



Spinal Cord Infarction Associated with Coronavirus Disease 2019: A Case Report with Magnetic Resonance Imaging Insights

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Coronavirus disease 2019 (COVID-19) has been associated with various neurological complications, including the rare occurrence of spinal cord infarction. In this report, we present the case of a 42-year-old man who developed sudden quadriplegia after being diagnosed with COVID-19. Initial magnetic resonance imaging (MRI) provided inconclusive results; however, subsequent imaging revealed diffusion restriction and vertebral body signal changes, indicative of ischemic changes in the spinal cord. The patient received anticoagulation and corticosteroid therapy followed by rehabilitation, resulting in partial recovery of motor function. This case illustrates the importance of considering spinal cord infarction in patients with COVID-19 who present with neurological symptoms. Furthermore, it highlights the crucial role of MRI, including diffusion-weighted imaging, in diagnosis.

Keywords: Spinal cord infarction; COVID-19; Diffusion-weighted imaging; Vertebral body infarction

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Introduction

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), presents with a wide range of clinical manifestations, from respiratory distress to multisystemic complications [1,2]. Growing evidence indicates a potential association between SARS-CoV-2 infection and various spinal cord disorders, including acute transverse myelitis, acute necrotizing myelitis, neuromyelitis optica spectrum disorder, spinal epidural abscess, and spinal cord infarction [1]. Although rare, spinal cord infarction poses a notable challenge, and its occurrence in the context of COVID-19 complicates the diagnosis and management of this condition.

Magnetic resonance imaging (MRI) is the preferred neuroimaging modality for suspected cases of spinal cord infarction. Since diffusion restriction can occur with both inflammatory eti-

ologies and infarction, the limited spatial resolution of diffusion-weighted imaging (DWI) has historically hindered its clinical utility in evaluating spinal cord lesions. However, recent technological advancements in DWI have improved its capacity to reveal ischemic changes in the spinal cord [3,4].

Herein, we report a case of spinal cord infarction occurring post-COVID-19, emphasizing the rarity of this complication and the pivotal role of MRI in its diagnosis. This case underscores the importance of considering spinal cord infarction in the differential diagnosis of neurological complications associated with COVID-19, advocating for the early application of MRI in clinical practice.

Case Report

A 42-year-old man with no significant medical history present-

ed with symptoms of upper respiratory infection, including cough and sputum. He was confirmed to have COVID-19 through reverse transcriptase-polymerase chain reaction testing of a nasal swab. Five days later, he experienced a sudden tingling sensation in his neck, followed by weakness in both upper limbs. During transfer to the hospital, he also developed weakness in both lower limbs.

Upon arrival at the hospital, the patient demonstrated grade 1 muscle strength in both upper extremities and grade 0 in the lower extremities, as measured by the Medical Research Council (MRC) scale. Additionally, the patient exhibited sensory impairment below the C3 dermatome.

Initial brain computed tomography and MRI revealed no abnormalities. Likewise, his serum laboratory results were unremarkable: D-dimer, 0.16 $\mu\text{g/mL}$ (reference, 0 to 0.5); fibrinogen, 438 mg/dL (reference, 200 to 495); fibrinogen degradation products, 0.72 $\mu\text{g/mL}$ (reference, 0 to 3.0), prothrombin time, 12.5 seconds (reference, 10.3 to 13.6), and activated partial thromboplastin time, 31.6 seconds (reference, 25.0 to 37.0). Cerebrospinal fluid analysis and immunological testing showed no significant abnormalities, except a positive lupus anticoagulant screening test (1.28; reference < 1.20). The follow-up confirmatory test for lupus anticoagulant was also positive (1.30). Since all other autoantibody tests yielded negative results, this elevation was attributed to a transient response to the viral infection rather than an underlying hypercoagulable state.

Cervical spine MRI indicated high signal intensity and swelling in the anterior aspect of the spinal cord, spanning from C3 to C7, on T2-weighted images (Fig. 1A). At the lesion site, diffu-

sion-weighted images (b 800) exhibited high signal intensity, while apparent diffusion coefficient images displayed low signal intensity. These findings are consistent with ischemic changes (Fig. 1B, C).

The patient was initiated on anticoagulation therapy, receiving 60 mg of enoxaparin subcutaneously every 12 hours. Concurrently, he was treated with 1 g/day of methylprednisolone for 5 days. Due to escalating difficulty with breathing and expectoration, a tracheostomy was performed.

On day 12, the patient's spinal MRI revealed high signal intensity on fat-suppressed images in the posterior one-third of the C7 vertebral body, indicative of bone infarction (Fig. 1D). Three months following the onset of symptoms, spinal MRI displayed persistent high signal intensity in the cervical cord on T2-weighted images (Fig. 1E). After 3 weeks of treatment with enoxaparin, the patient was transitioned to oral aspirin at a dosage of 100 mg/day.

The patient continued rehabilitation therapy, which included range-of-motion exercises, balance training, activities of daily living training, and gait training. At the 1-year follow-up, a slight improvement was noted in the strength of his distal upper extremities. His lower extremity strength had improved to grade 4 on the MRC scale, enabling him to walk with assistance. The patient's neurological status was classified as C5 grade D on the American Spinal Injury Association Impairment Scale. He was able to breathe spontaneously without the need for a tracheostomy or supplemental oxygen and could void on his own. However, intermittent catheterization was still necessary due to residual urine.

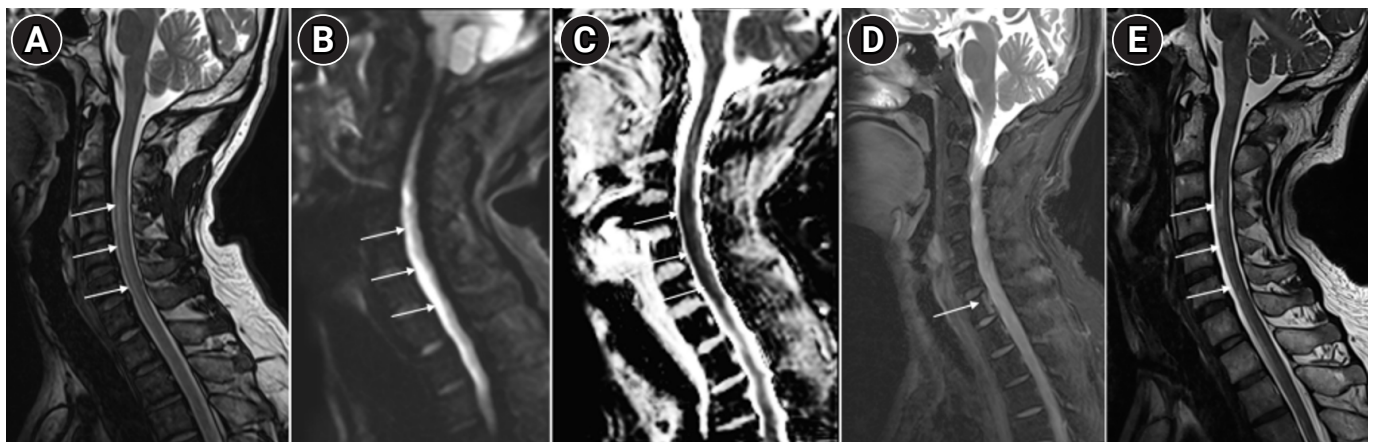


Fig. 1. Magnetic resonance imaging findings. (A) T2-weighted image reveals high signal intensity and swelling (arrows) in the anterior aspect of the spinal cord from C3 to C7. (B) The lesion site exhibits high signal intensity (arrows) on diffusion-weighted imaging (b 800). (C) Apparent diffusion coefficient image shows low signal intensity (arrows). (D) Fat-suppressed image demonstrates high signal intensity in the posterior one-third of the C7 vertebral body (arrow), indicative of bone infarction on day 12. (E) Three months after symptom onset, the cervical cord maintains high signal intensity (arrows) on T2-weighted imaging.

This study was approved by the Institutional Review Board Committee of Chung-Ang University Hospital (approval number: 2306-005-19473). Informed consent was waived by the board.

Discussion

The development of spinal cord infarction following COVID-19 infection underscores the potential of the virus to induce systemic vascular complications extending beyond the respiratory system. Although the pathophysiological mechanisms linking COVID-19 to spinal cord infarction are not fully understood, they may involve a multifaceted interaction of vascular endothelial damage, hypercoagulability, and inflammatory responses triggered by the viral infection [5].

The diagnostic challenges in identifying spinal cord infarction

are compounded by its rarity and the variability of clinical presentations, which may resemble other neurological conditions. We conducted an extensive search of the PubMed and Google Scholar databases using the following search terms: 'COVID-19 OR SARS-CoV-2 AND spinal cord.' We identified eight isolated cases and summarized their clinical findings in Table 1. Although COVID-19 has been considered a global pandemic for the past 4 years, published cases of spinal cord infarction associated with COVID-19 are extremely rare [3,4,6-12]. COVID-19 is known to be linked to a hypercoagulable state; however, the causal relationship between COVID-19 and spinal cord infarction remains unclear due to its rare incidence. In contrast, increasing reports have been made of COVID-19-associated transverse myelitis, now recognized as a significant neurological complication of the disease [1]. Notably, differentiating spinal cord infarction from transverse myelitis is challenging due to their similar clinical fea-

Table 1. Literature Review of All Published Cases of Spinal Cord Infarction in the Context of COVID-19

Reference/ Country	Age (y)/ sex	Comorbidity	Respiratory and systematic symptoms	Interval from COVID-19 to spinal cord infarction	Evidence of hypercoagulable state	Neurological manifestations	Lesions on neuroimaging	Treatment for spinal cord infarction	Outcome
Sampogna et al. (2020) [9]/Italy	69/Male	Hypertension Diabetes mellitus	Fever Cough Anosmia Respiratory failure requiring mechanical ventilator Pulmonary embolism with deep vein thrombosis Infarction of the left middle third posterior renal cortex artery	Approximately 34 days	Elevated D-dimer levels	Paraplegia with T10 AIS B	Ischemia from T8 to the conus medullaris on conventional MRI	None	Minimal assistance at a wheel-chair level
Eissa et al. (2021) [7]/Qatar	41/Male	None	Fever Initially moderate respiratory symptoms with 95% oxygen saturation progressing to ARDS requiring tracheostomy	Near simultaneous	Not described	Headache Right-side weakness followed by weakness in all four limbs	Diffusion restriction at the cervical and dorsal levels on DWI Restricted diffusion in right cerebellar and medulla oblongata on DWI Occlusion of the distal right vertebral artery on MRA	None	Not described
Bax et al. (2021) [10]/Italy	52/Female	None	Fever Desaturation	Simultaneous	Elevated D-dimer Reduced fibrinogen	Quadriplegia with AIS C Urinary retention	T2-weighted anterior cord hyperintensity from C6 to T1	Acetylsalicylic acid Methylprednisolone	AIS D Walker ambulatory

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Table 1. Continued

Reference/ Country	Age (y)/ sex	Comorbidity	Respiratory and systematic symptoms	Interval from COVID-19 to spinal cord infarction	Evidence of hypercoagulable state	Neurological manifestations	Lesions on neuroimaging	Treatment for spinal cord infarction	Outcome
Kahan et al. (2021) [3]/ USA	31/Male	None	Respiratory fail- ure requiring intubation	Approximately 20 days	Elevated D-di- mer and fi- brinogen lev- els	Quadriplegia with motor level C5/C5 and sensory level T4/T4	T2 hyperintensity and enhance- ment of the cervical spinal cord spanning C4 through C6, with corre- sponding re- stricted diffu- sion	Enoxaparin Methylpred- nisolone	Not de- scribed
Amalia (2021) [6]/Indone- sia	60/Male	Coronary ar- tery disease Hypertension Taking aspirin 81 mg once daily, bisoprolol 2.5 mg once daily, and atorvas- tatin 20 mg once daily	Fever Upper respiratory tract infection	14 days	Elevated D-di- mer and fi- brinogen lev- els	Complete pa- ralysis of lower limbs Loss of sensa- tion below the T12 level Urinary reten- tion	Drop in signal at the 12th tho- racic level on MRA	Heparin	Improve- ment in lower limb motor strength (walking with assis- tance)
Braglia et al. (2022) [11]/ Italy	44/ Female	Hypertension Uterine fibro- matosis Subacute thy- roiditis Idiopathic ele- phantiasis	Cough Fever Respiratory fail- ure requiring intubation	14 days	Elevated D-di- mer Normal fibrin- ogen	Acute back pain Syncope Paraplegia be- low the T12 level	Normal spine MRI	Enoxaparin Steroid	Assisted walking
Oleson et al. (2023) [8]/ USA	70/ Female	Undifferenti- ated con- nective tis- sue disease	Cough Fever	7 days	Not described	Paraplegia with T3 AIS A Urinary reten- tion Hypotension	T2 hyperintensity from T9 to the conus	Methylpred- nisolone	T6 AIS A
Xiao et al. (2024) [4]/ China	15/Male	Not described	Fever Dyspnea	Near simulta- neous	Elevated D-di- mer levels	Symmetric de- creased mus- cle tension of limbs	T2 hyperintensity in medulla ob- longata and cervical spinal cord and owl eye sign on conventional MRI	Not described	Not de- scribed
Present case	42/Male	None	Initially mild re- spiratory symptoms (i.e., cough and sputum) pro- gressing to re- spiratory fail- ure requiring tracheostomy	5 days	None	Quadriplegia with C3 AIS B	T2 hyperintensity of the cervical spinal cord spanning C3 through C7 with corre- sponding re- stricted diffu- sion	Enoxaparin Methylpred- nisolone	Quadriplegia with C5 AIS D

COVID-19, coronavirus disease 2019; AIS, American Spinal Injury Association Impairment Scale; MRI, magnetic resonance imaging; ARDS, acute respiratory distress syndrome; DWI, diffusion-weighted imaging; MRA, magnetic resonance angiography.

tures, such as the rapid onset of segmental neurological deficits and conventional MRI findings. These findings include T2 hyperintensities, which can appear longitudinally extensive or pencil-like on sagittal images and with an owl-eye appearance or extensive central involvement on axial images [3,4,9]. The clinical overlap between spinal cord infarction and transverse myelitis may contribute to an underdiagnosis of spinal cord infarction in the context of COVID-19. Among the nine cases of spinal cord infarction associated with COVID-19, including our case, six patients were male and three were female. Most spinal cord infarctions occurred within approximately 20 days following the onset of COVID-19 respiratory symptoms, with the infarctions located at the cervical or lower thoracic level.

In the present case, it is unclear whether COVID-19 infection directly caused the spinal cord infarction. A recent systematic review examining the link between COVID-19 and spinal cord ischemia identified coagulation abnormalities in five out of six patients. Our review found similar abnormalities in six of nine patients [12]. While coagulation-related abnormalities on laboratory tests may indirectly suggest a connection to spinal cord infarction, no specific biomarker currently exists for diagnosing spinal cord infarction caused by COVID-19. Classification criteria must be established for COVID-19-related spinal cord infarction, necessitating future research comparing the clinical characteristics, laboratory findings, and imaging features of spinal cord infarction cases associated with atherosclerosis and COVID-19.

Recent advances in DWI techniques for the spine have revolutionized diagnostic approaches, offering high sensitivity in detecting early ischemic changes within the spinal cord. This imaging modality enables the prompt initiation of therapies such as anticoagulation or antiplatelet treatment, which are crucial in mitigating irreversible neurological deficits. Theoretically, DWI is advantageous for detecting early ischemic changes because it can assess the restricted diffusion of water molecules in affected tissues. However, this diffusion restriction is not exclusive to infarcts; it can also occur under inflammatory conditions [3]. Other MRI findings of spinal cord infarction include signal changes within the vertebral bodies, which are diagnostically significant. Notably, infarction of the vertebral bodies at the same or adjacent levels as the spinal cord infarction is highly specific for diagnosing this condition, because the blood supply to the adjacent vertebral bodies and spinal cord originates from the same arteries. A recent study analyzing the MRI features of spinal cord infarction proposed that restricted diffusion and co-existing abnormalities of the vertebral body may represent key neuroimaging features for diagnosing spinal cord infarction [13]. Based on the clinical features and imaging findings, our patient was diagnosed with

spinal cord infarction following COVID-19 infection.

In conclusion, recognition and early intervention in cases of spinal cord infarction following COVID-19 is crucial, despite the rarity of this complication, to optimize patient outcomes. Our case report underscores the importance of clinical awareness among healthcare providers regarding this uncommon sequela and emphasizes the critical role of MRI, including DWI, as a non-invasive, rapid, and reliable diagnostic tool for COVID-19-related spinal cord infarction. Ongoing research and further case reports are necessary to deepen our understanding of the underlying mechanisms and to improve diagnostic and therapeutic approaches in this evolving clinical scenario.

Conflict of Interest

Du Hwan Kim is an editorial board member of the journal, but he was not involved in the peer reviewer selection, evaluation, or decision process of this article. No other potential conflicts of interest relevant to this article were reported.

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