Chyloperitoneum in Pediatric Peritoneal Dialysis: Rapid Remission After Introduction of Medium-Chain Triglyceride-Based Formula

Editor:

Chyloperitoneum (CP) is a rare complication of peritoneal dialysis (PD) (1,2). The diagnosis is based on a high level of triglycerides in effluent and the presence of chylomicrons. The most common causes of CP in children are congenital anomalies, lymphangioma, abdominal compression, and abdominal surgery with lesions of the lymphatic vessels (3,4). Only a few reports of CP as a complication of PD have been published (5). The condition is often a result of surgery, and it is more frequent in adults. Recurrent infections, immune deficiency, and hyperosmolar dialysate increase the risk of CP (2,4).

CASE DESCRIPTION

A 3-month-old boy with end-stage renal disease attributable to posterior urethral valves and renal dysplasia was on nightly intermittent PD. The diagnosis of posterior urethral valves was suspected at 17 weeks' gestational age. Renal sonography at day 1 revealed bilateral hydronephrosis, dysplastic kidneys with increased echogenicity, a posterior urethra dilated at 3 mm, and increased thickness of the bladder wall. Initial urine output was normal, but the massively dilated kidneys required bilateral nephrostomy, followed by bilateral ureterostomy. Despite renal drainage, renal insufficiency increased, requiring insertion of a (Tenckhoff) PD catheter. Continuous PD was started using 1.36% Physioneal solution (Baxter Healthcare Corporation, Deerfield, IL, USA). The patient had a first peritonitis attributable to Stenotrophomonas maltophilia requiring ciprofloxacin treatment for 3 weeks.

At 2 months of age, the boy underwent Nissen fundoplication and gastrostomy and a change of Tenckhoff catheter. After surgery, tube feeding was progressively introduced and was well tolerated, resulting in normal weight and height development.

One month later, the PD effluent had a milky appearance. Because of the recent gastrostomy, we first suspected intestinal perforation, with a milk outflow. The patient was pain-free and afebrile, and an abdominal examination was normal. Radiography of the abdomen eliminated pneumoperitoneum. The effluent lacked an appearance of digested milk, suggesting a chylous origin. Effluent analysis revealed very high triglycerides of 887 mg/dL, a white blood cell count of 53/mm³ (2% neutrophil granulocytes; 16% eosinophil granulocytes; 4% lymphocytes; 78% other cells). Bacterial and fungal analysis was negative. Numerous chylomicrons were present, but serum triglycerides remained normal. The foregoing results confirmed a diagnosis of chyloperitoneum, which is defined as triglycerides greater than 110 mg/dL.

A low-fat diet supplemented with medium-chain triglycerides (MCTs) was started in the first hours after the milky appearance of the dialysate. The formula was changed to Monogen (Nutricia, Schiphol, Netherlands), which contains a fat consisting of 90% MCTs (10.6 g MCT in 100 g Monogen). After 1 day, we supplemented Monogen with maltose dextrin powder and Lipigen [lipids: 50 g/100 mL, 98% MCTs (Lipigen, Oslo, Norway)] to enhance energy intake. On day 2, the effluent appeared less milky, with triglycerides of 98 mg/dL. Triglycerides were 26 mg/dL on day 4. Ten months later, the baby boy is doing well, with normal growth parameters, on ambulatory nightly PD.

Only a few cases of CP have been reported in pediatric PD patients (5). In all reported cases, exclusion of long-chain triglycerides (LCTs) was the first therapeutic strategy. The LCTs are directly absorbed and transported by the lymphatic system as chylomicrons; MCTs are absorbed by the intestine and transported by the portal system. The concentration of LCTs determines flow rate in the lymphatic system, with a postprandial increase from 1 mL/kg to 200 mL/kg per hour. As a result, LCT eviction slows lymph flow, resulting in faster restitution of lymph vessel integrity.

In the case of persistence after several weeks, octreotide, a somatostatin analog, is frequently used to reduce intestinal fat absorption and the lymphatic flow rate (6). In our case, the patient developed CP 1 month after a second surgery involving a change of Tenckhoff catheter. The LCT level in our patient was higher than has been reported in the literature. The MCT diet was introduced within 24 hours after the milky appearance of the effluent, producing an excellent response. In other reported CP cases, the diagnosis was established later, and a MCT diet was therefore started less rapidly. Indeed, most authors first suspect peritonitis, which

delays the diagnosis of CP. In the case of milky effluent, CP should be suspected, and a MCT diet should be started promptly.

CONCLUSIONS

Rapid introduction of a MCT diet may result in rapid recovery of lymphatic vessel lesions. This approach might facilitate rapid reintroduction of a normal diet without CP relapse, essential for optimal calorie intake in children on PD.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

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