prompt resolution of oxygen desaturations during sleep. Serial capillary blood gases showed improvement with partial pressure of carbon dioxide
between 39-44 mmHg. Polysomnography on room air three days after caffeine initiation demonstrated resolved hypoxemia, hypoventilation, periodic breathing, obstructive sleep apnea, and central sleep apnea (central AHI of 6.8). The patient was discharged home on caffeine and continuous pulse oximetry during sleep. At follow-up six weeks later, the patient had no oxygen desaturations and was successfully weaned off caffeine.
Conclusion: To our knowledge, there are no prior reports of term infants being treated with caffeine citrate for primary central sleep apnea of infancy. While caffeine is an established therapy for apnea of prematurity, it is typically discontinued at a postmenstrual age of 32 - 34 weeks. Our case demonstrates that in term infants with no underlying medical conditions and primary central sleep apnea of infancy, immature regulation of respiration should be suspected, and a trial of caffeine may be considered.
Support (If Any): None.

0823

ACDF - A HIDDEN ETIOLOGY OF OSA

Naila Manahil[1], Waiz Wasey[1], Asiya Mohammed[1]
Southern Illinois University School of medicine [1]

Introduction: OSA is characterized by recurrent episodes of upper airway collapse and obstruction during sleep, leading to oxygen desaturations. Risk factors include obesity, age, sex, family history, craniofacial abnormalities, stroke, diabetes, polycystic ovarian syndrome. This case illustrates symptomatic and clinically proven worsening of OSA after anterior cervical discectomy and fusion (ACDF), which can lead to narrowing of the airspace and dysfunction of the pharyngeal plexus leading to upper airway collapse.
Report of Cases: A 49-year-old female with cervical myelopathy was evaluated in the sleep clinic for snoring, witnessed apnea, and daytime somnolence. Her Epworth score was 11, and Mallampati score was 4, suggestive of underlying sleep-disordered breathing (SDB). A home diagnostic sleep test revealed mild OSA with AHI of 6.8/hr and was started on an Auto Continuous Positive Airway Pressure (APAP) device. She then reported worsening of her cervical pain leading to decreased use of APAP and total sleep time and underwent ACDF approximately 6 months after her diagnosis of OSA was made. Post-surgery she reported mild dysphagia, frequent nighttime awakening due to a choking sensation, and an increase in snoring. Following hospital discharge, the patient was re-evaluated in the sleep clinic for these complaints. Upon review, her BMI had decreased to 33.99 kg/m2, from 37.35 kg/m2, and the only medication change was the addition of hydrocodone-acetaminophen 5-325, 1-2 times daily as needed for pain. A repeat diagnostic home sleep study showed progression of OSA to moderate severity with an AHI of 24.5/hr, with no evidence of central sleep apnea or hypoventilation. Therefore, it was unlikely that the worsening of AHI was due to opioids. Thus, it was concluded that ACDF most likely led to the worsening of OSA.
Conclusion: Although there is no strong association in medical or sleep literature, this case demonstrates a strong association between ACDF and OSA. This highlights the importance of a timely diagnosis of new-onset or reassessment for worsening OSA in patients post-ACDF to improve sleep quality and prevent morbidity.
Support (If Any):

0824

CAN THE INTERFACE UTILIZED IN CONTINUOUS POSITIVE AIRWAY PRESSURE THERAPY SIGNIFICANTLY ALTER THE APNEA-HYPOPNEA INDEX IN CERTAIN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA?

Fauzieh Dabaja[1], Virginia Skiba[2], William Palmer[3]
Sleep Disorders and Research Center at Henry Ford Hospital [1] Sleep Disorders and Research Center at Henry Ford [2] Pulmonary and Sleep Medicine, Ascension Borgess [3]

Introduction: Multiple studies and analyses have demonstrated that use of an oronasal interface for delivery of continuous positive airway pressure (CPAP) therapy can lead not only to higher pressure requirements, but also a higher residual apnea hypopnea Index (AHI) in patients with obstructive sleep apnea (OSA).
Report of Cases: We record a 75-year-old man with severe OSA, AHI of 38, and treated with CPAP 16 cm water. On routine follow up, the residual AHI was 2.9, but he reported mouth-venting with nasal mask and stretched out the chinstrap. He was transitioned to an oronasal interface, and on subsequent follow-up his AHI was 19.6. The decision was made to change back to a nasal mask with a better-fitting chinstrap, which reduced his AHI to 4. To verify that the AHI recorded by the CPAP device was physiologic, the patient later used CPAP while wearing a home sleep apnea test (HSAT) utilizing peripheral arterial tonometry for 2 nights. On the night he used a nasal mask with a chinstrap, the AHI recorded on the CPAP was 1.4 and 3.9 on the HSAT. The following night, he wore the oronasal interface and the recorded AHI on CPAP was 31.6 and 18.5 on the HSAT.
Conclusion: Although oronasal masks are commonly used as an interface for CPAP, there is significant evidence that nasal delivery systems are more effective at controlling AHI in some individuals. Many studies show that oronasal masks not only correspond to an increase in AHI but also are associated with higher pressure requirements and a lower adherence compared to nasal interfaces. Imaging has shown greater retropalatal airway expansion when using a nasal interface compared to an oronasal device. Oronasal devices may also push the tongue posteriorly and cause increased occlusion of the airway. The consistent finding of worsening OSA control in the setting of oronasal masks has led many to consider CPAP with a nasal interface to be the gold standard for treating OSA. The clinician should therefore be mindful of these considerations when initiating or altering a CPAP interface.
Support (If Any):

0825

A CASE OF FAMILIAL RESTLESS LEGS SYNDROME AND RAYNAUD'S PHENOMENON

Ninad Maniar[1], Arthur S. Walters[2], Elias G. Karroum[1]
Department of Neurology & Rehabilitation Medicine, The George Washington University School of Medicine and Health Sciences, Washington, DC, USA [1] Department of Neurology, Vanderbilt University Medical Center, Nashville, Tennessee, USA [2]

Introduction: Restless legs syndrome (RLS) is a common sensorymotor disorder that frequently leads to sleep disturbances and reduced quality of life. Raynaud's phenomenon (RP) is characterized by episodes of reduced blood flow mainly to the fingers due to vasospasms, with subsequent pain and discoloration, usually triggered by cold exposure or by stress. We present an interesting case

report of co-occurrence and familial transmission of RLS and RP which has not been reported previously.
Report of Cases: A 64-year-old woman presented for evaluation of long-standing RLS. She had a history of RP, osteoarthritis (OA), and recurrent late pregnancy losses. The patient, and one brother and a sister had RLS and RP. She had another sister and two maternal first cousins with RLS. Her mother suffered from neither and her father and his brother both had RP. She has a daughter who also has RLS. The patient's mother was treated with Diethylstilbestrol (DES) and had five pregnancy losses. The patient also had 3 miscarriages and the patient's daughter miscarried her first pregnancy as well. The patient first experienced RLS symptoms at the age of 12 and has been treated with several medications since then, most recently clonazepam. Since she was taken off clonazepam, she experienced a worsening of RLS and was subsequently started on Pregabalin up to 300 mg at bedtime but without significant relief. She was then started on pramipexole, titrated up to 0.375 mg which resulted in significant improvement of RLS symptoms. Her RP symptoms were infrequent, occurring every few months and not bothersome. She did not report rheumatological complaints other than chronic OA from lifelong sports.
Conclusion: This case report suggests a possible shared genetic abnormality in the transmission of both RLS and RP in this family. This genetic association may be related to a vascular dysfunction common to both disorders. The co-occurrence of frequent miscarriages (although could be related to DES), in association with the presence of RLS and RP, may also suggest an underlying autoimmune disorder. Further genetic research is needed to confirm above findings with the potential uncovering of new therapeutic targets for RLS.
Support (If Any): None.

0826

A CASE OF PROPRIOSPINAL MYOCLONUS IN A PATIENT WITH MULTIPLE SCLEROSIS

Rebeca Maynard[1]
University of Michigan [1]

Introduction: Described first in 1991, by Brown et al, Propriospinal Myoclonus is a rare movement disorder of rhythmic, typically flexor, jerking motions most pronounced when lying supine. Although most cases are thought to be idiopathic, identified causes have been infection, spinal lesions, and psychogenic.
Report of Cases: A 38-year-old female with medical history of multiple sclerosis, anxiety and PCOS was referred for jerking body movements with sleep onset. Symptoms of nightly, full body jerking, beginning in her back, were described as "legs being pulled up towards the chest" and present only while lying supine in bed with symptom onset of 1 month. Difficulty initiating sleep and daytime sleepiness ensued as a result of these jerks. Work-up with in-lab polysomnogram failed to demonstrate obstructive sleep apnea (AHI 2.8/hr), however, did demonstrate periodic limb movements of sleep. MRI of the brain, cervical spine, and thoracic spine were negative for new or enhancing brain or spinal cord lesions. CBC, CMP, TSH and ceruloplasmin were ordered for a complete myoclonus evaluation and found to be within normal limits. She was started on Klonopin 0.5 mg at night. Three month follow-up revealed improvement of symptoms, with the frequency of her myoclonus decreased to 1-2 episodes per week with reported less intensity of each event.
Conclusion: While proper work-ups are always important, this case reminds us of the diligence required when certain conditions exist. Although our patient had a negative myoclonus work-up, and is thought to have idiopathic myoclonus, the presence of multiple sclerosis made the investigation increasingly relevant. Work-up should include an MRI to rule-out spinal cord lesions.
Support (If Any): None

0827

OBSTRUCTIVE SLEEP APNEA IN A CONGENITAL CENTRAL HYPOVENTILATION SYNDROME PATIENT POST-TRACHEOSTOMY VENTILATED WITH DIAPHRAGMATIC PACING

Victoria Cocozza[1], *David Kim*[2], *Shana Hansen*[2], *William Frey*[2]
Department of Pediatrics, San Antonio Uniformed Services Health Education Consortium [1] Department of Sleep Medicine, San Antonio Military Medical Center [2]

Introduction: Congenital central hypoventilation syndrome (CCHS) is a rare, autosomal dominant disorder associated with a genetic mutation in the PHOX2B gene. While the treatment of CCHS requires lifelong ventilatory support, advancements in management have allowed for life expectancies comparable to healthy individuals. Prolonged lifespan of CCHS patients presents new challenges with regards to the chronic management of the disease, including the concurrence of obstructive sleep apnea (OSA) in patients ventilated with diaphragmatic pacing post decannulation.
Report of Cases: A 23-year-old female with a past medical history of adenotonsillectomy and CCHS with nocturnal ventilatory support via diaphragmatic pacer (DP) was referred to sleep clinic by her pulmonologist for an evaluation of increased obstructive events and worsening nocturnal hypoxia. The patient did not require daytime ventilatory support and did not complain of any sleep-related symptoms. The DP was implanted at age 17 and the patient previously had a tracheostomy from age 3 months until decannulation at 18 years. She had a surgical closure a year later. The DP settings were titrated by her pulmonologist and monitored with repeat home sleep apnea testing (HSAT) to achieve optimal control of central hypoventilation. After an initial period of response, however, subsequent HSATs showed a progressive increase in obstructive breathing events associated with hypoxia. Further adjustments in the DP settings did not successfully correct the findings. An in laboratory polysomnography (PSG) confirmed moderate OSA with significant hypercapnia. At clinic follow-up, the patient was offered positive airway pressure therapy but chose to defer decision-making until pulmonary follow-up. The patient was also referred to ENT for an anatomic evaluation to look for potential causes contributing to upper airway obstruction.
Conclusion: DP remains a treatment option for select patients with CCHS. Limited studies have shown that OSA can occur in patients with CCHS using DP as their primary management modality. Our case demonstrates the importance of keeping a broad differential in evaluating the development of concurrent OSA in these patients. Potential contributors to developing OSA include weight gain, tracheomalacia or tracheal stenosis resulting from longstanding tracheostomy status, and effects of increased DP amplitude settings.
Support (If Any):