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Review Article

Rheumatological Manifestations in Inflammatory Bowel Disease

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Abstract

Inflammatory Bowel Disease (IBD) includes Crohn's Disease (CD) and Ulcerative Colitis (UC), chronic systemic inflammatory disorders primarily affecting the gastrointestinal tract. However, their impact extends beyond the gut, with extraintestinal manifestations occurring in up to 50% of patients. Among these, rheumatological manifestations are the most prevalent, significantly affecting quality of life. This article aims to provide a comprehensive review of the major articular manifestations associated with IBD, examining the shared immunological pathways, particularly the "gut-joint axis", and the clinical challenges in diagnosis. The presence of rheumatological manifestations emphasizes the systemic nature of IBD and the need for an integrated and multidisciplinary approach. Early recognition and targeted therapy are essential to optimize patient outcomes and prevent long-term functional impairment.

Keywords: Inflammatory Bowel Disease; Crohn's Disease; Ulcerative Colitis; Spondyloarthritis; Gut-Joint Axis

Abbreviations: IBD: Inflammatory bowel disease; CD: Crohn's disease; UC: Ulcerative colitis; SpA: spondylarthritis; TNF-α: Tumor necrosis factor-α; ASAS: Assessment of Spondyloarthritis International Society; MRI: Magnetic resonance imaging; US: Ultrasound

Introduction

Inflammatory bowel disease (IBD), including Crohn's disease (CD) and Ulcerative colitis (UC), is characterized by chronic, relapsing inflammation of the gastrointestinal tract resulting from an inappropriate immune response to environmental triggers in genetically susceptible individuals [1]. Despite their differences, both conditions are increasingly recognized as systemic diseases rather than localized intestinal disorders. In this context, extraintestinal manifestations are common and may precede the onset of intestinal symptoms, occur concurrently, or develop during periods of disease exacerbation or remission, reflecting the systemic nature of the underlying inflammatory process [2]. Articular manifestations, affecting approximately 2-46% of patients with IBD, represent the most frequent extraintestinal manifestations that fall under the family of spondylarthritis (SpA) [3]. The pathogenesis of articular involvement in IBD is driven by common immunological mechanisms that link gut and joint inflammation, collectively referred to as the "gut-joint axis" [4]. Musculoskeletal pain in IBD patients increases the overall disease burden and complicates treatment, as some therapies for spondyloarthritis can worsen intestinal inflammation. In patients with IBD, the initial presentation of spondyloarthritis often involves inflammatory musculoskeletal pain, including lower back pain and large joint arthralgias. These symptoms are usually underestimated, and without recognition of early signs, patients may continue with incorrect therapies, resulting in diagnostic delays and an increased risk of long-term musculoskeletal complications, such as chronic pain and joint dysfunction. Early identification and timely intervention are therefore essential to reduce these risks. Furthermore, articular manifestations in IBD often present

subtly, with symptoms overlapping those of other musculoskeletal conditions, making diagnosis difficult. This diagnostic complexity, combined with persistent pain and impaired mobility, can severely affect patients' quality of life and complicate the treatment of both IBD and its rheumatological complications.

Discussion

IBD is frequently associated with extraintestinal manifestations, among which rheumatological involvement is the most common. Musculoskeletal manifestations fall within the spectrum of spondylarthritis and include axial and peripheral arthritis, enthesitis and dactylitis. These manifestations may precede, parallel, or follow intestinal disease activity and contribute substantially to patient morbidity.

The concept of the gut–joint axis describes the bidirectional immunological and microbial crosstalk linking intestinal inflammation to extraintestinal musculoskeletal involvement in IBD. Disruption of the intestinal epithelial barrier, together with gut dysbiosis, promotes systemic immune activation and facilitates the translocation of microbial antigens. This process can trigger aberrant T-cell responses and the production of pro-inflammatory cytokines, including tumor necrosis factor-α (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23) [4]. TNF-α, in particular, is a key mediator of mucosal damage in the gut and synovial inflammation in the joints [5,6]. This dysregulated immune response, characterized by the overproduction of cytokines, underscores the interconnected nature of IBD and its associated rheumatological manifestations.

These shared inflammatory pathways contribute to synovial inflammation and enthesitis, explaining the frequent coexistence of spondylarthritis in patients with IBD. Genetic susceptibility further modulates this interaction, with HLA-B27 demonstrating the strongest clinical association, particularly in patients with axial involvement. In the overall IBD population, HLA-B27 carriage has been associated with a 10–40% increased risk of developing spondylarthritis. However, despite this strong association, the diagnostic utility of HLA-B27 in IBD remains limited. HLA-B27 is relatively prevalent in the general population, and only approximately 5% of HLA-B27– positive individuals develop clinically overt SpA [7]. Consequently, while HLA-B27 positivity may indicate increased susceptibility and aid in disease stratification, it should not be used in isolation for diagnosis and must be interpreted in conjunction with clinical assessment and imaging findings. The management of patients with concomitant IBD and rheumatologic manifestations requires close collaboration between the gastroenterologist and the rheumatologist, with the shared goal of achieving dual remission. Treatment strategies should control articular inflammation while preserving intestinal mucosal healing, ensuring that therapy for one condition does not compromise outcomes in the other.

Arthritis

Arthropathies associated with IBD are recognized as part of the spondyloarthritis spectrum, a group of inflammatory diseases primarily affecting the axial and peripheral joints. According to the Assessment of Spondyloarthritis International Society (ASAS) classification, SpA is divided into axial and peripheral forms, depending on the predominant pattern of symptoms [8]. In axial SpA, which primarily involves the sacroiliac joints and spine, the diagnosis hinges on the combination of clinical features of inflammatory low back pain and radiological evidence of sacroiliitis. Magnetic resonance imaging (MRI) is the preferred imaging modality to detect early sacroiliitis, particularly in patients under 40 years of age who present with inflammatory back pain lasting more than 3 months [9]. MRI is highly sensitive in detecting early inflammatory changes in the entheses, particularly in the spine and pelvis, often identifying subtle alterations before they are visible on X- ray. In contrast, X-rays may reveal enthesophytes or bone erosions, typically seen in the chronic stages of IBD-related SpA. Peripheral arthritis in IBD typically affects the large joints of the lower limbs, such as the knees, ankles, and occasionally, the wrists and elbows. These peripheral arthropathies are non-erosive and non-deforming, and they can be broadly categorized into Type 1 (pauciarticular) and Type 2 (polyarticular) arthritis [10].

Type 1 arthritis is a non-erosive, oligoarticular arthritis predominantly affecting large joints of the lower limbs, generally involving less than five joints (often knees and ankles) and it is usually asymmetric. There is a strong association with active IBD and symptoms tend to improve with control of the intestinal disease [10]. In contrast, Type 2 arthritis is a symmetric, polyarticular disease usually affecting five or more joints, often small joints of the hands and wrists symmetrically. This type of arthritis is independent of IBD activity, persisting even during intestinal remission. Joint deformity is absent, and rheumatoid factor (RF) is typically negative, distinguishing it from rheumatoid arthritis [10]. The diagnosis of peripheral arthritis in IBD is primarily clinical, based on characteristic features of joint inflammation, such as pain, swelling and morning stiffness. Imaging, including X-rays and MRI, helps confirm the absence of joint erosion and deformity, thereby distinguishing IBD-related arthritis from other inflammatory and degenerative conditions; in particular, MRI can identify synovitis or joint effusions, which are indicative of active inflammation. The prognosis for patients with IBD-associated arthropathy varies significantly based on the clinical phenotype. Type I peripheral arthritis is typically non-erosive and self-limiting, often resolving with the treatment of the underlying bowel inflammation. In contrast, Type II peripheral arthritis and axial SpA can follow a chronic, progressive course. If left untreated, axial involvement may lead to permanent structural damage, such as syndesmophyte formation and spinal ankylosis, severely impacting the patient's quality of life [11].

Enthesitis

Enthesitis is defined as inflammation at the sites of tendon, ligament, or fascia insertion into bone. In IBD, the most frequently affected areas include the Achilles tendon, plantar fascia, femoral trochanter and elbows. These sites are vulnerable due to mechanical stress and the inflammatory processes associated with IBD [12]. Clinically, enthesitis in IBD presents as pain, swelling

and tenderness at the affected enthesis, which worsens with movement or pressure. If left untreated, chronic inflammation can lead to bone erosions, enthesophytes (bone spurs), and fibrosis or calcification of the tendon or ligament attachments, causing stiffness and functional limitations [12]. Enthesitis may be clinically underrecognized, particularly when mild, but imaging modalities such as ultrasound (US) and MRI have improved its detection.

US can be very useful in revealing asymptomatic structural and inflammatory abnormalities of the entheses, especially in an early phase. With US the enthesis appears thickened and hypoechoic and power Doppler detects ethereal neo angiogenesis. MRI allows direct visualization of peri-ethereal inflammation and associated bone marrow enema, changes that reflect early inflammation. Fat-suppressed MRI sequences, including short tau inversion recovery (STIR), and contrast-enhanced T1-weighted imaging represent the most sensitive techniques for the detection of active enthesitis, enabling simultaneous evaluation of soft-tissue inflammation and adjacent subchondral bone involvement [13].

Dactylitis

Dactylitis, characterized by diffuse swelling of an entire digit, it is observed in approximately 2–4% of patients with IBD [14]. It reflects a combination of synovitis, tenosynovitis and enthesitis, and is generally associated with more active systemic inflammation. Although uncommon, its recognition is clinically relevant as it may indicate more severe musculoskeletal involvement and this manifestation is often correlated with IBD disease activity, with inflammation in the entheses exacerbating during periods of active CD or UC. Notably, CD patients exhibit a significantly higher propensity for developing dactylitis compared to those with UC, and the risk is closely linked to elevated intestinal inflammation. Imaging plays a key role in identifying dactylitis. US and MRI are the most commonly used techniques for evaluating this condition. The characteristic findings of dactylitis on imaging include flexor tendon tenosynovitis, joint synovitis, soft tissue thickening and extensor tendon thickening. MRI is particularly sensitive, revealing bone edema, which is not detectable on US. Furthermore, in US Doppler mode, hyperemia or tissue inflammation may be observed, reflecting increased blood flow to the affected areas [15].

Conclusion

Rheumatological manifestations represent the most frequent extra-intestinal complication of IBD, reflecting a complex interplay within the gut-joint axis. The underlying pathophysiology is driven by a dysregulated immune response involving shared cytokine pathways and a unique trafficking of immune cells from the inflamed gut mucosa to the synovial tissue. The complexity of these patients necessitates a shift from fragmented care to a multidisciplinary and integrated gastroenterologist and rheumatologist approach. Optimal management depends on the careful selection of therapies that address both systems simultaneously while avoiding agents that might benefit one organ but harm the other.

Acknowledgment

None.

Conflict of Interest

No conflict of interest.

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