# Pediatric Abdominal Tuberculosis

# 24

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## Abbreviation

| | |
|---|---|
| ADA | Adenosine deaminase |
| AFB | Acid-fast bacilli |
| ATB | Abdominal tuberculosis |
| ATT | anti-tubercular therapy |
| CBNAAT | cartridge-based nucleic acid amplification test |
| CD | Crohn's disease |
| FDC | Fixed drug combinations |
| FNAC | Fine needle aspiration cytology |
| GI | gastrointestinal |
| ITB | Intestinal tuberculosis |
| SAAG | serum ascitic albumin gradient |
| TB | Tuberculosis |

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V. Sharma (ed.), *Tuberculosis of the Gastrointestinal system*,
https://doi.org/10.1007/978-981-16-9053-2_24

**Key Points**

1. Pediatric abdominal tuberculosis is primarily a disease of the developing world.
2. It can have a varied presentation, frequently mimicking other diseases. Abdominal pain, fever, and weight loss are the triad of symptoms most commonly seen.
3. Multiple abdominal sites are frequently involved in children.
4. Establishing the diagnosis can be challenging. A unique aspect about childhood tuberculosis is the history of contact with an infected person.
5. Doses of anti-tubercular drugs per mg body weight is higher in children as compared to adults. Treatment duration for pediatric abdominal TB is usually 9–12 months.

## 24.1 Introduction

Children have been neglected in the fight against tuberculosis (TB) for years. Of the ten million cases of tuberculosis recorded worldwide in the year 2018, an estimated 1.1 million were children, of which an estimated 205,000 children died [1]. An alarming 96% of these deaths occurred among children who were untreated which highlights the fact that tuberculosis in children is frequently unrecognized [2]. Apart from this, children also represent a significant but underappreciated proportion of the multi-drug-resistant TB burden with an estimated 30,000 children each year [3]. India has by far the highest burden of tuberculosis in children, which is not surprising considering its large size, demographic composition, and moderate tuberculosis prevalence [4].

Pulmonary tuberculosis is overall the commonest site of tubercular involvement in children, and abdominal tuberculosis has been reported to comprise 0.3–4% of all cases of tuberculosis. [5–7] This is likely an underestimate. An autopsy study of children who died from TB showed that 15.7% children had abdominal involvement. [8] Abdominal tuberculosis is relatively rare in children when compared to adults. It has been reported mainly from developing countries, especially the Indian subcontinent and is rare in developed nations. Delisle et al. in a review spanning 70 years (1946–2014) found only a total of 45 cases reported in the literature form Europe, North America, New Zealand and Australia. Even among these all patients except one were from high-risk populations, including aboriginals, foreign born and those with a history of recent travel to endemic countries. [8].

Abdominal TB occurs in four forms: tuberculosis of the abdominal lymph nodes, peritoneal tuberculosis, gastrointestinal tuberculosis and visceral tuberculosis and may occur as a result of hematogenous spread from a primary complex elsewhere, by the ingestion of the tubercle bacilli or by contiguous extension from other adjacent organs.

The protean clinical manifestations make it a challenge for the physicians to establish the diagnosis, even more so in the pediatric age group where a child's

inability to define their problems accurately contribute to this challenge. It is important to remember that abdominal TB in a child is not like TB in a miniature adult. There are many important differences in the disease presentation, site and type of the disease, diagnostic evaluation and treatment, which have been highlighted in the next few sections.

## 24.2 Clinical Features

Abdominal TB usually presents in childhood in the age—group of 9–14 years and does not have a predilection to any gender. [7, 9–14] (Table 24.1) It is often initially confused with other conditions, and the diagnosis is usually delayed. A median delay of 4–6 months in diagnosis has been reported in literature even from the endemic areas where there is a general awareness of this condition. The clinical spectrum depends on the site of involvement. It ranges from nonspecific symptoms to those that may mimic Crohn's disease. Overall, abdominal pain (61.5–81%), fever (31–90%), and weight loss (40–74%) are the most frequent findings on presentation [7, 9–14]. In patients of the pediatric age group, loss of body weight is defined as >5% weight loss in the preceding 3 months. In a child presenting with these triad of symptoms, one should suspect abdominal TB. Table 24.1 includes the clinical features noted in the major series of patients with pediatric abdominal tuberculosis.

In 33–37% patients, extra-abdominal symptoms including respiratory (cough, breathlessness, etc.), neurological (headache, seizures), genitourinary symptoms, cervical/axillary lymphadenopathy, and dermatological manifestations (erythema nodosum) may be present.

### 24.2.1 Disease Distribution

There is considerable variability in the distribution of the disease within the abdomen reported from different centers. The reason for this variability includes differences in clinical setting (secondary care center vs tertiary referral center) and the specialties (pediatrics vs pediatric gastroenterology vs pediatric surgeon) from which the data is published. Overall, the spectrum of disease in children is different from adults, in whom peritoneal and lymph nodal involvement is more common than gastrointestinal disease [9].

In the largest series of cases of pediatric abdominal TB reported from Chandigarh, lymph nodal involvement was overall the commonest [12] (Table 24.2). Involvement of multiple abdominal sites is common. In the series from Chandigarh, a substantial number (54%) had involvement of multiple sites with a combination of intestinal and lymph nodal involvement being the commonest. In an autopsy series of 24 children with ATB, it was found that, in 82% (14/17) cases of intestinal TB, the intestinal lesion extended to the peritoneum, while 2/7 children with presumed isolated peritoneal TB had evidence of disease arising from other abdominal sites,

**Table 24.1** Clinical features of children with abdominal tuberculosis

| | Turkey ($n = 35$) | Taiwan ($n = 10$) | India Lucknow ($n = 38$) | India Chandigarh ($n = 218$) | India Ajmer ($n = 125$) | Tunisia ($n = 13$) | Europe, North America, NZ & Australia ($n = 45$) |
|---|---|---|---|---|---|---|---|
| Mean age | 9.7 ± 4.3 years | 14.7 years | 11 (4–16) Years | 10 (0.25–12) Years | 9.5 (2–14) Years | 9.8 (7–14) Years | 5–9 years–3<br>10–13 years–5<br>>14 years–11 |
| Gender (M) | 54.3% | 40% | 45% | 54% | 40% | 23% | 64% |
| Duration of symptoms | 109 days (10 days– 3 years) | – | 4.5 mths (15 days–5 years) | 4 mths (0.5–36) months | 54 day (7 days–9 months) | 4.2 mths (4 days–1 year) | – |
| Fever | 31.4% | 90% | 66% | 76% | 80% | 31% | 71% |
| Weight loss | 40% | 72% | 71% | 74% | 58% | 54% | 68% |
| Anorexia | – | – | 76% | 66% | 54% | 54% | 27% |
| Abdominal pain | 62.9% | 80% | 66% | 81% | 80% | 61.5% | 76% |
| Abdominal mass | – | – | 11% | 6% | 32% | 15.4% | 32% |
| Diarrhea | – | 0% | 18% | 21% | 14% | – | 29% |
| Intestinal obstruction | – | 0% | 21% | 16% | 36% | 7.7% | – |
| Abdominal distension | 45.7 | – | 63% | 43% | 38% | 61.5% | – |
| Doughy abdomen | – | – | – | 27% | 12% | – | – |
| Hepatomegaly | 16.1% | – | 42% | 32% | 6% | 7.7% | – |
| Splenomegaly | 16.1% | – | 21% | 15% | 4% | 16% | – |
| Ascites | 74.3% | 40% | 47% | 26% | 44% | 69% | 68% |
| Extra-gastrointestinal symptoms | 31.4% | 100% | 21% | 21% | 16% | 16% | 37% |
| Mortality | 2.8% | 10% | 8% | – | 0% | 0% | 9% |

**Table 24.2** Distribution of the disease within the abdominal cavity

| | Turkey ($n = 35$) | India Lucknow ($n = 38$) | India Chandigarh ($n = 218$) | India Ajmer ($n = 125$) | Tunisia ($n = 13$) | Developed ($n = 45$) |
|---|---|---|---|---|---|---|
| Peritonitis | 83% | 24% (39.4%) | 10.1% (35.3%)* | 44% | 38.4% | 42% |
| Gastro-intestinal | 14.3% | 16% (39.4%) | 16% (54.5%) | 20% | – | 49% |
| Lymph nodal | – | 16% (39.4%) | 17.9% (68.8%) | 36% | 7.7% | – |
| Visceral | – | 5% | 2% (8.2%) | – | – | – |
| Multiple sites | – | 32% | 54% | – | 54% | – |

(*)when those with involvement in multiple sites are also included

reiterating the fact that coexistence of TB at multiple sites of the abdomen is common in children [8]. Lymph nodal involvement varies from 7.7 to 68% of children with abdominal TB. The most commonly involved lymph nodes are the mesenteric nodes, peri-pancreatic, retroperitoneal, and omental nodes, and those along the celiac axis. However, it should be remembered that the presence of enlarged mesenteric lymph nodes alone does not mean that the child has abdominal TB as it is a common, non-specific finding in children. The diagnosis of TB should only be considered in the appropriate clinical context. The sonographic finding of oval and elongated lymph nodes with a short-axis diameter up to 10 mm in children should be considered a normal finding and should not be misdiagnosed as an early manifestation of tuberculosis [15].

Among children with intestinal TB (ITB), ileocecal involvement is the commonest and is seen in up to three-fourth of all children with ITB. Increased physiological stasis with a maximal period of contact, abundant lymphoid tissue, and minimal digestive activity in this region are reasons for this occurrence. The left-sided colon may be involved in ~40%. Clinical symptoms specific to ITB include abdominal pain (60%), diarrhea (40%), intestinal obstruction (20%), and blood in stools (10%) [16]. Growth failure is seen in up to 60%. The differentiation between ITB and Crohn's disease is often a challenge as microbiologic confirmation of ITB is possible only in around 40% cases. In a study by Singh et al., 20 children with ITB were compared with 23 children with CD. Features of subacute intestinal obstruction (20% vs 0%), ascites (30% vs 0%), and isolated ileocecal involvement (40% vs 8.7%) favored ITB. [16] The presence of blood in stool and left-sided colonic involvement were independent predictors of CD. On colonoscopy, the presence of deep, longitudinal ulcers with involvement of multiple segments is more suggestive of CD than TB. Perianal disease in children is seen exclusively in children with CD. This is in contrast to adults, where even though perianal disease is more common in CD it has been reported even in ITB. [16].

At times even after taking all the clinical, endoscopic, and biochemical parameters into account, it is not possible to conclusively differentiate between the two diseases. A therapeutic trial of anti-tubercular therapy (ATT) is safer than starting steroid in children in whom there is a diagnostic dilemma and up to one-third of children with CD receive ATT before a diagnosis of CD is made.

Overall, peritoneal involvement is reported in 35.3–83% of children with abdominal tuberculosis. Similar to adults, it includes the wet type (which is characterized by ascites formation), a dry fibrotic type (associated with a thickened peritoneum, adhesions, and omental thickening with little or no ascites), a mixed type (which is a combination of both), and abdominal cocoon (characterized by the presence of a membranous sac around the intestinal loops). Children with the wet type of peritoneal TB generally present with progressive diffuse or localized abdominal distension and pain in association with constitutional symptoms like fever, anorexia, and weight loss. Children with the dry fibrotic type or abdominal cocoon often present with features of abdominal pain and distension, vomiting, and constipation suggesting intestinal obstruction. In a series of 110 children with abdominal TB presenting to a pediatric surgical department with features of intestinal obstruction, an abdominal lump, or entero-umbilical fistula, 91% ($n$ = 100) were found to have adhesive (dry fibrotic) peritonitis. Out of these 100 patients, 23 also had mesenteric lymph nodal involvement [17]. Abdominal cocoon is also known as subacute encapsulating peritonitis which is a known cause of intestinal obstruction although it is rare in children. In a small series of 17 children all presented with features of small bowel obstruction [18].

Visceral (hepatic, splenic) tuberculosis is usually associated with an active pulmonary disease or miliary tuberculosis and isolated involvement (<2%) is rare. Fever, weight loss, right or left hypochondriac pain, and hepatosplenomegaly are the most frequently observed clinical findings. Jaundice is a very rare manifestation of tuberculous liver involvement and may be caused by extra- or intrahepatic obstruction.

Tubercular involvement of the stomach, duodenum, and esophagus in children is hitherto rare [12].

### 24.2.2 Congenital TB

A rare form of tuberculosis in children is congenital TB. By 2005, only ~400 cases had been reported in the literature [19]. TB during pregnancy may lead to infection of the placenta or the genital tract, which may then be transmitted to the fetus either hematogenously from the placenta to the umbilical vein or by aspiration or ingestion of contaminated amniotic fluid. In infants in whom the mode of acquisition is transplacental, the primary complex develops in the liver, while in those in whom it occurs by the ingestion of infected material the primary is in the gastrointestinal (GI) tract. As it primarily involves the liver and GI tract, i.e., has abdominal involvement, it has been included in this chapter.

The Cantwell criteria is used for the diagnosis and is as follows [20]:

Proven tuberculosis lesions in the infant plus one of the following:

1. Lesions occurring in the first week of life
2. A primary hepatic complex

3. Maternal genital tract or placental tuberculosis
4. Exclusion of postnatal transmission by thorough investigation of contacts

The average age at the onset of congenital tuberculosis is 24 days (range, 1–84 days). The symptoms are often nonspecific and include fever, irritability, poor feeding, hepatosplenomegaly, and respiratory symptoms. It is particularly hard to diagnose because it is seldom distinguishable from other neonatal and congenital infections seen in this age group. A majority (60–70%) of mothers of patients have no symptoms of TB, which makes this condition even difficult to recognize. Most mothers are diagnosed with tuberculosis only after the child has been diagnosed with tuberculosis [20].

A high index of suspicion is needed for the diagnosis of congenital TB. Tuberculin testing is generally normal. Abdominal imaging (ultrasonography, CT) is a useful tool in diagnosing lesions in the liver and spleen. The sensitivity of liver biopsy for the diagnosis of congenital tuberculosis is 100%; however, it is an invasive test [21]. One may first try isolating the bacilli from other samples such as gastric aspirates, ascitic fluid (if present), pleural fluid (if present), and/or cerebrospinal fluid as the disease is often disseminated. A proportion (~50%) of patients may have an abnormal chest radiograph which may suggest the diagnosis.

Historically, the prognosis of congenital TB has been poor with up to 50% mortality seen. A delay in diagnosis being the most important reason for it [22]. However, with timely diagnosis and initiation of treatment, outcomes have improved.

## 24.3 Diagnosis

Establishing the diagnosis of abdominal tuberculosis can be challenging in children. The paucibacillary nature of the disease in children, difficulties in obtaining adequate samples for analysis, and the technical problems in obtaining proper imaging are some of the things that contribute to this difficulty.

Diagnostic tests can be divided into two categories:

1. *Test for definitive diagnosis*
   (a) Demonstration of acid-fast bacilli on smear or culture

   It is the most definitive method to diagnose tuberculosis. Samples are obtained depending on the site of involvement.
   - Ultrasound-guided fine-needle aspiration cytology (FNAC) of enlarged lymph nodes, focal lesions in the liver and spleen, omentum, and thickened bowel may have a yield of 45–58% [23, 24].
   - Endoscopic biopsies from the lesions has an AFB detection rate of 36.1%. Ileocolonoscopy in younger children can be challenging and can be done only in tertiary centers where appropriate expertise and equipment (pediatric colonoscopies) are available.
   - Demonstration of AFB in ascitic fluid is seen in only ~10% children [25]. Ascites with a low serum ascitic albumin gradient (SAAG) and lymphocytic predominance is a characteristic of tuberculosis.

- The rates of bacteriological confirmation have remained at ~50% from 1976 to 2019. It appears that the advances in the diagnostic modalities have not translated into higher microbiological yields [11].

(b) Histopathology showing tubercular granuloma
- Tissue obtained during endoscopy may demonstrate the presence of caseating granulomas on histology in ~60% of children with intestinal TB.
- Laparoscopy is an invaluable tool when the diagnosis is unclear, with a pick-up rate of 85%. Thickened peritoneum with miliary yellowish white tubercles with or without adhesions may be seen. However, it is invasive and is generally done only when the imaging features raise doubts about the possibility of a malignancy, since a lymphoma in a child can mimic TB in every way or there continue to be progressive symptoms even after initiation of anti-tubercular therapy, i.e., failure of a therapeutic trial.
- In children with hepatic involvement, a liver biopsy may help in confirming the diagnosis.

(c) Cartridge-based nucleic acid assay (CBNAAT).

In all children with suspected abdominal TB, the appropriate specimen from the involved site should be collected and subjected to CBNAAT. It is rapid and fully automated and is based on polymerase chain reaction that detects deoxyribonucleic acid directly from the clinical specimens. It can also detect rpoB gene mutations that confer rifampicin resistance. In adults it has a pooled sensitivity and specificity of 23% (95% CI16–32%) and 100% (95% CI 52–100%), respectively, for intestinal TB [26]. Pediatric data are lacking.

2. *Investigations that support the diagnosis*

(a) Radiology

An abnormal imaging plays a pivotal role in the diagnosis of abdominal TB and has the highest yield. However, caution should be exercised in the interpretation of the radiological findings and a diagnosis of TB should only be considered in the appropriate clinical context. This is especially true in children with isolated mesenteric lymph node enlargement where unwarranted treatment is rife.

(b) Chest X-ray—It is a useful adjunct in establishing the diagnosis of tuberculosis. A third of patients may not have pulmonary symptoms and are detected to have pulmonary involvement incidentally on a chest radiograph. Routine chest X-ray is a part of the evaluation in children with suspected ATB.

(c) Ultrasound abdomen/computed tomography—The most common findings in children with abdominal TB are enlarged intra-abdominal lymph nodes [27]. The upper para-aortic and mesenteric groups are more often involved in TB than in lymphoma. Presence of large and/or conglomerate lymph nodes with central hypodensity is suggestive of TB. However, they may be non-enhancing or show areas of calcification. Calcification does not imply inactivity. Ascites (septate ascites), bowel-wall thickening, omental thickening "caking," clumped bowel loops, and solid organ involvement (tubercular abscess, calcifications) are other features seen. CT is superior to US because

of the ability to identify all the features in a single study. This is especially so in the detection of caseating lymph nodes, high-density ascites, and bowel-wall thickening. Bowel thickening is considered less common in children than in adults.

(d) Demonstration of AFB from extra-gastrointestinal sites.

(e) In children with concomitant pulmonary involvement, gastric lavage samples have a detection rate of 40–92% [28]. The specimen is collected after 4–6 hours of fasting. Induced sputum by 3% nebulized saline is another alternative. Whatever method one chooses to use, one needs to collect at least two, preferably three, samples.

In children with concomitant peripheral lymphadenopathy, fine-needle aspiration cytology (FNAC) specimen may demonstrate AFB in 20–70% cases [29].

(f) Adenosine deaminase

ADA activity in the peritoneal fluid has been proved to be a simple and reliable method for early diagnosis of tuberculous peritonitis. Sensitivity and specificity levels over 90% have been reported [30, 31]. Similar observations about the utility of ADA (cutoff 36–40 IU/L) in the diagnosis of tubercular peritonitis have also been made,

(g) Mantoux test

Tuberculin skin tests (Mantoux test) are examined 48–72 h after the intradermal injection of five tuberculin units of a purified protein derivative. Positive results have been seen in 17–90% children with abdominal TB.

(h) Contact

A unique aspect about childhood tuberculosis is the hunt for an index case, i.e., history of contact with an infected person. It has been reported in up to 21–65% children with abdominal TB [11, 32]. Risk of acquiring TB in the child is directly proportional to the number of bacilli to which he or she is exposed. Contagiousness is generally limited to subjects with pulmonary disease and is greater among the patients with positive sputum microscopy test results. Subjects with cavitating TB and cough with expectoration are more bacilliferous and contagious.

(i) Response to a therapeutic trial

At times when the diagnosis cannot be established even after exhausting all investigative modalities, one may have to give anti-tubercular drugs and assess the response. The follow-up of these patients is of extreme importance, and demonstration of an objective response to therapy secures the diagnosis. In children with a diagnostic confusion between TB and CD and who were initiated on ATT, the demonstration of endoscopic healing of the ulcers at the end of ATT helps in confirming the diagnosis [12, 16]. A mere subjective response, i.e., the resolution of symptoms, is not enough as some patients with CD may also have symptomatic improvement. The diagnostic yield of all the investigations have been summarised in Table 24.3.

**Table 24.3** Yield of various investigations for the diagnosis of pediatric abdominal tuberculosis

| | Taiwan | India–Lucknow | India- Chandigarh $n = 218$ | | | | Tunisia | Developed |
|---|---|---|---|---|---|---|---|---|
| | $n = 10$ | $n = 35$ | GI | L | P | V | $n = 13$ | $n = 45$ |
| Bacteriological ¶ | 40% | 47% | 36.1% | 29.3% | 29.8% | 44.4% | 23% | 73% |
| Histopathology | 50% | 19% ± | 54.7% | 45.3% | 72.2% | 72% | 46% | 18% |
| Radiology | 100% (60%)§ | 94.5% | 81.5% | 89.3% | 92.2% | 100% | 100% | – |
| ADA | – | 82% | 53.8% | | | | – | – . |
| Abnormal chest X-ray | 90% | 16% | 25.2% | | | | 15.3% | 24% |
| TB contact | 60% | 21% | 32.5% | | | | 7% | |
| Mantoux test | 17% | 45% | 64.7% | 68.7% | 67.5% | 72% | 61% | 90% |

± in these patients histopathology alone helped in the diagnosis
¶ AFB on smear, culture, or polymerase chain reaction, CBNAAT
§- Ultrasound only
*GI* gastrointestinal, *L* lymph node, *P* Peritoneal, *V* Visceral

In children with peritoneal, visceral, or lymph nodal TB, a repeat imaging at the end of therapy showing an improvement/resolution of the imaging features is warranted for an objective assessment. It is suggested to follow up these patients for ~2 years after completion of therapy to look for a relapse of symptoms.

Based on these investigations, the diagnosis of tuberculosis can be

- *Definitive* [demonstration of AFB (in a tissue smear, histopathology, or culture), positive CBNAAT or caseous granulomas on histopathology, and/or unequivocal response to ATT].
- *Probable* [compatible clinical features + radiology features/positive Mantoux/history of contact/raised ADA/demonstration of AFB from extra-gastrointestinal sites].

## 24.4 Treatment

In recent years, the pharmacokinetics of all first-line TB drugs have been revisited and there has been an upward revision of the dosages needed for children [33]. This is because the pharmacokinetic data suggest higher dosages for maximizing the area under curve above the minimum inhibitory concentration. Also, pediatric patients show a rapid metabolism of isoniazid and require a higher mg/kg body weight dose when compared to adults. The current dosages as per the revised RNTCP guidelines have been tabulated in Table 24.4. Fixed drug combinations (FDC) that incorporate multi-drug therapy are preferred due to safe and simplified treatment and to do away with the possibility of missing one or more of the combination drugs. The FDCs consist of four weight bands for adolescents and adults (25 kg to >70 kg) and six weight bands in children (4 to 39 kg). Dispersible tablets are available for children. For a newly diagnosed child with abdominal TB, intensive phase consists of 8 weeks of

**Table 24.4** Dosage for anti-tubercular dugs in children

| | Range (mg/kg/d) | Average (mg/kg/d) | Maximum dose (mg) |
|---|---|---|---|
| Rifampicin | 10–20 | 15 | 600 mg |
| Isoniazid | 7–15 | 10 | 300 mg |
| Pyrazinamide | 30–40 | 35 | 2000 mg |
| Ethambutol | 15–25 | 20 | 1500 mg |

isoniazid, rifampicin, pyrazinamide, and ethambutol. This is followed by 16–40 weeks of three drugs isoniazid, rifampicin, and ethambutol as a continuation phase.

The optimal duration of treatment for pediatric ATB is unclear. A shorter duration of treatment increases compliance and decreases the risk of toxicity of the drugs. However, it may also pose a risk for relapses. A review of three randomized controlled trials comprising 328 adult participants found a 6-month regimen (2-month intensive phase, 4-month continuation phase) to be efficacious, but whether it can be extrapolated to children or not is debatable [34]. Apart from including only adults, two of the three studies included only those who had intestinal tuberculosis, which may not be applicable to children where the majority have involvement of multiple abdominal sites. Moreover, the authors of the systemic review have conceded that the quality of evidence regarding the relapse estimate is very low, which is a cause for concern. Hence, more data are required before recommendations regarding the optimal treatment duration can be made. Till then treatment duration should be tailored according to the disease extent, treatment response, and treating physician's experience in managing such patients. Most centers treat for a total duration of 9–12 months. Anti-tubercular therapy-induced hepatotoxicity occurs less frequently in children than adults; it is by no means uncommon. It contributes to 4–8% and 8.7% pediatric cases of drug-induced liver injury in the West and India, respectively. It is important to keep it in the back of one's mind when evaluating a child on follow-up. [35].

Surgery is absolutely indicated when there is intestinal perforation. It constitutes ~15% of all children who present with perforating peritonitis to a tertiary center [36]. Partial intestinal obstruction, adhesive peritonitis, and entero-cutaneous fistulas are relative indications. In such children, it is prudent to first give a trial of ATT as a proportion of children may respond to it alone. In children with a tubercular enteric stricture, endoscopic dilatation may be attempted.

There has been a paradigm shift in the management, and the frequency of children who require surgery has gone down from 85–100% in the 1990s to 4% now.

## 24.5 Outcome

Most children respond well to therapy. Emergence of drug-resistant abdominal TB has recently been reported and should be considered in children who do not show an optimal response to anti-tubercular therapy. In a study from Mumbai, 12.5% children with abdominal TB had drug-resistant TB [37]. With a timely diagnosis,

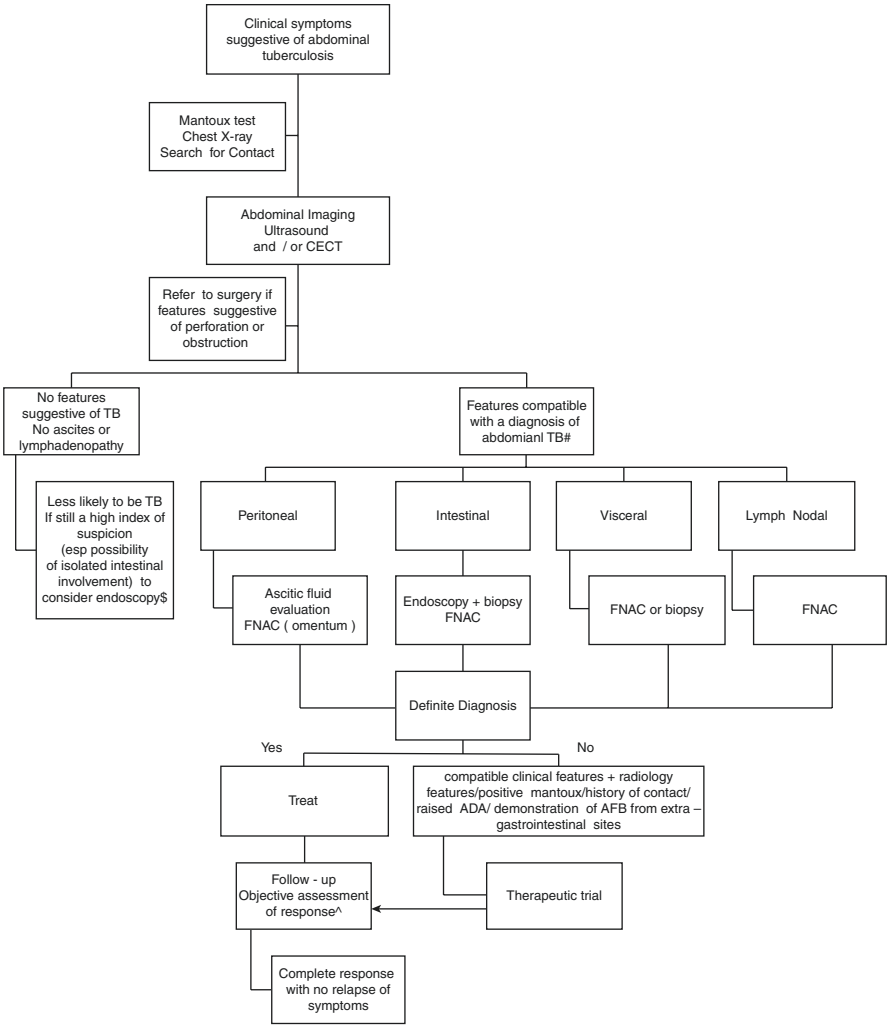


**Fig. 24.1** Algorithmic approach to pediatric abdominal tuberculosis. #—ascites (free or loculated), high density (on CT) with or without multiple, thin, complete, and incomplete septae; (•) lymphadenopathy (mesenteric, peri-pancreatic, periportal, and para-aortic groups of lymph nodes) seen as conglomerate masses. (•) and/or as scattered enlarged nodes with hypoechoic or anechoic centers (on USG)/peripheral rim enhancement, non-homogenous enhancement (on CT); (•) bowel-wall thickening, peritoneal thickening and nodularity, adhesions, mesenteric thickening, and irregular soft tissue densities in the omental area; and (•) tiny, low-density foci or multiple low-attenuation, 1–3 cm round lesions scattered in the liver and/or spleen. $—Isolated intestinal involvement has the lowest diagnostic yield on imaging. ^—If the child continues to have symptoms, then the diagnosis needs to be revisited. If previous tissue diagnosis has been inconclusive, one may consider re-obtaining/ repeating it . At this point, one may consider a laparoscopy to obtain better tissue samples for diagnostic evaluation. In children in whom the diagnosis is definite, the possibility of drug resistance has to be considered. Some children with tubercular intestinal strictures may continue to have pain even after ATT in spite of healing of lesions. This needs to be tackled endoscopically/ surgically. *CECT* Contrast-enhanced computed tomography, *FNAC* Fine-needle aspiration cytology, *ADA* Adenosine deaminase

mortality is rare; however, in children in whom the diagnosis is considerably delayed, up to 10% mortality has been reported.

An algorithm for approaching a child with suspected abdominal TB has been given in Fig. 24.1.

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