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Higher serum level of indoxyl sulfate is not associated with increased abdominal aorta calcification in patients on maintenance hemodialysis

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Background and Aims: Vascular calcification is strongly associated with an increase in all-cause and cardiovascular mortality, especially in patients with and stage renal disease. Results of numerous experimental studies have shown direct toxic effect of microbial-derived uremic toxins on vascular endothelial and smooth muscle cells which may contribute vascular calcification. Meanwhile, the effects of these metabolites on vascular remodeling in patients receiving dialysis treatment remains largely unclear. Here we aimed to investigate the relationship between serum level of the uremic toxin indoxyl sulfate and severity of abdominal aorta calcification (AAC) in patients on maintenance hemodialysis.

Method: This single-center, cross-sectional study included eighty (40 men, average age – 59,7±11,8 years) clinically stable patients receiving hemodialysis treatment for 52(21,3-120) months. 13(16,3%) participants had history of smoking, 27(33,8%) were diabetic, 63(78,8%) had high blood pressure, 17(21,3%) – previous history of ischemic heart disease, including 5(6,3%) suffered a myocardial infarction. Severity of AAC was determined by lateral lumbar radiography with Kauppila scoring. The total AAC score ranged from 0 to 24 points, and a higher AAC score indicated a more pronounced degree of calcification. Routine laboratory data were collected before hemodialysis session after fasting for 12 hours. Serum concentration of indoxyl sulfate was assessed by ELISA using commercially available kit (Cloud-Clone Corp., USA). Statistical analysis was performed with IBM SPSS Statistics 26 program. Non-parametric Spearman’s rank correlation method was used to evaluate the associations between the studied parameters. Value of p <0.05 was considered statistically significant in all tests.

Results: Median serum indoxyl sulfate was 2,1(1,4-3,0) μmol/l (the same parameter in relatively healthy individuals without impaired renal function is 0,1(0-0,3) μmol/l, p=0.001). Serum levels of calcium (Ca) was 2,27(2,1-2,5) mmol/l, ionized calcium (Ca++) – 1,15(1,1-1,3) mmol/l, phosphate (P) – 1,6(1,3-2,0) mmol/l, intact parathyroid hormone (iPTH) – 303(162,2-492,8) pg/ml, total cholesterol (TH) – 4,5(3,5-5,4) mmol/l, low-density lipoprotein (LDL) – 2,6(2,0-3,3) mmol/l. AAC score in hemodialysis participants was 4,5(0,0-9,0) and significantly correlated with age (r=0,693; p=0.001), Ca (r=0,208; p=0,04) and iPTH (r=0,418; p=0,021) but not with other indicators, including indoxyl sulfate concentration (r=0,164; p=0,147).

Conclusion: Our preliminary results do not allow to consider the elevated level of indoxyl sulfate as a factor contributing to increased AAC in patients on maintenance hemodialysis. Further prospective studies in larger populations should clarify the true role of indoxyl sulfate in vascular remodeling in individuals with chronic kidney disease.