RHEUMATOID ARTHRITIS OF THE THORACIC AND LUMBAR SPINE

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We report seven cases of patients with seropositive rheumatoid arthritis in whom involvement of the thoracic and lumbar vertebrae occurred. Histological corroboration is presented in four. Pathological lesions comprised various combinations of paravertebral joint erosions, erosive discitis, anteroposterior and rotatory instability, major lumbar nerve root compression, and vertebral collapse. Specific radiological features are presented, enabling a distinction to be made between pure degenerative spondylosis and rheumatoid spondylitis. We submit that subcervical rheumatoid spondylitis is commoner than is generally believed, though less common than rheumatoid involvement of the cervical spine.

Rheumatoid arthritis affecting the cervical spine has been well described in the literature, but scant attention has been paid to the less common rheumatoid involvement of the thoracic and lumbar regions. The diarthrodial facet joints are composed of rheumatoid target tissues so it was not unexpected that Lawrence et al., in a detailed study published in 1964, found an increased incidence of lumbar facet joint erosions and anteroposterior instability in 50 rheumatoid subjects when compared with 50 controls. Their radiological study also showed that the mean disc width was less in the rheumatoid subjects than in the control group. Bywaters (1981) reported on autopsy findings in thoracic spines of 114 rheumatoid arthritis subjects. In eight subjects who had not had complaints relating to their thoracic spines he found discitis associated with rheumatoid erosion of the costovertebral joints.

A search of the literature has revealed reports of eight patients with rheumatoid arthritis complicated by abnormalities in the thoracic and lumbar spinal regions and in whom histological proof of spinal involvement was obtained. Table I summarises these cases together with four of our own which we describe in this paper. We also present three cases without histological proof but in whom radiological and clinical features suggest the diagnosis of rheumatoid involvement of the lumbar spine. All seven patients were white (Caucasian).

The literature contains several other reports of "rheumatoid spondylitis" with destructive vertebral lesions and histological corroboration which are really accounts of ankylosing spondylitis and are not relevant to the present study.

CASE REPORTS

Definite rheumatoid spondylitis

Case 1. A man aged 60 years had suffered from active seropositive rheumatoid arthritis for 10 years when chest pain necessitated radiological investigation. A lytic lesion of the third rib was demonstrated, as well as collapse of the fifth thoracic vertebra (Fig. 1). He died of rheumatoid valvular disease.

Histological examination of the affected vertebra (Fig. 2) showed areas of fibrinoid necrosis with surrounding chronic inflammatory cell infiltration typical of rheumatoid arthritis. Bacterial culture was negative. The rib lesion showed similar features.

Case 2. A man aged 58 had a history of seropositive rheumatoid arthritis involving peripheral joints for 15 years. The disease had been quiescent for three years on chloroquine therapy when he developed pain in the left thigh with a reduced knee jerk and hypo-algesia in the third lumbar dermatome.

Radiographs showed minor displacement at L2/3 and L3/4 (Fig. 3). There was a destructive lesion in L4 and erosion of the L3 4 disc space with surrounding sclerosis. Needle biopsy of the disc and vertebra yielded no bacterial growth. Histology of an open biopsy specimen showed areas of focal bone necrosis alternating with sclerosis, surrounded by a chiefly polymorphonuclear cellular reaction (Fig. 4). A second bacterial culture, Weil-Felix, Widal and Brucella agglutinin tests were negative. Clinical and radiological deterioration (Fig. 5) led to myelography nine months later, and a partial block was demonstrated at the L2 3 level (Fig. 6).

At operation the histological findings and negative bacteriological tests were confirmed. Decompressive laminectomy and posterolateral fusion were performed from L2 to L4 and this was followed three weeks later by
Case 1
Figure 1 Collapse of the fifth thoracic vertebra in a 60-year-old male rheumatoid patient.
Figure 2 Histological section of autopsy specimen of the collapsed vertebra. Areas of fibrinoid necrosis are surrounded by inflammatory cell infiltration, chiefly round cells. Note also the area of focal bone necrosis.

Case 2
Figure 3 Minor spondylolisthesis of L2 on L3 and destructive erosions with retrolisthesis at the L3-4 disc space. Facet joint damage is present at both levels. Note that osteophytosis is minimal. Figure 4—Photomicrograph of the biopsy specimen taken from disc and adjacent bone at the L3-4 level. Areas of focal bone necrosis are surrounded by an inflammatory cell reaction, chiefly polymorphonuclear. Figure 5—Increasing erosion and collapse of the L3 and L4 vertebrae and the intervening disc space over a period of nine months. Figure 6—Myelogram showing a narrowed segment in the dye column opposite the L3 vertebra. Figures 7 and 8—Radiographs three years after decompressive laminectomy and posterolateral fusion. In spite of partial extrusion of the anterior strut graft, fusion appears to have occurred from L2 to L4.
Case 3. A man aged 53, with a 15-year history of seropositive polyarticular rheumatoid arthritis and on corticosteroid medication, presented with a three-month history of low back pain radiating down the right thigh. The right ankle jerk and knee jerk were absent, and plain radiographs (Figs 9 and 10) demonstrated spondylolisthesis of L4 on L5. The MRI (Figs 9 and 10) or discography (Crawford 1985) demonstrated spondylolisthesis of L4 on L5 with a marked lateral and rotational shift to the left. Myelography showed almost complete obstruction at the L4–L5 level (Fig. 11).

At operation, the paravertebral joints showed instability, loss of articular cartilage and hypertrophic pinkish synovium. Histological examination of this material (Fig. 12) showed a non-specific inflammatory infiltration. Culture was negative. The fourth and fifth lumbar laminae were removed. No granulation tissue or disc material was found in the spinal canal but the cauda equina was compressed by the lamina and inferior facets of L4 vertebra, and the L4 nerve root was kinked around the pedicle of the vertebra. After decompression, posterolateral fusion was done from L3 to L5. Four years later the fusion was radiologically solid at L4–5 but there was a pseudarthrosis at L3–4. He still had low back pain but no nerve root symptoms.

Case 4. A woman aged 50 with a 10-year history of seropositive rheumatoid arthritis had a complaint of low back pain for six months. Radiographs showed sclerosis and narrowing of the L1–2 disc with a suggestion of erosions of the end-plates a year after her first presentation. Figures 13 to 16 show the evolution of the lesion over two years. A technetium bone scan showed increased uptake at the level of involvement. Culture from a needle biopsy produced no organisms.

Microscopy (Fig. 17) revealed bone with some necrotic trabeculae and fibrous tissue with a predominantly polymorphonuclear cellular exudate. Weil–Felix, Widal and Brucella agglutinin tests were negative. No antibiotics were given. Two years later she is painfree and the radiograph shows better definition of the sclerotic end-plates but early retrolisthesis is now developing.

Probable rheumatoid spondylitis

Case 5. A woman aged 74, with a 15-year history of seropositive rheumatoid arthritis in partial remission on chrysotherapy, complained of increasing low back pain for three years. The radiographs showed spondylolisthesis of L3 on L4 with some lateral subluxation of L4 on L5 (Figs 18 and 19). Both disc spaces were narrow and sclerotic and showed minor erosions. No further investigations were justified since her pain was controlled with supportive therapy.
Case 4. Figures 13 to 16—Serial radiographs over a two-year period showing fairly rapid loss of disc space culminating in early retrolisthesis. Note the loss of definition of the end-plates, unlike degenerative disease, especially in the 1983 picture (Fig. 14). Note also the gas shadows in the disc space, similar to degenerative disease. Figure 17—Histological section of end-plate bone showing necrotic trabeculae surrounded by fibrous tissue with a cellular reaction, predominantly polymorphonuclear.

Case 5. Radiographs showing retrolisthesis of L2 3 and spondylolisthesis of L3 4. The third lumbar vertebra is also displaced laterally. Note the osteophytic build-up on the right, the side of the greatest narrowing.
Case 6. A man aged 63 years, on corticosteroid therapy for chronic obstructive lung disease and seropositive rheumatoid arthritis, presented with multiple joint involvement and increasing back ache. Radiographs of the cervical spine showed atlanto-axial instability. In the lumbar spine the lateral view showed a characteristic ragged and finely eroded appearance of the end-plates and retrolisthesis of L3 on L4 (Fig. 20). He refused further investigations and his symptoms were partly controlled by wearing a corset.

Probable degenerative and rheumatoid arthritis

Case 7. A woman aged 78, with aggressive seropositive rheumatoid arthritis of 22 years' duration, presented with moderate mechanical low back pain. Radiographs (Figs 22 and 24) showed lumbar scoliosis with wedging, displacement and rotation of the body of L2. In addition to the characteristic “soft”, woolly and eroded appearance of the end-plates of the three central lumbar disc space margins, there is pronounced osteophytosis and sclerosis.

A typical degenerative lumbar disc (Fig. 21) compared with rheumatoid discitis in Case 7 (Fig. 22). Contrast the smooth, “hard” sclerotic margins of the degenerative end-plates with the rheumatoid ones which are vague and blurred with a “moth-eaten” appearance from inflammatory erosion. The osteophytes seen in the rheumatoid vertebral margins do not affect the diagnosis.

A degenerative scoliotic mid-lumbar scoliosis (Fig. 23) contrasted with a rheumatoid spine with similar deformity (Fig. 24). Note the blurred outline of the end-plates in the latter.
DISCUSSION
The term "rheumatoid spondylitis" has sometimes been used synonymously with ankylosing spondylitis, but a clear distinction should be drawn between these entirely separate disease entities. "Rheumatoid spondylitis" should be confined to the condition described in this paper where patients with proven rheumatoid arthritis have vertebral involvement (Table I).

The histological findings of Cases 1, 2, 3, and 4, taken in conjunction with the clinical and serological data, suggest strongly that the spinal lesions are due to rheumatoid arthritis. The polymorphonuclear reaction seen histologically in Case 4 may raise doubts and suggest pyogenic infection, but this is unlikely to be the case as bacteriological culture was negative, white cell count was normal, and the symptoms remained controlled without antibiotics.

Several radiological features distinguish these patients from those with degenerative spondylitis. Of these the most important is the ill-defined, blurred and eroded margin of the vertebral end-plates. We have found the facet joints to be ill-defined, and their eroded state can usually be deduced only through their inability to prevent major vertebral displacement either forwards, backwards or laterally; this occurred in all our cases. Sims-Williams, Jayson and Baddeley (1977) were able to demonstrate rheumatoid erosions in apophyseal joints only on stereoscopic radiographs. The presence of osteophytes does not negate the diagnosis of rheumatoid spondylitis.

In Cases 5, 6 and 7 there was no justification for invasive diagnostic methods so the diagnosis of rheumatoid spondylitis rests on the presence of the characteristic radiological features outlined above, in conjunction with their generalised rheumatoid arthritis. The reader is referred to Figures 21 to 24 where radiographs of our rheumatoid patient (Case 7) are juxtaposed and compared with those of the far commoner condition degenerative spondylitis. In this case it is quite likely that degenerative changes were present at her advanced age, but the radiological features suggested that her aggressive rheumatoid arthritis had superimposed inflammatory changes on the already existing degenerative process.

Based on the original findings of Lawrence et al. (1964), Bywaters (1981) and our own observations, we proposed three pathways in the pathogenesis of subcervical rheumatoid spondylitis. Synovitis probably starts in the apophyseal joints, slowing eroding cartilage and subchondral bone in exactly the same way as it does in peripheral joints. Erosion of the facet joints produces functional incompetence with resultant anteroposterior and lateral instability as seen in all our patients with involvement of the lumbar spine.

The next lesion, rheumatoid discitis, probably starts at the discovertebral junction as an enthesopathy: this pathogenesis was postulated by Shichikawa and

Table I. Summary of published reports in which clinical data are linked to positive histological identification of rheumatoid tissue

<table>
<thead>
<tr>
<th>Authors</th>
<th>Vertebral level</th>
<th>Sex</th>
<th>Local pathological findings</th>
<th>Neurological abnormalities</th>
<th>Origin of histological specimen</th>
</tr>
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<tbody>
<tr>
<td>Baggenstoss, Bickel and Ward 1952</td>
<td>T12</td>
<td>M</td>
<td>Vertebral collapse</td>
<td>Nil</td>
<td>Vertebral body</td>
</tr>
<tr>
<td>Linquist and McDonnell 1970</td>
<td>L3 4</td>
<td>M</td>
<td>Facet joint granuloma</td>
<td>Nil</td>
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<tr>
<td>Friedman 1970</td>
<td>L5</td>
<td>M</td>
<td>Intraspinal rheumatoid nodule</td>
<td>L5, S1 root signs</td>
<td>Spinal canal, attached to ligamentum flavum</td>
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<tr>
<td>Magnaes and Hauge 1978</td>
<td>Case 1: L3, 4, 5 F</td>
<td></td>
<td>Facet joint granulations</td>
<td>Right leg &quot;weakness&quot;: spinal claudication</td>
<td>Facet, lamina, ligamentum flavum</td>
</tr>
<tr>
<td></td>
<td>Case 2: L4, 5  F</td>
<td></td>
<td>Facet joint granulations</td>
<td>Weakness both legs, hypoalgesia; spinal claudication</td>
<td>Facets, laminae, ligamenta flava</td>
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<tr>
<td>Shichikawa et al. 1978</td>
<td>Case 1: L1, 2, 3 F</td>
<td></td>
<td>Vertebral collapse (steroid medication)</td>
<td>Nil</td>
<td>Disc and vertebral body</td>
</tr>
<tr>
<td></td>
<td>Case 2: L2, 4, 5 F</td>
<td></td>
<td>Vertebral collapse (steroid medication)</td>
<td>Nil</td>
<td>Disc and vertebral body</td>
</tr>
<tr>
<td>Hauge, Magnaes and Skulderud 1980</td>
<td>L4</td>
<td>F</td>
<td>Spondylolisthesis; destruction of facet joints and disc</td>
<td>Cauda equina compression; left L5, S1 sensory loss</td>
<td>Disc and facet joint</td>
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<td>Present series 1986</td>
<td>T5</td>
<td>M</td>
<td>Vertebral collapse</td>
<td>Nil</td>
<td>Vertebral body, disc</td>
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<tr>
<td></td>
<td>L2, 3, L3/4</td>
<td>M</td>
<td>Spondylolysis</td>
<td>Left L3 root signs</td>
<td>Facet joint, vertebral body</td>
</tr>
<tr>
<td></td>
<td>L4, 5</td>
<td>M</td>
<td>Spondylolysis</td>
<td>Right L4 root signs</td>
<td>Facet joint capsule</td>
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<tr>
<td></td>
<td>L1 2</td>
<td>F</td>
<td>Discitis</td>
<td>Nil</td>
<td>Disc and end-plate</td>
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REFERENCES


