Poor ultrafiltration shortly after peritoneal dialysis initiation

Case

A 63-year-old male with end-stage renal disease due to nephroangiosclerosis started continuous ambulatory peritoneal dialysis on May 28, 2001 after 1 month of haemodialysis. A swan-neck Missouri coiled peritoneal catheter had been inserted under general anaesthesia 12 days earlier without complication. The patient’s medical history included long-lasting elevated blood pressure, hypercholesterolaemia, a triple aorto-coronary bypass together with aortic valve replacement in 1995, and atrial flutter since 1999. His maintenance therapy included warfarin, 1 mg or 2 mg o.d. on alternate days, calcium carbonate, 1 g t.d.s. and simvastatin, 20 mg o.d. Daily residual urine output was less than 500 ml.

At the first peritoneal dialysis exchange, a 5-cm fibrin clot was removed with the effluent (Figure 1). Next exchanges were clear. Four 2-l exchanges were performed daily (1.36% glucose bag once, 2.27% glucose bag twice during the day-time and one bag of icodextrin, during the night dwell, respectively). With this regimen, net ultrafiltration averaged 600 ml/24 h: ultrafiltration obtained with a 2.27% glucose bag was less than 250 ml while each icodextrin solution did not induce an ultrafiltration above 100 ml. After 48 h, the patient replaced, first, one of the 2.27% by a 3.86% glucose bag and secondly, the 1.36% glucose by a 2.27% glucose bag. Ultrafiltration over the next 3 days averaged 750 ml daily while patient’s weight increased progressively from 81 to 83.5 kg. Clinical examination did not show peritoneal leaks. One session of sequential ultrafiltration was performed and allowed the removal of 4 l. Peritoneal dialysis was restarted with one 2.27% and two 3.86% glucose bags twice during the day-time, and one icodextrin bag during the night dwell, without a significant improvement in ultrafiltration capacity (900 ml/day). After 4 days with this regimen, daily ultrafiltration was systematically insufficient to maintain the body weight below 82 kg.

Questions

What is your explanation for the poor ultrafiltration capacity observed in this patient?
What would be the tests you would perform to establish the diagnosis?
What would be your therapeutic approach?

Fig. 1. The 5-cm fibrin clot removed with the effluent of the first peritoneal dialysis exchange.
In this patient recently started on peritoneal dialysis, urine output was low (less than 500 ml/day) and fluid intake probably excessive. Daily net ultrafiltration obtained with one 1.36% glucose, two 2.27% glucose and one icodextrin bags was insufficient to maintain euvolaemia. Use of 3.86% hypertonic dialysate slightly improved net ultrafiltration but could not prevent weight gain. Interestingly, icodextrin did not significantly increase net ultrafiltration.

A major increase in the effective peritoneal surface area (and thus, an early dissipation of the osmotic gradient due to a rapid glucose absorption) is unlikely to explain the poor ultrafiltration capacity of this patient since (i) this condition mainly occurs in long-term peritoneal dialysis patients [1] and (ii) is characterized by the high potential of icodextrin to enhance ultrafiltration [2]. For the same reasons, a defect in the aquaporin function cannot be suspected [3]. Finally, there was no clinical reason to suspect the presence of peritoneal leaks or of an augmented fluid resorption from the peritoneal cavity into the plasma [4]. Poor ultrafiltration occurring in the beginning of peritoneal dialysis is rather due to a mechanical problem, either a dislodgement of the catheter or its obstruction by a fibrin clot [5,6]. To exclude the first hypothesis, the patient was given laxatives to mobilize the catheter, without improvement in ultrafiltration. Plain abdomen X-rays radiography showed adequate catheter location. Catheter obstruction was therefore suspected; its opacification with 5 ml of iodinated contrast medium (Omnipaque 350, Nycomed, Oslo, Norway), injected under fluoroscopy, showed the lack of progression of the dye product’s within the last 8 cm of the catheter while it diffused through lateral holes (Figure 2). Obstruction due to a fibrin clot was suspected as a clot had already been removed after the initial exchange. A heparin shot (2000 UI within 20 ml saline solution) did not improve catheter permeability. Therefore, a 2-h urokinase (Actosolv, Hoechst Marion Roussel, Brussels, Belgium) (75 000 UI) continuous infusion was performed. Immediate improvement of the catheter permeability could be seen with opacification of both lateral holes and total catheter length (Figure 3). To prevent recurrence, administration of 2500 UI heparin/bag was made for the next 2 days. Daily ultrafiltration then averaged 1200 ml using two 2.27% glucose and one 3.86% glucose bags daily and one icodextrin bag during the night dwell. Ultrafiltration obtained with icodextrin bags ranged between 140 and 755 ml.

Unexpectedly, the patient suddenly died at home 30 days later. He had been seen the day before at the peritoneal dialysis outpatient clinic and was well. A peritoneal equilibration test had not yet been performed.

The poor drainage capacity observed in this case was most likely due to the incomplete removal of the fibrin clot initially seen observed in the first dialysate bag. Catheter obstruction by fibrin clots has been previously reported [7–9]. Heparin administration seldom dissolves the clots. By contrast, urokinase infusion (with or without Fogarty, catheter desobstruction) has been proven to be more efficient [7]. An infusion accelerator or the use of endoluminal brush has also been successful to recanalize an obstructed catheter [10].
In summary, (i) poor ultrafiltration occurring shortly after peritoneal dialysis initiation should first suggest a mechanical problem rather than a membrane failure [11], (ii) the diagnosis of catheter obstruction is established by contrast opacification, (iii) catheter occlusion by a fibrin clot is best treated by urokinase infusion.

References

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