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Renal Damage in Rheumatological Diseases

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Abstract The kidney is a classic target organ for rheumatologic diseases. The importance of identifying kidney damage in rheumatic diseases is due to the steady progression of renal pathology. This article presents the results of an analysis of scientific sources by foreign researchers in recent years on the study of the development of chronic kidney disease in rheumatological patients. The prevalence, diagnostic criteria, and markers of kidney damage in these patients are described.

Keywords Chronic kidney disease, Drug-induced nephropathy, Rheumatological disease

1. Introduction

Kidney damage in systemic diseases is very common. Most often, secondary nephritis develops with systemic lupus erythematosus, systemic vasculitis, gout, systemic scleroderma, and much less often with arthritis.

The unfavorable prognostic significance of kidney damage in rheumatoid arthritis (RA) has actively attracted the attention of researchers in recent years [1]. Some clinical variants of kidney involvement in the pathological process in rheumatoid arthritis are observed in most patients [2]. Various types of kidney damage in rheumatoid arthritis have been described, in particular, glomerulonephritis, amyloidosis, vasculitis, as well as iatrogenic forms (analgesic tubulopathies, membranous nephropathy, etc.) [3-5]. It is noteworthy that in real clinical conditions, such patients may not undergo morphological verification of renal pathology for a long time for a number of objective reasons. Early manifestations of functional renal disorders, especially when they are moderate, do not always attract the attention of clinicians, while the progression of chronic kidney disease (CKD) in RA can be rapid, especially when associated with cardiovascular pathology [1,6].

Kidney damage can be a clinical manifestation and complication of therapy for rheumatic diseases. The importance of identifying kidney damage in rheumatic diseases is due to the steady progression of renal pathology in the absence of adequate treatment (Panevin).

A quarter of all nephrological patients with rheumatological pathology are patients with SLE; this is the most common pathology. At the same time, hemorrhagic and other vasculitis (11.2 and 1.5%, respectively) also make up a large group of observed ones. Less common are amyloidosis (17%), gout (9%), rheumatoid arthritis, rheumatism with kidney damage and other rheumatic diseases.

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When discussing kidney damage in rheumatological diseases, it is important to note that in practice there are various types of damage.

The first, most well-known option is kidney damage as a component of the disease. The most typical development of “renal vasculitis” is with systemic lupus erythematosus, systemic vasculitis (especially with polyarteritisnodosa, Wegener’s granulomatosis, hemorrhagic vasculitis), therefore manifestations of nephritis are the main criteria for diagnosing these diseases. Slightly less often, the kidneys become a target organ in rheumatoid arthritis, systemic scleroderma, gout, and mixed cryoglobulinemia.

The second, well-known option is complications of therapy and, above all, drug-induced nephropathies. Among drug-induced nephropathies in people with rheumatological diseases, the leading ones are:

- “golden kidney” — chronic tubulointerstitial nephritis that develops during treatment with gold preparations, limiting the use of this group of medications;
- acute renal failure associated with the use of non-steroidal anti-inflammatory drugs of various groups;
- chronic renal failure caused by the use of analgesics (analgin, various mixtures of analgin, aspirin, amidopyrine, phenacetin, codeine, etc.) and, rarely, anti-inflammatory drugs;
- arterial hypertension and progression of renal failure while taking cyclosporine, a drug widely used specifically in the treatment of immune nephropathies;
- urate nephropathy in secondary, drug-induced gout (cyclosporine, cytostatics, diuretics, etc.).

Much less often, acute renal failure associated with the use of ACE inhibitors, overdose of diuretics, antihypertensives, and radiocontrast drugs is noted.

Considering the variety of kidney lesions in systemic connective tissue diseases, to assess renal damage it is important to assess urinary syndrome, the function of the glomerular and tubular apparatus, the presence of “extrarenal

signs of activity" (hypertension, edema, diuresis, normochromic anemia), and determine the presence of immunoinflammatory syndrome.

CKD can also accompany the most common diseases of the osteoarticular system (osteoarthritis, ankylosing spondylitis, gouty arthritis, rheumatoid arthritis, etc.) [3,4,6,15], so screening for CKD is necessary already at the onset of rheumatological disease, before prescribing drug therapy, especially in patients with comorbid pathology (chronic tubulointerstitial nephritis, urolithiasis, diabetes mellitus, obstructive uropathy, glomerulopathies, cardiovascular diseases, metabolic syndrome, etc.) [14].

A decrease in the filtration function of the kidneys can be observed in patients with osteoarthritis (OA), which is also most often due to concomitant long-term use of NSAIDs and urolithiasis, therefore it is important to use less nephrotoxic drugs in treatment [2,3,4,16]. Treatment of OA is based on the use of slow-acting symptom-modifying drugs based on glycosamyglycans - SYSADOA, as well as the use of analgesics and non-steroidal anti-inflammatory drugs (NSAIDs).

The kidney is the classic target organ for hyperuricemia. Kidney damage can occur in four clinical and morphological variants: chronic tubulointerstitial nephritis, urate nephrolithiasis and acute uric acid nephropathy (renal acute kidney injury due to intratubular obstruction by uric acid crystals with rapid and abundant formation of uric acid, most often observed during the treatment of malignant neoplasms and increased protein catabolism) and immune complex glomerulonephritis [3,6,9]. According to various studies, kidney damage can occur in 25-75% of cases of gout, while significant dysfunction develops in 40% of cases [9].

In rheumatoid arthritis, kidney damage has a different genesis. What matters is the activity of the disease with a high level of proteins in the acute phase of inflammation and a long duration of the disease. The development of CKD in RA is significantly more common compared to the general population. According to various studies, the incidence of CKD ranges from 20 to 45% [12]. Among the types of kidney damage in RA, the most common amyloidosis with the gradual development of nephrotic syndrome and steadily increasing renal failure, various types of glomerulonephritis (membranous or membranous-proliferative) and glomerulonephritis as part of rheumatoid vasculitis, NSAID-associated tubulointerstitial nephritis, nephroangiosclerosis and fibrosis as an outcome are distinguished. arterial hypertension, concomitant diabetes mellitus [11].

Kidney damage in systemic connective tissue diseases (systemic lupus erythematosus, systemic vasculitis, thrombotic microangiopathy, antiphospholipid syndrome) has its own clinical, morphological and pathogenetic features of damage to the glomeruli and interstitium. Scleroderma kidney is characterized by the development of occlusive and thrombotic vasculopathy. Progressive immunoinflammatory changes in the kidneys during systemic diseases naturally lead to deterioration of kidney function and the development of end-stage renal failure (ESRD). The prognosis for 10-year renal survival with active pathogenetic therapy of systemic diseases, according to various authors, ranges from 28 to 52% [13]. The importance of identifying kidney damage in rheumatic diseases is due to the steady progression of renal pathology in the absence of adequate treatment, while at the same time nephroprotective therapy can prevent a further decrease in renal filtration.

2. Conclusions

The use of NSAIDs is steadily increasing in various fields of medicine, and therefore it is important to increase awareness of physicians and pharmacists regarding the risk of developing NSAID-associated renal side effects.

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