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Biological and Morphological Changes of the Brain in Alzheimer's Disease I

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Annotation: We review the key neurobiological processes observed in the development of Alzheimer's disease, including beta-amyloid deposition, pathological transformation of tau protein, disruption of synaptic connections, inflammatory responses, and structural atrophy. We consistently describe the biophysiological changes that occur in the early and late stages of the disease.

Keywords: Alzheimer's disease, beta-amyloid, amyloid plaques, tau protein, neurofibrillary tangles, microglial cells, synaptic dysfunction, inflammation, cognitive decline.

Alzheimer's disease is a complex neurodegenerative disease that causes degenerative changes in brain tissue, and is most common in older adults, particularly those over the age of 65. The symptoms of the disease include a decline in memory, thinking, speech, and activities of daily living, which progresses rapidly and continuously. There is no complete medical cure for Alzheimer's, but a deeper understanding of the cognitive and biological processes of the disease may help to detect the disease at an early stage and, if possible, slow it down.

In Alzheimer's disease, morpho-functional changes occur in various parts of the brain. They have several main processes:

Beta- amyloid accumulation and amyloid plaques - Beta- amyloid protein from APP (amyloid precursor protein) molecule harvest will be. Simple in case, A β molecules quickly metabolism to enter and disappear need But in Alzheimer's, A b of excess working release and accumulation is observed. A β collection first in line extracellular in the spaces (i.e., neurons between) exists will be. Such accumulations **amyloid plaques** as known structures harvest does. This disease main estimated reasons to the point enters.

Amyloid plaques:

1. Increases synaptic connections, which leads to a decline in **memory and cognitive abilities.**

2. Activates microglial cells, which in turn initiate inflammatory cascades.

Oxidative stress leading to cell death and increases **metabolic imbalance.**

The first plaques begin to form in the early stages of the disease, and later this process becomes clinically noticeable.

Tau protein and neurofibrillary tangles are - Tau Tau protein is a structural component of the internal skeleton of neurons, keeping them in their proper shape. However, in Alzheimer's, tau protein is **hyperphosphorylated,** this protein excess and its normal functions breaks down. As a result, tau protein neurons inside **neurofibrillary tissues (NFT)** is formed, this and neurons internal at the level destabilization does.

The role of tau protein and NFT:

a) Axonal transport is impaired, meaning the transmission of nutrients and chemical signals between neurons is slowed or stopped.

b) The internal metabolism of cells is disrupted, leading to the loss of neurons.

NFT formation and pathological formation of tau protein are associated with disease progression, a process that often causes significant damage to **the hippocampus and other cognitive centers of the brain.**

Inflammation and activation of microglial cells - In Alzheimer's, **microglial cells** - the brain's innate immune system - become overactive. This is mainly due to the accumulation of amyloid plaques. of collections increase and they with to fight attempt as a result to the surface Inflammation cells between and in the brain in tissues the damage strengthens, this and neurodegeneration accelerates.

Inflammatory processes:

a) Increased inflammatory cytokines,

b) High levels of microglial activity,

c) Increased cellular stress.

These processes not only cause local inflammation in the brain, but also lead to **synaptic disruptions** and neuronal death.

Selective degeneration of the hippocampus - The hippocampus is a brain structure that plays a central role in memory and learning processes. The hippocampus is the first to be damaged in AD. Neurons in the hippocampus eventually die and their structures are lost. This process is clinically manifested as short-term memory loss and difficulties in remembering new information.

Changes that occur in the hippocampus:

a) Early memory problems,

b) Decreased learning ability,

c) Weakening of the cardiovascular system.

Energy system and metabolic imbalance - in Alzheimer's neurons energy exchange is also important importance has. Healthy brain for neurons regular accordingly glucose energy source as uses. However In the AK **mitochondria** (cell energy working issuer organelle) activity weakens, this and neurons for necessary was of energy lack of take Energy deficiency :

a) It enhances synaptic transmission,

b) Accelerates cognitive decline,

c) Increases oxidative stress.

Changes in brain structures

Shrinkage of the cerebral cortex - Alzheimer's disease causes significant atrophy of the cerebral cortex. In the early stages, this is only noticeable in certain regions, but as the disease progresses, this process involves the entire cerebral cortex.

The main areas of atrophy in the cortex are:

a) **Frontal cortex** - decision-making and personality changes;

b) **Parietal cortex** - disorientation in time and space;

c) **Temporal cortex** - control of memory and linguistic functions

Ventricles and brain Volume - In AD, **the ventricles** dilate and the brain volume decreases. MRI scans can show this process as enlarged ventricles and thinning of the cerebral cortex.

a) **Ventricular enlargement** is a sign that the brain has decreased in size;

b) **Hippocampal atrophy** - indicates the severity of memory problems.

Conclusion: The biological and morphological changes that occur in Alzheimer's disease affect the structure and function of the brain in a wide range of ways. The main disease processes - amyloid deposition, tau protein changes, microglial inflammation, synaptic and structural changes - are the main components of the disease.

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