



The Brazilian Journal of INFECTIOUS DISEASES

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Letter to the Editor

Primary pneumococcal peritonitis in an immunocompetent child

Dear editor,

Primary or spontaneous peritonitis is an infection of the peritoneal cavity without an evident intra-abdominal source.¹⁻⁴ The route of entry of the infecting organism is presumed to be hematogenous spread frequently from the respiratory tract, lymphogenous, or transmural migration through the intact intestinal wall, or in females, the ascending spread from the genital tract.¹⁻⁴ In the preantibiotic era it accounted for 10% of abdominal emergencies; it now accounts for less than 1-2%, and is mainly reported in patients with nephrotic syndrome, liver cirrhosis, and HIV infection.¹ It occurs at any age but its peak incidence is between 4 and 9 years of age, and it is more common in girls than boys (ratio, 4:1).¹⁻⁴

A previously healthy 8-year-old boy was brought to the Emergency Department complaining of continuous abdominal pain, focused in the periumbilical area, diarrhea, vomiting and fever up to 40°C.

The physical examination of the abdomen revealed a positive rebound sign, with diffuse tenderness, especially in the lower abdominal region, meteorism and hypoactive bowel sounds. Chest auscultation did not reveal any pathologic signs.

A series of laboratory testing assays were performed, which evidenced a peripheral WBC count of 19,500/mm³ (neutrophils 96%), erythrocyte sedimentation rate 92 mm/h, and a C-reactive protein elevated at 10.7 mg/dL (normal values, 0.08-0.8 mg/dL). Urinalysis showed acid pH of 6.0 and low specific gravity of 1,005. The abdomen radiography revealed hydroaeric levels and free opaque fluid was evidenced in the ultrasonogram of the abdomen.

Antimicrobial empirical therapy was administered (ceftazidime IV 800 mg t.i.d., netilmicin IV 60 mg t.i.d., metronidazole IV 180 mg t.i.d) and within a few hours from admission, an exploratory laparotomy was performed.

At surgery, an odorless fibrino-purulent material was found in the abdominal cavity, covering the abdominal wall and the intra-abdominal organs. Although the appendix did not present signs of inflammation, appendicectomy was performed, and the resected tissue was sent for histological examination, which did not identify any kind of inflammatory process alterations.

Smears of the purulent secretion revealed the presence of leucocytes and Gram-positive diplococci, and the culture grew *Streptococcus pneumoniae* (serotype 7F), susceptible to beta-lactams, macrolides, quinolones, tetracyclines and glycopeptides. The antimicrobial regimen was changed to ceftriaxone IV 1 g b.i.d., which was administered for 10 days.

S. pneumoniae was not found in cultures of pharyngeal swab, in blood or in urine samples. Chest and abdomen radiography, as well as abdominal ultrasonogram performed on the 4th post-surgery day revealed no pathologic signs. Other laboratory analyses were within normal limits. The peripheral WBC count gradually decreased to 9,000/mm³ (neutrophils 65%), erythrocyte sedimentation rate at 38 mm/h, and C-reactive protein at 1.2 mg/dL. The patient was finally discharged on the 12th post-surgery day.

Several decades ago *S. pneumoniae* was the most commonly reported cause of primary peritonitis. Fowler⁵ reported 50 cases of pneumococcal primary peritonitis from 1925 to 1955 and only 6 cases from 1956 to 1970.

Although rarely encountered, primary pneumococcal peritonitis is a potentially life-threatening process that should be included in the differential diagnosis of acute abdominal pain in children.

Conflict of interest

All authors declare to have no conflict of interest.

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Received 10 August 2011

Accepted 23 August 2011

1413-8670

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