# BRITISH MEDICAL JOURNAL

**British Medical Journal**
**Volume 2, No 1., 2022**
**Internet address:** http://ejournals.id/index.php/bmj
**E-mail:** info@ejournals.id
Published by British Medical Journal
Issued Bimonthly
3 knoll drive. London. N14 5LU United Kingdom
+44 7542 987055
Chief Editor
**Dr. Fiona Egea**

*Requirements for the authors.*
*The manuscript authors must provide reliable results of the work done, as well as an objective judgment on the significance of the study. The data underlying the work should be presented accurately, without errors. The work should contain enough details and bibliographic references for possible reproduction. False or knowingly erroneous statements are perceived as unethical behavior and unacceptable.*
*Authors should make sure that the original work is submitted and, if other authors' works or claims are used, provide appropriate bibliographic references or citations. Plagiarism can exist in many forms - from representing someone else's work as copyright to copying or paraphrasing significant parts of another's work without attribution, as well as claiming one's rights to the results of another's research. Plagiarism in all forms constitutes unethical acts and is unacceptable. Responsibility for plagiarism is entirely on the shoulders of the authors.*
*Significant errors in published works. If the author detects significant errors or inaccuracies in the publication, the author must inform the editor of the journal or the publisher about this and interact with them in order to remove the publication as soon as possible or correct errors. If the editor or publisher has received information from a third party that the publication contains significant errors, the author must withdraw the work or correct the errors as soon as possible.*

*British Medical Journal Volume-2, No 1*
10.5281/zenodo.5978986

# Analysis of dysfunctioning podocytes and structural and functional changes in the nephrons (literature review)

Akhmedova Nilufar Sharipovna, Sulaymonova Gulnoza Tolqinjanovna
Bukhara State Medical Institute, Republic of Uzbekistan, Bukhara
e-mail: all_right_2010@mail.ru

**Abstract:** Cardiorenal continuum is the most discussed area in the field of internal medicine when it comes to comorbidity in the last 10 years. Cardiovascular diseases, obesity, type II diabetes, and kidney dysfunction are becoming more and more pandemics of the 21st century. In recent years, the main cause of renal dysfunction is not its primary disease, but hypertension, which means essential arterial hypertension and diabetes mellitius.

**Keywords:** diabetes mellitius, arterial hypertension, podocyt, nephropathy, comorbidity.

**Introduction**. According to experts of the World Health Organization, the increase in the prevalence of chronic non-communicable diseases is considered an epidemic of the XXI century [1,5,8, 55-60].

In recent years, special attention has been paid to diseases that are present or occur on the basis of the underlying disease and are different from it. Such cases were reported by American epidemiologist researcher A. It is referred to by the term comorbidity proposed by Feinstein in 1970 [2,22,31, 49-54].

Data on the prevalence of comorbid conditions vary somewhat and depend on a number of factors, including whether the patient is being treated in a primary care system or a specialized hospital, gender, age, propensity for medical examinations, and a number of other factors [5,9,12, 37-43].

In almost all studies, high levels of comorbidity have been reported to reduce quality of life, leading to impaired social adjustment and increased mortality [3,6,8,12,27, 28-32].

In addition, comorbidity significantly increases health expenditures. In the U.S., follow-up in patients with one, two, three, or more illnesses was 9, 15, 21, and 29%, respectively, according to the general practitioner, with hospitalizations at 1.7, 2.3,

2.9, and 3.2, and the total duration of hospitalizations at 11, 15. , 22 and 32 days, respectively, is a confirmation of this [4,9,23,27,33-36]. Also, the simultaneous occurrence of several diseases in patients dramatically reduces their propensity to treat them. In patients with HD, the intake of hypotensive drugs was found to be reduced by 57% in patients with bronchial asthma or chronic obstructive pulmonary disease, and by 50% in the presence of depression [12,15,18, 34].

The prevalence and increasing number of comorbidities indicates the importance of studying this problem for many countries, including Uzbekistan [7,5,10, 39].

When it comes to comorbidity in the last 10 years, the most discussed area in the field of internal medicine is the cardiorenal continuum. Cardiovascular diseases, obesity, type II diabetes, and kidney dysfunction are becoming more and more pandemics of the 21st century. In this case, the main cause of renal dysfunction is not its primary disease, but hypertension (HD), ie essential arterial hypertension (AH) and diabetes mellitus [11,23,29, 32]. According to the population register, cardiovascular disease and renal dysfunction without diabetes were 6.8%, in AH 15.2%, and in patients with HD and diabetes, the figure was 42% [6,13,28]. According to the NHANES III register, the presence of two risk factors for cardiovascular disease increased ball filtration (CFT) to 1 per minute, 73 m2 increases body weight <60 ml by 3.7 times [14,25,33, 46]. Systolic blood pressure 10 mm. Elevation of the mercury column increases the risk of developing chronic kidney disease (CHKD) by 6%. At high blood pressure (130 / 139-85 / 89 mm Hg), the risk of microalbuminuria is 2.13 times higher than at optimal blood pressure [1,5,36, 49].

According to 2011 statistics on the prevalence of diabetes, 360 million patients were registered and by 2030 their number is projected to reach 552 million.

It is known that in type II diabetes, irreversible severe changes occur in the target organs. Their number increases sharply in comorbid cases, including when accompanied by HD. Co-occurrence of diabetes mellitus and HD is detected in 60% of cases and is a serious risk factor for cardiovascular disease [1,5,8, 60].

GC accounts for 75% of cardiovascular diseases diagnosed in patients with diabetes mellitus [17,20, 29]. Only the presence of type II diabetes increases the

cardiovascular risk by 2 times in men and 3 times in women, and when AH is added to it, the risk increases by 4 times [35, 47].

Diabetes mellitus, like HD, is also a common disease among the population, and according to 2012 data, it affects 8.3% of the planet's population AHed 29-79 years. Over the past 10 years, the number of officially registered people with diabetes in the country has increased 1.5 times and is more than 1,500,000. 5% of the total population is predisposed to this disease [21,29,33].

A vascular complication of diabetes mellitus that develops gradually without clinical signs is diabetic nephropathy, and it is observed in 30-40% of cases when the disease is accompanied by HD [13,19,28]. A small decrease in CFT and the onset of albuminuria resulted in death and development of ChKD from all heart diseases, regardless of other cardiovascular diseases [29].

Reliable data were obtained in a major study conducted by The Chronic Kidney Disease Prognosis Consortium on the CHKD forecast. It was attended by more than 1 million people from 40 countries. The association between CFT and albuminuria has been studied in cases where the risk of developing CHKD is high. Observations have shown that the end of death from general and cardiovascular disease and renal disease is inversely related to CFT, and is directly related to albuminuria [24,26].

The ESSE-RF epidemiological study in the Russian Federation studied renal dysfunction and its association with AH and metabolic risk factors in the general population. CFTs of less than <90 ml per minute per 1.73 m2 of body surface area were detected in 76.8% of women and 23.2% of men. In this case, a decrease in CFT and CFT was more pronounced in cases where it is accompanied by impaired carbohydrate metabolism or diabetes mellitus [32].

The data presented confirm that co-occurrence of HD and diabetes mellitus is common in the population, and in this case nephropathy is more common than in each of them. Therefore, in these cases, early detection of changes in the kidneys before the onset of clinical signs will be of particular practical importance.

The number of patients with nephropathy is growing and the process is becoming a pandemic of the XXI century. Its primary cause is not kidney disease, but

HD and diabetes. In cases where these two diseases are combined, nephropathy has been reported in 43% of patients [34].

Despite the positive results and the introduction of effective antihypertensive drugs, experts from the World Health Organization predict that by 2025 the number of patients with HD will reach 1 billion 56 million people, accounting for almost 30% of the population over 20 years of AHe [1,9,22 ]

It has been more than 100 years since the first treatments for diabetes were introduced. In the twentieth century, a number of positive results have been achieved in its etiology, pathogenesis, the creation of new types of insulin. Nevertheless, the disease remains not only one of the most pressing problems in the world, but is becoming more and more widespread among the population, taking the form of a pandemic [13].

Consequently, the combination of these two common diseases and the growing number of patients with CHKD associated with them will become an important medical and social problem of the XXI century. Naturally, the detection of changes in the kidneys in this comorbid condition before the onset of clinical signs of dysfunction is one of the more pressing problems of medicine.

It is known that microalbuminuria is currently the most widely used method for early detection of nephropathy in various diseases, including HD and diabetes. However, a number of morphological studies have confirmed the presence of characteristic changes in the kidney tissue of patients diAHnosed with microalbuminuria (even, normoalbuminuria) in diabetes mellitus [7,31]. Therefore, structural-functional changes in the kidney occur until microalbuminuria is detected, and therefore it is not appropriate to consider it as a test to be observed before the clinical signs of nephropathy. There are also reliable data confirming that microalbuminuria is not only an early marker of nephropathy, but also a factor in its progression [11,18].

Therefore, the search for markers that allow the detection of nephropathy in various diseases before their early, clinical manifestations is of great practical importance.

The development of modern molecular medicine and experimental nephrology has broadened views on the mechanisms of occurrence of microalbuminuria and proteinuria. In this case, it was confirmed that podocyte cells, which are the main component of the diaphragm, are crucial [9,31].

Podocytes are a complex structural structure that provides its broad functions and adaptive processes in physiological conditions. It also makes cells very sensitive to damage [8,12].

In recent years, the existence of an organic link between albuminuria and ultrastructural and functional disorders of podocytes has been confirmed in a number of experimental and clinical scientific studies [5,8]. These changes have been shown to occur long before microaluminuria [14,32]. The data obtained confirmed that podocytes were involved in the processes much earlier and increased interest in it. This is because the detection of changes in this cell and in nephropathy makes it possible to transport the kidney damage until the clinical signs appear and to stop the process. Under the influence of a number of pathogens, in particular, hemodynamic, metabolic, immune, toxic factors, structural and functional changes in podocytes are observed, ie podocytopathy [9,11,15,19,24]. In this case, the factors affecting it may differ from each other in different diseases, or together occur in the predominance of one or the other. For example, hemodynamic in hypertension, metabolic in diabetes, immune in glomerulonephritis, and other conditions. Symptoms of podocytopathy are smoothing of the legs of podocytes, its hypertrophy, apoptosis, collapse of the basal membrane of the balls into the urinary cavity, manifested by a violation of the permeability of the diaphragm.

In this case, in the urine can be identified whole cells (podocyturia), as well as its protein structures (nephrin, podocytes, etc.). This process eventually leads to a decrease in podocytes in the balls, ie podocytopenia.

It has now been established that the flattening of the podocyte leg branches is a nonspecific reaction of epithelial cells to the above-mentioned pathogenic factors. These podocytes are associated with the remodeling of its dense network as a result of disruption of the actin cytoskeleton. As a result, the location of the diaphrAHm

10.5281/zenodo.5978986
hole shifts to the apical surface of the podocyte, the holes merge with each other, and the glomerular filter permeability is impaired. The grinding of the branches of the podocyte legs has been proven in a number of experimental and clinical scientific studies in diabetes mellitus and it has been confirmed that there is a direct correlation between these changes and the level of albuminuria [13, 14, 15, 19].

According to modern theories, the intercellular diaphragm serves as the main glomerular filter for plasma proteins. The detection of a large number of protein structures in the composition of podocytes indicates that the branches of its legs are a complex cellular structure. It was found that podocytes have a special adhesive property that forms a filter hole, and one of its main components is a structure consisting of transmembranous protein nephrin. On the one hand, it is involved in the binding of actin to the cytoskeleton of podocytes, on the other hand, it is involved in the formation of interstitial diaphragmatic openings as a result of the interaction of extracellular domains.

In the model of Heymanov nephritis called in the experiment, under the influence of the membrane-attacking complex (S56-9) in the immune damAHe of podocytes, its actin skeletal changes occur, the extracellular part of the nephrin is separated and excreted in the urine (nephrinuria). At the same time, before the development of proteinuria in the kidney tissue, under the electron microscope, the foci of podocytes are clearly visible, indicating the destruction of the diaphrAHm at the sites corresponding to the polished area. Later, when massive proteinuria develops, the number of defects increases sharply, alternating with the unevenly spaced diaphrAHm opening [13,14,20,31]. Changes have also been reported in diabetes mellitus with podocytopathy and nephrinuria [16,22,29]. According to co-authors Jim B. proteinuria and microalbuminuria were detected in 100% of diabetic patients and 54% of those with normoalbuminuria were diAHnosed with nephrinuria [10, 14, 15, 19]. These data have led to nephrinuria as a marker of early detection of nephropathy in diabetes mellitus [12, 17, 15, 19].

DamAHe to podocytes occurs in the urine not only with the formation of structural-functional proteins, but also with the appearance of the cells themselves.

10.5281/zenodo.5978986
Podocytes separated from the basal membrane of the glomeruli die as a result of disruption of cell-matrix interactions necessary to maintain their viability [30,31]. When the damAHing factor is exposed for a long time or strongly, the planned death of podocytes i.e. the process of apoptosis is accelerated and this is another mechanism of cell loss. Podocyte viability or death is controlled by pro and antiapoptic factors [13,35]. Angiotensin II (AT-II), AT1-receptor, b1-transforming growth factor (TGEF-b1), Smad-7, active oxygen radicals, separation of podocytes from the basal membrane of capillaries, mechanical elongation, reduction of cyclic activation of kinase-r27 and r21 inhibitors, the main growth factor of fibroblasts, apoptosis-inducing factor activates podocyte apoptosis. Cyclin I, nephrin protein, intracellular apoptosis, vascular endothelial growth factor, etc. have antiapoptic effects [19]. To date, the role of apoptosis in nephropathy has been confirmed in a number of experimental and clinical trials [31,33].

Activation of epithelial-mesenchial transdifferentiation mechanisms also contributes to the loss of podocytes. Under the influence of these processes, they lose their normal structure, cell polarity, intercellular communication, become mobile, resulting in increased migration of the balls from the basement membrane and the development of podocyturia. Transdifferentiated podocytes, like fibroblasts, have the ability to produce matrix proteins (fibronectin, collAHen, etc.) and accelerate the formation of glomerulosclerosis processes.

Podocytes are highly organized, last differentiated cells that have lost their ability to divide during evolution. The statement that nerve cells do not regenerate also applies to complete podocytes [7,13,17]. Another effect of podocytes AHainst damaging factors is hypertrophy [9,31]. A number of intracellular biochemical processes lead to an increase (not division) in the size of podocytes. In the early stages of the process, hypertrophy becomes adaptive. In this way, the balls try to close the exposed areas of the basement membrane. But over time, podocyte hypertrophy becomes less adaptive, as the mechanism of factors that induce it at the same time also exacerbates apoptosis. When 20-40% of podocytes in the balls are lost, synechiae are formed,

Thus, under the influence of the above factors, the destruction of podocytes and its structural and functional changes occur. It can be assumed that these processes become more pronounced when HD and diabetes occur in a comorbid state, and their study is of scientific and practical importance.

**References**

1. Akhmedova N.Sh. Current Approaches to early diAHnostics of chronic kidney disease and evaluated risk factors // European Science review - 2019, № 3-4, 76-79

2. Akhmedova N.Sh. The importance of proteinuria as a predictor of diAHnosis and a risk factor for the development of chronic kidney disease // European Sciences review 2018, 7-8, 84.

3. Akhmedova N.Sh., Khamdamov II Overweight as a risk factor chronic kidney disease and its modern approach to early diAHnosis // International Scientific and Practical CONFERENCE Trends in Science and Technology. Warsaw, Poland, 2018. - vol 3.- P 34-36.

4. Boltayev KJ, Naimova Sh.A. Risk factors of kidney damAHe at patients with rheumatoid arthritis // WJPR (World Journal of Pharmaceutical Research). - 2019. Vol 8. Issue 13. - P. 229 - 235.

5. Coresh J., Selvin E., Stevens L., Manzi J. at al. Prevalence of chronic kidney disease in the United States // JAMA 2007; 298 (17): 2038–2047.

6. Crowe E., Halpin D., Stevens P. NICE guideline: Early identification and manAHement of chronic kidney disease // Brit. Med. J. 2008. V. 337. - P. 1136–1145.

7. Eckardt KU, Coresh J., Devuyst O., Johnson RJ, Kottgen A., Levey AS, Levin A: Evolving importance of kidney disease: from subspecialty to global health burden // Lancet 2013, 382: r. 158–169.

8. Gansevoort RT, Correa-Rotter R., Hemmelgarn BR, Jafar TH, Heerspink HJ, Mann JF, Matsushita K., Wen CP: Chronic kidney disease and cardiovascular risk: Epidemiology, mechanisms, and prevention // Lancet 2013, r. 339–352.

9. Imai E., Matsuo S., Makino H., Watanabe T. et al. Chronic Kidney Disease Japan Cohort (CKD-JAC) Study: Design and Methods // Hypertens Res. - 2008. - Vol.31, N. 6. - P. 1101–1107.

10. KDIGO. Clinical Practice Guideline for the Evaluation and ManAHement of Chronic Kidney Disease // Kidney Int. 2013. V.3, No. l. P. 1–150.

11. Levey A., de Jong P., Coresh J. et al. Lower estimated glomerular filtration rate and higher albuminuria are associated with all-cause and c ardiovascular mortality. A collaborative meta-analysis of high-risk population cohorts // Kidney Int. - 2011. - N. 80. - P. 17–28.

12. Mills KT, Xu Y., Zhang W., Bundy JD, Chen CS, Kelly TN, Chen J., He J .: A systematic analysis of worldwide population-based data on the global burden of chronic kidney disease in 2010 / / Kidney Int - 2015. - Vol. 88 - P. 950–957.

13. Naimova Sh.A. Features Of Kidney DamAHe at Patients with Ankylosing Spondiloarthritis // Texas Journal of Medical Science (TJMS). - 2021. Vol.3. –P. 18-22.

14. Naimova Sh.A. Principles of early diAHnosis of kidney damAHe in patients of rheumatoid arthritis and ankylosing Spondiloarthritis // British Medical Journal - 2021. April. Volume 1. Issue 1. - P. 5-11.

15. Sulaymonova Gulnoza Tulkinjanovna, Raufov Alisher Anvarovich. The influence of defiency of microelements in children with bronchial hyperreactivity // ACADEMICIA: An International Multidisciplinary Research Journal (ISSN: 2249-7137) – 2020. April – Vol. 10, Issue 4, April –P. 846-853.

16. Toussaint N. Screening for early chronic kidney disease CARI (Caring for Australasians with Renal Impairment) National Recommendations // Kidney Health. - 2012. - P. 1–32.

17. Valerie A., Katherine R. Reducing major risk factors chronic kidney disease // Kidney International Supp. - 2017. - Vol 7. - P. 71–87.

18. Sulaymanova G. T., Amonov M. K. Regional Causes of Iron Deficiency Anemia, Pathogenesis And Use Of Antianemic Drugs // The American Journal of Medical

Sciences and Pharmaceutical Research (ISSN – 2689-1026) – 2021. April 30 – P. 165-170.

19. Axmedova N.Sh. Features of screening of renal function in ambulatory conditions // MEDICUS (International medical journal). - Volgograd, 2019, №2 (26) - pp. 17-21.

20. Axmedova N.Sh. Otsenka funktsionalnogo sostoyaniya pochek u patsientov s izbytochnoy massы tela i ojireniem // Biology and medical problems. Samarkand, 2018. - №4 (104). - S.15-18

21. Akhmedova N.Sh., Boltaev K.J., Sulaymonova G.T., Naimova Sh.A. Chronic kidney disease - early diAHnosis and prevention - Lambert Publishing 2020

22. Axmedova N.Sh., Shadjanova N.S. The importance of detection of microalbuminuria in the outpatient setting in the early diAHnosis of chronic kidney disease // Новый день в Медицине - New Day in Medicine 2018, 3 (23), 142-146.

23. Vyalkova A.A., Lebedeva E.N. and soavt. Kliniko-patoge-neticheskie aspekty povrejdeniya pochek pri ojirenii // Nefrologiya, 2014, T. 18, № 3. - p. 24–33.

24. Daminova K.M. Genotypic features of nephropathy in diabetes mellitus type 2 and metabolic syndrome / Dis. on soisk. uchen. step. kand. med. Science. Tashkent, 2011. - 149 p.

25. Kamilova D.N. Sotsialno gigienicheskoe issledovanie invalidizatsii naseleniya goroda Tashkenta po prichine nefro-logicheskix zabolevaniy / Dis. on soisk. uchen. step. kand. med. Science. Tashkent, 2011. 152 p.

26. Kim Yu.A. Klinicheskie aspekty techeniya xronicheskoy bolezni pochek u patsientov s arterialnoy gipertenziey // Vestnik AHIUV. Kazakhstan, 2016, № 1 - p. 6–16.

27. Kobalava J.D., Villevalde S.V., Borovkova N.Yu. and dr. soavt. The prevalence of markers of chronic kidney disease in patients with arterial hypertension: the results of epidemiological research CHRONOGRAPH // Cardiology, 2017, 57 (10) - p. 39–44.

28. Naimova Sh.A. Funktsionalnoe sostoyanie pochek u bolnyx rheumatoidnym arthritom, ankiloziruyushchim spondyloartritom i ix reabilitatsiya // Novyy den v meditsine. - 2021. № 2 (34). - S 316- 324.

29. National recommendations. Chronic bolezn pochek: osnovnыe printsipy skrininga, diAHnostiki, profilaktiki i podkhody k lecheniyu // Klinicheskaya nefrologiya, 2012, № 4. - C. 4–26.

30. Smirnov A.V., Dobronravov V.A., Kayukov I.G. and others. Chronic disease of the kidneys: the basic principles of screening, diAHnosis, prevention and approach to treatment. Natsionalnye rekomen-datsii // Nephrologiya. 2012, № 1. - p. 89–115.

31. Smirnov AB, Sedov V.M., Lxaaxuu O.E., Kayukov I.G. and dr. Snijenie skorosti klubochkovoy filtratsii kak nezavisimyy faktor riska serdechnososudistoy bolezni // Nefrologiya, 2006, T. 10, № 4, 7–17.

32. Smirnov A.V., Rumyantsev A.Sh. XXI vek - vremya integrativnoy nefrologii // Nefrologiya, 2015, T. 19, № 2 - p. 22–26.

33. Statsenko M.E., Turkina I.A. Vistseralnoe ojirenie kak marker riska multi organnogo porajeniya // Vestnik VolgGMU. - 2017, № 1. - S.10–14.

34. Shadjanova N.S., Sarkisova L.V., Safoeva D.X. Prichiny prezhdevremennyx rody i puti ix preduprejdenie // Jurnal "Novyy den v meditsiny". - 2019. - № 3. - C. 289-292.

35. Shadjanova N.S., Egamova S.Q. Factors of risk of acute myocardial infarction in young and middle-AHed patients // Journal "New day in medicine". - 2019. - № 2 (109). - S. 330 - 334.

36. Shilov E.M. Chronic bolezn pochek i nefropro-tektivnaya terapiya // Metod. rukov. dlya vrachey. M., 2012. - p. 1-70.

37. Amonov M.K. Risk factors for severe and critical patients with COVID-19 // Oriental Renaissance: Innovative, educational, natural and social sciences, 2021 Vol 1, № 10, pp 1080-1084

38. Amonov M.K. Orientation to Acute Kidney Injury in Covid-19 // journal of advanced research and stability (JARS), 2021, Vol. 1 No. 5.

39. Amonov, M. K. Acute Kidney Injury in Coronavirus Disease (Review Article). // *Central asian journal of medical and natural sciences* (2021): 378-382.

40. Abdullaeva U.K. Predicting the risk of atrophic transformation in chronic gastritis using serum pepsinogen // World journal of pharmaceutical research, Faculty of Pharmacy Medical University, Bulgaria, Vol. 8, Iss. 13, 2019, P. 219-228.

41. Abdullaeva U.K., Sobirova G.N., Karimov M.M., Aslonova I.J. The prevalence and possibilities of prevention of noncardial gastric cancer in the Bukhara region // American journal of medicine and medical sciences, 2020, 10(9), P. 679-681.

42. Sobirova G.N., Abdullaeva U.K., Nosirova M.S., Aslonova I.J. Evaluation of the gastrointestinal mucosa by the OLGA system in chronic atrophic gastritis // Journal of critical reviews, Kuala Lumpur, Malaysia, Vol. 7, Iss. 2, 2020, P. 409-413.

43. Karimov M.M., Sobirova G.N., Abdullaeva U.K., Aslonova I.Zh., Tulyaganova F.M. Possibilities of serological diagnosis of atrophic processes of the gastric mucosa // European Journal of Molecular & Clinical Medicine Vol. 7, Iss. 11, 2020, P. 2955-2960.

44. Karimov M.M., Sobirova G.N., Abdullaeva U.K. Chronic gastritis and carcinogenesis issues // Herald of Pancreatic Club, 2019. Iss. 45 (4). P. 65-70. [in Russian]

45. Sobirova G.N., Abdullaeva U.K. Immunopatogenesis of chronic gastritis and its role in carcinogenesis // Journal of Biomedicine and Practice, 1 (4). P. 40-44.

46. Karimov M.M., Sobirova G.N., Abdullaeva U.K., Aslonova I.Zh., Tulyaganova F.M. Possibilities of Serological Diagnosis of Atrophic Processes of the Gastric Mucosa // Annals of the Romanian Society for Cell Biology, , Vol. 25, Issue 1, 2021, Pages. 6168 – 6174.

47. Abdullaeva U.K., Shadjanova N.S. Using the OLGA system in chronic atrophic gastritis // New day in medicine, 2020, №2, P. 9-12.

48. Abdullaeva U.K. The value of interactive teaching methods in improving the level of clinical knowledge of students // Medical education and professional development. 2019, №1 (33), P. 29-32. [in Russian]

49. GN Sobirova, UK Abdullaeva Chronic gastritis and carcinogenesis issues // Central Asian Problems of Modern Science and Education. 2019, Iss. 4, №2, P. 159-172

50. Orziev Zavkiddin Mansurovich, Abdullaeva Umida Kurbanovna, Nurkhanova Nilufar Odiljonovna Study of the effectiveness of cholelitholytic therapy in patients with cholelithiasis, taking into account the type of violation of the contractility of the gallbladder // Science of the Young - Eruditio Juvenium. 2015. №4. P. 50. [in Russian]

51. Karimov M.M., Sobirova G.N., Abdullaeva U.K., Aslonova I.Zh., Tulyaganova F.M. Serological Diagnosis of Atrophic Processes of the Gastric Mucosa // The American Journal of Medical Sciences and Pharmaceutical Research, Vol. 2, Issue 12, 2020, Pages. 118-124

52. DB Mirzaeva, UK Abdullaeva, RR Boboeva The importance of interactive teaching methods in improving the level of clinical knowledge of students // Central Asian Problems of Modern Science and Education Vol. 4, Issue 2, 2019, Pages. 159-166

53. MM Karimov, ST Rustamova, ZhA Ismailova, UK Abdullaeva, ZZ Saatov Diagnostic efficacy of C14 breath test in Helicobacter pyloriosis // Cardiovascular therapy and prevention. 2019. Vol. 18, Issue S1, P. 85-86

54. Abdullaeva U.K., Jalolova V.Z. Study of the effectiveness of cholelitholytic therapy in patients with cholelithiasis, taking into account the type of violation of the contractility of the gallbladder // Bulletin of the Council of Young Scientists and Specialists of the Chelyabinsk Region. 2016. Vol. 5, Issue 4(15), P. 85-86 [in Russian]

55. Orziev Z.M., Abdullaeva U.K. Regional causes of extrahepatic "Subtransaminasemia" // Biology and integrative medicine 2016, №3. P. 28-40. [in Russian]

56. Abdullaeva U.K., Mirzaeva D.B. Regional prospects for metabolic therapy for stable senocardia Summary. // Bulletin of the South Kazakhstan Medical Academyю 2019. P. 74-76 [in Russian]

57. Orziev Z.M., Abdullaeva U.K., Yuldasheva D.H. Method for early prediction of the efficiency of cholelytic therapy based on dynamic control of bild pH indicators in patients with cholelystone disease // Innovative development of modern science. 2014. P.76-79 [in Russian]

58. Orziev Z.M., Abdullaeva U.K. Relationship between the effectiveness of cholelitholytic therapy and the state of contractility of the gallbladder // Bulletin of the Council of Young Scientists and Specialists of the Chelyabinsk Region. 2015. №3(10) [in Russian]

59. Orziev Z.M., Abdullaeva U.K. The effectiveness of cholelitholytic therapy for cholelithiasis // Health is the basis of human potential: problems and ways to solve them. 2015. Iss. 10. №2. P. 610-612. [in Russian]

60. Abdullaeva U.K. Predicting the risk of atrophic transformation in gastritis associated with chronic Helicobacter pylori // abstract of PhD dissertation on medical sciences. Tashkent. 2021. 46-p.