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The significance of hereditary factor in the development of chronic kidney disease (glomerulonephritis)

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The objective: **of the research was to evaluate the risk of chronic kidney disease: glomerulonephritis (CKD:GN) development by antigens of blood groups of AB0 and Rhesus (Rh) systems.**
***Materials and methods*. We examined 315 patients (166 men and 149 women) with CKD:GN which manifested by urinary syndrome (US) (asymptomatic proteinuria and/or hematuria). A survey of 1428 healthy individuals was conducted to determine the populational distribution of blood groups phenotypes of AB0 and Rh systems.**
***Results*. The value of the relative risk of CKD:GN with US occurrence in men with phenotype A (II) versus 0 (I) prevailed in 7.79 times the same in women and it was in 5.15 times higher in the latter with phenotype AB (IV) versus A (II) than the same in men. The common feature was a high chance to contract the studied pathology in carriers of phenotype AB (IV) Rh– versus 0 (I) Rh–. Individuals of both genders with group 0 (I) Rh– may be resistant to the development of the disease.**
***Conclusions*. Gender dimorphism consisted in the fact that men with phenotypes AB (IV) Rh– and A (II) Rh– versus 0 (I) Rh–, women with phenotypes AB (IV) Rh– and B (III) Rh– versus 0 (I) Rh– and A (II) Rh– had the highest risk to contract a disease. Somewhat lower risk of disease occurrence was possible in the presence of phenotypes A (II) Rh+ in men and AB (IV) Rh+ in women.**
Key words: *chronic kidney disease, glomerulonephritis, blood group, AB0 and Rh system, erythrocytic antigens.*

Currently, antigenic structure of human blood has been established to be complex, since all formed elements (erythrocytic, leukocytic, thrombocytic) and blood plasma proteins of different people differ in antigens composition. Antigenic structure of red blood cells (phenotype) is genetically determined (genotype) [8]. More than 250 erythrocytic antigens are known which form about 20 antigenic systems: AB0, Rhesus (Rh), Kell, Duffy, Kidd, Lewis, Lutheran, Auberger etc. [9]. Antigens pertaining to AB0 system, along with the antigens of Rh system, have the greatest value in medical practice and are considered to be important genetic markers [10].

One of the genetic methods is the establishment of the links between certain pathology and hereditary polymorphism of blood group factors of AB0 and Rh systems [4]. Blood-group specificity may be a risk factor in the development of various diseases in humans [6]. The proof of genes influence of AB0 and Rh blood groups on the occurrence and spread of diseases is known to be the comparison of frequencies of certain blood phenotypes presence in patients with any disease with the healthy. Increase in the number of carriers of any blood group phenotype among patients compared with the healthy can be regarded as a selective choice against this group and elimination of its carriers as vulnerable antigens from the population, as well as absence of changes or a slight decrease in dissemination of known antigen as a defense mechanism to the survival [4].

This problem attracts significant attention of scientists because the inheritance of AB0 and Rh blood groups is not associated with the gender, and their phenotypes are unchanged throughout life. The study of AB0 and Rh systems antigens provided an opportunity to find out the relationship between blood groups antigens and a number of diseases [5, 6, 7, 8, 10, 12]. However, such studies in case of glomerulonephritis (GN) were not found in the literature. In general, the study of genotype-phenotype in the population of patients with chronic kidney disease (CKD) is still at an early stage and requires continuous researches [11].

The objective: to evaluate the risk degree of CKD:GN development by antigens of blood groups of AB0 and Rh systems.

MATERIALS AND METHODS

We examined 315 patients (166 men and 149 women) with CKD:GN, which manifested by urinary syndrome (US) (asymptomatic proteinuria and/or hematuria). The mean age of patients was (36.77±12.49) years. The diagnosis was established according to the recommended criteria (the order of Ministry of Health of Ukraine № 593 from 12.12.2004). The patients underwent renal biopsy to verify the morphological form of the disease by using biopsy gun and disposable needles manufactured by the company Bard Magnum (USA) with the following research of renal biopsy material by the techniques of light and electron microscopy and immunohistochemistry. A survey of 1428 healthy individuals was conducted to determine the populational distribution of blood groups phenotypes of AB0 and Rh systems. All the surveyed were residents of Western Ukraine.

We used a statistical method for determining the relative frequency of risk (X) of a certain disease occurrence to analyze the associations between the development of CKD:GN with US and blood groups antigens of AB0 and Rh systems. For this purpose, the frequencies of two signs for example, A versus 0, were compared in two samplings (the patients and the control group) [3].

$$X = \frac{\text{A(pat.)} \bullet \text{0(contr.)}}{\text{0(pat.)} \bullet \text{A(contr.)}}$$

The association was absent if the ratio A/0 was the same in the two samplings. The X value was equal 1 in case of the absence of differences between compared groups of individuals. The X value was greater or lesser than 1 in the presence of association, moreover the degree of increase characterized the magnitude of the risk.

We used the chi-square test (χ^2) or Fisher's exact test (ϕ) and odds ratio (OR) for statistical analysis of the data [1, 2]. The critical level of statistical significance was considered at 5%.

RESULTS AND DISCUSSION

We analyzed quantitative and relative distribution of healthy persons and patients by blood groups phenotypes to investigate the genetic burdening to CKD:GN with US (Table 1).

Table 1

Quantitative and relative distribution of healthy individuals and patients with US in case of CKD:GN by blood groups antigens

Blood group of AB0 and Rh systems	Number of healthy individuals, n=1428	Number of patients, n=315	χ^2, p	OR
0 (I)	458 (32,07%)	72 (22,86%)	9,93; p=0,002	0,63 (0,47–0,84)
A (II)	565 (39,57%)	151 (47,94%)	7,13; p=0,008	1,41 (1,10–1,80)
B (III)	301 (21,08%)	58 (18,41%)	0,96; p=0,326	0,85 (0,62–1,16)
AB (IV)	104 (7,28%)	34 (10,79%)	3,39; p=0,067	1,55 (1,04–2,33)
Rh^+	1194 (83,61%)	282 (89,52%)	6,50; p=0,011	1,66 (1,13–2,43)
Rh^-	234 (16,39%)	33 (10,48%)	6,50; p=0,011	0,60 (0,41–0,89)

Table 2

The distribution of blood groups in patients with US in case of CKD:GN according to gender

Blood group of AB0 and Rh systems	Number of healthy individuals	Number of patients	χ^2, p	OR
Men				
0 (I)	146 (33,56%)	37 (22,29%)	6,69; p=0,010	0,57 (0,38–0,87)
A (II)	166 (38,16%)	88 (53,01%)	10,26; p=0,001	1,82 (1,27–2,62)
B (III)	92 (21,15%)	28 (16,87%)	1,12; p=0,289	0,76 (0,48–1,22)
AB (IV)	31 (7,13%)	13 (7,83%)	0,01; p=0,903	1,13 (0,58–2,19)
Rh^+	384 (88,28%)	148 (89,16%)	0,03; p=0,873	1,08 (0,61–1,89)
Rh^-	51 (11,72%)	18 (10,84%)	0,03; p=0,873	0,93 (0,53–1,63)
Women				
0 (I)	312 (31,42%)	35 (23,49%)	3,49; p=0,062	0,68 (0,45–1,01)
A (II)	399 (40,18%)	63 (42,28%)	0,16; p=0,691	1,09 (0,77–1,55)
B (III)	209 (21,05%)	30 (20,13%)	0,02; p=0,883	0,96 (0,62–1,46)
AB (IV)	73 (7,35%)	21 (14,09%)	6,93; p=0,008	2,10 (1,25–3,51)
Rh^+	810 (81,57%)	134 (89,93%)	5,75; p=0,016	1,96 (1,13–3,40)
Rh^-	183 (18,43%)	15 (10,07%)	5,75; p=0,016	0,51 (0,29–0,88)

Table 3

Quantitative and relative distribution of healthy individuals and patients with US in case of CKD: GN by blood groups antigens of AB0 and Rh systems

Blood group of AB0 system	Number of healthy Rh^+ carriers	Number of sick Rh^+ carriers	Number of healthy Rh^- carriers	Number of sick Rh^- carriers
0 (I)	377 (26,40%)	69 (21,90%) χ^2=2,51; p=0,113; OR=0,79 (0,59–1,05)	81 (5,67%)	3 (0,95%) χ^2=11,53; p=0,001; OR=0,19 (0,06–0,54)
A (II)	485 (33,96%)	137 (43,49%) χ^2=9,80; p=0,002; OR=1,50 (1,17–1,92)	80 (5,60%)	14 (4,44%) χ^2=0,47; p=0,493; OR=0,81 (0,45–1,43)
B (III)	255 (17,86%)	53 (16,83%) χ^2=0,12; p=0,724; OR=0,94 (0,68–1,29)	46 (3,22%)	5 (1,59%) χ^2=1,88; p=0,170; OR=0,53 (0,22–1,29)
AB (IV)	77 (5,39%)	23 (7,30%) χ^2=1,40; p=0,236; OR=1,40 (0,87–2,26)	27 (1,89%)	11 (3,49%) χ^2=2,40; p=0,122; OR=1,92 (0,96–3,87)

The disease was established to develop most often in the presence of phenotype A (II) constituting 47.94%. The second and third places by frequency were occupied 0 (I) – 22.86% and B (III) – 18.41% respectively. The carriers of antigens AB (IV) were registered the most rarely among the patients with CKD:GN comprising 10.79%. The specified order of phenotypes prevalence in the patients coincided with that in the population of healthy individuals. However, we detected a statistically significant increase in the frequency of phenotype A (II) in patients with the disease compared with healthy individuals (χ^2=7.13; p=0.008), and the appropriate decrease in the frequency of phenotype 0 (I) (χ^2=9.93; p=0.002). Meanwhile, an increase in Rh antigen carriers with probability of p=0.011 among patients and the appropriate reduction among those patients who did not have it (χ^2=6.50) was observed.

The next stage of the work was a study of blood groups distribution according to gender in the patients with US in case of CKD:GN (Table 2).

The gender differences in the distribution of specified parameters were established. Thus, statistically significant increase in the probability of investigated pathology occurrence in 1.39 times existed in men in the presence of antigen A (II) (χ^2=10.26; p=0.001) and accordingly in women – in 1.92 times in the presence of antigen AB (IV) (χ^2=6.93; p=0.008). The decrease in probability of CKD:GN with US development in 1.51 times (χ^2=6.69; p=0.010) was also observed in male patients with phenotype 0 (I). Comparative analysis of Rh carriers' frequency according to gender did not record statistical significance in men, however, it was defined in female patients: an increase of

Table 4

The distribution of blood groups antigens of AB0 and Rh systems in patients with US in case of CKD:GN according to gender

Blood group of AB0 system	Number of healthy Rh^+ carriers	Number of sick Rh^+ carriers	Number of healthy Rh^- carriers	Number of sick Rh^- carriers
Men				
0 (I)	129 (29,66%)	36 (21,69%) χ^2=3,44; p=0,064; OR=0,66 (0,43–1,01)	17 (3,91%)	1 (0,60%) ϕ=2,66; p=0,032; OR=0,22 (0,04–1,16)
A (II)	150 (34,48%)	77 (46,39%) χ^2=6,74; p=0,009; OR=1,64 (1,14–2,36)	16 (3,68%)	11 (6,63%) χ^2=1,80; p=0,180; OR=1,88 (0,87–4,08)
B (III)	82 (18,85%)	28 (16,87%) χ^2=0,20; p=0,657; OR=0,88 (0,55–1,41)	10 (2,30%)	–
AB (IV)	23 (5,29%)	7 (4,22%) χ^2=0,11; p=0,742; OR=0,83 (0,36–1,92)	8 (1,84%)	6 (3,61%) ϕ=1,24; p=0,227; OR=2,04 (0,72–5,75)
Women				
0 (I)	248 (24,97%)	33 (22,15%) χ^2=0,42; p=0,519; OR=0,86 (0,57–1,30)	64 (6,45%)	2 (1,34%) χ^2=5,29; p=0,021; OR=0,24 (0,07–0,87)
A (II)	335 (33,74%)	60 (40,27%) χ^2=2,16; p=0,141; OR=1,33 (0,93–1,89)	64 (6,45%)	3 (2,01%) χ^2=3,84; p=0,051; OR=0,34 (0,12–1,02)
B (III)	173 (17,42%)	25 (16,78%) χ^2=0,01; p=0,938; OR=0,97 (0,61–1,53)	36 (3,63%)	5 (3,36%) χ^2=0,01; p=0,943; OR=1,00 (0,40–2,49)
AB (IV)	54 (5,44%)	16 (10,74%) χ^2=5,44; p=0,020; OR=2,13 (1,19–3,80)	19 (1,91%)	5 (3,36%) ϕ=1,07; p=0,228 OR=1,90 (0,73–4,98)

Table 5

Associations between blood groups antigens of AB0 system and CKD:GN with US

Comparative groups	The value of relative risk versus control		
AB0 system	All patients	Men	Women
0 : A	0,588	0,478	0,710
0 : B	0,816	0,833	0,782
0 : AB	0,481	0,604	0,390
A : 0	1,700	2,092	1,408
A : B	1,387	1,742	1,100
A : AB	0,817	1,264	0,549
B : 0	1,226	1,201	1,280
B : A	0,721	0,574	0,909
B : AB	0,589	0,726	0,499
AB : 0	2,080	1,655	2,564
AB : A	1,223	0,791	1,822
AB : B	1,697	1,378	2,004
Rh^+ : Rh^-	1,675	1,092	2,018

frequency was observed in the Rh-positive and its reduction was noted in the Rh-negative (χ^2=5.75; p=0.016).

Further, we defined the distribution of patients with US in case of CKD:GN by blood groups antigens of AB0 and Rh systems (Table 3).

Comparative analysis of antigens combinations of AB0 and Rh systems detected differences in the examined patients from those in the control group. According to the data characteristics Rh-positive patients were distributed as follows: A(II)>0(I)>B(III)>AB(IV). The frequency of A (II) carriers was significantly higher compared to the population of healthy individuals (χ^2=9.80; p=0.002). Rh-negative patients constituted another distribution by consistent decrease in the frequency of different phenotypes of AB0 system: A(II)>AB(IV)>B(III)>0(I). Statistically considerable reduction of the frequency was shown in the latter with antigen 0(I) compared to the control (χ^2=11.53; p=0.001).

Thus, a greater probability of CKD:GN with US development was observed in carriers of blood group A (II) Rh+. An interesting

Table 6

Associations between combination of blood groups antigens of AB0 and Rh systems and CKD:GN with US

Comparative groups	The value of relative risk versus control		
AB0 and Rh systems	All patients	Men	Women
0 Rh^+ : A Rh^+	0,648	0,544	0,743
0 Rh^+ : B Rh^+	0,881	0,817	0,921
0 Rh^+ : AB Rh^+	0,613	0,917	0,449
A Rh^+ : 0 Rh^+	1,543	1,839	1,346
A Rh^+ : B Rh^+	1,359	1,503	1,239
A Rh^+ : AB Rh^+	0,946	1,687	0,604
B Rh^+ : 0 Rh^+	1,136	1,224	1,086
B Rh^+ : A Rh^+	0,736	0,665	0,807
B Rh^+ : AB Rh^+	0,696	1,122	0,488
AB Rh^+ : 0 Rh^+	1,632	1,091	2,227
AB Rh^+ : A Rh^+	1,057	0,593	1,654
AB Rh^+ : B Rh^+	1,437	0,891	2,050
0 Rh^- : A Rh^-	0,212	0,086	0,667
0 Rh^- : B Rh^-	0,341	-	0,225
0 Rh^- : AB Rh^-	0,091	0,078	0,119
A Rh^- : 0 Rh^-	4,725	11,688	1,500
A Rh^- : B Rh^-	1,610	-	0,338
A Rh^- : AB Rh^-	0,430	0,917	0,178
B Rh^- : 0 Rh^-	2,935	-	4,444
B Rh^- : A Rh^-	0,621	-	2,963
B Rh^- : AB Rh^-	0,267	-	0,528
AB Rh^- : 0 Rh^-	11,000	12,750	8,421
AB Rh^- : A Rh^-	2,328	1,091	5,614
AB Rh^- : B Rh^-	3,748	-	1,895

fact was that carriers of phenotype 0 (I) Rh– were registered much rarer among patients than in the healthy individuals. It may indicate a certain resistance to CKD:GN with US occurrence in such individuals.

Logical continuation of our work was a statistical analysis of the distribution of different blood groups phenotypes of AB0 and Rh systems according to gender (Table 4).

Comparative analysis of antigens combinations of AB0 and Rh systems detected differences in the examined patients according to gender compared with those in the control group. The same distribution as in the general population was marked among Rh-positive patients of both genders by frequency of phenotypes. Depending on gender, the Rh-negative patients by frequency of associations with the studied antigens constituted the following lines: men – A(II)>AB(IV)>0(I), women – B(III)=AB(IV)>A(II)>0(I). It should be noted that men with group B (III) were not found among Rh-negative patients in our study and the same number of women with groups B (III) and AB (IV) was noted constituting 3.36%. Moreover, the carriers of antigens A (II) Rh+ prevailed those in the population of healthy individuals (χ^2=6.74; p=0.009) among male patients, and on the contrary, the frequency of 0 (I) Rh– was lower (ϕ=2.66; p=0.032). Similar sign was noted in the female sampling of patients – the frequency of phenotype 0 (I) Rh– (χ^2=5.29; p=0.021) was reduced. Greater predisposition to the development of CKD:GN with US in the presence of antigens AB (IV) Rh+ (χ^2=5.44; p=0.020) was different in women.

Determination of the relative risk of CKD:GN with US development depending on the blood groups versus a control group was conducted in the general sampling of patients, and separately for males and females (Table 5).

The risk of investigated disease depended on the antigens of AB0 system. The closest association between the disease and phenotype AB (IV) compared with all other phenotypes especially versus 0 (I) and B (III) and also A (II) versus 0 (I) was found in the whole group of patients. Analysis of the gender features of CKD:GN with US development showed that the greatest risk of disease occurrence in men was observed in the presence of antigen

A (II), in particular versus 0 (I) and B (III). Somewhat lower probability of pathology formation was marked in AB (IV) patients versus 0 (I) ones. Similar to the general sampling of patients, the most significant associations of the studied pathology in women were noted in the presence of group AB (IV) in comparison with all other groups. Total risk to contract an illness in females with this blood group was in 1.67 times higher in comparison with male group. It should be noted that the lowest probability to contract an illness was found in the presence of phenotypes 0 (I) versus AB (IV) and A (II) and also B (III) versus AB (IV) for all examined patients. An interesting fact was also that the chance of CKD:GN with US occurrence in Rh-positive women was almost twice higher than in Rh-positive men.

The logical continuation of the work was a detection of associations between AB0, Rh antigens and CKD:GN with US (Table 6).

The greatest risk of the disease occurrence in the general group of patients was observed in Rh-negative carriers of antigens AB (IV) versus all groups, and also A (II) and B (III) versus 0 (I). Accordingly, the slightest risk existed in the individuals with phenotype 0 (I) Rh–. This confirms our previous results once again. In the sampling of all patients among Rh-positive individuals the close group associations were detected in the presence of antigen AB (IV) and A (II) versus 0 (I). However, a total value of the relative risk of CKD:GN with US in the Rh-negative carriers of AB (IV) prevailed in 4.14 times the same in the Rh-positive. Analysis of gender features of the disease formation depending on AB0 and Rh phenotypes showed gender dimorphism. The greatest risk of CKD:GN with US in men was observed among Rh-negative individuals in the presence of antigens AB (IV) and A (II) versus 0 (I). Significantly lower risk was among Rh-positive men with group A (II).

The most significant associations among Rh-negative women were found in the presence of antigens AB (IV) and B (III) versus 0 (I) and A (II). The greatest chance of pathology development in Rh-positive females was marked in blood group AB (IV) versus all other groups. A comparison of certain groups in Rh-negative individuals of different genders was interesting: the value of the relative risk of CKD:GN with US occurrence in men with phenotype A (II) versus 0 (I) prevailed in 7.79 times the same in women and it was in 5.15 times higher in the latter with phenotype AB (IV) versus A (II) than the same in men. The total value of the relative risk of CKD:GN with US development in Rh-positive women with group AB (IV) prevailed in 2.3 times the same value in men. It should be noted that the common feature in all three groups was a high chance to contract the studied pathology in carriers of phenotype AB (IV) Rh– versus 0 (I) Rh–. Rh-negative men and women with the group 0 (I) were also found may be resistant to the development of CKD:GN with US.

CONCLUSIONS

1. Comparative analysis of phenotypes frequency by AB0 antigens in case of CKD:GN with US determined the following distribution in Rh-positive patients of both genders: A(II)>0(I)>B(III)>AB(IV). The phenotypes distribution differed in Rh-negative patients: A(II)>AB(IV)>0(I) was in men and B(III)=AB(IV)>A(II)>0(I) was in women.

2. The research of the antigens combination of AB0 and Rh systems in patients with US in case of CKD:GN proved a significant preference of the carriers of phenotypes A (II) Rh+ and significantly lower frequency of patients with phenotype 0 (I) Rh–.

3. Gender dimorphism in the markers distribution of hereditary predisposition to CKD:GN with US consisted in the fact that men with phenotypes AB (IV) Rh– and A (II) Rh– versus 0 (I) Rh–, women with phenotypes AB (IV) Rh– and B (III) Rh– versus 0 (I) Rh– and A (II) Rh– had the highest risk to contract a disease. Somewhat lower risk of the disease occurrence was possible in the presence of phenotypes A (II) Rh+ in men and AB (IV) Rh+ in women. Individuals of both genders with group 0 (I) Rh– may be resistant to development of the disease.

Prospects for further researches in this direction consist in the study of the risk degree of CKD:GN development by blood groups antigens of AB0 and Rh systems depending on different morphological forms of the disease.

Значення спадкового фактора у розвитку хронічної хвороби нирок (гломерулонефриту)

В.Я. Камінський

Мета дослідження: оцінювання ризику розвитку хронічної хвороби нирок: гломерулонефриту (ХХН:ГН) за антигенами груп крові систем АВ0 і резус (Rh).

Матеріали та методи. Було досліджено 315 пацієнтів (166 чоловіків і 149 жінок) із ХХН:ГН, який проявлявся сечовим синдромом (СС) (асимптоматична протеїнурія та/або гематурія). Для встановлення розподілу фенотипів груп крові систем АВ0 і Rh серед населення було обстежено 1428 здорових осіб.

Результати. Величина відносного ризику виникнення ХХН:ГН із СС у чоловіків з фенотипом А (II) проти 0 (I) переважала у 7,79 разу таку саму у жінок, а в останніх із фенотипом АВ (IV) проти А (II) – у 5,15 разу таку саму у чоловіків. Загальною рисою була висока ймовірність захворіти на досліджувану патологію у носіїв фенотипу АВ (IV) Rh– проти 0 (I) Rh–. Особи обох статей із групою 0 (I) Rh– можуть бути стійкими до розвитку цього захворювання.

Заключення. Гендерний диморфізм полягав у тому, що найбільший ризик захворіти мають чоловіки з фенотипами АВ (IV) Rh– та А (II) Rh– проти 0 (I) Rh–, жінки – АВ (IV) Rh– та В (III) Rh– проти 0 (I) Rh– і А (II) Rh–. Дещо менший ризик формування патології можливий при наявності фенотипів А (II) Rh+ у чоловіків та АВ (IV) Rh+ у жінок.

Ключові слова: *хронічна хвороба нирок, гломерулонефрит, група крові, система АВ0 і Rh, еритроцитарні антигени.*

Значение наследственного фактора в развитии хронической болезни почек (гломерулонефрита)

В.Я. Каминский

Цель исследования: оценка риска развития хронической болезни почек: гломерулонефрита (ХБП:ГН) по антигенам групп крови систем АВ0 и резус (Rh).

Материалы и методы. Было исследовано 315 пациентов (166 мужчин и 149 женщин) с ХБП:ГН, который проявлялся мочевым синдромом (МС) (асимптоматическая протеинурия и/или гематурия). Для установления распределения фенотипов групп крови систем АВ0 и Rh среди населения было обследовано 1428 здоровых лиц.

Результаты. Величина относительного риска возникновения ХБП:ГН с МС у мужчин с фенотипом А (II) против 0 (I) преобладала в 7,79 раза такую же у женщин, а у последних с фенотипом АВ (IV) против А (II) – в 5,15 раза такую же у мужчин. Общей чертой была высокая вероятность заболеть исследуемой патологией у носителей фенотипа АВ (IV) Rh– против 0 (I) Rh–. Лица обоего пола с группой 0 (I) Rh– могут быть устойчивыми к развитию этого заболевания.

Заключение. Гендерный диморфизм заключался в том, что наибольший риск заболеть имеют мужчины с фенотипами АВ (IV) Rh– и А (II) Rh– против 0 (I) Rh–, женщины – АВ (IV) Rh– и В (III) Rh– против 0 (I) Rh– и А (II) Rh–. Несколько меньший риск формирования патологии возможен при наличии фенотипов А (II) Rh+ у мужчин и АВ (IV) Rh+ у женщин.

Ключевые слова: *хроническая болезнь почек, гломерулонефрит, группа крови, система АВ0 и Rh, эритроцитарные антигены.*

Сведения об авторе

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