CASE REPORT

Laparoscopic management of midgut malrotation and tuberculous peritonitis in an adolescent boy

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SUMMARY

An unusual case of miliary plastic peritonitis associated with midgut malrotation in an adolescent in whom various diagnostic investigations, medical management and staged initial laparoscopic diagnostic followed by therapeutic procedure was curative have been presented. Tuberculous miliary plastic peritonitis causes dense adhesions between bowel loops and prevents midgut volvulus in a pre-existing malrotation. Diagnosis can be a challenge and threshold for diagnostic laparoscopy should be low. Interval correction of malrotation by laparoscopy after complete resolution of tuberculosis is easy, safe and effective, and cosmetically more pleasing. Midgut malrotation is a congenital anomaly referring to either lack of or incomplete rotation of fetal axis around the axis of superior mesenteric artery during fetal development. Most patients present with bilious vomiting in the first month of life because of duodenal obstruction or a volvulus. This is an unusual case of midgut malrotation in association with tuberculous peritonitis in an adolescent boy.

BACKGROUND

Midgut malrotation is a congenital anomaly of rotation of the midgut and generally presents in early infancy. Its late presentation is usually connected with some associated pathology.¹ Midgut malrotation is a congenital anomaly referring to either lack of or incomplete rotation of fetal axis around the axis of superior mesenteric artery during fetal development. Most patients present with bilious vomiting in the first month of life because of duodenal obstruction or a volvulus. This is an unusual case of midgut malrotation in association with tuberculous peritonitis in an adolescent boy. We wish to describe a case of combined pathology of midgut malrotation associated with tuberculous miliary plastic peritonitis in an adolescent boy posing several diagnostic and therapeutic challenges. We describe the presentation and investigation of a rare case of midgut malrotation with concurrent abdominal tuberculosis. We emphasise the importance of considering primary peritonitis in this context, and review current methods of rapid diagnosis of abdominal tuberculosis, which in this instance led to timely appropriate treatment.

had recent weight loss and evening fever with night sweats.

On admission to our hospital he was febrile and weighed 66.6 kg. His chest was clear and had minimal ascites.

INVESTIGATIONS

His laboratory investigations were within normal limits except erythrocyte sedimentation rate being 30 mm/h. Mantoux test was negative. 'QuantiFERON TB gold' was positive making mycobacterium tuberculosis infection likely.

During investigation of the cause of his low-grade fever back home in his country, he was found to have small bilateral pleural effusion and ascites on abdominal ultrasound. Subsequent abdominal CT scan confirmed bilateral pleural effusion and ascites and also suggested that a 3.5 cm segment of small bowel was thickened and that the lumen was collapsed. Upper and lower gastrointestinal panendoscopy was normal and ascetic tap revealed a lymphocytic exudate.

Chest X-ray on admission showed collapse of the left lower lobe and raised left dome of diaphragm (figure 1). Abdominal ultrasound showed a small trace of ascetic fluid in the pelvis but no pleural effusion, lymphadenopathy or organomegaly. Barium follows through the evidence of malrotation with third and fourth parts of duodenum following an abnormal anterior path (figure 2) and a rather medially positioned caecum. There was no evidence of stricture or inflammatory change within the entire small bowel. No small bowel mucosal lesions were visualised on video capsule endoscopy.

DIFFERENTIAL DIAGNOSIS

Inflammatory bowel disease, gastrointestinal tuberculosis and disseminated carcinomatosis with midgut malrotation were considered in the differential diagnosis.

TREATMENT

The patient subsequently underwent diagnostic laparoscopy at which multiple peritoneal seedings were noted and the ascetic fluid seemed to be thick and turbid.

At laparoscopy, massive inflammatory change with peritoneal seeding of lesions with miliary tuberculosis with plastic peritonitis was observed. The omentum was inflamed, thick and adherent to the falciform ligament and all bowel loops were plastered to the abdomen posteriorly. It was very difficult to carry out Ladd's procedure. Biopsy of

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CASE PRESENTATION

A 15-year-old boy referred to us from overseas with symptoms of diarrhoea and non-bilious vomiting of 2 months duration which was followed by persistent low-grade fever and abdominal pain. He

Figure 1 Chest X-ray showing collapse of left lower lobe.

these miliary lesions revealed the presence of florid granulomatosis consistent with intra-abdominal tuberculosis. The ascitic fluid was taken for culture as well. Staining for acid-fast bacilli was negative for tissue and peritoneal fluid. Results of culture and PCR confirmed tuberculosis.

The patient has had a very dramatic response to antituberculosis therapy. He was given a combination of four antituberculous medicines for the first 2 months of treatment and thereafter prescribed at least a further 4 months of rifampicin and isoniazid. He was given a short course of steroids in an attempt to reduce intraperitoneal adhesion formation. He was started on prednisolone 60 mg once daily for 1 month reducing over the next 3 weeks to 20 mg once daily for a week, 10 mg once for a week and then 5 mg once daily for 1 week before stopping.

It was advised to screen the family back home on return. The patient had monthly liver function tests and full blood count which were normal. Follow-up abdominal ultrasound in 2 months showed resolution of the ascites and he underwent

laparoscopic correction of midgut malrotation with Ladd's procedure when the adhesions were minimal.

OUTCOME AND FOLLOW-UP

The patient's postoperative period was uneventful and he made a smooth recovery. At 6 years follow-up, he is asymptomatic and thriving well. He is under long-term follow-up at a local hospital overseas and his renal, hepatic and bone profiles are all normal.

DISCUSSION

Midgut malrotation has protean manifestations and typically presents in early infancy. The clinical presentation in later life is being reported with delayed presentation and non-specific features. Late presentation poses difficulties in the diagnosis and at times in treatment as demonstrated in our case.²⁻⁷ The presentation in our case was incidental to investigation of the ongoing abdominal symptoms and the treatment was impossible in the wake of extensive adhesive plastic tuberculous peritonitis.

Laparoscopy is always the best method for definitive and rapid diagnosis of tuberculous peritonitis: exudative ascites predominantly of lymphocytes, miliary nodules with peritoneal biopsies, adhesions between the peritoneum and organs and congestion is seen in most cases. Laparoscopically guided peritoneal biopsies detect caseating granulomas in 87% of cases and laparoscopic appearance of the peritoneum mimicking a carcinoma in 15% of cases. Miliary nodules and adhesions are the more frequent appearances in the peritoneal cavity in tuberculous peritonitis.⁸ Peritoneal tuberculosis and carcinomatosis are the most frequent aetiologies of exudative ascites and require rapid diagnosis and treatment.⁹ Aetiological diagnosis of exudative ascites remains difficult to establish. Peritoneal biopsies under video laparoscopy are currently the 'gold standard' for diagnosis.¹⁰

It is difficult to have hard laboratory evidence of the tuberculosis and soft indicators with newer investigations together with a therapeutic trial remains mainstay in the management of these patients. In the face of associated midgut malrotation, correction with active disease is fraught with dangers of bowel perforation and fistula formations and the remedy may be worse than the disease itself.

Figure 2 Upper gastrointestinal contrast showing midgut malrotation.

Moreover, steroids help in reducing the adhesions and fibrosis with treatment and may predispose to the malrotation complications of midgut volvulus at a later date. It is, therefore, advisable to get a check laparoscopy once the treatment of tuberculosis is nearing completion. At the same time the malrotation correction can be performed therapeutically at that stage.

Interestingly, dense adhesions between the bowel loops and adhesive peritonitis prevented midgut volvulus in pre-existing

malrotation thereby leading to late presentation during adolescence. Owing to atypical symptoms of malrotation in this age group, a high degree of clinical suspicion with a battery of clinical investigations is warranted.

Contributors All authors were involved in the clinical management of this patient (RVP, PJ, AZ and PDC) and in preparation, editing, appraising and finalising this manuscript (RVP, PJ, AZ and PDC).

Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ Association of midgut malrotation congenital anomaly and abdominal tuberculosis is a rare combination in the same patient.
- ▶ Tuberculous miliary plastic peritonitis causes dense adhesions between bowel loops and this adhesive peritonitis prevents midgut volvulus in a pre-existing malrotation.
- ▶ Diagnosis of abdominal tuberculosis can be a challenge and with availability of laparoscopy, threshold for diagnostic laparoscopy should be low.
- ▶ There is a temptation of malrotation correction at first laparoscopy but associated dense adhesions cause more bleeding and more readhesions and they get better with antituberculous treatment and addition of short-term steroid enhances its effect.
- ▶ Interval correction of malrotation by laparoscopy after complete resolution of tuberculosis is easy, safe and effective, and cosmetically more pleasing.

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