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Post COVID-19 Parkinsonism: A Case Report

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1. Abstract

1.1. Background: Parkinsonism is a syndrome that is characterized by a reduction in dopamine and basal ganglia functioning. Its etiology can be primary, secondary, atypical, or idiopathic. The primary clinical manifestations include rigidity, bradykinesia, resting tremor, and postural instability. The diagnosis is clinical and treatment must be individualized. Within this context, the present case is important since it involves a phenomenon that needs to be explored in depth: parkinsonism after COVID-19 infection.

1.2. Case Presentation: We report the case of a 58-year-old man diagnosed with parkinsonism, a condition triggered 20 days after starting treatment for COVID-19 infection (about 6 months ago). After clinical worsening as a result of psychiatric therapy and an unremarkable head MRI, we requested a neurological positron emission tomography (PET)/CT scan, which revealed a pattern of global cortical hypometabolism with a discrete bilateral superior frontoparietal predominance. Improvement in symptoms was observed 30 days after the beginning of treatment.

1.3. Conclusion: Much still needs to be explored regarding the agent that caused the pandemic in 2020, as COVID-19 infection has led to different short-, medium- and long-term sequelae. Further studies in this area are therefore necessary in order to encourage new discussion.

2. Introduction

Parkinsonism is defined as a syndrome in which two or more of the following cardinal symptoms are present: rigidity, bradykinesia, resting tremor, and postural instability [1-5]. Other possible clinical manifestations are akinesia, hypokinesia, and phenomenon of freezing [6, 7], in addition to shuffling gait, dysarthria, micrographia [2] and, rarely, ataxia [8]. Furthermore, the emergence of parkinsonian syndromes is possibly related to reduced basal ganglia functioning and a decrease in dopamine in the nigrostriatal tract, with subsequent changes in the medial globus pallidus or subthalamic nucleus [7, 9]. Etiologically, the disease can be atypical and derive from neurodegenerative diseases, idiopathic, primary, or secondary to numerous causes such as drug use, intoxication, metabolic disorders, and infections [2, 10].

The diagnosis of parkinsonism is primarily clinical but individual laboratory tests and head magnetic resonance imaging (MRI) can be used to rule out other differential diagnoses [3]. Thus, treatment consists of removing the underlying cause, supported by the administration of levodopa, anticholinergic agents, dopaminergic agonists, catechol- ortho-methyltransferase inhibitors, amantadine, neuroprotective therapies, brain stimulation, or even surgical measures [2, 3].

We report here the case of a 58-year-old man who developed parkinsonism as a result of a COVID-19 infection after another possible history had been ruled out.

3. Case Presentation

A 58-year-old male patient, a lawyer, presented at our service in August 2021 accompanied by family members. The patient had a

history of COVID-19 in February of the same year. At the time, he had complaints of anosmia, ageusia, high fever, and clinical bronchopneumonia. The patient received specialized therapy but began to experience mental confusion, stiff gait, and tremors in his hands after 20 days of treatment. The patient was thus referred for psychiatric treatment. A head MRI was unremarkable. According to information from the family, the prescription of an antipsychotic and mood stabilizer worsened his clinical condition and the family thus decided to seek neurological assessment.

At our service, we performed neurological examination and the diagnosis was parkinsonism. A brain positron emission tomography (PET)/CT scan was thus requested, which demonstrated slight, diffuse enlargement of the cortical sulci, brain fissures and enlargement of the supratentorial ventricular system, without lobar predominance (Figures 1 and 2). We thus initiated neuropsychological, physiotherapeutic, and drug treatment for parkinsonism. Thirty days after the beginning of treatment, the patient exhibited significant improvement in symptoms.

According to the family, before COVID-19, the patient was not undergoing treatment for any pathology, did not use medications, and had a completely normal professional, family, and social life.

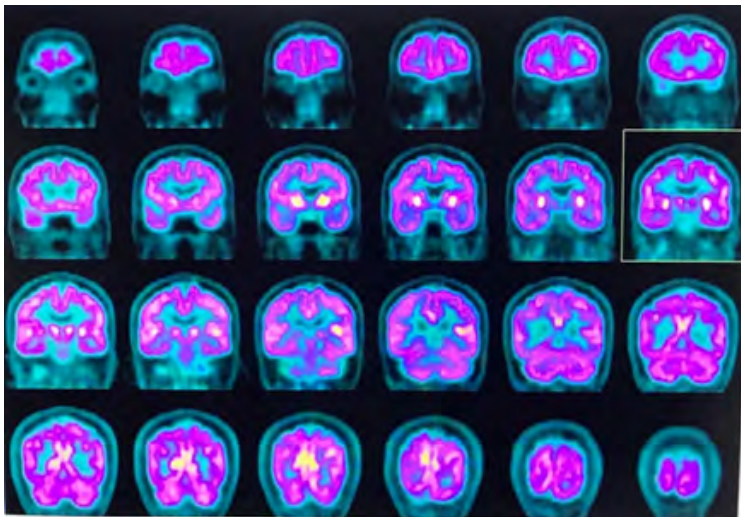

Figure 1: Neurological PET/CT coronal images.

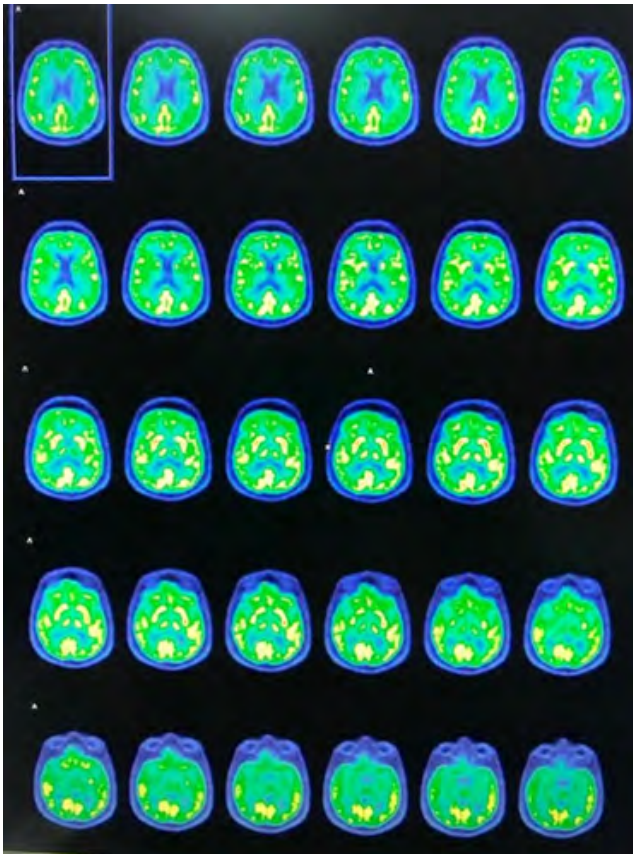

Figure 2: Neurological PET/CT axial images.

4. Discussion

In March 2020, the World Health Organization (WHO) declared the disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), known as COVID-19, a pandemic [11, 12]. Although the first findings mainly involved the respiratory tract, we have noted a progressive increase in neurological descriptions resulting from the disease [13].

The clinical presentation comprises different manifestations, including lesions in target organs, with changes in multiple systems such as the cardiovascular, gastrointestinal and neurological systems [14-16] and subsequent long-term consequences. These multiple sequelae affect a large percentage of individuals who contract COVID-19. One study demonstrated that 67.5% of patients who were not hospitalized and 59.7% of those who were had at least one post-COVID-19 symptom two years after acute infection [17]. Based on these findings, studies concluded that cognitive disorders could arise after COVID-19 infection, as well as changes in memory [18-20] and in the level of consciousness [15] and even movement disorders such as myoclonus-ataxia syndrome [21]. Other studies reported rarer findings such as Guillan- Barré syndrome, dementia, myasthenia gravis, and parkinsonian syndromes [11, 22-25]. Regarding the present case, we observed that the PET/CT scan was decisive in correlating the diagnosed parkinsonism with COVID-19 findings. Molecular imaging methods such as SPECT and PET are important since they are able to demonstrate decreases in the dopamine transporter (DAT) and in vesicular monoamine transporter type 2 (VMAT2), as well as in L-aromatic amino acid decarboxylase (L-AAAD) [26]. In addition to permitting the analysis of pre-synaptic dopaminergic functions, PET/CT can also detect conditions resulting from motor dysfunctions [27] and was therefore appropriate for the present patient.

The pattern of global cortical hypometabolism with discrete bilateral superior frontoparietal predominance detected on the images must be considered indeterminate because of its mild intensity. However, this pattern has been observed in cases of COVID- 19-related cognitive impairment. Some studies have shown neurocognitive impairment due to hypometabolism, especially in the frontoparietal region and in insular, subcortical, and limbic structures [28-30]. These findings are associated with the hyperinflammatory state, cognitive dysfunction, and reduced saturation seen in COVID- 19 [29]. Therefore, the mechanisms involved in the development of parkinsonism after COVID-19 include hypoxia-induced damage (in encephalopathy), damage to structures, and impairment of basal ganglia function. The already mentioned extensive inflammation may even lead to the discovery of initially asymptomatic Parkinson's disease [31].

Finally, considering the sequelae discussed above, we can confirm the development of parkinsonism secondary to COVID-19 infection in the present case since the patient was previously healthy and started to develop cognitive impairment and movement disorder only after exposure to the disease. This acute parkinsonism related to COVID-19, although rare [32], was confirmed by PET-CT imaging and elucidation of its mechanisms is increasingly necessary.

5. Conclusion

We conclude that the patient developed clinical signs and symptoms of parkinsonism as a result of COVID-19 infection. The mechanisms underlying the disease that caused the pandemic in early 2020 must still be explored, as well as its short-, medium- and long-term sequelae, in order to better understand the correlations between pathologies. Further studies in the area are therefore needed for better utilization of the results.

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