

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.

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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. ¹Department of Neurology, Hornsby Ku-ring-gai Hospital, Sydney, NSW, Australia; ²Department of Neurology, Concord Repatriation General Hospital, Sydney, NSW, Australia; ³University of Sydney, Sydney, NSW, Australia; ⁴University of Notre Dame, Sydney, NSW, Australia; ⁵Department of Neurology, The Sutherland Hospital, Sydney, NSW, Australia; ⁶Department of Neurology, Austin Hospital, Melbourne, VIC, Australia

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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

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

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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*

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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