Predictive modeling estimated that a 25% reduction in pNfL-c, similar to that observed with ozanimod 0.92 mg, predicts an ARR (standard error [SE]) of 0.18–0.23 (0.4), whereas a 13% reduction, similar to IFN, predicts an ARR (SE) of 0.29–0.37 (0.04).

**Conclusion**
Our findings support pNfL-c as a biomarker for relapsing MS disease activity. Ozanimod caused greater dose-dependent reductions in pNfL-c and ARR than IFN.

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**BRADYCARDIA AS A RARE NEUROCARDIAC PRODROME TO LEUCINE-RICH GLIOMA INACTIVATED-1 ANTIBODY ENCEPHALITIS**

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**Introduction**
Leucine-rich glioma inactivated-1 antibody encephalitis has been associated with bradycardia as a neurocardiac prodrome. Concurrent occurrence of cardiac arrhythmia and faciobrachial dystonic seizures have not previously been reported.

Case A 73-year-old male presented with a 6 week history of frequent episodes of an unpleasant sensation associated with sinus bradycardia requiring pacemaker implantation. Episodes continued despite pacemaker. He was diagnosed with a seizure disorder and commenced on levetiracetam without response.

Subsequently, on video EEG, subtle facial grimace and upper limb tonicity were captured, in keeping with faciobrachial dystonic seizures without an EEG correlate. MRI Brain showed no radiological evidence of encephalitis. Serum limbic encephalitis panel confirmed LGI1 antibodies. Other autoimmune and paracrine antibodies were negative. He was treated with a course of corticosteroids. Induction dose of intravenous immunoglobulin was prematurely terminated after one dose due to MRSA bacteraemia and tricuspid valve endocarditis, necessitating removal of the pacemaker with no recurrence of seizures or bradycardia at follow up without further treatment.

**Conclusion**
This case illustrates a rare presentation of LGI-1 antibody encephalitis with complete remission following incomplete induction course of intravenous immunoglobulin and corticosteroids. Neurocardiac prodrome as episodic bradycardia or asystole may precede the onset of encephalitis by approximately 2 months. There is a good response to immunotherapy, however relapse is common. This case illustrates that clinically atypical presentations of cardiac arrhythmia may warrant neurological review and raises a possibility that early initiation of immunosuppressive therapy may significantly alter the disease course of LGI-1 antibody encephalitis.

**REFERENCES**

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**HEMI-CORD INFARCTION FOLLOWING VERTEBRAL ARTERY DISSECTION IN A PATIENT WITH CONGENITAL HYPOPLASTIC VERTEBRAL ARTERY: A CASE REPORT**

1. Alanna Rottler, 1Yew Li (Michelle) Dang, 2Wai Foong Hooi, 3David A Burrows, 4Hong Kuan Kok, 1Douglas E Crompton. 1Department of Neurology, Northern Health, Epping, VIC, Australia; 2Department of Neurology, Eastern Health, Box Hill, VIC, Australia; 3Department of Neurology, Austin Health, Heidelberg, VIC, Australia; 4Department of Radiology, Northern Health, Epping, VIC, Australia; 5School of Medicine, Faculty of Health, Deakin University, Burwood, VIC, Australia; 6Department of Medicine, Melbourne University, Northern Health, Epping, VIC, Australia

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**Background**
While often causing posterior circulation strokes, vertebral artery dissections may also, more rarely, cause spinal cord infarction. This is the case report of a 39-year-old female with a right-sided high cervical hemi-cord infarction caused by vertebral artery dissection of a hypoplastic right vertebral artery.