



A Rare Case of Severe Long COVID-19 in Patient with Amyotrophic Lateral Sclerosis

Parul Kodan¹ Richa Aggarwal² Rakesh Kumar³ Satyendra Nawal²

¹ Department of Medicine, All India Institute of Medical Sciences, New Delhi, India

² Department of Critical and Intensive Care, Jai Prakash Narayan Apex Trauma Center, All India Institute of Medical Sciences, New Delhi, India

³ Department of Anaesthesiology, Critical Care and Pain Medicine, All India Institute of Medical Sciences, New Delhi, India

Address for correspondence Richa Aggarwal, MD, Department of Critical and Intensive Care, Jai Prakash Narayan Apex Trauma Center, All India Institute of Medical Sciences, New Delhi 110029, India (e-mail: pathakricha@yahoo.co.in).

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Introduction

There is growing interest on coronavirus disease 2019 (COVID-19)-associated neurological manifestations and the interplay of COVID-19 in patients with existing neurological illness.¹ We wish to share our rare case of COVID-19-associated neurological manifestations in a patient with amyotrophic lateral sclerosis (ALS). ALS is a neurodegenerative disease with very scarce information suggesting how COVID-19 affects patients with ALS.^{2,3} Case reports of COVID-19 in ALS are handful in literature and none of them highlights the acute deterioration in respiratory status with acute COVID-19 and the subsequent complications of long COVID-19. To the best of our knowledge, this is the first such reported case.

Case Report

A 40-year-old male with recently diagnosed ALS presented to the emergency department (ED) with a history of fever and worsening breathlessness for the last 4 days. The patient was followed up by neurology department for ALS in the last 6 months. He had baseline quadriparesis and was bedridden; however, he had no bulbar involvement and respiratory compromise with Revised Amyotrophic Lateral Sclerosis Functional Rating Scale (ALSF_{RS}-R) score of 27. He was on tablet riluzole. In the ED, he was conscious but tachypneic and his vitals were as follows: pulse rate of 120 /min, blood pressure, 130/86 mm Hg, respiratory rate of 32/min, and

oxygen saturation of 86% on room air. He was started on oxygen therapy and reverse-transcription polymerase chain reaction (RT-PCR) test for severe acute respiratory syndrome coronavirus 2 was done. However, his respiratory distress worsened rapidly within few hours and he was intubated and ventilated. As his RT-PCR test came out to be positive, he was shifted to intensive care unit (ICU) of COVID-19 center. He received steroids, anticoagulation, and supportive treatment as per institutional protocol. The initial chest X-ray was not suggestive of any COVID-19 features and was normal (→ Fig. 1). The computed tomographic scan could not be done; however, serial chest X-rays were done daily that did not show any radiological changes suggestive of COVID-19. Respiratory deterioration in this patient was due to respiratory muscle weakness rather than parenchymal abnormalities. He remained on ventilator due to poor respiratory efforts for the next 7 days. Tracheostomy was done at the end of first week in anticipation of possibility of prolonged respiratory support. Nutritional and physiotherapy support was provided for the holistic care and recovery.

In second week of illness, patient developed high-grade fever. The recorded axillary temperature was 104.6 F. There were no features of acute infection/sepsis or organ failure other than fever. All sepsis markers including procalcitonin were normal. All the inflammatory markers suggestive of severe COVID-19 were within the normal limits. Noninfective causes of fever in ICU like deep vein thrombosis, transfusion reaction, and acute cholecystitis were ruled out. There were no findings on ultrasonography chest and abdomen. All

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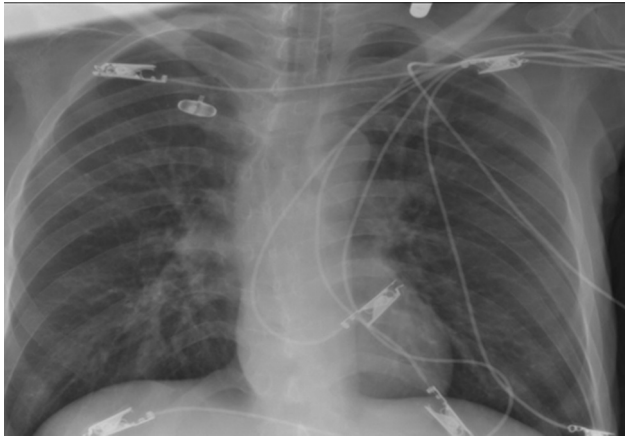


Fig. 1 Normal chest X-ray on day 1 of admission.

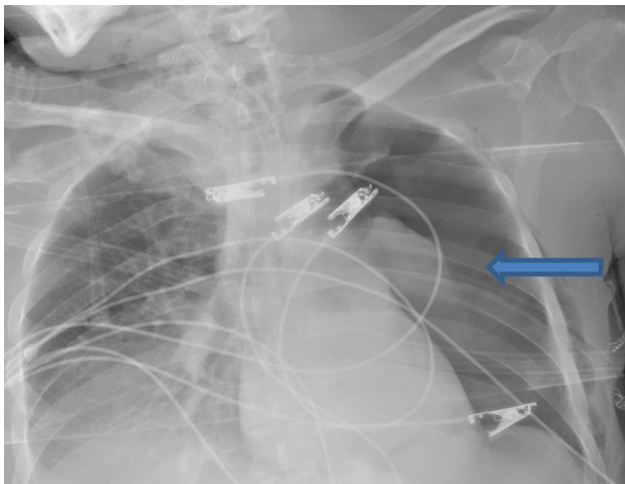


Fig. 2 Chest X-ray showing pneumothorax (blue arrow) in patient with severe coronavirus disease 2019 and amyotrophic lateral sclerosis with intercostal drain in situ.

cultures sent came out to be sterile. The possibility of drug fever was considered and riluzole was withheld and patient managed with symptomatic care. The fever resolved after stopping the drug. In due hospital course, the patient developed pneumothorax (→ **Fig. 2**) and was managed promptly with chest tube drainage and supportive care. The course of illness was further complicated by difficult weaning in this patient. In the third week of ICU stay, the patient developed ventilator-associated pneumonia secondary to *Acinetobacter baumannii* infection. He was treated as per culture sensitivity; however, unfortunately, the patient succumbed to illness.

It is important to understand how COVID-19 can affect patients with ALS especially because respiratory compromise is common in ALS patients and can be further complicated by the clinical course of COVID-19 that can lead to

respiratory failure and need for intubation.^{1,2} The literature is scarce on COVID-19 infection as a trigger to accelerate respiratory weakness in patients on ALS¹ and the association between two remains speculative. Similar to the reported cases,¹ our case also highlights a possibility of COVID-19-accelerated disease progression in ALS. However, the respiratory decline in our case occurred during the acute COVID-19 phase instead of post-COVID-19 phase as reported earlier.

Second, another learning point was regarding the drug riluzole. It has been documented to cause hypersensitivity reaction but drug fever has not been documented. However, in our case, the possibility of drug fever could not be ruled out.^{3,4} Moreover, no literature on drug interaction between remdesivir and riluzole exists and the same needs exploration.

Underlying neuromuscular condition and prolonged hospital course in background of COVID-19 pose a challenging scenario. We need to be aware that these patients may require prolonged ICU stay due to difficult weaning that has its own set of complications including secondary infections and barotrauma. The risk of pneumothorax in COVID-19 has been well documented.^{5,6}

This case adds to existent sparse literature on prolonged course of COVID-19 and its complications in a patient of ALS. Reporting of such cases and compilation of a COVID-ALS registry⁷ can throw more light about COVID-19 in ALS.

Author Contributions

P.K. and R.A. conceptualized and drafted the letter. R.K. and Satyendra contributed in drafting of the letter.

Conflict of Interest

None declared.

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