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Mini Review

Neurological Manifestations Related To Covid 19

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Abstract

The clinical manifestations related to sars-COV-2 Coronavirus infection appear to be very polymorphic and multi-systemic, far beyond the typical nosological framework described (respiratory picture: fever, cough, asthenia and respiratory discomfort). Currently, neurological damage is more and more frequent, sometimes inaugural and complex. Through a literature review, we focused on the central and peripheral nervous system manifestations of COVID-19 disease.

Keywords: *Neurological Manifestations*

Introduction

Since December 2019, an epidemic attributed to a new coronavirus has appeared in China (Wuhan). This new virus causes a respiratory symptomatology similar to the coronavirus identified in 2003, called SARS-CoV. The two viruses also have sequence similarity and also act on the same target, the angiotensin-converting enzyme 2 receptor (ACE2). As a result, the virus was named SARS-CoV-2 and in February 2020, the World Health Organization named this disease coronavirus disease 2019 (COVID-19) [1]. The clinical manifestations related to sars-COV-2 Coronavirus infection appear to be very polymorphic and multi-systemic, far beyond the typical nosological framework described (respiratory picture: fever, cough, asthenia and respiratory discomfort). Currently, neurological damage is more and more frequent, sometimes inaugural and complex. They can involve the central and peripheral nervous systems [2]. If anosmia and dysgeusia were initially the best known, other neurological disorders of varying severity have come to reinforce the neurotropism of SARS-COV-2. And, we will carry out a synthesis of the main neurological clinical pictures reported to date in the literature in order to deepen the knowledge of this disease.

Pathophysiology of neurological damage

The coronavirus can spread from the respiratory tract to the nervous system. Neuroinvasion requires interaction with specialized structures, such as the blood-brain barrier (BBB), consisting in particular of a monolayer of endothelial cells; or peripheral synapses that regulate CNS access to molecules, cells or pathogens that may have toxic effects [3,4]. Viruses reach the CNS through hematogenous or neural propagation. Nerve dissemination is by olfactory means, by the polarization of neurons. This property gives them the ability to receive and transfer information. The olfactory pathway begins in bipolar cells located in the olfactory epithelium. Its axons and dendrites extend to the olfactory bulb, where they synapse with the cells of the olfactory bulb. Then the olfactory nerve and divides into two branches towards the olfactory nucleus present in the pyriform cortex. This transport can be retrograde or anterograde and is facilitated by proteins called dynein and kinesin, which can be virus targets. Once entered the CNS, viruses can generate alterations in neurons and their functioning [5-7].

Neurological damage can not only be related to the neurotropism of the virus but also secondary to vascular lesions such as blood hypercoagulability, endothelial lesions, capillary vasoconstriction and BBB permeability. One can also have an immune mechanism related to the cytokine storm, the hyperinflammatory state and the presence of autoantibodies. Other explanations are lesions secondary to disorders of homeostasis, metabolic [2].

Central events

CNS involvement can be inaugural, complex and polymorphic about 34.8% - 56.3% of all neurological disorders [8].

Adult encephalopathies are the most frequent, which mainly affect female genders in their sixties, with comorbidities (obesity, ethylism, cardiovascular risk factor). Usually appears around the twentieth days of SARS-COV-2 infection. Encephalopathic signs can be associated with focal (sensory or motor) or pyramidal deficit, damage to the cranial nerves [9]. The PCR test by nasopharyngeal swab is positive and negative for other viruses (HSV, VZV, Nile, shingles...). Brain MRI shows abnormalities of signals at temporal levels, cerebral parenchymas, micro-bleeding. The EEG exam shows non-specific slowdowns and sometimes non-critical focal anomalies. They can be hypoxic encephalopathy, hypoxia (decreased PaO2) with laminar necrosis on MRI or necrotizing hemorrhagic encephalopathy by temporal lobe hemorrhages, subsular on MRI [8].

Encephalitis mainly affects young people, most often in their fifties, male gender with comorbidities (epilepsy, high blood pressure, stroke). It appears in the first week of the disease either in moderate or severe forms. The clinical presentation is marked by pneumonia, acute respiratory distress syndrome, coma and status epileptic disease. A biological inflammatory syndrome is very marked (elevation of CRP, VSH, D-dimers and lymphocytopenia) [2,8]. The PCR test (nasopharyngeal swab) is negative on the other hand positive in CSF with altered cells, hyperproteinorachia, pleiocytosis. The EEG shows a slowed pathological rhythm, microvolted, or epileptiform abnormalities. On imaging, the brain scan is normal but the chest scan shows an image of pneumonia. A hyposignal along the wall of the lower horn of the right lateral ventricle, and/or atrophy of the mesiotemporal region, or the hippocampus on MRI [7,9].

Acute myelitis in a patient with COVID 19 affects the sixty-year-old. It is preceded or associated with fever, aches and acute or subacute bilateral and symmetrical fever, aches and paralysis. The PCR test by nasopharyngeal swab is positive and a biological inflammatory syndrome (CRP, serum ferritin, VSH). MRI shows a spinal hypersignal depending on the course of the disease [2].

Stroke, typically by obstruction of the sylvian artery (ischemic stroke) can be inaugural or a complication of COVID-19. Often a man in his sixties with high cardiovascular risks having a severe form that has a pyramidal type deficit of a hemibody around the first week. The PCR test (nasopharyngeal swab) is positive and brain imaging gives the anatomical diagnosis. Other less common clinical forms appear as deep-localized hemorrhagic stroke or transient ischemic attack or cerebral thrombophlebitis [9,10].

Insomnia is a common neurological manifestation, in the mild or severe form of COVID-19. Often a young man, who can exist from the beginning of the disease and fluctuate up to 7 months in post-COVID. It is manifested by a fragmentation of sleep, irruptions of nightmares or hypersomnia. Insomnia is either referred to as a psychiatric symptom (depression, post-COVID anxiety) or neurological impairment of a sleep regulation center [11].

Peripheral events

Cranial nerve damage is common during COVID-19. Olfactory nerve (I) mononeuropathy is the mode of discovery of COVID-19. It can appear at the beginning of the disease and persists a few months later. The involvement ranging from hyposmia to anosmia. Damage to the olfactory nerve may be associated with damage to the taste nerve (VII). The PCR test (nasopharyngeal swab) is positive at the beginning of the olfactory and/or gustatory nerve damage. The evolution is marked by a spontaneous improvement between 3 to 60 days [2, 8].

Other peripheral nerve disorders, mononeneuropathy of the nerves (VII, VIII, III), multineuritis or multiple mononeuropathies (bilateral VII with oculomotor nerve damage) [9].

Guillain Barré syndrome is also a peripheral manifestation of COVID-19. It often affects a young man and appears around 30 days of COVID-19, a muscle weakness that rises in a few days. On clinical examination, a flaccid muscle deficit, predominantly proximal associated or not with a distal superficial sensitivity disorder. Osteotendinous reflexes (ROT) are abolished. The ENMG or electro-neuromyogram: confirms demyelination by lowering the conduction rate, distal latency elongation, the presence of motor conduction block; associated with albumino-cytological dissociation with examination of cerebrospinal fluid. Without treatment, the course is unfavorable by respiratory damage and death; with correct treatment, the evolution is favorable in the majority of cases [2].

Inflammatory myopathy may also be encountered during COVID-19. It is manifested by myalgia, sometimes muscle weakness during infection. On neurological examination, a predominantly distal muscle deficit, with or without amyotrophies and osteotendinous reflexes are present. The ENMG shows normal nerve conduction, exertion activities in favor of a myogenic trace at detection. Biological examination shows an elevation of CPK, CRP and VSH. With treatment, the course is favorable [8,9].

Conclusion

Neurological manifestations related to COVID-19 are the most common extrapulmonary manifestations. The pathophysiological mechanism is explained by virus neurotropism, from hematogenous dissemination via the nasal epithelium by olfactory receptor neurons and secondary to multi-systemic manifestations (vascular, immune, metabolic...) and many mechanisms remain undetermined. The involvement of the central nervous system is dominated by encephalopathies, encephalitis, myelitis, stroke and insomnia while mononeuropathies, multineuritis, Guillain Barré syndrome are common to the peripheral nervous systems. Admittedly, these manifestations are still not exhaustive, hence the need for additional studies to have more knowledge on the neurological manifestations related to the COVID-19 disease.

Conflicts of Interest

Virtual neroscopy of the Malagasy Society of Neurology (SMNe) jointly with the National Academy of Medicine of Madagascar. (07.09.2021).

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