A case of demyelinating myelopathy during treatment with Tumor Necrosis Factor -α blokers

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Introduction

Tumor Necrosis Factor-α (TNF-α) blokers are a known therapeutic choice in many inflammatory rheumatological diseases. With the widespread use of TNF-α blokers, a growing number of perypheral and central nervous system (CNS) demyelinating disorders have been reported (1). TNF-α is a cytochine with a complex role in immune system regulation and maintenance of self-tolerance (2). We describe a patient with clinical, radiological and laboratory analysis suggestive of CNS demyelination in the setting of TNF-α blokers therapy.

Case Report

A 41- year-old male with a history of HLA-B27 seronegative anchylosing spondylitis had received golimumab for six years. He presented numbness of the legs ascending bilaterally to the trunk and the hands over a week. The neurological examination revealed positive Lehermitte's sign, dysesthesias of the right leg and right truncal region up to C5 level , brisk tendon reflexes at lower extremities with bilateral Babinski sign. Routine blood tests and autoimmunity were unremarkable. Brain magnetic resonance (MR) showed few small periventricular T2 hyperintense lesions without gadolinium enhancement; spinal MR revealed an enhancing tumefactive lesion at C3 level **(Fig. 1).** Cerebrospinal fluid (CSF) analysis revealed mild pleocytosis, normal glucose and protein levels, raised Ig-G index, positive oligoclonal bands; coltural examination and PCR for viral nucleic acids on CSF were negative. PEV documented P100 latency with borderline latency in one eye. The treatment with golimumab was discontinued and the patient was treated with intravenous high dose methylprednisolone, followed by prednisone tapering. On discharge from the hospital his symptoms had almost completely resolved; methotrexate was started. At the control visit at three and six months he was mostly asymptomatic; brain and spinal MR at six months documented improvement of the cervical lesion without gadolinium enhancing and the absence of new T2 lesions. **(Fig. 2).**

Discussion

The clinical manifestations, the morphology and distribution of cerebral and spinal lesions and the CSF data argue for the diagnosis of demyelinating disease of the CNS. In previous reported series CNS demyelination induced by TNF-alfa blockers included transverse myelitis, optic neuritis, brainstem syndromes and even demyelinating features in asymptomatic patients (3). There is still a debate whether these events are coincidental or side effects of TNF-α blockers use, and whether these treatments unmask preexisting Multiple Sclerosis (MS) or may induce a de novo SNC demyelination.

Conclusions

Careful clinical and neuroradiolgical assessment of patients treated with TNF- α blockers may be helpful. In our case long term follow-up is required to point out the potential differences from a typical MS.

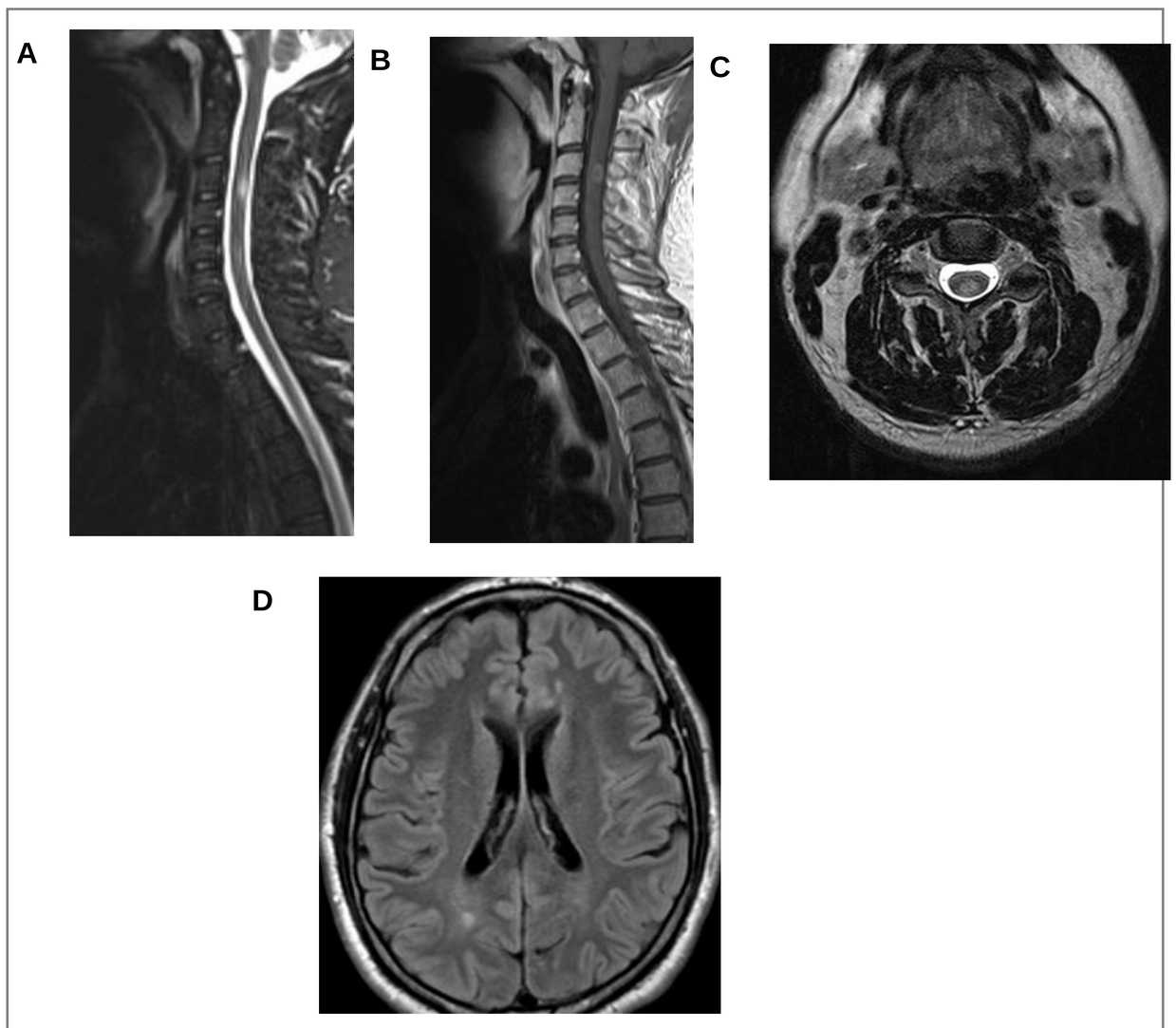

Figure 1. MRI at presentation. **Panel A:** sagittal,. T2 weighted spinal MRI. **Panel B:** sagittal,.T1 weighted , Gd + spinal MRI :**Panel C** axial, T2 weighted, spinal MRI **Panel D:** axial FLAIR brain MRI

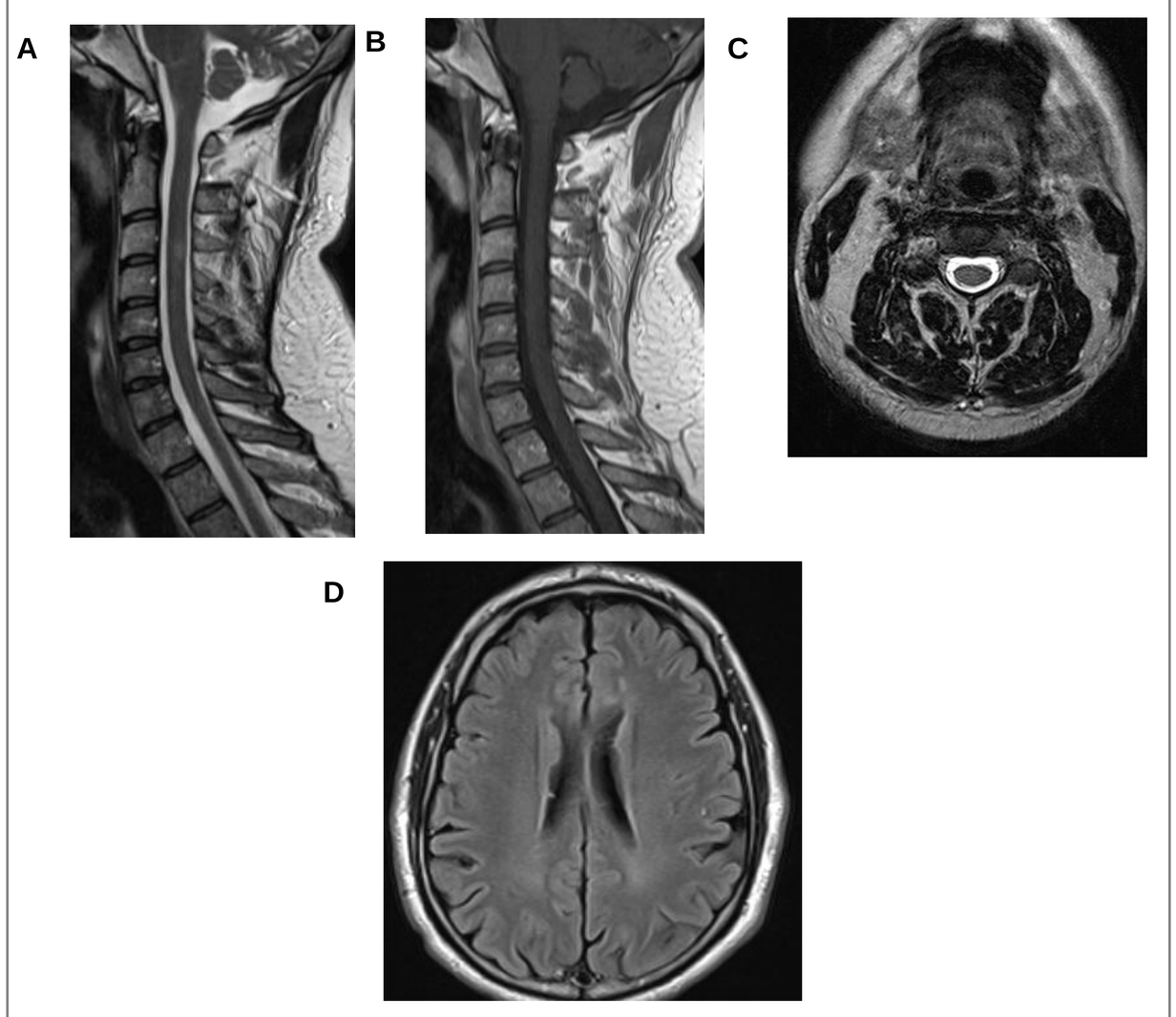

Figure 2. MRI at follow-up. **Panel A:** sagittal,. T2 weighted spinal MRI. **Panel B:** sagittal,.T1 weighted , Gd + spinal MRI :**Panel C** axial, T2 weighted, spinal MRI **Panel D:** axial FLAIR brain MRI

References

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