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EXPLODING HEAD SYNDROME AND HYPERSOMNIA

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Introduction: Exploding head syndrome (EHS) is a hypnagogic parasomnia where the patient experiences a loud noise or flash of light and often reports distress secondary to the stimulus. EHS is relatively uncommon, and to our knowledge, there has been only one published report of comorbid EHS and narcolepsy, describing a 38-year-old male. This report focuses on a pediatric case of EHS, evaluating possible comorbid narcolepsy and sleep apnea.
Report of Cases: Eight-year-old male was referred for excessive daytime sleepiness. He endorsed trouble staying awake in school, both in the morning and after lunch time. His Epworth in clinic was a 16, despite nightly sleep duration ≥8 hours. He also endorsed weakness secondary to strong emotions and was observed to have difficulty grasping and holding a pencil while tickled. Patient and his caregiver denied snoring, witnessed apnea, dream enactment, and symptoms of restless leg syndrome. However, the patient did admit to auditory hallucinations, occurring about twice per week during the transition from wakefulness to sleep. He described the hallucination as a banging sound and the sound of clinging bracelets, noting he has searched for the source of the stimulus, but has never been able to find a cause. Patient's mother denied hearing similar noises. No visual hallucinations or sleep paralysis noted. Polysomnography revealed 0 Central Sleep Apneas, 0 Mixed Apneas, 10 Obstructive Apneas, and 12 hypopneas. The mean duration of these events was 0 seconds. The patient's Apnea-Hypopnea Index (AHI) was 2.5 and had a REM AHI of 8.3. Patient was supine 75% of the night.
Conclusion: This case illustrates a unique hypnagogic parasomnia in a pediatric patient. At this point, the patient's symptoms would suggest EHS characterized by auditory hallucinations that involve hearing the banging sound, upon transition from sleep to wake. The patient also exhibits some narcolepsy type symptoms - hypersomnia, Epworth of 16, and subtle symptoms of cataplexy. In lab PSG – showed overall AHI of 2.5, REM AHI of 8.3-oxygen nadir 90%. Patient referred to ENT for tonsillectomy and adenoidectomy. With follow up NPSG and MSLT if still sleepy. He was referred to behavioral sleep medicine clinic to address anxiety secondary to EHS, but did not attend.
Support (If Any):

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LONGITUDINAL MANAGEMENT OF NARCOLEPSY WITH ATYPICAL PRESENTATION OF CATAPLEXY (UNILATERAL WITH SYMPTOMS ONLY ON THE RIGHT SIDE FOR THE PAST 13 YEARS)

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Introduction: Narcolepsy with atypical unilateral cataplexy is a rare phenotype. We would like to share single center, single patient experience over the last 13 years of managing this patient.
Report of Cases: 64-year-old female diagnosed with narcolepsy with unilateral cataplexy in 2008 with MSLT. Her cataplexy syndrome has been atypical, in that it has a strictly unilateral presentation for the past 13 years, with right face, arm, and leg weakness with strong emotions(anger/laughter). Her right-side extremities go limp for a few seconds to a minute. Her cataplexy syndrome has never evolved to become bilateral or unilaterally on her left side. Her physical examination has been largely unremarkable. For Narcolepsy, was on Modafinil (2008) which was up titrated to 600 mg with no side effects, eventually switched to Armodafinil 250 mg (2013). Eventually did not control EDS fully, methylphenidate (2014) was added. The patient reported cognitive impairment with gradual memory loss on stimulants (starting in 2012) and nocturnal insomnia on methylphenidate SR. Due to worsening cognition and memory, stimulants were discontinued in 2014 and patient referred to neurology and had neuropsychiatric evaluation for memory loss. Dementia lab workup and MRI with spectroscopy were unremarkable. Due to continuation of EDS was restarted on Modafinil until May 2021. In May 2021, she reported no longer fully controlled daytime sleepiness and Pitolisant was added with improvement in ESS from 17/24 to 9/24. For the management of unilateral cataplexy, patient was well controlled initially on venlafaxine (2008), eventually up titrated with an additional dose a few times. After cognitive impairment, patient stopped refilling it in 2014 and subsequently slept better without the venlafaxine. She reported that she would fall when she got very upset/mad so learnt not to get mad at people. She had many episodes of unilateral cataplexy mainly with laughing or excitement so was started on Fluoxetine 20 mg and eventually up titrated to 40 mg. Her unilateral cataplexy symptoms have been well controlled with the addition of Pitolisant.
Conclusion: Management of Narcolepsy with atypical unilateral cataplexy is challenging and requires use of multiple medications with different sites of action.
Support (If Any):

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NARCOLEPSY TYPE 1 IN A PEDIATRIC PATIENT WITH TEMPORAL LOBE EPILEPSY

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Introduction: Narcolepsy type-1 is clinically characterized by irrepressible daytime sleepiness and REM-sleep dissociation, including cataplexy. Cataplexy usually manifests as episodes of brief, symmetrical sudden loss of muscle tone with retained consciousness. It may be difficult to distinguish from seizure activity as they may share overlapping features. We discuss a pediatric patient with temporal lobe epilepsy with co-occuring Narcolepsy Type 1.
Report of Cases: A 17-year-old boy with obesity and a history of focal epilepsy secondary to intracranial hemorrhage in the neonatal period status-post right parieto-occipital resection at age 11, presented with worsening fall episodes and feeling of imbalance Following surgery, his seizures improved, but he was having excessive sleepiness. At age 13, he started experiencing episodes of slurred speech, feeling of imbalance, "body heaviness and tongue heaviness" with occasional falls. He denied loss of consciousness, body shaking, stiffening, or incontinence. At age 17, he presented to medical attention due to increasing frequency of such episodes. Physical exam was significant for BMI 42.7 kg/m , Mallampati 4, tonsils 3+ bilaterally, and homonymous hemianopia. Epworth Sleepiness Scale: 22/24. Video EEG was negative for epileptiform discharges