

Spontaneous Peritonitis in Cirrhosis of the Liver in a Child*

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Since reported cases of spontaneous peritonitis in cirrhosis of the liver in English literature¹⁻³ are fairly rare we would like to report this case which was diagnosed clinically.

Case Report

A. K. (143462) is a 13 year old boy who was seen with the complaints of jaundice and swelling of the abdomen on December 8, 1969. He had jaundice for two months a year previous to this for admission which he was hospitalized to a local hospital for 11 days. Since then he had been fairly well and was attending school. Fifteen days prior to hospital admission, his abdomen became distended, his urine darkened and jaundice reappeared. In ten days he lost his appetite. He had had epistaxis off and on over the last five years in minute amounts.

On physical examination: he looked somewhat underdeveloped for his age (height 150 cm., weight 35 kg.; 25 and 10 percentile, respectively) but alert and not in acute distress. Scleral jaundice was noticed, his liver was palpable 5 cm. below the right costal margin the spleen was not palpable and he had fairly large amount of ascites. He did not have fever, abdominal distress and gastrointestinal symptoms. B. P. was 120/80 and other findings, including complete neurologic examination, were not remarkable. Blood was obtained for CBC and liver function tests and he was sent home. That night at 2.30 A. M. he was brought back because

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of fever, abdominal discomfort, and vomiting. He was found in acute distress, his abdomen was distended and tender. Otherwise, physical examination was essentially the same. Since no focus was found for fever, abdominal paracentesis was performed, and opalescent ascites was obtained. The specific gravity and the protid of this fluid were 1014, and 790 mg. per 100 ml. respectively. Rivalta's reaction was positive and plenty of polymorph leucocytes were seen. He was given penicillin (4.000.000 u/day) and chloramphenicol (40 mg/kg/day). His symptoms subsided in three days. Ascitic fluid obtained on the seventh day of admission, did not contain pus cells and Rivalta's reaction was negative. Its specific gravity was 1010 and the protid 616 mg. per 100 ml. On the 16th day of admission liver needle biopsy was obtained which revealed postnecrotic cirrhosis for which he was treated symptomatically. His ascites decreased by the use of antidiuretic and aldosteron antagonists. On the 17th day when he was ready for discharge, he developed an infection and was taken from hospital inspite of medical advice. Later we have learned that he was doing fairly well.

Laboratory results at the time of admission: Hemoglobin 12.5 gr per 100 ml., white blood cell 11.000 per cmm with the differential of 64 % segments 32 % lymphocytes, 1 % monocytes, 2 % eosinophils (12 hours prior to admission WBC was 5.000/cmm). Urine analysis was negative including bilirubin determination, his electrolytes were normal (CO_2 : 24 mEq/L; Na: 140 mEq/L., K: 4,6 mEq/L, Cl: 109 mEq/L). SGOT; 580 u, SGPT: 190 u*, Thymol turbidity 2,5 u, cephalin cholesterol (4+) total bilirubin 2.2 mg % (1.4 mg % was conjugated). Total protein 7.2 gr. per 100 ml (albumin/globulin ratio was 4, 2/3). Serum electrophoresis: Alb: 31 %; alpha one 6.4 %; alpha two 10.3 %; beta 9.4 %; and gamma 42.7 %. Blood ammonia 120 gamma per 100 ml, prothrombin time 21 seconds (normal 10 seconds). L. E. cell negative (3 times), sweat chloride normal (23.9 mEq/L), venous pressure was found elevated (195 mm water).

Comments

This is a case of peritonitis in a boy with postnecrotic cirrhosis in whom no known cause was found for peritonitis. Although blood and ascites cultures stayed negative, this is not against this diagnosis, because of the appearance and type of the cell in the ascitis fluid. Since paracentesis was not performed prior to the abdominal findings, perforation of the abdominal viscus was not comparable with the prognosis of the pa-

* Karmen units.

tient, and no site of infection was found twelve hours prior to peritonitis. This is accepted as spontan peritonitis.

Peritonitis, found in the postmortem examination in patients with cirrhosis, is not a rare complication and usually gram-negative organisms are identified. Corticosteroids seems to increase these complications.⁴ But we have not seen this in any of the 72 patients with liver cirrhosis examined in the liver clinic during the last 18 months (Table I).

TABLE 1
PATIENTS WITH LIVER CIRRHOSIS SEEN IN THE LIVER CLINIC LAST 18 MONTHS

	Number of cases
Postnecrotic cirrhosis	17
Biliary cirrhosis	8
Portal cirrhosis	6
Willson's disease (cirrhosis)	7
Cirrhosis (type not specified)	34 (6)*
Total	72

(*) Diagnoses was verified by biopsy, in each case with the exception of six cases whose diagnosis was only clinical.

There have been several theories about spontananeous peritonitis,¹ but we do not know which one was the most likely possibillity for the present case.

In spite of severe liver disease this patient responded to treatment promptly without developing liver failure. We think this is due to very early diagnosis of peritonitis. For that reason, all the evidences of exudate was not present.

Summary

A boy with cirrhosis of the liver, who had spontananeous peritonitis, is reported. His peritonitis was diagnosed very early and responded to therapy promptly.

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