IMMUNOSUPRESSION, ENDOTHELIAL DYSFUNCTION, PROTEINURIA AND LEFT VENTRICULAR STRUCTURE IN PATIENTS WITH GLOMERULONEPHRITIS

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INRODUCTION AND AIM

Endothelial dysfunction could be related to the limited availability of nitric oxide (NO). NO is synthesized with the participation of NO synthase which activity is inhibited by asymmetric dimethylarginine (ADMA).
The aim of this study was to assess the relationship between plasma concentration of ADMA and proteinuria and kidney and heart function in patients with chronic glomerulonephritis (ChGN) undergoing medical treatment including RAAS blockade.

The type of treatment	Total(n-37)	Group A (n-17)	Group B (n-20)
ACE inhibitors	15 (40%)	6 (35%)	9 (45%)
Sartans	4 (11%)	0	4 (20%)
Double blockade of RAA	15 (40%)	11 (65%)	4 (20%)
Statins	9 (24%)	4 (24%)	5 (25%)
Diuretics	16 (43%)	1 (6%)	15 (75%)
Glucocorticosteroids	19 (51%)	0	19 (95%)
Cyclophosphamide	4 (11%)	0	4 (20%)
Cyclosporin	1 (3%)	0	1 (5%)

Table 1 The treatment schedule.

parameter	Mean ±SD 1 visit	Mean ±SD 2 visit	Mean ±SD 3 visit	P
ADMA (µmol/l)	n-37 0.77 ±0.26 A 0.76 ±0.21 B 0.78 ±0.30	n-37 0.56 ±0.20 A 0.54 ±0.18 B 0.59 ±0.21	n-37 0.40 ±0.14 A 0.42 ±0.14 B 0.38 ±0.15	<0.001* <0.001* <0.001*
Creatinine (mg/dl)	n-37 1.14 ±0.37 A 1.20 ±0.45 B 1.09 ±0.29	n-37 1.17 ±0.44 A 1.28 ±0.59 B 1.09 ±0.28	n-37 1.15 ±0.52 A 1.25 ±0.65 B 1.06 ±0.36	0.793 0.500 0.376
eGFR (MDRD) ml/min/1.73m^2	n-37 80.85 ±26.57 A 75.12 ±26.47 B 81.4 ±28.65	n-37 80.74±26.85 A 73.80 ±27.61 B 80.95 ±23.56	n-37 81.47±27.17 A 77.56 ±29.70 B 85.56 ±28.45	0.126 0.328 0.230
eGFR (CKD-EPI) ml/min/1.73m^2	n-37 83.80 ±26.1 A 77.82 ±24.97 B 84 ±27.55	n-37 83.68±26.78 A 76.47 ±27.99 B 84.84 ±24.76	n-37 84.44±27.03 A 80.38 ±29.81 B 88.11 ±28	0.055* 0.302 0.102
Daily protein urinary excretion (g/d)	n-37 4.13 ±4.34 A 1.53 ±0.96 B 6.3 ±4.87	n-37 1.76 ± 3.23 A 1.23 ± 0.88 B 2.19 ± 4.26	n-37 1.55 ±1.91 A 1.01 ±0.86 B 2.04 ±2.44	<0.001* 0.005* <0.001*

Table 2 The charge of biochemical parameters in 12 months of observation.

MATERIAL AND METHODS

The study included 37 patients (26M, 11F) at mean age 38.5+/- 11,5 (range 22- 64) years, divided into group A and group B, depending on the treatment. Group A was treated with renin angiotensin aldosterone system (RAAS) blockers. Group B was treated both with RAAS blockers and immunosuppressive drugs. Details in table 1.
In studied patients (groups A and B) such parameters as: ADMA, creatinine level and urine protein excretion were evaluated before and after 6 and 12 months.

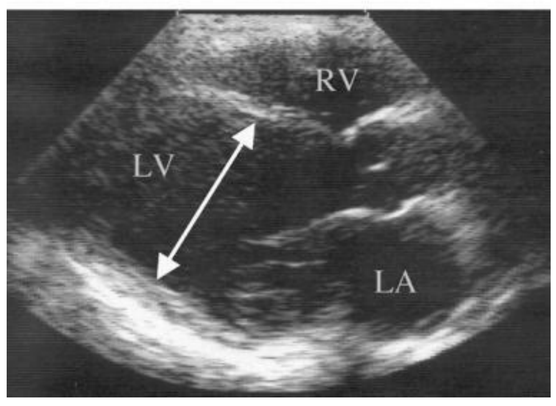
Additionally echocardiography (ECHO) was performed, where the structure of the left ventricle (LV) was assessed using the following parameters: IVSD (interventricular septal end diastole diameter), PWD (posterior wall thickness at end-diastole), LVEDD (left ventricle end diastole diameter), LVMI (left ventricle mass index), RWT (relative wall thickness).

parameter	Mean ±SD 1 visit	Mean ±SD 2 visit	Mean ±SD 3 visit	P
IVSD (mm)	n-37 10,54 ± 1,44 A 10,43 ± 1,16 B 10,64 ± 1,67	n-37 10,5 ± 1,36 A 10,33 ± 1,09 B 10,67 ± 1,54	n-37 10,76 ± 1,57 A 10,64 ± 1,43 B 10,87 ± 1,71	0,173 0,376 0,327
PWD (mm)	n-37 10,3 ± 1,11 A 10,18 ± 1,27 B 10,41 ± 0,96	n-37 10,15 ± 1,32 A 10,13 ± 1,41 B 10,16 ± 1,29	n-37 10,17± 1,35 A 9,88 ± 1,27 B 10,42 ± 1,41	0,213 0,409 **0,028****
LVEDD	n-37 45,0 ± 4,39 A 44,49 ± 5,12 B 45,47 ± 3,71	n-37 46,36 ± 4,33 A 44,38 ± 5,17 B 47,81 ± 2,95	n-37 47,07 ± 4,44 A 45,62 ± 4,22 B 48,36 ± 4,35	**0,078*** 0,257 0,157
LVMI (g/m^2)	n-37 91,58 ± 22,31 A 85,59 ± 21,08 B 89,53 ± 23,23	n-37 90,84 ± 22,0 A 80,64 ± 16,76 B 92,37 ± 19,5	n-37 91,11 ± 22,03 A 83,13 ± 18,65 B 98,56 ± 24,94	0,200 0,435 **0,081***
RWT	n-37 0,45 ± 0,06 A 0,47 ± 0,08 B 0,46 ± 0,05	n-37 0,44 ± 0,06 A 0,45 ± 0,08 B 0,42 ± 0,05	n-37 0,44 ± 0,06 A 0,44 ± 0,06 B 0,43 ± 0,06	**0,008*** 0,131 **0,003***

Table 3 The charge of echocardiographic parameters in 12 months of observation.

* ANOVA variation – model assumes linear function
** ANOVA variation – model assumes quadratic function

2D Echo COCM Diastole



RESULTS

In both studied groups during the whole period of observation statistically significant reduction of ADMA (0.77 vs 0.4 µmol/l; $p<0.05$) was noticed. Renal function remained stable during the whole study period. In both study groups a statistically significant reduction of daily urinary protein excretion was noticed (table 2).
ECHO parameters before the study were within the range (table 3). Although significant changes in such parameters as: LVMI, PWD and RWT were noticed, they remained within the reference ranges during the study period. There was not significant correlation between the concentration of ADMA in the plasma and ECHO LV structure parameters.

CONCLUSIONS

1. The significant reduction of ADMA serum concentration and proteinuria was noticed in both group during the study period, independently of the application of immunosuppresion.
2. The essential changes of ECHO parameters were observed and they still remained within normal range.

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