**POI06** RITUXIMAB IN NEUROPSYCHIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS: A RETROSPECTIVE STUDY OF THE CAMBRIDGE EXPERIENCE

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The monoclonal B cell depleting antibody, rituximab, has shown promise in open-label studies of refractory neuropsychiatric (NP) lupus. Here, we show our tertiary referral centre experience in patients with CNS syndromes probably causally related to active SLE, and in those with past CNS symptoms treated for non-NP SLE. Consensus criteria proposed by Hanly et al (Arth Rheum 2007) were used to classify patients into those likely to have attributable disease or not. Some patients have been previously reported (Smith et al Arth Rheum 2006). Four patients received rituximab for CNS symptoms attributable to active lupus (two psychosis, one neuropsychiatric syndrome, one neuromyelitis optica). The baseline clinical, imaging and laboratory features, clinical responses and adverse events are reported. A summary is provided of our other patients given rituximab for active non-NP SLE, with previous possible attributable NP disease, including anti-phospholipid syndrome. All patients had other organ disease, received prior and concomitant immunotherapy, and most were retreated. Rituximab appears effective with attributable disease and limited prior damage, with limited effect on symptoms due to static deficits. A randomised, double-blind placebo-controlled trial of rituximab, in patients with early attributable disease and limited established damage, is required. There is also a need to study the natural history of NP involvement in SLE and discover and develop biomarkers to aid attribution.

**POI07** RECURRENT N-METHYL-D-ASPARTATE RECEPTOR ENCEPHALITIS: A CASE REPORT

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**Introduction** Autoimmune encephalitis associated with N-methyl-D-aspartate (NMDA) receptor antibodies is increasingly recognised in intensive care but there have been no reports of recurrent cases.

**Case** A 22-year-old woman presented in 2001 with generalised tonic clonic seizures followed by a progressive encephalopathy with fluctuating consciousness and impaired memory. Routine blood tests, auto-immune screen and serology were negative, CSF examination showed a pleocytosis and MRI revealed atrophy of the left hippocampus. She had a prolonged illness but made a slow recovery, returning to her premorbid level of functioning. She continued to suffer occasional seizures. She had a similar illness 13 years previously at the age of nine with encephalopathy, abdominal pain, headache, confusion and a hyperkinetic movement disorder. She again had made a slow but full recovery. No definitive cause for her illness had been found. Following reports of NMDA receptor encephalitis in 2008, this patient’s serum tested positive for antibodies to the NMDA receptor. This confirmed the likely diagnosis of anti-NMDA receptor encephalitis.

**Conclusion** NMDA receptor encephalitis may develop in childhood and be recurrent. It can be useful to retrospectively test serum for antibodies in cases of undiagnosed encephalopathic illnesses.

**POI08** SERUM C4A LEVELS IN MULTIPLE SCLEROSIS CORRELATE WITH DISEASE ACTIVITY

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Complement is known to play a key role in MS and recent work utilising proteomic analysis identified raised serum levels of C4a in patients compared to controls which decreased following relapse. In order to replicate and validate this finding which has significant implications for the pathogenesis of MS, we have measured serum C4 and C4a in 107 patients with representation from all disease subgroups and 40 controls. We also examined serial samples on 45 patients following relapse to assess dynamic changes. C4 levels showed good correlation with C4a (r=0.27, p=0.001). C4 levels were raised in MS patients (mean 267 mg/l, SD 116) compared to controls (mean 248 mg/l, SD 79; p<0.001) with no discrimination between disease subgroups. C4a levels were not significantly raised in primary (mean 1.40 mg/l, SD 0.67; p=0.89) or secondary (mean 1.69 mg/l, SD 0.70; p=0.18) progressive patients or relapsing remitting patients in remission (mean 1.59 mg/l, SD 0.79; p=0.91) compared to controls; but were significantly increased in patients in acute relapse (mean 2.07 mg/l, SD 1.07; p<0.001) and decreased significantly after 2 months (mean 1.85 mg/l, SD 0.91; p=0.028). In summary, we were able to confirm dynamic changes in C4a levels in patients in acute relapse implying a systemic humoral inflammatory component at relapse ignition; however, because of low sensitivity/specificity between patient groups it is unlikely that C4 or C4a could be employed as reliable clinical diagnostic or disease state biomarkers.

**POI09** TRENDS IN BK AND JC POLYOMAVIRUS AND RELATIONSHIP TO CD4+/CD8+ RATIO IN PATIENTS WITH RELAPSING REMITTING MULTIPLE SCLEROSIS ON NATALIZUMAB THERAPY

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Natalizumab reduces clinical relapses and the risk of sustained progression of disability in patients with relapsing remitting multiple sclerosis (RR MS). It has been associated with the development of progressive multifocal leucoencephalopathy, a rare demyelinating disease caused by JC virus. BK virus is a related polyomavirus known to cause morbidity in the immunocompromised host. Studies in the HIV population have linked BK viruria and activation of JC virus. The risk of developing BK viruria, and significant immuno-suppression by sampling serum and urine specimens at baseline and at 3-monthly intervals. This is an ongoing prospective, longitudinal study that started in January 2007. A total of 86 subjects with active RRMS received natalizumab in our Department. We found no significant relationship between duration of natalizumab therapy and activation of JC virus. The risk of developing BK viruria, however, increased over time with the number of doses received. No significant reductions in CD4+ counts or in CD4+/CD8+ ratios were noted. These findings indicate that while there is a link between natalizumab and BK activation no significant immuno-suppressive effect was observed. Further studies on the implications of BK virus in MS remain to be done but these results may have
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