Successful Use of CAPD in a Patient with von Willebrand Disease

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Next Section

Editor:

Reasons for switching to peritoneal dialysis (PD) from hemodialysis include access failure, hypotension during hemodialysis due to fluid loss or heart failure, and neuropathy (1). We describe here a patient with end-stage renal disease (ESRD) and von Willebrand (VW) disease who was switched to PD due to bleeding complications during hemodialysis.

A 38-year-old man diagnosed with VW disease during childhood presented with ESRD. He underwent hemodialysis with a subclavian catheter for 2 months but experienced hemorrhage from the catheter site. Furthermore, we were unable to create an arteriovenous fistula. He was therefore started on continuous ambulatory PD (CAPD).

Two weeks after insertion of a Tenckhoff catheter we observed the formation of a fibrin clot that resulted in inflow and outflow failure. We therefore flushed the catheter with saline and heparin and, to prevent further catheter occlusion, we heparinized the dialysate at a dose of 300 units/L. Three months later, however, fibrin clot occlusions again occurred; these obstructions were managed with a Fogarty catheter. Five months after starting CAPD, a clot again formed in the PD catheter, resulting in outflow failure. However, the obstruction was not relieved by heparin flushes and a Fogarty catheter. We therefore replaced the catheter by laparotomy. To date, no further outflow failure has occurred but the patient has experienced recurrent episodes of hemoperitoneum. Instillation of heparin at a dose of 700 units/L in the dialysate was useful in preventing clot formation in the peritoneal catheter. However, the patient complained of epistaxis and gum hemorrhage following the intraperitoneal heparin administration. He had no history of peritonitis during CAPD. His CAPD regime consisted of two exchanges with 1.36% dextrose and two with 2.27% dextrose. Laboratory findings during PD showed hemoglobin concentration 10.3 g/dL, serum albumin 3.7 g/dL, weekly Kt/V 1.74, dialysate-to-plasma creatinine ratio 0.91, and normalized protein catabolic rate 0.95.

To our knowledge, this is the first description of a patient with both ESRD and VW disease managed with CAPD. During the first 6 months, he experienced complications from outflow

failure due to fibrin clot formation and several episodes of hemoperitoneum. CAPD was continued successfully; however, intraperitoneal heparin was required for prophylaxis of fibrin clot formation. We did not use tissue-type plasminogen activator for treatment of fibrin clots. Some studies have reported tissue-type plasminogen activator is useful for clots (2).

von Willebrand disease is the most common inherited bleeding disorder, with abnormal platelet function and reduced synthesis or activity of VW factor. Furthermore, uremia has been reported to be associated with abnormalities in VW factor (3) and with ineffective binding of VW factor to platelet membranes (4). Therefore, the co-occurrence of ESRD and VW disease makes renal replacement therapy difficult. Our patient had bleeding episodes following hemodialysis but CAPD was performed successfully, with good PD adequacy and nutrition status.

The patient complained of epistaxis and gum hemorrhage following the intraperitoneal heparin administration at a dose of 700 units/L. Although the transfer of heparin is minimal, Kaplan *et al*. reported a case of heparin-induced thrombocytopenia secondary to intraperitoneal heparin exposure (5). They speculated that absorption was via the peritoneal lymphatic system across the peritoneal membrane.

We conclude CAPD may be a suitable modality for renal replacement therapy for ESRD patients that also have bleeding tendency disorders such as von Willebrand disease.

Previous SectionNext Section

DISCLOSURES

The authors declare that no financial conflict of interest exists.

Previous Section

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