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Sporadic adult-onset neurodegeneration with brain iron accumulation: a distinct nosological entity. Neurodegenerative diseases are characterized by selective loss of neuronal subpopulations, mainly characterized by progressive brain atrophy and specific motor and cognitive deficits. In late stages, the neuronal loss is accompanied by abnormal glial tissue reactivity and the development of Alzheimer-type plaques, tau- and ubiquitin-containing neurofibrillary tangles, as well as amyloid- β (A β) deposits, mainly found in the neocortex. The majority of these neuropathological changes are morphological hallmarks of sporadic Alzheimer disease (AD). A small number of neurodegenerative disorders are characterized by neuronal loss and brain iron deposition. To date, only the neurodegenerative diseases Friedreich ataxia, Niemann-Pick type C disease, hereditary spastic paraplegia, and neuroaxonal dystrophy are known to have underlying causes of degeneration due to deficiency of proteins implicated in iron homeostasis. All these disorders have autosomal recessive inheritance and are characterized by adult-onset degeneration with clinically evident progressive disease. The term adult-onset motor neuron disease (AOMD) has been used to describe this group of disorders due to clinical and pathological homogeneity. In 2009, sporadic adult-onset (AO) progressive supranuclear palsy (PSP) was reported in patients with abnormal accumulation of iron and ferritin in neurons and glial cells. Later, a number of non-neurological (mechanistic) disorders with

spongiform degeneration and iron deposits in the brain were also described. The consensus is that the development of these non-neurological disorders depends on the genetic background of the affected person. Based on these data, it can be concluded that not all patients with progressive AOMD present brain iron accumulation, and therefore 6d1f23a050

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