Costovertebral and costotransverse joint involvement in rheumatoid arthritis

MICHAEL J. COHEN, JOAN EZEKIEL, AND ROBERT H. PERSSELLIN

From the Division of Rheumatology, Department of Medicine, and the Department of Radiology, The University of Texas Health Science Center at San Antonio, USA

SUMMARY Lesions of the costovertebral (CV) and costotransverse (CT) joints are distinctly unusual in rheumatoid arthritis. The patient presented had dramatic changes in these joints with destruction, ankylosis, and bony overgrowth. This led to a moderate respiratory impairment and a distinctive radiological presentation.

The costovertebral (CV) and costotransverse (CT) joints are diarthrodial joints containing hyaline articular cartilage and lined with synovial membrane and can be involved in inflammatory joint disease (Goldthwait, 1940; Dihlman and Frik, 1968; Zimmer, 1968; Jaffe, 1972). However, lesions of the ribs and their articulations with the vertebral column are rare. The following is a case with unique involvement of the CV and CT joints which has not been previously described.

Case report

The patient, a 28 year old, Mexican-American female, was admitted to Bexar County Hospital because of an exacerbation of her arthritis. She had a 5-year history of symmetrical polyarthritis involving peripheral joints with morning stiffness and high titre rheumatoid factor.

On admission the patient complained of pain and limitation of motion of all major joints and pain along the medial border of the scapulae. She specifically denied dyspnoea on exertion or chest pain, but did admit to prolonged morning stiffness and fatigue.

Physical examination revealed a thin women in moderate distress due to active synovitis. There was mild micrognathia and tender temporomandibular joints. There was full range of motion of the cervical spine, but some tenderness was elicited over the spinous process of C7. There was mild tenderness on palpation near the medial borders of the scapulae. Chest expansion measured at the fourth intercostal space was 1.5 cm. She had diffuse tenderness of multiple peripheral joints. There was extensive atrophy of the muscles of the hands. Erythema and tenderness were detected over the right achilles tendon. There were no rheumatoid nodules.

The haematocrit was 38 with a white blood cell count of 4900/mm³. The erythrocyte sedimentation rate was 50 mm/hr (Westergren); a sensitised sheep cell agglutination titre was positive at 1:3584, and an RA slide latex (Hyland) was 4+ positive (Cheng and Perssellin, 1971). The antinuclear antibody fluorescence test was positive and serum anti-DNA binding was negative (Kredich et al., 1973). HLA typing revealed A2, A28, B16, and BW40. Pulmonary function testing showed a moderate to severe restrictive ventilatory defect. Nerve conduction in the forearms and wrists was normal.

Chest radiology showed bulb-like lesions in the region of the CT joints bilaterally. There was bony overgrowth and ankylosis of the CT joints of ribs 5, 6, 7, and 8 on the left and 5, 6, and 7 on the right. There was also involvement of the adjacent CV joints which was best seen on tomograms. A dorsal lordotic view detailed this involvement of both CV and CT joints (Fig. 1). In addition, a small erosion of the superior margin of the posterior part of the left third rib was noted. The involved ribs were enlarged and the cortices extending from the CV and CT joints were thickened. The involved ribs were 33% wider at a point 5 cm from the midline compared to the uninvolved ribs. There was irregularity of the joint margin of the third and fourth left CT joints. Juxta-articular demineralisation and small erosions were noted at the radial aspect of the left third and
fourth and the right third and fourth proximal interphalangeal joints. Cervical spine, thoracic spine, and lumbosacral spine films showed only mild apophyseal sclerosis at T₁₁–T₁₂, but no changes of ankylosing spondylitis or degenerative joint disease. Rib notching was not found and the sacroiliac joints were normal.

After 7 days in hospital on high dose salicylate therapy, the patient developed chills, nausea, and malaise. SGOT was 855 IU, SGPT 644 IU alkaline phosphatase 136 IU, and bilirubin 0.4 mg/100 ml (6.8 μmol/l). The buffered aspirin was stopped and ibuprofen (Motrin) was substituted. Several days later these symptoms subsided and 1 week later the SGOT was 50 IU, SGPT 161 IU, alkaline phosphatase 130 IU, and bilirubin 0.2 mg/100 ml (3.4 μmol/l). Hepatitis B surface antigen was negative. The articular symptoms improved and she was discharged.

Discussion

Arthritis of the CT and CV joints is recognised as occurring in ankylosing spondylitis (Dihlman and Frik, 1968; Jaffe, 1972) and degenerative joint disease (Krauss, 1956; Zimmer, 1968), but has not been widely reported in rheumatoid arthritis (RA). The changes described in association with ankylosing spondylitis include irregularities of joint margins, periarticular calcification and destruction of the joint. These findings are usually seen in conjunction with the more typical axial skeleton changes of ankylosing spondylitis and have not been reported as occurring alone. Osteophyte formation of these joints has been seen in patients with degenerative joint disease. Only a brief comment of CV joint arthritis was made by Bywaters (1974) in a discussion of discitis in RA.

The CV joint consists of the head of the rib and facets on the upper and lower edges of the adjacent vertebral bodies at the disc margin, except for the CV joints of T₁₁ and T₁₂, which do not override a disc space. The CT joint consists of the tubercle of the rib and a facet on the anterior or superior surface of the transverse process (Fig. 2). Since both are lined by a synovial membrane and are in constant motion, they would be expected to be involved in a generalised inflammatory arthritis. Perhaps rheumatoid involvement is overlooked since this area cannot be easily discerned on a routine chest x-ray and special views are required.

The pathological process in our patient appears to have been an inflammatory synovitis which led to restriction of motion, bony overgrowth and ankylosis of the involved joints (Fig. 2). The bony overgrowth also involved the transverse processes and the ribs, which showed an increase in cortical thickness and total diameter extending laterally from the joint. Clinically the patient denied dypsnoea but did have a restrictive defect on pulmonary function testing.

There are several unusual features of this case. The axial predominance is suggestive of ankylosing spondylitis. However, our patient was female, had normal sacroiliac joints, no other manifestations of ankylosing spondylitis, and was HLA-B27 negative. Our patient had aspirin hepatotoxicity. Whereas this is more common in the juvenile form of rheumatoid arthritis (JRA) (Manso et al, 1956; Russell, 1972; Rich and Johnson, 1973; Athreya et al, 1975) it has been observed in the adult form as well (Seaman and Plote, 1976; Wilson, 1976). She had also micrognathia, suggestive of JRA. The peculiar bony overgrowth seen at the CV and CT joints is reminiscent of the enlarged joints in some JRA patients when inflammatory synovitis occurs near the epiphyseal plate (Ansell and Bywaters, 1956; Martel et al, 1962).

The diagnosis of rheumatoid arthritis in this patient was based on the chronic, symmetrical polyarthritis with erosive changes in the small joints of
the hands, morning stiffness, and the presence of rheumatoid factor. Our patient's chest wall symptoms began at age 22, before closure of the epiphyses of the ribs. The secondary ossification centers of the rib are at the tubercle and the vertebral epiphyses at the head of the rib. These appear about age 14 but do not fuse until age 25 (Caffey, 1967). Therefore, we conclude that our patient had involvement by adult rheumatoid arthritis of these joints before epiphyseal closure, leading to this surprising and unique radiological picture. The inflammatory changes with ankylosis led to atypical back pain and pulmonary dysfunction.

This study was supported in part by grants from the South Central Texas Chapter of the Arthritis Foundation, The Ruth and Vernon Taylor Foundation of Denver, Colorado, and by a Clinical Research Center grant from The Arthritis Foundation.

References


