Obstructive Sleep Apnea (OSA)

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Background and Presentation

Definition

OSA is characterized by periodic collapse of the upper airway leading to partial or full obstruction to airflow that occurs during sleep, which results in apnea or hypopnea. The episode is usually terminated by momentary arousal. This may cause intermittent hypoxemia, hypercapnia, disordered sleep, and sympathetic stimulation. OSA is associated with hypertension, cardiac, cerebrovascular, neurocognitive, and metabolic disease [1–3].

Incidence

1. The incidence of OSA in pregnancy is unknown, due to under diagnosis and lack of data. The incidence of OSA in obese parturients may be as high as 15.4% [4]. Up to 90% of women with OSA have not been diagnosed, and the prevalence increases as pregnancy progresses [5].
2. In the general population, the prevalence is both high and increasing.
   (a) Moderate-to-severe disease has an estimated prevalence of 10–17% in men and 3–9% in women. This is based on the apnea-hypopnea risk index (AHI), which defines moderate-severe apnea as ≥15 events per hour. These events last for 10s and are associated with desaturation [6].
   (b) When including patients with lower AHI classification, the prevalence is much higher.
   (c) OSA affects approximately 27% of women and 43% of men between the ages of 50 and 70 years old and 9% of women and 27% of men between the ages of 30 and 49 years old. Other authors offer a much higher prevalence of OSA [2, 7].
3. Patients at higher risk for OSA include obese patients who have a more structurally narrow airway with a propensity to pharyngeal airway collapse. Moderate-to-severe OSA occurs in 65% of obese males and in 23% of obese females. Others have suggested an even higher incidence of OSA in obese patients [8]. Pregnancy, like obesity, is also an independent risk factor for OSA [2, 6].

Symptoms

Patients may complain of loud snoring, gasping, disordered sleep, and daytime somnolence. Most symptoms develop at a more advanced age when patients may develop cardiac, cerebrovascular, psychiatric, cognitive, and metabolic complications. Arrhythmias, heart failure, coronary artery disease, sudden death, cor pulmonale, stroke, diabetes, and memory loss are some of the long-term effects of OSA.

Pathophysiology

Most patients with OSA have a crowded narrow oropharyngeal space. When the pharyngeal transmural pressure decreases, airway collapse occurs. This may be exacerbated by reduced ventilatory motor output to the muscles of the upper airway, a reduction in pharyngeal dilator activity, and decreased compensatory responses to airway obstruction. Other factors that contribute to upper airway collapse include gravitational forces and dynamic factors such as the Bernoulli effect, increased pharyngeal airway resistance, and increased pharyngeal wall adherence. The pathophysiology of OSA is complex and evolving. However, chronic intermittent hypoxia, hypercarbia, and negative

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intrathoracic pressures result in a number of changes that affect the development of pathologic states. These include autonomic system disarray, sympathetic system activation, increased aldosterone and renin, activation of the hypothalamic-pituitary-adrenal axis, induction of oxidative stress with production of reactive oxygen species, impaired glucose metabolism, enhancement of a pro-inflammatory and pro-thrombotic response, and endothelial dysfunction, to name a few [1, 9–15].

Testing [16]

1. OSA is diagnosed by polysomnography. Aspects of this testing may include pulse oximetry, airflow measurement, chest and abdominal movement, electroencephalography (EEG), electromyography, and electrooculography.
2. The AHI is often used to describe the severity of the OSA. An apnea event may be defined as absence of flow for at least 10 seconds, and hypopnea is a reduction in flow of more than 30% for at least 10 seconds. These episodes are followed by desaturation and EEG evidence of arousal. Five events per hour is within normal limits, and 5–14, 15–29, and 30 events per hour represent mild, moderate, and severe OSA, respectively.
3. Several questionnaires such as the Berlin, Epworth Sleepiness Scale, and the STOP-BANG scoring system have been used to help diagnose OSA. The STOP-BANG survey is commonly used to determine whether patients have any of eight signs and symptoms associated with OSA: loud snoring, daytime tiredness, observed obstruction during sleep, high blood pressure, a basal metabolic rate of >35 kg/m^2, age over 50, neck circumference >40 cm, and male gender. However, the specificity of most questionnaires is poor, leading to many false-positive findings. These scales may not to be sensitive or specific in pregnant women [17].
4. Other tests will be determined based on the duration, severity, and presence of comorbidities such as pulmonary hypertension in patients with OSA.

Interaction with Pregnancy

Effect on Pregnancy on OSA [18–20]

1. Self-reporting on questionnaires notes an increased incidence of snoring due to increased upper airway resistance during pregnancy.
2. Weight gain, elevation of the diaphragm, and decreased functional residual capacity could exacerbate symptoms of OSA.
3. Narrowed upper airways have been reported during pregnancy.
4. Estrogen-induced congestion and rhinitis, edema, and venous engorgement may contribute to OSA exacerbation by narrowing the airway and increasing airflow resistance during pregnancy.
5. Progesterone-induced hyperventilation and alkalosis could cause an increased respiratory drive with more negative upper airway inspiratory pressure leading to airway collapse.
6. Decreased functional residual reserve combined with caudal traction of the pharynx and trachea by the lung during inspiration augments upper airway collapse.
7. Patients may develop OSA during pregnancy, and women with OSA may have worsening of OSA during pregnancy [5, 20]. One study of nonobese pregnant women did not notice an increased prevalence of OSA [21]. Others have shown an increased prevalence of OSA particularly as pregnancy progresses with improvement postpartum [5].

Effect of OSA on Pregnancy

1. There is growing evidence that OSA has both maternal and fetal consequences.
2. OSA has been associated with gestational hypertension, preeclampsia, and diabetes [22–26].
3. One large cross-sectional retrospective study found that in addition to preeclampsia, eclampsia, and diabetes, pregnant women with OSA have an increased risk of cardiomyopathy, pulmonary edema, pulmonary embolism, and in-hospital mortality [27].
4. OSA is also a consistently cited risk factor for cesarean delivery (CD) [4, 25, 27–29].
5. Fetal growth restriction resulting in small for gestational age and low birthweight infants and preterm delivery (PTD) has been reported to occur in the offspring of pregnant women with OSA [4, 22, 24, 25, 29–32].
6. Many pregnant women with OSA are obese with one study noting an obesity rate of 40.23% compared to 7.87% in women with OSA [22]. Others report that up to 70% of obese patients have OSA [33]. Obesity is associated with maternal diabetes, preeclampsia, preterm labor, PTD, CD, and infection [34]. Thus, obese women with OSA represent a particularly high-risk group for adverse pregnancy outcomes [22, 27, 35].
7. Most studies are retrospective in nature, and many have not been able to adjust for confounding variables and to have sufficient power to detect rare events. Thus, further investigation is warranted to assess reports of poor cardiovascular and neonatal outcomes.

Treatment

Medical

1. The treatment for OSA consists of continuous positive airway pressure (CPAP) or bi-level positive airway pressure.
2. In nonpregnant women, CPAP has been shown to decrease cardiovascular risk factors [1, 9]. Preoperative and perioperative CPAP application may decrease a number of cardiovascular, airway, and respiratory complications as well as the length of the hospital stay [6, 17, 36].
3. Women who use CPAP prior to pregnancy should continue CPAP therapy during pregnancy. CPAP may need to be adjusted as gestational age increases, and compliance may be compromised during pregnancy given the presence of nasal congestion and discomfort [37].
4. CPAP appears to improve neurocognitive and cardiovascular impairment during pregnancy [5].There is evidence that CPAP decreases both systolic and diastolic blood pressures, particularly in pregnant women with hypertensive disorders. CPAP treatment may therefore reduce the need for antihypertensive medications. Decreases in blood pressure and increases in cardiac output have been noted in preeclamptic women using CPAP. CPAP may prevent at risk pregnant women from developing preeclampsia. There may also be decreased intrauterine growth retardation in fetuses born to women compliant with CPAP therapy. Thus, CPAP use during pregnancy likely improves cardiovascular and fetal outcomes. However, additional studies are needed to validate these findings [18, 37–40].

Anesthetic Management

1. General endotracheal anesthesia (GETA) in pregnant women with OSA may be complicated by difficulty with both ventilation and intubation. Pregnancy and obesity are both independent risk factors for difficult intubation [41]. The risk of failed intubation in obese pregnant women is at least eight times higher than in nonobese parturients [42]. OSA is also an independent risk factor for difficult ventilation and intubation. Two retrospective studies noted difficult intubations occurring in approximately 22–43% of patients with OSA compared to 2–3% without OSA, and 5% of patients could not be intubated [33, 43]. Thus, pregnant women with OSA represent a particularly high-risk group for airway mishap.
2. Neuraxial anesthesia should be strongly encouraged in pregnant women with OSA.
   (a) Neuraxial anesthesia mitigates potential airway misadventure, apnea, aspiration, sedation, and the need for postoperative patient-controlled analgesia (PCA) with opioids, which can all cause respiratory depression.
   (b) There is evidence that regional anesthesia results in decreased postoperative complications compared to GETA in the general population [44]. The guidelines established by the American Society of Anesthesiology endorse the use of regional anesthesia in patients with OSA [45]. Spinal, epidural, or combined spinal-epidural anesthesia could be used for CD.
   (c) Intrathecal opioid or patient-controlled epidural analgesia should be administered for postoperative pain management to avoid the respiratory depression seen with systemic opioids.
   (d) However, the anesthesiologist must be mindful that patients with OSA are also more sensitive to the respiratory depressant effects of neuraxial opioids, perhaps due to upregulation of opioid receptors induced by recurrent episodes of hypoxemia. Continuous epidural infusions with fentanyl and bupivacaine have been associated with adverse cardiorespiratory events [20]. The incidence of respiratory depression with neuraxial morphine in pregnant women ranges from 0 to 0.9% [46]. Thus, all patients with OSA who receive even low-dose intrathecal morphine should be monitored by pulse oximetry and capnography, if possible, in the postpartum period [37].
3. Labor epidural analgesia should be encouraged and placed early in labor. Obesity has been associated with both more painful labors and CD [37]. Placing labor epidural analgesia in obese patients with OSA may pose a technical challenge; however, labor epidural analgesia results in less opioid consumption, sedation, and respiratory depression than alternative forms of analgesia. In addition, epidural analgesia can be used to extend the block for CD anesthesia, if indicated. CPAP therapy can be continued during labor for women with moderate-severe OSA.
4. GETA
   (a) If GETA becomes necessary, preoxygenation with 100% oxygen with the application of CPAP is recommended. Aspiration prophylaxis should be administered prior to induction.
   (b) A difficult airway cart should be available, and a Troop Elevation Pillow may facilitate intubation.
   (c) Given that these patients are known to have a higher rate of difficult and failed intubation, consideration should be given to awake fiber-optic intubation or use of a video laryngoscope [33].

(d) Induction may be done in the head-up or sitting position. Short-acting anesthetic medications and less-soluble inhalation anesthetics should be used with careful titration of opioids and other sedatives. Short-acting opioids are preferred to long-acting medications.

(e) The patient should be extubated after complete reversal of neuromuscular blockade and when fully awake in the non-supine position. The use of CPAP during extubation may be beneficial.

(f) Patients with OSA may have decreased pain tolerance as well as increased sensitivity to opioids [44]. A closed claims analysis of causes of death and ischemic brain injury in the general population found that respiratory depression usually occurred during the first 24 h after surgery and that 40% of cases involved patients with OSA [33, 44]. Thus, if possible, either postoperative intrathecal opioid or epidural analgesia should be strongly considered.
- If PCA is necessary, a basal infusion rate should not be administered and alternative forms of analgesia should be strongly encouraged.
- Such alternative intravenous medications include dexmedetomidine and alpha-agonists such as clonidine. Anti-inflammatory medications, acetaminophen, tramadol, ketamine, and dexamethasone may be beneficial [37]. In addition, a transverse abdominis block could be performed.
- Extreme vigilance must be used in the postoperative period. These patients should recover in the non-supine position and have continuous cardiopulmonary monitoring for a prolonged period of time. Elevation to 45° has been recommended for pregnant women with OSA in the postpartum period [47]. CPAP can be employed and may decrease airway obstruction, postoperative complications, and the length of admission.

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