T-Cell Subsets in the Cerebrospinal Fluid and Peripheral Blood of Patients with Parkinson’s Disease


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There is recent evidence to suggest that immune functions may be disordered in patients with Parkinson’s disease (PD). Auto-antibodies reacting with neural structures have been found in a substantial proportion of these patients. However, it is possible that the presence of auto-antibodies may reflect a deregulation of hypothalamic-mediated immune modulation. The hypothalamus has been shown to play a central role in mediation and integration of both humoral and cell-mediated immune response /1,2/. The purpose of our study was to address the rate of T cells (CD2+), Helper/Inducer T cells (CD4+), Suppressor/Cytotoxic T cells (CD8+), HLA DR+ cells and the presence of interleukin-2 receptor (CD25+) in the peripheral blood (PB) and CSF of patients with PD by an immunocytochemical method that permits both morphological evaluation and immunological classification of cells.

Control CSF was taken from (orthopedic and proctologic) patients submitting to surgical procedures using spinal anaesthesia. PD CSF and PB were taken from 11 patients graded as III-IV on the Hohen and Yahr Rating Scale. Multispot slides, with 21 spots separated by water-repellent dimethylpolysiloxane were used for cell attachment, and coated with poly-L-lysine. Ten microliters of cell suspensions were added to each spot and sequentially incubated in the appropriate immunologic reagents. The cells were stained for enzyme activity (Streptavidin/Biotin-HRP-System).

Significant amounts of CD25+ lymphocytes were usually present in the CSF of PD patients and controls. However, CSF from PD patients showed higher levels of CD25+, HLA-DR+ and CD4+ cells. Taking into account that these patients are in later stages of the disease, not only had degeneration of neurons in substantia nigra and other pigmented brainstem nuclei occurred, but degeneration was also seen in hypothalamus and sympathetic ganglia, i.e. in the neural structures presently known to be involved in immune regulation. Surface marker levels, except CD8+ cells, were significantly higher in CSF than in PB of PD patients, particularly CD4+ and CD25+ cells (Figure). These results suggest selective cell crossing through the blood-brain barrier for these populations that show activated markers.

In summary, (i) in normal CSF 90% of cells are T lymphocytes; (ii) a high ratio of CSF lymphocytes from normal controls are CD25+ cells and (iii) significant differences were found in the percentage of CD4+, CD25+ and HLA-DR+ cells between CSF from PD patients and normal controls, as well as in the percentage of CD2+, CD4+, CD25+ and HLA-DR+ cells between CSF and PB from PD patients. These findings suggest that disordered neural-immune regulation should be considered as a potential cause of immune abnormalities occurring in PD patients.

**REFERENCES**

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