

With Oxygen, More is Not Necessarily Better

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Abstract

We present a case of a patient with obesity-hypoventilation, sleep apnea syndrome who developed respiratory failure with administration of high doses of oxygen. The case illustrates of administering oxygen without titration can have potentially adverse effects.

Case Presentation

History of Present Illness

A 48 year old male was admitted for shortness of breath. He had been discharged 2 days earlier after a 10 day hospitalization for presumed sleep apnea, obesity-hypoventilation syndrome, hypertensive urgency and non-ST segment myocardial infarction complicated by ventricular tachycardia and hypercapnic respiratory failure. Although he was prescribed continuous positive airway pressure (CPAP) at 10 cm H₂O during sleep, this was not used since his prior admission. He had a questionable history of asthma and had been using his albuterol inhaler every 1-2 hours without relief.

Physical Examination

He was alert, obese man (weight 148 kg) who was oriented and appeared in no acute distress. BP was elevated at 169/120. Occasional wheezes were heard on lung auscultation. He had 1+ pretibial edema.

Laboratory and Radiology

Pertinent laboratory findings include an elevated white blood cell count of 17,700 cells/microliter with a left shift. Arterial blood gases showed a PaO₂ of 79, PaCO₂ of 71 and a pH of 7.29 on oxygen administered at 2 l/min by nasal cannula. Brain natriuretic peptide was elevated at 145 pg/ml. Chest x-ray was considered normal.

Hospital Course

He was treated for an asthma exacerbation with albuterol by small volume nebulizer every 4 hours and prednisone 60 mg orally. He refused CPAP therapy and because his SpO₂ decreased to 87 his oxygen was increased to 4 l/min by nasal cannula. About 6 hours after admission he was lethargic, but arousable, and his oxygen was further increased to 6 l/min. He became unarousable except to painful stimuli. His arterial blood gases were repeated and showed a PaO₂ of

90, PaCO₂ of 92 and a pH of 7.20. He was evaluated for transfer to the intensive care unit for intubation by the pulmonary fellow, but rather than intubate the patient, the patient's oxygen was decreased to 2 l/min by nasal cannula. The patient's SpO₂ decreased to 88 but he became awake and alert within 30 minutes. He had uneventful subsequent hospital course and was discharged 3 days later on CPAP for nighttime use.

Discussion

This case illustrates the potential dangers of inappropriate use of oxygen therapy, a problem which is being increasingly recognized. Increasing oxygen in patients with hypercapnea, particularly the hypercapnic patient with a history of respiratory depression from high oxygen, is potentially dangerous. The patient presented is typical with probable obesity-hypoventilation and/or sleep apnea, hypercapnea and a history of respiratory depression from oxygen administration. Fortunately, the respiratory depression was recognized and reduction in oxygen and administration of noninvasive positive pressure ventilation likely averted endotracheal intubation and mechanical ventilation.

High doses of oxygen can cause a number of adverse effects including absorption atelectasis and increased ventilation/perfusion mismatch, which impairs elimination of carbon dioxide potentially leading to hypercapnea and respiratory depression. Suppression of hypoxic drive is probably less important than ventilation/perfusion mismatch (1).

The British Thoracic Society (BTS) has published guidelines on the use of oxygen in adults (2). The guidelines emphasize that oxygen is a treatment for hypoxemia, not breathlessness or dyspnea. Oxygen has not been shown to have any effect on the sensation of breathlessness in non-hypoxemic patients. Therefore, increasing oxygen therapy is not only ineffective, but as illustrated by the case above, potentially dangerous.

It has become increasingly recognized that oxygen should be prescribed according to a target oxygen saturation range. Oxygen saturation can now be readily measured in most settings through the use of pulse oximetry. BTS emphasizes that those who administer oxygen therapy should monitor the patient and keep within the target saturation range. BTS suggests oxygen should be prescribed to achieve a target saturation of 94–98% for most acutely ill patients or 88–92% for those at risk of hypercapnic respiratory failure. High-risk patients with a prior history of hypercapnic respiratory failure may usually be safely managed with an oxygen saturation in the range of 85%-88%.

In support of the concept that titration of oxygen improves outcomes, Austin et al. (3) recently compared nontitrated high flow oxygen treatment with titrated oxygen treatment in the prehospital (ambulance/paramedic) setting in patients with an exacerbation of COPD. They demonstrated that titrated oxygen titrated to an

SpO₂ of 88-92% had significantly reduced mortality, hypercapnia, and respiratory acidosis compared with high flow oxygen at 8-10 l/min in acute exacerbations of chronic obstructive pulmonary disease. The authors concluded that the results provide strong evidence to recommend the routine use of titrated oxygen treatment in patients with breathlessness and a history or clinical likelihood of chronic obstructive pulmonary disease in the prehospital setting.

Administration of high doses of oxygen to patients without hypercapnic respiratory failure may also have adverse consequences. Kilgannon et al. (4) studied patients admitted to the intensive care unit following resuscitation from cardiac arrest. Arterial hyperoxia defined as a PaO₂ of ≥ 300 mm Hg was independently associated with increased in-hospital mortality compared with either hypoxia or normoxia. Bellomo et al. (5) also found that hyperoxia after cardiopulmonary arrest was associated with higher mortality. However, once Cox proportional hazards modelling was applied, hyperoxia defined as PaO₂ > 400 mmHg was no longer predictive of hospital mortality.

The above emphasize the need to prescribe oxygen as a drug, especially in the acute setting. As with most drugs, the drug effect should be monitored, in this case by the oxygen saturation. Adjusting the oxygen to appropriate levels may well avert intubation, mechanical ventilation and even death in some patients.

References

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