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.

Objective 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.

Background Whilst 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.