rather than malignant infiltration. There was no evidence of spinal cord compression attributable to disc degeneration or prolapse.

The patient underwent a posterior spinal decompression from T8 to T10. Histological examination of intra-operative tissue samples showed a large number of macrophages and varying amounts of cystic change associated with fibrin deposition. There was lymphoplasmacytic infiltrate that is present around the periphery of the nodules, which were entirely consistent with the history of RA and synovitis. Six months following his surgery, the patient’s ataxia had improved dramatically. He was mobilizing with two crutches and could manage a short distance without. A year from surgery and he had improved further. He was ambulating independently with one stick.

Patients with chronic RA are widely known to commonly develop spinal problems, in particular of the cervical spine. These can range from upper cervical spinal instability and stenosis to soft tissue spinal cord compression. As a result, a number of these patients will present with myelopathic signs and symptoms requiring investigation and intervention. The majority will undergo imaging of their cervical spine with an MRI scan as first-line investigation. In the majority of cases, this line of investigation will identify the primary pathology and lead to appropriate intervention.

We present an unusual case of thoracic spinal cord compression as a result of facet joint synovitis in the thoracic spine. From the literature search, we have performed this is one of only a few such reported cases. With this case report, we hope to highlight a rarer cause for patients presenting with profound myelopathy. Failure to appreciate such a diagnosis could lead to continuity of symptoms, further deterioration in mobility and potentially inappropriate surgery as some patients may have incidental findings on MRI. We believe that in cases such as the one we present that consideration of imaging the whole spinal cord and not just the cervical cord should be made. This is relevant to both the rheumatologist, to whom a lot of these patients may present initially and also the spinal surgeons who may be referred the patient for surgical management. In this specific case, prompt diagnosis was made and the patient is very grateful for the improvement in his symptoms that he has had following surgery.

**Rheumatology key message**

- Thoracic spinal cord compression should always be considered in rheumatoid patients presenting with myelopathy.

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**Clinical analysis of anti-NR2 glutamate receptor antibodies and interleukin-6 with neuropsychiatric systemic lupus erythematosus**

Sir, A 14-year-old female was diagnosed as having SLE at the age of 13 years when she presented with fever,
arthralgia, headache and purpura. She had ANAs (1:2560). She was treated with steroids. When these were tapered, the fever and headache returned.

Written informed consent was obtained from the patient. The patient was admitted to the hospital with fever, headache, tremors and memory dysfunction. The peripheral blood counts revealed white blood cells at 2300/µl (segment 77.9%, lymphocytes 0.4%), haemoglobin at 11.1 g/dl and platelets at 48,000/µl. The CRP level was elevated (4.0 mg/dl) and the ESR was 54 mm/h. Anti-dsDNA antibodies, complements C3 and C4 were normal. ANAs were positive (titre, 1 : 160) as were anti-SS-A/Ro antibodies (99.4, <10 U/ml), IgG aCLs [11, normal range (NR) <10 U/ml] and the lupus anti-coagulant (1.43, NR < 1.3). The serum thrombomodulin was 8.8 FU/ml (NR < 4.5). Serum ferritin levels were high, at 1969 ng/ml. A bone marrow smear revealed histiocytic proliferation.

A brain MRI evaluation showed abnormalities at the right paralateral ventricle (Fig. 1A and B). Her cerebrospinal fluid (CSF) was sterile with normal protein, glucose and white blood cell counts of 6/µl. The CSF IL-6 was high at 228 pg/ml (normal < 4.0). Her serum and CSF IgG anti-NR2 glutamate receptor antibody titres were 24.0 U/ml (NR 5.63 [3.02]) and 8.6 U/ml, respectively. A psychological evaluation with the Wechsler Intelligence Test for Children (3rd edn) (WISC-III) test indicated that her non-verbal intelligence quotient (IQ) was 57, in the mild mental retardation range, and her verbal IQ was 79, which was in the borderline range. Her global IQ was 65, which indicates mild cognitive dysfunction. She was diagnosed with neuropsychiatric SLE (NPSLE) and haemophagocytic syndrome.

After diagnosis, we administered high-dose pulse i.v. steroids of methylprednisolone, ciclosporin A and an anti-coagulant, but her fever persisted, low platelet count and elevated ESR continued. We added treatments with IVIG and i.v. CYC. Her headache and tremors improved. Her CSF IL-6 level was 1.3 pg/ml. The serum and CSF anti-NR2 antibodies titres were 10 and 3.22 U/ml, respectively. Monthly i.v. CYC was continued for 6 months. A second WISC-III test indicated that her...
non-verbal IQ was 94, her verbal IQ was 87 and her global IQ was 90. Her cognitive impairment gradually decreased. One year later, brain MRI (Fig. 1C and D) showed improvement in the right paralateral ventricle.

The classification of NPSLE has been used to verify the clinical manifestations [1]. Her neuropsychiatric manifestations were cerebrovascular disease, headache, tremors and cognitive dysfunction. The pathogenesis of NPSLE remains unclear. The present case showed vascular abnormalities, aPLs and elevation in CSF IL-6. Previous studies have shown associations between increased serum levels of IL-6 and learning deficits in SLE [2, 3]. Horai et al. [4] have shown that the CSF IL-6 levels are useful in the diagnosis of NPSLE.

Recently the N-methyl-D-aspartate (NMDA) receptor subunit NR2 was shown to be a target of autoantibodies in SLE, supporting the notion of the antigenic target hypothesis as a mechanism of NP manifestations [5–8]. The NR2 receptors are expressed on neurons in the hippocampus and cortex, and bind the excitatory amino acid neurotransmitter glutamate. These receptors have been postulated to be important in mechanisms underlying learning and memory. Anti-NR2 antibodies may be produced only outside the CNS and enter the CSF as a result of damage to the blood–brain barrier. Arinuma et al. [6] showed that elevated levels of anti-NR2 antibodies in the CSF occurred frequently in the patients with diffuse NPSLE. Yoshio et al. [5] found an association between NP symptoms and CSF anti-NR2 antibodies compared with circulating anti-NR2 antibodies. Further, they described that steroid therapy may improve the damage to the blood-brain barrier and decrease the level of anti-NR2 antibodies entering the CSF. Anti-NR2 antibodies function as NMDA receptor agonists [7, 8]. IL-6 synthesis in neurons is dependent on activation of the NR2B subunit [9]. In conclusion, no gold standard diagnostic tests for NPSLE are available at present. The findings in the present case indicate that CSF anti-NR2 glutamate receptor antibodies and IL-6 levels may be associated with pathogenesis, and they might be useful as markers of NPSLE activity.

**Rheumatology key message**

- CSF anti-NR2 antibodies and IL-6 are useful in both diagnosis and activity of NPSLE.

**Disclosure statement:** The authors have declared no conflicts of interest.

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