(S140) CENTRAL NERVOUS SYSTEM Demyelination AND ANTI-TUMOR NECROSIS Factor Alpha Therapy

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Background: Tumor necrosis factor alpha (TNF-α) inhibitors have gained more importance in the treatment of Crohn's disease, rheumatoid arthritis, ankylosing spondylitis, and psoriasis. An association between anti-TNF-α therapy and peripheral and central nervous system (CNS) demyelination has been described, although the extent of this association and its impact on clinical practice have not been fully evaluated. However, anti-TNF-α therapy may need to be included in differential diagnosis considerations when assessing patients for possible demyelinating disease. Objectives: To assess the relevance of TNF-α inhibitor treatment in the differential diagnosis of possible CNS demyelination. Methods: Case presentation and literature review. Results: A 35-year-old man with severe Crohn's disease since age 12 was placed on infliximab (Remicade) at age 30. At age 33 years, he developed urinary urgency and incontinence and was found to have detrusor overactivity classified as neurogenic bladder by an experienced urologist, although no other neurologic symptoms or signs were found on history or clinical examination. Magnetic resonance imaging (MRI) of the brain showed minimal white matter disease; MRI of the cervical and thoracic spine was unremarkable. The second patient is a 39-year-old woman with severe psoriatic arthritis who has been on etanercept (Enbrel) since age 34. She presented with right-sided optic neuritis. Brain MRI showed very few punctate T2 signal abnormalities within the bilateral frontal white matter as well as along the deep surface of the corpus callosum without contrast enhancement. Conclusions: We present two cases of possible CNS demyelination and relevant literature review. Although the cases we present demonstrate that it is difficult to prove a causal relationship between TNF-α inhibitors and the development of CNS demyelination, it appears that patients treated with TNF-α inhibitors need to be assessed more frequently for neurologic side effects. Providers in neurologic practices and multiple sclerosis (MS) clinics should be aware of this possible consideration. As discontinuation of the anti-TNF-α therapy has led to improvement of neurologic symptoms in most cases reported in the literature, other disease-modifying treatments to prevent future attacks of MS may not be necessary.

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