# Neglected Fecal Impaction in HIV- Infected Patient leading to Stercoral Colonic Perforation with Fatal Peritonitis: A Case Report 

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#### Abstract

Background: Untreated constipation can lead to fecaloma loading in colon, stercoral colonic perforation and subsequently stercoral peritonitis. Stercolar peritonitis is relatively uncommon presentation with a grave prognosis. Case Presentation: We recently managed a 46 -year-old lady known HIV- infected who had a long standing history of constipation. She presented to us with history of acute generalized abdominal pain, fever, vomiting and signs of peritonism for four days. Abdominal radiography showed colonic fecal loading and pneumoperitoneum while abdominal ultrasound demonstrated free fluid in the peritoneal cavity. She underwent emergency exploratory laparotomy in which, we found a perforation at rectosigmoid junction with protruded fecaloma and significant feco-purulent peritoneal fluid. Evacuation of fecolomas, resection of perforated bowel and Hartmann's procedure was done. The patient was sent to a high dependent unit with standard post-operative orders. She unfortunately demised on day 2 post-operatively due to intractable sepsis. Conclusion: Fecal impactions should be promptly treated to avoid fatal complications like stercoral peritonitis. Judicious selection of patients with underlying risk conditions like HIV/AIDS for referral and expert definitive management can warrant relatively favorable surgical outcome.


Keywords: Fecal impaction, Stercoral perforation, Peritonitis, HIV.
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## Background

Chronic constipation can lead to fecal impaction. If left untreated, it can also rarely lead to catastrophic complications like colonic obstruction, stercoral perforation and peritonitis [1, 2]. Although conservative management is sufficient in most cases of fecal impaction, the possibility of stercoral peritonitis should be considered when patient presents with acute abdominal symptoms. Timely management of stercoral peritonitis by attaining optimal pre-operative preparation; immediate treatment of any underlying sepsis; removal of all stercoral ulcerated diseased colonic tissue; extensive peritoneal lavage; aggressive post-operative management and appropriate treatment of any co-morbid medical conditions can warrant relatively good prognosis.

We report a rare case of neglected fecal impaction in HIV-infected patient leading to stercoral
colonic perforation with fatal peritonitis in Northwestern Tanzania.

## Case Presentation

A 46-year-old lady known HIV-infected for the past nine years was admitted to the Emergency Department (EMD) of Bugando Medical Centre (BMC), a referral, teaching and consultant hospital in Mwanza, Northwestern Tanzania. She presented with history of constipation and abdominal discomfort for three weeks. She has been attending Care and Clinic (CTC) for the past 9 years in a district hospital for routine highly active antiretroviral therapies (HAART) re-fills and Peptic Ulcer Disease (PUD) treatment till four days when her condition deteriorated and started to experience worsening abdominal pain, vomiting and low grade fever prompting her to be admitted at BMC for further management.

Upon arrival at EMD she was full conscious, restless, with a cannula on hand running normal Saline 500 mls at the rate of 36 drops per minute and urethral catheter draining concentrated urine volume of 320 mls for the past 24 hours. She looked dehydrated with dry mucous membrane, mild pale, febrile with a temperature of $38^{\circ} \mathrm{C}$, pulse rate (PR) of 92 beats per minute, blood pressure (BP) of $98 / 78 \mathrm{mmHg}$, Saturation partial Pressure of Oxygen $\left(\mathrm{SPO}_{2}\right)$ of $96 \%$ in room air, Fasting Blood Glucose (FBG) $7.7 \mathrm{mmol} / \mathrm{l}$ and respiratory rate (RR) $26 / \mathrm{min}$.

Abdomen was uniformly distended with reduced movement during respiration. She had mild generalized abdominal tenderness on superficial palpation with dough like mass felt on each frank. On deep palpation there was severe tenderness, generalized guarding and marked rebound tenderness. Digital rectal examination revealed hard impacted stool in the rectum. Other systems were essentially normal.

An abdominal radiograph (Figure 1) reveled colonic fecal loading with mottled sign and pneumoperitoneum abdomino-pelvic ultrasound showed colonic fecal loading, absent peristalsis movement and free fluid in the peritoneum. She had electrolyte imbalance with severe Hypokalaemia \{Serum-Potassium [K] $1.47 \mathrm{mmol} / \mathrm{l}$, Sodium [Na] $136.95 \mathrm{mmol} / \mathrm{l}$, Chloride [Cl] $96.6 \mathrm{mmol} / \mathrm{l}$ and calcium [Ca] $1.9 \mathrm{mmol} / \mathrm{L}]\}$. Complete Blood Cell count (CBC) revealed Normochromic Microcytic Anaemia (Hb $8 \mathrm{~g} / \mathrm{dl}$ ), Leucocytosis (WBC $18000 \mathrm{~mm}^{3}$ ). Serum Urea level was normal with an elevated Serum Creatinine ( $128 \mathrm{mmol} /$ litre).

A diagnosis of peritonitis secondary to perforated hollow viscus; Anaemia and Hypokalaemia in HIV-Seroconverted patient was concluded.

We prepared her for an emergency exploratory laparotomy by rehydration with Ringers Lactate 3 Litres to run in 2 hours, management of severe hypokalaemia as per standard guideline and preoperative management of anemia with blood transfusion. Anaesthesiologist informed to review and later patient taken into operating theatre after attainment of optimal condition for an emergency exploratory laparotomy. She also received Ceftriaxone 1 gm and Metronidazole 500 mg intravenously 15 minutes before incision as pre-operative antibiotics in line with BMC treatment guidelines.

Intra-operatively, we found a 2 cm perforation at anti-mesenteric border of the rectosigmoid junction with an extra-murally protruded fecaloma (Figure-2), whole of colon was distended with palpable fecalomas from ascending colon to rectum and significant fecopurulent peritoneal fluid (about 1.5litres) with fibrinous process matting the ileum to colon. Most parts of bowel were edematous with reduced peristaltic movement.

Inspection of the colon did not reveal clinical features suggestive of neoplasms or diverticula. Other viscera were essentially normal.

An intraluminal lavage was performed and we managed to evacuate fecalomas of about 1.2 Kgs , (Figure-3) from the full length of larger bowel through the perforation. Suctioning feco-purulent fluid was done from peritoneal cavity followed by clearing of fibrinous process. Segmental resection of perforated sigmoid colon was done followed by Hartmann's procedure with rectal mucous fistula. Resected segment sent for histopathology examination. Abdominal lavage was done by copious irrigation with 10 litres of warm saline, we succeeded to achieve haemostasis and abdominal drainage tube inserted. Inspection of the full length small bowel from the ligament of Treitz to all sides of the mesentery to the ileocecal junction and larger bowel to the distal sigmoid colon was done. There were no any other perforations, obstructions or ischemic changes seen.

Abdominal wall closed using simple interrupted suture by using PDS no 1 to the fascia without skin closure. Estimated blood loss was 200 mls and she received one unit ( 450 mls ) of whole blood intra-operatively. Patient tolerated well the operation which took 3 hours and later sent to high dependency unit (HDU) following recovery from Anaesthesia.

In HDU, the patient was given intramuscular (IM) Pethidine 50 mg 6 -hourly for 24 hours, Intravenous (IV) Paracetamol 1 gram 8-hourly for 2 days, Ringers Lactate and Dextrose Saline IV 4.5 litres in 24hours, KCL20mEq IV in 500 mls of $0.9 \%$ Normal Saline, Enoxaparin 40 mg subcutaneously 12 hourly for 3 days, Ceftriaxone 1 gm IV once per days for 5 days, Metronidazole 500 mg IV 8hourly for 5days, Pantoprazole 40 mg IV 12 -hourly for 5 days. We continued to monitor for fluid input and output, BP, PR, RR and $\mathrm{SPO}_{2}$ after every 2 hours.

She progressed well despite spikes of fever after 6 hours and first day post Laparotomy and we instructed her to resume HAART. Her condition suddenly changed on second day, she become dyspnoeic (RR 27/min), hypotensive (BP 98/68mmhg), sinus tachycardia (PR $123 \mathrm{~b} / \mathrm{min}$ ), hypothermia temp $\left(35.0^{\circ} \mathrm{C}\right.$ ), Hyperglycaemia (FBG $7.7 \mathrm{mmol} / \mathrm{l}$ ) and SPO2 $92 \%$ in room air. We ordered Arterial Blood Gas (ABG) which showed PH 7.247 and serum electrolytes - [K] $1.5 \mathrm{mmol} / \mathrm{l}$, [ Na ] $153.0 \mathrm{mmol} / \mathrm{l}$ and [Cl] $120.0 \mathrm{mmol} / \mathrm{l}$.

We had an impression of septic shock and severe Hypokalaemia, blood sample was taken for Blood Culture and Sensitivity as well as control Complete Blood Count (CBC). She was kept warm and given supportive Oxygen therapy 5 to 7 litres per minute and later transferred to Intensive Care Unit
(ICU). We started management of severe hypokalemia again as per standard guideline. Control CBC showed Leukocytosis of $18,000 \mathrm{~mm} 3$ and thrombocytopaenia of Platelets of 80,000 per microliter of blood. Her condition kept on deteriorating and she succumbed 6
hours after being transferred to ICU. Histopathology results of a resected colon revealed a perforation and ulcers along the anti-mesenteric border with features suggestive of acute ischemic necrosis.


Figure-1: Supine abdomen radiography showed marked large bowel fecal loading with a mottled gas appearance and hyperlucency in the upper abdomen with gas is outlining both sides of the bowel wall ('Rigler's sign') in keeping with intraabdominal free air


Figure-2: The Image taken intraoperatively showing stercoral perforation at anti-messenteric border of recto-sigmoid junction with an extramural protruded fecoloma pointed out by a pair of forceps


Figure-3: The image showing Fecolomas milked out through a stercoral perforation of HIV-infected patient who had longstanding history of constipation

## Discussion and Conclusion

Stercoral colonic perforation is an infrequent acute abdominal emergency occurring due to the pressure effect of hard fecalomas on the wall of an otherwise normal colon, in the absence of any other established pathology [1]. The pathogenesis is due to poor hydration of the feces, resulting in hard and impacted feces, also called fecoloma. Progressive loading of fecoloma in bowel led to increases intraluminal pressure and causes ischemic necrosis of the colonic wall, stercoral ulcer formation, colonic perforation and subsequently stercolar peritonitis [2]. Literature review has revealed that chronic or intermittent constipation has been implicated to be the common cause of fecal impaction that may led to fecalomas [3, 4]. This finding is similar to our case, in which patient had history of constipation for more than three weeks without any appropriate medical or nutritional intervention. The other probable risk for constipation and eventually stercoral ulcer perforation in our patient is the history of longstanding use antacids for treatment of PUD. Erckenbrecht J et al., [5] in their study on effects of high dose antacids on bowel motility, associated use of antacid to constipation and eventually stercoral ulcer perforation. Other risk factors for stercoral ulcer perforation are use of non-steroidal
anti-inflammatory drugs (NSAIDs) or drugs-like Amitryptyline, Steroids, Codeine, and Heroin [5, 6].

Our patient had a perforation on the antimesenteric border of the rectosigmoid colon. This is one of the common sites of stercoral ulcer and/or perforation, other anatomical locations are the sigmoid colon and the anterior rectum (just proximal to the peritoneal reflection) [2, 7]. These sites are related to decrease perfusion at the anti-mesenteric border [2, 8] as well the narrow diameter of the distal colon, due to which it is difficult for hard stools to pass through, leading to increased intraluminal pressure and colonic ischemia [8].

Despite the wide range of laboratory and radiographic tests available and their utility in evaluation of abdominal pain, secondary peritonitis remains primarily a clinical diagnosis [9]. In our case patient presented with history of worsening abdominal pain, low grade fever and vomiting which supported with signs of peritonism (tenderness, guarding and rebound tenderness). These are minimal clinical features that warrant a diagnosis of peritonitis in most of resource limited setting. The history of constipation for 3 weeks and palpable hard stool on digital rectal
examination raised suspicion for stercoral peritonitis in our patient.

On imaging, supine abdominal radiographs usually show fecal impaction within the bowel and it can be utilized as confirmatory investigation in most of resource limited setting [10]. As presented in our case report, supine abdominal X-ray showed marked colonic fecal loading with mottled sign and pneumoperitoneum which signified that perforation has already occurred. However, chest x-rays demonstrate free-air under the diaphragm in only $30 \%$ of colonic perforations, so we didn't request chest $x$ - ray in our case which is usually ordered in patient with suspected visceral perforation. In resourceful setting, CT of the Abdomen and Pelvis should be considered in cases with suspected stercoral perforation for preoperative diagnosis which has a reported accuracy ranging from 82 and $90 \%$ [11].

The principles of management of secondary peritonitis due to stercoral perforation are optimal preoperative preparation; immediate treatment of any underlying sepsis; removal of all stercoral ulcerated diseased colonic tissue; extensive peritoneal lavage; aggressive post-operative management and appropriate treatment of any co-morbid medical conditions [9, 12]. In our case, we managed to attain optimal condition for emergency laparotomy by resuscitation and adequate correction of electrolyte imbalance. Treatment of intraabdominal sepsis was achieved by massive saline irrigation and perforation control with the intention of decreasing the bacterial load in the abdominal cavity and deterring the development of overwhelming sepsis which is similar to interventions recommended in literature review [13].

Broad spectrum antibiotics were initiated 15 minutes before incision and continued to be administered as part of post-operative care to combat sepsis caused by polymicrobial peritoneal cavity contamination resulting from intestinal perforation this concurred with several articles [11, 12]. Emergency exploratory laparotomy, resection of segment with perforation and Hartmann's procedure performed in our case. These are recommended surgical interventions that give an optimal clinical outcome and comparatively low mortality as reported in two review articles [14, 15] and one series [16]. In general, septic sequelae and death occur most frequently after anastomosis following colectomy rather than diversion [14].

Stercoral perforation in our case was not purely a surgical condition as it was probably complicated by immuno-compromised state which imparts a poor prognosis [12]. Our patient was known HIV-infected for 9 years and was on medications. We could not manage to get details of her recent CD4 count and viral load. However, studies of the value of viral load and CD4 counts (alone or in combination) in predicting operative morbidity and mortality did not
produce conclusive results and these tests are not ideal for every day practical use [17, 18]. Four factors that have been found to increase operative morbidity and mortality in HIV/AIDS patients are (a) a compromised physiological state - as in general surgery the best predictors of peri-operative morbidity and mortality appear to be scores that measure general health such as ASA (American Society of Anaesthesiology) risk classes or the Korsakoff's performance scale; (b) physiologically demanding surgery; (c) emergency surgery as opposed to elective procedures and; (d) operations in contaminated fields, such as anorectum or oral cavity $[18,19]$. Our patient demised on the second day post operation and she had almost all factors mentioned to increase operative morbidity and mortality. In general, stercoral peritonitis can be life threatening with mortality rates ranging between 32 to $57 \% ~[2,3,20]$.

This case report highlights how a neglected fecal impaction can lead to fatal stercoral peritonitis. Early and accurate diagnosis of a preventable and treatable cause such as constipation could have changed the prognosis, reduces resources used and time wasted in management. In our case report, delay in diagnosis and promptly treatment of constipation at the district hospital and late referral when a patient started to manifest with features of peritonitis could have contributed to poor outcome. This reflects the challenging state of health care in our developing world and that need to be addressed by the concerned authorities. We are recommending development of standard operating procedures for emergency surgical conditions that will guide health care workers in district hospitals on initial management and when to refer patient with this kind of condition.

## List of abbreviations

BMC: Bugando Medical Centre
CTC: Care and Treatment Clinic
EMD: Emergency Department
HDU: High Dependency Unit
ICU: Intensive Care Unit

## Declaration

Ethical approval and consent to participate: This case report was approved by institutional ethical review board of Catholic university of Health and Allied Sciences.

Consent for publication: The patient's family gave written consent for their personal, clinical details along with images to be published in this case report.

Availability of data and materials: All patient's information are retained at Medical record Department of Bugando Medical Centre.

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