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MONOCLONAL ANTIBODIES TARGETING CGRP IN MIGRAINE: CURRENT PERSPECTIVES AND CLINICAL GUIDELINES

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ABSTRACT

Aim: This review aims to explore the clinical relevance and therapeutic potential of monoclonal antibodies targeting the calcitonin gene-related peptide (CGRP) pathway in the preventive treatment of migraine. Emphasis is placed on their mechanism of action, efficacy, and safety as demonstrated in clinical trials, as well as on their practical implementation in routine care. By examining current evidence and real-world application, the review seeks to inform clinicians about the benefits and appropriate use of anti-CGRP monoclonal antibodies in patients with disabling migraine.
Methods: We conducted a comprehensive literature search in April and May 2025 using the PubMed and Google Scholar databases. The search included peer-reviewed articles published between 2015 and 2025, with a focus on studies investigating monoclonal antibodies and geptans used in the treatment of migraine. We prioritized original research involving human subjects and excluded unpublished abstracts and conference proceedings. Only articles available in English and Polish were considered.
Results: Recent studies confirm the sustained safety and effectiveness of all four CGRP monoclonal antibodies, supporting earlier access due to their good tolerability, reduced migraine frequency, and improved quality of life.
Conclusions: We should increase the awareness among specialists about the effectiveness and safety of anti-CGRP monoclonal antibodies so that more patients have the opportunity to benefit from treatment.

KEYWORDS

Migraine, Preventive Therapy, Monoclonal Antibodies Targeting CGRP, Guidelines

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1. Introduction

Migraine is a common, chronic and complex neurological disorder characterized by recurrent headache attacks [1]. This condition is the sixth most disabling disorder in the world, and first most disabling neurological disorder [2]. It is most commonly seen between the ages of 25 and 55. The disorder significantly affects quality of life, limits productivity, and restricts involvement in work, education, and social activities. Notably, around 75% of those affected are women, meaning the condition disproportionately impacts women of reproductive age (15–49 years) [6]. Migraine is commonly characterized by unilateral, pulsating head pain accompanied by hypersensitivity to movement and sensory stimulators, including visual and auditory inputs. A subset of patients experiences a premonitory phase, marked by symptoms such as fatigue, irritability, reduced concentration which may occur up to 48 hours before headache onset. Following the headache phase, many individuals report postdrome symptoms, typically involving persistent fatigue. Additionally, approximately one-third of patients experience transient neurological disturbances, collectively referred to as migraine aura, which are attributed to cortical dysfunction [2]. Given the significant impact of migraine on daily functioning and quality of life for a large patient population, it is essential to place greater emphasis on preventive therapies rather than focusing solely on the acute treatment of attacks. Anti-CGRP monoclonal antibodies represent a promising class of preventive medications [5]. It is crucial that clinicians consider their routine use in the management of patients with frequent or severe migraine, to reduce attack burden and improve long-term outcomes.

2. The role of CGRP proteins in the pathophysiology of migraine

The pathophysiology of migraine is complex and not yet fully understood. Migraine is considered a genetically determined channelopathy, with up to 200 genes potentially implicated in its development. A migraine attack is characterized by hyperreactivity of cerebral blood vessels, triggered by cortical spreading depression. These phenomena are directly linked to the occurrence of migraine aura and lead to the activation of the trigeminovascular system. This system comprises the vascular network, the trigeminal nerve along with its nucleus in the brainstem, and associated cortical centers. Activation of the trigeminovascular system results in the release of vasoactive neuropeptides—such as substance P, calcitonin gene-related peptide (CGRP), and vasoactive intestinal peptide (VIP)—outside the vascular bed [7].

Calcitonin gene-related peptide (CGRP) is one of the key factors in the pathophysiology of migraine. It has been identified in multiple regions of the central nervous system, and during a migraine attack, elevated levels of CGRP have been observed in the external jugular vein, suggesting an increase in its concentration within the central nervous system. CGRP concentrations normalized after the resolution of migraine [7,8].

This neuropeptide exists in two isoforms: alpha-CGRP, which is found in both the central and peripheral nervous systems, and beta-CGRP, which is primarily located in enteric nerve endings. One of its well-established functions is vasodilation [8]. The identification of CGRP's role in migraine pathophysiology has led to the development of targeted therapies, including small-molecule antagonists of the CGRP receptor (known as gepants, e.g. rimegepant, ubrogepant, atogepant) and monoclonal antibodies directed against the CGRP pathway (such as erenumab, fremanezumab, galcanezumab, and eptinezumab). [21]

3. Clinical Application of Anti-CGRP Monoclonal Antibodies

Pharmacological management of migraine may be implemented in two approaches: acute treatment, aimed at alleviating symptoms during an ongoing attack, and preventive therapy, intended to reduce the frequency, duration, and severity of future migraine episodes [5]. Preventive therapy is indicated for adult patients with episodic migraine, defined as experiencing migraine attacks on a minimum of four days per month, with each episode persisting for over 24 hours and demonstrating inadequate response to acute treatment. It is also recommended for individuals with chronic migraine, characterized by headache occurring on more than 15 days per month for a duration of at least three months, with migraine features present on at least eight of those days [9,10].

According to the current guidelines of the European Headache Federation (EHF), anti-CGRP monoclonal antibodies are recommended as a first-line therapy for the preventive treatment of chronic migraine [10] and also they have been shown to be effective in reducing the intensity of severe episodic migraine [12-15].

Anti- CGRP monoclonal antibodies may be divided into two groups: anti-CGRP receptor antibodies (erenumab) and anti-CGRP peptide antibodies (fremanezumab, galcenezumab, eptinezumab). These biologics differ also in terms of antibody structure, route of administration, and pharmacokinetic profile. Understanding

these differences is important for optimizing therapeutic strategies and individualizing treatment plans. Table 1 summarizes the key pharmacological and pharmacokinetic properties of the four currently approved anti-CGRP monoclonal antibodies: erenumab, galcanezumab, fremanezumab, and eptinezumab.

Table 1. Pharmacological and Pharmacokinetic Characteristics of Monoclonal Antibodies Targeting the CGRP Pathway

Feature	Erenumab [22]	Galcanezumab [23]	Fremanezumab [24]	Eptinezumab [25]
Target	CGRP receptor	CGRP ligand	CGRP ligand	CGRP ligand
Antibody type	Fully human IgG2	Humanized IgG4	Humanized IgG2	Humanized IgG2
Route of administration	Subcutaneous (s.c.)	Subcutaneous (s.c.)	Subcutaneous (s.c.)	Intravenous (i.v.)
Dosing regimen	70 or 140 mg every 4 weeks	120 mg monthly (after 240 mg loading dose)	225 mg monthly or 675 mg every 3 months	100 or 300 mg every 12 weeks
t_{max}	4–6 days	5 days	5- 7 days	Immediately after infusion
t_{1/2}	~28 days	~27 days	~30 days	~27 days
Distribution volume	Vz 3,86 l	Vc 7,3 l	Vc 6 l	Vc 3,7 l
Metabolism / Elimination	Proteolysis; not metabolized via CYP450	Catabolized similarly to endogenous IgG; minimal renal/hepatic clearance	Enzymatic proteolysis; minor renal excretion	Intracellular catabolism; not CYP450 metabolized
Effect of renal/hepatic impairment	No clinically significant effect	No clinically significant effect	No clinically significant effect	No clinically significant effect
Most common adverse events	Constipation, injection site reactions, muscle cramps	Injection site pain, upper respiratory tract infections, dizziness	Injection site reactions, fatigue	Nasopharyngitis, hypersensitivity, infusion-related reactions

4. Clinical Efficacy – Results from Randomized Controlled Trials

Randomized controlled trials (RCTs) have consistently demonstrated that monoclonal antibodies targeting the CGRP pathway—erenumab, eptinezumab, fremanezumab, and galcanezumab—are effective in both episodic and chronic migraine prevention. In individuals with episodic migraine, all four CGRP-mAbs showed significant benefits compared to placebo across several endpoints, including reduction in monthly migraine days (MMD), increased responder rates (≥50% reduction in MMD), decreased use of acute migraine medications, and favorable safety profiles, with no significant safety concerns reported [10][12–15][17]. Similar findings were observed in populations with chronic migraine, where the same four agents were associated with meaningful clinical improvements over placebo [10][13][16][18][19].

Furthermore, when directly compared to traditional preventive treatments, such as topiramate, erenumab demonstrated superior efficacy. In a 24-week head-to-head study, erenumab led to a greater reduction in MMD (−5.86 vs. −4.02; $p < 0.001$) and a higher responder rate (55.4% vs. 31.2%; OR 2.76; 95% CI 2.06–3.71; $p < 0.001$) than topiramate. Discontinuation due to adverse events was significantly lower in the erenumab group (10.6%) compared to topiramate (38.9%; OR 0.19; 95% CI 0.13–0.27; $p < 0.001$), with no major safety concerns reported for erenumab [10][20]. Meta-analysis comparing CGRP mAbs with topiramate and

divalproex, showing that while all are more effective than placebo, the oral antiepileptics are less well tolerated. This supports considering CGRP mAbs as first-line preventive treatment [36].

A large 2024 prospective study of 5,818 patients found that over half experienced a ≥50% reduction in monthly headache days. The study also indicated that unilateral pain, fewer migraine days, and lower disability at baseline predicted better treatment outcomes, suggesting that earlier initiation of CGRP mAbs could enhance success rates [37].

Recent studies evaluating anti-CGRP monoclonal antibodies as a drug class support the case for earlier access. A 2024 systematic review and meta-analysis demonstrated that eptinezumab, fremanezumab, galcanezumab, and erenumab significantly reduced the number of patients with medication overuse or medication overuse headache, particularly when using triptans or multiple medications, though not simple analgesics [35].

Efficacy is typically defined as achieving one or more of the following: ≥50% reduction in monthly headache days, a minimum 5-point improvement on the MIDAS scale (for baseline scores between 11–20), or a ≥30% reduction in MIDAS for scores closer to 20. Improvement may also be measured using tools such as the Migraine Physical Function Impact Diary (MPFID), HIT-6, or patient diaries[9]. Anti-CGRP monoclonal antibodies have proven to be a highly effective option for migraine treatment, as confirmed by numerous clinical studies demonstrating their therapeutic value [34].

5. Safety and Adverse Events

Current guidelines do not recommend the use of CGRP-targeting monoclonal antibodies in pregnant or breastfeeding women, in individuals with vascular diseases, or in those with a history of alcohol or drug addiction [10]. Additionally, there is insufficient safety data regarding their use in pediatric and elderly populations[10].

They are not recommended for women planning pregnancy, as their elimination from the body may take several weeks, corresponding to half-lives and we would not be able to determine whether the antibodies would be completely vanished before pregnancy (Table 1). It is recommended to discontinue therapy 6 months before the planned pregnancy [21].

Anti-CGRP therapies operate via two main mechanisms: erenumab blocks the CGRP receptor, while fremanezumab, galcanezumab, and eptinezumab bind directly to the CGRP ligand. Both approaches ultimately inhibit CGRP receptor activation. However, erenumab allows unbound CGRP to potentially interact with alternative receptors, such as the amylin 1 receptor [27], binding affinity for this receptor was shown in in vitro cell culture models. The clinical significance of these off-target interactions remains unclear [28]. Conversely, antibodies targeting CGRP may not block other peptides, such as adrenomedullin, from binding to the CGRP receptor [29].

The long-term cardiovascular safety of CGRP pathway inhibition remains uncertain, considering CGRP's protective role in the cardiovascular system [31,32]. CGRP also contributes to the maintenance of gastrointestinal mucosal integrity, and its inhibition may pose risks, especially in patients with inflammatory bowel disease [33].

Despite these limitations, CGRP antibodies have demonstrated excellent tolerability and safety profiles. Previous studies have not reported any major safety concerns. The most frequently observed adverse events were related to injection sites or infusion-related reactions [12–20]. Furthermore, the development of anti-drug antibodies (ADA) has been observed only in very rare cases, even with humanized antibodies. When present, these antibodies appeared in very low titers and had no impact on the clinical efficacy of the treatment. Due to their route of administration (subcutaneous or intravenous) and long half-life, are expected to show lower interindividual pharmacokinetic variability compared to gepants [26].

Overall, the available data support the favorable safety and efficacy profile of CGRP-targeted therapies, making them a promising option for migraine prevention in appropriately selected patients.

6. Current Guidelines and Recommendations

The latest guidelines from the European Headache Federation (EHF) recommend anti-CGRP monoclonal antibodies as a primary treatment option for the prevention of chronic migraine for patients >18 years old. Furthermore for patients with medication overuse, anti-CGRP monoclonal antibodies are recommended as a first-line preventive option [10].

Dosing Regimens of Anti-CGRP Monoclonal Antibodies:

Erenumab is administered subcutaneously (s.c.) at a dose of either 70 mg or 140 mg once every 4 weeks. It binds to the CGRP receptor and is a fully human monoclonal antibody. [22][26]

Eptinezumab is given intravenously (IV) at doses of 100 mg or 300 mg every 12 weeks. It binds directly to the CGRP peptide and is a humanized monoclonal antibody (90–95% human). [23][26]

Fremanezumab is administered subcutaneously either as 225 mg monthly or 675 mg quarterly. It targets the CGRP peptide and is a humanized monoclonal antibody (90–95% human). [24][26]

Galcanezumab is first given as a 240 mg subcutaneous loading dose, followed by monthly 120 mg subcutaneous injections. It also targets the CGRP peptide and is a humanized monoclonal antibody (90–95% human). [25][26]

In patients with episodic or chronic migraine starting treatment with a monoclonal antibody targeting the CGRP pathway, efficacy should be assessed after at least 3 consecutive months of therapy. A treatment pause may be considered after 12–18 months in patients with stable attack patterns and a ≥50% reduction in headache days compared to baseline [38]. While many experts currently support ongoing treatment as long as clinically needed, efficacy is typically evaluated after 3 or 6 months, depending on the dosing schedule. If migraine attacks worsen after discontinuation, therapy should be resumed. At present, there is insufficient evidence to support combined use of anti-CGRP mAbs with other preventive agents to improve clinical outcomes [9,10].

7. Conclusions

The introduction of monoclonal antibodies targeting the CGRP pathway has marked a significant advancement in the preventive treatment of episodic and chronic migraine. Clinical trials and long-term observational studies have consistently demonstrated their efficacy in improving quality of life, while maintaining a favorable safety and tolerability profile. Recent evidence supports their early use, and updated recommendations from international societies such as the American Headache Society (AHS) and the European Headache Federation (EHF) now endorse anti-CGRP mAbs as first-line therapies in appropriately selected patients. As ongoing research continues to refine our understanding of individualized response predictors and long-term outcomes, anti-CGRP therapies are becoming a cornerstone in the modern management of migraine.

Disclosures

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