difficulty accessing immunoglobulin treatment for patients diagnosed with Chronic Inflammatory Demyelinating Polyneuropathy (CIDP). Methods: A retrospective cross-sectional study was conducted with CIDP patients (n=16, 68.75% female, mean age 60.38 ± 11.32) recruited from three Montreal tertiary care institutions. Patients completed a questionnaire inquiring about changes in their immunoglobulin treatment during the pandemic and about their quality of life. We used weighted chi-squared statistical tests and Cramer's V correlation ratios to measure associations with treatment change. Results: Eighteen months after the pandemic started, 25% of our population were receiving immunoglobulin treatment at a different frequency, 6.3% were receiving a different dose, 12.5% were receiving a different dose and frequency, and 6.3% were receiving a different treatment. Reasons associated with treatment change were worsening of neurological condition (18.8%; Cramer's V=0.480; p-value=0.055), improvement of neurological condition (25%; Cramer's V=0.577; p-value=0.021) and reduced availability of treatment (6.3%; Cramer's V=0.258; p-value=0.302). There were no significant correlations between lower quality of life (p-value=0.323) or lower Rasch-built Overall Disability Scale score (p-value=0.574) and treatment change. Conclusions: Difficulty accessing immunoglobulin treatment was not significantly associated with treatment change for CIDP patients during the COVID-19 pandemic.

P.032

NMDA receptor encephalitis with severe orofacial dyskinesias treated tramadol and clonazepam

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Background: Anti-*N*-methyl-D-aspartate receptor (NMDAR) encephalitis is a neuroinflammatory disease mediated by antibodies targeting the GluN1 subunit of the NMDAR. It presents with well-defined neuropsychiatric symptoms, including psychosis, agitation, seizures, and memory disturbances. Movement disorders including orofacial dyskinesias are common, but often difficult to manage, with no specific published guidelines. Methods: A 23-year-old female was diagnosed with NMDAR encephalitis. She was treated with ovarian teratoma removal, corticosteroids, intravenous immunoglobulin therapy, rituximab, and tocilizumab. She continued to experience severe, self-mutilating orofacial dyskinesias. Tetrabenazine, haloperidol, and diazepam did not yield any sustained improvement. Tramadol was started based on a prior case report suggesting its efficacy. Results: Tramadol 50 mg po q6h led to immediate improvement in symptoms. Over the next 5 days, tramadol was increased to 150mg NG q6h and further reduced movements. When tramadol was held for one day, the movements significantly worsened and improved when it was restarted. Clonazepam 1mg NG QID also led to further improvement. Conclusions: Tramadol and clonazepam effectively treated severe orofacial dyskinesias in a patient with NMDAR encephalitis and refractory symptoms despite aggressive management. We propose early use of tramadol and clonazepam be considered for severe orofacial dyskinesias secondary to NMDAR encephalitis.

P.033

Detection of Myelin Oligodendrocyte Glycoprotein Immunoglobulin G (MOG-IgG) by live and fixed cell-based assays

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Background: MOG-IgG is associated with non-MS demyelinating disease of the optic nerves, spinal cord and brain. Specificity has been issue so we validated the live and fixed MOG-IgG CBAs against the Oxford Autoimmune Neurology Diagnostic Laboratory (OANG) live CBA as a comparator with high specificity. Methods: At BC Neuroimmunology lab (BCNI), 54 MOG-IgG serum samples previously positive by live-CBA at OANG and BCNI were blindly tested by commercial fixed CBA. All 54 MOG IgG positives came from MOG-IgG positive patients. In addition, 256 samples from healthy people and other neurolgic disease were tested. Results: The live MOG-IgG CBA performed at BCNI was 100% concordant (54/54) with OANG live CBA. In contrast, only 49/54 samples were found seropositive by the commercial fixed CBA. The BCNI live-CBA identified 3/256 control samples as positive while 6/256 controls were positive on the fixed commercial CBA. On this cohort the live CBA is 100% sensitive, 98.8% specific and has PPV of 95%. The commercial fixed MOG test is 91% sensitive, 97.6% specific and has PPV of 87.5%. Conclusions: BCNI live MOG-IgG CBAs are in 100% agreement with MOG-IgG. Three positive results in non-MOGAD associated clinical phenotype require further investigation. These data confirm the superiority of the live MOG CBA.

P.034

Temporal lobe epilepsy associated with autoimmune conditions: a review

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Background: Epilepsy mediated by immune cells must be identified early since immunotherapy has been associated with better clinical outcomes. This provides an overview of autoimmune TLE, emphasizing recent developments in its pathophysiology, imaging, and therapeutic interventions. Methods: Web-based research using advanced features of databases. Results: Epilepsy caused by immune dysfunction leads to inflammation of the brain. Inflammation play a role in the development of seizures. Proinflammatory molecules found to be overexpressed in neurons and glia of individuals with DRE, provoke a proinflammatory cytokines in the plasma and CSF. Autoimmune epilepsy is characterized by focal seizures refractory to ASMs accompanied by other neurological manifestations, as described by clinical scoring systems.Scoring systems are available to identify patients who are likely to be positive. The MRI findings include signal hyperintensities in the affected brain regions. EEG