Recognizing Sleep-Disordered Breathing in Hospitalized Patients: Review Questions

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QUESTIONS

Choose the single best answer for each question.

Questions 1 and 2 refer to the following case.

A 67-year-old man with type 2 diabetes mellitus, obesity, and hypertension is admitted to the intensive care unit for acute lower gastrointestinal bleeding. He undergoes an urgent prepped colonoscopy after volume resuscitation. He receives midazolam 1 mg and fentanyl 25 µg. His eyelids close, and he does not respond to voice. Within a few minutes, the nurse reports that the patient's pulse oximetry reading is falling. He is making an effort to breathe, but there are paradoxical movements of the chest and abdomen.

1. Which intervention will most likely correct the oxygen desaturation in this patient?

(A) Flumazenil
(B) Intubation
(C) Jaw thrust
(D) Naloxone

2. What is the most likely cause of this patient's response to conscious sedation?

(A) Aspiration pneumonitis
(B) Hemorrhagic shock
(C) Obstructive sleep apnea (OSA)
(D) Sepsis

Questions 3 and 4 refer to the following case.

A 56-year-old woman presents to the emergency department complaining of leg swelling and dyspnea. She is placed on diuretics but experiences little relief. The patient has a history of difficult-to-control blood pressure, currently treated with lisinopril, hydrochlorothiazide, and amlodipine. She reports that for the past several years she has been unable to sleep flat and has headaches on a regular basis. Pulmonary function testing shows a modest restriction with a total lung capacity of 70% and diffusion capacity for carbon monoxide (DLCO) of 70%. A recent pharmacologic stress test showed no perfusion defects and a normal left ventricular ejection fraction. Kidney function and spot urine protein screen are normal. The patient is admitted for further cardiac evaluation. On admission, blood pressure is 160/100 mm Hg, heart rate is 102 bpm, and peripheral oxygen saturation is 92% on room air. She is 5 ft 6 in and weighs 305 lb. HEENT examination reveals no jugular venous distention and a normal thyroid, but the tonsils and uvula cannot be seen. Cardiovascular examination reveals a regular rate and rhythm, no murmurs, and S_4 present at the left lower sternal border. The chest is clear to auscultation bilaterally with no retractions. Pulses in the extremities are normal, there is 2+ edema in the ankles, and there is no clubbing or cyanosis. Laboratory testing reveals a hemoglobin level of 15 g/dL, serum creatinine of 1.0 mg/dL, blood urea nitrogen of 14 mg/dL, and bicarbonate level of 30 mEq/L. Portable chest radiograph demonstrates clear lung fields. Arterial blood gases on room air include a pH of 7.40, $Paco_2$ of 50 mm Hg, Pao_2 of 62 mm Hg, and an oxygen saturation of 92%. Overnight oximetry is performed (**Figure**).

3. What is the most likely cause for this patient's symptoms?

(A) Hypothyroidism
(B) Obesity hypoventilation syndrome (OHS)
(C) Polycythemia vera
(D) Pulmonary fibrosis

4. Which of the following is the next best step in the management of this patient?

(A) Bilevel positive airway pressure (BiPAP) at 10/5 cm H_2O
(B) Cardiac catheterization
(C) Oxygen and intravenous furosemide
(D) Polysomnography

5. A 62-year-old man with a history of hypertension, type 2 diabetes, coronary artery disease, and chronic obstructive pulmonary disease (COPD) is admitted

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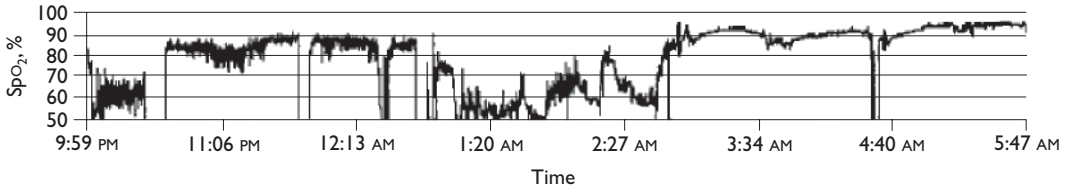

Figure. Overnight oximetry performed in the patient described in questions 3 and 4. Spo_2= oxygen saturation as measured by pulse oximetry.

to a hospital after a low-speed motor vehicle accident in which he hit a light post due to loss of consciousness. He was alert and oriented at the scene of the accident but could not recall anything after he got into his car to drive. Initial evaluation including cardiac enzymes, telemetry, and serial electrocardiograms (ECGs) is unremarkable. Examination findings include a body mass index (BMI) of 28 kg/m^2, hemoglobin level of 17.2 g/dL, bicarbonate level of 50 mEq/L, resting peripheral oxygen saturation of 88% on room air, droopy eyelids, clear but distant lung sounds, and trace edema. Thyroid-stimulating hormone is 4 µIU/mL. Blood gases on room air show a pH of 7.36, Pco_2 of 54 mm Hg, and Po_2 of 58 mm Hg. Computed tomography of the chest shows hyperinflated lungs, centrilobular emphysema, and prominent pulmonary arteries without evidence of pulmonary embolism. Telemetry shows no arrhythmias. Chemical stress testing shows no ischemia. Echocardiogram shows normal left ventricular function and evidence of pulmonary arterial hypertension with peak pressures of 48 mm Hg. Pulmonary function tests are significant for forced vital capacity (FVC) of 73% predicted, forced expiratory volume in 1 second (FEV_1) of 54% predicted, and FEV_1/FVC ratio of 60% and a diffusion capacity of 55%, confirming moderately severe COPD. Further history indicates excessive daytime somnolence, unrefreshing sleep, snoring, and witnessed choking episodes while sleeping. Which of the following is the most likely cause of this patient's loss of consciousness while driving?

(A) COPD exacerbation with hypercapnia
(B) OHS
(C) Pulmonary overlap syndrome
(D) Seizure disorder

ANSWERS AND EXPLANATIONS

1. **(C) Jaw thrust.** Conscious sedation is widely used in a variety of procedures, including endoscopies and minor surgical procedures. Usual medications include benzodiazepines (eg, midazolam), which act as amnestics and anxiolytics, and opiates (eg, fentanyl), which provide pain relief. These medications not only suppress respiratory drive centrally but also lower the tone of pharyngeal structures.[1] Although flumazenil and naloxone are reasonable options in this case, the first step should be a jaw thrust/chin lift maneuver to make the airway patent. The degree of difficulty in visualizing the tonsillar pillars, the soft palate, and the base of the uvula predicts difficulty with intubation.[2]

2. **(C) OSA.** Clinicians providing conscious sedation should be cognizant of the possibility of OSA syndrome. The prevalence of OSA in patients presenting for surgery has been estimated at 1% to 9%, although it may be more common but undiagnosed in certain populations.[2] Medications such as midazolam and fentanyl may provoke obstructive apneas or hypopneas. While central hypoventilation, or central apnea, is characterized by a decreased central effort to breathe, the hallmark of obstructive apnea is obstruction at the palato glossopharyngeal level. Often, the apneas can be mixed. Falling oximetry, continued effort to breathe, and paradoxical movements of the chest and abdomen after relatively modest doses of midazolam and fentanyl are suggestive of underlying OSA rather than aspiration pneumonitis, hemorrhagic shock, or sepsis.

3. **(B) OHS.** This patient has severe obesity (BMI, 49.2 kg/m^2), headaches likely due to hypercapnia, unrefreshing sleep, evidence of cor pulmonale, secondary erythrocytosis due to chronic hypoxia, and Malampati class 4 airway with nocturnal desaturations. Therefore, the most probable diagnosis is OHS, which is defined as a combination of obesity (ie, BMI ≥ 30 kg/m^2) and awake chronic hypercapnia (ie, $Paco_2$ > 45 mm Hg) accompanied by sleep-disordered breathing, usually severe OSA syndrome.[3] If left untreated, patients with OHS can develop pulmonary hypertension and cor pulmonale.[4] Patients with OHS have an elevated serum bicarbonate level due to the metabolic compensation for the chronic respiratory acidosis.[5] Compared

with patients with similar degrees of obesity, patients with OHS have increased medical resource utilization and are more likely to be hospitalized and require intensive care monitoring. Pulmonary hypertension is more common and more severe in patients with OHS than in those with OSA syndrome (50% versus 15%, respectively).[6] It is essential for clinicians to maintain a high index of suspicion for OHS, particularly because early recognition and treatment improve outcomes. Polycythemia vera, a myeloproliferative disorder, is less likely in this case because it typically involves elevated hematocrit and splenomegaly in the absence of chronic hypoxemic stimulus to erythropoietin production. Pulmonary fibrosis would be expected to cause a restrictive ventilatory defect, along with hypoxia and, in severe cases, cor pulmonale. The absence of velcro crackles and normal lung examination and clear lung fields in this patient along with relatively preserved DLCO further rules out the diagnosis of pulmonary fibrosis. This patient's normal thyroid examination and resting heart rate (102 bpm) argue against profound hypothyroidism; however, further biochemical testing is needed to rule this out.

4. **(D) Polysomnography.** An observed polysomnogram should be ordered to establish the severity of OSA syndrome and the degree of oxygen desaturation. In addition, results of polysomnography can be used to immediately and effectively titrate the positive airway pressure (PAP) to resolve obstructive events and correct the hypoxemia. Approximately half of patients with OHS require oxygen therapy in addition to PAP therapy upon initiation of treatment. Although continuous PAP alone may suffice in about half of cases, BiPAP and, in a significant number of cases, supplemental oxygen may be necessary.[7] Supplemental oxygen and furosemide are reasonable options; however, oxygen alone will not alleviate the hypoventilation and obstructive apneas. Furosemide may improve the edema but it will not resolve in the setting of hypoventilation syndrome. There is no role of cardiac catheterization in the absence of ischemia. BiPAP at 10/5 cm H_2O may not suffice if started empirically, as the degree of OSA contributing to hypoventilation is unknown and the amount of positive end-expiratory pressure needed to resolve the obstructive component of the disease can only be determined through accurately measuring respiratory effect, airflow, and oxygen saturations, all components of the polysomnogram.

5. **(C) Pulmonary overlap syndrome.** This patient has pulmonary overlap syndrome, a term used to describe the association between COPD and OSA. COPD and sleep apnea-hypopnea syndrome are both common diseases, affecting 10% and 5% of the adult population over age 40 years, respectively; their coexistence, which is called overlap syndrome, can be expected to occur in approximately 0.5% of this population.[8] Patients with overlap syndrome have an obstructive pattern on spirometry and are more likely to have hypoxemia, hypercapnia, and pulmonary hypertension compared with patients with simple OSA.[9] In overlap syndrome, hypercapnia develops in patients with a lower BMI and at a lower apnea/hypopnea index as compared with hypercapnic patients with OHS without obstructive defects and at a higher FEV_1 than hypercapnic patients with COPD alone. This patient certainly has COPD, which explains the resting hypoxia, secondary erythrocytosis, and pulmonary hypertension; however, COPD usually does not cause chronic hypercapnia at his level of obstruction. This patient also likely has OSA syndrome, but OSA alone is unlikely to cause this degree of hypercapnia and is not associated with moderate pulmonary hypertension unless associated with another cause for daytime hypoxia. This patient is not obese, which rules out OHS.

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