Unilateral diaphragm paralysis with COVID-19 infection

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SUMMARY
Neurological complications are well described in SARS-CoV-2, but for the first time we report a case of unilateral diaphragm paralysis occurring early in mechanical ventilation for respiratory failure due to such an infection. The patient subsequently required tracheostomy and ventilator support for 37 days, and had increased breathlessness and an elevated diaphragm at clinic review 9 months later. Dynamic chest radiography demonstrated persistent diaphragm paralysis with an accompanying postural change in lung volumes, and he subsequently underwent surgical plication. This case demonstrates that although persistent dyspnoea is a common feature following SARS-CoV-2 infection and is usually due to deconditioning or persistent parenchymal involvement, it can be due to other causes and needs to be investigated appropriately.

BACKGROUND
Lower respiratory tract involvement is a common feature of infection with the SARS-CoV-2 virus, most notably respiratory failure due to viral pneumonitis, but as the COVID-19 pandemic continues, long-term complications are emerging. Among these are involvement of the neurological tract1,2 and chronic lung disease, chiefly pulmonary fibrosis.3 We report a case of unilateral diaphragmatic paralysis in an individual with SARS-CoV-2 infection, and explore the possible contributory factors and learning points of this novel case.

CASE PRESENTATION
A 54-year-old Caucasian man was admitted with rapidly progressive dyspnoea due to PCR positive SARS-CoV-2 infection. He had a history of insulin dependent type 2 diabetes mellitus, obstructive sleep apnoea (OSA) managed with home continuous positive airway pressure (CPAP), primary hypertension and a raised body mass index (38.1 kg/m²). He reported no other respiratory history and was a non-smoker. After 3 days, due to refractory type 2 respiratory failure despite CPAP, he was intubated and placed on lung protective airway pressure release ventilation with intense neurological blockade. He also developed acute renal failure, for which he received temporary venovenous renal replacement therapy. Standard drug treatment in use for SARS-CoV-2 pneumonia at the authors’ unit at the time was given: intravenous spectrum antibiotics, anticoagulation and corticosteroids.

INVESTIGATIONS
The initial chest radiograph showed ground glass consolidation in the left lung, with normal bilateral haemidiaphragm position (figure 1A), but this progressed rapidly to bilateral, peripherally predominant ground glass change consistent with SARS-CoV-2 pneumonia. Bilateral consolidation and volume loss were seen early in the admission, with a progressively elevated right haemidiaphragm from day 7. He had received a right jugular central venous catheter (CVC) at the beginning of his admission, but this was placed without complication, replaced soon after by a contralateral jugular haemofiltration line and the elevated right haemidiaphragm did not become apparent for 4 days after placement. A tracheostomy was performed at 2 weeks, followed by a prolonged respiratory wean supported with CPAP, and he was liberated from mechanical ventilator support after 37 days. No iatrogenic injury to the neck was noted during this period. CT thorax at this point showed bilateral multifocal consolidation worse on the right, with an elevated right haemidiaphragm (figure 1B).
OUTCOME AND FOLLOW-UP

Following a period of rehabilitation, he was discharged at 61 days. At 4-month clinic review, he reported persistent dyspnoea and orthopnoea. CT revealed significant improvement of the consolidation, with a raised right haemidiaphragm and minor residual upper lobe linear atelectasis.

His symptoms persisted, and at 9 months dynamic chest radiography (a real-time large-field-view thoracic imaging system) demonstrated clear lung fields but a raised right haemidiaphragm with ipsilateral paradoxical motion on sniff manoeuvre (figure 1C,D). Spirometry showed a postural reduction in forced vital capacity of 43.5% from standing to lying. He subsequently underwent surgical plication.

DISCUSSION

The diaphragm is the primary muscle of respiration, and each haemidiaphragm is supplied by the phrenic nerve. Damage to this nerve or intrinsic weakness of the diaphragm muscle fibres can lead to diaphragmatic palsy, which may be traumatic, malignant, iatrogenic, neurological, inflammatory or idiopathic.

The SARS-CoV-2 virus has neuroinvasive potential, and infection is associated with numerous neuromuscular complications such as myasthenia gravis, Guillain-Barré syndrome and anosmia. To the authors’ knowledge, there has been only one previous case report of diaphragm paralysis following SARS-CoV-2 infection, not associated with mechanical ventilation.

Prolonged intubation and mechanical ventilation are associated with diaphragm weakness, likely as a consequence of critical illness polyneuropathy or mechanical trauma, and diaphragm dysfunction in ventilated patients carries a high mortality and morbidity. However, in our case, diaphragmatic paralysis occurred early in the disease course and no pruning manoeuvres or neck trauma took place, suggesting that it was not due to mechanical causes or critical illness. Phrenic neuropathy is well described in diabetes, and this may have been contributory. Although phrenic nerve palsy may be associated with trauma during jugular CVC insertion, this is extremely rare, and unlikely given the uncomplicated insertion and lack of temporal association with the development of haemidiaphragm paralysis.

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