Sleep Apnea Syndromes: A Potential Contraindication for Patient-controlled Analgesia

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This report describes a near-fatal episode of respiratory distress in a patient with obstructive sleep apnea syndrome while receiving patient-controlled analgesia (PCA). Although quite obese, this patient was not known to have symptoms of sleep apnea until after the incident. Our evaluation indicates that self-administration of morphine together with a propensity for airway obstruction and poor respiratory monitoring precipitated this event.

CASE REPORT

A 45-yr-old man (height 182.9 cm and weight 136 kg) was admitted for severe left flank pain and gross hematuria. Intravenous pyelogram and computer tomography revealed an upper pole of the left kidney, and the patient was scheduled for radical left nephrectomy.

Preoperative evaluation indicated no significant past medical history; there were no respiratory or cardiovascular symptoms noted, and the patient neither smoked cigarettes nor drank alcohol.

On admission, blood pressure (BP) was 150/90 mmHg, and pulse was 80 beats per min. Physical examination revealed an obese, middle-aged man in considerable distress. His neck and abdomen were exceptionally large. Preoperative labs were normal with the exception of hematuria. The ECG showed normal sinus rhythm and right bundle branch block.

Anesthesia was induced with thiopental, and oral intubation was accomplished without difficulty after administration of succinylcholine. Anesthesia was maintained with isoflurane in N2O and O2 and with fentanyl, and muscle relaxation was obtained with vecuronium. The only surgical complication was an inadvertent incision of the left pleura for which a chest tube was placed; the lung was not damaged. Anesthesia lasted from 8 AM to 3 PM and was uneventful. The patient left the operating room in stable condition with his trachea intubated.

In the S intensive care unit (SICU), ventilation was controlled with an Emerson ventilator at a FiO2 of 0.40, tidal volume 900 ml, intermittent mechanical ventilation 8, and PEEP of 5. At 3:45 PM the patient received a bolus of 1,000 ml lactated Ringer's solution and had a BP of 100/60 mmHg. His vital signs remained stable, and at 7 PM continuous positive airway pressure was instituted. At 8:15 PM his trachea was extubated without difficulty, and he was provided with 40% O2 via a face mask. Chest x-ray at 8:30 PM showed some left lower lobe atelectasis but no pneumothorax.

At 9:00 PM a Bard PCA pump was used to administer PCA, at a setting of 1.5 mg morphine per injection, with a 10-min delay between injections, and at a basal rate of 2 mg/h. At 9:50 PM the nurse reported the patient asleep and snoring loudly. The patient continued to sleep and had normal vital signs until awakened at 4:00 AM for laboratory tests and dressing changes.

At 7:30 AM the patient was found unresponsive, and an arterial blood gas analysis revealed pH = 7.02, arterial CO2 tension (PaCO2) = 94 mmHg, arterial O2 tension (PaO2) = 44 mmHg, base excess = -7.8 mEq, and hemoglobin O2 saturation of 56%. An ampule of naloxone was administered; the trachea was reintubated without difficulty; and controlled mechanical ventilation was reimstituted.

Analysis of the PCA pump revealed that it had functioned according to specifications. The patient had received a total of 47 mg morphine during the preceding 10.5 h. During the 2 h from 4 AM (when he was awakened) to 6 AM, he attempted 42 injections and received 15 mg morphine. There was only one additional attempt at injection during the next 1.5 h, the period just prior to his being found unresponsive.

DISCUSSION

There are several factors indicating that excessive self-administration of morphine was a contributing factor in this case of near-fatal respiratory depression. The patient's trachea had been extubated without difficulty, and his vital signs had remained stable until 1 or 2 h before the incident. None of the chest films taken after surgery or after the incident accounted for acute respiratory distress; although there was some atelectasis of the left lower lung there was no sign of a pneumothorax. In addition, the patient responded dramatically to intravenous (iv) naloxone by awakening and attempting more forceful respiration within minutes of its administration.

Our analysis suggests that several factors combined to produce this outcome. First, this patient appears to have had an obstructive sleep apnea syndrome. Although a definitive diagnosis can be made only through sleep laboratory studies, interviews with both the patient and his mother revealed symptoms that fit this profile. The patient reported awakening in the middle of the night short of breath, found it difficult to awaken in the morning, and was sleepy most of the day. His mother, who had lived with him for the preceding few years, related that her son snored loudly and had on numerous occasions awakened gasping for breath. In addition, the patient noticed in the hospital that when he lay down and put his head back, his "throat closes and his air is cut off."

Second, the patient self-administered a number of boluses of morphine just prior to the event. Patients with sleep apnea syndrome typically are obese and have obstructive apneic episodes secondary to upper airway obstruction.1 When these occur the patient is able to overcome them and resume breathing only when the PaCO2

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increases. Morphine blunts this response and thereby necessitates an even greater increase in P_{\text{aCO}_2} to stimulate ventilation. In our case, the patient was awakened at 4 AM for dressing changes. The pain and anxiety associated with this stimulation may have caused him to self-administer the maximum amount of morphine (42 button pushes and 15 mg of morphine). Once the dressing was completed, the patient went back to sleep, after having receiving a relatively large amount of morphine. This, coupled with his compromised airway, can easily account for his near-fatal respiratory distress.

Third, although this patient was in the SICU, he was not monitored adequately. A review of his chart found that during the critical 2 h before the patient’s respiratory distress, respiration was not monitored or charted because of a nursing shortage and emergencies in other parts of the unit.

Morbid obesity frequently is associated with respiratory impairment. In patients who suffer from the Pickwickian or obesity hypoventilation syndrome, arterial blood gas analysis reveals hypoxemia and hypercarbia; for this reason, it can be argued that these patients are not candidates for PCA. However, those suffering from obstructive sleep apnea syndrome frequently have normal blood gases while awake and may not appear to be short of breath. It is these patients who may experience difficulties with PCA.

How than should we deal with postoperative pain in the obese patient? It is these patients whose respiratory status benefit most from optimal analgesia, particularly after abdominal and thoracic surgery. In retrospect it may have been safer for our patient to have received PCA only, instead of PCA plus a 2-mg/h basal infusion. In addition, the Bard PCA pump can be programed with a 1-h limit on the amount of drug delivered; in our case this safety feature was not used. For obese patients, it may be prudent to use smaller boluses of drug or to employ longer lock-out times between injections. Finally, for very obese patients or for those who exhibit signs of sleep apnea syndrome, it may be wise to restrict PCA use to an intensively monitored setting or alternately to rely on nurse-administered injections of intramuscular analgesics.

REFERENCES


Hypotension during Transfusion of Autologous Blood

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Autologous blood transfusion is generally considered the safest and most preferable method of transfusion therapy, and its appropriate use is highly recommended.1–4 Among its advantages is a decrease in the risk of transfusion-associated reactions, including those related to infection, alloimmunization, and incompatibility.1–2,4 However, this technique is not without hazards, which may include volume overload, bacterial contamination, and incompatibility due to clerical errors.2,5,6 We report a case of repeated hypotension in association with transfusion of autologous blood, a situation that we do not believe has been reported previously.

CASE REPORT

A 58-yr-old woman presented for cholecystectomy, open liver biopsy, and possible right hepatic lobectomy. A hepatic mass had been detected on an abdominal ultrasound obtained to evaluate epigastric pain and diarrhea. A computed tomograph confirmed the finding. Her medical history was remarkable for cholelithiasis, hypercholesterolemia, well-controlled hypertension, and palpitations believed to be caused by paroxysmal supraventricular tachycardia. She had previously undergone total abdominal hysterectomy and bilateral salpingo-oophorectomy for ovarian cancer and was not aware of any prior anesthetic complications. Her medications included verapamil, gemfibrozil, conjugated estrogens,