limb movement disorders. While periodic limb movements (PLMs) can occur in pediatric patients, they are rare compared to their adult counterparts. Literature is limited regarding the presence of PLMs post-concussion in the pediatric population. We describe an unusual case of PLMs in the setting of mild TBI in a pediatric patient.

**Report of Cases:** A 6-year-old male with a past medical history significant for adeno-tonsillar hypertrophy and chronic cough was brought to the emergency department by ambulance after being found facedown secondary to a fall at school. Physical examination findings were significant for dried blood at the nares with an abrasion to the anterior nasal bridge. No other signs of trauma were noted, and his Glasgow Coma Scale (GCS) was 15. Computerized Tomography (CT) scan of the head was negative for any acute intracranial abnormality. He was diagnosed with a mild TBI and sent home with concussion precautions. Prior to his concussion, at the age of 4, he was diagnosed with obstructive sleep apnea (OSA) via polysomnography (PSG). Moderate OSA was noted with an apnea hypopnea index (AHI) of 8.1 per hour leading to adenotonsillectomy. Five weeks after his concussion, an evaluation by the pediatrician revealed complaints of restless sleep and worsened emotional lability prompting a referral to the sleep clinic. By comparison, the patient's post-TBI PSG at age 6 showed no evidence of sleep-disordered breathing (AHI of 1.48 per hour) but with new periodic limb movements and an elevated PLM index of 6.56 per hour. An iron panel is pending.

**Conclusion:** PLMs in the setting of pediatric TBI is a rarely diagnosed entity and, to our knowledge, has never been reported in the literature. Our case suggests that clinicians should have a high level of suspicion for sleep problems post-TBI and consider PSG to assess for PLMs which can affect recovery and the overall quality-of-life of the pediatric patient.

**Support (If Any):** Viola-Saltzman M, Watson NF. Traumatic brain injury and sleep disorders. Neurol Clin. 2012;30(4):1299-1312. doi:10.1016/j.ncl.2012.08.008

## 0813

### POST-PINEALECTOMY INSOMNIA AND MELATONIN THERAPY

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**Introduction:** Melatonin is a hormone produced in the pineal gland that has an important role in sleep; immune, neurologic, psychiatric, metabolic, and endocrinologic function; cardiac-autonomic regulation and even cancer risk. We present a case of insomnia, somnambulism, dream enactment, and periodic limb movements of sleep (PLMS) after a pinealectomy.

**Report of Cases:** A 40-year-old woman with a history of a complete pinealectomy due to a pineal cyst presented to the sleep medicine clinic. Shortly after the pinealectomy, she developed sleep onset and maintenance insomnia. Two years later she developed somnambulism, and four years later she developed dream enactment and PLMS. She reported no prior treatments for her sleep issues, including no history of melatonin use. On average, her total sleep time (TST) was 2-8 hours/night with awakenings every 2 hours. Sleep latency was 10-45 minutes. Polysomnograghy demonstrated an apnea-hypopnea index of 0.6/hr, PLM index of 68.1/hr, normal REM atonia, and no complex behaviors. The patient started 1mg immediate release (IR) melatonin, which did not help her insomnia, but parasomnias resolved. She had improvement in her PLMS with iron supplementation and melatonin. The melatonin dose was increased to 3mg IR which helped increase her TST to 4-8 hours. She was switched to 3mg extended release (ER) melatonin, and then increased to 4mg ER. She obtained the most benefit for her insomnia with 1mg IR plus 4mg ER with sleep latency reduced to 5-10 minutes and TST improved up to 7.5 hours with rare awakenings.

**Conclusion:** Pinealectomy in humans is rarely reported. Most data about the consequences of pinealectomy and pathophysiology of melatonin come from animal research. Melatonin level after pinealectomy is often undetectable or severely diminished. Current limited literature on patients with pinealectomy consists of case reports about patients who experienced insomnia, non-24-hour sleep-wake rhythm disorder [SSM1] and mood disorders. Melatonin doses ranging from 0.5mg to 14mg IR and up to 5mg ER have been trialed with most patients having symptomatic improvement with doses above 3mg. We found that a combination of 1mg immediate and 4mg extended release melatonin was the most beneficial for our patient.

**Support (If Any):**

## 0814

### THREE SLEEPY SIBLINGS

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**Introduction:** Narcolepsy type 1 is caused by destruction of hypocretin-producing neurons, likely via a T-cell mediated autoimmune process, and clinically identified either by CSF hypocretin deficiency or the presence of cataplexy. Individuals with narcolepsy type 1 have an underlying genetic predisposition attributed to the HLA DQB1*0602 gene. This genetic variant has been linked to increased propensity for sleepiness even in healthy adults. Relatives of patients with narcolepsy type 1 appear to be at increased risk for other disorders of hypersomnolence such as idiopathic hypersomnia. Here we describe three siblings, all positive for HLA DQB1*0602, who presented with distinct clinical features diagnostic for narcolepsy type 1, narcolepsy type 2, and idiopathic hypersomnia.

**Report of Cases:** A 15-year-old female was diagnosed with narcolepsy type 1 based on typical cataplexy, excessive daytime sleepiness, and mean sleep latency of 2 min with 2 SOREMPs on MSLT. Lumbar puncture (LP) performed 5 years after symptom onset showed low CSF hypocretin (9.3 pg/mL). Her older brother presented at age 21 with excessive sleepiness, somewhat atypical cataplexy, hypnagogic hallucinations, and sleep paralysis. MSLT showed mean sleep latency of 6 min with 5 SOREMPs. Despite the presence of cataplexy, LP performed 2 years after symptom onset showed normal CSF hypocretin (296.5 pg/mL) and he was diagnosed with narcolepsy type 2. Their younger sister presented at age 19 with progressive daytime sleepiness. PSG/MSLT showed mild OSA (RDI 9.2) and mean sleep latency of 6.5 min without SOREMPs. She does not have cataplexy, hypnagogic hallucinations, or sleep paralysis. The current findings are most consistent with idiopathic hypersomnia, although an LP to evaluate for hypocretin deficiency is an important next step. Similarly, a repeat LP in the brother might demonstrate change in hypocretin over time.

**Conclusion:** These cases may support a familial link between narcolepsy type 1, type 2, and idiopathic hypersomnia. The discordant hypocretin and cataplexy statuses of these siblings implies a mechanism for excessive sleepiness beyond hypocretin deficiency, possibly mediated by HLA DQB1*0602. Identifying the mechanisms of familial aggregation of sleepiness in the central disorders of hypersomnolence may shed light on the pathophysiology of these distinct disorders.

**Support (If Any):**