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Largest Experience of Safety and Efficacy of Patiromer in Solid Organ Transplant

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Dear Editors,

Hyperkalemia, a potentially life-threatening electrolyte abnormality, is commonly seen in solid organ transplant (SOT) recipients. The predisposing factors are renal dysfunction and polypharmacy. Many medications routinely used posttransplant (calcineurin inhibitors, trimethoprim in trimethoprim-sulfamethoxazole, and renin-angiotensin system inhibitors) alter renal and cellular potassium handling.[1,2] Hyperkalemia leads to discontinuation or dose reduction of these medications making the recipients prone to infections, rejections, and healthcare expenditures. Patiromer, a potassium-calcium cation exchanger, has been widely used for hyperkalemia in the general population, but studies are limited in SOT.[3-5]

A retrospective, single-center, case series was conducted in adult SOT recipients who received patiromer for at least 1 month from January 2015 to August 2019. The primary endpoints were change in potassium levels between baseline and 4 and 12 wk, and difference in calcineurin inhibitors levels from baseline to 4 wk. Gastrointestinal adverse effects, electrolyte abnormalities, and patiromer discontinuation were assessed as secondary outcomes.

Baseline characteristics and outcomes of the 36 SOT who met inclusion criteria are listed in Table 1. There was a statistically significant reduction in mean potassium levels between baseline at both 4 wk (5.4, 5.0; 95% confidence intervals [CI], −0.63 to −0.16) and 12 wk (5.4,

TABLE 1.

Baseline characteristics and outcomes in SOT recipients

Baseline characteristics	n = 36
Type of organ transplant, n (%)	
Kidney	26 (72)
Liver	8 (22)
Kidney-pancreas	1 (3)
Lung	1 (3)
Comorbidities, n (%)	
Diabetes	17 (47)
Hypertension	34 (97)
Medications present, n (%)	
CNI	35 (97)
TMP-SMX	18 (66)
Diuretics	9 (33)
SPS	7 (19)
Etiology of hyperkalemia, n (%)	
Serum creatinine >2 mg/dL	18 (50)
Supratherapeutic CNI level	5 (14)
Constipation	2 (6)
Maintenance CNI, n (%)	
Tacrolimus	28 (78)
Cyclosporine	7 (19)
None	1 (3)
D posttransplant patiromer initiation, mean (95% CI)	322 (142, 502)
D to first potassium <5.2 mmol/L, mean (95% CI)	25 (10, 41)
Switch from another K lowering agent, n (%)	11 (31%)
Potassium level (mmol/L) at baseline, mean (95% CI)	5.4 (5.2, 5.6)
Potassium level (mmol/L) at 4 wk, mean (95% CI)	5.0 (4.9, 5.2)
Potassium level (mmol/L) at 12 wk, mean (95% CI) n = 26	4.8 (4.6, 5.1)
CNIs	
Tacrolimus level (ng/mL) at baseline, mean (95% CI)	6.9 (6.3, 7.5)
Tacrolimus level (ng/mL) at 4 wk, mean (95% CI)	8.3 (7.1, 9.6)
Cyclosporine level (C2, ng/mL) at baseline, mean (95% CI)	456 (161, 750)
Cyclosporine level (C2, ng/mL) at 4 wk, mean (95% CI)	Missing = 1 423 (266, 580)
Magnesium	
Hypomagnesemia (Mg <2 mg/dL) before patiromer, n (%)	26 (72)
Hypomagnesemia (Mg <2 mg/dL) at 4 wk, n (%)	Missing = 3 24 (67)
Magnesium level (mg/dL) before patiromer, mean (95% CI)	1.8 (1.7, 1.9)
Magnesium level (mg/dL) at 4 wk, mean (95% CI)	Missing = 3 1.7 (1.7, 1.9)
Reason for discontinuation of patiromer	
Resolution of hyperkalemia, n (%)	8 (22)
Cost, n (%)	7 (19)
Adverse effect, n (%)	3 (8)
Alternative K lowering agent, n (%)	2 (6)

CI, confidence interval; CNI, calcineurin inhibitor; SOT, solid organ transplant; SPS, sodium polystyrene sulfonate; TMP-SMX, trimethoprim-sulfamethoxazole.

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4.8; 95% CI, −0.84 to −0.27). By 4 wk, 72% were able to achieve goal potassium of <5.2 mmol/L and 73% at 12 wk. A statistically significant increase was seen in the mean tacrolimus levels from baseline to 4 wk (6.9, 8.3; 95% CI, −2.5 to −0.37). However, a decrease in tacrolimus dose was required in 32% of patients, 47% required no adjustment, and 21% required a dose increase. There was no significant difference in cyclosporine (C2) levels from baseline to 4 wk after patiromer initiation (95% CI, −232 to 367).

Most patients had no reported gastrointestinal adverse effects. Hypomagnesemia was present in 72% of patients before patiromer initiation and 67% at 4 wk. Magnesium supplements were received in 11% at baseline and 11% started on magnesium after patiromer initiation. Hypercalcemia was present in 11% of patients at 4 wk but not at baseline. Ninety-seven percent of recipients remained on the same dose of 8.4 g daily throughout the treatment. Resolution of hyperkalemia, followed by cost, adverse effects, and switch to alternative potassium lowering agents, were the reasons for discontinuation of patiromer (Table 1).

This case series demonstrated that patiromer is moderately effective in treating hyperkalemia and no major safety events occurred in this small population of SOT recipients. Although hypomagnesemia is a common adverse effect of patiromer, the incidence was similar before and after patiromer initiation and may require management with magnesium supplementation. Many baseline potassium values were <5.5 mmol/L at patiromer initiation; this represents real-life situations in which prescribers try other potassium lowering agents first before committing to a daily potassium lowering agent of patiromer. Finally, transplant providers should be aware of the possible increase of tacrolimus levels but empirical dosage adjustments are not recommended when starting this agent for hyperkalemia.

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