subjects underwent neuroimaging (MRI), genetic testing for NOTCH3 mutations, and cerebrospinal fluid analysis. The severity of vestibulo-ataxic syndrome was assessed using the Scale for the Assessment and Rating of Ataxia (SARA), and cognitive function was evaluated through the Mini-Mental State Examination (MMSE).

The results of the research and their discussion

MRI findings demonstrated widespread white matter hyperintensities in all cases, predominantly in periventricular and subcortical regions. Five patients exhibited the characteristic anterior temporal lobe involvement associated with CADASIL, while three had deep lacunar infarcts suggestive of small-vessel disease. Genetic testing confirmed NOTCH3 mutations in four individuals, supporting a CADASIL diagnosis. The remaining six patients were categorized as having leukoencephalopathy of unknown origin. SARA scores correlated with disease progression, with decompensated cases showing significant gait disturbances. Cognitive decline was prominent in CADASIL-positive cases, with MMSE scores averaging 18/30. The findings highlight the challenge of diagnosing CADASIL solely based on clinical and imaging features, emphasizing the importance of genetic testing.

Conclusions

Leukoencephalopathy of unspecified genesis presents diagnostic difficulties, particularly when associated with vestibulo-ataxic syndrome and progressive neurological decline. CADASIL remains an important differential diagnosis, with genetic testing serving as a crucial tool for confirmation [2]. Early identification and supportive management are essential in mitigating disease progression and improving patient outcomes.

LITERATURE

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COMPARING THE EFFECTS OF EARLY-ONSET VS. LATE-ONSET ALZHEIMER'S DISEASE, A PROGRESSIVE NEURODEGENERATIVE DISORDER WORLDWIDE

Introduction

Alzheimer's disease (AD), a degenerative neurological disorder characterized by behavioral problems, memory loss, and cognitive decline, is the most common type of dementia in the world. In this progressive neurodegenerative disease, beta-amyloid plaques and neurofibrillary tangles accumulate, compromising neuronal function and leading to widespread brain atrophy. Beta-amyloid, particularly the lethal Aβ42 form, builds up between neurons to form plaques that interfere with synaptic transmission and cellular metabolism.

Meanwhile, abnormal tau protein breaks away from microtubules to form tangles inside neurons, which further disrupt synaptic function by obstructing nutrition transfer.

These degenerative alterations first damage memory-related areas such the entorhinal cortex and hippocampus before influencing behaviour, language, and cognitive functions in the cerebral cortex. Significant cognitive deterioration results from brain atrophy, which enlarges when neurones die and lose connections. Additionally, because dysfunctional microglia and astrocytes release toxic inflammatory chemicals instead of eliminating amyloid plaques and cellular debris, prolonged inflammation caused by these cells worsens neuronal damage [1].

Reduced blood flow, blood-brain barrier dysfunction, and altered glucose metabolism are examples of vascular contributions that exacerbate neurodegeneration by impairing the brain's capacity to remove harmful proteins and depriving it of oxygen and nourishment.

Inflammation, vascular damage, tau dysfunction, and amyloid pathology all play a part in Alzheimer's disease's progressive and ultimately lethal course.

While the majority of Alzheimer's cases (also known as late-onset Alzheimer's disease, or LOAD) affect individuals 65 and older, a sizable portion of patients experience early-onset Alzheimer's disease (EOAD) symptoms prior to that age. Recent research indicates that EOAD and LOAD may have different clinical presentations, progressions, and underlying molecular pathways, despite sharing fundamental pathological characteristics like tau neurofibrillary tangles and amyloid-beta plaques [2].

Goal

Compare and analyze the differences between Early-Onset Alzheimer's Disease (EOAD) and Late-Onset Alzheimer's Disease (LOAD) in terms of cognitive performance, neuropathological changes, disease progression, and demographic trends.

Material and methods of research

Data was gathered from research studies worldwide that report on cases that compare and analyze early-onset and late-onset of Alzheimer's Disease.

The results of the research and their discussion

Research shows that EOAD and LOAD may manifest different clinical findings, underlying molecular pathways and progressions even with sharing basic pathological characteristics like tau neurofibrillary tangles and amyloid-beta plaques.

Not only age contributes to the contrast among EOAD and LOAD, but other factors also play a significant role. Such as the pathophysiology of the disease, psychological variables a genetic predisposition. For example, EOAD is frequently linked to a more aggressive course of the illness and a higher prevalence of cognitive abnormalities including language and visuospatial issues that are not related to memory. Alternatively, LOAD often appear and do exists with age-related diseases and shows more severe memory-related symptoms.

Comprehending the differences between EOAD and LOAD is very important for developing diagnostic and treatment plans based on the patient's individuality. Special requirements of EOAD patients may get ignored Since most of the current methods of diagnosis, treatments are based on LOAD researches and findings.

Furthermore, the emotional toll of EOAD emphasizes the significance of particular interventions and support systems because it frequently impacts individuals in their prime working years. Our study intends to improve our knowledge of Alzheimer's disease and guide the development of specialized treatments for these various patient populations by clarifying the parallels and discrepancies between EOAD and LOAD. Additionally, the emotional toll of EOAD emphasizes the significance of particular interventions and support networks, as it frequently impacts individuals in their prime working years. Our study intends to improve our knowledge of Alzheimer's disease and guide the development of specialized treatments for these various patient populations by clarifying the parallels and discrepancies between EOAD and LOAD.

Patients with EOAD frequently have worse baseline cognitive function than those with LOAD when comparing cognitive performance at diagnosis. At the time of diagnosis, this involves more severe deficits in language, attention, memory, and visuospatial skills [2]. Additionally, LOAD patients usually have shorter lifespans, maybe due to age-related comorbidities, but EOAD patients usually survive longer after diagnosis, probably because of their earlier physical health. More extensive and severe neuronal loss in both cortical and subcortical regions is linked to neuropathological abnormalities in EOAD patients.

Neurochemical changes affect neurons containing GABA, somatostatin, norepinephrine, and LOAD in addition to the cholinergic system. Although neuronal loss and neurochemical changes are possible, they are usually not as severe as in EOAD [3].

Five to ten percent of all instances of Alzheimer's disease are EOAD patients, and the proportion of LOAD patients rises with age [2] The number of people suffering from dementias, such as Alzheimer's disease, rose from 0.736 million to 0.77 million between 1990 and 2021. Among men, the figure rose from 0.571 million to 0.589 million. Age-related differences exist in the incidence rates of EOAD patients [3]. The incidence is roughly 20.5 per 100,000 person-years for those between the ages of 30 and 64. For people 45–64 years old, this rate rises to 33.7 per 100,000 person-years. The incidence rates of LOAD patients in Europe are as follows: 3.4 for individuals 65–74 years old, 13.8 for individuals 75–84 years old, and 35.8 for individuals over 85.

Although EOAD and LOAD share many basic clinical characteristics, EOAD is distinguished by unique neuropathological alterations, a quicker rate of decline, and more severe early cognitive deficits. The aforementioned statistics illustrate the rising incidence of Alzheimer's disease across the globe and the necessity of early identification, efficient therapies, and continued research to manage and impede the disease's progression [2].

Conclusions

In conclusion we can see significant differences between Early-Onset Alzheimer's Disease (EOAD) and Late-Onset Alzheimer's Disease (LOAD) in terms of cognitive functions the progression of the disease and the neuropathological modifications.

When it comes to cognitive functions, Language, attention, memory, and visuospatial impairments are more severe in EOAD patients. Furthermore, a significant amount of neurotransmitter systems is impacted by changes of neuro chemical and death of the neurons. Even though EOAD patients are younger and have a better physical health compared to LOAD patients they undergo accelerated cognitive decline. Majority of Alzheimer's cases are LOAD and have a shorter survival rate according to statistics. And that is probably because of the impact of the age.

The growing incidence rates of Alzheimer's disease across age groups, which show the disease's increasing global prevalence, underscore the need for early identification, targeted therapy interventions, and ongoing research to better understand the distinct mechanisms behind EOAD and LOAD. These differences must be taken into consideration while creating ailored treatment programs and improving patient outcomes for those suffering from Alzheimer's disease. To reduce the growing burden of this debilitating condition, research and clinical treatment must continue.

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