Case Report

Lumbar Pannus Presenting as Cauda Equina Syndrome in a Patient with Longstanding Rheumatoid Arthritis

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ABSTRACT. Relatively little attention has been paid to lumbar spine involvement in rheumatoid arthritis (RA), and indeed it is generally considered to be an uncommon and usually clinically minor manifestation of the disease. We describe a case of acute right lower extremity weakness secondary to compression of multiple lumbar nerve roots by a large interforaminal rheumatoid pannus, and review the literature on this complication and other lumbar spine involvement in RA. (J Rheumatol 2001;28:627–30)

Key Indexing Terms:
RHEUMATOID ARTHRITIS LUMBAR SPINE

Chronic low back pain is common in the general adult population, with a prevalence between 28.4 and 50%[1-5]. Therefore, it is not unexpected that a significant percentage of patients with rheumatoid arthritis (RA) report chronic low back pain. Similarly, a survey of 503 RA outpatients in Leeds found that 43.2% complained of back pain[6]. However, except for osteoporosis, which is at least partly secondary to use of systemic corticosteroids, little attention has been paid to lumbar spine involvement in RA, and it is generally considered to be an uncommon and usually clinically minor manifestation of the disease. We describe a case of acute right lower extremity weakness secondary to compression of multiple lumbar nerve roots by a large interforaminal pannus. We review the literature on lumbar involvement in RA.

CASE REPORT

A 79-year-old man presented in October 1998 in the emergency room of London Health Sciences Centre with a complaint of pain and weakness in the right leg. He had a 57 year history of seropositive RA that had been managed medically with nonsteroidal antiinflammatory drugs only. He also had congenital nystagmus, hypertension, atrial fibrillation (not anticoagulated), and treated hypothyroidism. He had had bilateral knee arthroplasties, and a right total hip arthroplasty in 1996, as well as a cervical fusion.

His current right lower extremity pain had been preceded by one week of nonradiating low back pain, and 3–4 days of progressive right lower extremity weakness primarily involving hip flexion. On the day of presentation to the ER, he had fallen while riding a stationary bicycle, when his right lower extremity weakness became acutely worse. He was subsequently unable to bear weight or ambulate, both because of pain and weakness. A radiograph of the right hip did not reveal a fracture and his prosthesis appeared stable.

He was admitted to hospital and assessed by a rheumatologist. He denied any pain, but still complained of weakness in the right leg and was unable to walk. Examination revealed evidence of active peripheral synovitis, synovial thickening and/or effusions, and numerous deformities including severe ulnar deviation of the metacarpal phalangeal joints bilaterally, flexion deformities of the fingers, primarily in the right hand, and subluxation of both wrists. A subcutaneous nodule was noted over the left olecranon. He was weak in the right thigh muscles with only 3/5 strength in the hip flexors and abductors; weakness was also noted in the right quadriceps, and in dorsiflexion and plantar flexion of the right ankle. Deep tendon reflexes were asymmetrical with an absent right knee and left ankle jerk. Toes were downgoing.

Computerized axial tomography of the lumbar spine revealed extensive erosive changes of the right facet joint and transverse process at L4 (Figure 1). A paravertebral soft tissue mass was evident on the right. A magnetic resonance image (MRI) at L4-L5 also revealed a large paravertebral soft tissue mass, presumed to be a pannus arising from the facet joint and extending to the plane of the right L4 nerve root in the lateral recess (Figure 2). Spinal stenosis was considered to be marked (not shown). In addition, erosive changes were noted in the L2 vertebral body bilaterally (not shown). Probable inflammation of the interspinous ligament was noted at L1-L2, and possibly at L4-L5 and L5-S1 (not shown). Significant changes were also noted in the cervical spine, including pannus formation at multiple levels, subluxation of C6 on C7, and possible cord compression at C7-T1 (not shown).

A neurosurgical consultation was obtained; the neurosurgeon advised against a surgical intervention. The patient was discharged and followed in the outpatient department. His right thigh weakness became more severe so that he could not walk more than 10–12 steps even with a walker. Sulfasalazine was started at 500 mg/daily with the intention of increasing the dose gradually to 1 g twice daily. He died at home of an acute myocardial infarction in December 1998.

DISCUSSION

We performed an extensive literature review by accessing Medline from 1969 to 1999 using the identifiers “rheumatoid arthritis and lumbar,” “pannus and lumbar,” “lumbar

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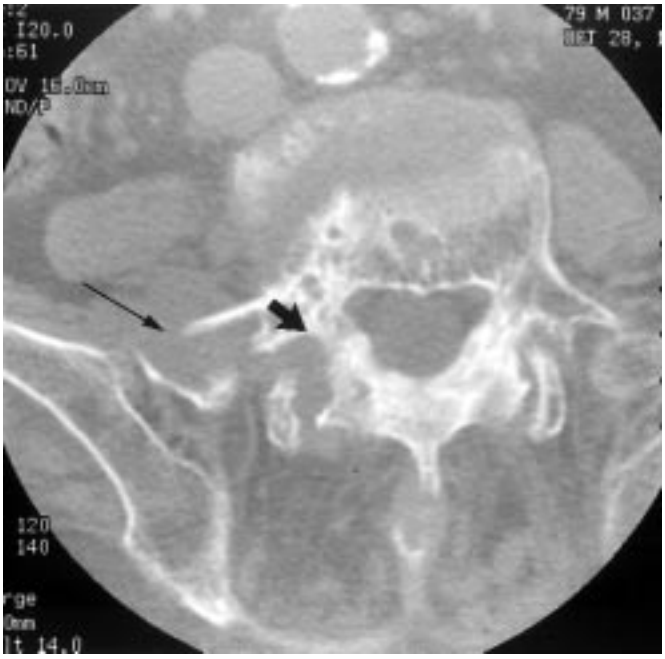

Figure 1. Computerized axial tomography, lumbar spine L4. Destructive changes are visible mainly on the right side, involving the facet joint and transverse process (short arrow). There is a paravertebral soft tissue mass on the right (long arrow).

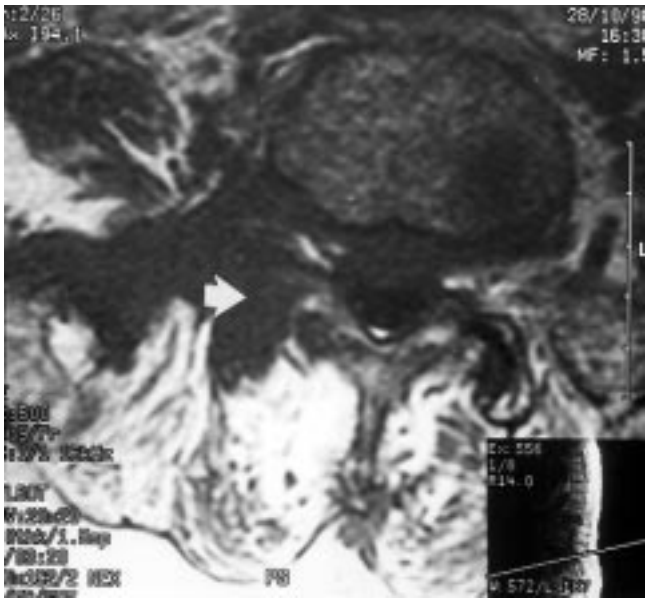

Figure 2. Magnetic resonance image, T1 weighted axial view at L4-L5. Soft tissue arising from facet joint extends to the plane of the right L4 root in the lateral recess (arrow).

synovitis," and "rheumatoid arthritis and cauda equina." All articles identified in Medline that appeared to pertain to rheumatoid involvement of the lumbar spine were reviewed in detail. Several additional articles pertaining to lumbar involvement in RA were identified by other means (e.g., bibliographies and article references).

Early reports on the involvement of the lumbar spine in RA are somewhat difficult to interpret because they speak of "rheumatoid spondylitis," often lumping together cases of ankylosing spondylitis and RA[7].

Lawrence, *et al* published an early report, and also a controlled study on the involvement of the lumbosacral spine in RA[8]. Radiographs were obtained from a group of 19 men and 31 women aged 55 to 64 years, with either clinical evidence of RA or a positive sheep cell agglutination test. These radiographs were compared with those of randomly selected, age matched controls, although details are scant. Disc narrowing was not significantly different between patients with RA and controls, but disc narrowing without bony proliferation was significantly more common in RA. Vertebral subluxation and apophyseal joint destruction also were more common in RA.

There are several limitations to this study. First, only 31 of the 50 "rheumatoid x-rays" were from patients with known arthritis. Second, the authors do not report which, if any, subjects complained of low back pain. Interestingly, the finding of more frequent disc space narrowing without osteophytosis in RA was reported again almost 30 years later by Helliwell, *et al*[6].

Several small series or case reports have been published of patients presenting with RA involvement of the lumbar spine[9-18]. We think that 21 of those cases, in addition to our patient, most likely had true RA, and did not have involvement secondary to osteoporosis or other causes not directly related to their disease.

Fifteen cases were female. The mean age was 59 years, with a range from 42 to 79. Disease duration ranged from 0 months (one patient presented with back pain and subsequently developed an inflammatory polyarthritis diagnosed as RA) to 40 years. Disease duration was reported in 21 of the 22 cases; RA had been present for 10 or more years in 15 patients (Table 1).

Thirteen patients were rheumatoid factor positive, 2 were seronegative, and the serologic status was unreported in 7. The majority of patients presented with chronic low back pain with or without lower extremity pain. Four patients, including our own, presented with lower extremity weakness in addition to their pain. The most frequently documented lesions were discovertebral and paravertebral erosions. In several cases the erosions were documented as being due to granulomatous formation[13,16,17]. One patient's symptoms were attributed to an intraspinal rheumatoid nodule, another to a large extradural cyst. One patient had anterior vertebral subluxation secondary to extensive erosive changes at the discovertebral interface. Three patients in addition to our own had a rheumatoid pannus compressing nerve roots.

These reports suggest that lumbar involvement due to RA is more likely to become manifest in individuals who have had longstanding RA. Since low back pain is a

Table 1. Case reports of RA involving the lumbar spine.

Reference	Sex	Age, yrs	Duration of RA, yrs	Serologic Status	Presenting Symptoms	Documented Lesions
Current Case	M	79	25	RF+	Acute LBP, LE weakness	Paravertebral joint pannus, discovertebral erosions
9	M	58	15	RF+	Acute LE pain	Discovertebral erosions
9	M	53	15	RF+	Chronic LBP, LE pain	Paravertebral joint pannus
9	F	50	10	RF+	Chronic LBP	Discovertebral erosions
9	F	74	15	RF+	Chronic LBP	Discovertebral erosions
9	M	63	?	RF+	LBP	Discovertebral erosions
9	F	78	22	RF+	LBP	Discovertebral erosions
10	F	43	16	RF+	Acute LBP	Paravertebral joint erosions
10	F	47	17	RF+	LBP	Paravertebral joint erosions
10	F	61	11	RF+	Chronic LBP	Paravertebral joint & discovertebral erosions
10	F	68	30	RF+	None	Paravertebral joint erosions
10	F	65	12	RF+	Acute LBP, LE pain	Paravertebral joint & discovertebral erosions
10	F	49	15	RF–	Chronic LBP	Paravertebral joint erosions
11	F	71	40	?	Acute LBP, LE pain	Paravertebral joint & discovertebral erosions, anterior body subluxation
12	F	61	3	?	Acute LBP, LE pain & weakness	Paravertebral joint pannus, discovertebral erosions
12	F	54	10	?	Acute LBP, LE pain & weakness	Paravertebral joint pannus, discovertebral erosions
13	M	47	0	?	Chronic LBP	Discovertebral erosions & pannus
14	M	42	7	?	LBP, LE pain & weakness	Intraspinal rheumatoid nodule
15	F	52	12	?	Lumbar mass, LE pain	Extradural rheumatoid cyst
16	M	56	3	?	Back pain	Vertebral body rheumatoid granuloma
17	F	73	6	RF–	Back pain	Vertebral body rheumatoid granuloma
18	F	62	9	RF+	Back pain, sciatica	Vertebral body and apophyseal joint erosion
22	15 F 7 M	59.4	13.4	13 RF+ 2 RF– 7 unknown		

RF: rheumatoid factor; LBP: low back pain; LE: lower extremity.

common symptom, its relation to RA will be unclear in most cases. Disc narrowing without osteophyte formation might suggest a greater likelihood of RA being responsible for the back pain[6,8]. However, RA should be strongly suspected as the cause of low back pain in those patients who have destructive changes in the vertebral body and/or apophyseal joint, particularly in those who present with lower extremity weakness and nerve root signs. In these individuals a rheumatoid pannus may be responsible, such as the one that was identified histologically in 2 cases in which there was extensive infiltration of granulation tissue with lymphocytes, plasma cells, pallisading epithelioid cells, and fibrinoid necrosis, all consistent with a rheumatoid pannus[12]. Our patient did not have surgical intervention that could confirm the presence of a rheumatoid pannus histologically. Nevertheless, the MRI findings were strongly suggestive.

It is not known where the pannus originates. The above case reports suggest that there may be 2 primary sites: (1) the discovertebral interface, and (2) the apophyseal joints. Extensive erosions and tissue destruction have been reported at both sites. It is more likely that a pannus would originate from facet joints since these articulations are synovial, whereas the discovertebral joints are not. Unfortunately, in each of the previously reported cases, the pannus was so extensive as to make identification of its source difficult. The MRI in our patient revealed lesions appearing to originate from multiple facet joints, especially at L4-L5 on the right. Perhaps MRI studies of the lumbosacral spine earlier in the course of the disease would allow for identification of the origin of a lumbar pannus. Savitz has published 2 reports on hypertrophic synovitis found incidentally at the time of surgery in 12 patients presenting with otherwise classic disc herniations[19,20]. These lesions were small and clearly originated from the facet joints. However, none of the cases was reported to have concomitant peripheral inflammatory joint disease, and the lesions themselves only contained a small number of hemosiderin laden macrophages.

It is not clear what the treatment of lumbosacral involvement in RA should be. It is possible that the disease would respond to aggressive treatment with disease modifying drugs, but this has not been tested. The results of surgical intervention remain unknown. However, patients presenting with lower extremity weakness may never recover their muscle strength and thus function may be lost, even if surgery is successful in permanently removing the pannus.

Severe involvement of the lumbosacral spine is a rare manifestation of RA. However, as our case demonstrates, it can result in sudden, severe loss of mobility and function. Further research is warranted to identify the site of the origin of the inflammatory tissue, the risk factors for this complication, and its natural history and treatment.

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