

ACUTE HYPOXEMIA AFTER REPOSITIONING OF PATIENT: A CASE REPORT

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Hypoxemia occurred after induction of anesthesia and repositioning in a patient undergoing hip pinning. The patient had previously presented to the emergency department with multiple fractures and hemodynamic instability sustained in a motor vehicle accident. Three days after admission to the intensive care unit the patient remained intubated with respiratory insufficiency and had developed acute respiratory distress syndrome with marginal oxygen saturation.

The patient was transported to the operating room for hip pinning, and anesthesia was induced with midazolam, fentanyl, vecuronium, and isoflurane. When the patient was turned to the left lateral position, oxygen saturation suddenly worsened from 94% to 78%, with Pao₂ from arterial blood gas measured at 54 mm Hg. The patient was returned

to the supine position, but despite maneuvers to improve oxygen saturation, the patient's saturation remained below 87% and pulmonary thromboembolism was suspected. However, other signs of pulmonary embolus such as hemodynamic deterioration and right ventricular dysfunction were not present.

Chest radiographs demonstrated severe left lung atelectasis, and surgery was postponed. Upon return to the intensive care unit, fiberoptic bronchoscopy was performed, and a large mucous plug was removed from his left upper and lower lobes, with subsequent improvement of Pao₂ to 77 mm Hg with an oxygen saturation of 94%.

Key words: Atelectasis, hypoxemia, ventilation-perfusion mismatch.

Anesthesia is associated with lower oxygen tensions, and occasionally oxygen desaturation and hypoxemia occur. Mechanisms for hypoxemia include ventilation perfusion mismatch, hypoventilation, diffusion impairment, and physiologic or anatomic shunt.¹ Clinical causes include equipment failure, endotracheal tube malfunction, hypoventilation, and decrease in functional residual capacity.² Patients having experienced trauma and hip fracture have a high incidence of venous thromboembolism and are at risk for pulmonary embolism and associated hypoxemia.³ However, atelectasis due to mucous plugging also has been reported in patients in this setting⁴⁻⁹ and should always be a consideration when hypoxemia is encountered.

Case summary

This patient was a 42-year-old, 128-kg, 168-cm (5 feet, 6 inches) morbidly obese male with a history of motor vehicle accident and fractured right hip 3 days earlier who had subsequently developed acute respiratory distress syndrome (ARDS) and respiratory insufficiency. After consultation between the intensivist, orthopedic surgeon, and anesthesia care providers, it was determined that intramedullary nailing would allow the patient to sit in bed and improve respiratory mechanics.

Preoperative assessment was remarkable for respi-

ratory insufficiency, morbid obesity, ARDS, and multiple fractures. Chest radiograph and electrocardiograph (ECG) demonstrated bibasilar infiltrates and significant laboratory values included hematocrit of 33% and PaO₂ of 68 mm Hg. Maintenance ventilator settings in the intensive care unit (ICU) immediately prior to transport were FiO₂, 0.6; tidal volume, 800 mL; respiratory rate, 14; and positive end-expiratory pressure (PEEP), 7 cm H₂O, with peak inspiratory pressures of 38 cm H₂O.

After transport to the operating room from the ICU with 100% oxygen, the patient was placed on the anesthesia machine ventilator with settings of FiO₂, 0.6; tidal volume, 800 mL; respiratory rate, 14; and PEEP, 10 cm H₂O. Standard monitors, including noninvasive blood pressure cuff, 5-lead ECG, and pulse oximeter probe (right middle finger) were applied. The arterial line in the left radial artery correlated well with the noninvasive blood pressure cuff and measured blood pressure was 138/75. Pulmonary artery pressures measured 44/23 mm Hg by continuous cardiac output pulmonary artery catheter, with right atrial pressure of 11 mm Hg. Initial heart rate measured 112 beats per minute and oxygen saturation was 94%, with peak airway pressure (PAP) at 45 cm H₂O. Midazolam, 5 mg; fentanyl, 250 µg; vecuronium, 10 mg; and isoflurane, 1%, were administered for induction, and the patient was moved to the operating table and placed in the left

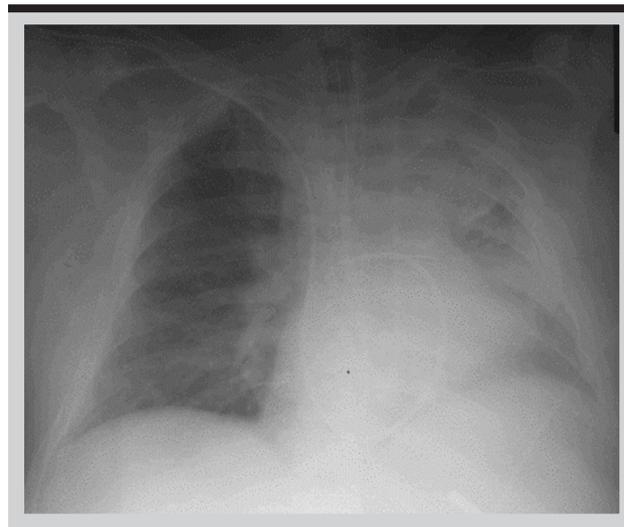
lateral decubitus position. An axillary roll was placed and bilateral breath sounds were distant but present.

Upon assuming the left lateral position the PAP increased to 68 cm H₂O and oxygen saturation decreased to 85% over a 2-minute period. The increase in PAP was assumed to be related to position, so the tidal volume was decreased to 600 mL and respiratory rate was increased to 16. Train-of-four after the previously administered vecuronium demonstrated 4 twitches, blood pressure was 128/76, and pulse was 118 beats per minute. Pulmonary artery pressure had increased to 51/26 mm Hg, and the right atrial pressure had increased to 15 mm Hg. Pulmonary artery diastolic occlusion pressure was unobtainable due to inability to wedge the balloon tip, and cardiac index was 2.5 L/min per meter.² Despite the reduction in tidal volume, PAP remained 65 cm H₂O. FiO₂ was increased to 100% and the patient was hand ventilated to improve pulmonary compliance. Vecuronium, 10 mg, and pancuronium, 10 mg, were administered to facilitate better control of ventilation, and an additional pulse oximeter probe was applied to the patient's ear to correlate with the finger probe.

Assessment of breath sounds was unchanged, and pulmonary toilet yielded moderate amounts of purulent secretions. The inspiration-to-expiration ratio was decreased from 1:3 to 1:2, the respiratory rate was increased to 20 breaths per minute to improve minute ventilation, and PEEP was increased to 15 cm H₂O. Respiratory therapy was consulted for use of an ICU quality ventilator to provide pressure support ventilation. Albuterol was administered via endotracheal tube, and inhalation agent was increased to provide bronchodilation in an effort to decrease airway resistance. Pulse oximetry continued to display low saturations, measuring 85% on the right middle finger, 85% on the right ear, and 82% on the right second toe. Arterial blood gases were drawn and sent to the laboratory for analysis, and revealed pH, 7.36; PaCO₂, 54 mm Hg; PaO₂, 55 mm Hg; SaO₂, 85%; and base excess, 2.9. Pulmonary toilet continued to yield thick, viscous secretions in moderate amounts but was infrequently performed due to the patient's precarious ventilatory status. While heart rate and blood pressure remained stable, oxygen saturation remained low, so the patient was turned supine to improve ventilation-perfusion ratios. The head of the bed was then elevated to minimize the effect of the abdomen on lung expansion, and consideration was given to performing the pinning in the supine position.

With the sudden decrease in saturation and increase in pulmonary artery and right atrial pressure, pulmonary embolus was suspected. However, blood

Figure. Chest radiograph demonstrating mucous plugging and left lung atelectasis



pressure and cardiac index were consistent with previous readings and the electrocardiograph was unchanged. End-tidal CO₂ was unchanged, and no new murmur was appreciated by auscultation of the chest. A chest radiograph was performed to rule out a pneumothorax or other issues, and consideration was given to performing pulmonary angiography to rule out pulmonary embolus.

While chest radiograph revealed no typical signs of pulmonary embolus such as wedge-shaped density or Westermark sign, severe atelectasis in the left lung field (Figure) with almost complete obliteration of the left upper and lower lobes was noticed. The decision was made to cancel the operation and return to the ICU for bronchoscopy and resolution of the patient's oxygenation issues. Fiberoptic bronchoscopy was performed, and a large mucous plug was removed from the left upper and lower lobes, subsequently returning oxygen saturation to 94%. Surgery was performed successfully in 2 days after vigorous pulmonary toilet and diuresis, and the patient eventually was discharged.

Discussion

During anesthesia and neuromuscular relaxation, ventilation is redistributed to the nondependent regions of the lungs due to atelectasis and airway closure in the dependent lung regions,¹⁰⁻¹⁴ resulting in increased alveolar-arterial oxygen gradients and hypoxemia.¹⁵ Assuming less favorable positions, such as the lateral position, further affects both ventilation and perfusion,^{16,17} and the addition of PEEP may actually be deleterious by forcing perfusion toward the dependent lung.¹⁸ Morbid obesity further increases ventilation perfusion mismatch,^{19,20} and

these changes are magnified when supine and/or lateral positions are assumed.²¹⁻²³ Further complicating the clinical picture is the onset of ARDS, which may occur following trauma such as a motor vehicle accident. Due to increased permeability of the capillary endothelium and alveolar epithelium, leakage of plasma and erythrocytes into the interstitial and alveolar spaces occurs along with pulmonary edema.^{24,25} While increasing inspired oxygen concentration initially improves oxygenation, as the disease progresses hypoxemia may not be corrected due to the right to left shunting resulting from perfusion of atelectatic or fluid filled alveoli. PEEP, lower tidal volumes, and optimal fluid management are necessary,²⁶ and the already compromised oxygenation status may be worsened by other factors creating additional ventilation-perfusion mismatch during anesthesia.

While pulmonary atelectasis is not uncommon in the postoperative setting,²⁷ massive intraoperative atelectasis and collapse of an entire lobe or lung segment is uncommon and may be potentially fatal. In this patient, acute atelectasis was due to mucous plugging, and while diagnosis may have been made by clinical signs, such as the sudden onset of hypoxemia and reduction or absence of breath sounds, the initial clinical presentation included low oxygen saturation. Also, distant breath sounds and tachycardia were consistent with the patient's initial presentation, and controlled ventilation masked any signs of respiratory difficulty or labored respiratory pattern. Further complicating the clinical picture was the presence of many factors, which could have caused arterial hypoxemia and desaturation, including ARDS and morbid obesity.

A very likely cause of hypoxemia in this patient could have been venous thrombosis and pulmonary embolus. Pulmonary embolism is common in critically ill patients requiring mechanical ventilation^{28,29} and is a common complication associated with trauma and hip fracture.^{3,30} Any unexplained hypoxemia of a sudden onset in a patient undergoing orthopedic surgery on a hip fracture would be suspect, especially in the setting of morbid obesity and immobilization. However, end-tidal CO₂ did not decrease, and arterial blood pressure and cardiac rhythm were unchanged. Also, no new right bundle branch block, right axis deviation, T wave inversion, murmur, increase in jugular vein distention, or other sign of right ventricular dysfunction were noted. Pulmonary artery pressures were high and elevated above initial readings, but increases in pulmonary artery pressures were a part of this patient's initial presentation and could have been affected by positioning and/or

worsening hypoxemia. In addition, increases in mean pulmonary artery pressure are dependent on degree of severity of pulmonary embolism and may vary by classification.³¹ While right atrial pressure was slightly elevated (15 mm Hg), cardiac index was consistent with values in the ICU and were within normal limits.

As the clinical picture of pulmonary embolism may vary according to severity of vascular obstruction, size and location of emboli and preexisting cardiopulmonary disease³² diagnosis by clinical assessment is difficult. While consideration was given to pulmonary arteriograms, venous ultrasonography and d-dimer enzyme-linked immunosorbent assay (ELISA)³³ for more definitive diagnosis, the chest radiograph revealed no focal abnormality or wedge-shaped density but rather almost complete left lung collapse. As more common causes of atelectasis, such as bronchial intubation, aspiration, and pneumothorax had been excluded, mucous plugging was suspected due to the sudden onset and presence of copious amounts of aspirated secretions from the patient's endotracheal tube.

While lung collapse and intraoperative hypoxemia have been reported previously, few involve mucous plugging and atelectasis. In reviewing the literature, we found 5 reports describing mucous plugging, all associated with orthopedic surgery following trauma⁴⁻⁶ and all diagnosed with chest radiograph. In these patients, the combination of recent trauma and surgery may have been factors contributing to intraoperative atelectasis and lung collapse from mucous obstruction. Four other cases of mucous plugging have been reported in quadriplegic patients initially diagnosed with pulmonary embolism.⁷⁻⁹ However, chest radiograph was inconclusive, and diagnosis was made in these cases only by the use of ventilation scintiscans.

In summary, mucous plugging should be a consideration in any previously intubated trauma patient presenting for surgery. While deep vein thrombosis of the lower extremities is a common complication following trauma³ and should always be considered as a potential cause of sudden hypoxemia, sudden desaturation with any position change should immediately warrant suctioning and assessment for this potentially life-threatening clinical incident. Further, once patients at risk for intraoperative or pulmonary complications are identified, pulmonary assessment and optimization should be used prior to surgery and may include fiberoptic bronchoscopy, pulmonary lavage, and diuresis.

REFERENCES

1. West JB. *Pulmonary Pathophysiology: The Essentials*. 6th ed. Baltimore, Md: Williams and Wilkins; 2000:603-610.
2. Benumof J. Respiratory physiology and respiratory function during anesthesia. In: Miller RD. *Anesthesia*. 5th ed. Philadelphia, Pa: Churchill Livingstone; 2000.

3. Geerts WH, Code KI, Jay RM, Chen E, Szalai JP. A prospective study of venous thromboembolism after major trauma. *N Engl J Med*. 1994;331:1601-1606.
4. Pivalizza EG, Tonnensen AS. Acute life-threatening intraoperative atelectasis. *Can J Anaesth*. 1994;41:857-860.
5. Samuels SI, Brodsky JB. Profound intraoperative atelectasis. *Br J Anaesth*. 1989;62:216-218.
6. Samuels SI, Clark RW. Profound atelectasis during anesthesia. *Anesth Analg*. 1980;59:792-795.
7. Dee PM, Suratt PM, Rose CE Jr. Mucous plugging simulating pulmonary embolism in patients with quadriplegia. *Chest*. March 1984;85:363-366.
8. Bray ST, Johnstone WH, Dee PM, et al. The "mucous plug syndrome": a pulmonary embolism mimic. *Clin Nucl Med*. 1984;9:513-518.
9. Pham DH, Huang D, Korwan A, Greyson ND. Acute unilateral pulmonary nonventilation due to mucous plugs. *Radiology*. October 1987;165:135-137.
10. Rehder K, Hatch DJ, Sessler AD, Fowler WS. The function of each lung of anesthetized and paralyzed man during mechanical ventilation. *Anesthesiology*. 1972;37:16-26.
11. Rehder K, Sessler AD, Rodarte JR. Regional intrapulmonary gas distribution in awake and anesthetized-paralyzed man. *J Appl Physiol*. 1977;42:391-402.
12. Klingstedt C, Baehrendtz S, Bindslev L, Hedenstierna G. Lung and chest wall mechanics during differential ventilation with selective PEEP. *Acta Anaesthesiol Scand*. 1985;29:716-721.
13. Hedenstierna G, Bindslev L, Santesson J. Pressure-volume and airway closure relationships in each lung in anesthetized man. *Clin Physiol*. 1981;1:479-493.
14. Hedenstierna G, Bindslev L, Santesson J, Norlander OP. Airway closure in each lung of anesthetized human subjects. *J Appl Physiol*. 1981;50:55-64.
15. Bindslev L, Hedenstierna G, Santesson J, Gottlieb I, Carvallhas A. Ventilation-perfusion distribution during inhalation anesthesia. *Acta Anaesthesiol Scand*. 1981;25:360-371.
16. Coonan TJ, Hope CE. Cardio-respiratory effects of change of body position. *Can Anaesth Soc J*. 1983;30:424-437.
17. Klingstedt C, Hedenstierna G, Baehrendtz S, et al. Ventilation-perfusion relationships and atelectasis formation in the supine and lateral positions during conventional mechanical and differential ventilation. *Acta Anaesthesiol Scand*. 1990;34:421-429.
18. Hedenstierna G, Baehrendtz S, Klingstedt C, et al. Ventilation and perfusion of each lung during differential ventilation with selective PEEP. *Anesthesiology*. 1984;61:369-376.
19. Damia G, Mascheroni D, Croci M, Tarenzi L. Perioperative changes in functional residual capacity in morbidly obese patients. *Br J Anaesth*. 1988;60:574-578.
20. Adams JP, Murphy PG. Obesity in anaesthesia and intensive care. *Br J Anaesth*. 2000;85:91-108.
21. Vaughan RW. Pulmonary and cardiovascular derangements in the obese patient. In: Brown BR, ed. *Anesthetics and the Obese Patient*. Contemporary Anesthesia Practice Series. Philadelphia, Pa: FA Davis; 1982:19.
22. Perilli V, Sollazzi L, Bozza P, et al. The effects of the reverse Trendelenberg position on respiratory mechanics and blood gases in morbidly obese patients during bariatric surgery. *Anesth Analg*. 2000;91:1520-1525.
23. Salem MR, Joseph N, Lim R, et al. Respiratory and hemodynamic response to PEEP in grossly obese patients [abstract]. *Anesthesiology*. 1984;61:A511.
24. Demling RH. The pathogenesis of respiratory failure after trauma and sepsis. *Surg Clin North Am*. 1980;60:1373.
25. Ware LB, Matthay MA. The acute respiratory distress syndrome. *N Engl J Med*. 2000;342:1334-1349.
26. Tobin MJ. Culmination of an era in research on the acute respiratory distress syndrome. *N Engl J Med*. 2000;342:1360-1361.
27. Pederson T, Viby-Mogensen J, Ringsted C. Anaesthetic practice and postoperative pulmonary complications. *Acta Anaesthesiol Scand*. 1992;36:812-818.
28. Attia J, Ray JG, Cook DJ, Douketis J, Ginsberg JS, Geerts WH. Deep vein thrombosis and its prevention in critically ill adults. *Arch Intern Med*. 2001;161:1268-1279.
29. Cook D, McMullin J, Hodder R, et al. Prevention and diagnosis of venous thromboembolism in critically ill patients: a Canadian survey. *Crit Care*. 2001;5:336-342.
30. Anderson FA, Spencer FA. Risk factors for venous thromboembolism. *Circulation*. 2003;107:19-16.
31. Stein PD, Henry JW. Clinical characteristics of patients with acute pulmonary embolism stratified according to their presenting syndromes. *Chest*. 1997;112:974-979.
32. Dalen JE. Clinical diagnosis of acute pulmonary embolism. When should a V/Q scan be ordered? *Chest*. 1991;100:1185-1186.
33. Goldhaber SZ. Pulmonary embolism. *N Engl J Med*. 1998;339:93-104.

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