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Conus myelitis associated with Covid 19 infection - a rare complication

ISHMA AIJAZI, FADIL MUSTAFA ABDULLA, POURNAMY SARATHCHANDRAN, INSHIA BEGUM

DUBAI HOSPITAL INTERNAL MEDICINE DEPARETMENT (DUBAI HEALTH AUTHORITY)

ABSTRACT

Covid 19 pandemic has taken away millions of lives. Our understanding of this disease, till to date, is not complete. This disease has a wide variety of neurological manifestations. Acute transverse myelitis is one such rare neurological complication of Covid 19. The exact etiology is not clear. Auto immunity might be one of the possible mechanisms. We report a case of 39-year-old lady, who had recent history of high-grade fever and cough. This was followed by weakness of both legs and in- ability to pass urine. SARS-CoV-2 (PCR) from nasopharyngeal swab was positive. She was found to have features of acute non compressive myelopathy. MRI brain and MRI cervical spine with contrast was normal. MRI dorso lumbar spine with contrast was suggestive of diffuse hyper intensity of conus medullaris with contrast enhancement suggestive of conus myelitis. CSF analysis ruled out infection and autoimmune causes. She was pulsed with high dose steroids. There was some transient improvement in symptoms.

Learning points:

1) Physicians should not consider covid as a respiratory illness only. It can present with a variety of extra pulmonary manifestations

2) Acute transverse myelitis is a rare complication of covid 19 infection. Timely recognition and treatment can prevent permanent neurological damage and residual disability.

3) Conus myelitis might not present with classic upper motor neuron signs. Any new onset bladder dysfunction in a setting of a recent covid infection should be taken seriously and requires urgent imaging of the spine.

Keywords: Covid 19, extra pulmonary manifestations, acute transverse myelitis, conus medullaris

***Correspondence to Author:**
ISHMA AIJAZI
DUBAI HOSPITAL INTERNAL MEDICINE DEPARETMENT (DUBAI HEALTH AUTHORITY)

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Introduction

Covid 19 infection can present with neurological symptoms namely headache, anosmia and dizziness. [1]. Other neurological manifestations include acute encephalitis, Guillain -Barre syndrome, large vessel strokes and acute transverse myelitis [2]. ATM associated with covid 19 was initially reported in a 66-year-old man from Wuhan who developed par paresis one week after the onset of fever [3]. We present a case of a young lady who developed covid 19 infection, followed by paraparesis and urine retention. MRI spine revealed involvement of the conus medullaris. To our knowledge this is the first case reported in literature of “conus myelitis - associated with covid 19 infection”

Case description

39-year-old lady from Philippines presented to the hospital with 1 day history of numbness and weakness in legs followed by inability to walk and difficulty in passing urine .4 days prior to this she had complaints high grade fever and mild cough. She presented to the hospital with urine retention. On examination, she was afebrile with normal vitals. CNS examination revealed intact higher mental functions, neck was supple. Cranial nerve examination was normal. Power in upper limb was 5/5, lower limb 3/5 with brisk reflexes and bilateral up going planters. There was impairment in pain and touch sensations till T3. Joint position sensation was impaired in toes and ankles. Joint position sensation was normal in upper limbs limb. Other systemic examination was unremarkable.

Her labs revealed normal full blood count with mild lymphopenia. Urea electrolytes and creatinine, liver function tests were in normal range. Ferritin 73.7 ng/ml (15-150 ng/ml), c reactive protein 17.2 mg/l (<5mg/l), d dimer 1.34 ug/ml (<0.5 ug/ml). Interleukin 6 (9.7pg/ml). Serum folic acid was 5.4ng/ml (3.1-20 ng/ml), vitamin B12 323 pg/ml (187-883 pg/ml). Autoimmune panel was negative.

Serology for Epstein–Barr virus, cytomegalovirus, hepatitis B and C, HIV and mycoplasma was negative. Urine and blood culture revealed no growth.

SARS-CoV-2 (PCR) from nasopharyngeal swab was positive

MRI brain with contrast was normal. MRI whole spine with contrast revealed diffuse hyper intensity of the conus medullaris with contrast enhancement suggestive of conus myelitis.

She was immediately catheterized for urine retention. A diagnosis of non-compressive myelopathy was made. Urgent CSF analysis was done to rule out infection. She was pulsed with high dose steroids (methyl prednisolone) and was empirically started on iv acyclovir, which was later stopped after CSF herpes simplex and VZV PCR came negative. She was also empirically started on iv immunoglobulins (400mg/kg/day).

Detailed CSF analysis revealed a mildly elevated protein 49mg/dl (15-45 mg/dl), normal glucose and, normal LDH levels. CSF micro and culture was negative. PCR for all virus and tuberculosis was negative. Auto immune anti bodies were negative.

Patient had transient improvement in power. At discharge she was able to ambulate with support. However, the bladder dysfunction continued, and she was discharged on catheter.

Table:1 Detailed CSF analysis

Macroscopic appearance: slightly turbid – colorless. Blood not visible: coagulum: not visible
CSF cell count: wbc 600/mm3 (0-5) Rbc 80/mm3 Poly morph 5%, lymphocytes 95% Gram stain: no organism seen
Micro and culture: negative
CSF glucose 69 mg/dl (40-76) protein 49 mg/dl (15-45) LDH 38U/L
Herpes simplex virus (1 and 2) DNA- PCR negative
Herpes simplex virus (6 and 7) DNA – PCR negative
Varicella zoster virus (VZV) DNA -PCR negative
Enterovirus (DNA) -PCR - negative

TB – PCR - negative
AFB smear: negative AFB: culture: no growth
Auto immune antibodies Aquaporin -4 antibodies (Anti NMO- antibodies) < 1.1 (1.1) Antibodies for anti -myelin associated glycoprotein (MAG) IgM and Ig G <1.1 (1.1)
Ig G intrathecal synthesis: negative **Csf oligoclonal bands negative** CSF albumin 270mg/l (<350mg/l) Immunoglobulin G (Ig G/CSF) 32 mg/l (<34mg/l) **Serum oligoclonal band negative** Serum albumin 36.4 gm/l (35-52 gm/l) Immunoglobulin G (Ig G/ serum) 20.5gm/l Albumin ratio (csf/s) 7.4 (up to 6.7)

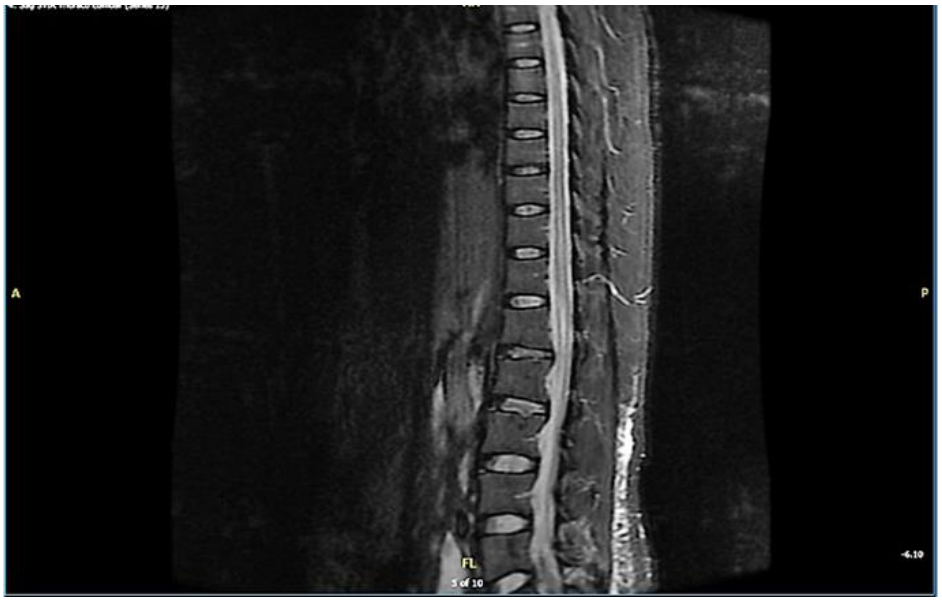

Image 1: Sagittal STIR image showing hyperintensity of conus epi-conus with central hyperintensity extending up Indicating cord edema.

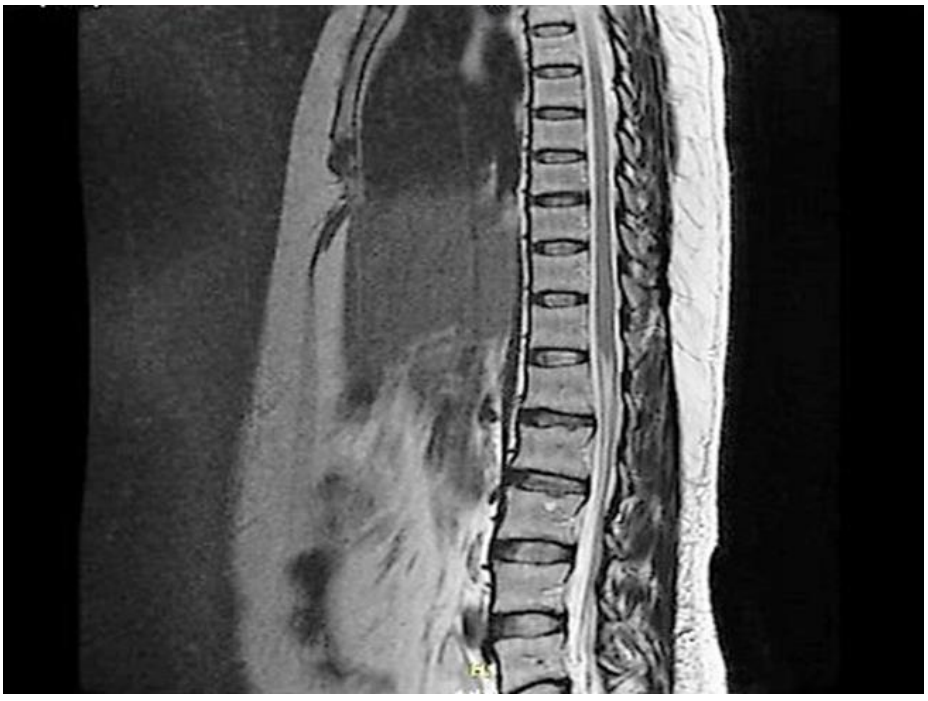

Image 2: T2 sagittal images showing hyperintensity of the conus and epi conus

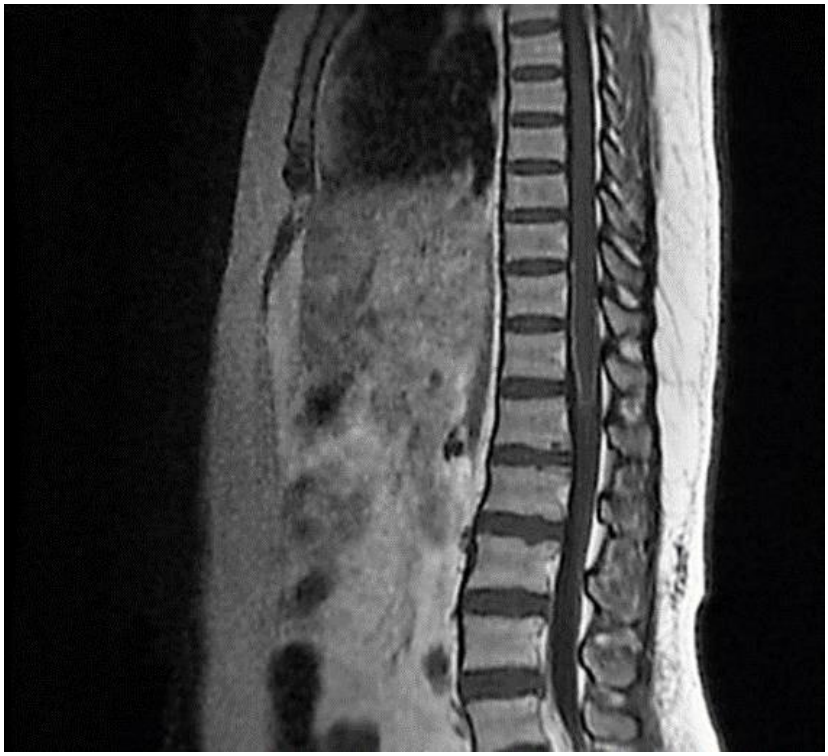

Image 3: Contrast T1 images showing enhancement of the epi-conus -conus.

Discussion.

Cauda equina and conus medullaris syndromes are very rare and can co -exist together. The estimated prevalence is 1 in 30,000 to 100,000 people per year. [4] Annual incidence ranges between 1.5 to 3.4 per million people. [4]

Lesions of the conus medullaris can have a combination of upper and lower motor neuron signs. Our patient had isolated conus medullaris syndrome; hence the neurological deficit was bilateral and symmetrical. Patients can have upper motor neuron signs like hyperreflexia and upgoing Babinski. Bladder dysfunction occurs in 92% of conus medullaris lesions followed by bowel dysfunction (occurring in 72% patients). Conus medullaris can be affected by HIV- related myelopathy, transverse myelitis, multiple sclerosis, syringomyelia, spinal cord arteriovenous malformations. [4]

Our patient had transverse myelitis involving conus medullaris (conus myelitis). She had a sensory level at T3, which was a false localizing sign because of cord edema. Longitudinal extensive transverse myelitis (LETM) (involvement of 3 spinal cord segments has been reported with covid 19 infection [5]. Number of mechanisms have been postulated, namely

a) During active phase, direct invasion of central nervous system through blood circulation and also through the olfactory bulb. b) acute covid can present as viral encephalitis. [6] c) other than immune mediated and hypoxic pathways can induce neuronal injury [6].

Covid 19 can trigger an autoimmune response which may be responsible for pathogenesis of Guillain Barre associated like picture and transverse myelitis [7]. Also, SARS-CoV-2 virus has strong affinity for ACE2 receptors in the central nervous system which triggers a systemic inflammatory response through pro inflammatory cytokines like interleukin 6. This results in increase permeability of the blood brain barrier and there is also immune mediated CNS inflammation. [8] There is an increase in acute phase proteins like CRP and fibrinogen [8]

In our patient ferritin and interleukin 6 at admission were not high. Patient only had mild self-limiting respiratory symptoms. It seems that in our patient the transverse myelitis is most likely immune mediated rather than direct CNS invasion of SARS- COV virus. Rapid improvement of symptoms with steroids further supports this. There were no visual symptoms such as eye pain or vision loss that are suggestive of multiple

sclerosis or neuromyelitis Optica. Furthermore, CSF was negative for immunoglobulin G autoantibodies or oligoclonal bands that are the hallmark of these diseases [9]

Similar cases have been reported in literature. Example a 28-year-old female patient who presented with features of ATM after COVID-19 infection and she had marked improvement in symptoms after receiving intravenous corticosteroids and plasma exchange. [10]

CONCLUSION

Covid 19 patients can develop serious neurological complications after initial recovery from novel corona virus and these complications are most likely immune mediated. Any patient presenting with bladder dysfunction in the setting of recent covid infection should alert physicians to this complication. To our knowledge this is the first case of conus myelitis following covid 19 infection reported in literature.

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