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Dopaminergic agonist pramipexole enhances regulatory T cell response after one-year treatment in Parkinson disease patients

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Statement of the Problem: The loss of dopaminergic neurons in Parkinson disease (PD) is associated to a neuro inflammatory process leading to neurodegeneration. PD treatment compensates the lack of dopamine resulting from the death of dopaminergic neurons. The dopaminergic agonist pramipexole has shown a neuroprotective effect by mechanisms yet unknown. On the other hand, several studies have shown that both inflammatory and anti-inflammatory immune cells can be stimulated by dopamine and dopaminergic agonists. In fact, regulatory T cells (Tregs), critical in restraining the inflammatory response, express dopamine receptors. This work is aimed to determine the effect of pramipexole on Treg-mediated immunoregulatory responses.

Methodology & Theoretical Orientation: 30 untreated PD patients (scoring 1-2 in the Hoen & Yar scale, H&Y) and 22 healthy controls were included. Peripheral blood samples were taken at inclusion and one year after treatment. The levels of classical CD4+CD25+FOXP3+CD127low/-, resting CD4+CD45RO-FOXP3low, non-Tregs CD4+CD45RO+FOXP3med, activated CD4+CD45RO+FOXP3hi, Tr1 CD4+CD25hiIL-10+ and Th3 CD4+CD25hiTGF-beta+Treg cells were determined. Patient status was assessed by the UPDRS and H&Y scales. Treatment consisted of the dopamine agonist pramipexole alone or combined with levodopa.

Findings: At inclusion, PD patients showed significantly lower levels of classical, activated and Tr1 Tregs than controls. After treatment, no differences were found in these populations between patients and controls. As expected, the patients showed a significant increase in the levels of classical, activated, and Tr1 cells after treatment. A decrease in the scores of the UPDRS and H&Y scales was also observed.

Conclusion & Significance: PD is characterized by decreased Treg levels. Our results at inclusion show the specific subpopulations diminished (classical, activated, and Tr1). Treatment promoted an increase in Treg levels, suggesting a recovery in the regulatory immune response that could contribute to suppress neurodegeneration-related inflammatory processes. Interestingly, Treg recovery coincided with decreased scores in the UPDRS and H&Y scales.

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