Brief Communication

Spontaneous Perforation of the Colon
Clinical Review of Five Episodes in Four Patients

Sabah Al Shukry

Abstract

Spontaneous perforation of the colon is rare and is classified as "Idiopathic" and "Stercoral." Four patients with a total of 5 episodes of spontaneous perforation of the large bowel treated at the department of surgery, Rustaq hospital over the past 5 years are presented in this study as well as a literature review of clinical and pathological characteristics of each case. Spontaneous perforation of the colon should be considered in the differential diagnosis of patients with acute peritonitis and free gas under the diaphragm. The condition is usually associated with chronic constipation and the use of NSAIDs. Early surgical intervention reduces morbidity and mortality.

From the Department of Surgery, Rustaq Hospital, South Batina, Oman

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Address correspondence and reprint requests to: Dr. Sabah Al-Shukry, Department of Surgery, Rustaq Hospital, South Batina, Oman.
E-mail: alshukry@yahoo.com

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Spontaneous perforation of the colon is a rare condition. In 1984, J.A. Berry classified spontaneous perforations into "stercoral" and "idiopathic" perforations on the basis of etio-pathological causes of lesions. Stercoral perforation is associated with ulcerative lesion, often in the sigmoid colon or rectum, or rarely in the cecum.[1,2] The Stercoral perforation is a "round" or an "ovoid" hole with necrotic and inflammatory edges. Idiopathic perforation is a linear "tear" with a normal appearance of the colonic wall.[3] The natural history of the 2 types have many similarities. Both entities are infrequently diagnosed preoperatively and are associated with a high mortality rate. A high index of suspicion is required for early diagnosis and treatment. But generally, stercoral perforations of the colon may be preventable.[4]

Four patients with 5 episodes of spontaneous perforations, were seen over 5 years, and are presented in this study.

Patient No. 1

ASM; A 35-year-old male presented with sudden abdominal pain progressively increasing in severity over 18 hours duration. The patient had been passing mucus with normal stools for 3 days. On examination; temperature was 37°C, pulse rate was 92/min, BP was 145/79 mmHg and his chest was clear while his abdomen was rigid with generalized tenderness and sluggish bowel sounds. A plain film of the abdomen showed free gas under both domes of the diaphragm. The laboratory findings were as follows: (Table 1)

Table 1: Patient no. 1 laboratory findings

Laboratory Test	Results
Hb	14.5 gm/dl
WBC	9.1 k/ul
Glucose	7.6 mmol/l
Blood Urea Nitrogen	5.6 mmol/l
Na	137 mmol/l
K	3.8 mmol/l
Cl	100 mmol/l

Hb: Hemoglobin; WBC: White Blood Cell; Na: Sodium; K: Potassium; Cl: Chloride

Laboratory findings.
-continued

Diagnosis of perforated gut was made. Treatment was started with IV fluids and broad spectrum antibiotics (Rocephin, Gentamycine & Metronidazol). Informed consent was obtained from the patient for urgent laparatomy. At laparotomy, there was a 3cm perforation on the anti-mesenteric border of the lower sigmoid, with copious amount of purulent fluid in the peritoneum and in the pelvis. Resection of an 8cm segment of sigmoid including the site of perforation with primary end-to-end anastamosis was performed. The patient had an uneventful recovery and was discharged on the 4th postoperative day. The pathological report revealed congested sub mucosa and serosa with marked edema in the central region around the perforation. There was no evidence of inflammation.

The same patient was presented 3.5 years later with a history of acute abdominal pain, constipation and vomiting for 2 days. On examination, the abdomen was distended with generalized rigidity, tenderness and rebound tenderness, absent bowel sounds but his temperature was normal. The patient's blood pressure (BP) was 140/80 mmHg and his pulse rate was 90/min. A plain X-ray of the abdomen showed free air under both domes of the diaphragm. A blood test revealed; Hb 12.4 gm/dl and White Blood Cells (WBC) 7.2 k/ul. Blood urea nitrogen and electrolytes were within normal range. The patient was treated with IV fluids initially, followed by

N/G tube insertion with suction and broad spectrum antibiotics (Rocephin, Gentamycine & Metronidazol). Informed consent for urgent laparotomy was obtained from the patient. Laparotomy showed seropurulent fluid with free gas in the peritoneum, dilated loops of small bowel and few fecal lumps in the sigmoid colon. A 3cm perforation with irregular margins was seen in the sigmoid colon near the area of previous anastamosis. There was no fecal matter in peritoneum. Resection of a 7 cm segment including the perforation was performed as well as Primary anastamosis. The patient recovered uneventfully and was discharged on the 8th postoperative day. Pathological report revealed brown discoloration of the bowel wall adjacent to the perforation with mucosal edema and inflammatory granulation tissue with necrotic tissue extending to the serosa, edema of mucosa and fibrinuos exudates on serosa in the rest of the bowel segment. The patient was asymptomatic on follow up.

Patient No. 2

KSM; A 70 year old patient with Post herpetic neuralgia and dermatitis was treated for 7 months with local steroids, oral Carbamazepin and Diclofenac sodium. He was admitted to the medical ward for treatment of infected dermatitis and post herpetic pain control. After 2 weeks in hospital, he developed acute abdominal pain, distention, rigid abdomen with tenderness, rebound tenderness and absent bowel sounds. On examination, the patient's BP was 100/50 mmHg, pulse rate was 100/min and temp was 38°C. Laboratory findings revealed the following: (Table 2)

Table 2: Patient no. 2 laboratory findings

Laboratory Test	Results
Hb	8 gm/dl
WBC	16 k/ul
Blood Urea Nitrogen	15 mmol/l
Cretinine	150 mmol/l
Na	130 mmol/l
K	5 mmol/l
Cl	90 mmol/l

Hb: Hemoglobin; WBC: White Blood Cell; Na: Sodium; K: Potassium; Cl: Chloride

Abdominal X-ray showed a large amount of free gas under the diaphragm. Informed consent for a high risk urgent laparotomy was signed by the next of kin. At laparotomy; a large amount of fecal matter was present in the peritoneum and there were multiple large perforations in the sigmoid colon. A meticulous peritoneal lavage was performed with resection of unhealthy 15cm segment of the sigmoid with end colostomy and closure of the distal end. The patient had a stormy postoperative course in the Intensive Care Unit complicated by wound infection, burst abdomen, septicemia and pneumonia. He died one week after the surgery. Pathological report revealed a segment of the large bowel with 4 defects on the anti mesenteric border. The rest of the mucosa showed irregular ulcerations with dark green exudates. Microscopy showed ulceration and perforations; probably ischemic, with scattered inflammatory cells in the sub mucosa, serositis with fecal matter on the surface.

Patient No. 3

SNA; A 15 year old Female with cerebral palsy & mental retardation. She was bed ridden with chronic constipation and was admitted with a three day history of vomiting, abdominal pain, and distention. She was passing little stools and on examination she was found to be jaundiced, dehydrated and had cold sweats. Her temperature was 38°C, BP was 97/54 mmHg and her pulse rate was 101/min. The patient's abdomen was markedly distended with guarding, tenderness and rebound tenderness. Bowel sounds were absent and the rectum was full of faeces. Laboratory findings were as follows: (Table 3)

Table 3: Patient no. 3 laboratory findings

Laboratory Test	Results
Hb	15.4 gm/dl
WBC	8.0 k/ul
Blood Urea Nitrogen	5.6 mmol/l
Creatinine	74 umol/l
Na	127 mmol/l
K	4 mmol/l
Cl	108 mmol/l

Hb: Hemoglobin; WBC: White Blood Cell; Na: Sodium; K: Potassium; Cl: Chloride

Diagnosis of advanced peritonitis was made. Informed consent for high risk laparotomy was signed by the patient's guardian. At laparotomy, fibrinous adhesions with multiple loculi of purulent fluid in the peritoneum were observed. There was fecal matter in the pelvis and there was a sealed perforation in the rectum with edematous indurations of the wall of the rectum and the sigmoid colon was full of hard faeces. Peritoneal lavage and loop colostomy were performed with evacuation of the rectum and sigmoid. Post operatively, the patient never recovered from septic shock. She was kept on a ventilator and inotropic drugs with antibiotics (Imipenim & Cephtazedime). The patient then developed a progressive

derangement of blood counts and biochemistry and died after 4 weeks. (Table 4)

Table 4: Patient no. 3 laboratory findings after laparotomy

Laboratory Test	Results
Hb	8.2 gm/dl
WBC	2.9 k/ul
Blood Urea Nitrogen	295 mmol/l
Creatinine	295 umol/l
Na	130 mmol/l
K	3.4 mmol/l
Cl	1.93 mmol/l

Hb: Hemoglobin; WBC: White Blood Cell; Na: Sodium; K: Potassium; Cl: Chloride

Pathology of the resected segment of the colon showed inflammatory granulation tissue in the mucosa above the perforation, mucosal ulcerations, congestion and haemorrhage, plus fungal hyphae were also present on the mucosal surface.

Patient No.4

SMS; A 61 year old male who was presented with acute generalized abdominal pain, vomiting, constipation and progressive abdominal distention for three days. On examination, the abdomen was markedly distended with guarding, tenderness, rebound tenderness, and sluggish bowel sounds. However, the rectum was empty. Laboratory findings were as follows:

Table 5: Patient no. 4 laboratory findings

Laboratory Test	Results
Hb	15.0 gm/dl
WBC	10.1 k/ul
Blood Urea Nitrogen	127.5 mmol/l
Creatinine	3.4 umol/l
Na	95 mmol/l
K	4 mmol/l
Cl	108 mmol/l

Hb: Hemoglobin; WBC: White Blood Cell; Na: Sodium; K: Potassium; Cl: Chloride

Informed consent for high-risk laparotomy was signed by the patient's son. The laparotomy showed fecal peritonitis with a 2 cm perforation at the apex of the sigmoid loop with adhesions of omentum and small bowel loops at the site of perforation. Resection and primary anastamosis were performed with delayed closure of the laparotomy wound 2 days later. The patient had slow recovery and was discharged after 3 weeks in good condition. Pathological findings from sections of the sigmoid colon showed ulceration & perforation with edematous submucosa, congested blood vessels, and numerous proliferating vascular channels. The serosa also showed congested blood vessels and inflammatory cells. Adjacent normal looking bowel showed extensive serosal inflammation.

Stercoral perforation of the large bowel is rare. Less than 100 cases have been reported in the literature. Idiopathic perforation is much less frequently reported. In the first patient the findings are in favor of the diagnosis of idiopathic perforation particularly in the first episode where there was no obvious pre-existing lesion or precipitating factor. There was no apparent reason for the second episode of perforation near the site of previous anastamosis. The absence of fecal matter in the peritoneum and very little faeces in the colon excluded the "stercoral" factor.

Recurrent perforation is very rare. One case of 3 episodes of idiopathic perforations in different parts of the large bowel has been reported in the literature.[5] However, there are no report of perforation at the same part of the colon as seen in patient number 1. One patient with type 4 Ehler-Danlos Syndrome has also been reported with developed idiopathic perforation of the sigmoid.[6] Another case report of a 14 year old male with type IV EDS died with multiple bowel and abdominal vessel ruptures.[7]

In the other 3 patients, the perforations are stercoral with fecal peritonitis. Several reports of large bowel perforations have been associated with chronic use of non-steroidal anti-inflammatory drugs and chronic constipation.[8-11] These 2 factors may exert their deleterious effects on the lower gastrointestinal tract through both local and systemic actions. Systemic effects are caused by the inhibition of cyclooxygenase and reduction of protective prostaglandins. The local damage of the intestinal mucosa in the distal bowel segments seems to be caused by sustained release formulation with a high enterohepatic circulation.[8] Severe untreated chronic constipation may, on rare occasions, cause free perforation of the sigmoid colon, and much less frequently of the cecum.[2, 12]

Surgical treatment is standardized and post-operative survival is over 60%. However, the morbidity and mortality rates depend on peritoneal contamination.[1] Early surgical eradication of the affected part of the colon including all stercoral ulcers and aggressive therapy for peritonitis leads to low mortality.[12] Timely intervention to prevent and/or treat any associated sepsis along with extensive peritoneal lavage and surgical removal of diseased colonic tissue at the primary stercoral ulceration site coupled with aggressive therapy for peritonitis are key treatment modalities in salvaging patients presented with stercoral perforation of the

colon.[13]

Primary resection with anastamosis and Hartmann procedure are not competing operations but are situation-dependent therapeutic concepts in spontaneous colonic perforation.[14]

The deaths of patients 2 and 3 were mainly due to sepsis associated with fecal peritonitis. Prolonged use of NSAIDs with steroid creams in case 2 may be a precipitating factor combined with longstanding constipation leading to multiple perforations. The fourth patient was diagnosed three days after the first symptoms but the radical aggressive treatment contributed to the recovery.

Left large bowel perforation by non-diverticular disease is associated with high mortality and morbidity. The prognosis is determined by the development of septic shock and colonic ischaemia as underlying disease may influence patient's survival.[15]

Colonic perforation can be a critical complication after renal transplantation.[16] In a review of 713 renal transplant patients at the Medical College of Wisconson resulted in 17 (2.3%) patients with spontaneous colonic perforations. The cause seemed to be nonocclusive ischaemia in nine cases. Hence, early detection was associated with improved survival.[17]

During the same period, three patients were successfully treated for traumatic perforations of the sigmoid colon. In two cases, the perforations were self-inflicted by passing an object through the anus and rectum resulting in the perforation of the sigmoid colon. The third was an iatrogenic injury to the sigmoid and small bowel during curettage for postpartum hemorrhage (Unpublished reports). There are no records of patients with perforated divericulosis or perforated malignant tumor of the colon seen at the hospital over the same period.

Overall, prevention of stercoral perforation may be achieved by:

1. Increasing the awareness of the public as well as the medical community on the possibility of spontaneous perforation of the bowel occurring from long standing constipation.
2. Careful monitoring of bowel habits of the debilitated, bed ridden patients, patients with cerebral palsy, paralysis, dementia or mental abnormalities.[4]
3. Regular rectal and abdominal examination of bedridden patient to ensure that the rectum is not full of hard fecal matter. Manual evacuation is a useful procedure to stimulate bowel motions
4. Limiting the use of nonsteroidal anti-inflammatory drugs to minimum period and using smaller doses of NSAIDs in chronically constipated patients.

Generally, spontaneous perforation of the colon is rare in western countries compared to perforations associated with the more prevalent diverticulosis and malignancy.[15] Diverticular disease is rare among Asians and Africans.[18] Consequently the incidence of perforations is rare in this part of the world. Spontaneous perforation of the colon should be considered in the differential diagnosis of patients with acute peritonitis and free gas under diaphragm. The idiopathic type is less common than stercoral type but the prognosis is better because of the minimum degree of fecal contamination. The stercoral type is associated with chronic constipation and the use of NSAIDs. Patients who had organ transplantations, those on long term steroids therapy and those with congenital diseases like type IV Ehlers- Danlos syndrome are most likely to develop spontaneous perforations than the rest of the population.

The diagnosis can be delayed but early surgical intervention is important in order to reduce morbidity and mortality. On the other hand, stercoral perforation of colon and rectum can be prevented.

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