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Patterns of Renal Manifestations in Patients with Inflammatory Bowel Disease: A Comprehensive Review

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ABSTRACT

Background: Renal involvement represents an important but often under-recognized extraintestinal manifestation of inflammatory bowel disease (IBD), encompassing both Crohn's disease (CD) and ulcerative colitis (UC). The spectrum of renal disorders ranges from glomerular disease and tubulointerstitial nephritis to nephrolithiasis and amyloidosis. These conditions can occur as a direct consequence of systemic inflammation, immune-mediated mechanisms, metabolic alterations, or secondary to therapeutic agents. Despite their clinical relevance, renal manifestations in IBD remain underexplored, with variable prevalence reported across different populations. The aim of this review is to evaluate the existing evidence regarding the patterns of renal involvement in IBD patients, highlighting epidemiological trends, underlying mechanisms, and clinical subtypes. Special emphasis is placed on glomerular diseases, tubulointerstitial injury, drug-related nephrotoxicity, renal calculi, and amyloidosis. Furthermore, we explore the diagnostic challenges posed by overlapping renal and intestinal symptoms and review strategies for timely detection and management. By integrating findings from epidemiological and clinical studies, this review identifies key research gaps, particularly the lack of large-scale prospective studies assessing renal outcomes in IBD cohorts. Recognition of renal manifestations is essential not only for reducing morbidity but also for optimizing therapeutic decision-making in IBD patients. Ultimately, this review underscores the need for a multidisciplinary approach between gastroenterologists and nephrologists to improve patient outcomes.

Keywords*: Renal Manifestations , Inflammatory Bowel Disease*

Introduction

Inflammatory bowel disease (IBD), which encompasses Crohn's disease (CD) and ulcerative colitis (UC), is a chronic, relapsing-remitting disorder of the gastrointestinal tract with systemic implications. Over the past decades, its burden has increased worldwide, affecting millions of individuals across diverse populations [1]. While gastrointestinal involvement is the hallmark of IBD, extraintestinal

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manifestations (EIMs) are observed in up to 40% of patients, impacting multiple organ systems [2]. Among these, renal involvement remains underappreciated despite its potential to significantly influence prognosis and quality of life.

The renal manifestations of IBD exhibit remarkable heterogeneity, ranging from glomerulonephritis and tubulointerstitial nephritis to nephrolithiasis and amyloidosis. Some are driven by systemic inflammation and immune dysregulation, whereas others are consequences of malabsorption, metabolic derangements, or adverse drug reactions [3]. Importantly, renal manifestations may precede or parallel intestinal disease activity, complicating diagnosis and management.

Despite growing awareness of EIMs, systematic research into renal involvement remains scarce compared to musculoskeletal or dermatological manifestations of IBD. Consequently, clinicians may fail to detect renal complications early, leading to chronic kidney disease or irreversible renal impairment [4]. The aim of this review is therefore to synthesize current knowledge on the epidemiology, pathophysiology, and clinical spectrum of renal manifestations in IBD patients. We focus particularly on the patterns of renal affection, exploring their frequency, clinical presentation, and underlying mechanisms.

By delineating these patterns, this review aims to bridge the gap between gastroenterology and nephrology, encouraging timely recognition and integrated management of renal disease in IBD. Ultimately, this may improve both intestinal and systemic outcomes in affected patients [5].

A. Epidemiology of Renal Involvement in IBD

Renal manifestations in IBD are relatively uncommon but clinically significant. Their reported prevalence varies widely across studies, reflecting differences in population characteristics, study design, and diagnostic criteria. Estimates suggest that renal involvement occurs in 4–23% of IBD patients [6]. The spectrum includes both glomerular and nonglomerular pathologies, with nephrolithiasis being one of the most frequently reported complications.

Several population-based studies have documented a higher risk of chronic kidney disease (CKD) among patients with IBD compared to the general population. A large cohort study from Denmark demonstrated that IBD patients had a 2-fold increased risk of CKD, independent of traditional risk factors such as hypertension and diabetes [7]. Another multicenter study highlighted that renal disease may develop at any stage of IBD, although it is more commonly reported in longstanding disease [8].

The type of IBD may also influence renal involvement. Crohn’s disease appears more strongly associated with nephrolithiasis and metabolic complications due to extensive small bowel involvement, malabsorption, and surgical resections [9]. Conversely, ulcerative colitis has been more frequently linked to interstitial nephritis, often related to drug exposure such as 5-aminosalicylates

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[10]. These differences underscore the importance of distinguishing between UC and CD when evaluating renal complications.

Epidemiological trends further suggest geographic and ethnic variability. For example, studies from Western populations report higher rates of nephrolithiasis in CD, whereas reports from Asia emphasize glomerular disease, particularly IgA nephropathy [11]. Such variability may reflect genetic, environmental, and dietary factors, as well as differential therapeutic approaches across regions.

Overall, while renal involvement remains a minority manifestation in IBD, its clinical impact is substantial. It increases the risk of hospitalization, reduces quality of life, and may contribute to long-term renal impairment. Hence, understanding the epidemiological patterns is crucial for targeted screening and early intervention in at-risk patients [12].

B. Pathophysiological Links between IBD and Renal Manifestations

The pathophysiological relationship between IBD and renal involvement is multifactorial, reflecting the interplay of immune dysregulation, systemic inflammation, metabolic derangements, and therapeutic exposures. Both Crohn's disease and ulcerative colitis are characterized by chronic mucosal inflammation driven by genetic susceptibility, altered microbiota, and environmental triggers. These mechanisms extend beyond the gut, leading to systemic immune activation that may contribute to renal injury [13]. Circulating immune complexes and aberrant T-cell responses have been implicated in glomerular damage, while proinflammatory cytokines such as TNF-α and IL-6 mediate tubulointerstitial inflammation and fibrosis [14].

Immune complex deposition is one of the leading mechanisms underlying glomerular involvement in IBD. Elevated mucosal immune activity can lead to the overproduction of IgA, which deposits in the glomeruli and triggers mesangial proliferation, resulting in IgA nephropathy—the most commonly reported glomerulonephritis in IBD patients [15]. Similarly, autoantibodies and complement activation may contribute to other glomerular pathologies, including membranous and minimal change disease. These immunological phenomena highlight the systemic nature of IBD beyond the gastrointestinal tract [16].

Another major pathway involves metabolic and absorptive disturbances related to intestinal disease activity and surgery. Patients with Crohn's disease often undergo extensive small bowel resections, which predispose them to malabsorption of oxalate, magnesium, and citrate, thereby creating a milieu conducive to nephrolithiasis [17]. Enteric hyperoxaluria is a particularly important contributor, as increased intestinal permeability allows excessive oxalate absorption, ultimately promoting calcium oxalate stone formation [18]. These metabolic alterations represent indirect yet clinically significant links between bowel and renal pathology.

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Medications used in the management of IBD also play a central role in renal involvement. 5-aminosalicylates (5-ASA), commonly prescribed in ulcerative colitis, are well recognized for their association with interstitial nephritis, often presenting as insidious renal impairment [19]. Biologic agents such as anti-TNF therapies have also been implicated in rare cases of glomerular injury, likely due to immune-mediated mechanisms [20]. Thus, both disease-related and treatment-related factors must be considered when evaluating renal complications in IBD patients.

Overall, the pathogenesis of renal involvement in IBD is complex and multifaceted, involving overlapping immune, inflammatory, metabolic, and iatrogenic mechanisms. This underscores the importance of integrated care, with gastroenterologists and nephrologists collaborating to distinguish disease-related renal pathology from drug-induced injury [21].

C. Patterns of Glomerular Diseases in IBD

IgA Nephropathy

IgA nephropathy is the most frequently reported glomerular disorder associated with IBD, particularly Crohn's disease. The underlying mechanism is thought to be related to excessive mucosal immune activation, leading to increased IgA production and mesangial deposition in the kidneys [22]. Clinically, patients present with microscopic hematuria, proteinuria, or rarely nephrotic syndrome. Histologically, mesangial proliferation and immune complex deposition are typical findings. Importantly, disease activity in IBD often parallels the renal course, suggesting shared inflammatory drivers [23]. The presence of IgA nephropathy in IBD patients not only reflects systemic immune dysregulation but also underscores the need for renal surveillance in those with persistent urinary abnormalities.

Minimal Change Disease

Minimal change disease (MCD), although rare in IBD, has been documented in both adults and children with Crohn's disease and ulcerative colitis. The pathogenesis remains unclear, but immune-mediated mechanisms, including T-cell dysfunction and cytokine release, are suspected contributors [24]. Some reports describe an association between the onset of nephrotic syndrome and anti-TNF therapy, raising the possibility of treatment-induced MCD [25]. Clinically, MCD manifests with severe proteinuria, hypoalbuminemia, and edema. Fortunately, most patients respond well to corticosteroids, though relapse can occur, especially if intestinal inflammation is uncontrolled. Recognition of this entity is crucial since untreated proteinuria contributes significantly to morbidity in IBD patients.

Membranous Nephropathy

Membranous nephropathy is another glomerular lesion occasionally reported in IBD patients. Its etiology may involve circulating immune complexes, drug-induced injury, or idiopathic mechanisms unmasked by systemic inflammation [26]. The disease typically presents with nephrotic-range

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proteinuria and can progress to chronic kidney disease if not addressed. Case reports have implicated both 5-ASA therapy and biologics as potential triggers [27]. Although less common than IgA nephropathy, membranous nephropathy emphasizes the importance of considering iatrogenic as well as autoimmune mechanisms when evaluating renal dysfunction in IBD.

Other Glomerular Disorders

In addition to these common entities, other glomerulopathies such as focal segmental glomerulosclerosis (FSGS), membranoproliferative glomerulonephritis (MPGN), and pauci-immune crescentic glomerulonephritis have been described in the context of IBD [28]. These are far less frequent but clinically significant due to their association with progressive renal impairment. For example, vasculitis-related glomerulonephritis has been linked with ulcerative colitis in rare cases, highlighting the systemic autoimmune overlap [29]. Because such conditions are rare, evidence is largely derived from case reports and small case series, underscoring the need for prospective cohort studies to clarify their prevalence and outcomes.

Clinical Implications

The spectrum of glomerular disease in IBD is wide and variable, but certain patterns can be distinguished. IgA nephropathy is the dominant entity, while minimal change and membranous nephropathy represent smaller but important subgroups. Less common forms, including FSGS and vasculitic glomerulonephritis, highlight the systemic autoimmune nature of IBD. Importantly, disease recognition requires a high index of suspicion, as renal manifestations are often subtle and overshadowed by gastrointestinal symptoms. Regular urinalysis and renal function monitoring are essential components of comprehensive IBD management [30].

D. Tubulointerstitial Involvement

Drug-Induced Nephrotoxicity: 5-Aminosalicylates

The most common cause of tubulointerstitial nephritis in IBD is drug-induced toxicity, particularly related to 5-aminosalicylates (5-ASA), such as mesalamine, sulfasalazine, and olsalazine. Although generally well tolerated, these drugs can cause idiosyncratic, immune-mediated interstitial nephritis, typically presenting as slowly progressive renal insufficiency [31]. Importantly, nephrotoxicity can occur even after years of treatment and is often reversible if detected early and the offending agent is withdrawn [32]. Routine monitoring of renal function, including serum creatinine and urinalysis, is therefore strongly recommended for patients on long-term 5-ASA therapy [33].

Biologics and Immunosuppressive Agents

Beyond 5-ASA, biologics and immunosuppressants also contribute to tubulointerstitial damage. Anti-TNF agents such as infliximab and adalimumab have been associated with acute interstitial nephritis and, in rare cases, glomerulonephritis [34]. Azathioprine and cyclosporine, used in refractory disease,

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may cause dose-dependent nephrotoxicity through direct tubular injury or vasoconstrictive effects [35]. Distinguishing drug-induced injury from disease-related renal involvement is challenging, highlighting the need for close pharmacovigilance and timely kidney biopsy in selected cases [36].

Idiopathic and Immune-Mediated Interstitial Nephritis

Not all cases of interstitial nephritis are drug related. Some appear to be immune-mediated, reflecting systemic inflammation in IBD itself. Reports describe idiopathic interstitial nephritis with lymphocytic infiltration, granulomas, and fibrosis in patients without recent nephrotoxic drug exposure [37]. These cases suggest that tubulointerstitial nephritis may also be part of the broader autoimmune spectrum of IBD, paralleling its systemic manifestations in other organs. Corticosteroids are often effective in such scenarios, although chronic injury can lead to permanent impairment [38].

Chronic Tubulointerstitial Damage and CKD

Regardless of etiology, tubulointerstitial involvement carries a significant risk of progression to chronic kidney disease (CKD). Chronic interstitial nephritis is characterized by tubular atrophy, interstitial fibrosis, and impaired renal concentrating ability. Repeated subclinical episodes of nephrotoxicity, combined with systemic inflammation, may gradually erode renal reserve in IBD patients [39]. This long-term risk reinforces the importance of regular renal monitoring, particularly in patients receiving multiple nephrotoxic agents or with long disease duration.

Clinical and Diagnostic Considerations

Clinically, interstitial nephritis often presents with nonspecific features such as mild proteinuria, sterile pyuria, or subtle renal impairment, making early diagnosis difficult. Definitive confirmation usually requires renal biopsy, which reveals interstitial inflammatory infiltrates and tubular injury [40]. Since clinical presentation overlaps with glomerular and metabolic complications, a systematic diagnostic approach—including careful drug history, laboratory monitoring, and imaging when needed—is essential for accurate attribution. Early withdrawal of offending drugs and prompt initiation of corticosteroids can preserve renal function in many cases, emphasizing the need for awareness among treating physicians [41].

E. Renal Calculi and Metabolic Abnormalities

Epidemiology of Nephrolithiasis in IBD

Nephrolithiasis represents one of the most common renal complications in inflammatory bowel disease, with prevalence estimates ranging from 4% to 23%, depending on study population and disease subtype [42]. Patients with Crohn's disease are particularly predisposed, especially those who have undergone small bowel resection or suffer from extensive ileal involvement [43]. In contrast, ulcerative colitis carries a comparatively lower risk, likely due to limited small bowel pathology. Stone

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formation not only contributes to morbidity but may also signal underlying metabolic disturbances requiring targeted intervention.

Enteric Hyperoxaluria and Calcium Oxalate Stones

The most important mechanism linking IBD to nephrolithiasis is enteric hyperoxaluria. In Crohn's disease with ileal involvement or resection, bile salt malabsorption leads to increased free fatty acids in the gut lumen. These fatty acids bind intraluminal calcium, reducing its availability to complex with oxalate. The resulting increase in free oxalate absorption in the colon predisposes patients to calcium oxalate stone formation [44]. This mechanism explains the high incidence of oxalate nephrolithiasis in Crohn's disease and underscores the importance of dietary counseling and metabolic screening in at-risk individuals [45].

Hypocitraturia and Metabolic Contributions

Beyond hyperoxaluria, hypocitraturia and hypomagnesuria also play significant roles in stone pathogenesis. Chronic diarrhea and metabolic acidosis, common in IBD, reduce urinary citrate excretion, thereby diminishing citrate's natural inhibitory effect on calcium crystallization [46]. Similarly, hypomagnesuria decreases the binding of oxalate, further promoting stone formation. Together, these metabolic abnormalities synergize to elevate the risk of recurrent nephrolithiasis, especially in patients with longstanding or surgically altered Crohn's disease [47].

Uric Acid Stones in IBD

Uric acid calculi, while less frequent than calcium oxalate stones, are also observed in IBD, particularly in patients with chronic diarrhea and acidic urine [48]. Chronic bicarbonate loss via the gastrointestinal tract contributes to persistent acidification of urine, creating an optimal environment for uric acid precipitation. Additionally, dehydration due to diarrhea exacerbates stone risk by reducing urinary volume and concentrating solutes. Awareness of this pattern is essential, as uric acid stones may be preventable with alkalinization therapy and adequate hydration strategies [49].

Clinical Implications and Prevention

The development of nephrolithiasis in IBD patients is not only a source of acute morbidity but also a risk factor for long-term renal impairment. Recurrent stones may cause obstructive uropathy, chronic infections, and loss of renal function over time. Preventive strategies are therefore critical, including adequate hydration, dietary modification, calcium supplementation to bind oxalate in the gut, and in selected cases, pharmacological interventions such as potassium citrate [50]. Given the high recurrence rates, clinicians should maintain vigilance for nephrolithiasis in IBD patients, particularly those with Crohn's disease and a history of bowel resection.

F. Amyloidosis in IBD

Epidemiology and Clinical Significance

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Secondary (AA) amyloidosis is a rare but severe renal complication of inflammatory bowel disease, particularly Crohn's disease. The condition arises due to chronic systemic inflammation, which promotes persistent elevation of serum amyloid A (SAA), an acute-phase reactant that deposits as insoluble fibrils in the kidneys and other organs [51]. The reported prevalence of amyloidosis in IBD ranges from 0.5% to 3%, with most cases occurring in patients with longstanding, poorly controlled disease [52]. Although less frequent than nephrolithiasis or drug-induced nephritis, amyloidosis carries a grave prognosis due to its association with progressive renal dysfunction.

Pathogenesis of Amyloidosis in IBD

The pathogenesis of renal amyloidosis in IBD reflects the interplay between chronic intestinal inflammation and systemic immune activation. Proinflammatory cytokines such as IL-1, IL-6, and TNF-α stimulate hepatic synthesis of SAA, which accumulates in tissues when overproduced and incompletely degraded [53]. In IBD, repeated disease flares and inadequate control of inflammation accelerate this process, leading to amyloid deposition in glomeruli, blood vessels, and interstitium. The kidney is the most commonly affected organ, explaining why renal manifestations dominate the clinical picture [54].

Clinical Presentation and Outcomes

Renal amyloidosis typically manifests as proteinuria, often in the nephrotic range, accompanied by progressive decline in glomerular filtration rate. Patients may also present with edema, hypertension, and, in advanced cases, end-stage kidney disease [55]. The prognosis is closely tied to the control of intestinal inflammation; remission of IBD often slows amyloid progression, whereas refractory disease accelerates renal decline. Unfortunately, once significant renal damage occurs, therapeutic options are limited, and many patients progress to dialysis or require kidney transplantation [56].

Management Considerations

The cornerstone of management for renal amyloidosis in IBD is aggressive control of intestinal inflammation. Immunosuppressive and biologic therapies, particularly anti-TNF agents, have been reported to stabilize or improve proteinuria in some patients by reducing SAA production [57]. Supportive nephrological care, including the use of renin-angiotensin-aldosterone system (RAAS) inhibitors, is crucial to mitigate proteinuria and slow renal decline. Kidney transplantation remains an option for patients with end-stage renal disease, although recurrent amyloidosis in the graft remains a risk if IBD remains uncontrolled [58].

G. Impact of Surgical Interventions and Nutrition on Renal Outcomes

Effects of Bowel Resection and Short Bowel Syndrome

Surgical interventions are frequently required in Crohn's disease, with nearly 50% of patients undergoing bowel resection within 10 years of diagnosis [59]. Such resections, especially involving

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the ileum, predispose to profound metabolic alterations that affect renal outcomes. Short bowel syndrome (SBS) following extensive resection results in chronic diarrhea, dehydration, and electrolyte imbalance, all of which can impair renal function [60]. Moreover, altered bile salt metabolism promotes enteric hyperoxaluria, leading to a markedly increased risk of recurrent calcium oxalate nephrolithiasis [61].

Urinary Volume and Dehydration

Patients with ileostomies or high-output stomas are particularly prone to dehydration and chronic hypovolemia. Reduced urinary output not only increases stone risk but may also accelerate the progression of chronic kidney disease (CKD) [62]. Continuous fluid and electrolyte losses, particularly of sodium, bicarbonate, and magnesium, exacerbate metabolic derangements that place additional stress on the kidneys. This makes hydration therapy and meticulous monitoring of electrolyte status critical components of postoperative management in IBD patients [63].

Nutritional Deficiencies and Metabolic Bone Disease

Malnutrition is a well-recognized consequence of IBD and intestinal resections, with direct and indirect implications for renal health. Vitamin D deficiency, common in these patients, exacerbates bone loss and predisposes to secondary hyperparathyroidism, which can worsen calcium metabolism and stone risk [64]. Likewise, deficiencies in magnesium and citrate reduce natural inhibitors of urinary crystallization, further favoring nephrolithiasis [65]. Nutritional counseling and supplementation therefore play a key role in preventing renal complications.

Clinical Implications of Surgery-Nutrition-Renal Axis

Overall, surgical and nutritional factors exert a substantial impact on renal outcomes in IBD patients. Those with short bowel syndrome, recurrent dehydration, and poor nutritional status face a disproportionately higher risk of renal stones, tubulointerstitial injury, and CKD progression [66]. Early identification of high-risk individuals, combined with proactive measures such as hydration protocols, dietary counseling, and supplementation, may significantly reduce the burden of renal disease in this subgroup. A multidisciplinary approach involving gastroenterologists, nephrologists, and dietitians is essential to optimize care.

H. Diagnostic Considerations in IBD Patients with Renal Affection

Importance of Early Screening

Renal manifestations in IBD are frequently subtle and may remain undetected until significant renal impairment develops. Early screening is essential, especially in patients with long-standing disease, those receiving potentially nephrotoxic drugs, or individuals with previous bowel resections [67]. Periodic urinalysis for hematuria and proteinuria, along with serum creatinine and estimated glomerular filtration rate (eGFR), is recommended for all IBD patients, particularly those on 5-

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aminosalicylates or biologic therapies [68]. These simple measures provide the first line of defense against delayed recognition of renal complications.

Laboratory Evaluation

Laboratory investigations should be tailored to the suspected renal pathology. Persistent proteinuria or hematuria warrants quantification of protein excretion and, when relevant, serological evaluation for autoantibodies such as ANCA or anti-GBM antibodies [69]. Metabolic evaluation, including 24-hour urine collection for oxalate, citrate, magnesium, and uric acid, is crucial in patients with nephrolithiasis [70]. Electrolyte monitoring also provides valuable insights, particularly in patients with chronic diarrhea or ileostomy, where hypokalemia, hypomagnesemia, and metabolic acidosis may complicate the clinical picture [71].

Imaging Approaches

Imaging modalities play a complementary role in diagnosis. Renal ultrasound is a safe, non-invasive method useful for detecting nephrolithiasis, hydronephrosis, and chronic structural changes [72]. In selected cases, non-contrast CT scanning offers superior sensitivity for small renal calculi. MRI may be reserved for evaluating complex renal pathology in patients who cannot tolerate radiation exposure. Imaging findings should always be interpreted in the context of clinical and laboratory data to guide further diagnostic steps [73].

Role of Renal Biopsy

Renal biopsy remains the gold standard for diagnosing glomerular and interstitial pathologies in IBD. It is particularly indicated in cases of unexplained proteinuria, hematuria, or rapidly deteriorating renal function [74]. Histopathological analysis not only identifies the specific renal lesion—such as IgA nephropathy, minimal change disease, or interstitial nephritis—but also helps distinguish between disease-related and drug-induced injury. Biopsy findings directly inform management decisions, including the need for immunosuppressive therapy versus drug withdrawal [75].

Multidisciplinary Diagnostic Approach

The diagnosis of renal complications in IBD requires close collaboration between gastroenterologists, nephrologists, and radiologists. Given the overlapping etiologies—autoimmune, metabolic, infectious, and iatrogenic—multidisciplinary care ensures comprehensive evaluation and reduces the risk of misdiagnosis [76]. Incorporating routine renal monitoring into IBD management protocols can facilitate early detection and timely intervention, ultimately improving long-term renal and gastrointestinal outcomes.

I. Management Strategies and Outcomes

Control of Intestinal Inflammation

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The cornerstone of preventing and managing renal manifestations in IBD is optimal control of intestinal inflammation. Persistent systemic inflammation contributes to glomerular disease, interstitial nephritis, and amyloidosis. Effective IBD therapy—using corticosteroids, immunomodulators, and biologics—has been shown to stabilize or improve renal outcomes by reducing systemic immune activation [77]. For example, anti-TNF agents have not only improved gut inflammation but also reported benefits in secondary amyloidosis by reducing serum amyloid A production [78]. Thus, treating the underlying intestinal disease is critical for renal protection.

Drug Withdrawal and Pharmacovigilance

When drug-induced nephrotoxicity is suspected, prompt discontinuation of the offending agent is essential. Withdrawal of 5-ASA in cases of interstitial nephritis, or azathioprine in cases of dose-related tubular toxicity, frequently results in stabilization or improvement of renal function [79]. Corticosteroids may accelerate recovery in immune-mediated interstitial nephritis, although outcomes vary depending on the chronicity of injury. Importantly, clinicians must balance the need for intestinal control with nephrotoxicity risk, often requiring alternative therapies to avoid disease flares [80].

Supportive Nephrological Management

Supportive care plays a central role in the management of renal involvement. Renin-angiotensin-aldosterone system (RAAS) inhibitors are recommended for patients with proteinuric glomerular disease to reduce proteinuria and slow progression to chronic kidney disease [81]. In nephrolithiasis, preventive measures include maintaining high fluid intake, calcium supplementation to bind intestinal oxalate, and potassium citrate to correct hypocitraturia [82]. Patients with advanced renal dysfunction may require renal replacement therapy, and in select cases, kidney transplantation offers long-term survival, though recurrent disease remains a risk [83].

Multidisciplinary Approach and Monitoring

Given the multifactorial nature of renal involvement, a multidisciplinary approach involving gastroenterologists, nephrologists, urologists, and dietitians is essential. Routine monitoring of renal function, urinalysis, and metabolic parameters should be integrated into IBD care pathways [84]. Special vigilance is needed for patients with extensive bowel resections, chronic diarrhea, or exposure to nephrotoxic drugs. Early recognition of abnormalities allows timely intervention, reducing the risk of irreversible kidney damage.

Prognosis and Long-Term Outcomes

The prognosis of renal involvement in IBD varies widely depending on the underlying pathology. Drug-induced interstitial nephritis often improves if recognized early, whereas chronic cases may lead to irreversible CKD. Nephrolithiasis carries a high recurrence rate, particularly in Crohn’s disease, unless preventive strategies are adopted. Amyloidosis, though rare, remains a severe complication

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associated with poor renal and overall survival outcomes [85]. Importantly, most renal complications are modifiable if detected promptly, emphasizing the need for proactive monitoring and integrated management strategies.

J. Conclusion

Renal manifestations in inflammatory bowel disease, though less common than other extraintestinal features, carry significant clinical implications. Their spectrum is broad, ranging from glomerular diseases such as IgA nephropathy to tubulointerstitial nephritis, nephrolithiasis, and secondary amyloidosis. While prevalence rates vary, the burden of renal involvement is underestimated, in part due to nonspecific symptoms and under-recognition in clinical practice. Early diagnosis is critical, as many renal complications are reversible or modifiable if detected promptly.

The pathogenesis of renal involvement in IBD is multifactorial, involving immune dysregulation, systemic inflammation, metabolic abnormalities, surgical and nutritional consequences, and drug-induced nephrotoxicity. These overlapping mechanisms highlight the complexity of disease management and the importance of distinguishing between IBD-related and treatment-related renal pathology. Routine renal monitoring, especially in patients on long-term 5-ASA or biologics, is therefore essential to reduce morbidity.

Ultimately, the optimal management of renal complications in IBD requires a multidisciplinary approach that integrates gastroenterologists, nephrologists, urologists, and dietitians. Advances in IBD therapies offer hope for reducing systemic inflammation and, by extension, renal sequelae. However, more prospective studies are needed to clarify epidemiological trends, validate biomarkers for early detection, and establish evidence-based guidelines for renal monitoring in IBD. By increasing awareness of these patterns and fostering cross-specialty collaboration, clinicians can significantly improve both intestinal and renal outcomes for patients with inflammatory bowel disease.

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