Antipsychotic Use in the Elderly: Overview and Evidence-Based Management
Helen C. Kales, MD, Mary Blazek, MD, Susan M. Maixner, MD, and Laura M. Struble, PhD, GNP-BC

Abstract
- **Objective:** To present an overview of the current evidence base for the use of antipsychotics in older adults, particularly those with neuropsychiatric symptoms of dementia.
- **Methods:** Review of the literature.
- **Results:** Antipsychotics are among the most widely prescribed psychotropic drugs for the elderly population; it has been estimated that as many as 23% of older adults will experience psychotic symptoms at some point. The neuropsychiatric symptoms of dementia are one of the most common indications for which atypical antipsychotics are prescribed in the elderly, and include agitation, aggression, mood syndromes, paranoia, delusions, and hallucinations. Besides dementia, late-life syndromes with psychotic symptoms include schizophrenia, mood disorder (depression or mania) with psychosis, and delirium. The U.S. Food and Drug Administration has not approved any medication for the treatment of the neuropsychiatric symptoms of dementia, and in 2005 and 2008 warned that use of both atypical and conventional antipsychotics to treat neuropsychiatric symptoms of dementia was associated with increased mortality. Several subsequent studies highlighted the risk of antipsychotic medications in older adults in general.
- **Conclusion:** Clinicians contemplating the prescription of an antipsychotic for an elderly patient need to consider the risks and benefits of such medications with a knowledge of the current evidence base for their usage in older adults.

CASE STUDY
Initial Presentation
A 75-year-old woman presents to her primary care physician after an episode of getting lost in her car on her way to a doctor’s appointment.

History
During this episode, the patient ended up in another state and was found by a policeman who contacted her husband, who came to pick her up. The husband states that he then realized the significance of his wife's memory problems, although there had been indications of cognitive concerns prior to this, including “forgetfulness” and word-finding problems. Past medical history is significant only for hypertension and hypercholesterolemia.

Physical and Mental Status Examination
Findings on physical examination are unremarkable. Mental status examination is significant for gross evidence of short-term memory loss and word-finding problems as well as symptoms of depression and anxiety. The patient's score on the Mini-Mental State Exam (MMSE) is 19 (maximum, 30). The physician initiates a workup to look for reversible causes of dementia that includes laboratory work, neuropsychological testing, and brain magnetic resonance imaging. Folate and B₁₂ levels, VDRL, thyroid-stimulating hormone, complete blood count, basic metabolic and liver panel, and urinalysis are all within normal limits. Imaging shows white matter hyperintensities.

Clinical Impression
Based on the patient’s history, physical/neurological examination, mental status testing, laboratory results, and neuropsychological report, the physician’s impression is that the patient has Alzheimer’s dementia with accompanying noncognitive neuropsychiatric symptoms of significant depression and anxiety.

- How common is dementia and the neuropsychiatric symptoms of dementia?
Dementia is one of the largest health problems facing our aging society. The dementia syndrome refers to a group of symptoms related to a sustained decrease in memory in addition to decline in 1 or more other cognitive areas (eg, aphasia, apraxia, agnosia, or executive functioning) accompanied by loss of function [1]. It is a devastating syndrome that is estimated to affect over 5 million people in the U.S. [2]. Causes of dementia include Alzheimer’s disease (AD), vascular disease, Lewy body disease, frontotemporal dementia, dementia due to other neurodegenerative illnesses such as Huntington’s and Parkinson’s disease, as well as other less common causes. The cost of the dementia syndrome has been estimated at up to $140 billion per year in caregiving costs, lost productivity, and medical and institutional care [2]. The risk of dementia increases with age, doubling in prevalence every 5 years after age 60 [3], and because the proportion of the population over 65 is rapidly increasing, dementia is emerging as a major public health problem.

Although cognitive impairment is the clinical hallmark of dementia, noncognitive symptoms are exceedingly common and often dominate disease presentation [4]. Thirty percent of the cost of caring for community-dwelling patients with AD is directly attributable to the management of neuropsychiatric symptoms (NPS) [5]. NPS commonly occur in dementia of all types and include depression, anxiety, apathy, agitation, sleep problems, and psychosis (delusions, paranoia, hallucinations). Such symptoms have been noted in 60% to 90% of patients with dementia [6–8] and may fluctuate over the course of the illness. In terms of psychotic symptoms, data from a study of the cumulative incidence of hallucinations and delusions in patients with probable AD suggested that half of patients with clinically diagnosed AD will manifest hallucinations or delusions within 4 years [9]. Psychotic symptoms are found in the majority of patients with dementia with Lewy bodies [10], where visual hallucinations may be one of the first manifestations of the illness and systematized persecutory delusions are common.

It is the NPS, as opposed to the core cognitive symptoms of dementia, that tend to create problems for patients, families, caregivers, and providers and that commonly lead to earlier placement in nursing homes [11–14]. Paranoia and agitation appear to be particularly important factors in the genesis of caregiver burden and patient institutionalization [15]. Other negative outcomes associated with the NPS of dementia include excess morbidity and mortality, increased hospital lengths of stay [16], caregiver stress and depression, and reduced caregiver employment income [17–20].

The assessment of a person suspected of having dementia and/or new-onset psychiatric symptoms such as depression or psychosis should include careful history, physical/neurological examination as well as selected laboratory testing. Review of any new medical symptoms, such as symptoms of a urinary tract infection (which can cause both mental status changes as well as behavioral symptoms) should be performed. Knowing the timeline of the new symptoms is also useful; for example, delirium tends to develop acutely (eg, the family may say a change happened “overnight”), depression develops over weeks to months, and in many cases, the onset of dementia is insidious (months to years). With the new onset of psychotic or manic symptoms in later life, secondary causes such as underlying dementia, delirium, or iatrogenic causes (eg, recent steroid burst) should be thoroughly explored. In cases where memory loss has been gradual, it may be helpful to prompt family members by asking about functional problems such as getting lost while driving or having trouble remembering how to prepare previously known recipes. In addition, all medications (including over-the-counter and herbal medications) should be reviewed, including recent medication changes with a particular emphasis on drugs that may be associated with adverse neuropsychiatric effects (eg, anticholinergic medications). In addition to urinary infections and medications, other common contributing causes to new NPS in dementia include pain, constipation, dehydration, upper respiratory infection, or other medical illness [21]. Other stressors can also precipitate symptoms and may include environmental changes, difficulties in the caregiving relationship, or psychological needs that are unmet.

On examination, focal neurologic problems should be identified and the patient should be screened for depression as well as memory problems. The latter is usually screened for with the MMSE [22], a cognitive screening test that can be easily performed by physicians during an office visit. Importantly, the MMSE neither confirms nor rules out the presence of dementia; however, scores below a certain cut-off (initially 24, but some studies now suggest a higher cut-off of 26 [23,24]) indicate the need for further workup and attention. A baseline MMSE score also enables the provider to track change over time. In some cases, particularly in patients with high levels of education, the MMSE score may be close to normal in early dementia despite functional difficulties and family report of memory loss; here, alternative screening methods may be useful, such as the Montreal Cognitive Assessment (MOCA) [25]. The MOCA is also a rapid screening instrument for mild cognitive dysfunction and assesses different cognitive domains: attention and concentration, executive functions, memory, language, visuoconstructual skills, conceptual thinking, calculations, and orientation. Time to administer the MOCA is similar to the MMSE and
the total possible score is also 30 points (a score of 26 or above is considered normal).

Laboratory screening for thyroid disease and vitamin B₁₂, and folate deficiency is also recommended; other basic laboratory work that may be helpful can include a complete blood count (as anemia can mimic symptoms of depression) and basic metabolic and liver functions. Urinalysis is also performed in the assessment of confusion, agitation, or other new psychiatric symptoms to rule out a urinary tract infection. Imaging of the brain, such as a noncontrast computerized tomography or magnetic resonance imaging scan is performed to rule out neurologic abnormalities (eg, stroke, normal pressure hydrocephalus, subdural hematoma) as well as to assist in determining the underlying etiology of dementia symptoms (eg, Alzheimer’s vs. vascular dementia). Neuropsychological testing is used both to examine patterns of cognitive impairment that may be helpful in distinguishing among the types of dementia as well as establishing a baseline of cognition for the individual patient.

**Patient Follow-up**

The patient’s primary care physician starts the patient on donepezil 5 mg per day for her cognitive symptoms as well as citalopram 10 mg per day for mood lability as well as occasional passive suicidal ideation. Donepezil is increased to 10 mg per day after 1 month, while citalopram is increased in 10-mg increments over a period of 1 year to 40 mg. The patient has a formal driving evaluation by the state and based on the recommendations of the examiner agrees to surrender her license.

Despite taking the antidepressant for a number of months, the patient continues to be depressed and is referred to a geriatric psychiatrist. When seen by the geriatric psychiatrist, it is noted that her symptoms are consistent with a partially treated depression with particular symptoms of diurnal variation, guilt symptoms and feeling like a burden, and mood lability as well as occasional passive suicidal ideation. In addition to the medications noted above, the patient was taking hydrochlorothiazide 12.5 mg (for hypertension) and lovastatin 20 mg (for hypercholesterolemia). Citalopram is increased to 10 mg per day after 1 month, while citalopram is increased in 10-mg increments over a period of 1 year to 40 mg. The patient has a formal driving evaluation by the state and based on the recommendations of the examiner agrees to surrender her license.

During the same period, the patient becomes increasingly agitated, labile and tearful, and paranoid toward her caregiver and sharply diminishes her oral intake. Her primary care physician starts her on haloperidol 0.5 mg per day. A few days later, the patient has a syncopal episode and an emergency department visit followed by a psychiatric admission for stabilization and safety.

- What are the risks and benefits of antipsychotic medication in the elderly?
- What is the evidence for risk of mortality with use of antipsychotics in the elderly?

As compared with research to treat cognitive symptoms, research examining the treatment of NPS of dementia is modest, and no medication is approved by the U.S. Food and Drug Administration (FDA) for this indication. Nevertheless, conventional antipsychotics have long been used to treat behavioral symptoms, and their overuse in U.S. nursing homes in the 1980s led to federal regulations requiring their oversight [26]. However, Teri et al [27] found no significant differences between haloperidol, trazodone, behavioral management techniques, and placebo in treating agitation in Alzheimer’s disease.

Following the introduction of atypical antipsychotics in the 1990s, with lower reported rates of parkinsonism and tardive dyskinesia, there was a significant shift from the use of conventional antipsychotics to atypicals [28]. In 2001, over 70% of atypical antipsychotic prescriptions in the United States were written for off-label indications [29], and atypical antipsychotics accounted for 82% of antipsychotics written for older patients in Canada in 2002 [30]. At present, atypical antipsychotics have largely replaced conventional antipsychotics as the preferred treatment modality for NPS of dementia [31].

The shift from conventional to atypical antipsychotics in the elderly is thought to be due to several factors [31]: efficacy evidence from clinical trials; perceived safety advantages; and expert clinical opinion [32]. Schneider et al’s meta-analysis was based on 15 published and unpublished randomized, placebo-controlled, double-blind, parallel-group trials of atypical medications in patients with Alzheimer’s disease or dementia (drug = 3353 patients, placebo = 1757) [31]. After statistically combining the trials, they observed the following: (1) evidence for symptomatic efficacy of aripiprazole and risperidone; (2) olanzapine was not associated with efficacy overall; and (3) the evidence for quetiapine was inconclusive (trials used different selection criteria and outcomes could not be statistically combined). The magnitude of standardized mean differences for atypicals was similar to those observed in meta-analyses of the cognitive effects of cholinesterase inhibitors and memantine. There were smaller effects for less severe dementia, outpatients, and patients selected for psychosis. A Cochrane review of
atypical trial data for dementia supported the effectiveness for olanzapine and risperidone as compared with placebo for aggression and risperidone for psychosis, although both drug-treated groups had a significantly higher incidence of serious adverse events, including stroke [33]. More recently, the National Institute of Mental Health-sponsored Clinical Antipsychotic Trial of Intervention Effectiveness-Alzheimer’s disease (CATIE-AD) showed limited efficacy for atypical antipsychotic medications over placebo [34]. A subsequent study published from the CATIE-AD effectiveness trial indicated that antipsychotics may be more effective for management of particular symptoms such as anger, aggression, and paranoid ideation, but that the medications do not appear to improve patient functioning, care needs, or quality of life [35]. There have been no other trials specifically designed to assess the efficacy of antipsychotic medications for individual dementia symptom clusters (eg, psychotic vs. other syndromes) [4,36]. Non-antipsychotic medications used to treat NPS of dementia include antidepressants, mood stabilizers, and benzodiazepines. However, there is little clinical trial support for non-antipsychotic medications for treatment of NPS in dementia [4]. Cognitive enhancers (eg, cholinesterase inhibitors such as donepezil and memantine) have been reported to have small but positive effects on NPS of dementia in clinical trials; however, the data supporting these conclusions are from trials in which NPS were secondary outcomes [21].

Treating older patients is challenging due to issues common in the aging population: comorbid illness; polypharmacy; and age-related changes in pharmacokinetics and pharmacodynamics, which may include changes in drug response and ability to metabolize drugs. All of these factors may cause the elderly to be more susceptible to treatment-emergent adverse events [37]. The treatment of elderly patients is even more difficult when the patient has dementia [38]. The mortality rates of patients with dementia are twice those of the older population without dementia, and the risk of mortality increases with dementia duration [39].

While antipsychotics may have some sedating properties for older patients, their use in patients without psychotic symptoms is not recommended in light of their side-effect profiles as well as recent evidence that they may be associated with a significant risk of increased mortality in the elderly. Antipsychotic use can lead to extrapyramidal symptoms, gait instability, falls, and hip fractures [40–42]. At the same time, their use in a population where polypharmacy is common may lead to greater problems of drug-drug interactions and drug toxicities [43]. Finally, a number of critical studies have linked the use of both conventional and antitypical antipsychotics with cardiac arrest and heart failure [44,45]. Among conventional antipsychotics, the risk of adverse events is even higher than among those prescribed atypical antipsychotics [46]. Furthermore, adverse events frequently occur within the first month of being prescribed an antipsychotic [46].

Concerns about cerebrovascular adverse events (CVAEs) with the use of antipsychotics in dementia patients began to arise in 2002 [47]. Later estimates of pooled data from risperidone trials suggested that the drug was associated with approximately 3 times the risk of CVAEs over placebo in this population [48]. Subsequently, the manufacturers of olanzapine presented data from randomized controlled trials that indicated that the risk of CVAEs was also 3 times higher in treated than placebo patients with dementia and NPS. Consequently, safety information updates were published in Canada [49], the United States [50–52], and the United Kingdom [53].

In 2005, the FDA issued a “black box” warning that the “treatment of behavioral disorders in elderly patients with dementia with atypical (second-generation) antipsychotic medications is associated with increased mortality [54].” This advisory was based on an FDA analysis of data from 17 placebo-controlled trials (only 4 of which had been published) performed with atypicals (aripiprazole, olanzapine, quetiapine, and risperidone) in 5106 patients with dementia-related behavioral disorders. In the analyses, atypical agents showed increased mortality (approximately 1.7-fold) in drug-treated compared to placebo-treated patients. Specific causes of death were mostly cardiac-related events (eg, heart failure, sudden death) or infections (primarily pneumonia). A similar warning for conventional antipsychotics followed in June 2008 [55]. During this same time period, a number of research reports also (1) confirmed the concern for mortality with antipsychotics in both dementia [56–58] and in the elderly [59,60]; and (2) indicated that conventional antipsychotics were no safer, and in a number of studies appeared to confer higher risk, than atypicals. The association between mortality and antipsychotics is not well understood, and may be due to a direct medication effect or to the pathophysiology underlying neuropsychiatric symptoms that prompt antipsychotic use.

More recent studies have linked atypical antipsychotics with the risk of sudden cardiac death [61] and reaffirmed stroke risk [62]. Finally, the recent Dementia Antipsychotic Withdrawal Trial (DART-AD) randomized 165 AD patients to either continue antipsychotic treatment or receive placebo; those who continued antipsychotic medication had a significantly higher risk of mortality than those allocated to placebo, with the difference in mortality more pronounced after 1 year [63].

Thus, the risk-benefit profile for antipsychotics for other than FDA-indicated conditions (eg, schizophrenia and bipolar disorder) in later life would appear to be very heavily weighted toward the risks.
Is there any difference in risk of mortality among individual atypical antipsychotic agents?

Most studies up to this point have focused on antipsychotic agents as classes (e.g., conventionals or atypicals). Thus, there is very limited information about mortality with individual antipsychotic agents. Kryzanovskaya et al found no significant differences in mortality between olanzapine and risperidone (0.89, −0.33 to 2.11) in analyses adjusted for exposure-related differences [38]. However, the number of patients who died during this trial was small (n = 6 and n = 4, respectively) and confidence intervals were very wide.

In the mortality meta-analysis of Schneider et al [56], there was no increased risk of death found with any individual atypical antipsychotic; however, there may have been inadequate power to detect significant differences between agents after controlling for confounding variables between trials. Differences between trials included nursing home versus outpatients; trial length; severity of cognitive impairment; and key inclusion criteria (Alzheimer’s vs. mixed dementias, symptom clusters included, fixed vs. variable dosing). Also unknown are differences in medical burden between patients in various trials. Further studies are needed with larger sample sizes controlling for key covariates that can be assembled in observational cohorts.

Expert consensus is that second-generation antipsychotics are not a homogenous class. Although most of these agents possess potent antagonism at the serotonergic receptor, they exhibit significant differences in activity at muscarinic, cholinergic, histaminergic, noradrenergic, and other serotonergic receptors [64]. In the elderly, these differences have import for adverse events. For example, the elderly are significantly more sensitive to anticholinergic toxicity, which may impact adversely on already impaired cognition [37].

Atypical antipsychotics also have different movement disorder adverse effects; in patients with Parkinson’s disease, olanzapine and risperidone are both associated with worsened motor function, while quetiapine is not [65]. Risperidone and olanzapine have both been associated with extrapyramidal side effects in patients with dementia; there is less experience with quetiapine in this patient population [66]. In Schneider et al’s efficacy and adverse effects meta-analysis [31], only risperidone was significantly associated with increased extrapyramidal symptoms, although olanzapine was associated with abnormal gait. There was also a significantly increased risk of CVAEs with risperidone.

In an ongoing retrospective cohort study using VA national healthcare data (NIMH R01MH081070), our group is comparing 12-month mortality risks for individual antipsychotic agents in older patients with and without dementia diagnoses, determining cause of death in patients who die in the 12 months following a new antipsychotic prescription, and performing analyses among patients with dementia to examine the impact of cognitive impairment/dementia stage and behavioral disturbance on medication preference and mortality. Figure 1 outlines our conceptual framework.

**Patient Follow-up**

During the psychiatric admission, haloperidol is discontinued and replaced with olanzapine 2.5 mg.
at bedtime. Citalopram is cross-tapered to venlafaxine. After discharge when seen by the geriatric psychiatrist, the patient shows improvement but has ongoing agitation and paranoia toward her caregiver and depressive symptoms. Olanzapine is titrated to 5 mg and venlafaxine titrated to 225 mg total per day.

- **If antipsychotic medications are used, what should be the approach?**

The lack of efficacy and side-effect issues with antipsychotics leave clinicians in a difficult situation given the prevalence of NPS in dementia patients, the desperation of families (either attempting to keep a family member with difficult behaviors in the home, or trying not to lose a nursing home placement), and the lack of true pharmacologic alternatives. Despite concern about adverse effects associated with antipsychotic medication use in dementia patients, in clinical practice some families may be less concerned with the FDA and other warnings and more focused on the need to manage complex neurobehavioral disturbances in their loved ones with dementia.

Both the American Association for Geriatric Psychiatry [21] as well as the FDA [54] have urged clinicians and families to adopt a risk-benefit approach to determining when to use antipsychotic medications in patients with dementia, which includes (1) use of antipsychotics only for psychotic symptoms (eg, hallucinations, paranoia, delusions) that potentially endanger the patient or family (eg, patient has a delusion that family is poisoning him and won’t eat); (2) targeted treatment for NPS clusters (eg, use antidepressants for depression symptom cluster; use anticonvulsants for agitated/labile cluster); and (3) conducting a thorough evaluation of behavioral antecedents (eg, aggression occurring in association with a particular caregiver or activity) and use of behavioral management strategies.

It is also very important to note that the NPS of dementia tend to be a “moving target.” What works today may not work tomorrow. In addition, behaviors may extinguish over time or manifest in other ways. Thus, medications to treat such symptoms should not simply be renewed indefinitely, and attempts at periodic drug withdrawal are recommended. See Figure 2 for a recommended approach to management of NPS. Specialty care (geriatric psychiatry, neurology or geriatric medicine) is usually indicated for the long-term management of these cases.

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**Figure 2.** Managing neuropsychiatric symptoms (NPS) of dementia. ECG = electrocardiogram.
**Patient Follow-up**

Examination of antecedents of agitation and paranoic behavior centers on interactions with a female caregiver. The patient believes that this caregiver patronizes her; the patient perceives that there might be something “going on” between the caregiver and her husband. Specific recommendations are made to the caregiver to improve interactions with the patient, including (1) to avoid using a child-like tone or language, which might seem condescending to the patient; (2) to offer choices rather than directions; (3) to defer to the patient’s wishes unless safety is a concern; and (4) to address underlying concerns rather than arguing or confronting inaccurate accusations. The patient shows marked improvement over the next few weeks with much improved mood and affect, decreased agitation and resolution of paranoia, and improved oral intake and appropriate weight gain.

**What is the role of behavioral management in dementia?**

NPS of dementia cause significant distress to patients and their caregivers. The manifestations of these symptoms are complex, diverse, and often dependent on the environment and the situation. Therefore, an individualized approach, with knowledge of the patient’s life experience is essential [67]. Careful observation, creativity, and problem-solving are keys to successful behavioral interventions [68]. As previously discussed, every pharmacologic intervention carries some risk of side effects or adverse reactions. Behavioral interventions, when applied judiciously and appropriately, have a reduced risk of side effects and no drug-drug interactions or adverse reactions [69]. Customizing available interventions to the history, needs, and abilities of the patient may lead to greater benefits. Not every behavioral intervention works with every patient, and not every intervention works all of the time. While more studies are needed to explore specific efficacies and patterns of response for NPS of dementia, 2 major evidenced-based behavioral models provide guidance and understanding into the meaning of such symptoms and a different way of thinking about “problem” behaviors.

**Need-Driven Dementia-Compromised Behavior Model**

In the Need-Driven Dementia-Compromised Behavior (NDB) model [70], NPS of dementia are viewed as an expression of unmet needs or goals (physical, psychological, emotional, or social). The loss of ability to express needs verbally leads the person to communicate and express needs through behavior. The NDB model emphasizes the interaction between individual characteristics and fluctuating environmental factors that may cause stress or discomfort. The management strategies are highly individualized and arise out of comprehensive assessment data. The model also recognizes that passive “nonproblematic” behaviors can also be the result of unmet needs [71] and lack of meaningful activity may develop into unmet needs [72].

To apply this model to our case example, the patient’s suspiciousness and paranoia could represent unmet emotional needs for affection and security in their marriage. The husband, caregiver, and patient could be educated to recognize and address these needs. They could develop strategies to emphasize the dyad of the husband and wife couple rather than the husband and caregiver team.

**Progressively Lowered Stress Threshold Model**

This model [73] was developed to organize observations, make care decisions, and plan care by modifying stress-inducing triggers. The premise is that all behavior has meaning and that NPS result from excess environmental stress. Older adults with dementia have a decreased ability to process stimuli. This decrement lowers the stress threshold, which produces higher levels of anxiety/frustration, and if unabated, develops into anxiety and severe agitation. Stress is caused by physical conditions (hunger, thirst, pain), changes in routine, too many competing stimuli, and physical and social environmental changes. Behavioral strategies are based on adapting the patient’s environment to reduce potential trigger reactions. The following factors in the model contribute to stress: physical stressors (pain, acute illness, depression), fatigue, change in environment, routine or caregiver, misleading stimuli or inappropriate stimulus levels, and demands that exceed functional ability [74].

To apply the second model to our case example, the patient is suspicious of the interaction between her husband and caregiver. On further exploration, he expresses a wish for independent time out of the home. Decreasing the time that both the husband and caregiver are present with the patient could avoid misinterpretation of the situation. The caregiver could also wear a uniform/name badge to provide a visual cue, defining her role.

Many alternative methods have been explored and demonstrated to be at least somewhat effective. In a systematic review of non-drug treatments for dementia, the Leeds Institute of Health Sciences lists 3 evidence-based approaches that are likely to be beneficial [75]. These are music or music therapy, hand massage or gentle touch, and physical activity or exercise. However, evidence is mixed or limited even for these interventions. The authors caution that these interventions do work in reducing some but not all behaviors. Other practices, including acupuncture, animal-assisted therapy, light therapy, reality orientation, and reminiscence therapy, light therapy, reality orientation, and reminiscence
therapy, lack sufficient evidence and are categorized as “might work” [75]. Perhaps the most fruitful intervention involves education of the family and caregiving team. Teri et al [76] developed a practical evidenced-based systematic approach to caregiver training in community-dwelling caregivers to reduce mood and behavior disturbances in older adults with Alzheimer’s disease. In a randomized control trial, 95 family caregivers and Alzheimer’s disease care recipients were randomly assigned either to the intervention or the control group. Consultants were trained on the rationale and use of the “A-B-C” (antecedents-behavior-consequences) problem-solving approach to behavior change [77,78]. The study demonstrated improvement in the frequency and severity of care recipient behavior problems and improved quality of life. In addition, family caregivers showed significant improvements in depression, level of burden, and reactivity to behavioral problems.

CONCLUSION

Our case provides an excellent illustration of the coordination of pharmacologic and nonpharmacologic approaches to the management of NPS of dementia. The patient suffered from depressive symptoms and psychosis secondary to Alzheimer’s disease. These symptoms responded well to a regimen of an antidepressant and antipsychotic added to the cholinesterase inhibitor. However, nonpharmacologic interventions led to further improvement. The precipitant to the agitation and paranoia (eg, suspiciousness of the caregiver) was addressed. Education of the caregiver and the patient’s husband included specific suggestions in their approach to the patient. This led to better enjoyment of personal interactions for all, decreased caregiver stress, and improvement in the patient’s general health as evidenced by appropriate weight gain.

In summary, antipsychotic medications are widely used in the elderly, particularly for the NPS of dementia. The questionable efficacy for the wide spectrum of NPS and side-effect issues with antipsychotics leave clinicians in a difficult situation given the prevalence of NPS, the desperation of families, and the lack of true pharmacological alternatives. In some cases, antipsychotics may be viewed as the only option for patients with harmful behaviors, particularly given that there is little clinical trial data supporting the use of non-antipsychotic medications for treatment of NPS of dementia. It is useful for clinicians and families to adopt a risk-benefit approach to determining whether and when to use antipsychotic medications in dementia. Nonpharmacologic interventions can be effective in alleviating or decreasing NPS of dementia, either independently, or when combined with pharmacologic agents, providing decreased distress to both patients and their caregivers.

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Corresponding author: Helen C. Kales, MD, Dept. of Psychiatry, Univ. of Michigan, 4250 Plymouth Rd., Box 5765, Ann Arbor, MI 48109, kales@umich.edu.
Financial disclosures: Dr. Maixner has been on the speakers bureau of AstraZeneca and Pfizer-Eisai and a consultant for Pfizer-Eisai.
Author contributions: conception and design, HCK, MB; drafting of article, HCK, MB, LMS; critical revision of the article, HCK, MB, SMM, LMS.

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