PHAGOCYTIC ACTIVITY AND OXIDATIVE BURST OF NEUTROPHILS AND MONOCYTES IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Introduction:

Bacterial infections are a common cause of morbidity and mortality in patients on dialysis treatment. The key role in the first line of antimicrobial immunity play phagocytic cells - neutrophiles (N) and monocytes (M). Various functions of phagocytes were the subject of many studies and the abnormal functions of the cells were reported. However most of the studies concerned the patients with end stage renal disease on renal replacement therapy, making difficult to distinguish the effect of factors associated with impaired renal function from those caused by treatment procedures [1-5].

Objectives:

The aim of the study was to evaluate the phagocytic activity and oxidative burst of N and M in patients at different stage of chronic kidney disease (CKD) treated conservatively compared to the control group.

Methods:

The study involved 48 patients (31 M, 17 F) with CKD with a mean serum creatinine 3.62 ± 1.8 mg/dL (eGFR 27,86 ± 4,24 ml/min) who were treated conservatively. The control group consisted of 43 healthy volunteers.

The quantitative assessment of phagocytes functions was determined by Phagotest and Phagoburst using flow cytometry. The data were presented as percentage [%] of N and M phagocyting opsonised $_{(OPS)}$ and nonopsonised $_{(NOPS)}$ E.coli bacteria, and the mean fluorescence intensity [MFI], correlating with the number of bacteria absorbed by phagocytic cell. The oxidative burst after stimulation with E.coli bacteria was determined as percentage phagocytes producing reactive oxygen species $_{(ROS)}$ and the enzymatic activity of cells [MFI].

Results:

Tab.1. The ability of M and N to phagocyte opsonised E.coli bacteria in CKD patients and the control group.

	CKD	Controls	p
N_{OPS} [%]	86.87±17.5	78.56±18.74	p <0.05
N_{OPS} [MFI]	424.36±162.1	334.68±112.1	p<0.05
M_{OPS} [%]	52.98±20.95	49.64±19.39	ns
M_{OPS} [MFI]	192.6±101.05	122.8±75.92	p<0.01

Tab.2. The oxidative burst of M and N in CKD patients and the control group.

	CKD	Controls	p
N_{ROS} [%]	90.85±14.74	89.88±18.3	ns
N_{ROS} [MFI]	57.03±27.23	59.99±21.72	ns
M_{ROS} [%]	75.56±17.15	74.84±17.45	ns
M_{ROS} [MFI]	24.55±6.24	28.17±7.13	p<0.01

The N of patients with CKD were characterized by significantly higher ability to phagocyte opsonised E. coli in comparison to the control group. The percentage of M phagocyting opsonised bacteria did not differ from the control group, but the M from CKD patients were absorbing bacteria more actively (Tab. 1).

The phagocytosis of nonopsonised bacteria by N and M was not different between the groups.

The oxidative burst of N in CKD patient did not differ from the control group, but M showed lower enzymatic activity in comparison to healthy subjects (Tab. 2).

Conclusions:

There was no evidence of impaired phagocytic activity of N and M in nondialysed CKD patients in relation to the control group. The oxidative burst of M in CKD was decreased in comparison to the healthy subjects, which may partly explain the increased incidence of bacterial infections in these group of patients.

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