COVID-19 infections may lead to neurologic complications

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COVID-19 may affect brain function in some people who become infected. Neurological symptoms can present at any time during the illness or even after infection has been definitively diagnosed.[1] These symptoms may be seen in mild forms of the illness but seem to be more common in severe cases.[2] Less severe neurological symptoms include loss of sense of smell and taste, headaches, fatigue, dizziness, and muscle ache/pains (myalgia).[2] See the table below for how severe symptoms may be used to help guide clinicians in their neurological workup.

Table: COVID-19-related neurological phenotypes and associated tests

Neurological phenotypes	Symptoms, findings, and associated conditions	Related testing
Encephalopathies/ Neuroinflammatory syndromes	Encephalitis—delirium, psychosis, confusion, disorientation, acute demyelinating encephalomyelitis, seizures, myelitis, numbness, various CT and/or MRI findings suggestive of other neuroinflammatory illnesses	• Encephalitis Antibody Evaluation with Reflex to Titer and Line Blot, Serum • NMDA Receptor (NR1-subunit) Autoantibody • Autoimmune Neurology Antibody Comprehensive Panel with Reflexes, Serum
Peripheral nervous system	Guillain-Barré Syndrome—weakness and tingling in the feet and legs or paralysis, muscle weakness, difficulty moving eyes or face, chewing, talking or swallowing	• Sensory Neuropathy Antibody Panel (Ganglioside) • Sensory-Motor Neuropathy Complete Antibody Panel • SensoriMotor Neuropathy Profile with Recombx®-Complete • Sensory-Motor Neuropathy Antibody Panel (Ganglioside) • Sensory Neuropathy Complete Antibody Panel
Stroke	Hypercoagulability, increased D-dimer, pulmonary embolism, CT or MRI evidence of a thrombus	• D-Dimer, Quantitative • Prothrombin Fragment 1.2 • CardioIQ® Fibrinogen Antigen, Nephelometry • Plasminogen Activator Inhibitor-1 (PAI-1) • Protein C and S Activity with Reflex to Protein C and/or S Antigen

Who is at increased risk?

Older patients with severe disease and patients of any age with a history of obesity, hypertension, ischemic or hemorrhagic stroke, dementia, or autoimmune conditions.

What can I do as a clinician?

Monitor patients' risk for coagulopathies (PT, aPTT, D-dimer, fibrinogen, ADAMTS13, APOE).

Conduct symptom-driven neuroimmunologic evaluation and monitoring with appropriate laboratory testing, neuroimaging studies, and physical and mental assessments.

How can clinicians incorporate this information into patient care?

Blood- or CSF-based biomarker assessments (AChr, NMDAr, titin, RyR, Musk, MOG, NfL, etc) may assist with the differential diagnosis of a patient's presenting/emerging neurologic symptoms and inform appropriate clinical interventions and risk mitigation.

Evidence shows that patients with neurologic complications from COVID-19 usually lack typical autoantibody signatures in serum and CSF (except for NfL), but do show increased levels of CSF proteins, lactate, or white blood cells indicative of autoimmune encephalitis. Additionally, antineuronal autoantibodies in the CSF (IgG) have been identified that are not normally included in standard assays.[3]

For more information, go to **QuestDiagnostics.com/testdirectory**

References

1. Paterson RW, Brown RL, Benjamin L, et al. The emerging spectrum of COVID-19 neurology: clinical, radiological and laboratory findings. *Brain*. ePub ahead of print. July 8, 2020. doi:10.1093/brain/awaa240/5868408
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3. Franke C, Ferse C, Kreye J, et al. High frequency of cerebrospinal fluid autoantibodies in COVID-19 patients with neurological symptoms. *medRxiv*. ePub ahead of print. July 6, 2020. doi:10.1101/2020.07.01.20143214