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Understanding Dementia: A Review

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ABSTRACT :
According to the World Health Organization (WHO), dementia is "an umbrella term for several diseases affecting memory, other cognitive abilities and behavior that interfere significantly with a person's ability to maintain their daily living activities. It is not a normal part of aging". Various diseases can cause dementia; these will be discussed below. Since dementia is not caused by a single disease, the primary cause of the disease is unknown and thus we remain powerless to treat it. The medicaments only help in relieving some of the symptoms.The reason stems from our incomplete understanding of the deep biology of the contributing diseases and associated epigenetic/ ecogenetic influences. After a brief introduction of the disease, I will elaborate on the different types of dementia, its causes,signs and symptoms (early, middle and late), risk factors, and different progressive stages of dementia. I will also review the treatment for the disease. I will further discuss the main contributors to dementia (Alzheimer disease; the various dementias: vascular, Lewy body, Parkinson, frontotemporal, and; normal pressure hydrocephalus; and Creutzfeldt-Jacobs disease) and other contributors. Management of the disease and the associated psychopharmacotherapy are also detailed, although the medications used have little or no effect on the underlying disease process. Lastly complementary and preventive measures are outlined.

Keywords:
Dementia, Memory loss, Alzheimer's, Parkinson's, Care for patients with dementia, Treatment, Therapy.

INTRODUCTION:

Dementia is a syndrome,in which there is deterioration in cognitive function beyond what might be expected from the usual consequences of biological ageing.

Cognitive domains such as language,Visuo-spatial or executive function are affected,which are sufficient to interfere with social or occupational functioning in an alert person. It affects memory,thinking, orientation, comprehension,calculation,learning capacity,language and judgement.

The impairment in cognitive function is commonly accompanied and occasionally preceded by changes in mood,emotional control,behaviour or motivation.

There are various/ multiple diseases which can cause the syndrome of dementia. The large majority of people with dementia have neurodegenerative disease or cerebrovascular ischemia as the underlying cause.

Between 60%-70% of people with the dementia syndrome have Alzheimer's disease,about 20% - 30% have vascular or mixed vascular and Alzheimer's disease.

A smaller number have other causes such as Lewy body dementia , frontal dementia, Parkinson's disease, Hypothyroidism and Vitamin B12 deficiency.

The term Dementia means "being out of one's mind." The term dementia was used since 13th century but its was mentioned in the medical community only in the 18th century.

Types of behavioural andpsychological symptoms of dementia (BPSD): Aggression,Agitation, Apathy,Biting, Delusion, Depression,Hallucinations, Kicking, Restlessness, Screaming,Shaking, Poor sleep, Wandering.

Types:

Alzheimer's Disease

Alzheimer's disease is a progressive, neurodegenerative disease.It is irreversible in nature. Alzheimer occurs when nerve cells, present in the brain die. As a result it negatively impacts cognitive abilities, such as thinking skills and reasoning and is responsible for a loss in memory. There are two types of Alzheimer's disease:

Early-onset Alzheimer's: The people with age group 30-60 are mostly affected.

Late-onset Alzheimer's: More prominent in patients with 60 years or more.

Alzheimer's disease accounts for 50% to 80% of cases, causing dementia.

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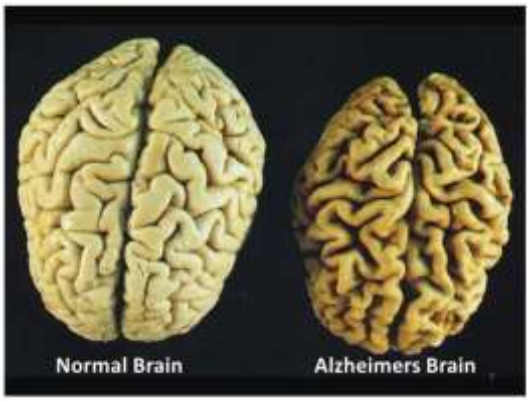
Causes

Alzheimer’s disease is thought to be caused by the abnormal build up of proteins in an around brain cells. One of the protein involved is called Amyloid, which when deposited forms “ PLAQUES” around the brain cells. The other protein is called as “TAU”, deposits of which form tangles within the brain cells. Also,it has been observed that the levels of “Acetylcholine” decreases, which is a neurotransmitter; involved in sending signals between the brain cells. Over the time, shrinking of different areas of the brain is observed. The very first region of the brain to be affected, is the HIPPOCAMPUS – which is responsible for forming memories and spatial navigation. People with DOWN SYNDROME are at higher risk of developing Alzheimer’s.



Treatments

There is no cure forreversing the destruction of brain cells caused by Alzheimer's disease but there are different treatments available to slow or manage the symptoms of the disorderinorderto help improve the quality of life for both the patient and the patient’s family and friends.

Medication helping to treatmemory loss, communication skills, certain behavioral problems, and other symptoms of Alzheimer's diseaseare: donepezil (Aricept), rivastigmine (Exelon), galantamine (Razadyne), and memantine (Namenda). All the four are approved by the USFDA.

There are also ways of managing symptoms of Alzheimer's without the use of drugs, including different types of therapies, like art or music therapy, which can stimulate the senses and improve a patient's mood and behavior. Taking supplemental remedies, like omega-3 fatty acids, coral calcium, and ginkgo biloba, have likewise been shown to promote memory and delay symptoms associated with Alzheimer's disease. Furthermore, studies have shown that continuous cognitive training and regular exercise may help slow the progression of Alzheimer's symptoms.

Parkinson's Disease

Parkinson's disease is a chronic and progressive neurodegenerative disorder that affects movement and is caused by destroyed nerve cells in the brain. It initially causes tremor in one hand and stiffness or slowing of movement.The 14kDa protein ALPHA SYNUCLEIN is strongly associated with PD. There is no cure for Parkinson's disease, but symptoms can be treated, and in some cases improved, with certain medications and other forms of treatment.

It is estimated that approximately 50% to 80% of those with Parkinson's disease will eventually develop at least mild dementia, while up to 20% will develop full-blown dementia. Symptoms of dementia may not be apparent until up to a decade after a Parkinson's diagnosis.

Causes

The pathological changes in patients with parkinson’s disease are mainly neuronal damage in the substantia nigra compact area and abnormal accumulation of Lewy body (LB) in the brain, resulting in a decrease in dopamine content in the brain and an imbalance of dopamine and acetylcholine, causing related clinical motor symptoms.

Treatments

Levodopa and carbidopa (Duopa, Rytary, Sinemet) .Levodopa, also known as L-DOPA, has been long been and continues to be the most effective drug in treating Parkinson disease symptoms. Others are DOPAMINE AGONISTS (Merapexin), MAO-B INHIBITORS (Azilect), COMT INHIBITORS (Comtess), Amantadine, ANTICHOLINERGICS (Kemadrin).

If medication proves ineffective in treating an individual's symptoms of Parkinson's disease, there is a surgical procedure, known as deep brain stimulation (DBS) surgery that may be performed. DBS surgery involves implanting electrodes on specific areas of the brain as well as an impulse generator in the chest. The generator can then be adjusted to send electrical impulses to the brain in order to reduce involuntary movement problems, tremors, and other symptoms of Parkinson's disease. As with all types of surgery, there are

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certain risks involved, including infections, stroke and bleeding, so talk with a doctor first about the risks, complications, and expectations of the procedure.

Huntington's disease

Huntington's disease is a progressive disorder that causes the brain to lose nerve cells, affecting the part of the brain that regulates mood, movement and cognitive skills. About 30,000 people in the United States have Huntington's disease. To date, there is no known cure, so themanagement of symptoms is the primary focus of treatment.

Causes

Huntington's is caused by a genetic “stutter" on one of the 23 chromosomes containing the human genetic code. A person whose mother or father had Huntington's disease has a 50 percent chance of inheriting the genetic defect that causes the disorder.

Treatment

Medication, psychotherapy and occupational therapy are a few of the treatments used to help treat the conditions associated with Huntington's disease. In addition, caregivers can do a great deal to help patients manage their symptoms.

According to the Alzheimer's Disease Association, atypical antipsychotic drugs or SSRI antidepressants can be used to treat anger and threatening behavior. Obsessive-compulsive behavior, depression and anxiety can also be treated with medication. Psychotherapy can help Huntington's patients to develop coping skills, communicate more effectively with family members and caregivers and control behavioral issues.

Vascular Dementia

Vascular dementia is a decline in thinking and cognitive skills caused by a blockage or reduced blood flow to the brain, which deprives the brain of much needed oxygen and nutrients. It is the second most common cause for dementia after Alzheimer's disease, accounting for about 10% of all cases. Vascular dementia is most frequently associated with stroke, but high cholesterol, high blood pressure and diabetes may also contribute to vascular dementia.

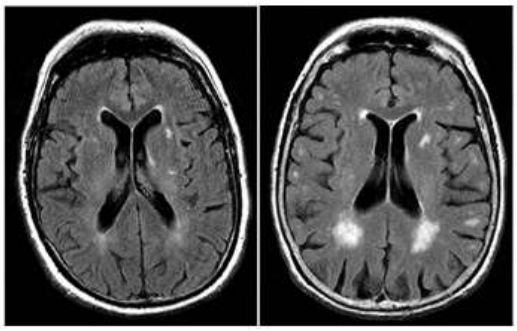
Causes

Vascular dementia is caused by brain damage due to impaired blood flow to the brain. The most common cause is stroke, but vascular dementia can also be the result of any condition that narrows or damages blood vessels, including brain hemorrhage, diabetes, and the normal wear-and-tear of aging. Transient ischemic attacks, or "mini strokes," can also increase the risk of developing vascular dementia.

Treatments

Medicines such as low dose aspirin or clopidogrel to reduce the risk of blood clots and further strokes.Anticoagulant medicines such as warfarin,which also reduce the risk of blood clots and further strokes.

The Mayo Clinic recommends certain lifestyle changes that may help reduce the risk of vascular dementia, such as regular exercise, a low-fat diet, quitting smoking, and controlling high blood pressure or diabetes.



Normal Pressure Hydrocephalus

Normal pressure hydrocephalus (NPH) is a type of dementia caused by a buildup of cerebrospinal fluid in the ventricles of the brain. This fluid buildup leads ventricles to enlarge and eventually damages surrounding brain tissue, leading to cognitive impairment and symptoms like loss of bladder control and difficulty walking.

Causes

While there is no known Personality and behavior changes
Apathy and withdrawal
Speech problems
Mood change

Brain surgery complications

singular cause for normal pressure hydrocephalus, professionals have identified high risk factors for

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developing the affliction. It's been identified that NPH can occur because of:
Meningitis
Other brain injuries, infections, or inflammation.

Treatments

While many NPH symptoms are similar to Alzheimer's disease symptoms, the good news is that unlike Alzheimer's disease, NPH is treatable and reversible to a certain point. While there are currently no effective medication-based or nonsurgical treatments for the affliction, a surgical procedure involving the insertion of a tube to drain excess brain fluid has shown very positive clinical results. Though NPH is not completely curable, this surgical procedure usually leads to substantial improvements in walking abilities, and minimal improvements in bladder control and cognitive impairment. For those unwilling or unable to go through surgery, medication can help alleviate and control symptoms.
Treatment for NPH involves surgical placement of a shunt in the brain to drain excess CSF into the abdomen where it can be absorbed as part of the normal circulatory process. This allows the brain ventricles to return to their normal size.
Acetazolamide is the most suitable drug alone or in combination with furosemide for treatment of hydrocephaly.

Lewy Body Dementia

Lewy body dementia (LBD), the second most common form of dementia after Alzheimer's Disease, is a brain disorder that results in irreversible cognitive decline and movement problems similar to Parkinson's Disease. Lewy bodies are an alpha-synuclein protein that develops in areas of the brain involved in thinking and motor control. When they build up they can have a negative impact on the brain, affecting memory, thinking skills, movement, mood, and behavior. These abnormal proteins are also found in the brains of Alzheimer's patients, leading experts to believe there may be a Lewy body variant of Alzheimer's, or that a person can have both. The similarities among these types of dementia make diagnosis difficult.

Lewy body dementia refers to both dementia with Lewy bodies and Parkinson's disease dementia.Diagnosis depends on which symptoms occur first. In dementia with Lewy bodies, the patient starts with cognitive problems and later loses control of their movements.

Causes

Lewy body dementia is a disease associated with abnormal deposits of a protein called alpha-synuclein in the brain.These deposits called Lewy Bodies.

Treatment

This complex disease demands a comprehensive treatment plan since every patient is unique in how their symptoms are expressed and how they react to certain medications. Whatever the case, individuals with LBD should never take ANTIPSYCHOTIC, as they are extremely sensitive to these drugs and taking them may worsen symptoms. Talk with a doctor about potential side effects before starting any medication, since Lewy body patients are more likely to have negative reactions to drugs used to treat individual symptoms (e.g. antidepressants, antispasmodics, tranquilizers and surgical anesthetics).

Frontotemporal Dementia

There are multiple kinds of degenerative dementia, all of which affect different parts of the brain in different ways. Frontotemporal Dementia, which has several subtypes of its own, is one of these conditions, almost exclusively affecting the frontal lobe of the brain.

Frontotemporal Dementia (FTD), including a variety commonly referred to as Pick's disease, is a form of dementia that is characterized by a degeneration of the brain's frontal lobe, which sometimes expands into the temporal lobe.

Causes

Many degenerative neurological diseases do not have a strong genetic component, but FTD is believed to be an exception, with a high familial component compared to other instances of dementia. Unlike in other forms of dementia, however, there are no nutritional deficiencies or other habits that increase the likelihood of developing FTD.
Instead, risk factors for developing FTD include:
Mutations in the MAPT and/or GRN genes of chromosome 17
A family history of FTD
Treatments

Like other varieties of degenerative dementia, there is no cure or treatment that can eradicate, prevent or stop the disease from progressing. With proper support, behavioral issues can be managed, though the average length of survival is seven years from the onset of the condition.

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FTD differs from other forms of degenerative dementia, because of its manifestation in the frontal lobe of the brain, and tendency to produce primitive reflexes and inappropriate behaviors. With a genetic correlation and no known cure, the disease can only be symptomatically managed with medical support.

Creutzfeldt-Jakob Disease

Creutzfeldt-Jakob disease, a brain disorder characterized by the introduction and spread of incorrectly folded proteins, causes a degeneration of brain capacity and ultimately leads to death. Unfortunately, there is no current treatment for the disease, but by understanding the scope of the condition, you can better comprehend and support the condition in a loved one.

Creutzfeldt-Jakob disease (CJD) is a degenerative neurological condition sometimes referred to as the human form of Mad Cow disease because of the similarities in symptoms, though the two diseases are completely unrelated. In CJD, the tissue of the brain begins to become spongy because of the presence of prions—incorrectly folded proteins, which replicate by infecting their correctly folded counterparts.

Causes

There are three varieties of the Creutzfeldt-Jakob disease:

Sporadic, in which a person has no previously known risk factors.
Familial, in which the disease manifests from a genetic predisposition.
Variant, the most common form, in which the disease stems from another medical condition or treatment.
Approximately 5 to 10 percent of patients acquire CJD genetically, from a genetic mutation that codes for the particular prion protein. This information is inherited, promoting the incorrectly folded protein to infect others and induce the disease.

The mutated protein can also be transmitted in harvested humanbrain products, including corneal grafts, electrode implants, or intravenous immunoglobulins. The traditional sterilization methods of surgical equipment do not necessarily inactivate prions, so medical equipment can transfer the infectious proteins to new patients if the proper precautions are not taken. Rarely, the disease can manifest through the use of Human Growth Hormone that has been obtained from the pituitary glands of a person who died from CJD. Blood transfusions and sperm donation can transmit these prions as well, leading to heavy restrictions in the donation process.

Treatments

Though research continues, there is no accepted treatment for this fatal disease. Many experiments have attempted to find a means of halting the disease's progression, but no method has successfully prevented the further loss of brain tissue and function.

Further research is pointing toward RNA interference as a possible means of stopping the disease. The RNA may block the production of proteins that turn into prions. This hypothesis is currently being investigated in mice, though it is many years away from any kind of human testing.

Wernicke-Korsakoff Syndrome

Alcohol is linked to the development of Wernicke-Korsakoff syndrome, a brain disorder resulting from thiamine (vitamin B1) deficiency. Wernicke-Korsakoff syndrome is actually a two-stage brain disorder in which Karsakoff syndrome (also known as Korsakoff psychosis) develops due to permanent brain damage as symptoms of Wernicke encephalopathy wane.

Symptoms

Wernicke-Korsakoff Syndrome cannot be diagnosed while a patient is in withdrawal, or experiencing medical complications from their alcohol abuse. A patient must be sober and still experiencing symptoms to be diagnosed with Wernicke-Korsakoff Syndrome.

Causes

Alcohol use can be a risk factor in developing any type of dementia. A recent report from the BBC showed that even moderate alcohol consumption placed people at greater risk for developing dementia.

Wernicke-Korsakoff syndrome is the combination of two conditions with separate stages: Wernicke's encephalopathy and Korsakoff syndrome. Both are caused by damaged parts of the brain due to a lack of vitamin B1 (thiamine). Alcoholics are prone to developing this condition because they usually have poor nutrition, and a decreased ability to properly absorb nutrients. Alcoholism can lead to severe deficiencies in thiamine, which helps the brain process and produce energy from sugar. Without thiamine, the brain cannot create enough energy to sustain itself,

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leading to serious damage and memory loss, among other symptoms. This condition has also been observed in people who lacked diets with thiamine, but were not alcoholics.

Treatment

Unfortunately, doctors and researchers have found that much of the damage wrought by alcohol abuse is permanent; making it very difficult to treat Wernicke-Korsakoff syndrome. People with alcohol-related dementias improve their quality of life at half the rate of those with Alzheimer's disease or vascular dementia.

Experts recommend abstaining from alcohol as a large part of treating these conditions. A second, equally important component, is maintaining a healthy, balanced, nutritional diet to slow the progression of alcohol related dementias. Alcoholics Anonymous and other similar support groups can help those changing the role of alcohol in their lives.

If you have any concerns about you or your loved one's alcohol use and change in behavior, please contact your doctor or a treatment specialist.

Mixed dementia

Mixed dementia, sometimes known clinically as "Dementia - multifactoral", is a dementia condition characterized by symptoms and abnormalities of more than one type of dementia at once. Usually, mixed dementia entails a combination of Alzheimer's disease and vascular dementia. In other cases, mixed dementia involves characteristics from both Alzheimer's disease and Lewy bodies, and sometimes mixed dementia can even entail a combination of all three of these diseases.

Recent research is discovering that mixed dementia may be much more common than previously presumed. This research shows that a large proportion of dementia sufferers over the age of 80 may have mixed dementia, with characteristics of both Alzheimer's disease and vascular dementia.

Causes

Little is known about the specific causes and risk factors of mixeddementia as compared to other dementia conditions. However, the high incidences of mixed dementia in older individuals may suggest that age is a contributor to developing mixed dementia. Risk factors for Alzheimer's disease, vascular dementia, and lewy body dementia all factor in to the incidence of mixed dementia.

As mixed dementia often involves vascular dementia, vascular risk factors may be central to the development of mixed dementia. For example, hypertension and high blood pressure may increase the risk of developing mixed dementia.

Treatments

Unfortunately mixed dementia cannot be cured, though research on possible treatment continues. There are, however, alternative treatments and medications that can be used to treat symptoms of mixed dementia. Specific treatments and medication formixed dementia will depend on the dementia conditions in question.

As mixed dementia cannot be cured or slowed, risk reduction is currently one of the most important areas of dementia research. Current research suggests that diet, alcohol abuse, exercise, cardiovascular disease, cholesterol levels, incidences of mild cognitive impairment, diabetes, and smoking are all contributors to the development of dementia. Positive lifestyle changes, especially during middle age, can be integral to dementia prevention.

What Causes Dementia?

Reversible Dementias

Dementia is generally caused by damage to or disruptions of brain cells, particularly in the cerebral cortex (the part of the brain which controls memory, perception, consciousness, and language). In some cases, this damage or disruption isn't permanent, causing reversible dementia conditions that can be slowed or cured with proper treatment.

Delirium

Especially in the older population, infections and fevers can cause delirium, a state of extreme disorientation that is often confused with dementia because it shows many of the same symptoms. Delirium is noticeably more abrupt and sudden than dementia and can worsen or improve drastically in a matter of days or hours. Delirium is not caused by gradual damage to the brain but rather by external factors like infections (UTIs or influenza), withdrawal, strokes, or adverse reactions to medications.

Vitamin B12 Deficiency

Vitamin B12 assists in the production of red blood cells which transport oxygen to the brain, so impaired absorption of vitamin B12 or a vitamin

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Volume 7, Issue 5 Sep-Oct 2022, pp: 335-345 www.ijprajournal.com ISSN: 2456-4494

B12 deficiency can lead to dementia caused by pernicious anemia. Symptoms of pernicious anemia include fatigue, yellowed skin, pica, and shortness of breath along with dementia symptoms like confusion and difficulty concentrating. Usually, pernicious anemia isn't caused by dietary issues or a lack of vitamin B in the diet but rather by an inability to properly absorb and retain the vitamin.

Pernicious anemia is sometimes passed down genetically, through it more frequently results from atrophic gastritis (weakened stomach lining) or autoimmune conditions. Vitamin B12 supplements or injections can reverse pernicious anemia, eliminating the dementia symptoms that accompany it. However, pernicious anemia is not completely curable, so medications must be taken consistently to prevent the onset of dementia symptoms.

Subdural Hematomas

Subdural hematomas, or blood clots in the brain, are caused by bruising from severe head trauma, most commonly from trauma received in vehicle accidents. In the older population, blood clots can occur even as a result of very minor head injuries which may be unnoticed or forgotten. Hematomas can cause dementia and mimic symptoms of Alzheimer's disease, though quickly treating and removing the blood clot can eliminate symptoms and restore brain function.

Thyroid Disease

When the thyroid gland doesn't produce enough thyroid hormones (or overproduces thyroid hormones) overall body functioning decreases, possibly leading to dementia. Thyroid-related dementia affects females up to five times as often as it affects males. Thyroid supplements can fully restore functioning and eliminate dementia symptoms, but life-long treatment and consistent medication is needed to manage thyroid disease.

Tumors

Though it's uncommon, tumors and cancers in certain parts of the brain can damage surrounding tissues and cause dementia. Tumor-related dementia will usually be accompanied by symptoms like headaches, vomiting, intracranial pressure, or rapid personality changes. Treating or removing these tumors will cause dementia symptoms to significantly reduce or disappear.

Toxic Reactions to Drugs or Chemicals

Certain medications or interactions between medications, along with types of poisoning like carbon monoxide, mercury, lead, or other heavy metal poisoning can lead to dementia symptoms which will diminish once exposure to the chemicals or medications ceases.

Heavy drug use and alcoholism (especially alcohol poisoning) can also lead to dementia symptoms that may decrease after the substance abuse is stopped. However, with serious or long-term substance abuse permanent damage may occur, resulting in a condition known as substance-induced persisting dementia.

Irreversible Dementias

Alzheimer's Disease
Vascular Dementia
Huntington's Disease
Parkinson’s Disease
Infectious Dementias
Creutzfeld-Jakobs Disease
AIDS-induced Dementia

Neurosyphilis

Syphilis is a sexually transmitted infection caused by the bacteria Treponema pallidum and spread through direct contact with syphilis sores. Neurosyphilis can develop if syphilis goes untreated long enough for the infection to reach the spinal cord and brain. Syphilis is easy to treat and prevent, and it can take 10-20 years for syphilis to progress into neurosyphilis. Only 25-40% of untreated syphilis cases develop into neurosyphilis, but if the Treponema pallidum bacteria infects the brain or spinal cord it can cause dementia symptoms which mimic Alzheimer's disease.

The Seven Stages Of Dementia

Stage 1: No Cognitive Decline

A patient generally does not exhibit any significant problems with memory, or any cognitive impairment. Stages 1-3 of dementia progression are generally known as "pre-dementia" stages.

Stage 2: Age Associated Memory Impairment

This stage features occasional lapses of memory most frequently seen in:
Forgetting where one has placed an object
Forgetting names that were once very familiar
Oftentimes, this mild decline in memory is merely normal age-related cognitive decline, but it can also be one of the earliest signs of degenerative dementia.

Stage 3: Mild Cognitive Impairment

Clear cognitive problems begin to manifest in stage 3. A few signs of stage 3 dementia include:

International Journal of Pharmaceutical Research and Applications
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Getting lost easily
Noticeably poor performance at work
Forgetting the names of family members and close friends
Difficulty retaining information read in a book or passage
Losing or misplacing important objects
Difficulty concentrating
Patients often start to experience mild to moderate anxiety as these symptoms increasingly interfere with day to day life. Patients who may be in this stage of dementia are encouraged to have a clinical interview with a clinician for proper diagnosis.

Stage 4: Mild Dementia
At this stage, individuals may start to become socially withdrawn and show changes in personality and mood. Denial of symptoms as a defense mechanism is commonly seen in stage 4. Behaviors to look for include:
Decreased knowledge of current and/or recent events
Difficulty remembering things about one's personal history
Decreased ability to handle finances, arrange travel plans, etc.
Disorientation
Difficulty recognizing faces and people
In stage 4 dementia, individuals have no trouble recognizing familiar faces or traveling to familiar locations. However, patients in this stage will often avoid challenging situations in order to hide symptoms or prevent stress or anxiety.

Stage 5: Moderate Dementia
Patients in stage 5 need some assistance in order to carry out their daily lives. The main sign for stage 5 dementia is the inability to remember major details such as the name of a close family member or a home address. Patients may become disoriented about the timeand place, have trouble making decisions, and forget basic information about themselves, such as a telephone number or address.

While moderate dementia can interfere with basic functioning, patients at this stage do not need assistance with basic functions such as using the bathroom or eating. Patients also still have the ability to remember their own names and generally the names of spouses and children.

Stage 6: Moderately Severe Dementia
When the patient begins to forget the names of their children, spouse, or primary caregivers, they are most likely entering stage 6 of dementia and will need full time care. In the sixth stage, patients are generally unaware of their surroundings, cannot recall recent events, and have skewed memories of their personal past. Caregivers and loved ones should watch for:

Delusional behavior
Obsessive behavior and symptoms
Anxiety, aggression, and agitation
Loss of willpower
Patients may begin to wander, have difficulty sleeping, and in some cases will experience hallucinations.

Stage 7: Severe Dementia
Along with the loss of motor skills, patients will progressively lose the ability to speak during the course of stage 7 dementia. In the final stage, the brain seems to lose its connection with the body. Severe dementia frequently entails the loss of all verbal and speech abilities. Loved ones and caregivers will need to help the individual with walking, eating, and using the bathroom.

By identifying the earliest stages of dementia as they occur, you may be able to seek medical treatment quickly and delay the onset of later stages. Though most cases of dementia are progressive, some may be reversible, and sometimes dementia-like conditions may be caused by treatable underlying deficiencies or illnesses. The more reactand seek help, either for yourself or for a loved one.

How common is dementia:
Currently more than 55 million people live with dementia worldwide.There are nearly 10 million new cases every year. In England, 800,000 people currently live with dementia older the age,the more common it is.Around 1 in 14 people aged 65 or more has a form of dementia.

Treatment of dementia:
There are currently three ChEIs regularly used in the UK that are licensed for the treatment of mild to moderate AD:donepezil, galantamine, and rivastigmine.. Donepezil (Aricept) comes in two doses (5 and 10 mg) and until recently had the unique advantage of once a day prescribing. The dose for rivastigmine (Exelon) ranges from 1.5–12 mg a day and has a buturylcholinesterase action in addition to acetylcholinesterase. Galantamine (Reminyl) has a nicotinic receptor allosteric
action and has just become available in an extended release daily capsule.

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NICE guidance for the treatment of Alzheimer'sdisease

Alzheimer's disease must be diagnosed in a specialist clinic where assessments should include cognitive and behavioural functioning, activities of daily living, and likelihood of compliance with treatment.

Treatment should be initiated by specialists but may be continued by general practitioners.

The carer's views should be sought before and during treatment.

Treatment should only be commenced in patients with mild or moderate Alzheimer's disease whose MMSE score is above 12 points.

The patient should be assessed 2–4 months after a maintenance dose is established. Treatment should only continue if the MMSE has improved or not changed and if behavioural or functional assessment shows improvement.

The patient should be assessed every 6 months. Treatment should usually only continue if MMSE score remains above 12 and it is considered that treatment is having a worthwhile effect on functioning and behaviour. MMSE= mini mental state examination.

Initiating treatment with cholines teraseinhibitors

Establish diagnosis of Alzheimer's disease using standardised criteria (for example, ICD-10) through interviewing the patient, taking a collateral history, and undertaking a cognitive assessment. The patient should have a full dementia blood screen and neuroimaging, when appropriate, to identify co-morbidities and reversible causes of cognitive impairment.

Explain and discuss diagnosis, prognosis, and treatment options with patient and carer.

Agree a treatment plan with involvement from a multidisciplinary team. Suggest and encourage the patient and carer to contact the local branch of the Alzheimer's Society.

Establish baseline scores for cognitive function, global functioning, neuropsychiatric symptoms, and activities of daily living using standardised rating scales.

Commence cholinesterase inhibitor.

Make sure an ECG has been conducted recently to exclude problems with cardiac conduction.

Monitor the patient's progress regularly using clinical skills and standardised rating scales.

ECG, electrocardiogram; ICD-10, International classification of disease.

Main side effects of cholinesterase inhibitors

Nausea,Vomiting,Diarrhoea,Anorexia, Abdominal pain, Headache, Dizziness, Tremor, Weightloss, Fatigue. The inhibition of AChE may increase parasympathetic tone therefore ChEIs should be used with great caution in patients with Bradycardia, sick sinus syndrome or cardiac conduction disturbances.

Use of memantine in dementia:

The initial dose is 5 mg once a day which should be increased in 5 mg increments at intervals of at least one week until a maximum dose of 10 mg twice a day is reached.

Memantine is generally better tolerated than the ChEIs.

COMBINATION THERAPY

A donepezil–memantine combination performed significantly better than donepezil–placebo on cognition, activities of daily living, and behavioural neuropsychiatric scales in a randomised controlled trial. The combination was well tolerated.

OTHER DRUG TREATMENTS FOR DEMENTIA

ChEIs and memantine are the mainstay of treatment, but other agents have been tried which have generally beenunsuccessful.

Ginkgo biloba—There have been mixed results in the trials, but meta-analysis shows a small but statistically significant benefit on cognition relative to placebo.18 The effect is smaller than the effect found with ChEIs.

Non-steroidal anti-inflammatory drugs (NSAIDs)—Observational studies have found an association between anti-inflammatory drugs and lower risk of AD, but the result of clinical trials are negative.19 Oestrogens—Trials have not been successful and several safety problems have arisen, including increasing risk of venous thrombosis.

Selegiline—A systematic review of selegiline concluded that although there is some evidence of improvement in cognition and activities of daily living in dementia in the short term,the magnitude was not clinically important.20 Vitamin E—Vitamin E may maintain the functional status of people with AD.21 There is no evidence to suggest it can help prevent dementia or improve cognitive function.

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Non-pharmacological approaches forthe treatment of BPSD

Verbal therapies
Behavioural therapy
Cognitive behavioural therapy
Interpersonal therapy
Reality orientation
Reminiscence therapy
Validation therapy
Non-verbal therapies
Aromatherapy
Bright light therapy
Exercise and activities
Multisensory therapy
Music therapy.
Future perspective:

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