Refractory hypercapnia in asthma exacerbation. Utility of extra-corporeal carbon dioxide removal ($ECCO_2R$) to facilitate respiratory weaning: a case report

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Introduction

Asthma exacerbations can be life-threatening and require ICU admission. Treatment strategies focus on promoting bronchodilatation with inhaled b2-agonists, muscarinic antagonists and magnesium sulphate, reducing inflammation with systemic corticosteroids and reducing dispnea and work of breathing. Correction of hypoxemia and hypercapnia is a key in managing life-threatening asthma. In severe hypoxic/hypercapnic cases which fail conservative therapies and non-invasive ventilation (NIV), endotracheal intubation and mechanical ventilation should not be delayed. However, mechanical ventilation in these patients often requires controlled hypoventilation, adequate sedation and occasional use of muscle relaxation to avoid dynamic hyperinflation, which can result in barotrauma or volutrauma[1]. In these cases, extra-corporeal carbon dioxide removal ($ECCO_2R$) in combination with continuous renal replacement therapy (CRRT) could improve respiratory acidosis and facilitate clinical stabilization and respiratory weaning.

Case report

A 53 years-old female, smoker, affected by COPD, presented to emergency department (ED) with a history of dyspnea and a mild hypoxic respiratory failure (PaO_2 47 mmHg). Empiric antibiotic therapy started despite no pneumonia evidence at CT scan. Due to a worsening in respiratory mechanics and acidosis (pH 7.19, $PaCO_2$ 91 mmHg), despite a maximal medical therapy with bronchodilators (ipratropio bromide, albuterol, topic steroid) and systemic corticosteroids and because of a NIV trial failure, endotracheal intubation and ICU admission were performed.

In ICU, dynamic hyperinflation persisted (autoPEEP 21 cmH2O with PEEP 0) despite maximal bronchodilator therapy with endobronchial (eb) and intravenous (iv) epinephrine, magnesium sulfate iv, albuterol iv and inhalatory Sevoflurane, in addiction to Midazolam and Ketamine sedation and neuromuscular blockage. At this point, $ECCO_2R$-CRRT treatment was introduced (*Figure 3*). A continuous venovenous hemodiafiltration (CVVHDF) has been set with standard parameters to correct mixed acidosis (*Figure 1*). For decarboxylation, a small standard hollow-fiber gas exchanger membrane (multi$ECCO_2R$) was applied, integrated after dialysis filter. The main aim was to reduce mechanical ventilation and thus avoid barotrauma and volotrauma by directly removing CO_2 with $ECCO_2R$ and by increasing serum bicarbonate with CRRT. Maximal blood flow and reduced minute ventilation (8mL/kg predicted body weight (PBW) with protective pressure plateau) have been set, observing a gradual decreasing in $PaCO_2$ (pH 7.33, $PaCO_2$ 51 mmHg) (*Figure 2*).

On the seventh day of treatment hemofilter thrombosis occured so the CO_2 membrane was removed. Immediate clinical worsening occurred for evidence of respiratory acidosis (pH 7.06, $PaCO_2$ 107 mmHg), so $ECCO_2R$-CRRT has been restored with respiratory stabilization (pH 7.46, $PaCO_2$ 48 mmHg) (*Table 1*).

Owning a positive response from the applied treatments, $ECCO_2R$-CRRT was continued for two weeks, in conjunction with respiratory weaning and de-escalation of medical therapy (stop of neuromuscular blockade, epinephrine eb/iv, albuterol iv, etc.). Percutaneous tracheotomy was performed and home assisted ventilation started due to a severe critical illness polyneuropathy (CIP) requiring long lasting ventilation.

Finally just over a month, the patient was discharged from ICU and she was hospitalized in sub-respiratory department. The patient is still alive in the one year follow up.

Figure 1

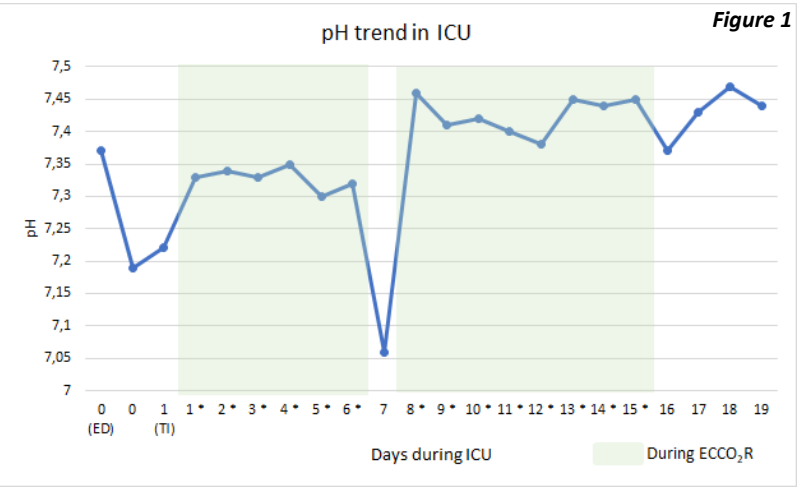
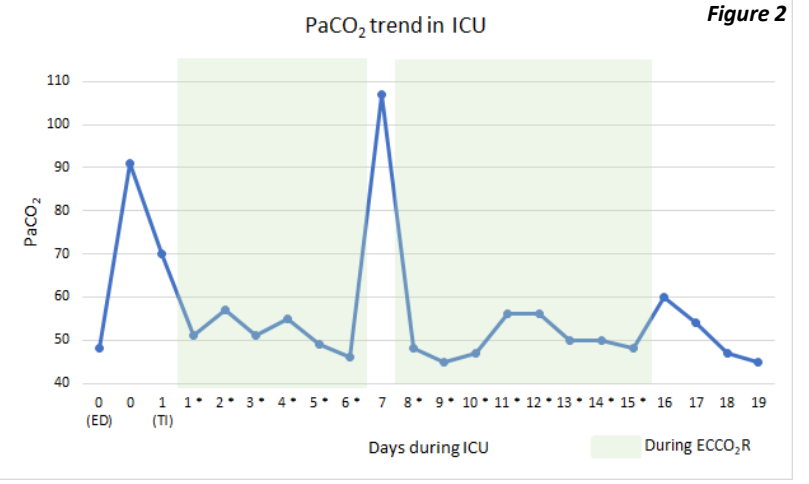


Figure 2



Conclusion

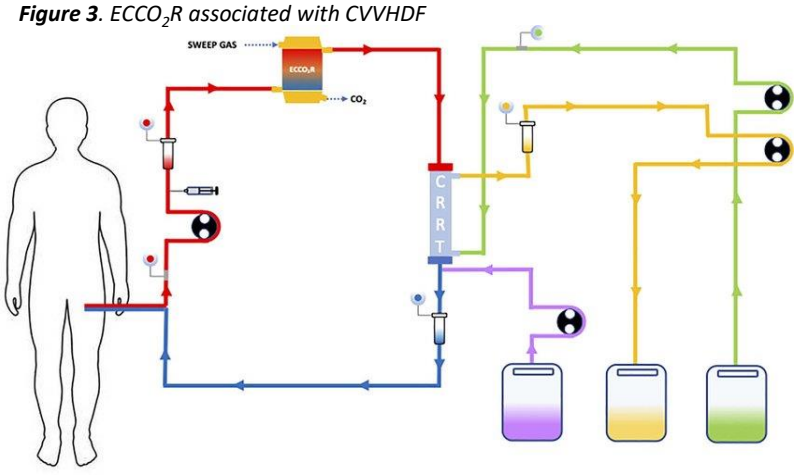
$ECCO_2R$-CRRT treatment has proven to be effective in improving CO_2 elimination and facilitating acid-base balance. This device also has found application during sedative and respiratory weaning, supporting arousal and passage to spontaneous breathing. Furthermore, it has proven to be safe, well tolerated without any adverse event.

Table 1. *Ventilatory and ABG data during ICU stay*

Days from ED	pH	$PaCO_2$ (mmHg)	PaO_2 (mmHg)	HCO_3^- (mmol/L)	TV (mL)	VE (L/min)	PEEP (cmH_2O)	PEEPi (cmH_2O)	Ppeak (cmH_2O)	Pplat (cmH_2O)
0	7,37	48	47	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
0 (ED)	7,19	91	235	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
1 (TI)	7,22	70	127	28	400	6,4	n.a.	+21	n.a.	n.a.
1 *	7,33	51	163	27	420	7,6	0	+15	32	n.a.
4 *	7,35	55	128	27	480	6,7	0	+12	28	18
6 *	7,32	46	106	23	460	7,8	0	+13	32	22
7	7,06	107	131	29	460	7,8	0	+10	n.a.	n.a.
8 *	7,46	48	83	34	540	7,6	0	+11	33	19
11 *	7,4	56	116	30	520	7,3	0	+10	30	17
15 *	7,45	48	97	32	540	7,6	0	+11	22	18
16	7,37	60	92	34	PSV	n.a.	+4	+11	30	n.a.
19	7,44	45	92	30	520	7,3	0	+7	n.a.	n.a.

*n.a. = not available, * = during ECCO2R-CRRT*

Figure 3*. $ECCO_2R$ associated with CVVHDF*



Reference [1] *Garner O, Ramey JS and Hanania NA. Management of Life-Threatening Asthma. CHEST 2022; 162(4):747-756*