2736 ACUTE DISSEMINATED ENCEPHALOMYELITIS AND ACUTE INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY FOLLOWING ASTRA ZENECA COVID19 VACCINATION

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Objectives To describe a fatal case of fulminant acute disseminated encephalomyelitis with concurrent acute inflammatory demyelinating polyneuropathy secondary to Astra Zeneca COVID vaccination.

Background Acute disseminated encephalomyelitis and acute inflammatory demyelinating polyradiculoneuropathy are known uncommon post-vaccination complications. Both concurrently have not been reported secondary to vaccination.

Case A 67-year-old with a background of depression, alcohol misuse and emphysema presented with diplopia, dysarthria, ataxia, and back pain. Astra Zeneca COVID19 vaccination was administered one week prior. The next day he developed acute respiratory failure necessitating intubation.

Examination confirmed severe global weakness and areflexia. The patient was commenced on intravenous immunoglobulin with nerve conduction study confirming a diffuse demyelinating polyneuropathy.

GGT was elevated, and there was a mild thrombocytopenia but renal function, electrolytes, folate, vitamins A, B1, B6, B12, D and E, selenium, copper, zinc, ammonia, and thyroid function tests were normal. ANA, dsDNA, ANCA, AQP4, MOG and anti-ganglioside antibodies were negative.

CSF was inflammatory with an elevated protein of 1.05g/L and leucocytes of 19x10^6/L (90% mononuclear). Extensive microbiological testing was negative. MRI Brain and cord demonstrated florid neural axis white matter centric inflammation consistent with acute disseminated encephalomyelitis, with associated radiculitis of the cauda equina.

The patient deteriorated despite high dose corticosteroids, plasma exchange and intravenous immunoglobulin. He died 3 weeks after presentation. Limited post-mortem assessment of the brain and spinal cord was consistent with catastrophic fulminent acute disseminated encephalomyelitis.

Conclusion We describe the first case of fulminant peripheral and central nervous system demyelination following Astra Zeneca viral vector COVID19 vaccination.

2737 SJOGRENS'S SYNDROME PRESENTING WITH PURE MOTOR TRIGEMINAL NEUROPATHIES

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Objectives To describe a case of Sjogren's syndrome presenting with weight loss secondary to a motor trigeminal neuropathy

Background Trigeminal neuropathy is well-recognised in Sjogren's syndrome; typically a slowly progressive unilateral sensory neuropathy. Pure motor trigeminal neuropathies are rare.

Case Report An 85-year old woman with chronic kidney disease, hypertension, osteoarthritis, sleep apnoea and a pacemaker for sick sinus syndrome presented with two weeks of diplopia and 10kg weight loss over three months.

She was found to have a left trochlear nerve palsy and bilateral optic disc swelling. ESR was elevated upto 110. High dose steroids were prescribed for putative giant cell arteritis, however subsequent temporal artery biopsy was negative.

Neurology consultation revealed additional examination findings of normal corneal and facial sensation, with bilateral masseter and pterygoid weakness. The patient described being unable to eat, with difficulty chewing and sucking. She later developed appendicular sensory ataxia, and xerostomia.

MRI brain did not reveal any acute pathology. ANA was positive (homogenous and speckled pattern), with positive SCL-70, and Ro52 antibodies. The remainder of laboratory tests were unremarkable. Nerve conduction studies confirmed a sensory neuropathy.

The patient improved following intravenous immunoglobulins. She remains on intravenous immunoglobulins, mycophenolate and low dose prednisolone.

Conclusion We present a case of Sjogren's syndrome presenting with bilateral motor trigeminal neuropathies, without sensory involvement.

2740 POST-SPINAL SURGERY ISCHEMIC OPTIC NEUROPATHY AND THROMBOSED ORBITAL VARIX – CASE REPORT

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Non arteritic ischemic optic neuropathy (NAION) with consequent visual loss is recognised and serious outcome following spinal and cardiac surgery. We describe a patient in her 70s who developed sequential bilateral complete blindness and a thrombosed orbital varix post spinal surgery. This case highlights the atypical presentation for NAION and the role of prone positioning, ocular blood flow and hypoxia.

A lady in her 70s was transferred to a tertiary hospital for management of an L1 fracture and cauda equina. She had worsening lower limb weakness and urinary incontinence post a fall 6 months ago prior to the presentation. She underwent posterior decompression + fusion of T12-L2 in a prone position. Immediately post-op, the patient developed blurred vision in the upper quadrant of her left eye. The postoperative period was also complicated by an episode of reduced consciousness level which was thought to be secondary to opioids. A CT head identified a left superior ophthalmic vein varix with potential thrombosis. Ophthalmology review noted mild inferior blurred discs, but examination was considered within normal limits and CT findings were considered incidental.

Two weeks later she reported to staff that the vision in her right had become dull and blurred over the last 24 hours. Ophthalmology assessment which showed bilateral optic swelling and was then admitted for further work-up of her bilateral vision loss. Her vision continued to deteriorate during her stay. Lumbar puncture, MRI Brain/Spine, temporal artery ultrasound and temporal artery biopsy did not reveal any obvious abnormality.