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STRATIFICATION OF NEPHROCEREBRAL AND CARDIOVASCULAR RISK IN CHRONIC GLOMERULONEPHRITIS (LITERATURE REVIEW)

Abstract

This article analyzes the literature data concerning the origin and progression of cerebrovascular and cardiac diseases in renal dysfunction. Cardiovascular diseases and chronic kidney disease have common "traditional" risk factors, while the growth of patients with renal impairment population currently occurs mainly due to secondary renal damage on the background of socially important diseases such as obesity, hypertension, atherosclerosis, type 2 diabetes mellitus, ischemic heart disease, and chronic heart failure. The presented data of scientific researches show the direct correlation between the decrease of the renal function and the increased risk of cardio- and cerebrovascular diseases and death, irrespective of other risk factors. Obesity and associated biological substrates are independent risk factors for persistent impairment of renal function. An increase in the body mass index causes both direct damage to the kidneys due to the disrupted synthesis of cytokines with nephrotoxic action by adipose tissue, and indirect damage by inducing the development of type 2 diabetes mellitus and hypertension, which are the most frequent risk factors for chronic kidney disease and cardiovascular diseases. The data are presented on the role of endothelial dysfunction in impaired renal function, which contributes to the formation of atherosclerosis, and the increase in the severity of the atherosclerotic process contributes to an increase in the severity of renal failure. Literature data on the place of the heart rate are also presented. The increase in the heart rate can lead to atherosclerotic induration of the arteries, which is associated increase in pulse wave velocity a violation of the mechanisms of autoregulation of the blood flow in the brain and kidneys.

Key words: *chronic kidney disease, glomerular filtration rate, risk factors, cardiovascular risk, cerebrovascular diseases*

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AP — atheromatous plaque; LVH — left ventricular hypertrophy; IHD — ischemic heart disease; MI — myocardial infarction; BMI — body mass index; LA — left atrium; CS — cerebral stroke; MRI — magnetic resonance imaging; DM —

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diabetes mellitus; GFR — glomerular filtration rate; CVD — cardiovascular diseases; CVC — cardiovascular complications; CIMT — carotid intima-media thickness; LVEF — left ventricular ejection fraction; AF — atrial fibrillation; RF — risk factors; CKD — chronic kidney disease; CG — chronic glomerulonephritis; HECGM — Holter ECG monitoring; Ch — cholesterol; CeVD — cerebrovascular diseases; HR — heart rate

The onset and progression of cerebrovascular and cardiac diseases in renal dysfunction are becoming relevant due to the widespread prevalence of chronic kidney disease (CKD), in particular chronic glomerulonephritis (CG). Recent studies have found that even a mild decrease in renal function is associated with an increase in the risk of cardio- and cerebrovascular diseases (CeVD) and death, regardless of other risk factors (RF) [1, 2]. Cardiovascular diseases (CVD) and CKD have common "traditional" RF, while the growth of the population of patients with renal impairment currently occurs mainly due to secondary kidney damage in the context of socially significant diseases: obesity [3, 4], hypertension [5, 6], atherosclerosis [7, 8], type 2 diabetes mellitus (DM) [9, 10], ischemic heart disease (IHD) [11, 12], and chronic heart failure [13, 14]. CVD prevalence in a population of patients with renal dysfunction is 64% higher than in individuals with normal renal function [15]. An independent inverse relationship was shown between glomerular filtration rate (GFR) < 60 ml/min / 1.73 m^2 and increased risk of death, cardiovascular complications (CVC), and hospitalization. The overall prevalence of CKD among obese people is 3.94% (3.62% among men and 4.25% among women), with a tendency to increase with age [16]. At the same time, the researchers confirmed that a history of hypertension, DM, myocardial infarction (MI) and cerebral stroke (CS) among the participants was associated with a higher risk of developing renal dysfunction, regardless of gender. Obesity and the associated biological substrates are independent RF of persistent deterioration of renal function: a 10% increase in body mass index (BMI) leads to an increase in the probability of 1.27-fold GFR decline [17]. In recent years, the incidence of glomerulopathy associated with obesity has practically increased 10-fold [18]. M. C. Foster et al. (2011) showed an association of albuminuria among men with an increase in visceral adipose tissue measured by computed tomography [19]. Most researchers agree that an increase in BMI causes direct kidney injury due to impaired synthesis of various nephrotoxic cytokines by adipose tissue, and also causes indirect kidney injury due to the induction of the development of type 2 DM and hypertension, which are the most frequent RF of CKD and CVD [18, 20]. Potential mechanisms of damage to both cardiovascular and renal systems in obesity are also realized through the effects of adipokines, primarily leptin, on the myocardium, the vessel wall and kidney tissue, with the development of generalized endothelial dysfunction [21, 22].

Due to the low resistance of the cerebral and renal arterial blood vessels, these organs in hypertension are subjected to hemodynamic stress due to a deeper penetration of the accelerated pulse wave. Therefore, hypertension contributes to a significant acceleration of the development and progression of atherosclerotic lesions of the main arteries of the brain and kidneys [23, 24]. In turn, a tense hemodynamic situation in case of elevated BP leads to the fact that the formation of atherosclerotic plaque (AP) can be complicated by its destabilization due to the loss of integrity of the plaque cap and the appearance of ulcerations of the plaque surface with parietal thrombus formation, as well as the development of hemorrhage into the plaque with an increase in its volume and obstruction of the lumen of the vessel that feeds the brain and kidneys [25]. Unstable AP can cause the development of cardiocerebral events and the progression of ischemic CKD, especially in elderly persons [26, 27].

An increase in BMI can often be accompanied by an increase in BP associated with the activation of sympathetic tone caused by the development of insulin resistance and atherogenic dyslipidemia [28]. Absolutely, these changes lead to progression of renal dysfunction and CVD. The presence of white matter hyperintensities and silent cerebral infarction is accompanied by an elevated risk of CS, cognitive impairment and dementia [29, 30-32]. Magnetic resonance imaging (MRI) performed on patients with hypertension and without obvious CVDs showed that silent cerebrovascular lesions are even more common (44%) than subclinical

cardiac and kidney damage (21% and 26%, respectively), and are often found in the absence of signs of damage to other organs [33]. In addition, typical for hypertension asymptomatic small deep brain infarcts, leukoaraiosis, atrophic changes in the form of dilation of the subarachnoid spaces and the brain ventricular system, are also a morphological substrate of vascular cognitive impairment.

The role of hypertension in the prognosis of cerebrovascular and cardiac complications in patients with CKD can hardly be overestimated. Timely and adequate correction of hypertension reliably postpones the onset of dialysis-dependent stage of renal dysfunction. According to some researchers, the frequency of hypertension is up to 40% at stage 1-2 of CKD, that is close to the frequency of hypertension in the general population [34, 35]. Diastolic dysfunction and/or left ventricular hypertrophy (LVH) developing in hypertension causes overload and dilation of the left atrium (LA), distension of ostia of pulmonary veins, which is a morphological prerequisite for the onset of cardiac arrhythmias, in particular, atrial fibrillation (AF) [36, 37]. On the one hand, high group ectopic electrical activity of the myocardium is a predictor of AF, and on the other hand, it is a predictor of the development of the LV geometry impairment [37, 38]. According to P. Kirchhof et al. (2016), in CKD, the risk of AF development is 2.5% at the 1st and 2nd stages of the disease, and the probability of AF is increased to 68% if GFR is ≤ 60 ml/min [38]. In another study by J. P. Piccini et al. (2013), a further decline in GFR (≤ 58 ml/min) by 5 ml/min was found to be accompanied by an increase in the development of CS by 9% [39]. It has been established that an increase in the risk of CS in AF is inversely related to the rate of decline in GFR [40], the progression of the disease, the development of the dialysis-dependent stage of CKD in the presence of AF [41], and an increased risk of MI [42]. It is important to note that timely, rational antihypertensive therapy reduces the relative risk of recurrent CS by 19% and coronary events by 20–25% [43, 44]. An ultrasound examination of the carotid arteries with measurement of carotid intima-media thickness (CIMT) and the assessment of the presence of plaques allows us to predict both cerebrovascular diseases (CeVD) and IHD, regardless of traditional cardiovascular RF [45, 46].

Endothelial dysfunction is already present in the early stages of CKD [47]. As CKD and endothelial dysfunction progress renal impairment contribute to the formation of atherosclerosis, an increase in the severity of the atherosclerotic process contributes to an increase in the severity of renal failure. At the same time, activation of inflammation processes and endothelial dysfunction occur concurrently with a decrease in GFR, an additional laboratory manifestation of which can be an increased CRP level in plasma. Higher concentrations of CRP are associated with accelerated loss of renal function on the one hand, and progression of endothelial dysfunction on the other hand [48]. Activation of inflammation occurs in parallel with the enhancement of the mechanisms of apoptosis with the underlying CKD [49]. APs are often detected in the carotid arteries when CIMT value is normal [50, 51]. The appearance of APs and an increase in CIMT were obtained in the study by O. V. Piyankina et al. among the patients with CKD at the predialysis stage of the disease [52]. Moreover, an *in vivo* study of the APs structure revealed its increased vulnerability in case of renal dysfunction [53].

Heart rate (HR) is a specific marker of life expectancy, reflecting the state of metabolism in the body [54]. Slowing the heart rate improves the balance between myocardial oxygen supply and demand in patients with IHD and significantly reduces the risk of cardiovascular complications and death. Increased HR is one of the predictors of hypertension and kidney hemodynamic stress development [55]. In the Framingham Heart Study, the overall mortality and mortality from CVD in people with hypertension almost doubled with an increase in HR for every 40 beats per min, regardless of additional RF [56]. At the same time, an increase in heart rate at rest can be a marker of imbalance of the autonomic nervous system, i.e. suppression of vagal activity or increasing sympathetic activity [57]. High HR increases the risk of AP damage due to hydrodynamic disorders, which underlies the development of acute cardiovascular and nephrocerebral events [58]. The mechanism of anti-atherosclerotic action of low HR is probably due to a positive effect on arterial stiffness. In contrast, the increase in HR can lead to atherosclerotic induration of the arteries, which is associated with

an increase in pulse wave velocity. Certainly, autoregulation of blood flow in the brain and kidneys is disturbed due to non-uniform elasticity, the presence of multiple arterial branches and low resistance of blood vessels.

The negative effect of an increase in HR is realized by several mechanisms, including an increase in myocardial oxygen consumption, a decrease in coronary blood flow during the diastole, an increased fibrillation threshold, stimulation of atherogenesis and AP ruptures, and possibly a reduction in renal blood flow (especially in the elderly patients). Conversely, HR reduction in myocardial ischemia reduces myocardial oxygen demand, prolongs the diastole, increases the blood supply to the damaged myocardial areas (in particular, the subendocardium), prevents AP rupture, has a beneficial effect on the ischemic myocardium and supports the contractile function of the heart, thereby inhibiting GFR decline. According to Copie et al. [59], HR assessed during Holter ECG monitoring (HECGM) has a prognostic value even higher than LVEF, which is usually used as a prognostic index. K. H. Bonaa et al. (1992) studied the relationship between HR and levels of cholesterol (Ch) and its fractions in plasma in more than 19,000 women and men of young and middle age [60]. Correlation of heart rate with the severity of coronary atherosclerosis was found in people who had myocardial infarction at a young age [61]. In this study, the increase in HR at rest by 5 beats per min corresponded to the progression of the lesion from 0.21 to 0.27 points [62]. Thus, summing up the data of literature analysis, we note that a comprehensive clinical and instrumental examination is necessary in case of renal dysfunction in order to identify potential RF of nephrocerebral and cardiovascular disorders to prevent serious cardiovascular and renal complications.

Conclusion

The complex integrative relationship of RF of nephrocerebral and cardiovascular disorders in renal dysfunction gives us grounds for further scientific research, which is of undoubted importance for improving the effectiveness of preventive measures, as well as improving the prognosis of disease and life in this category of patients.

Conflict of Interests

The authors declare no conflict of interests.

References

1. Moiseev V.S., Mukhin N.A, Smirnov A.V. Cardiovascular risk and chronic kidney disease: cardio-nephroprotection strategies. Journal of Cardiology. 2014; 8: 7-37. DOI:10.15829/1560-4071-2014-8-7-37 [In Russian].
2. Mark P.B. Strategies to manage cardiovascular risk in chronic kidney disease. Nephrology Dialysis Transplantation. 2017; 33(1): 23-25. DOI:https://doi.org/10.1093/ndt/gfx329.
3. Stenvinkel P., Zoccali C., Ikizler T. A. Obesity in CKD-what should nephrologists know? Journal of the American Society of Nephrology. 2013; 24(11): 1727-1736. DOI:10.1681/ASN.2013040330.
4. Bouquegneau A., Vidal-Petiot E., Moranne O. et al. Creatinine-based equations for the adjustment of drug dosage in an obese population. British journal of clinical pharmacology. 2016; 81(2): 349-361. DOI:https://doi.org/10.1111/bcp.12817.
5. Levey A.S., Rocco M.V., Anderson S. et al. K/DOQI clinical practice guidelines on hypertension and antihypertensive agents in chronic kidney disease. American Journal of Kidney Diseases. 2004; 43: 5. Suppl.1.
6. Chazova I.E., Nedogoda S.V., Zhernakova Iu.V. et al. Guidelines for management of patients with arterial hypertension and metabolic disorders. Cardiological bulletin. 2014; 9(1): 3-57. [In Russian].
7. Lamprea-Montealegre J.A., Astor B.C., McClelland R.L. et al. CKD, plasma lipids, and common carotid intima-media thickness: results from the multi-ethnic study of atherosclerosis. Clinical Journal of the American Society of Nephrology. 2012; CJN. 02090212.DOI:10.2215/CJN.02090212.
8. Volkov M.M., Degtereva O.A., Smirnov A.V. et al. Atheroclerosis as the main factor of calcinosis of the heart valve apparatus in the predialysis period of chronic kidney disease. Nephrology. 2007; 11(4): 47-54. [In Russian].
9. Algorithms of specialized medical care for patients with diabetes mellitus. Edited by I.I. Dedov, M.V. Shestakova, A.Yu. Mayorov. 8th edition. M. 2017. FSBI «NMIC Endocrinology of the Ministry of Health of Russia» [Electronic resource. date of the application: 28.08.2018]. 20:1S:1-112. DOI: 10.14341/DM20171S8. [In Russian].
10. Whaley-Connell A.A., Sowers J.R., McCullough P.A. et al. KEEP Investigators. Diabetes mellitus and CKD awareness: the kidney early evaluation program (KEEP) and national health and nutrition examination survey (NHANES). American Journal of Kidney Diseases. 2009; 53(4): S11-S21. DOI: https://doi.org/10.1053/j.ajkd.2009.01.004.
11. Sarnak M.J., Levey A.S., Schoolwerth A.C. et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. Circulation. 2003; 108: 17: 2154-2169. DOI:https://doi.org/10.1161/01.CIR.0000095676.90936.80.

12. Levin A. The clinical epidemiology of cardiovascular diseases in chronic kidney disease: clinical epidemiology of cardiovascular disease in chronic kidney disease prior to dialysis. Seminars in dialysis. Oxford, UK: Blackwell Science Inc. 2003; 16(2): 101-105. DOI:https://doi.org/10.1046/j.1525-139X.2003.16025.x.
13. Serov V.A., Shutov A.M., Menzorov M.V. et al. Epidemiology of chronic kidney disease patients with chronic heart failure. Nephrology. 2010; 14(1): 50-55. [In Russian].
14. Crowley M.J., Diamantidis C.J., McDuffie J.R. et al. Clinical outcomes of metformin use in populations with chronic kidney disease, congestive heart failure, or chronic liver disease: a systematic review. Annals of internal medicine. 2017; 166(3): 191-200. DOI:10.7326/M16-1901.
15. Go A.S., Chertow G.M., Fan D. et al. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. New England Journal of Medicine. 2004; 351(13): 1296-1305. DOI:10.1056/NEJMoa041031.
16. Dong Y., Wang Z., Chen Z. et al. Comparison of visceral, body fat indices and anthropometric measures in relation to chronic kidney disease among Chinese adults from a large-scale cross-sectional study. BMC nephrology. 2018; 19(1): 40. DOI:https://doi.org/10.1186/s12882-018-0837-1.
17. Despres J.P. Abdominal obesity: the most prevalent cause of the metabolic syndrome and related cardiometabolic risk. Eur Heart J. 2006; 8(suppl.B): 4-12. DOI:https://doi.org/10.1093/eurheartj/sul002.
18. Kambham N., Markowitz G.S., Valeri A.M. et al. Obesity-related glomerulopathy: an emerging epidemic. Kidney Int. 2001; 59(4): 1498-1509. DOI:https://doi.org/10.1046/j.1523-1755.2001.0590041498.x.
19. Foster M.C, Hwang S.J., Massaro J.M. et al. Association of subcutaneous and visceral adiposity with albuminuria: the Framingham Heart Study. Obesity (Silver Spring). 2011; 19(6): 1284-1289. DOI:https://doi.org/10.1038/oby.2010.308.
20. Kovesdy C.P., Furth S., Zoccali C. World Kidney Day Steering Committee.Obesity and kidney disease: hidden consequences of the epidemic. Journal of Endocrinology, Metabolism and Diabetes of South Africa. 2017; 22(1): 5-11. DOI:https://doi.org/10.1080/16089677.2017.1299975.
21. Malyszko J. Mechanism of endothelial dysfunction in chronic kidney disease. Clinica chimica acta. 2010; 411(19-20): 1412-1420. DOI:https://doi.org/10.1016/j.cca.2010.06.019.
22. Briffa J.F., McAinch A.J., Poronnik P., & Hryciw D.H. Adipokines as a link between obesity and chronic kidney disease. American Journal of Physiology-Renal Physiology. 2013; 305: 12: F1629-F1636. DOI:https://doi.org/10.1152/ajprenal.00263.2013.
23. Diagnostics and treatment of arterial hypertension. Russian recommendations (the fourth revision). Systemic hypertension. 2010; 3: 5–26. [In Russian].
24. Mathew R.O., Bangalore S., Lavelle M.P. et al. Diagnosis and management of atherosclerotic cardiovascular disease in chronic kidney disease: a review. Kidney international. 2017; 91(4): 797-807. DOI:https://doi.org/10.1016/j.kint.2016.09.049.
25. Yu F.P., Zhao Y.C., Gu B. et al. Chronic Kidney Disease and Carotid Atherosclerosis in Patients with Acute Stroke. The neurologist. 2015; 20(2): 23-26. DOI:10.1097/NRL.0000000000000044.
26. Fomin V.V., Moiseev S.V., Shvetsov M.Yu. et al. Arterial hypertension in clinical assessment of ischemic renal disease. Ther. archive. 2005; 77(6): 27-32. [In Russian].
27. Fomin V.V. Ischemic kidney disease: clinical and pathogenetic features, diagnostics, treatment. Clinical medicine. 2008; 86(4): 8-14. [In Russian].
28. Siew E.D., Ikizler T.A. Insulin resistance and protein energy metabolism in patients with advanced chronic kidney disease. Seminars in dialysis.Oxford, UK: Blackwell Publishing Ltd. 2010; 23(4): 378-382. DOI:https://doi.org/10.1111/j.1525-139X.2010.00763.x.
29. Longstreth W.T.Jr., Manolio T.A., Arnold A. et al. Clinical correlates of white matter findings on cranial magnetic resonance imaging of 3301 elderly people. The Cardiovascular Health Study. Stroke. 1996; 27(8): 1274–1282. DOI: 10.1161/01.STR.27.8.1274.
30. Vermeer S.E., Longstreth W.T.Jr., Koudstaal P.J. Silent brain infarcts: a systematic review. Lancet Neurology. 2007; 6(7): 611-619. DOI:https://doi.org/10.1016/S1474-4422(07)70170-9.
31. Wong T.Y., Klein R., Sharrett A.R. et al. Cerebral white matter lesions, retinopathy and incident clinical stroke. JAMA. 2002; 288(1): 67–74. DOI:10.1001/jama.288.1.67.
32. Buyck J.F., Dufouil C., Mazoyer B. et al. Cerebral white matter lesions are associated with the risk of stroke but not with other vascular events: the 3-City Dijon Study. Stroke. 2009; 40(7): 2327–2331. DOI: 10.1161/STROKEAHA.109.548222.
33. Kearney-Schwartz A., Rossignol P., Bracard S. et al. Vascular structure and function is correlated to cognitive performance and white matter hyperintensities in older hypertensive patients with subjective memory complaints. Stroke. 2009; 40(4): 1229-1236. DOI: 10.1161/STROKEAHA.108.532853.
34. Rao M.V., Qiu Y., Wang C., Bakris G. Hypertension and CKD: Kidney Early Evaluation Program (KEEP) and National Health and Nutrition Examination Survey (NHANES), 1999-2004. Am. J. Kidney Dis. 2008; 51(4): S30-S37. DOI:https://doi.org/10.1053/j.ajkd.2007.12.012.
35. Rimoldi S.F., Scherrer U., Messerli F.H. Secondary arterial hypertension: when, who, and how to screen? Eur. Heart J. 2013; 35(19): 1245-1254. DOI:https://doi.org/10.1093/eurheartj/eht534.
36. Franczyk B., Gluba-Brzózka A., Banach M., & Rysz J. The Problem of Atrial Fibrillation in Patients with Chronic Kidney Disease. Current vascular pharmacology. 2016; 14(3): 260-265.
37. Naser N., Dilic M., Durak A. et al. The Impact of Risk Factors and Comorbidities on The Incidence of Atrial Fibrillation. Materia socio-medica. 2017; 29(4): 231-236. DOI:10.5455/msm.2017.29.231-236.
38. Kirchhof P., Benussi S., Kotecha D. et al. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. European Heart Journal. 2016; 37: 2893-2962. DOI:https://doi.org/10.1093/eurheartj/ehw210.

39. Piccini J.P., Stevens S.R., Chang Y. et al. Renal dysfunction as a predictor of stroke and systemic embolism in patients with nonvalvular atrial fibrillation. Circulation. 2013; 127(2): 224-232. DOI: 10.1161/CIRCULATIONAHA.112.107128.
40. Wu G., Cheng M., Huang H. et al. A variant of IL6R is associated with the recurrence of atrial fibrillation after catheter ablation in a Chinese Han population. PloS one. 2014; 9: 6: e99623. DOI:https://doi.org/10.1371/journal.pone.0099623.
41. Bansal N., Xie D., Tao K. et al. Atrial Fibrillation and Risk of ESRD in Adults with CKD. Clinical Journal of the American Society of Nephrology. 2016; CJN. 10921015. DOI:10.2215/CJN.10921015.
42. Olesen J.B., Lip G.Y., Kamper A.L. et al. Stroke and bleeding in atrial fibrillation with chronic kidney disease. New England Journal of Medicine. 2012; 367(7): 625-635. DOI: 10.1056/NEJMoa1105594.
43. The European Stroke Organization (ESO) Executive Committee; ESO Writing Committee. Guidelines for management of ischaemic stroke and transient ischaemic attacks. 2008. Cerebrovasc Dis. 2008; 25(5): 457–507. DOI: 10.1159/000131083.
44. Rashid R., Leonardi-Bee J., Bath Ph. Blood pressure reduction and secondary prevention of stroke and other vascular events. A systematic review. Stroke. 2003; 34(11): 2741-2748. DOI: http://dx.doi.org/10.1161/01.STR.0000092488.
45. Nambi V., Chambless L., Folsom A.R. et al. Carotid intima-media thickness and presence or absence of plaque improves prediction of coronary heart disease risk: the ARIC (Atherosclerosis Risk in Communities) study. J Am Coll Cardiol. 2010; 55(15): 1600-1607. DOI: 10.1016/j.jacc.2009.11.075.
46. Zanchetti A., Bond M.G., Hennig M. et al. Calcium antagonist lacidipine slows down progression of asymptomatic carotid atherosclerosis: principal results of the European Lacidipine Study on Atherosclerosis (ELSA), a randomized, double-blind, long-term trial. Circulation. 2002; 106(19): 2422–2427.
47. Perticone F., Ceravolo R., Pujia A. et al. Prognostic significance of endothelial dysfunction in hypertensive patients. Circulation. 2001; 104(2): 191-196.
48. Tonelli M., Sacks F., Pfeffer M.et al. Biomarkers of inflammation and progression of chronic kidney disease. Kidney Int. 2005; 68(1): 237-245. DOI:https://doi.org/10.1111/j.1523-1755.2005.00398.x.
49. Dounousi E., Koliousi E. Mononuclear leukocyte apoptosis and inflammatory markers in patients with chronic kidney disease. Am J Nephrol. 2012; 36(6): 531-536. DOI:https://doi.org/10.1159/000345352.
50. Murkamilov I.T., Aitbaev R.A., Fomin V.V., Yusupov F.A. Subclinical lesion of carotid arteries in chronic glomerulonephritis. The Russian Archives of Internal Medicine. 2017; 7(4): 300-305. DOI: https://doi.org/10.20514/2226-6704-2017-7-4-300-305. [In Russian].
51. Murkamilov I.T., Sabirov I.S., Fomin V.V., Murkamilova Zh.A., Aytbaev K.A., Rayimzhanov Z.R. Evaluation of nephrocerebral risk using cystatin C in patients with chronic kidney disease. Zhurnal nevrologii i psikhiatrii imeni SS Korsakova. 2018; 118(9): 10-16. DOI:https://doi.org/10.17116/jnevro201811809110. [In Russian].
52. Pyankina O. V., Tatarintsev P. B., Ragozin O. N. Influence of uremic factors in the processes of remodeling of peripheral vessels in patients with chronic kidney disease. Modern problems of science and education. 2013; 1: URL: https://science-education.ru/en/article/view?id=8509. Electronic resource. Date of the application: 28.08.2018. [In Russian].
53. Pelisek J., Hahntow I.N., Eckstein H.H. Impact of chronic kidney disease on carotid plaque vulnerability. J Vasc Surg. 2011; 54(6): 1643-1649. DOI:https://doi.org/10.1016/j.jvs.2011.05.049
54. Levine H.J. Rest heart rate and life expectancy. Journal of the American College of Cardiology. 1997; 30(4): 1104-1106.
55. Levy R.L., White P.D., Stroud W.D., Hillman C.C. Transient tachycardia: prognostic significance alone and in association with transient hypertension. JAMA. 1945; 129(9): 585-588. DOI:10.1001/jama.1945.02860430001001.
56. Gillman M., Kannel W., Belanger A., D`Agostino R. Influence of heart rate on mortality among persons with hypertension: The Framingam study Am Heart J. 1993; 125(4): 1148-1154. DOI: https://doi.org/10.1016/0002-8703(93)90128-V.
57. Schwartz R.J. The neural control of heart rate and risk stratification after myocardial infarction. Eur Heart J. 1999; 1 (Suppl H): H33-H43.
58. Fusrer V., Badimon L., Badimon J.J., Chesebro J.H. The pathogenesis of coronary artery disease and the acute coronary syndromes (I). N Engl J Med. 1992; 326(4): 242-250. DOI: 10.1056/NEJM199201233260406.
59. Copie X., Hnatkova K., Staunton A. et al. Predictive Power of Increased Heart Rate Versus Depressed Left Ventricular Ejection Fraction and Heart Rate Variability for Risk Stratification After Myocardial Infarction. J Am Coll Cardiol. 1996; 27: 270-276. DOI: 10.1016/0735-1097(95)00454-8.
60. Bonaa K.H., Arnesen E. Association between heart rate and atherogenic blood lipid fractions in a population. The Tromso Study. Circulation. 1992; 86: 394-405. PMID:1638708.
61. Perski A., Hamsten A., Lindvall K., Theorell T. Heart rate correlates with severity of coronary atherosclerosis in young postinfarction patients. Am Heart J. 1988; 116(5): 1369-1373. DOI:https://doi.org/10.1016/0002-8703(88)90469-3.
62. Perski A., Olsson G., Landou C. et al. Minimum teart rate and coronary atherosclerosis: independent relations to global severity and rate of progression of angiographic lesion in men with myocardial infarction at a young age. Am Heart J. 1992; 123(3): 609-616. DOI:https://doi.org/10.1016/0002-8703(92)90497-J.

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