

Rheumatoid Arthritis and Ankylosing Spondylitis Occurring Together

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Abstract: We report a case of old aged woman who initially presented with inflammatory pain affecting small and large joints of upper and lower limbs associated with morning stiffness with deformities. Later, she developed inflammatory low back pain with restriction of movement and enthesopathy. So, though the incidence is very rare the co-existence of rheumatoid arthritis and ankylosing spondylitis in this case would be a possibility.

Key words: Ankylosing Spondylitis • Rheumatoid Arthritis

INTRODUCTION

Ankylosing spondylitis (AS) is a chronic inflammatory disease of the axial skeleton manifested by back pain and progressive stiffness of the spine. It characteristically affects young adults with a peak age of onset between 20 and 30 years. AS is the most common of the spondyloarthritis (SpA) that an association with the human leukocyte antigen HLA-B27. AS is a disease of often insidious onset. The plain film radiologic diagnosis is somewhat observer-dependent. In addition, patients with milder disease and involvement of peripheral joints may have been misdiagnosed as having seronegative rheumatoid arthritis [1]. Ankylosing spondylitis (AS) is a systemic rheumatic disease, meaning it affects the entire body. Approximately 90% of AS patients express the HLA-B27 genotype, meaning there is a strong genetic association. However, only 5% of individuals with the HLA-B27 genotype contract the disease [2]. Tumor necrosis factor- α (TNF α) and IL-1 are also implicated in ankylosing spondylitis. Autoantibodies specific for AS have not been identified. Anti-neutrophil cytoplasmic antibodies (ANCA) are associated with AS, but do not correlate with disease severity. In a study of 40 patients with AS, ANCA was an infrequent finding, being present

in only six patients [3]. Rheumatoid arthritis (RA) is a symmetric, inflammatory, peripheral polyarthritis of unknown etiology [4]. It typically leads to deformity through the stretching of tendons and ligaments and destruction of joints through the erosion of cartilage and bone. RA should be suspected in the adult patient who presents with inflammatory polyarthritis [5]. RA and AS are considered to be separate and unrelated diseases. Diagnostic confusion may occasionally arise when signs and symptoms of the one disease overlap those of the other or, rarely, when both entities coexist within the same patient. The chance of this association in the same person is about 1 in 50000 to 1 in 200000. Thus, diagnostic precision can be achieved by an awareness of this potential association. We report one case of coexisting RA and AS.

Case Report: Case -This 70-year-old woman initially presented with peripheral arthritis in the small joints of hands, feet, wrists, knees and ankles with significant morning stiffness from 12 years ago when she was 58. On examination was found to have extensive polyarthritis that affecting proximal interphalangeal joints, metacarpophalangeal joints, wrists, metatarsophalangeal joints, wrists and knees. There were no nodules and there

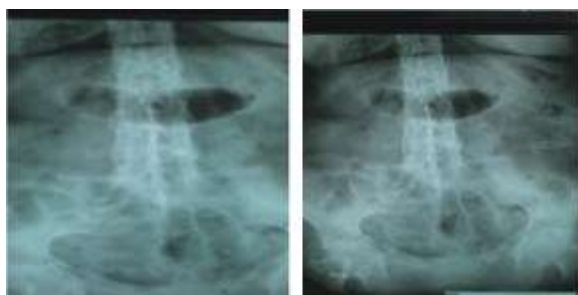


Fig. 1, 2: Radiograph of sacroiliac and lumbar fusion



Fig. 3: Lateral lumbar with complete bridging

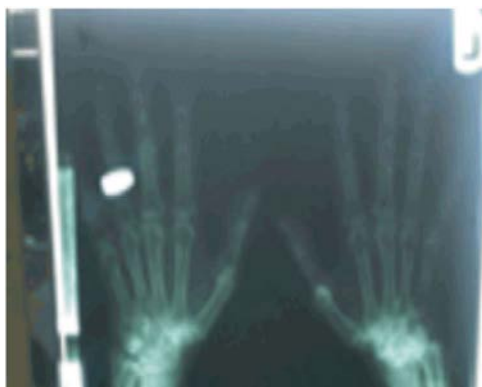


Fig. 4: Radiograph of right and left wrist shows severe erosive arthritis

was no spinal and eye involvement. X-ray examinations showed extensive peripheral erosive arthropathy. The rheumatoid factor and anti CCP was positive. ESR was 85 mm in one hour and CRP was 3+. She was treated with diagnosis of RA on the basis of American college of Rheumatology criteria. She admitted in the hospital because of low back pain. On examination she was mildly anemic with pulse rate: 84/min, Blood pressure: 110/70 mm of Hg and cardiac and lungs examination show no abnormality. On rheumatologic examinations she has grossly limited spinal movement and deformities in spine

including: exaggerated thoracic kyphosis and loss of lumbar lordosis. Tenderness is present over thoracic and lumbar spine; movement is painful and restricted in all directions. Schober's test is positive. (Expansion <2cm). There was ulnar deviation at metacarpophalangeal joints with subluxation, thumb 'Z' deformity with ulnar deviation of digits and extensive hand muscle wasting. She also has polyarthritis affecting proximal interphalangeal joints metacarpophalangeal joints, wrists, metatarsophalangeal joints and knees. On laboratory ESR was 35 mm in one hour, CRP was +3, liver function test was normal, brucella IgG was negative and HLA-B27 antigen was positive. Radiographs showed complete sacroiliac fusion with spinal ankylosis extending to the lumbar and thoracic region (Fig. 1, 2), severe spinal ankylosis and complete bridging (Fig. 3) and extensive erosive changes in hands, wrists (Fig. 4). Chest x-ray and chest CT-scan show no abnormality.

DISCUSSION

RA and AS are differentiated in most cases on the basis of characteristic clinical, laboratory and radiographic manifestations. Briefly the arthritis of AS is characterized by genetic predisposition, earlier onset, male predominance and axial distribution. In order to reduce the delay in the diagnosis of AS as well, an algorithm was developed recently by Rudwaleit *et al.* including inflammatory back pain, HLA-B27 and a family history of spondyloarthritis in order to detect early cases of AS [5]. About 9% of a rheumatoid population would be expected to exhibit HLA-27 histocompatibility antigen, but this does not mean that these patients will all have sacroiliitis any more than such a finding would in the ordinary population [6]. Although HLA-B27 and rheumatoid factor provide sensitive disease markers, they may be present in apparently normal individuals and in other disease states and are therefore nonspecific [7]. Atypical varieties of rheumatoid arthritis and ankylosing spondylitis may create diagnostic confusion; sacroiliitis complicates rheumatoid arthritis in 20% of patients and a peripheral arthropathy is present in 50% of spondylitics [8]. Rheumatoid factor or HLA-B27 may be undetected in otherwise classic examples of these diseases or, as previously mentioned, are identified in individuals who have no other stigmata of either disease. Radiography is especially useful in such atypical cases, since subtle, but distinctive differences exist between the two entities in their appearance and distribution. Thus, a chronic, erosive peripheral arthritis is rarely seen with ankylosing

spondylitis; if present, it occurs in an asymmetrical distribution and is associated with proliferative bony changes [8]. Sacroiliitis is rarely the initial manifestation of rheumatoid arthritis and lumbar involvement eventuating in extensive ankybosis is distinctly unusual [9]. Another possible source of diagnostic error arises when both diseases coexist in the same patient. On the basis of prevalence rates of rheumatoid arthritis and ankylosing spondylitis in the general population, such an association has been conservatively estimated to occur in 1:100, 000 persons [10]. Conlon *et al.*, have shown that it is rare for ankylosis to occur in rheumatoid arthritis [11]. Our case fulfilled the diagnostic criteria of the American Rheumatism Association for both rheumatoid arthritis and ankylosing spondylitis. One explanation of this phenomenon is that rheumatoid arthritis had occurred by chance in patients already suffering from ankylosing spondylitis or, conversely, that ankylosing spondylitis had developed by chance in patients already suffering from rheumatoid arthritis. The paucity of reported cases of coexisting rheumatoid arthritis and ankylosing spondylitis is probably due to (1) lack of awareness of this potential association and (2) a natural tendency to attribute the observed abnormalities to a single disease process. It is likely that in many cases of coexisting rheumatoid arthritis and ankylosing spondylitis, the characteristic differentiating features of one or the other entity are lacking. Either disease is then diagnosed on the basis of the dominant abnormalities. Although coexistence of rheumatoid arthritis and ankylosing spondylitis can be strongly suspected on radiographic findings alone, a firm diagnosis requires logical integration of radiographic, clinical and laboratory data. In contrast with RA, DMARDs are proven to be not effective in AS, except for sulfasalazine which is beneficial in case of peripheral arthritis [12, 13]. NSAIDs play different roles in the management of patients with RA and AS. In RA, NSAIDs are considered only as symptomatic drugs and should not be used as a sole treatment, because they do not prevent structural damage of joints. In AS, NSAIDs are drugs of first choice, not only because of the high symptomatic efficacy, but also because of the possible potential to retard radiographic progression in the spine (and maybe elsewhere) when used continuously. Considering younger age of AS patients, lower prevalence of comorbidities and concomitant medication intake (e.g., glucocorticoids) in comparison to RA in general, patients with AS have probably lower risk of NSAID side effects during short- and long-term therapy. Thus, based on the available data, AS patients

considered for long-term treatment with NSAIDs can and should be informed about the potential risk of such a treatment, which is relatively low [14, 15].

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