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Clinical Corners in Sleep Medicine

Management of obstructive sleep apnea in acromegaly

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A 38-year-old female presented with hand and foot growth, headaches and facial pain, blurry vision, galactorrhea, snoring, and fatigue. Her body mass index was 29 kg/m^2, her neck circumference was 15 in, and her oropharynx revealed a Mallampati classification III. Her facial features were coarsened and frontal bossing was apparent. She had elevated serum levels of growth hormone (59 ng/ml) and insulin-like growth factor-1 (1254 ng/ml) and glucose loading failed to suppress growth hormone levels. Her brain magnetic resonance imaging (MRI) scanning (Fig. 1) revealed a multilobulated enhancing sellar mass consistent with a pituitary macroadenoma and she was diagnosed with acromegaly.

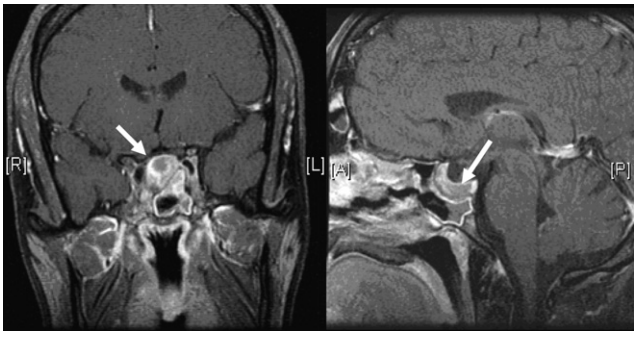

Fig. 1. Coronal (left) and sagittal (right) gadolinium enhanced T1 weighted MRI images revealing a pituitary macroadenoma (white arrows). The adenoma extends into the left cavernous sinus surrounding the internal carotid artery and approaches the optic nerves anteriorally.

One week after partial transphenoidal resection she was evaluated with polysomnography (Fig. 2). Her apnea–hypopnea index was 31 events/h with an oxygen saturation nadir of 89%. After an appropriate amount of time the patient underwent a continuous positive airway pressure (CPAP) titration (Fig. 2) and was started on a CPAP of 9 cm. Her residual pituitary adenoma was treated with 4500 cGy of fractionated radiation therapy, octreotide, and bromocriptine.

Initially, the patient had a positive response to CPAP with improved sleep and reduced fatigue and sleepiness. However, after 4 months her symptoms returned as did her acromegaly. She also began having problems wearing her CPAP due to acromegaly associated facial pain and nasal congestion.

Discussion questions

1. Can cure or adequate treatment of acromegaly subsequently cure associated obstructive sleep apnea?
2. How soon following transphenoidal surgery can a patient safely be treated with CPAP without causing pneumocephaly?
3. Are patients with acromegaly treated with radiation therapy at risk for developing sleepiness as a complication of treatment?
4. Lastly, is there a role for surgical therapy in the treatment of acromegaly associated obstructive sleep apnea?

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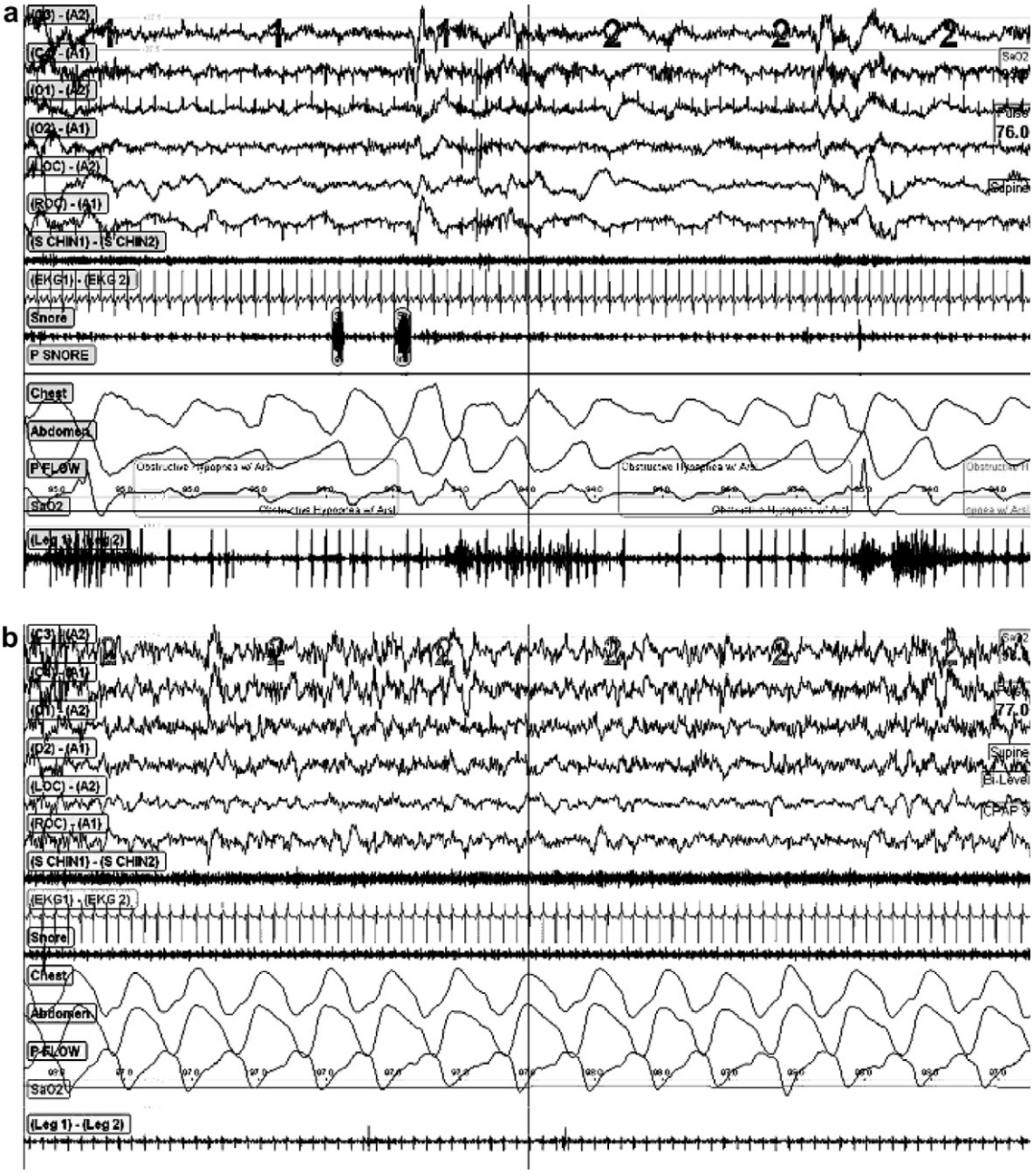

Fig. 2. (a) Sixty-second diagnostic polysomnography fragment revealing snoring, obstructive hypopneas with arousals, and paradoxical chest/abdomen movement characteristic of obstructive sleep apnea. Central (C3-A2, C4-A1) and occipital (O1-A2, O2-A1) electroencephalogram (EEG), left (LOC-A2) and right (ROC-A1) electrooculogram (EOG), submental electromyogram (S Chin 1–S Chin 2), electrocardiogram (EKG1–EKG2), microphone (Snore) and vibration detected snoring (P Snore-not functioning during this study), chest (Chest) and abdominal (Abdomen) respiratory impedence plesthymography, nasal airflow (P Flow), pulse oximetry (SaO_2), and left and right anterior tibialis electromyography (Leg 1–Leg 2). (b) Sixty-second CPAP titration polysomnography fragment revealing cessation of snoring, resolution of apneas and hypopneas, and consolidated uninterrupted sleep on a CPAP pressure of 9 cm. Chest and abdomen movements are now synchronized. Central (C3-A2, C4-A1) and occipital (O1-A2, O2-A1) electroencephalogram (EEG), left (LOC-A2) and right (ROC-A1) electrooculogram (EOG), submental electromyogram (S Chin 1–S Chin 2), electrocardiogram (EKG1–EKG2), microphone (Snore) detected snoring, chest (Chest) and abdominal (Abdomen) respiratory impedence plesthymography, nasal airflow (P Flow), pulse oximetry (SaO_2), and left and right anterior tibialis electromyography (Leg 1–Leg 2).