0858

UNDER REPORTING OF APNEA HYPOPNEA INDEX (AHI) IN PATIENTS WITH PREDOMINANT REM SLEEP DISORDERED BREATHING ON ANTIDEPRESSANTS.

PRATAP REDDY[1], DARON KAHN[2]
TOWER HEALTH SLEEP MEDICINE FELLOWSHIP PROGRAM AT READING HOSPITAL [1] TOWER HEALTH SLEEP MEDICINE FELLOWSHIP PROGRAM [2]

Introduction: The prevalence of Rapid Eye Movement (REM) related Sleep Disordered Breathing (SDB) ranges from 13.5% to 36.7% in patients suspicious to have SDB.1 It is reported that patients with REM related SDB often have associated depression and are commonly on anti-depressants.2 Antidepressants suppress overall REM sleep duration. Significant sleep disordered breathing is known to occur during REM sleep. These patients commonly present with excessive daytime sleepiness and fatigue. Obstructive sleep apnea (OSA) diagnosis is often missed in these patients secondary to REM sleep suppression and subsequent underreporting of AHI.1. Conwell W, Patel B etal 2012 PMID: 21614575. 2. Geckil AA, Ermis H. 2020 PMID: 30949927.
Report of Cases: Case 1 is a 53-year-old obese white female with stable depression on citalopram 20mg presented with witnessed loud snoring and apneas, sleep onset and maintenance insomnia, daytime fatigue and hypersomnolence. Her Epworth Sleep Score (ESS) was 13/24. Her in lab baseline polysomnogram demonstrated overall AHI 3.8/hour and REM only AHI 24.3/hour. To further explore her persistent daytime sleepiness a Mean Sleep Latency Test (MSLT) was ordered. Citalopram was held 2 weeks prior to MSLT. In the PSG her overall AHI increased to 7.6/hour diagnosing her with mild OSA. PAP therapy was initiated that improved patients nocturnal sleep, daytime fatigue and hypersomnolence. Case 2 is a 54-year-old overweight white female with past medical history of chronic stable depression on Wellbutrin SR 100 mg daily presented with significant day time fatigue and hypersomnolence (ESS 15/24). Her baseline PSG reported AHI 1.7/hour with REM only AHI 10.7/hour. However due to persistent excessive daytime sleepiness MSLT was ordered. Her Wellbutrin was held 2 weeks prior to testing. Her overnight PSG demonstrated increase in her AHI to 7.9/hour and REM AHI to 34.2/hour. Mild obstructive sleep apnea was diagnosed, and PAP therapy was initiated that improved her hypersomnolence.
Conclusion: These cases illustrate that antidepressants likely mask obstructive sleep apnea by suppressing REM sleep. Given the underreported AHI in patients with REM SDB who are on antidepressants it may be prudent to hold anti-depressants for 2 weeks to establish an accurate diagnosis of sleep apnea.
Support (If Any):

0859

AVAPS TO THE RESCUE: A CASE OF SEVERE OBSTRUCTIVE SLEEP APNEA WITH TREATMENT RESISTANT TO UPPP AND CPAP IN A PEDIATRIC PATIENT WITH PRADER-WILLI SYNDROME

Nauras Hwig[1], Victor Peng[2], Montserrat Diaz-Abad[3], Anayansi Lasso-Pirot[1]
University of Maryland Medical Center [1] UMMC [2] University of Maryland School of Medicine [3]

Introduction: Prader-Willi syndrome (PWS) has a prevalence of 1/10,000 to 1/30000 and is the most common syndromic form of obesity. Prader-Willi syndrome is defined by a multitude of features that develop from early childhood to adolescents, but one of the primary developments is obesity in the setting of hyperphagia during early childhood. Due to the development of obesity, there is a high prevalence of obstructive sleep apnea (OSA) among PWS patients.
Report of Cases: A 6-year-old boy with PWS with a 2-year history of loud snoring underwent polysomnography (PSG) which showed severe OSA with Apnea-Hypopnea Index (AHI, events/h) 133.7. The patient underwent surgical intervention with a combined uvulopalatopharyngoplasty and adenotonsillectomy and a follow up PSG showed a residual AHI of 37. On CPAP titration the AHI improved to 1.2 with CPAP 14 cm H2O and the patient was discharged on home nasal CPAP. Over the next several years, the patient had suboptimal CPAP compliance. At 11 years of age, the patient was admitted to the intensive care unit (ICU) with volume overload –with a normal cardiac workup- and respiratory failure requiring high flow nasal cannula oxygen. Venous blood gas (VBG) showed pH 7.34 and severe hypercapnia with pCO2 73 mmHg c/w obesity hypoventilation syndrome (OHS). The patient was started on CPAP then changed to bilevel PAP ST with supplemental oxygen. Due to persistent hypercapnia in the high 60s noninvasive ventilation (NIV) was switched to Average Volume-assured Pressure Support with auto titrating EPAP (AVAPS-AE) with significant improvement in hypercapnia and overall tolerance. Discharge VBG showed pH 7.35 and PCO2 56 mm Hg. The patient was discharged home, is doing well and has avoided readmission for at least 6 months.
Conclusion: This case highlights the potential for newer modes of noninvasive ventilation with autotitrating EPAP to treat severe OSA with OHS and hypercapnic respiratory failure in patients with PWS and other conditions. With the growing obesity epidemic, rates of treatment failures with CPAP and traditional bilevel PAP therapy may increase and consideration of alternative modes of NIV therapy should be considered.
Support (If Any):

0860

BURST SUPPRESSION DUE TO DIFFUSE ENCEPHALOPATHY

Shilpa Pandey[1], Rohini Coorg[2], Sherrill Mohan[3], Kevin Kaplan[3]
Baylor College of Medicine [1] Pediatric Neurology, Texas Children's Hospital [2] Pediatric Sleep Medicine, Texas Children's Hospital [3]

Introduction: Burst suppression is a finding on electroencephalography (EEG) associated with a severe encephalopathy associated with coma, severe infantile-onset epilepsy syndromes, hypothermia, or may be medically induced by general anesthesia. It presents as a pattern of alternating high-voltage, 75-250µV, activity separated by periods of amplitude dampening, less than 5µV, of electrical brain activity1. The duration of the burst's activity is 1-20 seconds while the suppression lasts longer than 10 seconds1. When not medically induced, burst suppression is a known marker of poor prognosis.
Report of Cases: An 18-year-old male with cerebral palsy, spastic quadriplegia, and static encephalopathy secondary to hypoxemia ischemic injury in the perinatal period presents with excessive sleepiness during therapy sessions. He was empirically placed on non-invasive ventilatory support with BPAP ST 8/4 cm H2O with a rate of 10 breaths per minute for chronic respiratory failure during sleep. While awake he shows no evidence of hypoxemia or hypercapnia on room air. A polysomnogram was ordered showing moderate obstructive sleep apnea (oAHI 9.73) and central sleep apnea (5.84 events per hour) without hypoxemia (SpO2 nadir 90%), or hypercapnia (TcCO2 max 48 mmHg). The study was scored as REM/NREM as specific sleep architecture was not identified. Diffuse burst suppression was observed. No epileptiform abnormalities were recorded. A MRI of the brain shows diffuse