O2 at 3-4 L/min. Per the parents, the patient has been maintaining her oxygen saturation in the absence of BIPAP therapy with oxygen use. Due to COVID, patient was unable to follow up but will be scheduled for a repeat PSG in the near future. She followed with Neurosurgery for Arnold Chiari II and they recommended no surgical intervention at this time due to functional VP shunt.
Conclusion: This is an atypical presentation of Biot's breathing in the absence of CNS infections and opioid use in a patient with Arnold Chiari malformation II. Patient has complex sleep apnea, initially well controlled with BiPAP ST, but developed BiPAP intolerance. She is on oxygen with good control of hypoxemia in the absence of BiPAP therapy.
Support (If Any):

0837

AVERAGE VOLUME-ASSURED PRESSURE SUPPORT (AVAPS) AFTER CPAP FAILURE IN A PEDIATRIC PATIENT WITH SEVERE OBSTRUCTIVE SLEEP APNEA AND SLEEP-RELATED HYPOVENTILATION

Xinhang Tu[1], Victor Peng[2], Anayansi Lasso-Pirot[3], Montserrat Diaz-Abad[3]
Medstar Health Baltimore Residency Program, Department of Medicine [1] University of Maryland Medical Center [2] University of Maryland School of Medicine [3]

Introduction: Obstructive sleep apnea (OSA) has become an increasingly pervasive sleep disorder in the pediatric population. Current mainstream treatments include adenotonsillectomy and positive airway pressure therapy. Average volume-assured pressure support (AVAPS) is a relatively new mode of non-invasive ventilation, which has been increasingly used in the treatment of respiratory failure and hypoventilation syndromes. Here we present a case of a pediatric patient with severe OSA and sleep-related hypoventilation who was successfully treated with AVAPS after failure of CPAP therapy.
Report of Cases: A four year old boy with history of severe OSA, severe obesity, asthma, and allergic rhinitis underwent polysomnography one year after adenotonsillectomy and nasal turbinate reduction due to continued symptoms of sleep-disordered breathing. Results showed elevated residual apnea-hypopnea index (AHI = 30.4 events/hour), sleep-related hypoventilation (T ETCO2≥50 = 228.3 minutes), and sleep-related hypoxemia (T≤90% = 7 minutes). Therefore the patient underwent repeated adenotonsillectomy and turbinate reduction, with post-operative course complicated by pulmonary edema requiring intubation. He was extubated and weaned to nocturnal CPAP. Following discharge, CPAP titration failed to control AHI at maximal pressure (AHI 54.5 on 20 cm H2O, T≤90% = 15.3 minutes). The patient was then started on AVAPS with auto-titrating EPAP (AVAPS-AE, settings Pmax 20 cm H2O, PS 2-10 cm H2O, EPAP 5-10 cm H2O, RR auto, room air) with subsequent improvement of snoring and witnessed apneas, as well as reduction of daytime sleepiness. Afterwards, AVAPS-AE titration confirmed resolution of obstructive sleep apnea, sleep-related hypoxemia, and sleep-related hypoventilation (AHI = 2.5, T≤90% = 1.2 minutes, T ETCO2 ≥50 = 6.5 minutes.) The patient has since remained stable on AVAPS-AE until age ten, with the most recent AVAPS titration demonstrating continued resolution of sleep-disordered breathing.
Conclusion: AVAPS was an effective treatment for a pediatric patient with severe OSA and sleep-related hypoventilation who had failed CPAP therapy.
Support (If Any): None.

0838

CHEYNE-STOKES BREATHING IN A PEDIATRIC PATIENT WITH DILATED CARDIOMYOPATHY AND MUSCULAR DYSTROPHY PRIOR TO HEART TRANSPLANT

LeQuan Dang[1], Kevin Kaplan[2], Amee Revana[3]
Baylor College of Medicine [1] Baylor College of Medicine, Texas Chidlren's Hospital [2] Baylor College of Medicine, Texas Children's Hospital [3]

Introduction: Cheyne-Stokes breathing (CSB) has rarely been identified in the pediatric population. Neuromuscular diseases (NMD) such as Duchene Muscular Dystrophy (DMD) can predispose patients to sleep-disordered breathing including central sleep apnea (CSA) and CSB. Sleep-disordered breathing in children with NMD may not have symptoms; thus, treatment can be delayed. Currently, there is limited data to support resolution of CSB in DMD with dilated cardiomyopathy post-transplant.
Report of Cases: We present a 15-year old female with a significant history of both dilated cardiomyopathy and DMD who presented with acute on chronic heart failure. Due to her disease progression, she was listed for heart transplant. Prior to her transplant, she completed an inpatient polysomnography (PSG) to rule out sleep-disordered breathing due to concerns of snoring and dyspnea during sleep. Her Pediatric Daytime Sleepiness Scale score (PDSS) was 8. The polysomnogram recorded moderate obstructive sleep apnea (OSA) and central sleep apnea (CSA) consistent with Cheyne-Stokes breathing along with rare premature ventricular contractions (PVCs). Patient was started on BPAP of 13/8 cm H2O with a back-up rate of 12 breaths per minute after titration study. The patient subsequently received a heart transplant in which the patient's dyspnea and snoring resolved. Post-transplant PSG pending to reassess the severity of sleep-disordered breathing.
Conclusion: Though CSA can be seen in children, CSB is rarely seen in children with either heart failure or muscular dystrophy. When CSB is observed, the cornerstone of treatment is correcting the underlying cause. This patient demonstrated CSB with symptoms that improved with BPAP and now post-heart transplant. When both heart failure and neuromuscular disease are involved, close monitoring for clinical symptoms along with screening for CSB is important and may affect overall quality of life and recovery.
Support (If Any):

0839

COMPLEX SLEEP APNEA IMPROVED WITH DECOMPRESSION OF A CHIARI I MALFORMATION IN A PEDIATRIC PATIENT

Matthew McKee[1], Lacie Petitto[1], Kevin Kaplan[1]
Pediatric Sleep Medicine, Texas Children's Hospital [1]

Introduction: Chiari malformation (CM) occurs when a portion of the cerebellum herniates through the foramen magnum. CM is categorized as two types. Type 1 involves the cerebellar tonsils and type 2 involves the cerebellum and brain stem. Those with CM can be asymptomatic to having debilitating neurologic symptoms such as dysphagia, tinnitus, emesis, balance difficulty, muscle weakness, and/or headache. Central sleep apnea (CSA) and obstructive sleep apnea (OSA) have been associated with CM. It is postulated that

the sleep disordered breathing (SDB) is due to compression of the medulla which houses the breathing center and cranial nerves that play a role in nocturnal breathing. OSA has been thought to be related to muscle weakness of the lower airway in those with CM. After resolving CM with surgical decompression, residual OSA is common requiring treatment with positive airway pressure therapy.
Report of Cases: A 3-year-old male with a history of OSA and tonsillar hypertrophy. Polysomnography (PSG) was performed after tonsillectomy and adenoidectomy indicating severe OSA (oAHI 17.7/hr) and CSA (CSAI 29.9/hr). A titration study was conducted and bilevel positive airway pressure spontaneous/timed (BPAP ST) 10/6 cmH2O with a backup rate of 12 breaths per minute was utilized further resolving his SDB and daytime sleepiness. After months of BPAP ST therapy, he presented to an urgent care with symptoms of somnolence, emesis, and nystagmus of the left eye. Magnetic resonance imaging of the brain revealed a 2 cm herniation of the cerebellar tonsils indicating CM type 1. Neurosurgery performed surgical decompression without complication. Postoperative PSG indicated significant improvement of OSA (oAHI of 3.45/hr) and resolution of CSA (0.9/hr). BPAP ST therapy was discontinued with the resolution of his daytime sleepiness and SDB.
Conclusion: This case demonstrates a patient with severe OSA and CSA due to an undiagnosed CM type 1. OSA and CSA are associated with CM; however, residual OSA typically exists status post decompression and can require positive airway pressure therapy for treatment. Our case demonstrates the need for consideration of CM in patients with complex sleep apnea and that surgical decompression can improve both OSA and CSA in these patients.
Support (If Any):

0840

BE WARY OF ADJUSTMENT DISORDER WITH ANXIETY IN PATIENT WITH INSOMNIA AND DISCOMFORT WITH POSITIVE PRESSURE AIRWAY THERAPY

Saad Bin Jamil[1], Talar Kachechian[1], Karl Doghramji[1], Zhanna Fast[1]
Jefferson Sleep Disorders Center, Thomas Jefferson University [1]

Introduction: Insomnia is a common complaint. When it occurs in the context of treatment with continuous positive airway pressure (CPAP), it can complicate treatment and lead to dissatisfaction with CPAP therapy. Adjustment disorder with anxiety (ADWA) is a condition that develops following an identifiable stressor and can be associated with insomnia. We describe a case of ADWA causing disrupted sleep and dissatisfaction with CPAP therapy.
Report of Cases: The patient is an 80-year-old man with history of severe obstructive sleep apnea (OSA) and chronic obstructive lung disease, under stable management with CPAP. During a routine follow up visit, he reported the recent onset of frequent, and often prolonged, nocturnal awakenings associated with daytime sleepiness and fatigue. Insomnia was associated with discomfort with breathing, especially during exhalation, while using CPAP. His machine report indicated a significant decrement in compliance. Upon questioning, he reported that a few of his family members had died unexpectedly, following which he began to experience significant anxiety. His wife noted that he had become moody and irritable. On examination, the patient was visibly anxious; his speech was accelerated and animated, and he displayed psychomotor activation. Affect also displayed despondency. The patient was diagnosed with ADWA leading to insomnia and discomfort with CPAP use. He was prescribed buspirone 10 mg twice daily. On two week follow up patient reported no improvement in symptoms and was switched to escitalopram 10 mg daily. Following approximately three weeks, his clinical evaluation revealed significant improvement in mood; anxiety had dissipated, and sleep was more continuous with infrequent and brief awakenings. Daytime alertness had been restored. Of interest was the temporary return of anxiety and insomnia symptoms following the brief discontinuation of escitalopram because of hospitalization.
Conclusion: ADWA can occur in the setting of recent stressors and can result in sudden onset of insomnia, compromise CPAP adherence and decrease subjective benefit from PAP therapy. This case highlights the importance of questioning patients for the possibility of recent stressors and traumatic events when they develop difficulties with PAP use. These may indicate the presence of ADWA, whose management can restore CPAP compliance and improve sleep quality and daytime functioning.
Support (If Any):

0841

CASE REPORT: CAN LSD BE ASSOCIATED WITH CHRONIC EXPLODING HEAD SYNDROME?

Feby Puravath Manikat[1], Clete Kushida[2]
Sleep Medicine, Stanford University Medical Center [1] Stanford University Medical Center [2]

Introduction: Exploding head syndrome is a rare phenomenon characterized by a loud imagined noise at sleep onset. Less commonly, it is associated with a simultaneous stab of pain in the forehead. It may result in recurring arousals and anxiety regarding the events. The underlying pathophysiology remains unknown.
Report of Cases: A 27 year old male with no past medical history presented to the sleep medicine clinic with a 2-year history of sleep-onset pain between his eyes, like "a flash of lightning," associated with moderate to severe pain. He experiences "brain zaps" or "a jolt of electricity between his eyes every night." He would yell and jolt out of bed due to this sensation. These episodes occur approximately 60 to 90 minutes after sleep onset almost every night, jolting him from sleep and lasting approximately 1 minute. He also has difficulty seeing in the dark and also eye twitching. He experiences palpitations, diaphoresis, and a sense of fear often with these episodes. He denies any neurological deficits or muscle jerks. He is not on any daily medications. He does not regularly use any sleep aids but has tried melatonin 3 mg in the past. Evaluation by ophthalmology has ruled out ophthalmologic and neuro ophthalmic causes. He does report use of illicit substances including lysergic acid diethylamide (LSD), marijuana, methylenedioxymethamphetamine (MDMA), and cocaine. He did not experience any unpleasant effects after his first use of LSD, however within minutes of his second ingestion, he felt a sharp pain on his nasal bridge and forehead 10/10 in intensity. He states that since then, this pain has recurred almost every night. He does not have a family history of any neurologic disorders or migraines. The current plan for workup includes an in lab polysomnography with seizure montage and, if negative, a trial of a tricyclic antidepressant is planned.
Conclusion: This case illustrates the potential issues after LSD use causing severe dopaminergic and serotonin surge with drug use and the value of a complete history. Awareness of a possible correlation is clinically useful and may serve as a caution in the use of recreational drugs.
Support (If Any):