.com/review-history/73364

Case study

Received 28 June 2021
Accepted 08 September 2021
Published 11 September 2021

ABSTRACT

Introduction: Acute massive lower gastrointestinal bleeding (LGIB) is a rare and serious manifestation of obstructive colitis that requires urgent therapeutic intervention. Here, we report a case of LGIB due to obstructive colitis in an adult patient.
Presentation of Case: A 34-year-old man with large bowel obstruction secondary to sigmoid colon cancer underwent laparotomy and Hartmanns procedure (resection of rectosigmoid colon with a proximal end colostomy). Post-operatively, he had recurrent episodes of severe bleeding from the colostomy that required transfusion of a total of eleven units of packed cells and four units of fresh frozen plasma over the next two days. Urgent oesophagogastroduodenoscopy showed pan gastritis and insignificant superficial gastric erosions. Colonoscopy via the colostomy showed stigmata of recent bleed but failed to identify the exact site of bleeding. Computed tomography angiogram failed to localize the site of bleeding. A re-laparotomy was performed. On-table colonoscopy through the end colostomy followed by completion total colectomy and ileorectal anastomosis was done. The patient recovered uneventfully after the surgery with no further episode of rectal bleeding. Histology

**Corresponding author: E-mail: szeli18@yahoo.com;*

findings of the resected colon were compatible with obstructive colitis. He remains well at five years follow-up with no recurrence.
Discussion: The case highlights the rare occurrence of acute massive LGIB as a life-threatening complication of obstructive colitis. The diagnosis should be considered in patients who present with large bowel obstruction.
Conclusion: A high index of suspicion is key to early diagnosis and an extended resection of the colon is necessary to arrest bleeding.

Keywords: Obstructive colitis; sigmoid cancer; intestinal obstruction; lower gastrointestinal bleeding; obstructing colon cancer.

1. INTRODUCTION

Acute massive lower gastrointestinal bleeding (LGIB) is a rare manifestation of obstructive colitis (OC) which is serious and requires urgent therapeutic intervention. Surgical resection is often necessary to arrest bleeding and establish pathological diagnosis [1]. Obstructive colitis refers to ulceroinflammatory lesions occurring in the colon proximal to a completely or partially obstructing lesion such as colorectal cancer or diverticula disease [2,3]. It has been referred in various terms such as ulcerative [4] or pseudo-ulcerative colitis [5], acute necrotizing colitis [1,6], ischaemic colitis [7], acute gangrenous colitis [8] or nonspecific colitis [9]. It has been reported to be associated with 0.3 to 7% of all colorectal cancer [2]. It can occur at any site in the colon, but the majority involves the left side of the colon, as the most common obstructions occur in the rectosigmoid junction [3,10]. Obstruction can either be benign due to diverticulitis or malignant due to carcinoma as the most common causes [3]. The clinical presentation is non-specific with some symptoms attributable to obstructed colon [1].Here, we report a 34-year-old man who developed acute massive LGIB after an emergency Hartmanns procedure for an obstructing sigmoid colon cancer. A completion total colectomy and ileorectal anastomosis was performed to arrest the bleeding.

2. PRESENTATION OF CASE

A 34-year-old man was admitted to medical ward with a three-week history of intermittent abdominal pain and fever, associated with loss of appetite and weight. Ten days prior to admission, he had constipation and alternating diarrhoea. Otherwise, he did not have any history of malaena or per rectal bleeding. He had no significant past medical or surgical history, and there was no family history of malignancy.

Physical examination revealed a normotensive gentleman with a pulse rate of 84 beats per minute and a temperature of 37^0 C. The abdomen was slightly distended, soft and non-tender. Digital rectal examination showed brownish stool with no palpable mass. The rest of the examinations were normal. Laboratory investigations showed a normocytic normochromic anemia with hemoglobin of 12.9 g/dl; a normal white cell count of 6.4 x 10^9 /L and normal platelet count of 251 x 10^9 /L. Serum urea, electrolytes, creatinine and liver function tests were all normal. C-reactive protein was negative. His viral serology tests were negative for Human Immunodeficiency Virus (HIV), Hepatitis B and C virus. He was suspected of having typhoid colitis with Widal test showing a positive reaction for the *S. typhi* O antigen (1:160) and H antigen (1:40). The patient was treated on intravenous fluids and intravenous Ciprofloxacin 400mg twice daily whilst his other results were pending. Two consecutive blood cultures returned negative.

The patient was unwell for the next few days with increasing abdominal distension and high nasogastric output. A surgical consult was made as he was suspected of having bowel perforation secondary to typhoid colitis. An urgent computed tomography (CT) scan of the abdomen revealed a circumferential tumour in the sigmoid colon causing large bowel obstruction (Fig. 1). He was then taken to theater for emergency laparotomy with the diagnosis of acute large bowel obstruction secondary to sigmoid colon cancer.

During laparotomy, it was noted that there was an obstructing tumour of the sigmoid colon causing proximal dilatation of the colon and small bowels up to the mid-jejunum. Apart from dilatation, the proximal colon and small bowels looked normal and well - perfused externally. The colon distal to the obstructing tumour was collapsed. There were enlarged mesenteric lymph nodes seen adjacent to the tumour and there was minimal clear ascitic fluid. The liver and the rest of the peritoneal cavity were otherwise normal. The dilated small bowel was

decompressed via enterostomy and a Hartmanns procedure (resection of rectosigmoid colon with a proximal end colostomy) was performed. Histopathological examination confirmed a moderately differentiated adenocarcinoma of the sigmoid colon with a staging of T3N1M0 (stage III) according to the 7th edition of the American Joint Committee on Cancer (AJCC) staging system, 2010. The tumour had a proximal margin of 50 mm which the colonic mucosal reported as unremarkable.

Post-operatively, he had recurrent episodes of severe bleeding from the colostomy that required transfusion of a total of eleven units of packed cells, and four units of fresh frozen plasma over the next two days. An urgent oesophagogastroduodenoscopy showed pan gastritis and superficial gastric erosions which excluded bleeding from an upper gastrointestinal source. Colonoscopy via the colostomy showed stigmata of recent bleed but failed to identify the exact site of bleeding. CT angiogram was performed twice, but could not demonstrate the source of bleeding. Due to the recurrent bleeding episodes with high transfusion requirements, he was taken back to theater for emergency re-laparotomy. On-table colonoscopy through the end colostomy revealed the whole length of colon was filled with blood clots. After irrigation with warm water, parts of the transverse colon were noted to have multiple diffuse haemorrhagic mucosal areas. The terminal ileum was intubated and was normal with no evidence of proximal bleeding. The whole colon and a short segment of terminal ileum were resected and a primary ileorectal anastomosis was performed to restore continuity. The patient recovered uneventfully after the surgery with no further episode of rectal bleeding and was discharged on the ninth day after the second surgery. He subsequently completed adjuvant chemotherapy and was followed up for 53 months with no recurrence.

Histology examination of the resected specimen revealed acute colitis of a length of 220 mm, starting from the stoma end. The mucosal was described as having focal areas of glandular epithelium with alternating areas of ulceration and pseudopolyps comprising of hyperplastic epithelial mucosa. There was marked inflammatory changes with infiltration of mixed inflammatory cells transmurally. It was associated with congestion and scattered areas of haemorrhage (Fig.2). There was distortion and destruction of the muscularis propria layer. However, there were no tumour deposits seen. There was also evidence of peri-appendicitis with the serosa of the appendix infiltrated with mixed purulent and granulomatous inflammatory cells.

3. DISCUSSION

We described a case where the patient developed massive LGIB following a Hartmanns procedure for obstructing sigmoid colon cancer. To the best of our knowledge, this is the first report of massive LGIB after Hartmann caused by obstructive colitis. The case highlights several important learning points. First, massive LGIB can be a presentation of OC. Second, there was the diagnostic difficulty in identifying OC during the first surgery as the colon appeared macroscopically normal. The diagnosis was only established by histology after surgical resection. Nevertheless, the surgeon should be alerted to the possibility of this uncommon clinical entity in patients who present with large bowel obstruction.

The presentation of OC is highly variable. Patients can present as acute intestinal obstruction [11] or symptoms which are indistinguishable from colorectal cancer such as per rectal bleeding, abdominal pain, altered bowel habit, nausea and vomiting [2,10]. Our patient presented with abdominal pain, fever, loss of weight and appetite. He had history of altered bowel habit but the diagnosis of obstructing colon cancer was not immediately apparent most probably because of the patients age and the history of undocumented fever. The diagnosis was only established via CT scan that showed an obstructing sigmoid tumour.

The histopathologic diagnostic features of OC are the presence of normal mucosal distal to the obstruction and normal mucosal of varying length between the obstruction and the colitis [3,10,11]. These features are to distinguish it from ulcerative disease and other forms of colitis which has a continuity of mucosal involvement proximal to the obstructing tumour [3,12]. In addition, the rectum is always involved in ulcerative colitis [3]. There is considerable variation in the type, extent and depth of necrosis and ulceration in the involved colon, ranging from early mucosal to transmural necrosis & acute to chronic ulcers [3]. The colour of the serosa is unchanged in the majority of cases with transmural necrosis and fibrinous exudate only noted in some [3]. In our case, the histology from the first surgery showed a normal proximal margin of 5 cm from the tumour. However, the

histology from the second surgery demonstrated an ulcerative segment of the involved colon with a length of 22 cm, starting from the stoma end. The microscopic findings of mucosal ulceration and marked transmural inflammation, together with congestion and haemorrhage of the involved colon segment were compatible with OC.

The features of OC are suggestive of an ischaemic origin [2,10]. It is most probably due to hypoperfusion following raised intramural pressure, distension of colonic wall and other factors which impair adequate perfusion [3,12]. Altered faecal flora, as a result of colonic inflammation and ischaemia,may also have a direct or indirect role in its pathogenesis [12]. According to Laplace, tension in the wall of a sphere is the product of the pressure times the radius of the chamber [13]. The high intramural pressure and bowel dilatation proximal to the intestinal obstruction compromise the blood flow to the colonic wall, resulting in secondary ischaemia [2,10].

OC can present as a diagnostic challenge to the surgeons as the apparently normal external appearance of the involved segments of colon may be masking extensive mucosal damage [14]. As in our case, the colon appeared deceptively normal from the outside during the first surgery, thus, escaping the attention of the operating surgeon. The underlying pathophysiology appeared to be the shunting of blood from mucosa and muscularis to submucosal and subserosal layers during the increase in intraluminal pressure [14]. Consequently, the metabolically active mucosal is the first layer to suffer ischemic damage while the serosal blood flow remains intact [3,14].

Extended bowel resection is often necessary as the ulcero-inflammatory changes involve a variable length of colon, ranging from 8 to 25 cm with an intervening normal colon of 2.5 to 35 cm length [12]. Failure to perform extended resection in critically ill patients with OC resulted in poor outcomes, often contributing to the patients death [5]. The proximal bowel should always be examined after transection [2]. Delayed diagnosis could lead to complications such as peritonitis, perforation, breakdown of anastomoses and bleeding as in our case [2]. It has been recommended to open the resected bowel during surgery to examine the proximal colon after transection [2,4]. If ulcers are seen, a frozen section examination of the proximal margin of the resected colon can be performed to determine the viability of tissues [4]. Otherwise, extended resection of the colon should be done until the healthy mucosal margin is achieved [2]. In our case, completion total colectomy was performed as it was considered risky to limit the extent of the colitis to what was seen endoscopically. There were copious amounts of blood clots that limited accurate assessment of the mucosa. It was fortunate that an ileocolic anastomosis was possible without untoward anastomotic complications.

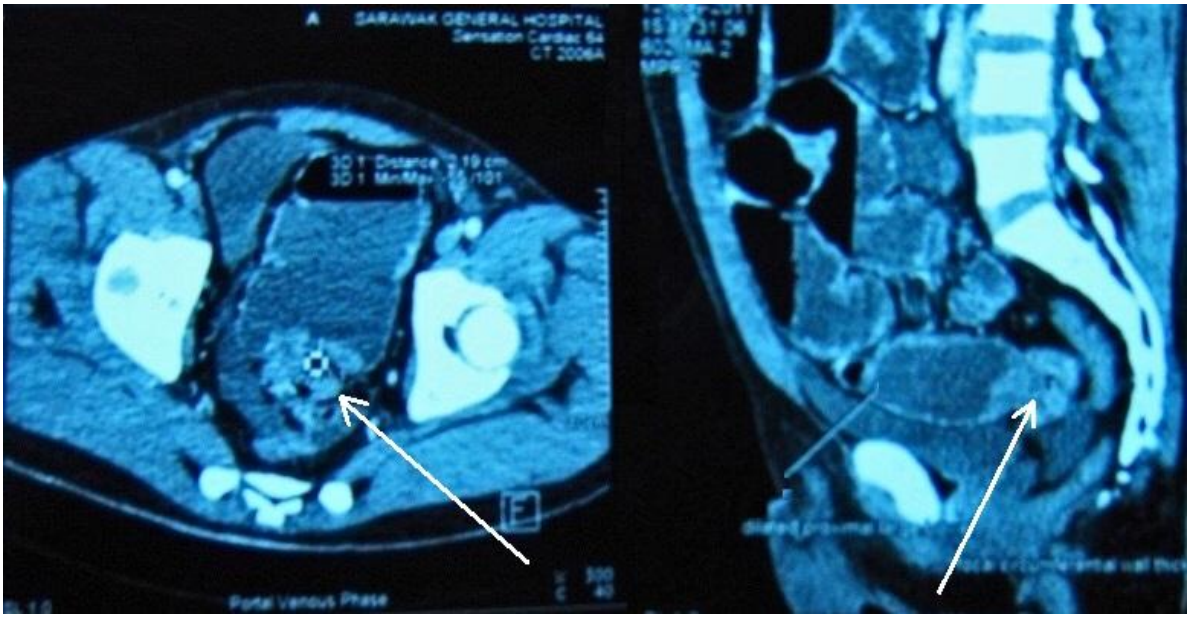

Fig. 1. Computed tomography scan of the abdomen & pelvis showing a circumferential sigmoid colon tumour (white arrow) causing large bowel obstruction

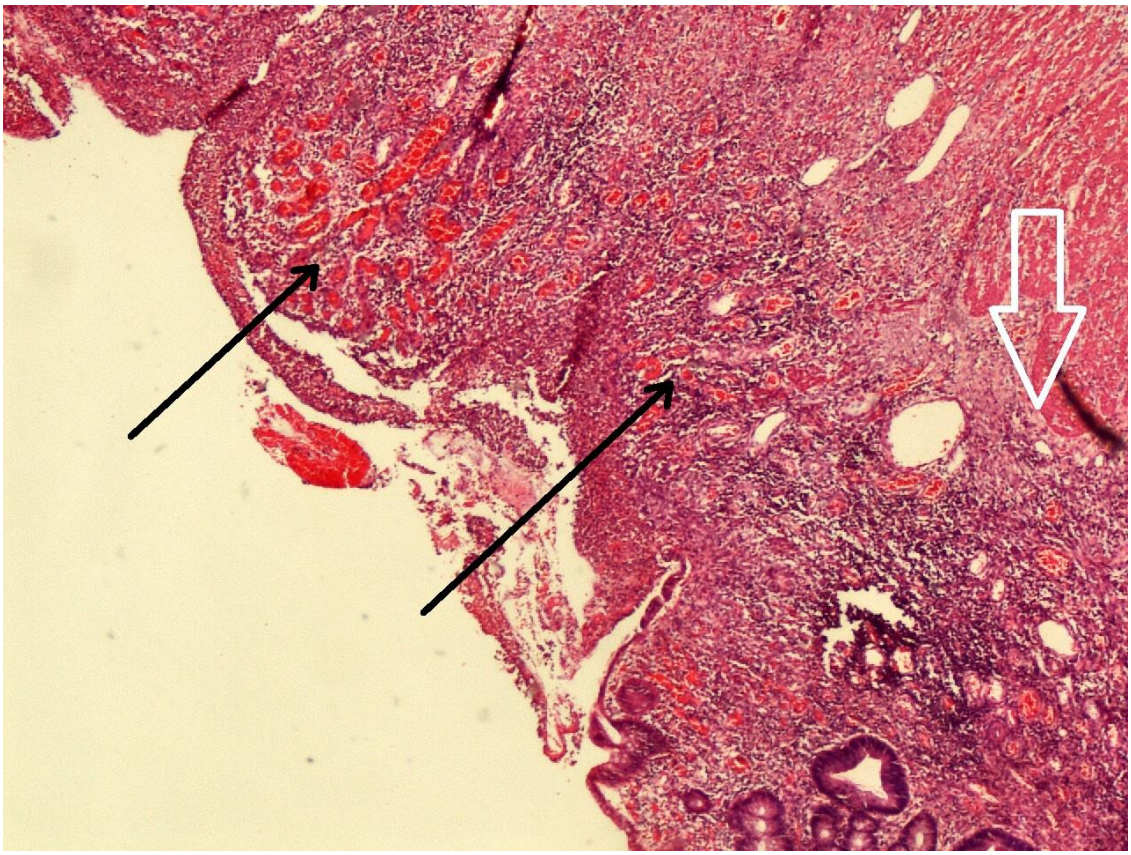

Fig. 2. Histopathological findings of the affected colon revealing mucosal ulceration with marked transmural inflammation (arrow head) and scattered area of haemorrhages (arrow)

4. CONCLUSION

This is a case of OC complicated with massive LGIB that was managed successfully with resection of the affected colon. Massive LGIB is a rare life-threatening complication of OC and may occur in patients presenting with obstruction of the large bowel. A high index of suspicion is key to early diagnosis and an extended resection of the colon is necessary to arrest bleeding.

CONSENT

Informed consent was obtained from the patient to be included in the study.

ETHICAL APPFROVAL

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinski Declaration of 1975, as revised in 2008(5).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Teasdale C, Mortensen NJ. Acute necrotizing colitis and obstruction. Br J Surg. 1983;70:44-47.
2. Chang HK, Min BS, Ko YT, et al. Obstructive Colitis Proximal to Obstructive Colorectal Carcinoma. Asian J Surg. 2009; 32:26–32.
3. Gratama S, Smedts F, Whitehead R. Obstructive colitis: an analysis of 50 cases and a review of the literature. Pathology. 1995;27:324-329.
4. Feldman PS. Ulcerative disease of the colon proximal to partially obstructive lesions: report of two cases and review of the literature. Dis Colon Rectum. 1975; 18:601-612.
5. Rutledge RH. Pseudo-ulcerative colitis proximal to obstructing colon carcinoma. Am Surg. 1969;35:384-388.
6. Matsunaga H, Shida D, Kamesaki M, Hamabe Y. Acute necrotizing colitis due to sigmoid colon cancer. World J Surg Oncol. 2014;12:19.

7. Ganchrow MI, Clark, Benjamin HG. Ischemic colitis proximal to obstructing carcinoma of colon. Dis Colon Rectum. 1970;14:38-42.
8. Harada T, Umezawa I, Mogami K, Itoh A. Acute gangrenous colitis proximal to obstructive cancer of the sigmoid colon. Jpn J Surg. 1975;5:39-47.
9. Tietjen GW, Markowitz AM. Colitis proximal to obstructing colonic carcinoma. Arch Surg. 1975;110:1133-1138.
10. Tsai MH, Yang YC, Leu FJ. Obstructive colitis proximal to partially obstructive colonic carcinoma: a case report and review of the literature**.** Int J Colorectal Dis. 2004;19:268-72.
11. Levine TS, Price AB. Obstructive enterocolitis: a clinico-pathological discussion. Histopathology. 1994;25:57-64.
12. Toner M, Condell D, OBriain DS. Obstructive colitis. Ulceroinflammatory lesios occurring proximal to colonic obstruction. Am J Surg Pathol. 1990; 14:719-728.
13. Stillwell GK. The Law of Laplace. Some clinical applications. Mayo Clin Proc. 1973; 48:863-869.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle4.com/review-history/73364