

# Acute Spinal Cord Dysfunction Following COVID-19 Infection: A Case Report

Luca Braglia<sup>1</sup> | Gabriele Vandelli<sup>2</sup> | Angioletta Manenti<sup>3</sup> | Stefano Sacchi<sup>4\*</sup>

\*Correspondence: Prof Stefano Sacchi

Address: <sup>1</sup>Research Fellow, University of Modena and Reggio Emilia, Italy; <sup>2</sup>Neurology Fellow, University of Modena and Reggio Emilia, Italy; <sup>3</sup>Primary Care Physician, Mantova, Italy; <sup>4</sup>Senior Professor, University of Modena and Reggio Emilia, Italy

e-mail ✉: [stefano.sacchi@unimore.it](mailto:stefano.sacchi@unimore.it)

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## ABSTRACT

Here, we describe a 44-year-old female that developed abrupt symptomatology, including lower-limb paralysis with sensory involvement at the T12 level, two weeks after the resolution of COVID-19 bilateral pneumonia. Based on the neurological symptoms and the neurophysiological investigations, a clinical diagnosis of thoracic spinal cord dysfunction was made. However, all the complementary tests performed showed normal results. In particular, repeated MRIs showed no alterations; the cerebrospinal fluid analysis showed normal results: the CT scan of the brain was normal and the CT scan of the abdominal and thoracic aorta showed a normal size and course. Cases of spinal cord involvement have rarely been described, in which the MRIs never revealed cord signaling changes. We hypothesize that a small vessel spinal cord stroke might explain the pathogenesis and the absence of changes detectable on an MRI along with the patient's modest recovery during the long follow up. However, it cannot be excluded that the severe clinical course of SARS-CoV-2 infection, may suggest other different etiologies that could be related to prolonged hospitalization.

**Keywords:** COVID-19, Spinal Cord Dysfunction, Lower-limb Paresis

## Introduction

The spinal cord is the main pathway of communication between the brain and the rest of the body. The spinal cord controls the voluntary muscles of the trunk and limbs, and it receives sensory input from these areas. Spinal cord disorders may originate outside the cord or, less commonly, within the cord. Extrinsic factors include injuries and compression, such as vertebral fractures or compression from a tumor, hematoma, or abscess. Among the causes within the cord, the most common are infection (syphilis, viruses, particularly Human Immunodeficiency Virus [HIV]), vitamin B12 and copper deficiencies, tumor, inflammation, and demyelinating disorders, such as multiple sclerosis. With the outbreak of the COVID-19 pandemic, several reports have described neurologic complications associated with SARS-CoV-2 infection. These complications affected both the central and peripheral nervous systems (Ellul *et al.*, 2020; Nath, 2020; Studart-Neto *et al.*, 2020). Although spinal cord involvements have been widely reported, the complications seem to be relatively rare (Eissa *et al.*, 2021; Garg *et al.*, 2021;

Sampogna *et al.*, 2020). This case report describes a 44-year-old female that developed abrupt symptomatology, including lower-limb paralysis with sensory involvement at the T12 level, two weeks after the resolution of COVID-19 bilateral pneumonia.

## Case Report

After a few days history of dry cough, dyspnea, and fever, a female patient was admitted to the emergency room. She was 42 years old, weighed 114 kg, and was 178 cm tall. Her medical history included arterial hypertension, uterine fibromatosis, which was treated surgically, and subacute thyroiditis, treated with steroids, when the patient was 20 years old. She had long suffered from modest bilateral leg edema with no other sign and symptoms diagnosed as idiopathic elephantiasis. On admission, on March 22, 2021, she had low oxygen saturation, and a chest X-ray showed bilateral interstitial pneumonia. A nasopharyngeal swab was tested with a SARS-CoV-2 polymerase chain reaction (PCR) test, and the result was positive. Coagulation parameters and troponin levels were normal. The Inflammatory markers were significantly increased and mild anemia was observed.

The patient entered the respiratory assistance unit for patients with COVID, where she underwent non-invasive ventilation. Then, when oxygen saturation levels decreased further to 88%, she was admitted to the intensive care unit (ICU) and underwent an orotracheal intubation (OTI). The patient was treated with antibiotics, steroids, convalescent plasma, and enoxaparin.

On April 4, the oxygen saturation level was 96% and the mechanical ventilation was removed, and she began the rehabilitation process. SARS-CoV-2 PCR tests on two nasopharyngeal swabs performed before and after the OTI, showed negative results. During a rehabilitation course, on April 13, she experienced acute back and lumbar pain, then lipothymia, where she fell down, due to momentary loss of consciousness. Immediately after falling, she woke up with lower-limb paresis and anesthesia below the T12 level. A neurological examination revealed paraplegia and areflexia of the lower limbs, with tactile and painful hypoesthesia at the T12 level, suggesting spinal cord dysfunction.

To explore the causes of these neurological symptoms, between 13 and 30 April, she underwent several examinations. On April 19, her C-reactive protein (CRP) was 20 mg/L, and her D-dimer level was 2800 ng/mL. She had normal values for prothrombin time, partial thromboplastin time, fibrinogen, platelets, and white blood cell count and differential, but her hemoglobin was low, at 10.2 g/dL. An extensive serology work up, including an HIV test, showed negative results. An MRI of the cervical, dorsal, and lumbar spine with contrast showed a preserved vertebral alignment, a normal-size vertebral canal, and some modest cervical disc protrusions, at the level of passages D4-D5; however, no spinal cord

structural changes or signaling abnormalities were detected. A CT scan of the brain showed normal results. Electromyography excluded neuromuscular abnormalities or neuropathy. The evoked potential tests showed normal results in the upper limbs, and altered results in the lower limbs, which suggested a thoracic spinal cord injury. A CT scan of the abdomen with contrast excluded masses that could compress the aorta. A lumbar puncture was performed, and the cerebrospinal fluid (CSF) analysis showed normal results, which excluded inflammation and pathologies linked to bacterial or viral agents. Further serologic tests showed mild anemia, modest neutrophilic leukocytosis, normal levels of vitamins and copper, and the CRP level had dropped to normal. Finally, CT angiography of the abdomen showed that the aorta size and course were normal and the aorta was free of any pathology. The Adamkiewicz artery was not detectable, probably due to the inherent limitations of the method. During May and June, the patient continued physiotherapy rehabilitation. Enoxaparin therapy was continued, and steroid therapy was slowly reduced. There was a modest improvement in lower-limb strength, but moderate spastic paraparesis persisted.

On May 7, the blood counts were normal and the patient was discharged after withdrawing from enoxaparin. She was advised to start taking aspirin and to continue physiotherapy.

On December 20, she underwent a dorsal and lumbosacral MRI with contrast, but no spinal cord structural changes or signaling abnormalities were observed.

On March 14, 2022, she underwent a new neurological examination. She remained unable to walk without double support, and frequently needed to use a wheelchair.

On June 8, 2022, the patient continued to experience lower limb hypoesthesia, lower limb paraparesis, and severe back and leg pain, despite continuing physiotherapy and motor rehabilitation. Several analgesic drugs were prescribed, but they had little effect. Currently, she walks with crutches, but often has to use a wheelchair.

Summary of clinical history, laboratory tests, radiologic examinations and the timing of the most important clinical events are reported in [Table 1](#) and [Figure 1](#).

## Discussion

Since the beginning of the COVID-19 pandemic, cases of spinal cord involvement have been described, including myelitis/encephalomyelitis, hypoxic and autoimmune myelopathy, spinal cord infarction, and spinal epidural abscesses. A total of 58 patients with spinal cord involvement have been described in two recent reviews (Garg *et al.*, 2021; Mondal *et al.*, 2021) and in other case reports (Eissa *et*

*al.*, 2021; Sampogna *et al.*, 2020). Some potential mechanisms for spinal cord involvement might be a direct viral invasion (Eissa *et al.*, 2021), cytokine storm (Tang and Zheng, 2022), coagulopathy (Szegedi *et al.*, 2020), or an autoimmune response, which induced a demyelinating disease after a SARS-CoV-2 infection (Khair *et al.*, 2022), mediated by either direct neurotropism or an aberrant immune-mediated injury.

Typically, these patients had been hospitalized for severe COVID-19 and then experienced symptoms related to different types of myelitis or spinal cord ischemia. All patients showed alterations on CT/MRI scans or CSF analyses.

Our patient experienced sudden lower-limb paralysis with a loss of sensation below the T12 level, two weeks after the COVID-19 pneumonia had resolved and the SARS-CoV-2 PCR test produced a negative result. Based on the neurological symptoms and the neurophysiological investigations, a clinical diagnosis of thoracic spinal cord dysfunction was made, and the patient experienced lower-limb paraparesis with sensory involvement at the T12 level. However, all the tests that we performed to inform a more precise diagnosis showed normal results. In particular, repeated MRIs showed no alterations; the CSF analysis showed normal results; the CT scan of the brain was normal and the CT scan of the abdominal and thoracic aorta showed a normal size and course.

Recently, five other cases have been described in the setting of post-COVID-19 (Abrams *et al.*, 2021). Those patients also displayed moderate symptoms of clinical myelopathy and negative radiographic results (Abrams *et al.*, 2021). Moreover, some aspects of our patient's medical history were similar to those described by Abrams *et al.* (Abrams *et al.*, 2021). In all cases, the patients presented with an acute or subacute onset of lower-limb paresis and sensory changes, the MRIs never revealed cord signaling changes, and all patients experienced a modest recovery. However, Abrams's patients had experienced mild or asymptomatic SARS-CoV-2 infections, and most showed neurological symptoms months after the SARS-CoV-2 infection. In contrast, our patient was hospitalized for severe COVID-19 and manifested lower-limb paresis and anesthesia only three weeks after a SARS-CoV-2 PCR test showed a positive result.

Although radiologic findings may be absent in up to 40% of patients with inflammatory myelopathies (Scotti and Gerevini, 2001), it is unusual that a spinal injury as serious as that observed in our patient would occur without any alterations detectable on an MRI, notwithstanding the previous description by Abrams *et al.* (Abrams *et al.*, 2021). Our patient experienced minimal clinical recovery during the long follow up and repeated MRIs showed no alterations. For these reasons, we hypothesize that a small vessel spinal cord stroke might explain the pathogenesis and the absence of changes

detectable on an MRI along with the patient's modest recovery during the long follow up. Indeed, SARS-CoV-2 infections have been shown to cause micro hemorrhages and small vessel micro-thromboses, due to endothelial damage and coagulopathies (McFadyen *et al.*, 2020; Salamanna *et al.*, 2020). However, we cannot absolutely exclude that the severe clinical course of SARS-CoV-2 infection, which required recovery in an ICU, may also suggest other different etiologies that could be related to prolonged hospitalization.

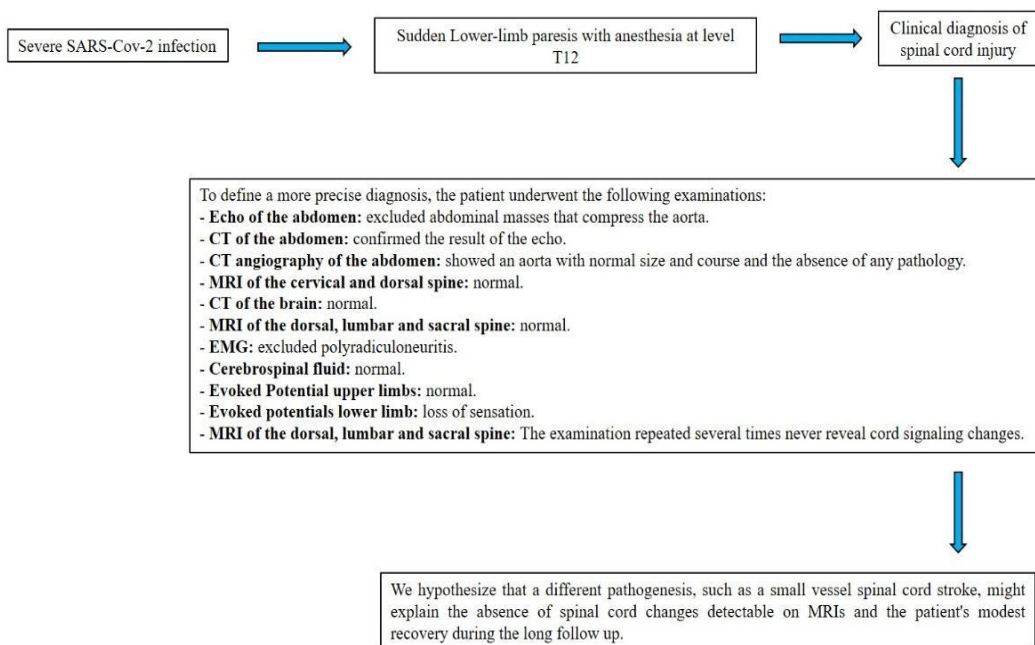
## Disclosure

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**Conflict of Interest:** The author declares that there are no conflicts of interest regarding the publication of this article.

**Consent:** The patient has given permission for publication of her clinical details.

**Ethics Approval and Consent to Participate:** Not applicable.



**Figure 1:** Flow chart of the most important clinical events, the main tests and the presumed pathogenesis of the spinal cord dysfunction.

**Table 1:** Summary of clinical history and of laboratory and radiologic examinations.

Proposed test	Timing of clinical events	Clinical diagnosis and symptoms	Blood Parameters	Imaging	Neuroimaging	Other Tests	Treatment
Emergency room	21-Mar-21	SARS-CoV-2 infection	Protein: 5601 (7.4), normal value: 150 to 4600 (0.4-1.1) LXN: 11813 X.N, normal value: 188 to 113 PTT: 11.1 s, normal value: 13 to 16.6 (4.4)	13 days of the illness showed bilateral increased parenchyma		SARS-CoV-2 PCR positive Organic Seroconversion: 95% (normal value: 96 to 100%)	Antibiotics
		Bilateral increased parenchyma	556.11 (1.4), normal value: 13 to 16.6 (4.4)				
		Dyspnoea	Protein: 521 (6.2), normal value: 150 to 4600 (0.4-1.1)				
		Pain	D-Dimer: 322 (0.9), normal value: <0.50 (0.1-0.2) (0.1-0.2) Fibrinogen: 607 (0.4), normal value: <450 (0.1-0.2)				
			CRP: 12.2 (0.2), normal value: <5 (0.1-0.2)				
			Protein: 1714 (0.4), normal value: 150 to 4600 (0.4-1.1)				
Covid Test	23-Mar-21	Worsening dyspnoea				Organic Seroconversion: 95% (normal value: 96 to 100%)	Not started treatment (NTI)
							Start
							Antibiotics
Intensive care unit	29-Mar-21	Patient worsening dyspnoea				Organic Seroconversion: 95% (normal value: 96 to 100%)	Empiric
Covid Test	4-Apr-21	Proteinuria 346 (0.4)	Protein: 5501 (7.4), normal value: 150 to 4600 (0.4-1.1) LXN: 11713 X.N, normal value: 188 to 113	Edema of the abdomen: colicoid abdominal masses that compress the vein		SARS-CoV-2 PCR: not required Organic Seroconversion: 95% (normal value: 96 to 100%)	Operative antibiotic Two days of corticosteroids Pain-physiotherapy session
		Improvement in oxygen saturation	556.7 (1.4), normal value: 13 to 16.6 (4.4)				Mechanical ventilation started
			CRP: 62 (0.2), normal value: <5 (0.1-0.2)				
	13-Apr-21	Improvement in oxygen saturation Laparotomy: severe abdominal back and lumbar pain Cervical lordosis at level T11 and T12 Pain: severe daily pain: not described at level T11					
	14-Apr-21	Pain: severe daily pain: not described at level T11				EMG: isolated polyradiculopathy	
Neurology	17-Apr-21	Pain: severe daily pain: not described at level T11		CT of the abdomen: confirmed the result of the echo	CT of the lumbar: normal		
	18-Apr-21	Pain: severe daily pain: not described at level T11				Orthopedic limb: normal	
	18-Apr-21	Pain: severe daily pain: not described at level T11	Protein: 5501 (7.4), normal value: 150 to 4600 (0.4-1.1) LXN: 11013 X.N, normal value: 188 to 113				
			556.10 (1.4), normal value: 13 to 16.6 (4.4)				
			D-Dimer: 350 (0.9), normal value: <0.50 (0.1-0.2) (0.1-0.2)				
			CRP: 20 (0.2), normal value: <5 (0.1-0.2)				
			Protein: 701 (0.4), normal value: 150 to 4600 (0.4-1.1)				
	14-Apr-21	Pain: severe daily pain: not described at level T11		MR of the cervical and thoracic spine: showed the presence of small cervical disc protrusions and normal vertebrae, without spinal cord signaling changes		Isolated Proteinuria: normal	
	26-Apr-21	Pain: severe daily pain: not described at level T11		CT angiography of the abdomen: showed an acute sub-renal vein thrombosis and normal contrast, without renal cord signaling changes		Isolated proteinuria: lower limb loss of sensitivity	
				MR of the thorax and lumbo-sacral spine: normal		SARS-CoV-2 PCR: negative	
	6-May-21	Slight improvement in proteinuria and dyspnoea	Protein: 4501 (5.4), normal value: 150 to 4600 (0.4-1.1)			SARS-CoV-2 PCR: negative	Physiotherapy from May to August 2021
			556.11 (1.4), normal value: 13 to 16.6 (4.4)			Organic Seroconversion: 95% (normal value: 96 to 100%)	
			D-Dimer: 407 (0.9), normal value: <0.50 (0.1-0.2) (0.1-0.2)				
			CRP: 13 (0.2), normal value: <5 (0.1-0.2)				
The patient was discharged	7-May-21						Physiotherapy from May to August 2021
	15-May-21	Pain: severe daily pain: not described at level T11					Empiric antibiotic New physiotherapy session
	19-May-21	Worsening of painful symptoms					Aspirin
	30-May-21	Slight improvement of pain antibiotic					Pain therapy
							Outpatient as needed
							Duloxetine
	30-Dec-21	Slight improvement of pain antibiotic					Pain therapy
	14-Mar-22	Slight improvement of pain antibiotic					Pain therapy
	8-Jun-22	Pain: severe daily pain: not described at level T11					New physiotherapy session
							Pain therapy

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