

Case Reports

Case reports are accepted under this heading. These reports should be short and concise and contain a minimum of figures, tables and references.

SPONTANEOUS BACTERIAL PERITONITIS IN A CHILD WITH ABDOMINAL LYMPHOMA

Spontaneous bacterial peritonitis (SBP) is defined as an infection of preexisting ascites in absence of an obvious intraabdominal source (1). Though SBP is common in patients with transudative ascites of liver diseases and nephrotic syndrome it is rare in exudative ascites of abdominal malignancy (2). We here report a well-documented case of SBP with abdominal lymphoma. Timely recognition and appropriate antibacterial treatment are important before instituting antineoplastic therapy.

Case report. A 5-year-old girl presented in February, 1993 with fever, abdominal distension, anorexia and weight loss of 45 days' duration. Examination revealed a cachectic child, weighing 13 kg (normal 18 kg), mild pallor, pedal oedema, tense ascites, two palpable lumps (firm, nontender and nodular), one each in epigastrium and right iliac fossa. No abdominal tenderness, guarding, and rigidity could be elicited.

Investigations showed haemoglobin of 109 g/l, total leukocyte count (TLC) $7.5 \times 10^9/l$, polymorphonuclear cells (PMN) 80%, platelets $390 \times 10^9/l$; normal biochemical liver function tests. Blood urea nitrogen, serum creatinine and uric acid were 12 $\mu\text{mol/l}$, 61 $\mu\text{mol/l}$ and 0.65 mmol/l respectively. Blood and urine cultures were sterile. Erect abdominal x-ray examination showed only ascites and no gas under the diaphragm. Chest x-ray was normal. Ultrasonogram and contrast-enhanced CT scan of abdomen showed ascites with multiple large retroperitoneal lymph nodes, and extensive small bowel thickening without evidence of liver involvement. Small bowel barium series revealed irregular thickening of small intestinal folds without evidence of obstruction or perforation. Fine-needle aspiration of right iliac fossa mass and ascitic fluid cytology revealed lymphoblastic cells, suggestive of malignant lymphoma. Ascitic fluid analysis showed TLC of $7.2 \times 10^9/l$ (PMN 80%), protein 270 g/l, glucose 3.9 mmol/l and absence of bacilli, cocci or mycobacteria in Gram's and acid fast stained smears. Culture grew *Pseudomonas aeruginosa*. Intravenous ciprofloxacin was started in accordance with sensitivity report. Examination of ascitic fluid repeated after 48 h of antibiotic therapy revealed TLC $3.8 \times 10^9/l$ (80% PMN), and no growth in aerobic and anaerobic cultures. Intravenous fluid and allopurinol were started. Serum creatinine and uric acid repeated at this stage were 79 $\mu\text{mol/l}$ and 0.5 mmol/l respectively. Chemotherapy with cyclophosphamide, vincristine and prednisolone was started four days later. The patient died suddenly 4 h after initiation of chemotherapy which might be related to the advanced malignancy and/or sepsis.

Discussion. Diagnosis of SBP in our patient was established by standard criteria (3) i.e. ascitic fluid PMN count $> 0.25 \times 10^9/l$, positive culture in absence of any recognizable secondary cause, such as hollow viscous perforation, which was ruled out by absence of leak in barium series, monomicrobial infection, ascitic fluid glucose > 2.8 mmol/l, fall in PMN count $< 50\%$ of baseline value and sterile culture after 48 h of treatment (4). These criteria have been found to differentiate between spontaneous and secondary peritonitis caused by gut perforation with high degree of sensitivity and specificity (4).

SBP is a common complication in patients having ascites due to cirrhosis of liver or nephrotic syndrome (1) but rare in malignant

ascites; rarity of SBP in these patients is thought to be due to normal complements, opsonic and bactericidal activity in the ascitic fluid. However, SBP has been reported in patients with malignant ascites due to carcinoma of the stomach (5, 6) and colon (7). There has been one previous report in association with malignant lymphoma in which the criteria for SBP were not fulfilled and it is thus a report of spontaneous bacterascites instead of SBP (8).

SBP develops as a result of translocation of bacteria from gut into the ascitic fluid; this is evidenced by frequent isolation of gut-derived bacteria in SBP (1). Low protein ascites in cirrhotic patients favour bacterial proliferation due to deficiency of complements and opsonic activity; the latter being normal in exudative ascites of malignancy, SBP develops only when particularly virulent bacteria enter into the fluid (like *Pseudomonas aeruginosa* in our patient). Furthermore, patients with malignancy may have depressed phagocytic function and impaired Kupffer cell function particularly in presence of hepatic involvement (5). Antimalignant chemotherapy may further exacerbate these immunological dysfunctions.

SBP, though rare, does occur in malignant ascites. We report this case because awareness of such a rare complication of malignant ascites, its early diagnosis and appropriate antimicrobial therapy, is essential before starting antineoplastic therapy. Moreover, if ascitic fluid examination is undertaken in isolation, the presence of SBP itself may take the clinicians away from primary diagnosis of malignancy considering rarity of this association.

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October 1994

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