

COVID-19 Associated Neuroimaging Findings

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Since the first detection of Corona virus in December 2019, there had been a rapid increase in the number of cases, progressing to a pandemic state. Although respiratory system involvement is most well-known, the central nervous system may also get affected. The etiopathogenesis may be related to the following:

- Direct neural transmission via the olfactory pathway.
- Hematogenous spread.
- Angiotensin converting enzyme 2 receptor.
- Hypoxemia.

Neurological symptoms may be vague especially in the elderly with multiple comorbidities and in children. These include altered mental status, headache, anosmia, cerebellar ataxia, seizures, hemiparesis, and syncope. Encephalitis or a Guillain-Barré syndrome like presentation may occur.

CT (computed tomography) and MRI (Magnetic resonance imaging) are the most common imaging modalities used. Medial temporal lobe has been shown to be a common site of involvement. Confluent or non-confluent white matter T2 and FLAIR hyperintensities with or without myelitis may occur. The findings in adults may include any of the following:

- Acute disseminated encephalomyelitis (ADEM)
- Myelitis
- Infarcts
- Transient splenic lesions

- Intracranial haemorrhage
- Acute haemorrhagic necrotizing encephalopathy.

What is interesting to note is that neurological manifestations of COVID-19 infection may occur in the absence of any respiratory symptoms. Another finding is that CSF analysis may be unremarkable in many patients.

Both the above-mentioned findings have been true in my experience. The common CNS findings seen in recent times include infarcts, hemorrhage and two cases of encephalomyelitis with multifocal, non-enhancing and non-restricting demyelinating lesions in cerebral and cerebellar white matter and in the cervico-thoracic spinal cord.

There have been increasing number of reported cases of children developing systemic inflammatory response (pediatric multisystem inflammatory syndrome temporally associated with COVID-19). A further group of children with a far less severe, Kawasaki-like disease, who respond to a variety of immunomodulatory treatments has been identified. A toxic shock syndrome like presentation may occur. Despite the typically mild acute infection, children may be at high risk of a secondary inflammatory syndrome.

Since majority of pediatric CNS cases are of asymptomatic to mild degree, clinicians need to be alert. The neuroimaging findings in children may include:

- Acute infarcts
- Acute necrotizing encephalopathy

- Diffuse cerebral edema.

The differential diagnosis may include other viral encephalitis- Herpes, Varicella, HIV associated diseases, Flavivirus, influenza, and enterovirus. A thorough clinical and laboratory investigation is needed to rule out other causes of overlapping presentations such as meningitis, electrolyte disturbance related myelinolysis and opportunistic infections related to immunocompromised state. Previous history of infection, malignancy and treatment and drug history is required. Immunization history in children is important. CSF analysis for cytology and culture should be done to rule out alternative plausible causes. Other causes of infarcts like arterial or venous thrombosis may have to be ruled out. RT- PCR test should be carried out in patients after the basic set of investigations even if chest imaging is normal.

Hence a high index of suspicion is required for the diagnosis and to prevent morbidity and mortality [1-9].

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