

## —Report on Experiments and Clinical Cases—

A Case Report of Rapidly Progressing Cauda Equina Symptoms  
due to Rheumatoid Arthritis

Hidemi Kawaji, Masabumi Miyamoto, Yoshikazu Gembun and Hiromoto Ito

Department of Orthopaedic Surgery, Nippon Medical School

**Abstract**

Although rheumatoid involvement of the lumbar spine is relatively rare, we report a patient with rapidly progressing cauda equina symptoms due to rheumatoid diskitis.

A 72-year-old woman was admitted to our hospital because of motor weakness below the iliopsoas muscle and sensory disturbance beneath the level of L2. Plain X-ray films, computed tomography, and magnetic resonance imaging demonstrated destruction of the L2/3 intervertebral disc and endplates with subluxation of the facet joints. The dural sac was compressed. Based on a diagnosis of spinal canal stenosis due to rheumatoid diskitis, we performed partial laminectomy and posterolateral fusion with pedicle screws. The neurological deficits improved immediately.

The mechanism of intervertebral disc destruction in this case is thought to be rheumatoid nodes or enthesitis. Destruction of the facet joints and the intervertebral disc might have led to severe instability and spinal canal stenosis.

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**Key words:** rheumatoid arthritis, spondylodiskitis, cauda equina syndrome, spinal fusion

**Introduction**

Rheumatoid involvement of the lumbar spine is relatively rare. We report a patient with rheumatoid diskitis who became unable to walk due to rapidly progressing cauda equina symptoms.

**Case Report**

A 72-year-old woman was admitted to a local hospital because of motor weakness and drift while walking. On magnetic resonance imaging (MRI), she was diagnosed as having cauda equina syndrome due to spinal canal stenosis at the level of L2/3 and

referred to our hospital in September 1999. She had been treated with corticosteroids for rheumatoid arthritis (RA) for 20 years. She was in Steinbrocker's stage IV and class 2 at the time of admission. There was no contributory family history.

Neurological examinations demonstrated significant muscle weakness below the iliopsoas muscle and sensory disturbance beneath the level of L2, and the patient could not walk at all. Deep tendon reflexes of the bilateral lower extremities disappeared and pathological reflexes were not observed. Bowel and bladder functions were normal.

On blood examination, the white blood cell count was 4,400/mL, the hemoglobin concentration was 12.1 g/dL, and erythrocyte sedimentation rate was



Fig. 1 Plain X-ray films on admission

The intervertebral disc space between L2 and L3 was narrowed, and the endplates of both vertebral bodies showed sclerotic changes.

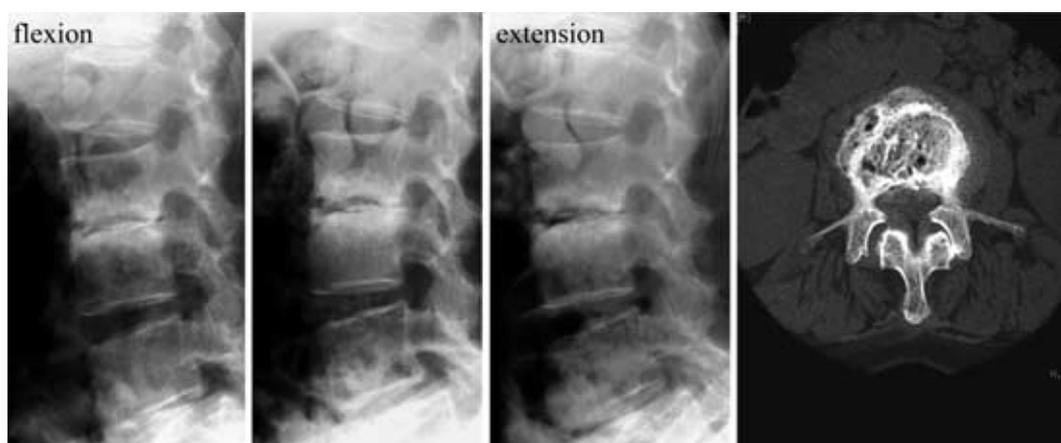


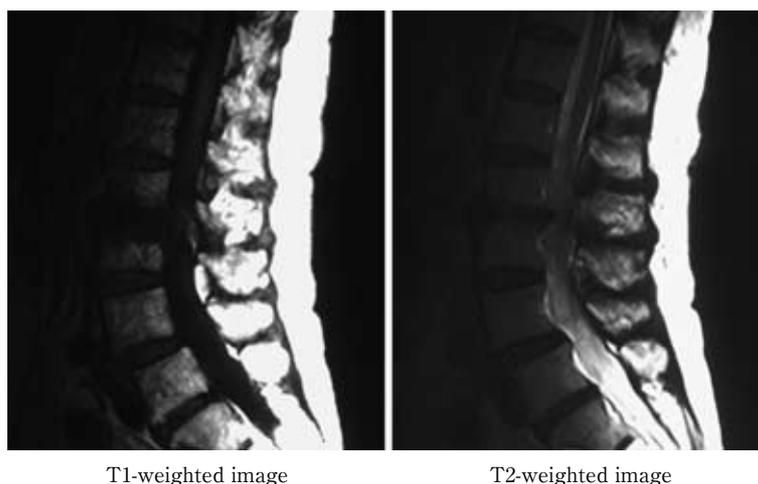
Fig. 2 Functional X-ray film and CT on admission

Functional X-ray film demonstrated severe instability on both flexion and extension. CT of L2/3 showed destruction of the intervertebral disc and asymmetry of the facet joints.

11 mm/hour, and no signs of infection or severe inflammation were seen. Rheumatoid factor was high at 96.7 IU/mL and RAPA (rheumatoid arthritis particle agglutination) was positive ( $\times 80$ ), but other findings were within the normal range.

Plain X-ray films showed narrowing of the L2/3 intervertebral disc, poorly defined endplates with erosions and sclerotic changes, and lateral listhesis of

the L2 vertebral body (Fig. 1). On functional view, instability of L2/3 was evident. Computed tomography (CT) demonstrated irregularity of the subchondral bones and subluxation in the L2/3 facet joints (Fig. 2). On MRI, we recognized narrowing and irregular signal intensity of the L2/3 disc. Wavy images with low signal intensity were seen on both T1- and T2-weighted images at the caudal endplate



T1-weighted image

T2-weighted image

Fig. 3 MRI on admission

The L2/3 intervertebral disc was narrowed, and signal intensity within the disc was irregular. The endplates of the L2 and L3 vertebral bodies showed wavy images with low signal intensity on both T1- and T2-weighted images. Significant spinal canal stenosis was recognized at the L2/3 level.

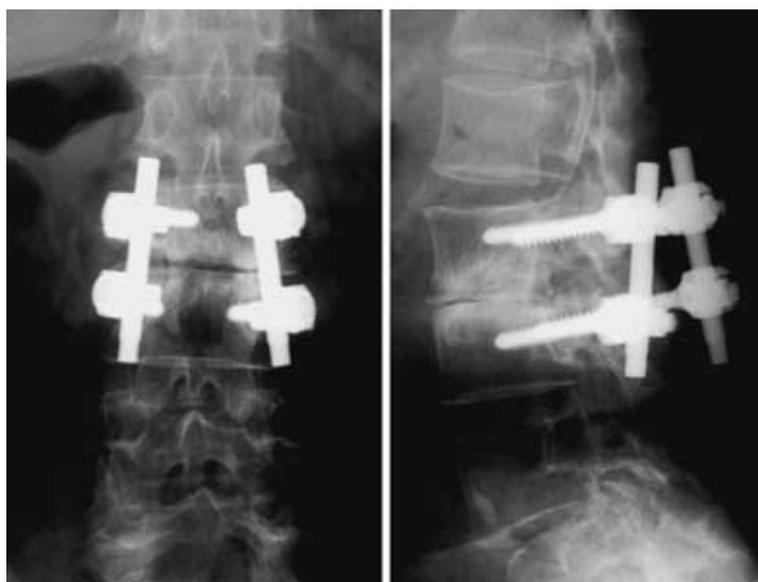


Fig. 4 Postoperative X-ray films

Posterolateral fusion and instrumentation were performed by a posterior approach.

of L2 and the cephalad endplate of L3 (**Fig. 3**).

We diagnosed spinal canal stenosis due to rheumatoid diskitis based on the absence of infectious signs, history of RA, and diagnostic characteristics on various images.

By a posterior approach, after partial laminectomy of L2 and L3, posterolateral fusion and facet fusion of L2/3 were performed with an autologous bone graft,

then instrumentation with pedicle screws was added (**Fig. 4**). On intraoperative observation, the bilateral capsules of the L2/3 facet joints were swollen, and synovial fluid had leaked. Cartilage of the facet joints had completely disappeared with debris floating in the joint fluid. Significant instability was recognized. These findings indicated rheumatoid synovitis of the facet joints. No space-occupying lesion was found in

the spinal canal. We believe that spinal canal stenosis had developed owing to instability of the intervertebral disc and had led to the development of neuroparalysis.

The postoperative course was good and there were no complications seen. The neurological deficits of both lower extremities improved immediately and muscle strength had almost completely recovered by 1 week after surgery. The patient could walk with a T-cane when discharged.

### Discussion

The cervical spine is more frequently involved in RA, and atlantoaxial subluxation is well known. However, destructive vertebral body lesion and aseptic diskitis in the thoracolumbar spine have been reported in patients with RA, although in small numbers. Heywood and Meyers investigated 746 patients with definitive and classic RA and recognized thoracolumbar involvement in 7 patients (0.94%). Of 12 previously reported cases, 4 showed destructive vertebral body lesion, 4 showed aseptic diskitis and 4 demonstrated local lesions only in the facet joints<sup>1</sup>.

Concerning the pathogenesis of aseptic diskitis, two mechanisms have been proposed<sup>1-3</sup>. The first one is a facet joint lesion in which erosions of the cartilage and subchondral bone occurs in the same manner as arthritis in the peripheral joints. This lesion causes insufficiency of the facet joints, which finally may lead to instability of the intervertebral disc. The second is inflammation at the attachment of the ligament that binds the vertebral body and disc. Shichikawa et al reported a case of enthesitis in the anterior and posterior longitudinal ligaments which spread to the surrounding tissue<sup>4</sup>. These changes are followed by destruction of the disc structure and may cause narrowing of the disc and erosive changes in the endplates.

Since simultaneous development of these lesions in the disc and facet joints leads to significant instability, listhesis of the vertebral body in the anteroposterior or lateral directions occurs. Furthermore, rotational displacement may disturb the root or cauda equina with stenosis of the spinal

canal.

On plain X-ray films, there are characteristic features of narrowing of the disc and unclear endplates with erosive changes. CT can show erosions in the facet joints that might not be seen on radiographs. Nakase et al have reported MRI features, such as narrowing and irregular signal intensity of the disc and wavy images at the borders between the discs and bodies<sup>5</sup>. This wavy image shows low signal intensity on both T1- and T2-weighted images, although inflammation is usually seen as low signal intensity on T1-weighted images and high signal intensity on T2-weighted sequences, which may indicate invasion of the pannus or rheumatoid node from the border between the discs and endplates.

On clinical diagnosis of our reported case, rapidly progression of destruction of the L2/3 disc and facet joints caused instability and lateral listhesis of the L2 body, and finally spinal canal stenosis developed along with paralysis of both lower extremities. On the basis of absent signs of infection, a history of RA, and imaging findings of the spine that did not contradict RA, we diagnosed rheumatoid diskitis.

We determined that decompression and spinal fusion were necessary to treat this case. Therefore, partial laminectomy and posterolateral fusion with pedicle screws were performed by a posterior approach. The reason we did not select anterior intervertebral body fusion is that solid fusion may induce spontaneous fusion of vertebral bodies, because there was no destruction of the vertebral bodies themselves and the intervertebral disc space had markedly narrowed.

In patients with RA, who have received corticosteroids for a prolonged period, the risk of instrumentation loosening after surgery is believed to be high. However, in patients with a mutilating form of RA, other parts of the spine may become involved and activities of daily life may be disturbed. In our case, there have been no postoperative complications. Pain in the lumbar region and the lower extremities disappeared, and neurological deficit recovered immediately. X-ray films showed that spinal fusion was achieved in L2/3 and stability was acquired. The patient could resume her daily

activities, but within 5 years, progression of RA cause atlantoaxial subluxation and destruction of the right knee and left ankle joint. Furthermore, she sustained a left-sided femoral neck fracture in a fall and now requires a wheelchair.

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