

# Acute transverse myelitis in a HIV-positive patient with COVID-19

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## Case Report

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# Abstract

Immunocompromised status keeps on being a challenge for a neurologist, especially in the context of the coronavirus disease – 19 (COVID-19) pandemic. We report a clinical case of a human-immunodeficiency virus (HIV) - positive patient who developed an acute transverse myelitis. Magnetic Resonance Imaging (MRI) examination showed longitudinally extensive spinal cord abnormality, and laboratory tests confirmed SARS-CoV-2 infection. The patient responded to methylprednisolone pulse therapy and therapeutic plasma exchange. No cases of HIV-positive patients with myelitis and COVID-19 has been reported yet.

## Introduction

Transverse myelitis (TM) is a focal disorder of the spinal cord presenting in acute or subacute manner (hours/days) resulting in motor, sensory, and autonomic dysfunction of varying degrees of expression depending on the type, location and surface of the lesion longitudinally and transversely. The term longitudinally extensive transverse myelitis (LETM) is used when the spinal cord lesion extends over three or more vertebral segments.

The possible causes of myelitis are as follows: infections (viruses, bacteria, fungi, parasites), post-infectious or post-vaccination, paraneoplastic, demyelinating (multiple sclerosis (MS), neuromyelitis optica (NMO), acute disseminated encephalomyelitis, other inflammatory disorders (neurosarcoidosis, systemic lupus erythematosus, Sjögren syndrome, Behçet's disease), congestive edema due to dural arterio-venous fistula, and tumor [1, 2]. TM may be idiopathic in 15-30% of cases. Post-infectious and idiopathic myelitis incidence varies between 1.3 - 8 cases / 1 million, but can reach 24.6 cases / million in patients with MS [3]. Recently case reports of myelitis associated with the new coronavirus infection were published, but the mechanism of spinal cord injury remains unclear [4,5].

Human-immunodeficiency virus (HIV) causes spinal cord injury both by direct HIV virus invasion, presenting in vacuolar myelitis in most of cases, as well as a manifestation of opportunistic virus-induced infections (Herpes simplex virus (HSV), Cytomegalovirus (CMV), Epstein-Barr virus (EBV), Varicella Zoster virus, or bacteria (Mycobacterium tuberculosis, Treponema pallidum), but can also be due to vitamin B12 deficiency. Vacuolar myelopathy (VM) is symptomatic in 5-10% of acquired immune deficiency syndrome (AIDS) patients, has a progressive evolution and is pathologically characterized by the presence of intralamellar white matter vacuoles in the posterior and lateral columns of the thoracic spinal cord [6].

SARS-CoV-2 causing the COVID-19 pandemic is proven to be neurotropic and may affect the central and peripheral nervous system. The mechanism of the new type of coronavirus neuroinvasion has not yet been fully identified. [7]. Short- and long-term health effects for patients who have been infected have not yet been assessed. The changes induced by the new type of coronavirus in the coagulation status

resulted in an impressive number of fatal thromboembolic events, including the cerebral ones. Guillain-Barré syndrome reported in COVID-positive patients is explained as rather a possible parainfectious manifestation, and cases of necrotizing hemorrhagic meningoencephalitis, altered mental status, and respiratory distress syndrome are referred to both direct virus action on brain neurons, as well as cytokine storm syndrome [8,9].

## Case Report

A 27-year-old male, with known HIV infection for the past 1 year on anti-retroviral therapy, was hospitalized on June 7, 2020 due to paresthesia and numbness in legs and in right arm, paralysis in lower extremities, bladder and bowel dysfunction (retention). All these symptoms developed quickly and reached the nadir in 15 hours.

Clinical evaluation revealed normal cranial nerves function, spastic tetraparesis, with 4/4,5 Medical Research Council (MRC) in upper and 0.5/2MRC in lower extremities, Th7 superficial and C7 deep sensory level disturbances. The patient was subfebrile 37.5C without other systemic abnormalities.

An extensive paraclinical workup was obtained. Routine laboratory data showed a slight general blood inflammatory abnormalities (WBC  $13.7 \times 10^9$ , ESR 14 mm/h) but with negative CRP. His CD4 count at admission was 310 cells/ $\mu$ l, and viral load was less than 40 copies/ml. Cerebrospinal fluid (CSF) analysis was normal. Blood serology and CSF polymerase chain reaction (PCR) for HSV 1,2,6, CMV, EBV, Borrelia burgdorferi, Treponema pallidum, Toxoplasma gondii, Chlamydia trachomatis, Mycoplasma pneumoniae, Ureaplasma urealyticum were negative. Autoimmune markers tests (autoantibodies ANA, ANCA), tumor marker tests (CA 19.9, Ca 15.3, PSA, CEA, Alpha Fetoprotein) and hepatitis viral serology all were negative. Thyroid function tests were normal. First swab for SARS-CoV-2 at admission was negative. CSF oligoclonal bands (OCB) and serum anti-aquaporin-4 antibody (AQP4-IgG), anti-MOG antibodies were negative. Vitamin B12, methylmalonic acid and angiotensin-converting enzyme levels were normal. Folic acid level test was 4,62 nmol/L (normal range 6.00-39.0). Brain MRI did not show any abnormalities. Spinal cord MRI revealed an extensive C4-Th5 lesion mainly in posterior columns and right lateral column without gadolinium enhancement (Figure 1).

The patient was treated with IV methylprednisolone 1g/day for 5 days, followed by five plasma exchanges. His condition improved with a significant reduction in paresis. Lung CT performed on 19<sup>th</sup> day of illness showed a slight patchy ground-glass opacity basal on the left side. No other typical symptoms for CoV-2 infection were noted. A repeated swab for SARS-CoV-2 was positive, followed by negative COVID-19 PCR in the CSF. A one month follow-up spinal MRI didn't reveal significant changes.

## Discussion

The presented case is unique for several reasons. All these circumstances - transverse myelitis, HIV infection and COVID-19 may be a random combination, as well as a range of three successive elements

or an interrelationship that influenced the onset of clinical signs and imaging changes. Normal CSF examination does not meet the TM diagnostic criteria [10]. Negative data for OCB and AQP4-IgG, and normal brain imaging disclaim the most common demyelinating causes of TM - MS and NMO, but do not rule out a possible isolated clinical syndrome or NMO spectrum disorders.

Spinal cord injury presented in posterior and lateral columns assumed a subacute combined degeneration (SCD) due to vitamin B12 deficiency. Neurosyphilis and neurosarcoidosis were excluded by laboratory and imaging studies.

Despite the patient's CD4 cells count does not indicate a severe degree of immunosuppression, the risk of opportunistic infections as a cause of myelitis has to be considered, however the absence of CSF pleocytosis and normal infectious disease tests did not confirm this hypothesis. HIV-associated spinal cord disease cannot be excluded, particularly vacuolar myelitis, which is slowly progressive, predominantly in the AIDS stage. The clinical examination usually demonstrates motor and sphincter disorders, sensitive ataxia, however without a sensory level, as well as normal or discretely modified MRI and often develops simultaneously with cognitive impairment [6].

The patient's SARS-CoV-2 infection role is controversial. This could be an accidental co-infection during hospitalization, fact supported by the negative test result at admission and the lack of typical blood changes. At the same time a false-negative result cannot be denied. High-dose corticosteroids therapy and plasma exchange might influence the relatively benign course of SARS-CoV-2 pneumonia. Myelitis may have features of a direct virus injury, or a parainfectious autoimmune process, as well as a result of molecular mimicry, the fact sustained by the positive response to corticosteroids therapy.

## **Declarations**

### **Compliance with ethical standards**

#### **Ethical approval**

The patient consented to publish his anonymized health data.

#### **Conflict of interest**

The authors declare that they have no conflict of interest.

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## Figures



**Figure 1**

Sagittal and axial MRI shows the spinal cord segmental myelopathy area at C4-T5 level, ~15 cm in length, with predominant involvement of the posterior and right lateral columns, without contrast uptake.