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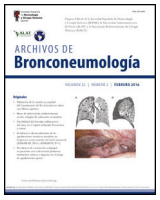

Case Report

# Severe COVID-19 Pneumonia and Delayed Bilateral Vocal Cord Paralysis

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Coronavirus disease 2019 (COVID-19) can cause severe acute respiratory distress (ARDS) and long-term complications.[1] We report a case of delayed bilateral vocal cord paralysis (BVCP) as an exceptional complication of severe COVID-19 pneumonia.

A 41-year-old patient was admitted for severe ARDS caused by bilateral SARS-CoV-2 pneumonia that required orotracheal intubation for 6 days. The patient was extubated, but reintubation was required for laryngeal edema that was treated with corticosteroids, following which he was successfully extubated. Fiberoptic laryngoscopy showed mild laryngeal edema and vocal cord pathology was ruled out. Seventy-six days after discharge, the patient consulted for progressive dysphonia and inspiratory stridor, so corticosteroid treatment was started. Respiratory function tests showed amputated flow/volume curves. Fiberoptic laryngoscopy was repeated, confirming complete paralysis of both vocal cords. A subsequent neck and chest CT scan ruled out other lesions, so we opted for speech therapy and respiratory physiotherapy that resulted in clinical improvement.

BVCP has a broad etiology: surgery, cancer, and neurological disorders are the most common causes, while intubation and viral infection are much more infrequent.[2] Up to 50% of cases are classified as idiopathic but may conceal a viral etiology.[2] Vocal cord involvement has been reported in several viral diseases. Influenza A,[2] for example, has been associated with delayed postviral vagal neuropathy at 22 weeks and a good prognosis.[2,3]

The absence of other possible causes and the timeframe of the case suggested a viral etiology. Severe SARS-CoV-2 infection can cause delayed cranial nerve changes[4] and there is evidence of the neuroinvasive potential of the virus, possibly as a result of direct neurotoxicity mediated by ACE-2 receptors that allow access to the central nervous system (CNS).[4,5]

Once the virus has accessed the CNS it is difficult to eliminate due to the absence of major histocompatibility complex antigens and the homeostatic characteristics of the CNS cells.[4] This situation, along with a progressive inflammatory cascade and persistent immune hyperreactivity, could affect various cranial and peripheral nerves that, due to their more caudal location, would account for delayed involvement in a very limited number of cases.[4,5] Furthermore, retrograde transport of SARS-CoV-2 along the vagus nerve to the brainstem causing delayed BVCP has been mooted.[4]

The real incidence of BVCP caused by SARS-CoV-2 is unknown, but it is clearly very rare.[2] The few reported cases involved a serious event that appeared 3–40 days after discharge, although in our case, BVCP developed even later.[2–4]

The possibility of BVCP in the late stages of a serious SARS-CoV-2 infection must be recognized to facilitate prompt diagnosis and treatment of this potentially life-threatening condition. It can be diagnosed by assessing patient background, clinical history, respiratory function tests, and direct endoscopic images, and appropriate treatment must be administered in line with severity and clinical impact.

## Conflict of Interests

The authors state that they have no conflict of interests.

## Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.arbres.2023.05.019.

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