

Original Article

Permissive hypercapnia combined with low-level PEEP in bullous emphysema patient undergoing cardiac surgery: a case report and literature review

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Abstract: Background: Bullous emphysema is characterized by destruction of lung parenchyma, reduction in elasticity of the lung, hyperinflation, and ventilation/perfusion mismatch. Ventilatory management of the patient with bullous emphysema is challenging, and especially so for the patient undergoing cardiac surgery. Case report: A 47-year-old man with severe diffuse bullous emphysema requested general anesthesia for tricuspid valve replacement under cardiopulmonary bypass (CPB). Preoperative physical examination detected signs of right heart dysfunction, arterial blood gas (ABG) analysis revealed the partial pressure of CO₂ in arterial blood (PaCO₂) was 62 mmHg. Echocardiogram detected severe tricuspid regurgitation and pulmonary hypertension. After induction of anesthesia, the lungs were ventilated using volume-controlled ventilation, with a low-tidal volume (6 mL/kg) and a low-level PEEP (5 cmH₂O). Under these conditions, ABG analysis revealed PaCO₂ 64 mmHg. The patient underwent tricuspid valve replacement under CPB. ABG analysis was performed at 20, 60, and 95 minutes following initiating CPB, the PaCO₂ was 43, 42, and 36 mmHg, respectively. Forty-five minutes after weaning CPB, ABG analysis showed that the PaCO₂ was 64 mmHg. The postoperative period was uneventful and there was not marked ventilator-associated pulmonary complications. Conclusions: Permissive hypercapnia combined with a low level of PEEP as a ventilatory strategy provided adequate oxygenation and minimized ventilator-induced lung injury in the patients with bullous emphysema undergoing cardiac surgery.

Keywords: Bullous emphysema, permissive hypercapnia, positive end-expiratory pressure, cardiac surgery, cardiopulmonary bypass

Introduction

Bullous emphysema is defined as an alveolar space with a diameter more than 1 cm, and characterized by destruction of lung parenchyma, reduction in elasticity of the lung, hyperinflation, and ventilation/perfusion mismatch [1, 2]. In these patients, maintaining a normal partial pressure of carbon dioxide in arterial blood (PaCO₂) requires a high respiratory airway pressure and a large tidal volume, both of which can lead to ventilator-induced lung injury (VILI).

Permissive hypercapnia, a ventilatory strategy providing low tidal volume and low inspiratory airway pressure, has increasingly been accepted as a protective ventilatory strategy to prevent VILI in patients with severe pulmonary diseases [3-6]. Here, we describe the successful

use of permissive hypercapnia and positive end-expiratory pressure (PEEP) in a patient with severe diffuse bullous emphysema who underwent tricuspid valve replacement under cardiopulmonary bypass (CPB), and we include a review of the anesthetic concerns that should be considered during the operation.

Case report

A 47-year-old man (height, 176 cm; weight, 52 kg) with severe tricuspid valve regurgitation was admitted to our hospital for tricuspid valve replacement. Eight years previously, he had undergone a mitral valve replacement under CPB at another institution. For the last year, he had complained of palpitations and shortness of breath after exercise. On admission, he presented with mild respiratory distress while walking.

Physical examination revealed bilateral pitting pedal edema up to the knees, and an increased anteroposterior diameter of the chest wall. The jugular venous pulsation was elevated up to the mid neck. Breath sounds were muffled with prolonged exhalation. The blood pressure was 108/64 mmHg, heart rate was irregular at 71 beats/min and respiratory rate was 21 breaths/min. Arterial blood gas (ABG) analysis via nasal cannula at oxygen flow 1.5 L/min revealed a pH of 7.39. The partial pressure of oxygen in arterial blood (PaO_2) was 99 mmHg, and the PaCO_2 was 62 mmHg. The remainder of the physical examination and laboratory studies was normal.

The patient's 12-lead electrocardiogram showed atrial fibrillation, right-axis deviation, incomplete right bundle branch block and right ventricular hypertrophy. Chest X-ray revealed bilateral hyperinflated lungs and increased cardiac markings. Echocardiographic examination demonstrated enlargement of the right atrial and ventricular chambers, severe tricuspid regurgitation, and pulmonary hypertension. Left ventricular ejection fraction could not be determined due to wall motion abnormalities. No chest computed tomography or pulmonary function testing data were available. The patient was scheduled for tricuspid valve replacement under general anesthesia.

No sedative premedication was given. Standard monitors (electrocardiogram, pulse oximeter) were applied in the operating room. Intravenous access and arterial blood pressure monitoring were established before induction of anesthesia. After pre-oxygenation for 5 min, anesthesia was induced with intravenous 0.5 $\mu\text{g}/\text{kg}$ of sulfentanil, 0.1 mg/kg of midazolam, 0.3 mg/kg of etomidate. Tracheal intubation was facilitated by vecuronium. After confirmation of tube placement, 2% sevoflurane in 100% oxygen was administered with a Dräger Fabius GS anesthesia machine (Dräger Medical, Lübeck, Germany). The lungs were ventilated using volume-controlled ventilation, with a tidal volume of 6 mL/kg, respiratory rate of 15 breaths/min, an inspiratory to expiratory (I:E) ratio of 1:2.5, and a PEEP of 5 cmH_2O . Under these conditions, the peak and plateau airway pressures were 27 and 19 cmH_2O , respectively. A 7F 3-lumen catheter was inserted into the right internal jugular vein for continuous measure-

ment of central venous pressure and intravenous infusion, and a urinary catheter was introduced into the bladder to measure urine output. Nasopharyngeal and rectal temperatures were monitored with thermistor probes. Anesthesia was maintained with sevoflurane and sulfentanil, and repeated doses of vecuronium as required. Ten minutes after intubation, ABG analysis revealed pH 7.42, PaO_2 392 mmHg, and PaCO_2 64 mmHg. At that time, the patient's vital signs were normal.

The operation was performed through a median sternotomy approach. CPB was established with an arterial cannula in the ascending aorta. A heparinized membrane oxygenator was used, and heparin (3 mg/kg body weight) was administered to maintain activated clotting time of more than 600 seconds. As the patient was cooled to 29°C, the ascending aorta was cross-clamped and mechanical ventilation was stopped. Meanwhile, the lung was kept inflated by delivery of 100% oxygen 0.6 L/min at 8 cmH_2O pressure. Repeated cold, potassium-enriched blood cardioplegia was used for myocardial arrest protection.

The patient underwent tricuspid valve replacement. During CPB, mean arterial pressure in the radial artery was 64-80 mmHg, central venous pressure was 10.2-16.0 cmH_2O , the flow rate was regulated at 60-80 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, and mean hematocrit was maintained at ~30%. ABG analysis was performed at 20, 60, and 95 minutes following starting CPB, the PaCO_2 was 43, 42, and 36 mmHg, respectively. The patient was weaned from CPB without any difficulty, and no rhythm disturbance was observed. After stopping CPB, mechanical ventilation was resumed with 100% oxygen at a fresh gas flow of 1 L/min and 5 cmH_2O PEEP, and continuous intravenous infusion of alprostadil (0.02 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), nitroglycerin (0.5-1 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and dopamine (2-5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) were used for maintenance of hemodynamic stability. Forty-five minutes after stopping CPB, ABG analysis showed that the PaCO_2 was 64 mmHg.

After opening the pleura, lung volume reduction surgery involving resection of 20% of the most diseased areas of the lung was performed. After manual inflation with pressure of 35 cmH_2O detected no air leaks, anterior and posterior chest tubes were inserted. CPB time and

Table 1. Perioperative arterial blood gas analysis data

	pH	PaCO ₂ (mmHg)	PaO ₂ (mmHg)	SaO ₂ (%)
Preoperative	7.39	62	99	94
After induction				
10 min	7.42	64	392	100
CPB				
20 min	7.51	43	334	100
60 min	7.55	42	220	100
95 min	7.59	36	276	100
After CPB				
45 min	7.34	64	437	100
After operation				
3 h	7.42	52	124	99
1 day	7.32	59	118	98
2 days	7.38	50	116	98
3 days	7.39	42	117	98

PaCO₂: Partial pressure of carbon dioxide in arterial blood; PaO₂: Partial pressure of oxygen in arterial blood; SaO₂: Arterial oxygen saturation; CPB: Cardiopulmonary bypass.

aortic cross-clamp time were 131 and 50 min, respectively. Total operation time was 360 min.

After the operation, the patient was transferred to the intensive care unit for continuous mechanical ventilation using 50% oxygen in air. Initial ventilatory setting included a tidal volume of 7.5 mL/kg, respiratory rate of 15 breaths/min, an I:E ratio of 1:2, and a PEEP of 5 cmH₂O. On the 2nd postoperative day, the patient was weaned from mechanical ventilation and the endotracheal tube was removed without difficulty. The perioperative ABG analysis data are listed in **Table 1**. Despite hypercapnia, the perioperative period was uneventful. The patient was discharged from the hospital on the 25th postoperative day. Subsequent pulmonary function tests revealed improvement in his condition.

Discussion

Bullous emphysema arises from a destruction of alveolar tissue usually associated with chronic obstructive pulmonary disease (COPD). An effective ventilatory strategy is required to optimize adequate oxygenation and avoid VILI, especially for our patient, who underwent cardiac surgery known to complicate ventilatory management. In the present case, permissive

hypercapnia with low PEEP was selected to maintain oxygenation and avoid VILI without ill effects to the patient.

Ventilatory strategies used in patients with bullous emphysema to prevent the potential risks of VILI have not been standardized. Heres et al. [7] reported the use of high-frequency ventilation in a patient with severe bullous emphysema during emergency coronary artery bypass grafting under CPB. Eagle et al. [8] suggested positive-pressure ventilation should be avoided in patients with bullae. However, Hillier et al. [9] believed that pressure-controlled or pressure-limited ventilation were preferred over volume-controlled ventilation.

The use of PEEP in patients with COPD remains controversial. The results of some studies suggest that in patients with emphysema ventilation with a 10 cmH₂O PEEP may further deteriorate the impaired distribution of ventilation and thus worsen gas exchange [10]. Nieszkowska et al. [11] reported that ventilation with a 15 cmH₂O PEEP markedly increased the volume of emphysematous lung regions. Alternatively, some authors stated that an optimal level of PEEP should decrease expiratory resistance and improve expiratory airflow, without causing further hyperinflation [12]. Furthermore, several reports suggest low levels of PEEP did not significantly increase the end-expiratory lung volume and did not cause further hyperinflation [13, 14]. Therefore, some authors believe low tidal volume with a 5 cmH₂O of PEEP may keep the lung open without overdistention, and has a lung-protective effect [15].

Given the high risks of VILI during mechanical ventilation, in our patient we utilized volume-controlled ventilation involving a low tidal volume, a moderate ventilator rate and a long I:E ratio, combined with a low level of PEEP, to prevent excessive lung stretch and maintain adequate gas exchange. Long expiratory phases may alleviate trapped gas in the bullae and decrease compression of the normal lungs. As expected, the result of ABG analysis at 10 min after intubation indicated that this ventilatory strategy if continued would result in an unavoidable hypercapnia. Therefore, in order to avoid VILI and maintain adequate oxygenation, we used permissive hypercapnia combined with a low level of PEEP as a ventilatory strategy in our patient during general anesthesia.

Although the safe level of hypercapnia remains to be determined, permissive hypercapnia has been accepted more and more as a key component of lung-protective strategies in critically ill patients. Some authors believed that acute progression of CO₂ retention up to 100 mmHg did not lead to any serious adverse effects, as long as SaO₂ was normal [5]. Mutlu et al. [6] reported two extreme cases of permissive hypercapnia related to ventilating patients in severe status asthmaticus. PaCO₂ levels > 150 mmHg and pH levels < 7 persisted for 16 hrs in the first patient and for 9 hrs in the second; both patients recovered with no major consequences despite extreme and prolonged hypercapnia. In fact, a respiratory acidosis (pH > 7.25) is acceptable as long as adequate oxygenation and cardiovascular stability are maintained [16]. In the present case, we carried out moderate permissive hypercapnia to maintain the PaCO₂ level at 60-70 mmHg, and keep arterial oxygenation at a normal level during the operation.

Excessive hypercapnia on the other hand may have potential deleterious effects, such as hemodynamic instability, hypoxemia, and cerebral injury [4, 17]. Hypercapnia also increases pulmonary arterial pressure, which causes right heart failure. Permissive hypercapnia may thus be considered a relative contraindication in our patient, who has pulmonary hypertension and right heart dysfunction. However, in our patient all factors that increase pulmonary vascular resistance (PVR) were avoided and corrected during the perioperative period. Etomidate, which maintains systemic hemodynamic stability without affecting PVR, was used for anesthetic induction. In addition, pulmonary hypertension was managed with intravenous vasodilators. Alprostadil, a prostaglandin E1 analogue, is a product of arachidonic acid metabolism and it increases cAMP. Prostaglandin E1 has been shown to produce significant pulmonary vasodilation and decrease PVR after protamine infusion, but does not affect arterial blood pressure or cardiac output [18].

It is commonly accepted that hypercapnia can induce cerebral vasodilation, which may increase intracranial volume and aggravate preexisting intracranial hypertension. Some authors have reported that permissive hypercapnia can cause subarachnoid hemorrhage and cerebral edema [19, 20]. However, it should

be noted that hypercapnia has not been associated with cerebral injury if adequate oxygenation is maintained [21]. Fortunately, our patient did not present with hypoxemia and no unilateral or bilateral pupillary changes were detected during the perioperative period, and he fully recovered without detectable neurological deficit after surgery.

In summary, the patient with severe diffuse bullous emphysema undergoing cardiac surgery under CPB presents many problems to the anesthesiologist. Herein we described the use of permissive hypercapnia combined with a low level of PEEP as a ventilatory strategy to avoid the risks of VILI in a patient with severe diffuse bullous emphysema undergoing tricuspid valve replacement under CPB. Although it may be argued that hypercapnia is best avoided in patients with pulmonary hypertension and right heart dysfunction, our ventilatory strategy provided adequate oxygenation and minimized VILI during the perioperative period.

Disclosure of conflict of interest

None.

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