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Anti-CGRP monoclonal antibodies in migraine treatment

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Abstract

Migraine is a prevalent neurological disease connected with headache that affects a vast number of people and makes everyday life very difficult. Despite the enormous progress that has been made in migraine treatment in recent years, the disease often remains poorly controlled. The CGRP protein turns out to be very important in migraine's pathogenesis, allowing it to be used as a drug target. One of the newest drugs used in the treatment of migraine are anti-CGRP monoclonal antibodies, which are becoming more and more widely used. Currently, three monoclonal antibodies against the CGRP ligand are available: eptinezumab, fremanezumab, galcanezumab, and one against the CGRP receptor - erenumab. This review will discuss the effectiveness and applicability of CGRP mAbs. *(Farm Współ 2025; 18: 81-87) doi: 10.53139/FW.20251823*

Keywords: Migraine, Anti-CGRP antibody, eptinezumab, erenumab, fremanezumab, galcanezumab

Introduction

Migraine is a neurological disorder associated with headaches caused by hyperexcitability of the central nervous system [1]. This disease is very burdensome for both the patient and their surroundings. It is also a common problem because it affects 18% of women and 6% of men. As much as 2% of the world's population is troubled by chronic migraine [2]. The diagnosis of migraine is made primarily based on a properly conducted interview with the patient [2]. This disease is very burdensome for both the patient and their environment. Patients complain about difficulties in work, family life, and social activities [1]. Unfortunately, the exact pathophysiology of migraine remains unknown. However, the progress observed in recent years in explaining the anatomical and functional changes that accompany a migraine attack or the transformation of episodic migraine into chronic migraine, as well as the genetic factors that may influence susceptibility to migraine, is encouraging [3].

CGRP and its role in migraine pathomechanism

Calcitonin gene-related peptide (CGRP) has two isoforms (α and β) found in sensory neurons throughout the body and in neuroendocrine cells and motor neurons [4]. It is a neuropeptide with various physiological functions. CGRP has been shown to have the ability to activate adenylate cyclase in smooth muscle cells, which results in a substantial dilation of arterioles in the brain.

In addition, when there is a local decrease in cerebral blood flow, CGRP is released by the trigeminal nerves in the trigeminovascular reflex to dilate the vessels and restore normal cerebral blood flow.

During spontaneous migraine attacks, CGRP is selectively released from the trigeminal system, and some drugs can inhibit this process.

It can be assumed that CGRP is involved in the pathophysiology of migraine [5]. CGRP has become an essential target for monoclonal antibodies (mAbs). Currently, four mAbs are available for the preventive treatment of migraine: eptinezumab, fremanezumab, galcanezumab, and erenumab.

The mechanism of action of monoclonal antibodies on CGRP and its receptor is shown in Figure 1.

The use of erenumab or galcanezumab is associated with significant plasma CGRP reduction in migraine patients [6].

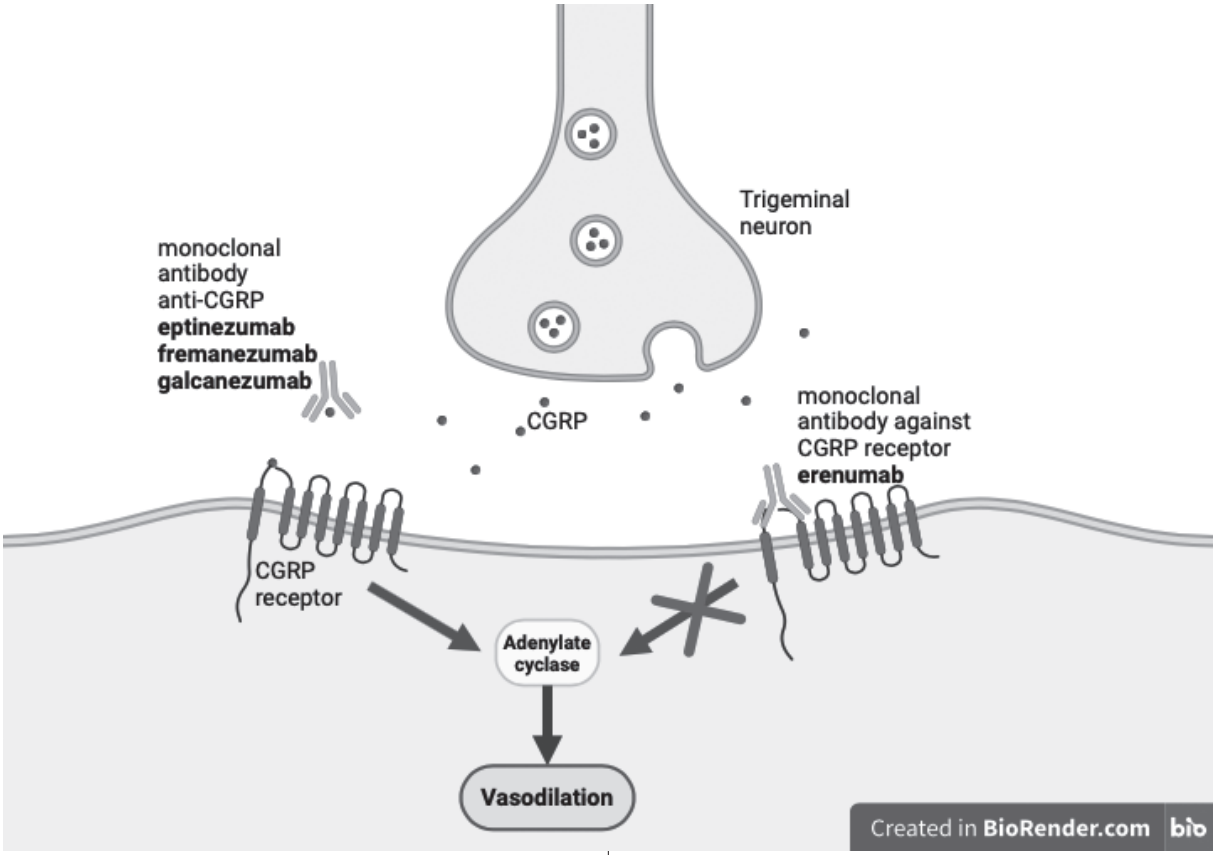

Figure 1. Mechanism of action of monoclonal antibodies on CGRP and its receptor

Erenumab

Erenumab is a human monoclonal antibody against the CGRP receptor. One study also suggests that the possibility of erenumab antagonism towards the AMY1 receptor should be considered [7]. A critical study on erenumab was ARAISE. In this study, the effectiveness of 70 mg of Erenumab was compared to placebo. The percentage of patients who achieved > 50% reduction in monthly migraine days was 39.7% (for the erenumab group). The incidence of adverse events was similar in both groups [8].

In another similar study, STRIVE, Erenumab 70 mg, and 140 mg were compared to placebo. A > 50% reduction in monthly migraine days was achieved in 43.3% for the 70 mg dose and 50.0% for the 140 mg dose. In this case, there were no significant differences in the incidence of adverse events in the study groups [9].

A similar safety profile was observed in children and adolescents as in adults. [10]. Further studies have confirmed strong evidence of erenumab's effectiveness and safety [10,11]. Erenumab demonstrated durable efficacy even in patients with episodic migraine with 2-4 prior treatment failures [12]. The APOLLON study showed that the deterioration of migraines associated with drug discontinuation is reversible, and the reintroduction of treatment has effects similar to those of the initial treatment [13]. Erenumab was also shown to significantly reduce the frequency of migraine and the need for pain medication in patients with refractory chronic migraine and medication overuse headaches [14].

Erenumab was also more effective than rimegepant in monotherapy [15], although it was noted that gepant could be used with good results in patients already treated with erenumab [16].

Patients treated with erenumab reported significant improvement in the impact of headaches on quality of life compared with topiramate [17]. It has also been shown that in patients in whom 1 or 2 pre-

ventive treatments were ineffective, earlier initiation of erenumab is more effective and safer than continuous use of oral migraine prophylactic medications [18].

There is also one case report of the beneficial effect of erenumab on the symptoms of osteoarthritis [19].

It is worth being particularly careful regarding blood pressure in patients treated with erenumab, as it may move to a higher stage in 23.3% of patients who started treatment [20].

In contrast, another study showed that treatment with erenumab, fremanezumab, or galcanezumab for one year did not increase the incidence of hypertension compared with trends in the general population [21].

Fremanezumab

Fremanezumab is a humanized monoclonal antibody that targets a-CGRP and b-CGRP ligands.

One of the first studies on the efficacy of fremanezumab compared a monthly dose of 225 mg and 675 mg every 3 months with a placebo. The percentage of patients with > 50% reduction in monthly migraine days was 41% (for 225 mg every month) and 38% (for 675 mg every 3 months). The most commonly reported adverse event was injection site pain [22]. The efficacy of these doses was assessed in a similar study, where > 50% reduction in monthly migraine days was achieved in 47.7% (for the dose of 225 mg every month) and 44.4% (in the group treated with a dose of 675 mg every 3 months). In this case, the most frequently reported adverse event was also a reaction at the drug injection site [23].

Other studies also confirm the efficacy and safety of fremanezumab [24], as well as in the case of drug-resistant migraine [25].

Fremanezumab has also been used with good results in patients with previous treatment failures, even in cases of drug overuse, and in patients with comorbid psychiatric disorders [26].

Fremanezumab compares favorably with drugs such as topiramate, valproate, and propranolol [27].

Fremanezumab may also be considered when treatment with other mAbs used in migraine has been ineffective [28].

The possible effectiveness of fremanezumab in headaches due to intracranial neoplasia has also been described [29].

Table I. Efficacy and safety of anti-CGRP mAbs

mAb	Study	Dose	The percentage of patients with >50% monthly migraine days reduction	The percentage of patients with treatment-emergent adverse events	References
Erenumab	ARAISE	70mg	39.7%	48.1% Placebo 54.7%	[8]
	STRIVE	70mg 140mg	43.3% 50.0%	57.3% 55.5% Placebo 63.0%	[9]
Fremanezumab		225mg 675mg	41% 38%	71% 70% Placebo 64%	[22]
		225mg 675mg	47.7% 44.4%	66.2% 66.3% Placebo 58.4%	[23]
Galcanezumab	EVOLVE 1	120mg 240mg	62.3% 60.9%	65.5% 67.7% Placebo 60.4%	[30]
	EVOLVE 2	120mg 240mg	57% 59%	65.0% 71.5% Placebo 62.3%	[31]
Eptinezumab	PROMISE 1	30mg 100mg 200mg	50.2% 49.8% 56.3%	58.4% 63.2% 57.6% Placebo 59.5%	[39]
	PROMISE 2	100mg 300mg	57.6% 61.4%	43.5% 52.0% Placebo 46.7%	[40]

Galcanezumab

Galcanezumab, humanized monoclonal antibody, inhibits both α and β ligand forms of CGRP.

The EVOLVE 1 study assessed the percentage of patients with > 50% reduction in migraine frequency for 240 mg and 120 mg of galcanezumab compared with placebo. This was 60.9% (240 mg group), 62.3% (120 mg group). The most commonly reported adverse drug reaction was an injection site reaction, but the number of adverse drug reactions related to the drug was not statistically different from placebo [30].

The EVOLVE 2 study was very similar. Galcanezumab 120 mg and 240 mg were compared with placebo. The percentage of patients with >50% reduction in migraine frequency for 240 mg was 59%, and for 120 mg, it was 57%. The most commonly reported adverse drug reaction was an injection site reaction [31].

Further studies have confirmed the effectiveness of galcanezumab [32], even when prophylactic drugs were ineffective [33] and in the treatment of menstrual migraine [34].

Galcanezumab ensures quick onset of action and long-term effectiveness [35].

However, galcanezumab was not more effective than rimegepant [36].

The use of galcanezumab also showed improvement in sleep quality [37].

Eptinezumab

Eptinezumab is the newest humanized monoclonal antibody, which binds α and β forms of CGRP.

It is the only mAb used in migraine that is administered intravenously, which may be associated with a faster onset of action and higher drug concentration in plasma. Still, as many as 62% of patients are afraid of intravenous injection of the drug. Once treatment begins, this number drops to 14% [38].

One of the first studies to evaluate the effectiveness of epinezumab was the PROMISE 1 study, in which doses of 30 mg, 100 mg, and 300 mg were compared with placebo. A > 50% reduction in monthly migraine days was 50.2% for the 30 mg dose, 49.8% for the 100 mg dose, and 56.3% for the 300 mg dose. In all eptinezumab treatment groups, the most commonly reported adverse events were upper respiratory tract infection (10.5%), nasopharyngitis (6.8%), and sinusitis (3.6%) [39]. In the subsequent PROMISE 2 study, the efficacy of 100 mg and 300 mg of eptinezumab was tested compared to placebo, and a > 50% reduction in monthly migraine days was achieved, 57.6% for the 100 mg dose and 61.4% for the 300 mg dose. In this group treated with eptinezumab, the reported adverse events were not statistically different from placebo [40].

The effectiveness and safety of the drug were confirmed by subsequent studies [41], also in patients in whom preventive treatment proved ineffective [42], and in patients with drug overuse headaches [43]. Unfortunately, in patients who have not previously responded to other anti-CGRP mAbs, eptinezumab is less effective [44].

There is a chance that eptinezumab may be effective in drug-resistant migraine, but the evidence is limited [45].

Table II. Characteristic of individual anti-CGRP monoclonal antibodies [46]

mAb	Class	Target	Administration	Dose	Interval between administrations	Tmax	T1/2	Production
Erenumab	humanized IgG2	CGRP receptor	Subcutaneous injection	70 or 140 mg	4 weeks	5.5 days	21-23 days	Chinese hamster ovary
Fremanezumab	humanized IgG2	α and β CGRP ligand	Subcutaneous injection	225 mg or 675 mg	4 or 12 weeks	5-7 days	31 days	Chinese hamster ovary
Galcanezumab	humanized IgG4	α and β CGRP ligand	Subcutaneous injection	240 mg loading dose, then 120 mg	4 weeks	7-13 days	28 days	Chinese hamster ovary
Eptinezumab	humanized IgG1	α and β CGRP ligand	Intravenous infusion	100-300 mg	12 weeks	2-5 hours	27 days	Yeast

Conclusions

The comparison of the features of individual anti-CGRP monoclonal antibodies is presented in Table 2. Undoubtedly, anti-CGRP monoclonal antibodies in treating migraine were a great discovery. Monoclonal antibodies against CGRP and its receptor are another effective therapeutic option when other drugs have proven ineffective or poorly tolerated. They are more effective in the treating chronic migraine than OnabotulinumtoxinA with comparable safety [47]. As research on anti-CGRP mAbs develops, we learn about their increasingly broader applications. For example, in the prevention of menstrual migraine, mAbs, including erenumab and galcanezumab, are more effective than triptans [48]. However, among the new evidence, there remain many inconsistencies regarding these drugs, which will require more profound research in the future. For example, one study showed that anti-CGRP monoclonal antibodies effectively reduced the average monthly number of aura days [49]. In turn, another study showed that the frequency of aura attacks did not change with a shorter or absent pain phase [50]. Also, the prevention of migraine in children and adolescents still leaves a great need for research. One study highlighted the potential of anti-CGRP monoclonal antibodies in treating adolescents and young adults. However, data on the use of these drugs in this group remains limited [51]. We also lack sufficient evidence to switch antibodies, but changing to another class of antibodies, i.e., a CGRP receptor blocker to a CGRP ligand blocker and vice versa, seems rational, and these changes should be considered no earlier than after 6 months of treatment [52]. Research on the use of monoclonal antibodies should be continued for the further development of headache treatment.

Conflict of interest
None

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