

Spontaneous "true" ileal perforation of tuberculous origin: a report of two cases

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Abstract

Majority of the tuberculous intestinal perforation are associated with a distal stricture and are considered to be blow outs due to distension. Perforations not associated with strictures or adhesive bands which can cause proximal dilatation are only considered as true perforations. Some of the perforations are associated with initiation of antituberculous therapy. Herewith we report 2 cases of tuberculous ileal perforation which are associated with neither strictures nor antituberculous therapy. Hence these are considered as spontaneous and true.

Key Word: tuberculous, Spontaneous.

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INTRODUCTION

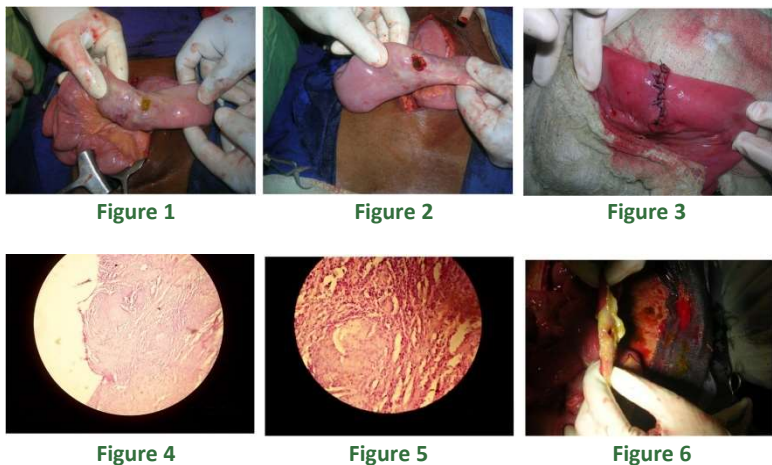
Free ileal perforation of tuberculous ileal ulcer is rare. Because of the chronic granulomatous reaction and fibrosis, perforation is usually contained within adhesions. Herewith we report two cases of spontaneous true ileal perforation.

CASE REPORT

CASE 1

A 68 year old male, farmer by occupation presented to the hospital with a history of pain abdomen of 1 day duration. Pain was sudden in onset, continuous, located in the lower abdomen and aggravated by movements. There was no history of vomiting or fever. No history of drug intake and no history of trauma. He has been smoking and consuming alcohol for the past 30 years. On examination, patient was afebrile. Vital signs are: pulse rate 98/minute, BP 130/84mm Hg and RR 24/minute. On examination

there was tenderness in the right iliac fossa and hypogastric region. There was localized guarding in the right iliac fossa. Rebound tenderness is present in the right iliac fossa. Liver dullness was obliterated. Per rectal examination showed anterior wall bulge indicating fluid collection in the pelvic cavity. X-ray abdomen erect view showed free air under the diaphragm. Laboratory investigations were within normal limits. HIV test was negative. A clinical diagnosis of hollow viscus perforation was made and laparotomy was done. On opening the abdomen, about 500 ml of purulent fluid was present. Appendix was normal. There was a 1x1 cm perforation about 40cm from ileocaecal junction. The perforation was transverse in orientation (Fig.1). There were no enlarged mesenteric lymph nodes. Complete laparotomy revealed no stricture or adhesive band distal to the perforation. The edges of the perforation were excised and the perforation site closed in two layers (Fig.2 and3). The excised specimen was sent for histopathologic examination. Microscopic examination of the specimen showed small intestinal mucosa with ulceration, necrosis and dense inflammatory cell infiltration with nodular collection of epithelioid cells, giant cells, polymorphs and lymphocytes (Fig.4and5). The impression was tuberculous lesion of the ileum with ulceration and necrosis. The post operative period was uneventful and the patient was discharged on 10th postoperative day. He was started on antituberculous treatment.



Legend:

- Figure 1:** A transversely oriented perforation in the ileum. The adjacent bowel appears indurated.
- Figure 2:** The edges are excised and bleeding healthy tissue is seen
- Figure 3:** Perforation closed in two layers
- Figure 4:** Low power view showing granuloma
- Figure 5:** High power view showing granuloma with epithelioid cells and giant cells.
- Figure 6:** Ileal perforation with longitudinal orientation. The HPE report was tuberculosis. When only the Payers’ patches is involved, the ulcer is longitudinal. Only when spread through the lymphatics which are transversely oriented occurs, the ulcer attains the classical transverse orientation.

CASE 2

A 17 year old male patient was admitted with history of pain abdomen for 2 days. The onset was sudden and the pain was present all over the abdomen. It was severe and not related to drugs. No history of vomiting. Continuous fever was present from the time of onset of pain. Not associated with rigors. On examination, the patient was conscious, oriented and febrile. His pulse rate was 130/min, blood pressure was 96/60 mm Hg, and respiratory rate was 28/min. the temperature was 100° F. abdomen was guarded and there was tenderness all over the abdomen. Liver dullness was obliterated. Per rectal examination revealed bulging rectovesical pouch indicating fluid collection in the pelvic cavity. Laboratory investigation revealed increased blood urea and serum creatinine (56mg/dl, and 1.2mg/dl respectively). Serum electrolytes were within normal limits. HIV test was negative. X-ray abdomen erect revealed free air under diaphragm. A diagnosis of hollow viscus perforation was made and laparotomy was performed. Abdomen was opened through a midline incision. About 250 ml of purulent fluid was present. There was a 1cm x 0.5 cm sized perforation about 15cm from the ileocaecal junction (Fig.6). The ulcer edges were excised and the perforation was closed in layers. Complete laparotomy revealed no stricture or adhesive band distal to the perforation. After thorough peritoneal lavage, the abdomen was closed. The postoperative period was uneventful and the patient was discharged on 10th postoperative day. This ulcer was longitudinal in orientation and to our surprise the HPE

suggested tuberculosis. The histopathology report of the excised wound edges came as: section shows completely ulcerated mucosa with necrosis, fibrosis with infiltration of inflammatory cells with a focal granuloma consisting of epithelioid cells and Langerhans giant cell. Diagnosis is granulomatous lesion suggestive of tuberculosis. The patient was on antituberculous therapy and followed up.

DISCUSSION

Intestinal tuberculosis can be ulcerative, hypertrophic or ulcero-hypertrophic. Ulcerative form is more common, but the ulcers rarely perforate. In the ulcerative type, the ulcer bearing segment is moderately indurated and the mesenteric fat in the affected region is increased. Nodules may be present on the serosa. The ulceration is relatively superficial and usually does not penetrate the muscularis propria. The thickness of the wall underlying the ulcer bed is variable and may appear hypertrophic and scarred¹. The tuberculous ulcer is caused by the spread of the bacilli to the lymphoid tissue of the bowel, and the ulcer presents as one or more annular, circular or oval ulcers lying transversely². The annular or transverse orientation is due to the transversely running lymphatics. However, if the Payer’s patch alone is involved, the ulcer may be longitudinal³. The chronic inflammatory process of tuberculosis results in fibrosis and formation of adhesions to adjacent organs. This form protection against free perforation and perforations are rare in intestinal tuberculosis^{4,5}. The reported incidence of perforation in patients with intestinal tuberculosis varies from 1-11%.

But majority of these perforation (70-80%) are not truly perforations of tuberculous ulcers, but really are blow outs of small bowel, secondary to distension of the bowel due to strictures or adhesions just distal to the ulcer^{6,7}. Kakar *et al.*,⁸ has reported 22 cases acute small bowel perforations of tuberculous etiology of which 16 patients had a stricture immediately distal to the perforation. Both of our patients did not have any distal stricture or adhesions. Most of the reported perforations are contained due to adhesions to the surrounding structures and very few show free leakage into the peritoneal cavity. In a series of 28 tuberculous perforations, only 5 are free perforations as reported by Nagi *et al.*⁹. A higher incidence of tuberculous ulcer perforation has been reported during anti-tuberculous therapy^{10,11,12}. This has been attributed to reduction in the intestinal wall inflammation before a sufficient fibrous response occurs. Both of our patients had free perforation which is rare. Tuberculous intestinal perforations account for about 5-9% of all small bowel perforations in India and is the second commonest cause after typhoid fever^{13,14}. Clinical features of intestinal tuberculosis and tuberculous intestinal perforations are non-specific¹⁵ and a preoperative diagnosis is almost impossible unless the patient is on antituberculous therapy. Hence the operating surgeon should have a very high index of suspicion to make a diagnosis. The orientation of the perforation whether longitudinal or transverse cannot rule out tuberculous cause, because some of the tuberculous ulcer may be longitudinally oriented as pointed out earlier. Hence, excision of wound edges and submitting them to biopsy in all cases of intestinal perforation is the only way to diagnose without missing any case. Minimum treatment of any intestinal perforation is adequate excision of ulcer edges so that unhealthy, diseased tissue is removed and healthy, viable tissue should be approximated. When this principles are ignored, catastrophe can occur. Sweetman and Wise¹⁶ describe 20 cases of perforated tuberculous ulcer which were operated. Most of these had been treated with treated with simple suture and the mortality rate is 50%. These authors attribute the high mortality to the poor tissue in the ulcer edges and the presence of distal stenosis. There are no definitive guidelines for management of tuberculous perforation. Single perforation without much contamination can be managed by adequately excising the edges and suturing healthy, bleeding tissues. When the ulcers are multiple and the general condition of the patient is favourable, resection and anastomosis can be done. If the general condition is poor, exteriorization or ileostomy with mucus fistula has to be attempted.

Perforation with associated stricture requires resection of the involved segment of the bowel^{17,18}.

CONCLUSIONS

Preoperative or intraoperative diagnosis of tuberculous ulcer perforation is difficult. The surgeon should have a high index of suspicion. Biopsy of ALL intestinal perforations is mandatory to diagnose etiology and initiate appropriate treatment. Minimum treatment is adequate excision of wound edges and only healthy bleeding tissues should be approximated.

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