Neuropathology of Alzheimer’s disease

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Alzheimer's disease is a complex neurodegenerative disorder characterized by progressive cognitive decline and memory impairment. The neuropathology of Alzheimer's disease involves several key features, including the presence of amyloid plaques, neurofibrillary tangles, and neuronal loss.

Amyloid plaques's

Amyloid plaques are abnormal protein deposits that accumulate between nerve cells in the brain. These plaques primarily consist of a protein called beta-amyloid, which is derived from the breakdown of a larger protein called amyloid precursor protein (APP). In a healthy brain, beta-amyloid is cleared away and broken down, but in Alzheimer's disease, there is an imbalance between the production and clearance of beta-amyloid, leading to its accumulation. The beta-amyloid aggregates into plaques, which can disrupt communication between neurons and contribute to inflammation and cell death.

Neurofibrillary tangles

Neurofibrillary tangles are another hallmark feature of Alzheimer's disease. These tangles are formed by the abnormal accumulation of a protein called tau, which is normally involved in stabilizing the internal structure of neurons. In Alzheimer's disease, tau proteins become hyperphosphorylated, causing them to aggregate and form tangles within the neurons. These tangles disrupt the normal functioning of the neurons and contribute to their degeneration and eventual death.

Neuronal loss

In addition to the presence of amyloid plaques and neurofibrillary tangles, Alzheimer's disease is also characterized by significant neuronal loss. As the disease progresses, there is a gradual and widespread degeneration of neurons, particularly in brain regions involved in memory, such as the

hippocampus. The loss of neurons results in a progressive decline in cognitive function and the development of symptoms associated with Alzheimer's disease.

Other pathologies in Alzheimer Disease

The neuropathology of Alzheimer's disease is not only limited to these three features. Other pathological changes, such as inflammation, oxidative stress, and synaptic dysfunction, also contribute to the progression of the disease. Moreover, there is a complex interplay between these pathological changes, making the understanding of Alzheimer's disease a continuing area of research.