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An adult with ARDS managed with high-frequency oscillatory ventilation and prone position

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Introduction

A protective strategy to prevent ventilator-induced lung injury has been proved to improve outcome in patients with acute respiratory distress syndrome (ARDS) [1,2]. With this strategy, low tidal volume is used to minimize alveolar stretch, resulting in various degrees of hypercapnia. High-frequency oscillatory ventilation (HFOV) is another mode of ventilation for lung protection. HFOV has been reported to improve the outcomes in neonates with respiratory distress syndrome [3] and in pediatric patients with acute respiratory failure [4]. Recently, its clinical use has been increasing in adult patients [5,6].

We experienced an adult patient with ARDS managed with HFOV and prone position. Ventilation improved immediately with HFOV, and oxygenation improved with prone position during HFOV. The duration of HFOV was about 38 h, and no adverse effects were observed.

Case report

A 76-year-old male patient (body weight, 67 kg) underwent subtotal esophagectomy for esophageal cancer. After the operation he was mechanically ventilated with a PB-7200 ventilator (Puritan-Bennett, Carlsbad, CA, USA) in the intensive care unit. He had received chemotherapy (but no radiation therapy) before the operation. The preoperative chemotherapy had been started 11 days before the operation and had finished 9 days before the operation.

On the first postoperative day, a chest radiograph revealed bilateral infiltrates, and he was diagnosed as having acute lung injury (ALI), or ARDS, according to the definition of the American-European consensus conference on ARDS [7]. On the second postoperative day, marked neutropenia was observed (neutrophils, 200/μl; leukocytes, 1800/μl), and granulocyte colony-stimulating factor (G-CSF; 75 μg/day) was administered subcutaneously for 2 days. On the third postoperative day, oxygenation and hemodynamics deteriorated after the second administration of G-CSF. One hour before the second administration of G-CSF, arterial oxygen tension (PaO_2) was 73.3 mmHg and arterial carbon dioxide tension ($PaCO_2$) was 39.6 mmHg with pressure support ventilation; fractional inspired oxygen (FIO_2) was 0.7, positive end-expiratory pressure (PEEP) was 7 cmH_2O, and pressure support, 18 cmH_2O (PaO_2/FIO_2 (P/F) ratio, 104.7). Two hours after the second G-CSF, PaO_2 was 47.6 mmHg and $PaCO_2$ was 36.4 mmHg with the same ventilator settings (P/F ratio, 68.0). Arterial blood pressure was 75/45 mmHg, and central venous pressure was 15 mmHg with dopamine, 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and dobutamine, 4 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Although his hemodynamics improved with norepinephrine, 0.2 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and dopamine, 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$, urine output transiently decreased and continuous hemodiafiltration (CHDF) was started. Oxygenation did not improve and ventilation worsened in spite of the use of aggressive ventilator settings under conditions of sedation (continuous infusion of fentanyl and propofol) and muscle relaxation. PaO_2 was 62.6 mmHg and $PaCO_2$ was 52.4 mmHg (pH 7.28) with pressure-controlled ventilation (PCV); FIO_2, 1.0; PEEP, 10 cmH_2O; distending pressure, 25 cmH_2O; respiratory frequency, 30/min. This resulted in a tidal volume of 0.35 l, minute volume of 10.5 l/min, mean airway pressure (MAP) of 22.5 cmH_2O,

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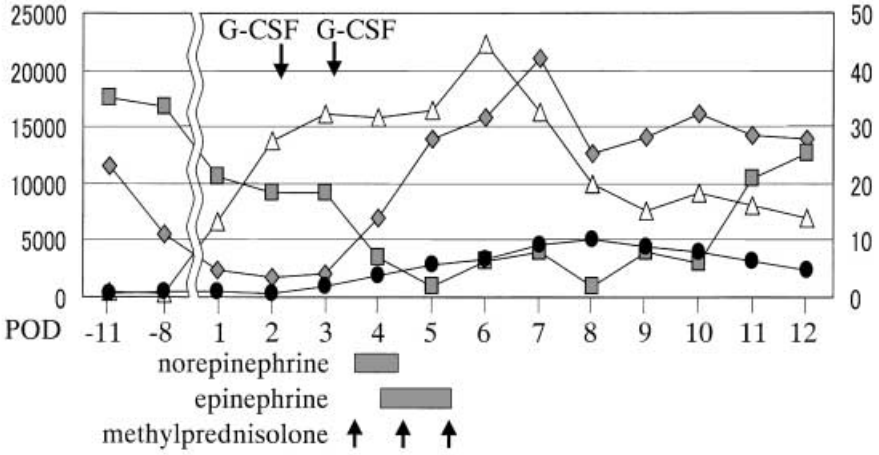

Fig. 1. Changes in leukocyte count (per μl; *gray diamonds*), platelet count (×10/μl; *gray squares*), C-reactive protein (mg/dl; *white triangles*), and total bilirubin (mg/dl; *black circles*). *G-CSF*, Granulocyte colony-stimulating factor, *POD*, postoperative day. Leukocyte and platelet counts shown on the *left*; C-reactive protein and total bilirubin on the *right*

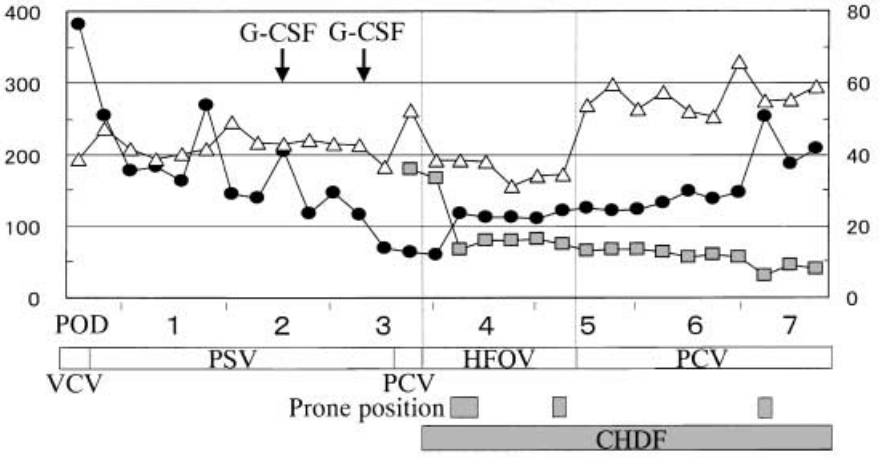

Fig. 2. Changes in arterial oxygen tension (PaO_2)/fractional inspired oxygen (FIO_2) (P/F) ratio (mmHg; *black circles*, on the *left*), and arterial carbon dioxide tension ($PaCO_2$; mmHg; *white triangles*, on the *right*), and oxygenation index (OI; *gray squares*, on the *right*). OI = Mean airway pressure × FIO_2 × 100/PaO_2. *G-CSF*, Granulocyte colony-stimulating factor; *POD*, postoperative day; *VCV*, volume-controlled ventilation; *PSV*, pressure support ventilation; *HFOV*, high-frequency oscillatory ventilation; *PCV*, pressure-controlled ventilation, *CHDF*, continuous hemodiafiltration

and an oxygenation index (MAP × FIO_2 × 100/PaO_2; OI) of 35.9.

We decided to use HFOV for lung protection. We used a newly developed prototype HFO ventilator for adults (Suzuki-Metran, Tokyo, Japan). Informed consent was obtained from the patient's family. The initial settings of the HFOV were: FIO_2, 1.0; MAP, 20 cmH_2O; frequency, 9 Hz; and stroke volume (SV), maximum. Although oxygenation did not improve with HFOV, ventilation improved immediately. Two hours after HFOV, PaO_2 was 63.7 mmHg and $PaCO_2$ was 37.0 mmHg (pH 7.32), with FIO_2 1.0; MAP, 20 cmH_2O; frequency, 9 Hz; and SV, 85% of maximum. Gas exchange did not change for the subsequent 9 h; the patient was then placed in the prone position for 6 h on the fourth postoperative day. Twenty minutes after he had been placed in the prone position, oxygenation had improved (PaO_2, 110.1 mmHg). Then FIO_2 and MAP decreased, to 0.75 and 18 cmH_2O, respectively. The placement in the prone position was repeated for 3 h on the fifth postoperative day.

After the placement of the patient in the prone position for the second time, HFOV was switched to PCV. The duration of HFOV was about 38 h, and no adverse effects were observed. Before the switching to PCV, PaO_2 was 84.2 mmHg and $PaCO_2$ was 40.2 mmHg with HFOV; FIO_2 was 0.7; MAP, 18 cmH_2O; frequency, 9 Hz; and SV, 85% of maximum (P/F ratio, 120.4; OI, 14.9). Three hours after the switching to PCV, PaO_2 was 84.9 mmHg and $PaCO_2$ was 51.4 mmHg with conventional PCV; FIO_2 was 0.65; PEEP, 10 cmH_2O; distending pressure, 20 cmH_2O; respiratory frequency, 20/min. This resulted in a tidal volume of 0.38 l, minute volume of 7.6 l/min, and MAP of 16.0 cmH_2O (P/F ratio, 130.6; OI, 12.2).

Figure 1 shows the changes in laboratory data and Fig. 2 shows the changes in the P/F ratio, $PaCO_2$, and OI. There were no apparent infections during the early postoperative period. On the seventh postoperative day, the P/F ratio exceeded 200 and FIO_2 was decreased to 0.5. The patient was weaned from CHDF on the eighteenth postoperative day. Mechanical ventilation

was needed until the forty-second postoperative day because of left phrenic nerve paralysis and bacterial pneumonia, and then the patient was transferred to a general ward.

Discussion

In our patient, HFOV immediately improved ventilation, but not oxygenation. In HFOV, "a high-lung volume strategy", using a higher MAP than that of conventional mechanical ventilation, has proven to be effective and important in improving oxygenation [3–5]. Because of the patient's unstable hemodynamics, we used a lower MAP than that of PCV. We performed sustained inflation, using a pressure of 35 cmH_2O for 30 s when starting HFOV. This pressure was same as the plateau pressure during PCV. As a result, oxygenation with HFOV was same as that with PCV (same level of P/F ratio, but lower OI). This means that the number of lung units that were open and ventilated was not increased with HFOV. A recent animal study, embracing the open lung concept, [8] has shown that HFOV and PCV had the same effect on oxygenation when an identical MAP was used after the same recruitment maneuver. This study well explains why oxygenation was not improved in our patient. To improve oxygenation with HFOV, not only a higher MAP but also sustained inflation using higher pressure may be essential to recruit more lung units and keep them open.

Oxygenation was immediately improved with the first trial of the prone position during HFOV. The effect of the prone position is generally thought to be brought about by better recruitment and/or better ventilation/perfusion matching [9–11]. Because improved oxygenation continued after the patient was returned to the supine position, it is conceivable that better recruitment was obtained with the prone position and was maintained with HFOV after the return to the supine position. This may explain why the second trial of the prone position was less effective in improving oxygenation than the first trial. Three hours after the switching of HFOV to PCV, oxygenation had improved further. This may have occurred because we did not perform sustained inflation periodically during HFOV. If sustained inflation had been performed periodically, some further recruitment and further improvement of oxygenation may have been obtained. Although CHDF was performed during and after HFOV, the patient's clinical course shows that CHDF had no direct relationship to the improvement of oxygenation during or immediately after HFOV.

With the changing of PCV to HFOV, ventilation improved immediately. The prototype HFOV machine we used produces a maximum SV of 140 ml at 9 Hz and a maximum SV of 80 ml at 15 Hz with an 8-mm endotracheal tube. The reason that we used 9 Hz is that it is the lowest frequency in this machine. In HFOV, a frequency of 10 to 15 Hz has been used for neonates [3], 5 to 10 Hz for pediatric patients [4], and around 5 Hz for adults [5,6]. The reason that around 5 Hz has been used for adults [5,6] is that it was difficult to obtain enough ventilation at a higher frequency, because of the limited capacity of the machine used. In HFOV, the SV required to get the same alveolar ventilation becomes lower as the frequency becomes higher. On the other hand, the ventilatory capacity of the HFOV machine is reduced when a higher frequency is used because the SV produced by the machine becomes much lower with higher frequency. If a lower SV produces less ventilator-induced lung injury in HFOV, a higher frequency may be beneficial for ARDS lungs.

Although G-CSF has been used safely in patients in intensive care units [12,13], there are a few reports that suggest that G-CSF can induce ARDS [14–16]. Takahashi et al. [16] showed that the use of G-CSF increased the incidence of ARDS caused by pulmonary infection in patients with hematological malignancy. In our patient, because the onset of ARDS occurred before the administration of G-CSF, the cause of ALI/ARDS was thought to be systemic inflammatory response syndrome (SIRS) following surgical stress. However our patient's status deteriorated rapidly after the second administration of G-CSF. Therefore, the G-CSF administered may have played a role as an exacerbating factor. We used methylprednisolone (1 g/day) for 3 days after the second administration of G-CSF. Steroid pulse therapy was reported to be very useful for the treatment of ARDS related to G-CSF administration [16], thus, the steroid treatment we employed may have contributed to the patient's successful outcome.

In conclusion, we have reported an adult with ARDS that was successfully managed with HFOV and the prone position. We suggest that HFOV may be a useful new tool for adult patients with ARDS.

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