

involved, and intracellular crystals can be demonstrated in the synovial fluid. Rheumatoid arthritis and other systemic autoimmune diseases usually manifest with symmetrical polyarthritis of the upper and lower extremities associated with abnormal serologies such as rheumatoid factors, anti-cyclic citrullinated peptide (CCP) antibodies, or antinuclear antibodies. Predominately axial spondyloarthritis must be differentiated from indolent infections of the sacroiliac joints, vertebrae, or intravertebral disks; degenerative disease of the spine and disks (i.e., spondylosis); and diffuse idiopathic skeletal hyperostosis (DISH).

The radiographic features of the spondyloarthritis are highly specific and, in the correct clinical setting, greatly increase the certainty of the diagnosis. Sacroiliitis is usually the earliest radiographic sign of spine disease and results in sclerosis and erosions of the sacroiliac joints with eventual bony fusion (Fig. 78-2A). Many radiographic changes result from chronic spondylitis, including ossification of the annulus fibrosus, calcification of spinal ligaments, bony sclerosis and squaring of vertebral bodies, and ankylosis of apophyseal joints. These changes can lead to vertebral fusion and a bamboo spine appearance (see Fig. 78-2B).

Radiographic findings progress over many years of illness and may not be apparent in early disease. However, during this pre-radiographic period, MRI demonstrates bone inflammation (i.e., osteitis) and erosion at the sacroiliac joints and vertebral bodies, and CT shows bony sclerosis and joint erosions.

Bone erosions, sclerosis, and new bone formation may occur at sites of enthesitis. Erosions at bone-cartilage interface (i.e., subchondral erosions), sclerosis, and bone proliferation are hallmarks of spondyloarthritis involving peripheral joints. In severe cases such as the arthritis mutilans form of psoriatic arthritis, total or subtotal bone resorption (i.e., osteolysis) of a phalange may occur.

TREATMENT

No cure has been found for any form of spondyloarthritis, but effective treatment for many of the manifestations is available. Patient education regarding the disease is essential and allows identification of affected family members and early detection of urgent clinical features such as uveitis. Physical therapy, including a daily stretching program, postural adjustments, and strengthening, helps to maintain proper bony alignment, reduce deformities, and maximize function, particularly for those with axial disease. Selective use of orthopedic surgery may be highly effective in correcting significant spinal deformities or instability.

Nonsteroidal anti-inflammatory drugs (NSAIDs) can provide significant relief of spinal pain and stiffness, and many patients take these drugs continually for years (Centre for Evidence Based Medicine; level 1 evidence). No clear evidence indicates that systemic glucocorticoids benefit patients with spondyloarthritis, and these agents are usually avoided. Intra-articular glucocorticoid injection into the sacroiliac or other involved joints may provide temporary relief. Similarly, the role and efficacy of older immunosuppressive agents in the treatment of axial spondyloarthritis have not been established. In contrast, clinical trials have shown that the peripheral manifestations of spondyloarthritis improve with sulfasalazine and methotrexate (level 2).

TNF- α blockers (i.e., infliximab, etanercept, adalimumab, and golimumab) represent a substantial breakthrough in the treatment of spondyloarthritis. The efficacy of these agents is well established, and they have rapidly become the treatment of choice for patients with axial inflammation who do not satisfactorily or fully respond to NSAIDs and physical therapy (level 1 evidence). TNF- α blockers can significantly reduce pain, improve function, and improve quality of life. They may also prevent or

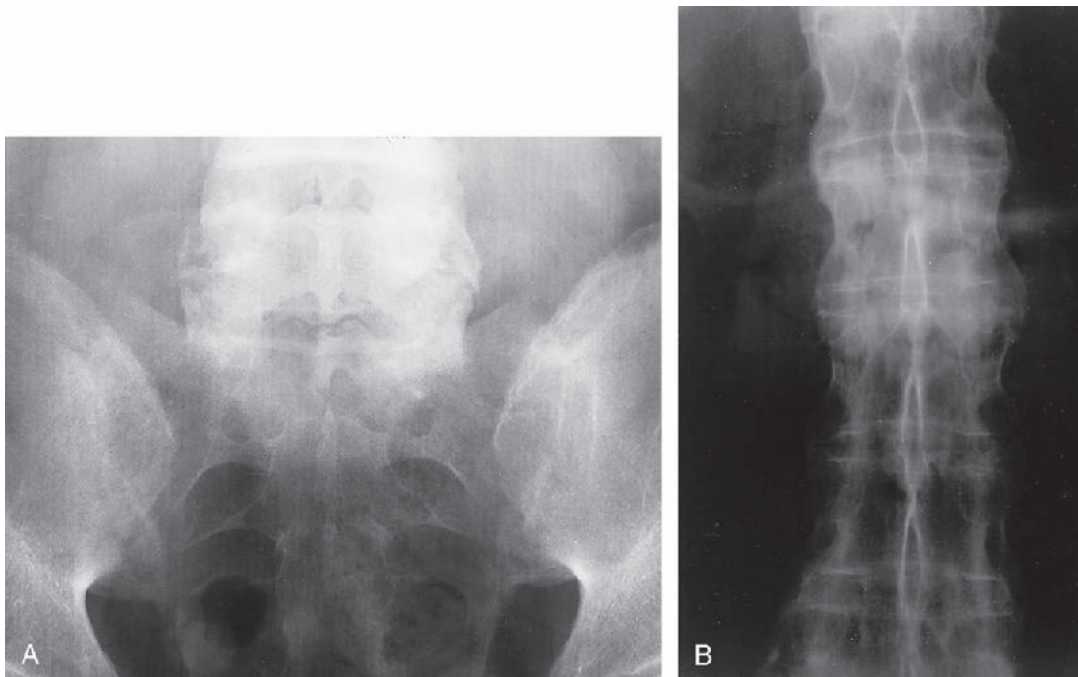


FIGURE 78-2 **A**, Bilaterally symmetrical sacroiliitis in ankylosing spondylitis. **B**, Lumbar spondylitis in ankylosing spondylitis with symmetrical, marginal bridging syndesmophytes and calcification of the spinal ligament. (From Cush JJ, Lipsky PE: The spondyloarthropathies. In Goldman L, Bennett JC, editors: Cecil textbook of medicine, ed 21, Philadelphia, 2000, Saunders, pp 1499–1507.)