Normal Peritoneum After Nine Years of Peritoneal Dialysis with Biocompatible Dialysate: A Case Report

Editor:

Recent advances in clinical practice and improved biocompatibility of dialysate should reduce the risk of peritoneal damage in peritoneal dialysis (PD) patients.

Morphology changes in the peritoneum of long-term PD patients include denudation of peritoneal mesothelial cells, interstitial sclerosis, and hyalinizing vasculopathy (1). Most long-term PD patients show abnormal peritoneal histology (2,3). Introduction of biocompatible dialysate with a neutral pH and lesser concentrations of glucose degradation products (GDPs) into recent Japanese PD practice might exert a favorable effect on peritoneal morphology (4–7).

Here, we report an 85-year-old male patient whose peritoneal morphology was preserved during 9 years of PD with the use of biocompatible dialysate.

CASE REPORT

An 85-year-old Japanese man with end-stage renal disease because of hypertensive nephrosclerosis was initiated on PD in 2003. He was treated with continuous cycling PD for the first 2 years and was then transferred to combination therapy in 2005 because of anuria. His regimen of combined therapy was daily PD (7 days per week) and once-weekly hemodialysis. Because of hemodynamic instability attributable to aortostenosis, fluid status was maintained with PD.

The dialysate used by the patient was Midpeliq L (Terumo, Tokyo, Japan), a dual-chamber dialysate with concentrations of GDPs that are about one fifth of those in conventional glucose solution (4,7). The final concentrations of electrolytes and buffer in the solution

are Na^+ 135 mEq/L, Ca^{2+} 2.5 mEq/L, Mg^{2+} 0.5 mEq/L, Cl^- 98.0 mEq/L, and lactate 40.0 mEq/L, with a pH between 6.3 and 7.3. Osmolality of the 1.35% glucose solution is 350 mOsm/L, and that of the 2.5% glucose solution is 414 mOsm/L. The patient's regimen throughout the entire treatment period was automated PD with 7.5 L of 1.35% glucose dialysate and 2.5 L of 2.5% glucose dialysate (mixed in the cyclor for a final glucose concentration of about 1.9%) with 6 exchanges during a 4-hour dwell. Peritoneal function was mostly preserved throughout PD therapy (dialysate-to-plasma creatinine in peritoneal equilibration tests was constant at approximately 0.5 – 0.6).

Our patient did not experience peritonitis until a refractory peritonitis in 2012 that led to catheter removal. The patient's first peritonitis episode in February 2012 was caused by *Klebsiella pneumoniae*, which was successfully treated with intravenous vancomycin. After that episode, peritonitis occurred twice more within about 2 months each time. The final episode of peritonitis occurred in July 2012. Although all episodes were promptly controlled with antibiotic therapy, we removed the catheter because we thought the episodes indicated refractory peritonitis.

Microscopic examination of parietal peritoneum obtained at the time of catheter removal revealed an almost intact peritoneum. The mesothelium was lined with a cell monolayer, and interstitial fibrosis or hyalinizing vasculopathy were not observed. We also found eosinophils in capillary lumina, probably as a result of eosinophilia (2340/ μL) induced by administration of antibiotics (Figure 1).

Because the peritoneal morphology was preserved, catheter reinsertion was performed in August 2012.

Figure 1 — Peritoneum at the time of catheter removal. Microscopic examination revealed unremarkable changes in parietal peritoneum. Peritoneal mesothelial cells covered the peritoneum in a single layer. Interstitial fibrosis was minimal, and vasculopathies were not observed. Hematoxylin and eosin stain.

The patient restarted PD and continued the technique without any trouble.

DISCUSSION

It is well reported that, after several years of PD, the peritoneum in nearly all patients is damaged and that the damage progresses with time on PD (1). Morphology change in the peritoneum includes denudation of peritoneal mesothelial cells, interstitial sclerosis, and hyalinizing vasculopathy (1). None of those changes were observed in our patient.

Evidence suggests that several factors contribute to peritoneal damage in PD patients, including chronic uremia, peritonitis, and long-term use of bioincompatible fluids (3). Recent approaches to improving PD technique survival in Japan have included introduction of combination therapy, especially in patients with anuria (5); prevention of peritonitis; and use of biocompatible dialysis solutions with low concentrations of GDPs and a neutral pH. We recently reported that those approaches preserved peritoneal morphology for more than 5 years of PD in 11 patients (6) and for 10 years of PD in 1 patient (7).

In the patient reported here, peritoneal function remained stable throughout PD therapy (his dialysate-to-plasma ratio of creatinine remained constant at approximately 0.5 – 0.6). Most regimens of combination therapy include 1 or 2 days of peritoneal rest (PD for 5 or 6 days per week, and hemodialysis 1 day per week) (5). Our patient had no peritoneal rest, and so we could for the first time directly see the effect of long-term continuous exposure to biocompatible dialysate.

CONCLUSIONS

We have observed a PD patient whose peritoneal morphology was almost intact after more than 9 years of PD with biocompatible dialysate. Longer and larger studies of such fluids are needed.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

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doi:10.3747/pdi.2012.00306