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

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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?

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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):

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