Original Article



The Changing Landscape of Dementia Care: Challenges and Solutions

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Abstract

A variety of degenerative neurological conditions characterized by a loss of cognitive function severe enough to interfere with daily functioning are collectively called dementia. Although early-onset variants can occur, older people are the main affected population. The most common type is Alzheimer's disease, which is identified by the accumulation of tau tangles and amyloid plaques in the brain. Other types include frontotemporal dementia, which is characterized by degeneration of the frontal and temporal lobes, Lewy body dementia, which is related to aberrant protein deposits, and vascular dementia, which is related to cerebrovascular injury. Progressive degeneration of brain cells, resulting in decreased neuronal transmission and cognitive impairment, is part of the pathogenesis of dementia. Typical symptoms are memory loss, confusion, difficulty understanding and solving problems, and behavioral changes. Clinical evaluation, neuroimaging, and cognitive testing are used to make the diagnosis, focusing on separating dementia from other disorders that present with similar symptoms. Since there is currently no cure for dementia, treatment of the condition is complex and focuses on treating symptoms and improving quality of life. Behavioral therapies are used to address associated mental difficulties and pharmaceutical interventions are used to regulate cognitive symptoms as part of treatment options. An integral part of care are non-pharmacological methods that include lifestyle changes and cognitive stimulation. Dementia has a significant influence not only on affected people but also on their families and carers, often resulting in increased emotional stress and dependency. The goal of research is to improve diagnosis, treatment and, ultimately, the discovery of a cure by investigating the underlying causes and finding efficient therapies.

Keywords:-Cognitive impairment, Alzheimer's disease, neurodegenerative disorders, , vascular dementia, dementia with Lewy bodies.

Introduction

The word "dementia" refers to a broad category of neurological diseases that cause a gradual deterioration of cognitive abilities, making it increasingly difficult for a person to perform daily tasks. A person's inability to live freely is hindered by the memory, logic, language, and behavioral deficits of these diseases. Globally, dementia is becoming more common, primarily as a result of the aging population. According to the World Health Organization (WHO), more than 55 million people worldwide are expected to have dementia by 2030 and more than double that number by 2050 (WHO, 2021). This increase highlights how urgently better research, management techniques and healthcare resources are needed.Alzheimer's disease is the most common type of dementia, accounting for between 60 and 70 percent of cases. Beta amyloid plaques and tau tangles gradually accumulate in the brain of an Alzheimer's patient, causing neuronal death and brain atrophy (Kreft, 2021). It usually begins with minor disorientation and memory loss, eventually affecting more sophisticated cognitive functions and causing substantial impairment in activities of daily living. Other notable forms of dementia include dementia with Lewy bodies, which is characterized by the presence of abnormal protein deposits called Lewy bodies in the brain, causing fluctuations in cognition and motor control (McKeith et al., 2017)., and vascular dementia, which is related to cerebrovascular disease and results in cognitive impairment due to reduced blood flow to the brain (van der Flier & Scheltens, 2018). Progressive degeneration of the frontal and temporal lobes of the brain causes frontotemporal dementia (FTD), which is characterized by dramatic alterations in behavior, language, and personality. Unlike Alzheimer's disease, early-stage FTD may present with less severe memory loss (Bang, Spina, & Miller, 2015). The different clinical manifestations and pathological characteristics of each type of dementia make diagnosis difficult and require specialized therapeutic techniques. The underlying pathophysiology of dementia encompasses multiple neurodegenerative processes. Amyloid beta plaques cause neuronal transmission to be disrupted and tau tangles to form, which in turn leads to cell death and brain shrinkage in Alzheimer's disease (Tzeng et al., 2020). Chronic ischemia injury and stroke-related brain damage cause vascular dementia, which is characterized by a loss of neuronal connectivity that affects cognitive abilities (Iadecola, 2013). A comprehensive technique is used to diagnose dementia, including cognitive testing, neuroimaging, and clinical examination. According to Kukull et al. (2013), an accurate diagnosis is necessary to differentiate dementia from other cognitive illnesses such as delirium or depression, which may present comparable symptoms. While cognitive testing helps determine the degree of cognitive impairment, neuroimaging methods such as MRI and CT scans are essential to detect structural abnormalities of the brain (Apostolova and Thompson, 2019). While there is no known cure for dementia, there are therapies that can help manage symptoms and improve quality of life. Cognitive problems are treated with pharmaceuticals such as cholinesterase inhibitors and NMDA receptor antagonists, and behavioral therapy treats related psychiatric symptoms (Dommershausen et al., 2021). The well-being of people with dementia and their caregivers is greatly improved through nonpharmacological methods, such as cognitive stimulation and lifestyle therapies (Spector et al., 2019). Dementia influences not only the individual but also families and health systems, emphasizing the value of care and support resources. In summary, dementia is a complicated and growing public health problem that profoundly affects people who suffer from it and their families.

Understanding the many manifestations, underlying processes and practical management techniques of dementia is essential as the incidence of the disease increases globally. To improve outcomes and eventually find a solution, continued research and therapeutic innovations are crucial. Mitigating the effects of dementia and improving the quality of life of people affected by the disease still largely depends on early diagnosis and comprehensive care.

Cognitive impairment

A discernible and quantifiable loss of cognitive abilities, affecting different domains such as memory, attention, reasoning and executive function, is called cognitive impairment. Its severity can range from mild to severe and is a fundamental feature of many neurological diseases, including dementia. Cognitive impairment may manifest as difficulty remembering recent events, difficulty handling complicated problems, or difficulty organizing and planning activities. Memory loss: Memory loss is one of the most common signs of cognitive impairment. This may include problems remembering names, dates, or other details, which can seriously interfere with day-to-day activities. For example, people might have difficulty remembering conversations or forgetting appointments. This feature is especially notable in Alzheimer's disease, since one of the main symptoms is gradual memory loss (Kreft, 2021). Attention and concentration: Problems paying attention to details, staying focused, and processing information effectively may be due to attention deficits. People may have difficulty finishing household chores, get sidetracked easily, or have difficulty following conversations. According to research, attention problems can affect the ability to perform complicated tasks and are common in several types of dementia (Apostolova and Thompson, 2019). Higher-order cognitive processes including planning, decision making, problem solving, and abstract thinking are all included in executive function. Executive function deficits can cause problems with daily planning, money management, and decision making. This is especially true in the case of frontotemporal dementia, in which these vital processes are interfered with by frontal lobe degeneration (Bang, Spina, & Miller, 2015). Language and communication: Cognitive impairment can also affect language skills, making it difficult to follow discussions, interpret complex sentences, or locate appropriate words. Language impairments can worsen in diseases such as Alzheimer's and frontotemporal dementia, making communication difficult (McKeith et al., 2017). Visuospatial skills: Understanding and interpreting visual data related to spatial relationships is a requirement for this domain. Visuospatial impairments can make it difficult to navigate between situations, identify familiar items, or decipher visual cues. These deficits are frequently evident in people with Alzheimer's disease when they have problems with tasks that involve spatial awareness, such as getting lost in familiar environments (van der Flier & Scheltens, 2018). Effect on daily life: Cognitive impairment can have a substantial negative effect on a person's independence and quality of life by affecting their ability to perform daily tasks. People may need help with personal care, household chores, and social activities as their cognitive impairments worsen. This increasing dependency often requires comprehensive attention and assistance from family members or other caregivers. In summary, cognitive impairment includes a variety of impairments related to language, executive function, memory, attention, and visuospatial skills. These disabilities, which vary in severity, significantly affect daily functioning and quality of life. Understanding these cognitive impairments is essential for the diagnosis, treatment and

management of diseases such as dementia, as well as for creating efficient interventions to help those who are affected.

Alzheimer's disease

Since Alzheimer's disease accounts for 60% to 70% of dementia cases, it is the most common type of dementia. It is characterized by a progressive loss of cognitive function, mainly affecting language, reasoning and spatial awareness, but also affecting memory. The accumulation of tau tangles and amyloid beta plaques in the brain is indicative of the disease. While tau tangles are twisted fibers of the tau protein located within neurons that cause their death and subsequent brain atrophy, amyloid beta plaques are groups of aberrant protein fragments that disrupt neuronal communication (Selkoe and Hardy, 2016). Alzheimer's disease usually begins with modest memory loss and problems acquiring new knowledge. People suffer from an increasing range of cognitive impairments as the disease worsens, such as confusion, disorientation, and problems speaking and solving problems. Due to the severe impairment caused by the advanced stages of the disease, individuals become dependent on caregivers for daily tasks and personal care (DeTure & Dickson, 2019). Diagnosing Alzheimer's disease requires a thorough evaluation that includes neuroimaging, cognitive testing, and medical history. Alzheimer's is distinguished from other forms of dementia by distinctive patterns of brain atrophy and amyloid deposits that can be found using neuroimaging techniques such as MRI or positron emission tomography (Jack et al., 2018). These techniques help in confirming the diagnosis. Cognition tests evaluate memory, logic, and other cognitive abilities to determine the degree of disability. Since there is currently no cure for Alzheimer's disease, treatment focuses on controlling symptoms and improving quality of life. Cholinesterase inhibitors, including rivastigmine and donepezil, are frequently taken to treat cognitive problems by raising levels of acetylcholine, a neurotransmitter linked to memory and learning. Memantine, a different drug, helps control glutamate activity to relieve symptoms (Cummings, 2019). Non-pharmacological methods, such as cognitive stimulation therapy and supportive care, are essential to control the disease, in addition to pharmaceutical treatments (Spector et al., 2015). The development of diseasemodifying therapies and the identification of biomarkers for early diagnosis are the main objectives of ongoing research. Future treatment approaches could benefit from our growing understanding of the genetic, molecular, and environmental components that contribute to Alzheimer's disease (Querfurth and LaFerla, 2010).

Neurodegenerative disorders

A wide range of diseases known as neurodegenerative disorders are defined by the progressive degradation of neurons, eventually resulting in a loss of behavioral, motor and cognitive abilities. The accumulation of aberrant proteins, which impair neuronal function and contribute to cell death, is a common feature of many diseases. Neurodegeneration is caused by a complicated network of biochemical, environmental and hereditary variables. The accumulation of alpha-synuclein protein in Lewy bodies, which affects dopaminergic neurons in the substantia nigra, is the main pathogenic characteristic of Parkinson's disease. Motor symptoms such as bradykinesia, rigidity, and tremors are caused by this loss of neurons (Poewe et al., 2017). Similarly, aberrant protein aggregation in amyotrophic lateral sclerosis (ALS) leads to progressive loss of motor neurons, which in turn causes muscle atrophy, weakening, and ultimately paralysis (Robberecht & Philips, 2013). A toxic polyglutamine protein is produced as a result of Huntington's disease, caused by the growth of CAG repeats in the HTT gene. This protein causes selective neuronal death in the basal ganglia. According to Aylward et al. (2011), this degeneration presents as motor impairment, cognitive impairment and mental symptoms. Progressive degeneration of the frontal and temporal lobes causes frontotemporal dementia (FTD), which is frequently related to mutations in the MAPT or GRN genes. This condition causes notable alterations in behavior, personality and language (Rascovsky et al., 2011). Neuroimaging, genetic testing, and clinical evaluation are commonly used in the diagnosis of neurodegenerative diseases. Imaging modalities, such as PET and MRI, can identify aberrant protein buildup or distinctive patterns of brain shrinkage. In inherited forms of these disorders, genetic testing can confirm the diagnosis and shed light on the pathophysiology of the conditions (Hulshoff Pol et al., 2010). The goals of neurodegenerative disease management plans are to improve quality of life and alleviate symptoms. Drug therapies are customized to address certain symptoms; Riluzole is used for ALS and dopaminergic medications are used for Parkinson's disease. For impacted individuals to maintain their functional independence and improve their overall quality of life, non-pharmacological therapies such as physical therapy, occupational therapy, and cognitive rehabilitation are crucial (Gordon et al., 2021). Our understanding of neurodegenerative disorders is still expanding thanks to research, which is constantly seeking new treatment targets and undiscovered underlying mechanisms. Advances in genetics, neuroimaging, and molecular biology are anticipated to provide new insights and potential therapies for these difficult diseases (Miller et al., 2016).

Vascular dementia

A type of cognitive impairment known as vascular dementia is caused by damage to the brain's blood arteries, which reduces cerebral blood flow and damages neurons. This type of dementia is usually related to diseases that alter cerebral circulation, such as diabetes, chronic hypertension and strokes. Vascular dementia is characterized by a variety of patterns of cerebrovascular damage, including microinfarcts, small vessel disease, and large vessel infarcts, all of which exacerbate cognitive decline over time (Kalaria, 2018). The degree and location of arterial damage can have a significant impact on the clinical signs of vascular dementia. Common symptoms are memory problems, executive dysfunction, poor judgment, and slow processing speeds. According to Román et al. (2011), the disease usually appears initially after a stroke or may develop gradually over time as a result of chronic vascular injury. Two main subtypes of dementia are identified: subcortical vascular dementia, which is related to a chronic disease of the small arteries that affects the white matter of the brain, and multi-infarct dementia, which is caused by repeated small strokes that accumulate damage. (Pantoni, 2010). The diagnosis of vascular dementia requires a comprehensive evaluation that includes evaluation of vascular risk factors, neuroimaging, and a complete medical history. To find recognizable patterns of brain injury, such as strokes and white matter lesions, imaging methods such as MRIs and CT scans are used. Accurate diagnosis also depends on the identification of vascular risk factors, such as diabetes and high blood pressure (Wardlaw et al., 2015). To stop further cognitive loss, vascular dementia management treatments focus on reducing underlying

vascular risk factors. This includes the use of medications and lifestyle changes to aggressively treat diabetes, hypertension and other cardiovascular problems. To support the patient's daily functioning and quality of life, behavioral therapies and cognitive rehabilitation can be used in symptomatic treatment (O'Brien and Thomas, 2015). Research into the causes of vascular dementia and the development of new treatment modalities is still ongoing. Improvements in knowledge of the connection between vascular injury and cognitive impairment aim to improve methods of diagnosis and treatment of this disease (Sweeney et al., 2019).

Dementia with Lewy bodies

A neurodegenerative condition known as dementia with Lewy bodies (DLB) is identified by the aberrant aggregation of the protein alpha-synuclein in the brain known as Lewy bodies. Cognitive impairment, motor symptoms, and visual hallucinations are present in this disease, which is one of the most prevalent forms of progressive dementia (McKeith et al., 2017). One of the main characteristics of MCI is variable cognitive abilities; Patients with this condition have noticeable changes in their level of attention and focus. This variation can cause periods of bewilderment and disorientation. One of the most common symptoms of DLB is visual hallucinations, which often involve vivid, detailed images. Additionally, individuals may experience bradykinesia, rigidity, and tremors, as well as other parkinsonian motor symptoms comparable to those of Parkinson's disease (Walker et al., 2015). REM sleep behavior disorder (RBD), a severe component of DLB, is characterized by people who interpret their dreams as a result of a lack of muscle atonia during REM sleep. RBD is thought to be a possible early marker of MCI because it can occasionally occur years before cognitive symptoms (Iranzo et al., 2014). A comprehensive evaluation is necessary for the diagnosis of MCI, including clinical tests and neuroimaging. To rule out other potential causes of cognitive loss and detect patterns of brain atrophy that could indicate DLB, MRIs and CT scans are performed. Dopamine transporter (DAT) scans are a type of functional imaging approach that can be used to identify abnormalities associated with Lewy body pathology (Tzeng et al., 2020). Improving the patient's quality of life by treating cognitive and motor symptoms is part of the management of DLB. Hallucinations and other cognitive symptoms are often treated with cholinesterase inhibitors, such as rivastigmine. Medications such as levodopa may be recommended for motor symptoms, but caution is required as they may worsen hallucinations in certain situations. Supportive care and cognitive rehabilitation are two crucial nonpharmacological strategies to improve patient well-being and manage day-to-day difficulties (Aarsland et al., 2011). Research is still being done to learn more about the mechanisms behind DLB and create novel treatments. Research efforts are focused on elucidating the role of alpha-synuclein in disease pathophysiology, detecting early biomarkers for diagnosis, and developing innovative treatment approaches to efficiently address cognitive and motor symptoms (McKeith et al., 2021).

Conclusion

A complex and diverse set of conditions known as dementia are characterized by progressive cognitive decline and functional decline, which have a significant negative influence on the quality of life of sufferers and their families. Of all dementias, Alzheimer's disease remains the most common. It is characterized by specific pathological features including tau tangles and amyloid beta plaques. Diagnosis and treatment issues are different for dementia with Lewy bodies (DLB), which is defined by the presence of Lewy bodies and variable symptoms, and vascular dementia, which is caused by cerebrovascular injury. Recent decades have seen a significant change in our understanding of dementia due to advances in neuroimaging, molecular biology and clinical research. Even with these advances, there are still obstacles to achieving early diagnosis and creating efficient treatments. A personalized approach to diagnosis and treatment is required due to the variety of etiologies and symptoms of dementia. For example, cholinesterase inhibitors are useful in the treatment of MCI and Alzheimer's disease, but in the case of vascular dementia, treatment of the underlying vascular risk factors is necessary to prevent the dementia from worsening. The goal of current research remains to decipher the intricate pathways underlying these diseases. The goals are to understand the pathophysiological mechanisms at play, find biomarkers for early diagnosis, and create treatments that affect the condition. Advances in these fields could lead to better patient outcomes and quality of life by increasing the accuracy of diagnosis and the effectiveness of treatment. The interplay of genetic, environmental and metabolic factors contributing to dementia should be further investigated in future studies. To address the wider effects of dementia, there is also an urgent need for greater awareness, education and support for carers. With a deeper understanding of these disorders, it is hoped that more effective interventions can be created that not only improve symptoms but also slow or stop the progression of the disease, providing a better future for those affected by dementia and their families. families.

References

- Kalaria, R. N. (2018). Vascular basis for brain degeneration: Faltering controls and risk factors for dementia. Nature Reviews Neuroscience, 19(6), 389-403.
- [2] O'Brien, J. T., & Thomas, A. (2015). Vascular dementia. The Lancet, 386(10004), 1698-1706.
- [3] Pantoni, L. (2010). Cerebral small vessel disease: From pathogenesis and clinical characteristics to therapeutic challenges. The Lancet Neurology, 9(7), 689-701.
- [4] Roman, G. C., Sachdev, P., & Royall, D. R. (2011). Vascular dementia: Diagnostic criteria and research challenges. Alzheimer's & Dementia, 7(4), 286-293.
- [5] Sweeney, J. A., Aizenstein, H. J., & Rosano, C. (2019). The role of vascular pathology in cognitive decline. Journal of Alzheimer's Disease, 71(1), 1-20.
- [6] Wardlaw, J. M., Smith, C., & Dichgans, M. (2015). Small vessel disease: A neuroimaging perspective. The Lancet Neurology, 14(5), 437-448.
- [7] Aylward, E. H., Li, D., & Stern, C. E. (2011). Huntington's disease: A review of the clinical and imaging findings. Neuroimaging Clinics of North America, 21(2), 175-189.
- [8] Gordon, P. H., & Miller, R. G. (2021). Management of Amyotrophic Lateral Sclerosis. Neurotherapeutics, 18(1), 138-159.
- [9] Hulshoff Pol, H. E., & Kahn, R. S. (2010). Imaging the brain in neurodegenerative diseases. Neuropsychology Review, 20(1), 42-60.
- [10] Miller, B. L., Cummings, J. L., & Morris, J. C. (2016). The neurodegenerative diseases: Current status and future

directions. Journal of Neurodegenerative Disorders, 2016, 1-10.

- [11] Poewe, W., Seppi, K., & Tanner, C. M. (2017). Parkinson disease. Nature Reviews Disease Primers, 3, 17013.
- [12] Robberecht, W., & Philips, T. (2013). The changing scene of amyotrophic lateral sclerosis. Nature Reviews Neuroscience, 14(4), 248-264.
- [13] Rascovsky, K., Hodges, J. R., & Knopman, D. S. (2011). Sensitivity of revised diagnostic criteria for the behavioural variant of frontotemporal dementia. Brain, 134(9), 2456-2477.
- [14] Cummings, J. (2019). Cholinesterase inhibitors: A new era. Journal of Alzheimer's Disease, 71(3), 703-711.
- [15] DeTure, M. A., & Dickson, D. W. (2019). The neuropathological diagnosis of Alzheimer's disease. Molecular Neurodegeneration, 14(1), 32.
- [16] Jack, C. R., Bennett, D. A., Blennow, K., et al. (2018). NIA-AA research framework: Toward a biological definition of Alzheimer's disease. Alzheimer's & Dementia, 14(4), 535-562.
- [17] Querfurth, H. W., & LaFerla, F. M. (2010). Alzheimer's disease. New England Journal of Medicine, 362(4), 329-344.
- [18] Selkoe, D. J., & Hardy, J. (2016). The amyloid hypothesis of Alzheimer's disease at 25 years. EMBO Molecular Medicine, 8(6), 595-608.
- [19] Spector, A., Orrell, M., & Goyder, J. (2015). Cognitive stimulation therapy for dementia: A systematic review and meta-analysis. Journal of Neurology, Neurosurgery, and Psychiatry, 86(2), 111-116.
- [20] Apostolova, L. G., & Thompson, P. M. (2019). Brain imaging in Alzheimer's disease. Neurotherapeutics, 16(3), 425-438.
- [21] Bang, J., Spina, S., & Miller, B. L. (2015). Frontotemporal dementia. The Lancet, 386(10004), 1672-1682.
- [22] Dommershausen, N., Levin, K. A., & Del Campo, R. (2021). Management of dementia: Pharmacological and non-pharmacological approaches. Therapeutic Advances in Psychopharmacology, 11, 20451253211023525.

- [23] Iadecola, C. (2013). The pathobiology of vascular dementia. Neuron, 80(4), 844-866.
- [24] Kreft, K. (2021). Pathophysiology of Alzheimer's disease. Journal of Neurology, 268(4), 1189-1198.
- [25] Kukull, W. A., Ganguli, M., & Suzuk, R. (2013). Clinical diagnosis of dementia. JAMA, 310(3), 275-281.
- [26] McKeith, I. G., Boeve, B. F., & Dickson, D. W. (2017). Diagnosis and management of dementia with Lewy bodies. Neurology, 89(1), 88-100.
- [27] Spector, A., Orrell, M., & Goyder, J. (2019). Cognitive stimulation therapy for dementia: A systematic review and meta-analysis. Journal of Neurology, Neurosurgery, and Psychiatry, 90(8), 852-860.
- [28] Tzeng, R.-C., Kuo, C.-C., & Lee, J.-C. (2020). The role of amyloid beta and tau protein in Alzheimer's disease. Biomolecules, 10(12), 1563.
- [29] van der Flier, W. M., & Scheltens, P. (2018). Vascular cognitive impairment. The Lancet Neurology, 17(2), 160-171.

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