Superimposed High Frequency Jet Ventilation for the Treatment of Cardiogenic Pulmonary Edema in a Case of Viral Myocarditis

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High frequency jet ventilation (HFJV) has been used to deliver adequate alveolar ventilation in patients with bronchopleural fistula[1] or in patients undergoing laryngomicrosurgery with a narrow endotracheal tube. It has also been reported to improve gas exchange in patients with postoperative respiratory failure[2]. However, the effectiveness of HFJV for the treatment of severe hypoxia unresponsive to conventional mechanical ventilation (CMV) with PEEP has not been reported. We report a patient with severe hypoxia due to cardiogenic pulmonary edema, who was successfully treated with superimposed HFJV (SHFJV) on the CMV.

Case Report

A 33-year-old man was admitted to a private hospital with the complaints of fever for 6 days and dyspnea for 3 days. Both oxygen therapy by mask and CMV under tracheostomy failed to improve his respiratory distress. He was then transferred to our intensive care unit (ICU) on the same day. He had no history of cardiac or pulmonary disease.

On admission to ICU, he had tachypnea (50 breaths·min^{-1}) and severe cyanosis despite of 100% oxyen via the tracheal cannula. Pink frothy sputum was aspirated. A chest X-ray showed diffuse bilateral pulmonary infiltrates consistent with pulmonary edema. The cardiothoracic ratio was 0.5. Initial arterial blood gas analysis revealed severe hypoxemia: Pa_{O_2}, 34 mmHg; Pa_{CO_2}, 31 mmHg; and pH, 7.44 (F_{IO_2}=1.0). The blood pressure was 115/60 mmHg and the pulse rate was 124/min. His temperature was 37.8°C. We immediately started mechanical ventilation using a Puritan Bennet 7200a ventilator in the intermittent mandatory ventilation (IMV) mode with PEEP of 10 cmH_2O and pressure support. Furosemide was used to facilitate diuresis. Dopamine was given at 7 to 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ to maintain the mean arterial blood pressure above 70 mmHg. One hour later, little improvement was observed in arterial blood gases: Pa_{O_2}, 38 mmHg; Pa_{CO_2}, 33 mmHg; and pH, 7.49 (F_{IO_2}=1.0). Hemodynamic measurements showed pulmonary artery pressure of 50/27 mmHg (mean, 37 mmHg), pulmonary artery occlusion pressure of 30 mmHg

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and right atrial pressure of 15 mmHg, all indicating severe pulmonary hypertension and left ventricular failure. The cardiac index was 2.9 $l \cdot min^{-1} \cdot m^{-2}$ and the mixed venous P_{O_2} was 23 mmHg. The alveolar to arterial oxygen tension difference ($A\text{-}aD_{O_2}$) and the intrapulmonary shunt (Qs/Qt) were 640 mmHg and 53%, respectively. Electrocardiography showed severe left ventricular ischemia. Laboratory studies revealed abnormal levels of GOT, GPT, bilirubin, BUN, creatinine, LDH and CPK. The white blood cell count, erythrocyte sedimentation rate, and C-reactive protein level were also elevated.

PEEP was increased so as to reduce the inspiratory oxygen concentration. At that time, the real ventilatory mode was pressure support ventilation with PEEP, because he was often breathing faster than the IMV rate. In addition to mechanical ventilation, pharmacological support was continued, including inotropic agents (dopamine, dobutamine), diuretics, and prostaglandin E_1 for pulmonary vasodilation.

Despite our usual regimen for pulmonary edema, he still showed poor oxygenation 24 hours after admission. The Pa_{O_2} was only 66 mmHg with 20 cmH_2O of PEEP and 20 cmH_2O of pressure suport on a $F_{I_{O_2}}$ of 0.6. At this stage, we superimposed HFJV on the CMV. He was then ventilated in the IMV mode at a rate of 14 per minute with PEEP of 20 cmH_2O, in combination with a Mera HFO Jet Ventilator set at a rate of 420 breaths$\cdot min^{-1}$, a driving pressure of 1 kgf$\cdot cm^{-2}$, and an insufflation time of 50%. The tidal volume on the CMV system was decreased during SHFJV to give the same peak airway pressure as during CMV, and saline (10 ml$\cdot hr^{-1}$) was dripped onto the tip of the HFJV system for humidification.

Soon after SHFJV was initiated, rapid rise of the continuously monitored venous oxygen saturation was observed. Within 30 minutes, the Pa_{O_2} increased from 66 mmHg to 144 mmHg on the same $F_{I_{O_2}}$. Simultaneously, $A\text{-}aD_{O_2}$ and Qs/Qt decreased from 321 mmHg to 249 mmHg and from 33% to 18%, respectively. Then the inspired oxygen fraction was reduced to 0.5. During SHFJV, the mean airway pressure was the same as during CMV and the Pa_{CO_2} remained constant at around 32 mmHg. Thereafter, the Pa_{O_2} gradually increased, but an attempt to return to CMV after 2 or 3 hours of SHFJV failed to maintain a satisfactory Pa_{O_2}. We kept inotropic support constant and the hemodynamic state including diuresis did not change before and during SHFJV.

Twelve hours after the start of SHFJV, its combined use had to be discontinued because hyperinflation of the lungs was detected by chest X-ray. The abrupt return to CMV at this time did not have an adverse influence on arterial blood gases, so SHFJV was discontinued and weaning from CMV was started carefully. Echocardiography performed on the third day after admission showed general akinesia and poor function of the left ventricle, so that myocarditis was strongly suspected. In the following days, the hemodynamic disorders and all laboratory findings gradually returned to normal. Throughout the course of his illness, the patient was alert, cooperative, and able to tolerate each mode of ventilation. On the 8th day after admission he was extubated and was transferred to the ward on the 10th day without any sequelae.

Serological examination revealed an elevated antiviral antibody titer to parainfluenza 3 virus that was more than fourfold above the control value.

Discussion

This case was diagnosed retrospec-

tively as cardiogenic pulmonary edema due to acute viral myocarditis. The possible etiologic agent in this case, parainfluenza 3 virus, is a very rare cause of myocarditis[3].

The usual active treatment for cardiogenic pulmonary edema, which consists of mechanical ventilation with PEEP, hemodynamic support using inotropic agents, and diuretics, failed to achieve sufficient oxygenation in this case. Although a very gradual increase in Pa_{O_2} was observed during CMV with PEEP, the persistent hypoxemia would have caused further damage to organs such as the liver, kidneys, and heart. Furthermore, high levels of PEEP greater than 20 cmH_2O would have depressed cardiac performance[4]. This is one of the reasons why we chose SHFJV in this patient. There are reports that HFJV or SHFJV do not have any effects on hemodynamics[5,6], and we also found no change of hemodynamics in this patient during SHFJV. The reason for the improvement of oxygenation in this case was not due to improved cardiac perfomance, pharmaceutical interventions nor effect of the detected mean airway pressure, because they did not change during the different modes of ventilation. Though the mechanism of the improvement in oxygenation induced by SHFJV remains unclear, it was probably due to alveolar PEEP (auto-PEEP)[2,7,8]. Rouby et al.[2] have demonstrated that HFJV can improve oxygenation with increasing levels of mean alveolar pressure. Thus SHFJV might increase alveolar pressure more markedly compared to CMV with PEEP. SHFJV certainly contributed to accelerate oxygenation in this case, it required, however, more than 2 or 3 hours for the alveolar reexpansion to be accomplished.

No previous report has shown that SHFJV is effective for cardiogenic pulmonary edema. Our experience might therefore shed some light on an alternative ventilaroty strategy for patients with cardiogenic pulmonary edema in whom appropriate oxygenation can not be obtained by CMV alone.

In summary, a marked improvement of oxygenation was observed during SHFJV in a patient suffering from severe hypoxemia due to cardiogenic pulmonary edema arising from acute viral myocarditis. This improvement could not be achieved by CMV alone.

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