Obstructive sleep apnea (OSA) results from a structural compromise of the upper airway combined with decrease in muscle tone during sleep. Overt upper airway pathology is rare, however a variety of pharyngeal tumors have been well described as a cause of OSA. We describe a case of a mass originating in the carotid body resulting in severe OSA with hypersomnia resistant to positive pressure ventilation.

**Keywords:** Obstructive sleep apnea (OSA), carotid body tumor.

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**REPORT OF CASE**

A seventy-six (76) year-old man, a retired lawyer, was diagnosed with OSA by a polysomnogram 3 years prior, and continuous positive airway pressure (CPAP) was prescribed. He was intolerant of it and therefore noncompliant. He had worsening of his excessive daytime hypersomnia, snoring, and apneas, and he sought a second opinion about his disease. He had history of coronary artery disease, diabetes mellitus, stroke, and depression. On physical examination, he weighed 200 lbs (91 kg), height was 73 inches (185.42 cm), BMI was 26.2, and neck circumference was 16 inches (40.6 cm). The rest of the examination was significant for a muffled voice with slurred speech, an elongated facial height, slight anterior overbite, and a mild left septal deviation of the nose. The soft palate and uvula were elongated but not notably obstructing the upper airway.

Polysomnography identified significant hypoxia reaching 61%, with an apnea hypopnea index (AHI) of 33. Treatment with CPAP and even bilevel positive airway pressure did not resolve REM associated desaturations and hypopneas.

After failure of acclimatization to bilevel pressure, he was referred to the otolaryngology service for possible surgery. Fiberoptic laryngoscopy revealed normal lingual tonsils, larynx, and supraglottic area. Hypopharyngeal examination showed fullness in the left lateral wall of hypopharynx in the sitting position, but a marked obstruction was noted when supine with severe obstruction at the level of the epiglottis. CT scan (figure 1) followed by MRA revealed a 3 cm X 2 cm growth with a mass effect on the lateral hypopharyngeal wall consistent with a carotid body tumor. His 24-hour urine epinephrine, plasma metanephrine, and normetanephrine levels were all normal.

A multidisciplinary tumor board advised resection, but the patient declined the surgical option.

**DISCUSSION**

OSA has been reported to occur with lipomas, retention cysts, palatine hemangiomas, tonsillar lymphomas, and salivary gland tumors. Paraganglioma occur in 1/30,000 of head and neck tu-
mors, making them very rare tumors. Carotid body tumors are usually asymptomatic, grow slowly, originating in the chemoreceptors of the carotid body, and can go unnoticed for many years. Mayer-Brix et al reported pharyngeal tumors to be the cause of OSA in 3 out of 336 patients who underwent thorough evaluation for the cause of OSA. Hoijer U et al evaluated 6 patients with pharyngeal tumors for evidence of OSA and found that all patients had either polysomnographic or oximetric evidence of OSA with average ODI of 24 (range 10-58) with complete resolution after treatment.

While neoplasms of the upper airways can rarely cause (and present with) OSA as outlined previously; early diagnosis may be delayed as in our patient. Careful history taking and physical examination of the upper airways, assisted by imaging techniques of the head and neck using computed tomography, magnetic resonance, and occasionally endoscopy is crucial to early diagnosis and intervention.

Most patients with conventional OSA should improve with application of CPAP therapy, making it a very effective and noninvasive modality of treatment if patients comply with treatment. Compliance rates are reported to be between 40%-60%. Poor adherence to CPAP can be due to multiple factors, including psychological (claustrophobia), nasal dryness, difficulty adapting to pressure, and mask discomfort. It is well accepted that interventions such as patient education, close follow-up, and improving mask fit and other technical problem are important in maximizing patient compliance.

This patient is, to our knowledge, one of 3 cases reported in which OSA appeared to be caused by a carotid body tumor. As in the initial case report, because of patient request, no resection was attempted. It has been suggested by expert opinion that surgery not be performed on patients >50 years because of high morbidity associated with surgical resection and low chance of growth and malignancy of these tumors.

This case highlights the importance of a comprehensive evaluation of OSA patients that represent a challenge to the sleep clinician. Our case also emphasizes the great value of positional laryngoscopy and imaging studies in patients with refractory OSA to assess structural and motor dysfunction of the upper airway.

REFERENCES: