International Journal of Allied Medical Sciences and Clinical Research (IJAMSCR)

ISSN:2347-6567

IJAMSCR |Volume 8 | Issue 1 | Jan - Mar - 2020
www.ijamscr.com

Review article *Medical research*

A review on dementia: epidemiology, risk factors, diagnosis, types and management

S. Salini[1], K. Vishal[1], N. Sriram[1]. Dr. S. Selvakumar[2]

[1]*HITS College of Pharmacy, Bogaram(Vill.), Keesara (Mandal), RR Dist. Telangana.*
[2]*Cherraans College of Pharmacy, Coimbatore -39.*
***Corresponding Author**: **S. Salini**
Email id: shalinisontyana22@gmail.com

ABSTRACT

Dementia is a clinical syndrome that has difficulty in memory, language and behavior that leads to impairment in daily activities, majorly occurring in older ages. Diagnosis of dementia is difficult and much complicated, it is differentiated based on vascular, infectious and genetic causes.. Management of dementia includes pharmacological, non pharmacological treatments.. Even for today, a definitive diagnosis, pathophysiology and treatment of dementia are unclear.

Keywords: Alzheimer's Dementia, Cognitive impairment, Cholinesterase inhibitors.

INTRODUCTION

Dementia is a new functional dependence based on progressive cognitive decline and representing, as its form Latin origins suggest, a previous mental functioning. The incidence of dementia rises with age as it's a common phenomenon within our ageing population. People with dementia are more dependent and vulnerable, both socially and in terms of physical and mental health, presenting evolving challenges to society. Despite the seemingly, the clinical diagnosis of dementia can be difficult with newly functional impairment often obscured by physical foible, comorbid psychiatric symptoms such as depression. Dementia is one of the major causes of disability in ageing groups. The arising of symptoms decades into the pathophysiological process slow down targeted disease therapy. A myriad of research initiatives is underway to identify potential biomarkers of disease processes earlier. It is estimated that the older adult population aged 65 and over is projected to increase from 16 per cent in 2008. Once the diagnosis is authoritative, prediction measures are required and are still lacking, as disease trajectories between individuals can vary greatly. Globally, governments are recognizing these challenges. Drugs contribute symptomatic benefit are available and memory service structures exist to diagnose dementias and guide management. The personal impact of dementia on patients and families is also being recognized [42].

Prevalence, Epidemiology and Socio-Economic Implications

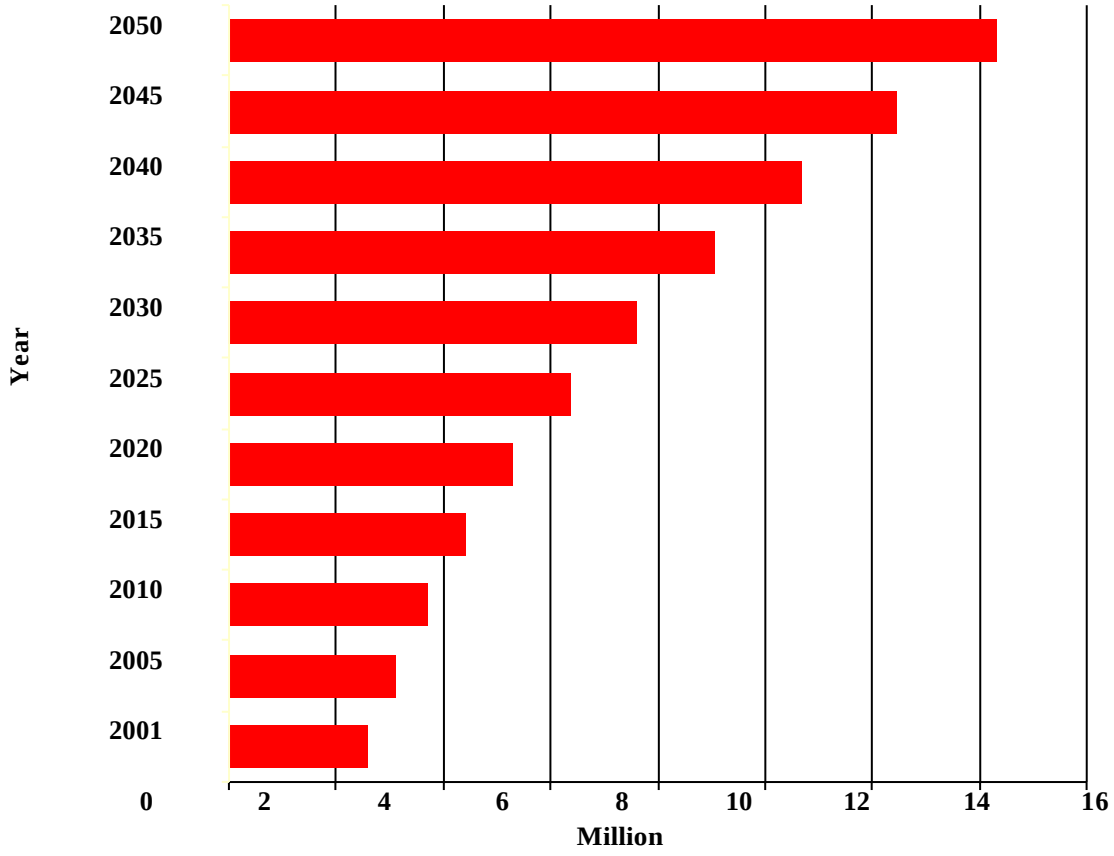
The situation in India with regards to dementia prevalence has not been researched thoroughly though there have been indications of prevalence according to the 10/66 Dementia study which was conducted in seven low- and middle-income

countries in eleven sites which included both rural and urban India. This study was conducted on the background of previous literature that low socioeconomic countries harbour less of dementia compared to higher economic countries [43].

In assessing and diagnosing dementia there are several issues when countries like India are considered. The educational background, social status, urban/rural living, understanding of assessment process and validation of the assessment tools used are to be taken into account when diagnosing somebody with a condition which means little to people but harnesses itself most overwhelmingly. Though the percentage figures from these studies look small in numbers one could imagine the prevalence of Schizophrenia, which is considered to be 1% and still presents with significant psychiatric morbidity costing the Government a fortune [43].

The census conducted in 2001 has shown that the elderly population of India accounted for 77 million compared to 24 million in 1961, 43 million in 1981 and 57 million in 1991. This trend projects rise from 5.63% of older adults in 1961 to 6.58% in 1991, reaching 7.5 per cent in 2001. Global estimates of a doubling in the dementia population every 20 years giving an estimated 115 million people with dementia by 2050 were revised further upwards in 2013, to take account of the likely further increases in lower- and middle-income countries [42].

Estimation of the number of people with dementia over 60 years in India between 2001 and 2050[48]



Risk factors

- Age
- Vascular diseases(majorly in Vascular Dementia)
- Genetic factors
- Minor risk factors are smoking, alcohol consumption and diabetes [51].
- Vitamin D deficiency was also identified as an independent risk factor for the development of dementia of any cause, and supplementation is

recommended for patients in whom deficiency is diagnosed [2]

Diagnosis

The role of primary care is to exclude treatable factors like depression, vit.B12 deficiency or thyroid disturbance; refer to a specialist if unusual symptoms persist like neurological, psychiatric, or behavioral changes.

Investigations and brief cognitive tools that are used in the assessment of dementia

Blood test

Blood test includes full blood count, erythrocyte sedimentation rate, urea and electrolytes, thyroid function, vitamin B12, and folate.

Brief cognitive assessment tools

General practitioner assessment of cognition

Takes no longer than five minutes to administer and comprises two components: a six-item cognitive assessment with the patient and an informant questionnaire (if the cognitive assessment score is equivocal: 5-8 inclusive). Scores >8 are deemed to represent cognitive impairment and <5 intact cognition. Sensitivity 82-85%; specificity 83-86%

6 item cognitive impairment test

Takes 3-4 minutes to perform and consists of six questions on orientation and memory, although the test may be susceptible to influences of language and education. Scores of 0-7 are considered normal and ≥8 suggest cognitive impairment. Sensitivity 78.5-83%; specificity 77-100%

Mini-cog assessment instrument

Takes 2-4 minutes to complete and consists of two components, a three-item recall and the clock drawing test. Cognitive impairment is considered to be present if people are unable to recall any of the three items or if they recall only one or two items and draw an abnormal clock. Sensitivity 76-99%; specificity 89-96%

Memory impairment screen

Takes around four minutes to complete and is a brief four-item delayed free recall and cued recall memory impairment test. A score of ≤4 indicates possible dementia. Sensitivity 74-86%; specificity 96-97% [41]. MMSE (Mini-Mental State Examination) scale can be used in detecting cognitive dysfunction of dementia, but the MMSE scale alone cannot be used alone in the diagnosis of MMSE [5].

Types of Dementia

Various types of diseases, infections and genetic disorders can be represented by term Dementia. The most common type of disease includes Alzheimer's disease. Other types include Frontotemporal Dementia, Dementia with Lewis bodies and Vascular Dementia. HIV or Creutzfeldt-Jacob disease are the infections that result in Dementia. Huntington's disease is a genetically inherited syndrome that results in Dementia. [1]

Alzheimer's Disease [AD]

Major cases of Dementia includes Alzheimer's disease accounting for about 60-80% of total Dementia diagnosis [2] That includes 11% of those age 65 and older, one-third of those 85 and older. Female has high risk than male at old age [51]. Alzheimer's disease has a clinical duration of around 8-10 years, it is an irreversible and progressive neurodegenerative disorder that affects cognition and behavioral changes. More than 80 million people may be affected by AD in 2050. Memory loss, depression, apathy, aggression and psychosis are the major neuropsychiatric symptoms (NPS). Individual NPS vary with structural and metabolic features of AD brain [4]. Three stages of AD can be noticed as early, middle and last stage.

Early-stage AD

At this stage person with AD can function independently. Symptoms in early-stage include problems coming up with the right word or name, forgetting material that was just read, trouble remembering names when introduced to new people, increasing trouble with planning or organizing, challenges performing tasks in social or work settings, Losing or misplacing a valuable object.

Middle-stage AD

It is the longest period and lasts for many years, a person at this stage requires great level care. Forgetfulness of events or about one's personal history, confusion about where they are or what day it is, feeling moody or withdrawn especially in socially or mentally challenging situations, trouble controlling bladder and bowels in some individuals,

being unable to recall their address or telephone number or the high school or college from which they graduated, need for help choosing proper clothing for the season or the occasion, changes in sleep patterns, such as sleeping during the day and becoming restless at night, personality and behavioural changes, including suspiciousness and delusions or compulsive, repetitive behaviour like hand wringing or tissue shredding, An increased risk of wandering and becoming lost.

Last-stage AD

At this stage person with AD loses the ability to control their movements, cannot carry on conversations, unable to respond to the environment, they become vulnerable to infections like pneumonia, need personal care and assistance round the clock.

Neurological examination includes brain imaging like CT-scan and MRI which helps to identify strokes and tumours. Multifactorial risk factors are present in developing AD, which includes preventable [smoking, hypertension, type 2 diabetes, sedentary lifestyle, obesity and head injury] and non-preventable [age and genetics] risk factors. Association of more than 20 genetic loci is a main genetic risk for AD. 60% of individuals with AD have an e4 allele gene for apolipoprotein E(ApoE). Intracellular neurofibrillary tangles (NFTs), extracellular amyloid plaques, synaptic deterioration, and neuronal death are the core neuropathologic findings of AD. [3] Majorly symptomatic treatment is considered as use of Serotonin reuptake inhibitors (SSRIs: fluoxetine, sertraline, paroxetine, citalopram, fluvoxamine) are largely considered to be among the most efficient antidepressants to treat comorbid depression in AD dementia [6]. They can also be used in the treatment of agitation and psychosis. Cholinesterase inhibitors like donepezil, rivastigmine, galantamine are used in mild-moderate and severe AD. Memantine which is having action both dopamine antagonist and non-competitive N-methyl-D-aspartate receptor antagonist is used in the treatment of moderate to severe AD, who show difficult in alertness and attention. Nutraceutical huperazine A can also be used to improve the memory of patients [2]. Currently, only cholinesterase inhibitors and memantine are used in the treatment of Dementia.

Vascular Dementia (VaD)

Cerebrovascular disease either alone or in combination with Alzheimer's disease is a leading cause of cognitive impairment. VaD is caused by a decrease in the supply of oxygen and nutrients to the brain, results in the decline of thinking skills caused by conditions that block or reduce blood flow to the brain. All types of vascular pathologies lead to VaD. Cognitive dysfunction can be clinical [ischemic, haemorrhagic stroke] and subclinical or silent stroke which ranges from subjective memory complaints that cannot be detected even by detailed neuropsychological examination, to full-blown dementia [9]. Increased age, diabetes, hypertension, atherosclerosis, and stroke are the major risk factors of VaD [40]. VaD includes cortical vascular dementia; hypoperfusion dementia; subcortical ischemic dementia; hemorrhagic dementia: strategic-infarct dementia; and dementias resulting from specific arteriopathies. Frontotemporal Dementia, Lewy body Disease superseded VaD which is previously considered as the second most common type of Demetia [8] causing around 15% of total Dementia cases. It is more common in men than in women. Ageing in vessels, cardiovascular risk factors, cerebrovascular risk factors are the three major risk factors in VaD. 4-5 years is the average survival after diagnosis and patient die often with the cardiovascular or cerebrovascular event. Management includes the use of anticoagulants, antihypertensives and lipid regulators [HMG-CoA reductase inhibitors] and antipsychotics, antidepressants are also used. Having a proper diet and prevention of smoking are preventive measures [1].

Dementia with Lewy Body

Dementia with Lewy bodies (DLB) is an age-associated neurodegenerative disorder producing progressive cognitive decline that interferes with normal life and daily activities, accounting for 10-15% of total dementia cases [12]. Neuropathologically, DLB is characterised by the accumulation of aggregated α-synuclein protein in Lewy bodies and Lewy neurites, similar to Parkinson's disease (PD). Extrapyramidal motor features characteristic of PD, are common in DLB patients but are not essential for the clinical diagnosis of DLB. Dementia associated with cortical Lewy bodies (DCLB), LB dementia, LB variant Alzheimer's disease(LBVAD), diffuse LB

disease (DLBD), senile dementia of LB type (SDLT) is the previously used clinical diagnosis, which was now preferred as DLB [11]. Symptoms of DLB has core features and supportive features; core features includes recurrent detailed hallucinations, progressive cognitive decline effective social and occupational functioning, spontaneous parkinsonism (eg. Dystonia), 2 core features are required for probable DLB and 1 for possible DLB. Supportive features include loss of consciousness (fainting), falls, depression, sleep disorders specifically altered phases of rapid eye movement sleep, SPET and PET imaging can be done to demonstrate low dopamine transporter in basal ganglia. Probable DLB can be made if 1 or more of supportive features in addition to 1 core feature is present, possible DLB is present if only 1 supportive feature and no core features are present [13]. There is no specific drug treatment for DLB. As acetylcholine is important to increase the cognitive function of the brain, Cholinesterase inhibitors can be used to treat DLB as its pharmacological action is to increase the acetylcholine in the brain. As persons with DLB may also have some parkinsonian movement symptoms, in such cases levodopa which is a dopamine agonist can be used as a treatment. REM sleep behaviour disorder can be treated by using melatonin up to a dose of 15mg at bedtime is required. Clonazepam can be used in severe REM sleep behaviour disorder in case melatonin is unsuccessful. Medical benefits must be weighed against possible adverse effects from the use of these medications when considering the use of medication in DLB treatment. In the last 2 years in USA clinical trails especially for DLB has begun in developing two novel therapeutics known as intepirdine and nelotanserin. Intepirdine is a selective 5-HT6 receptor antagonist which may have efficacy in reducing parkinsonian symptoms and improving cognitive symptoms as well as caregiver's interpretation of change and occurrence of adverse events. While Nelotanserin is an inverse agonist of serotonin receptors of the subtype 5-HT2A, trails are going on to observe changes in extrapyramidal symptoms and frequency of visual hallucinations [14].

Frontotemporal Dementia (FTD)

FTD is the third most leading cause of Dementia after Alzheimer's disease and Dementia with Lewy body [15]. FTD is a clinical spectrum of neurodegenerative disorders affecting primarily the frontal and/or temporal lobes which cause disturbances in language, personality and behaviour changes [20]. In persons less than 65 years old FTD is the second-most cause of Dementia. Almost 25% of FTD cases can be in persons above 65 years of age having Dementia. It is estimated FTD has an incidence of about 1.61 to 4.1 Per one hundred thousand annually [16]. The average for the cause of FTD is 45-65 years. Major clinical syndromes of FTD, behavioural change (behavioural variant frontotemporal dementia) which accounts half of the cases, and the remainder present with language decline (primary progressive aphasia) characterized either by impaired speech production (progressive non-fluent aphasia) or by impaired word comprehension and semantic memory (i.e., memory for meaning) (semantic dementia) [17], motor changes (rigidity, muscle atrophy, extraocular movement abnormalities, bradykinesia) [22]. Mutation in three genes progranulin (GRN), microtubule associated protein tau (MAPT), and chromosome 9 open reading frame 72 (C9orf72; also known as C9orf72-SMCR8 complex subunit) is a genetic risk factor for FTD [18]. All the neuropathological entities that lead to a cause of FTD are characterized by selective degeneration of frontal and temporal cortices. Four major symptoms like impairment in the regulation of personal conduct, social interpersonal conduct, loss of insight and emotional blunting enabled the researchers and clinicians for clinical diagnosis of FTD [19]. Psychotic symptoms like hallucinations and delusions are considered as core symptoms, psychosis or depression are the common symptoms. Evidence-based medicine that would support an FDA indication for the treatment of FTD requires large-scale, randomized, double-blind, placebo-controlled trials that do not currently exist [24]. Treatment majorly includes symptomatic [22]. Psychoactive medications are primarily used for the management of FTD as it involves various behavioural symptoms [23]. Pharmacological treatment of cognitive symptoms includes acetylcholinesterase inhibitors (donepezil, rivastigmine, and galantamine,), Selective serotonin reuptake inhibitors (SSRI) can be prescribed in FTD patients as they have been successfully used in treating clinical symptoms in psychiatric patients that resemble some of the

problematic FTD behaviours [24]. Carbamazepine and valproic acid are anti-epileptics with mood-stabilizing effects were reported to improve behavioural symptoms.

Creutzfeldt-Jacob Disease (CJD)

CJD belongs to a family of fatal neurodegenerative disorders like prion diseases or transmissible spongiform encephalopathies (Tse) which can cause several fatal neurodegenerative disorders in humans and animals [25]. CJD is a rare, degenerative, progressive brain disorder where the nerve cells in the brain break very quickly which is having 100% mortality [28]. Early symptoms include memory problems, vision problems, behaviour/personality changes, depression, loss of muscle coordination, insomnia (difficulty falling asleep or staying asleep), unusual sensations. As the disease progress, it leads to dementia. ~1 per million will be affected by CJD every year in the US and worldwide [26]. CJD exists an inherited (familial), acquired (variant and iatrogenic), and spontaneous (sporadic) forms [27]. Sporadic CJD (sCJD) is the most common type which accounts for about 80%-85%. 65 years is the mean onset of the age of sCJD [29]. Cause of sCJD is unknown, as reported most of the patients to become fatal in 6months after the cause [30]. Inherited (familial) CJD is believed to be caused by several mutations in the PRNP gene. Diagnosis of CJD is difficult and there is no particular method used to diagnosis as symptoms of CJD are similar to all types of neurodegenerative disorder. Current developments leading to the diagnosis of CJD based on a combination of the clinical picture, MRI and EEG findings together with the detection of protein 14-3-3 in CSF which is released into spinal fluid when brain cells die [31]. CJD is not transmitted by air or most form of casual contact [33], several reports suggest that oral transmission is through by taking contaminated food or brain and in few cases suggest CJD is also caused by eating infected squirrels and wild goats [32]. There is no specific treatment for CJD, symptomatic treatment is suggested in many cases. Recently trails and observational studies are being conducted in the treatment of sCJD early with doxycycline, but the results are unclear [31], concluding a larger trial of doxycycline should be performed in persons in early stages of sCJD and is recommending doxycycline can be used for treatment in early stages of sCJD as a useful therapeutic option unless another treatment option is available [34].

HIV Associated Dementia (HAD)

The survival rate in HIV increased dramatically in recent times due to advances in treatment by long term antiretroviral therapy [37]. By the introduction of HAART in 1996, has result in the reduction of HAD to about 50% [38]. HAD is the type of HIV-associated neurocognitive disorders (HANDs) along with other types like asymptomatic neurocognitive impairment, mild neurocognitive disorder (MND) which is caused by HIV 1 infection majorly and HIV 2 in very rare cases, which causes neurocognitive complications [35] occurs majorly in older ages of HIV infected persons. HAD is occurred at the beginning of the AIDS epidemic primarily in patients with advanced HIV disease and low CD4 cell counts, as HIV spreads to the brain causing encephalopathy(a disease condition which affects the brain function) leading to dementia. Major symptoms include memory impairment along with other complications like psychomotor slowing, inattention and apathy. Motor symptoms include lack of coordination [36]. Treatment including antiretroviral drugs in combination with symptomatic treatment. A study was conducted believing Abacavir, a "CNS active" agent would suppress a potential reservoir of HIV in the CNS, where other drugs, especially protease inhibitors, might not be effective. Zidovudine, the earliest available nucleoside reverse transcriptase inhibitor, seemed to improve the motor functions of people with HAD when given in higher doses than normally used for the treatment of systemic HIV disease. An observational data showed that dementia prevalence in the West dropped after zidovudine became available, suggesting that zidovudine prevented HAD. Even so, the burden of mild cognitive impairment in HIV remained substantial. But the study on Abacavir concludes an effective understanding the natural history and interrelationships is essential in the treatment of dementia and milder forms of neurocognitive impairment [39].

Huntington's Disease (HD)

Huntington's disease is a rare, autosomal, neurosomal disorder which is inherited. Prevalence of 3.6 to 5.7 per 100,000 in regions mainly comprising residents of Caucasian descent. HD is characterized by Many though many cognitive and

motor symptoms are present memory loss in HD is taken as basic criteria for the diagnosis of Dementia in HD [50]. According to WHO the occurrence of HD in western countries is about 5 to 7 per 1000 individuals are between 35 to 45 years. Neuropathology majorly includes neuronal loss in striatum and cortex. No specific treatment is present to treat HD, Tetrabenazine (xenazine) approved by the FDA to treat jerky involuntary movements (or) chorea associated with Huntington disease [48].

Management of Dementia

Pharmacological

Offending medications, in particular, those with anticholinergic properties should be reconsidered and stopped where possible. It is important to note that even over the counter medications can affect cognition. An association with benzodiazepines has been suggested by observational work and these too should be reconsidered [42].

The pharmacological treatment of dementia is associated with important challenges such as complexities in the clinical presentation and diagnosis, non-availability of therapeutic agents with robust effectiveness and issues related to the tolerability of medications used in the treatment of dementia [44].

- Cholinesterase inhibitors prevent the breakdown of acetylcholine in the brain, a key neurotransmitter involved in learning and memory, thus increasing the level of acetylcholine in the brains of individuals with dementia [46].
- Donepezil has been approved for all stages of Alzheimer's dementia. Rivastigmine and Galantamine have been approved for mild to moderate Alzheimer's dementia [46].
- Memantine has been approved for moderate to severe Alzheimer's dementia [46].
- Combination of Donepezil and Memantine has been approved for moderate to severe Alzheimer's dementia [46].
- Rivastigmine Transdermal patch (4.6mg, 9.5mg or 13,3mg per 24 hours) has been approved for mild to moderate Alzheimer's dementia. The extent of adverse effects with Rivastigmine is lesser in transdermal patch than oral formulation [46].

The benefit of cholinesterase inhibitors is difficult to establish at the individual patient level because of the progressive nature of the illness and the possibility of contribution to cognitive decline from multiple factors [44].

Nonpharmacological

Non-pharmacological management strategies have an important role in the management of dementia of any type. It is particularly helpful in elderly patients who may not tolerate pharmacological agents due to the development of adverse effects even in smaller doses [44].

- Cognitive-behavioural therapy and interpersonal therapy may have significant benefits in the treatment of depression in dementia but are likely to be limited in patients with severe dementia [47].
- Although various cognitive training and exercise programs have been proposed to improve or preserve cognition and function in patients with mild to moderate dementia, multiple studies have not provided sufficient evidence to support any particular beneficial intervention. However, while exercise does not improve cognition, neuropsychiatric symptoms of depression, it may improve the ability to perform activities of daily living in individuals with dementia [46].
- Many other non-pharmacological therapies have been shown to result in improvements, including RO, RT, VT, music therapy, and recreational activities. Most of these therapies are simple to implement and should probably be the first choice in treating mild-moderate depressive symptoms [47].
- These interventions can be delivered by health and social care staff and volunteer with proper training and supervision. The response to each form of therapy should be monitored and the care plan should be reviewed from time to time as there may be individual variations in the response to each of these modalities [44].

PREVENTION OF DEMENTIA

As in any chronic medical illness, the objective of intervention in dementia is to prevent the onset or postpone and eliminate the disease or control the symptoms in the diseased population. Prevention can be categorized into three levels.

Primary Prevention

Primary prevention focuses on reducing the incidence of dementia by addressing risk factors.

Secondary Prevention

It focuses on early detection before the emergence of overt dementia and halts the progression.

Tertiary Prevention

It focuses on timely diagnosis and treatment of cognitive, behavioural, and psychological symptoms along with decreasing caregiver burden and improving quality of life [45].

CONCLUSION

As it became difficult to differentiate types of dementia, major advancements in the scientific field must be done to have a proper diagnosis, to find a proper pathology which is helpful in the treatment of all types of dementia.

REFERENCES

[1]. *Andrew Husband and Allen Wosley,* The Pharmaceutical Journal 277, 2006, 579-580.

[2]. Current understanding of Alzheimer's disease diagnosis and treatment [version 1; referees: 2 approved] Jason Weller, Andrew Budson 1,2 Department of Neurology, Boston VA Hospital, 150 South Huntington Street, Jamaica Plain, MA, 02130, USA Department of Neurology, Boston University School of Medicine, 72 East Concord Street C-309, Boston, MA, USA

[3]. *MaileenGlorianeUlep, Simrit Kaur Saraon, and Samantha McLea*; Alzheimer Disease The Journal For Nurse Practitioner's 14(3), 2018, 129-135

[4]. *Xiao-Ling Li, Nan Hu, Meng-Shan Tan, Jin-Tai Yu, and Lan Tan*; Behavioral and Psychological Symptoms in Alzheimer's Disease; 2014, 9. https://doi.org/10.1155/2014/927804

[5]. *Sid E. O'Bryant, Joy D. Humphreys, et. al*; Detecting Dementia with the Mini-Mental State Examination (MMSE) in Highly Educated Individuals; Arch Neurol. 65(7), 2008, 963–967. doi:10.1001/archneur. 65.7.963.

[6]. *Konstantina G. Yiannopoulou and Sokratis G. Papageorgiou* Current and future treatments for Alzheimer's disease; Ther Adv Neurol Disord. 6(1), 2013, 19–33.doi: 10.1177/1756285612461679PMCID: PMC3526946 PMCID: PMC3526946

[7]. *Catherine McVeigh and Peter Passmore*; Vascular dementia: prevention and treatment Clin Interv Aging. 1(3), 2006, 229–235. Published online doi: 10.2147/ciia.2006.1.3.229PMCID: PMC2695177 PMID: 18046875

[8]. *Amos D. Korczyn*; What is new in vascular dementia?; BMC Med. 14, 2016, 175. PMCID: PMC5094035 PMID: 27806713.

[9]. *F-E de Leeuw and J. van. Gijn*; Department of Neurology, University Medical Centre Utrecht Practical Neurology, 2003, 86–91; Pract Neurol: first published as 10.1046/j.1474-7766.2003.04132.x

[10]. *Efraim Jaul and Oded Meiron Front*Systemic and Disease-Specific Risk Factors in Vascular Dementia: Diagnosis and Prevention; *Aging Neurosci., 17, 2017. https://doi.org/10.3389/fnagi. 2017.00333*

[11]. *Tiago Fleming Outeiro et.al*; Dementia with Lewy bodies: an update and outlook; PMCID: PMC6341685; PMID: 30665447; 2019. doi: 10.1186/s13024-019-0306-8

[12]. Ian McKeith, MD; Dementia with Lewy Bodies; FMedSci 6(3), 2004, 333–341
PMCID: PMC3181810; PMID: 22033743

[13]. *Melanie Zupancic, MD, Aman Mahajan, MD, and KamnaHanda, MD*; Dementia With Lewy Bodies: Diagnosis and Management for Primary Care Providers; doi: 10.4088/PCC.11r01190; PMCID: PMC3267516 PMID: 22295275

[14]. *Samuel D. Capouch. Martin R. Farlow. Jared R. Brosch*; A Review of Dementia with Lewy Bodies' Impact, Diagnostic Criteria and Treatment; Published online: 9, 2018

[15]. *Juan Joseph Young, Mallika Lavakumar, Deena Tampi* et al; Frontotemporal dementia: latest evidence and clinical implications; Ther Adv Psychopharmacol, 8(1), 2018, 33–48 DOI: 10.1177/ 2045125317739818

[16]. *Nicholas T. Olney, MD, Salvatore Spina, MD, PhD, and Bruce L. Miller, MD*Frontotemporal Dementia; Neurol Clin. 35(2), 2017, 339–374.
doi: 10.1016/j.ncl.2017.01.008; PMCID: PMC5472209; NIHMSID: NIHMS859441; PMID: 28410663

[17]. *Jason D Warren, Jonathan D Rohrer et.al*; Frontotemporal dementia; Cite this as: BMJ 2013, 347:f4827
[18]. *Katrina M Moore*; Age at symptom onset and death and disease duration in genetic frontotemporal dementia: an international retrospective cohort study; Lancet Neurol 19, 2020, 145–56;
[19]. *jee bang MD., et.al.;* Frontotemporal dementia;*Volume 386(10004), 2015, 24–30 1672-1682*
[20]. *Andreas johnen; Maxime Bertoux*; Psychological and Cognitive Markers of Behavioral Variant Frontotemporal Dementia–A Clinical Neuropsychologist's View on Diagnostic Criteria and Beyond; Front. Neurol., 7, 2019 , https://doi.org/10.3389/fneur.2019.00594
[21]. *Maria LandqvistWaldö, Lars Gustafson, et al* ; Psychotic symptoms in frontotemporal dementia: a diagnostic dilemma?; doi:10.1017/S1041610214002580
[22]. *Gregory A Jicha and Peter T Nelson;* Management of frontotemporal dementia: targeting symptom management in such a heterogeneous disease requires a wide range of therapeutic options; *PMCID: PMC3172080; NIHMSID: NIHMS303718; PMID: 21927623;* Neurodegener Dis Manag. 1(2), 2011, 141–156.; doi: 10.2217/nmt.11.9
[23]. *Mendez MF*; Frontotemporal dementia: therapeutic interventions; Front Neurol Neurosci. 24, 2009, 168-178. doi: 10.1159/000197896. Epub 2009 Jan
[24]. *Richard M. Tsai, MD MBA and Adam L. Boxer, MD PhD;*Treatment of Frontotemporal Dementia; doi: 10.1007/s11940-014-0319-0.
[25]. *Kranthi K. Sitammagari, WajeedMasood ;*Creutzfeldt Jakob Disease StatPearls - NCBI Bookshelf.
[26]. *William A. Rutala and David J. Weber;* Creutzfeldt-Jakob Disease: Recommendations for Disinfection and Sterilization.
[27]. *Richard Knight*;Creutzfeldt-Jakob Disease: A Rare Cause of Dementia in Elderly Persons; National Creutzfeldt-Jakob Disease Surveillance Unit, Edinburgh, United Kingdom.
[28]. *Marc Manix MD, Piyush kalakoti,* et al.; Creutzfeldt-Jakob disease: updated diagnostic criteria, treatment algorithm, and the utility of brain biopsy; http://thejns.org/doi/abs10.3171/2015.8.FOCUS15328
[29]. *Gotaro Kojima MD; Brent K. Tatsuno MD; et al;* Creutzfeldt-Jakob Disease: A Case Report and Differential Diagnoses.
[30]. *Marie-Clare Por ter MBBS MRCP, Michelle LeemansMbChB FRCA;* Creutzfeldt–Jakob disease*;* Continuing Education in Anaesthesia, Critical Care & Pain j 13(4), 2013, 119-124.
[31]. *Aseel Al-Ansari, N. P. Robertson*; Creutzfeldt–Jacob disease: new directions in diagnosis and therapeutics; J Neurol 264, 2017, 1029–1031 DOI 10.1007/s00415-017-8473-4.
[32]. *Kamin M, Patten BM*; Creutzfeldt-Jakob Disease. Possible transmission to humans by consumption of wild animal brains; Am J Med. 76(1), 1984, 142-5.
[33]. "Creutzfeldt-Jakob Disease Fact Sheet", NINDS, Publication date; NIH Publication No. 18, 2018, NS-2760
[34]. *Daniela Varges,1 HenrikeManthey; et.al;* Doxycycline in early CJD: a double-blinded randomised phase II and observational study J Neurol Neurosurg Psychiatry 0, 2016, 1–7. doi:10.1136/jnnp-2016-313541
[35]. *Marcus Kaul*; HIV-1 associated dementia: update on pathological mechanisms and therapeutic approaches; CurrOpin Neurol. 22(3), 2009, 315–320. doi:10.1097/WCO.0b013e328329cf3c.
[36]. Dennis *L. Kolson, Francisco González-Scarano*; HIV and HIV dementia; J Clin Invest. 106(1), 2000, 11-13. https://doi.org/10.1172/JCI10553.
[37]. *Jane KasoziNamagga, et.al;* Prevalence and Risk Factors of HIV-Associated Neurocognitive Disorders in Rural Southwestern Uganda; J Assoc Nurses AIDS Care. 30(5), 2019, 531–538. doi: 10.1097/JNC.0000000000000036
[38]. *Ahmad M. Yakasai;* Prevalence and Correlates of HIV-Associated Neurocognitive Disorders (HAND) in Northwestern Nigeria; Hindawi Publishing Corporation Neurology Research International, Article ID 486960, 2015, 9. http://dx.doi.org/10.1155/2015/486960
[39]. *Ronald J. Ellis*; Evidence-Based Treatment for HIVAssociated Dementia and Cognitive Impairment: Why So Little?; www.plosclinicaltrails.org; 2007, e15
[40]. *Juhyun Song,Won Taek Lee, et al;* Association between Risk Factors for Vascular Dementia and Adiponectin; Biomed Res Int. 2014, 261672.; Published online doi: 10.1155/2014/261672; PMCID: PMC4016875;PMID: 24860814

[41]. *Louise Robinson, Eugene Tang, John-Paul Taylor*; Dementia: timely diagnosis and early intervention; BMJ. 2015; 350: h3029. Published online 16, 2015. doi: 10.1136/bmj.h3029;PMCID: PMC4468575;PMID: 26079686

[42]. *Cunningham , McGuinness , Herron , Passmore* ; Dementia; published online on Ulster Med J 84(2), 2015, 79-87

[43]. *Farooq Khan;* Dementia in India: it's high time to address the need!;AP J Psychol Med. 12(2), 2011, 64-8

[44]. K.S. Shaji, *et al;*Clinical Practice Guidelines for Management of Dementia; Indian J Psychiatry. 60(3), 2018, S312–S328. doi: 10.4103/0019-5545.224472: 10.4103/0019-5545.224472.

[45]. *Ramanathan Sathianathan, Suvarna Jyothi* Kantipudi; The dementia epidemic: Impact, prevention, and challenges for India; 20, 2020, 43.228.93.88.

[46]. *Silvia Duong, PharmD, Tejal Patel, BScPhm, PharmD, and Feng Chang, BScPhm, PharmD*; Dementia What pharmacists need to know; Can Pharm J (Ott). 150(2), 2017, 118–129. Published online doi: 10.1177/1715163517690745.

[47]. *R Overshott, A Burns*; treatment of dementia; J Neurol Neurosurg Psychiatry 76(V), 2005, v53–v59. doi: 10.1136/jnnp.2005.082537.

[48]. *Shaji KS, Jotheeswaran AT, et al*; Alzheimer's & Related Disorders Society of India. The Dementia India Report: prevalence, impact, costs and services for Dementia: Executive Summary 2010.

[49]. , *Vishal K et al* / Int. J. of Allied Med. Sci. and Clin. Research 2(2), 2018, 127-130

[50]. *Mark W. Jacobson, et al;* Cognitive and Functional Decline in Huntington's Disease: Dementia Criteria Revisited; Mov Disord. 25(9), 2010, 1163–1169. doi: 10.1002/mds.22953; PMCID: PMC2910142,NIHMSID: NIHMS160637 PMID: 20629124

[51]. *W M van der Flier, P Scheltens*; Epidemiology And Risk Factors Of Dementia; J Neurol Neurosurg Psychiatry 76(V), 2005, v2–v7. doi: 10.1136/jnnp.2005.082867.

How to cite this article: S. Salini, K. Vishal, N. Sriram. Dr. S. Selvakumar. A review on dementia: epidemiology, risk factors, diagnosis, types and management. Int J of Allied Med Sci and Clin Res 2020; 8(1): 110-119.

Source of Support: Nil. **Conflict of Interest:** None declared.