

ascites and had milliary mottling on chest X-ray. In such a setting antitubercular drug therapy was fully justified without subjecting this case to unnecessary diagnostic laparotomy. Diagnosing abdominal tuberculosis by definitive criteria, i.e., tissue demonstration of caseation granuloma and/or AFB positivity is not always possible; instead one may go by probable criteria(2,3).

Further the value of ascitic fluid adenosine deaminase (ADA) determination has been highlighted in the diagnosis of tubercular peritonitis. A raised level of ascitic fluid ADA has a very high sensitivity (98-100%) and specificity (96-97%) in diagnosing tubercular peritonitis(2). Therefore, antitubercular drug therapy in an appropriate clinical setting, exudative ascites with high ascitic fluid ADA level will obviate diagnostic laparotomy and its attendant morbidity and mortality in patients with tubercular peritonitis.

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Reply

The child had features of acute abdomen with toxic paralytic ileus. Analysis of ascitic fluid was confirmatory of spontaneous primary peritonitis (SPP) due to *Staphylococcus aureus*. Histopathology of mesentric lymph nodes and peritoneal tissue confirmed tuberculosis as mentioned in the report.

Though the patient had clinical features to suggest disseminated tuberculosis, factors like acute and rapidly progressing illness, presence of SPP with paralytic ileus, and failure to respond to conservative therapy even after 72 hours, prompted us to subject the child to exploratory laprotomy. The procedure served the main purpose of performing peritoneal toiletting and to carry out tissue diagnosis. Surgical exploration in SPP is indicated if multiple organisms are seen on Gram stain, free air is demonstrated on abdominal roentgenogram, or after 48 hours of parenteral antibiotics either the child's condition deteriorates or the physical findings persist and show localization(1).

The priority for treatment in this patient was for SPP. Anti tubercular therapy alone would not have benefited the child. We have not generalized the statement that diagnosis of tubercular peritonitis is by exploratory laprotomy. It is only in those cases where the onset is acute mimicking, acute abdomen and other positive evidence for tuberculosis is not present.

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Generalized BCG Tuberculosis

Fatal generalized BCG tuberculosis in association with BCG vaccination is extremely rare. Congenital agammaglobulinemia was used to be considered as one of the conditions associated with disseminated BCG infection(1). Immune system abnormalities associated with dissemination of live vaccines include thymocytic deficiency without agammaglobulinemia or hypogammaglobulinemia, and leukocyte and monocyte deficiency disease(2). We report a case of generalized BCG tuberculosis.

A 3-month-old boy was brought to this hospital with swelling on the left side of neck about 20 days after receiving BCG vaccination. The patient had fever, irritability, poor feeding and excessive crying for the last 2 months. There was no history of contact with tuberculosis and he was breastfed. The antenatal, natal and postnatal history was insignificant. On examination BCG scar was present about 2.5 cm from the acromion tip. Eight lymph nodes of the left upper cervical group were palpable, measuring 0.5-2 cm, they were

firm, mobile, not-matted and non-tender. Abdominal examination showed a hepatomegaly of 5 cm, firm, smooth, non-tender and splenomegaly of 6 cm, firm with round margin and non-tender. The hemoglobin, total and differential counts were within normal limits; SGPT was 292 IU/l. The chest roentgenogram showed bilateral miliary mottling (*Fig.*). Lymph node biopsy revealed tubercular granuloma consisting of areas of caseation surrounded by epitheloid cells, lymphocytes and Langhans giant cells. The child was treated with rifampicin 10 mg/kg, INH 10 mg/kg and ethambutol 20 mg/kg along with steroid 1

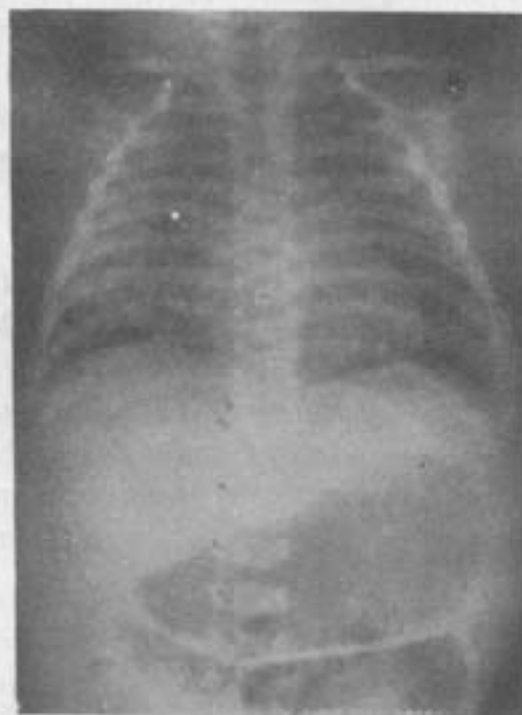


Fig. Chest X-ray showing diffuse miliary mottling suggestive of miliary tuberculosis.

mg/kg. He came back after about 5 months with progressive enlargement of swelling in the neck and abscess was drained with excision of some caseating lymph nodes.

Progressive BCG infection is extremely