Obstructive Sleep Apnea in Pregnancy

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A 25-year-old woman, gravida 4 para 2, at 37 weeks gestation was evaluated and treated for preeclampsia. Overnight, the patient had a witnessed apneic episode with maternal oxygen desaturation and concurrent fetal heart rate deceleration. She subsequently delivered an infant that was small for gestational age. This is the first case described with confirmed obstructive sleep apnea by formal polysomnography and witnessed maternal desaturation with fetal heart rate decelerations. Recognizing obstructive sleep apnea (OSA) early in gestation will help dictate treatment options and may prevent adverse maternal fetal outcomes. Continuous positive airway pressure (CPAP) seems to be a safe treatment with minimal adverse effects. Questioning of patients at the first prenatal visit and monitoring for increased snoring during gestation may help detect early signs and symptoms of OSA. Treatment of OSA with CPAP might improve perinatal outcomes. (J Am Board Fam Pract 2004;17:292–4.)

Obstructive sleep apnea (OSA), characterized by upper airway obstruction and nocturnal hypoxemia during sleep, occurs in 2% of women in the general population.[1] This condition has only rarely been reported in pregnancy. We found 7 cases of documented OSA in pregnancy. Two patients had infants confirmed small for gestational age, 3 had infants of normal size, and 2 did not comment on the growth of the infant. Four cases were associated with preeclampsia and one case reported pulmonary hypertension.[1,2] We present a preeclamptic gravida with previously undiagnosed obstructive sleep apnea who demonstrated a fetal heart rate deceleration associated with an apneic episode and who subsequently delivered an infant with intrauterine growth restriction (IUGR).

Case Presentation

A 25-year-old woman, gravida 4, para 2, presented at 37 weeks, and 3 days of gestation, presented to labor and delivery with complaints of worsening lower extremity edema, headache, dizziness, and scotomata. On physical examination, the patient's vital signs were: pulse, 106 beats/min; respirations, 16 breaths/min; temperature, 98°F, blood pressure, 152/52 mm Hg. Laboratory results were: platelets, 221 $\times$ 10^3/mm^3; hemoglobin, 11.4 g/L; hematocrit, 0.324; white blood cell count, 11.9 $\times$ 10^3/mm^3; sodium, 137 mEq/L; potassium, 2.9 mEq/L; serum urea nitrogen, 3 mg/dL; creatinine, 0.7 mg/dL; uric acid, 4.3 mg/dL; and magnesium, 1.2 mg/dL. Twenty-four hour total urinary protein was 390 mg/dL. Patient weight was 203 pounds after gaining 32 pounds during gestation.

Past medical history was significant for anxiety/panic disorder, sleepwalking, asthma, supraventricular tachycardia, gastroesophageal reflux disease, and seasonal allergies. Present medications included albuterol, atenolol, and fluticasone propionate. Family history was significant for hypertension. Obstetrical history included preeclampsia in the previous pregnancy.

The patient was admitted for observation and further evaluation of her presenting symptoms. During the overnight hours, the nursing staff noted that the patient snored loudly when she was asleep and they observed several apneic episodes of unknown duration. Fetal heart tones were auscultated at 90 to 100 beats/min during one such episode when the maternal oxygen saturation had fallen to 80%. The patient was awakened, and the fetal heart tones returned to a satisfactory baseline and were reactive.

A serial labor induction over 3 days using intravenous Pitocin was conducted. Persistent late decelerations appeared, and a cesarean section was performed. Blood pressure at that time was 141/91 mm Hg. A 4-lb, 4-oz (1.7 kg) male infant, who was

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small for his gestational age, was delivered with Apgar scores of 8 and 9. Cord pH was 7.21. Head circumference was 31 cm, length was 40.7 cm, and abdominal circumference was 24.2 cm. Placental weight was 335 g. The infant exhibited hypoglycemia, poor weight gain, hypospadias, pyelocaliectases, and a heart murmur. An echocardiogram revealed a left branch pulmonary stenosis. At 4 months old, the infant appeared to be growing and developing normally.

The patient received a formal sleep study before leaving the hospital and was found to have severe obstructive sleep apnea with an apnea-hypopnea index of 160 (normal, ≤5; severe, >30). She also had episodes of sinus bradycardia with the obstructive events during rapid eye movement sleep. She was placed on continuous positive airway pressure (CPAP), and there was marked clinical improvement of her apnea-hypopnea index and daytime symptoms.

Discussion

Ours is the first case with polysomnographically confirmed OSA and documented fetal heart rate changes during an apneic episode associated with maternal oxygen desaturation. OSA was first reported in pregnancy in 1978.[3] Joel-Cohen and Schoenfeld[3] reported 3 cases of clinical OSA, not confirmed by polysomnography, in pregnancy with apnea-associated changes in fetal heart rate. Only 7 additional cases of OSA in pregnancy could be found; however, none documented fetal heart rate changes with apneic episodes. As in our patient, preeclampsia was noted in 4 cases and all reported infant weights were less than that of the 50th percentile.[1]

The prevalence of OSA in pregnancy is unknown, although it has been suggested that pregnancy may precipitate or exacerbate this condition.[4] The physiologically high levels of progesterone in pregnancy are thought to act as a ventilatory stimulant and perhaps increase pharyngeal muscle tone. Increased upper airway resistance may occur in pregnancy secondary to diffuse pharyngeal edema.[5] These normal physiologic changes could potentially worsen OSA in pregnant women.[4]

Hypertension is often associated with OSA;[6] 5 of 7 reported cases have also had preeclampsia. Habitual snoring, the most common symptom of OSA, has been associated with hypertension, and during pregnancy, it has been associated with preeclampsia and intrauterine growth restriction.[7] Nasal CPAP is the mainstay of therapy for OSA and has been shown to significantly reduce blood pressure and uric acid levels in women with preeclampsia.[8] CPAP has been successfully used during pregnancy. Theoretically, it is possible that there could be adverse effects on uteroplacental perfusion,[9] although these have not been reported.

Maternal apneic episodes have now been associated with fetal cardiotocographic changes in 4 cases. Maternal oxygen desaturation during apnea may result in fetal hypoxia as demonstrated with resultant fetal heart rate abnormalities. Episodic fetal hypoxia may result in poor fetal growth. This has been reported in 3 other cases, as well as in the present case; however, our patient was exposed to antenatal atenolol, a drug known to be associated with IUGR.

Primary care physicians are likely to have patients with OSA and are in a unique position to identify those at risk for OSA. Patients with obvious signs and symptoms of OSA are usually sent for formal sleep studies. However, patients with mild to moderate disease may be underdiagnosed.[10] Our patient had a history of sleep walking, anxiety/panic disorder, and supraventricular tachycardia, all possible sequelae from OSA. She had risk factors of obesity, snoring, pregnancy, and seasonal rhinitis. No set of criteria has been developed that accurately predicts OSA, although male gender, body mass index >30, witnessed apnea, excessive daytime sleepiness, and hypertension are predictive.[11] Even if a patient has all five factors, the positive predictive value is only 61%. At this time, only the physician's overall clinical impression can be used to assess the need for formal polysomnography. This is caused by the significant overlap of symptoms in patients without sleep apnea.[11]

Future studies are needed to determine the true prevalence of OSA in the pregnant population and its association with preeclampsia and IUGR. If studies continue to be consistent with our findings, consideration should be given to questioning patients at the first prenatal visit and subsequent visits for symptoms of OSA, with the potential to diagnose, treat, and subsequently reduce the associated IUGR and preeclampsia. Any development or worsening of symptoms should prompt the physician to send the patient for formal polysomnogra-

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phy testing. Nasal CPAP has demonstrated no adverse effects during pregnancy[1] and may be beneficial in conjunction with serial assessment of fetal well-being.

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