Injury (AKI) in COVID-19 is unclear. The pathophysiology of acute kidney injury is associated with poor outcome.

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Sir,

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The AKI in both patients was non-oliguric, urine analysis unremarkable, the respiratory distress improved over three days (Figure 1). The respiratory distress was stopped and renal functions showing improvement in four days. The patients favipiravir dose as in patient one along with enoxaparin 40 mg subcutaneous for 10 days and favipiravir at 1600 mg daily. The favipiravir was stopped, creatinine in creatinine with good urine output increasing 48 hours after favipiravir showing a progressive decreasing trend in creatinine with good urine output.

The favipiravir was stopped, creatinine within a period of one year and 2 months, SARS-CoV2.
There was no sensory deficit. Upon investigations, the reverse-transcriptase polymerase chain-reaction (RT-PCR) of nasopharyngeal swabs identified SARS-CoV2. Interleukin-6 (IL-6) was 11.37 pg/ml; C-reactive protein 98.1; D-Dimer 1.76 mcg/ml; ferritin 702 ng/ml; lactate dehydrogenase (LDH) 243 U/L. High resolution computed tomography (HRCT) of thorax showed ground-glass opacity in posterior segment of right lower lobe. Gadolinium enhanced magnetic resonance imaging (MRI) of spine demonstrated patchy, heterogeneous T2/STIR hyperintensity in the anterior aspect of conus medullaris extending from D11 to the lower endplate of L1 vertebral body, consistent with Conus Myelitis (Figure 1). Cerebral MRI was normal. Blood for anti-Aquaporin-4 antibody, myelin-oligodendrocyte glycoprotein (MOG) antibody, anti-nuclear antibody (ANA), SSA-RO (soluble substance A) IgG, SS-B-La (soluble substance B) IgG were negative. Angiotensin converting enzyme (ACE) was 16.64 U/L (N: 12-68). CSF analysis revealed cell count 9/ cu mm (10% polymorphs, 90% lymphocytes), glucose 55 mg/dl and protein elevated to 120 mg/dl.

Based upon the clinico-radiological findings coupled with temporal association with Covid-19 infection established the diagnosis in our case of “Covid-19 associated Conus Myelitis.” The patient received a 5-day course of intra-venous pulse methylprednisolone (1G/day) followed by tapering dose of oral steroids. The neurological deficits recovered over the next one month.

In Conus myelitis the inflammatory lesion is strictly confined to conus-epiconus region of spinal cord. The characteristic clinical manifestations include sphincter dysfunction and symmetrical or asymmetrical motor weakness in legs restricted to L4, L5 and sacral segments. Sensory deficit is limited to the sacral dermatomes but is often absent. Covid-19 related myelitis is an immune-mediated disorder. SARS-COV2 binds strongly to the ACE2 receptors of spinal cord neurons and adjacent vascular endothelium. This generates exaggerated immune-mediated reaction leading to acute myelitis.

References