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Perspective

Pathophysiology of Alzheimer's Disease

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DESCRIPTION

Alzheimer's disease (AD) is the most prevalent form of dementia that affects at least 27 million individuals worldwide and represents for 60% to 70% of all dementia cases. In addition to having a significant economic impact on society, the prevalence of this condition has a significant impact on the patient's family. From an anatomopathological point of view, AD is characterized by two prototypical lesions.

1) Senile plaques, which are extra-cellular lesions composed of a nucleus of amyloid protein accumulation and

2) Intraneuronal neurofibrillary tangles composed of phosphorylated tau protein (P-tau).

Pathophysiology

The accumulation of amyloid protein can also damage capillary walls, arteries, and arterioles, resulting in amyloid cerebral angiopathy, which reduces blood flow and promotes vascular wall component degradation while increasing the risk of intraparenchymal haemorrhages. The main characteristics of AD are extracellular plaques of the insoluble peptide -amyloid and Neuro Fibrillary Tangles (NFT) of P-tau in the cytoplasm of neurons. The deposits are considered to produce atrophy and death of neurons due to excitotoxicity processes (excessive stimulation of neurotransmitter receptors in neuronal membranes), collapse in calcium homeostasis, inflammation, and depletion of energy and neuronal factors. Although the mechanisms by which these changes lead to cognitive decline are still under the process. The aforementioned cognitive decline is the consequence of this process, which affects the neurons and synapses involved in memory processes, and other cognitive activities. The main event that causes the disease, according to the amyloid cascade theory (one of the most widely accepted theories about AD pathogenesis), is the cerebral accumulation of Aβ-peptide, which results from an imbalance between the production and clearance of this protein. The Amyloid Precursor Protein (APP), a naturally produced protein that is crucial formaintaining brain homeostasis, and is converted into the 36-43 amino acids long Aβ peptides. The APP gene is situated on chromosome 21, which explains, why Down syndrome patients and individuals with APP gene region duplication-an uncommon form of early onset of hereditary origin have greater rates of early-onset AD. It is thought that increased APP expression causes cerebral Aβ-peptide levels to rise and subsequently leads to its deposition. A non-amyloidogenic secretase-mediated pathway and an amyloidogenic and secretase-mediated pathway are currently recognised as the two primary mechanisms for APP processing. When APP is broken down by -secretase, a soluble molecule called sAPP is produced. This molecule probably has neuroprotective properties, as it aids in the survival and plasticity of neurons as well as their protection against excitotoxicity. Aβ-secretase breaks down APP to produce the Aβ peptide. In this mechanism, secretase inactivates APP to produce a soluble fragment of APP (sAPP, a mediator of neuronal death), as well as a carboxy-terminal complex associated with cell membrane. APH-1 (formerly known as pharynx-defective-1) and PEN-2 (Presenilin Enhancer-2) are the four proteins that constitute the-secretase complex, which cleaves and produces the A peptide. The 40 amino acid form (Aβ 40), followed by the 42 amino acid form (Aβ 42), and dominates the generation of Aβ peptides, which range in size from 38 to 43 amino acids. The amyloidogenic and non-amyloidogenic pathways exist in equilibrium during physiological circumstances, with the latter being promoted more than the prior.

CONCLUSION

Alzheimer's disease is the most common type of dementia and typically manifests through a progressive loss of memory and cognitive function. Acquired factors such as cerebrovascular diseases, diabetes, hypertension, obesity and dyslipidemia increase the risk of AD development.

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