861

VASCULAR DEMENTIA PRESENTING AS RAPIDLY PROGRESSIVE DEMENTIA SECONDARY TO ZOLPIDEM

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Introduction: Rapidly progressive dementia is a condition with a wide differential which remains difficult to accurately diagnose. The potential pathologies responsible include thyroid, vitamin, and electrolytes abnormalities, infectious, and malignant causes. Vascular dementia, however, typically has a slow and insidious presentation. Zolpidem (Ambien) is among the top 50 prescribed medications in the US.
Report of case(s): An 84-year-old Caucasian male with a past medical history of insomnia, and sleep apnea who is noncompliant with CPAP presented after a fall associated with altered mental status. He has taken zolpidem 10 mg nightly for over six years. The patient and wife reported notable personality changes beginning six months prior, as well as four months of progressively worsening auditory and visual hallucinations. Additionally, the patient noted developing urinary incontinence, and worsening gait steadiness with recurrent falls. The patient then developed sleep-wake inversion during the three weeks prior to his fall, and an outpatient referral to neurology was subsequently sent for dementia evaluation. On the night prior to his presentation, the patient took his usual nighttime zolpidem at 22:00 and later fell and was unable to get up. Subsequent testing was negative for reversible causes of dementia and MRI Brain revealed only chronic microvascular disease. His zolpidem dose was decreased to 5 mg and scheduled earlier which resulted in the resolution of his hallucinations, gait abnormalities, and acute encephalopathy.
Conclusion: One month later, the patient presented to the hospital after a repeat fall secondary to taking his zolpidem at his previously scheduled time. Once more, his dosage was further decreased to 2.5 mg and scheduling earlier, resulting again, in the complete resolution of his symptoms. Zolpidem, has an increased potential for delirium in elderly patients and especially those with dementia. Chronic use of zolpidem with insidiously progressive vascular dementia led to a worsening delirium which resolved after adjustment of timing and reduction of zolpidem dosing.
Support (if any):

862

VOLUME-ASSURED PRESSURE SUPPORT IMPROVES OUTCOMES IN A PATIENT WITH CONGENITAL CENTRAL HYPOVENTILATION SYNDROME

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Introduction: Congenital Central Hypoventilation Syndrome (CCHS) is a condition caused by a mutation of the PHOX2B gene and an incidence of 1 in 50,000 live births. Clinically the condition is characterized by autonomic nervous system dysfunction, the most prominent feature of which is the failure of respiratory homeostasis during sleep. In patients severely affected, life-long ventilatory support is required. This might start as early as the newborn period. Subsequent adjustments are required due to their growth and development. The role of Volume-Assured Pressure Support (VAPS) ventilation in treatment of CCHS was only described in a couple reports before.
Report of case(s): A 17-year-old female patient born at term and diagnosed with CCHS at birth at our center with a PHOX2B mutation confirmed. Her daytime ventilatory support was weaned at age 18 months and the tracheostomy was removed at age 10 years old. She relocated to another state, was lost to follow-up, and returned this year for adjustments of her ventilator whose settings had not been adjusted for several years. She was on a Trilogy 100 ventilator, in pressure-controlled mode with settings of EPAP 5, IPAP 20, and a rate of 22/min without supplemental oxygen. Her measured weight is 69kg, body mass index (BMI) 27. The patient complained of difficulty breathing while on those settings and reported decreased desire to use the machine. The patient was empirically switched to VAPS due to titration availability limitations during the COVID-19 pandemic. Initial AVAPS settings were: EPAP 4–7, PS 3–12, breath rate 16/min, TV 350mL. Upon implementing the changes, the patient reported improved comfort and increased usage. Average minute ventilation decreased from 10.5 to 5.8 L/min, patient triggered breaths increased from 1.1% to 12.3%, average breaths per minute decreased from 22.0/min to 16.2/min, the average peak flow 31.8 to 26.2L/min, tidal volume decreased from 463 to 355mL.
Conclusion: AVAPS ventilation can be successfully used in managing patients with CCHS, and it might be superior to pressure-controlled mode in certain cases, improving patient comfort and compliance.
Support (if any): Department of Internal Medicine, University of New Mexico School of Medicine, Albuquerque, NM.

863

WHEN SLEEP TAKES YOU TO UNWANTED PLACES?

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Introduction: Somnambulism is a parasomnia occurring in non-rapid eye movement sleep, and is characterized by ambulation as a disorder of arousal regulation. Sleep deprivation, alcohol abuse, fragmented sleep and certain medications can increase the risk of sleep walking.
Report of case(s): Here we present a 35-year-old man with multiple triggers for sleep walking, resulting in recurrent parasomnia events over fifteen years. He had a history of bipolar disorder, post-traumatic stress disorder (PTSD), chronic insomnia, moderate untreated obstructive sleep apnea (OSA), anxiety with violent daytime behaviors, and prior alcohol abuse status post six years of sobriety. He was sent to our clinic due to increased frequency and severity of events, with nightly events for the last five years. Episodes were characterized by walking around the home, leaving the home, and driving on occasion. He reported at least one minor car accident as a result of sleep driving. He also reported an injury resulting from a fall in his home while sleep walking. Several security measures were implemented, including door gates, door alarms, and hiding car keys. His family slept in different bedrooms with locked doors for safety. The patient's chronic insomnia improved with cognitive behavioral therapy, leading to an average sleep time of five hours per night with no reported hypersomnia or daytime fatigue. After his initial evaluation, he was referred for a mandibular advancement device for treatment of his OSA, due to prior poor compliance with positive airway pressure therapy related to his PTSD. Optimizing his OSA helped decrease arousals that might trigger sleep walking events. He also maintained close follow up with mental health for pharmacotherapy and psychological therapy. Treatment with clonazepam 0.25 mg at bedtime was initiated given the severity of his somnambulism.
Conclusion: The use of a benzodiazepine can reduce slow wave sleep duration by its effect on the inhibitory neurotransmitter gamma aminobutyric acid (GABA). Our patient had multiple risk factors for parasomnias, with severe, frequent episodes of sleepwalking leading to self-injury. His treatment involved both pharmacotherapy as well as optimization of underlying triggers.
Support (if any):