

A case of localized cervical bony ankylosis due to ulcerative colitis

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Localized neck pain is an uncommon presenting complaint of ulcerative colitis (1).

A 43-year-old man presented with a 10-year history of cervical pain. He had occasional diarrhea without bloody stool or abdominal pain for 20 years. As this abdominal symptom was mild, he did not seek medical attention. There was no family history of spondyloarthritis, psoriasis, or inflammatory bowel diseases. On examination, his neck was ankylotic and could not be moved in any direction. Peripheral joint examination revealed no enthesitis or synovitis. The modified Schober test result was negative. Skin and nail examinations indicated no evidence of psoriasis. Blood test results revealed elevated inflammatory marker levels (C-reactive protein level, 26.0 mg/L; erythrocyte sedimentation rate, 65 mm/h). Test results for the rheumatoid factor and anti-citrullinated protein antibody were negative. The test result for human leukocyte antigen (HLA)-B27 was negative, but the result for HLA-B60, which is associated with spondyloarthritis in the Asian population, was positive (2, 3). Computed tomography of the cervical spine was performed, revealing bony ankylosis of both facet joints (Figure 1). Magnetic resonance imaging revealed no evidence of sacroiliitis or lumbar spondylitis. Underlying inflammatory bowel disease (IBD) was suspected; thus, colonoscopy was performed and showed mucosal edema and erythema of the rectum and sigmoid colon. Biopsy findings indicated lymphoid aggregates with crypt disarray. Accordingly, spondyloarthritis due to ulcerative colitis was diagnosed. After adalimumab treatment, his neck pain and diarrhea resolved, although his neck remained immobile.



Figure 1. Computed tomography of the neck showing fused facet joints from C2 to C7



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Spinal involvement occurs in up to 20% patients with IBD, with the involvement of any facet joint (1, 4). Spinal symptoms may precede intestinal symptoms or develop later; spinal symptoms do not always correlate with intestinal symptoms. Spinal involvement is often silent. On the other hand, silent IBD can be found on biopsy in patients with spondyloarthritis, such as our case (5). Thus, colonoscopy with histological exploration should be performed in a case of spondyloarthritis with unknown origin.

In enteropathic spondyloarthritis, the prevalence of HLA-B27 is only between 53% and 75%; this is lower than that in ankylosing spondylitis (1). HLA-B60 is prevalent in patients with HLA-B27-negative ankylosing spondylitis and undifferentiated spondyloarthritis among the Asian population (2, 3). HLA-B60 may be correlated with enteropathic spondyloarthritis.

There are no specific blood tests for confirming a suspicion of IBD-related arthritis. This report highlights the importance of colonoscopy in addition to careful history and clinical examination of a patient in whom spondyloarthritis with unknown origin was found.

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