Demyelinating disease during anti-tumor necrosis factor α therapy – case report

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Tumor necrosis factor alpha (TNFα) is a cytokine that plays a key role in inflammatory response in various autoimmune diseases also in the central nervous system (CNS). This potent agent with pleiotropic actions can be present both as a transmembrane protein as well as soluble cytokine. Biological effects of both forms are mediated through interaction with receptors TNFR1 and TNFR2 with distinct functions. Multiple sclerosis (MS) is a chronic and progressive disease of the CNS with a complex etiology. Its main pathological features include neuroinflammation, demyelination and axonal loss. There is strong evidence of role of tumor necrosis factor alpha (TNFα) in pathogenesis of the disease. Despite data suggesting that TNF-α intrinsically causes primary demyelination, apoptosis and neurological damage previous attempts of treatment of MS with TNFα antagonists led to an increase of disease activity. Moreover, neurological adverse events have been reported among patients which received anti-TNFα treatment for other autoimmune and inflammatory diseases. Here we present a case of patient suffering from rheumatoid arthritis treated with recombinant monoclonal human anti-TNFα antibody (adalimumab). During therapy neurological symptoms developed suggestive of the CNS demyelination. Neurological deficits correlated with magnetic resonance imaging showing hyperintensities on T2-weighted images without gadolinium enhancement. Cerebro-spinal fluid analysis supported hypothesis of present neuroinflammation with positive oligoclonal bands however with normal IgG index. In this study we also discuss dualistic role of TNFα in the process of CNS myelin damage and repair with emphasis on different roles of TNFR1 and TNFR2.