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Splenic Abscess and Peritonitis in a Continuous Ambulatory Peritoneal Dialysis (CAPD) Patient

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A 26-year-old female was on continuous ambulatory peritoneal dialysis (CAPD) because of diabetic endstage renal failure. She developed an acute peritonitis that relapsed repeatedly despite appropriate antibiotic treatment. Investigations showed the presence of a splenic abscess, and splenectomy and peritoneal cannula removal were required. The patient died of myocardial infarction two weeks postoperatively.

This is the first recorded case of peritonitis secondary to splenic abscess in a CAPD patient. Autopsy findings suggest that the abscess developed from infection of a splenic infarct.

KEY WORDS: Peritonitis; splenic abscess.

p eritonitis in continuous ambulatory peritoneal dialysis (CAPD) patients is occasionally secondary

to intra-abdominal pathology. Most common underlying causes include perforation of the large bowel or appendix, diverticulitis, and gangrenous cholecystitis (1).

We report a previously undocumented association of relapsing acute peritonitis and splenic abscess occurring in a young diabetic female on CAPD.

CASE REPORT

A 26-year-old female was transferred to this hospital for management of relapsing acute peritonitis. She had a 25year history of insulin-dependent diabetes mellitus and had been on CAPD for treatment of diabetic end-stage renal disease for 18 months. She had advanced diabetic retinopathy.

Two weeks earlier she had presented elsewhere with her first episode of peritonitis on CAPD. The peritoneal dialysate quickly cleared after treatment with i.p. cephalothin and netilmicin. All cultures were negative.

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However, pyrexia persisted over the ensuing 12 days. Extensive investigations were negative except that a splenic cyst was noted on ultrasonography. On the ninth hospital day antibiotics were discontinued and on the 14th day peritonitis recurred. *E. cali* was cultured from the dialysate and treatment with the same antibiotics was again successful.

On admission to this hospital the patient's condition was stable. There was low-grade fever. Abdominal and chest examination was unremarkable. Cardiac examination showed left ventricular enlargment. There was no evidence of endocarditis.

Fever persisted and, two days after admission, despite continuation of antibiotics, peritonitis flared again with abdominal and left shoulder pain. The patient improved with peritoneal lavage. Computerised tomographic scanning showed an irregular, fluidfilled splenic cyst 4 cm in diameter (see Figure 1). Percutaneous aspiration of the cyst yielded 120 mL of foul-smelling pus from which *E. cali* was cultured. The patient improved with percutaneous drainage but fever persisted and five days later she was taken to the operating room where splenectomy and removal of the dialysis cannula were performed. At operation, the spleen contained a necrotic cavity with residual pus. There was no evidence of bowel pathology .There was extensive atheroma in the splenic arterial tree with evidence of recent thrombotic occlusion of one of the endarteries.

The patient was maintained on haemodialysis and initially did well. However, seven days postoperatively she had an acute myocardial infarction with cardio genic shock and died eight days later.

Autopsy revealed no additional findings except for cardiac hypertrophy, extensive coronary atheroma, and recent myocardial infarction. There was no residual intraabdominal pathology.

DISCUSSION

This is the first recorded association of splenic ab scess and peritonitis in a CAPD patient.

We suspect, on the basis of the pathological find ings, that the initial event was a splenic infarct sec



Figure 1-Computerised tomographic scan of splenic cyst.

ondary to atheroma and superimposed thrombosis in one of the endarteries supplying the spleen. This infarct may then have become infected with *E. cali* perhaps secondary to a transient bacteraemia such as is known to occur intermittently in healthy people (2). Thus, a splenic abscess resulted and recurrently seeded the peritoneal dialysate giving rise to relapsing episodes of acute peritonitis.

The occurrence of splenic abscess on the basis of infection of splenic infarcts is a recognised complication in infective endocarditis and in sickle cell disease (3). Other cases of splenic abscess have occurred secondary to urinary tract infection, appendicitis, trauma, contiguous neoplasm, and typhoid fever (3). N one of these conditions was present in our patient but she did have severe vascular disease secondary to longstanding diabetes mellitus.

There is one previously recorded case of splenic

abscess in a patient on acute peritoneal dialysis (3). This resulted from a perforation of the small bowel by the dialysis trocar with consequent intra-abdominal sepsis.

It could be argued that the splenic abscess in our patient was the consequence rather than the cause of the initial presentation with acute peritonitis. Such a sequence of events cannot be excluded but has not previously been noted and we think that it is less likely in view of the pathological evidence of atheroma and acute thrombosis in the splenic arterial tree as an underlying cause for splenic infarction.

Our patient's nonspecific clinical presentation is not atypical for this condition and highlights the importance of ultrasonography and, especially, of computerised tomographic scanning in the diagnosis of splenic abscess (4).

Once the abscess was diagnosed, the management of the patient was based on the belief extrapolated from the experience in other secondary causes of peritonitis that definitive surgical intervention with splenectomy and cannula removal would be essential to cure the infection. It was planned to reinsert a cannula and restart the patient on peritoneal dialysis after a four-week interval but this was prevented by the patient's death due to associated cardiac disease.

We conclude that the possibility of a splenic abscess should be kept in mind in patients with relapsing acute peritonitis especially in the presence of a predisposing cause.

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