

associated with renal cell carcinoma and anti-NMDAR and EBV DNA detected in cerebrospinal fluid (CSF).

**Methods** This was a personal case of the authors, with documentation and investigations reviewed from patient medical records at Sir Charles Gairdner Hospital.

**Results** A 55 years old immunocompromised female patient presented with two weeks of increasing ataxia, oscillopsia and tremor, preceded by 3 weeks of headache, nausea and vomiting. This was on a background of three failed renal transplants for IgA focal segmental glomerulosclerosis and recent resection of stage I renal clear cell carcinoma. Examination was consistent with opsoclonus-myoclonus-ataxia syndrome. There were no features on encephalopathy apart from mild emotional lability. CSF results: lymphocytosis with an elevated protein, positive EBV using polymerase chain reaction and positive anti-NMDAR. Treatment: Plasma exchange, Rituximab, intravenous acyclovir for two weeks followed by oral acyclovir for 3 months. Improvement was marked, although with residual myoclonus on 2 month follow-up.

**Conclusions** This rare case of paraneoplastic double-positive EBV and anti-NMDAR opsoclonus-myoclonus syndrome in an immunocompromised patient demonstrates the broadening clinical phenotype of anti-NMDAR and highlights the contentious issue of EBV pathogenicity and treatment in an immunocompromised patient.

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#### CONTACTIN-1-MEDIATED CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY (CIDP) PRESENTING AS AN ACUTE CASE OF GUILLAIN BARE SYNDROME (GBS)

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New and emerging evidence describing the role of autoantibodies to nodal and paranodal proteins in the pathogenesis of chronic inflammatory demyelinating polyneuropathy (CIDP) has arisen over the past decade, with significant diagnostic, therapeutic and prognostic implications. Although rare, CIDP with anti-contactin-1 (CNTN1) IgG4 antibodies has a distinct pathogenesis and clinical phenotype that differs from both typical CIDP and its other atypical variants. Here, we report the case of a young normally well 49-year-old man from rural Australia with severe refractory anti-CNTN1-mediated CIDP who presented like a case of Guillain-Barré syndrome (GBS) who improved dramatically after

chemoimmunotherapy with Rituximab and who also had an unexpected late response to subsequent treatments with intravenous immunoglobulin (IVIg). In reporting this case, we hope to highlight important considerations in the diagnosis and treatment of patients with severe refractory CIDP, and especially those patients with anti-CNTN1 seropositive disease.

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#### NEUROFILAMENT LIGHT CHAIN CONCENTRATION PREDICTS RISK OF RELAPSE IN PARTICIPANTS WITH RELAPSING MULTIPLE SCLEROSIS IN PHASE 3 OZANIMOD TRIALS

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**Objectives** Plasma neurofilament light chain concentration (pNfL-c) is increased in patients with multiple sclerosis (MS) and may serve as a biomarker for neurologic damage and disease activity in relapsing MS. We analyzed changes in pNfL-c and on-treatment risk of relapse with ozanimod vs interferon  $\beta$ -1a (IFN).

**Methods** In this post hoc analysis of the phase 3 SUNBEAM (NCT02294058;  $\geq 12$  months) and RADIANCE (NCT02047734; 24 months) trials, pNfL-c was measured at baseline and after 12 and 24 months of treatment with oral ozanimod 0.46 or 0.92 mg/d or intramuscular IFN 30  $\mu$ g/wk. Poisson generalized linear models were used to fit the number of relapses as a function of baseline pNfL-c and treatment group with an offset for duration. Predictive modeling of expected annualized relapse rate (ARR) was calculated using median percentage change in pNfL-c from baseline.

**Results** At end of treatment, median pNfL-c was reduced from baseline by 20%–23% ( $P < 0.01$ ) and 23%–27% ( $P \leq 0.0001$ ) with ozanimod 0.46 and 0.92 mg, respectively, and by 13%–15% with IFN. Higher baseline pNfL-c was associated with more relapses ( $P < 0.0001$ ), and greater median reductions in pNfL-c from baseline were associated with lower ARR.