in Victoria (13.0%). Oral DMTs were most common in NSW (44.0%) and least common in Tasmania (29.3%). Infusion DMTs were most common in Tasmania (47.4%) and least common in ACT (27.7%).

Conclusion We have demonstrated marked variation in DMT use in different Australian states and territories during the study period. The use of DMTs in Australia has evolved over time,[3] but differences between states/territories remain unexplained. We plan to investigate the observed clinical variation in future studies.

REFERENCES

1. Department of Health. Pharmaceutical Benefits Scheme. Canberra: Australian Government; 2021. https://www.pbs.gov.au/(accessed 19 Feb 2021).
2. WHO Collaborating Centre for Drug Statistics Methodology. ATC-DDD Toolkit: Defined Daily Dose (DDD). Geneva: World Health Organization; 2021. https://www.who.int/tools/atc-ddd-toolkit/about-ddd (accessed 19 Feb 2021).
3. Goudarzi MH, *et al*. Disease modifying therapies for relapsing-remitting multiple sclerosis: use and costs in Australia (1996-2019). *Mult Scler Relat Disord* 2021;**50**:102835.

090 PAINLESS PROGRESSIVE MONONEURITIS MULTIPLEX SECONDARY TO AML ASSOCIATED NEUROLEUKAEMIOSIS

[1]Nimalan Harinesan, [1]Rajiv Wijesinghe, [2,3]Nicole Wong-Doo, [1,3]Katrina Morris, [1]James D Triplett. *[1]Neurology and Neurophysiology Department, Concord Repatriation General Hospital, Sydney, NSW, Australia; [2]Haematology Department, Concord Repatriation General Hospital, Sydney, NSW, Australia; [3]Concord Clinical School, The University of Sydney, Sydney, NSW, Australia*

10.1136/bmjno-2021-ANZAN.90

Objectives To report the clinical history, imaging and neurophysiology findings of a case of mononeuritis multiplex caused by acute myeloid leukemia (AML) neuroleukaemiosis

Case A 58-year-old male presented with painless and progressive mononeuropathies after completing high dose cytarabine consolidation treatment for AML. Nine days following chemotherapy a right third nerve palsy developed followed by right 5th, right 7th, left 3rd and left radial, ulnar and peroneal neuropathies.

Serial MRI and PET imaging was unremarkable and 2 cerebrospinal fluid (CSF) were normal. Nerve conduction studies demonstrated abnormal right blink responses, a peroneal neuropathy and evidence of conduction block at a non-compressible site within the left ulnar nerve, however nerve ultrasound did not demonstrate any causative lesion.

Differentials considered included opportunistic fungal infections and a paraneoplastic neuropathy. A third CSF sample performed on day 24 demonstrated myeloblasts, consistent with central nervous system leukaemic infiltration. A diagnosis of neuroleukemiosis was made and intrathecal chemotherapy (initial methotrexate [MTX] and cytarabine, followed by alternating MTX/cytarabine twice a week) plus systemic chemotherapy (fludarabine, cytarabine, idarubicin) and granulocyte-colony stimulating factor was commenced resulting in partial resolution of pre-treatment symptoms.

Conclusions Progressive neuropathies in patients with leukemia are rarely reported and can be diagnostically challenging. Mononeuritis multiplex associated with AML may be painless and focused imaging may fail to demonstrate significant abnormalities. A high index of clinical suspicion is required as the differential diagnoses of neuroleukaemiosis is broad including paraneoplastic syndromes, infection and inflammatory conditions. As in this instance multiple CSF examinations maybe required to confirm its diagnosis.

091 THE IMPACT OF DEVICE-ASSISTED THERAPY INITIATION ON THE GUT MICROBIOME IN PARKINSON'S DISEASE

[1,2]Michal Lubomski, [3]Xiangnan Xu, [4]Andrew J Holmes, [3]Jean Yang, [1,2]Carolyn M Sue, [1]Ryan L Davis. *[1]Department of Neurogenetics, Kolling Institute, University of Sydney and Royal North Shore Hospital, Sydney, NSW, Australia; [2]Neurology Department, Royal North Shore Hospital, St Leonards, NSW, Australia; [3]School of Mathematics and Statistics. Sydney Precision Bioinformatics, University of Sydney, Camperdown, NSW, Australia; [4]School of Life and Environmental Sciences, The Charles Perkins Centre, University of Sydney, Sydney, NSW, Australia*

10.1136/bmjno-2021-ANZAN.91

Objectives Several studies have evaluated the impact of oral medication on the gut microbiome (GM) in Parkinson's disease (PD). However, the impact of PD device-assisted therapies (DAT) on the GM remains to be investigated. We profiled acute temporal GM stability around the initiation of PD DAT.

Methods The GM of 21 PD patients initiating either Deep Brain Stimulation (DBS) or levodopa-carbidopa intestinal gel (LCIG) were compared to 10 spousal healthy control (HC) subjects. 16S amplicon sequencing of the V3-V4 region of stool bacterial DNA was used to compare temporal GM stability between groups and with clinical outcome measures, including disease alternations relative to therapy initiation. GM response to therapy in the PD group was assessed by comparing pre-therapy (-2 and 0 weeks) with post-therapy initiation timepoints (+2 and +4 weeks) and HCs at baseline (0 weeks).

Results Altered GM compositions were noted between the PD and HC groups at various taxonomic levels, including specific differences for DBS and LCIG therapies. Beta diversity changes were also identified across the 4 week post-treatment initiation period, implying a therapy-effect on the GM.

Conclusions We present the first acute longitudinal assessment of GM response to PD DAT. The pre-treatment PD-specific GM (consistent with previous studies) was altered following DAT initiation, indicating DATs have a modulatory impact on the GM in PD.

092 CRYOLIPOLYSIS-INDUCED RADIAL MONONEUROPATHY

Nimalan Harinesan, Rajiv Wijesinghe, Michael Hayes, James D Triplett. *Neurology and Neurophysiology Department, Concord Repatriation General Hospital, Sydney, NSW, Australia*

10.1136/bmjno-2021-ANZAN.92

Objectives To report an association between cryolipolysis or 'fat-freezing' in the upper arm and development of an acute radial neuropathy likely secondary to a combination of thermal and pressure effects.

Results A 31-year-old female presented with an acute right wrist drop that occurred following a cryolipolysis procedure to the upper right arm. Paraesthesia and numbness occurred towards the end of a 40-minute cryolipolysis procedure, with weakness reported within 24 hours. Examination of the arm 1 week following symptom onset revealed a significant ecchymosis at application site of the fat freezing device. In addition,

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.

REFERENCE

1. Jong Gyu Baek, Jung A Park, Jung Im Seok. Radial neuropathy after cryolipolysis. *Journal of the Korean Neurological Association* 2017;**35**:1, 30–32.

093 CJD AND MOTOR NEURON DISEASE: A GROWING ASSOCIATION

Adam P Cooper, Wilson Vallat. *Neurology Department, Lyell MEwin Hospital, Adelaide, SA, Australia*

10.1136/bmjno-2021-ANZAN.93

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

[1,2,3]Leon Edwards, [1,2,3]Christopher Blair, [4]Sulekha Rajagopalan, [4]Madhura Bakshi, [5,6,7]Nathan Manning, [1,2,3]Alan McDougall. *[1]Neurology and Neurophysiology, Liverpool Hospital, Liverpool, NSW, Australia; [2]Ingham Institute for Applied Medical Research, Ingham Institute, Liverpool, NSW, Australia; [3]South Western Sydney Clinical school, University of New South Wales, Liverpool, NSW, Australia; [4]Clinical genetics, Liverpool Hospital, Liverpool, NSW, Australia; [5]Institute of neurological sciences, Prince of Wales Hospital, Randwick, NSW, Australia; [6]Neurointervention, Liverpool Hospital, Liverpool, NSW, Australia; [7]The Florey institute of neurosciences, Melbourne, VIC, Australia*

10.1136/bmjno-2021-ANZAN.94

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

Samuel SK Kwok, James JH Hughes, Dark LD Lisa. *Tamworth Hospital, Oxley Vale, NSW, Australia*

10.1136/bmjno-2021-ANZAN.95

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