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P0216 The potential role of the CCR5 and CXCR3 receptors in the lymphocyte migration in tick-borne encephalitis

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Background: In tick-borne encephalitis (TBE) the cerebrospinal fluid (CSF) and central nervous system (CNS) tissue mononuclear cell infiltrates consist mainly of CD3+CD4+ (Th) and CD3+CD8+ (Tc) lymphocytes. The chemokines interacting with of CCR5 and CXCR3 receptors on lymphocytes have been proposed to drive the migration of these cells into CNS. Especially, CCR5 expression is supposed to be protective in TBE and West Nile encephalitis based on the genetic association studies. We have compared the expression of both receptors in CSF and blood lymphocytes from TBE patients, to verify if they are present and enriched in the CSF lymphocyte population and possibly involved in its accumulation.

Materials/methods: We have studied simultaneous blood and CSF samples from 20 patients with serologically confirmed TBE (14 with meningitis, 6 with meningoencephalitis/meningoencephalomyelitis) and 8 patients with non-TBE viral meningitis. Expression of CCR5 and CXCR3 receptors was measured cytometrically with fluorochrome-stained monoclonal antibodies on gated activated (CD45RO+) CD3+CD4+ and CD3+CD8+ lymphocytes. The fractions of positively staining cells and mean fluorescence indexes (MFI) for CCR5 and CXCR3 were used as measures of receptor expression and their CSF/blood ratios were calculated. The data were analyzed with non-parametric tests with $p<0.05$ considered significant.

Results: The CCR5 expression was significantly increased in CSF compared to blood in both CD4+ and CD8+ lymphocytes. Still, CCR5 was expressed by only 14% of CSF CD4+ and 47% of CD8+ lymphocytes. CXCR3 was expressed by about half of CD4+ and CD8+ lymphocytes, but its expression tended to be similar or lower in CSF than in blood. There was no difference between the meningitis and meningoencephalitis/myelitis TBE patients. The expression of CCR5 and CXCR3 was not increased in CSF of non-TBE meningitis patients, who had the CSF/blood ratios for both receptors significantly lower than TBE patients.

Conclusions: CCR5, but not CXCR3, is likely to contribute to CD4+ and CD8+ lymphocyte migration into CSF in TBE. Additional chemotactic axes should be involved in the accumulation of the CSF CD3+CD8+ and especially CD3+CD4+ cells, majority of which express neither CCR5 nor CXCR3.