Mild cognitive impairment

Mild Cognitive Impairment (MCI) is a condition characterized by a noticeable decline in cognitive abilities, such as memory, thinking, and language skills, that is greater than expected for a person's age and educational background. However, the changes in cognition are not severe enough to interfere significantly with daily functioning or independence.

MCI is considered an intermediate stage between the cognitive changes associated with normal aging and the more serious decline seen in dementia, such as Alzheimer's disease. While not everyone with MCI will develop dementia, it is recognized as a risk factor and an early sign of potential progression to dementia.

Types

Amnestic MCI: This subtype primarily affects memory, leading to difficulties in remembering recent events or important information.

Non-amnestic MCI: This subtype involves impairments in other cognitive domains, such as language, attention, executive function, or visuospatial skills.

Etiology

The exact causes of MCI are not fully understood, but several factors may contribute to its development, including age, genetics, certain medical conditions (e.g., cardiovascular disease, diabetes), lifestyle factors (e.g., lack of physical exercise, smoking), and certain medications. However, MCI can also occur without an identifiable cause.

It is essential for individuals with MCI to undergo a comprehensive evaluation by a healthcare professional, typically a neurologist or geriatrician, to determine the underlying cause and monitor any potential progression.

Treatment

Treatment options for MCI include lifestyle modifications (e.g., regular physical exercise, cognitive stimulation, healthy diet), managing underlying health conditions, and in some cases, medications may be prescribed to manage specific symptoms.

The association of white matter hyperintensity burden with amyloid positivity and conversion to dementia in people with mild cognitive impairment (MCI) is unclear. The aim of the study was to expand on this research by examining whether a change in white matter hyperintensity burden over time differs in amyloid-negative (Aβ-) and amyloid-positive (Aβ+) people with MCI who either remain stable or convert to dementia. To examine this question, they compared regional white matter

hyperintensity burden in four groups: amyloid positive (Aβ+) progressor, amyloid negative (Aβ-) progressor, amyloid positive (Aβ+) stable, and amyloid negative (Aβ-) stable.

Participants with MCI from the Alzheimer's Disease Neuroimaging Initiative were included if they had APOE ε4 status and if amyloid measures were available to determine amyloid status (i.e., amyloid positive, or amyloid negative). Participants with a baseline diagnosis of MCI, had APOE ε4 information, and amyloid measures were included. An average of 5.7 follow-up time points per participant were included, with a total of 5054 follow-up time points with a maximum follow-up duration of 13 years. Differences in total and regional white matter hyperintensity burden were examined using linear mixed-effects models.

A total of 820 participants (55-90 years of age) were included in the study (Aß+ Progressor, n= 239; Aß- Progressor, n= 22; Aß+ Stable, n= 343; Aß- Stable, n= 216). People who were Aß- stable exhibited reduced baseline white matter hyperintensities compared to Aß+ progressors and Aß+ stable at all regions of interest (β belongs to [.20 -.33], CI belongs to [.03 -.49], p<.02), except Deep white matter hyperintensities. When examining longitudinal results, compared to Aß- stable, all groups had steeper accumulation in white matter hyperintensity burden with Aß+ progressors (β belongs to [-.03-.06], CI belongs to [-.05-.09], p<.01) having the largest increase (i.e., largest increase in white matter hyperintensity accumulation over time).

These results indicate that white matter hyperintensity accumulation contributes to conversion to dementia in older adults with mild cognitive impairment who are amyloid-positive and negative people [1)].

Mild cognitive impairment is a nonmotor complication in Parkinson's disease (PD) that have a high risk of developing dementia. White matter is associated with cognitive function in PD and the alterations may occur before the symptoms of the disease. Previous diffusion tensor imaging (DTI) studies lacked specificity to characterize the concrete contributions of distinct white matter tissue properties. This may lead to inconsistent conclusions about the alteration of white matter microstructure. Here, we used neurite orientation dispersion and density imaging (NODDI) and the white matter fiber clustering method to uncover local white matter microstructures in PD with mild cognitive impairment (PD-MCI). This study included 23 PD-MCI and 20 PD with normal cognition (PD-NC) and 21 healthy controls (HC). To probe specific and fine-grained differences, metrics of NODDI and DTI in white matter fiber clusters were evaluated using along-tract analysis. Our results showed that PD-MCI patients had significantly lower neurite density index (NDI) and orientation dispersion index (ODI) in white matter fiber clusters in the prefrontal region. Correlation analysis and receiver operating characteristic (ROC) analysis revealed that the diagnostic performance of NODDI-derived metrics in the cingulum bundle (2 clusters) and thalamic-frontal (2 clusters) were superior to DTI metrics. The study provides a more specific insight to uncover local white matter abnormalities in PD-MCI, which benefit understanding the underlying mechanism of cognitive decline in PD and predicting the disease in advance [2)].

1)
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2)
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