there was severe weakness of right elbow, wrist and finger extension with an anaesthetic patch over the anatomical snuff box. Ultrasonography showed fascicular oedema of the radial nerve in the upper arm. Nerve conduction studies confirmed an acute axonotmetic radial neuropathy at the spiral groove. The patient was referred for hand therapy and at 4 months regained most of the function in her hand, with some mild persistent sensory impairment.

Conclusions Cryolipolysis is a cosmetic treatment that aims to locally reduce subcutaneous fat. The procedure is performed using a vacuum applicator to cool the selected area to temperatures as low as -9 degrees Celsius. Peripheral neuropathies following the procedure have been rarely described[1] but, to our knowledge, this is the first report of an acute neuropathy developing during the procedure. The causative mechanisms of cryolipolysis-induced nerve injury in this case were likely due to nerve compression related to local oedema and thermal effect on the radial nerve.

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093 CJD AND MOTOR NEURON DISEASE: A GROWING ASSOCIATION

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Introduction Amyotrophy in Creutzfeldt-Jakob Disease (CJD) is rarely a conspicuous clinical finding. The overlap of CJD (a prionopathy) and motor neuron disease is reported in the literature but it remains to be established whether the neuronopathy is an integral part of CJD presentation, or, whether this represents a distinct variety. Our case describes a male patient with clincopathological diagnosis of sporadic CJD along with evidence of motor neuronopathy on nerve conduction studies.

Case Summary At presentation to our neurology service, the patient was a 72-year-old male, living at home with his wife. He was initially referred for progressive short-term memory loss, personality change, and gait disturbance. On review, it was noted that in addition to gait and limb ataxia, and cognitive impairment, he demonstrated prominent generalised fasciculation. Nerve conduction and electromyography studies showed normal nerve conduction but fasciculations in proximal and distal muscle groups of the left upper and lower limbs, in keeping with a motor neuronopathy. CSF 14-3-3 and EEG provided little bearing. MRI demonstrated progressive T2-hyperintense, diffusion-restricted lesions in the bilateral basal ganglia, thalami and medial frontal cortices consistent with CJD. Post-mortem examination demonstrated spongiform encephalopathy and immunohistological staining (12F10) in-keeping with diagnosis of CJD.

Conclusion In our patient, the combination of clinical and neurophysiologic features of motor neuron disease and a confirmed diagnosis of Creutzfeldt-Jakob Disease raises the vexed question of whether this represents a distinct overlap syndrome or an infrequent manifestation of the same pathology. Further research is required to establish this.

094 NOVEL *NOTCH1* VARIANT IN A PATIENT WITH SPONTANEOUS INTERNAL CAROTID ARTERY DISSECTION

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Introduction We report a case of spontaneous left internal carotid artery (ICA) dissection associated with a novel *NOTCH1* variant.

Case A 43-year-old lady presented with a 3-day history of severe headache and transient expressive dysphasia. There was no history of preceding trauma. CT brain (CTB) and carotid angiography (CTA) demonstrated small areas of established subcortical infarction with occlusion of the M1-segment of the left middle cerebral artery (LMCA). CT cerebral perfusion (CTP) displayed a region of increased mean transit time and cerebral blood volume consistent with a large ischaemic penumbra.

Digital subtraction angiography confirmed an occlusion of the LMCA with luminal irregularity of the supraclinoid ICA suggestive of arterial dissection. An intracranial stent was deployed from the superior M2-division of the LMCA to the cavernous ICA. Progress CTA and CTP demonstrated reperfusion of the LMCA territory.

Six-months later, she remains well with no recurrence of symptoms or detectable neurological signs. Targeted gene panel demonstrated a novel heterozygous missense variant (c.56C>T;p.Ala19Val) in exon-1 of the *NOTCH1* gene. Segregation testing demonstrated an identical variant in her mother.

Conclusion Spontaneous intracranial ICA dissection is a rare condition described mostly in single case reports. Mortality rates have been reported of up to 75%. The NOTCH-signalling pathway is involved in the embryonic development of arterial endothelium. *NOTCH1* variants have been associated with autosomal dominant bicuspid aortic valve aortopathy, and rarely in extracranial arterial dissection. To our knowledge; this is the first reported case of intracranial dissection where a previously undescribed *NOTCH1* variant is identified.

095 'NO END IN SIGHT', MANAGEMENT DILEMMA OF REFRACTORY MOG ANTIBODY POSITIVE OPTIC NEURITIS

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Background The patient is a 38-year-old lady who presented with impaired visual acuity in her right eye which was accompanied with pain on extra-ocular movements. Her symptoms initially resolved with high dose steroid therapy. This is on a background of eosinophilic asthma which is refractory to maximal inhaler therapy and IL-5 monoclonal antibody therapy.

Methods/Results The patient had unremarkable blood results and inflammatory makers and a normal CSF study. She was subsequently found to be myelin oligodendrocyte glycoprotein

(MOG) antibody positive but negative for (Aquaporin -4) AQP-4 antibodies. Radiological findings on MRI of the brain and spine was suggestive of multiple sclerosis (MS). Whilst receiving intravenous immunoglobulin (IVIG) as a bridging therapy to Multiple Sclerosis (MS) modifying therapy, the patient developed severe adverse reaction to IVIG with desquamation of her hands and legs. Furthermore, she did not achieve satisfactory disease control with Ocrelizumab. Has cushingoid features secondary to long term high dose steroid therapy.

Conclusions To highlight the challenges and difficulty in managing a patient with refractory MOG antibody optic neuritis and its impact on the quality of life of the patient. To demonstrate the gap in available clinical evidence in the management of recurrent MOG antibody positive optic neuritis. To initiate an open discussion regarding the treatment of these challenging group of patients

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096 INTRAVENTRICULAR MIGRATION OF INTRAOCULAR SILICONE OIL

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Introduction We report a case of intraventricular migration of intraocular silicone oil.

Case A 53-year-old male presented with a decreased level of consciousness. His past medical history included bilateral retinal detachment, atrial fibrillation and ischaemic stroke. Initial blood tests and chest X-ray were consistent with a community acquire pneumonia. He was started on a 5-day course of IV ceftriaxone and azithromycin with improvement in his symptoms.

A CT brain was performed upon presentation given his conscious state. This demonstrated a high density in the right globe with tracking along the optic nerve and bilateral homogenous isointense spherical masses in the frontal horns of the lateral ventricles. Imaging was initially reported as bilateral vitreous haemorrhages, an optic chiasm lesion and intraventricular haemorrhage. Follow-up MRI brain confirmed the biventricular masses with evidence of a chemical shift artefact on T1 and T2 sequences. There was disappearance on the masses on fat-suppressed T1-sequences. Images were reviewed in the combined neurology-neuroradiology meeting with the diagnosis made of cerebral migration of silicone oil.

Conclusion Intraventricular migration is a rarely reported complication following intraocular silicone endotamponade. The precise mechanism of migration is unknown. As silicone appears hyperintense on CT, it can mimic a haemorrhage or mass. Cases of misidentification of intraventricular silicone have resulted in protracted hospital admissions and surgical biopsy. Typical MRI findings and a comprehensive clinical history allow for accurate identification. Early and accurate recognition is critical to avoiding unnecessary investigation and patient morbidity.

097 CASE REPORT: A ROLE FOR OCRELIZUMAB FOR MULTIPLE SCLEROSIS WITH OCULAR SARCOIDOSIS?

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Introduction Multiple sclerosis causes a variety of clinical neurological features which may overlap with sarcoidosis with central nervous system involvement. Rarely, concurrent diagnosis of MS and sarcoidosis in an individual has been reported. Currently, best management strategy for these individuals with this dual pathology remains unknown.

Case A 54-year-old man with a 14-year history of isolated ocular sarcoidosis, as well as type 2 diabetes mellitus, hypertension, and dyslipidemia, presented with ongoing right-hand paresthesia. Gadolinium-enhanced neuroaxis MRI imaging revealed comparatively stable non-enhancing appearances of previously known numerous punctate supratentorial subcortical white matter hyperintensities, and a new non-enhancing right ventrolateral C3/4 cervical spinal cord hyperintense lesion. The radiological pattern in combination with positive oligoclonal bands and elevated protein levels on cerebrospinal fluid testing, with normal serum ACE level, was thought to favour a diagnosis of multiple sclerosis (with McDonald criteria fulfillment), rather than neurosarcoidosis. In addition to his sarcoidosis treatment comprising prednisolone and hydroxychloroquine (later changed to mycophenolate), Ocrelizumab was selected as initial disease modifying therapy for his new relapsing remitting multiple sclerosis, due to postulated benefits for his sarcoidosis as well based on ocrelizumab's similarity to rituximab, an established treatment option in refractory sarcoidosis.

Conclusion This case highlights several key issues:

- Recognising the possibility of co–occurrence of CNS demyelinating disease in sarcoidosis
- The diagnostic challenge of ascertaining new neurological changes in sarcoidosis
- The challenge of managing concurrent inflammatory conditions of distinctly separate aetiology

We propose that Ocrelizumab has potential utility as an option in streamlining therapeutic management of this dual pathology.

098 STROKE-LIKE MIGRAINE ATTACKS AFTER RADIATION THERAPY: BE SMART AND ORDER AN EEG

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Background Seizures may be a feature of SMART (stroke-like migraine attacks after radiation therapy) syndrome but their role in the pathophysiology and clinical presentation is unclear.