therefore performed. Both the nerve and muscle biopsies showed noncaseating granulomatous inflammation consistent with neurosarcoidosis. An FDG-PET scan did not show sarcoidosis involving other organs.

Conclusions This case shows that sarcoidosis can be limited to just the nerves and muscles without clear systemic manifestations. It also provides a case for neurosarcoidosis to be considered as a differential in future presentations of subacute onset polyneuropathy.

REFERENCES

1. Kidd DP. Neurosarcoidosis: clinical manifestations, investigation and treatment. *Practical Neurology*. 2020;**20**(3):199. doi:10.1136/practneurol-2019-002349
2. Cohen Aubart F, Abbara S, Maisonobe T, *et al*. Symptomatic muscular sarcoidosis. *Neurology – Neuroimmunology Neuroinflammation*. 2018;**5**(3):e452. doi:10.1212/NXI.0000000000000452

2403 SEVERE AXONAL NEUROPATHY FOLLOWING NITROUS OXIDE MISUSE

[1,2,3,4]Stephanie L Barnes*, [2,5]Rajiv Wijesinghe, [2,6]Jessie Chen, [2,3]James R Burrell. *[1]Department of Neurology, Hornsby Ku-ring-gai Hospital, Sydney, NSW, Australia; [2]Department of Neurology, Concord Repatriation General Hospital, Sydney, NSW, Australia; [3]University of Sydney, Sydney, NSW, Australia; [4]University of Notre Dame, Sydney, NSW, Australia; [5]Department of Neurology, The Sutherland Hospital, Sydney, NSW, Australia; [6]Department of Neurology, Austin Hospital, Melbourne, VIC, Australia*

10.1136/bmjno-2022-ANZAN.146

Background Nitrous oxide misuse causes severe neurotoxicity due primarily to functional vitamin B12 (cobalamin) deficiency. Peripheral neuropathy is a less frequent presentation than subacute combined degeneration of the cord (SCDC).

Methods Review of a patient with severe axonal neuropathy secondary to nitrous oxide misuse.

Results An 18-year-old Chinese male presented with subacute bilateral foot drop, distal numbness and neuropathic pain. There was transient finger weakness and no proximal weakness, symptoms of CNS dysfunction or constitutional symptoms. He was misusing nitrous oxide over several months and self-initiated oral cobalamin supplementation.

On examination, there was mild abductor pollicis brevis weakness bilaterally and bilateral foot drop (2/5 power on dorsiflexion, ankle eversion and great toe extension). Ankle jerks were absent. Plantar responses were flexor. Coordination was normal. Sensation was reduced distally. Gait was high-stepping and tandem was unsteady. Romberg's could not be performed.

Nerve conduction studies demonstrated severe axonal motor neuropathy with active denervation change on electromyography. MR spine showed subtle T2/FLAIR dorsal column hyperintensity. Somatosensory evoked potentials revealed abnormal large-fibre sensory conduction rostral to the cauda equina. Bloodwork demonstrated mild macrocytosis, low red-cell folate and normal cobalamin. Neuropathy/vasculitic screens and CSF analysis were unremarkable.

He received high-dose parenteral cobalamin and supportive care. He did not return for follow-up.

Conclusions Nitrous oxide misuse can cause severe axonal neuropathy, in addition to the more frequent SCDC, and can be the predominant clinical phenotype, even in the setting of oral supplementation. This syndrome is profoundly disabling but potentially reversible with abstinence and high-dose cobalamin replacement.

2404 THE TEMPORAL DISTRIBUTION OF INTERICTAL DISCHARGES IN JUVENILE ABSENCE EPILEPSY (JAE) SHOW CYCLES DURING SLEEP WITH BURSTS SUGGESTING COUPLING TO A SLEEP-PHASE GENERATOR

[1]Peter Hingston*, [2]Anita Dharan*, [1,3]Ewan Nurse, [1,4]Udaya Seneviratne, [1]Wendyl J D'Souza. *[1]Department of Medicine, St Vincent's Hospital, The University of Melbourne, Fitzroy/Melbourne, VIC, Australia; [2]Melbourne School of Psychological Sciences, The University of Melbourne, Parkville, VIC, Australia; [3]Seer Medical, Melbourne, VIC, Australia; [4]Department of Medicine, School of Clinical Sciences at Monash Health, Monash University, Clayton, VIC, Australia*

10.1136/bmjno-2022-ANZAN.147

Objectives Idiopathic Generalised Epilepsy is not a benign condition with increasing evidence of enduring cognitive deficits beyond seizures. We aimed to describe the temporal distribution, duration, and relationship to sleep of interictal discharges (IEDs) using outpatient ambulatory 24-hour EEG recordings (aEEG) in juvenile absence epilepsy (JAE).

Methods We retrospectively identified 15 patients with JAE undergoing treatment adjustment with at least three aEEGs between 2012–2020. JAE was classified with onset of first or predominant absence seizures after age 9 years. We used a published automated detection algorithm to assist aEEG review and recorded the timing and length of IEDs. Following training, PH/AD independently marked IEDs with any uncertain or discordant IEDs resolved by WD. For each individual recording, the timing and length of the discharges was plotted against 24-hour clock.

Results 15 patients (onset age 9 to 16 years), had a total of 14701 IEDs (median 104; IQR 11 – 403). 9917 (67.5%) IEDs occurred between 22:00 and 07:00 (individual aEEG median 63%; IQR 49.8% – 92.3%). IEDs show an overall pattern of clustered discharges during sleep compared to a more sporadic frequency in wakefulness. In addition, during sleep IEDs oscillate between high frequency peaks and quiescent periods throughout the night.

Conclusion JAE demonstrates a cyclical pattern in the distribution of IEDs with two-thirds occurring during sleep. The oscillating pattern of IEDs during sleep has not been previously reported in humans and suggests coupling to a sleep phase generator, a critical time for memory encoding.

2407 AN INTERESTING CASE OF ATYPICAL PARKINSONISM

Grace Oh*, Beatriz Romero, Jan Schepel, Allan Thomas, Matthew Phillips. *Neurology, Waikato DHB, Hamilton, New Zealand*

10.1136/bmjno-2022-ANZAN.148

Functional movement disorders (FMD) are thought to account for around 10% of new patients at large movement disorder clinics. Among patients with FMD, only 5% have functional parkinsonism. Functional progressive supranuclear palsy (PSP) likely accounts for a very small proportion of these patients. Despite a literature search, I was not able to find any case description of functional PSP. I report an interesting and rare case of functional parkinsonism very closely mimicking PSP.

A 70-year-old NZ European man presented to the Emergency Department following a collapse and progression of symptoms related to his previously diagnosed PSP, making him

BMJ Neurology Open: first published as 10.1136/bmjno-2022-ANZAN.148 on 2 February 2024. Downloaded from https://neurologyopen.bmj.com on 10 October 2024 by guest. Protected by copyright.

unable to manage at home. He had frequent falls, shuffling gait, slow movements, right sided limb tremor along with difficulty with eye movements, dysphagia and several collapses. On examination, he had gait and other features of parkinsonism (tremor, bradykinesia, rigidity and postural instability). He also had ophthalmoparesis, suggestive of PSP.

However, there were some incongruencies that prompted for a more detailed examination. This revealed subtle but definite features in keeping with functional parkinsonism. The patient was referred to physiotherapy and cognitive behavioural therapy. He is also awaiting a dopamine transporter imaging.

During my oral presentation, I will be showing videos of this patient's examination findings and present a literature review. This case demonstrates how easy it is to miss the signs if they are not carefully and actively looked for. It also highlights the importance of challenging and digging deeper when things do not quite fit.

2408 THE FOREST NOT THE TREES: A PRESENTATION OF A CHALLENGING CASE

Grace Oh*, Hannah Higgins, Allan Thomas, Matthew Phillips. *Neurology, Waikato DHB, Hamilton, New Zealand*

10.1136/bmjno-2022-ANZAN.149

A 37-years-old NZ European man presented with a history of five years of progressive neurological deficits. These included dysarthria, hyper-salivation, vivid dreams, sleep disordered breathing, elevated hemi-diaphragm, left upper limb paraesthesia, syncopal episodes, lethargy, irregular bowels, difficulty passing urine, difficulty with temperature regulation, locked and painful jaw, anxiety, reduced sleep and headaches. On examination, he had mild dysarthria, wasted tongue with deviation to left and weakness on the left. The reflexes were brisk in the lower limbs with an up-going plantar response on the left. MRI revealed an ill-defined heterogeneous enhancement of the medulla extending to the right cerebellar peduncle. The patient underwent extensive work up and treatment trial with steroids with a working diagnosis of Neuro-sarcoidosis. He developed several further symptoms of tremors and further paraesthesia over the next three months. The patient had a sudden death and the final diagnosis of Alexander disease was revealed in his autopsy.

Retrospectively, the symptoms collectively clearly point to a neurological disorder, but during the five years of the disease progression, his complaints were approached individually as separate issues by multiple specialities. Due to this, despite the numerous red flags, these were unrecognised and the patient presented to Neurology with an advanced illness. This is a valuable case for learning and it reminds us how an eye for detail and careful observation in history and examination is critical, especially for patients presenting as a diagnostic challenge. This very fact is also what makes Neurology such a fascinating and intriguing specialty.

2409 AN ATYPICAL PRESENTATION OF AUTOIMMUNE GLIAL FIBRILLARY ACIDIC PROTEIN (GFAP) ASTROCYTOPATHY WITH EXTENSIVE SPINAL CORD DISEASE WITHOUT BRAIN INVOLVEMENT

Grace Oh*, Allan Thomas, Jan Schepel, Matthew Phillips. *Neurology, Waikato DHB, Hamilton, New Zealand*

10.1136/bmjno-2022-ANZAN.150

Autoimmune glial fibrillary acidic protein (GFAP) astrocytopathy is a novel central nervous system disorder that presents with one or more of meningitis, encephalitis and myelitis. 95% of patients present with either meningoencephalitis or meningo-encephalo-myelitis. Myelitis without cerebral involvement is rare and thought to represent only about 5% or less of cases.

We report, along with review of literature, a rare presentation of autoimmune GFAP astrocytopathy, who presented with myelitis without encephalitis and experienced initial misdiagnosis and a delay in the diagnosis.

A 25-year-old male, kiwi packer, migrant from India presented with meningism (fevers, headache, neck stiffness, photophobia, nausea and vomiting) with subsequent development of urinary retention and progressive weakness and sensory change in the limbs. CSF examination revealed the GFAP-IgG with significantly elevated lymphocytes and protein. Magnetic resonance imaging revealed a rare finding of longitudinally extensive myelitis extending from the C2 to T11 level without any brain lesions. He had significantly elevated lymphocytes and protein in the CSF with the presence of GFAP-IgG. Interestingly, He was initially diagnosed with viral meningitis and had multiple re-presentations to the hospital with ongoing deterioration in clinical status despite antibiotic and antiviral therapy. This led to further investigations and immunotherapy (IV steroids and plasma exchange) with good recovery.

This is a valuable case for learning, which reports an uncommon presentation of a rare disorder. It highlights the importance of detailed history and examination, having broad differentials in mind and early re-evaluation of diagnosis when things do not go as planned.

2413 DOES SERUM NEUROFILAMENT LIGHT CHAIN LEVEL CONTRIBUTE TO THE PREDICTION OF TREATMENT RESPONSE IN MULTIPLE SCLEROSIS?

[1]Nahid Moradi*, [1]Sifat Sharmin, [1]Charles B Malpas, [2]Jens Kuhle, [2]Pascal Benkert, [2]David Leppert, [3]Eva Havrdová, [3]Dana Horáková, [3]Pavlina Kleinová, [3]Tomas Uher, [4]Jan Hillert, [4]Tomas Olsson, [4]Ali Manouchehrinia, [5]Bruce V Taylor, [6]Michael H Barnett, [7]Trevor J Kilpatrick, [1]Tomas Kalincik. *[1]CORe, Neurology, Royal Melbourne Hospital, University of Melbourne, Parkville, VIC, Australia*; *[2]Neurologic Clinic and Policlinic, MS Centre and Research Centre for Clinical Neuroimmunology and Neuroscience, University Hospital Basel, University of Basel, Basel, Switzerland*; *[3]First Faculty of Medicine, General Faculty Hospital, Charles University, Prague, Czech Republic*; *[4]Department of Clinical Neuroscience, Karolinska Institute, Stockholm, Sweden*; *[5]Menzies Institute for Medical Research, University of Tasmania, Hobart, TAS, Australia*; *[6]Brain and Mind Centre, The University of Sydney, Camperdown, NSW, Australia*; *[7]Melbourne Neuroscience Institute, University of Melbourne, Parkville, VIC, Australia*

10.1136/bmjno-2022-ANZAN.151

BMJ Neurology Open: first published as 10.1136/bmjno-2022-ANZAN.148 on 2 February 2024. Downloaded from https://neurologyopen.bmj.com on 10 October 2024 by guest. Protected by copyright.