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Effect of Cinacalcet for treatment of hypercalcemic hyperparathyroidism after kidney transplantation

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Effect of Cinacalcet for treatment of hypercalcemic hyperparathyroidism after kidney transplantation **Introduction:** Hypercalcemia after kidney transplantation(KT) is frequently caused by secondary hyperparathyroidism. Several medical therapies have been tried for the treatment of secondary hyperparathyroidism and hypercalcemia. We reviewed our experience of cinacalcet treatment in post KT patients with hypercalcemic hyperparathyroidism. **Methods:** From October 2013 to August 2016, eight KT patients with hypercalcemic hyperparathyroidism had been treated with cinacalcet (daily dose of 25mg to 50mg) for over 12 months in our institute. We analyzed the efficacy of cinacalcet with serum calcium, phosphorus, intact parathyroid hormone, alkaline phosphatase and creatinine for a period of 12-months. **Results:** During the treatment period, all patients showed significant reduction in the serum calcium concentration (11.8±0.50 to 10.55±0.60 mg/dL, p=0.048). Intact parathyroid hormone levels were also decreased but statistically not significant(242.8±113.96 to 177.6±39.02 pg/mL, p=0.110). Serum phosphorus(2.46±0.39 to 2.97±0.61 mg/dL, p=0.245), alkaline phosphatase(643.1±383.73 to 428.0±129.78 U/L, p=0.136) and creatinine(1.08±0.37 to 1.12±0.33 mg/dL, p=0.451) levels were not changed. None of eight patients reported any clinical side effects. **Conclusion:** This preliminary experience suggests that cinacalcet may be useful in the treatment of post KT hypercalcemic hyperparathyroidism without evidence of renal function deterioration or significant side effects.

Figure 1. Levels of biomarkers after cinacalcet treatment for 12 months of follow up. Values are shown as mean ± SD

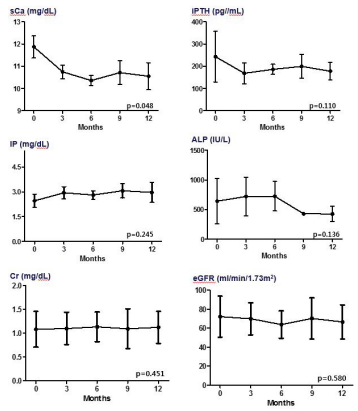

Table 1. Baseline characteristics of patients at begining of cinacalcet therapy

Patient	Sex	Age	Months after KT	Immunosuppression	eGFR	Etiology	sCa	PTH
1	m	34	34	BSX+MPD+MMF+FK	64	undetermined	11.7	192
2	f	63	23	ATG+RTX+MPD+MMF+FK	75	hypertensive	11.7	206
3	f	45	26	ATG+MPD+FK	39.7	undetermined	12.8	426
4	f	48	3	BSX+MPD+MMF+FK	106.3	undetermined	11.5	232
5	f	65	9	BSX+MPD+MMF+FK	72.9	hypertensive	12.2	407
6	f	57	7	BSX+MPD+MMF+FK	94.6	undetermined	12.2	222
7	m	60	5	BSX+MPD+MMF+FK	50.7	undetermined	11.7	128
8	m	45	12	BSX+MPD+MMF+FK	73.3	ADPKD	11.2	130

KT, Kidney transplantation; BSX, basiliximab; MPD, methylprednisolone; MMF, mycophenolate mofetil; FK, tacrolimus; ATG, anti-thymocyte globulin; eGFR, estimated glomerular filtration rate (in ml/min/1.73m^2); sCa, serum calcium level (in mg/dL); PTH, serum parathyroid hormone level (in pg/mL)

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Serum retinol binding protein 4 is associated with type 2 diabetic nephropathy

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Background/Aims: Serum retinol binding protein 4(RBP4) is known to be associated with insulin resistance, obesity, and type 2 diabetes. Whether it can be used as a biomarker of diabetic complications remains undetermined. **Methods:** We investigated the association of RBP4 level in type 2 diabetic nephropathy. Patients were divided into 4 groups; 1) normal non-diabetic control, 2) normoalbuminuric diabetic group, 3) microalbuminuric diabetic group and 4) macroalbuminuric diabetic group. Furthermore, we examined the RBP4 effect on renal specific cells. **Results:** Total four groups including non-diabetic controls showed similar basal characteristics in age, sex, BMI, eGFR, Hb, serum albumin, CRP, lipid levels. Interestingly, patients with macroalbuminuria showed higher serum RBP4 level compared to patients without diabetic nephropathy or with normoalbuminuria or with microalbuminuria. It is notable that RBP4 level was also increased in patients with lower creatinine clearance.In univariate and multiple regression analysis, 24-h albuminuria and systolic blood pressure were positively correlated with logRBP4 level. BMI was negatively correlated with that level. In case of other vascular complications of type 2 diabetes, RBP4 level was not increased in the patients with retinopathy and peripheral neuropathy. However, the patients with higher RBP4 had significantly increased risk of cardiovascular, peripheral arterial and cerebrovascular disease. Podocytes expressed their uptake of RBP4 strongly compared to other renal cells including mesangial and tubular cells. Treatment of RBP4 into podocytes induced the activation of insulin receptor substrate-1(IRS-1). **Conclusions:** RBP4 may contribute to pathogenesis of diabetic nephropathy as well as used as a biomarker of diabetic nephropathy.