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Primary Peritonitis

M. Reza Jahadi, MD, and John G. Whitcomb, MD*

COMMON prior to 1940, primary peritonitis has diminished in incidence to only one or two cases per year at Henry Ford Hospital. This decrease is almost certainly related to the use of antibiotics.

When we recently encountered a case of idiopathic peritonitis in a child with nephrosis, we reviewed the literature and found that primary peritonitis still presents problems of diagnosis and treatment. It still carries a rather high mortality rate and hence, in spite of its rarity, should interest the clinician.

Case Report

A six-year-old Black boy presented to the Emergency Unit of Henry Ford Hospital with a 12-hour history of generalized adbominal pain and vomiting. The patient was reported to have had a "cold" about a week earlier. His abdominal pain was constant and aggravated by activity. Two episodes of vomiting preceded his admission.

His past health history was significant in that a diagnosis of nephrosis was made at age of 18 months. Since then he has been treated intermittently with Prednisone, but not in the past six months.

Physical examination revealed an acutely ill child with a temperature of 39.5°C and a pulse rate of 148 per minute. His blood pressure was 90/60 mm Hg. His abdomen was distended. There was generalized adbominal tenderness, more marked in his right lower abdomen. There was slight rigidity and some guarding. Bowel sounds were absent.

Laboratory studies showed a hemoglobin of 12.7 gm% and a white blood corpuscle count of 18,800 per cubic mm with a shift to the left. Urine showed a specific gravity of 1018, pH of 6.5 and a three-plus albuminuria. Blood urea nitrogen, serum amylase and serum electrolytes were within normal limits. Sickle cell prep was negative. A roentgenogram of the chest showed no evidence of pneumonia, but abdominal roentgenogram showed evidence of ileus.

The patient was treated with nasogastric suction and intravenous fluid. Throat, urine and several blood cultures were obtained and he was started on a large dose of aqueous penicillin. In a few hours his temperature went up to 40.1°C and his abdomen became more rigid and tender.

At operation, it was found that the abdominal cavity was filled with a rather thick creamy white, non-odorous fluid. Mesenteric lymph nodes were pale and slightly prominent. Appendix was grossly normal and there was no evidence of Meckle's diverticulum. An appendectomy was done and the peritoneal cavity lavaged. A Penrose drain was left in his pelvis after abdominal closure.

Postoperatively, large doses of aqueous penicillin were continued. His throat as well as his urine cultures were negative. Peritoneal fluid showed no growth on culture, however, Pneumococcus was isolated from the blood on two different samples. Failure to isolate Pneumococcus from the peritoneal culture was most likely due to the concurrent antibiotic therapy. The patient had an uneventful recovery and was discharged from the hospital in a satisfactory condition.

Discussion

Primary peritonitis was first reported in a case with glomerulonephritis in 1885.[1] Since then a vast literature has accumulated. Although frequently documented in the older literature, nephrosis, per se, did not seem to pre-

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dispose to this type of infection and the relationship is still obscure. The disease has been described in all age groups, even the elderly,[3] but it is largely a disease of infancy and early childhood. The responsible organism in most cases is a Pneumococcus or hemolytic Streptococcus. In some instances, bacteriological studies of the peritoneal fluid yield no growth.[2,4] The inflammatory process diffusely affects the parietal and visceral peritoneum. The peritoneal exudates are usually fibropurulent, odorless and creamy-colored fluid. The mesenteric lymph nodes are swollen, pale, and prominent.

The portal of entry for bacteria still remains uncertain. Some authors have concluded that the genital tract is the main portal of entry. This theory fails to explain the mode of infection in male patients. The gastrointestinal tract may serve as a source of infection in rare cases. The transdiaphragmatic lymphatics have also been incriminated. However, not all cases present with a preceding pneumonia or acute upper respiratory infection. Most authors agree that the blood stream is probably the most common pathway of invasion.

The illness commonly begins with an upper respiratory tract infection. The patient may complain of diffuse severe abdominal pain. The child appears extremely sick and dehydrated. Temperature is generally elevated and pulse is rapid. The abdomen is distended, diffusely tender and often has a "doughy" consistency. The white blood corpuscle count is almost invariably elevated with a shift to the left.

Primary peritonitis should be differentiated from generalized peritonitis secondary to ruptured appendicitis and pelvic peritonitis. Pneumonia, especially in an infant, may be difficult to differentiate from peritonitis.

Treatment advocated for this disease varies from agressive surgery to conservative antibiotic therapy. Gross[2] advocates a middle course including a small right lower quadrant exploratory incision for all suspected cases and drainage of the abdominal cavity.

Establishing the diagnosis of primary peritonitis preoperatively is extremely difficult. Also, because of the chance of overlooking a peritonitis from an appendicitis, we feel that the operative therapy in these cases should not be abandoned. We recommend in all suspected cases surgical exploration through a small right lower quadrant incision. After assessing the diagnosis, a quick appendectomy should be done and the abdominal cavity should be routinely drained. Appendectomy does not seem to complicate the case and the morbidity and mortality would not be increased.

Summary

Primary peritonitis in a six-year-old boy with nephrosis was presented with a review of literature. The etiology and pathology of this disease suggest surgical treatment.

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