

Correspondence on: 'Interleukin-6 blockade with sarilumab in severe COVID-19 pneumonia with systemic hyperinflammation—an open-label cohort study' by Della-Torre *et al*

We read with deep interest the article by Della-Torre *et al*,¹ which was aimed at assessing the safety and efficacy of interleukin (IL)-6 blockade with sarilumab in patients with severe COVID-19 pneumonia and systemic hyperinflammation. The results indicated that at day 28, overall clinical improvement and mortality were not significantly different between sarilumab and standard of care. Sarilumab was associated with faster recovery in a subset of patients who showed minor lung consolidation at baseline. This conclusion might be of great significance for alleviating the current COVID-19 pandemic.

However, we noticed that most of the patients in this study met the diagnostic criteria for acute respiratory distress syndrome (ARDS,² according to the baseline demographic and clinical characteristics of the patients' cohort, there were 22 patients with a PaO₂/FiO₂ ratio of 100–200 and 30 patients with a PaO₂/FiO₂ ratio <100; the duration of symptoms before enrolment (days) was 7 days; and bilateral pneumonia was radiologically documented, although there were no respiratory distress data and no cardiogenic pulmonary oedema data). The pathological findings of COVID-19 also confirmed that it was associated with ARDS,³ but most patients did not receive invasive mechanical ventilation (MV) or the authors did not consider MV as the main observation target and, therefore, did not show the data of the use of MV. However, in our opinion, it is important to understand how to reduce the use of MV in patients with severe COVID-19 pneumonia.

ARDS is a life-threatening form of respiratory failure characterised by inflammatory pulmonary oedema resulting in severe hypoxaemia.⁴ Non-invasive ventilation (NIV) improves pulmonary hypoventilation as a result of persistent strong spontaneous inspiratory efforts, which simultaneously increases tissue stress. This leads to an increase in pulmonary transvascular pressure, vascular flow and fluid leakage, resulting in rapid deterioration of lung function.⁵ The guidelines on the management of critically ill adults with COVID-19 recommend the use of MV in case of ARDS as early as possible; in mechanically ventilated adults with COVID-19 and ARDS, the guidelines recommend the use of low tidal volume (Vt) ventilation (Vt 4–8 mL/kg of predicted body weight),⁶ higher PEEP (<15 cm H₂O) and prone positioning while minimising oxygen consumption and possible hypercapnia.⁷ In mechanically ventilated adults with COVID-19 and ARDS, the guidelines suggest the use of systemic corticosteroids, as opposed to not using corticosteroids.⁶

Furthermore, ventilator-assisted breathing, regardless of whether it is MV or NIV, has an adverse effect on blood pressure. For NIV to work normally, good cooperation is required between the patient and the ventilator, which means that it is difficult for patients with consciousness weakness to receive NIV; however, in the demographic and clinical characteristics baseline of the patients' cohort, there were no such key data, including the patient's basic blood pressure, state of consciousness (Glasgow score), respiratory rate, arterial blood gas (pH, PaCO₂, PaO₂), peripheral oxygen saturation (SpO₂), and the number of patients who changed to MV during the course of treatment. Additionally, the data did not indicate whether the patients had to use vasoactive drugs after using the ventilator.

More details regarding how the authors used NIV to help patients to go through such severe hypoxia will be of great help to COVID-19 epidemic areas, particularly those facing a shortage of medical devices.

We respect the significant contributions of the authors and look forward to the follow-up results of this study.

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