0639

POLYSOMNOGRAPHIC BIOMARKERS OF SLEEP DISRUPTION AND SLEEP DISORDERED BREATHING IN MIGRAINE: A LARGE MATCHED CASE CONTROL CLINICAL REGISTRY-BASED STUDY

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Introduction: Sleep disruption and sleep architectural changes in relation to migraine are not well characterized. Disruption in sleep may serve as a risk for migraine or result from migraine. We leveraged a large clinical cohort to examine the hypothesis that those with migraine have greater degrees of sleep architectural alterations and sleep disordered breathing (SDB).
Methods: This was a polysomnogram-based retrospective case (migraine) control (non-migraine) study of patients aged >18 matched 1:3 on age, sex, race, body mass index (BMI), and year of polysomnogram. Two domains were considered: 1)Sleep architecture (arousal index:AI, (primary predictor), total sleep time (TST), percentage of sleep stage time) and 2)SDB (apnea hypopnea index (AHI:primary predictor), mean oxygen saturation) were considered. Comparisons were performed by two-sample t-test or Wilcoxon rank sum test for continuous variables, and chi-square test or Fisher's exact test for categorical variables.
Results: 4,783 migraine cases (47.5 ±13.3 years, 76.4% Caucasian, body mass index:BMI 33.7 ±8.6kg/m2) were matched to 14,287 controls. In migraine patients vs those without, TST was lower (359.0[307.0, 421.0] minutes vs 363.0[306.0, 432.5] minutes,p=0.01), percentage of N2 was higher (67.8%[59.6, 75.6] vs 67.0%[58.4, 74.8],p<0.001), percentage of REM was lower (16.7% [10.0, 22.0] vs 17.0% [11.1, 22.2],p=0.012), AHI was lower (7.4 [2.6, 17.0] vs 9.5 [3.7, 22.1],p<0.001), AI was lower (19.6 [12.8, 30.9] vs 22.6 [14.7, 34.9],p<0.001), and mean oxygen saturation was higher (93.7%±2.4 vs 93.3±2.6,p<0.001).
Conclusion: In this largest study of its kind, we identify novel associations of migraine in relation to curtailed sleep and sleep architectural alterations, i.e. increase in N2 and reduced REM sleep and lower AI compared to contemporaneously matched controls. Directionality of these relationships requires further elucidation given the cross-sectional nature of this study. Interestingly, we observed lower degree of sleep apnea and hypoxia burden in patients with migraine. As Calcitonin Gene-Related Peptide (CGRP), a neuropeptide increased during and between migraine attacks in migraine patients, and serotonin are implicated in arousals due to apnea-related increases in CO2, there is biologic plausibility for migraines patients to exhibit potential protection from SDB. Further investigation is needed to confirm these findings.
Support (If Any):

0640

NORTH AMERICAN PRODROMAL SYNUCLEINOPATHY CONSORTIUM: BASELINE CHARACTERISTICS IN 251 PATIENTS WITH REM SLEEP BEHAVIOR DISORDER

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Introduction: Introduction: Rapid Eye Movement (REM) Sleep Behavior Disorder (RBD) is characterized by a lack of muscle atonia during REM sleep with dream enactment. RBD is regarded as a prodromal synucleinopathy as a high proportion of patients eventually phenoconvert to Parkinson's Disease and related synucleinopathies, suggesting RBD may be an early non-motor symptom of disease. Accordingly, patients with RBD are ideally situated to test potential therapeutic interventions to prevent phenoconversion to synucleinopathy. However, RBD itself, and associated patient registries, are rare. The North American Prodromal Synucleinopathy Consortium (NAPS) establishes a multisite registry of RBD patients with standardized neurological, neuropsychiatric, and neuropsychological assessments and biomarker collection. The present work reports baseline characteristics of this RBD patient database at its current state.
Methods: Methods: Participants >18 years of age with overnight polysomnogram-confirmed RBD by ICSD-3 criteria who did not meet criteria for the diagnosis of PD, dementia, MSA, or narcolepsy were enrolled from 10 sites across North America (8/2018 to 4/2021). Data collection included family and personal history of RBD and related symptoms, as well as standardized assessments related to cognitive, motor, sensory and autonomic function. Additionally, all subjects have contributed blood, and a subset of subjects have contributed cerebrospinal fluid samples to the National Centralized Repository for Alzheimer's Disease and Related Dementias for future analysis.
Results: Results: A total of n=251 participants were enrolled. Outcomes are reported based on sex (n=202 male, n=49 female). Data were further examined based on participants' history of antidepressant use (n=142 with, n=103 without) and based on participants' extent of synucleinopathy burden (n=70 defined as isolated RBD, n=181 defined as RBD+ [i.e., exhibiting ≥1 abnormality]). Any observed sex differences among the data did not persist after correction for antidepressant use.
Conclusion: Conclusions: This prospective, cross-sectional data on history, demographic, cognitive, motor, sensory, and autonomic function in n=251 participants with RBD highlight the lack of sex differences and the high preponderance of concomitant neurological abnormalities with RBD, and provide a valuable registry for future longitudinal studies and neuroprotective clinical trials.
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