Advances in the Treatment of Dementia

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Educational Objectives

- Describe the economic impact of dementia.
- Review the available pharmacological treatments for dementia in memory and behavior disturbance and for disease-modifying treatment.

Introduction

Dementia is a common debilitating disease that is characterized by cognitive impairment, especially as it affects memory, and by behavioral disturbances that interfere with the functional ability of the individual.

Types of irreversible dementia include Alzheimer’s dementia, vascular (multi-infarct) dementia, frontotemporal lobe dementia, Parkinson’s disease–related dementia, and dementia with Lewy bodies (DLBs). Alzheimer’s dementia and vascular dementia represent the majority of dementia cases seen in clinical practice. Together they account for more than 80% of all cases.

Alzheimer’s disease (AD) is the most prevalent type of irreversible dementia in the U.S.,¹ with a prevalence of 1% to 2% of the population at or below age 65 and increasing to 33% to 50% by age 85 years and older. AD accounts for approximately 65% to 80% of all cases of dementia. AD currently affects three to four million people in the U.S., and by the year 2030, it is estimated that approximately 8.5 million Americans will have the disease.²,³ AD is the fourth leading cause of death among adults in the U.S.

The Economic Impact of Alzheimer’s Disease

AD is a very expensive disease, costing more than $100 billion annually in the U.S. The disease severity greatly affects the cost of treatment because it determines the site of care and the total number of health care resources utilized.

As AD progresses from mild to severe, the cost of treatment doubles.⁴ The length of time spent in a long-term care facility has, by far, the greatest impact on the cost of care for AD patients. Estimated nursing home charges for these patients are between $4.3 and $6.4 million ($35,000 to $52,000 per nursing home patient). The median length of stay in nursing homes for residents with AD has been found to be more than 10 times the national average for all diagnoses, including cerebrovascular accidents, hip fractures, and pneumonia. In the U.S., caregiving costs approach $20 billion, just in patients with dementia who are over age 70. With the increase in the severity of dementia, caregiving requirements increase from five hours of care per week in the mild stage to 40 hours a week in the severe stage.

In a 1997 study by Ernst et al.,⁵ the cost savings from treatment were small for patients with mild and severe dementia. For moderately to severely demented home-dwelling patients with AD, the prevention of a two-point decline in Mini-Mental State Examination (MMSE) scores would save about $3,700 annually, and a two-point increase in MMSE scores would save about $7,100 annually. From an economic perspective, an important goal of Alzheimer’s therapy is to enhance cognition and to improve functional abilities, thus potentially increasing the time it takes for a patient to present with more severe symptoms and postponing the need for institutionalization.

Treatment of Dementia

Although no cure exists for AD, improving or enhancing cognitive function is a primary treatment goal because of the enormous economic, clinical, and social ramifications. For the patient to derive the most benefit in cognitive and functional abilities, it is important to start the treatment of AD early in the course of the disease before neuronal loss occurs.⁶

Mild cognitive impairment (MCI) was described by R. C. Petersen as memory complaints, normal activities of daily living, normal general cognitive functioning, abnormal memory for one’s age, but no dementia.⁷ Mild cognitive impairment refers to a clinical condition between normal aging and AD in which people experience memory loss to a greater extent than one would expect for their age; yet they do not meet currently accepted criteria for clinically probable AD. When these individuals are observed longitudinally, they progress to clinically probable AD at a considerably accelerated rate compared with healthy age-matched individuals. Consequently, the condition has been recognized as suitable for possible therapeutic intervention, and several multicenter international treatment trials are under way.⁸
Because progression to AD occurs more frequently in patients with mild cognitive impairment than in normal elderly populations, the new diagnostic challenge is to identify which patients with mild cognitive impairment will eventually develop AD. It is possible that accurate and early diagnosis of AD might minimize the use of costly medical resources and give patients and caregivers more time to plan for medical, financial, legal, and personal needs.

Imaging studies such as computed tomography (CT), magnetic resonance imaging (MRI), single photon emission CT (SPECT), and positron emission tomography (PET) are used to measure brain volume, especially the size of the hippocampus. It is thought that these modalities might be a useful tool to predict which patients with mild cognitive impairment will progress to AD; in patients with mild cognitive impairment, the hippocampus is smaller, compared with that in healthy people of similar age, and patients with a diagnosis of mild cognitive impairment are more likely to progress to AD compared with those with a larger hippocampus.1,10

Additional studies have focused on identifying AD in a preclinical state before the condition can be confirmed with the use of consensus diagnostic criteria. This approach has several potential benefits. When early detection assessment findings are negative, people with mild memory complaints can be reassured that their forgetfulness reflects a normal age-related change and probably will not progress.

Pharmacological Therapy

Pharmacotherapy for dementia has three major goals:

- treatment of memory disturbance
- treatment of behavioral disturbance
- disease-modifying treatment

Treatment of Memory Disturbance

Cholinesterase Inhibitors

It is well established that the brains of people with AD exhibit a loss of cholinergic function. This loss is caused by a reduction in the amount of acetylcholine and by a decrease in the activity of choline acetyltransferase, an enzyme involved in the production of acetylcholine. There are two types of cholinesterases: acetylcholine esterase and butyrylcholine esterase; the latter appears to become more prominent as the disease progresses.

The most successful pharmacological approach has been to inhibit cholinesterase, thereby raising the levels of acetylcholine and, potentially, facilitating cholinergic transmission. Cholinesterase inhibitors have been demonstrated to enhance cognitive function in patients with mild to moderate AD.1,11 These are the only drugs that have been shown to delay disease progression.12 More recently, preclinical evidence has emerged suggesting that acetylcholine may play a role in cerebrovascular perfusion and that cholinergic stimulation might protect against neuronal ischemic damage.13

Because antidementia treatments are more likely to delay the dementia process than to reverse it or to prevent death, patients with mild memory loss who are at risk for progression of AD are ideal candidates for antidementia interventions. Although cholinergic treatment has been shown to result in symptomatic rather than disease-altering effects, it would certainly be of interest to initiate treatment very early when searching for a disease-modifying effect.

Tacrine (Cognex, Parke-Davis), an acetylcholine esterase inhibitor, was approved for the treatment of AD in 1986. In practice, its hepatotoxicity, up to 30%, and its four-times-daily dosing schedule severely limited its acceptance by physicians, patients, and caregivers, and it is rarely used.14

Donepezil (Aricept®, Eisai America), an acetylcholine esterase inhibitor, was approved for the treatment of AD in 1996. Its plasma half-life is 70 hours, which allows for once-daily dosing. Donepezil is a very selective acetylcholine esterase inhibitor, and this property appears to be associated with fewer adverse side effects, which are relatively limited and are primarily cholinergic, such as nausea, vomiting, diarrhea, and muscle cramps. Side effects are minimized when the drug is titrated from an initial dose of 5 mg daily to 10 mg daily over one month or longer. Vivid dreams and sleep problems are seen in some patients with long-term use.

In a 24-week study, the donepezil group showed significant benefits in cognition, as measured by the AD Assessment Scale–Cognitive Subscale (ADAS-Cog),15 and in behavior and psychiatric function, as measured by the Neuropsychiatric Inventory (NPI). Several trials have provided evidence to support the use of donepezil over extended periods and in more severely affected patients and have shown that using the high dose of 10 mg is more efficacious. These results suggest that donepezil continues to have beneficial effects in patients who have progressed to more severe stages of AD.

Rivastigmine (Exelon®, Novartis), an inhibitor of both acetylcholine esterase and butyrylcholine esterase, was approved by the FDA in April 2000 for the treatment of cognitive deficits in patients with mild to moderate AD. This drug has been found to confer significant beneficial effects on cognition.

In a double-blind, placebo-controlled study, patients in the higher-dose rivastigmine group (up to 12 mg/day) experienced the largest magnitude of treatment effect on ADAS-Cog scores to date.16 With its rapid, forced titration with weekly dosage increases to the maximum tolerated dose, however, rivastigmine caused cholinergic side effects of nausea, vomiting, diarrhea, and muscle cramps. These effects generally abated with continued dosing of the drug. A slower rate of titration every 2 to 4 weeks can minimize side effects. Other occasional side effects of rivastigmine that might be clinically significant in underweight or frail patients are anorexia and weight loss.

Rivastigmine has been beneficial in the treatment of dementia with Lewy bodies. A multicenter, double-blind, placebo-controlled study demonstrated significant beneficial effects on cognition, and almost twice as many patients taking rivastigmine, compared with those taking placebo, showed at least a 30% improvement from baseline values.17 More recently, rivastigmine...
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has also demonstrated clinical benefit in patients with vascular dementia. Because this drug is an inhibitor of both acetylcholine esterase and butyrylcholine esterase, it might provide advantages in clinical effects or in delaying disease progression in patients with severe AD.

**Galantamine** (Reminyl®, Janssen Research), an acetylcholine esterase inhibitor, also acts as a modulator of nicotinic receptors in the brain. The second property may provide advantages in delaying symptoms or disease progression. In 2001, the FDA approved the use of galantamine for the symptomatic treatment of patients with mild to moderate AD.

Studies of this compound have shown substantial dose-related cholinergic side effects that can be avoided by starting with a low dose. A study published in 2001 demonstrated significant efficacy with doses of 16 mg/day and 24 mg/day on both cognition and behavioral symptoms. Occasional adverse effects were anorexia and weight loss. Galantamine also showed significant benefits in cognition, activities of daily living, behavior, and global functioning in patients with probable vascular dementia or AD combined with cerebrovascular disease. Acting as a nicotinic receptor enhancer, galantamine might theoretically facilitate acetylcholine binding to receptors, causing cholinergic neurons to release more acetylcholine.

In addition to improving cognitive abilities, cholinesterase inhibitors appear to relieve behavioral symptoms and to improve daily functioning, and are effective across a broad range of disease stages than had been originally apparent from early pivotal trials. They also appear to be effective in patients with dementia with Lewy bodies and vascular dementia. Whether one of these drugs is significantly superior to another can be determined only by head-to-head clinical trials.

Evidence continues to accumulate that the beneficial effects of acetylcholine esterase inhibitors persist beyond the first 1 or 2 years of therapy, as supported by the extension study done by Doody et al. and also by Raskin et al. Therefore, it is recommended that therapy with cholinesterase inhibitors be continued until unacceptable side effects occur or until there is clear evidence of dementia progression.

Many tests are used to measure the progression of AD, including the ADAS-Cog score, quality-of-life (QOL) assessment, and the dementia rating scale (DRS). These cognitive tests are often used in clinical trials. However, patients and caregivers place a higher value on clinical improvements in mood, behavior, and activities of daily living. Rosenthal suggested using the patient’s Folstein Mini-Mental State Examination as a guide and continuing the medication until the patient scores lower than 12 points out of a possible 30 on this mental test. The final decision to stop or continue treatment with cholinesterase inhibitors depends on the risk–benefit evaluation and is judged on a case-by-case basis.

In general, cholinesterase inhibitors have not been associated with clinically significant drug–drug interactions. No liver toxicity has been reported for donepezil, rivastigmine, or galantamine.

**Glutamate Antagonists**

Recent studies suggest that the excitatory neurotransmitter glutamate may play a role in the pathogenesis of AD. Dysfunction of this neurotransmitter can cause a prolonged excitatory effect, resulting in degeneration and death of cortical neurons. There are three major varieties of glutamate receptors: N-methyl-D-aspartate (NMDA), α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA), and kainate. The NMDA glutamate receptor appears to mediate excitotoxic neuronal death.

**Memantine**, a moderate-affinity NMDA antagonist that appears to block neuronal toxicity associated with prolonged glutamate release, is approved for treating symptoms of dementia in Germany. In a large double-blind, multicenter trial conducted in Europe, memantine delayed the cognitive and functional deterioration in patients with moderate to severe end-stage AD or vascular dementia. A similar large trial in the U.S. conducted only in patients with moderate to severe AD showed similar effectiveness. More studies are needed to determine whether glutamate antagonists are clinically beneficial cognition enhancers alone or in combination with acetyl cholinesterase inhibitors.

**Interventions for Neurobehavioral Aspects of Dementia**

Most dementias are associated with a range of neurobehavioral disturbances, affecting as many as 80% to 90% of demented patients at some point over the course of their illness. Behavioral symptoms, including apathy, agitation, depression, emotional instability, irritability, delusions (paranoia), hallucinations, verbal outbursts, sleep–wake cycle disturbances, and eating problems, may be the eventual determinant of residential placement.

A nonpharmacological approach is often the most appropriate intervention and includes behavioral management and environmental modifications. Behavior management techniques have produced improvement in alleviating agitation and depressive behaviors, which suggests that a nonpharmacological intervention (Table 1) should be routinely considered.

Pharmacological intervention is often necessary to treat neurobehavioral symptoms associated with dementia that are of significant intensity and severity, such as aggressive and dangerous behaviors, delusions, and hallucinations. Treatment options for these symptoms have improved; they currently range from atypical antipsychotic agents, buspirone, and trazodone to the selective serotonin reuptake inhibitors (SSRIs) and the mood stabilizers. Neither the SSRIs nor the mood stabilizers have received FDA approval for the treatment of neurobehavioral symptoms of AD.

Conventional antipsychotic agents (haloperidol, thioridazine) have shown modest efficacy but have been associated with severe extrapyramidal signs, including parkinsonism and tardive dyskinesia. Atypical antipsychotics (risperidone, olanzapine, quetiapine, clozapine) represent a distinct class of agents with the potential for improving behavioral dysfunction associated with AD. In contrast to conventional antipsychotic drugs, the atypical antipsychotics bind more specifically to dopamine receptors and exhibit greater activity in other neurotransmitter systems, especially with serotonin.
Risperidone (Risperdal®, Johnson & Johnson) appears to be the most widely prescribed agent for the treatment of dementia-related psychosis, probably because it is the most studied drug in the treatment of dementia-related behavioral problems. Compared with haloperidol, risperidone is more efficacious in treating dementia. The presence and severity of extrapyramidal signs for patients given risperidone were equivalent to those in patients receiving placebo and less than those noted in patients receiving haloperidol.34

The recommendation for dosing is to start with 0.25 to 0.5 mg at bedtime, with a target dose range of 0.75 to 1.25 mg at bedtime. Side effects with risperidone can be significantly reduced by using a 1-mg daily dose.34–37 Risperidone has also been effective in patients with AD, vascular dementia, and mixed dementia.36

A six-week, randomized, double-blind, placebo-controlled study of 206 patients (61% women and with a mean age of 85.8 years) showed that low-dose olanzapine (5 and 10 mg/day) was significantly superior to placebo and well tolerated in treating agitation, aggression, and psychosis in patients with AD; however, olanzapine (Zyprexa®, Eli Lilly) at 15 mg/day showed no significant efficacy. Somnolence was significantly more common among patients receiving olanzapine, and gait disturbance occurred in those receiving 5 or 15 mg/day. The incidence of extrapyramidal signs and central anticholinergic effects at any olanzapine dose was similar in patients given placebo.38

A 10-week, randomized, double-blind, placebo-controlled study of long-term care residents with AD and psychosis compared the effects of quetiapine (Seroquel®, AstraZeneca) with those of haloperidol (Haldol®, Ortho-McNeil). Results showed no improvement of psychosis with either antipsychotic agent, although both drugs relieved agitation significantly. The adverse effects of quetiapine were similar to those of placebo, and patients given quetiapine at doses up to 100 mg/day experienced less somnolence compared with patients given haloperidol.39

Clozapine (Clozaril®, Novartis) may be the least likely choice for patients with AD, given its anticholinergic side effects and its tendency to produce agranulocytosis. Clozapine may be beneficial in treating psychosis associated with Parkinson’s disease or dementia with Lewy bodies.40 The recommended initial dose is 6.25 to 12.5 mg at bedtime.

Ziprasidone (Geodon®, Pfizer) is the newest atypical antipsychotic agent approved for the treatment of schizophrenia. No studies are available regarding its use in elderly patients with AD; use of the drug could be limited by a prolonged QT interval and the increased risk of ventricular arrhythmias and sudden death.

Antidepressants

Neurobehavioral features of depression, apathy, irritability, and emotional instability frequently coexist with AD. Agitation in a patient with dementia sometimes presents as “masked depression,” in which case the patient may have only vague physical symptoms, such as weakness, fatigue, headache, and complaints of pain.

SSRIs are now considered to be first-line medications for treatment of depressive symptoms in AD. Several SSRIs, namely fluoxetine, sertraline, and fluvoxamine, have demonstrated efficacy for reducing agitated behavior in patients with dementia, but they are not approved for this treatment.

Citalopram (Celexa®, Forest) is effective in the treatment of depression, anxiety, irritability, restlessness, and destructive vocalization that occurs with AD.41,42 It appears to produce fewer drug–drug interactions than the other SSRIs,43 a favorable feature for elderly patients likely to be taking multiple medications. Fluoxetine, fluvoxamine, and paroxetine are more likely to be involved in significant drug–drug interactions compared with citalopram or sertraline.43

Trazodone (Desryl®, Lemmon) was compared with haloperidol in treating agitation in patients with dementia. Both treatment groups demonstrated equal reduction in agitation, repetition, verbal aggression, and oppositional behaviors.44 Trazodone had a better safety profile than haloperidol. Because of its sedating quality, trazodone may also be effective in relieving agitation in patients with dementia.44

Anticonvulsants

Anticonvulsants are being used with increasing frequency in older people with agitation, aggression, and behavioral disturbances. Valproate (Depacon®, Abbott) can be effective and well tolerated for the treatment of aggressive behavioral disturbances associated with AD.45 Dosing is usually initiated at 125 mg/day and titrated in 125-mg increments in the usual range of 500 to 1,500 mg/day given at bedtime or two times per day. The role of monitoring drug levels is unclear in the elderly. The drug is titrated to clinical effect. Monitoring of liver function tests and platelets is required because hepatotoxicity and thrombocytopenia are major side effects of valproate. Gastrointestinal intolerance and excessive sedation may limit its utility, especially at higher doses. Divalproex (Depakote®, Abbott) may have a better side-effect profile than valproic acid.46

Although carbamazepine (Tegretol®, Novartis) has demonstrated significant improvement in terms of decreased agitation and aggression, compared to placebo in patients with advanced dementia, its use is limited owing to its risk for drug–drug interactions.47

### Table 1 Nonpharmacological Interventions in Dementia

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Antianxiety Agents

Anxiety is commonly associated with dementia, and some reports suggest that anxiolytics may be helpful for reducing agitation, anxiety, and impaired sleep. Treatment with benzodiazepines may be effective in the short term. For short-term use, a benzodiazepine with a short half-life is recommended (e.g., lorazepam [Ativan®, Wyeth-Ayerst], oxazepam [Serax®, Wyeth-Ayerst], alprazolam [Xanax®, Pharmacia & Upjohn]), especially for elderly patients, in whom doses are more likely to accumulate.

The long-term usage of anxiolytics is limited by a gradual decline in efficacy and by the potential side effects of sedation; decreased cognitive function, including learning and memory; loss of coordination; unsteady gait, which may lead to falls; and paradoxical agitation. The nonbenzodiazepine anxiolytic buspirone of coordination; unsteady gait, which may lead to falls; and parado-

cial agitation. The nonbenzodiazepine anxiolytic buspirone (BuSpar®, Bristol-Myers Squibb), which produces considerably fewer side effects compared with benzodiazepines—mainly headaches and dizziness—may be effective for some patients with restless and anxiety, although its delayed onset of action (two to three weeks) may limit its use.

Disease-Modifying Treatments

Anti-inflammatory Approaches

Changes in the level of acute-phase reactants, complement proteins, and inflammation-mediating cytokines (interleukin-1, interleukin-6) have all been associated with AD.51

Case-control studies of patients taking nonsteroidal anti-inflammatory drugs (NSAIDs) regularly for arthritis demonstrated a reduced risk for the development of AD.52 Several large epidemiologic studies have suggested that the use of NSAIDs might decrease the risk of AD in healthy elderly people by 30% to 70%. The recent introduction of selective cyclooxygenase-2 (COX-2) inhibitors, such as celecoxib (Celebrex®, G. D. Searle) and rofecoxib (Vioxx™, Merck & Co.), which may cause fewer gastrointestinal side effects, has led to the initiation of therapeutic trials in AD. In early trials, patients with AD did not demonstrate any significant benefits from these drugs.53 The rofe-
toxic properties and that inhibit the formation of free radicals might prevent or retard the pathophysiological processes of AD.60 In one study, vitamin E and selegiline were efficacious in delaying nursing-home placement, functional decline, and death by 25%; however, no prevention of cognitive decline was reported.61 The effect of vitamin E in mild AD has not been studied. A government advisory group has recently recommended that vitamin E be given in daily dosages between 60 and 1,000 IU but suggested that the higher dosages might increase the risk of coagulation disorders.

It has been purported that the herbal preparation Gingko biloba may have antioxidant properties. In one trial of the herbal extract, a very modest improvement in cognitive function was reported.62 The use of Ginkgo is not recommended because of its limited efficacy and the variability in the dosing and contents of herbal extracts.63 Ginkgo may also increase the risk of bleeding in patients taking vitamin E and/or warfarin. Other antioxidants such as vitamin C and alpha-lipoic acid have not been well-studied.

Interventions for Families and Caregivers

Individuals charged with the caring for patients afflicted with AD exhibit greater rates of physical and psychiatric morbidity and report higher levels of psychosocial distress than do noncaregivers. Furthermore, there appears to be a relationship between the level of the caregivers’ physical and psychological distress and the decision to institutionalize patients with AD.

It is generally felt that most families and caregivers would benefit from basic intervention or receiving education regarding AD as well as from participation in support groups. At times, specific symptoms may require intervention consisting of psychotherapy or even pharmacotherapy. Sessions of family therapy have proved beneficial.
Prevention of Dementia

The prevention of AD involves many modalities, including lifestyle modification, such as pursuing a higher educational level and staying mentally active in the later stages of life. Modifying vascular risk factors also plays a major role in preventing dementia of AD, vascular dementia, and AD with vascular dementia. Such modifications include controlling blood pressure, eating a low-cholesterol diet, consuming alcohol in moderation, and taking vitamin B₆, vitamin B₁₂, folate, and vitamin E. Epidemiologic data suggest that NSAIDs, estrogen replacement therapy, and perhaps the use of statins might play a role in preventing dementia. A recent study showed that treatment with statins decreased AD risk by 79%.64

Promising New Therapies for the Future

Because elevated levels of amyloid beta peptide (Abeta) 1-42 (amyloid β-42) are evident in the brain tissues of patients with early dementia and are strongly correlated with cognitive decline, the β-amyloid vaccine was developed with the intention of decreasing deposits of amyloid by stimulating its immune clearance. The β-amyloid vaccine study was discontinued because of cases of encephalitis reported with the use of the vaccine.

Inhibition of beta or gamma secretase may be a promising treatment for AD. Inhibitors of the gamma-secretase and neuronal growth factors (leteprinim potassium) are being developed. Other promising new therapeutic modalities include inhibition of both cholinesterase and butyrylcholinesterase, and blockade of the glutamate receptor. Large multicenter trials of glutamate AMPA receptor antagonists are likely to begin in 2002. These therapies may dramatically improve AD management but remain years away from general clinical use.

Conclusion

The cholinesterase inhibitors, the only FDA-approved agents for the treatment of AD, can provide modest efficacy for cognitive, behavioral, and functional stability. Other medications, such as atypical antipsychotic agents, SSRIs, anxiolytics, and mood stabilizers, are being used off-label for excess neurobehavioral features. The appropriateness of nonpharmacological psychosocial interventions for patients or caregivers should be routinely assessed and implemented as needed.

The list of potential and approved treatment options has grown over the past five years. With the addition of newer acetylcholine esterase inhibitors, more patients showed benefit from early recognition of dementia and earlier treatment. Patients with other forms of dementia other than AD may benefit from acetylcholine esterase inhibitors.

Treatment with cholinesterase inhibitors is currently limited to improving and transiently maintaining cognitive abilities in patients with mild to moderate AD and, at best, may delay the progression of the disease. Consequently, examining ways to prevent dementia, in addition to developing new drugs, has become very important, with the potential of affecting the course of the disease process or perhaps even providing a cure.

References


