ALBUMINURIA DURING AND AFTER PARICALCITOL TREATMENT IN CHRONIC KIDNEY DISEASE PATIENTS

[1]Ekart Robert, [2]Hojs Nina, [2]Bevc Sebastjan, [2]Hojs Radovan

Department of [1]Dialysis, [2]Nephrology, Clinic for Internal Medicine, University Clinical Centre Maribor, Maribor, Slovenia

INTRODUCTION AND AIMS: Albuminuria is associated with chronic kidney disease (CKD) progression, cardiovascular disease and death. Reduction of albuminuria is one of the goals in the management of CKD patients. Therefore the aim of our study was to evaluate the effect of paricalcitol treatment on albuminuria in non-dialysis CKD patients.

METHODS: Non-dialysis CKD patients with secondary hyperparathyroidism and albuminuria were included in our study. All patients were examined at the beginning of our study, after 6 months of paricalcitol treatment (1 μg/day), 3 and 6 months after drug withdrawal. Three patients stopped treatment after 3 months because of low parathyroid hormone. We measured urine albumin/creatinine ratio (UACR), 24-hour albuminuria (24hA) and cystatin C. 24-hour ambulatory blood pressure monitoring was done at the beginning, after 6 months of treatment and 6 months after drug withdrawal. 24-hour mean ambulatory blood pressure (MAP) was used for statistical analysis. Fixed doses of angiotensin converting enzyme inhibitors and/or angiotensin receptor blockers and/or statins were kept for 3 months before and during the study. None of the patients received a vitamin D analogue at least 1 month before the beginning of the study.

RESULTS: Forty-two non-dialysis CKD patients (29 men, 13 women), aged 62.3±11.9 years (range 31-84 years), all of white race, were included in our study. To get an approximately normal distribution, laboratory data for albuminuria were log transformed. Paricalcitol treatment significantly reduced UACR (p=0.001) and 24hA (p=0.001) (figure 1 and 2). Three months after drug withdrawal only UACR (p=0.023) significantly increased, while 6 months after drug withdrawal UACR (p=0.014) and 24hA (p=0.032) significantly increased compared to albuminuria at the end of paricalcitol treatment. Paricalcitol treatment (p=0.443) and withdrawal (p=0.239) did not affect MAP. Cystatin C significantly increased during treatment (p=0.001), but 3 (p=0.312) and 6 (p=0.311) months after drug withdrawal it stayed stable.

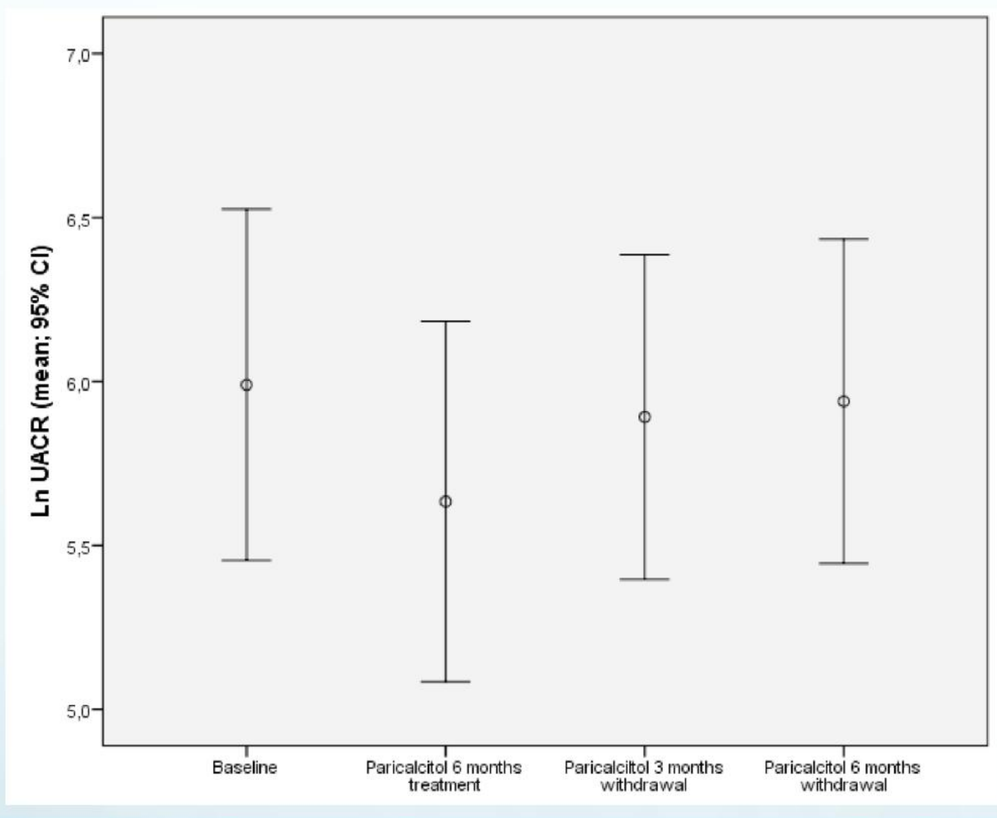

Figure 1. Log transformed data for urine albumin/creatinine ratio (LnUACR) at baseline, after 6 months of paricalcitol treatment, 3 and 6 months after paricalcitol withdrawal.

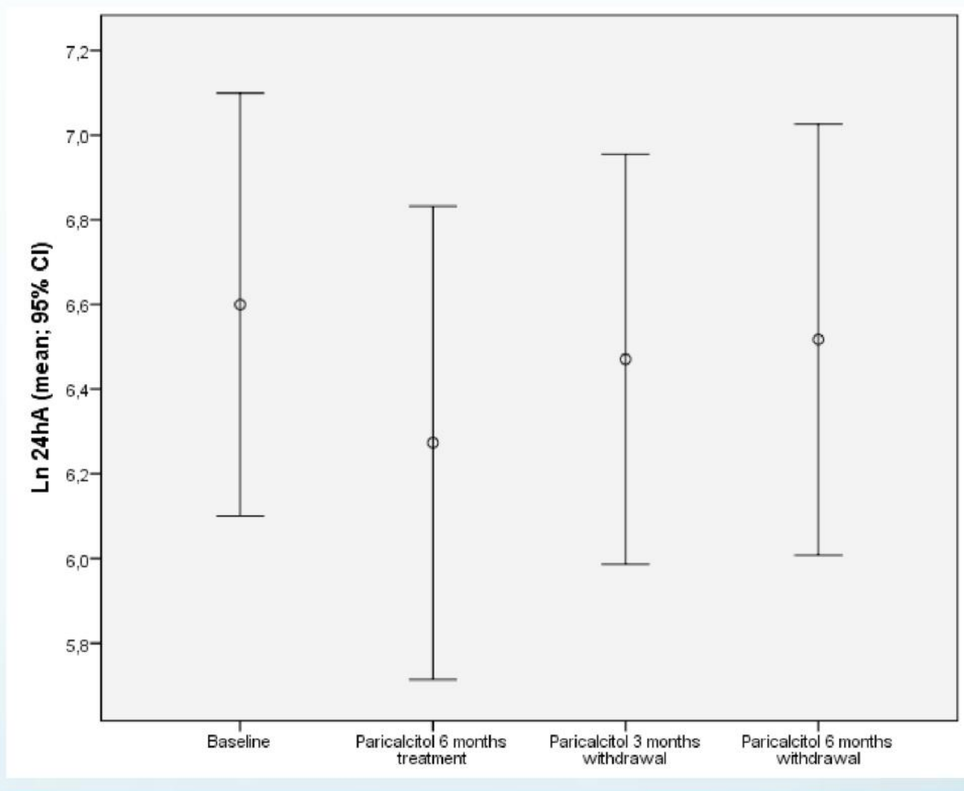

Figure 2. Log transformed data for 24-hour albuminuria (Ln24hA) at baseline, after 6 months of paricalcitol treatment, 3 and 6 months after paricalcitol withdrawal.

CONCLUSIONS: Paricalcitol treatment (1 μg/day) in non-dialysis CKD patients significantly reduced albuminuria (UACR, 24hA). Six months after drug withdrawal UACR and 24hA increased. Paricalcitol treatment did not affect MAP. Cystatin C significantly increased during treatment, after drug withdrawal it stayed stable.

REFERENCES:

• Agarwal R, Acharya M, Tian J, et al. Antiproteinuric effect of oral paricalcitol in chronic kidney disease. Kidney Int 2005;68(6):2823-8.
• Alborzi P, Patel NA, Peterson C, et al. Paricalcitol reduces albuminuria and inflammation in chronic kidney disease: a randomized double-blind pilot trial. Hypertension 2008;52(2):249-55.
• de Zeeuw D, Agarwal R, Amdahl M, et al. Selective vitamin D receptor activation with paricalcitol for reduction of albuminuria in patients with type 2 diabetes (VITAL study): a randomised controlled trial. Lancet 2010;376(9752):1543-51.
• De Nicola L, Conte G, Russo D, et al. Antiproteinuric effect of add-on paricalcitol in CKD patients under maximal tolerated inhibition of renin-angiotensin system: a prospective observational study. BMC Nephrol 2012;13:150.
• Hojs N, Bevc S, Balon BP, et al. Paricalcitol reduces proteinuria in non-dialysis chronic kidney disease patients. Ther Apher Dial 2013;17(4):368-72.

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Nina Hojs
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