Alzheimer’s disease is the most common cause of dementia and increases in prevalence exponentially with age, with trends in the United States likely to worsen in ensuing decades. The pathology in Alzheimer’s disease is characterized by an increase in extracellular amyloid plaques and intraneural neurofibrillary tangles, with neuronal destruction in several areas of the brain, and biochemically by a deficiency in acetylcholine; clinical manifestations include progressive loss of memory, change in personality, and behavioral disturbances. Pharmacotherapy includes the use of cholinesterase inhibitors and memantine; addressing the many behavioral manifestations of the disease, especially in advanced stages, imposes tremendous burden to caregivers and healthcare resources. [AHDB: 2009;2(1):39-47.]

Alzheimer’s Disease: A Healthcare Burden of Epidemic Proportion

T.S. Dharmarajan, MD, FACP, AGSF; Srinivas G. Gunturu, MD

Alzheimer’s disease is the most common cause of dementia and increases in prevalence exponentially with age, with trends in the United States likely to worsen in ensuing decades. The pathology in Alzheimer’s disease is characterized by an increase in extracellular amyloid plaques and intraneural neurofibrillary tangles, with neuronal destruction in several areas of the brain, and biochemically by a deficiency in acetylcholine; clinical manifestations include progressive loss of memory, change in personality, and behavioral disturbances. Pharmacotherapy includes the use of cholinesterase inhibitors and memantine; addressing the many behavioral manifestations of the disease, especially in advanced stages, imposes tremendous burden to caregivers and healthcare resources. [AHDB: 2009;2(1):39-47.]

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Neuropathology

The core of AD pathology involves formation of amyloid neuritic plaques and neurofibrillary tangles (NFTs). Beta-amyloid (Aβ) 42 is derived from proteolysis of amyloid precursor protein, which undergoes polymerization to form abnormal, insoluble, sticky amyloid plaques, resulting in inflammation and loss of neurons in areas of the brain involving memory and cognition (Figure 2). Hyperphosphorylation of tau protein leads to formation of insoluble intraneuronal NFTs and to neuronal destruction. Senile plaques and NFTs are markers of AD. In patients with AD, senile plaques are seen throughout the neocortex, whereas NFTs are seen in the amygdala, hippocampus, thalamus, association areas, and the cortical region. Although amyloid plaques and NFTs occur with normal aging, it is the distribution, density, and neuronal loss in the association areas and the cerebral cortex that characterize Alzheimer’s dementia. Because these plaques and tangles are insoluble, future therapy should focus on prevention strategies.

Risk Factors

Of the different risk factors that have been linked to AD and dementia, aging is the most important factor. Data are inconsistent for most of the other risk factors, which include genetic factors, white race, female sex, low education level, poor physical status, a history of head trauma, depression or postoperative delirium, family history of dementia, elevated C-reactive protein, and lower household income.

Predisposition to dementia in general is linked to low thyroid-stimulating hormone, hypertension, folic acid or vitamin B12 deficiency, elevated homocysteine levels, hyperlipidemia, smoking, diabetes mellitus and metabolic syndrome, and cerebrovascular disease.

Although no single risk factor is known to cause Alzheimer’s dementia, it is tempting to speculate an interplay between aging and genetics, modified by environmental factors. The link between AD and genes is evident in early- and late-onset AD, but no single genetic etiology is known to cause AD. The neuropathology of AD mimics Down syndrome; patients with Down syndrome who live to age 40 manifest neuropathology identical to Alzheimer’s dementia. In early-onset familial AD, genes PS1, APP, and PS2 are linked to chromosomes 1, 14, and 21; late-onset AD is linked to the APOE gene and to chromosome 19. The €4 allele of the APOE gene is implicated in increased risk of late-onset AD, whereas €2 allele appears protective, and €3 allele is neutral.

Figure 1 Projected Annual Incidence of Alzheimer’s Disease in the United States

Initiating early interventions in community-living patients with AD often is associated with lower overall costs compared with costs for institutional placement.10 Table 1 outlines the economic impact of AD and dementia on the healthcare system.11
Clinical Features of Dementia in AD

The onset of dementia in AD is insidious, with a progressive decline in functioning level. Possibly the earliest stage is mild cognitive impairment (MCI), which is evident without impairment in social or occupational functioning. Cognitive impairment without dementia is more prevalent than dementia; the Aging, Demographics and Memory Study found that 22% of adults older than 71 years in the United States had cognitive impairment without dementia; 8% died and 11.7% advanced to dementia annually. The conversion rate is 1% to 2% annually. MCI is common and is considered a precursor to dementia, but not necessarily Alzheimer’s dementia. A diagnosis at this early stage helps delay onset of dementia and nursing home placement.

The 3-stage classification categorizes Alzheimer’s dementia into mild, moderate, and severe stages. A diagnosis is often missed by the first physician consulted, and “normal aging” is a common misdiagnosis.

In mild dementia, patients have memory loss for recent events and are unable to learn new facts; well-educated patients may mask their difficulties. Subtle personality changes are evident. Behavioral and motor changes are not evident, and patients are independent for activities of daily living (ADLs).

In moderate dementia, worsening of recent, remote, and recall memory is more readily apparent, along with significant cognitive impairment and personality changes. Behavioral changes include agitation, aggressive behavior, anxiety and depression, with partial dependence for ADLs.

With severe or advanced Alzheimer’s dementia, memory, language, and cognition are markedly impaired, requiring complete caregiver dependence. The patient is incontinent, unable to feed or swallow, resulting in poor nutrition, and speech deteriorates to a few words and can include echolalia and palilalia. Patients are lost in familiar environments; wandering, falls, and accidents are common. At this stage, nursing home placement and means for nutrition are at stake.

Cognitive Assessment

The Mini-Mental State Examination (MMSE) is a common screening instrument used to evaluate cognition, and is administered in less than 10 minutes. A score higher than 24 does not rule out dementia nor does a score below 24 confirm dementia; age and educational status influence the score. MMSE scores deteriorate at a rate of about 4 points annually in Alzheimer’s dementia.

Table 1 Economic Impact of Alzheimer’s Disease, 2000

<table>
<thead>
<tr>
<th>Economic costs</th>
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</thead>
<tbody>
<tr>
<td>- Dementia increased the cost of care 3-fold compared with care for patients without dementia (about $13,000 vs $4500 per person)</td>
</tr>
<tr>
<td>- Patients with dementia stayed in the hospital 3.4 times longer than cognitively intact patients</td>
</tr>
<tr>
<td>- Hospital stays for dementia cost 3.2 times more compared with patients without dementia</td>
</tr>
<tr>
<td>- When additional comorbidity occurred (coronary heart disease, heart failure, diabetes, chronic obstructive lung disease), costs for patients with dementia doubled or more compared with patients without dementia</td>
</tr>
<tr>
<td>- Medicare home care costs were 3.8 times higher than patients without dementia</td>
</tr>
<tr>
<td>- More physician visits were required for patients with dementia than for other Medicare beneficiaries</td>
</tr>
<tr>
<td>- Costs are high for care of Alzheimer’s dementia patients in all settings: at home, in daycare, in an assisted-living facility, and in a nursing home</td>
</tr>
</tbody>
</table>

Health plan policy recommendations from the Alzheimer’s Association

- Include affordable prescription drug benefits
- Include medication management, education of family or caregiver, multidisciplinary care conferences, and help access community resources
- Provide benefits to include home visits by nursing or other professionals
- Targeted coordinated care to benefit those with dementia

The Clock Drawing Test (CDT) is easily administered. The Mini-Cog test is a composite of the CDT combined with a “3-item recall”; its strengths are brevity and ease of administration. The Mini-Cog test takes no more than 5 minutes and appears more valid in better-educated individuals. The Functional Assessment Staging and Global Deterioration Scale help evaluate functional deterioration and dependency for assistance.

Evaluation

Most important in the evaluation of dementia in AD is the initial focused history (and physical examination), with the history obtained separately from the patient and from a family member or caregiver who truly knows the patient. A diagnosis of Alzheimer’s dementia can be made
Drug-Induced Memory Loss

Several medications can lead to memory impairment; a detailed review of over-the-counter drugs and supplements is indicated, especially products with anticholinergic activity that are known to worsen cognition. Decline in cognition results from the drugs blocking muscarinic receptors; the resulting worsening of working memory, speed of processing, and praxis predict overall performance and cognitive status, as well as its impairment.¹⁵

A recent study of 107 medications revealed moderate-to-severe anticholinergic activity with many frequently used medications (oxybutinin, tolterodine, diphenhydramine, amitriptyline, thioridazine, among others) warranting caution with their use in dementia.¹⁵ These drug-induced anticholinergic effects can cause cognitive deficits that resemble dementia and have the potential to worsen memory in those with MCI.

Diagnosis

A patient with dementia should be offered neuroimaging at least once, to exclude treatable causes; a computed tomography scan or magnetic resonance imaging will demonstrate common structural alterations in the brain. No specific pattern in imaging is diagnostic of Alzheimer’s dementia. Brain atrophy in neuroimaging is common in older adults; although seen in AD, atrophy by itself is not diagnostic of cognitive impairment. Lately, there is increasing awareness of the role of positron emission tomography (PET) imaging in early diagnosis of Alzheimer’s dementia through mea-
measurement of decreased localized glucose metabolism correlating with loss of cognition. PET scans have the potential to help make the diagnosis before advanced clinical manifestations develop, with reasonable sensitivity (84%-93%) and specificity (93%) even in the stage of MCI. The scan also helps distinguish Alzheimer’s dementia from other dementias.

Although no single laboratory test is diagnostic of AD or dementia, routine and some individualized tests can be used to delineate any treatable cause. Genetic testing for AD is not routinely offered at this time.

Finally, the differential diagnosis of Alzheimer’s dementia includes other causes of dementia, including vascular dementia, Lewy body dementia, frontotemporal dementia, dementia of Parkinson’s disease, normal pressure hydrocephalus, and reversible causes, such as hypothyroidism and B12 deficiency, among others. Delirium, a short-term disorder that causes alteration in level of mentation and difficulties in sustaining attention, as well as depression, can mimic or complicate dementia; these are reversible and their treatments differ from that of Alzheimer’s dementia.

Management

Early diagnosis of Alzheimer’s dementia helps plan the future, and initiation of therapy with cholinesterase inhibitors (ChEIs) to maximize benefit in preserving functional status. Besides cognition, many aspects of AD demand attention.

Behavioral manifestations of dementia are foremost in AD, demanding caregiver attention and difficulties in management of wide-ranging moods; in one study, apathy, agitation, aggressive behavior, and depression were most common, whereas hallucinations and delusions, anxiety, and wandering were less common. Sudden alterations in behavior warrant a search for causes that precipitate delirium, such as infection, pain, electrolyte imbalance, or medication-related adverse effects. Approaches to treatment include empathy, tact, ensuring safe environments, and psychotherapy. Therapeutic touch (namely, a meditative, compassionate hand touch to help or heal) can help minimize agitation.

Visual and hearing impairments are common with aging and contribute to even greater handicaps in Alzheimer’s dementia. Because they may be a cause of behavioral changes, glasses and hearing aids should be available at all times.

Several simple measures can be instituted to provide a safe environment in the home, hospital, or institution. Measures for environment safety minimize falls. These include appropriate carpeting (avoid scatter rugs); avoidance of slippery floors (in bathrooms) and clutter; use of cordless phones, handrails, and proper lighting; and so on.

Difficult decisions may lead to frustration and agitation. When patients are agitated, arguments are pointless. Restraints are discouraged, and should be used short-term, weighing the benefit against the burden. Cognitive training may benefit daily functional activities. In aimless wanderers, precipitating factors need to be looked for and rectified.

Sleep disturbances are handled through regular sleep schedules, evening exercises,避免 of bright light and noise, and limiting fluids in the late hours to decrease bathroom visits at night. Regular ongoing physical activity correlates with cognitive improvement.

As the disease progresses, dietary restrictions are minimized, with quality of life (QOL) gaining greater importance. In terminal stages, a common consideration is the insertion of a percutaneous endoscopic gastrostomy (PEG) tube for enteral feeding, a decision fraught with ethical dilemmas. Studies have not confirmed that PEGs prolong life, improve QOL or nutritional status, or lower the likelihood for pressure ulcers and aspiration pneumonia. Nearly half of terminal AD patients who receive PEG tubes die within a year.

Advance directives are ideally implemented early when the patient still has some capacity to make decisions. A durable power of attorney for healthcare (healthcare proxy) can make decisions on behalf of the patient who loses capacity in advanced disease. Hospitals and institutions are required to document whether patients have advance directives and if they do not, offer an opportunity to execute one. In general, we address advance directives inadequately; in our hospital, we have demonstrated that efforts by health providers result in vastly improved responses from patients.

Pharmacotherapy

Goals of pharmacotherapy (Table 2) in AD are to preserve cognition and function to the extent that is possible, slow progression of the disease, and address behavioral disturbances.

Cholinesterase Inhibitors

Several ChEIs (acetylcholinesterase, butyrylcholinesterase) are the mainstay of treatment based on the principle of enhancing neurotransmitter function and
acetylcholine availability at the neurons. No agent prevents neuronal death.

Tacrine (Cognex), the first ChEI developed, is no longer used because of its adverse effects; 3 additional ChEIs are generally similar in effectiveness. Clinicians should choose a pharmacologic agent based on tolerability, adverse effects, cost, and ease of use; the evidence to compare the effectiveness is weak based on review of studies presented in recent guidelines. They differ in pharmacokinetics and side effects. Significant anecdotal and unproved use of combinations also exist. Peak efficacy is attained in 3 months.

In mild-to-moderate forms of AD, a single ChEI is typically used. In moderate-to-severe stages, a noncompetitive N-methyl-D-aspartate (NMDA) receptor antagonist, memantine (Namenda) may be indicated with or without a ChEI to enhance cognition, function, and behavior-related measures. Memantine is an antiglutamatergic agent; glutamate is a neurotransmitter essential for learning and memory through facilitation of NMDA receptors.

For those with dysphagia, liquid formulations are available. Another option is transdermal rivastigmine (Exelon), associated with better gastrointestinal tolerance and potentially improved adherence to regimen.

Withdrawal of a drug may lead to rapid loss of efficacy and clinical deterioration, whereas appropriate use can delay nursing home placement, a stated priority for caregivers, and help manage adverse behaviors.

### Antipsychotics

When behavioral disturbances become worse and nonpharmacologic approaches fail, medications may be tried for that purpose. General principles of psychotrop-

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**Table 2: Pharmacology of Alzheimer’s Disease**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of action</th>
<th>Dose</th>
<th>Indication, additional requirements</th>
<th>Food interactions</th>
<th>Metabolism</th>
<th>Half-life</th>
<th>Elimination</th>
<th>Adverse effects</th>
<th>Approx. cost per month, $</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tacrine</td>
<td>AChE and BuChE* inhibitor</td>
<td>10-40 mg q6h</td>
<td>Follow liver function tests, no longer used</td>
<td>Food decreases absorption</td>
<td>CYP1A2</td>
<td>2-4 h</td>
<td>Hepatic</td>
<td>Withdrawal (up to 55%), maximum side effects, abnormal liver function, nausea, vomiting, diarrhea</td>
<td>310-360</td>
</tr>
<tr>
<td>Donepezil</td>
<td>AChE inhibitor</td>
<td>5-10 mg/d</td>
<td>Mild-to-moderate dementia</td>
<td>None</td>
<td>CYP2D6</td>
<td>70-80 h</td>
<td>Hepatic</td>
<td>Withdrawal (0%-57%), nausea, vomiting, diarrhea</td>
<td>170-190</td>
</tr>
<tr>
<td>Rivastigmine</td>
<td>AChE and BuChE* inhibitor</td>
<td>1.5-6 mg bid</td>
<td>Mild-to-moderate dementia, also available as transdermal patch</td>
<td>Delays</td>
<td>Cholinesterase-mediated hydrolysis</td>
<td>1.5-2 h</td>
<td>Renal</td>
<td>Withdrawal (12%-29%), nausea, vomiting, diarrhea, confusion, insomnia</td>
<td>184-193</td>
</tr>
<tr>
<td>Galantamine</td>
<td>AChE inhibitor and nicotine modulatory receptor effect</td>
<td>4-12 mg bid</td>
<td>Mild-to-moderate dementia</td>
<td>Decreases</td>
<td>CYP2D6, CYP3A4</td>
<td>5-7 h</td>
<td>Renal and hepatic</td>
<td>Withdrawal (8%-54%), nausea, vomiting, diarrhea, anorexia</td>
<td>180-380</td>
</tr>
<tr>
<td>Memantine</td>
<td>NMDA receptor antagonist</td>
<td>5-20 mg/d</td>
<td>Moderate-to-severe dementia</td>
<td>None</td>
<td>Nonhepatic</td>
<td>60-80 h</td>
<td>Renal (active tubular secretion)</td>
<td>Withdrawal (95%-12%), nausea, diarrhea, dizziness, agitation</td>
<td>130-160</td>
</tr>
</tbody>
</table>

AChE indicates acetylcholinesterase; BuChE, butyrylcholinesterase; NMDA, N-methyl-D-aspartate; CYP, cytochrome P 450. *Average cost in the United States (not standardized).

ic therapy that apply to the elderly include a “start low, go slow” approach, understanding the pharmacokinetic changes with aging, and monitoring for side effects.

Drug therapy should be considered short-term (3-6 months), with intent to terminate therapy after resolution of symptoms, as adverse events evolve with time. Antipsychotics include the traditional haloperidol (Haldol) and risperidone (Risperdol) and the atypical antipsychotics olanzapine (Zyprexa) or quetiapine (Seroquel). The antidepressants paroxetine (Paxil), sertraline (Zoloft), and citalopram (Celexa); valproic acid (Depakote); and others are used for agitation or difficult behavior. Keeping the dose low minimizes adverse effects, such as movement disorders. Some atypical drugs have been linked to the development of glucose intolerance and vascular complications with long-term use.

Caregiver Burden

The majority of patients (70%) with AD—with or without dementia—live in the community and are dependent on caregivers, typically a woman (wife or daughter); hence the need to understand the existence of “caregiver burden” requiring support.

Caregivers need education and counseling to understand the course of AD and the means to deal with the family member who has the disease. A pilot study demonstrated that home care agencies can assess specific caregiver needs and help improve caregiver mastery to decrease strain. Caregivers of demented individuals tend to devote precious time for caregiving, often at the expense of their own occupational or leisure activities, and suffer from physical, mental, and emotional stress. Caregivers often suffer from depression, musculoskeletal disorders, and hypertension.

Institutionalization of patients with AD associated with dementia results from 1 or more variables that are patient-related (aggressive behavior, incontinence, feeding difficulties, terminal dementia) or caregiver-related (poor health, financial impact, poor support, depression, impaired QOL).

Medicare Coverage for Care in AD

The majority of patients with AD and dementia are likely to be older than 65 years of age and eligible for Medicare, which includes coverage for:

- Hospital stay at variable rates (which is influenced by illness severity, management strategies, and duration of stay)
- Skilled nursing care only after a hospital stay of 3 days or more (and demonstrating a need for skilled care)
- Home healthcare and “reasonable and necessary” physician visits.

The 2003 Medicare Modernization, Improvement, and Prescription Drug Act (Part D) introduced voluntary prescription drug coverage applicable to Medicare beneficiaries. The Centers for Medicare & Medicaid Services requires health plans to cover essentially all protected drug classes used in AD (eg, antipsychotics and antidepressants), as well as two thirds of the drugs used in AD in nonprotected classes.

Although ChEIs are covered by Medicare to the tune of 67% to 100%, prior authorization is required 12% to 25% of the time. Similar formulary coverage is available for antipsychotics.

Although ChEIs are covered by Medicare to the tune of 67% to 100% (depending on a given drug within the class), prior authorization is required 12% to 25% of the time. Similar formulary coverage is available for antipsychotics. Nursing homes and pharmacies identify a set of preferred drugs in each class, with some choices available.

Medicare provides coverage for PET scans, based on documentation of recent dementia and cognitive decline of at least 6-month duration, and when the patient meets diagnostic criteria for AD and frontotemporal dementia, as ascertained by the provider. Finally, it is apparent that patients with Alzheimer’s dementia manifest more comorbidity than matched controls, and therefore cost more to care for. Annual costs are 34% higher for patients with Alzheimer’s dementia compared with those without dementia; outpatient pharmacy is a major cost factor.

Future Perspectives

Earlier diagnosis of AD may help alter its course. Detection of tau protein and Aβ42 measurements in cerebrospinal fluid have the potential to discriminate early Alzheimer’s dementia from other causes of memory loss. The role for PET scans and genetic testing in the diagnosis may change in the future. Exploring the region-specific decline in cerebral glucose metabolism in AD may be a novel approach in the future.

Data on the role for testosterone, estrogen, vitamins E and C, and beta-carotene are inconsistent with
regard to benefits in Alzheimer’s dementia. And the evidence that gingko biloba has a role in preventing or delaying cognitive decline is similarly not convincing.46

Tarenflurbil (Flurizan), a selective AB42-lowering agent, is an investigational agent with a new mechanism of action and is currently in clinical trials. It promises benefits for ADLs and global functioning in mild AD.47

In preliminary studies, nonsteroidal anti-inflammatory drugs were believed to lower AB production by modulation of gamma-secretase activity or through anti-inflammatory actions. However, they are yet to deliver these expected benefits in randomized controlled trials. Similarly, a potential was suggested for beta- and gamma-secretase inhibitors to decrease formation of insoluble AB or alternatively the upregulation of alpha-secretase to increase soluble amyloid formation; these are promising concepts, but are yet far from reality.

Testosterone supplementation in those with low testosterone levels failed to improve either cognition or function for patients with AD,48 but a recent study indicated that serum bioavailable (not total) testosterone had lower risk for MCI.49

Peroxisome proliferator-activated receptor-gamma agonists, such as rosiglitazone (Avandia), may be novel therapeutic agents for improved memory and cognition.50

Immunotherapy (eg, vaccination) to target AB protein and tau protein has been tried, but active immunization was fraught with development of aseptic meningoencephalitis in humans. Passive immunization trials are now under way.51

Statins show promise in slowing cognitive decline. The mechanisms postulated include cholesterol lowering, lessening vascular damage, and anti-inflammatory and antioxidant effects. Epidemiologic data suggest lower incidence of Alzheimer’s dementia for those using statins.

Anecdotal reports suggest that the Mediterranean diet and fruits and vegetable juices delay the progression of AD. Finally, maintaining appropriate folate levels may prove useful, and acetyl-L-carnitine may also be beneficial.

Conclusion

AD with dementia is characterized by gradual memory loss and cognitive impairment, followed by personality changes and behavioral problems. Although several risk factors are implicated, a precise etiology is not delineated. AD may be the result of interplay between aging, genes, and environment. The result is a severe burden to caregivers and to society, placing a heavy toll on healthcare resources. And as the prediction is for AD to become more prevalent, we need to plan wisely to allocate appropriate resources to meet the demands of the disease, while continuing to channel research funds to delay onset, slow progress, or reverse the disease.

References

characteristics. Presentation at the American College of Physicians Internal Medicine Annual Meeting; May 15-17, 2008; Washington, DC.

Stakeholder Perspective
The Challenge of Value-Based Benefit Design in Alzheimer’s Disease

Payers: A coordinated approach spanning the healthcare and disease progression continuum presents a challenge to payers when designing value-based benefits for the treatment of Alzheimer’s disease (AD). The main goals of AD therapy are delaying the progression of the disease and mitigating the sequelae and complications of advanced disease. Dementia is the most common manifestation of and most economically burdensome in AD, yet it is not necessarily manifested in all cases with the disease. When dementia does manifest in AD, it is predominantly in the later years of a patient’s life. Although the economic burden, both direct and indirect, is up at the top of the national healthcare expenditures, the largest share of this burden is borne by Medicare plans, because of the typically older age-group that is affected by AD.

Some evidence suggests that early intervention may delay or slow the manifestations of the disease, but it is difficult to predict which patients may benefit most from intervention, and which patients may never progress to dementia. In addition, among patients who do progress to AD, it is difficult to determine if the intervention slowed that process, and to what extent.

Finally, payers must decide if they are willing to pay for interventions without any assurances of positive outcomes, especially when their obligation may soon be transferred to a Medicare provider. Until clearly defined screening mechanisms for prediction of outcomes or consensus guidelines are introduced, payers will continue to struggle with the development of a value-based benefit offering for members with AD.

Patients: The process of losing control of the functional aspects of one’s life and personal independence is probably one of the most anxiety-producing events a patient and his or her loved ones can experience. Although all those affected would seek to thwart or at least slow that process, no clear direction from either the medical literature or the payer stakeholders exists to help with such a plan. In fact, the lack of direction may cause the patient in the early stages of AD and his or her family to bear a significant portion of the financial burden for a management plan suggested by the treating physician.

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