

Central Nervous System Demyelination Associated to Rheumatoid Arthritis

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ABSTRACT

Introduction: Patients with rheumatoid arthritis (RA) may have central nerve system (CNS) comorbidities such as demyelinating lesions. In current practice, this clinical situation raises an etiological problem of this demyelination; is it linked to treatments; anti TNF drugs, which is the most frequent case? or is it a primary demyelination of the CNS?

Case Report: We reported the case of a 52-year-old woman followed for seronegative Rheumatoid arthritis (RA) treated with methotrexate that presented clinical and radiological multifocal inflammatory demyelinating lesions. No etiology related to RA or its treatment could explain the radioclinical presentation of the patient. Magnetic resonance imaging lesions fulfilling MC Donald's criteria for Multiple Sclerosis (MS) (2017) and analysis of cerebrospinal fluid showed intrathecal synthesis. All the difficulty of this medical condition lies in the etiological assessment. The diagnosis of primary demyelination, specifically multiple sclerosis associated with RA, was retained.

Conclusion: Certainly it is an infrequent situation in the absence of anti-TNF treatment which makes the originality of our case.

Keywords: Rheumatoid Arthritis, Multiple Sclerosis, demyelination

CASE REPORT

A 52-year-old woman followed since the age of 37 years for seronegative Rheumatoid arthritis (RA). She was initially treated with corticoid and methotrexate and achieved remission. 2 years ago, she suffered from recurrent symptomatology associating weakness of the lower limbs, dizziness, paresthesia of the face as well as progressive numbness of the lower limbs. Neurological examination showed paraparesis, quadriparymidal syndrome and central vestibular syndrome. No extraneurological signs were found except those related to her RA. Cerebral Magnetic resonance imaging (MRI) revealed several hyperintense FLAIR signals in the periventricular and sub-cortical white matter with gadolinium enhancement of one lesion (Figure 1). Medullary MRI also showed hyperintensities on T2 images in cervical and dorsal lateral spinal cord which were not enhanced by gadolinium. She underwent several investigations including Serological studies for Lyme, Human immunodeficiency virus, Hepatitis and Syphilis that were negative. Immunological assessment

containing rheumatoid factor (RF), antinuclear antibody, anti- NMO antibodies were also within the normal range. Cerebrospinal fluid examination (CSF) revealed normal cytology and biochemistry, but evidenced Oligoclonal bands and intrathecal synthesis of IgG with IgG index at 1,36. Visual evoked potentials were normal. According to The Mc Donald's criteria 2017, the diagnosis of multiple sclerosis (MS) was made. The patient was treated with high-dose of intravenous methylprednisolone (1g/day IV during 5 days). The Methotrexate was stopped after mutual agreement between neurologist and rheumatologist because of the remission of RA in our case and immunomodulatory drug (Interferon-beta-1b) was initiated.

DISCUSSION

Rarely the involvement of the central nervous system was associated with RA especially if it is a primary demyelination of the central nerve system type MS. Although this association between RA and MS has been described, the diagnosis remains difficult: Neurological

manifestation of RA? Iatrogenic demyelination? Authentic MS? RA and MS are both autoimmune diseases that share similar pathogenesis particularly a T cell mediated process (1). Although there are increasing reports that MS develops in patients with RA treated with anti-tumor necrosis factor-alpha (anti-TNF-alpha) (2), the comorbidity of RA and MS in patients with no anti-TNF-alpha treatment has been rarely reported (3,4). In our case, diagnosis of MS was made according to clinical features and exclusion of other autoimmune diseases. Furthermore multifocal inflammatory demyelinating lesions revealed by

MRI and fulfilling MC Donald's criteria, were suggestive of MS. In RA, MTX is given in small doses and no anomalies on brain and medullar imaging have ever been reported. Moreover, the development of MS after the onset of RA is rare, especially in patients being treated commonly with medications such as methotrexate that have potential immunosuppressive effects (5). Thus, the development of ambiguous neurologic symptoms consistent with MS in patients with RA whether or not they are receiving anti-TNF α should lead to consider in this context the possibility of associated MS disease.

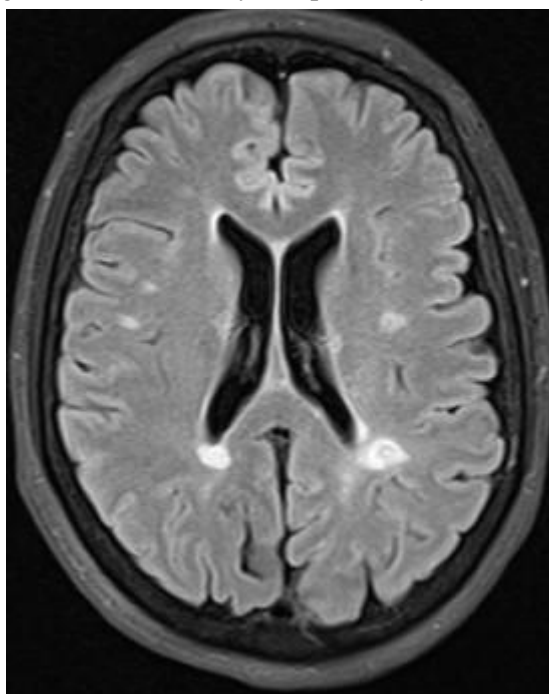


Figure1. Magnetic resonance imaging (MRI) of brain of index patient: Axial Fluid Attenuated Inversion Recovery (FLAIR) image showing several ovoid hyperintensities in periventricular and subcortical white matter

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