Lewy pathology is not the first sign of degeneration in vulnerable neurons in Parkinson disease

The goal: To determine whether Lewy body pathology is a marker of neuronal degeneration in Parkinson disease.

Methods: In a study of 11 cases of Lewy body disease and 20 cases of Parkinson disease, we assessed the number and density of Lewy bodies in the substantia nigra and compared them with controls with similar dopamine levels and pathology.

Results: Lewy bodies were present in a similar percentage of Parkinson disease patients and controls, and the density of Lewy bodies correlated with the degree of neuronal loss.

Conclusions: These results do not support the theory that Lewy body disease is the first sign of neuronal degeneration in Parkinson disease.

Neurology® Arabic Translation
doi: 10.1212/WNL.0b013e318278fe32
December 11, 2012 vol. 79 no. 24 2307-2314
Objective: To determine whether evidence of neuronal dysfunction or demise preceded deposition of Lewy pathology in vulnerable neurons in Parkinson disease (PD).

Methods: We examined the extent of nigral dysfunction and degeneration among 63 normal, incidental Lewy body disease (ILBD), and PD cases based on tyrosine hydroxylase (TH) immunoreactivity and neuron densities, respectively. The relationship between these markers and Lewy pathology (LP) burden in the substantia nigra (SN) and Braak PD stage was assessed.

Results: Compared with normal subjects, ILBD cases displayed a significantly higher percentage of TH-negative cells and lower neuronal densities in the SN as early as Braak PD stages 1 and 2, before LP deposition in the nigrostriatal system. ILBD nigral neuron densities were intermediate between normal subjects and PD cases, and TH-negative percentages were higher in ILBD than either normal or PD cases. Furthermore, neuron density and neuronal dysfunction levels remained relatively constant across Braak PD stages in ILBD.

Conclusions: These results suggest that significant neurodegeneration and cellular dysfunction precede LP in the SN, challenging the pathogenic role of LP in PD and the assumption that ILBD always represents preclinical PD.

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