LETTER TO THE EDITORS

Safety and efficacy of patiromer use with tacrolimus in kidney transplant recipients

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Dear Editors,

Hyperkalemia is a life-threatening condition that may affect kidney transplant recipients due to delayed graft dysfunction (DGF), kidney allograft failure, or as a side-effect of medications. Calcineurin inhibitors and trimethoprim are commonly prescribed to transplant recipients and can cause hyperkalemia [1].

Patiromer is a cation-exchange nonabsorbed potassium binder licensed for the treatment of hyperkalemia in adults [2]. The nonabsorbed polymer binds potassium in exchange for calcium, mainly in the distal colon where potassium has the highest concentration in the gastrointestinal tract [3]. Therefore, it increases the fecal excretion of potassium and results in a lower serum potassium level. Drug–drug interactions are a major concern with patiromer use in kidney transplantation. Prescribing information is to avoid oral medications at least 3 h prior to or after administration of patiromer; therefore, scheduling medications is challenging and may affect compliance [4].

With these concerns, the use of patiromer remains limited in kidney transplant recipients, despite its favorable profile compared to sodium polystyrene sulfonate that uses sodium as the exchange cation. We present two cases of successful use of patiromer with tacrolimus.

A 34-year-old Asian woman with end-stage renal disease (ESRD) secondary to IgA nephropathy received a second kidney transplant from a deceased donor in October 2016. The patient was highly sensitized (cPRA of 100%). Two weeks post-transplant, the patient was

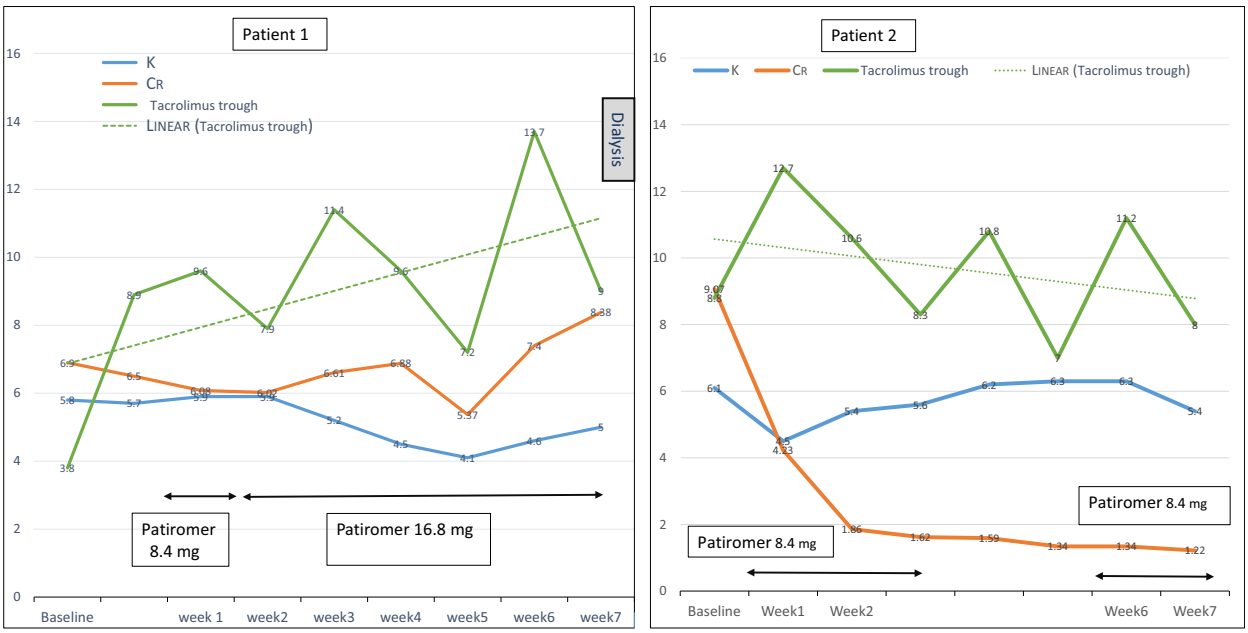

Figure 1 Levels of creatinine (mg/dl), tacrolimus trough (ng/dl), and potassium level (mEq/l) with patiromer use (mg).

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treated for acute antibody-mediated rejection. Tacrolimus LCP target trough levels were increased to 10.0–12.0 ng/ml. Kidney function improved, and creatinine went down to 2.5 mg/dl. Nine months later, the patient had acute cellular rejection. Her creatinine and potassium peaked at 6.0 mg/dl and 6.0 mEq/l, respectively. Phosphate was elevated at 6.7 mg/dl and was started on sevelamer. Dialysis initiation was discussed, but the patient refused dialysis. Patiromer 8.4 mg daily was prescribed (taken 4 h after tacrolimus dose) and titrated to 16.8 mg daily to treat hyperkalemia to allow time for the patient's readiness for dialysis initiation (Fig. 1). Within days of dosage adjustment, her hyperkalemia improved, and she was able to maintain a potassium level at 4.0–5.5 mEq/l until she started to develop uremic symptoms and was started on hemodialysis.

The second patient is a 58-year-old Caucasian man with ESRD secondary to diabetic nephropathy who received a deceased donor kidney in January 2018. Postoperative care was complicated by delayed graft function. Patient continued his pretransplant phosphate binder (sucroferric oxyhydroxide) for the first 10 days post-transplant with a phosphate level between 6 and 7 mg/dl. Despite improving allograft function, he remained hyperkalemic with a potassium level between 5.5 and 6.5 meq/l. Tacrolimus LCP target level was 8–12 ng/ml as the patient was highly sensitized (cPRA 100%). Patiromer 8.4 mg daily was started (taken 4 h after tacrolimus dose). Hyperkalemia resolved but returned once patiromer was discontinued (5.5–6.5 mEql/l). Patiromer was restarted, and the patient was maintained on patiromer until renal function improved without the need for adjustment of the tacrolimus dose (Fig. 1).

Our two cases demonstrate that patiromer use is effective in treating hyperkalemia and does not affect tacrolimus tough level if administered at least 3 h after the tacrolimus dose. In addition, there were no side effects reported with the use of patiromer in both patients. In the first case, decreasing the K level was beneficial in delaying the initiation of dialysis until the patient started to have symptoms and was able to pass the first year mark of transplant. In the second case, controlling potassium level was helpful in avoiding frequent emergency room visits in a patient with a DGF while maintaining tacrolimus in a therapeutic level in a highly sensitized patient. Our center does not check mycophenolic acid level; therefore, we are not able to comment whether patiromer has an impact on the absorption of mycophenolate mofetil. More studies are needed to examine the pharmacokinetics of tacrolimus with the use of patiromer.

Conflicts of interest

The authors declare no conflict of interests

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