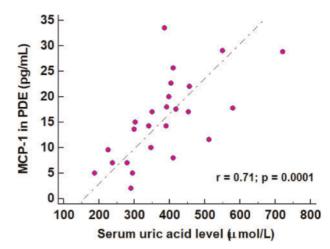
Abstracts



CONCLUSIONS: The results of this study have provided the preliminary evidence that hyperuricemia is significantly associated with dialysate TNF- α and MCP-1. Further, well-designed clinical trials are required to establish the impact of high level of sUA on intraperitoneal inflammation in PD patients.

FP575 URIC ACID INDUCES INTRAPERITONEAL INFLAMMATION IN PERITONEAL DIALYSIS PATIENTS

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INTRODUCTION: It has been shown a contributory role of elevated serum uric acid (sUA) to metabolic syndrome, insulin resistance and cardiovascular diseases which in turn are closely associated with peritoneal dialysis (PD) outcomes. Moreover, the association between high level of sUA and PD technique failure has been recently demonstrated. But, in fact, little is known about the underlying mechanism of hyperuricemia impact on PD adequacy.

The present study was undertaken to investigate the association of serum UA with intraperitoneal inflammation in PD patients.

METHODS: In this observational cross-sectional study, there were analyzed the associations between sUA concentration and intraperitoneal production of tumor necrosis factor- α (TNF- α) and monocyte chemoattractant protein -1 (MCP-1) in 25 non-diabetic PD patients (average age 51.6 ± 11.9). All patients had been undergoing continuous ambulatory PD (CAPD) for more than 3 months (median was 33.8 [22.3-54] months).

Concentration of TNF- α and MCP-1 in peritoneal dialysis effluent (PDE) was analyzed using ELISA. Hyperuricemia was defined as sUA concentration above 416.4 μ mol/L or 7 mg/dL (it was measured using automated enzymatic methods).

For the statistical analysis, we used the Student's t-test and Pearson's rank correlation test. The average values (M) and standard deviation (SD) were calculated according to a normal distribution. All statistical analyses were performed using MedCalc.

RESULTS: PDE levels of TNF- α and MCP-1 in the patients with hyperuricemia were significantly higher compared with the normal sUA level patients: 9.8 \pm 3.2 vs 6.0 \pm 2.6 pg/ml, p =0.006 and 21.3 \pm 6.3 vs 13.6 \pm 8.4, p = 0.046 pg/ml, respectively. sUA concentration was directly correlated with PDE levels of TNF- α (r = 0.79, p <

such concentration was directly correlated with FDE levels of TNF- α (r = 0.79, p < 0.0001) and MCP-1 (r = 0.71, p = 0.0001).

