2631 EARLY ONSET OF EFFICACY WITH ATOGEPANT FOR THE PREVENTIVE TREATMENT OF CHRONIC MIGRAINE: RESULTS FROM THE PROGRESS TRIAL

[1]Jessica Ailani, [2,3]Uwe Reuter, [4]Brad C Klein, [5]Sung Yun Yu, [6]Krisztian Nagy, [7]Hua Go, [8]Kate Watts*, [6]Joel Trugman. *[1]MedStar, Georgetown University Hospital, Washington DC, USA; [2]Charité Universitätsmedizin Berlin, Berlin, Germany; [3]Universitätsmedizin Greifswald, Greifswald, Germany; [4]Island Health, Brentwood Bay, BC, Canada; [5]Abington Neurological Associates, Abington, PA, USA; [6]AbbVie, Maddison, NJ, USA; [7]AbbVie, Budapest, Hungary; [8]AbbVie, Mascot, NSW, Australia *This individual has been added to the author by-line, with the permission of the original authors, for the express purpose of conducting the presentation at a local congress. He/she did not contribute to the content of the publication*

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Objectives Evaluate the time course of efficacy of atogepant for the preventive treatment of Chronic Migraine (CM).

Methods PROGRESS was a phase 3 trial which assessed the efficacy and safety of atogepant in patients with CM. This analysis evaluated the change from baseline in mean monthly migraine days (MMDs) during 4-week intervals, change in weekly migraine days during weeks 1–4, and the proportion of participants with a migraine on each day during the first 7 days of treatment.

Results 755 participants were included. Baseline MMDs ranged from 18.6 to 19.2. During weeks 1–4 of treatment, mean changes in MMDs were −6.6 for atogepant 30mg BID, −6.2 for atogepant 60mg QD, and −3.7 for placebo ($P<0.001$). This decrease was maintained during weeks 5–8 ($P<0.001$). Baseline mean weekly migraine days ranged from 4.6 to 4.8. During each week of the 1–4 weeks, mean reductions in weekly migraine days were greater in both atogepant groups compared with placebo ($P\leq0.009$). During the baseline period, daily rates of participants reporting a migraine day ranged from 66.3% to 68.4%. On the first day after treatment initiation, atogepant participants were less likely to have a migraine than placebo ($P\leq0.03$). TEAEs were reported by 56.4–63.2% participants taking atogepant, compared with 49.4% participants taking placebo. The most common TEAEs for atogepant were constipation (10.0–10.9%) and nausea (7.8–9.6%).

Conclusion Atogepant demonstrated early and sustained reduction in migraine days. Results showed a statistically significant effect as early as the first full day after study drug initiation. Atogepant was safe and well tolerated.

2632 AN ATYPICAL CASE OF NEUROPATHOLOGICALLY PROVEN SPORADIC CREUTZFELDT-JAKOB DISEASE

Laurence Preen*, Andrew Clarke. *Royal Adelaide Hospital, Adelaide, SA, Australia*

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Creutzfeldt-Jakob disease (CJD) is a rare neurodegenerative human prion disease with a worldwide incidence of 1-per-million. CJD typically is rapidly progressive and invariably fatal, usually within a year of diagnosis in 90% of cases. It is suggested that some cases may go under-reported due to atypical subtypes, negative supplementary testing and the low rates of brain autopsy in this population.

A 53-year-old female presented with a 3-year history of progressive dementia, and physical decline which had progressed to near complete functional incapacity. Clinical examination at this late stage revealed advanced dementia, akinetic mutism, generalized myoclonus, dystonia, rigidity and pyramidal dysfunction. Magnetic Resonance Imaging (MRI) of her brain demonstrated global parenchymal atrophy without typical T2-weighted hyperintense signal changes or diffusion restriction changes. Electroencephalography (EEG) demonstrated mild intermittent slowing without periodic sharp wave complexes. Her Cerebrospinal Fluid (CSF) demonstrated normal protein, glucose, microscopy and total Tau-protein with negative 14–3-3 protein and real-time quaking-induced conversion (RT-QuIC) assay. She underwent genetic testing with a phenotype driven exome analysis, which did not identify a disease-causing variant and specifically did not reveal a prion protein gene (PRNP) mutation. Post-mortem neuropathological examination demonstrated extensive spongiform changes with moderate degree of prion protein plaque-like aggregates diagnostic of CJD.

This case highlights the importance of the post-mortem neuropathological examination when clinical suspicion for CJD exists, but there is an atypical timeline and supplementary testing is not supportive.

2634 CYCLICAL ROZANOLIXIZUMAB TREATMENT IN GENERALISED MYASTHENIA GRAVIS (GMG): POOLED ANALYSIS OF MYCARING (PHASE 3 STUDY) AND TWO OPEN-LABEL EXTENSION STUDIES

[1,2]Jeremy M Welton*, [3]Vera Bril, [4]Artur Drużdż, [5]Julian Grosskreutz, [6]Ali A Habib, [7]Renato Mantegazza, [8]Kimiaki Utsugisawa, [9]John Vissing, [10]Tuan Vu, [11]Marion Boehnlein, [12]Maryam Gayfieva, [11]Bernhard Greve, [11]Franz Woltering, [13]Henry J Kaminski. *[1]UCB Pharma, Malvern, VIC, Australia; [2]Department of Medicine, St Vincent's Hospital, University of Melbourne, Fitzroy, VIC, Australia; [3]University Health Network, Toronto, Canada; [4]Department of Neurology, Municipal Hospital Poznań, Poznań, Poland; [5]Precision Neurology, Department of Neurology, University of Lübeck, Lübeck, Germany; [6]MDA ALS and Neuromuscular Center, University of California, Irvine, Orange, California, USA; [7]Fondazione Istituto di Ricovero e Cura a Carattere Scientifico, Istituto Nazionale Neurologico Carlo Besta, Milan, Italy; [8]Department of Neurology, Hanamaki General Hospital, Hanamaki, Japan; [9]Department of Neurology, Rigshospitalet, University of Copenhagen, Copenhagen, Denmark; [10]Department of Neurology, University of South Florida Morsani College of Medicine, Tampa, Florida, USA; [11]UCB Pharma, Monheim, Germany; [12]UCB Pharma, Slough, UK; [13]Department of Neurology and Rehabilitation Medicine, George Washington University, Washington DC, USA*

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Objective To assess efficacy and safety of cyclical rozanolixizumab treatment, based on study MG0003 with the open-label extensions (OLEs) MG0004 (NCT04124965) and MG0007 (NCT04650854).

Methods MG0004 was an OLE study of ≤52 weekly rozanolixizumab subcutaneous infusions. In MG0007, after an initial cycle, cycles were 'symptom-driven', administered on symptom worsening (at investigator's discretion, e.g., MG-ADL increase ≥2/Quantitative Myasthenia Gravis [QMG] increase ≥3). Data were pooled for patients with ≥2 symptom-driven cycles across MycarinG, MG0004 (first 6 weeks) and MG0007 (interim data) (efficacy); ≥1 cycle across MycarinG (symptom-driven) and MG0007 (fixed/symptom-driven) (safety).

Results 127 (64.8%) patients received ≥2 symptom-driven rozanolixizumab cycles (7mg/kg, n=69; 10mg/kg, n=58). Treatment response was consistent across cycles 1–6; Day 43 mean change from baseline (CFB) in MG-ADL score for all rozanolixizumab-treated patients: Cycle 1 (n=127): −3.7; Cycle 2 (n=127): −3.9; Cycle 3 (n=98): −3.4; Cycle 4 (n=75): −3.8; Cycle 5 (n=51): −3.9; Cycle 6 (n=32): −4.5. Day 43 mean CFB in QMG score was: Cycle 1 (n=127): −5.4; Cycle 2 (n=125): −4.7; Cycle 3 (n=97): −4.7; Cycle