

Tandem cervical and lumbar canal stenosis in an elderly woman with long-standing rheumatoid arthritis

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ABSTRACT

Tandem spinal stenosis involving both cervical and lumbar regions is typically degenerative and may be accelerated by chronic inflammatory arthropathies such as rheumatoid arthritis (RA). We report the case of a 71-year-old woman with a 10-year history of RA who developed progressive cervical and lumbar radiculopathy due to inflammatory changes in the vertebral canal. In 2015, she developed an acute systemic illness with high-grade fever, severe joint pain, and marked swelling of the knees and wrists on both sides. She was diagnosed with RA and initiated on disease-modifying anti-rheumatic drugs. From 2024 onward, she developed progressive gait imbalance, limb weakness, and neuropathic symptoms. Magnetic resonance imaging of the spine revealed multilevel degenerative spondylosis with narrowing of the vertebral canal in cervical (C4–C7) and lumbar levels (L3–L5) with disc bulges. This case highlights the cumulative impact of RA-related inflammation and age-related degeneration on the development of tandem stenosis and the challenges of management in elderly patients.

Key words: Myelopathy, Rheumatoid arthritis, Tandem stenosis

Rheumatoid arthritis (RA) is a chronic systemic inflammatory disease that primarily affects bones, synovial joints, and ligaments. In 1890, Garrod first noted cervical spine involvement in 178 (35%) of 500 patients with RA [1]. Cervical myelopathy occurs in 2.5% of patients suffering from RA for more than 14 years [2]. Tandem spinal stenosis (TSS) is defined as the coexistence of cervical and lumbar spinal canal narrowing, originally described by Dagi *et al.* [3]. The condition produces intermittent neurogenic claudication and progressive gait disturbances [3-6]. The incidence of TSS varies from 0.12% to 28%, reflecting differences in diagnostic criteria and imaging sensitivity. Although the radiological prevalence is significantly higher, many cases remain unrecognized due to overlapping symptoms that mask stenosis at another level [3,4]. Chronic RA may coexist with osteoporotic changes, facet arthropathy, and spondylitis deformities, making the interpretation of lumbar and cervical imaging complex [1,7-9].

The present case reported here showed cervical spondylitis with posterior disc-osteophyte complexes at C4–C7 along with lumbar degenerative disease at the L3–L5 on magnetic resonance imaging (MRI). Additionally, the presence of cerebral small-vessel

disease and diffuse cerebral atrophy in this patient further impaired gait stability and neurological reserve, compounding the clinical presentation of tandem stenosis.

CASE HISTORY

A 71-year-old woman presented to our hospital with a 1-year history of progressive gait imbalance, lower limb weakness associated with lower back pain, a forward bent posture, and recurrent falls. She also had tingling and numbness in both upper limbs and neck stiffness. In 2015, she developed an acute systemic illness characterized by high-grade fever, severe joint pain, marked swelling of the knees and wrists of both sides, and an inability to walk for nearly three months. She was diagnosed with RA and initiated on disease-modifying anti-rheumatic drugs (DMARDs)-hydroxychloroquine, leflunomide, tramadol hydrochloride, and iguratimod, following which, her symptoms improved significantly. She regained full mobility and remained clinically stable, without recurrent joint swelling or functional impairment, for nearly a decade (2015–2025). Beginning in early 2024, she noticed new neurological symptoms, including numbness and tingling in both upper limbs. Around the same time, she developed gradually worsening

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lower back pain radiating to both lower limbs. Her posture progressively changed, and she began walking with a forward-flexed stance. Over subsequent months, she experienced increasing difficulty in walking. From late 2024 to early 2025, her lower-limb weakness progressed steadily. She began to lose balance frequently and had multiple falls due to loss of motor control of the lower limbs. By mid-2025, she was no longer able to stand or walk without support. The weakness was symmetrical, gradual in onset, and associated with radiating pain in both lower limbs. Past medical history was notable for long-standing hypertension. RA is well controlled without recent flare-ups and no active inflammatory symptoms. Her functional capacity declined significantly over the past year, and she became dependent for daily activities.

On general examination, her blood pressure was 180/90 mmHg. Systemic examination revealed normal cardiovascular findings with audible S1 and S2 and no added sounds, and normal respiratory findings. The patient was conscious, well-oriented, with preserved higher mental function. Formal cognitive assessment using the Mini-Mental State Examination showed a grade of 30/30. Cranial nerve examination was normal. On motor examination, the power in both upper limbs was moderately reduced (4/5) with markedly weak bilateral hand grip. There was no atrophy of intrinsic muscles of the hand with increased tone along with brisk deep tendon reflexes (biceps, triceps, and supinator). Sensory examination showed reduced sensation over C6–T1 dermatomes. Lower-limb power was markedly decreased, with proximal muscle strength at the hips (3/5), while the distal strength was (4/5) bilaterally. Deep tendon reflexes at the knees and ankles were diminished (+1) bilaterally. Plantar responses were flexor bilaterally. Sensory examination revealed reduced sensation over L4–S1 dermatomes. Cerebellar signs were absent. Spine examination revealed intermittent right-sided neck stiffness and lumbar paraspinal muscle tightness with no scoliosis or deformity.

Laboratory investigations revealed mild anemia (hemoglobin 11.6 g/dL) and hyponatremia (serum sodium 130 mEq/L). Her cardiac evaluation revealed mild left ventricular systolic dysfunction (ejection fraction 45%), making her a high-risk candidate for surgical decompression. Vitamin B12 levels were within normal range. Nerve conduction studies were not performed in this patient.

MRI brain showed cerebral small vessel disease and diffuse cerebral atrophy. MRI of the cervical spine revealed multilevel spondylotic changes characterized by disc-osteophyte complexes and uncovertebral hypertrophy at C4–C5, C5–C6, and C6–C7, causing mild-to-moderate canal narrowing and foraminal stenosis. The official radiology report didn't describe any definite

intramedullary signal abnormality. MRI of the lumbar spine demonstrated diffuse disc herniation at L3–L4 with an anteroposterior (AP) canal diameter of 7 mm, and a broad-based disc bulge at L4–L5 (AP diameter 9 mm), along with ligamentum flavum hypertrophy and facet arthrosis, leading to significant thecal sac indentation and compression of cauda equina nerve roots. A block vertebra at D3–D4 and sacralisation of L5 vertebra were also noted. The radiologist did not report the Lumbar spondylolisthesis in the image (Figs. 1 and 2).

Due to her advanced age and medical comorbidities, surgical intervention was not pursued, and she was managed conservatively with analgesics, muscle relaxants, supportive care, and physiotherapy. Despite medical treatment, her neurological function did not improve, and she remained severely weak with limited mobility.

DISCUSSION

Multilevel involvement is well recognized in RA, where cervical spine disease is reported in up to 80% of the patients affecting atlantooccipital, atlantoaxial, or subaxial C3–C7 joints and may progress insidiously before presenting with significant neurological deficits [1,10,11]. In severe or long-standing RA, cervical instability, pannus formation, and ligamentous hypertrophy may precipitate acute or progressive neurological deterioration, often exacerbated by chronic inflammation and systemic disease activity [6,12].

Castañero-Quintero *et al.* [11] reported a case of an 80-year-old hypertensive male patient, who was also a chronic smoker. He presented with paraesthesia and hypoesthesia of fingers and forearm for the last 5 months. Later, there was progressive dysesthesia of the lower limbs, and the patient was bedridden for 2 months. Clinical examination findings were symmetrical quadriparesis, generalised hyperreflexia, bilateral Babinski, and generalised hypoesthesia with C3 level sensitivity. MRI cervical spine showed an odontoid pannus with narrowing of the canal and spinal compression and signs of acute myelopathy. The laboratory report showed Rh factor positive (113 IU/mL). The patient returned home by walking after surgical decompression and C1–C2 arthrodesis. Janssen *et al.* [2] reported a retrospective analysis of a case series of 11 cases with RA. Nine patients (4 male, 5 female) who underwent surgery developed myelopathy. All patients presented with clinical myelopathy, and additionally, few had further neurological deficits such as tetraparesis in 2, hemi or mono paresis in 4, incontinence in 1, and pain in 3 cases. There was atlantoaxial instability of the median atlantoaxial joint, combined with retrodental pannus in 4 cases, anterior atlantoaxial subluxation in 2 cases, and basilar invagination in 3 cases. According



Figure 1: Whole-spine sagittal T2-weighted magnetic resonance imaging showing cervical canal stenosis from C3 to C7 with cord compression and lumbar canal stenosis at L3-L4 and L4-L5, consistent with tandem spinal stenosis

to Ranawat classification (Table 1), 2 cases were Class II, 5 Class III A, and 2 Class III B.

Treatment with DMARDs and biologics prevents the development, but it is insufficient once the clinical manifestations involve neurological deficit with myelopathy. Such progression requires surgical management [11]. While cervical involvement in RA is well recognised, lumbar spine manifestations are less clearly defined and are often difficult to distinguish by imaging from degenerative spondylosis. Pain is often the main symptom and main reason for seeking care; the most common regions include the lower back, buttocks, and thighs [13].

Similar cases in the literature have reported cervical myelopathy in patients with long-standing RA, often presenting with progressive quadriparesis or gait dysfunction. However, reports describing concurrent cervical and lumbar stenosis in RA patients remain limited. This overlap can obscure clinical localization, as cervical myelopathy may mask lumbar symptoms, or vice versa. Plain radiographs are limited in evaluating

Table 1: Showing the Ranawat classification [1,2]

Class	Description
I	No pain, no neurological deficits
II	Subjective weakness, hyperreflexia, dysesthesia
III	Objective weakness, long tract signs
III A	Ambulatory
III B	Non ambulatory

soft tissue pathology such as pannus formation. MRI remains the imaging modality of choice in symptomatic patients, due to its ability to detect soft tissue abnormalities [2,6,12]. In the present case, the coexistence of cervical and lumbar canal stenosis likely contributed to the mixed neurological presentation and progressive functional decline. The case belongs to type I according to Ranawat classification (Table 1).

These overlapping degenerative and inflammatory processes highlight the diagnostic complexity of TSS in elderly patients with a history of RA and emphasize the need for whole-spine evaluation in individuals presenting with mixed upper and lower motor neuron signs.



Figure 2: Axial magnetic resonance images of the lumbar spine revealed multilevel disc bulges with significant central canal narrowing and bilateral lateral recess stenosis, most pronounced at L4–L5

CONCLUSION

This case illustrates tandem cervical and lumbar spinal stenosis in an elderly patient with long-standing RA, highlighting the role of chronic inflammatory and degenerative processes. Whole spine evaluation should be considered in patients presenting with progressive neurological symptoms to enable timely diagnosis and appropriate management.

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