*ejpmr, 2024, 11(2), 259-261*

**SJIF Impact Factor 6.222**

**EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH**

www.ejpmr.com

Case Study

ISSN 2394-3211

EJPMR

# PRIMARY MULTIDRUG RESISTANCE ABDOMINAL TUBERCULOSIS

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**Article Received on 05/12/2023** **Article Revised on 25/12/2023** **Article Accepted on 15/01/2024**

## ABSTRACT

Tuberculosis has been a major cause of morbidity and mortality around the world. Abdominal Tuberculosis constitute 3%-5% of extra-pulmonary tuberculosis [EPTB] which accounts for approximately 20% of all cases with TB in immunocompetent individuals. The preferred site of involvement by TB is the ileocaecal region because of abundance of lymphoid tissue, relative physiologic stasis, and minimal digestive activity which permits greater contact time of acid-fast bacilli [AFB] with the mucosal surface of the ileocecum. Other sites of involvement that are common include the colon [where the segmental decrease of frequency occurs from the ascending colon to the rectum], jejunum, stomach, duodenum and oesophagus. Abdominal tuberculosis shows some nonspecific features of the disease which causes delay in the diagnosis and development of complications. This condition therefore is known as a great mimicker of other abdominal pathology. The treatment for multidrug resistance tuberculosis is complicated as most of the second line anti TB drugs are weak and toxic. Because of this reason MDR treatment generally takes 18-24 months. Patients of abdominal tuberculosis suffers from various sequelae such as intestinal obstruction, perforation, fistulas and fibrosis that increases morbidity if left untreated.

**KEYWORDS:** Abdominal tuberculosis, MDR-TB, Ileocecum.

## INTRODUCTION

Tuberculosis bacteria reach the gastrointestinal tract via haematogenous spread, ingestion of infected sputum, or direct spread from infected contiguous lymph nodes and fallopian tubes. The gross pathology is characterized by transverse ulcers, fibrosis, thickening and stricturing of the bowel wall, enlarged and matted mesenteric lymph nodes, omental thickening, and peritoneal tubercles.[1]

The diagnosis of abdominal TB is usually made by adequate radiological and histopathological studies. The methods of biopsy include endoscopic GI mucosal biopsy, image-guided percutaneous biopsy, endoscopic ultrasound guided biopsy, and surgical (open or laparoscopic) biopsy. The caseation necrosis in granulomas is the histologic hallmark of TB. In intestinal tuberculosis the granulomas are multiple, larger (more than 200 μm) and coalescent in mucosa and submucosa. Hematologic findings are nonspecific and include raised erythrocyte sedimentation rate, anaemia and hypoalbuminemia.[2]

The main causes of the spread of resistant TB are weak medical systems, amplification of resistance patterns through incorrect treatment, and transmission in communities and facilities. Although patients harbouring MDR and XDR strains present a formidable challenge for treatment, cure is often possible with early identification of resistance and use of a properly designed regimen.[3]

## CASE REPORT

A 28-year-old female presented with a 7-month history of generalized abdominal pain and lowgrade fever for 6 months. She had no history of weight loss, loss of appetite, night sweats, malaise, and fatigue. There was no history of abdominal trauma. Patient was non diabetic and non-hypertensive. There is no previous history of pulmonary tuberculosis. Three days before admission she reported of vomiting, abdominal distension, abdominal colic pain. Palpation showed tenderness in the right lower quadrant with an abdominal mass. Systemic examination did not reveal any other abnormalities. The initial laboratory investigations showed a normal haemogram, erythrocyte sedimentation rate of 80 mm/h, random plasma glucose 120 mg/dl, and positive tuberculin test (11 mm induration with 5 TU of purified protein derivative (PPD). Contrast-enhanced computed tomography (CT) of the abdomen demonstrated diffuse short segment of reduced distensibility with circumferential wall thickening with few ulcerations within it is seen in the terminal ileum and IC Junction, measuring approx. 1.4 cm in maximum thickness. Another short segment of Irregular wall thickening with

enhancement is seen in the mid ileal loop, measuring approx. 1,1cm in thickness and extending for a length of 2 cm. Gut loops proximal to it are prominent, measuring upto 2.9 cm. Another segment of wall thickening is seen in the mid ileal loop, measuring approx. 0.8cm in thickness intervening gut loops are normal stomach, duodenum, jejunum and colon are normal. Multiple enlarged lymph nodes are seen in the mesentery, the largest measuring approx. 2.7x1.3 cm in size. Enlarged lymph nodes are also seen at the porta and portocaval regions, the largest measuring approx. 3.8x1.5 cm in size. Upper GI endoscopy shows multiple duodenal erosions. Colonoscopy shows terminal ileal and IC valve ulcers. Histopathology report shows the features of necrotizing granulomatous inflammatory pathology favouring tuberculous aetiology- Ileocaecal valve ulcer. The gene expert (xPERT MTB/RIF) of ileocaecal valve tissue demonstrated mycobacterium tuberculosis with rifampicin resistance. The patient was started on longer oral MDR regime with bedaquilin, levofloxacin, linezolid, cycloserine, clofazimine.

## DISCUSSION

Abdominal tuberculosis is a type of extrapulmonary tuberculosis which involves the abdominal organs such as intestines, peritoneum and abdominal lymph nodes. It can either occur in isolation or along with a primary focus (such as the lungs) in patients with disseminated tuberculosis.[4]

Some features, including free or loculated ascites with thin-mobile septa, smooth peritoneal thickening and enhancement, misty mesentery with large lymph nodes, smudged omental involvement, and advanced ileocecal changes demonstrated by US, CT, or gastrointestinal series are deemed suggestive radiological findings. The diagnosis still requires a high index of suspicion, once the suggestive features have been demonstrated by imaging modalities.[5]

Grouping of anti-TB drugs and steps for designing longer MDR-TB regimen.

Group A- Include all three medicines
Levofloxacin Lfx or
Moxifloxacin Mfx
Bedaquiline Bdq
Linezolid Lzd.

Group B- Add one or both medicines
Clofazimine Cfz
Cycloserine Cs
Terizidone Trd.

Group C- Add to complete the regimen and when medicines from Group A and B cannot be used
Ethambutol E
Delamanid Dlm
Pyrazinamide Z
Imipenem-cilastatin or Ipm-Cln
Meropenem Mpm
Amikacin Am (OR Streptomycin S)
Ethionamide Eto or
Prothionamide Pto
p-aminosalicylic acid PAS.[6]

Diagnosis is challenging and is often delayed due to its non-specific presentation. GI TB responds well to standard anti-tuberculous drugs. Surgery is only required in cases that develop complications such as strictures or obstruction, not responding to medical therapy.[7]

Tuberculosis (TB) is still a major health problem worldwide. Especially, multidrug-resistant TB (MDR-TB), which is defined as TB that shows resistance to both isoniazid and rifampicin, is a barrier in the treatment of TB. Globally, approximately 3.4% of new TB patients and 20% of the patients with a history of previous treatment for TB were diagnosed with MDR-TB. The treatment of MDR-TB requires medications for a long duration (up to 20-24 months) with less effective and toxic second-line drugs and has unfavorable outcomes. However, treatment outcomes are expected to improve due to the introduction of a new agent (bedaquiline), repurposed drugs (linezolid, clofazimine, and cycloserine), and technological advancement in rapid drug sensitivity testing.[8]

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