Dementia – The Presence of Mind

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Dementia is a topic that is aired with increasing frequency in the media – perhaps due to raised awareness but certainly as a response to demographic change. The subject also arises in the context of life and health insurance product development, underwriting and claims.

The UK government launched its Dementia Challenge in March 2012 to drive improvements in dementia health, care and research. The ambition is that, by 2015, two-thirds of the estimated number of people in England with dementia should have a diagnosis and appropriate support. From April 2013, everyone aged 65 - 74 offered an NHS Health Check was told of the availability of help with dementia diagnosis and management. Almost 1 million people took up the offer of a Health Check in the months to December 2013, up 1.5% from the previous year and by May 2014, when Dementia Challenge progress was reviewed, diagnosis rates had increased by 6%. In a bid to boost numbers, it was announced in October last year that General Practitioners (GPs) would receive £55 for every new diagnosis measured by the net increase in the dementia register by the end of March 2015. On top of the £66 million, the government has committed to dementia research by 2015, a further £15 million of public money has been pledged to kick-start the hunt for innovative treatments.

Despite all this activity raising awareness, there remains debate about who can actually make a dementia diagnosis. The source of this dilemma is the National Dementia Strategy launched in 2009. In a bid to ensure fewer diagnoses were made only at the point of crisis, the strategy removed responsibility for dementia management from GPs and passed it instead to national network of “memory clinics”. An unforeseen side effect has been that memory has become a “privileged” symptom that may only be assessed by specialists. This effect has been made worse by GPs’ inaccurate encoding of diagnoses in individuals’ computerised health records. Other viewpoint is that dementing illness is not solely about memory and that shackling the diagnostic and support services to this emotive word creates further stigma in the minds of those who would most benefit from accessing them.

However, the fervour surrounding dementia must be viewed against a background of falling prevalence partly explained by reductions in smoking and improved treatment of hypertension and diabetes that have acted to curb the incidence of vascular risk. Commonly, estimates of future dementia numbers assume age-specific
prevalence will remain constant but a 30% reduction has been observed in three English regions. If this fall were to be replicated nationally it would significantly ameliorate the concerns about providing for the care needs of the growing numbers of older persons.

**Memory**
The word “dementia” is frequently used synonymously with Alzheimer’s disease (AD) – almost as if the latter were the proper name of the former. This error is understandable because AD is the most common type of dementia. Dementia is also commonly assumed to be a condition defined solely by poor memory, a simplification that is also inaccurate. In practice, many different factors combine to make distinct dementia types, and the borders between them are frequently indistinct.

Let’s deal with memory first. Memory is one of the most important cognitive functions, helping us to think, to know, to remember. The normal process of ageing reduces our visual acuity, hearing and motor speed but also slows information processing capability. This combination of inconveniences should not be confused with cognitive impairment or dementia. Many things cause memory difficulties in older people – low mood, decreased initiative, vitamin deficiency, low thyroid function, sleep apnoea, pain, the side effects of medicines or just being unwell. This is not dementia.

The space between the memory changes experienced in normal ageing, and those of true dementia, is occupied by a syndrome labelled Mild Cognitive Impairment (MCI). Having MCI increases the risk of dementia but does not guarantee it will develop. MCI causes people minor problems with planning, visuospatial skills and day-to-day memory, effects that may worsen, stabilise or improve. This is not dementia either.

Dementia is, however, an umbrella term that describes a syndrome that includes memory loss, mood changes and problems with communication and reasoning. Dementia is acquired (i.e. you are not born with it), progressive (to distinguish from the static damage from head injury or stroke), chronic (to distinguish from the acute cognitive problems resulting from infection or other process), and impairs a number of cognitive processes, not memory alone.

Ageing is the greatest risk factor for the development of dementia. Whilst the prevalence is relatively low between age 65 and 74, there is an exponential rise thereafter. Risk factors for all dementias are genetic, vascular or related to lifestyle. Genetics cause small numbers of early and late onset dementia. High blood pressure, raised levels of cholesterol, diabetes and smoking represent the vascular risk factors. Lifestyle contributors include traumatic head injury, alcohol and other toxic exposures, physical activity, diet and education.

**Types**
The different forms of dementia have varying, but often overlapping, symptom profiles arising from a variety of underlying physical processes. This means that making a certain diagnosis can be difficult at times. Alzheimer’s disease (AD) stems from a combination of risk factors and develops with a gradual and progressive pattern. Patients experience progressive, disabling cognitive impairment and eventually need constant care and supervision. The brain changes that contribute to the development of the symptoms are the accumulation of an abnormal protein (beta-amyloid) in plaques outside of the brain cells and a second abnormal protein (tau) as tangles within the cells. These processes interfere with the cells’ ability to communicate and eventually cause cell death. Other forms of dementia have different mechanisms of destroying the communications between cells and causing cell death. A small genetic component cannot be ruled out; for example, people with Down’s syndrome who live into their 50s and 60s are at particular risk of AD.

Vascular dementia is the second most common type after AD and is the result of multiple tiny strokes, often symptomless, that cause oxygen deprivation in multiple areas of brain tissue. Strokes, whether due to a blood clot (ischaemic stroke) or a burst blood vessel (haemorrhagic stroke), cause immediate and permanent irreversible death of brain tissue. Vascular dementia classically develops in a step-wise pattern as tissue damage progresses. The key effect of so many cerebrovascular impacts is to worsen memory but symptoms of stroke (weakness or paralysis), seizures and periods of acute confusion are also
common. There are several sub-types of vascular dementia that differ due to the cause and the focus of resulting brain damage.

Mixed dementia occurs when more than one type of dementia exists simultaneously. For example, and most commonly, the abnormal protein deposits of AD co-exist with blood vessel problems linked to vascular dementia. This combination of pathologies creates significant difficulties with daily functioning because of problems with thinking, planning and memory. In autopsy studies, up to 50% of brains that met the pathological criteria for AD were found to have evidence of one or more coexisting dementia.

Dementia with Lewy bodies (DLB) shares the symptoms of Parkinson’s disease and AD. The Lewy bodies are tiny protein deposits formed in nerve cells that are linked to low levels of acetylcholine and degeneration of brain tissue. Lewy bodies at the base of the brain cause motor symptoms, the main feature of Parkinson’s. Lewy bodies in the outer layers of the brain cause cognitive symptoms, characteristic of DLB. The motor and cognitive symptoms can occur together. In addition to the cognitive decline, the illness frequently presents with falls and florid hallucinations.

Fronto-temporal dementia (FTD) occurs when nerve cells in the lobes at the front and the side of the brain die and the pathways that connect them change. Loss of important chemical messengers occurs and, over time, the brain tissue in the frontal and temporal lobes shrinks. These areas of the brain control the domains of behaviour, emotion and the use and understanding of language. There are three FTD variants, each causing different symptoms in these domains. There may be overlap with motor disorders like progressive supranuclear palsy. FTD is more common amongst those whose dementia starts before the age of 60.

Dementia can also occur when infectious agents, known as prions, attack the central nervous system and invade the brain causing dementia. The best-known prion disease is Creutzfeldt-Jakob disease. Korsakoff’s syndrome is a brain disorder that is usually associated with long-term alcohol abuse and causes loss of short-term memory. People with HIV and AIDS sometimes develop HIV-related cognitive impairment, particularly in the later stages of their illness.

**Diagnosis and treatment**

No single test is diagnostic of dementia. The result of a brief memory test alone, including the Mini Mental State Exam (MMSE), commonly used in an underwriting context as a convenient screening tool, is insufficient to make a dementia diagnosis. The cognitive tests available to clinicians are often sensitive to the diagnosis but not specific, leading to a high false positive rate. A lone brain scan result is also not enough. Diagnosis is made only after combining a medical assessment and cognitive tests with careful history-taking (including from an informant). A full medical examination and blood testing is needed to rule out, or identify, underlying illness. Questioning the person about recent events, their past memories and investigating their thinking skills is a further diagnostic requirement. Talking with family members to seek out clues of changes in patient health over time is integral. Serial neuroimaging with computerized tomography (CT) and/or magnetic resonance imaging (MRI) may reveal developing physical changes in the brain. This is all time-consuming and expensive.

The time it takes to confirm a dementia diagnosis once symptoms are recognised varies depending on the attitudes of the person with dementia and his or her family, but also on what health structures they can access for medical help.

Drug therapy cannot cure AD but it can slow the effects. Currently five drugs are available: four acetylcholinesterase inhibitors (ACIs) and one N-methyl-D-aspartate (NMDA) receptor antagonist. The ACIs stop the breakdown of the chemical messenger acetylcholine. The NMDA blocks the messenger glutamate and is used in more severe AD cases. These are supportive and palliative therapies rather than curative or disease-modifying treatments. There is little to offer medically for dementias other than AD, although DLB can respond well to ACIs. Managing vascular risk is vital for those with vascular dementia, but there is no specific treatment.

No new treatments have become available to treat AD, or any other form of dementia, for more than a decade. The brain changes of AD are believed to begin many years before the onset of clinical symptoms, so identifying biomarkers in at-risk people would be a major first step to developing...
significant disease altering treatments. To date, population screening for dementia has not been considered cost-effective in any country. Progress has been made in neuroimaging, genetics and blood testing but these remain a long way from clinical utility.

Parting thoughts

Dementia has a lower prevalence in developing countries. Incidence rates are also much lower, partly explained by cultural indifference to mild disease and by lower levels of exposure to dementia risk factors. It is easy to dismiss dementia in countries where few people living in extreme poverty survive to age 65, but global patterns of morbidity and mortality are likely to converge, leading to an increased burden of the illness in poorer countries. Even now, most people with dementia live in developing countries; 60% in 2001 and expected to rise to 71% by 2040. It is estimated that over 35 million people worldwide had dementia in 2010 and that numbers will exceed 115 million by 2050.7

The G8 Dementia Summit held in December 2013 agreed a lofty ambition to identify a “cure” for dementia by 2025 – itself a worrying oversimplification.8 More likely, a disease-modifying therapy will result from significantly increased investment and international research cooperation. The importance of research into dementia cannot be overstated. Significant strides have been made to pull together the disparate research entities across Europe, with the hope that significant benefits will soon be translatable from the laboratory to routine clinical practice.

The task for insurers continues to be to define dementia appropriately within the domains of policy design and claims while identifying those with the disease at underwriting stage. When considering the appropriate approach to this devastating illness in an insurance context, it is important to ensure the underlying causes are well understood. Keep in mind that dementia is a memory problem that interferes with day-to-day activities but there are several different types. Remember, not all dementia is Alzheimer’s and not all memory problems are dementia.

Endnotes

2  http://dementiachallenge.dh.gov.uk/2014/05/07/champion-groups-letter/.
6  Rush Memory and Aging Project conducted by the Rush Alzheimer’s Disease Center and the Rush Institute for Healthy Aging in Chicago and funded by the National Institute on Aging (NIA).

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