

The Prospects of Dementia Pathology: An Extensive Analysis of Developments and Advancements

Tim Carter*

Short Communication

Department of Psychology, University of Psychiatric Studies, Adelaide, Australia

*Corresponding author: Tim Carter, Department of Psychology, University of Psychiatric Studies, Adelaide, Australia, Email: Carter_tim123@pd.usp.au

Received: 22-Apr-2024, Manuscript No. JADP-24-138320; **Editor assigned:** 24-Apr-2024, PreQC No. JADP-24-138320 (PQ); **Reviewed:** 08-May-2024, QC No. JADP-24-138320; **Revised:** 15-May-2024, Manuscript No. JADP-24-138320 (R); **Published:** 22-May-2024, DOI: 10.4172/2161-0460.1000606

Citation: Carter T (2024) The Prospects of Dementia Pathology: An Extensive Analysis of Developments and Advancements. J Alzheimers Dis Parkinsonism 14: 606.

Copyright: © 2024 Carter T. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Description

Dementia is a complex and multifaceted syndrome characterized by a significant decline in cognitive functions, surpassing what might be expected from normal aging [1]. This decline affects several cognitive domains including memory, thinking, orientation, comprehension, calculation, learning capacity, language and judgment. Dementia is not a single disease but a collective term for various neurodegenerative disorders that share the common feature of progressive cognitive deterioration. The dementia is substantial and growing with the World Health Organization estimating that around 50 million people worldwide were living with dementia in 2019, a number expected to triple by 2050 due to population aging [2].

Dementia encompasses a range of neurodegenerative disorders characterized by a progressive decline in cognitive function, impacting memory, thinking, orientation, comprehension, language and judgment [3].

Alzheimer's disease

Alzheimer's disease is characterized by two primary pathological features: amyloid plaques and neurofibrillary tangles [4].

Amyloid plaques: Amyloid plaques are extracellular deposits primarily composed of amyloid-beta (A β) peptides [5]. These peptides are derived from the Amyloid Precursor Protein (APP) through sequential proteolytic processing by β -secretase and γ -secretase enzymes [6].

Neurofibrillary tangles: Neurofibrillary tangles are intracellular aggregates composed of hyperphosphorylated tau protein [7]. Tau is a microtubule-associated protein that when abnormally phosphorylated forms paired helical filaments that aggregate into tangles. These tangles disrupt the normal function of neurons and contribute to cell death [8].

Vascular dementia

Vascular dementia results from conditions that impede blood flow to the brain leading to ischemic and hemorrhagic damage [9]. Common pathological features include:

Lacunar infarcts: Small deep cerebral infarcts caused by occlusion of penetrating arteries.

Large vessel infarctions: Due to major stroke events.

Microbleeds: Resulting from small vessel disease.

White matter lesions: Often associated with chronic hypertension and small vessel disease.

Lewy body dementia

Lewy Body Dementia (LBD) encompasses Dementia with Lewy Bodies (DLB) and Parkinson's Disease Dementia (PDD) [10]. The primary pathological feature is the presence of Lewy bodies, which are intracytoplasmic inclusions, composed of alpha-synuclein protein.

Frontotemporal dementia

Frontotemporal Dementia (FTD) is a heterogeneous group of disorders characterized by degeneration of the frontal and temporal lobes [11]. The major pathological subtypes include:

Tauopathies: Including Pick's disease and corticobasal degeneration characterized by abnormal tau protein deposition.

TDP-43 proteinopathies: Characterized by deposits of Transactive Response (TAR) Deoxyribonucleic Acid (DNA) binding Protein 43 (TDP-43).

FUS proteinopathies: Involving Fused in Sarcoma (FUS) protein aggregates.

Other dementias

Huntington's disease: Huntington's disease is an autosomal dominant disorder caused by a mutation in the *Huntingtin (HTT)* gene leading to abnormal expansion of Cytosine, Adenine, Guanine (CAG) repeats and resulting in mutant huntingtin protein accumulation. [12].

Creutzfeldt-Jakob disease: Creutzfeldt-Jakob Disease (CJD) is a prion disease characterized by the accumulation of misfolded Prion Protein (PrP^{s_c}).

Conclusion

The pathology of dementia is involving various neurodegenrative processes that challenge the development of effective disease-modifying therapies. Key pathological features such as protien misfolding, neuro inflammation and synaptic dysfunction are common across many forms of dementia and represent critical targets. Continued exploration of these mechanisms is essential for creating targeted treatments that address the specific needs of each dementia type. Early Early diagnosis and timely intervention, supported by advancements in neuroimaging and biomarker identification for improving patient outcomes. Additionally, preventive strategies focusing on lifestyle modifications and management of cardiovascular risk factors hold the potential in reducing the incidence and impact of dementia. Ultimately a multifaceted approach combining early detection, preventive measures and innovative study is necessary to mitigate the burden of dementia on individuals and society.

Acknowledgement

Special thanks are extended to Department of Psychology for providing the necessary resources and support.

Conflict of interest

The authors declare no conflicts of interest.

References

- Brownell M, Sehar U, Mukherjee U, Reddy PH (2024) Creating Cultural and Lifestyle Awareness about Dementia and Co-morbidities. J Alzheimers Dis Rep 8(1):747-764.
- Wortmann M (2012) Dementia: a global health priority-highlights from an ADI and World Health Organization report. Alzheimers Res Ther 4:1-3.
- Dugger BN, Dickson DW (2017) Pathology of neurodegenerative diseases. Cold Spring Harb Perspect Biol 9(7):a028035.
- Chien DT, Szardenings AK, Bahri S, Walsh JC, Mu F, et al. (2014) Early clinical PET imaging results with the novel PHF-tau radioligand [F18]-T808. J Alzheimers Dis 38(1):171-184.

- Silva J, Monge-Fuentes V, Gomes F, Lopes K, dos Anjos L, et al. (2015) Pharmacological alternatives for the treatment of neurodegenerative disorders: Wasp and bee venoms and their components as new neuroactive tools. Toxins 7(8):3179-3209.
- Gorelick PB, Scuteri A, Black SE, deCarli C, Greenberg SM, et al. (2011) Vascular contributions to cognitive impairment and dementia: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 42(9):2672-2713.
- Goedert M (1999) Filamentous nerve cell inclusions in neurodegenerative diseases: tauopathies and alpha-synucleinopathies. Philos Trans R Soc Lond B Biol Sci 354(1386):1101-1118.
- McKhann GM, Albert MS, Grossman M, Miller B, Dickson D, et al. (2001) Clinical and pathological diagnosis of frontotemporal dementia: report of the Work Group on Frontotemporal Dementia and Pick's Disease. Arch Neurol 58(11):1803-1809.
- 9. Frank S (2014) Treatment of Huntington's disease. Neurother 11(1): 153-160.
- Gibbs Jr CJ, Gajdusek DC, Asher DM, Alpers MP, Beck E, et al. (1968) Creutzfeldt-Jakob disease (spongiform encephalopathy): transmission to the chimpanzee. Science 161(3839):388-389.
- Wang YJ, Zhou HD, Zhou XF (2006) Clearance of amyloid-beta in Alzheimer's disease: progress, problems and perspectives. Drug Discov Today 11(19-20):931-938.
- 12. Auning E, Rongve A, Fladby T, Booij J, Hortobágyi T, et al. (2011) Early and presenting symptoms of dementia with Lewy bodies. Dement Geriatr Cogn Disord 32(3):202-208.