Aliskiren-Induced Chyloperitoneum in a Patient on Peritoneal Dialysis

Editor:

Chylous ascites is rarely observed in patients undergoing peritoneal dialysis (PD) (1,2), but it can be caused by medication. In 1993, Yoshimoto and colleagues reported manidipine-induced turbid peritoneal dialysate with mean dialysate triglyceride levels of $20 \pm 9 \text{ mg/dL}$ (3). Since then, several reports have been published, but most have been related to calcium channel blockers (4–6). Here, we present the first case report of chyloperitoneum in a PD patient induced by the direct renin inhibitor aliskiren.

A 58-year-old man with diabetic nephropathy had been receiving PD therapy since May 2007. He had 1 PDrelated peritonitis episode in September 2008. He had been receiving telmisartan, cilnidipine, spironolactone, trichlormethiazide, and furosemide for anti-hypertensive therapy for more than 2 years. Despite that regimen, his blood pressure was poorly controlled, and we prescribed aliskiren starting 1 November 2010.

Three days after starting aliskiren, the patient experienced cloudy effluent (Figure 1) and visited our hospital. He had no abdominal symptoms or findings to indicate peritonitis. Abnormal findings were not detected in the catheter tunnel or at the exit site. The dialysate white blood cell count was 2/mm³, and no organisms were detected with gram-stain and culture. Space-occupying lesions, such as malignancies or infectious lesions, were



Figure 1 — Cloudy dialysate effluent 3 days after the patient took aliskiren.

not observed on abdominal computed tomography imaging. Although his serum triglyceride concentration was not high (40 mg/dL), elevated levels of triglycerides were noted in dialysate (17 mg/dL). We therefore diagnosed chyloperitoneum related to aliskiren, and discontinued aliskiren. Fortunately, the cloudy effluent disappeared immediately after discontinuation of the aliskiren, and dialysate triglycerides became undetectable.

After a 2-week interruption, the patient was restarted on aliskiren because his blood pressure could not be controlled. Cloudy effluent reoccurred 16 days after resumption of the aliskiren. As in the previous episode, no evidence to support peritonitis was observed, and dialysate triglyceride levels increased to 19 mg/dL. Dialysate triglyceride levels in this patient were not much higher than levels previously reported (5), which might have been because of earlier detection of cloudy effluent.

Our patient was taking cilnidipine, and an interaction between aliskiren and cilnidipine may have induced chyloperitoneum by elevating cilnidipine blood concentrations. However, that scenario is unlikely, because several reports have found that aliskiren has little interaction with amlodipine, which is metabolized in the same manner that cilnidipine is (7,8). Based on our findings, aliskiren apparently induced chyloperitoneum in this patient.

Although details of the mechanism by which calcium channel blockers induce chyloperitoneum are unclear, numerous studies have reported that these agents prevent peritoneal fibrosis and increase ultrafiltration (9,10). A published report has also discussed the relationship between the direct renin inhibitor aliskiren and peritoneal membrane function (11). The present case is interesting, because it suggests that aliskiren has some effect on the peritoneum. It is also important to note that aliskiren may induce chyloperitoneum in some patients.

DISCLOSURES

The authors have no financial conflicts of interest to disclose.

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