

remains lymphoma and previous treatment with steroids might cover pathological features. Although the exact cause remains elusive, treatment for PACNS could be considered in unilateral white matter lesion not responsive to pulse steroids.

**2987** A FRESH PERSPECTIVE ON STROKE-LIKE MIGRAINE ATTACKS AFTER RADIOTHERAPY (SMART) SYNDROME: SERIES OF FOUR CASES

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**Background/Objectives** Patient receiving cranial radiotherapy can experience reversible hemispheric dysfunction because of delayed toxicity to the brain. Many patients do not fulfil the criteria for a diagnosis of stroke-like migraine attacks after radiotherapy (SMART) syndrome due to the requirement for distinctive imaging findings, and it is likely there is a broader spectrum of disease than previously believed. The aim of this study was to collect cases of stroke-like attacks which did not fulfil the diagnostic criteria to further characterise the typical clinical features, investigations findings and potential management of this condition.

**Methods** Cases were collected retrospectively from two South Australian hospitals of patients presenting with a history of cranial radiotherapy and an acute onset of neurological symptoms without fulfilling the diagnostic criteria for SMART syndrome.

**Results** Four patients with transient hemispheric dysfunction post-radiotherapy, who did not fulfil the diagnostic criteria for SMART syndrome, were identified. All patients presented with acute-onset focal neurological deficits, including expressive dysphasia (75%), hemiparesis (50%) and hemineglect (25%). No patients presented with MRI findings suggestive of SMART syndrome. Patients received either supportive management or pharmacotherapy with anti-seizure drugs, l-arginine, or a combination of medications, and were all discharged without residual neurological deficits relating to their episode.

**Conclusion** SMART syndrome likely exists on a spectrum, which includes patients exhibiting characteristic clinical features without imaging findings. More cases of stroke-like episodes post-radiotherapy need to be identified to further characterise this condition and establish a more inclusive set of diagnostic criteria.

**2989** NMDAR AUTOIMMUNE ENCEPHALITIS AND FULMINANT RELAPSE OF MULTIPLE SCLEROSIS TREATED WITH ALEMTOZUMAB: A RARE OVERLAP SYNDROME

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**Background** Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis is an autoimmune disease that has been rarely associated with anti-aquaporin-4 immunoglobulin G (AQP4 IgG) and anti-myelin oligodendrocyte glycoprotein IgG (MOG IgG) associated diseases, and even more rarely with multiple sclerosis (MS).

**Case A** woman in her 40s presented akinetic, mute, and newly bed-bound following three weeks of rapid functional and cognitive decline. She had been diagnosed with relapsing-remitting MS sixteen years prior and was treated with two courses of Alemtuzumab six and seven years prior. The initial impression was of fulminant relapse of multiple sclerosis, although the severe abulia and mutism were considered atypical, and magnetic resonance imaging of head and spine showed new extensive post gadolinium contrast enhancement throughout regions of pre-existing demyelinating lesions. Serum and cerebrospinal fluid tested positive for NMDAR antibodies, and electroencephalography showed only encephalopathic features. She received IV methylprednisolone and five sessions of therapeutic plasmapheresis before an induction course of Ocrelizumab, and she subsequently made a slow and partial recovery, returning to live in the community with supports.

**Conclusions** We systematically reviewed similar reported cases (23 in total), finding a lower than anticipated prevalence of detected underlying malignancy (0 of 20), and relatively favourable prognosis (64% making a good recovery), compared to isolated NMDAR encephalitis. These findings suggest a rare overlap syndrome with some distinct clinical features.

**2991** CENTRAL AND BRANCH RETINAL ARTERY OCCLUSION AETIOLOGY, PROGNOSIS AND OCULAR NEOVASCULAR COMPLICATIONS: A RETROSPECTIVE COHORT STUDY

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**Background/Objectives** Retinal artery occlusion (RAO) due to central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO) is an incompletely understood, rare and sight-threatening condition with limited treatment options. The aim of this retrospective study was to determine the aetiology, treatment and outcomes of CRAO and BRAO in a South Australian cohort.

**Methods** Patients attending the Central Adelaide Local Health Network between a 12-month period with a new presentation or previous diagnosis of CRAO/BRAO were included. Data were collected on aetiology, treatment, visual acuity outcomes and the development of neovascular glaucoma (NVG).

**Results** The cohort comprised 80 patients, of whom 55 were diagnosed with CRAO and 25 with BRAO. Aetiology was identified in 21.3% of cases, with vasculitis (10%) and carotid artery thromboembolism (6.3%) being the most common confirmed causes respectively. Median visual acuity at presentation was >6/60 and 6/9 in CRAO and BRAO subgroups respectively. Visual acuity at presentation was the only predictive factor significantly associated with follow-up visual acuity for both CRAO ( $p = 0.047$ , coefficient 0.45, 95%CI 0.007–0.884) and BRAO ( $p < 0.001$ , coefficient 1.03, 95%CI 0.585 – 1.468). Ten patients with CRAO developed NVG and one patient with BRAO had NVG. No factors associated with the development of NVG after CRAO/BRAO were identified.

**Conclusion** Visual acuity outcomes for patients with RAO are poor. The only significant predictor of visual acuity prognosis was visual acuity at the time of the initial assessment. A thorough aetiological workup is essential as most cases have no specific aetiology identified.