Alzheimer's Disease has a High Rate of Early Onset

Maryem Bezine*

Editorial Office, Neurology and Neurorehabilitation, Germany

Corresponding Author*

Maryem Bezine

Editorial Office

Neurology and Neurorehabilitation

Germany

E-mail: nneurorehabilitation@gmail.com

Received date: 12-January-2022; Manuscript No. NNR-22-52255; **Editor assigned:** 14-January-2022; Pre QC No. NNR-22-52255(PQ); **Reviewed:** 15-January-2022; QC No. NNR-22-52255(Q); **Revised Date:** 20-January-2022; Manuscript No. NNR-22-52255(R); Accepted: 26-January-2022; **Published:** 29-January-2022; DOI: 10.37532/nnr.22.4.1.5-6

Abstract

While late-onset Alzheimer's disease Late-Onset Alzheimer's Disease (LOAD) is more common, Early-Onset Alzheimer's Disease (EOAD) with onset before 65 years of age differs significantly from LOAD. EOAD accounts for around 5% of all cases of Alzheimer's disease and is linked to delayed diagnosis, an aggressive course, and age-related psychosocial demands. In addition to cognitive impairments, the phenotypic variations feature atrophy and white matter alterations that match physically to the cognitive changes, and they appear to involve different brain networks than conventional Alzheimer's Disease (AD). The treatment of EOAD is similar to that of LOAD, but more attention should be paid to the specific cognitive regions implicated, as well as more age-appropriate psychosocial support and education. Early-onset Alzheimer's disease has many of the same symptoms as "late-onset" Alzheimer's disease and is not caused by known genetic abnormalities. Persons in their 30s or 40s can acquire non-familial early-onset AD, but this is exceedingly unusual; most people affected are in their 50s or early 60s. The treatment of EOAD is similar to that of LOAD, but more attention should be paid to the specific cognitive regions implicated, as well as more age-appropriate psychosocial support and education.

Keywords: Early-onset Alzheimer's disease • Alzheimer's disease • Late-onset Alzheimer's disease • lvPPA

Introduction

The most frequent kind of early-onset neurodegenerative dementia is EOAD. According to the few epidemiological studies on EOAD, the vast majority of cases are non-familial, accounting for about 4%-6% of all AD with an annual incidence rate of about 6.3/100,000 4 and a prevalence rate of about 24.2/100,000 in the 45-64year age group 5, resulting in between 220,000 and 640,000 Americans. As patients approach the age of 65, the incidence and prevalence rates of EOAD increase exponentially. Early childhood cognitive development includes the development of thinking, attention, memory, and problem solving skills, all of which aid children in their understanding of the world around them. Early childhood development, which is mentioned as a fundamental component of Sustainable Development Goal-4, is the basis of adult health and welfare. Alzheimer's disease that develops before the age of 65 is known as early-onset Alzheimer's disease or younger-onset Alzheimer's disease. About 60% have a positive family history of Alzheimer's disease, and 13% are inherited as an autosomal dominant trait. As the condition advances, the patient begins to experience increasingly significant symptoms, such as mood swings and the inability to do sophisticated tasks like driving. Confusion, poor judgement, language difficulty, agitation, withdrawal, hallucinations, seizures, Parkinsonian characteristics, increased muscular tone, myoclonus, incontinence, and mutism are some of the other prevalent symptoms. A citation to a medical source is required. They lose their ability to conduct simple tasks, such as combing their hair, and require full-time care in their latter phases. There has been evidence of a link between chronic stunting and cognitive development. A recent meta-analysis of 29 LMICs found a link between linear growth and cognitive development in the first two years of life, but the meta-analysis was unable to incorporate environmental, educational, or follow-up data to describe the link between stunting and cognitive impairment. Early-onset dementia is related with psychological issues such as the impacts of unanticipated loss of independence, grieving, and a sense of "out-of-step" deterioration in middle age, difficulties managing ongoing duties, and relatively retained insight with sadness and anxiety. EOAD patients, on the other hand, had less general comorbidities such diabetes, obesity, and cardiovascular problems than LOAD patients.

Phenotypes of Variant EOAD

Non-amnestic phenotypic variants of EOAD, such as logopenic variant primary progressive aphasia, posterior cortical atrophy, progressive ideomotor apraxia, behavioral/dysexecutive AD, corticobasal syndrome, and others, affect up to 50% of patients with the disease. One of the most notable features of EOAD is that it manifests itself as a variety of non-amnestic, variable phenotypes, which may support its classification as "Type 2" AD. The young tail of the normally distributed age of AD onset curve is represented by these variants [1]. Non-amnestic phenotypic variants of EOAD, such as logopenic variant primary progressive aphasia, posterior cortical atrophy, progressive ideomotor apraxia, behavioral/dysexecutive AD, corticobasal syndrome, and others, affect up to 50% of patients with the disease. Non-amnestic variant phenotypes account for 22%-64 % of EOAD cases, and they differ from typical amnestic AD (either EOAD or LOAD) not only in non-memory presentations but also in the lower prevalence of the Apolipoprotein E (APOE) 4 allele and early posterior cortical Neurofibrillary Tangles NFTs with hippocampal sparing. Others claim that a biparietal phenotype with Progressive Ideomotor Apraxia (PIA), as well as visuospatial and other deficits, is a common form of EOAD. The presence of a behavioral/dysexecutive variation, commonly referred to as "frontal variant AD50," is highlighted in the literature. Furthermore, up to 25% of patients with corticobasal syndrome, which is characterised by progressive limb apraxia and motor changes, have AD at autopsy [2].

Primary Progressive Aphasia Logopenic Variant

When people with logopenic variation PPA (lvPPA, also known as PPA-L) communicate, they have trouble finding words. As a result, individuals may talk slowly and pause frequently while searching for the appropriate word. They can still remember the meanings of words, unlike persons with semantic variant Primary Progressive Aphasia (PPA). The gradual deterioration in language known as lvPPA is a significant EOAD phenotypic variation. Patients with this condition have trouble locating words, have reduced sentence repetition, and have irregularities in echoic memory, as well as phonological buffer deficiencies. The existence of episodic memory and visuospatial abilities difficulties helps to identify lvPPA from other PPAs [3]. Furthermore, individuals with lvPPA frequently have a history of dyslexia, indicating a pre-existing vulnerability in language networks. In one study, 25% of lvPPA patients reported delays in spelling to themselves or informants. Glial cells (particularly astrocytes and microglia), beta-amyloid, and proinflammatory chemicals are among [4] the key substances implicated in these pathways [5]. Connections between networks of neurons may break down as neurons are harmed and die across the brain, and numerous brain areas begin to shrink [6].

Conclusion

When compared to individuals with LOAD, patients with EOAD had more parietal atrophy, more white matter abnormalities, and less hippocampus volume loss. In comparable cognitive regions of the brain, the phenotypic variations show atrophy and white matter alterations. Patients with EOAD exhibit significant regional amyloid and tau buildup in the posterior neocortex on neuropathology. When compared to conventional AD, abnormal tau drives this neocortical pathology, with more posterior cortical NFTs per grey matter atrophy. There is a well-established link between stunting in early childhood and cognitive performance in later life. Early childhood cognitive development has been linked to biological and behavioural risk factors such as stunting, poverty, and a bad family

environment. The left hemisphere language regions in lvPPA and the visual neocortex in Posterior Cortical Atrophy (PCA) have a higher focal neocortical load of NFTs. In comparison to conventional AD, the variations tended to spare the hippocampi, and in later stages, the pattern of atrophy converged across the variants.

References

1. Maurer, K., et al. "Auguste D and Alzheimer's disease." Lancet 349.9064 (1997): 1546-1549.
2. Terry, R. D., & Peter D. "Dementia of the Alzheimer type." Annu Rev Neurosci 3.1 (1980): 77-95.
3. Bertram, L., & Rudolph E. T. "Thirty years of Alzheimer's disease genetics: The implications of systematic meta-analyses." Nat Rev Neurosci 9.10 (2008): 768-778.
4. Zhu, X. C., et al. "Rate of early onset Alzheimer's disease: A systematic review and meta-analysis." Ann Transl Med 3.3 (2015).
5. Bickel, H., et al. "Presenile dementia in memory clinics-incidence rates and clinical features." Nervenarzt 77.9 (2006): 1079-1085.
6. Harvey, R. J., et al. "The prevalence and causes of dementia in people under the age of 65 years." J Neurol Neurosurg Psychiatry 74.9 (2003): 1206-1209.

Cite this article: Bezine M. Alzheimer's Disease has a High Rate of Early Onset. Neurol Neurorehabilit. 2022, 4 (1), 001-004.