Case Report

Permissive hypercapnia: Is there any upper limit?

Sunil Kumar Garg

Abstract

A 19-year-old male presented with history of massive hemoptysis. The patient was kept on mechanical ventilation because of severe hypoxia. Lung protective ventilation with low tidal volume was given in view of very poor pulmonary compliance. During the course of treatment, the patient developed a very high CO_2 level of 373 mmHg. The patient was successfully weaned off on the 9th day without any obvious adverse consequences.

Keywords: Lung protective ventilation, permissive hypercapnia, supercarbia

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Introduction

Current protective lung ventilation strategies commonly involve hypercapnia. This approach has resulted in an increase in a shift in clinical paradigms regarding hypercapnia from avoidance to tolerance, with hypercapnia increasingly permitted in order to realize the benefits of low lung stretch.[1]

The reported levels of $PaCO_2$ (mean maximum $PaCO_2$ 67 mmHg) in the study conducted by Hickling and co-workers reflects typical levels observed with the institution of this technique.[2] No consensus, however, has been established in terms of its upper limits and the optimal CO_2 level for mechanically ventilated patients with acute lung injury (ALI).[3,4] The concept of an optimal CO_2 concentration is essential as most physiological systems are saturable and it is therefore reasonable that an effective upper limit of CO2, a point beyond which advantages shift toward harmful effects, exists.[3]

Case Report

A Nineteen -year-old male presented to the emergency department with massive hemoptysis. He was treated for pulmonary tuberculosis for the full duration and stopped ATT only few months back. In view of the patient's drowsiness and hypotension, he was intubated in emergency and shifted to the Intensive Care Unit (ICU). His arterial blood gas (ABG) analysis showed severe hypoxia and hypercarbia and therefore he was kept on mechanical ventilation. Sedation and paralysis was initiated. On mechanical ventilation (MV), the patient's airway pressures were very high. To prevent lung injury, the tidal volume was decreased to 150 mL with monitoring of end-inspiratory plateau pressure. Because the patient was retaining CO2, his respiratory rate was increased to 35. On admission, his laboratory results included hemoglobin of 10.3, TLC 30.1, platelet count 413, blood urea 16, creatinine 0.60, sodium 135 and potassium 4.4. His liver functions tests were normal. Chest X-ray on admission showed bilateral diffuse heterogeneous opacities [Figure 1]. His initial ABG analysis was pH 7.007, pO_2 38 and pCO_2 100. His endotracheal secretion showed presence of AFB.

There was no further episode of bleeding. He was started on broad-spectrum antibiotics with anti-tubercular medication. Lung protective ventilation with low tidal volume was given in view of poor pulmonary compliance. During mechanical ventilation, PEEP was always kept at 5–6 and end-inspiratory plateau pressure was kept at around 30. Other ventilator parameters include TV varying between 150 and 180 mL for the initial 48 h; thereafter, as the pulmonary

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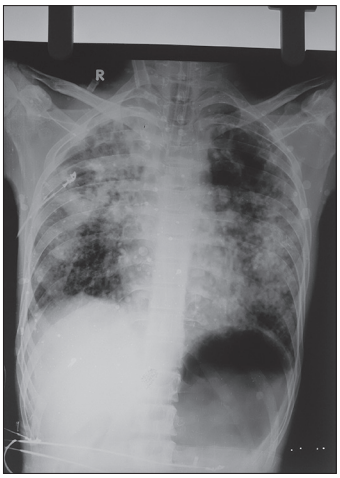

Figure 1: CXR on the day of admission

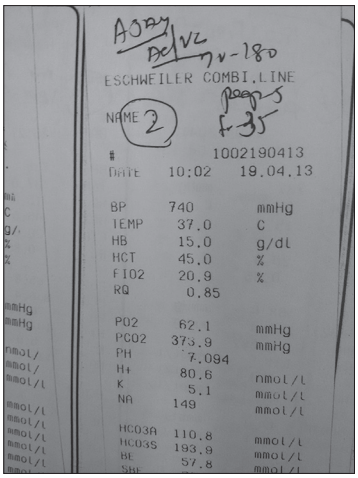

Figure 2: ABG showing pCO_2 value

compliance improved, TV was increased. Flow pattern was square wave.

The patient developed further rise in CO_2 as expected and fall in pH with maintained blood pressure and organ perfusion. After about 24 h, the patient was started on Nor Epinephrine support because of falling blood pressure. During this time, his ABG analysis was pH 6.96, pCO_2 219 and pO_2 54 on mechanical ventilation setting of F 35, TV 180, FiO_2 0.6 and PEEP 5, while the plateau pressure was 30. He was started on soda bicarbonate infusion around 36 h after admission in view of further fall in pH to 6.88 and continued need of vasopressors to raise blood pressure. The remainder of the ABG analysis was pCO_2 230 mmHg and pO_2 68. After 12 h of soda bicarbonate infusion at the rate of 50 mL/h, his blood gas analysis showed pH 7.09, pCO_2 373 and pO_2 62 on TV 180, F 35, FiO_2 0.75 and PEEP 5 [Figure 2]. The soda bicarbonate infusion was stopped around after 17 h. At that time, the patient's pH rose to 7.29, with CO_2 of 141 mmHg and pO_2 of 92 on MV settings of F 35, TV 240 mL, FiO_2 0.65 and PEEP 6 while the plateau pressure was 30. Over the period of the next 48 h, Nor Epinephrine was tapered and stopped. He was weaned off from the ventilator on the 9th day of admission and shifted out of the ICU on the 18th day in a stable condition.

Discussion

Permissive hypercapnia has become a central component of protective lung ventilatory strategies.[5] This strategy has resulted in an increase in the clinical acceptability of elevated carbon dioxide tension in order to avoid the deleterious effects of high lung stretch.[1] These strategies invariably involve a reduction in tidal volume and/or transalveolar pressure. These protective lung ventilation strategies have been demonstrated to improve survival in patients with ARDS. In current practice, mean maximum $PaCO_2$ and pH associated with permissive hypercapnia are around 67 mmHg and 7.2, respectively,[2] and are reported to be well tolerated as long as tissue perfusion and oxygenation are preserved and there are no contraindications.[6-8]

In our patient, we observed a maximum documented pCO_2 level of 373 mmHg. While the rise in carbon dioxide tension could be contributed by soda bicarbonate infusion, it appears that a major rise was because of protective lung ventilation and underlying diseased lung as the pCO_2 value before starting ventilation and sodium bicarbonate infusion was 100 mmHg and 230 mmHg, respectively, and the CO_2 value before stopping sodium bicarbonate infusion was 141 mmHg. His recovery was without any obvious adverse consequences. Several reports emphasize the potential for tolerance to extreme levels of hypercapnia, termed supercabia. Mazzeo *et al*. described complete clinical recovery after an episode of prolonged life-threatening status asthmaticus in an 8-year-old boy. Despite maximal medical therapy involving both conventional and last resort bronchodilator therapies, a progressive respiratory acidosis developed, with a nadir pH of 6.77 and pCO2 of 293 mmHg recorded 10 h after admission. Hemodynamic stability was maintained throughout and no neurologic or other organ dysfunction was detected after eventual complete resolution of status asthmaticus.[9]

Urwin *et al*. reported survival without adverse sequelae after extreme hypercapnia (pCO_2 233 mmHg) in an older woman with acute decompensation of chronic obstructive airway disease.[10] To date, however, human trials studying the effect of CO_2 *per se* on outcome in patients with lung injury have not been performed.

Conclusion

Ventilatory strategies involving hypercapnia are widely used in the setting of acute respiratory failure, with the aim of realizing the benefits of reduced lung stretch. No consensus, however, has been established in terms of its upper limits and the optimal CO_2 level for mechanically ventilated patients with ALI. It appears to be well tolerated as long as oxygenation and tissue perfusion are maintained and there is no contraindication of hypercapnia.

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