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Oral Presentation

Central Nervous System Involvement in Rheumatoid Arthritis: Possible Role of Chronic Inflammation and TNF Blocker Therapy

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Abstract

Rheumatoid arthritis (RA) is a chronic disease, the etiology of which has yet to be clarified, which causes activation of proinflammatory pathways that bring about joint and systemic inflammation. In recent years, the pathophysiology of CNS involvement that can occur in RA has attracted a great deal of attention. Emphasis has focused on the possibility that CNS involvement occurs due to blood-brain barrier (BBB) damage associated with chronic inflammation. The present study was performed to investigate the possible effects of BBB dysfunction and tumor necrosis factor (TNF) blocker therapy on BBB function, which may cause CNS damage in patients with RA. 90 RA patients [65 females, 25 males] and 40 healthy controls [25 females, 15 males] were included in the study. All RA patients were on synthetic DMARD therapy at the beginning. 55 patients continued DMARD therapy, and 35 patients with high disease activity were started on TNF blocker therapy. All demographic characteristics of the patients were recorded. Disease activity was evaluated using the Disease Activity Score 35-joint count C reactive protein. The Mini-Mental State Examination was used to evaluate cognitive function, and the Fazekas scale was used to assess cranial lesions visualized by magnetic resonance imaging (MRI). Patients' peripheral blood S100β, glial fibrillary acidic protein (GFAP), claudin, interleukin (IL)-17, and IL-1β levels were measured at the beginning of the study and after 6 months. Demographic characteristics (including sex, age, and body mass index) were similar in the RA and control groups. S100β and GFAP levels were significantly higher in the patient group than in the control group. In the group that was started on TNF blocker therapy, S100β and GFAP levels were significantly decreased 6 months after commencement of treatment. No difference was observed between the RA and control groups in terms of hyperintense lesions seen on cranial MRI. The S100β levels increased with lesions in the deep white matter seen on cranial MRI in patients with RA. next to decreasing disease activity and joint erosions by suppressing inflammation, anti-TNF therapy in RA can also suppress potential CNS involvement linked to BBB (blood-brain barrier) dysfunction.

Keywords: Blood-Brain-Barrier; Rheumatoid Arthritis; S100 beta

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