WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 9, Issue 5, 688-698. Review Article ISSN 2277– 7105

A REVIEW ON COGNITIVE IMPAIRMENT AND DEMENTIA

Ranjana Bohra*

Department of Pharmacy, Invertis University Bareilly, U.P. 243123.

Article Received on
02 March 2020,

Revised on 23 March 2020,
Accepted on 12 April 2020,

DOI: 10.20959/wjpr20205-17350

***Corresponding Author**
Ranjana Bohra
Department of Pharmacy,
Invertis University Bareilly,
U.P. 243123.

ABSTRACT

Cognition is group of several mental processes that includes memory, learning, language, attention, focus, decision making and mild mental impairment of cognition may be sign of a disease that lead to dementia. Dementia is a syndrome that is characterized by progressive deterioration of cognitive functions. Patients with dementia may also exhibit behavioral and psychological symptoms. Prevalence of dementia increases with age. Dementia can cause by thyroid dysfunction, vitamin deficiencies, disturbed sleep and depression. Treatment of cognitive impairment mainly focuses on several sign and symptoms which included improving quality of life, cognition, motivation, mood and behavioral impairment.

KEYWORDS: *Dementia, cognitive impairment, Alzheimer disease, Memory, learning.*

INTRODUCTION

Cognition: Cognition is the processes supporting human thinking and experiences. It concern with the process of selecting, thinking, identifying, translating, storing, and using information to make sense of and interact with the social world, to perform everyday activities, and to plan and perform the course of occupational life.[1] cognitive function can also be describe as a perception, language, intelligence, attention, memory, executive function (initiating, planning, organizing, controlling and evaluation of thinking and acting) and psychomotor speed.[2] Cognitive impairment and dementia highly affect the daily activity of one's life (and their relatives) in a significant way.

Factors affecting cognitive function

Age is the greatest and one of the most concern risk factor.[3] After that several diseases also affect cognitive function such as depression, stress-related diseases, sleeplessness, long-term

alcohol abuse, lack of folic acid and vitamin B12, cardiovascular diseases, diabetes and multimorbidity. Neurodegenerative diseases such as Parkinson disease, Alzheimer disease (AD), frontotemporal dementia and multiple sclerosis.[4] Diet is also play an important factor in mentioned as an important factor cognitive impairment.

MEMORY AND LEARNING

Memory of an individual is the ability to record sensory stimuli, information, event etc., and keep in mind for short or long periods of time and recall the same at a later date when needed. Poor memory, slow recall and lower retention are common problem in today's stressful and competitive world. Age, stress, emotions are conditions that may lead to memory loss, amnesia, high blood pressure, anxiety, dementia or to more ominous threats like schizophrenia and Alzheimer's disease.[5] Learning is the process by which one can obtain knowledge about the world and memory is the process by which that knowledge is encoded, stored and later retrieved. Learning is classified into two – long term learning and short term learning. Accordingly long term memories are those that we can recall days, months or year after they were originally stored. Short term memories are those that last second to hours and are vulnerable to disruption.[6]

DEMENTIA: Dementia refers to a syndrome that is characterized by progressive deterioration of cognitive functions also characterized by cognitive or memory impairments not involving any alteration in consciousness or alertness. In other words dementia is a syndrome that is characterized by progressive deterioration of cognitive functions. It's a mental disorder characterized by loss of intellectual ability which interfere with one's occupational and social activity. Prevalence of dementia increases with age. The incidence of dementia is increasing worldwide.[7] Neuropsychiatric symptoms such as apathy, agitation and depression are common which also include emotional problems like problem with language and decrease in motivation. Alzheimer disease and cerebrovascular ischemia (vascular dementia) are the two most common cause and some cases of dementia involve both of these disorders. Although some potentially reversible conditions such as hypothyroidism or vitamin B12 deficiency are often thought to cause dementia, no more than 1.5% of cases of mild to moderate dementia are fully reversible.[8] The most prevalent type of dementia is Alzheimer's disease, which makes up 50% to 70% of cases. Other types include lewy body dementia (15%), vascular dementia (25%) and frontotemporal dementia. Less common causes includes Parkinson's disease, syphilis, normal pressure hydrocephalus and

Creutzfeldt-Jakob disease among others.[9]

The cognitive impairment in dementia include memory loss, inability to carry out motor activities despite adequate motor function, difficulty in understanding or using words and failure to identify or recognize objects. It is the mental or psychological disorder characterized by loss of intellectual ability. People with dementia usually experience impairments in occupational and social functioning and may present behavioral disturbances. Dementia is characterized by the accumulation of abnormally folded protein fragments i.e. amyloid beta peptide and tau that precipitate in amyloid plaques and neuronal tangles respectively. Characteristic feature of dementia is progressive loss of memory.[10] Neuropsychiatric symptoms such as apathy, agitation and depression are also common. Many diseases can cause dementia syndrome. Alzheimer disease and cerebrovascular ischemia are two most common causes and some cases of dementia involve both of these disorder. Although some potentially reversible conditions such as hypothyroidism or vitamin-B12 deficiency are often thought to cause dementia, no more than 1.5% of cases of mild to moderate dementia are fully reversible.[11]

Prevalence of dementia increases with age. The incidence of dementia is increasing worldwide. The transition in world age demography toward older age is associated with an increased risk of neurodegenerative diseases, such as Alzheimer's disease.[12] Dementia of Alzheimer disease accounts for 50% to70% of dementia cases.[13] It has been estimated to be approximately 6% to 10% of individuals aged 65 years or older prevalence increases with age, rising from 1% to2% among those aged 65 to 74 to 30% or more of those aged 85 or older.[14] In another investigation, 40% of those aged 90 to 94 were reported to suffer from dementia, with the commonness of dementia peaking at 58% among individuals older than 94 years.[15] It is not worthy that, at present the majority of older person do not experience dementia. Nevertheless, with older person projected to represent a greater proportion of the U.S. population, for people with dementia the cost of caring will become an increasingly important public health consideration. Dementia increases the mean annual health care cost per older patient with 75% of these increased costs for hospitalization and expenditures on skilled nursing facilities.[16] Some forms of dementia are reversible. Timely recognition and intervention are key to the optimal care of older patient with dementia, which may be attributable to a number of causes.

a) Reversible causes of dementia

A complete medical history, physical examination and medication review are important components of the initial assessment of individuals presenting the memory cognitive impairments characterizing dementia. Thyroid dysfunction, Vitamin deficiencies, and normal pressure hydrocephalus have all been identified as potentially reversible causes of dementia.[17] Only about 9% of cases of dementia are reversible and appear to have decreased in prevalence[18] identifying and treating potentially reversible causes of dementia remains the greatest importance.[19]

b) Depressive symptoms

Depressive disorders may feature cognitive symptoms which is similar to those observed in dementia. When the recognition tests are delayed, individuals with dementia were reported to make intrusion errors (false positive errors), while individuals with depression tended to under report recognition of items actually presented[20] (false negative errors). In one more investigation, depression was associated with disturbed sleep and early morning awakening whereas dementia was associated with shortage in rapid eye movements during sleep and with sleep-disordered breathing.[21] Decreased sex drive and increased anxiety were found to be more common among depressed individuals, whereas those with dementia were found to apparent greater disorientation to time and difficulty completing self-care tasks.[22] Any person whose cognitive decline is due to depression may experience restoration of cognitive function once their depression has been effectively treated. There is complex relationship between depression and dementia and not entirely understood. Depressive symptoms have been a risk factor among older patients with mild cognitive impairment for subsequent development of dementia. There is brain structure abnormalities in older patient with depressive symptoms and cognitive impairment have been reported[23] and similarly abnormal cerebral blood flow observed in patient with dementia. Treatment of depression may prevent further functional deterioration among people with the “depression-executive dysfunction” syndrome of late life.[24]

c) Delirium

Dementia must also be differentiating from delirium, a condition characterized by impairments in consciousness and attention, as well as by the impairments in memory and cognition observed in dementia. The onset of delirium is usually sudden, whereas the presentation of dementia is characteristically deceptive. Delirium can be precipitated by

illness or in toxicities or it can be induced by medication.[25] Often presenting as an acute confusional state, delirium is a medical emergency associated with a high risk for morbidity and mortality. However, delirium typically resolves if the underlying cause is effectively treated, although older patient may be slow to return to their pre morbid level of functioning. Individuals with delirium may gain symptomatic relief if they are provided with a comfortable environment and anti-psychotic medication if needed. These reports show that the necessity of an initial medical evaluation of older people experiencing cognitive impairment.

d) Mild cognitive impairment

In the last decade efforts to better understand decline in cognitive abilities among older people not meeting criteria for dementia. Older adults experiencing mild cognitive impairment (MCI) typically present with subjective memory complaints (ideally verified by an informant) and also show objective affirmation of memory deficits as measured by neuropsychological tests. However, the degree of cognitive impairment experienced by people with MCI does not interfere with their ability to perform activities of daily living.[26] In a multicenter population study of older adults, 19% of participators are younger than 75 and 29% of participators are older than 85 were identified as having MCI. Longitudinal research has revealed that between 23% and 47% of adults aged 75 and older who were initially dementias free but who manifested "ageing associated cognitive decline" (a construct similar to MCI except that it does not require subjective memory impairment) developed dementia over a 2.6-year period.[27] Individuals with MCI who have the epsilon 4 allele of *APOE*, the gene for apo lipoprotein E, appear to be at increased risk of progressing to dementia. American Academy of Neurology recommends that people with MCI receive clinical monitoring and evaluation including neuropsychological testing. Although MCI has been proposed as a potential starting point for involvement, the effectiveness of treatments for MCI is currently under evaluation.

e) Alzheimer's disease

Researchers have estimated that approximately 75% of individuals with dementia have Alzheimer's disease (AD). AD can be distinguished from other forms of dementia by its deceptive and progressive course, although plateaus in the progression of AD may occur. Depression, insomnia, lack of self-restraint, delusions and hallucinations may be manifest as the disease progresses, and some neurological signs including sudden muscle contraction and

gait disturbance.[28] As its initial presentation is subtle, information provided by someone close to the individual with mild AD may be essential to its early recognition. Deterioration in recall of items on the Mini-Mental State Examination, names of relatives, difficulty with calculation (such as balancing a checkbook or maintaining household finances) making repetitious statement and exhibiting poor judgment are symptoms associated with early AD. Characteristic brain pathology of AD includes the presence of neuro fibrillary tangles (inter weavings of filaments within the body of the nerve cell (neuron) and plaques indicative of neuron degeneration. Degeneration is particularly prominent among neurons that release the neurotransmitter acetylcholine. Abnormalities in the transport of glutamate, the chief excitatory neuro transmitter in the brain, may also underlie the development of AD.[29] Recent research has suggested that the Cerebrospinal fluid of patients with AD has altered levels of proteins associated with neuron degeneration. However the utility of CSF as a risk indicator for AD awaits further evaluation. To date identified risk factors for AD include a positive family history of the disorder, limited education, head injury, *APOE* genetic endowment and age.[30]

f) Vascular dementia

Vascular dementia (VaD) has been estimated to account for 15% to 20% of all dementias among older adults and is precipitated by some form of cerebro vascular disease. Mostly, blood vessels blockage in the brain yields the death of tissue or infarction, in the affected region. Necrosis underlying dementia may involve a single, strategic blood vessel or numerous smaller ones (multiple infarct dementia). Traditionally, VaD has been distinguishing by sudden onset, stepwise progression, and focal neurological deficits associated with the region of the brain affected. The results of research undertaken during the last decade have revealed that an estimated 20% of cases of VaD are characterized by a deceptive onset and a steadily progressive course.[31] Postmortem examinations of the brains of individuals with dementia have revels that the coexistence of VaD with AD is not uncommon. Laboratory tests and brain imaging techniques like EEG, PET, MRI, fMRI can be used in the diagnosis of VaD. Atherosclerosis Hypertension, diabetes, age, and male sex are probable risk factors for vascular dementia. As several of these factors have also been associated with an increased risk of AD, identification of potential vascular components of AD is growing. Frontotemporal dementia initially affecting the regions of the brain governing planning, social behavior, and language perception, fronto temporal dementia (FTD) includes the syndrome commonly referred to as Pick's disease. As compared to other dementias, FTD

is characterized by a younger age of onset, with its presentation after age 75 being rare. FTD can also be distinguished from other dementias by a unique constellation of symptoms. In contrast to AD, older adults with FTD initially evident less memory impairment[32] but more changes in speech and personality, indicative of disinhibition and poor social awareness. The behavioral presentation of FTD may include inappropriate swearing, impulsive changes in eating habits and deficits in self-care may also be present. FTD may also include progressive deterioration of language function. Older adults with FTD may present with difficulties in word usage, precipitating reading and writing impairments that may culminate in autism. Recent research has suggested FTD may be more prevalent than was previously believed. A family history of dementia and mutation of the gene that produces tau protein have been associated with FTD.[33]

g) Lewy body dementia

Lewy bodies are common among individuals with Parkinson's disease. Lewy body dementia (LBD) is distinguish by the presence of Lewy bodies, proteins in the cerebral cortex (which governs thought processing) and brain stem (which coordinates movement). Although prevalence estimates differ, some researchers have estimated that LBD accounts for 15% to 20% of all cases of dementia.[34] As might be expected, people with LBD have been found to clear signs of Parkinsonism, such as difficulties in initiating movements, slowness of movement, muscular rigidity and tremor. Although its clinical presentation may be similar to that of AD, individuals with LBD can be distinguished from individuals with AD by marked cognitive fluctuations, prominent hallucinations and the presence of Parkinsonism. Recent research suggests that LBD can also be differentiated from AD by behaviors that the individual exhibits during periods of cognitive slowing. Daytime drowsiness despite adequate sleep, sleeping for 2 or more hours during the day, staring into space for long intervals of time, and periods of nonsensical or disorganized speech are more common in LBD than AD.[35] In addition to the presence of Lewy bodies, pathology characteristic of AD (i.e., plaques, and, to a lesser degree, neurofibrillary tangles) is also frequently present in LBD. However, differentiating these two forms of dementia is crucial, because individuals with LBD are highly sensitive to the adverse effects of antipsychotic drugs[36] which may be administered with the intent of providing relief from distressing hallucinations.

h) Dementia interventions

Although dementia remains a source of great suffering for many older adults, the results of

recent research suggest that there are reasons for hope about the prospects of improved care of older adults with cognitive impairment. Some forms of dementia, albeit a minority, can be reversed through timely intervention. Moreover, not all forms of cognitive impairment among older adults are dementia. Some are attributable to other conditions that are controllable to treatment. Public health efforts to heighten awareness of the importance of early evaluation of older adults showing signs of cognitive impairment are clearly warranted, as are efforts to educate the public about the heterogeneity and potential reversibility of cognitive impairment. Perhaps most significantly, dementia is increasingly recognized as the endpoint of a continuum of cognitive decline among older adults, with the detection of mild cognitive impairment suggesting new opportunities for intervention.

CONCLUSION

Dementia is associated with significant physical or mental impairment that disturbed quality of life among older adults. It is the mental disorder characterized by loss of intellectual ability. Characteristic feature of dementia is progressive loss of memory, difficulty in understanding or using words, inability to carry out motor activities and failure to identify or recognize objects. Timely detection and intervention are key to the optimal care of older adults with dementia. Early detection of cognitive impairment and dementia is essential to reducing inactivity and improving or retaining a person's activity level and thus quality of life. This is beneficial for the individual as well as for society and health care.

REFERENCES

1. Kielhofner, G. Conceptual foundations of occupational therapy practice. (4th ed.). Philadelphia: F.A Davis Company, 2009.
2. Wahlund, L. O., Nilsson, C. & Wallin, A. Kogntiv medicin. Norsted, 2011.
3. Blennow, K., De Leon, M. J. & Zetterberg, H. 2006. Alzheimer's disease. The Lancet, 2006; 368: 387-403.
4. Fastbom, J., Fors, S. & Lövdén, M. Åldrande och minne - Naturligt eller sjukligt glömsk? Forskning i korthet. Tryck: Tryckeri AB Orion, 2014.
5. Parle, M. and Vasudevan, M. Memory enhancing activity of abana; An Indian ayurvedic Poly – herbal formulation. Journal of health sciences, 2007; 43- 52.
6. Bear, M.F., Connors, B.W. and Paradiso, M.A. (2011). Neuroscience; exploring the brain, 2011; 15-22.
7. Prince, M., Bryce, R. and Ferri, C. The benefits of early diagnosis and intervention.

London Alzheimer,s disease international, 2011.

8. Boustain, M., Peterson, B.H., Harris, R. and Krasnov, C. Screening for dementia. Rockville, 2002; 1-164.
9. Gautheir and Serge. Clinical diagnosis and management of Alzheimer disease 3rd edition Abingdon, Oxon: Informa healthcare, 2006; 53-54.
10. Dao, A.T., Zagaar, M.A., Levine, A.T., Salim, S., Eriksen, J.L. and Alkadhi, K.A.Treadmill exercise prevents learning and memory impairment in Alzheimer's disease-like pathology, Curr. Alzheimer. Res, 2013; 507-515.
11. Boustain, M., Peterson, B.H., Harris, R. and Krasnov, C. Screening for dementia. Rockville, 2002; 1-164.
12. Stephan, B.C., Matthews, F.E. and Ma, B.Alzheimer and vascular neuropathological changes associated with different cognitive States in a non-demented sample, J. Alzheimers, 2012; 309-318.
13. Singh, B., Sharma, B., Jaggi, A. S., and Singh, N. Attenuating effect of lisinopril and telmisartan in intracerebroventricular streptozotocin induced experimental dementia of Alzheimer's disease type: possible involvement of PPAR-g agonistic property, J. Renin Angiotensin Aldosterone Syst, 2013; 124-136.
14. Hendrie, H.C. Epidemiology of dementia and Alzheimer's disease. Am J Geriatr Psychiatry, 1998; 6: S3-S18.
15. Ebly, E.M., Parhad, I.M., Hogan, D.B. and Fung, T.S. Prevalence and types of dementia in the very old: results from the Canadian study of health and aging. Neurology, 1994; 1593-1600.
16. Hill, J.W., Futterman, R., Duttagupta, S., Mastey, V., Lloyd, J.R. and Fillit, H. Alzheimer's disease and related dementias increase costs of comorbidities in managed medicare. Neurology, 2002; 62-70.
17. Espino, D.V., Jules-Bradley, A.C., Johnston, C.L and Mouton, C.P. Diagnostic approach to the confused elderly patient. Am Fam Physician, 1998; 57: 1358-1366.
18. Clarfield, A.M. The decreasing prevalence of reversible dementias: an updated meta-analysis. Arch Intern Med, 2003;163: 2219-2229
19. Wivel, M.E. NIMH report. NIH consensus conference stresses need to identify reversible causes of dementia. Hosp community psychiatry, 1988; 39: 22-23.
20. Gainotti, G and Marra, C. Some aspects of memory disorders clearly distinguish dementia of the Alzheimer's type from depressive pseudo-dementia. J Clin Exp Neuropsychol, 1994; 16: 65-78.

21. Hoch, C.C. Reynolds CF 3rd. Electroencephalographic sleep in late-life neuropsychiatric disorders. Int Psychogeriatr, 1989; 1: 51-62.
22. Reynolds, C.F., Hoch, C.C., Kupfer, D.J., Buysse, D.J., Houck PR, Stack JA, *et al*. Bedside differentiation of depressive pseudodementia from dementia. Am J Psychiatry, 1988; 145: 1099-103.
23. Pearlson, G.D., Rabins, P.V., Kim, W.S., Speedie, L.J., Moberg, P.J., Burns, A. Structural brain CT changes and cognitive deficits in elderly depressives with and without reversible dementia (pseudodementia). Psychol Med, 1989; 19: 573-84.
24. Alexopoulos, G.S. The depression-executive dysfunction syndrome of late life: a specific target for D3 agonists? Am J Geriatr Psychiatry, 2001; 9: 22-29.
25. Samuels, S.C. and Evers, M.M. Delirium: pragmatic guidance for managing a common, confounding, and sometimes lethal condition. Geriatrics, 2002; 57: 33-38.
26. Petersen, R.C., Stevens, J.C., Ganguli, M., Tangalos, E.G., Cummings, J.L. and DeKosky, S.T. Practice parameter: early detection of dementia: mild cognitive impairment (an evidence-based review): Report of the quality standards subcommittee of the American academy of neurology. Neurology, 2001; 56: 1133-42.
27. Busse, A., Bischkopf, J., Riedel-Heller, S.G. and Angermeyer, M.C. Mild cognitive impairment: prevalence and predictive validity according to current approaches. Acta Neurol Scand, 2003; 108: 71-81.
28. Richards, S.S., Hendrie, H.C. Diagnosis, management, and treatment of Alzheimer disease: a guide for the internist. Arch Intern Med, 1999; 159: 789-798.
29. Thai, D.R. Excitatory amino acid transporter EAAT-2 in tangle-bearing neurons in Alzheimer's disease. Brain Pathol, 2002; 12: 405-411.
30. Hall, K., Gureje, O., Gao, S., Ogunniyi, A., Hui, S.L. and Baiyewu, O. Risk factors and Alzheimer's disease: a comparative study of two communities. Aust N Z J Psychiatry, 1998; 32: 698-706.
31. Erkinjuntii, T., Sulkava, R. Diagnosis of multi-infarct dementia. Alzheimer Dis Assoc Disord, 1991; 5: 112-121.
32. Binetti, G., Locascio, J.J., Corkin, S., Vonsattel, J.P. and Growdon, J.H. Differences between Pick disease and Alzheimer disease in clinical appearance and rate of cognitive decline. Arch Neurol, 2000; 57: 225-32.
33. Rosso, S.M., Donker Kaat, L., Baks, T., Joosse, M., de Koning I. and Pijnenburg .Y. Frontotemporal dementia in The Netherlands: patient characteristics and prevalence estimates from a population based study. Brain, 2003; 126: 2016-2022.

34. Campbell. S., Stephens., S. and Ballard. C. Dementia with Lewy bodies: Clinical features and treatment. Drugs aging, 2001; 18: 397-407.
35. Ferman, T.J., Smith, G.E., Boeve, B.F., Ivnik, R.J., Petersen, R.C. and Knopman, D. DLB fluctuations: specific features that reliably differentiate DLB from AD and normal aging. Neurology, 2004; 62: 181-187.
36. McKeith, I.G., Galasko, D., Kosaka, K., Perry, E.K., Dickson, D.W. and Hansen, L.A. Consensus guidelines for the clinical and pathologic diagnosis of dementia with Lewy bodies (DLB): report of the consortium on DLB international workshop. Neurology, 1996; 47: 1113-1124.