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Chylous Ascites in a Patient Receiving Peritoneal Dialysis

Chun-Hsien Wu[1], Hsuan-Hwai Lin[1], Tsung-Neng Tsai[1] and Jin-Shuen Chen[2*]

[1]Department of Internal Medicine, [2]Division of Nephrology, Department of Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, Republic of China

Cloudy dialysate in a peritoneal dialysis (PD) patient may be due to infection or other causes. In patients without infection, chylous ascites is an infrequent complication. Here we report a 69-year-old Asian woman with end-stage renal disease resulting from diabetic nephropathy who developed chylous ascites one week after Tenckhoff catheter insertion. A low-fat diet was prescribed to treat the complication, with the chylous ascites subsiding after four weeks of treatment. The literature on possible etiology and treatment of chylous ascites in peritoneal dialysis patients is reviewed.

Key words: chylous effusion, chyloperitoneum, peritoneal dialysis, low-fat diet.

INTRODUCTION

The appearance of cloudy dialysate fluid most often heralds infectious peritonitis in peritoneal dialysis patients. The diagnosis of infectious peritonitis is usually readily established by positive dialysate cultures and leukocytosis of the peritoneal dialysis fluid. However, infection does not explain all cases of increased dialysate turbidity. The differential diagnosis of noninfectious cloudy peritoneal dialysis fluid is heterogenous and based on the identification of cellular or noncellular constituents enhancing the turbidity of the dialysate[1]. The abnormally increased constituents include polymorphonuclear cells due to intra- or juxtaperitoneal inflammation, eosinophils associated with an allergic reaction to the dialysis, malignant cells and triglyceride seen in the chyloperitoneum.

Chylous ascites develops when there is disruption of the lymphatic system because of traumatic injury or obstruction (from benign or malignant causes). Other nonmechanical causes of chylous ascites include inflammatory disorders of the abdominal cavity, heart failure, cirrhosis and postoperative complications[2]. Chylous ascites is an infrequent complication of peritoneal dialysis and easily confused with peritonitis because of their characteristic and undifferentiated turbid appearance. Chyloperitoneum in PD had been defined as a triglyceride concentration over 110 mg/dL and detectable chylomicrons by lipoprotein electrophoresis in the dialysate effluent[3]. In PD patients, chylous ascites caused by lymphoma, tuberculosis, pancreatitis, superior vena cava syndrome and drugs of the dihydropyridine-type calcium channel blockers have been reported[4-9]. We describe a uremia patient who developed chylous ascites one week after insertion of a Tenckhoff catheter and was treated successfully with a fat-free and low-fat diet. The literature relating to chylous ascites in peritoneal dialysis patients is also reviewed.

CASE REPORT

A 69-year-old woman had been noted with diabetes mellitus and hypertension for 10 years and had generalized edema two weeks before admission. The patient had not had recent surgery, internal jugular vein catheterization, trauma or underlying malignancy. Her family history was noncontributory.

On physical examination, her blood pressure was 148/90 mmHg, pulse rate 88 beats per min, respiratory rate 24 per min and temperature was 36.5 °C. Her consciousness was alert. Cardiac examination revealed no murmur, gallops or rubs. The patient's lungs were clear on auscultation. There was no abdominal pain or rebounding tenderness. Bilateral grade II pitting edema was present in the lower legs. The neurological examination was normal. The remainder of the physical examination was unremarkable.

A complete blood count revealed normocytic anemia with hemoglobin 8.6 g/dL, mean corpuscular volume 88.1 fL, white cell count 4400/μL and platelets 174,000/μL. Serum biochemistry showed blood urea nitrogen 94 mg/

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*Corresponding author: Jin-Shuen Chen, Division of Nephrology, Department of Medicine, Tri-Service General Hospital, 325, Cheng-Gong Road, Section 2, Neihu 114, Taipei, Taiwan, Republic of China. Tel:+886-2-8792-7213; Fax:+886-2-8792-7134; E-mail: dgschen@ndmctsgh.edu.tw

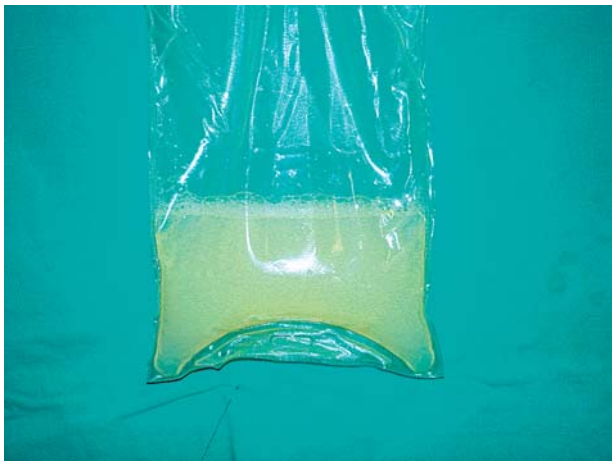

Fig. 1 Appearance of cloudy dialysate from our patient.

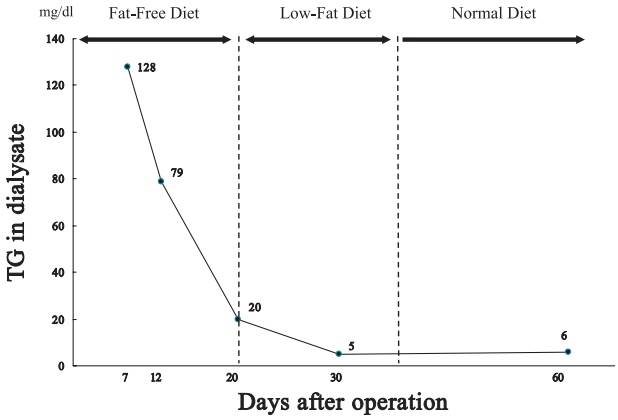

Fig. 2 Serial changes in dietary fat content and triglyceride concentrations of the dialysate effluent. TG: triglyceride

dL, creatinine 11.3 mg/dL, total cholesterol 324 mg/dL, triglyceride 165 mg/dL and albumin 2.5 g/dL. The urine creatinine clearance was 2.4 mL/min and daily protein loss was 4.3 g. Abdominal sonography demonstrated bilateral small-sized kidneys without ascites. Diabetic nephropathy with nephrotic syndrome and end-stage renal disease was diagnosed.

The Tenckhoff peritoneal dialysis catheter was placed smoothly by a surgeon for continuous ambulatory peritoneal dialysis (CAPD) on the third hospital day and then she was discharged with regular outpatient department follow-up. The peritoneal dialysis fluid was clear and sterile initially but became turbid one week after the catheter placement (Figure 1). The patient had mild abdominal pain without rebounding tenderness or fever. Cell counts of the cloudy dialysis effluent revealed 80 leukocytes/μL with 32% polymorphonuclear cells and 68% mononuclear cells. Biochemical analysis of the PD fluid revealed a triglyceride level of 128 mg/dL. The diagnosis of chylous ascites was made. Cytological examination was negative, as were bacterial, fungal and acid-fast bacterial cultures. An abdominal computed tomography with intravenous injection of contrast medium revealed bilateral pleural effusion and ascites but no enlarged lymph nodes, pancreas or mass lesions.

A fat-free diet was instituted for two weeks followed by a low-fat diet for another two weeks. Home PD was performed regularly during the period. After four weeks of treatment, the PD fluid was clear and the triglyceride concentration was lowered to 5 mg/dL. No recurrence of chyloperitoneum was noted after cessation of diet restriction for one year and receiving regular CAPD. The courses and response to treatment are summarized in Figure 2.

DISCUSSION

Chylous ascites is one of the numerous causes of cloudy peritoneal fluid and is a rare occurrence in patients receiving PD[1]. The incidence of chylous ascites in PD patients was 0.5% in one review article[10] but this reflects the acute complication rate of insertion. Chylous ascites is the extravasation of milky chyle in the peritoneal cavity and has a variety of causes, including inflammation, neoplasm, postoperative complication, trauma and obstruction of the lymphatic system[3]. The most common cause of chylous peritoneal dialysate is lymphatic obstruction secondary to malignancy, in particular lymphoma[4]. However, our patient appeared to have none of these conditions and her dialysate was characterized by normal leukocyte counts, normal red blood cell counts, no organisms and no malignant cells. The nephrotic syndrome is known to be associated with chylous ascites and chylothorax[11]. The pathogenesis of chylous ascites in nephrotic syndrome is not understood, but hypoalbuminemia-induced bowel edema with resultant lacteal leakage or malabsorption has been offered as a speculative explanation. In our patient, the initial diagnosis was nephrotic syndrome without ascites, which made nephrotic syndrome associated chylous ascites an unlikely cause. Levy et al. reported the development of chylous ascites two weeks after catheter placement and speculated that repeated mild trauma to the lacteals or the thoracic duct at the time of catheterization or movement of the catheter could be the cause[2]. A flush of PD fluid leads to elevated abdominal pressure and increased net transport of lymph into the peritoneal cavity[12]. In this case, the development of chylous ascites one week after Tenckhoff catheter insertion may have been caused by

incidental lymphatic vessel trauma during the operation and precipitated by an early flush. Further analysis of more cases is needed to elucidate the possible mechanism and ways of preventing chylous ascites in PD patients.

PD patients with chylous ascites are at greater risk of developing hypoalbuminemia and malnutrition because of protein and lymphocyte loss from the peritoneal effluents. This may exacerbate the immunocompromised state in end-stage renal patients. Therefore, early recognition and management of the condition is imperative. Once the diagnosis of chylous ascites is established from an analysis of the turbid dialysate, a low-fat diet with medium-chain triglycerides (MCT) and dietary restriction of long-chain triglycerides (LCT) is the first line treatment. The LCT are hydrolyzed within the intestinal lumen to monoglycerides and free fatty acids (FFA), which are transported from intestinal cells as chylomicrons via the intestinal lymph ducts. MCT have less than 12 carbon atoms, are absorbed directly into the intestinal cells and are transported as FFA and glycerol directly to the liver via the portal vein. The use of a low-fat diet with medium-chain triglyceride supplementation therefore reduces the production and flow of chyle. The resolution of chylous ascites through conservative treatment was achieved by diet alone in only 43% of patients[2]. Patients who do not respond to the above measures should be fasted to reduce lymph flow and started on total parenteral alimentation. Some reported cases have used somatostatin and octreotide to treat chylous effusion in patients with postoperative lymphatic leakage. It has been speculated that somatostatin was able to decrease intestinal fat absorption and reduce both the triglyceride level in the thoracic duct and lymphatic flow in the major lymphatic vessels[13].

In conclusion, culture negative, cloudy peritoneal dialysate results from a diverse group of etiologies. The possibility of infective peritonitis should be excluded by clinical and laboratory findings. Benign conditions such as Tenckhoff catheter insertion-related chylous ascites as in our case could be the cause. A systematic approach may reduce unnecessary antibiotic use, and management with diet modification could avoid malnutrition and immunocompromising the PD patient.

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