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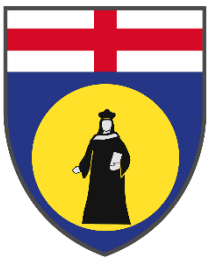

Tesi sperimentale per la Prova Finale

EVALUATION OF T LYMPHOCYTES INFILTRATION INTO THE BRAIN PARENCHYMA OF PATIENTS AFFECTED FROM BIPOLAR DISORDER TYPE I

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Abstract

Bipolar Disorder (BD) is a serious brain disorder, included between the 50 most dangerous pathologies worldwide, whose etiology is still not clear. The type I (BD I) of this pathology is particularly important because it can be characterized by manic episodes that can lead to suicide attempts. Recent observations have revealed that there could be a correlation between the incidence of the disease and the generation of chronic inflammation in the brain. Particularly, different microstructural abnormalities have been detected in the brain white matter of bipolar patients, which could be related to the uncontrolled activity of microglia. Different studies have revealed a large increase in CD4+ and CD8+ T lymphocytes in the Central Nervous System, due to increased permeability of the Blood-Brain Barrier. This has been observed mainly for the subpopulations of effector memory, terminally differentiated effector memory (CD8+ CD28- CD45RA+) and CD8+ INFγ+ T cells, which have shown significantly different concentrations between the peripheral blood and the inflamed areas of the brain in patients with BD I.

The following study has been performed to evaluate the infiltration of T lymphocytes in the Cingulate Cortex (CC) of patients with BD I.

An histological analysis of brain specimens taken from the superior Anterior Cingulate Cortex (sACC) and ventral Anterior Cingulate Cortex (vACC) areas has been performed. Then, it has been measured the expression of two inflammatory markers, CD161 and CD45RA, typical of the terminally differentiated effector memory (T_{EMRA}) and other infiltrating T lymphocytes. Finally, the expression of CD45RA and CD161 genes have been compared between patients and healthy controls using statistical analysis.

The results of this work show an important increase of T_{EMRA} in the ACC of individuals with BD I with respect to healthy controls. This could corroborate the hypothesis that inflammation can contribute to the development of the disease. However, further investigations are needed to clarify the relation between T lymphocytes infiltration and brain structural modifications that characterize patients with BD I.