Chylous ascites due to bile duct tumour in a patient receiving automated peritoneal dialysis

Sir,

Bacterial peritonitis is the most common cause of cloudy dialysate seen in patients with peritoneal dialysis (PD) [1]. However, the differential diagnoses include sterile ‘allergic’ peritonitis, excess fibrin and haemoperitoneum (e.g. menstrual blood loss). A rare cause is chyloperitoneum [2] caused by leakage of chyle. For the first time, we present a case of chylomperitoneum in a PD patient secondary to an obstructive bile duct carcinoma.

History. This 72-year-old male with hypertensive nephrosclerosis commenced CAPD in May 1994. He chose to switch to automated peritoneal dialysis (APD) in 1996. In January 1998 he commenced a 3 month cruise, but whilst on holiday became progressively unwell with nausea, vomiting and general malaise. He was admitted to the in-patient unit immediately on return. He had lost 7 kg in weight, was vomiting and had dizziness on standing. On examination he was deeply jaundiced with signs of extra-cellular fluid depletion. Systemic and abdominal examination were otherwise unremarkable. Biochemical investigations were consistent with obstructive jaundice—alkaline phosphatase of 777 IU/ml (normal range = 40–140), alanine transaminase of 220 IU/l (normal range = 40–140) and serum bilirubin of 44 μmol/l (normal range = 5–17). Abdominal ultrasound showed multiple biliary calculi and a dilated common bile duct of 14 mm.

At ERCP, a malignant stricture of the common bile duct was found and a stent was inserted. An abdominal CT scan showed no intra-pancreatic pathology and the diagnosis was of inoperable cholangiocarcinoma. He remained on APD but then presented with a cloudy PD bag that was analysed by white cell count and microbiological culture according to the standard ISPD protocol. He was commenced on intraperitoneal vancomycin and gentamicin but in fact the white cell count was 10 leucocytes/μl and subsequent culture was negative. PD bags became increasingly ‘milky’ over the next 3–4 days, and he developed increasing problems with postural hypotension secondary to excessive ultrafiltration (>1 l/2 l PD fluid) even with hypotonic PD exchanges. Biochemical analysis of PD fluid showed: total protein 1.64 mg/dl, amylase 15 μmol/l, LDH 36 IU/l and cholesterol was 0.04 mmol/l. Ultra-centrifugation of the PD fluid demonstrated chylomicrons. A diagnosis of chylous ascites secondary to malignant obstruction of the common bile duct was made.

Maintenance of fluid balance using PD proved impossible because of the excessive fluid loss even with hypotonic PD exchanges. He was transferred to haemodialysis but he still required regular drainage of 2–3 l of chylous ascites at each dialysis session. Four weeks after presentation with the cloudy exchange he was re-admitted for terminal care and dialysis was discontinued.

Discussion. This patient presented with chylous ascites several years after commencement of PD, with intra-abdominal malignancy and management was difficult because of the intense changes in fluid balance that developed as a consequence of the problem. The incidence of chylous ascites in chronic PD patients is unknown. One review [3] of 192 PD catheter insertions reports an incidence of 0.5%, but this reflects the acute complication rate of insertion. A review [2] of 12 cases among 230 patients in two centres showed malignancy (lymphoma and ovarian carcinoma) in two cases and a variety of other causes in seven patients, in three cases a microtrauma from the catheter was suspected. In four cases the chylous ascites lasted for more than 2 years and a change in dialysis modality was not required. Other individual cases have been reported, including lymphoma [4] and tuberculous peritonitis [5].

The diagnosis of chyloperitoneum should be suspected when the dialysate has a typical milky appearance and is confirmed if chylomicrons are detected or dialysate triglyceride levels are higher than the plasma level. Management of chylous ascites is difficult and based on anecdotal evidence. Timing in relation to PD catheter insertion is important since this can contribute by presumed micro-trauma to lymphatic vessels. In these cases, supportive management is indicated along with a change in diet. Long-chain fatty acids are absorbed from the bowel directly into the lymphatic system and contribute to chyle flow. Medium-chain triglycerides are absorbed directly into the bloodstream and decrease the chyle flow. Thus, a low-fat diet containing medium-chain triglycerides can be employed and over several months has shown to be effective in reducing chylous flow. Long-term total parenteral nutrition is an alternative approach although not without practical problems. However, this case demonstrates that chylous ascites developing remote from catheter insertion can be due to underlying malignant disease. After confirmation of the diagnosis by demonstration of chylomicrons and/or elevated triglyceride levels in PD fluid, investigation is essential to exclude malignancy/infection.

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