Case Report

Extra corporeal carbon dioxide removal: A reliable modality in refractory hypercapnia to prevent invasive ventilation

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Abstract

Extracorporeal carbon dioxide removal (ECCO₂R) is a valid alternative to consider in hypercapnic respiratory failure in chronic obstructive pulmonary disease (COPD) patients to avoid invasive ventilation when noninvasive ventilation fails. Here we report a similar case, after obtaining informed consent, where a patient suffering from severe hypercapnic respiratory failure due to COPD, was selected for ECCO₂R and improved remarkably.

Keywords: Chronic obstructive pulmonary disease, hypercapnia, respiratory failure

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Introduction

Extracorporeal carbon dioxide removal (ECCO₂R) is a partial form of extracorporeal lung assist (ECLA), which can be used in the management of refractory hypercapnic respiratory failure.^[1] It is a valid alternative to consider in hypercapnic respiratory failure in chronic obstructive pulmonary disease (COPD) patients to avoid invasive ventilation when noninvasive ventilation fails.^[2]

Here we report a similar case, after obtaining informed consent, where a patient suffering from severe hypercapnic respiratory failure due to COPD, was selected for ECCO₂R and improved remarkably.

Case Report

We present a case report of a 73-year-old male (91.5 kg, height - 184 cm) referred to our hospital with acute on

chronic COPD exacerbation and Type 2 respiratory failure not responding to conventional noninvasive ventilation. Later patient developed multiple organ dysfunction syndrome with Type 2 respiratory failure with mixed acidosis.

His presenting blood gases showed profound acidemia. pH - 7.057, base excess - 7.5, PCO₂-11.23 kPa, PaO₂-13.7 kPa on FiO₂-50%. P: F ratio was around 200. His inflammatory markers were high (total leukocyte count [TLC] $26 \times 10^9/L$, procalcitonin 11.2 mcg/L, C-reactive protein [CRP] 104 mg/L) and renal functions worsened and he was oliguric.

He was started on appropriate antibiotics and noninvasive ventilation. He was started on noradrenaline infusion to target mean arterial pressure of 70 mm Hg. Continuous veno-venous hemodiafiltration instituted to manage his renal failure with an aim for a negative fluid balance of 1.5 L. Hemodynamic monitoring was done by pulse-induced contour cardiac output.

After 12 h, he continued to be acidotic profoundly because of raised PaCO₂-10.86 kPa. Failure to remove CO₂ was noted despite being on non-invasive ventilation. His chest was clear B/L on auscultation. His vasopressors

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