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KIDNEY LESIONS IN HIV-INFECTED PATIENTS

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Introduction. *HIV* prevalence is one of the most important issues of contemporary medicine. Over a 30-year history of this disease more than 75 million people have been infected with HIV, nearly 30 million adults and children of died. In the future decades, its significance in world premature mortality rates continues to rise. The objective of the study was to establish clinical and laboratory features of kidney lesions in HIV infection.

Methods. The study involved 292 HIV-infected patients, who were managed outpatiently at the Chernivtsi Regional AIDS Center. Taking into account the main markers of kidney lesions: persistent proteinuria and glomerular filtration rate <60 mL/min/1.73 m², 48 persons were diagnosed with chronic kidney disease (CKD), which was very frequently accompanied by dysfunction of these organs.

Results. Increasing proteinuria rate is accompanied by a significant renal dysfunction and more frequently is combined with arterial hypertension as well as hematuria without significant differences in the incidence of opportunistic diseases. The mean reciprocal correlation between the levels of proteinuria and glomerular filtration rate (r=-0.562, p<0.01), as well as between the levels of proteinuria and hemoglobin (r=-0.596, p<0.01) have been established as well.

Conclusions. *Kidney lesions in HIV-infected are most often characterized by tubulointerstitial lesions. At the same time, glomerular kidney lesion, which is much less common, is accompanied by a significantly higher level of HIV RNA.*

KEY WORDS: **HIV-infection**; chronic kidney disease; tubulo-interstitial lesion; glomerular lesion of kidneys.

Introduction

HIV prevalence is one of the most important issues of contemporary medicine. Over a 30year history of this disease more than 75 million people have been infected with HIV, nearly 30 million adults and children died [1, 2]. In the future decades, its significance in world premature mortality rates continues to rise.

The kidneys lesion, which is often characterized by severe clinical manifestations, can significantly affect the life expectancy in HIVinfected patients [3, 4]. Considering the increasing number of HIV-infected people in the world and a rise in the life expectancy of such patients, an increase in the number of HIVinfected people in need of expensive substitution renal therapy as well as kidney transplantation is expected.

The world scientific literature points out the factors associated with renal impairment in HIV infection: history of kidney disease, uncontrolled HIV infection, time spent on HAART, older age, female sex, African origin (APOL1 genetic va-

Corresponding author: Margaryta Andrushchak, Bukovinian State Medical University, Chernivtsi, Ukraine Phone number +380996019597 e-mail: margaritaassistent@gmail.com riant), CD4⁺ lymphocytes <200 cells/ml, as well as the use of nephrotoxic drugs [5].

However, despite a large number of foreign publications concerning this topic, the issue of the kidney lesion in HIV infection is studied insufficiently in Ukraine.

The objective of the study was to establish clinical and laboratory features of kidney lesions in HIV infection.

Methods

The study involved 292 HIV-infected patients, who were managed outpatiently at the Chernivtsi Regional AIDS Center (Chief Physician V. M. Mochulskyi).

In establishing the diagnosis, clinical and epidemiological data as well as findings of the laboratory examination methods: serological and immunological (including determination of CD4⁺-lymphocyte contents), were taken into account. The initial screening of HIV-infected people was carried out, when they were registered for monitoring in accordance with the clinical protocol No. 551, dated July 12, 2010.

The average age of all patients was (29.3 ± 8.2) years (ranged from 19 to 55 years). There were 188 (64.4%) men and 104 (35.6%)

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women among the patients. The study mostly involved young patients (25-44 years old). Among the patients, who were included in the study, 26 (8.9%) were diagnosed with the first stage of HIV infection, 40 (13.7%) individuals – with the second one, 108 (37.0%) patients – with the third, and 118 (40.4%) were diagnosed with the fourth clinical stage of the disease.

The screening of kidney lesion markers with albuminuria/proteinuria test systems by means of urinary strips (Aution Sticks-2EA) was performed. With the presence of proteinuria \geq 1+ in the screening test, corresponding to a gradation of 30 mg/l, repeated urinalysis was performed with a quantitative protein determination by means of MIKROLAB-600 spectrophotometer using UNI-TEST-BM reagents separated in the period from 3 days to one week.

The functional status of the kidneys was evaluated by the integral index, which characterized the degree of active nephrons mass maintenance/loss. A decrease in glomerular filtration rate (GFR) <60 mL/min per 1,73 M^2 was a criterion of renal dysfunction [6, 7]. Chronic kidney disease was diagnosed when proteinuria or that in combination with a decrease in GFR for 3 months or more was revealed.

A screening study to identify the kidney lesion markers (permanent proteinuria, reduction in GFR that was detected for 3 or more months) in HIV-infected patients was conducted in accordance with the recommendations of the Kidney Disease Outcome Quality Initiative, K / DOQI, 2002, and Infectious Diseases Society of America, IDSA, 2005 [4, 7]. Among the surveyed patients there were 105 (36.0%) people with markers of kidney lesion: albuminuria/ proteinuria. Based on the main markers of kidney lesion: persistent proteinuria (PU) and GFR <60 mL/min/1.73 m², in 16.4% of cases chronic kidney disease (CKD) was diagnosed, which was very frequently accompanied with renal impairment.

HIV-associated nephropathy was revealed in 48 out of 292 (16.4%) patients (31 men and 17 women), in whom the markers of kidney lesion: persistent proteinuria or proteinuria combined with a decrease in GFR, were identified and confirmed in the course of the examination.

Statistical processing of the received data was carried out using the package of applications STATISTICA 6.1 (StatSoft, USA) and Microsoft Excel 2007 programs.

The normal dissemination of the signs was determined by the graphical method, the Lilliefors criterion and the W-criterion of Shapiro-Wildlife. Dispersion of attributes was evaluated using the F-criterion in the ANOVA dispersion analysis procedure. To describe the selective normal distribution of quantitative attributes, the arithmetic mean (M) and standard deviation (m) were calculated. If the dissemination of the sign differed from normal, for its description the median (Me) and the interquartile scale with the boundaries of the segment [25%; 75%] was developed.

When comparing several independent groups, the Crackel-Wallis dispersion analysis was used (to avoid multiple comparisons). Nonparametric methods were used to compare two independent groups: the Mann-Whitney U-test and the Kolmogorov-Smirnov test, and the two dependent groups were for the Wilcoxon criterion.

The correlation analysis of two quantitative attributes was carried out using Spierman's rank method: the relationship between the indicators was considered weak in case r<0.3, moderate – at 0.3<r<0.7, strong – at r>0.7.

The comparison of groups by qualitative features was carried out by nonparametric method through analyzing 2×2 conjugation tables using a two-sided exact Fisher or χ^2 for unrelated groups.

Multivariate logistic regression analysis was used to identify the predictors of kidney impairment. Statistical differences were significant at p<0.05, very significant at p<0.01, the most significant at p<0.001 and insignificant at p>0.05.

When describing qualitative signs, the percentage of patients with the presence or absence of the analyzed sign from the total number of patients in the group is presented. The results of studies, processed statistically and presented in tables or diagrams, allow establishing the dynamics of the parameter, reliability, as well as the relationship with the changes in other parameters in accordance with existing requirements.

Results

48 HIV-infected patients with kidney lesion had the following distinctive clinical symptoms and syndromes of CKD:

 – urinary syndrome characterized by isolated proteinuria of varying degrees, by proteinuria in combination with hematuria/leukocyturia;

- arterial hypertension (AH);
- acute nephritic syndrome;
- nephrotic syndrome;

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- chronic renal insufficiency.

It was established that in every fourth HIVinfected person with CKD the urinary syndrome was characterized by isolated PU (27.1%). PU was most often combined with changes in the urine sediment: erythrocyturia and leukocyturia (17 persons – 35.4%) or hematuria (14 patients – 29.2%), with the latter most often accompanied by PU>1.0 g/day compared with the group of patients with a lower level of protein in the urine (90.5 and 51.9% respectively, p<0.01). In 4 patients (8.3%) PU was combined with leukocyturia. It should be noted that in more than half of patients transient non-bacterial leukocyturia was evidenced – more often at PU \leq 1.0 g/day.

AH was diagnosed in 15 patients (31.3%) in the presence of proteinuria compared with 2.5% in its absence (p<0.001). Acute nephritic syndrome was revealed in 5 patients (10.4%), nephrotic syndrome – in 7 (14.6%), reduction of GFR<60 mL/min/1.73 m² – in 23 individuals (47.9%).

According to the analysis of complaints, anamnestic information and clinical symptoms of kidney lesion, the patients were divided into 2 groups. The first group consisted of 31 (64.6%) out of 48 persons with tubulointerstitial and the second one – of 17 (35.4%) patients with glomerular diseases (Table 1). The presented data confirm that HIV-infected kidney lesions are most often characterized by tubulointerstitial lesion.

Chronic tubulointerstitial diseases of kidneys were characterized by a minimal or insignificant PU (0.4 [0.3; 0.8] g/day) and only in 4 (12.9 \pm 6.0)% of patients it exceeded 1 g/day. PU only was evidenced in 9 – (29.0 \pm 8.1)% of cases, but in most people PU was combined with changes in urine sedimentation. For instance, PU was accompanied by hematuria, manifested by isomorphous erythrocytes and leukocyturia in 8 (25.8 \pm 7.9)% of patients, hematuria – in 2 (6.5 \pm 4.4)% and leukocyturia – in 4 (12.9 \pm 6.0)% of cases.

In tubulointerstitial diseases, in comparison with the glomerular pathology of kidneys, the renal function impairment was diagnosed much less frequently (32.3 ± 8.4) against (76.5 ± 10.3) (p<0.01), as well as AH – (9.7 ± 5.3) and $(70.6\pm11.0)\%$ respectively (p<0.001).

Glomerular kidney lesion was characterized by a significantly lower glomerular filtration rate – 48.7 [30.2; 78.9] vs. 84.5 [52.6; 107.2] mL/

	Damage to			
Criterion	Tubulointerstitial diseases (n=31)	Glomerular diseases (n=17)	All patients (n=48)	
GFR, mL/min/1.73 m ² , median [25%; 75%]	84.5 [52.6; 107.2]	48.7 [30.2; 78.9]*	60.2 [34.4; 80.3]	
≥90, n (M%±m%)	15 (48.4±9.0) %	1 (5.9±5.7) %*	15 (31.3±6.7) %	
60-89, n (M%±m%)	8 (25.8±7.9) %	3 (17.6±9.2) %	10 (20.8±5.9) %	
30-59, n (M%±m%)	7 (22.6±7.5) %	8 (47.1±12.1) %	16 (33.3±6.8) %	
15-29, n (M%±m%)	1 (3.2±3.2) %	2 (17.6±9.2) %	3 (6.3±3.5) %	
<15, n (M%±m%)	0 (0.0±0.0) %	3 (17.6±9.2) %	4 (8.3±4.0) %	
Renal impairment, n (M%±m%)	10 (32.3±8.4) %	13 (76.5±10.3) %*	22 (45.8±7.2) %	
Proteinuria, g/day, median [25%; 75%]	0.4 [0.3; 0.8]	1.3 [1.4; 3.0]*	0.8 [0.34; 1.42]	
≤1 g / day, n (M%±m%)	27 (87.1±6.0) %	0 (0.0±0.0) %*	27 (56.3±7.2) %	
>1 g / day, n (M%±m%)	4 (12.9±6.0) %	17 (100.0±0.0) %*	21 (43.8±7.2) %	
Isolated proteinuria, n (M%±m%)	9 (29.0±8.1) %	3 (17.6±9.2) %	12 (25.0±6.3) %	
Proteinuria and hematuria, n (M%±m%)	2 (6.5±4.4) %	11 (64.7±11.6) %*	14 (29.2±6.6) %	
Proteinuria, hematuria, leukocyturia, n (M%±m%)	8 (25.8±7.9) %	9 (52.9±12.1) %*	17 (35.4±6.9) %	
Proteinuria and leukocyturia, n (M%±m%)	4 (12.9±6.0) %	0 (0.0±0.0) %*	4 (8.3±4.0) %	
Acute Nephritis Syndrome, n (M%±m%)	0 (0.0±0.0) %	5 (29.4±11.0) %*	5 (10.4±4.4) %	
Nephrotic syndrome, n (M%±m%)	0 (0.0±0.0) %	7 (41.2±11.9) %*	7 (14.6±5.1) %	
Arterial hypertension, n (M%±m%)	3 (9.7±5.3) %	12 (70.6±11.0) %*	15 (31.3±6.7) %	
Hemoglobin, g/l, median [25%; 75%]	124.0 [112.5; 133.0]	99.1 [83.0; 123.6]*	111.5 [85.5; 131.0]	

Notes: * – significant difference between the groups of patients with glomerular and tubulointerstitial diseases (p<0.05-0.001).

min/1.73 m² (p<0.05). Accordingly, only 1 person with glomerular lesion had GFR higher than 90 mL/min/1.73 m², which was significantly lower than the corresponding frequency of this feature in tubulointerstitial pathology – (48.4±9.0)% (p<0.001). At the same time, the final stage of CKD was in 3 (17.6±9,2)% of patients, 2 of whom were recommended substitution renal therapy by hemodialysis program.

In cases of glomerular kidney lesion there was also a significantly higher level of PU – 1.3 [1.4; 3.0] vs. 0.4 [0.3; 0.8] g/day (p<0.05). For instance, it exceeded 3.0 g/day in 8 patients and reached 8.0 and 9.0 g/day in 2 of them. The combination of PU with hematuria (64.7 ± 11.6) and (6.5 ± 4.4)%, respectively (p<0.001), with hematuria and aseptic leukocyturia (52.9 ± 12.1) and 25.8 ± 7.9)%, respectively (p<0.05) were present much more frequently than in the patients with tubulointerstitial diseases. Thus, in the majority of cases there was microhematuria, manifested by dysmorphic erythrocytes, whereas episodic macrohematuria was evidenced in 2 patients.

7 (41.2 \pm 11.9)% patients were diagnosed with nephrotic and 5 (29.4 \pm 11.0)% people suffered from acute nephritic syndromes. It is noteworthy, that these syndromes were not revealed in any representative of the group with tubulointerstitial disease. Expectedly, the level of hemoglobin in glomerular kidney lesion was reliably lower: 99.1 [83.0; 123.6] vs. 124.0 [112.5; 133.0] g/l (p<0.05). This may point to the direct effect of HIV on the glomerular apparatus, whereas tubulointerstitial kidney lesions are most likely due to the influence of opportunistic infections and drugs with nephrotoxic potential, as well as the use of psychotropic drugs and the uncontrolled administration of nonsteroidal anti-inflammatory drugs, which these patients often abuse of.

The mean number of CD4⁺ lymphocytes in serum of the patients with proteinuria is much lower than in the HIV-infected individuals without markers of kidney lesion: 185.5 [25-60.9; 75% – 318.0] vs. 312.0 [25% – 175.5; 75% – 469.0] cl/µl respectively (p<0.05). A decrease in CD4⁺ lymphocytes level ≤200 cl/µl was found in 52.1±7.2% of patients with proteinuria and in 30.0±7.2% of those without it (p<0.05). The difference between the ratios of CD4⁺/CD8⁺lymphocytes in the studied groups was also quite significant: 0.2 [0.1; 0.4] and 0.4 [0.2; 0.6] respectively (p<0.05) (Table 2).

The data concerning the HIV RNA level and type of CKD are presented at Fig. 1.

Depending on the level of proteinuria, the patients were divided into two groups. The first group consisted of 27 out of 48 (56.3%) patients with PU less than 1.0 g/day, the second group – 21 (43.7%) patients with PU more than 1.0 g/day, in 7 of them it reached the nephrotic level – more than 3.0 g/day.

There were more males in both groups (70.4 \pm 8.4) and (57.1 \pm 10.9)% respectively, and people aged 25-44 (66.7 \pm 9.1) and (61.9 \pm 10,6)%

	Damage to			
Criterion	Tubulointerstitial diseases (n=31)	Glomerular diseases (n=17)	All patients (n=48)	
Viral load (RNA of HIV), copies/ml	22 000	250 000	135 000	
	[5 125; 308 000]	[35 225; 690 500]*	[14 027; 460 000]	
HIV RNA was not detected, n, (M±m, %)	1 (3.2±3.2) %	2 (11.8±7.8) %	4 (8.3±4.0) %	
≤100 000, n (M±m, %)	11 (35.5±8.6) %	8 (48.1±12.1) %	18 (37.5±7.0) %	
>100 000, n (M±m, %)	19 (61.3±8.7) %	7 (41.2±11.9) %	26 (54.2±7.2) %	
CD4⁺ (median [25 %; 75 %])	220.4 [34.6; 280.5]	197.5 [54.3; 309.0]	185.5 [60.9; 318.0]	
≤200, n (M±m, %)	16 (51.6±9.0) %	8 (47.1±12.1) %	25 (52.1±7.2) %	
201-350, n (M±m, %)	8 (25.8±7.9) %	5 (29.4±11.0) %	13 (27.1±6.4) %	
>350, n (M±m, %)	7 (22.6±7.5) %	4 (23.5±10.3) %	10 (20.8±5.9) %	
Correlation CD4 ⁺ /CD8 ⁺ (mediana [25 %: 75 %])	0.2 [0.1; 0.5]	0.2 [0.1; 0.4]	0.2 [0.1; 0.4]	
Duration of HIV infection, years, (mediana [25 %; 75 %])	5.0 [3.5; 8.0]	6.0 [2.5; 7.5]	5.5 [3.0; 8.0]	

Table 2. Number of RNAs of HIV, CD4⁺ lymphocytes and the ratio of CD4⁺/CD8⁺ in the patients with different variants of clinical kidney damage

Notes: * – significant difference between the groups of patients with glomerular and tubulointerstitial diseases (p<0.05-0.001).

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respectively, the same as in the total number of HIV-infected people.

According to Table 3, in both groups of patients, fungal diseases were the most common (51.9 ± 9.6) and (52.4 ± 10.9)% respectively, the same as the diseases of viral etiology – (18.5 ± 7.5) and (33.3 ± 10.3)%, respectively, without significant differences in their incidence (p>0.05).

Clinical description of the HIV-infected patients with different levels of proteinuria is presented in Table 4.

According to Table 4, the HIV-infected patients with a level of PU more than 1 g/day were much more frequently diagnosed with arterial hypertension (52.4 ± 10.9) versus (14.8 ± 6.8)% in the patients with proteinuria not exceeding the indicated level (p<0.01). Thus, the relationship between the level of PU and the presence of arterial hypertension was established. It should be noted that according to the level of blood pressure, the patients in the groups were distributed as follows: the first degree AH was diagnosed in 3 (11.1%) patients of the first group and in 3 (14.3%) patients of the second group; the second degree AH was

revealed in 2 (7.4%) and 4 (19.0%) patients respectively, and the third degree AH was only found in one (3.7%) patient of the first group and in 3 (14.3%) persons with proteinuria >1 g/day.

The incidence of hypercholesterolemia and hypoalbuminemia in the comparable groups was approximately the same (p>0.05). At the same time, the level of hemoglobin (99.0 [78.0, 123.0]) (median [interquartile scale]) was expectedly much lower in the patients with a higher level of PU vs. (124.0 [93.0; 139.0]) g/l (p<0.05), the incidence of hematuria was much higher as well (90.5 \pm 6.4) vs. (51.9 \pm 9.6)% (p<0.01).

It is noteworthy that with the increase in PU the GFR levels decreased significantly from (72.0 [38.3; 99.6]) to (48.3 [30.5; 61.8]) mI/ min/1.73 m² (p<0.01), and only in the group of patients with PU less than 1 g/day the GFR did not drop off below 30 mI/min/1.73 m² (Fig. 2).

There were statistically significant intergroup differences in the severity of kidney lesion, depending on the level of proteinuria. For instance, the preserved renal function (GFR \geq 90 mL/min/1.73 m²) was more frequently evidenced





Fig. 1. HIV RNA level in the HIV-infected patients with different variants of kidney damage.

Fig. 2. Correlation among the levels of proteinuria and glomerular filtration rate.

Table 3. Frequency of opportunistic diseases in the HIV-infected patients with different levels of proteinuria

	Proteinuria level				
Opportunistic infections	≤1 g/day (n=27)		>1 g/day (n=21)		р
	n	M±m, %	n	M±m, %	
Bacterial	3	11.1±6.0	2	9.5±6.4	>0.05
Viral	5	18.5±7.5	7	33.3±10.3	>0.05
Fungal	14	51.9±9.6	11	52.4±10.9	>0.05
Parasitic	3	11.1±6.0	2	9.5±6.4	>0.05
Tuberculosis	4	14.8±6.8	6	28.6±9.9	>0.05

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Indicator	Proteinu						
Indicator	≤1 g/day (n=27) >1 g/day (n=21)		- p				
Arterial hypertension, n (M±m, %)	4 (14.8±6.8)	11 (52.4±10.9)	<0.01				
AT Systolic, mm Hg (median [25%; 75%])	135 [100; 170]	145 [110; 180]	>0.05				
Diastolic blood pressure, mm Hg (median [25%; 75%])	90 [85; 100]	95 [90; 110]	>0.05				
Cholesterol, mmol/L (median [25%; 75%])	4.1 [3.2; 5.4]	4.3 [3.4; 5.8]	>0.05				
Hypercholesterolemia, n (M±m, %)	5 (18.5±7.5)	4 (19.0±8.6)	>0.05				
Hypoalbuminemia, n (M±m, %)	7 (25.9±8.4)	7 (33.3±10.3)	>0.05				
Albumin, g/l (median [25%; 75%])	36.9 [30.3; 42.8]	34.1 [28.7; 38.5]	>0.05				
Hemoglobin, g/l (median [25%; 75%])	124.0 [93.0; 139.0]	99.0 [78.0; 123.0]	<0.05				
Hematuria, n (M±m, %)	14 (51.9±9.6)	19 (90.5±6.4)	<0.01				
GFR, ml/min /1.73 m ² (median [25%; 75%])	72.0 [38.3; 99.6]	48.3 [30.5; 61.8]	<0.01				
≥90, n (M±m, %)	13 (48.1±9.6)	2 (9.5±6.4)	<0.01				
60-89, n (M±m, %)	9 (33.3±9.1)	3 (14.3±7.6)	>0.05				
30-59, n (M±m, %)	5 (18.5±7.5)	10 (47.6±10.9)	<0.05				
15-29, n (M±m, %)	0 (0.0±0.0)	3 (14.3±7.6)	<0.05				

 $0(0.0\pm0.0)$

Table 4. Clinical and laboratory characteristics of the HIV-infected patients with different levels of proteinuria

in the patients of the 1st group (48.1±9.6)% and much less frequently in those with proteinuria >1 g/day - (9.5±6.4)% (p<0.01). On the contrary, the GFR, which corresponded to the 3rd stage of CKD, was evidenced in half of patients with proteinuria >1g/day (47.6±10.9)% and only in 18.5 \pm 7.5% of patients with PU ≤1 g/day (Fig. 3). Accordingly, the terminal renal insufficiency

<15, n (M±m, %)

(GFR<15 mL/min/1.73 m²) was revealed only in the patients of the 2nd group, in 2 of them proteinuria exceeded 3.0 g/day.

3 (14.3±7.6)

< 0.05

The mean reciprocal correlation between the levels of proteinuria and the glomerular filtration rate (r=-0.562, p<0.01), as well as between the levels of proteinuria and hemoglobin (r=-0.596, p<0.01) have been also established.





Discussion

Thus the clinical manifestations of kidney lesion in the studied patients coincide with the typical ones for various pathologies in the general number of nephrology patients.

There are numerous experiments on the study of a number of renal diseases in the HIVinfected individuals worldwide [4, 8, 9]. For instance, the studies conducted in the United States have revealed that, according to the renal biopsy, 52.7% of the patients with nephrotic PU were diagnosed with HIV-associated nephropathy. They were all African Americans. The high incidence of this pathology is associated with racial affiliation, as well as with a specific variant of the antigen/receptor to Duffy chemokines, which are found in the renal tissue [10]. According to the results of multicenter studies in France and Italy, where most patients were of the Caucasian race, among morphologically verified diagnoses, immune deposit diseases were prevalent in the HIV-infected patients with kidney pathology [11, 12].

Proteinuria is established to be one of the major laboratory criteria for CKD. Therefore, the next stage of the work was the establishment of clinical and laboratory features of renal impairment depending on the level of protein in urine. Thus, the analysis proved that the increase in PU levels was accompanied by a significant renal dysfunction and a more frequent combination with arterial hypertension and hematuria without significant differences in the frequency of opportunistic diseases. The inverse correlation between the level of proteinuria, GFR and hemoglobin value has been established.

According to other indicators characterizing the course of HIV infection in people with different clinical variants of chronic kidney lesion, there were no reliable differences.

Conclusions

Kidney lesions in HIV-infected are most often characterized by tubulointerstitial lesions. At the same time, glomerular kidney lesion, which is much less common, is accompanied by a significantly higher level of HIV RNA.

An increase in proteinuria level is accompanied by a significant renal dysfunction and a more frequent combination with arterial hypertension and hematuria without significant differences in the incidence of opportunistic diseases. The mean reciprocal correlation between the levels of proteinuria and glomerular filtration rate (r=-0.562, p<0.01), as well as between the levels of proteinuria and hemoglobin (r=-0.596, p<0.01) have been established as well.

УРАЖЕННЯ НИРОК У ВІЛ-ІНФІКОВАНИХ

М. О. Андрущак

БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ, ЧЕРНІВЦІ, УКРАЇНА

Вступ. Однією з найважливіших проблем сучасності є епідемія ВІЛ-інфекції. За 30-річну історію цієї хвороби ВІЛ уразив понад 75 мільйонів людей, з них майже 30 мільйонів дорослих і дітей померли. У найближчі десятиліття, як і раніше, вони відіграватимуть істотну роль у світових показниках передчасної смертності.

Мета роботи – встановити клінічні та лабораторні особливості ураження нирок при ВІЛ-інфекції. Методи. Обстежено 292 хворих на ВІЛ-інфекцією, які перебували на амбулаторному спостереженні в Чернівецькому обласному центрі з профілактики та боротьби зі СНІДом. На підставі основних маркерів пошкодження нирок (персистентна протеїнурія та швидкість клубочкової фільтрації <60 мл/хв/1,73 м²) у 48 осіб діагностовано хронічну хворобу нирок, яка з великою частотою супроводжувалася порушенням функції цих органів.

Результати Встановили, що у ВІЛ-інфікованих ураження нирок найчастіше характеризується їх тубулоінтерстиційним ураженням. Водночас гломерулярне ураження нирок, що буває значно рідше, супроводжується достовірно вищим рівнем РНК ВІЛ.

Підвищення рівня протеїнурії супроводжувалося достовірно значущим порушенням функції нирок і частішим поєднанням з артеріальною гіпертензією і гематурією за відсутності достовірних відмінностей у частоті опортуністичних захворювань. Встановлено зворотну середньої сили кореляцію між рівнями протеїнурії і швидкістю клубочкової фільтрації – (r=-0,562, p<0,01), а також між рівнями протеїнурії та гемоглобіну (r=-0,596, p<0,01). Висновки У ВІЛ-інфікованих ураження нирок найчастіше характеризується їх тубулоінтерстиційним ураженням. Водночас гломерулярне ураження нирок, що буває значно рідше, супроводжується достовірно вищим рівнем РНК ВІЛ.

КЛЮЧОВІ СЛОВА: **ВІЛ-інфекція; хронічна хвороба нирок; тубулоінтерстиційне ураження; гломерулярне ураження нирок.**

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