Abstracts

Wake-up stroke is not associated with sleep-disordered breathing in women

**Background** We sought to investigate the frequency of wake-up stroke (WUS) and its association with sleep-disordered breathing (SDB) in women.

**Methods** Within a population-based study, women with acute ischemic stroke were asked about their stroke symptom onset time. SDB screening was performed with the well-validated ApneaLink Plus device; SDB was defined by a respiratory event index ≥10. Logistic regression was used to test the association between SDB presence and severity and WUS unadjusted and adjusted for potential confounders including prestroke depression and sleep duration.

**Results** Among 466 participants, the median age was 67.0 years (interquartile range [IQR] 58.0–77.0), 55% were Mexican American, and the median initial NIH Stroke Scale score was 3.0 (IQR 1.0–6.0). Stroke symptom onset occurred during nocturnal sleep (25.3%), during a nap (3.9%), during wakefulness (65.9%), or unknown (4.9%). In those with SDB screening performed (n = 259), a median of 11 days (IQR 5–17) poststroke, WUS was not associated with the presence or severity (respiratory event index) of SDB in unadjusted or adjusted analysis.

**Conclusions** In this population-based study, WUS represented about 30% of all generally mild severity ischemic strokes in women and was not associated with SDB.

Molecular genetic testing for hereditary ataxia: What every neurologist should know

**Purpose of review** Because of extensive clinical overlap among many forms of hereditary ataxia, molecular genetic testing is often required to establish a diagnosis. Interrogation of multiple genes has become a popular diagnostic approach as the cost of sequence analysis has decreased and the number of genes associated with overlapping phenotypes has increased. We describe the benefits and limitations of molecular genetic tests commonly used to determine the etiology of hereditary ataxia.

**Recent findings** There are more than 300 hereditary disorders associated with ataxia. The most common causes of hereditary ataxia are expansion of nucleotide repeats within 7 genes: ATXN1, ATXN2, ATXN3, ATXN7, ATXN8, CACNA1A (spinocerebellar ataxia type 6), and FXN (Friedreich ataxia). Recent reports describing the use of clinical exome sequencing to identify causes of hereditary ataxia may lead neurologists to start their clinical investigation with a less sensitive molecular test providing a misleading "negative" result.

**Summary** The majority of individuals with hereditary ataxias have nucleotide repeat expansions, pathogenic variants that are not detectable with clinical exome sequencing. Multigene panels that include specific assays to determine nucleotide repeat lengths should be considered first in individuals with hereditary ataxia.

Practice Current

Our survey on the topic, “How do you treat neuromyelitis optica?” received over 500 responses from over 60 countries. Explore this topic and others on our newly redesigned website: compare your practice with peers and see survey results displayed on an interactive world map.

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