Progressive Dementia with ‘Diffuse Lewy-Type Inclusions’ in Cerebral Cortex

A Case Report

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Summary. A 69-year-old male suffering from progressive dementia died 3 years after the beginning of his disease. The neuropathology of this case revealed the coexistence of senile changes, typical for Alzheimer’s disease, and the characteristics of Parkinson’s disease, namely, numerous senile plaques and neurofibrillary tangles in the cerebrum and neuronal loss with depigmentation in the substantia nigra and locus caeruleus. Lewy-type inclusions were distributed not only in the pigmented brain stem nuclei, but also diffusely in the CNS. The close nosological relationship between paralysis agitans and Alzheimer’s disease is discussed.

Key words: Lewy bodies – Dementia – Senile changes – Parkinson’s disease.


Introduction

Parkinsonism with dementia (Alvord et al., 1974; Hakim and Mathieson, 1979), or conversely, dementia with parkinsonism (Pearce, 1974) are frequently reported. In our case of progressive dementia, neuropathological findings are characterized by numerous Lewy-type inclusions in the cerebral cortex in addition to the well-known changes of Alzheimer’s and Parkinson’s diseases.

Case Report

The patient (a 69-year-old male) had spent his last 2 years, up to the time of hospitalization, with his daughter’s family. During this time slight forgetfulness had been noticed which had progressed gradually. At first, the patient was capable of harmonious integration into the family system, but approximately 6 months before his death his condition worsened rapidly; exhibiting confusion, disorientation, and delirium, especially at night. A month later, he was admitted as an ambulatory inpatient to a mental hospital. On admission, the patient seemed to be alert but restless, he was incontinent and disoriented, and was confused in speech. His mood was rather euphoric. Neurological examination revealed no particular abnormalities. The physical history only mentioned a record of chronic bronchitis with emphysema. There was no history of hypertension. Hepatomegaly and sinus rhythm with extrasystoles were found. He had a habitual intake of two or three 0.331 bottles of beer per day before his hospitalization. Clinical diagnosis was early senile dementia and suspected predelirant state resulting from alcohol abuse. The physical and mental condition of the patient deteriorated further and he was confined to bed for about 3 months before his death. During this time, somnolentia had alternated with intervals of conscious state with aggressiveness. Later he suffered from recurrent attacks of fever due to therapy-resistant bronchitis and decubitus. He died of circulatory failure caused by septic fever, approximately 3 years after the first memory disturbances.

Neuropathological Findings

Only the brain was available for examination. The brain weighed 1292 g after 4% buffered formalin fixation. There were mild frontal, temporal, and parietal atrophies on the cerebral convexities. The basilar arteries indicated no arteriosclerotic change. Upon coronal section, the atrophy was found to be more predominant in the temporal region. The lateral ventricle was remarkably dilated. No obvious macroscopic changes were noticed in the mammillary bodies, substantia nigra, locus caeruleus, or elsewhere. Histological findings were itemized as follows:

1. Senile Changes. Many senile plaques and neurofibrillary tangles were distributed in the temporal region and were especially numerous in the fusiform parahippocampal and hippocampal gyri (Fig. 2), but not in Sommer’s sector. These changes were also sporadically found in other cerebral-cortical regions. Abundant nerve cells with granulovacuolar degeneration were seen in Ammon’s horn. In the subcortical nuclei only the amygdaloid nucleus and the supraoptic part of the hypothalamic region were affected by numerous neurofibrillary tangles.