

BASELINE SLEEP QUALITY PREDICTS DIFFERENTIAL RESPONSE TO MIGRAINE PROPHYLAXIS: AN INTERACTION ANALYSIS ON MOOD OUTCOMES

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Background. Preventive migraine treatments may differentially affect psychiatric comorbidities, such as anxiety and depression. However, comparative data across oral preventive therapies, botulinum toxin type A (BoNT-A), and anti-calcitonin gene-related peptide (CGRP) monoclonal antibodies (mAbs) are lacking. We hypothesized that baseline sleep quality may modify the relative effectiveness of these therapeutic classes on mood outcomes, particularly in patients with comorbid insomnia.

Methods. We prospectively evaluated a cohort of 148 patients initiating preventive migraine treatment at baseline and after 6 months of therapy. Patients were grouped according to the preventive treatment received: antidepressants (amitriptyline, n=15), antiseizure medications (topiramate, n=16), beta-blockers (propranolol, n=14), calcium channel blockers (flunarizine, n=12), BoNT-A (n=21), and anti-CGRP monoclonal antibodies (erenumab, n=21; fremanezumab, n=22; galcanezumab, n=27).Changes in anxiety and depressive symptoms were assessed using the Hospital Anxiety and Depression Scale (HADS) and its anxiety (HADS-A) and depression (HADS-D) subscales, as well as the Beck Depression Inventory-II (BDI-II). Multivariable linear regression models were used to examine the interaction between treatment class and baseline Pittsburgh Sleep Quality Index (PSQI) score, adjusting for baseline mood scores, headache frequency, sex, and age. Analyses were further stratified according to a clinically relevant PSQI cutoff (≥10 vs. <10).

Results. After 6 months, a significant interaction between oral preventive therapies and baseline PSQI was observed for BDI-II scores ($\beta = -0.93$, $p = 0.029$), with a trend toward significance for HADS-D ($\beta = -0.37$, $p = 0.092$). For each one-point increase in baseline PSQI score, oral preventive therapies were associated with an additional 0.93-point reduction in BDI-II score compared with anti-CGRP mAbs. Among patients with insomnia (PSQI ≥10), oral preventive therapies produced greater improvements in depressive symptoms than anti-CGRP mAbs (ΔBDI-II: −10.7 vs. −6.4; $p = 0.05$). Conversely, among patients without insomnia, anti-CGRP mAbs yielded greater improvements (ΔBDI-II: −4.7 vs. +1.9 with oral preventive therapies; $p = 0.006$; ΔHADS-D: −2.0 vs. +2.1; $p = 0.001$).

Conclusions. Baseline sleep quality significantly modifies the comparative effectiveness of preventive migraine treatments on mood outcomes. In patients with good sleep quality, anti-CGRP monoclonal antibodies were the most effective treatment for improving all mood-related outcomes.