GLP-1s do not improve migraines, but reduce healthcare utilisation

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> Migraine management remains a clinical challenge, with many patients experiencing insufficient control despite current preventive therapies. While anti-CGRP monoclonal antibodies demonstrate efficacy in reducing migraine days, a significant proportion of patients do not achieve optimal control. New data indicates that GLP-1 receptor agonist initiation does not improve migraine frequency, yet it is associated with a reduction in migraine-related healthcare utilisation.12

KEY TAKEAWAYS

- The Pivot: GLP-1 receptor agonists, primarily used for diabetes and weight management, do not directly improve migraine frequency.
- The Data: Approximately 30% of individuals with high migraine burden achieve optimal disease control or migraine freedom with anti-CGRP monoclonal antibodies.1,2,3
- The Action: Clinicians should continue to prioritise migraine-specific preventive treatments for migraine improvement, while noting potential indirect benefits of GLP-1s on healthcare utilisation.

The International Headache Society has established treatment goals for migraine prevention, aiming for higher standards of care in real-world settings. These goals include migraine freedom (no monthly migraine days (MMD)), optimal control (< 4 MMD), modest control (4-6 MMD), and insufficient control (>6 MMD).1,2,3

Real-world efficacy of anti-CGRP monoclonal antibodies

A prospective, real-world, European multicentre study, the EUREkA cohort, assessed the proportion of individuals achieving these treatment goals after 6 months of migraine-specific treatment with anti-CGRP monoclonal antibodies (MAbs). The study included 5,818 adults with migraine, of whom 4,963 had 6 months of data. The cohort was predominantly female (82.3%, 4,086/4,963) with a median age of 48.0 years (interquartile range: 40.0-55.0).1,2,3

At baseline, participants had a high migraine burden, with a median monthly headache days (MHD) of 20.0 (13.3-28.0) and MMD of 15.0 (10.0-20.0). All participants were classified as having insufficient headache control (>6 MMD) at baseline.1,2,3

After 6 months of anti-CGRP MAb treatment, the distribution across treatment goal categories was as follows:1,2,3

• Migraine freedom: 6.9% (342/4,963)
• Optimal control: 22.9% (1,137/4,963)
• Modest control: 24.6% (1,223/4,963)
• Insufficient control: 45.6% (2,261/4,963)

Within the insufficient control group, 27.1% (613/2,261) achieved a ≥50% reduction in MMD. These findings indicate that high standards of care, defined as optimal disease control or migraine freedom, are achieved in approximately 30% of individuals with a high migraine burden in real-world settings with anti-CGRP MAbs.1,2,3

The study highlights the need to expand global access to these treatments and suggests that initiating migraine-specific preventive treatments earlier could potentially further reduce residual migraine days in responders, thereby enabling a larger proportion of patients to achieve optimal disease control.1,2,3

Migraine is a prevalent neurological disorder characterized by recurrent headaches, often accompanied by symptoms such as throbbing pain, photophobia, phonophobia, and nausea. It significantly impacts quality of life and productivity. Globally, migraine affects a substantial portion of the population, with a higher prevalence in women. Chronic migraine, defined as 15 or more headache days per month for over 3 months, with at least 8 days meeting criteria for migraine, represents a particularly debilitating form of the disorder. The economic burden of migraine includes direct healthcare costs and indirect costs related to lost productivity. Effective preventive treatments are crucial for managing this condition and improving patient outcomes.

Anti-CGRP monoclonal antibodies target the calcitonin gene-related peptide (CGRP) pathway, which plays a key role in migraine pathophysiology. CGRP is a neuropeptide widely distributed in the nervous system, involved in nociceptive transmission and vasodilation. During a migraine attack, CGRP levels increase in the jugular vein. Anti-CGRP MAbs work by either binding to the CGRP ligand itself or to its receptor, thereby preventing CGRP from activating its receptor and mediating its effects. This mechanism of action differs from traditional migraine preventives, which often have broader pharmacological targets and a less specific effect on migraine pathways.

The EUREkA cohort study specifically focused on adults with a high migraine burden, indicated by the median monthly headache days and migraine days at baseline. This patient population often represents individuals who have failed previous preventive treatments or have particularly severe and frequent migraines. The study's multicentre design across Europe contributes to the generalizability of its findings within a European context, reflecting diverse clinical practices and patient demographics. Data collection involved standardized protocols to ensure consistency across participating sites, capturing detailed information on migraine frequency, severity, and treatment response over the 6-month follow-up period. The real-world nature of the study provides valuable insights into the effectiveness of anti-CGRP MAbs in routine clinical practice, complementing findings from controlled clinical trials.

Despite the positive outcomes observed in a significant proportion of patients, the study also identified limitations. Nearly half of the participants remained in the insufficient control group after 6 months of treatment, underscoring the persistent challenge of achieving optimal migraine management for all patients. This could be due to various factors, including individual patient variability in response to treatment, adherence issues, or the presence of comorbidities that complicate migraine management. The study's observational design, while providing real-world data, does not allow for causal inferences to the same extent as randomized controlled trials. Furthermore, the follow-up period of 6 months, while sufficient for assessing initial response, may not fully capture long-term treatment effects or the impact of treatment adjustments over extended periods. Future research could explore predictors of response to anti-CGRP MAbs and investigate strategies to improve outcomes for those who do not achieve optimal control, including combination therapies or alternative treatment approaches.

For a comprehensive and practical guide to understanding and managing neurological conditions, including persistent migraines, further insights can be found in the Oxford Handbook of Neurology.

CLINICAL IMPLICATIONS

The EUREkA cohort data provides a clear benchmark for the real-world effectiveness of anti-CGRP monoclonal antibodies in migraine prevention. While a 30% rate of optimal control or migraine freedom is a significant achievement for patients with high migraine burden, it also underscores that nearly half of patients still experience insufficient control. This necessitates a continued focus on optimising existing migraine-specific therapies and exploring new avenues for the substantial proportion of patients who do not respond adequately.

The observation that GLP-1 receptor agonists do not improve migraine frequency, despite reducing healthcare utilisation, presents an interesting dichotomy. It suggests that any perceived benefit in healthcare resource use is likely indirect, perhaps related to improvements in comorbidities such as obesity or diabetes, rather than a direct antimigraine effect. Clinicians should be cautious not to conflate these indirect benefits with a primary migraine treatment effect. The evidence remains clear: for migraine improvement, specific migraine preventive treatments are paramount.

For the pharmaceutical industry, the EUREkA findings reinforce the market for anti-CGRP MAbs while also highlighting the unmet need for more effective treatments for non-responders. Investment in research for novel migraine-specific mechanisms, or strategies to enhance response rates to current therapies, remains critical. The data also implicitly cautions against overstating the migraine benefits of drugs primarily indicated for other conditions, even if they show ancillary reductions in healthcare visits.

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