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A Case Report: Viral Meningitis Secondary to Reactivation of HSV after the Second Dose of the Vaccine During SARS-CoV-2 Infection.

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Abstract:

Case Report: A 21-year-old female nursing student with no known co-morbidities and a recent history of HSV type 2 infection, as well as a previous history of viral meningitis, presented to the Acute Covid Assessment Unit (ACAU) with a 1-day history of frontal headache and severe photophobia, and a 1-day history of vomiting and mild myalgia. However, no respiratory symptoms are associated. On physical examination, she was alert, and oriented, with a Glasgow coma scale (GCS) of 15/15. She had a temperature of 37.5 C and no meningeal signs or skin rashes suggestive of meningitis. The patient tested positive for SARS-CoV-2 infection one week after receiving the second dose of the ®Pfizer vaccine (mRNA vaccine).

Introduction:

In 2019, a global health emergency was declared by The World Health Organization (WHO) (1).
After the outbreak of COVID-19 is the new public health pandemic threatening the world with the spreading of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) or the novel coronavirus (2019-nCOV) (1) (2). According to a retrospective case series study in Wuhan, China, several COVID-19 patients can manifest neurological symptoms as well as the usual presentation of respiratory symptoms (3).

COVID-19 is highly associated with common manifestation symptoms of mild to moderate respiratory symptoms such as non-productive dry cough, fever, and fatigue. However, in some cases, the respiratory presentation might be associated with neurological manifestations for example anosmia, hyposmia, headache, dysgeusia, encephalitis, meningitis, and cerebrovascular accident during the infectious period. Moreover, the neurological manifestation may be the initial manifestation of COVID-19 patients in rare cases. Additionally, patient may develop one or more neurological symptoms (1) (4) (5). Recent studies have shown that cases with severe COVID-19 infection experience more neurological manifestations as skeletal muscle damage, cerebrovascular accidents, meningitis, and altered level of consciousness (5). Li et al. suggested that the acute respiratory failure in COVID-19 patients can be due to direct damage to the lungs, as well as partial selective damage to the brain stem by the virus. Therefore, Li et al. raises the question of how the SARS-CoV-2 enters the brain (6) (7) (8).

Certain theories support how SARS-CoV-2 could affect the CNS. SARS-CoV-2 is one of the coronaviruses family which contains positive-sense single-stranded RNA viruses in the genome as well as on its surface a number of spike membrane glycoproteins (9). The similarity of genetic structures between SARS-CoV-2 and SARS-CoV is 79%, where the major function receptor is acted by the angiotensin-converting enzyme-2 (ACE2), and between SARS-CoV-2 and MERS-CoV the similarity is 50% (5) (10) (11). SARS CoV-2 attacks the ACE2 receptors which may be found on neurons, glial tissues, and brain vessels. Since ACE2 is a cardio-cerebral vascular protecting factor, its damage causes a leak of the virus in the CNS (5). Therefore, the mechanism of how the blood-brain-barrier (BBB) is prohibiting the virus from entering the brain is yet to

studied (6) (7).

On the other hand, herpes simplex virus type 2 (HSV-2) is a DNA virus that is a part of the neurotropic herpesvirus family (12). The formulation of latent infection occurs after primary infection. However, in the presence of immunosuppression, the virus could become active and involve multiple organs (cutaneous, kidney, liver, and brain) (12) (13).

Several studies performed in severe cases of COVID-19 infections suggest that HSV-2 reactivations are frequent as the severe forms of SARS-CoV-2 are associated with acquired forms of immunosuppression biological and/or clinical signs, for instance, lymphopenia (14) (15). As a result, viral reactivations are inclined to occur due to immunodeficiency. Furthermore, SARS-CoV-2 patients suffer from septic shock with the typical biological and/or clinical pictures (15). There are variable immunological aspects of patients with SARS-CoV-2. Severe cases might present with immunosuppression and cytokine storm syndrome (16), which indicates the existence of an irregular immune response and exhaustion of cytokines by attacking T lymphocytes (CD4 cells, CD8 cells, and NK cells) (17); this unbalanced response could explain the reactivation of latent viral infection such as HSV-2 and this could explain also the sudden worsening of symptoms during the recovery (16) (17).

The prevalence of infection with HSV-2 between adults is around 25% in the United States and between 4-18% in Western Europe (18). HSV is transmitted at the sub-clinical shedding phase (19) (20). Most patients with seropositive HSV-2 report no history of genital lesions (21) (15). The acquired infection transmission of HSV-2 is high among persons with no history of genital herpes infection (22). As a result, viral shedding is frequent in seropositive patients; in spite of having a history of genital herpes or not. Additionally, women might asymptomatically shed HSV-2 “internally” (cervix and vagina), and this can explain the undergoing unnoticed reactivations of infection (15).

Case Report:

A 21-years-old female nursing student with no known co-morbidities and a recent history of HSV- 2 DNA infection and viral meningitis was presented to the Acute Covid Assessment Unit (ACAU) for COVID-19 infection with a 1-day history of frontal headache and severe photophobia, and a 1-day history of vomiting and mild myalgia. She denied any cough, shortness of breath, neck stiffness, and diarrhea. Initially, the patient was known as COVID-19 positive after one week from receiving the second dose of the ®Pfizer vaccine. Due to her illness upon presentation, COVID-19 PCR was ordered which came back positive.

The patient was admitted for viral meningitis related to COVID-19 infection. On admission, her initial temperature was 37’8 C, respiratory rate was between 12 and 18, Oxygen saturation was 98% on Room Air, blood pressure was 130/72. She was awake, alert, and coherent. She followed commands well and was oriented to name, place, time, and situation. On examination, the patient was neurologically intact with a GCS of 15, normal cranial nerves, and no motor or sensory deficits, she had a normal tone, bulk, and strength. Additionally, negative meningeal signs Brudzinski and Kernig's and absence of meningeal rash. Her chemistry was within normal limits, as were her liver and renal function. ECG showed no acute ischemic changes and CK was within normal range. Her chest x-ray was clear. CT brain, without contrast, showed no acute intraparenchymal changes. On the other hand, lumbar puncture (LP) was done and her cerebral spinal fluid (CSF) analysis revealed 243 white blood cells with 96% mononuclear cells and 4% polymorphs. CSF red cells were 207. In addition, CSF virus screening was not detected for SARS-CoV-2; however, it was detected to HSV-2 DNA.

The patient was hospitalised for 5 days and had received supportive treatment (Paracetamol and IV fluids). Meanwhile, she showed good progress during the hospitalisation. She was symptoms-free on discharge.

Discussion

In our case, we report a case of a COVID-19 patient with reactivation of HSV-2 due to the patient’s status of immunosuppression associated with SARS-CoV-2 infection. In this case, the initial presenting symptoms seemed to be exclusive to meningitis, in spite of the patient received the second dose of the COVID-19 vaccine before she became infected with COVID-19. Meningitis is the inflammation of the coverings of the brain and spinal cord. A case of SARS-CoV-2 related meningitis /encephalitis has been reported in Japan (8), where a young patient presented with an altered level of consciousness and a single episode of seizures.

This case report draws to light the possibility of patients manifesting merely neurological symptoms without respiratory distress or severe respiratory illness. Nevertheless, the role of the COVID-19 vaccine in preventing immunosuppression in patients with latent infection and in providing immunity in complicated cases yet to be study.

In our case, the CSF showed positive results of HSV type 2 infection and negative results of SARS-CoV-2 from the same sample, which most likely indicates reactivation of latent infection with HSV-2 due to immunosuppression status post-COVID-19 vaccination. The acquired infection transmission of HSV-2 is high among persons with no history of genital herpes infection. As a result, viral shedding is frequent in seropositive patients; in spite of having a history of genital herpes or not. For people affected with SARS COV-2 infection, there is an increased risk of CNS infections due to reactivation of neurotrophic agents, which raises the question of its particular role in the brain barrier cross. As a result, this may lead to acute new infections with neurological manifestations, not associated with respiratory symptoms. However, the role of the blood-brain-barrier in averting SARS-CoV-2 from entering the brain is yet to be established.

It is important to increase awareness of these rare presentations in physicians and healthcare workers and facilitate early diagnosis and management to prevent further complications and outbreaks of the disease.

Conclusion

A literature review revealed that in addition to COVID-19 infection common presentation of fever, fatigue, and mild

respiratory symptoms such as dry cough and shortness of breath, patients may also manifest a range of neurological manifestations which may include headache, anosmia, hyposmia, dysgeusia, meningitis, encephalitis, and acute cerebrovascular accidents during the course of the disease.

Finally, HSV-2 is a latent infection, the viral shedding is frequent in seropositive patients despite developing no genital lesions. Due to a deficiency in the immune system in patients with latent infection, the reactivation of the virus might occur with a range of manifestations. The infection with the new SARS CoV-2 could reactivate the latent viruses, and cause worsening of the initial symptoms, or even manifest new mild to severe symptoms. Therefore, the mechanism of how the blood-brain-barrier (BBB) is prohibiting the virus from entering the brain is yet to be studied. As a result, viral screening is highly recommended for patients with a previous history of viral infections whether the patient is symptomatic or not.

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