Transient Horner syndrome associated with COVID-19: A case report

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ABSTRACT

The Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2), the virus that causes coronavirus disease 2019 (COVID-19), has spread as a global pandemic with significant morbidity and mortality. As the prevalence of COVID-19 has risen, so has the diversity of its clinical presentation. SARS-CoV-2 is considered to have neuroinvasive and neurotropic qualities that can lead to central and peripheral nervous system manifestations. We describe a 65-year-old woman who developed new-onset unilateral ptosis and miosis following a diagnosis of COVID-19. To our knowledge, this is the first reported case describing transient Horner syndrome in association with COVID-19.

1. Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus was first detected in Wuhan City, Hubei Province of China on December 31, 2019 [1]. This virus causes coronavirus disease 2019 (COVID-19), a contagious respiratory disease that has rapidly become a global pandemic. Common symptoms include fever, non-productive cough, dyspnea, diarrhea, headache, and myalgia [2].

SARS-CoV-2 enters cells via fusion with the angiotensin-converting enzyme 2 (ACE-2) receptor, expressed on the surface of type II alveolar epithelial cells in human lungs, as well as neurons and glial cells [3,4]. SARS-CoV-2 has been shown to enter the central nervous system (CNS) through endothelial and epithelial cells of the blood brain barrier (BBB) as well as retrograde axonal transport of cranial nerves and peripheral nerves [5]. The release of inflammatory cytokines and chemokines in response to infection is thought to promote increased BBB permeability [6].

In a systematic review of neurological complications of COVID-19 patients, headache, dizziness, olfactory/gustatory impairment, and altered consciousness were the most frequently described symptoms [7]. Less frequent neurological complications include cerebrovascular events, seizures, meningoencephalitis, and immune-mediated neurological diseases. To our knowledge, there have been no reports of Horner syndrome (HS) in association with COVID-19.

HS is a constellation of symptoms attributed to a disruption of the oculosympathetic pathway, which can manifest clinically as ipsilateral ptosis, miosis, and in some cases facial anhidrosis [8].

2. Case presentation

A 65-year-old woman with a past medical history of inflammatory bowel disease, hypothyroidism, rheumatoid arthritis, Factor V Leiden Deficiency, and a remote history of smoking was admitted to the emergency department with dyspnea, fever, body aches, and generalized weakness. The day prior to admission, a reverse transcription polymerase chain reaction (RT-PCR) assay of nasal and pharyngeal samples was found to be positive for SARS-CoV2.

On examination, the patient was in respiratory distress, tachypneic, with a respiratory rate of 32, and hypoxic with an SpO2 of 85% on room air. Vitals signs were otherwise normal. She had posterior oropharyngeal erythema. There were no other pertinent physical examination findings. Her neurological examination, including the cranial nerve examination, was normal. The patient denied any previous history or current headaches. The laboratory results are shown in Table 1. Electrocardiogram was normal. Computed tomography of the chest with contrast revealed patchy, diffuse ground-glass infiltrates consistent with COVID-19.

Table 1
Systemic laboratory data and imaging findings.

| Blood | Patient value | Normal range |
|-------|---------------|--------------|
| Hemoglobin g/dL | 12.7 | 11.7–15.3 |
| Leukocyte count K/μL | 9.8 | 3.8–11.8 |
| Neutrophils % | 88.8 | 42.7–76.8 |
| Lymphocytes % | 6.9 | 16.0–45.9 |
| Glucose mg/dL | 126 | 70–100 |
| Blood urea nitrogen mg/dL | 9 | 7–22 |
| Creatinine mg/dL | 0.81 | 0.50–1.50 |
| Sodium mmol/L | 131 | 135–145 |
| Potassium mmol/L | 3.5 | 3.5–5.1 |
| Aspartate aminotransferase IU/L | 65 | 1–42 |
| Alanine aminotransferase IU/L | 34 | 1–50 |
| C-reactive protein mg/dL | 16 | ≤1.0 |
| Erythrocyte sedimentation rate mm/h | 5 | ≤20 |

Imaging modality | Findings |
|-----------------|----------|
| Electrocardiogram | Normal |
| Computed tomography of the chest with contrast | Findings consistent with pneumonitis due to COVID-19 |

| Computed tomography of the head with contrast | Normal |
| Computed tomography angiography of the neck with perfusion | Normal |
| Magnetic resonance imaging of the head both with and without contrast | Normal |

* Performed the day of admission.

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ptosis and miosis without anhidrosis, and was diagnosed with HS. It should be noted that the absence of anhidrosis was simply observed and treated with dexamethasone, remdesivir, and plasmapheresis. Her respiratory status improved, though she remained on supplemental oxygen. One day following her admission, the patient developed right sided ptosis and miosis without anhidrosis, and was diagnosed with HS. It should be noted that the absence of anhidrosis was simply observed and not formally evaluated. Cranial nerve examination was otherwise normal. Further workup including computed tomography of the head without contrast, computed tomography angiography of the neck with and without contrast, computed tomography angiography of the neck with gadolinium contrast were all normal (Fig. 1, Table 1). This negative evaluation included the absence of cervical lymphadenopathy, ganglion enhancement, third cranial nerve enhancement, and orbital neuropathies. Lumbar puncture (LP) was performed, which revealed a normal opening pressure with clear, colorless cerebrospinal fluid. Full LP results are listed in Table 2. Cerebrospinal fluid SARS-CoV2 polymerase chain reaction (PCR) and antibody testing were not available at the time of evaluation. Her ptosis and miosis began to resolve three days following symptom onset, and completely resolved within eight days.

4. Conclusion

In summary, SARS-CoV-2 is considered to have neuro-invasive and neurotropic qualities that can lead to CNS and PNS manifestations. To our knowledge, this is the first reported case describing transient HS in association with COVID-19.

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Table 2

Lumbar puncture results.

| Component                           | Value     | Reference | Units |
|-------------------------------------|-----------|-----------|-------|
| CSF Total Nucleated Cells           | 0         | 0–5       | /mm3  |
| CSF Red Blood Cells (1st tube)      | 3060      | 0         | /ul.  |
| CSF Red Blood Cells (2nd tube)      | 403       | 0         | /ul.  |
| CSF Glucose                         | 68        | 40–70     | mg/dL |
| CSF Protein                         | 28        | 12–60     | mg/dL |
| CSF Gram Stain                      | Negative  |           |       |
| CSF Culture                         | Negative  |           |       |
| CSF Meningitis/Encephalitis Panel² | Negative  |           |       |

CSF = Cerebrospinal Fluid.
² Included herpes simplex virus, Venereal Disease Research Laboratory test, bacterial, and fungal cultures.

Fig. 1. Clinical timeline of the patient.
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