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# Cognitive damage after COVID-19 in a patient with Parkinson's disease and dementia: A case report

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***Abstract*** *— Introduction: The human disease associated with the new coronavirus, called COVID-19, was initially discovered in Wuhan, China. Although the existing literature on cognitive damage resulting from this injury is still very scarce, recent studies have shown dysfunction in the field of sustained attention, memory, verbal fluency and executive function, especially in patients with baseline neurocognitive fragility, such as Parkinson's disease (PD). These tend to have exacerbation of symptoms related to PD, in addition to strong psychological distress. Therefore, the aim of the present study is to report the case of a patient with PD and Dementia who evolved with cognitive decline during SARS-CoV-2 infection. Case Report: We present the case of a 63-year-old male patient with Parkinson's disease and dementia diagnosed 1 year before the condition, who presented significant cognitive decline during the SARS-CoV-2 infection. Discussion: The presentation of COVID-19 reported here consists of an atypical manifestation of the disease in a patient susceptible to cognitive damage due to the underlying dementia. It is believed that this pathology can cause patients with cognitive impairments that last from months to years after the infection, as observed in SARS-CoV and MERS-CoV infections. Conclusion: the effects of SARS-CoV-2 on cognitive function are gradually more apparent. Still, clinical trials in the short, medium and long term are essential to determine the main risk and vulnerability factors associated with cognitive impairment by COVID-19.*

## I. INTRODUCTION

The human disease associated with the new coronavirus (SARS-CoV-2), called COVID-19, was initially discovered in Wuhan, China. Such injury spread quickly to different continents, becoming a pandemic[1]. Although this pathology leads predominantly to symptoms of the respiratory system, different studies have reported neurological manifestations secondary to this infection, which affect at least 36% of patients, justifying the neurotropic potential of the virus [1,2].

Although the existing literature on cognitive impairment from COVID-19 is still very scarce, recent studies show dysfunction in the field of sustained attention, memory, verbal fluency, and executive function[3-5]. Since the virus was recently discovered, there are obviously no long-term studies that demonstrate its chronic consequences in individuals who have recovered from the disease. Even so, short and medium term neurological deficits are already observed in surviving patients of COVID-19[6].

Lesions of the Central Nervous System (CNS) resulting from infection with the new coronavirus are associated with three possible mechanisms: cerebral hypoxia resulting from breathing difficulties, vascular changes resulting from the state of hypercoagulability, or direct attack of the virus on the CNS through direct transport to brain tissue by breaking down the blood-brain barrier or through the mucous membranes of the nasal cavity[6-10]. Each of these factors can trigger brain damage, affecting patients cognitive ability.

The impact of the COVID-19 pandemic on the physical and mental well-being of the world's population is indisputable. However, this repercussion tends to be experienced more intensely in patients with chronic diseases, such as Parkinson's disease (PD)[11]. Although there are no reports regarding the greater probability of infection in PD patients when compared to the general population, it is known that these, when affected by SARS-CoV2, will experience worsening of their symptoms, especially greater psychological suffering, which in turn this time worsens motor symptoms and neuropsychiatric symptoms, such as: anxiety, lack of concentration, and lower scores on their cognitive skills[11]. Thus, the objective of the present study is to report the case of a patient with Parkinson's Disease and Dementia who started a cognitive decline during the SARS-CoV-2 infection, and through this, proposes a provocative discussion regarding the main evidence available in the literature regarding cognitive decline and COVID-19.

## II. CASE REPORT

FASM, male, white, 63 years old, married, born and resident of the city of Rio de Janeiro. He started to lower his level of consciousness with lethargy and headache, exacerbation of unmotivated laughter, too much anxiety, emotional lability, visuospatial dysfunction, executive dysfunction, constructive dysfunction, dysnomy, in addition to impaired recent and past memory, requiring help for walking and feeding, without any respiratory complaints or signs of infection. Previous history of Parkinson-Demencia complex diagnosed a year ago with significant speech impairment, undergoing multidisciplinary treatment with speech therapists, physiotherapists, psychologist and neurologist. Recent neuropsychological report provided by the psychologist shows that the patient had gradual and sustained improvement in attention, executive function, and episodic memory, up to 15 days before the onset of symptoms. However, after the onset of the condition, he presented a worsening in the performance of tasks and activities, severe attentional difficulty, as well as in the understanding of the commands, functions hitherto performed with some ease. After the patient's cognitive decline, his wife started suffering from the Flu Syndrome and then the suspicion of COVID-19 was started as a triggering factor of the condition now presented by the patient. The PCR performed on 26/10/2020 on the patient's nasal SWAB sample detected genetic material compatible with SARS-CoV-2, and a serology performed on 11/09/2020 revealed IgM and IgG reagents. There was no need for hospitalization or mechanics. Using Prolopa BD 25 mg, Prolopa HBS 200 mg, Alois 10 mg, Brintellix 10 mg, QueraLP 0.375 mg, Cronobê 1 ampoule per month, Proians 1 capsule per month and Extima 1 sachet per month.

## III. DISCUSSION

The presentation of COVID-19 reported here consists of an atypical manifestation of the disease in a patient susceptible to cognitive damage due to the underlying dementia. Such presentation is similar to previous reports of COVID-19 in patients with previous dementia. The aspect of SARS-CoV-2 infection in patients with dementia consists of changes in mental status, such as: agitation, confusion, disorientation, hyporexia and refusal of care[12-14].

Although the existing literature on cognitive damage from SARS-CoV-2 infection is still scarce, much can be learned from similar viral injuries. As observed in infections with other coronaviruses, SARS-CoV and MERS-CoV, it is believed that survivors of COVID-19

can manifest neurological damage from months to years after infection[15,16]. A study carried out in Chicago, demonstrated that 24% of the participants hospitalized by COVID-19 had short-term memory loss[17]. In addition to these, 6 patients from a UK cohort had neurocognitive disorder[18].

A detailed review of the literature revealed a report showing a prevalence of dysexecutive syndrome in 36% of 39 patients with COVID-1919 (10). In addition to this, several articles allude to confusion and difficulty in attention in infected patients, which also suggests disexecutive syndrome[19,20]. The dysexecutive syndrome is covered by deficits in attention, planning, abstraction, behavioral control and guidance, and is therefore compatible with the case presented here[21]. In both the acute and long-term phases, executive dysfunction can be predicted to be part of the neurological consequences of this viral infection[21].

More than a third of the participants in an observational study conducted in France showed cognitive impairment after discharge from the Intensive Care Unit (ICU), especially Disexecutive Syndrome[22]. In addition to this study, cognitive impairment was also observed in a series of 4 cases, and was manifested as memory deficit and frontal syndrome after discharge from the ICU[23]. Although the patient in this report did not need to be admitted to the ICU, symptoms similar to those described above were observed, which corroborates that such conditions do not result only from the treatment conditions of COVID-19, but from the evolution of the disease itself.

Hypoxemia resulting from lung injury caused by SARS-CoV-2 probably contributes indirectly to neuronal injury and underlying cognitive decline[6]. Although patients with COVID-19 pneumonia have relatively well-preserved pulmonary mechanics, they can exhibit severe hypoxemia, and subsequent neurological changes[6,7]. In addition, the hypercoagulable and hyperinflammatory state observed in COVID-19 contributes to delirium and cognitive decline, given that both inflammation and coagulopathy are independent risk factors for delirium[6-8]. It is worth noting that the neuroinflammation resulting from the rupture of the blood-brain barrier can facilitate delirium in the short term, in addition to severe cognitive deficits in the long term[10].

Delirium consists of cytokine-mediated activation of microglia and astrocytes, associated with acute brain dysfunction[24]. It is characterized by hyperactive, hypoactive or mixed states. The hyperactive state is characterized by anxious or agitated behavior with increased psychomotor activity, while the hypoactive state presents as depression or decreased psychomotor activity[24,25]. Delirium can trigger fluctuations in attention (focused, sustained or displaced) and awareness (orientation) and can also involve cognitive disorders[25].

An Egyptian study carried out in 38 patients with Parkinson's Disease (PD) showed higher levels of stress and lack of concentration during the pandemic in these patients when compared to previously healthy ones, emphasizing that the stress burden related to COVID-19 increased the severity of symptoms in these patients and this effect was mediated by the degree of psychological distress[11]. Therefore, patients with PD, when affected by SARS-CoV2, experience greater psychological suffering, and worsening of motor and neuropsychiatric symptoms, such as: anxiety, lack of concentration, and lower scores on their cognitive skills[11].

The risk factors for a worse prognosis associated with infection by the SARS-CoV-2 virus involve advanced age, the existence of chronic diseases, and smoking[26-28]. Likewise, these factors are associated with an increased risk of cognitive decline[29]. All of these associated factors correspond to baseline neurocognitive frailty, which increases patients' susceptibility to cognitive complications both during injury and after the hyperinflammatory state[30]. Thus, the individuals most susceptible to severe infection by COVID-19 also represent the population most susceptible to cognitive decline in the context of COVID-19 inflammation.

## IV. CONCLUSION

Although the effects of SARS-CoV-2 on cognitive function are increasingly apparent, neurocognitive syndromes as a consequence of COVID-19 have not yet received adequate attention. However, in view of the above, it is believed that it is highly likely that such injury may manifest itself with deficits in attention, memory and executive function, as well as psychomotor or visuospatial performance. Thus, clinical trials that follow in the short, medium and long term, the cognitive function of patients infected with beta corona virus are essential to determine the main risk and vulnerability factors associated, as well as identify ways to mitigate the potential negative effects in the cognitive functioning of these patients.

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