

## Fludrocortisone corrects tacrolimus associated hyperkalemia in renal transplant patients

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### Introduction

Hyperkalemic metabolic acidosis is commonly observed following kidney transplantation. This is often due to calcineurin inhibitors which are known to cause type 4 renal tubular acidosis either due to hyporeninemic hypoaldosteronism or due to direct effect on aldosterone responsive potassium secretion in the distal nephron.

### Methods

We report five post-renal transplant patients (5 males, all within 4-8 weeks post-transplant) on tacrolimus with hyperkalemia treated with daily doses of either 50 mcg (n=3) or 100 mcg (n=2) of fludrocortisone. None of the patients were on ACEI or ARB and all patients were on oral sodium bicarbonate at the time of starting fludrocortisone. We retrospectively collected data at three time points before and after administration of fludrocortisone on serum concentrations of sodium, potassium, bicarbonate, creatinine and tacrolimus as well as eGFR and blood pressure. We recorded emergency admissions and length of stay for treatment related to hyperkalemia. Data are presented as mean +/-SD) and analysed with a paired students t-test.

### Results

Serum potassium was  $6.3 \pm 0.3$  mmol/L and following fludrocortisone decreased to  $5.1 \pm 0.3$  mmol/L ( $p=0.0018$ ). Pre and post-fludrocortisone serum concentrations for venous bicarbonate were  $18.4 \pm 1.8$  mmol/L and  $20.4 \pm 2.0$  mmol/L ( $p=0.1079$ ); sodium  $135 \pm 1.6$  mmol/L and  $135 \pm 2.2$  mmol/L ( $p=0.8757$ ); creatinine  $184 \pm 12.2$   $\mu$ mol/L and  $155 \pm 10.6$   $\mu$ mol/L ( $p=0.0579$ ); eGFR  $39 \pm 3.4$  ml/min and  $47 \pm 4.2$  ml/min ( $p=0.0349$ ); blood tacrolimus levels  $9.8 \pm 2.1$  ng/mL and  $11.2 \pm 1.0$  ng/mL; blood pressure was  $133/69 \pm 12/9$  mmHg and  $129/70 \pm 8/6$  mmHg before and after fludrocortisone respectively. We were able to either reduce or stop sodium bicarbonate after starting fludrocortisone due to increase in serum bicarbonate levels. In two patients we measured urinary K excretion and serum chloride levels before starting fludrocortisone and in both of them urinary potassium was low ( $<20$ mmol/l) and both demonstrated mild hyperchloremic normal anion gap metabolic acidosis.

Before starting fludrocortisone there were 6 episodes of serum potassium  $\geq 6.5$  mEq/L. Three patients required either renal day case or A&E visits/hospital admissions ( $n = 6$ ) for management of hyperkalaemia with length of stay of between 1-3 days. Following fludrocortisone administration there was 1 admission (length of stay = 1 day) for hyperkalemia. Reduction in potassium levels to 'safe levels' were noted within 24-48h of starting Fludrocortisone.

### Discussion

Hyperkalemia was a significant problem in patients described in this report requiring hospital visits for treatment and it occurred with tacrolimus levels in target range. Treatment of hyperkalemic metabolic acidosis with low dose fludrocortisone resulted in normalization of serum potassium levels. There were no adverse effects on blood pressure, serum sodium levels or clinical evidence of fluid retention after commencing fludrocortisone. Instigation of fludrocortisone prevented emergency admissions for treatment

of hyperkalemia and allowed the clinicians to run adequate tacrolimus levels. Fludrocortisone can be a cheap, safe and effective option for the treatment of hyperkalemia in renal transplant patients on tacrolimus.