Procedia of Engineering and Medical Sciences
Volume: 01 Issue: 01 | 2022

Functional and Morphological State of the Kidneys in Rheumatoid Arthritis

Sh.A. Mustafayeva | *Bukhara State Medical Institute*

Abstract. The scientific review is devoted to one of the urgent problems of modern medicine -kidney damage in rheumatoid arthritis. The issues of prevalence and risk factors of chronic kidney disease are considered. The kidneys are affected by rheumatoid arthritis more often than it is diagnosed. As a result, early diagnosis of kidney damage in RA patients has important clinical and prognostic significance. In rheumatoid arthritis, the occurrence of chronic kidney disease depends primarily on the duration of the disease and the nature of the inflammatory process. The problem of kidney damage in rheumatoid arthritis has been little studied and requires further research.

Keywords: rheumatoid arthritis, chronic kidney disease, glomerulonephritis, amyloidosis, cardiovascular pathology.

Rheumatic diseases are the oldest human pathology, and are considered the most common ailments of the XXI century. In recent decades, there has been some progress in the field of theoretical and clinical rheumatology. According to E.A. Galushko and E.L. Nasonov, rheumatic diseases include more than 80 diseases and syndromes. [43]

Rheumatoid arthritis (RA) is an autoimmune disease characterized by the development of chronic destructive polyarthritis with frequent involvement of other systems in the pathological process. Extra-articular systemic lesions in RA can have a serious impact on the prognosis of the disease [7,55]. Large-scale studies conducted in recent years have demonstrated the association of RA with a high risk of chronic kidney disease (CKD) and cardiovascular complications, which is associated with an increase in mortality in this category of patients [8,33,20].

The growing population of people with rheumatic diseases creates considerable difficulties for practical healthcare. Due to the multifactorial origin, complex and not fully understood pathogenesis, rheumatoid arthritis (RA) remains in the focus of attention of researchers [40,51,52, 53, 21, 30].

The formation of extra-articular systemic lesions in RA largely determines the severity and prognosis of the disease [42]. At the heart of RA is a chronic progressive lesion of the connective tissue of mainly peripheral (synovial) joints by the type of erosive-destructive polyarthritis.

Earlier in the works of V.A. Nasonova it was noted that women are more often ill with RA than men everywhere (4:1). Moreover, in women, the incidence of RA increases with age [56]. In addition, a higher incidence of RA was found among relatives of patients with the first degree of kinship than in the general population. These data are fully confirmed at the present time [3].

The formation of nephropathy in RA is multifactorial in nature, which presents a variety of their clinical and morphological variants with minor, non-specific changes in urine tests. The course of rheumatoid nephropathy, as well as other chronic kidney diseases, is progressive with the development of nephrosclerosis and a decrease in the bridgehead of functioning nephrons, with an outcome in chronic renal failure, with an extremely unfavorable prognosis, which determines the importance of early diagnosis and treatment of nephropathies in RA.

Renal pathology is detected in RA with a high frequency - about 60%, according to various authors [49,26].

Patients with RA may have various renal diseases: secondary amyloidosis of the kidneys, glomerulonephritis, interstitial nephritis, renal vascular vasculitis, nephrosclerosis, and in some cases their combinations [41,50]. Etiologically, kidney lesions in patients with RA can be divided into 2 groups: firstly, nephropathy as one of the extraarticular manifestations or complications of RA itself, for example, renal vascular vasculitis, chronic glomerulonephritis, secondary amyloidosis, and secondly, as a complication of drug therapy P A: analgesic nephropathy (AN), drug glomerulonephritis. The pathogenesis of such different kidney diseases cannot be the same. Renal vascular vasculitis and glomerulonephritis have an immune nature, mainly immunocomplex; in severe cases, signs of an autoimmune process are recorded. The toxic effects of long-term NSAID intake on the enzyme systems of the epithelial cells of the renal tubules and interstitium underlie the development of AN.

Among the factors determining the progressive loss of renal functions, regardless of the etiology of nephropathies, hemodynamic factors, such as hyperfiltration, systemic and intraclubular hypertension, as well as metabolic factors (dyslipidemia, hyperuricemia, hyperhomocysteinemia) are primarily important [38,47,62], as well as metabolic factors (dyslipidemia, hyperuricemia, hyperhomocysteinemia) [39,64]. A certain contribution to the progression of chronic kidney disease is made by disorders in the hemostasis system, endothelial dysfunction [57, 59,28],the frequency of exacerbations of the disease, the presence of half-moons and the severity of tubulointerstitial changes in the nephrobioptate [24].

In patients with rheumatoid renal vascular vasculitis, a slight transient decrease in renal function is more often detected along with transient hematuria, indicating local inflammation, and severe renal insufficiency is rarely observed [54,1].

The spectrum of renal pathology underlying CKD in RA is quite wide. Secondary amyloidosis for many years occupied the main position among the variants of nephropathy in RA patients [61,35]. According to some studies, there is a tendency to change the structure of kidney damage in RA [5].

Many researchers have noted that in RA patients, the development of CKD and the severity of its manifestations are determined by the duration and activity of the underlying disease, age, the presence of arterial hypertension (AH), lipid metabolism disorders and hyperglycemia [65, 2,17].

The unfavorable prognostic significance of kidney damage in rheumatoid arthritis (RA) has been actively attracting the attention of researchers in recent years [9].

Certain clinical variants of involvement of the kidneys in the pathological process in rheumatoid arthritis are noted in most patients [45].

https://procedia.online/ ISSN-2795-563X

Various variants of kidney damage in rheumatoid arthritis are described, in particular, glomerulonephritis, amyloidosis, vasculitis, as well as iatrogenic forms (analgesic tubulopathy, membranous nephropathy, etc.) [48, 37,44].

It is noteworthy that in real clinical conditions, morphological verification of renal pathology may not be performed for a long time in such patients for a number of objective reasons. Early manifestations of functional renal disorders, especially with their moderate severity, do not always attract the attention of clinicians, while the progression of chronic kidney disease (CKD) in RA can be rapid, especially in old age, as well as in association with cardiovascular pathology [10,15].

According to some researchers, the development of CKD in RA may be associated with cardiovascular damage to a greater extent than with the activity of RA itself [16].

It is noteworthy that the amount of data on factors contributing to the development of cardiovascular pathology, as well as various variants of nephropathies and chronic kidney disease in RA is insufficient, and the available information is scattered, somewhat contradictory [32,19].

It is noteworthy that in recent years, leading world experts have proposed to isolate RA in the elderly – with a debut older than 60 years (the so-called elderly-onset rheumatoid arthritis) [27], and there is a tendency to increase the occurrence of this form [36]. This variant of the disease has some differences from RA with a debut at a younger age (less high activity of arthritis, more frequent seronegativity, usually a more favorable course), at the same time, it should be noted that the features of the formation of cardiovascular and renal pathology in both early and late onset of RA continue to be studied.

In-depth scientific studies devoted to the problems of kidney damage in RA, note that among patients with RA, renal dysfunction during life is diagnosed only in 52% of cases [18]. In terms of the frequency of kidney damage, RA is in third place, behind diseases such as systemic lupus erythematosus and systemic vasculitis. Some researchers report that the frequency of immunocomplex vasculitis in RA is 64% according to skin biopsy data, increasing as the duration of the disease increases [63].

According to the literature, men suffering from RA are more predisposed to the development of CKD than women [34].

Apparently, this is due to the higher prevalence of risk factors (smoking, hypercholesterolemia, obesity, hypertension) among males.

Currently, the leading pathogenetic mechanism for the development of glomerular and tubulointerstitial changes in the kidneys is chronic inflammation. In particular, elevated levels of C-reactive blood protein (CRP) in patients with RA cause glomerular vascular endothelial dysfunction and trigger the synthesis of proinflammatory cytokines. The prognostic significance of an increase in the level of inflammatory markers and a decrease in glomerular filtration rate (GFR) in RA individuals have been noted in few studies [23,29]. Previously published studies have shown that in RA patients treated with cytokine inhibitors, kidney function remained stable for a long time [22]. According to other data, in RA and amyloidosis of the kidneys, therapy with tumor necrosis factor alpha inhibitors led to a simultaneous decrease in proteinuria [4,6].

The study of the pathogenesis of glomerulonephritis continues, since existing therapies do not have the desired effectiveness [46,14]. The connection of glomerulonephritis with changes in the equilibrium of cytokine synthesis associated with the mechanisms of the immune response has been proven [13,25].

It has been established that cytokines take part in the regulation of proliferative processes, differentiation,

growth, and cell activity [11,31]. Cytokines help regulate the nature and duration of the immune response and inflammation. The quantitative content of cytokines and their ratio reflect the dynamics of the pathological process, correlate with the activity of the disease, which allows us to judge the effectiveness of the therapy and predict the outcome of the disease [12,58].

However, the degree of cytokine involvement in the development of kidney diseases, including glomerulonephritis, has not been sufficiently studied. Reports on the study of cytokine interactions in glomerulopathy, especially in children, are few, their results are contradictory. Based on the high importance of glomerulonephritis and the significant importance in the pathogenesis of this disease of the immune system, the study of the cytokine profile in immune inflammation in the kidney remains relevant and promising.

Bibliography

1. *Bacon P.A., Moots R.J.* Extra-articular rheumatoid arthritis // In Koopman W.J. Arthritis and allied conditions. 13-th. Ed. -1997. P.1071-1089.
2. Crowson CS, Matteson EL, Myasoedova E, et al. The lifetime risk of adult-onset rheumatoid arthritis and other inflammatory autoimmune rheumatic dis-eases // Arthritis Rheum. 2011 Mar;63(3):633-9.doi:10.1002/art.30155
3. England BRM, Mikuls TR. Epidemiology of, risk factors for, and possible causes of rheumatoid ar-thritis.2020.
4. Fernández-Nebro A, Tomero E, Ortiz-Santamaría V, et al. Treatment of rheumatic inflamma-tory disease in 25 patients with secondary amyloidosis using tumor necrosis factor alpha antagonists // Am J Med. 2005;118(5):52-556.doi:10.1016/j.am-jmed.2005.01.028
5. Gois M, Carvalho F, Sousa H, et al. Renal involvement in rheumatoid arthritis: analysis of 53 renal biopsies. *Port J Nephrol Hypert.*2017;31(1):25-30.
6. Gottenberg JE, Merle-Vincent F, Bentaberry F, et al. Anti-tumor necrosis factor alpha therapy in fif-teen patients with AA amyloidosis secondary to inflam-matory arthritides: a followup report of tolerability and efficacy // Arthritis Rheum. 2003;48(7):2019-2024. doi:10.1002/art.11163
7. Hickson LJ, Crowson CS, Gabriel SE, et al. Development of reduced kidney function in rheumatoid arthritis. *Am J Kidney Dis.* 2014;63:206-13. doi: 10.1053/j.ajkd.2013.08.010
8. Hickson LJ, Crowson CS, Gabriel SE, et al. Development of reduced kidney function in rheumatoid arthritis. *Am J Kidney Dis.* 2014;63:206-13. doi: 10.1053/j.ajkd.2013.08.0101,
9. Hickson LJ, Crowson CS, Gabriel SE. Development of Reduced Kidney Function in Rheumatoid Arthritis. *Am J Kidney Dis* 2014;63(2): 206-213
10. Hickson LJ, Crowson CS, Gabriel SE. Development of Reduced Kidney Function in Rheumatoid Arthritis. *Am J Kidney Dis* 2014;63(2): 206-213,
11. Ifuku M., Miyake K., Watanebe M. et al. Various roles of Th cytokine mRNA expression in different formsof glomerulonephritis. *Amer. J. Nephrol.* 2013; 38 (2):115–123. DOI: 10.1159/000353102.
12. Ifuku M., Miyake K., Watanebe M. et al. Various roles of Th cytokine mRNA expression in different forms of glomerulonephritis. *Amer. J. Nephrol.* 2013; 38 (2): 115–123. DOI: 10.1159/000353102.
13. Imig J.D., Ryan M.J. Immune and inflammatory role in renal disease. *Compr. Physiol.* 2013; 3 (2): 957–976.DOI: 10.1002/cphy.c120028

14. Imig J.D., Ryan M.J. Immune and inflammatoryrole in renal disease. *Compr. Physiol.* 2013; 3 (2): 957–976.DOI: 10.1002/cphy.c120028.
15. Jesky M, Lambert A, Burden A. The impact of chronic kidney disease and cardiovascular comorbidity on mortality in a multiethnic population: a retrospective cohort study. *BMJ Open* 2013;3(12). doi 10.1136/bmjopen-2013-003458
16. Jesky M, Lambert A, Burden A. The impact of chronic kidney disease and cardiovascular comorbidity on mortality in a multiethnic population: a retrospective cohort study. *BMJ Open* 2013;3(12). doi 10.1136/bmjopen-2013-003458
17. Kapoor T, Bathon J. Renal manifestations of rheumatoid arthritis // Rheumatic Disease Clinics.2018;44:4:571-584.doi:10.1016/j.rdc.2018.06.008
18. Karie S, Gandjbakhch F, Janus N, et al. Kidney disease in RA patients: prevalence and implication on RA-related drugs management: the MATRIX study // Rheumatology.2008;47:3:350-354.doi:10.1093/rheumatology/kem370
19. Karie S, Gandjbakhch F, Janus N. Kidney disease in RA patients: prevalence and implication on RA-related drugs management: the MATRIX study. *Rheumatology (Oxford)* 2008;47(3):350-354–10
20. Kim HW, Lee CK, Cha HS, et al. Effect of anti-tumor necrosis factoralpha treatment of rheumatoid arthritis and chronic kidney disease.*Rheumatol Int.* 2015;35(4):727-34. doi: 10.1007/s00296-014-31464
21. Kim HW, Lee CK, Cha HS, et al. Effect of an-titumor necrosis factor alpha treatment of rheumatoid arthritis and chronic kidney disease // Rheumatol Int.2015;35(4):727-34. doi:10.1007/s00296-014-3146-4
22. Kim HW, Lee CK, Cha HS, et al. Effect of an-titumor necrosis factor alpha treatment of rheumatoid arthritis and chronic kidney disease // Rheumatol Int.2015;35(4):727-34. doi:10.1007/s00296-014-3146-4
23. Kochi M, Kohagura K, Shiohira Y, et al. In-flammation as a Risk of Developing Chronic Kidney Disease in Rheumatoid Arthritis // PLOS ONE. 2016. doi:10.1371/journal.pone.016022
24. *Koseki Y., Terai C., Moriguchi M. et al.* A prospective study of renal disease in patients with early rheumatoid arthritis // Ann. Rheum. Dis. 2001. V. 60 (4). P. 327—331.
25. Kurts Ch., Panzer U., Anders H.-J., Rees A.J. The immune system and kidney disease: basic concepts and clinical implications. *Nature Rev. Immunol.* 2013; 13: 738–753. DOI: 0.1038/nri3523.
26. *Nacano M., Veno M., Nishi S.* et al. Analisis of renal patology and drug histori in 158 Japanese patients with rheumatoid arthritis // Clin Nephrol. -1998. - Vol.50. - P. 154-166.
27. Rasch EK, Hirsch R, Paulose-Ram R. Prevalence of rheumatoid arthritis in persons 60 years of age and older in the United States: effect of different methods of case classification. *Arthritis Rheum* 2003;48:917–926
28. *Sanchez-Lorada I.G., Tapia E., Avila-Cazado C.* Mild hyperuricemia induces glomerulas hypertension in normal rats // Am. J. Physiol. Renal Physiol. 2002. V. 2823 (5). P. 1105—1110.
29. Shankar A, Sun L, Klein BE, et al. Markers of inflammation predict the long-term risk of developing chronic kidney disease: a population-based cohort study // Kidney Int. 2011;80(11):1231-8.doi:10.1038/ki.2011.283
30. Song L, Yin Q, Kang M, et al. Untargeted metabolomics reveals novel serum biomarker of renal damage

in rheumatoid arthritis // Journal of Pharma-ceutical and Biomedical Analysis.2020;180:113068. doi:10.1016/j.jpba.2019.113068

31. Suárez-Fueyo A., Bradley S.J., Klatzmann D.,Tsokos G.C. T-cells and autoimmune kidney disease.*Nature Rev. Nephrol*. 2017; 13: 329–343. DOI: 10.1038/nrneph.2017.34.
32. Toblli JE, Bevione P, Di Gennaro F. Understanding the mechanisms of proteinuria: therapeutic implications. *Int J Nephrol* 2012;546039. Doi 10.1155/ 2012/546039
33. Tokoroyama T, Ando M, Setoguchi K, et al. Prevalence, incidence and prognosis of chronic kidney disease classified according to current guidelines:a large retrospective cohort study of rheumatoid arthritis patients.*Nephrol Dial Transplant.* 2017;32:2035-42. doi:10.1093/ndt/gfw315
34. Tokoroyama T, Ando M, Setoguchi K, et al. Prevalence, incidence and prognosis of chronic kidney disease classified according to current guidelines: a large retrospective cohort study of rheumatoid arthritis patients // Nephrol Dial Transplant. 2016;0:1-8. doi:10.1093/ndt/gfw315
35. Varshavskij VA, et al. Osobennosti techeniya AA-amiloidoza u bol`ny`kh revmatoidny`m artritom. *Therapeutic Archive*. 2006;78(5):31-6 (In Russ.)
36. Villa-Blanco JI, Calvo-Alen J. Elderly onset rheumatoid arthritis: differential diagnosis and choice of first-line and subsequent therapy. *Drugs Aging* 2009;26:739-750
37. Zhigalov SA, Marasaev VV, Bazhina OV. Glomerulyarnye porazheniya pochek pri revmatoidnom artrite. *Klin nefrol* 2013;6:42-45
38. *Азарова В.В., Фирсов Н.Н., Козловская Л.В. и др.* Нарушение реологических свойств крови у больных гломерулонефритами // 1-й съезд нефрологов России: тез. докл. Казань, 1994. С. 5.
39. *Азарова В.В., Фирсов Н.Н., Козловская Л.В. и др.* Нарушение реологических свойств крови у больных гломерулонефритами // 1-й съезд нефрологов России: тез. докл. Казань, 1994. С. 5.
40. Александров ВА, Шилова ЛН, Александров АВ. Особенности определения скорости клубочковой фильтрации при оценке функции почек у больных ревматоидным артритом // Медицинский алфавит. 2020;(15 (2020)):44-48. doi:10.33667/2078-5631-2020-15-44-48
41. *Балабанова Р. М.* Ревматоидный артрит // В кн.: Руководство по внутренним болезням. Ревматические болезни.Под ред. В. А. Насоновой, Н. В. Бунчука. - М .: Медицина, 1997. - С. 257-295.1
42. *Балабанова Р.М.* Ревматоидный артрит // Ревматические болезни: руководство для врачей / под ред. В.А. Насоновой, Н.В. Бунчука. М.: Медицина, 1997. С. 257—294. , 66. *Нефрология:* руководство для врачей / под ред. И.Е. Тареевой. М.: Медицина, 2000. 688 с.
43. Галушко ЕА, Насонов ЕЛ. Распространенность ревматических заболеваний в России. Альманах клинической медицины. 2018;46(1):32–39.doi:10.18786/2072-0505-2018-46- 1-32-39.
44. Гасанов МЗ, Батюшин ММ, Терентьев ВП, Садовничая НА. Особенности протеомного зеркала мочи пациентов с гломерулонефропатиями различного генеза. *Кубанский научный медицинский вестник* 2012:4:37-42 [Gasanov MZ,Batyushin MM, Terent'ev VP, Sadovnichaya NA. Osobennosti proteomnogo zerkala mochi pacientov s glomerulonefropatiyami razlichnogo geneza. *Kubanskij nauchnyj medicinskij vestnik* 2012:4:37-42
45. Демидова НВ, Гусева ИА, Каратеев ДЕ. Клинико-иммунологические аспекты раннего ревматоидного артрита.*Тер арх* 2010; 5:71-77 [Demidova NV, Guseva IA, Karateev DE.Kliniko-

immunologicheskie aspekty rannego evmatoidnogo artrita.*Ter arh* 2010; 5:71-77

46. *Детская нефрология*. Руководство для врачей.Под ред. М.С. Игнатовой. Изд. 3-е, перераб. и доп.М.: МИА. 2011; 696 с. [*Detskaya nefrologiya.* (Pediatricnephrology.) Ed. by M.S. Ignatova. 3d ed. Moscow: MIA. 2011; 696 p. (In Russ.)
47. *Дун Б.Р., Андерсон Ш., Бреннер Б.* Гемодинамические основы прогрессирования почечных болезней // Современная нефрология: сб. тр. Междунар. нефролог. семинара. М., 1997. С. 162—172.
48. Жигалов СА, Марасаев ВВ, Бажина ОВ. Гломерулярные поражения почек при ревматоидном артрите. *Клин нефрол* 2013;6:42
49. *Крель А.А.. Варшавский В.А.. Каневской М.З., Семейкина О.В.* Поражение почек у больных ревматоидным артритом // Тер. архив. - 1990. - № 6. - С. 104- 113.6, 15.*Boers M.* Renal disorders in rheumatoid arthritis II Arthr. Rheum. - 1990. - Vol.20. - P.57-68.
50. *Крель А.А.. Варшавский В.А.. Каневской М.З., Семейкина О.В.* Поражение почек у больных ревмато идным артритом // Тер. архив. - 1990. - № 6. - С. 104- 113.6, 15.*Boers M.* Renal disorders in rheumatoid arthritis II Arthr. Rheum. - 1990. - Vol.20. - P.57-68.
51. Мамасаидов АТ, Абжамилова ЖА, Абдурашитова ДИ. Ассоциация аутоимунного тиреодита и спонтанной активности В-лимфоцитов при ревма-тоидном артрите // Наука. Образование. Техника. 2019;1(64):103-107
52. Мамасаидов АТ, Эшбаева ЧА, Калматов РК. О патогенетическом и клиническом значении антигенспецифической В-клеточной активации при раннем ревматоидном артрите // Современная наука: актуальные проблемы теории и практики. Серия: Естественные и технические науки. 2018;10:79-82
53. Мурадянц АА, Шостак НА, Кондрашов АА. Остеопороз у больного мужского пола с ревматоидным артритом (клиническое наблюдение) // Клиницист.2014;1:71-76.
54. *Насонов Е.Л., Баранов А.А., Шилкина Н. П.* Вас- кулиты при ревматических заболеваниях // В кн. Васкулиты и васкулопатии. Ярославль: «Верхняя Волга», 1999.-С.515 - 540.
55. Насонов Е.Л., Каратеев Д.Е., Балабанова Р.М. Ревматоидный артрит. В кн.: Ревматология. Национальное руководство. Под. ред.Е.Л. Насонова, В.А. Насоновой. М.: ГЭОТАР-Медиа, 2008; с. 290-331 [Nasonov EL, Karateev DE, Balabanova RM. Rheumatoid arthritis. In: Rheumatology. National Guide. Nasonov EL, Nasonova VA, ed. Moscow: GEOTAR-Media, 2008; p. 290-331 (In Russ.)
56. Насонова ВА, Астапенко МГ. Клиническая ревматология: Руководства для врачей. АМН СССР. М. Медицина, 1989.С.592.
57. *Наточина Н.Ю.* Тромбоцитарное звено гемостаза и коррекция его нарушений при гломерулонефритах у детей: дис. канд. мед. наук. СПб., 2000. 160 с.
58. Орадова А.Ш., Устенова Г.О., Стабаева Г.С. Методы исследования цитокинов (обзорная статья). *Medicine.* 2014; (10): 84–87. [Oradova A.Sh., Ustenova G.O., Stabaeva G.S. Methods of cytokine research (review article). *Medicine.* 2014; (10): 84–87. (In Russ.)\
59. *Ратнер М.Я., Серов В.В., Варшавский В.А. и др.* Прогностические факторы ускоренного прогрессирования хронического гломерулонефрита и хронических невоспалительных

нефропатий // Терапевт. арх. 1998. № 6. С. 7—11.

60. Саркисова И.А., Рамеев В.В., Варшавский В.А. и др. Особенности течения АА-амилоидоза у больных ревматоидным артритом. *Терапевтический архив.* 2006; 78(5):31-6 [Sarkisova IA, Rameev VV,
61. Саркисова И.А., Рамеев В.В., Козловская Л.В. Ревматоидный артрит как основная причина развития АА-амилоидоза. *Клиническая геронтология.* 2009;15(2):14-20 [Sarkisova IA, Rameev VV, Kozlovskaya LV. Revmatoidnyj artrit kak osnovnaya prichina razvitiya AAamiloidoza.*Klinicheskaya gerontologiya.* 2009;15(2):14-20 (InRuss.)
62. *Сигидин Я.А., Гусева Н.Г., Иванова М.М.* Диффузные болезни соединительной ткани: руководство для врачей. М.: Медицина, 2004. 542 с.
63. Современные принципы диагностики и лечения хронической болезни почек: методическое руководство для врачей [Электронный ресурс] / М.Ю. Швецов, И.Н. Бобкова, И.Б. Колина, Е.С. Камышова; под ред. Е.М. Шилова. Саратов, 2011.Ч. 2. 50.
64. *Ставская В.В., Рябов С.И., Клемина И.К.* О клиническом значении тубулоинтерстициальных изменений при хроническом гломерулонефрите // Клинич. медицина. 1988. № 10. С. 125—130.
65. Щаднева СИ, Калягин АН. Поражения почек при ревматических заболеваниях // Современные проблемы ревматологии.2014;6:6:10-26.