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 naturetic 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 does 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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