2731 DISSEMINATED ASPERGILLOSIS: A RARE CAUSE OF ACUTE ISCHAEMIC STROKE

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Introduction Aspergillus species are ubiquitous fungal species that rarely cause a significant burden of disease in immunocompetent individuals. Invasive aspergillosis however, in an immunocompromised host, has high rates of morbidity and mortality. Initial infection via airway colonisation and dissemination via vasculature, can occur with significant consequences including stroke.

Case A 55 year old female presented as a stroke code, outside thrombolysis window, aphasic, dense right hemiparesis, forced left gaze deviation with reduced level of consciousness and NIHSS 32. Initial stroke series imaging with CT brain, CT carotid angiogram and CT perfusion scan identified an established left middle cerebral artery stroke with a left M1 thrombus. She was transferred to an endovascular clot retrieval centre and underwent thrombectomy. Macroscopically the fresh thrombus appeared white, histopathology identified fungal hyphae septated and branching at 45-degree angle, consistent with aspergillus. CT chest abdomen and pelvis identified cavitating left apical lung lesions, the largest 2.6cm in diameter. Transthoracic echocardiogram demonstrated moderate tricuspid regurgitation with raised pulmonary pressures, transoesophageal echocardiogram confirmed a patent foramen ovale. She was managed with intravenous voriconazole which was changed to amphotericin due to QT prolongation. Immunoglobulin levels, inclusive of IgA, IgG, IgM and IgE, were within normal range and Human immunodeficiency virus (HIV), hepatitis B (HBV), hepatitis C (HCV), Syphilis and mycobacterium tuberculosis (TB) cultures were negative. Her admission was complicated by several acute issues and was prolonged by complex discharge planning.

Conclusion This case highlights a rare case of disseminated aspergillosis causing an acute ischaemic stroke.

2732 RHABDOMYOLYSIS ASSOCIATED WITH CROHN'S DISEASE

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Rhabdomyolysis refers to acute breakdown of muscle; often the precipitating factor is not immediately apparent. We report an unusual temporal association between rhabdomyolysis and Crohn's disease, a systemic and traditionally considered a non-neurological autoimmune disorder. We present a 20-year-old male who was admitted for a two-day history of severe upper limb and chest tenderness after a light gym workout. The creatinine kinase at presentation was significantly elevated to 55,453 unit/L with normal K and renal function. Urine myoglobulin was also positive and he was subsequently diagnosed with rhabdomyolysis. Extensive serum workup for myopathy was negative and the symptoms of rhabdomyolysis had completely resolved within six weeks. Two months after this admission, he presented to emergency with two weeks of colicky abdominal pain and bloody diarrhoea. He was diagnosed with Crohn's disease following colonoscopy but concurrently had no myalgia.

Crohn's disease has been associated with myopathy, albeit rarely, and we propose that this patient, may have been predisposed to developing rhabdomyolysis after light exercise due to an underlying autoimmune process.

2733 A CASE OF SPORADIC CREUTZFELDT-JAKOB DISEASE (SCJD) FOLLOWING VIRAL VECTOR COVID-19 VACCINATION

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A previously well and highly functioning 39-year-old male, had progressive short term memory loss and low mood, one month following first dose of AstraZeneca COVID-19 vaccination. He developed diplopia secondary to an acquired esotropia. Investigations were spuriously positive for anti-MUSK antibody and low positive VGKC antibody (negative CASPR2 and LGI1 antibody). Infective and autoimmune encephalitis screens were negative. MRI demonstrated diffuse cortical diffusion restriction with CSF negative for 14-3-3 and Rt-QuIC. Brain and whole body PET-CT showed global diffuse reduced activity, without occult malignancy. EEG showed nonspecific slowing. His condition deteriorated despite high dose steroids and IVIG.

The patient progressed to a rapidly progressive dementia with memory impairment, dysphasia, dysphagia, and myoclonus, 15 months following initial vaccination. Further treatment with plasmapheresis, IVIG and weekly IV methylprednisolone (Mayo Clinic autoimmune dementia protocol) did not improve his symptoms. Genetic testing showed homozygosity of methionine at PRNP codon 129 which confers a higher risk of sporadic CJD. Subsequent biopsy showed multifocal synaptophysin-like prion staining and focal perineuronal prion positive staining in keeping with the definitive diagnosis of Creutzfeldt-Jakob disease.

Conclusions We postulate that virus vector COVID vaccination may have accelerated or triggered sporadic CJD, in a genetically susceptible individual. Studies have highlighted a possible casual relationship between COVID-19 infection and CJD and other neurodegenerative disorders, due to a promotion of neuroinflammation and protein misfolding. Animal studies demonstrate accelerated transition from pre-clinical to clinical stages of prion disease with co-infection. This is to our knowledge the first case report of vaccine associated CJD.