Potential for treatment benefit of small molecule CGRP receptor antagonist plus monoclonal antibody in migraine therapy

Kathleen Mullin, MD, David Kudrow, MD, Robert Croop, MD, et al.

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Study objective
This study describes the use of 2 calcitonin gene-related peptide (CGRP) therapies to treat refractory migraine, and the results showed that rimegepant was effective for acute treatment during concomitant treatment with erenumab for preventive treatment.

Classification of evidence
Class IV.

What is known and what this paper adds
CGRP-targeting monoclonal antibodies are approved for the preventive treatment of migraine, and small molecule CGRP receptor antagonists, known as gepants, are approved for the acute treatment of migraine. This report suggests that using a gepant for acute treatment in combination with a monoclonal antibody for preventive treatment is a viable therapeutic strategy for refractory migraine.

Participants and setting
Patient 1 was a 44-year-old white woman, and patient 2 was a 36-year-old white woman. Both patients reported experiencing ≥19 years of suboptimal responsiveness to multiple antimigraine medications. Both participated in a multisite clinical trial (NCT03266588) investigating the safety of 75-mg rimegepant tablets.

Design, size, and duration
Patient 1 had been taking rimegepant for 6 months when she began receiving monthly subcutaneous 70-mg erenumab injections. Patient 2 had been taking rimegepant for 2 months when she began receiving monthly subcutaneous 140-mg erenumab injections. While taking both rimegepant and erenumab, the patients recorded their experiences with treating acute migraine attacks.

Primary outcome measures
The primary outcomes were the patients’ evaluations of whether rimegepant alleviated their acute migraine attacks while they were receiving preventive treatment with erenumab.

Main results and the role of chance
Both patients reported that taking rimegepant as an acute treatment while taking erenumab as a preventive provided substantial relief from all acute migraine attacks.

Bias, confounding, and other reasons for caution
The present study presents anecdotal reports from only 2 patients.

Generalizability to other populations
The focus on 2 middle-aged white women may limit the generalizability of the results to dissimilar patients.

Study funding/potential competing interests
This study was funded by Biohaven Pharmaceuticals. Some authors report being Biohaven Pharmaceuticals employees; owning stock or stock options in Biohaven Pharmaceuticals and eNeura Therapeutics; serving on the editorial boards of journals, including Neurology®; receiving consulting fees, honoraria, committee appointments, and funding from healthcare companies, including Biohaven Pharmaceuticals; receiving funding from the NIH and foundations; and receiving publishing royalties. Go to Neurology.org/N for full disclosures.

Table
Numbers of migraine attacks experienced and numbers successfully treated

<table>
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<tr>
<th>Patient</th>
<th>No. of migraine attacks while using rimegepant and erenumab</th>
<th>No. of attacks successfully treated with rimegepant</th>
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<tr>
<td>1</td>
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Neurology® 2020;94:900. doi:10.1212/WNL.0000000000009280

In the article “Potential for treatment benefit of small molecule CGRP receptor antagonist plus monoclonal antibody in migraine therapy” by Mullin et al.,¹ first published online January 13, 2020, the last sentence of the abstract’s Results should read “While using rimegepant alone or together with erenumab, patients reported no related adverse events.” The sentence appears correctly in the May 19, 2020, issue. The authors regret the error.

Reference

Retinal defect in children with infantile spasms of varying etiologies
An observational study

Neurology® 2020;94:900. doi:10.1212/WNL.0000000000009279

In the article “Retinal defect in children with infantile spasms of varying etiologies: An observational study” by McFarlane et al.,¹ the percentage of vigabatrin-naive children (59 out of 312) should be 18.9% in the Abstract Results, in the Results section under the heading, “Prevalence of retinal defect in vigabatrin-naive children,” and in table 3. Further, the second and third sentences in the Discussion should read, “We found that nearly a fifth of vigabatrin-naive children (before treatment with vigabatrin or <4 weeks of vigabatrin treatment) with IS showed evidence of a retinal defect on the 30-Hz flicker ERG. Nearly a quarter of vigabatrin-naive children with a structural-acquired perinatal cause of spasms had an abnormal ERG.” Finally, the first sentence of the last paragraph of the Discussion section should read, “The 30-Hz flicker ERG response is reduced in almost 19% of children under 3 years of age with IS who are naive to vigabatrin.” The initial online version of the article has been republished with the corrected text along with a supplement (links.lww.com/WNL/B51) highlighting the errors, and the final version appears without the errors. The authors regret the errors.

Reference