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Abdominal Tuberculosis Presenting as Acquired Megacolon

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Authors' contributions

This work was in collaboration between both authors. Author VICN designed the study, wrote the first draft of the manuscript. Both authors managed the literature searches, read and approved the final Manuscripts.

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Case Study

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ABSTRACT

Background: Tuberculosis is a chronic granulomatous infection caused by the bacilli Mycobacterium tuberculosis. Decades after the discovery of effective treatment, the infection persists till date as a re-emerging communicable disease. It is preventable by childhood vaccination. Its presentation may be protean and mimic many other conditions. Abdominal tuberculosis presents a diagnostic challenge hence the need to expose the various shades of presentation to aid prompt diagnosis and proper management to reduce attributable morbidity and mortality. We report a case of abdominal tuberculosis presenting as acquired Megacolon

Case Report: A 28-year-old mother of three presented with six months history of recurrent constipation, marked weight loss, gross abdominal distension and colicky abdominal pain. She was evaluated and a clinical impression of acquired Megacolon was made but at laparotomy features of abdominal tuberculosis were found and confirmed by histopathologic examination of biopsied granulomatous lesions on the colon and lymph nodes. A temporary loop colostomy was fashioned and later closed after six-month successful treatment with antituberculosis drugs.

Conclusion: Abdominal tuberculosis should be considered in the differential diagnosis of adult patients presenting with chronic constipation and distension.

Keywords: Abdominal tuberculosis; abdominal distension; constipation; Megacolon.

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1. INTRODUCTION

Abdominal tuberculosis, is the sixth most frequent form of extrapulmonary tuberculosis and constitutes a diagnostic challenge to clinicians as it has no specific clinical features [1-6]. It is seen in 10 – 30% of patients with pulmonary tuberculosis [2]. It is usually found in adults from endemic areas of the Low and Middle-income countries of the world as well as the immigrant populations of advanced countries and dwellers of overcrowded urban slums [5-6]. In 2013, the World Health organization (WHO) estimated the global burden of tuberculosis to be about 26 million with greater than 50% found in India, China, the Russian federation and South Africa [1]. Other factors promoting the re-emergence of tuberculosis include Human Immunodeficiency Virus infection, Diabetes Mellitus, malnutrition, Alcoholism.

Tuberculosis is primarily a pulmonary disease but abdominal tuberculosis is present in 15 -25% of cases [1]. The local prevalence of abdominal tuberculosis is difficult to estimate as it is a diagnosis of exclusion. The incidence is observed to be increasing in the population of both immuno-competent and immuno-compromised due to co-infection with HIV/AIDS [1]. The age incidence is equal. Routes of involvement of the abdomen may be through ingestion of infected milk or sputum, haematogenous from pulmonary or other extra-pulmonary foci, direct spread to peritoneum from adjacent involved affected abdominal organs or through lymphatic channels from affected lymph nodes [1]. It may present as a chronic or acute on chronic intestinal obstruction [2]. The clinical features are non-specific including abdominal pain, distension, constipation, anaemia and weight loss. It presents in many pathological forms and combinations usually seen during surgical exploration viz ulcero-stricturous small intestinal disease, ulcero-hypertrohic colonic and ileocaecal disease, enlargement of mesenteric lymph nodes (tabes mesenterica) thickened omentum studded with nodules (pancake omentum) matted loops of intestine, colonic strictures, ascites [1,2,4-6]. Clinical experience and a high index of suspicion are essential for diagnosis. It is usually amenable to medical therapy but when complicated by intestinal obstruction, perforation or haematochezia, surgery becomes a necessary adjunct to treatment. Massive abdominal distension and long duration of colorectal obstruction due to abdominal tuberculosis closely mimicking acquired megacolon disease, is rarely described in literature.

2. CASE REPORT

A non-gravid twenty-eight-year-old mother of three presented at the Surgical Out Patient Clinic of our hospital with a six-month history of colicky abdominal pain, inability to defaecate and progressive abdominal distension. She had normal bowel movement of once or twice a day, passing formed stool prior to onset until it became progressively scanty and later ceased as her distension worsened. She had no painful defaecation and admitted to intermittent use of local herbal enema without improvement. There was no associated soilage of under wears. She had never been hospitalized prior to this episode. She denied any history of cough, night sweats nor received treatment for chest infection. She denied any history of nausea or vomiting. Her children, husband and parents were alive and well and do not have prolonged cough.

On examination, we found an emaciated young lady with massively distended and shiny abdomen. The abdomen was the size of 39 weeks with polyhydramnios and demonstrated numerous frequent peristaltic waves on the surface of the shiny abdomen. Palpation revealed moderate tenderness but no masses could be felt on account of the tense abdominal wall.

(See Fig 1. Below). Rectal examination revealed only an empty rectum.

An impression of chronic intestinal obstruction with megacolon was made and the investigations requested included plain abdominal X-ray, abdomino-pelvic ultra-sonography, serum electrolytes and blood grouping/crossmatching. Contrast enema was not requested in view of Intestinal obstruction. The patient was admitted to the ward, optimized and operated upon electively.

At surgery, under general endo-tracheal anesthesia, the abdomen was opened via a midline incision through which massively distended loops of bowel tumbled out. (See Fig. 2 below).

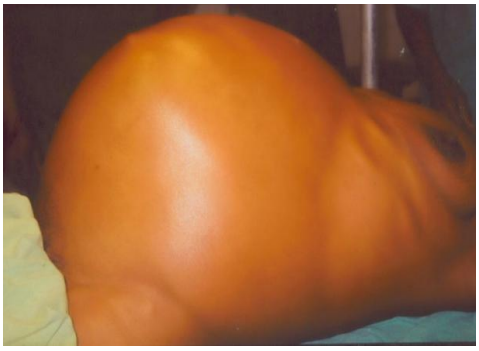

Fig. 1. Shiny, grossly distended abdomen

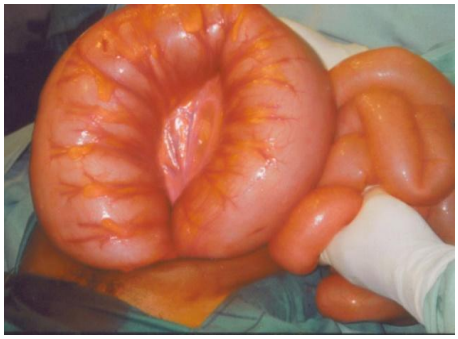

Fig. 2. Intestines gushing out at laparotomy

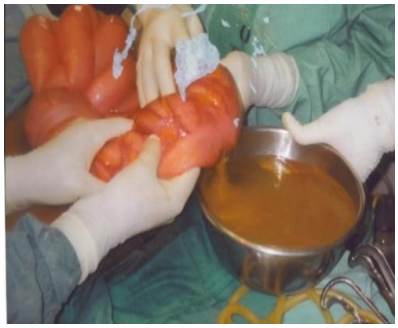

Fig. 3. Brownish fluid stool after enterotomy

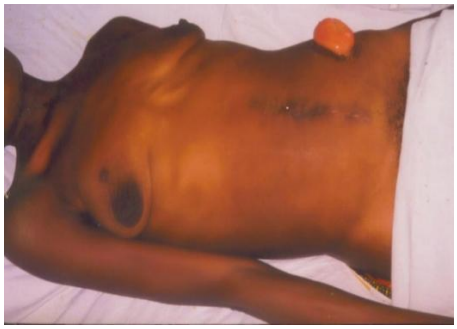

Fig. 4. Sigmoid Colostomy before take down

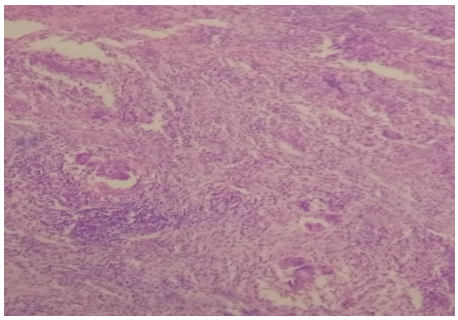

Fig. 5. H&E X100 – TB of the Peritoneun

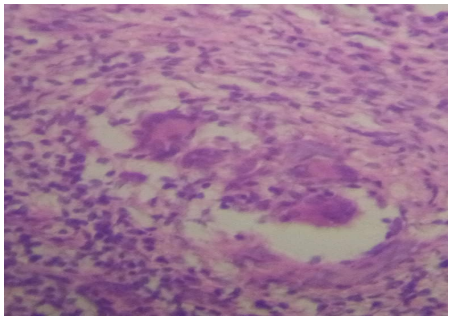

Fig. 6. H&E X 400 – TB of the Peritoneum

A careful exploration of all the viscera was conducted. The liver, spleen and stomach were normal but the omentum/mesentery was thickened and studded with multiple peritoneal nodules. The mesenteric lymph nodes were enlarged and matted together while the visceral and parietal peritoneum were dotted with granulomatous nodules. There was little ascitic fluid. A fibro-granulomatous lesion could be felt at the rectosigmoid junction which was biopsied along with the mesenteric lymph nodes.

A decompression colotomy was done on the antimesenteric border of a segment of the sigmoid colon through which 13.5 Litres of thick brownish fluid stool was evacuated (Fig. 3). It was later brought out as a loop colostomy (Fig. 4).

The patient was treated with antituberculosis drugs including Rifampicin, Isoniazid and Ethambutol post-operatively for six months following histological confirmation of tuberculosis as shown in the photomicrograph (Figs 5, 6). The colostomy was closed six months later after confirming distal continuity of bowel and she has remained well and gaining weight on follow up.

3. DISCUSSION

Abdominal tuberculosis is a term used to describe the various manifestations of tuberculosis in the gastrointestinal tract and its accessory organs, the liver, pancreas and the peritoneum as well as the abdominal lymph nodes and spleen singly or in combination [2-7]. It is caused by Mycobacterium tuberculosis, an acid-fast bacillus (AFB) on Ziehl Nielsen (ZN) stain. The organism is difficult to culture and the finding of typical caseous necrosis with giant cell formation on histopathology is diagnostic.

The tuberculosis bacilli reach the gastrointestinal tract via several routes from primary lung focus, ingestion of bacilli in sputum form active pulmonary focus, direct spread from adjacent organs, and through lymph channels from infected nodes and the fallopian tubes [5-7]. The commonest abdominal site of involvement is the ileocaecal region [4-7]. Colonic and rectal tuberculosis in the form of ulcero-strictural lesions present with pain, changes in bowel habits, haematochezia, intestinal obstruction and constitutional disturbance [6,7]. Ascites, usually mild to moderate is a feature of wet peritonitis as noted in our case but could not account for the massive abdominal distension depicted. The complete rectosigmoid stricture in the patient caused obstruction, leading to maximal colonic dilatation and our initial suspicion of acquired megacolon. However, trypanosomiasis caused by T.cruzi is relatively uncommon in our setting.

We believe that caecal bursting was averted by presence of incompetent ileocaecal junction probably distorted by the presence of tuberculous granulomas as the small bowel was also distended thus averting a closed loop situation. The caecal diameter was about 12 cm while the sigmoid diameter was more than 7cm in addition to stretching, qualifying it as a megacolon.

In the case of our patient, the stricture involved the rectosigmoid junction with complete occlusion of the lumen with many other areas of fibrotic adhesions. Chest X-ray reveals concomitant pulmonary lesions in less than 25% of patients with abdominal TB thus a normal chest X-ray as in this patient does not rule out abdominal tuberculosis. Her plain abdominal X-ray showed dilated loops of colon with air fluid levels suggesting intestinal obstruction necessitating exploration. Abdominal ultrasonography was done but showed no pathology of the solid organs. Invasive surgical procedures are not indicated if diagnosis is proven as anti-tuberculosis drugs are able to resolve the lesion within six months of therapy [8-11]. However, in cases with intestinal obstruction, urgent surgical relief is invited. In our index patient, a temporary decompressing colostomy was created to ward off caecal bursting. It was closed after six months of antituberculosis therapy and establishment of distal bowel continuity. In the absence of specific clinical features and active pulmonary tuberculosis, clinical diagnosis of abdominal tuberculosis will continue to elude clinicians especially as it may mimic a host of other abdominal diseases.

3.1 Biopsy of Peritoneal Nodule

3.1.1 Microscopy

Caseating granuloma comprising of numerous mononuclear inflammatory cells mainly lymphocytes, plasma cells, epitheloid cells and multinucleated giant cells of Langerhan – Tuberculosis of the peritoneum.

4. CONCLUSION

Abdominal tuberculosis is an elusive but treatable communal disease. It lacks specific clinical features thus causing low suspicion with capacity to invite surgical intervention.

Our report highlights the importance of thorough clinical evaluation of patients with chronic constipation and abdominal distension. A high index of suspicion of abdominal tuberculosis in such patients is vital, especially in resource poor regions where it may mimic other disease entities.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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