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Current Views about Ulcerative Colitis and its Impact on the Kidneys

Mirakhmedova Nargisa Rizoevna
Bukhara State Medical Institute

Abstract: Increasing interest in the problem of inflammatory bowel diseases (IBD) among medical practitioners and researchers is due to the variety of extraintestinal manifestations, including kidney pathology. In this area, tubulointerstitial nephritis, nephrolithiasis, glomerulonephritis, IgA and IgM nephropathies, and renal amyloidosis are distinguished. The mechanisms of development of tubulointerstitial nephritis in IBD remain the subject of debate. Some researchers view it as an extraintestinal manifestation of IBD related to its activity. It is noted that specific proteins found in the urine may indicate damage to the renal tubular apparatus, often associated with the activity of ulcerative colitis and Crohn's disease.

Keywords: ulcerative colitis, inflammatory bowel diseases, nephritis, glomerulonephritis, kidneys.

Relevance. Tubulointerstitial nephritis is often considered an undesirable effect of basic therapy for IBD. Data on the nephrotoxicity of basic 5-ASA therapy drugs are contradictory. It has been noted that the incidence of tubulointerstitial nephritis in patients with IBD reaches 1%; the pathology is associated with taking 5-ASA [Patel H 2009]. Dose-dependent renal damage due to cyclooxygenase inhibition or a hypersensitivity reaction is suspected. However, the risk of tubulointerstitial nephritis in patients with IBD does not depend on the specific 5-ASA drug. An individual reaction to sulfasalazine is also possible, unrelated to the dose and duration of taking the drug [Patel H 2009]. They indicate the presence of concomitant autoimmune pathology in patients with UC and CD, which becomes a trigger for renal damage and the development of tubulointerstitial nephritis [Ramos-Casals M 2008]. Cyclosporine and tacrolimus prescribed for severe exacerbations of IBD or as second-line therapy have side effects , including dose-dependent nephrotoxicity associated with intense arteriolar vasoconstriction, leading to a decrease in GFR and culminating in interstitial fibrosis [Sereno J 2014]. Cases of tubulointerstitial nephritis during therapy with immunomodulators or anti-TNF drugs have not been described to date.

The diagnosis of kidney pathology in IBD should include nephrolithiasis, which occurs more often in IBD than in healthy people. Low urine pH and decreased urine volume in patients undergoing colon surgery initiate and maintain excess formation and excretion of uric acid [Parks J.H 2003]. Malabsorption of bile salts in patients with ileal surgery, increased permeability of the intestinal wall to oxalates, and a decrease in the number of Oxalobacter formigenes involved in the catabolism of oxalates in the intestine can cause hyperoxaluria [Nazzal L 2015]. IBD activity plays a significant role in the development of nephrolithiasis, increasing dehydration and electrolyte losses, especially in patients with UC. In IBD, oxalate stones are more likely to form. In turn, oxalates have a toxic effect on nephrocytes, changing the permeability of cell membranes, mitochondrial activity, increasing the amount of reactive oxygen species [Dídia B.C 2013]. In recent years, there has been a growing interest among both practitioners and researchers in inflammatory bowel diseases (IBD), including any extraintestinal manifestations. The range of extraintestinal manifestations of IBD is quite wide [Ivashkin VT 2017]. These may include kidney damage, with the incidence of chronic kidney disease and renal failure estimated at 2–15% [Clinical protocols of MH RK, 2017].

In IBD, various morphological variants of glomerulonephritis have been described: membranous, glomerular, membranoproliferative, IgA and IgM glomerulonephritis, focal and segmental glomerulosclerosis [Ambruzs J.M 2014]. The development of glomerulonephritis, especially in CD, is explained by the commonality of genetic (HLA-DR1 and HLA-DR1/DQw5 loci), immunological

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mechanisms of chronic inflammation, impaired production and transport of IgA [Kiryluk K 2014]. The time of occurrence of glomerulonephritis often coincides with the onset of IBD; the clinical picture is largely determined by the morphological variant of glomerulonephritis. Glomerulonephritis activity is associated with IBD activity; with adequate treatment of exacerbations, regression of renal damage is observed [Vegh Z 2017].

Renal amyloidosis is a rare manifestation of IBD, which, as a rule, is diagnosed 10–15 years after verification of the diagnosis. AA amyloidosis is more common, which is clinically manifested by proteinuria, nephrotic syndrome, which is progressive, progressing to renal failure [Iñarrairaegui B.M 2004]. The diagnosis of amyloidosis is complex with a nephrobiopsy or biopsy from the rectum with staining for amyloid. Glomerulonephritis can be classified as a rare extraintestinal manifestation of both ulcerative colitis and Crohn's disease. Lakatos L. et al. when describing the results of a 25-year study with dynamic observation of patients with IBD in Hungary, only 3 cases of glomerulonephritis are cited (1 with ulcerative colitis and 2 with Crohn’s disease) [Lakatos L 2003].

The same publication mentions earlier work by D.S. Pardi et al., devoted to the issue of urological and nephrological complications of IBD, indicating that in the literature there is only a description of clinical cases of this combined pathology [Pardi DS 1998]. In the literature you can find a description of membranous nephropathy associated with familial ulcerative colitis in 12 -year-old girl

A study of the literature data on ulcerative colitis and its effect on the kidneys revealed the following results:

1. Incidence of renal complications in inflammatory bowel disease (IBD):-The incidence of chronic kidney disease and renal failure in IBD is estimated to range from 2% to 15%.
2. Morphological variants of glomerulonephritis in IBD: - Various variants of glomerulonephritis have been described, such as membranous, glomerular, membranoproliferative, IgA and IgM glomerulonephritis, focal and segmental glomerulosclerosis.
3. Factors contributing to the development of glomerulonephritis in IBD: - The commonality of genetic and immunological mechanisms of chronic inflammation, impaired production and transport of IgA can contribute to the development of glomerulonephritis in IBD.
4. Renal amyloidosis as a rare manifestation of IBD: -AA amyloidosis is most often diagnosed 10–15 years after verification of the diagnosis of IBD and can progress to renal failure.

The study results support an association between inflammatory bowel disease and renal complications, such as glomerulonephritis and renal amyloidosis. - It is important to consider that the activity of glomerulonephritis often correlates with the activity of IBD, and adequate therapy for IBD may lead to regression of renal damage. - Rare cases of glomerulonephritis associated with IBD ulcerative colitis and Crohn's disease indicate the need for further research in this area.

Diagnosis of renal amyloidosis in IBD requires a multidisciplinary approach, including nephrobiopsy or rectal biopsy with amyloid staining. - Cases of membranous nephropathy in patients with ulcerative colitis highlight the need for close monitoring of the kidneys in patients with IBD.

These results and discussion highlight the importance of further research and the development of effective strategies for diagnosing and treating renal complications in patients with inflammatory bowel disease.

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