TUBERCULOUS ILEAL PERFORATION IN A HIV POSITIVE PATIENT 
A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT
Background: Tuberculosis is prevalent worldwide. Even in developed countries there is a resurgence of tuberculosis mainly due to increasing HIV infection. Tuberculous ileal perforation is uncommon. It is, however, a potentially fatal complication of intestinal tuberculosis especially in HIV/AIDS patient.

Aim: To highlight tuberculosis ileal perforation as an underestimated complication of intestinal tuberculosis in an HIV patient presenting with acute abdomen.

Method: A 42 year old HIV positive long distance truck driver with tuberculous ileal perforation is presented and related literatures reviewed.

Conclusion: Intestinal perforation due to abdominal tuberculosis is an aetiological factor in acute HIV abdomen. High index of suspicion remains the key to diagnosis.

Key Words: Tuberculosis, Ileal Perforation, HIV.

INTRODUCTION
Tuberculosis (TB) is one of the leading causes of HIV related morbidity and mortality throughout Africa. The resurgence of TB in developed countries is mainly due to increasing HIV infection. In developing countries, where poor diagnosis and improper treatment of TB have resulted in multiple drug resistance, more cases of severe intra-abdominal TB catastrophes are now seen. Abdominal TB represents about 6% of extra pulmonary disease in Caucasians. There are few literatures on TB ileal perforation especially in sub Saharan Africa. Ileal perforation is an uncommon but potentially fatal complication of intestinal tuberculosis. Prognosis is worse if diagnosis is delayed. We hereby present our experience in the management of TB perforation of the ileum in an HIV patient.

Case Report
A 42 year old long-distance truck driver presented to us with three months history of relative constipation and dull aching lower abdominal pain, low grade fever and night sweats. Three days prior to presentation, abdominal pain became colicky and generalized with progressive abdominal distention. No history of vomiting. There was history of anorexia and progressive weight loss, but no cough or chest pain or contact with chronic cough patient. He was diagnosed HIV positive four years before index presentation but defaulted to Anti Retroviral therapy (ART). There was a history of cigarette smoking and alcohol consumption. On clinical examination he was chronically ill-looking, pale, not dyspnoic, severely dehydrated, no significant peripheral lymphadenopathy. The chest was clinically clear. The abdomen was distented with tenderness at right iliac fossa and periumbilical regions. There was a vague mass in the right iliac fossa. No demonstrable ascites. The bowel sound was high pitch. The rectum was empty. He was resuscitated using intravenous fluids, nasogastric tube for decompression and urethral catheter for urine out put monitoring. Parenteral Ceftiraxone 1g 12 hourly and Metronidazole 500mg 8hourly were given. The investigations carried prior to surgery were; Full blood count showed PCV 34%, WBC 15 x 10³/l, ESR 140mm/hr. Chest x-ray was normal. Plain abdominal x-rays showed evidence of peritonitis but no air under the diaphragm. Abdomino-pelvic scan showed ill-defined mass in the lower abdomen. Manteaux test Negative. HIV I positive. CD4 count 287cells/ul. His wife was also HIV I positive. At laparotomy there were multiple tubercles on the omentum walling off a perforated ileum 20cm from the ileo-caecal junction. Adhesiolyis was done, perforation edge was excised and closed in two layers using chromic cutgut 2/0 and silk 3/0 and then the peritoneum was lavaged using normal saline. The excised perforation edge tissue histopathology examination revealed tuberculous granuloma (fig.1). The patient developed faecal fistula two weeks post-operation. He was placed on high protein diet and colostomy bag was used to

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collect the fistula effluent. He was commenced on anti TB therapy; tabs INH 150mg, caps Rifampicin 600mg, tabs Pyrazinamide 1.5g, and tabs Ethambutol 800mg daily, and pyridoxine 10mg daily, ten days postoperatively and continued for four weeks before commencing the Highly Active Antiretroviral Therapy (HAART). The fistula healed and he was discharged home and presently being followed up.

Figure 1: Photomicrograph of Tuberculosis Granuloma of the Ileum.

DISCUSSION
The advent of HIV/AIDS has rekindled interest in tuberculosis world wide. Intestinal tuberculosis is usually caused by the human strain of mycobacterium tuberculosis but in areas where people consume fresh unpasteurised milk, as still obtains in many tropical countries, bovine strain infection is common. Usually the infective process is arrested leading in many cases to glandular calcification. If it progresses, however, three main types of lesions may be produced: ulcerative, hypertrophy or ulcero-hypertrophic. These may occur anywhere along the intestinal tract with caecal lesions accounting for 50% of cases. Perforation of ulcerative ileocaecal tuberculosis is rare because of the mounted immunological reaction leading to the thickening of the ulcerated segment leading to stricture from progressive cicatrisation. Perforation of the ulcer may be encountered in HIV infection due to the defect in immune response caused by HIV virus co-infection. Hence the patient will present with features of Acute AIDS abdomen posing a new challenge to surgeons. Plain abdominal x-rays showing free peritoneal air or multiple gas fluid levels with dilated intestinal loop may confirm perforation. If the plain films are normal and acute abdominal pain is present, Ultrasound scan may show mesenteric and Para-aortic lymphadenopathy. CT scan may show the site of perforation. Manteaux test may be non reactive because of immune suppression by HIV virus infection. Full blood count may show anaemia and neutrophilia. However, in this case the investigations were normal except the ultrasound scan that showed a vague mass in lower abdomen. A high index of suspicion is therefore important in the establishment of the diagnosis.

Emergency abdominal surgery in acutely ill AIDS patient carries about 100% morbidity and 70% mortality with 40% hardly leaving the hospital alive. Our patient developed faecal fistula two weeks in the postoperative period and was managed conservatively. From previous studies, early initiation of anti-tuberculous therapy using directly observed treatment short-course strategy and HAART markedly improves the outcome of severely HIV-infected patients with TB. Though there is no evidence regarding the appropriate time for initiating HAART in patients with HIV-TB co-infection, in this patient Antituberculous therapy was started ten days post surgery and continued for four weeks before initiating HAART so as to minimize drug reactions. It's our conclusion that TB intestinal perforation is likely cause of acute HIV abdomen and a high index of suspicion remains the key to diagnosis.

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REFERENCES


