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**AN ASTRA-NOMICAL HEADACHE**

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**Introduction**

We present a case of myelin-oligodendrocyte glycoprotein antibody disease (MOGAD) requiring long-term immunosuppression triggered by a dose of the AstraZeneca COVID-19 vaccination. Relapsing MOGAD is thus far an unknown complication of COVID-19 vaccination.

**Case Description:** A 58-year-old lady developed headache, nausea, dizziness, facial numbness, ataxia and slurred speech 8 days after the COVID-19 AstraZeneca vaccination. Her imaging showed acute disseminated encephalomyelitis (ADEM) with a white matter lesion in the left cerebellum and bilateral smaller lesions. Her cerebrospinal fluid showed 38 white cells and elevated protein. She initially responded well to steroids, however relapsed with optic neuritis 7 months later, requiring long-term immunosuppression with mycophenolate mofetil.

**Discussion**

Although there have been some case reports of MOG following COVID-19 infection, to our knowledge this is only the second reported case of MOG following vaccination against COVID-19, and the first such case to require long-term immunosuppression. The other reported case also occurred following the COVID-19 AstraZeneca vaccine, and also presented with ADEM. This is in contrast to reported cases of MOG following COVID-19 infection, where adults mostly presented with optic neuritis. We wanted to highlight the possibility of this vaccine-related neurological complication occurring, particularly in the context of potentially frequent ongoing COVID-19 booster vaccinations.

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**CHANGING PHENOTYPES, A SPECTRUM OVER 10 YEARS**

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Our patient first presented with progressive unsteadiness and slurred speech at the age of 67 years. The initial working diagnosis was progressive ataxia. There were minimal cerebellar changes on MRI head and extensive laboratory tests including common spinocerebellar ataxia screen was negative. He subsequently developed distal muscle wasting and weakness, and pathologically brisk reflexes, with normal CK levels. Motor neuron disease (MND) was then clinically suspected, however, further neurophysiological studies and a nerve biopsy revealed changes consistent with an axonal neuropathy. Generalised muscle wasting, bilateral scapular winging, eyelid ptosis and complex ophthalmoplegia were identified 8 days after initial clinic review. The complex evolving neurological phenotype prompted a muscle biopsy to investigate for mitochondrial disease. Whilst some evidence of mitochondrial dysfunction was identified, the mitochondrial DNA maintenance nuclear gene panel was negative. He was enrolled to the 100k genome project which revealed a heterozygous KIF5A pathogenic variant. Mutations in KIF5A are associated with a wide phenotypic spectrum including CMT neuropathy, hereditary spastic paraplegia and MND-like syndrome. Our case highlights the diagnostic conundrum of evolving neurological manifestations of KIF5A disease that demonstrates overlapping cardinal features with mitochondrial disease.

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**CASE PRESENTATION COMPETITION |5 BACK TO BASICS**

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10.1136/jnnp-2022-abn2.43

Serotonin syndrome associated with clozapine withdrawal and concurrent selective serotonin reuptake inhibitor (SSRI) use has previously been reported.

A 56-year-old female with schizophrenia was admitted for pyrexia, rigidity, and altered mental state after her second dose of clozapine restart. She had discontinued her long-term clozapine 2 weeks prior. She developed ventilatory failure, reduced consciousness, eye deviation, and worsening rigidity, requiring ICU support. Examination showed a right upper motor neurone syndrome with absent ankle reflexes.

She had raised inflammatory markers and creatine kinase. Serum neuropathy, encephalitis screen, and COVID PCR were negative. Respiratory investigations were unfruitful. MRI head and spine did not show brain or cord signal change to correlate to signs. Lumbar puncture showed a quiet CSF, negative culture, viral PCR, and encephalitis antibodies. EEG showed bihemispheric background slowing.

Despite clinical improvement, repeat examination showed persistent signs. She was diagnosed with serotonin syndrome after developing a bilateral tremor. Treatment with cyproheptadine correlated with an improvement in her signs, cognitive state, and EEG.

Serotonin syndrome can present with reversible neuromuscular signs. With clozapine withdrawal, it can require a prolonged time course of recovery in contrast with classical serotonin syndrome. Cyproheptadine can cause agranulocytosis and this delays clozapine restart.

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**CRYPTIC CONFUSION**

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Neurotoxicity is often a late consideration, particularly if the neurotoxic agent is given with therapeutic intent, because the poisoning occurs without the diagnostic clues typical of an intentional attack. We present the case of a 76 year old woman with renal failure, who was admitted with confusion and hal- lucinations. Several days previously, she had started Aciclovir because of a vesicular rash. Her condition was attributed to varicella zoster encephalitis and she was switched...
A young male presented with a one week history of left leg weakness and sensory loss. On examination there was severe UMN weakness of the left leg and reduced sensation. An MRI head revealed a large right frontal lobe inflammatory lesion, with two further smaller lesions. He subsequently developed severe weakness in both legs, mild weakness in the left arm and urinary retention. He had brisk reflexes and a sensory level at T8. His MRI spine revealed diffuse long segment cord signal change between C6 and T4 in keeping with neuromyelitis optica so he was treated with high dose then weaning steroids. We considered inflammatory, infective, and neoplastic differentials. His HIV screen was positive, he had a low CD4 count (242 cells/mm3) but also a low HIV viral load which is unusual for HIV 1; HIV 2 PCR was negative. HIV broadened the differential to vacuolar myelopathy, infections of the immunocompromised, and CNS lymphoma. Serum MOG and aquaporin 4 antibodies were negative. CSF testing for infections and haematological malignancies was negative and his viral load was low. He is improving with therapies, steroids and antiretrovirals. Currently the working diagnosis is seronegative neuromyelitis optica spectrum disorder.

## Poster presentations

### 001 AN UNUSUAL CAUSE OF LYMPHOCYTIC MENINGITIS

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A 36-year-old Spanish man, with a background of ketamine use, presented with a 10-day history of severe headache, photophobia, nausea, unsteadiness and slurring of speech. His CT head was normal. CSF white cell count was 151 (lymphocytes), protein 0.7, glucose 2.3 (serum 3.6). He was treated with IV acyclovir for two weeks with no clinical improvement.

On examination he remained distressed by headache and photophobic with severe dysarthria, bidirectional nystagmus and ataxia.

Blood tests were normal or negative including HIV, serology for syphilis, mycoplasma, lyme and brucella, ANA, ENA, dsDNA, ANCA, ACE, LGI1 and NMDA antibodies, anti-Hu, Ri and Yo. IGRA was positive. MRI brain scan was normal. Serial lumbar punctures showed a variable CSF white cell count, with a negative extended virology panel, TB PCR, bacterial cultures, cytology and immunophenotyping.

There was no improvement with pulsed steroids or antimicrobial cover for listeria. After 5 plasma exchanges there was some improvement in his CSF white cell count and headache, but he remained very ataxic.

An extended paraneoplastic panel revealed positive anti-Tr antibodies. This is a well recognised cause of paraneoplastic cerebellar degeneration, normally associated with Hodgkin’s lymphoma. There have been no previous reported cases of anti-Tr antibodies associated with meningitic symptoms.

### 003 A CASE OF SARS-COV-2 OMICRON VARIANT ASSOCIATED ACUTE ENCEPHALOMYELITIS

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10.1136/jnnp-2022-abn2.47

With the ongoing pandemic of SARS-CoV-2 many neurological complications in relation to COVID-19 infection as well as immune-mediated and vaccine-associated phenomena have been described. To our knowledge, there has been no publication of a case of SARS-CoV-2 Omicron variant associated acute encephalomyelitis.

We present a case of a 73-year-old woman with no relevant background history who is otherwise fit and well and fully vaccinated. She suffered from mild COVID symptoms and had a positive PCR test with presumptive Omicron variant on day 2. Five days into her respiratory illness she developed in quick succession sensory disturbances of hands and feet, bilateral asymmetric flaccid leg weakness, and mild arm weakness. She had absent deep tendon reflexes in the legs and diminished deep tendon reflexes in the right arm. MRI of brain and spine showed signal changes in the brainstem, cervical and low thoracic cord in keeping with acute encephalomyelitis. Her CSF showed an inflammatory picture with raised protein of 1.27g/L and no cells. At the time of abstract submission, the patient received treatment with five days of intravenous steroids followed by ongoing plasma exchange and no comment on treatment response can be made at this stage.