

Awareness of Dementia

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ABSTRACT

Dementia is said to be a major global health problem and in the absence of a cure there has been increasing focus on the risk reduction, timely diagnosis, followed by an early intervention. It is characterized by a collective syndrome rather than a specific disease. It's an overall term that describes a wide range of symptoms associated with a decline in memory or difficulty in recollecting things, problem-solving and reduced command in language, severe enough to reduce a person's ability to perform everyday activities. Therefore the word dementia describes a combination of symptoms which causes a decline in cognition that is significant enough to interfere with normal independent, daily performances. The causes of dementia are multifactorial ranging from primary neurological deficit, neuropsychiatric and medical conditions. Different types of dementia have been identified with different clinical manifestations and underlying pathologies. It is important to identify the factors that may delay the onset, slow the progression and prevent cognitive decline. These multiple underlying diseases may contribute to the onset of dementia on the long run. Neurodegenerative dementias, like Alzheimer disease and dementia with Lewy bodies, are most common in the elderly, while traumatic brain injury and

brain tumors are common causes in younger adults. It has been an ongoing process for researchers and clinicians from various disciplines and medical specialties aggressively trying to identify the root cause attempting to treat the contributing factors by prescribing medications. The main objective is to improve cognitive decline and motor symptoms, promote evidence-based brain-healthy behaviors and improve overall quality of life for patients and families. This paper highlights several challenges that needs to be revisited and addressed in order to provide adequate and effective care for people with dementia as the condition deteriorates.

Key words: Alzheimer's disease, Cognitive decline, Neurological deficit, Lewy bodies, Brain tumours.

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INTRODUCTION

The term "Dementia" is described as a collection of signs and symptoms, which is referred to an acquired decline in memory and some cognitive function. Dementia is a progressive neurodegenerative disease characterized by impairment in memory, language sensory awareness and changes in personality.¹⁻² It is an illness of progressive deterioration leading to eventual death. However, the trajectory of dementia is unpredictable with average life expectancy ranging from 3 to 10 years.³⁻⁴

The series of events occurring in dementia is only evident over longer periods when it is chronic in nature with mild to severe cognitive decline, hallucinations and delusions occurring against a background of clear consciousness.⁵ Dementia is not a single disease in itself; it is a collective symptom of several underlying diseases and brain disorders. In general, the term describes symptoms related to impairments of memory, communication and thinking.⁶ The loss of this mental ability is severe enough to interfere with normal daily activities lasting more than six months, most definitely not present since birth and not associated with a loss or alteration of consciousness. While the likelihood of having dementia increases with age, it is not a normal part of aging process. Light cognitive impairments, such as reduced short-term memory, can happen as a normal part of aging. This is known as age-related cognitive decline rather than dementia because it does not cause significant problems. This condition is not considered as a critical issue by health and social care practitioners. On the other hand people with severe dementia have complex physical and psychological need. While the overwhelming number of people with dementia is elderly, dementia is not an inevitable part of aging; instead, it is often believed to be caused by specific brain diseases. There are more than hundred types of dementia and the most common is said to be Alzheimer's disease (AD), followed by vascular or multi-infarct dementia.⁷

The prevalence of dementia is difficult, as it has been mentioned before partly because of the differences in the contributing factors and lack of studies among researchers and specialist, which includes diagnostic criteria and different mean population ages. According to different

studies, there is also some normal decline in functional ability with age. The prevalence roughly doubles for every five years of age beginning at age 60. Dementia incidence increases with age between the ages of 65 and 90 years and doubles approximately every 5 years.⁸ Dementia affects about 1% of people between ages 60 and 64, 5-8% of all people between ages 65 and 74, up to 20% of those between ages 75 and 84 and between 30% and 50% of those age 85 and older. The prevalence doubles with every five years of age within that range, with few differences taking into account secular changes, age, gender and place of living.⁹ According to studies done approximately 60% of patients staying in nursing home usually develop dementia. The prevalence of dementia also varies worldwide. The prevalence of dementia roughly doubles for every five years of age beginning at age 60.¹⁰ Prevalence and incidence reveals that the number of people with dementia will continue to rise, particularly among the older individuals. The total number of people with dementia worldwide is projected to nearly double every 20 years, to 65.7 million in 2030 and 115.4 million in 2050. The total number of new cases of dementia each year worldwide is nearly 7.7 million, implying one new case every four sec. The number of people with dementia worldwide is expected to grow substantially as the population ages and exposure to the contributing factors.¹¹ Thus in the category of non-communicable diseases, dementia, have been recognized as the major threat to the world population. The World Health Organization estimates that 35.6 million people live with dementia and it is anticipated to triple by 2050.¹²

Dementia: Types of Dementia

Dementia in latin is described as a departure from previous mental functioning. It is a clinical diagnosis requiring new functional dependence on the basis of progressive cognitive decline. The most common of these is Alzheimer's disease (50-75%) followed by vascular dementia (20%), dementia with Lewy bodies (5%) and frontotemporal lobar dementia (5%).¹³ There are many other conditions that can cause symptoms of

dementia, including some that are reversible, such as thyroid problems and vitamin deficiencies. Some of the common type of dementia are:

1. *Dementia associated with Alzheimer's disease*

Dementia may result from a brain damage due to an underlying disease, such as Alzheimer's disease or a series of strokes. Alzheimer's disease is the most common cause of dementia but not all dementia is due to Alzheimer's. Currently, 30 million people worldwide suffer from Alzheimer's dementia and the World Health organization projects that this number will triple over the next 20 years.¹⁴ Alzheimer's disease is the most common form of dementia and may contribute to 60–70% of cases. Vascular dementia, which occurs after a stroke, is the second most common dementia type. It is characterized by “plaques” between the dying cells in the brain and “tangles” within the cells. These structures are due to protein abnormalities. The brain tissues in a person with AD have progressively fewer nerve cells and connections due to shrinkage of total brain size.¹⁵

2. *Dementia with Lewy bodies*

Dementia with Lewy bodies is a neurodegenerative condition and it is said to be the second leading cause of dementia after Alzheimer's disease. Dementia with Lewy bodies (DLB) accounts for 15% to 35% of dementia cases. The changes in the brain involves a protein called alpha-synuclein. When these protein develops in the cortex, this will result in dementia. Alpha-synuclein also aggregates in the brains of people with Parkinson's disease but the aggregation pattern may differ from dementia with Lewy bodies. The core symptoms seen in DLB like cognitive decline, visual hallucinations and Parkinsonism, may not always be present as a triad and therefore clinicians may be unaware of other associated symptoms. Thus, the diagnosis is frequently missed by primary care providers and often misdiagnosed as Alzheimer's disease, Parkinson's disease or a primary psychiatric illness.¹⁶

3. *Mixed dementia*

It is a condition where changes representing more than one type of dementia occur simultaneously in the brain. In the most common form, the plaques and tangles associated with nerve cells in Alzheimer's disease are present along with blood vessel changes associated with vascular dementia.¹⁷ Vascular changes typically occur in elderly people, whose brains may be affected by age-related degenerative changes and additional diseases. Thus in many cases, the pathogenesis of the dementia is complex, with vascular lesions interacting with primary neurodegenerative processes.¹⁸ Mixed dementia refers to a diagnosis of two or three types occurring together. The person may show both AD and vascular dementia at the same time with each of the abnormality contributing to the onset of dementia.

4. *Parkinson's disease (PD)*

This is one of the most common neurodegenerative movement disorder which affects approximately 1% of the population over 60 years of age.¹⁹ This is usually marked by the presence of Lewy bodies. Although PD is often considered a disorder of movement and coordination, there are several non-motor signs and symptoms that may cause a considerable amount of burden to the individual leading to the onset of dementia symptoms. According to various study, dementia is common and affects approximately 40% of PD patients during the course of the illness. As the condition progresses, it is most likely results in a progressive dementia similar to dementia with Lewy bodies or AD.²⁰

5. *Huntington's disease*

A condition in which contributes to progressive brain disorder caused by a single defective gene on *Chromosome 4*. It is a rare neurodegenerative disorder of the central nervous system characterized by specific type of uncontrolled choreatic movements, behavioral and psychiatric disturbances and leading to dementia.²¹

6. **Other disorders**

Leading to symptoms of dementia include:

- I. Frontotemporal dementia (also known as Pick's disease)
- II. Normal pressure hydrocephalus
- III. Posterior cortical atrophy (resembles changes seen in AD but in a different part of the brain)
- IV. Down syndrome (increases the likelihood of young-onset AD).²²

Causes of Dementia

Dementia is usually caused by degeneration of brain cells in the cerebral cortex which is the part of the brain responsible for thoughts, memories, actions and personalities. Death of brain cells in this region leads to the cognitive impairment, which is collectively known as dementia.

The most common cause of dementia is AD with progressive brain cells death, accounting for one-half to three-fourths of all cases.²³ The brain in a person with AD becomes clogged with two abnormal structures called neurofibrillary tangles and senile plaques. Neurofibrillary tangles are twisted masses of protein fibers inside nerve cells (neurons) while senile plaques are composed of parts of neurons surrounding a group of proteins called beta-amyloid deposits. The exact etiology for the accumulation of these deposits is still unknown, however current research studies indicates several possible factors like inflammation, restriction in blood flow and molecular fragments known as free radicals maybe responsible for the development of dementia.²⁴

In addition to that several genes have been associated with higher incidences of AD but the exact role of these genes is still unclear. Researchers in Duke University discovered in the early 1990s, that potentially the most important genetic link to AD is chromosome 19. A gene on this chromosome called APOE (apolipoprotein E), codes for a protein involved in transporting lipids (fats) into neurons. Certain variations of this gene appear to increase the possibility of developing AD and/or lower the age at which symptoms occur.²⁵ It is also said that there are as many as seven other AD risk-factor genes also exist. In 2007, scientists identified a possible risk factor in four new AD-related regions in the human genome. In these regions, one out of several hundred genes may be a risk factor. One of the genes called SORL 1 has drawn particular research attention. This gene is involved in regulating the transport of certain proteins in the cell.²⁶ Another possible cause of dementia is vascular dementia, which is estimated to cause approximately 5-30% of all dementias. As the name suggest, it occurs from a decrease in the blood flow to the brain, depriving brain cells of oxygen and this leads to a series of small strokes as in multi-infarct dementia.²⁷ Other cerebrovascular causes include vasculitis from syphilis, Lyme disease or Systemic Lupus Erythematosus (SLE), Subdural hematoma and subarachnoid hemorrhage. Vascular dementia is usually sudden in nature and therefore symptoms related to this tend to begin more abruptly than those of Alzheimer's dementia. These symptoms may progress step-wise with the occurrence of new strokes. Unlike AD, the incidence of vascular dementia is usually lower after the age of 75.

Moreover, dementia can also be caused by certain types of traumatic brain injury, particularly if it's repetitive in nature, ex. sports players like boxing. These sport injuries have been linked to certain dementias appearing later in life. However, a single brain injury raises the likelihood of having a degenerative dementia such as AD.²⁸

Other conditions that may cause dementia include, Creutzfeldt-Jakob disease, brain tumors, hydrocephalus, multiple sclerosis, prolonged abuse of alcohol or drugs, vitamin deficiency of thiamin, niacin or B12, hyperthyroidism and hypercalcemia.

Symptoms of Dementia

Dementia comes in many forms, mainly it is a progressive disorder of memory loss and impaired cognitive ability. The Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV defines dementia as a decline in memory with impairment of at least one other cognitive function, such as skilled movements (limb apraxia), language (aphasia) or executive function (e.g., planning, attention and abstract reasoning). This decline mentioned above should represent a change from his/her previous behavior; it should impair social and/or occupational functioning; and cannot be accounted for by other psychiatric conditions such as depression, mood disorders or psychosis. This combination of complex symptoms is often noticed by the affected individual and sometimes may be picked up by caregivers or health care workers.²⁹

1. Recent memory loss

Memory loss usually is the first symptom noticed. One of the most common signs is that they might be asking the same question repeatedly. It usually begins with misplacing valuables such as a wallet or car keys, then progresses to forgetting appointments and then to more substantive daily normal issues such as forgetting where the car was parked or the route back home. More profound carelessness, loss of memory will follow, such as forgetting the names and faces of family members.³⁰

2. Impaired abstraction and planning

The affected person may lose the ability to perform familiar tasks, plan activities and draw simple conclusions from facts. For example, difficulty completing familiar tasks may be as simple as making a drink or cooking a meal.³¹ The affected person may not be able understand or adapt into a certain situation and act accordingly in a rational manner.

3. Language and comprehension disturbances

People with dementia may not be able to understand simple instructions or follow the logic of moderately complex sentences. This may further lead to not understanding his or her own sentences and have difficulty forming thoughts into words. For example, forgetting simple words, sentences expressing him or herself or using the wrong ones which may not be relevant to the subject matter. It is often confusing as to what they are trying to say or respond to a conversation.³² Language deficits are frequent in dementia and among other signs involved is usually word-finding problems (anomia), sentence comprehension deficits and lack of cohesion in discourse. Aphasias that are due to focal brain damage differs from the language deficits in dementia which occurs in the context of multiple cognitive impairments.³³

4. Impaired orientation ability

The affected individual may not be able to identify the time of day, may not recognize his or her location, despite living in the same familiar area for a long time. This disability may stem partly from losses of memory and partly from impaired abstraction. This combined disorientation with regards to time, place and person is a classic triad often seen in people with dementia and certain neurodegenerative disorders.

5. Poor judgment

They may not recognize the consequences of his or her actions or be able to evaluate the appropriateness of their behaviour or level and the risk. The behaviour may become rude, overly friendly or aggressive. Personal appearance and hygiene may be ignored.³⁴ Attention span is markedly reduced associated with restlessness whereby the affected person may begin as impulsive activity and soon after quickly loose interest in it. For example, he or she may begin to cook something on the stove, then become distracted without any specific reason and walk away while the food is still being cooked not realising their action.

6. Behavioural changes and psychosis

Personality and mood changes often surfaces without apparent reason such as becoming irritable, suspicious, lack of confidence, confused, emotional and fearful. They may lose interest in the same activities which was part of their lives and become more passive, goes on to develop sleep disorders like insomnia, depressed or anxious. In addition to that delusions, suspicion, paranoia and hallucinations may occur later as the disease progresses. (Having said these symptoms above, we know that it is a combination of various cognitive decline which may or may not be brought about by a predisposing factor or any underlying neurological disorder.³⁵

Diagnosis of Dementia

Taking a history and the examination of the memory impaired patient is vital. It is very important to obtain a detailed history from a relative or close friend in addition to the patient's presentation if they can provide one.

Therefore diagnosis of dementia involves a proper history taking, clinical examinations and also a few laboratory tests.³⁶ During the history taking, it is mandatory to test the patient's mental status as well as his or her memory performances. The level of alertness and cooperation during the interview should be assessed. If the patient appears poorly responsive, delirium as opposed to dementia should be considered. Some of the red flags or important features to note in the history include:

- The onset of symptoms.
- The impact on work and family life.
- Family history of dementia.
- Past medical history.
- Risk factors.

The first step in testing memory performance and cognitive health involves standard questions. Asking simple questions, such as "What is your father's name?" "Where do you live?" will give an indication of whether there is dementia or not and help to decide whether further investigation is needed. Moreover, simple word knowledge tests and drawing tasks are included alongside with memory questions. Diagnosing dementia can be difficult owing to its insidious onset, symptoms resembling "normal ageing" memory loss and a diversity of other presenting symptoms—for example, difficulty in finding words or making decisions. Research also has shown that dementia cannot be diagnosed without using the simple standard tests below. Upon completing the test and recording all the answers, we can draw some conclusion leading to early diagnosis of the condition. However, diagnosis of dementia also takes account of other pathology as well which was discussed earlier.³⁷

1. Cognitive dementia tests

Cognitive dementia tests is currently widely used and has been verified as a reliable way of indicating dementia. The test has been changed a little since being established in the early 1970s. The abbreviated mental test score has 10 questions, which includes as follows:

- What is your age?
- What is the time, to the nearest hour?
- What is the year?
- What is your date of birth?
- etc.

Each correct answer will be awarded one point; hence scoring six points or fewer suggests cognitive impairment.³⁸

2. General Practitioner Assessment of Cognition (GPCOG) test

This includes an added element for recording the observations of relatives and caregivers. It is designed for doctors as this sort of test may be the first formal assessment of a person's mental ability. The second part

of this test probes someone close to the patient such as family members or friends and includes few questions to find out whether the patient has:

- Unable to remember recent events or conversations.
- Struggles to find the right words or using inappropriate ones.
- Finds difficulty managing money or medications.
- Needs more help with transport or getting around.

If the test does indicate memory loss, standard investigations is recommended, including routine blood tests and a CT brain scan. Clinical tests will identify or rule out treatable causes of memory loss and help to narrow down the potential cause's.

3. Mini-mental state examination (MMSE)

This is a cognitive test which measures:

- Orientation to time and place
- Word recall
- Language abilities
- Attention and calculation
- Visuospatial skills

The MMSE is used to help diagnose dementia caused by AD and also to rate its severity and whether drug treatment is needed.³⁹

4. Structural Magnetic Resonance Imaging (MRI)

Structural imaging should be carried out at least once in the diagnostic work-up of patients with cognitive impairment which serves at least three purposes: to exclude other potentially treatable diseases, to recognize vascular lesions and to identify specific findings to help distinguish different forms of neurodegenerative types of dementia. MRI is currently the imaging modality of choice for assessing subjects with suspected dementia. However, where MRI is not available or contraindicated, computed tomography (CT) scans can be useful instead to exclude major space occupying lesions, large infarcts and hydrocephalus. Multi-detector row CT is the best alternative for patients who cannot undergo MRI.⁴⁰

5. Functional Imaging

Although typical cases of dementia may not benefit from routine Single Positron Emission Computed Tomography (SPECT) or Positron Emission Tomography (PET) imaging, these tools are usually recommended in cases where diagnosis remains in doubt after clinical and structural MRI work-up and in particular clinical settings. Functional imaging can be of value to diagnose (or exclude) a neurodegenerative dementia in those subjects with cognitive impairment presenting with severe psychiatric disturbances (ex. depression and agitation) and in cases where proper cognitive testing is difficult, that is, with no language in common with the clinician who is handling the case.⁴¹

Since dementia usually progresses slowly, diagnosing it in its early stages can be very difficult. However, prompt intervention and treatment has been shown to help slow the effects of certain types of dementias. This goes on to specify that early diagnosis is important to facilitate appropriate and prompt treatment.

Treatment of Dementia

Dementia is characterized with brain cell death and brain cell death cannot be reversed, so there is no known cure for degenerative dementia.

However, management of AD is particularly focused on providing care and treating symptoms rather than their underlying cause (Table 1). If dementia symptoms are due to a reversible, non-degenerative cause, then, treatment may be possible to prevent or halt further brain tissue damage. Examples include injury, medication effects and vitamin deficiency. It is therefore important to identify potentially reversible dementias and treatment should be considered, even if the symptoms are not sufficiently severe to meet the clinical criteria for dementia.⁴² In treating AD,

symptoms can be reduced by some medications. There are four drugs, known as cholinesterase inhibitors which have been used for decades to treat AD. They are:

- Donepezil
- Alantamine
- Rivastigmine
- Tacrine

A different kind of drug, memantine, an NMDA (N-Methyl-D-aspartate) receptor antagonist may also be used, alone or in combination with a cholinesterase inhibitor.⁴³ In addition to the above drugs, psychotic symptoms including paranoia, delusions and hallucinations may be treated with antipsychotic drugs such as haloperidol, risperidone, quetiapine and olanzapine. Side effects of these drugs can be significant. Antianxiety drugs such as buspirone may improve behavioral symptoms, especially agitation and anxiety. Depression is treated with antidepressants, usually selective serotonin reuptake inhibitors (SSRIs) such as sertraline or paroxetine.⁴⁴ Sleep disorders can also be treated although many drugs for insomnia are recommended for short-term use only. Medications generally should be administered cautiously and in the lowest possible effective doses to an individual with dementia in order to minimize the side effects.

Caregivers are also advised accordingly to actively supervise the administration of all the medications.⁴⁵

On the other hand, alternative treatment is also available for dementia. Antioxidants, which act to protect against oxidative damage caused by free radicals, have been shown to inhibit toxic effects of beta-amyloid in laboratory tissue cultures. In addition, vitamin E which is an antioxidant, is thought to delay AD onset. However, it is still unclear whether this is due to the specific action of vitamin E on brain cells or to an increase in the overall health of those taking it. Research is still being conducted to determine if vitamin E or other antioxidants may delay or prevent AD.⁴

Prevention of Dementia

The most effective approach to delay the onset and potentially reduce the number of new cases mainly involves in the reduction of certain risk factors that are known to be associated with dementia. However, age is the biggest predictor.⁴⁸

Table 1: Management of Dementia.

	Concentrate on what you can still do/would like/need to do
	Approach each task positively with a calm manner.
For the person with dementia:	Do things at times when you feel good, e.g. in the morning, afternoon or evening.
	Take your time with no deadline or restrictions.
	Simplify tasks and break them down into several stages.
	Allow yourself to grieve the loss of a skill.
	Try to plan ahead.
	Anticipate difficulties based on what you notice or suspect may be difficult for the person with dementia.
	Remember that you may need to adapt your solutions again and again. Be flexible and above all with extreme patience.
For the carer:	Encourage the person with dementia to do as much as possible by themselves to give them confidence.
	Don't rush the person but allow more time involving a particular task.
	If the person with dementia makes a mistake, don't ask why and expose them and certainly never to criticize their faults. ⁴⁷

Some of the risk factors which most likely contributes to the onset of dementia include:

- Smoking and alcohol use
- Atherosclerosis (a cardiovascular disease causing the arteries to be narrow)
- High levels of “bad” cholesterol (low-density lipoprotein)
- Above-average blood levels of homocysteine (a type of amino acid)
- Diabetes
- Mild cognitive impairment can sometimes, but not always, lead to dementia.

Besides, it is important to implement public health interventions and awareness based on knowledge of risk factors for AD. Use of interventions to modify known risk factors is even more important in the absence of effective pharmacological treatments for AD.

ABBREVIATIONS

AD: Alzheimer’s Disease ; **DBL:** Dementia with Lewy Bodies; **PD:** Parkinson’s Disease; **APOE:** Apolipoprotein E; **SLE:** Systemic Lupus Erythematosus; **DSM:** Diagnostic and Statistical Manual of Mental Disorders; **GPCOG:** General Practitioner Assessment of Cognition; **MMSE:** Mini Mental State Examination; **SPECT:** Single Positron Emission Computed Tomography; **PET:** Positron Emission Tomography; **NMDA:** N-Methyl-D -Aspartate; **SSRI:** Selective Serotonin Reuptake Inhibitors.

CONFLICT OF INTEREST

Authors declare that there no conflict of interest.

REFERENCES

1. Colin M, Matilde L. Global burden of dementia in the year 2000: summary of methods and data sources. *Global Burden of Disease*. 2000.
2. Debbie T, Yves R, Sandrine A, Jean PA, John B, Athanase B, *et al*. International Association of Gerontology and Geriatrics: A Global Agenda for Clinical Research and Quality of Care in Nursing Homes. *Journal of the American Medical Directors Association*. 2011;12(3):184-9.
3. Claire G, Catherine E, Jane W, Katherine F, Vari D, Elizabeth S, *et al*. End of life care for community dwelling older people with dementia: an integrated review. *Int J Geriatr Psychiatry*. 2010;25(4):329-37.
4. Shega JW, Levin A, Hougham GW, Cox HD, Luchins D, Hanrahan P, *et al*. Palliative Excellence in Alzheimer Care Efforts (PEACE): a program description. *J Palliat Med*. 2003;6(2):315-20.
5. Jenny TVDS. Dying with Dementia: What We Know after More than a Decade of Research. *Journal of Alzheimer’s Disease*. 2010;22(1):37-55.
6. Markus M. Dementia: Symptoms, stages and types. *Medical News today*. 2017.
7. Chengxuan Q. Epidemiology of Alzheimer’s disease: occurrence, determinants and strategies toward intervention. *Dialogues Clin Neurosci*. 2009;11(2):111-28.
8. Jorm AF, Jolley D. The incidence of dementia: a meta-analysis. *Neurology*. 1998;51(3):728-33.
9. Henderson S. Epidemiology of dementia. *Annales de Medecine Interne*. 1998;149(4):181-6.
10. Liara R, Idiane R, Matheus RC. Global Epidemiology of Dementia: Alzheimer’s and Vascular Types. *BioMed Research International*. 2014. Article ID 908915.
11. Ferri CP, Prince M, Brayne C, *et al*. Global prevalence of dementia: a Delphi consensus study. *The Lancet*. 2005;366(9503):2112-7.
12. World Health Organization. Dementia A Public Health Priority. World Health Organization. 2012.
13. Cunningham EL, McGuinness B, Herron B, Passmore AP. Dementia Ulster *Med J*. 2015;84(2):79-87.
14. Wimo A, Jonsson L, Winblad B. An estimate of the worldwide prevalence and direct costs of dementia in 2003. *Dement Geriatr Cogn Disord*. 2006;21(3):175-81.
15. Brent IG, El-Agnaf OM, Ganesh MS, Dominic MW. Protein Aggregation in the Brain: The Molecular Basis for Alzheimer’s and Parkinson’s Diseases. *Mol Med*. 2008;14(7-8):451-64.
16. Zupancic M, Mahajan A, Handa K. Dementia With Lewy Bodies: Diagnosis and Management for Primary Care Providers. *The Primary Care Companion to CNS Disorders*. 2011;13(5):PCC.11r01190.
17. Raj NK, Timo E. Small Vessel Disease and Subcortical Vascular Dementia. *J Clin Neurol*. 2006;2(1):1-11.
18. Korczyn AD. Mixed dementia—the most common cause of dementia. *Ann NY Acad Sci*. 2002;977(1):12934.
19. Dodel R. Dementia in Parkinson’s Disease. *Orphanet Encyclopedia*. 2004. <http://www.orpha.net/data/patho/GB/uk-PDD.pdf>
20. Diego I, Geraci-Erck M, Marcie LR, Charles HA, Geidy S, Thomas GB. Parkinson disease and incidental Lewy body disease. *Neurology*. 2015;85(19):1670-9.
21. Raymond ACR. Huntington’s disease: a clinical review. *Orphanet J Rare Dis*. 2010;5:4.
22. Alzheimer’s Society’s factsheets at www.alzheimers.org.uk/factsheets.
23. Rossen D, Martin K, Bruno M, Johannes T. Neuronal death in Alzheimer’s disease and therapeutic opportunities. *J Cell Mol Med*. 2009;13(11-12):4329-48.
24. Holger J. Memory loss in Alzheimer’s disease. *Dialogues Clin Neurosci*. 2013;15(4): 445-54.
25. Julie H, Mary G. Dementia and Cognitive Impairment: Epidemiology, Diagnosis and Treatment. *Clin Geriatr Med*. 2014;30(3):421-42.
26. Daniel WH, Jacob LM, Amy EC, Lynne LM, Perry CG, Lan J. Journal title??? *Am J Med Genet B Neuropsychiatr Genet*. 2006;141B(2):160-6.
27. Langa KM, Foster NL, Larson EB. Mixed dementia: emerging concepts and therapeutic implications. *JAMA*. 2004;292(3):2901-8.
28. Ramalho J, Castillo M. Dementia resulting from traumatic brain injury. *Dement Neuropsychol*. 2015;9(4):356-68.
29. Scott, Barrett. Dementia syndromes: evaluation and treatment. *Expert Rev Neurother*. 2007;7(4):407-22.
30. Carl AG. Memory loss in Alzheimer’s disease: implications for development of therapeutics. *Expert Rev Neurother*. 2008;8(12):1879-91.
31. Corina S, Luiza G, Carlos T. Planning ability impairments in probable Alzheimer’s disease patients: Evidence from the Tower of London test. *Dement Neuropsychol*. 2017;11(2):137-44.
32. Steven HF, Martin F. Language impairment in Alzheimer’s disease and benefits of acetylcholinesterase inhibitors. *Clin Interv Aging*. 2013;8:1007-14.
33. Daniel K, Mira G. Language and Dementia: Neuropsychological Aspects. *Annu Rev Appl Linguist*. 2008;28:73-90.
34. Soumya H, Ratnavalli E. Capacity issues and decision-making in dementia. *Ann Indian Acad Neurol*. 2016;19(Suppl 1):S34-S39.
35. Nilamadhab K. Behavioral and psychological symptoms of dementia and their management. *Indian J Psychiatry*. 2009;51(Suppl1):S77-S86.
36. Cooper S, Greene JDW. The Clinical Assessment of The Patient With Early Dementia. *J Neurol Neurosurg Psychiatry*, 2005;76(Suppl V):v15-v24.
37. Alan MA, Mel PD. Initial Evaluation of the Patient with Suspected Dementia. *Am Fam Physician*. 2005;71(9):1745-50.
38. Bart S. Assessment scales in dementia. *Ther Adv Neurol Disord*. 2012;5(6):349-58.
39. Kelvin KFT, Joyce YCC, Hoyee WH, Samuel YSW, Timothy CYK. Cognitive Tests to Detect Dementia. A Systematic Review and Meta-analysis. *JAMA Intern Med*. 2015;175(9):1450-8.
40. Scheltens P, Fox N, Barkhof F, Carli CD. Structural magnetic resonance imaging in the practical assessment of dementia: beyond exclusion. *Lancet Neurol*. 2002;1(1):13-21.
41. Filippi M, Agosta F, Barkhof F, Dubois B, Fox NC, Frisoni GB, *et al*. EFNS task force: the use of neuroimaging in the diagnosis of dementia. *Eur J Neurol*. 2012;19(12):e131-40.
42. Manjari T, Deepti V. Reversible dementias. *Indian J Psychiatry*. 2009;51(Suppl1):S52-S55.
43. Howard R, McShane R, Lindesay J, Ritchie C, Baldwin A, Barber R, *et al*. Donepezil and Memantine for moderate-to-severe Alzheimer’s disease. *N Engl J Med*. 2012;366(10):893-903.
44. James MF. SSRI Antidepressant Medications: Adverse Effects and Tolerability. *Prim Care Companion J Clin Psychiatry*. 2001;3(1):22-7.
45. Steven DT. Treating Psychotic Symptoms in Elderly Patients. *Prim Care Companion J Clin Psychiatry*. 2001;3(4):156-63.
46. Sucholeiki, Roy, *et al*. Dementia: Overview of Pharmacotherapy. *eMedicine.com*. 2007. Retrieved from <http://emedicine.medscape.com/article/1136306-overview> accessed 18 Feb 2017.
47. Managing everyday tasks. *Alzheimer Europe 2009*. Retrieved from <http://www.alzheimer-europe.org/Living-with-dementia>.
48. Rodolfo S, Ronald CP. Prevention of Dementia. *Psychiatr Clin North Am*. 2011;34(1):127-45.