839

NOT YOUR EVERYDAY DAYTIME SLEEPINESS: TWO PEAS IN A POD

Elena Stuewe,[1] Peter Ostrow,[1] Aarti Grover,[1] Greg Schumaker,[1] Joel Oster,[1] Rajesh Zacharias[1]
[1]Tufts Medical Center

Introduction: Obstructive sleep apnea (OSA) and narcolepsy are both causes of excessive daytime sleepiness (EDS). OSA is a more prevalent diagnosis, but it can coexist with narcolepsy and confound diagnosis. We present a case of a delayed diagnosis of type 2 narcolepsy in a patient with known OSA.
Report of case(s): A 31-year-old man with depression treated with sertraline and prior history of severe OSA diagnosed at an outside facility presented to our clinic for residual excessive daytime sleepiness. He demonstrated adequate adherence to continuous positive airway pressure (CPAP) of 13 cmH2O over a period of one year, good sleep hygiene and adequate sleep duration. He reported vivid dreams and sleep paralysis in the past, but none recently. There was no history of a delayed sleep phase. He denied hypnagogic or hypnopompic hallucinations or cataplexy. An in-lab polysomnogram (PSG) followed by multiple sleep latency test (MSLT) was ordered for further evaluation. Sertraline was held 2 weeks prior to the study. Overnight PSG on CPAP showed adequate treatment of OSA on CPAP pressures of 13–16 cmH2O. MSLT showed 3/5 sleep-onset rapid eye movement periods with a mean sleep latency of 5.8 minutes. A diagnosis of coexisting type 2 narcolepsy was made. Treatment was initiated with modafinil; however, his symptoms of EDS persisted and he was changed to methylphenidate with subsequent improvement.
Conclusion: The case above highlights the importance of maintaining a broad differential when investigating the etiology of EDS. In particular, patients with narcolepsy often experience a significant delay between onset of symptoms and receiving a diagnosis. Diagnosis can be confounded by a lack of classic symptoms and/or the presence of another sleep-related breathing disorder, as in the patient above. Residual EDS can be seen in patients with adequately treated OSA. There is sparse data regarding the co-prevalence of narcolepsy as the etiology of residual EDS in adequately treated OSA. Patients should still be screened for symptoms suggestive of narcolepsy. Persistence of EDS symptoms in young adults with adequately treated OSA should raise suspicion for another sleep-related disorder and merits further investigation.
Support (if any):

840

OBSTRUCTIVE TO CENTRAL: A SWITCH IN SLEEP APNEA TYPE AFTER ARTERIOVENOUS MALFORMATION RUPTURE

Meredith Greer,[1] Lynn Marie Trotti,[2] Nancy Collop[1]
[1]Emory University, [2]Emory University Hospital

Introduction: Sleep apnea and stroke have long been shown to be linked, with sleep apnea increasing the risk for stroke and stroke leading to sleep apnea. When the latter occurs, it can present as central sleep apnea (CSA), often in the form of Cheyne-Stokes breathing (CSB), and has been shown to resolve over time. We present a patient with persistent CSA after severe hemorrhagic stroke secondary to rupture of a temporal/thalamic arteriovenous malformation (AVM).
Report of case(s): A 33-year-old man with a history of obstructive sleep apnea (OSA) presented to our clinic for re-evaluation of his disease. He was diagnosed with OSA in 2006 at which time he was 270 pounds with a body mass index (BMI) of 36.7, thus the OSA was thought to be secondary to obesity. When he presented to our clinic 10 years later, he had lost approximately 80 pounds after suffering multiple strokes. In 2014, he had a left temporal lobe hemorrhage due to rupture of a left temporal/thalamic AVM and required decompressive hemicraniectomy. In 2015, he had a re-bleed of this AVM, with new hemorrhage extending inferiorly into the left cerebral peduncle and pons, and superiorly into the left parietal periventricular white matter anteriorly along the optic tract. Ultimately, he was treated with stereotactic radiotherapy to the AVM nidus with no residual AVM. However, he has chronic encephalomalacia of the left basal ganglia, thalamus, temporal, parietal, and occipital lobes with extension into the left cerebral peduncle and changes consistent with radiation necrosis. His residual symptoms are aphasia and right-sided hemiplegia and although his snoring resolved with weight loss, his mother noticed pauses in his breathing overnight. A repeat sleep study done in 2016 showed 27 central apneas and 0 obstructive apneas with an AHI of 5.4 events/hour. He was subsequently studied on ASV with residual AHI of 0.4 events/hour.
Conclusion: Although patients with OSA may be at higher risk for stroke, it is important to re-evaluate their sleep apnea after such an event to ensure appropriate diagnosis and treatment going forward.
Support (if any):

841

PAP THERAPY DATA PROVIDES CLUES TO COMORBID SLEEP DISORDERS

Bethany Bartley,[1] Alice Cai,[1] Lawrence Epstein[2]
[1]MassGeneral Brigham, [2]Brigham and Women's Hospital

Introduction: Obstructive sleep apnea (OSA) is a common condition and positive airway pressure (PAP) therapy is the treatment of choice. Treatment guidelines recommend monitoring objective PAP usage data to track treatment efficacy. A typical report includes the percentage of days/month and cumulative hours/day a PAP device was used. In addition, PAP therapy timing can be graphically viewed with use plotted by time/day. This latter presentation reveals patterns of usage that can identify comorbid sleep disorders.
Report of case(s): Case 1: A 65-year-old male with OSA presented with sleep inertia despite compliance with PAP therapy. Therapy timing data revealed a delayed circadian phase, with bedtime of 4 am and wake time of 1 pm. Circadian phase advancement therapy was added. Case 2: An 86-year-old male with OSA presented with daytime sleepiness after years of excellent PAP usage. Two years earlier, he was diagnosed with Parkinson's disease and developed increasing afternoon sleepiness. Compliance data showed a mild reduction in PAP usage. However, therapy timing data revealed an irregular sleep-wake rhythm disorder with sleep scattered around the clock, consistent with neurodegenerative disease. Case 3: A 70 year-old-female with PTSD and OSA presented with persistent tiredness and difficulty initiating and maintaining sleep despite good compliance with PAP therapy. Her timing data showed irregular sleep times with long gaps in usage suggestive of sleep maintenance insomnia. After starting a behavioral treatment regimen her pattern regularized and awakenings reduced, seen by consistent PAP use. Case 4: An 80-year-old female with OSA complained of early morning awakenings despite PAP use with good compliance. Review of PAP therapy timing revealed a pattern suggestive of advanced sleep-wake phase disorder. Circadian phase delay therapy was started.
Conclusion: Patients with OSA on PAP therapy may have comorbid sleep disorders impacting sleep quality. PAP therapy data can provide information on sleep-wake behavior similar to actigraphy to help diagnose these conditions and track treatment response.

Support (if any): None

842

PAP THERAPY IN A PANDEMIC: MANAGEMENT OF SEVERE MIXED APNEA PREDOMINANT OSA & CSA DURING THE COVID-19 PANDEMIC

Ugorji Okorie,[1] Rupa Koothirezhi,[2] Pratibha Anne,[1] Oleg Chernyshev,[1] Cesar Liendo,[2] Brittany Monceaux[1]
[1]LSU Health Shreveport, [2]Ochsner-LSU

Introduction: Introduction/Background: A new protocol and standard of care was created amidst the COVID-19 Pandemic that began in 2020. Traditional split night studies fell out of favor and were replaced by solely diagnostic studies with placement on Auto-PAP therapy if treatment of sleep disordered breathing was required. Some patients, however, required a more tailored approach if diagnostic polysomnogram (PSG) was particularly concerning. Our case report describes the treatment of a patient with severe Mixed Apnea Predominant Obstructive Sleep Apnea (OSA) with accompanying Central Sleep Apnea (CSA) using COVID-19 Precautions.
Report of case(s): Case Description: A 48 year old AAM patient with a PMH of HTN, pre-diabetes, GERD, obesity and tobacco abuse initially presented to Sleep Medicine in late January 2020 with complaints of snoring, witnessed apneas, waking up gasping, excessive daytime sleepiness, fatigue, and non-restorative sleep for many years with ESS 24 and FSS 48 on initial evaluation. Diagnostic PSG showed AHI 76.9 with O2 desaturation to 59% and demonstrated the presence of severe Mixed Apnea predominant OSA and CSA with worsening during REM sleep. Because of the severity, he underwent a PAP titration in August 2020 using the AASM COVID-19 sleep study precautions which included use of a negative pressure room. Optimal control of snoring, apneic respiratory events and oxygen desaturations was achieved at 14 cm H2O in the supine body position during REM sleep. Follow up with Sleep Medicine in October and December 2020 showed objective compliance over a 30 day period not completely at goal due to issues with mask desensitization and sleep hygiene, however the patient subjectively reported that he noticed great improvement in snoring, excessive daytime sleepiness and fatigue.
Conclusion: Discussion/**Conclusion:** With a diagnosis of Severe Mixed Apnea Predominant OSA as well as CSA noted during the study, the differential diagnosis included CHF, Chiari malformation, opioid abuse and idiopathic CSA as the cause. Despite a dangerous pandemic, appropriate therapy for certain patients must still be attained. Special protocols developed during the COVID-19 Pandemic allowed for our patient to receive adequate treatment, while ensuring the safety of all involved.
Support (if any): References COVID 19: FAQs for Sleep Clinicians. AASM official website. https://aasm.org/covid-19-resources/covid-19-faq/

843

PARADOXICAL WAKING HYPOXEMIA THAT IMPROVES WITH SLEEP

Jessica Cho,[1] David Dai,[1] Constance Fung[2]
[1]University of California - Los Angeles, [2]VA Greater Los Angeles Healthcare System

Introduction: We present a case of paradoxically worsened hypoxia during wake phase of polysomnography while undergoing a CPAP titration study. Nighttime hypoxemia is a common feature in obstructive sleep apnea, due to obstructive events that manifest while sleeping. Excluding OSA, there remains an extensive differential for disease processes that cause hypoxemia while asleep; however, none of these processes can explain waking hypoxemia that improves upon sleeping.
Report of case(s): A 70 year old male with severe OSA diagnosed by home sleep test (REI 46.5, nadir O2=76%) underwent polysomnography with PAP titration and demonstrated several hours of interrupted sleep without hypoxia and minimal obstructive events on CPAP 9–13 cmH2O. During the study, while awake at CPAP of 14 cmH2O, he developed hypoxia to mid-high 80s and supplemental oxygen bleed in was added starting at 3L and increased to 5L during a prolonged period of wakefulness. On CPAP 15 cmmH2O with 5L bleed-in, the patient fell asleep and oxygen saturation again increased to low 90s. He underwent an extensive workup for other cardiopulmonary causes of hypoxemia, with pulmonary function testing showing moderate obstructive ventilatory defect and mild DLCO impairment. An echocardiogram with saline contrast bubble study was relatively unremarkable, without evidence of right to left shunting. He underwent a chest CTA which was negative for pulmonary embolism, though it did reveal an enlarged pulmonary artery consistent with pulmonary hypertension. His chronic hypoxemia was treated with 2L supplemental oxygen during the day and bleed-in with CPAP at night.
Conclusion: Though nocturnal hypoxemia is common with OSA, polysomnography with paradoxical hypoxemia during wake phase has not been reported. Notably, the patient was without prolonged hypoxia during his sleep phase while on CPAP treatment with minimal apneic/hypopneic events. Pulmonary hypertension can also present as nocturnal hypoxemia, but it should worsen with sleep, rather than improve. There are case reports of right to left shunting worsened by PAP, though his hypoxemia persisted despite PAP. His paradoxical worsening hypoxemia with wakefulness is still unexplained.
Support (if any):

844

PEDIATRIC VAGUS NERVE STIMULATOR-INDUCED OBSTRUCTIVE SLEEP APNEA

Nouraddin Nouraddin,[1] Louella Amos[2]
[1]Medical College of Wisconsin Affiliated Hospitals, [2]Medical College of Wisconsin

Introduction: Vagus nerve stimulation (VNS) is an adjunct treatment for seizures refractory to medications. VNS in children with epilepsy can reduce seizures by up to 90%. VNS settings include stimulation on-time, off-time, frequency and output current. Complications of VNS include sleep-disordered breathing due to laryngopharyngeal dysfunction, which can also cause voice alteration, hoarseness, and cough. Both obstructive apneas (more common) and central apneas can be seen in those patients who have VNS-induced sleep-disordered breathing.
Report of case(s): A 14-year-old male with Lennox-Gastaut syndrome treated with multiple antiepileptic drugs and VNS was admitted to the PICU with worsening seizures. He developed acute respiratory failure due to status epilepticus, requiring intubation. After extubation, he was observed to have repetitive respiratory obstruction at regular intervals, occurring throughout the day and night, and associated with mild oxygen desaturations. Polysomnography showed cyclical obstructive respiratory events lasting 30 seconds followed by approximately 2-minute intervals of regular breathing. Interrogation of his VNS device revealed the following settings: output current of 1.75 mA, 30 seconds on, and 1.8 minutes off. CPAP therapy improved his oxygen saturations, but he continued to clinically exhibit the repetitive obstructive apneas even on positive pressure. However, after his VNS device settings were decreased, repeat polysomnography showed resolution of his obstructive breathing.