

day discharge. The MRI result changed management in 88% of patients.

**Conclusion** A significant proportion of patients experienced delay to discharge due to delay of inpatient MRI. The result of MRI changed treatment plans for most patients, and allowed same day discharge in almost one third of cases. Timely access to inpatient MRI is a critical and potentially modifiable variable that may reduce length of stay and expedite treatment for neurology inpatients.

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#### A CASE REPORT OF MYELIN OLIGODENDROCYTE GLYCOPROTEIN ANTIBODY POSITIVE ENCEPHALITIS MIMICKING HANDL SYNDROME WITH SUBSEQUENT ACUTE OPTIC NEURITIS

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**Background** A 22 year-old right-handed female presented with acute onset of speech disturbance. There was no visual/motor deficit or clinical seizure activity. CT brain/angiography/perfusion demonstrated hypoperfusion in the left temporo-parieto-occipital region (not conforming to a single vascular territory).

There was subsequent improvement in her speech, and she reported a severe left temporal headache with associated nausea and photophobia. An urgent MRI brain was normal. She was diagnosed with migraine with dysphasic aura and treated symptomatically with ongoing improvement. On review the next day she reported mild ongoing headache, but her neurological examination was normal with no dysphasia.

2 days later, she developed right retro-orbital pain, which was followed a further 3 days later by right eye monocular visual impairment. Clinical examination was consistent with acute right optic neuritis. Repeat MRI brain/orbits demonstrated new changes of right optic neuritis with perineuritis and a left thalamic T2-hyperintense lesion. CSF studies demonstrated a monocytic pleocytosis.

The clinical impression was of possible myelin oligodendrocyte glycoprotein (MOG) antibody disease leading to optic neuritis and unilateral cortical encephalitis, manifesting as cortical spreading depression/migraine aura.

She was commenced on IV methylprednisolone followed by an oral prednisolone taper with complete symptom resolution.

Her serum subsequently tested positive for MOG antibody.

**Conclusions** MOG-antibody disease can cause a unilateral encephalitis that may cause attacks of cortical spreading depression. This case demonstrates that this may occur in the absence of the previously reported significant unilateral cortical MRI changes – we hypothesise that some patients previously diagnosed with ‘HANDL syndrome’ may have MOG-antibody disease.

#### REFERENCES

- Ogawa R, Nakashima I, Takahashi T, et al. MOG antibody-positive, benign, unilateral, cerebral cortical encephalitis with epilepsy. *Neurol Neuroimmunol Neuroinflamm* 2017;4(2):e322. Published 2017 Jan 16. doi:10.1212/NXI.0000000000000322
- Matoba, S, Inoue, M, Morihata, H, Takeshima, T. Case report of myelin oligodendrocyte glycoprotein antibody-positive encephalitis mimicking hemiplegic migraine. *Neurol Clin Neurosci* 2020;8:323– 325. <https://doi.org/10.1111/nncn.12420>

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#### FEASIBILITY OF INSTITUTING GRADUATED HIGH INTENSITY TRAINING FOR PARKINSON DISEASE (FIGHT-PD); A NON-CONTACT BOXING EXERCISE STUDY

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**Objectives** Preliminary evidence suggests non-contact boxing exercise is feasible and possibly beneficial for Parkinson Disease (PD). Current studies lack detailed description of component elements and documentation of exercise intensity. We present the protocol of FIGHT-PD; which explores the feasibility, tolerability and safety of a non-contact boxing exercise program for PD, developed by a neurologist (who has PD), a professional boxing trainer, a neurophysiotherapist and exercise physiologists.

**Methods** Twenty early stage (Hoehn and Yahr 1 and 2) PD subjects will undergo baseline evaluations of PD and cardiac stress testing. Training includes quantifiable balance and movement drills, high intensity aerobic bursts, and sequences of punches using the Fightmaster training machine.

Over 15 weeks, three 30-60 minute workouts per week will be conducted in three, 4 week blocks separated by rest weeks. Block one focuses on technique; the second escalates the physical intensity, and the third adds cognitive challenges. Rate of perceived physical exertion (RPE) and mental exertion will be measured by the Borg scale for every component of each workout, and heart rate continuously recorded by Polar monitors. Numerous standardised PD scales and a body chart discomfort scale will be administered at each workout, monitoring the development of pain or injuries. These observations will provide the primary outcomes of tolerability and safety, and secondary outcomes of quantified heart rate measuring exercise intensity, and effect on quality of life. Feasibility details including recruitment, retention and adherence rates will be measured.

**Conclusions** This trial should provide essential details to plan future exercise-based studies.

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#### PEMBROLIZUMAB INDUCED LAMBERT-EATON MYASTHENIC SYNDROME

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**Case Report** Lambert-Eaton Myasthenic Syndrome (LEMS) is a neuromuscular disorder caused by antibodies directed to the presynaptic voltage-gated calcium channel. It is often paraneoplastic, most commonly associated with Small Cell Lung Cancer (SCLC).

This report outlines the case of a patient who developed LEMS secondary to pembrolizumab treatment for metastatic melanoma.

An 82 year-old female presented to hospital 1 week after cycle 2 of pembrolizumab treatment for metastatic melanoma.

On examination, she was found to have dysphagia, ocular muscle weakness and generalised weakness (most markedly weakness in hip flexors). Her weakness was fatigable and she