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DEMENTIA: WHAT IT IS, WHEN YOU SHOULD BECOME CONCERNED, AND HOW YOU CAN HELP OTHERS

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Dementia is a clinical syndrome caused by a range of diseases or injuries to the brain. Worldwide, 47.5 million people have dementia. Given the dramatic growth of the population of older people, the number of people living with dementia worldwide is expected to double every 20 years, going up to 135.5 million by 2050. According to a 2020 report published by the Alzheimer's and Related Disorders Society of India, there are around 5 million people in India living with dementia.

Anant (name changed), an 80-year-old retired district judge, came to our institute with increasing forgetfulness for 18 months. He would misplace commonly used items such as keys, his mobile phone and pens. He would then keep searching for these items. He had also started to lose his way in and around his house. He would be frequently found in his neighbourhood trying to find his bearings. (His son then made him wear a GPS armband.) His social engagements had shrunk, as had his vocabulary. He also had wild mood swings and had become more irritable and aggressive over time.

After he became paranoid and started to think his family members were "out to get him", they consulted me. The provisional diagnosis was Alzheimer's disease with behavioural and psychological symptoms of dementia.

Clinical presentation

The most common cause of dementia is Alzheimer's disease. It is implicated in up to 70% of dementia diagnoses. The tragic combination of symptoms in Alzheimer's has a profound and resource-intensive impact on patients, family, friends, and carers.

Early symptoms include absent-mindedness, difficulty recalling names and words, difficulty retaining new information, disorientation in unfamiliar surroundings, and reduced social engagement. More atypical symptoms include impairment in recognising visually presented objects (visual agnosia) despite a normal visual field, acuity and colour vision. Some might also experience word-finding difficulties (anomic aphasia).

As the disease progresses, there is marked memory loss and loss of other cognitive skills, including a reduced vocabulary and less complex speech patterns. This may be accompanied by mood swings, apathy, a decline in social skills, and the emergence of psychotic phenomena.

Advanced disease is characterised by monosyllabic speech, psychotic symptoms, behavioural disturbance, loss of bladder and bowel control, and reduced mobility.

Evaluating dementia

Doctors diagnose dementia on clinical grounds using neuroimaging and neuropsychological tests. The first and foremost step is to obtain a comprehensive medical history of the individual from a reliable informant. A reliable informant is one who knows the individual well; it is typically a family member. Informants themselves can be influenced by their own mental states, such as depression or being in denial, so it is useful to speak with more than one informant to confirm or clarify the individual's narrative.

A slowly progressive dementia over years with insidious onset may point to Alzheimer's dementia. A dementia that progresses rapidly over months may point to dementia due to prion disease. It is useful to determine when the individual was last well instead of determining when the symptoms first showed themselves. Informants frequently minimise early symptoms by attributing them to "normal ageing".

Dementia affects cognition – the mental processes used to obtain knowledge and which inculcate an awareness of our environment. These mental processes also facilitate one's interactions with the environment. They include perception, complex attention, judgement, memory, language, imagination, social awareness, organisation, and learning.

Conducting a cognitive assessment is central to the evaluation of dementia. Common tools for this include the mini-mental state examination (MMSE), the modified mini-mental state examination (3MS) and the Montreal cognitive assessment (MoCA). Such neuropsychological tests can help differentiate dementia from milder cognitive syndromes and/or from normal ageing. The severity of dementia can be 'mild', 'moderate' or 'severe' based on the MMSE score, informed by the degree of impairment in the individual's functioning.

Further work-up using laboratory studies and brain-imaging will be required in most instances. These include a metabolic panel, liver test, blood counts, thyroid test, and vitamin B12 and folate tests. As clinical presentation and history demand, doctors may also ask for additional tests, such as heavy-metal screens, HIV test, syphilis serology, toxicology, electrocardiogram, computed tomography (CT scan), magnetic-resonance imaging (MRI), and chest radiography.

As of today, there is no genetic or biomarker test that can be used to diagnose dementia.

Preventing dementia

The WHO has identified preventing Alzheimer's disease to be a key element in the strategy to fight the world's dementia epidemic. Economic analyses have found that delaying the onset of the disease by even one year could reduce its prevalence by 11%, while a delay of five years could halve it.

Prevention programmes usually focus on lifestyle risk factors – such as sedentary behaviour, unhealthy diet, smoking, and excessive alcohol use – together with mental wellbeing and risk of cardiovascular diseases.

The Goteborg Longitudinal Study and the Honolulu Asia Aging Study have both demonstrated a strong relationship between midlife hypertension and dementia in later life. Aggressively managing vascular risk factors (e.g. keeping systolic blood pressure below 160 mm Hg), high cholesterol, diabetes, and obesity can go a long way in preventing dementia.

Current smokers have a 50% higher risk of developing dementia relative to those who have never smoked. (Smoking cessation is known to reduce dementia risk to the level of never-smokers.)

Regular exercise helps offset cardiovascular, and in fact broader, health risks. Exercise improves cerebral perfusion, has anti-inflammatory properties, improves synaptic function, and stimulates the growth of new brain cells in the hippocampus. Exercise also has a social and cognitive element that are broadly protective for mental health.

In addition, there is a robust link between depression in late life and the incidence of sporadic dementia. Having depression increases the risk of developing dementia by nearly twofold. The deleterious effects of depression on memory, sleep, and social functioning are well-established. Treating depression in persons with established cognitive impairment is vital.

Higher educational and occupational attainments have consistently been implicated as protecting against developing dementia later in life. One recent study reported that those who attain higher education have a 40% lower risk. The protective effects of education may be due to the longer period of learning, which in turn stimulates the development of larger or more complex neural networks.

The cognitive reserve theory posits that these brain reserves, accumulated by learning, compensate for the underlying dementia pathology and delay the onset of clinical symptoms.

Dementia care

The first pillar of care is to manage the important aspects of the disease, with a goal to reversing their effects or to delay its progression in the brain. The second is to manage the cognitive, neuropsychiatric, and functional symptoms of the disease.

The other two pillars involve providing systematic, evidence-based supportive care to patients and to carers.

An optimal dementia care-team includes the treating psychiatrist, occupational and physical therapists, a nurse, a psychologist, and a social worker.

Except in emergency situations, non-pharmacological interventions form the first line of therapy. But over time, medicines often become necessary, even an integral part of symptom management.

Cognitive symptoms associated with dementia are treated with drugs called cholinesterase inhibitors. Acetylcholine is a neurotransmitter that has been hypothesised to be important in cognition. Two enzymes – acetylcholinesterase and butyrylcholinesterase – degrade acetylcholine. Cholinesterase inhibitors thus increase the concentration of acetylcholine in the brain. They provide modest and temporary stabilisation of the disease process. These don't reverse or stop the degenerative process, however.

These medicines lead to notable but temporary symptomatic improvements in 10-15% of persons with dementia. The symptomatic improvements last for 6-12 months.

Despite the modest immediate benefits associated with these drugs, some additional benefits may be seen in those receiving long-term treatment. In the Donepezil and Memantine in Moderate to Severe Alzheimer's Disease (a.k.a. DOMINO) study, researchers examined the effects of continuing symptomatic treatment beyond the stage of moderate to severe dementia.

The group that discontinued symptomatic treatment experienced more rapid cognitive decline, worse functional outcomes, and were admitted to care sooner. The trial led to the prevailing consensus that cholinesterase inhibitors shouldn't be stopped just because the point of severe dementia has been reached.

The behavioural and psychological symptoms of dementia include depression, psychosis, agitation, aggression, disturbed sleep, wandering, apathy, and a variety of socially inappropriate behaviours. Together, they precipitate a loss of independence, add to the carer's responsibilities, and result in early placement in nursing care. Non-pharmacological interventions are used to address these problem behaviours.

Neuropsychiatric symptoms of dementia respond modestly to aromatherapy, bright light therapy, music therapy, controlled multisensory stimulation, animal-assisted therapy, physical therapy, occupational therapy, and speech therapy.

Future trends

The Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (a.k.a. FINGER) trial was an initiative to understand the secondary prevention of dementia. Researchers recruited persons without dementia aged 60-77 years but who had an elevated dementia risk score (based on age, sex, education, blood pressure, BMI, cholesterol levels, and physical activity) as well as a minor degree of cognitive impairment.

The trial selected an ultra-high-risk population for dementia and trialled multi-domain interventions, involving changes to nutrition, physical activity, education, and cognitive training. The intervention group's cognitive outcomes improved 25-150% compared to the control group, which only received health advice.

Future studies will aim to demonstrate the benefit of such interventions on the principal public health outcome: time to dementia onset. Despite the large societal gains that may accrue from controlling risk factors, we will still need disease-modifying therapies to reduce the global burden of dementia.

We will also need a cultural transition in the public perception of Alzheimer's and other related dementias, before we can reap the full benefits of preventative and therapeutic strategies. Moving from dementia to a framework of brain health will destigmatise cognitive decline, empower people to take more responsibility towards prevention, and encourage society more broadly to adopt inclusive solutions to maintain functional independence.

Let's reimagine dementia care in terms of brain-health centres rather than in terms of memory clinics.

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