



## Resident fellow section: Clinical vignette

# Differential diagnosis of suddenly developed motor weakness in bilateral lower extremities of a 79-year-old male patient

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Received: October 21, 2022 • Revised: November 1, 2022 •

Accepted: November 5, 2022 • Published online: January 9, 2023

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J Yeungnam Med Sci 2023;40(4):457-460  
<https://doi.org/10.12701/jyms.2022.00787>

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## Patient information

A 79-year-old man visited the outpatient clinic of a university hospital with bilateral lower extremity weakness, which suddenly developed two weeks prior to presentation. The patient also experienced constipation and voiding difficulty, which manifested as urinary incontinence. He was taking medications for hypertension and dyslipidemia (bisoprolol fumarate, 2.5 mg, once daily; telmisartan, 40 mg, once daily; and (S)-amlodipine besylate, 2.5 mg, once daily). In addition, the patient had undergone radiofrequency catheter ablation at the cardiology department for atrial fibrillation 1 year prior

to this visit and was taking oral anticoagulants (rivaroxaban, 20 mg, once daily) and antiarrhythmics (flecainide acetate, 50 mg, twice daily). The patient was admitted to the Department of Physical Medicine and Rehabilitation for further evaluation and treatment. This study conforms to all RFS-CARE (Resident and Fellow Section-CAse REport) guidelines, and the required information has been reported accordingly ([Supplementary Checklist](#)).

## Clinical findings

Physical examination revealed symmetric motor weakness (manual muscle testing scores on bilateral hip flexors, 3; bilateral knee extensors, 4; bilateral ankle dorsiflexors, 2; bilateral great toe extensors and plantar flexors, 1). Light touch and pin-prick sensations were impaired below the bilateral L2 level. Voluntary anal contraction was impaired, and perianal and deep anal sensations were absent. Motor and sensory functions in the upper extremities were normal, but bilateral knee and ankle jerks were reduced. Furthermore, the Babinski reflex and ankle clonus were not observed.

## Diagnostic assessment

Vital signs and laboratory findings, including complete blood count, electrolytes, transaminase, creatine phosphokinase, C-reactive protein (CRP), erythrocyte sedimentation rate, and urine analysis, were within normal ranges. However, his hemoglobin A1c level was 6.6%, and the patient was diagnosed with diabetes through an oral glucose tolerance test.

### 1. Differential diagnosis

The following diagnoses were considered.

#### 1) Spinal cord infarction

Sudden onset weakness in the bilateral lower extremities increases the possibility of a spinal cord infarct. Because the deep tendon reflexes had decreased, the pathological site included the conus medullaris. Notably, a medical history of atrial fibrillation increases the likelihood of developing this disorder [1].

## 2) Spondylotic myelopathy

Although symptoms of spondylotic myelopathy often develop insidiously and gradually aggravate, they can occasionally occur suddenly and rapidly. Spondylotic myelopathy is commonly observed in clinical practice.

## 3) Spinal cord tumor

A spinal cord tumor was considered; however, the sudden occurrence of weakness in our patient made this diagnosis less likely.

## 4) Diabetic amyotrophy

The patient was diagnosed with diabetes. In addition, his deep tendon reflexes in the bilateral lower extremities had decreased. Therefore, the possibility of diabetic amyotrophy was considered. However, the patient's weakness pattern was symmetric, which differs from the asymmetrical neuropathy observed with diabetic amyotrophy [2].

## 5) Vasculitic neuropathy

CRP levels were within the normal range; thus, the possibility of vasculitic neuropathy was low. In addition, sensorimotor deficits from vasculitic neuropathy usually develop asymmetrically [3].

## 6) Guillain-Barré syndrome

Guillain-Barré syndrome was considered because the patient presented with symmetrical motor weakness in both lower extremities. The patient may have been in the early stages of Guillain-Barré syndrome, which usually begins in the lower extremities and spreads to the upper extremities. However, 2 weeks after the initial manifestation of the patient's symptoms, his motor weakness was still confined to the lower extremities. Therefore, the possibility of Guillain-Barré syndrome was low.

## 2. Diagnosis and management

Whole-spine magnetic resonance imaging (MRI), including diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) mapping, was performed. T2-weighted MRI showed focal swelling with enhanced signal intensity in the central grey matter of the conus medullaris (Fig. 1). Signal hyperintensity at

the conus medullaris was found on ADC maps, with no significant restricted diffusion on DWI (Fig. 1). On cervical and thoracic spine MRI, no significant abnormalities were found above the T12 level, and spondylotic myelopathy was excluded. In addition, electrodiagnostic studies, including nerve conduction studies, electromyography, and central motor conduction time, revealed no abnormalities. Therefore, disorders of the peripheral nervous system, such as diabetic amyotrophy, vasculitic neuropathy, and Guillain-Barré syndrome, were excluded.

The patient was diagnosed with subacute spinal cord infarction [4]. We performed transthoracic echocardiography (TTE) to evaluate cardiac risk factors and computed tomography angiography (CTA) to exclude aortic dissection. No abnormal findings were observed on TTE or CTA. We increased the dose of the oral anti-coagulant (rivaroxaban, 20 mg per day to dabigatran etexilate mesylate, 150 mg, twice daily). Exercises for strengthening the lower extremity muscles and improving standing balance and walking ability were conducted (Monday to Friday, 2 hours/day).

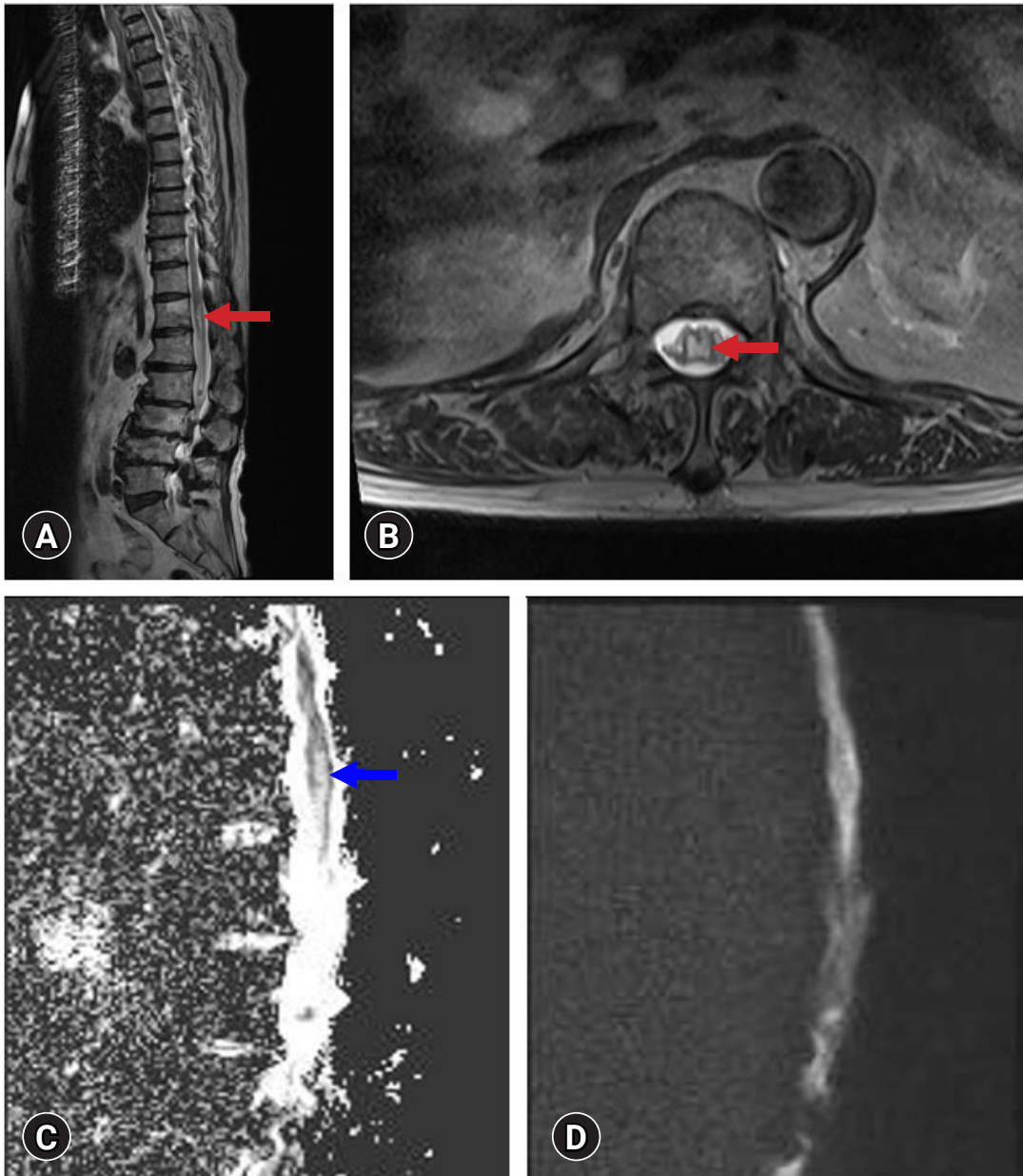
## Clinical course

At the 3-month follow-up after diagnosis, weakness in the right lower extremity of the patient improved (manual muscle testing score on bilateral hip flexors, 4; bilateral knee extensors, 4; bilateral ankle dorsiflexors, 3; bilateral great toe extensors and plantar flexors, 3).

## Discussion

Our patient was diagnosed with subacute spinal cord infarction. Spinal cord infarction occurs much less frequently than cerebral infarction and accounts for only 1% of all strokes [4]. The most frequent type of spinal cord infarction is anterior spinal artery syndrome, which presents with bilateral weakness, impairment of spinothalamic sensation, and preservation of deep sensations. Rarely, posterior infarcts that spare spinothalamic sensation and involve lemniscal sensation are encountered [5].

Numerous etiologies have been implicated in spinal cord infarction, including aortic disease, vertebral artery



**Fig. 1.** (A) Sagittal T2-weighted and (B) axial T2-weighted magnetic resonance images show focal swelling, with the enhanced signal intensity of the central grey matter of the conus medullaris (red arrows). (C, D) Signal hyperintensity of the conus medullaris (blue arrow) is found on apparent diffusion coefficient mapping, with no significant restricted diffusion on diffusion-weighted imaging.

dissection, arterial or cardiac embolism, fibrocartilaginous embolism, hypercoagulable states (e.g., sickle cell disease, antiphospholipid syndrome, and malignancy), decompression sickness, vasculitis, systemic hypotension or global hypoperfusion from cardiac arrest, radicular artery compression from the disc, and trauma [6,7]. The most common identifiable causes are aortic diseases such as aortic dissection or aortic aneurysm [6,8]. CTA of the chest/abdomen should be conducted in patients with thoracic cord infarcts or infarcts of the conus

medullaris to evaluate the presence of aortic dissection or aneurysm [7]. Atrial fibrillation is associated with an increased risk of subsequent spinal cord infarction. Therefore, evaluation of potential cardioembolic sources, including echocardiographic evaluation, is needed in patients with unexplained spinal cord infarction [9].

To date, there are no consensus guidelines regarding the management of spinal cord infarcts. In some previous case studies, anticoagulation and antiplatelet agents were administered in the acute phase of spinal cord in-

farction in patients who were suspected to have an atherosclerotic etiology. However, the effect of these agents on recovery after spinal cord injury has not been evaluated [7,10]. Similar to other spinal cord injuries, early rehabilitation is important for independence and physical function. It is also necessary to prevent complications from injuries, such as neurogenic bladder and bowel, urinary tract infections, pressure ulcers, orthostatic hypotension, spasticity, deep vein thrombosis, autonomic dysreflexia, and pulmonary problems.

## Conclusion

Clinicians should consider the possibility of spinal cord infarction in patients who present with sudden onset weakness in their bilateral extremities, especially in patients with a history of atrial fibrillation or aortic disease.

## Supplementary materials

Supplementary Checklist can be found via <https://doi.org/10.12701/jyms.2022.00787>.

## Notes

### Ethical statements

This study was approved by the Institutional Review Board (IRB) of Yeungnam University Hospital (IRB No: 2022-06-031-001). Written informed consent was obtained for the publication of this report.

### Conflicts of interest

Mathieu Boudier-Revéret has been an editorial board member of *Journal of Yeungnam Medical Science (JYMS)* since 2021. Min Cheol Chang has been an associate editor of *JYMS* since 2021. They were not involved in the review process of this manuscript. Otherwise, there is no conflict of interest to declare.

### Funding

None.

### Author contributions

Conceptualization, Methodology: all authors; Data curation: SYK, MCC; Formal analysis, Investigation, Resources, Supervision, Validation: MCC; Visualization: SYK; Writing-original draft: all authors; Writing-review & editing: all authors.

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