HER-MES CLINICAL TRIAL

HER-MES is the first randomized, double blind, head-to-head study of Aimovig® (erenumab) against topiramate in patients with episodic and chronic migraine[1]

KEY CLINICAL TRIAL DETAILS

Patients:

777 patients suffering from ≥ 4 migraine days/month and who were naïve to, not suitable for or had previously failed up to three prophylactic migraine treatments

Endpoints:

Primary:
Superior tolerability of Aimovig (erenumab) compared with topiramate assessed by treatment discontinuation due to adverse events during the double blind treatment period

Secondary:
Superiority of Aimovig (erenumab) compared with topiramate in terms of at least a 50% reduction in monthly migraine days (MMDs) over the last three months (months 4, 5, and 6) of the double blind treatment period

Dose:

Aimovig (erenumab):
70 mg or 140 mg

Topiramate:
Highest-tolerated dose (50-100 mg/day), starting with a six-week titration phase

Site:

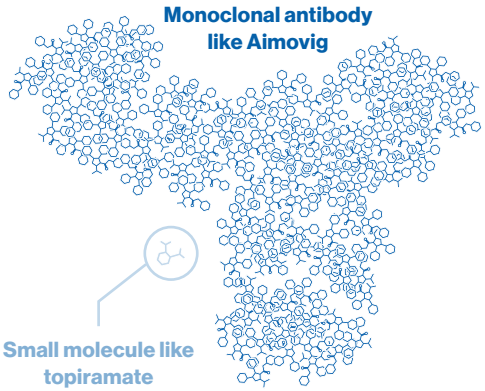
The study was conducted in 82 centers in Germany

RESULTS

Aimovig (erenumab) had a **superior tolerability and efficacy profile** compared with topiramate, with a significantly lower treatment discontinuation rate due to adverse events (10.6% versus 38.9%, respectively)

55.4% of patients in the Aimovig (erenumab) treatment arm experienced **≥50% reduction in MMDs** compared with 31.2% in the topiramate arm

HOW ARE AIMOVIG (ERENUMAB) AND TOPIRAMATE DIFFERENT?



Both Aimovig (erenumab) and topiramate are migraine prevention treatments but they work differently:

Aimovig (erenumab) is a monoclonal antibody and topiramate is a small molecule, with very different mechanisms of action. While small molecules work acutely, antibodies are heavy molecules that are able to bind to target proteins for a longer time and provide long lasting therapeutic action[2]

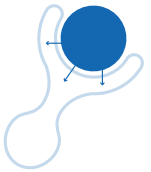
Aimovig (erenumab) is engineered to specifically block calcitonin gene-related peptide (CGRP) receptors to prevent migraines, whereas topiramate is an anti-convulsant also used for migraine prevention[3]

MIGRAINE AND ITS PATHOGENESIS

Migraine can be triggered by a variety of elements, including environmental factors (such as light, sound, certain food or stress)

Once triggered, the body releases neurotransmitters, one of them is called calcitonin gene-related peptides (CGRPs)

When released, CGRPs look for their docking stations (CGRP receptors), which are located in and around the brain



Migraine pain and other accompanying symptoms are then set off once CGRPs connect to their docking station[4,5]

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References

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4. Bigal ME, Walter S, Rapoport AM. Calcitonin gene-related peptide (CGRP) and migraine current understanding and state of development. Headache. 2013;53(8):1230-44.
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