

Bilateral vocal cord paralysis post-COVID-19 infection

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SUMMARY

COVID-19 infection is a pandemic that began in Wuhan, China since December 2019. It is a viral illness that can have multisystem manifestations. Cranial nerve involvement including vocal cord palsy has been increasingly recognised. We report a case of a 63-year-old gentleman with bilateral vocal cord abductor paralysis resulting from the denervation of recurrent laryngeal nerve post-COVID-19 infection. He presented with right hemiplegia, right hemianopia and expressive aphasia, correlating with a left middle cerebral artery infarct. These were preceded by 12 days history of upper respiratory tract infection symptoms. The clinical and laboratory data showed evidence of severe COVID-19 infection requiring high oxygen support and close monitoring at COVID-19 isolation ward. He developed complications during hospitalisation such as bilateral vocal cord paralysis, deep vein thrombosis and pulmonary embolism. He was treated conservatively for vocal cord paralysis by the otorhinolaryngology team prior to discharge. Unfortunately, he was readmitted 1 month later for worsening shortness of breath, requiring emergency tracheostomy. This case report highlights the rare occurrence of bilateral vocal cord paralysis during the recovery phase of COVID-19 independent of the stroke.

INTRODUCTION

COVID-19 infection is a global threat to public health. By 27th of February 2022, there have been 430 million cases of COVID-19, including 5.9 million deaths, reported to the World Health Organization (WHO). The COVID-19 infection results in a spectrum of signs and symptoms resulting from the involvement of multiple organs. The incidence of COVID-19 infection has rapidly spread to approximately 221 countries globally with presence of neurological symptoms in certain cases. One case series study by Mao et al. showed that neurological symptoms were present in 36.4% of patients and were more common (45.5%) in patients with severe infections.¹

There is a myriad of neurological complications that are associated with COVID-19 infection. They include headache, altered sensorium, losing smell and taste, encephalopathy, neuromuscular disorder, acute cerebrovascular disorder and cranial neuropathy.²

In terms of cranial neuropathy, it may occur as mononeuropathy or polyneuropathy, either unilateral or bilateral, with or without central nervous system (CNS) involvement. There are a few mechanisms by which the

COVID-19 infection can inflict damage to the nervous system. For example, direct invasion by retrograde transport via axon to the brain, immunological reaction due to molecular mimicry, hypoxic injury and neurotoxic COVID-19 drugs.^{3,4}

Vocal cord is innervated by branches of vagus nerve. Its involvement in the context of COVID-19 has already been described by authors around the world as shown in Table I.

CASE REPORT

A 63-year-old gentleman, hospitalised after 12 days of intermittent fever, productive cough, shortness of breath and 1 day of right-sided body weakness. He has comorbidities including diabetes mellitus, hypertension and ischaemic heart disease. Upon arrival at hospital, he was febrile with Glasgow Coma Scale of E4V2M5, expressive aphasia, right hemiplegia, right hemianopia and left gaze preference. He was sent for urgent computed tomography (CT) brain which showed showed left middle cerebral artery (MCA) infarct correlating with his right-sided weakness (Figure 1) and chest X-ray revealed ground glass changes at bilateral peripheral and lower zones of lungs. COVID-19 polymerase chain reaction (PCR) assay was positive for the throat sample. Cardiovascular and abdominal examinations were unremarkable. The pulmonary system revealed bilateral crepitations on auscultation. Hence, he was admitted to COVID-19 isolation ward with oxygen support of 3L/min.

However, on the day 3 of admission, the general condition deteriorated with respiratory distress and required high-flow oxygen support of 15L/min. He was given supportive care including intravenous dexamethasone, antibiotics for possible aspiration pneumonia and adequate hydration. Laboratory investigations revealed severe COVID-19 infections. On day 11 of admission, he was transferred back to the general neurology ward for the continuation of post-stroke care. He developed inspiratory stridor with noisy breathing requiring urgent review by otorhinolaryngologist.

Bedside flexible nasopharyngolaryngoscopy showed bilateral vocal cord abductor paralysis during inspiration (Figure 2), even so he was able to maintain oxygen saturation without oxygen support. Throughout admission, he underwent intensive chest and limb physiotherapy but did not show any remarkable improvement. He was deemed unfit for a posterior cordectomy under general anaesthesia by the otorhinolaryngology and anaesthetic team. His family was not keen as well for any surgical intervention because he was

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Table 1: Available literature on cases of vocal cord palsy post-COVID-19 infection

Author	Study type	Title	Number of Patients	Intervention	Year of Publication
Jungbauer et al.	Case report	Bilateral Palsy of The Vocal Cord After COVID-19 Infection	1	IV Methylprednisolone, oxygen therapy, laterofixation, laser posterior cordectomy	2021
Korkmaz et al.	Case report	Unilateral Vocal Cord Paralysis Case Related to COVID-19	1	Voice therapy and injection laryngoplasty	2021
Rapoport et al.	Case Series	Acute Vocal Fold Paresis And Paralysis After COVID-19 Infection	16	All patients given voice therapy, 4 patients given in-office injection laryngoplasty using hyaluronic acid	2022
Zamzam et al.	Case Series	Impact of COVID-19 On Vocal Cord mobility	6	All patients given speech therapy, 1 required vocal cord medialisation by hyaluronic acid injection	2021

Herein, we describe a case of post-COVID-19 with bilateral vocal cord paralysis.



Fig. 1: Computed tomography brain showed left middle cerebral artery infarct (white arrow) correlating with his right-sided weakness

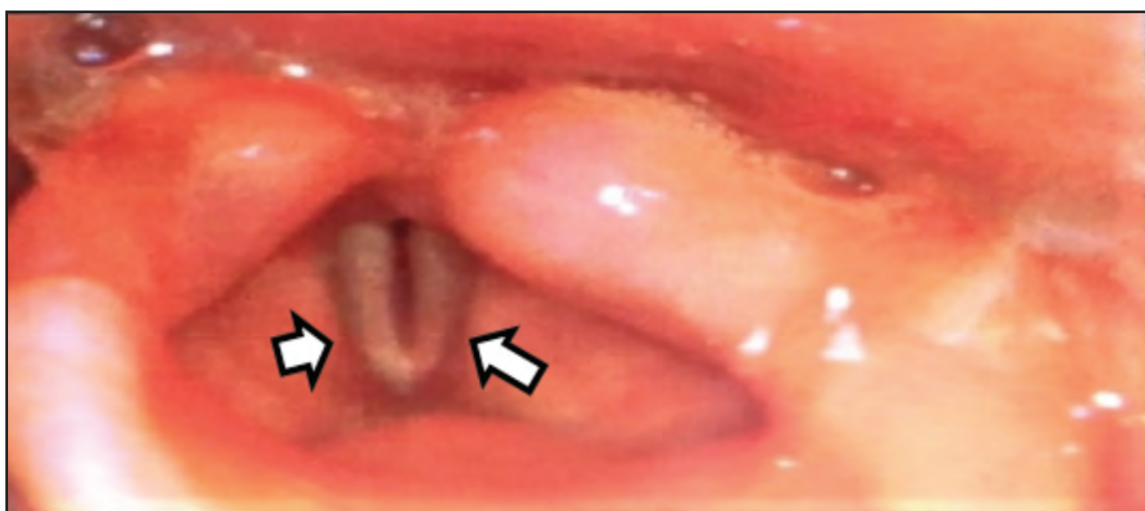


Fig. 2: Bedside flexible nasopharyngolaryngoscopy shows bilateral vocal cord abductor paralysis (white arrows) during inspiration

able to maintain oxygen saturation under room air. Thus, he was treated conservatively. He was discharged home with a Modified Rankin Scale of 5 after 1.5 months of hospitalisation. Two weeks later, he revisited our emergency department in view of respiratory distress, which required emergency tracheostomy intervention. Subsequently, he is on regular otorhinolaryngology and rehabilitation team follow-up.

DISCUSSION

Common causes of bilateral vocal cord paralysis include iatrogenic causes such as endotracheal intubation, neck surgery, brainstem stroke and malignant infiltration. Our patient did not have any upper airway interventions such as endotracheal intubation or neck surgery.

The patient developed stroke at the second week and then stridor at around 3 weeks after first onset of COVID-19 symptoms. It is tempting to think that the acute left MCA infarct could have caused the vocal cord paralysis. However, the territory of the stroke does not correlate to his vocal cord paralysis. Moreover, the presentation of stridor is subacute rather than at the onset of acute stroke.

Therefore, it seems that the most likely cause of the patient's vocal cord paralysis is due to COVID-19 infection based on clinical symptoms and available information. There is no lumbar puncture to look for evidence of COVID-19 infection in the CNS due to refusal of consent.

There are a few theories which can explain the pathophysiology of COVID-19 infection in the nervous system. Direct viral invasion can happen via neuronal pathway whereby the viruses migrate by infecting sensory or motor nerve ending.⁴ Since January 2020, angiotensin-converting enzyme 2 (ACE2) was found to be the functional receptors for COVID-19 which exists in organs such as nervous systems and skeletal muscles. Viral spike proteins from COVID-19 could interact with ACE2 receptors expressed in capillary endothelium and enter the CNS by damaging the blood-brain barrier.⁵ Furthermore, viral particles have been found in axons and neurons from autopsy repeatedly.⁶

Nervous system damage may also be mediated by the immune system. Molecular mimicry between the microbial and nerve antigens is one of the postulated hypotheses for neuro-immunological phenomenon. This happens when immunological reactions against the virus secondarily affect neuronal structures.³ Conditions such as Guillain-Barre syndrome, acute transverse myelitis and Bell's palsy share similar pathophysiology.^{7,8}

The third hypothesis is neurotoxic effect from the COVID-19 drugs. Drugs known to cause neuropathy and commonly given to COVID-19 patients include daptomycin, linezolid, lopinavir, ritonavir, hydroxychloroquine, cisatracurium, clindamycin, tocilizumab and glucocorticoid.⁹

CONCLUSION

Given the fact that COVID-19 infection may present with neurological symptoms, physicians need to be vigilant in considering COVID-19 as a differential diagnosis of vocal cord paralysis or other cranial neuropathies.

INFORMED CONSENT

Informed consent for publication was obtained from the patient and son.

CONFLICT OF INTEREST

None.

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