MRI revealed nine T2 periventricular and juxtacortical demyelinating lesions. Two lesions exhibited diffusion restriction indicating acuity. The CSF protein was 3.36 g/L, glucose was 1.4 mmol/L, with 1695 polymorphs per ul and no oligoclonal bands. Neisseria meningitidis was cultured from the CSF. She was treated with dexamethasone and ceftriaxone with complete recovery and without neurological sequalae. A follow up brain MRI performed six months later showed resolution of the lesions with no further clinical or radiological developments.

Conclusion CNS demyelination in the context of bacterial meningitis has not been described before. Given that *N meningitidis* is extracellular, induction of oligodendrocyte cell death by endotoxins, or molecular mimicry to bacterial porins with complement activation are plausible pathomechanisms. In this case, the inciting demyelinating agent evidently disappeared with the pathogen perhaps providing a clue as to what promotes relapsing demyelination.

2461 SERUM INHIBITORY INTERNEURON ANTIBODIES IN PATIENTS WITH MULTIPLE SCLEROSIS

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Introduction There has been recent interest in excitotoxicity as a contributor to neuronal cell death and progression in MS. Histopathology studies imply a specific loss of inhibitory interneurons in the cortex of MS patients, and CSF parvalbumin (exclusive to interneurons) is associated with progression. We investigated MS sera for interneuron antibodies.

Methods Serum from 103 patients with MS was analysed using indirect immunofluorescence on paraformaldehyde fixed frozen rat brain. The cerebellum, brainstem and cortex were viewed using deconvolution microscopy. Neurons were identified by counterstaining with neurofilament, parvalbumin and by morphology.

Results Ten of 103 (9.7%) patients exhibited high titre (>1:640) antibodies against interneurons. The IgG binding pattern was the same in all patients – vesicular intracellular components of basket cells, golgi cells, stellate cells and bipolar interneurons in the cortex and brainstem. Co-binding studies with parvalbumin confirmed inhibitory interneurons as the target and affiliated binding to the renal principal cell, indicated the IgG specificity was the same in positive patients. The female to male ratio was 4:1. The median age was 44 years at the time of antibody detection. At that point there was not a significant difference in EDSS, distribution of lesions or type of MS between antibody positive and negative patients. We will provide 10-year follow up data on the outcomes of these patients in this paper.

Conclusions 10% of patients with MS have inhibitory interneuron antibodies in the blood and work is required to determine whether this leads to neuronal cell loss and cerebral atrophy in MS.

2462 WHERE THERE'S SMOKE, THERE'S FIRE: A SLOW BURN

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Objectives To describe a case of long-standing cryptogenic medication-resistant focal epilepsy diagnosed as FLAIR-hyperintense lesions in anti-MOG associated encephalitis with seizures (FLAMES).

Methods/Background The first cases of MOG antibody disease manifesting as unilateral encephalitis with seizures were described in 2017 (Ogawa et al., 2017). Subsequently, additional cases were used to define a clinical-radiographic syndrome of FLAMES (Budhram et al., 2019).

Results A 40-year-old female presented with sleep-related focal epilepsy in 2010. She started having focal aware seizures in wakefulness with the semiology lateralising to the right hemisphere. After good control, seizures became medication resistant, and a new focal seizure semiology emerged. In 2018 a right occipital cortex lesion was noted on MRI consistent with the new seizure semiology. A CSF examination identified intrathecal oligoclonal bands. Empirical immunotherapy had a modest effect. Worsening seizure frequency in 2021 led to inpatient evaluation. Over 200 right occipital onset focal aware seizures were recorded. A repeat MRI of the Brain identified multiple new regions of cortical FLAIR hyperintensity in the right hemisphere that corresponded to regions of increased glucose metabolism on FDG PET imaging. A brain biopsy showed an inflammatory process with lymphocytes densely aggregated around leptomeningeal vessels. CSF testing was positive for MOG IgG.

Conclusion While most FLAMES cases reported are subacute presentations, this case demonstrates FLAMES may also present as medication-resistant cryptogenic focal epilepsy with a chronic progressive course.