P0176 **REGULATION OF OXALATE HOMEOSTASIS BY OXALATE-DEGRADING ACTIVITY IN FECAL MICROBIOTA IN DIALYSIS PATIENTS**

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Background and Aims: Despite evidence suggesting that a lack of fecal oxalate-degrading bacteria colonization is a risk factor for calcium oxalate stone formation, little is known about the oxalate-degrading activity (ODA) in fecal microbiota in end-stage renal disease (ESRD) patients. In addition, to date, there has been a general lack of research on the effect of fecal ODA on oxalate homeostasis in dialysis patients.

The present pilot cross-sectional study was performed to compare the oxalate homeostasis profiles depending on ODA in fecal microbiota in ESRD patients.

Method: The data of a cross-sectional pilot study examining ODA in fecal microbiota, plasma oxalate concentration (POx) and urinary oxalate excretion (UOx) in 32 ESRD patients were represented in this study. Among the patients, there were 21 hemodialysis (HD) patients and 11 peritoneal dialysis (PD) patients. The average age of the patients was 52.5 [39; 65] years.

The redoximetric titration with KMnO4 was adopted to evaluate total ODA in fecal microbiota. The results were expressed in % oxalate degradation per 0.01 g of feces. POx concentration and UOx excretion were measured spectrophotometrically using a commercially available kit (MAK-315, Sigma, Spain) and an oxalate oxidase/peroxidase reagent (BioSystems, Spain), respectively. Predialysis plasma samples were collected from HD patients.

For further analysis, the patients were allocated to 2 groups according to ODA in feces. Group 1 included the patients with $\geq$ 1 % oxalate degradation per 0.01 g of feces. Group 2 included the patients with negative ODA ($\leq$ 0 % /0.01 g).

For the statistical analysis, we used the nonparametric Kruskal-Wallis test. The median (Me) and interquartile ranges [Q25; Q75] were calculated. The Spearman test was used for the correlation analysis. Univariate logistic regression analysis was used for predicting hyperoxalemia. All statistical analyses were performed using MedCalc.

Results: ODA in fecal microbiota ranged from -23 to 24 %/0.01 g of feces in ESRD patients and was statistically higher in HD patients compared with PD patients (3.2 [-0.5; 16] vs -4 [-6.5; 6.2], p=0.05). Negative ODA in fecal microbiota ($\leq$ 0 % /0.01 g) was observed in 5/21 (23.8%) HD patients and 7/11 (63.6%) PD patients (χ^2=3.9, p=0.04). Consequently, it might be associated with the negative effects of peritoneal dialysis solution.

High POx concentration and low UOx excretion were diagnosed in patients with negative ODA in fecal microbiota (Group 2): 30.7 [25.5; 41.5] vs 50 [43.3; 75.5] µmol/L, p=0.01 and 60.9 [51; 65] vs 34.2 [24.4; 39] mg/d, p=0.0002, respectively (Fig. 1). Fecal ODA was directly associated with daily UOx excretion (r=0.85; p<0.0001) (Fig. 2) and had an inverse correlation with POx concentration (r=-0.36; p=0.04) (Fig. 3). In univariate logistic regression analysis, negative fecal ODA was determined as an independent risk factor for high POx concentration (OR: 40; 95% CI: 4.8-331, p<0.0001).

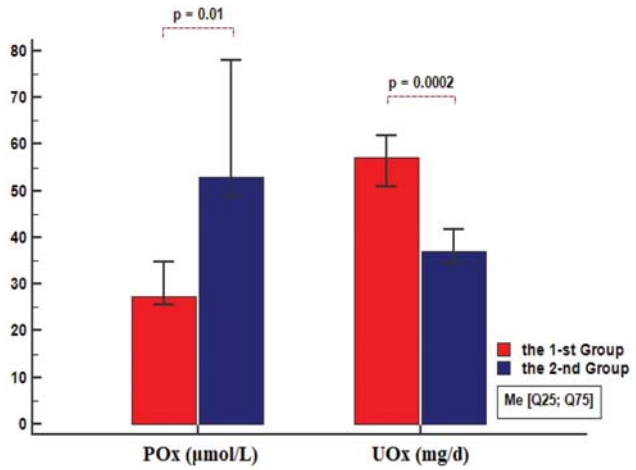

Figure 1: The concentration of plasma and urinary oxalate depending on ODA in ESD patients.

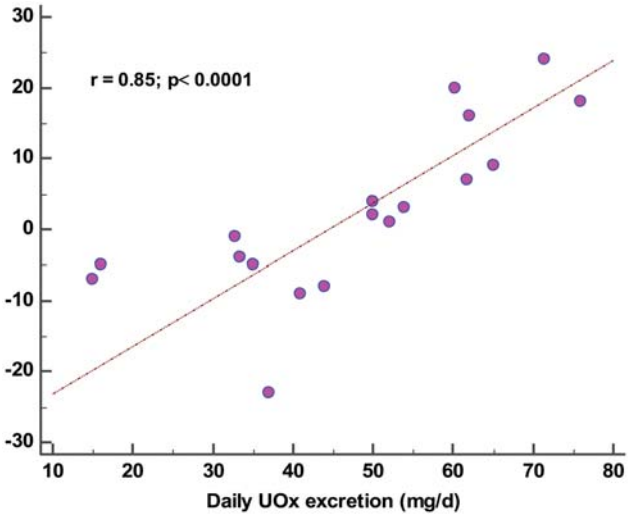

Figure 2: The association between ODA and daily UOx excretion in ESRD patients.

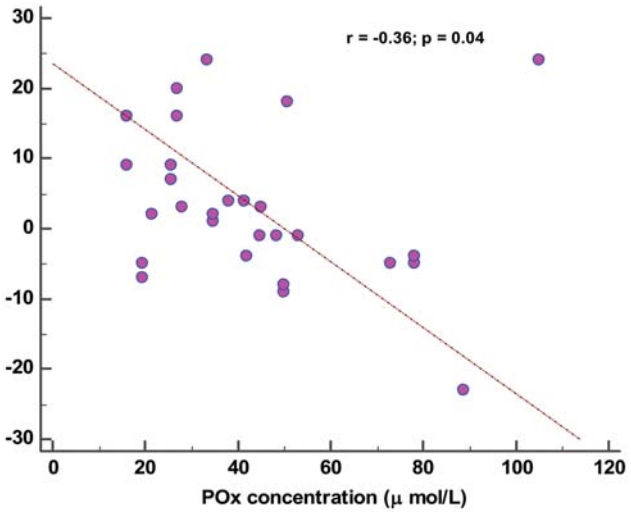

Figure 3: The association between ODA and POx concentration in ESRD patients.

Conclusion: Our pilot cross-sectional study firstly demonstrated a close association between ODA in fecal microbiota and oxalate homeostasis in ESRD patients: less ODA in fecal microbiota was, higher POx concentration and lower UOx excretion occurred. We suppose that the potential significance of our findings provides preliminary information on the feasibility and necessity of further research in this area.