ния, основанного на результатах динамического наблюдения и точки зрения специалистов, такое ортопедическое лечение больных с частичным отсутствием зубов необходимо и в дальнейшем активно использовать в стоматологической практике. Проанализировав научные работы многих исследователей, можно сделать вывод о том, что преимущества фиксации съемных протезов с помощью телескопических систем определяются рядом их возможностей. К ним относятся и распределение жевательного давления на опорные зубы в соответствии с их продольными осями, положительно влияя на пародонт и снижая степень атрофии тканей протезного ложа по сравнению с другими системами фиксации, а также приобретение «идеального» эстетического вида конструкций. Широкая возможность выбора конструкционных материалов и технологий.

Summary

CLASSICAL AND MODERN TELESCOPIC CONNECTION SYSTEMS

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Key words: partial removable prostheses, telescopic attachment system, double crowns.

The purpose of this work is to identify, evaluate and compare the scientific views on the role of telescopic crowns in fixing partial removable dentures. Materials and methods. Analytical study of relevant scientific sources on the issue of the restoration of dentition defects with dentures retained by telescopic crowns. To replace defects in dentition, partial removable dentures are the best option due to their optimal ratio between such parameters as aesthetics, quality and price. The analysis of the relevant literature has demonstrated that using telescopic attachment is the best choice for restoring chewing efficacy, functioning and aesthetics of the maxillofacial system, for removing the deformities of the occlusive surface of the dentition and traumatic occlusion in patients with partial loss of teeth. All of the above mention demonstrates that telescopic dentures have many advantages supported by observing the dynamics of follow up period in individuals who underwent the correction with partial removable dentures and by the opinions of experts. Having analyzed numerous reports of national and international researchers, we can conclude that the advantages of retaining removable dentures by telescopic systems are determined by a number of their capabilities that include the distribution of chewing pressure on the abutment teeth in accordance with their longitudinal axes, providing positive effect on the periodontium and reducing the atrophy of the tissues of the prosthetic bed compared with other retaining systems, as well as by providing a "perfect" aesthetic appearance of the dental appliance.

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KIDNEY DAMAGE IN PATIENTS WITH RHEUMATOID ARTHRITIS

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The purpose of this work is to perform a general analysis of relevant literature on the issue of kidney damage in patients with rheumatoid arthritis. Kidney damage in patients with rheumatic diseases is potentially dangerous, as it can lead to the development of terminal renal failure that may require replacement renal therapy. Amyloidosis often leads to kidney failure in patients with rheumatoid arthritis. Renal amyloidosis more often develops in patients with acute course of rheumatoid arthritis and under maximal immunological disorders. In patients with renal amyloidosis against the background of rheumatoid arthritis, manifestations of joint affection decrease, while the renal-uremic syndrome takes a predominant role. Signs of nephrotic syndrome and chronic renal failure develop gradually. Kidney damage can be caused by medications for rheumatoid arthritis. The choice of the optimal scheme of individual-centred therapy is vitally important for patients, since every aggravation of both rheumatic disease and secondary renal damage leads to the progression of chronic renal failure.

Key words: rheumatoid arthritis, kidney, renal failure, amyloidosis, glomerulonephritis

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Rheumatoid arthritis (RA) is one of the most common and serious diseases of connective tissue (approximately 1% of population in the world and 0,4% of population in Ukraine) [4, 13]. Systemic manifestations of RA determine the severity of its course, prognosis and can result in disability and death of patients [2, 13]. Renal pathology in RA patients ranks a leading position due to its occurrence rate (20 –60% of patients). It depends on the duration of the disease, the intensity of the pathological process, the presence of hemodynamic and metabolic disorders. [1, 2, 6, 15]. It is known that arterial hypertension, anaemia, dyslipidemia are interdependent conditions that can negatively affect the kidneys. [2, 3,13]. The interrelation between the kidney damage, the frequency rate and duration of NSAIDs, i. e. basic therapy drugs, and the age of patients has already been established. The struc-

ture of rheumatoid nephropathy is complex, multifactorial, and can be presented by various clinical and morphological variants. It should be noted that today there are little or no accurate data on the prevalence and occurrence of kidney damage in RA patients. Most of the data is based on the results of biopsies and pathoanatomical studies that indicate the relevance of this problem. The purpose of the work is to carry out generalized analysis of literature on the issue of kidney damage in patients with rheumatoid arthritis.

Approximately 2/3 of kidney damage in RA patients is recognized during the life of patients. There are so-called "specific" clinical forms of kidney damage in RA patients (secondary amyloidosis, rheumatoid glomerulonephritis, while rheumatoid granulomatosis and rheumatoid kidney vasculitis) are less often manifested, as well as iatrogenic ones, which may develop due to the symptomatic or basic therapy with some drugs (drug-induced tubulointerstitial nephritis, membranous nephropathy). At the same time, it should be noted that active therapy in the RA cases can provoke infectious diseases (pyelonephritis, apostematic nephritis, etc.) and vascular complications (systemic vasculitis). As a result, chronic renal failure (CRF) develops, but acute kidney damage is also possible (as with the medical genesis of nephropathy).

It is necessary to emphasize that the literature analyzed presents the contradictory data on the prevalence of various pathologies of the kidneys in RA patients [5, 15]. Nosological diagnosis, most often, is made based on the presence of clinical and laboratory criteria, the most common and the most important of which is proteinuria. Experts do not always pay enough attention to early manifestations of functional renal disorders, especially to moderate proteinuria. The verification of the diagnosis is made on the basis of an intravital biopsy of the kidneys and a histological examination of autopsy material.

The most adverse and threatening condition for the affected kidney is secondary reactive renal amyloidosis, type AA [12, 18]. The analysis of the morbidity statistics in recent years has demonstrated that among the causes of secondary AA-amyloidosis, RA takes the leading place. According to different authors, the numbers of such patients have increased from 35 to 60% [26]. Time-life diagnosis of renal amyloidosis has been established in only 37% of cases. [21], 7 – 8% of RA patients have asymptomatic amyloidosis [18]. It has also been observed carriers HLA-DRB1*04 (alleles*0401, *0405, *0410) and the patients with mutations of alleles R408Q i P369S have been more often diagnosed to have the disease, by 1,7% [25]. Amyloidosis typically develops several years later since the onset of the disease, and naturally, the duration and course of the underlying pathology play an important role as risk factors for the development of this complication. As a rule, it develops in people of different age groups, but more often in aged men. A typical feature of amyloidosis in RA patients is its systemic nature manifested by an increase in the liver, spleen, lymph nodes and damage to other organs. In such patients, articular and febrile RA manifestations reduce and uremic syndrome with a successive change in the stages of amyloidal nephropathy, clinical and laboratory signs of nephrotic syndrome (NS) and the development of chronic renal failure are coming to the fore [2]. A combination of renal amyloidosis with a tubulointerstitial component and vasculitis has been shown as an unfavourable prognostic sign for its progression [27]. Data on the survival of RA patients after confirming AA-amyloidosis are different and ranges from 2 to 5 years. The patients predominantly die from infectious complications and terminal renal failure. Along with clinical signs of kidney damage in RA patients, nephrobiopsy confirmed the diagnosis of renal amyloidosis in 10 – 15% of cases in the USA, in 22% of observations in Japan, and in 30% of cases in Finland [17, 19]. According to national data, amyloidosis is confirmed in 28,6% of cases [13]. An immunofluorescent examination of renal tissue biopsy along with amyloidal deposits revealed accumulations of IgA in the mesangium and IgG along the capillary walls of the glomeruli [19]. Renal amyloidosis in RA patients is often accompanied by the formation of extracapillary crescents, which resemble those in patients with rapidly progressive glomerulonephritis [24].

Rheumatoid glomerulonephritis (RGN) is far less frequently clinically diagnosed, approximately in 37.5% of RA cases and characterized by high intensity of the process, and in some cases indicates the presence of rheumatoid vasculitis. The prognosis for RGN, in contrast to renal amyloidosis, is more favourable, but in 30% of patients persistent hypertension may develop with progressive chronic renal failure [2, 13]. Urinary syndrome and transient proteinuria sometimes with microhematuria are considered as clinically typical. At the same time, the nature of morphological changes in glomerular lesions is variable, more often presented by mesangioproliferative glomerulonephritis (MPGN) that occurs in 70-80% of cases, by mesangiocapillary glomerulonephritis (MCGN) that occurs in 20-30% and membranous glomerulonephritis. In this case, the last two diseases are characterized by a rapidly progressive, severe clinical course [13]. Among other forms of RGN, focal segmental sclerosis-hyalinosis and subacute rapidly progressive extracapillary glomerulonephritis are detected.

There have been distinguished three morphological types of MCGN: classical, with massive subepithelial deposits; membranous and proliferative – a disease of dense deposits and mixed deposits. But the existence of the latter is not generally accepted. Membranous forms of MGN are particularly commonly detected during the iatrogenic development of glomerulonephritis. It is considered that its main morphological features are: "spikes" of capillary basement membranes on light microscopy

(Johnson's colouring), and characteristic "splitting" and "doubling" of the basal membranes in advanced stages. It should be noted that the basis of immune pathogenesis of kidney damage in RA patients is based on two mechanisms: the effect of nephrotoxic antibodies on the structural elements of the glomerular basal membrane (anti-GBM antibody) and the deposition of circulating immune complexes (CIC) in the subendothelial and mesangial zones of the renal glomeruli [13]. The connection of tissue changes in the kidneys with microcirculatory mechanisms that develop according to the general scheme has been proven: immune mechanisms trigger the immune response process, which is accompanied by release of pro-inflammatory cytokines, activation of lysosomal enzymes, kallikrein-kinin, reticuloendothelial systems, and complement system. This stimulates the proliferation of mesangial cells and the production of mesangial matrix (extracellular substance), and attracts neutrophils, lymphocytes, monocytes, and platelets to the area of inflammation from the bloodstream. As a result, the permeability of endothelium membranes increases and results in interstitial lymphoid mononuclear infiltration, fibroblast activation with induction of myofibroplastic phenotype and stimulation of synthetic processes in the extracellular matrix with proliferation of endothelial cells, development of the capillary basal membrane interposition of the glomerular capillaries (BMC) with the development of glomerular hypercellularity, hypertrophic changes in the tubules and stroma, followed by atrophy of the tubule epithelium, development of capillaries, vasculitis, as well as vasoconstrictor and hemocoagulation effects, which leads to the lesion of the kidney tissue [13]. It should be noted that all mechanisms are interconnected by the type of "vicious circle". At the same time, the initial kidney damage in RA patients initiates tubulointerstitial changes that increase the progression of the disease by maintaining pathological changes in the glomerular basement membrane, closing the vicious circle of pathological changes. The morphological form of glomerulonephritis does not affect the severity of damage to certain structures of the kidneys. Although regression analysis shows the relationship between the severity of morphological changes of the glomeruli, tubules, stroma and blood vessels and the level of immonoglobulin deposition in the renal structures. The median glomerular lesion ratio is related to the IgA and IgG in the capillaries, IgG in the stroma, IgM in mesangium, severity of tubular changes is characterized by IgG in the stroma and IgM in mesangium, IgG and IgM in the interstitium as well as IgM in mesangium, and IgA in capillaries.

The data obtained allow us to draw the following conclusion: the deposition of immunoglobulins in the kidney structures is a prognostically unfavourable sign for glomerular and stromal damage and reflects the severity of the process. Correlation analysis reveals that there is the direct relationship between the level of IgA deposition in stroma and IgM in mesangium and the severity of all the individual morphological signs of glomerulonephritis. Thickening of the basement membrane of the glomerular capillaries correlates with the level of IgA in mesangium, IgM and IgG in capillaries. The degree of proliferation of capillary endotheliocytes positively correlates with IgM in capillaries and in mesangium, IgG in capillaries and IgM in stroma, while lymphohistiocytic infiltration of interstitium with IgM and IgG deposition in it.

The constant use of basic drugs by RA patients leads to the necessity of monitoring of side effects, one of which is the development of tubulointerstitial nephritis (TIN) [4,9,11], whose occurrence rate is 47-49% [7, 28]. The basic treating of RA include cytostatics, gold compounds, sulfosalazine, hydroxychloroquine, D-penicylamine, infliximab and other drugs. Symptomatic remedies include NSAIDs. GCS therapy has symptomatic and pathogenetic properties. [16]. Each of these drugs can cause changes in the kidneys.

The nephrotoxicity of the "gold standard" basic therapy with methotrexate depends on the dose of the drug and the rate of elimination. When administered in large doses (1-12 g/м^2), under acidic conditions of urine, precipitation of metabolite crystals inside the tubules occurs, and after 1-2 days a crystal-induced non-oliguric and non-proteinuric renal failure develops. With a high concentration of the drug in the blood in 47% of cases the glomerular filtration rate halves. Risk factors causing increased methotrexate-mediated nephrotoxicity are older age, male gender, high-dose basic medication, low creatinine clearance and co-administration of antibiotics and proton pump inhibitors.

Patients with kidney damage are not recommended to take D-penicillin and antimalarial drugs (hydroxychloroquine) [9,14,22]. Significant place among the basic RA preparations takes sulfosalazin, which is not prescribed for severe impaired renal function.

The development of extramembranous glomerulonephritis with NS is possible with prolonged use of NSAIDs, since they induce prolonged renal tissue ischemia by inhibiting the synthesis of renal prostaglandins and ultimately lead to the development of diffuse sclerosis and chronic renal falure [23]. Cases of papillary necrosis and pyelonephritis are described [3,8]. Combination of TIN with other types of kidney damage in RA patients can accelerate the chronic renal failure progression. Clinical signs of interstitial nephritis are polyuria and nocturia, an increase in the level of nitrogenous substances (creatinine and urea) in the blood, electrolyte disturbances and a decrease in the secretory-excretory kidney functioning during a radionuclide study in the absence of proteinuria but in the presence of b2-microglobulinuria.

Diagnostic significance is the expression of tubulointerstitial disorders depending on the degree of albuminuria. It has been proven that even in pa-

tients with normoalbuminuria, a statistically significant increase in alpha-1-microglobulin, compared with healthy individuals indicates the presence of tubular lesions, even at an early stage [22].

It should be noted that long-term therapy with leflunomide, one of basic medications, helps to reduce the content of amyloidal protein in the blood that suggests the rationale for prescribing leflunomide in case of reactive kidney amyloidosis [25].

Some hope in the treatment of secondary amyloidosis rests on anticytokine drugs of biological therapy (adalimumab, infliximab, rituximab, etanercept), which have anti-amyloid action [12].

For the early diagnosis of kidney damage in all RA patients, it is necessary to assess the state of tubulointerstitial tissue in addition to investigation the standard "renal" markers indicating cardiovascular risk as glomerular filtration rate and urine albumin excretion. The earliest marker of kidney damage in RA patients is alpha-1-microglobulin, the increase in which indicates the length of the disease is an average up to 5 years. The goal of optimal nephroprotection consists in the early detection and treatment of anaemia, dyslipidemia, and arterial hypertension.

Subclinical kidney damage in RA patients can also develop at earlier stages of the disease but often remain undiagnosed in clinical practice. Early detection of renal dysfunction can contribute greatly to correcting the therapy chosen, and, thus, can improve the prognosis for RA patients.

In order to reduce the risks due to some nephrotoxicity of basic therapy with methotrexate, it is recommended to carry out hyperhydration balanced in electrolyte composition and osmolarity solutions in a dose 40-60 ml/kg/day and alkalization of urine by prescribing sodium bicarbonate (the reaction should be alkaline on the day of administration and in the next 2-3 days). While maintaining a high concentration of the drug in the blood, regardless of the level of azotemia, high-flux haemodialysis or hemofiltration / diafiltration are indicated based on the calculation of individual clearance depending on the serum methotrexate level. When performing extracorporeal hemocorrection, the methotrexate level reduces by 75% approximately. During active hydration and alkalization of urine, the toxicity of methotrexate can be reduced by 15%. Sodium bicarbonate not only alkalizes urine, but also inhibits the crystal formation. Administration of "loop" diuretics and sodium chloride solution will maintain a high level of filtration and non-toxic level of methotrexate (less than 0.1 mmol / l).

To reduce the frequency and severity of side effects of the tool, patients should be advised to take methotrexate according to the “evening-morning-evening” scheme with a possible transition to the parenteral administration. Methotrexate can not be prescribed simultaneously with aspirin, diclofenac, trimethoprim, alcohol and caffeine are excluded.

In cases of the formation of terminal renal failure against the background of nephropathy due to systemic pathology, it is vital to switch to hemo-or peritoneal dialysis, kidney transplantation that can significantly prolong the life expectancy of patients.

Prospects for further research

Treatment of rheumatic diseases and comorbid kidney damage is still remaining to be one of the most challenging issues of modern clinical medicine.

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Реферат

УРАЖЕННЯ НИРОК У ПАЦІЄНТІВ З РЕВМАТОЇДНИМ АРТРИТОМ

Ткаченко Л.А., Ярмола Т.І., Пустовойт Г.Л., Талаш В.В.

Ключові слова: ревматоїдний артрит, нирки, ниркова недостатність, амілоїдоз, гломерулонефрит

Мета даної роботи – узагальнюючий аналіз даних літератури щодо проблеми ураженя нирок у хворих на ревматоїдний артрит. Ураження нирок у пацієнтів з ревматичними хворобами є потенційно небезпечним, оскільки може призвести до розвитку термінальної ниркової недостатності і необхідності застосування замісної ниркової терапії. До ниркової недостатності нерідко призводить амілоїдоз нирок у хворих на ревматоїдний артрит. Частіше амілоїдоз нирок розвивається у хворих з гострим перебігом ревматоїдного артриту та максимальними імунологічними порушеннями. У хворих з амілоїдозом нирок на фоні ревматоїдного артриту суглобові прояви зменшуються, і на перший план виступає нирково-уремічний синдром. Поступово розвиваються ознаки нефротичного синдрому та хронічної ниркової недостатності. Ураження нирок при ревматоїдному артриті можуть бути і медикаментозні, на фоні лікування. Вибір оптимальної схеми індивідуалізованого лікування життєво важливий для пацієнтів, оскільки кожне загострення як ревматичного захворювання, так і вторинного ураження нирок приводить до прогресування хронічної ниркової недостатності.

Реферат

ПОРАЖЕНИЕ ПОЧЕК У ПАЦИЕНТОВ С РЕВМАТОИДНЫМ АРТРИТОМ

Ткаченко Л.А., Ярмола Т.И., Пустовойт А.Л., Талаш В.В.

Ключевые слова: ревматоидный артрит, почки, почечная недостаточность, амилоидоз, гломерулонефрит

Цель данной работы - обобщающий анализ данных литературы по проблеме поражений почек у больных ревматоидным артритом. Поражение почек у пациентов с ревматическими болезнями является потенциально опасным, поскольку может привести к развитию терминальной почечной недостаточности и необходимости применения заместительной почечной терапии. К почечной недостаточности нередко приводит амилоидоз почек у больных ревматоидным артритом. Чаще амилоидоз почек развивается у больных с острым течением ревматоидного артрита и максимальными иммунологическими нарушениями. У больных с амилоидозом почек на фоне ревматоидного артрита суставные проявления уменьшаются, и на первый план выступает почечно-уремический синдром. Постепенно развиваются признаки нефротического синдрома и хронической почечной недостаточности. Поражение почек при ревматоидном артрите может быть и медикаментозным, на фоне лечения. Выбор оптимальной схемы индивидуализированного лечения жизненно важен для пациентов, поскольку каждое обострение как ревматического заболевания, так и вторичного поражения почек приводит к прогрессированию хронической почечной недостаточности.