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Sleep disordered breathing as a delayed complication of iatrogenic vocal cord trauma

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Abstract

A case of a 55 year old woman with iatrogenic vocal cord trauma and sleep related symptoms is reported. In particular this case highlights sleep disordered breathing as a delayed complication after iatrogenic vocal cord trauma. The patient developed acute stridor from a contralateral vocal cord hematoma following vocal fold injection for right vocal cord paralysis. Acute respiratory symptoms resolved with oxygen, steroids and nebulized therapy, but nocturnal symptoms persisted and polysomnography revealed sleep-related hypoventilation and mild obstructive sleep apnea. Positive pressure therapy was successfully used to ameliorate her symptoms and treat sleep disordered breathing until her hematoma resolved. In addition to the typically acute respiratory symptoms that may result from vocal cord dysfunction, sleep disordered breathing may also present as a significant sub-acute or chronic problem. Management of the acute respiratory symptoms is relatively well established, but clinicians should be alert for more subtle nocturnal symptoms that may require further study with polysomnography.

Keywords

vocal cord paralysis; cancer; sleep-related hypoventilation; obstructive sleep apnea; vocal cord dysfunction; continuous positive airway pressure; sleep disordered breathing

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Introduction

A 55-year old woman with metastatic breast cancer presented with dysphonia and a weak cough. Laryngoscopy revealed right vocal cord paralysis and she underwent injection laryngoplasty (calcium hydroxylapatite gel) under stroboscopic guidance. Within 24 hours of the intervention, she developed stridor and dyspnea and was empirically placed on racemic epinephrine, dexamethasone and oxygen. She rapidly improved clinically and was discharged home on supplemental oxygen.

At her one week clinic follow up visit, she complained of persistent daytime fatigue, dyspnea and nocturnal “sounds”. In addition, she reported spontaneous arousals, anxiety and insomnia resulting in sleep disruption. Physical examination revealed a thin, anxious woman (BMI 20 kg/m^2) with 100% oxygen saturation at rest. There was audible stridor, but her lung fields were clear to auscultation. Repeat laryngoscopy showed a small glottic gap, with minimal bilateral abduction during inspiration and a hematoma that had dissected from the right side injection site to the left vocal cord. Given her persistent sensation of dyspnea and vocal cord dysfunction (VCD), tracheostomy was offered, but the patient refused.

Polysomnography (PSG) and positive airway pressure (PAP) titration were performed to evaluate her nocturnal symptoms. Significant sleep fragmentation was noted with reduced sleep efficiency of 76%, suboptimal REM sleep (13%) and delta sleep (13%), and an elevated arousal index of 25 arousals/hour of sleep. Significant sleep-related hypoventilation and mild obstructive sleep apnea (OSA) was also noted. PAP demonstrated improvement in sleep disordered breathing (SDB) with bi-level positive airway pressure (BIPAP). In the 3.6 hours at the optimal pressure, sleep disruption improved with sleep efficiency of 86% and arousal index of 14 arousals/hour of sleep. Patient reported resolution of dyspnea and anxiety with BIPAP. Her vocal cord hematoma resolved over time, and she was able to discontinue BIPAP after 7 to 14 days without symptom recurrence.

Image analysis

Figure 1 is a hypnogram revealing elevated transcutaneous end-tidal carbon dioxide ($ETCO_2$) throughout PSG. Figure 2 reveals pulmonary function testing at baseline and 1 week after procedure with abnormal flow-volume loop. Figure 3 displays PET-CT scan with narrowed glottis and left vocal cord inflammation and hematoma.

Discussion

VCD may have a significant impact in terms of respiratory symptoms and SDB. The use of PAP for acute respiratory insufficiency related to VCD has been well described. In contrast, there is a paucity of literature on VCD associated SDB. Intuitively, decreased glottic aperture may result in sleep-related hypoventilation due to airflow limitation. Associated laryngeal dysfunction can cause OSA similar to pharyngeal obstruction.(1)

In summary, we present the first description of the development of PSG documented SDB after iatrogenic vocal cord injury. PAP resulted in symptomatic improvement, both due to treatment of underlying SDB and facilitation of air entry through a narrowed glottis.

Following resolution of the hematoma, PAP was successfully discontinued. Since the injury was iatrogenic, the contralateral cord was not truly paralyzed; rather the hematoma reduced the glottic aperture despite maximal abduction with resulting functional flow restriction. PAP can be considered for the management of acute symptomatic VCD to forestall intubation or tracheostomy and may also have a role in treating associated SDB.

Acknowledgments

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References

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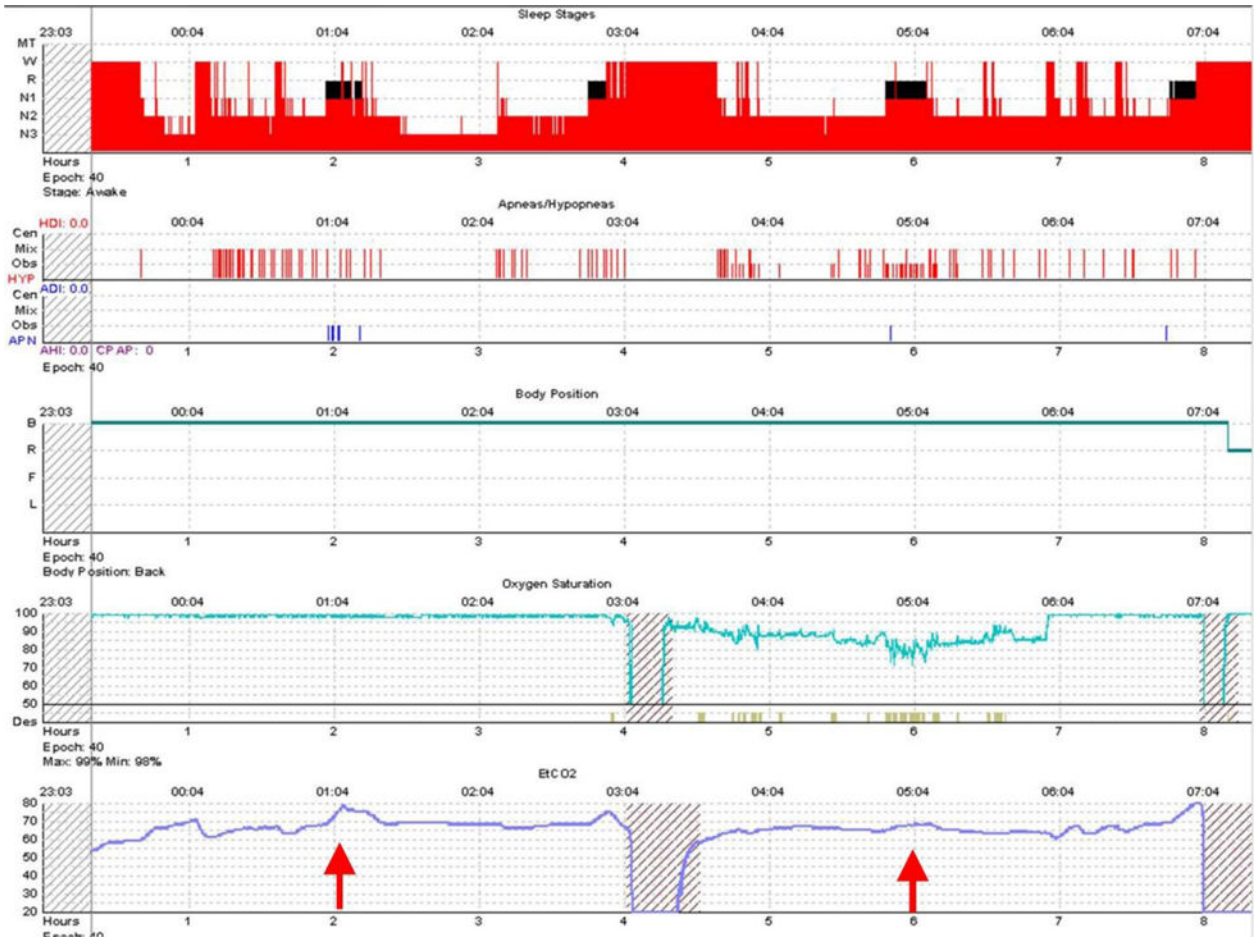

Figure 1.

Hypnogram from baseline polysomnogram revealing sleep stages, respiratory events, body position, oxygen saturations and transcutaneous end-tidal CO2 ($ETCO_2$) measurements. An apnea hypopnea index (AHI) of 6.1 episodes/hour of sleep with REM AHI 29 episodes/hour of sleep, and an oxygen saturation nadir (SaO_2) nadir of 71% was noted. $ETCO_2$ remains elevated throughout the study (red arrows). Baseline $ETCO_2$ was 52 mm Hg, mean REM $ETCO_2$ 68 mm Hg, and mean NREM $ETCO_2$ 62 mm Hg. Supplemental oxygen was used in the first part of the study then discontinued.

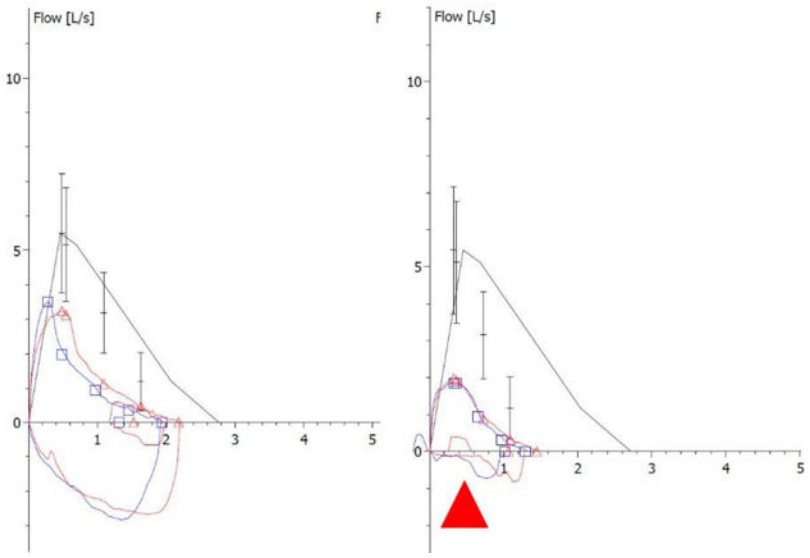

Figure 2.

Pulmonary function testing at baseline (A) and 1 week after procedure (B). Flattening of inspiratory loop (red arrowhead) is consistent with extrathoracic obstruction.

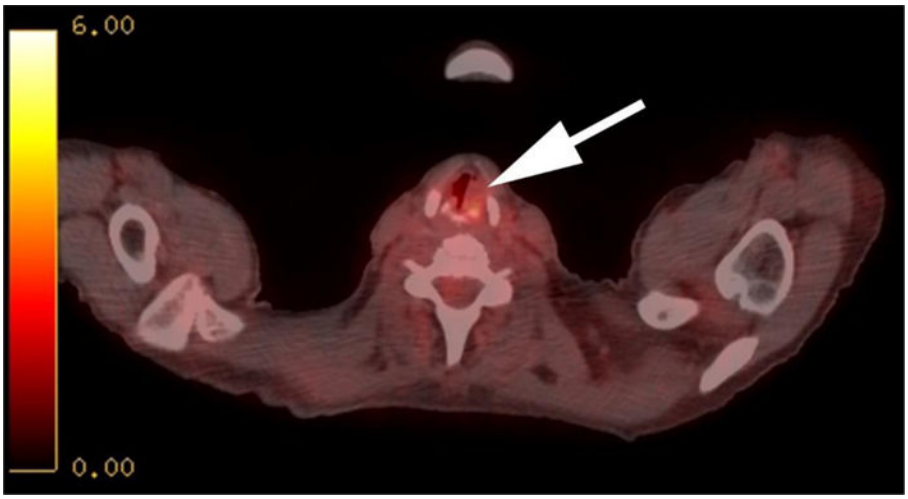

Figure 3.
PET-CT scan displaying narrowed glottis and left vocal cord (white arrow) with increased fluorodeoxyglucose (FDG) uptake consistent with inflammation and hematoma.