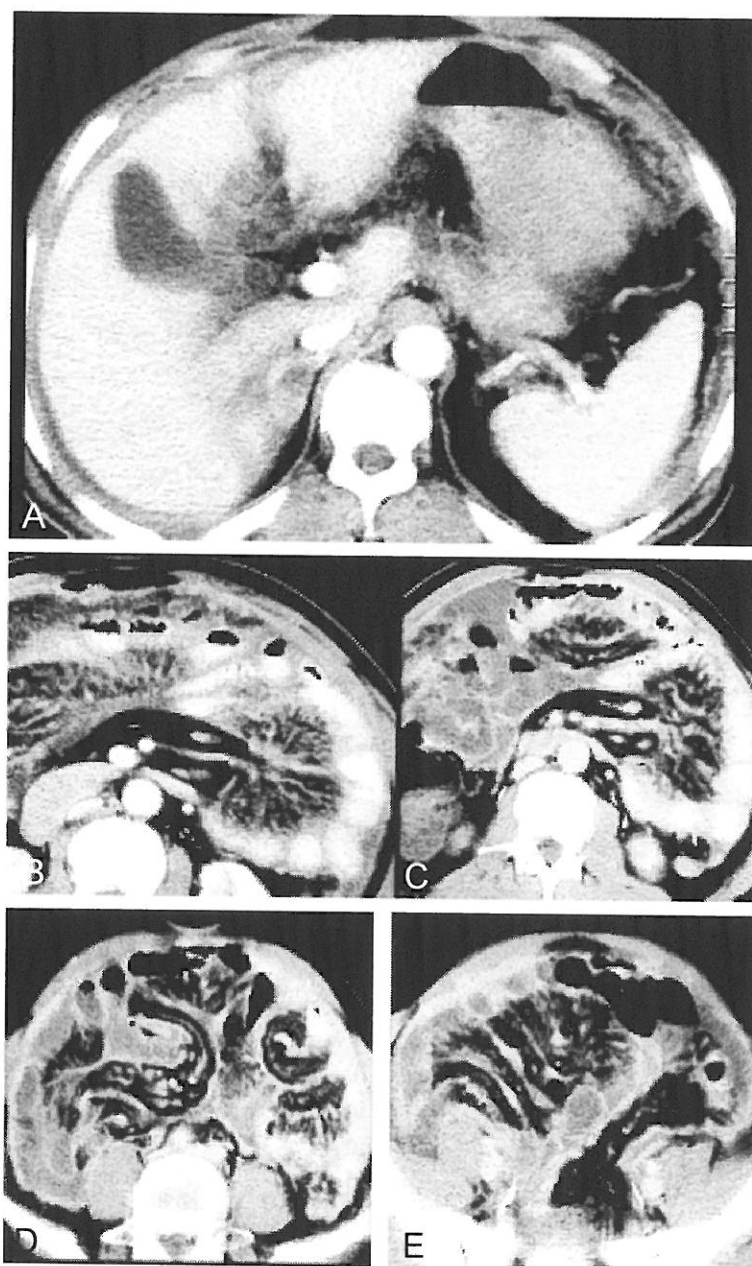


PERITONEAL TUBERCULOSIS IN AN AFRICAN IMMIGRANT

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Key-word: Tuberculosis, gastrointestinal.

Background: A 52-year-old black male originating from Guinea was admitted in poor general condition. The patient had undergone a laparotomy three months previously in his home country for unknown reason.



	1A	
Fig.	1B	1C
	1D	1E

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Work-up

On admission, the patient presented diffuse and severe abdominal pain without fever, weight loss of 24 kg and constipation associated with sporadic rectorrhage. Physical examination was unremarkable except for abdominal distension.

Inflammatory tests (CRP: 39 mg per liter and leucocytosis) and viral hepatitis B and C markers were positive. The blood level for CA 19-9 reached 68 IU/L. Serology for AIDS and syphilis was negative. Blood, expectoration and urine samples were sterile. Abdominal CT scan was obtained for suspicion of acute hepatologic disorder.

Radiological diagnosis

Helical CT scan of the abdomen. (fig. 1) included a section at the level of the liver and lesser sac (A) which showed regular micronodules in the lesser sac and hepatic hilum and thickening of the parietal peritoneum at the left upper quadrant. Sections at the level of the peritoneal cavity (B-E) showed regular micronodules of the same size on the serosal surface of the mesentery and thickening of the parietal peritoneum. Fixed small bowel loops in all peritoneal quadrants without luminal dilatation, stasis, or intramural abnormalities were observed. In the subsequent views, ascites with elevated density (+/- 30 HU) was noted in the right paracolic gutter (B). Linear thickening in the mesenteric leaves corresponding to small amounts of ascites and enlarged mesenteric vessels were also seen (B,C) and a large amount of ascites entrapped within peritoneal thickening was depicted (E,D).

On laparoscopic view of the peritoneum (fig. 2), multiple small granular nodules on the mesentery and parietal peritoneum were seen.

Based on the findings, the diagnosis of peritoneal carcinomatosis was raised first, but no primary tumor was recognized. Among other differentials, peritoneal tuberculosis or other benign granulomatous, or infectious diseases were mentioned. Chest examination was negative. Aspiration of peritoneal fluid showed thick fluid with 65 gr per liter of proteins and lymphocytes accounted for 72%.

At histology, specimens revealed *peritoneal tuberculosis* with granulomas without necrosis. Antibiotic treatment was given with a favorable response.

Discussion

Peritoneal tuberculosis accounts for 4% of extrapulmonary locations of the disease. The pure form without gastrointestinal, genital or renal involvement seems to be a very rare entity. Episodic reports were published in the literature. There is no consensus regarding etiology and pathogenesis. Confusion between peritoneal and intestinal tuberculosis is a cause of controversy. Primary peritoneal location is understood as a hematogenous complication following primary infection. Gastrointestinal tuberculosis originates from swallowed expectoration or has a bovine origin. According to this theory, intestinal tuberculosis is an ulcerative jejuno-ileo-colitis and ascites a complication due to fistulae and perforation. For peritoneal tuberculosis, ascites is usually the first clinical sign. Three categories of peritonitis are reported in the literature: a "wet" type with exudative ascites, a "dry" type with lymph nodes and adhesions, and a "fibrotic" type with abdominal mass and fixation of small bowel loops. In the experience of the authors, this classification lacks precision. In all of their five cases, patients presented with ascites. Follow-up during treatment showed progressive fibrosis leading to intestinal occlusion in two cases. Fluid collections in the peritoneal cavity were also identified in all of the cases reported in the literature, which suggest that dry and fibrotic forms, in fact, reflect advanced disease. The deposition and growth of secondary seeded infectious implants on the peritoneum reflect the physiologic pathway of flow of ascites within the peritoneal recesses and distribution depends on mesenteric reflections.

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