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.

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

Sai Nagaratnam, Yun T Hwang, Elizabeth Reyneke. *Neurology, Gosford Hospital, Gosford, NSW, Australia*

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Introduction Leucine-rich glioma inactivated-1 antibody encephalitis has been associated with bradycardia as a neurocardiac prodrome.[1] 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 paraneoplastic 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.[1 2] There is a good response to immunotherapy, however relapse is common.[3] 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.

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069 A PUTATIVE MECHANISM FOR SUBCORTICAL APHASIA

[1,2]Shadi El-Wahsh, [3]David Greenup, [4]Gemma White, [5,6]Michael J Fulham, [3,7]Arun Aggarwal, [1]G Michael Halmagyi. *[1]Neurology, Royal Prince Alfred Hospital, Sydney, NSW, Australia; [2]South Western Sydney Clinical School, University of New South Wales, Sydney, NSW, Australia; [3]Department of Rehabilitation Medicine, Balmain Hospital, Sydney, NSW, Australia; [4]Speech pathology department, Balmain Hospital, Sydney, NSW, Australia; [5]Department of Molecular Imaging (PET and Nuclear Medicine), Royal Prince Alfred Hospital, Sydney, NSW, Australia; [6]Sydney Medical School, University of Sydney, Sydney, NSW, Australia; [7]Neurology, Concord Repatriation General Hospital, Sydney, NSW, Australia*

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Objectives The role of subcortical structures in language function are still poorly understood. We aim to provide a putative mechanism for subcortical aphasia through a structural and functional imaging-based case discussion.

Methods We present a case of subcortical aphasia due to basal ganglia hypertensive haemorrhage and discuss serial MRI and PET imaging findings to elucidate the mechanism of profound language impairment in acute subcortical pathology.

Results A 71-year-old right-handed architect presented with acute onset global aphasia and right-sided hemiparesis. CT imaging showed a flame-shaped left-sided basal ganglia haemorrhage. MRI brain showed a left basal ganglia haemorrhage without ischaemic or haemorrhagic damage to the overlying fronto-parietal cortex. FDG-PET imaging showed profound left fronto-parietal cortex hypometabolism, as well as ipsilateral caudate, putamen, thalamic and pontine hypometabolism. MR tractography identified truncation of the arcuate fasciculus around the left angular gyrus as well as disconnection of the left fronto-parietal association fibres. Over 12 weeks of rehabilitation, the patient began to generate verbal output and was discharged home with ongoing word finding difficulties, nominal aphasia, and semantic paraphasias. Progress PET imaging revealed persistent hypometabolism in the aforementioned regions.

Conclusion We believe this is an important educational case for neurologists regarding the presentation of aphasia due to isolated subcortical lesions and raises some interesting hypotheses regarding a putative mechanism for subcortical aphasia due to dominant hemisphere cortical inactivation.

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

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

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Background Whilst often causing posterior circulation strokes, vertebral artery dissections may also, more rarely, cause spinal cord infarction.[1] 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.