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SLEEP-RELATED HICCUPS: A CASE REPORT OF ANTIDEPRESSANT ASSOCIATED HYPNIC JERKS

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Introduction: A hiccup is a sudden activation of inspiratory muscles, followed by paradoxical glottic closure, producing a familiar sound [1]. Antidepressants are commonly prescribed and hypnic jerks and hiccups have been reported as a rare adverse drug reaction [2]. Few reports have described pronounced hypnic hiccups with subsequent audio/video polysomnogram confirmation.
Report of Cases: A 44-year old man with a history of depression, anxiety, and alcohol use disorder presented to clinic with a long history of violent hypnic jerks associated with a loud hiccup. These episodes started 30 minutes before he fell asleep and continued throughout the night every 30 minutes with forceful hiccups that were so loud they wake others in his house. We believe that these episodes were likely induced by venlafaxine since they were noted initially 8 years ago when he started taking venlafaxine and there was complete remission after discontinuation of the medication. He was later prescribed escitalopram 20 mg daily, restarted on venlafaxine XR 150 mg daily, and later sertraline 100 mg daily due to refractory depression and anxiety recurrence. Episodes progressively became more frequent and pronounced that he avoided sleep and they impacted his ability to hold a relationship. Gabapentin and clonazepam did not previously improve symptoms. He took trazodone 50 mg every other night to aid falling asleep and reported to have less frequent hypnic jerks. Upon presentation to our clinic, subsequent PSG demonstrated severe obstructive sleep apnea (AHI 63, HI 52, CI 11) with significant sleep fragmentation (sleep efficiency 46%) with very frequent sudden axial myoclonic contractions with head and neck movements along with vocalizations likely representing hiccups. Video and audio demonstrated the loud hiccups described. These events occurred while the patient was resting awake and transitioning to sleep and was less frequent but persisted in N2. The patient was prescribed CPAP for sleep apnea and recommended to follow up with psychiatry to consider other medications.
Conclusion: Pronounced hypnic jerks and sleep-related hiccups can significantly impact total sleep time, sleep quality, and quality of life. Clinicians should be aware of these potential side effects in patients on antidepressants.
Support (If Any): 1. Askenasy JJ. About the mechanism of hiccup. Eur Neurol. 1992;32:159-63.2. Bagheri H, Cismondo S, Montastruc JL. Hoquet d'origine médicamenteuse: enquête à partir de la Banque Nationale de Pharmacovigilance [Drug-induced hiccup: a review of the France pharmacologic vigilance database]. Therapie. 1999 Jan-Feb;54(1):35-9. French. PMID: 10216420.

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WORSENING CENTRAL SLEEP APNEA: A SOLE DIAGNOSTIC MARKER OF A BRAIN TUMOR IN A CHILD.

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Introduction: Central sleep apnea (CSA) in children is a relatively uncommon and under-studied sleep disorder. A small subset of patients may have intracranial anomalies, but clinical presentation varies. We present a case of malignant intracranial tumor diagnosed primarily due to the unusual presentation and progression of CSA.
Report of Cases: The presented patient is a 12-year-old developmentally appropriate female with a history of obstructive sleep apnea (OSA) status post adenotonsillectomy at two years of age who presented with snoring without witnessed apnea, sleep interruptions, and unrefreshing sleep. Other sleep-related history was unremarkable. No daytime sleepiness or behavioral or learning concerns were noted. Physical examination was unremarkable with normal growth parameters. Initial diagnostic PSG demonstrated severe OSA with an obstructive apnea-hypopnea index (OAHI) of 16/h and mild CSA (CAI:6.7/h); therefore, PAP therapy was pursued after ruling out adenoid regrowth. However, during the first PAP titration study(incomplete study due to mask intolerance), treatment-emergent central apneas were noted (OAHI: 9.8/h; CAI:25.8/h), which were noted even during the second titration study (OAHI:2.7/h; CAI:59.2/h). Even though ordered earlier, the brain MRI was not performed until after the repeat PSG one year later showed persistence of severe CSA (OAHI of 4.5/h and CAHI of 39.5/h) after much persuasion. Interestingly, snoring had reportedly improved with no daytime symptoms or neurologic complaints. The brain MRI demonstrated compressing brainstem lesion highly suspicious for glioma. The patient was emergently sent to neurosurgical care and had chosen hospice care after a few weeks.
Conclusion: CSA can be an early or the only finding in patients with brainstem tumors, even before neurologic signs and symptoms. The PSG findings changed from mild CSA to treatment-emergent CSA to severe CSA, possibly with the progression of underlying disease. Close follow-up and ensuring patient compliance are essential in CSA patients. CSA severity should prompt MRI brain even with an intact neurological examination. More research is needed to fully understand the link between cerebral disease and polysomnographic data. This could aid in early diagnosis and treatment.
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LATE ONSET NARCOLEPSY WITH CATAPLEXY

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Introduction: NT1 is characterized by EDS, cataplexy, sleep related paralysis, and hallucinations. Cataplexy is defined as weakness precipitated by emotions, more commonly with positive emotions and is associated with narcolepsy.
Report of Cases: 70 yo with history of OSA on APAP referred with year history of worsening EDS, dream enactment, and cataplexy. Initially CPAP titration followed by MSLT was performed. Unfortunately sleep logs/actigraphy were not performed. CPAP was titrated 7-10 cm demonstrating suboptimal control (AHI 30) and presence of RSWA. MSLT demonstrated MSL of 2.9 minutes with 5 SOREMPS, however was confounded by THC use, suboptimal OSA control, and Trazodone. Considering above, patient was started on 11-16cm, and reevaluation was recommended. Patient was seen by neurology and started on Fluoxetine 20mg with some relief in symptoms (4-5episodes/day to 1-2/day). Imaging was unremarkable. Reevaluation was planned. He stopped Fluoxetine and THC 3 weeks prior. Actigraphy demonstrated significant movement. PSG demonstrated SOREMP with optimal OSA control (15-17cm), but there was recurrent evidence of vocalization with RSWA. MSLT was significant for MSL of 2.5 minutes, and 4 SOREMPs. Patient was restarted on Fluoxetine 40mg and melatonin 15mg for RBD. With optimal CPAP pressure daytime symptoms improved, however cataplexy worsened (3-4/day) triggered by laughter. Fluoxetine dose was increased with some relief