LONG-CHAIN FATTY ACIDS: A RARE CAUSE OF CHYLOUS ASCITES IN ADULT CONTINUOUS AMBULATORY PERITONEAL DIALYSIS PATIENTS?

Qijun Wan, Yongcheng He, Dan Zhu, Jing Li, Huili Xu

Shenzhen Second People's Hospital, Shenzhen, Guangdong Province, P.R.C

Objectives:

Continuous chyle leak can cause a decrease in immunity and loss of fat, resulting in recurrent infections, malnutrition and even obstruction of the peritoneal catheter among peritoneal dialysis patients. Chylous ascites typically occurs due to congenital anomalies of the lymphatic system or secondary to abdominal trauma or surgery in a child. However, is Long-Chain Fatty Acids a rare cause of chylous ascites in adult continuous ambulatory peritoneal dialysis (CAPD) patients? The literature has not been seen.

Methods:

A 63-year-old female was treated with CAPD for end-stage renal disease 4 weeks due to chronic glomerulonephritis. Four weeks after catheter insertion via an abdominal operation, she was initiated on an intravenous infusion of 10 % Medium and Long chain Fat Emulsion Injection (produced by Baxter, 250 ml containing long-chain fatty acids 12.5 g IV) and oral administration of Enteral Nutritional Emulsion (TP-HE, 500 ml containing long-chain fatty acids 8 g, orally) once daily. The indication for this was poor nutritional intake. Three days later, the peritoneal effluent became "milky". The milky effluent was most apparent on the first drain of each day. She had not been prescribed calcium channel blockers and she had no history of liver disease, portal hypertension, nephrotic syndrome or pancreatitis.

Results:

Peritoneal effluent cell counts were normal and cultures were negative. Peritoneal effluent triglyceride levels were 220 mg/dl which was higher than the diagnosis criterion of chyloperitoneum (defined as triglyceride levels > 110 mg/dl). Investigations to detect a cause of chyloperitoneum were negative. The intravenous infusion of Long-chain fatty acids and the oral administration of Enteral Nutritional Emulsion were stopped and the chyloperitoneum rapidly resolved. It was decided to transfer the patient from peritoneal dialysis to hemodialysis for two weeks to allow her peritoneum to rest. She subsequently resumed peritoneal dialysis with no recurrence of her chylous ascites. Because of the poor nutritional status of the patient, the two fatty acid products were reused. Then the chyloperitoneum appeared again. From then on, the two fatty acid agents were not used any more for the patient. The patient did not suffer any side-effects from this and, in particular, did not develop peritonitis, lymphopenia or worsening malnutrition.

Conclusions:

We highlight a new cause of chyloperitoneum in adult CAPD patients. In this case, the etiology was high doses of Long-chain fatty acids. This is supported by the fact that the chyloperitoneum disappeared after these agents were stopped and recurred when they were reused because of the poor nutritional status of the patient. To our knowledge, this is the first report of such an occurrence. The conservative treatment is effective enough in chylous ascites in our patient.

References:

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