Spring 3-2016

Dementia Care: Confronting Myths in Clinical Management

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Abstract

Every day, patients with dementia, their families, and their physicians face the enormous challenges of this pervasive life-changing condition. Seeking help, often grasping at straws, victims, and their care providers are confronted with misinformation and myths when they search the internet or other sources. When Persons with Dementia (PWD) and their caregivers believe and/or act on false information, proper treatment may be delayed, and ultimately damage can be done. In this paper, we review commonly misunderstood issues encountered in caring for PWD. Our goal is to equip Primary Care Practitioners (PCPs) with accurate information to share with patients and families, to improve the outcomes of PWD to the greatest extent possible.

While there are innumerable myths about dementia and its causes and treatments, we are going to focus on the most common false claims or misunderstandings which we hear in our Internal Medicine practice at Marshall Health. We offer suggestions for busy practitioners approaching some of the more common issues with patients and families in a clinic setting.

Introduction

The numbers are now familiar but remain staggering – 5.3 million Americans have Alzheimer's disease, and around 36,000 of them are in West Virginia. Within 10 years, those numbers are going to increase to 7 million and 44,000.[1] Clearly, every PCP has PWD on his or her patient panel. Recent data analysis shows that documented diagnosis of Alzheimer's disease in rural counties is substantially (11%) lower than in urban counties, so in West Virginia and other rural states, it is likely that each PCP has even more such patients than they realize.[2]

While it is extremely important that all parties have adequate knowledge of the disease process, and potential sources of information are widely available, these sources can include some gross misinformation. The internet, TV, and second-hand reports of "what John's doctor told him" can influence families to make bad decisions and in other ways lead to poor care. PCPs, therefore, need to be ready with accurate information to share during office visits.

It is universally true that PCPs are pressed for time, and do not have the luxury of either extensive literature reviews or long educational sessions with their patients and families. We identify the more common and egregious myths about Alzheimer's and other dementias and offer suggestions for efficient and effective ways to debunk them.

Myths vs. Clinical Truths for PWD

Myth I: "My mother had it, my grandmother had it. I'm doomed."

The Truth: *There is a genetic component to Alzheimer's disease and other dementias. Risk-conferring mutations which may or may not eventually cause disease are rather common, but determinative genetic mutations which inevitably lead to the disorder are quite rare.*

While age remains the greatest risk factor for dementia, family history *is* significant. Having a single first-degree relative with dementia increases an individual's risk by 10 to 30 percent[3], although the excess risk is less if the affected family member was older than 85. Recent advances in medical genomics offer great promise in terms of risk quantification. However, most of this information are still unevaluated "big data".

Certain scenarios are clearer than others. Young-onset Alzheimer's dementia, with initial symptoms between the ages of 30 and 60, has been extensively characterized in terms of genetic loci involved. Family history is quite relevant in such cases, as the disease in most young-onset cases follows an autosomal dominant pattern with high penetrance. For most other patients, however, the development of dementia depends on multiple risk factors, of which family history is only one. Family history of late-onset Alzheimer's *is* a risk factor for the development of dementia. However, hypertension, diabetes, hyperlipidemia, physical inactivity, depression, obesity, and smoking are also risks. Some evidence suggests that up to a third of cases of Alzheimer's disease are attributable to these factors[4]. Risk factor modification (particularly in middle adulthood) may be key in dementia risk mitigation; seemingly mundane education in primary prevention could eventually yield substantial benefits.

In many cases, patients and families ask for expensive genetic tests to gauge or decrease risk. Two points should be emphasized:

- First, family history of dementia does not mean an individual is going to develop dementia.
- Second, lack of family history does not guarantee that a person is going to escape dementia.

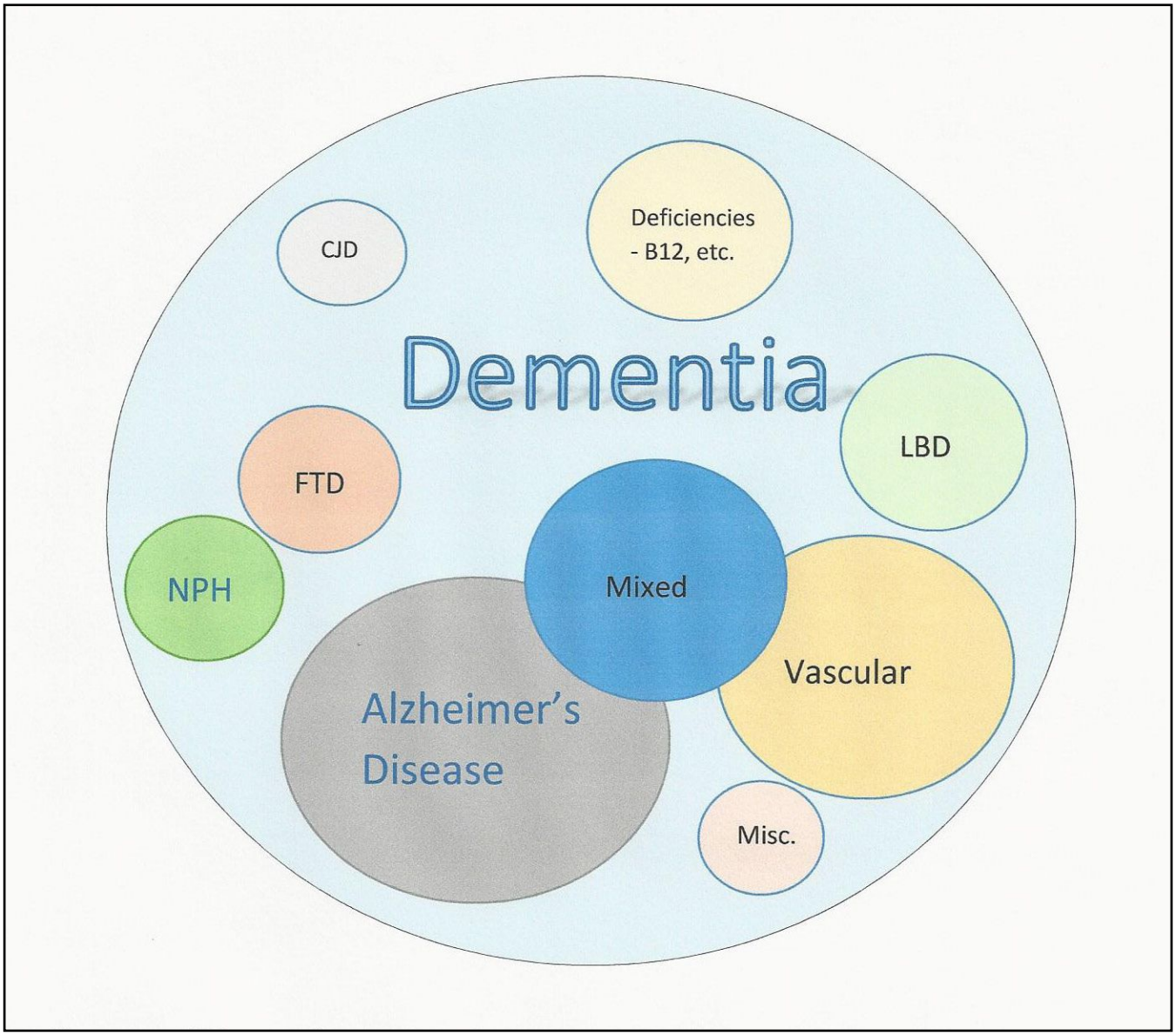
It may be worth specifically pointing out to families who request testing - with currently available genetic testing, one can only confirm what one already knows, that is, whether one is at higher risk or not. If a person is a member of one of the rare families with a known determinative mutation, testing can confirm the presence or absence of the gene in a given individual, but again these are rare cases, and counseling must be available to those who choose to be tested.

Myth II: "I've lost my keys again, I need Aricept™!"

Truth: *Memory lapses, such as losing your keys, forgetting someone's name, or forgetting why you entered a room are common with normal aging. They do not imply dementia nor predict it.*

By definition, dementia is a *significant decline* in cognition that interferes with one's ability to carry out normal daily functions. Subjective memory complaints rarely correlate to dementia. Once a patient becomes concerned about their memory, the PCP needs to be efficiently prepared to decide if there is a real concern or if the patient is in fact quite normal.[1] An effective first step toward validating a patient's worry about their subjective memory concerns is to discern if the memory lapses have caused interference with their daily activities.[5] Practical differentiation can be made along these lines:

Figure 1. *Illustration of "dementia" as the general term encompassing memory loss disorders of various etiologies.*



- A *normal memory lapse* might be forgetting the name of someone whom you met only once or twice, or forgetting why you went into a particular room.
- *Mild cognitive impairment* is forgetting the name of someone you see daily and not remembering it later. Mild cognitive impairment (MCI) (now known as Neurocognitive Disorder- Minor) does not significantly interfere with daily activities. It has more symptoms and disability than normal forgetfulness; it is described as the patient and the examiner both knowing that something is different or wrong, but still the patient can carry on with his normal life.
- In the case of actual *dementia* (Neurocognitive Disorder – Major), affected persons may *not* complain of memory loss but indeed may deny that they have any symptoms.[6] Observers can tell that these individuals have cognitive problems, which can be manifested by not just forgetting names, but forgetting the function of common objects and getting lost.

Patients worry about such memory lapses, not because they interfere with their activities,

but because they fear they are going to develop dementia, but there appears to be no link between normal forgetfulness and the development of MCI or dementia. MCI, on the other hand, may progress; on average, 10% per year of persons identified with MCI may progress to a diagnosis of actual dementia. The medications we currently have available for dementia are not useful for normal forgetfulness nor MCI though trials are underway to assess the benefits of treating minimal impairment.

Myth III: "Well, if it's Alzheimer's, at least, it's not dementia."* and *"So if it's dementia now, when will it go into Alzheimer's?"

The Truth: *While they are obviously related, Alzheimer's disease is not a stage of dementia, nor is dementia a stage of Alzheimer's.*

People have usually been trying to research the possible diagnoses by the time they present for evaluation; this can lead to major confusion for those with insufficient background knowledge. The two most common misunderstandings are 1) that AD and dementia are two separate and distinct disorders, or 2) that Alzheimer's is an early stage of dementia or vice versa.

PCPs should explain that "Dementia" is the umbrella term for chronic cognitive impairment disorders, and that Alzheimer's is only one type of dementia. It may be useful to demonstrate the concept with a diagram such as Fig. 1. Alternatively the analogy "All dogs are four-legged animals but all four-legged animals aren't dogs" may be used. Understanding the relationship between these terms helps considerably in future attempts to learn about the disease.

Myth IV: "The scan showed it was Alzheimer's disease."

The Truth: *At this point, no scan, no lab test, and no examination can unequivocally confirm a specific etiology of dementia in most cases.*

Extensive research is bringing us closer to the day when a premortem test can definitively diagnose Alzheimer's and other etiologies of dementia. However, it is a disservice to patients and families to imply to them that anything more than a *probable* diagnosis can be made currently in the majority of cases.

The National Institute on Aging – Alzheimer's Association workgroup published updated diagnostic criteria in 2011, the first significant revision since 1984.[7] They begin with core clinical criteria for the diagnosis of all-cause dementia. Once a patient meets these basic standards and is considered to have a diagnosis of dementia, further criteria are described for classifying diagnoses of 1) Probable Alzheimer's dementia, 2) Possible AD dementia, and 3) Probable or possible AD dementia with evidence of AD pathology. These criteria explicitly state that the latter category is intended for research purposes only; the reason being that neither imaging nor lab studies which can produce "evidence of AD pathology" are specific, sensitive, or reliable enough to be used in clinical practice.

A brain imaging study is recommended in the workup of a patient with memory impairment, even though it is not diagnostic. CT scans are useful for ruling out conditions which may not have been suspected or which cannot be proven by examination alone, for example, small or symmetrical subdural hematomas or even tumors. Normal Pressure Hydrocephalus may sometimes be diagnosed with CT scanning. MRI of the brain can be helpful to document the extent of vascular disease or the disproportionate atrophy seen in different areas with various dementias. PET scanning with either of two agents can be very supportive of an AD diagnosis. With the "Pittsburgh Compound" marker, amyloid deposition can be seen, and with fluorodeoxyglucose (FDG)-PETs, loss of glucose uptake in damaged neurons is seen. However, because amyloid can be seen in PET scans long before clinical symptoms occur, and because quantitative analysis of FDG-PET results is not standardized, these imaging studies cannot yet be considered diagnostic.

Biomarkers, such as CSF levels of tau protein and Beta-amyloid, are also insufficiently standardized to be used clinically for confirmation of diagnosis. (Low CSF $A\beta_{42}$, or elevated CSF total tau or phosphorylated tau, are positive markers of probable Alzheimer's.) In addition to the lack of standardization, there is limited access to this testing in most community settings. At this time, these tests are an option for clinicians but are used only in clinical trials. Carefully applied clinical criteria are sufficient for diagnosis in most cases without the expense of biomarker testing or the potential problems of lumbar puncture.[7]

Myth V: "I told her over and over that her dad has been dead for 30 years and is not coming to get her. However, she still asked me the same question a hundred times."

The Truth: *Repetitiveness, and believing that people from the past are still present, are among the most common manifestations of Alzheimer's and other dementias. These behaviors and beliefs cannot be corrected.*

Table 1. Complementary and Alternative Medicine Agents Promoted for Use in Dementia

Entity	Mechanism of Action	Evidence
Acetyl-l-carnitine	Lipid metab.; membrane structure; potent direct cholinergic agonist	2 large trials and Cochrane meta-analysis of 16 studies: **No benefit**
Ashwagandha (winter cherry; Indian ginseng)	Evergreen herb; antioxidant	Indian traditional medicine; limited animal studies; possible benefit for anxiety: **No benefit shown for dementia**
Caprylic acid (Ketasyn™, Axona™); Coconut oil	Triglyceride from coconut or palm kernel oil –"alternative energy source for brain cells"	Testimonials; **Phase II trial – no effectiveness; No Phase III trials done:** Alz. Assoc. – **not enough evidence to assess possible benefit as medical food**
Co-Enzyme Q 10, Ubiquinone	Antioxidant	Small trial of synthetic version: **Not effective**
Coral Calcium	Calcium carbonate plus trace minerals	**FDA and FTC filed complaints** against the product for exaggerated claims
Curcumin	Turmeric derivative; Anti-inflammatory; Antioxidant	Traditional Indian spice: **No clinical trials**
Gingko Biloba	Antioxidant; Anti-inflammatory	Traditional Chinese medicine; Small trials and meta-analysis in 199's and 2000's suggested benefit: **Phase III trial – no benefit, and GEM (Gingko Evaluation and Memory trial) – no benefit**
Huperzine A	Properties similar to cholinesterase inhibitors	Traditional Chinese medicine (moss extract); Small trials ongoing: **Alz. Disease Coop. Study (ADCS)- no benefit over placebo**
Lecithin	Choline phospholipid	Several trials: **No benefit**
Nicotinamide	Water soluble B vitamin	**No evidence of benefit**
Omega-3 fatty acids	DHA is chief Omega-3 in brain; Anti-inflammatory; Decrease risk of heart disease	FDA – Supportive but not conclusive evidence for DHA reducing risk of CAD: **Trials inconclusive, marginal benefit in dementia**
Periwinkle (vinpocetine)	Possible vasoactive and neuroprotective	Folk remedy in various areas of the world; Cochrane analysis: **Inconclusive and does not support use**
Phosphatidylserine	Stabilizes neuron membranes	FDA: **Little scientific evidence to support claim of reduced dementia risk**
Piracetam	GABA derivative	One well designed study and Cochrane analysis: **No benefit**
Tramiprosate (Alzhemed™, Vivimed™)	Modified amino acid taurine	FDA: **No benefit in Phase III trial**

Caregivers of PWD must deal with a number of disturbing symptoms. Which of the myriad behavioral and psychological symptoms of dementia can occur in a given person cannot be predicted, but some are more common than others. These include repetitiveness, loss of memory of current relationships with reversion to the belief that one's parents and siblings are as they were in the person's childhood, excess agitation over small matters, and losing/hiding items but believing they were stolen.

The main thing that caregivers need to know is that these behaviors are not volitional, and they should not argue with the patient. The PWD is not deliberately being difficult; rather, due to their brain damage, they are "existing in a different reality" at that moment. Arguing or trying to convince them otherwise may only escalate the agitation. Asking for a long dead person is particularly sensitive. No matter how many times they are told, they will not "learn" that the person is gone, and being so informed over and over can actually induce new grieving each time.

A full review of communication techniques and behavioral treatment is beyond the scope of this paper. However, PCPs should be prepared to teach caregivers that the PWD cannot make new memories, and therefore "reality orientation" is not going to work. Diversion and non-committal responses to repeated questions (even frankly bizarre statements), may calm the person for at least a while, whereas arguing, scolding or loss of temper serves

no purpose. As long as the behavior is not overtly dangerous, the PWD need not be "corrected".

Myth VI: "I saw on Facebook that we need to give her coconut oil. It cured all those people."

The Truth: *As for all disorders in the age of the internet, Google is full of postings about complementary, alternative, herbal, and homegrown remedies for dementia. There is no significant scientific evidence to support the use of any of them.*

Complementary or Alternative Medicine (CAM) is a conundrum for practitioners. Often CAM is viewed by practitioners as not conforming to the mainstream of medical thought and practice, but there *has* been progress in understanding a proper place for CAM in our practices. Also, further credence for proper placement of these agents in our treatment plans is developing as we learn about mechanisms of action and potential clinical effects.

However, like so many fashionable ideas, much hope has not defined clear benefit. Testimonials and traditional practices do not constitute scientific evidence upon which clear recommendations of benefit or cost-benefit can be defined. Billions of dollars per year are spent on CAM treatments in the hope of benefit for patients suffering from dementia. Families and patients must recognize that there is no regulation or standardization applied to supplements or "medical foods" by governing organizations such as FDA. This absence of regulation and oversight allows for propagation of unproven claims of effectiveness and safety, and for unclear parameters of purity. Additionally, there is a possibility of potential direct harm, such as interactions with prescribed medications. CAM, therefore, must be acknowledged to be occasionally safe, but sometimes to lead to harm to the unsuspecting patient, all with no accurate data regarding effectiveness.

The impact of dementia is profound for the patient and family, so it is completely understandable that all options for possible benefit will be explored and even entertained. While there is a growing number of complementary, alternative, herbal, folk, supplemental remedies or "medical foods" which lay claim to benefiting our patients, *none* of these unregulated or non-standardized agents hold clearly documented evidence-based benefit for our patients. While hope abounds, there are no agents today with proven efficacy in dementia. Table 1 lists several agents which are commonly promoted as useful CAM agents for dementia[8], along with the most recent assessment of their degree of effectiveness.

Summary

Caring for people with dementia is an integral part of the role of a primary care provider. As difficult as it is to define and treat the disorders, we all strive to do it well. Caregivers are searching for knowledge, but frequently encounter poor information and erroneous assumptions. This paper offers a review of common situations in which families and patients may have been misled or may have drawn incorrect conclusions from confusing data. By being prepared to address quickly questions or statements, PCPs can go a long way toward getting all the participants in the care of PWD to speak the same language, which in turn can decrease errors, increase patient safety, and raise understanding of what still needs to be addressed through research.

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