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Understanding the Risk of Rapidly Progressive Dementia in Humans

Guoyou Qin*

Department of Psychiatry, Korea University, Korea

DESCRIPTION

Rapidly Progressive Dementia (RPD) is a heterogeneous group of diseases that includes an abnormally rapid presentation of immune-mediated, infectious, and metabolic encephalopathies, as well as prion diseases and more common neuro-degenerative diseases. Some of these symptoms can be treated, while others may be infectious and require prompt diagnosis. Although prion diseases are considered the archetypal RPD, epidemiological reports over the past two decades and the identification of various encephalitis-mediated antibodies have increased the recognition of other brain disorders as potential causes of rapid cognitive decline. Knowledge of RPD etiology, syndromes, and diagnostic work-up protocols will help clinicians make early and accurate diagnoses, thereby increasing morbidity and mortality, especially in immune-mediated dementia and other reversible dementias rate can be reduced. In recent years, Rapidly Progressive Dementia (RPD) is increasingly recognized as a distinct clinical syndrome present in atypical non-AD dementias, human prion diseases, and related disorders considered in the differential diagnosis of these conditions. A common definition is that the time from the first disease-related symptoms to the onset of dementia syndrome is usually less than 1 or 2 years, but it can take several weeks depending on the cause of RPD, such as encephalitis or metabolic encephalopathy. May progress to dementia within In addition, specific definitions of RPD in neurodegenerative diseases, such as changes in Mini-Mental State Examination (MMSE) scores in rapidly progressing AD, using either total disease duration or rate of cognitive decline measures Suggested.

Rapid disease progression in dementia syndrome can be broadly classified as primary or secondary. The major rapid disease progression occurs in prion diseases, other rapidly progressing types of neurodegenerative dementias, encephalitis, and other diseases that usually cause severe neurological damage in a relatively short period of time. Rapid secondary disease progression is predominantly slow-progressing as a result of complications such as stroke or in the presence of coexisting CNS lesions (AD with cerebrovascular disease or Lewy body lesions). It can occur in CNS diseases. In addition, severe non-CNS lesions may contribute to neuronal damage, overall clinical manifestations pain, dyspnea, or tumorigenesis, or reduced survival time.

Treatment options for RPD patients are as diverse as the underlying etiology. For neurodegenerative diseases such as AD, cholinesterase inhibitors and memantine are among the established drugs. The recent FDA approval of aducanumab could represent a breakthrough treatment for AD, but remains controversial. Data on the efficacy of these agents in rapidly progressing neurodegenerative dementia are not yet available. Although there is no cure for prion disease, immunotherapy or treatments that alter protein expression may be an option in the near future. Various antibiotics and antivirals are well established and effective in treating infectious encephalitis, depending on the infectious agent identified. Treatment options for immune-mediated encephalitis include steroids, immunoglobulins, or plasmapheresis as first-line or bridging therapy and other immunotherapy agents such as rituximab as maintenance therapy.

Continued improvements in imaging modalities, the development of biomarkers, including blood-based biomarkers, for diagnosing brain pathologies, and the detection of an expanding spectrum of immune-mediated diseases, along with improved diagnostic options, are providing clinicians with Provides valuable information tools for making early differential diagnoses.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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Corresponding author Guoyou Qin, Department of Psychiatry, Korea University, Korea, E-mail: qin_g@gmil.com

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