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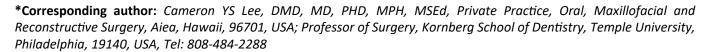
CASE REPORT

Acute Bell's Palsy: A Neurological Manifestation of the Severe **Acute Respiratory Syndrome (SARS-CoV-2)**

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Abstract

The COVID-19 pandemic caused by the severe acute (SARS-CoV-2) coronavirus-2 syndrome infection affects multiple organ systems with varied clinical presentation. The most frequently reported signs and symptoms associated with COVID-19 infection involve the cardiovascular and respiratory systems and include fever, cough, and shortness of breath. However, clinicians should be aware that neurological manifestations could be the only clinical sign and symptom reported by patients infected with the coronavirus. To the author's knowledge, there is a paucity of case reports documenting Bell's palsy in patients diagnosed with COVID-19 infection. We describe a patient who presented to the office with an acute, sudden onset of left facial muscle weakness without other complaints. Five days later, the patient reported that he tested positive for COVID-19 infection. Recognition of Bell's Palsy without other symptoms should be considered as part of the clinical presentation of COVID-19 infection that could easily be overlooked. Prompt diagnosis is critical to early appropriate management and decreases the potential long-term adverse clinical affects, psychological and social distress associated with this peripheral nerve palsy.

Keywords

Bell's palsy, Facial nerve, COVID-19 infection, Neurologic manifestations

Introduction

Bell's palsy is an acute idiopathic lower motor neuron peripheral nerve palsy of the facial nerve that innervates the muscles of facial expression [1,2]. The facial nerve also provides parasympathetic innervation to the salivary glands of the oral cavity, lacrimal glands,

and sensory innervation to the anterior two-thirds of the tongue, tympanic membrane, and inner ear canal [3]. The affected side results in facial muscle weakness or paralysis without other neurologic symptoms. The result is inability to move the muscles of facial expression. The incidence of this idiopathic palsy is estimated at 15 to 30 per 100,000 individuals. There is no predilection for age and gender [4].

Neurological Manifestations

The severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) is a multisystem disorder that can affect the cardiovascular, respiratory, neurological, gastrointestinal, and renal systems [5-8]. It remains unclear if SARS-CoV-2 demonstrates neurotropism, but there could be a correlation between neurologic manifestations and SARS-CoV-2 [9-11]. Neurologic manifestations with COVID-19 infection reported include large vessel cerebrovascular convulsions, Guillain-Barre syndrome, encephalopathy and loss of taste and smell [12-14]. Bell's palsy has also been reported in patients infected with the coronavirus (COVID-19) [15-17]. We report of a case of a patient who presented to the office with an acute, sudden onset of left facial muscle weakness. Five days later, the patient reported that he tested positive for COVID-19 infection.

Case Report

A 42-year-old Asian male urgently presented to the office with an acute onset of left sided facial muscle weakness. He was unable to smile on the affected side



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that was noticed by his wife when he woke up in the morning. The patient also was not able to close his left upper eye lid. He reported no other neurologic complaints. Medical history was unremarkable. He was not taking any medications and denied any allergy to medications. Maxillofacial examination revealed facial asymmetry with facial droop of the cheek and angle of the mouth, loss of the nasolabial fold and excess pooling of saliva. The patient had difficulty smiling, wrinkle the left forehead and close the upper lid of the eye with verbal commands all consistent with a lower motor neuron facial nerve palsy. He denied any specific cardiovascular or respiratory symptoms, such as chest pain, shortness of breath, cough, or fatigue. The remaining physical examination was unremarkable, and no other neurological deficits observed. A diagnosis of acute Bell's palsy (House-Brackmann Grade 4) without other medical or neurologic etiology was made. To regain function of the facial muscles and avoid potential long-term complications such as facial asymmetry, anosmia, speech impairment, permanent eye injury and quality of life, the patient was prescribed a 7-day course of oral antiviral medication (acyclovir 400 mg five times per day) and a steroid regimen of prednisone 40 mg per day [18]. To prevent corneal abrasion, the patient was prescribed artificial tears and an eye patch to cover the eye. Five days after the consultation and examination, the patient called the office that he refused to take the Covid-19 vaccine and tested positive for coronavirus by real time polymerase chain reaction (RT-PCR) yesterday but, remains asymptomatic.

Discussion

Sudden acute facial paresis or paralysis that appears in 72 hours or less without any definitive etiology is consistent with the diagnosis of Bell's palsy [3]. Although the pathogenesis of Bell's palsy remains unclear, different theories have been postulated such as viral infection, inflammation, ischemia, and autoimmune states [19]. Further, reactivated neurotropic viruses such as herpes simplex virus (HSV), Varicella-zoster virus, Epstein-Barr virus have been associated with Bell's palsy resulting in inflammation and demyelination of the facial nerve [1,4,19,20]. Neurological manifestations are part of the clinical spectrum of COVID-19 and acute facial paresis or paralysis is being reported in greater frequency [15-17]. It is hypothesized that the coronavirus could enter the central nervous system (CNS) by direct invasion, or through the circulatory system [21]. Moriguchi, et al. [22] isolated RNA from COVID-19 infected patients in the CSF further supporting the view that the coronavirus can directly infect the CNS.

Angiotensin-converting enzyme 2 (ACE2) is the primary functional host receptor for SARS-CoV-2 and plays a key role in the pathogenesis of COVID-19 infections as it is expressed in various tissues of the human body, including the heart, intestine, kidney,

and pulmonary alveolar (type II) cells [23-25]. Entry of the coronavirus into human cells occurs through the interaction of a receptor-binding domain on the viral spike glycoprotein ectodomain with the ACE2 receptor [24,25]. High levels of ACE2 receptors in the glial cells and neurons may provide a mechanism for attachment of the spike protein of the coronavirus and the observed neurological symptoms [26,27].

Conclusion

As COVID-19 infection is a multisystem inflammatory disorder, Bell's palsy may be a spectrum of neurological manifestations that is being reported in greater frequency. Recognition of Bell's palsy without other symptoms should be considered as part of the clinical presentation of COVID-19 infection that could easily be overlooked. Facial expression is critical to an individual's psychological well-being and interaction in the community. Prompt diagnosis is critical to early appropriate management and decrease the psychological and social distress associated with this peripheral nerve palsy.

Conflict of Interest

None.

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