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Brain Age Gap in Neuromyelitis Optica Spectrum Disorders and Multiple Sclerosis

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Abstract

Neuromyelitis Optica Spectrum Disorders (NMOSD) and Multiple Sclerosis (MS) are distinct autoimmune diseases that primarily affect the central nervous system. While both conditions involve demyelination and inflammation, their underlying pathophysiological mechanisms and clinical manifestations differ significantly. One intriguing aspect that has garnered recent attention is the concept of brain age gap – a phenomenon where the brain's apparent age, as determined by neuroimaging techniques, deviates from the chronological age of an individual. This phenomenon has been observed in various neurological disorders and may provide valuable insights into the neurodegenerative processes occurring within the brain.

This review aims to explore the presence and significance of the brain age gap in NMOSD and MS. We will delve into the existing literature to comprehend how these disorders contribute to accelerated brain aging and whether the brain age gap holds diagnostic, prognostic, or therapeutic implications. By comparing the pathophysiological mechanisms, clinical presentations, and neuroimaging findings of NMOSD and MS, we seek to unravel whether the brain age gap is a shared feature or distinct between the two diseases. This investigation is crucial not only for advancing our understanding of disease mechanisms but also for developing tailored interventions that target the unique neurodegenerative processes occurring in NMOSD and MS.

Introduction

Neuromyelitis Optica Spectrum Disorders (NMOSD) and Multiple Sclerosis (MS) are autoimmune disorders that predominantly affect the central nervous system (CNS). NMOSD is characterized by severe attacks of optic neuritis and longitudinally extensive transverse myelitis, often involving the aquaporin-4 water channel autoantibody[1]. MS, on the other hand, is characterized by multifocal demyelination and inflammation in the CNS, leading to a wide range of neurological symptoms. Recent advances in neuroimaging techniques have unveiled a fascinating phenomenon known as the "brain age gap." This phenomenon involves the utilization of machine learning algorithms to estimate the age of an individual's brain based on structural and functional neuroimaging data. Comparing this estimated brain age with the individual's chronological age can reveal whether the brain appears older or younger than expected. The concept of brain age gap has gained attention as a potential biomarker of neurodegeneration and a predictor of cognitive decline in various neurological disorders, including Alzheimer's disease and Parkinson's disease.

In the context of NMOSD and MS, understanding the presence and significance of the brain age gap could provide valuable insights into the neurodegenerative processes occurring within the CNS. Given the distinct pathophysiological mechanisms and clinical manifestations of these disorders, investigating whether the brain age gap is a shared feature or varies between NMOSD and MS could have important diagnostic and therapeutic implications. Moreover, unraveling the relationship between disease activity, disability progression, and the brain age gap might shed light on the underlying factors driving accelerated brain aging in these disorders [2,3].

This review aims to comprehensively explore the current state of knowledge regarding the brain age gap in NMOSD and MS. By synthesizing findings from neuroimaging studies, clinical observations, and mechanistic research, we seek to elucidate whether the brain age gap can serve as a unifying marker of neurodegeneration or if it presents distinct patterns in these two autoimmune CNS disorders. Such insights could pave the way for the development of novel therapeutic strategies targeting the specific neurodegenerative processes associated with NMOSD and MS.

Methods

To investigate the brain age gap in Neuromyelitis Optica Spectrum Disorders (NMOSD) and Multiple Sclerosis (MS), a comprehensive literature search was conducted using electronic databases such as PubMed, Web of Science, and Google Scholar. The search was performed using relevant keywords and phrases, including "brain age gap," "neuromyelitis optica," "multiple sclerosis," "neuroimaging," "neurodegeneration," and "machine learning." Studies published up to the date of this review were considered for inclusion.

Inclusion criteria encompassed studies that utilized neuroimaging data to estimate brain age, compared brain age with chronological age, and examined the brain age gap in individuals with NMOSD and MS [4]. Studies that explored the association between the brain age gap and clinical outcomes, disease severity, or treatment responses were also included.

Discussion

The brain age gap, as revealed by neuroimaging techniques and machine learning algorithms, has emerged as a novel and promising marker of neurodegeneration in various neurological disorders. This phenomenon reflects the complex interplay between chronological

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aging, disease-related processes, and individual susceptibility to neuronal damage. In the context of NMOSD and MS, the presence and significance of the brain age gap offer intriguing insights into the underlying pathophysiological mechanisms of these autoimmune CNS disorders.

Shared and distinct features

Studies investigating the brain age gap in NMOSD and MS have unveiled both shared and distinct features. Shared characteristics include regions of the brain that exhibit accelerated aging in both disorders. These regions often coincide with areas vulnerable to inflammation and demyelination, suggesting that ongoing immune-mediated processes contribute to the observed discrepancies between brain age and chronological age. However, differences in the specific patterns and extent of accelerated aging between NMOSD and MS have also been reported. These differences might reflect disease-specific mechanisms, such as the involvement of aquaporin-4 autoantibodies in NMOSD and distinct immune pathways in MS [5].

Clinical implications

The brain age gap holds potential clinical implications for NMOSD and MS. In NMOSD, the presence of a pronounced brain age gap might signify a heightened risk of cognitive impairment or neurological decline. Integrating the brain age gap into disease assessment could aid in identifying individuals who require closer monitoring and more aggressive treatment strategies [6]. In MS, the brain age gap could serve as a prognostic marker for disease progression and disability accumulation. Additionally, the brain age gap might help in monitoring treatment responses and evaluating the efficacy of disease-modifying therapies.

Future directions

Further research is warranted to elucidate the mechanisms driving the brain age gap in NMOSD and MS. Longitudinal studies examining the evolution of the brain age gap over time could shed light on its dynamic nature and its relationship with disease progression [7-10]. Additionally, investigations into the genetic, immunological, and environmental factors influencing the brain age gap could provide a more comprehensive understanding of its determinants. The brain age gap in NMOSD and MS offers a unique perspective on the neurodegenerative processes occurring within the CNS. While both disorders exhibit accelerated brain aging, the specific patterns and underlying mechanisms differ, reflecting their distinct pathophysiological characteristics. Incorporating the brain age gap into clinical practice could enhance disease assessment, prognostication, and treatment strategies for individuals with NMOSD and MS. However, continued research is essential to unravel the intricate associations between the brain age gap, disease mechanisms, and clinical outcomes in these autoimmune CNS disorders.

Conclusion

The investigation of the brain age gap in Neuromyelitis Optica Spectrum Disorders (NMOSD) and Multiple Sclerosis (MS) has provided valuable insights into the complex interplay between neurodegenerative processes, immune-mediated mechanisms, and clinical outcomes within the central nervous system. Neuroimaging techniques coupled with machine learning algorithms have illuminated the potential of the brain age gap as a sensitive marker of accelerated brain aging, enabling a deeper understanding of these autoimmune disorders. While both NMOSD and MS demonstrate a notable brain age gap, the patterns and extent of accelerated aging exhibit both shared and distinct features. Regions vulnerable to inflammatory and demyelinating processes seem to be common targets of accelerated aging, underscoring the role of immune responses in driving neurodegeneration. However, the specific brain regions affected and the magnitude of the brain age gap appear to differ, reflecting the unique pathophysiological underpinnings of each disorder.

The clinical implications of the brain age gap are multifaceted. In NMOSD, a pronounced brain age gap could serve as an early warning sign of cognitive decline and neurological deterioration. Incorporating the brain age gap into clinical assessments might allow for tailored interventions and closer monitoring of those at higher risk. In the case of MS, the brain age gap holds promise as a prognostic indicator for disease progression and disability accumulation, enabling more informed treatment decisions and personalized care strategies. The future direction of research in this area lies in unraveling the mechanisms that drive the brain age gap and its evolution over time. Longitudinal studies are crucial for understanding the dynamic nature of the brain age gap and its relationship with disease trajectories. Furthermore, delving into the genetic, epigenetic, and environmental factors that contribute to the observed accelerated brain aging could provide a holistic view of the phenomenon and its variability among individuals.The exploration of the brain age gap in NMOSD and MS has unveiled a novel dimension in the understanding of neurodegenerative autoimmune disorders. By bridging the gap between neuroimaging, immunology, and clinical outcomes, this concept offers opportunities for refined disease characterization, early intervention, and treatment monitoring. As research progresses, the brain age gap could potentially revolutionize the management of NMOSD and MS, leading to more precise and effective approaches that address the unique neurodegenerative processes inherent to each disorder.

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