Thalamic ^{123}I FP-CIT uptake in a patient with clinical diagnosis of Parkinson's disease and depression

Abstract

In Parkinson's disease (PD), the central pathologic process is a rather selective degeneration of the dopaminergic neurons in the pars compacta of the substantia nigra, leading to anterograde loss of the ascending nigrostriatal projections and their nerve endings. However it is becoming increasingly evident that many of the basic non-motor phenomena (e.g., psychiatric, cognitive, and autonomic features) may be caused by involvement of non-dopaminergic neurotransmitters, including serotonin [5-hydroxytryptamine (5HT)], norepinephrine, and acetylcholine. A 70 years old man was referred to our center for a further worsening of both depressive and motor symptomatology. No signs of cognitive impairment have been found after neuropsychological examination. Before patient's discharge, a levodopa test (125mg) has been performed with good response: basal unified Parkinson's disease rating scale motor section **(**UPDRS III**)** score was 42; after levodopa administration UPDRS III the score was 24. The presence of a focal area of extrastriatal ^{123}I-FP-CIT binding was evident examining images. By means of using the co-registered anatomic mapping CT, the anatomic correspondence of this area was the left thalamus, homolateral to the site of lower presynaptic projections impairment in semi-quantitative analysis. Even if a depressive symptomatology was one of the main clinical features of our patients, our data were consistent with a thalamic overexpression of 5-HTT rather than its reduction, as reported by others. *In conclusion*, the study of 5-HTT in thalamus could be of interest in PD for its possible role in the physiopathological basis of the illness, in the differential diagnosis and for possible therapeutic strategy.

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