kappa paraprotein, leading to a new diagnosis of monoclonal gammopathy of undetermined significance (MGUS). IgLON5 autoimmunity was considered the likely explanation for the peripheral neuropathy, as sural nerve biopsy findings were not typical for MGUS-related neuropathy. He received IVIg, oral prednisolone, plasma exchange and Rituximab. During follow-up, he progressed to multiple myeloma and commenced lenalidomide and dexamethasone.

Conclusion Our two cases and the few published reports suggest an association of peripheral neuropathy and IgLON5 autoimmunity. We recommend cases of IgLON5 autoimmunity undergo routine neurophysiological studies.

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082 FULMINANT ADEM MIMICKING A GLIAL TUMOUR

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10.1136/bmjno-2021-ANZAN.82

Introduction We describe an atypical case of fulminant acute disseminated encephalomyelitis (ADEM).

Case A 47 year-old Southeast Asian lady presented after developing headache, aphasia and right hemiparesis over four hours, preceded by dry cough for one week and fevers for two days. CT brain noted vasogenic oedema without enhancement in the left frontoparietal lobe, midline shift and incidental upper lobe consolidation and calcified hilar lymph nodes on CT chest. A provisional diagnosis of cerebral tuberculosis was made. MRI brain noted gross mass effect and T2 hyperintensity localised to the white matter, crossing the midline and extending directly to the pons without significant restricted diffusion. Ill-defined enhancement was noted without tuberculomas or leptomeningeal enhancement. MRI spine was unremarkable, as were extensive tests for infectious aetiologies on serum, sputum and CSF. A glial tumour was suspected; FDG-PET-CT did not show regions of increased metabolism. As the patient rapidly deteriorated, empirical corticosteroids, plasmapheresis and IVIg were commenced just prior to decompressive craniectomy and biopsy four days post-presentation. The biopsy demonstrated reactive astrocytosis and perivascular macrophages localised to the white matter, as well as perivascular and perivenular demyelination consistent with ADEM. Absence of a significant lymphocytic infiltrate may have been influenced by the short time to biopsy. The patient made a remarkable recovery following cyclophosphamide, achieving independence in mobility and driving within two months.

Conclusion Atypical features of fulminant ADEM highlight the need for a high index of clinical suspicion and early institution of aggressive immunosuppressive therapy for a favourable outcome.

083 AN ATYPICAL CASE OF IDIOPATHIC INTRACRANIAL HYPERTENSION

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10.1136/bmjno-2021-ANZAN.83

Idiopathic Intracranial Hypertension (IIH) is an increasingly common condition that usually presents with younger obese female patients. Studies report between 74-94% of patients with a BMI>30,[1–5]95% with ages under 50 [(7)] and over 87-91% of the patients being overwhelmingly females.[8 9]

In this case report, we present a 66-year-old man with a BMI of 24.7 kg/m^2 who was referred by his ophthalmologist with bilateral papilledema on the ophthalmic examination and OCTs. The 30-2 Humphrey Visual Field testing showed significant loss of his inferior field on the right side. There were also early field losses noted on the left side. Lumbar puncture showed a borderline elevated CSF opening pressure of 25 cmH_2O. Initial and subsequent MRI brain and orbits have shown constellation of findings consistent with idiopathic intracranial hypertension. Extensive investigations were carried out to identify any secondary cause. These included CT venogram, CT neck, chest abdomen and pelvis, serum and CSF testing for inflammatory/autoimmune, paraneoplastic, infectious and metabolic causes. His non-compliance with Acetazolamide led to clinical deterioration and optic atrophy on the right side. After 2 years of the onset, the patient is clinically stable on 250 mg TDS of Acetazolamide with normal CSF opening pressure on repeat testing recently.

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