

## Neurology in COVID-19 Era !

In December 2019, a new disease called novel coronavirus disease, or COVID-19, was spreading around Wuhan, China, and had become a worldwide pandemic.

Although pneumonia-like symptoms predominate, many patients are still at risk of having neurological manifestations at acute presentation, such as stroke, Guillain-Barre Syndrome, epilepsy, and encephalopathy.

In many patients with COVID-19, intensive care unit (ICU) treatment and long-term ventilatory support are required, increasing the risk of critical illness myopathy (CIM) and polyneuropathy (CIN).

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## How To Diagnose CIN/CIM ? ( Clinical and Neurophysiological Criteria )

The patient is critically ill and has limb weakness or difficulty weaning from the ventilator after non-neuromuscular causes such as heart and lung disease have been excluded.

For CIN, evidence of axonal motor and sensory polyneuropathy in nerve conduction studies and for CIM, needle electromyography of short-duration, low-amplitude motor unit potentials.



## How Much COVID-19 related CIN/CIM Is There ?

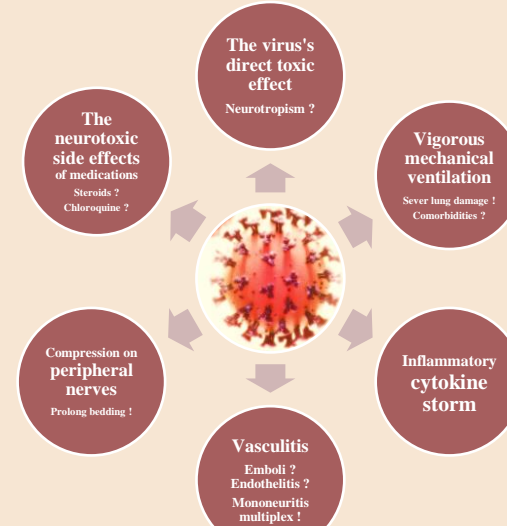
<b>Martinez et al. 2020</b> Case series	12 COVID-19 cases with ICU acquired weakness → 4 had CIN and 7 had CIM
<b>Frithiof et al. 2021</b> Cohort study	<ul style="list-style-type: none"> <li>111 COVID-19 ICU patients → 14 with weakness → 11 (10%) had CIN (7) / CIM (4)</li> <li>10 non-COVID ICU weakness → (7) CIM / (Zero) CIN</li> </ul>
<b>Bocci et al. 2021</b> Case series	8 COVID-19 cases with ICU acquired weakness → (6) had CIN
<b>Pérez et al 2021</b> Cross sectional	22 COVID-19 ICU patients with weakness → (17) had CIM, (4) had CIN, (12) had focal neuropathies
<b>Bagnato et al. 2021</b> Cross sectional	21 COVID-19 ICU patients with weakness → (5) had both CIM and CIN, (4) had CIN and (4) had CIM
<b>Hasrat et al. 2022</b> Cross sectional	11 Survivors COVID-19 ICU patients with weakness → (3) had CIN (Axonal Motor Sensory Polyneuropathy)

The discrepancy between the results (CIN? vs. CIM?) may be due to the duration of hospitalization, different therapeutic approaches and disease severity, varied ages, and small sample sizes.

## What Is Specific To COVID-19?

CIN/CIM usually occurs as a result of multiple organ failure, muscle immobility, corticosteroids, and neuromuscular blocking medications.

**What are the possible COVID-19 mechanisms ?**



## Is There Any Potential Biomarker ?

High neurofilament light chain (NfL) and glial fibrillary acidic protein (GFAP) levels were detected in COVID-19 patients who later developed CIM or CIN, in contrast to a previous studies suggesting that diagnosis of non COVID-19 ICU-acquired weakness is not possible using plasma NfL levels.

There is a correlation between the impairment of neurographic parameters (amplitude) with NfL and GFAP levels, thus suggesting CIN may be a consequence of COVID-19 itself rather than an effect of the severe illness. (Neurotropism?).

Elevated interleukin-6 at admission is a risk-predictor biomarker for CIN developing in COVID-19 (Cytokine storm theory?).

Skeletal muscle biopsies in CIM with COVID-19 patients showed non-specific changes such as degenerative-regenerative process with muscle atrophy, and preferential myosin loss.

## Why Is the Diagnosis of CIN/CIM Important in COVID-19 Circumstances?

One of the medical challenges in treating COVID-19 patients is the high number of patients requiring prolonged mechanical ventilation and unusually high sedation requirements, which predisposes to ICU associated weakness as well as the development of CIN/CIM can lead to prolonged hospital stay, delayed weaning from the ventilator, and an unfavourable outcome.

CIN and CIM are important to identify since survivors often present with severe residual disability and persistent exercise limitations several years afterwards.

There is a clear distinction between the outcomes of CIN versus CIM, where patients with CIN have a slower or incomplete recovery and a higher mortality rate, whereas patients with CIM often show complete recovery within 6 months.