

Unexplained hypoxia requiring prone ventilation in a patient with acute disseminated encephalomyelitis

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Introduction

Acute Disseminated Encephalomyelitis (ADEM) is a rare, immune-mediated, demyelinating disorder of the central nervous system characterized by acute encephalopathy with neurologic deficits and MRI findings consistent with multifocal demyelination requiring immunosuppression for therapy.^{1,2}

Patients seldom develop hypoxia due to impaired oxygenation during the course of the illness, requiring prone ventilation to improve oxygenation which is the first line of therapy and a proven standard of care in patients with ARDS.^{3,4}

Objective

We would like to present a case of ADEM where a patient developed unexplained hypoxia requiring prone ventilation.

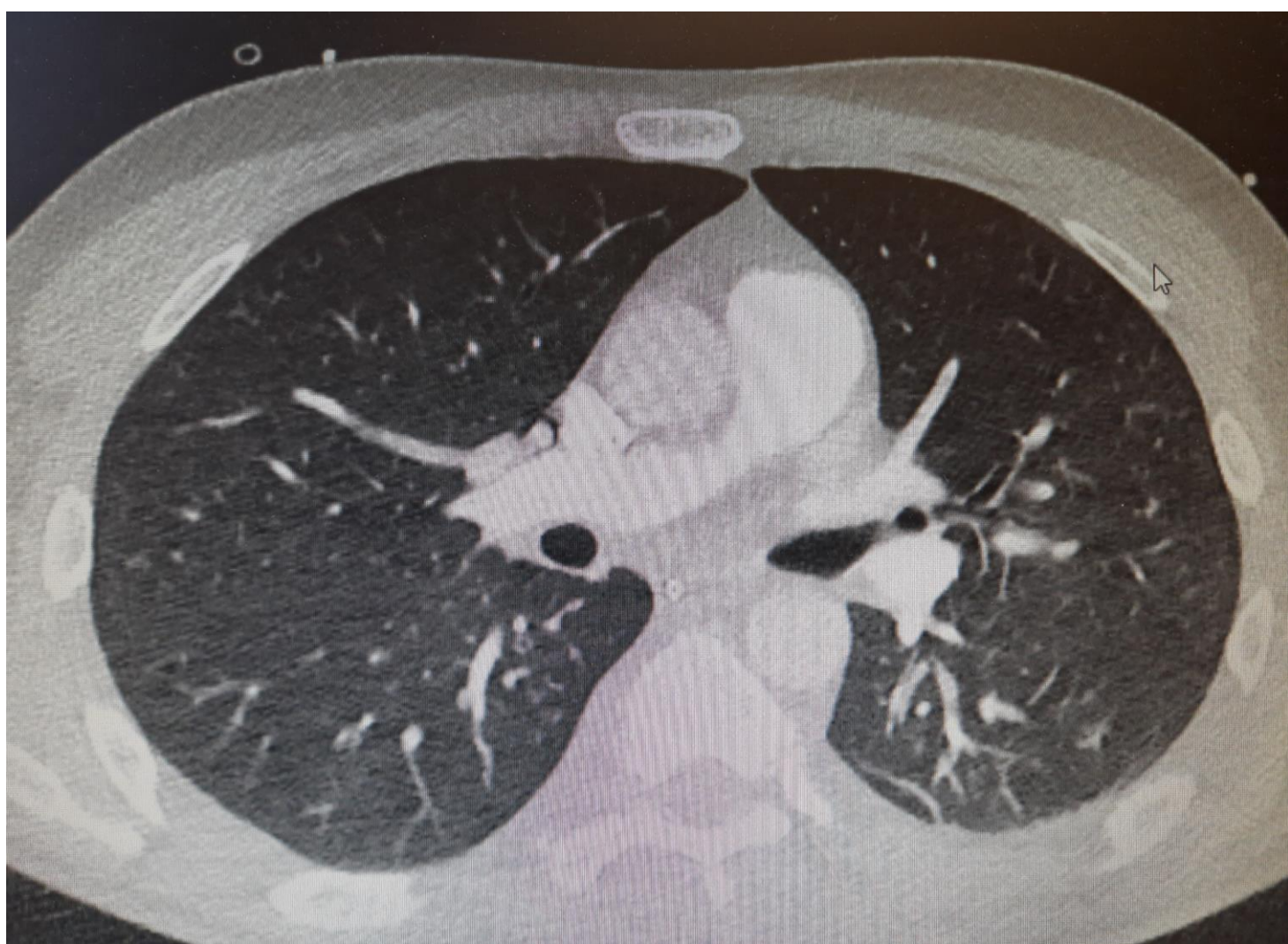


Fig 1: Day 9: CT Pulmonary angiogram: Normal lung parenchyma & no evidence of pulmonary embolism

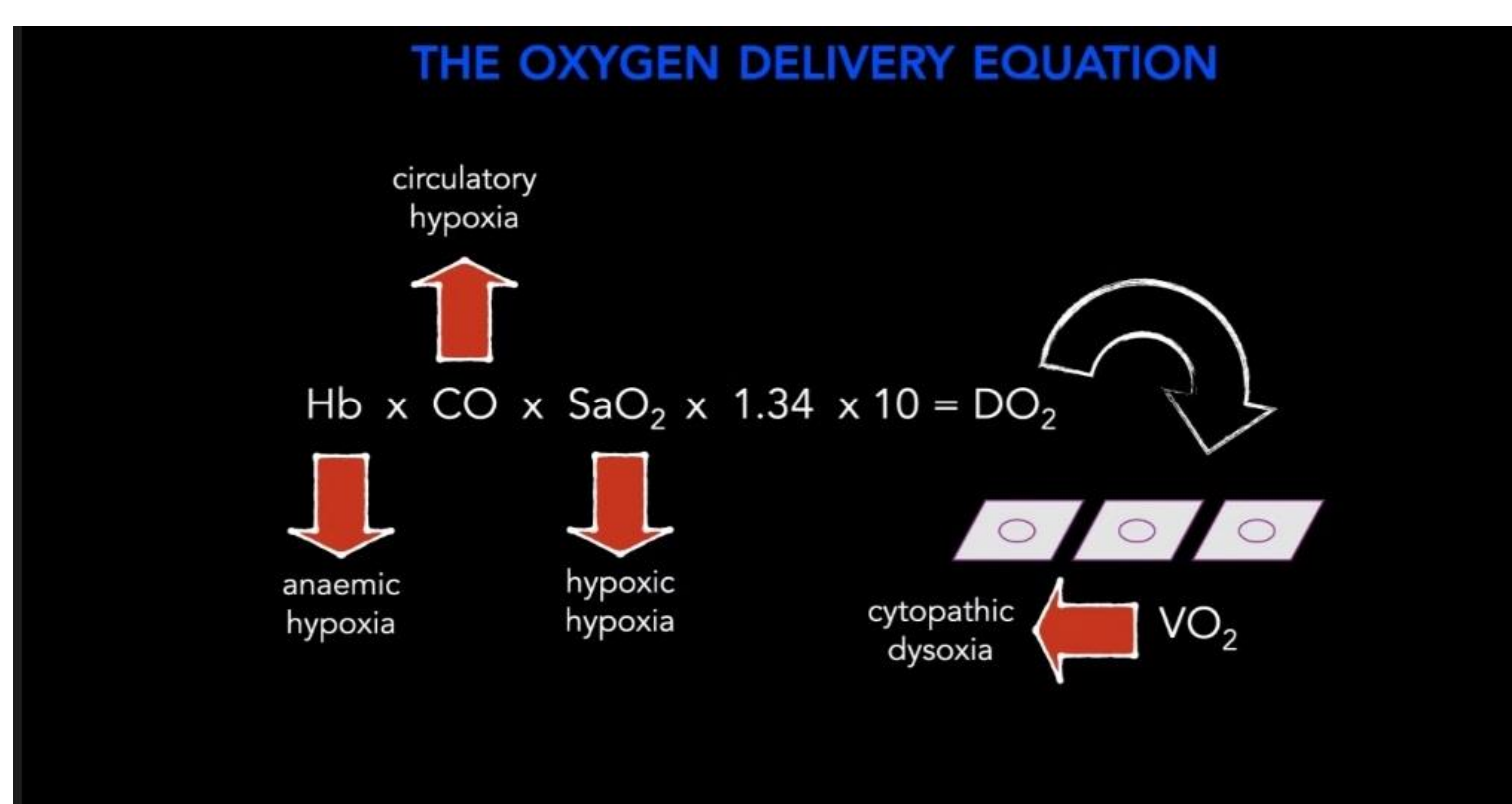


Fig 2: The Oxygen delivery equation – Pathophysiology of sepsis – Hypoxia - Merwyn Singer

Case Description

A male in his mid 30's with no significant past medical history presented to our neuro-specialist centre with **one day history of severe lower back pain associated with lower limb weakness and numbness.**

His symptoms, which commenced **10 days post his Covid vaccination**, rapidly progressed over 2 days of hospital admission to involve right upper limb & facial weakness.

MRI scan of brain and spine showed features of **ADEM** and pulse Methylprednisolone was initiated. CT thorax and abdomen on admission was unremarkable.



He was **transferred to the critical care** unit in view of progressive ascending paralysis and was **intubated on his 5th inpatient day** due to involvement of respiratory muscles.

Following 4 cycles of plasma exchange with albumin (day 6,7,9 and 10 of hospital admission), he **developed unexplained hypoxic episodes** which eventually resulted in sustained hypoxia, requiring 100% oxygen.



Airway pressures and lung compliance were within normal range. Bedside ultrasound demonstrated good lung sliding in all lung fields and good left ventricular contractility with no evidence of right ventricular dilatation. There was no evidence of pericardial/pleural effusion.



CT thorax repeated on day 9 showed no features of acute thromboembolic disease and there were no signs of lung parenchymal involvement.

Formal echocardiography with bubble test showed normal heart with no evidence of patent foramen ovale. **Multi-disciplinary discussions** involving cardiology, respiratory, neurology teams and regional ECMO centre **could not explain the enigma of impaired oxygenation.**

The patient responded well to 16 hours of prone ventilation on day 10 with decreasing oxygen requirements.



In the subsequent 3 months of his inpatient stay, he was weaned off oxygen and was tracheostomised in view of his neurological illness.

He continues to receive physiotherapy and neuro rehabilitation which had led to clinical improvement.

Conclusion: The possible reason for hypoxia could be impaired tissue oxygenation post plasma exchange.^{5,6} However, it could be a coincidental finding and there is not much literature to explain this phenomenon and warrants further research.

References

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