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RESEARCH ARTICLE

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COVID-19 AS A TRIGGER FOR DYSAUTONOMIC DISORDERS: A CASE REPORT

Rodrigo Silva de Brito^{1,*}, Marco Orsini², Jacqueline F. do Nascimento³, Nicolle Nunes³, Antonio Marcos da S. Catharino³, Marcos R.G. de Freitas⁴, Marco Antônio A. Azizi³, Thais de R. Bessa-Guerra³, Thiago R. Gonçalves³, Renata R. T. Castro³, Paulo Henrique de Moura³ and Adalgiza M. Moreno³

¹Universidade Federal Fluminense, Faculdade de Medicina, Niterói, RJ, Brasil

²Universidade Iguazu, Programa de Pós-Graduação em Neurologia e Neurociências da Universidade Federal Fluminense, RJ, Brasil

³Universidade Iguazu- UNIG, RJ, Brasil

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*Corresponding author:

Rodrigo Silva de Brito

ABSTRACT

The Covid-19 outbreak caused a joint movement of researchers from around the globe in a way never before seen in the history of modern science, with the purpose of elucidating the entire pathophysiology of the disease, caused by the Sars-Cov-2 virus, and its systemic alterations that go beyond the involvement of the respiratory system. There is now widespread interest in investigating a relationship between Sars-Cov-2 infection and the involvement of other systems, such as the central nervous system, on triggers for manifestations such as Guillain-Barré Syndrome, and dysautonomia. The present work reports the case of a 42-year-old physician, who in December 2020 started with symptoms of flu-like syndrome, later diagnosed with Covid-19 by RT-PCR, presented dysautonomic symptoms, with intense episodes of sweating without triggering factors physiological factors, which directly affected their quality of life.

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INTRODUCTION

MO, male, 41 years old, neurologist, high performance athlete, without pre-existing diseases, reports that on 12/30/2021 he developed a dry cough that lasted two days, which later evolved to myalgia and fever that was not measured, which persisted for 72 hours. He also claims hyposmia and hypogeusia on the fifth day after the onset of symptoms. Patient performed on 04/01/2021, RT-PCR exam for Sars-Cov-2 with detectable result. reports that during the manifestation of signs and symptoms of infection by Sars CoV2, he had started to present anarchic sweating alterations, regardless of the temperature of the places where he was. The episodes of sweating break out at varying times of the day and are unrelated to vital signs. "I feel myself sweating without usual triggering factors such as, for example, heat and/or stress."

Patient presents vital signs during clinical evaluation and physical examination: Axillary body temperature: 36.1°C; heart rate: 77 bpm; respiratory rate: 16 Irpm; sitting blood pressure: 120x80mmhg; orthostasis blood pressure: 118x80mmhg. Electrocardiogram: sinus rhythm, no changes, Exodopplecardiogram: No change; Chest computed tomography: no change. Complete Laboratory: Normal, including thyroid function, electrolytes, cortisol, among other markers. He states that eventually he has to change clothes 4-5 times a day due to intense sweating, directly affecting his quality of life. The condition is very localized on the trunk and lower limbs, rarely affecting the face (Fig. 1). During some episodes patient reports a sensation similar to faintness and alertness. "Sometimes, associated with these manifestations, a sensation of faintness and slowness of reasoning occurs - something as if the brain functions underwent a kind of reprogramming - download.



(Source: patient's personal collection)

Fig. 1. Episode of intense sweating recorded by the patient himself, affecting mainly the trunk regions

The picture lasts on average a few minutes, causing the patient to cease his activities and last for a few minutes in a cold bath. There is no specific reason that is a trigger for these manifestations during their daily activities. Questioned about anxiety, fear, and depression, he reaffirms that there is no such relationship. A tilt test was requested. Cardiac Evaluation: no changes. Neurological Examination: no changes.

DISCUSSION

There is still no means of stating precisely when the Sars-Cov-2 virus began infecting humans and being transmitted among their peers. However, it was in December 2019 that the disease, a pneumonia of undetermined etiology causing acute respiratory syndrome in many of the cases, had its first documented reports in Wuhan Province, China⁴. The disease, which aroused health interest worldwide, has advanced in a devastating way all over the globe, being declared a Covid-19 pandemic on March 11, 2020⁵. In Brazil alone, as of February 17, 2021, 9,921,981 cases of Covid-19 have already been reported, with a total of 240,940 deaths as a result of the disease⁶. Sars-cov-2 binds through the angiotensin-converting enzyme, ACE-2, which is abundantly expressed in the lung alveolar tissue, which explains the primarily respiratory involvement, but is also expressed in endothelial cells of several organs, justifying, in part, the extrapulmonary involvement in patients with Covid-19⁷. The disease may manifest itself severely, due to an exacerbation of the inflammatory response; autopsy findings show diffuse alveolar damage, in addition to an abundance of neutrophils, lymphocytes, monocytes, and macrophages, and thrombosis of the microcirculation⁸. Endothelial involvement has generated great interest in the academic community, especially due to the increased incidence of vascular and microvascular diseases, many of them of thrombotic origin, which, through direct viral action, generate endothelial dysfunction that leads to thrombotic phenomena such as activation of the coagulation cascade and increased activity of pro-inflammatory cytokines⁸. The virus also seems to have a predilection for cells of the nervous system, mainly due to high frequency neurological symptoms, such as headache, anosmia, ageusia, syncope, and myopathy, among others². Furthermore, the scientific literature has already described findings that prove the tropism of the new coronavirus also for the central nervous system, such as the detection of viral RNA in the cerebrospinal fluid by RT-PCR in a patient with encephalitis⁹. Another important finding is dysautonomia, a disorder in the autonomic nervous system that can manifest itself in multiple ways, such as exacerbated sweating, tachycardia, syncope, postural orthostatic tachycardia syndrome, etc¹⁰.

It is suggestive that Covid-19 affects small myelin fibers, however, a central origin of dysautonomia cannot be excluded. There is no evidence that the virus can cause direct damage to peripheral nerves, however, it is more likely that this effect is caused indirectly through a cytokine storm or due to a non-immunomediated mechanism. Autonomic dysfunction is a not very frequent event in Sars-Cov-2 infection, with a prevalence of 2.5% among Covid-19 cases recorded in the ALBACOVID study.

CONCLUSION

The pathophysiology of Covid-19 is becoming better understood by the scientific community; however, a complete elucidation of its mechanisms is still a distant prospect, especially in the current scenario where new variants are emerging and bringing with them even more questions. In this scenario of uncertainties, all signs and symptoms that make up the picture of the disease deserve to be investigated, even if in low prevalence. In this context are the dysautonomic phenomena, especially profuse sweating experienced by the patient in question, which lacks much information in the scientific literature. Considering the fact that the patient does not belong to any risk group for complications of Covid, this finding becomes even more relevant, since it is a manifestation that significantly alters his quality of life and interferes with his social activities, changing his daily life and affecting the patient months after healing from the acute Covid-19 condition.

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