

Rescue therapy with high-frequency oscillatory ventilation in severe acute respiratory distress syndrome

S.M.Khoo, K.H.Lee

Abstract

There is emerging evidence on the effectiveness and safety of high-frequency oscillatory ventilation in patients with acute respiratory distress syndrome. We described two patients with severe acute respiratory distress syndrome in whom low tidal volume ventila-

tion with conventional mechanical ventilator failed to achieve physiological improvement. The initiation of high-frequency oscillatory ventilation as rescue therapy in these patients resulted in remarkable improvement in oxygenation and hypercapnia.

Keywords: High-frequency oscillatory ventilation, acute respiratory distress syndrome, low tidal volume ventilation.

Introduction

With increased understanding of the pathophysiology of acute respiratory distress syndrome (ARDS) and the adverse consequences of ventilator induced lung injury, other strategies of respiratory support including high frequency oscillatory ventilation (HFOV) have been used in the hope of improving outcomes in these patients. In the ARDSnet trial, low tidal volume ventilator strategy (6mL/kg) was found to be associated with 8.8% reduction in absolute mortality compared with the traditional tidal volume (12mL/kg) [1]. HFOV, a ventilation modality which achieves gas exchange utilizing subdeadspace tidal volume, offers a less traumatic method of recruiting and stabilizing lung volumes than other conventional modalities.

We present two case reports of the successful use of HFOV as rescue therapy in patients with ARDS who had physiological failure on low tidal volume conventional mechanical ventilation.

Case Reports

Case 1

A 58-year-old man was brought to the emergency room with a history of cough, fever and dyspnoea. He was found to be in shock with blood pressure of 68/39 mmHg, heart rate 115/minute, respiratory rate 28/minute and temperature 35.3 degree Celsius. Chest X-ray showed bilateral basal opacities. Haemoglobin was 12.6 g/dL, white cell count was $12.65 \times 10^9/L$, platelet was $125 \times 10^9/L$, Sodium was 145 mmol/L, potassium was 3.8 mmol/L, urea was 5.5 mmol/L and creatinine was 238 mmol/L, glucose 11.4 mmol/L, lactate was 12.8 mmol/L. Arterial blood gas done when patient was on non-rebreather mask showed pH 7.19, arterial carbon dioxide tension (PaCO₂) of 39.5 mmHg, arterial oxygen tension (PaO₂) of 161.5 mmHg, base excess ~ 12.5 mmol/L and standard bicarbonate 14.8 mmol/L. A diagnosis of severe community acquired pneumonia with septic shock was made. Aggressive fluid resuscitation, inotropic support and intravenous antibiotics were commenced immediately. Despite that, he progressed rapidly to multi-organ failure and acute respiratory distress syndrome. He was intubated and put on mechanical ventilation. On day one in the intensive care unit, he was ventilated on a conventional ventilator on assist-control mode with a mean airway pressure (mPaw) of 39 mmHg and positive end-expiratory

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pressure (PEEP) of 17 cmH₂O. Delivered tidal volume (V_t) of 500mL (6.5 mL/kg). While breathing fraction of inspired oxygen (FiO₂) of 1.0 his arterial blood gas showed pH 7.23, PaCO₂ of 57.2mmHg, PaO₂ of 53.4 mmHg, base excess ~6.1 mmol/L and standard bicarbonate 19.3 mmHg. The patient was already deeply sedated and a neuromuscular blocking agent was administered. On day two, his respiratory status worsened despite a mPaw of 39 cmH₂O and decreased tidal volume. Because of concern about worsening oxygenation and increasing PaCO₂, he was placed on a high-frequency oscillatory ventilator (HFOV) (SensorMedics 3100 B, Yorba Linda, CA). The mPaw was initially set at 44 cmH₂O, 5 cmH₂O above the mPaw applied during conventional mechanical ventilation. Serial mPaw, FiO₂, arterial blood gas results are as shown in table one (patient 1). Few hours after initiation of HFOV, the patient's oxygenation improved (PaO₂/FiO₂ improved from 53 to 89), but the PaCO₂ also increased. The amplitude of oscillation was increased from 44 cmH₂O to 76 cmH₂O and FiO₂ was decreased to 0.6. Over the next few days, he continued to respond well to HFOV with remarkable physiological improvement. He was returned to conventional mechanical ventilation on day seven and successfully extubated on day fifteen. Unfortunately, he developed nosocomial pneumonia and passed away after being in the hospital for one and a half months.

Case 2

A 43-year-old man with a history alcohol and intravenous drug abuse was admitted to this hospital with cough, yellow sputum production, weight loss and fever. Chest X-ray showed bilateral diffuse infiltrate and left upper zone cavitating pneumonia. Subsequent investigations confirmed extensive pulmonary tuberculosis and tuberculous pericarditis. He was started on rifampicin, isoniazid and pyrazinamide. Despite that, his condition worsened and he developed type II respiratory failure. His arterial blood on non-rebreather mask was pH 7.24, PaCO₂ 128 mmHg, PaO₂ 101.7 mmHg, base excess 10 mmol/L and standard bicarbonate 31.5 mmol/L. His chest X-ray showed worsening bilateral infiltrates. He was intubated and ventilated on conventional ventilator. Assist-control mode was used with a mean airway pressure of 37 cmH₂O and a PEEP of 12 cmH₂O. Delivered tidal volume was 390 mL (6.5mL/kg). While breathing FiO₂ of 1.0, his blood gas showed a pH of 7.27, PaCO₂ of 71 mmHg, PaO₂ of 42 mmHg, base excess of 4.6 mmol/L and standard bicarbonate of 28 mmol/L. In view of the worsening oxygenation, high PaCO₂ despite high mean airway pressures, HFOV (SensorMedics 3100 B, Yorba

Linda, CA) was initiated. The mPaw was initially set at 34 cmH₂O and amplitude of 50 cmH₂O. Remarkable improvement in oxygenation and hypercapnia was observed (Table 1, patient 2). He was subsequently put back on conventional mechanical ventilation. Unfortunately, he developed intractable shock and passed away seven days after his admission to intensive care unit.

Discussion

We described two patients with severe ARDS in whom the use of low tidal volume with conventional mechanical ventilator failed to achieve physiological improvement. The initiation of HFOV in these patients as rescue therapy resulted in remarkable improvement in oxygenation and hypercapnia. To the best of our knowledge, this is the first report that showed the effectiveness of HFOV as a rescue therapy in ARDS patients who continued to deteriorate despite the application of low tidal volume ventilation (6.5mL/kg in both cases) with conventional ventilator.

ARDS is a common clinical disorder characterized by injury to the alveolar epithelial and endothelial barriers of the lung, acute inflammation and protein rich pulmonary oedema leading to acute respiratory failure. Conventional mechanical ventilation commonly used in this group of patients, which involves high tidal volume and low PEEP is likely to contribute to progressive lung injury [1]. The benefit of low tidal volume ventilation was confirmed in the ARDSnet trial which found that low tidal volume ventilation was associated with 8.8% reduction in absolute mortality. In a randomized controlled trial by Amato et al, lung-protective strategy (tidal volume 6mL/kg; PEEP targeted above the lower inflection point on the quasi static pressure-volume curve) was associated with improved 28-day survival [2]. These data suggest that an ideal ventilator strategy should be one that avoids both over-distension and derecruitment of the atelectasis-prone lung in ARDS.

Even though it is possible to achieve lung-protective strategy with conventional ventilator, it is not always easy to identify the ideal level of PEEP in patients with ARDS. This is because many patients with ARDS have an upward concavity of the pressure/volume curve without a clear inflection point, possibly suggesting a broad distribution of opening pressure in an inhomogeneously affected lung [3]. Study had shown that the PEEP levels that maintained alveolar re-expansion using a conventional ventilatory method were scattered along a steeply sloping portion of the deflation limb of the pressure/volume curve [4]. HFOV is able to maintain

Table 1

	ICU Day	Vent Mode	mPaw (cmH ₂ O)	FiO ₂	pH	PaCO ₂ (mmHg)	PaO ₂ (mmHg)	PaO ₂ /FiO ₂
Patient 1	1	NRM	-	1.0	7.19	39.5	161.5	161
	2	AC						
	10:00	PEEP17	39	1.0	7.23	57.2	53.5	53
	12:30	HFOV	44	1.0	7.14	72.2	89.0	89
	14:00	HFOV	39	0.6	7.22	59.8	157.8	263
	04:00	HFOV	27	0.3	7.32	54.2	176.6	588
	Patient 2	1	AC					
16:00		PEEP12	37	1.0	7.27	71	42	42
2								
20:00		HFOV	34	1.0	7.36	58	50	50
04:00		HFOV	39	1.0	7.25	58	169	169
21:00		HFOV	25	0.4	7.37	55	143	357

mPaw = mean airway pressure
 FiO₂ = fraction of inspired oxygen
 PaCO₂ = arterial carbon dioxide tension
 PaO₂ = arterial oxygen tension
 NRM = non-rebreather mask
 AC = assist-control mode
 PEEP = positive end-expiratory pressure
 HFOV = high-frequency oscillatory ventilation

the end-expiratory lung volume higher up on the relatively flat portion of the deflation pressure/volume relationship, without inducing concurrent overdistention, because of the much smaller tidal volumes. HFOV potentially offers the ideal combination of minimum tidal volume while maintaining maximal recruitment (the open lung), provided sufficient end expiratory volume is maintained. It involves the application of rapid oscillatory pressure at 5 to 15 Hz, of 300 to 900 breaths per minute, superimposed on a continuous distending pressure.

In the two cases described, both patients developed worsening hypercapnia when ventilated on low tidal volume with conventional ventilator. The benefit of using low tidal volume in conventional mechanical ventilation is realized at the expense of hypercapnia (permissive hypercapnia). However, the pulmonary and systemic effects of hypercapnic acidosis could adversely affect the outcomes of patients with ARDS and multi-organ failure [5]. The ability of HFOV to maintain gas exchange despite the small tidal volumes means that the benefit of

lung-protective strategy can be achieved without the cost of hypercapnia and the systemic effects associated with it.

There are few data on the use of HFOV in adult patients. In an observational study of 17 patients with ARDS, HFOV was reported to be effective and safe [6]. Mehta and co-workers found in a prospective trial that HFOV was effective as a rescue therapy in patients with severe oxygenation failure and early institution of HFOV may be beneficial [7]. A recently published multicentre, randomized, controlled trials comparing HFOV and conventional mechanical ventilation in 148 patients with ARDS showed that HFOV was associated with early improvement in PaO₂/FiO₂ and a non-significant trend toward reduced mortality at 30 days and 6 months [8].

Despite the demonstrable benefit of lung-protective conventional mechanical ventilation based on low tidal volume and high PEEP, there still may be ways to reduce morbidity and mortality even further [9]. HFOV, by way of its unique mechanical properties, may accomplish all of the goals of lung protection and thus

could be more effective at mitigating ventilator-induced lung injury. Further studies are needed to determine the role of HFOV in patients with severe ARDS who fail to respond to lung-protective conventional mechanical ventilation.

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