EDITORIAL

REVISTA MEXICANA DE CIRUGÍA DEL APARATO DIGESTIVO

Renal insufficiency in patients with inflammatory bowel disease: An often underestimated complication

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Insuficiencia renal en pacientes con enfermedad inflamatoria intestinal: una complicación a menudo subestimada

REVISTA MEXICANA DE CIRUGÍA DEL APARATO DIGESTIVO / Vol. 15 Núm. 4 / Octubre-Diciembre, 2026 / p. 125-127

INTRODUCTION

Inflammatory bowel disease (IBD), which primarily encompasses Crohn's disease and ulcerative colitis, comprises a group of chronic inflammatory disorders of the gastrointestinal tract characterized by a relapsing course, systemic manifestations, and extraintestinal complications that may significantly affect patients' quality of life and prognosis. Although clinical attention has traditionally focused on controlling intestinal inflammation, renal abnormalities are increasingly recognized as relevant extraintestinal manifestations, the timely identification of which represents a challenge for gastroenterologists, nephrologists, and primary care physicians.

Renal insufficiency in patients with IBD is a multifactorial clinical condition. It may present as acute kidney injury (AKI), generally associated with dehydration, electrolyte disturbances, infections, or adverse effects of pharmacological treatment, or progress to chronic kidney disease (CKD) as a consequence of inflammatory, immunological, metabolic, and obstructive mechanisms. The heterogeneity of these abnormalities, together with their frequently subclinical presentation, contributes to delayed diagnosis and increases the risk of irreversible deterioration in renal function.

From a nephrological perspective, it is essential to recognize that renal involvement in IBD should not be interpreted exclusively as a treatment-related complication. In some patients, it represents a direct manifestation of systemic inflammatory disease, whereas in others, it results from interacting pathophysiological mechanisms that may exacerbate kidney injury.

Epidemiology and clinical relevance

Renal and urological manifestations of IBD occur with variable frequency depending on the population studied, disease duration, diagnostic criteria, and the presence of concomitant risk factors. Reported abnormalities include nephrolithiasis, tubulointerstitial nephritis, glomerulopathies, secondary amyloidosis, and obstructive urinary tract complications.

Crohn's disease, particularly when associated with small bowel involvement, extensive intestinal resections, or stoma formation, is associated with an increased risk of dehydration, enteric hyperoxaluria, and kidney stone formation. Conversely, both Crohn's disease and ulcerative colitis may be associated with glomerular and tubulointerstitial abnormalities of presumed immunological origin.

The clinical importance of these manifestations lies in the fact that impaired kidney function may remain unrecognized for prolonged periods. Furthermore, the coexistence of IBD and CKD complicates therapeutic decision-making, particularly when potentially nephrotoxic medications are required or immunomodulatory treatments need to be adjusted.

Identifying predictors of kidney injury and implementing systematic surveillance strategies are therefore essential components of comprehensive patient care.

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https://doi.org/10.62640/CAD2615401ed

Pathophysiological mechanisms of kidney injury

The relationship between the intestine and the kidneys has gained increasing attention through the concept of the gut–kidney axis, which describes interactions among the intestinal microbiota, epithelial barrier integrity, systemic immune responses, and renal function.

In IBD, increased intestinal permeability facilitates immune system exposure to bacterial products and proinflammatory mediators. Persistent activation of cytokines, including tumor necrosis factor-alpha and various interleukins, may contribute to glomerular and tubulointerstitial inflammation. Likewise, intestinal dysbiosis and alterations in the metabolism of microbiota-derived substances may promote oxidative stress and endothelial dysfunction. Although these mechanisms provide a biologically plausible explanation, their specific causal contribution to kidney injury in humans remains under investigation.[1]

From a clinical standpoint, AKI represents one of the most important complications during periods of active intestinal disease. Persistent diarrhea, gastrointestinal fluid losses, reduced oral fluid intake, and electrolyte disturbances promote hypovolemia and decreased renal perfusion. When severe or prolonged, these abnormalities may lead to acute tubular injury, particularly in patients with preexisting kidney disease.

Among glomerular manifestations, immunoglobulin A nephropathy occupies a prominent position. Its association with IBD suggests the involvement of immunological mechanisms related to the intestinal mucosa. Persistent hematuria, proteinuria, or an unexplained decline in glomerular filtration rate should prompt a targeted nephrological evaluation.

Tubulointerstitial nephritis represents another particularly relevant abnormality. It may be associated with intestinal inflammatory activity or exposure to medications, especially 5-aminosalicylic acid derivatives. This dual etiological possibility requires careful assessment before attributing renal deterioration exclusively to the underlying disease or its treatment.[2]

Nephrotoxicity associated with the treatment of inflammatory bowel disease

Pharmacological treatment of IBD has undergone substantial advances over recent decades. However, some medications may cause renal adverse effects that require specific monitoring.

Aminosalicylates, particularly mesalazine, have been associated with acute and chronic interstitial nephritis. Although this complication is uncommon, its clinical relevance lies in its potentially insidious development without obvious clinical manifestations. The absence of urinary symptoms does not exclude kidney injury; therefore, periodic measurement of serum creatinine and estimation of glomerular filtration rate are essential.

Calcineurin inhibitors, such as cyclosporine and tacrolimus, used in selected clinical circumstances, may cause renal vasoconstriction and acute or chronic nephrotoxicity. Similarly, uncommon renal abnormalities have been reported in association with certain biological therapies, including glomerulopathies and immune-mediated reactions.

Importantly, the development of renal insufficiency during IBD treatment does not necessarily establish a causal relationship with pharmacological therapy. Clinical evaluation should consider intestinal disease activity, hydration status, infection, renal history, and concurrent exposure to other nephrotoxic agents.

The systematic review by van Hoeve and Hoffman highlights that kidney injury in these patients results from complex interactions among disease-related manifestations, metabolic complications, and treatment-related adverse effects. This observation reinforces the importance of individualizing therapeutic decisions and avoiding the unjustified discontinuation of effective medications without appropriate clinical assessment.[3]

Chronic kidney disease and prognosis

Progression to CKD represents one of the most concerning consequences of renal involvement associated with IBD. A systematic review has suggested that approximately 5 to 15% of adults with IBD may develop CKD during the course of their disease, although these estimates vary substantially across studies and populations.[3]

Persistent systemic inflammation, recurrent episodes of AKI, unrecognized interstitial nephritis, complicated nephrolithiasis, and glomerulopathies may contribute to progressive nephron loss. In less frequent cases, secondary AA amyloidosis associated with sustained chronic inflammation may cause significant proteinuria, nephrotic syndrome, and progressive deterioration of kidney function.

The importance of histopathological diagnosis should be emphasized in patients presenting with significant proteinuria, active urinary sediment, or progressive loss of kidney function without an evident cause. Kidney biopsy, when indicated, allows differentiation among distinct patterns of renal injury and guides disease-specific treatment.

Studies involving patients with IBD and biopsy-confirmed kidney disease have demonstrated that the extent of chronic histological damage and kidney function at the time of diagnosis have important prognostic implications. These findings emphasize that timely clinical recognition may be decisive in preserving long-term renal function.[4]

Nephrological surveillance and opportunities for prevention

One of the principal challenges in clinical practice is establishing renal surveillance strategies that enable early identification of functional and structural abnormalities in patients with IBD.

Initial evaluation should include serum creatinine, estimated glomerular filtration rate, urinalysis, and assessment of albuminuria or proteinuria when appropriate. In patients with persistent diarrhea, intestinal resections, a history of nephrolithiasis, arterial hypertension, diabetes mellitus, or exposure to potentially nephrotoxic medications, intensified surveillance is reasonable.

The 2024 Kidney Disease: Improving Global Outcomes (KDIGO) international guidelines emphasize the importance of integrating glomerular filtration rate and albuminuria into the identification, classification, and prognostic assessment of CKD. These principles are applicable to patients with IBD, although no universally validated interval for renal monitoring has been established specifically for this population.[5]

From a preventive perspective, maintaining adequate hydration, promptly correcting electrolyte disturbances, preventing recurrent episodes of AKI, and avoiding unnecessary exposure to nonsteroidal anti-inflammatory drugs and other nephrotoxic agents are essential. In patients with enteric hyperoxaluria or recurrent nephrolithiasis, urinary metabolic evaluation and individualized dietary interventions may help reduce recurrence.

Early nephrology consultation should be considered in cases of persistent deterioration of kidney function, clinically significant proteinuria, hematuria suggestive of glomerular disease, or suspected interstitial nephritis. Interdisciplinary management allows clinicians to balance the need for effective control of intestinal inflammation with the preservation of renal function.

CONCLUSIONS

Renal insufficiency in patients with inflammatory bowel disease is a clinically relevant complication that deserves greater attention in contemporary medical practice. Its multifactorial origin, diverse manifestations, and potentially silent progression represent major obstacles to timely diagnosis.

Available evidence indicates that renal involvement should not be regarded solely as a consequence of dehydration or drug toxicity. Immunological abnormalities, systemic inflammation, and interactions between the intestine and kidneys contribute to a spectrum of disorders that may permanently compromise renal function.

A comprehensive approach to IBD should be promoted, in which nephrological assessment becomes an integral component of clinical surveillance, particularly in patients with active disease, exposure to potentially nephrotoxic treatments, or additional risk factors for kidney disease.

Preservation of kidney function should be an explicit objective in the comprehensive management of inflammatory bowel disease. Early recognition of kidney injury, identification of its underlying etiology, and close collaboration between gastroenterology and nephrology provide meaningful opportunities to prevent complications, reduce progression to CKD, and improve long-term patient outcomes.

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