

Spondyloarthritis and the Gut Characteristics

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Editorial

The associations that link Spondyloarthritis (SpA) to the gut have been known for many decades and are nearly genealogical, since the rheumatic manifestations associated with Chronic Inflammatory Bowel Disease (CIBD) are among the spondyloarthritides. Evidence supporting these links has accumulated over time, as a result of both advances in test methods and new insights into immunopathological mechanisms. This progress opens up practical avenues for patient management and the development of potential treatment options. The backdrop: classical links between spondyloarthritis and the gut. Gut involvement in spondyloarthritis CIBD is diagnosed in about 5% to 10% of all patients with Ankylosing Spondylitis (AS). CIBD accounts for 19% of all extra articular manifestations of spondyloarthritis and is among the criteria used in the Amor classification system. In the 1980s, studies involving routine colonoscopy showed microscopic inflammation without clinical bowel symptoms in 50% of patients with SpA. Two patterns of inflammation were documented: acute inflammation (of the type associated with infections), chiefly seen in patients with transient arthritis; and chronic inflammation (such as in Crohn's disease), usually associated with persistent arthritis. Among patients with SpA and histological evidence of inflammation, 6.5% develop CIBD within 5 years; in the event of chronic inflammation, the proportion is 20% and the risk of progression to AS is higher. Other classical links include the frequent correlation between the degree of joint inflammation (chiefly in peripheral joints) and gut inflammation, the increased intestinal permeability documented in AS, and the involvement of mucous-membrane humoral immunity (IgA) in spondyloarthritis.

Experimental studies in the transgenic HLA-B27 rat model have demonstrated not only rheumatic manifestations, but also chronic colonic inflammation.

Animals raised in a sterile development remain free of joint and gut inflammation, which develops after the introduction of normal gut bacteria. Rheumatic manifestations in CIBD have a prevalence of about 50/100,000. Joint involvement has been reported in 15% to 33% of cases and is associated with chronic involvement, the extent of the enteropathy, and younger age. Several patterns of peripheral joint involvement have been reported in patients with CIBD: pauci or oligoarticular peripheral arthritis (Type 1 in the Orchard classification, 4%–8%) involves the large joints and lower limbs, runs a self-limiting course, and correlates with the course of the CIBD; whereas polyarticular disease (Type 2) is less common (1–3%), characterized by a symmetric topographic distribution predominating in the small joints and upper limbs, and runs a chronic course uncorrelated to that of the CIBD. Inflammatory enthesopathy (5%–10%) and dactylitis (2%–4%) have been reported and may occur as components of spondyloarthritis. Arthralgia without local objective abnormalities has been reported in 8% to 30% of cases. Axial forms classically include isolated sacroiliitis detected by imaging studies (2%–30%), inflammatory back pain (5%–30%). Incorporation within criteria sets CIBD in the patient or family is among the criteria used in the new SpA classification systems. The CIBD must have been diagnosed by a physician. The rheumatic manifestations of CIBD are clearly among these criteria. Associations linking gut inflammation to spondyloarthritis the research group based in Ghent, has continued to work on gut inflammation in SpA. They confirmed that 46% of their cohort of patients with SpA had histological evidence of gut inflammation. Variables independently associated with histological gut inflammation were male gender high disease activity as evaluated using the BASDAI, decreased mobility as assessed using the BASMI, and younger age. In contrast, no associations were documented with the HLA-B27 antigen, arthritis, enthesitis, uveitis, psoriasis, use of nonsteroidal anti-inflammatory drugs, or a family history of SpA.

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