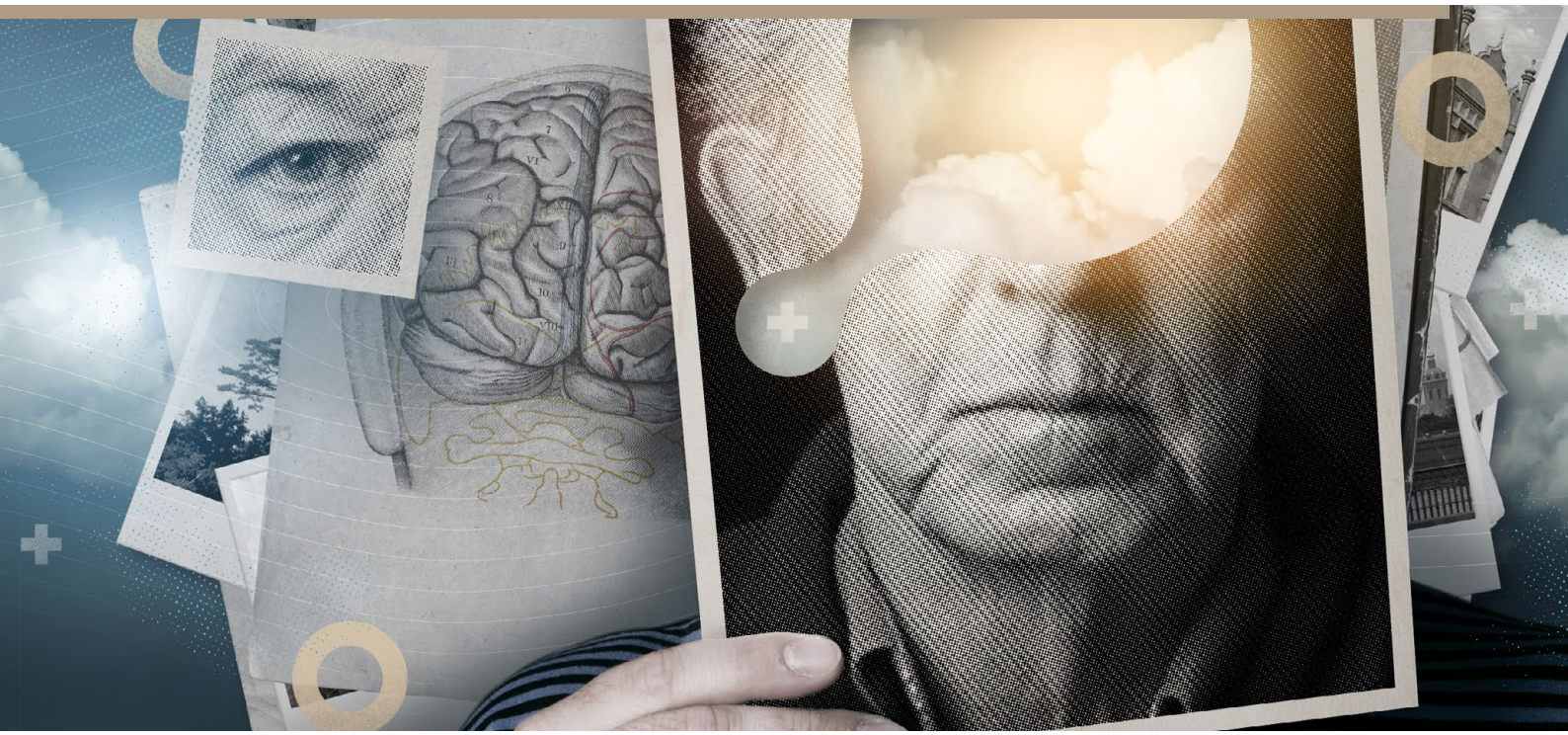


Recognising and managing early dementia

Dementia (mate ware ware – see below) is a growing healthcare challenge. There are an estimated 70,000 people with dementia in New Zealand; this number is predicted to increase to over 170,000 by 2050, due to factors such as population growth and increased longevity.* Without a curative treatment or ability to prevent progression of dementia, the main management goal, as for other terminal conditions, is to help people maintain their quality of life for as long as possible. Early diagnosis enables patients and their family/whānau to access support, information and appropriate symptomatic treatments, and allows time to plan for the future.

* Estimates from the Alzheimers New Zealand/Deloitte Dementia Economic Impact report (2016)

KEY PRACTICE POINTS:

- Discussions about cognitive decline and dementia (mate wareware) can be difficult; patients or their family/whānau may be reluctant to disclose symptoms due to fear, embarrassment, shame or denial
- Symptoms of cognitive decline should be assessed when first reported or noticed. In many cases, reassurance that the symptoms are due to age-related cognitive decline will be appropriate. However, if the symptoms are indicative of a potentially clinically significant change in cognitive function or are affecting the person's activities of daily living, they should be assessed for dementia.
- The initial consultation should focus on the clinical history (obtained from the patient and someone who knows them well), investigations to exclude other causes of cognitive impairment (e.g. medicine adverse effects, delirium, depression) and evaluation with a cognitive assessment tool
- Most patients with dementia can be diagnosed and managed in primary care. Referral to secondary care is appropriate if there is diagnostic or management uncertainty.
- Allow additional time when discussing a dementia diagnosis with a patient and their family/whānau to explain what the diagnosis means, how it was made, the management plan and where to access support
- Strongly encourage early engagement with the local branch of Alzheimers New Zealand or Dementia New Zealand as these organisations are often the main providers of personal support, information, dementia service navigation, "living well" services and programmes such as cognitive stimulation treatment
- There are currently no treatments available that can cure or prevent the progression of the common subtypes of dementia such as Alzheimer's disease or vascular dementia. Non-pharmacological and pharmacological interventions may help to delay or slow the development of cognitive and functional symptoms.

Mate wareware (pronounced "*ma*-te wah-ree wah-ree"), meaning to become forgetful and unwell, was identified as a preferred term in te reo Māori for dementia in a large qualitative study including 223 kaumātua (elders).[1]

Part 1: Making a dementia diagnosis

Dementia is not an inevitable part of ageing and not all changes in cognitive function are indicative of dementia, however, it is important that symptoms are investigated when first reported, e.g. by the patient or a family/whānau member, or noticed, e.g. by the primary care team.[2] Other scenarios where assessment of cognitive function should be considered in older patients include after a fall or other significant medical event, motor vehicle accident or a safety incident at home, e.g. unattended cooking causing a fire. Patients or their family/whānau may be reluctant to discuss the symptoms, e.g. due to fear, shame or denial, however, timely assessment enables primary care to provide reassurance to people who are experiencing normal age-related cognitive decline, prompt treatment to those with reversible causes of cognitive impairment, and earlier diagnosis in those with dementia.

The 15-minute consultation for a person with suspected dementia

The time constraints of primary care consultations can be particularly challenging when assessing a person with suspected dementia and it is likely that multiple consultations will be required when establishing a diagnosis and management plan. The key areas of focus of the initial consultation should be:

- Take a clinical history – establish the type, duration, pattern of symptoms and the impact on daily functioning; clinicians who have cared for patients and their family/whānau over some years will often already have an good understanding of the history and circumstances.
- If dementia is suspected, carry out a brief cognitive test, e.g. using the GPCOG (see: Cognitive testing with the GPCOG"). If impairment is indicated (GPCOG score ≤ 4), consider a follow-up appointment with a practice nurse for a more comprehensive cognitive assessment, e.g. with the Mini-ACE (see: "Comprehensive cognitive assessment"). However, Mini-ACE is much quicker to administer than the MoCA so it may be able to be completed during the first consultation.
- Exclude other causes of cognitive impairment, i.e. cardiovascular or neurological examination, depression screening, arrange appropriate investigations (see: "Consider other explanations for changes in cognitive function")

Investigating the cause of cognitive impairment will usually require at least two consultations: an initial consultation where the patient history and clinical investigations are undertaken and a follow-up where the outcome is discussed.

Initial consultation: take a clinical history and investigate reversible causes

The initial assessment of a person presenting with suspected cognitive decline should focus on the clinical history to establish the:

- Type of symptoms – cognitive, behavioural, psychological and neurological (see: "Distinguishing age-related cognitive impairment from mild cognitive impairment and dementia")
- Duration of symptoms, i.e. when did they start, are they constant or intermittent
- Pattern of symptom onset, i.e. sudden or gradual
- Impact of the symptoms on the person's functioning in their daily life
- Any other information that might suggest an alternative explanation for cognitive impairment, e.g. medicine adverse effects, delirium, depression (see: "Consider other explanations for changes in cognitive function")

If possible, information should also be obtained from someone who knows the person well, e.g. a family/whānau member. A questionnaire, e.g. the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE) or Functional Activities Questionnaire (FAQ), may be used to supplement this discussion (see below for links).[3]

Patient assessment questionnaires for family/whānau are available from:

- IQCODE: **www.alz.org/professionals/health-systems-medical-professionals/clinical-resources/cognitive-assessment-tools**
- FAQ: **www.alz.org/careplanning/downloads/functional-activities-questionnaire.pdf**

Consider other explanations for changes in cognitive function

There are many possible causes of cognitive impairment that must be considered when a person presents with memory loss or other cognitive changes, including:[5]

- Medicines use, e.g. medicines with anticholinergic action or adverse effects, opioids, many psychotropic medicines, steroids
- Psychological causes, e.g. depression, anxiety, stress, psychosis

- Delirium – many potential causes including dehydration, infection, e.g. urinary tract infection or pneumonia, sensory impairment, adverse medicine reaction, immobility, metabolic disturbances, pain, chronic or severe constipation
- Alcohol or drug misuse – may also cause delirium
- Metabolic causes, e.g. vitamin B1 or B12 deficiency, folate deficiency, hyperglycaemic hyperosmolar ketosis, hyperthyroidism, hypothyroidism, hyponatremia, hypercalcaemia, hepatic encephalopathy, uraemia
- Structural brain disease, e.g. subdural haematoma, mass lesion, normal pressure hydrocephalus
- Neurological infections, e.g. HIV or syphilis
- Hearing or vision impairments
- Sleep apnoea – has been associated with white matter abnormalities and impaired cognition, mood and daytime alertness[5, 6]

Also see: "Differentiating dementia, depression or delirium as the cause of cognitive impairment in older people"

Investigations to rule out other potential causes of cognitive decline include:

- Five-minute neurological examination
- Depression screening (see: "Differentiating dementia, depression or delirium as the cause of cognitive impairment in older people")
- Blood tests as appropriate:[2, 7]
  - Full blood count
  - Biochemistry tests – electrolytes, creatinine, corrected calcium, glucose, HbA_{1c}, renal and liver function
  - Thyroid function tests
  - Serum vitamin B12 and folate
  - C-reactive protein (CRP)
  - HIV and syphilis testing
- Mid-stream urinalysis – if a UTI is suspected

If a treatable cause is identified, manage this as appropriate and then reassess the patient's cognitive function. It is possible that a treatable cause may have exacerbated the symptoms of previously unidentified dementia.

Distinguishing age-related cognitive impairment from mild cognitive impairment and dementia

The main distinctions between cognitive impairment due to ageing and cognitive impairment due to dementia are that:

- Age-related cognitive impairment does not affect daily functioning or the ability to live independently
- Age-related cognitive impairments are less severe than those associated with dementia
- Dementia is always progressive

Mild cognitive impairment is a "grey area" between normal age-related memory loss and dementia where there is some impact on the person's daily functioning, but it is not considered significant by the person or the people close to them. Approximately half of people with mild cognitive impairment will progress to a dementia syndrome.

If memory loss is accompanied by other signs of cognitive impairment, this may be suggestive of a more advanced stage of dementia and further investigation is necessary.

Symptoms and signs include:[4]

- Aphasia (impairment in producing and understanding speech)
- Apraxia (difficulty in performing motor tasks)
- Agnosia (inability to recognise familiar people, places and objects)
- Disturbance in executive function (difficulty sequencing, organising, abstracting, planning)
- Change in behaviour or mood (i.e. agitation, apathy, anxiety, disinhibition)
- Physical signs including gait disturbance, extrapyramidal symptoms, focal or lateralising neurological signs

N.B. Some of these symptoms and signs could indicate another neurological diagnosis, particularly if focal neurological signs are present; consider whether referral to secondary care is indicated.

Differentiating dementia, depression or delirium as the cause of cognitive impairment in older people

Delirium and depression share common symptoms with dementia (the "3Ds") and often co-exist in older people. When an older person presents with cognitive impairment, depression and delirium must be ruled out as potential causes before a diagnosis of dementia is made. Table 1 shows some differential features. A key distinguishing feature is that delirium has a sudden onset, whereas dementia onset is insidious. If the clinical history suggests depression, consider using a depression screening tool validated for use in older people, e.g. Geriatric Depression Scale.

For information on identifying and managing depression in older people, see: **www.bpac.org.nz/BPJ/2011/July/contents.aspx**

The geriatric depression scale is automatically selected when using the *bestpractice* decision support Depression module in an older patient. The GDS is also available from: **www.bpac.org.nz/BPJ/2011/July/appendices.aspx**

Table 1. Differentiating features of dementia, depression and delirium.[9]

Feature	Dementia	Depression	Delirium
Timing of onset	Chronic and generally insidious	Variable, may coincide with life events/illness	Usually a sudden change from normal
Duration	Months to years	Weeks to months	Hours to days (less than one month)
Progression	Slow, progressive	Variable, uneven	Rapid, fluctuates. Can be normal at times.
Attention or concentration	Generally normal, or mildly affected	May be impaired	Severely affected; fluctuates, distractible
Psychomotor activity	Normal	Normal or reduced	Agitated, lethargic or swings between both
Sleep	Sometimes disturbed	Unrefreshing, early morning wakening	May be drowsy or alert, often with day/night reversal
Speech	Normal in early stages	May be slowed	Often incoherent, muddled, slow or rapid
Orientation	Usually impaired (unless very mild)	Normal	Disorientated
Thought content	Scarcity of thought, words hard to find	Often themes of hopelessness	Disorganised, incoherent

Consider arranging a head CT

Brain imaging with computed tomography (CT) is recommended when assessing people for dementia to exclude structural cerebral pathologies or potentially reversible conditions, and to assist with subtyping, management planning and as a clinical baseline if a dementia diagnosis is made.[8] If subtyping will not change the management plan or the prognosis, e.g. a person of advanced age with established severe dementia, then a head CT may not be necessary.[8]

N.B. Referral processes, eligibility criteria and access to head CT varies across DHBs; refer to your local HealthPathways or seek advice from a geriatrician or neurologist.

Cognitive screening and assessment: when and how to administer

The history, observation and examination will generally guide the clinician as to whether a formal cognitive assessment is indicated. Cognitive assessments can be used to help confirm and quantify cognitive impairments, and to monitor changes in the patient's cognitive function over time.

Consider a brief test first, followed by a comprehensive cognitive assessment

If cognitive impairment is suspected but you are unsure, a brief cognitive test can be used initially as a screening tool, e.g. the General Practitioner Assessment of Cognition (GPCOG see: "Cognitive testing with the GPCOG"). The GPCOG takes less than five minutes to administer and is validated for use in primary care. If the results suggest impairment or it is already apparent that some impairment is present, a more comprehensive evaluation should be undertaken. The recommended test for use as part of this assessment is the Mini-Addenbrooke's Cognitive Examination (Mini-ACE or M-ACE - see "Comprehensive cognitive assessment"). This test also takes about five minutes to administer and is validated for use. Mini-ACE will be the recommended test on the Cognitive Impairment Pathway on local Community HealthPathways platforms once they are updated on 1 September, 2020.

Consider the potential limitations of cognitive testing

There are limitations to cognitive testing, particularly in relation to potential biases that may arise due to the person's educational level, language or cultural identity. A culturally appropriate cognitive assessment tool for Māori that incorporates knowledge from te ao Māori (Māori worldview) is under development. Another limitation of cognitive testing is that it is possible for a person to score quite well and still have significant cognitive impairment; conversely, a person who functions well can score poorly on a cognitive test, e.g. if they are anxious or have mild dysphasia. Therefore, cognitive assessment tools should not be used in isolation to diagnose dementia.

Types of dementia

Dementia is a syndrome with a variety of causes. The symptoms of dementia and the rate of progression vary with subtype (Table 2). Alzheimer's disease is thought to be the most common subtype of dementia in New Zealand, followed by vascular dementia, but mixed pathology is also common.[11] Most patients with dementia can be diagnosed and managed in primary care.

When to refer to secondary care for diagnosis or management advice

Referral to secondary care is appropriate if there is diagnostic or management uncertainty, e.g. due to complexity (see below for examples), or to access management supports that are not available in primary care.

Complexities in people with suspected dementia where referral to secondary care is appropriate include:[7]

- Severe behavioural or psychological symptoms, including psychotic symptoms
- Rapidly deteriorating cognitive function
- Younger age, e.g. aged < 65 years
- Atypical presentation
- History of a significant head injury
- Chronic neurological disorder, e.g. Parkinson's disease, Huntington's disease, Motor Neurone disease or multiple sclerosis
- Intellectual disability
- Specific deficits, e.g. speech only

Follow-up consultation: discussing the diagnosis

When delivering a dementia diagnosis to the patient and their family/whānau, the discussion should cover:[2, 7]

- That the cognitive problems they have been experiencing are more than just normal ageing. It is important to use the terms dementia or mate wareware, rather than euphemistic descriptions, because this enables the patient and their family access to information, support and services.
- How the diagnosis was made, i.e. by explaining the information obtained from the history, assessment tool(s) and investigations
- General discussion about the course and prognosis of dementia
- Non-pharmacological and pharmacological treatment options for symptomatic management
- Referral for a Needs Assessment, if indicated

Cognitive testing with the GPCOG

The GPCOG has two components: a six-item cognitive assessment conducted with the patient (takes less than five minutes to administer) and an informant questionnaire (not always required).

GPCOG patient examination:[10]

Unless specified, each question should be only asked once

Recall

1. Give the patient a name and address and ask them to repeat it and remember it as you will ask them to recall it again e.g. John Brown, 42 West Street, Kensington.
   (Allow a maximum of four attempts to repeat the address).

Time orientation

2. What is the date? *1 point. Exact only*

Clock drawing (visuospatial functioning) Use a page with printed circle

3. Please mark in all the numbers to indicate the hours of a clock. *1 point. Correct spacing required.*
4. Please mark in hands to show 10 minutes past 11 o'clock. *1 point*
5. Ask the patient to tell you something that happened in the news recently (in the past week) *1 point*

Recall

6. Ask the patient to recall the name and address from Question 1.
   1 point for each of: John, Brown, 42, West Street, Kensington

Score = 9 no cognitive impairment, interview not necessary
Score = 5–8 proceed to informant interview
Score = 0–4 cognitive impairment, interview not necessary

GPCOG informant interview:[10]

Ask the informant: *"Compared to a few years ago":*

1. Does the patient have more trouble remembering things that have happened recently?
2. Does he or she have more trouble recalling conversations a few days later?
3. When speaking, does the patient have more difficulty in finding the right word or tend to use the wrong words more often?
4. Is the patient less able to manage money and financial affairs (e.g. paying bills, budgeting)?
5. Is the patient less able to manage his or her medication independently?
6. Does the patient need more assistance with transport (either private or public)?

Score one point for each "no" answer

Score = 4–6 no cognitive impairment
Score = 0–3 cognitive impairment detected

The original version of the GPCOG is available from: **www.patient.info/doctor/general-practitioner-assessment-of-cognition-gpcog-score**. For a PDF version, see: **www.comprehensivecare.co.nz/wp-content/uploads/2013/01/GPCog-.pdf**

- How to access information and support from their local dementia organisation; either Alzheimers New Zealand or Dementia New Zealand, depending on location
- Medico-legal issues to consider, e.g. driving, appointing an Enduring Power of Attorney, developing or updating an advance care plan, preparing a will. N.B. These discussions are likely to be ongoing and may not be considered in detail, if at all, at the first follow up appointment.

Allow additional time for this type of consultation. The pace should be guided by the patient and their support people, it is often not possible or advisable to cover everything in one consultation. Provide written information that the patient and their family can take home with them. Alzheimers New Zealand has "About dementia", "Living well with dementia" and "Supporting a person with dementia" booklets, available from: **www.alzheimers.org.nz/information-and-support/information/booklets-and-fact-sheets**. Information can also be accessed from Dementia New Zealand: **www.dementia.nz**

Acknowledge the impact of a dementia diagnosis

A diagnosis of dementia can have a significant impact on a person's self-esteem, relationships, employment and future plans. When learning about the diagnosis, the patient and their family/whānau may experience a range of feelings including shock, disbelief, anger, fear, hopelessness, despair and grief.[14] Some may also feel relief at having an explanation for the changes that have been occurring. Empathy, understanding and sensitivity toward the person and their family/whānau are imperative when discussing the diagnosis and prognosis for a person with dementia. Although the realities of dementia must be discussed, this needs to be balanced by encouraging the person and their family/whānau to focus on what they can do and to keep actively engaged in life.

Diagnosing dementia early allows people to make decisions about their future care and to access support. It also allows for early engagement with interventions that may help to preserve their quality of life for as long as possible. For carers, early diagnosis allows more time for them to adjust to the patient's changes in function, mood and personality, and to transition into their caregiving role.[14] Ensuring that carers have access to support and maintain their own health and wellbeing is a core component of dementia care.

Carers for a person with dementia can access support through the Supporting Families organisation, see: **www.supportingfamilies.org.nz**

Perspectives from patients who are living with dementia and their carers are available from: **cdn.alzheimers.org.nz/wp-content/uploads/2021/05/Report-This-is-our-story-1.pdf**

Comprehensive cognitive assessment

Mini-Addenbrooke's Cognitive Examination (Mini-ACE or M-ACE) will now be the recommended test for assessing cognitive function in New Zealand. Until recently, the Montreal cognitive assessment (MoCA) was the most frequently used cognitive screening tool in primary care in New Zealand and the preferred cognitive assessment tool listed by HealthPathways. However, from 1 September, 2020, the MoCA test will no longer be freely available and in New Zealand the recommended test will now be the Mini-ACE. The MoCA test can continue to be used if clinicians have paid for training and certification through the MoCA Institute.

For further information on the Mini-ACE, see: **www.nzdementia.org/mini-ace**. This website includes information on:

- How to download the New Zealand Mini-ACE test, and an administration and scoring guide
- Complete online training
- The full report from the Cognitive Impairment Assessment Review (CIAR) Working Group on how the recommendation was made
- Some alternative language versions (although these are not yet adapted for New Zealand)

N.B. A new Māori Assessment of Neuropsychological Abilities (MANA) tool is being developed and the aim is for this to be integrated into HealthPathways alongside the Mini-ACE in 2022. Currently there is not a te reo Māori version of Mini-ACE.

Rowland Universal Dementia Assessment Scale (RUDAS). The RUDAS is designed to minimise the effects of cultural learning and language diversity of cognitive performance. The test takes approximately 20 minutes and a score of ≤ 22 out of 30 is indicative of cognitive impairment.

For further information on the RUDAS and to access the test, see: **www.dementia.org.au/resources/rowland-universal-dementia-assessment-scale-rudas**

Table 2. Common types of dementia and the distinguishing symptoms and signs[12,13]

Dementia subtype	Symptoms and signs	Progression/prognosis
Alzheimer's disease	■ Short-term memory loss and difficulty finding words, followed by behavioural changes and impaired functioning, with reduced insight ■ Typically gradual onset. It can be difficult to determine exactly when symptoms began. May appear to be sudden onset if a triggering event, e.g. illness or stress, uncovers the underlying decline.	■ Progression is typically slow, i.e. changes occur over several years ■ Life expectancy is an average of eight to ten years following symptom onset, although this varies depending on the patient's age and other risk factors
Vascular dementia	■ Wide range of symptoms and signs depending on the extent, location and severity of cerebrovascular disease ■ Sudden onset of symptoms after a stroke or more insidious onset with small vessel disease ■ Memory loss can be a symptom but typically less noticeable than in Alzheimer's dementia ■ Deficits may be found in language, decision-making and information and visuospatial processing ■ Mood changes and apathy are common symptoms ■ Can be co-present with Alzheimer's disease (referred to as 'mixed dementia')	■ Progression depends on the type of disease: small vessel disease progresses slowly, like Alzheimer's disease, while vascular dementia following a stroke ("multi-infarct dementia") often follows a stepped progression with long periods of remaining the same and short periods where symptoms worsen ■ Life expectancy is approximately five years after symptom onset, however, there is wide variability. Often the cause of death is stroke or myocardial infarction.
Frontotemporal dementia (FTD)	■ Younger age of onset (e.g. age 50–60 years) ■ Symptoms include changes in personality and behaviour, may include disinhibition and impulsiveness. Memory is usually intact early in the disease. ■ Diagnosis can be easily missed; may require more specialised tests of social awareness or behaviour	■ Rate of progression can vary greatly ■ Life expectancy is an average six to eight years after symptom onset
Dementia with Lewy Bodies or Parkinson's disease with dementia	■ Suspect if dementia with marked fluctuations during the day, parkinsonian features, visual hallucinations, autonomic symptoms, e.g. postural hypotension, incontinence, sexual dysfunction, or falls ■ Tremor may be less evident in people with dementia with Lewy Bodies than with Parkinson's disease ■ Sleep disturbances, e.g. rapid eye movement sleep behaviour disorder (shouting out or moving while asleep), can occur many years before the onset of dementia ■ Up to 80% of people with Parkinson's disease will develop dementia. Symptoms are similar to dementia with Lewy bodies, but the motor symptoms occur before the cognitive symptoms by definition.	■ Dementia with Lewy bodies typically develops and progresses slowly, like Alzheimer's disease ■ In Parkinson's dementia, motor symptoms typically develop a year or more before the cognitive and psychiatric symptoms. ■ Life expectancy of a person with dementia with Lewy Bodies is an average of 6–12 years from symptom onset

Other causes or sub-types of dementia include:[13]

- Alcohol-related dementia
- Posterior cortical atrophy (an Alzheimer's disease variant)
- Creutzfeldt-Jakob disease
- HIV-related cognitive impairment
- Huntington's chorea
- "Parkinson's Plus" dementias, e.g. corticobasal syndrome, progressive supranuclear palsy
- Multiple sclerosis
- Niemann-Pick disease type C
- Normal pressure hydrocephalus
- Tertiary syphilis

Providing dementia care for Māori

Caring for Māori patients with mate wareware (dementia) and their whānau requires knowledge, respect and engagement with their beliefs, values and practices. The concepts of wairua (spirituality) and whānau (family) in particular are central to Māori understanding and experience of mate wareware.[1]

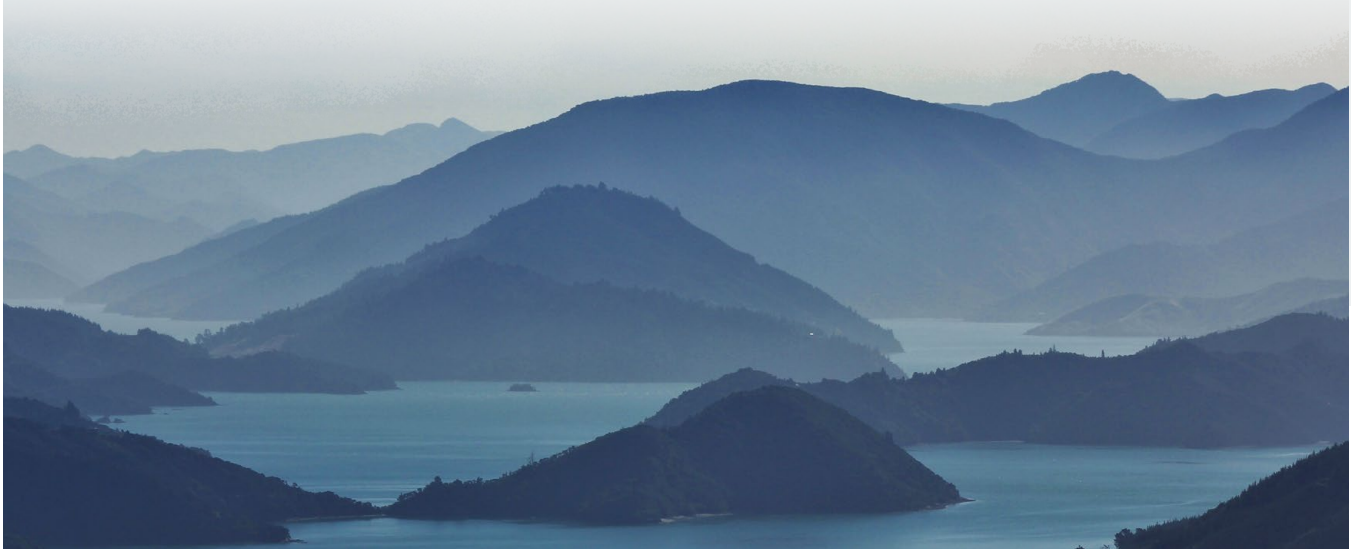
Further discussion about Māori understanding of mate wareware is available from: **www.nzma.org.nz/issue-id/vol-132-no-1503-4-october-2019**

The Goodfellow Unit has developed a free online course for primary healthcare professionals on providing dementia care for Māori. Key points from the course have been summarised below; the full course is available from: **www.goodfellowunit.org/group/111**

Key considerations when caring for Māori patients with dementia and their whānau include:

- Māori may try to hide or diminish symptoms of cognitive impairment, e.g. memory problems, to maintain their mana (prestige, respect, authority). Whānau are often more willing to share information and should be included in the consultations as appropriate.
- The symptoms of cognitive impairment may be described in nuanced ways, e.g. whānau may express that their loved one can still recite the entire whakapapa (genealogy) of their whānau, but struggle to recall other things that should be remembered, e.g. what they did earlier that day. It is important to understand the significance of whakapapa, that it represents deep, long-term knowledge that persists the longest.
- Establish clear and authentic lines of communication when working with whānau
- Ensure sufficient time and space to build relationships and gather information from the patient and the whānau. Make sure the patient knows they can bring as many whānau members with them as they wish and take time at the beginning of a consultation to connect with the patient and their whānau.
- Consider the roles that the patient has within their whānau and what effect it will have on the whānau when they can no longer perform those roles
- Engage with patients around their beliefs in relation to traditional Māori healing practices, and to understand how those beliefs may be interacting with proposed plans of care
- Some Māori living with dementia may be able to engage more in te reo Maori if this is their first language. Introducing te reo Māori into the consultation could improve engagement with patients.
- Whānau play a role in maintaining the wairua (spirituality) of their loved one through waiata and karakia once a person with dementia loses the ability to consciously maintain their wairua themselves. Some whānau may not be connected to tikanga Māori, but whānau can also utilise other means to create a collective spirit around their loved one, which strengthens and maintains wairua.

For further information on Māori health and wellbeing: **www.goodfellowunit.org/group/204**



Part 2: Managing early-stage dementia

Management should focus on slowing symptom development and maintaining quality of life

There are currently no interventions that can cure or prevent the progression of the more common causes of dementia, such as Alzheimer's disease or vascular dementia. Therefore, the aim of dementia management is to slow the rate of symptom development and help the person maintain their best quality of life (also see: "Providing dementia care for Māori"). Key areas of focus for primary care should include:[2]

- Management of co-morbidities and reducing cardiovascular disease (CVD) risk, e.g. stopping smoking, limiting alcohol intake, managing hypertension, diabetes and obesity, optimising diet (e.g. recommend the Mediterranean diet or Dietary Approaches to Stop Hypertension [DASH] diet [see link below]). Reducing CVD risk is particularly important in people with vascular dementia.
- Ensuring annual vision and hearing checks, and timely management of sensory impairment; enlist the help of a partner or family/whānau member if they cannot arrange an appointment themselves
- Assessing nutrition and hydration (e.g. using the Mini Nutritional Assessment **www.mna-elderly.com**). People with dementia are at risk for undernutrition (either generalised protein-energy malnutrition or specific micronutrient deficiency, especially B12 and folate) due to problems with meal planning, shopping, preparing food and eating regularly.
- Medicine reconciliation, i.e. reviewing medicines regimens and adjusting or stopping treatment as appropriate
- Risk assessment, e.g. safety while cooking, using electrical appliances, heavy machinery or firearms, driving, falls
- Reviewing how the patient is managing at home. Recommend strategies to help the patient manage memory loss (see link below). Refer for a Needs Assessment if there is significant carer stress or the patient needs support to remain living independently.
- Monitoring mental health, stress and coping – both the patient and their caregiver

Ideally, people with dementia, accompanied by their family/whānau or carer, should be reviewed every three to six months to monitor the management plan and address any concerns.

For further information on lifestyle strategies to slow cognitive decline, see: **www.bpac.org.nz/2020/cognitive.aspx**

For practical tips on how to manage memory loss symptoms, see: **www.alzheimers.org.nz/information-and-support/support/living-well-with-dementia/managing-your-symptoms**

Non-pharmacological interventions

Physical activity

People with dementia should be encouraged to engage in physical exercise both for their general health and wellbeing and as a way of slowing cognitive decline; this should ideally include a mix of aerobic and muscle strengthening exercises. Aerobic exercise, e.g. brisk walking, running, cycling, dancing, aerobics, swimming or aqua jogging, has been shown to be most beneficial in terms of cognitive functioning.[15, 16] A meta-analysis of 18 Randomised Control Trials (RCTs) including 802 people with dementia found that exercise interventions that included aerobic exercise (walking, running, cycling or dancing) improved cognition, independently of intervention frequency (i.e. <150 or >150 minutes per week).[16] Although the same cognitive benefits were not found with non-aerobic exercise interventions (Tai Chi, strength, balance or flexibility training), these exercises should still be recommended for falls prevention.[16]

Psychosocial stimulation

Cognitive Stimulation Therapy (CST) is a group or individual talking-based intervention recommended for people with mild to moderate dementia.[3] CST uses reminiscence (discussing past activities and events), stimulation and reality orientation (understanding the present using visual prompts) tasks, and focuses on opinions and discussions to stimulate language, thoughts and associations.[17] A meta-analysis of 14 RCTs including 657 people with dementia found that CST significantly improved cognitive function, communication and social interaction, self-reported quality of life and wellbeing.[18] A 2015 pilot of group CST in people with dementia in Auckland found that CST (14 sessions) reduced symptoms of depression, improved quality of life (reported by families and caregivers but not the patients themselves) and showed a trend towards an improvement in memory.[19]

Availability of CST around New Zealand is variable; check with the local Alzheimers New Zealand or Dementia New Zealand branch to see if a programme is available in your area.

Remaining mentally and socially active is important. All people with dementia should be encouraged to engage in cognitively and socially stimulating activities that are tailored to suit their interests and abilities, e.g. reading, quizzes, crosswords, sudoku, playing cards or board games, learning something new, playing music, dancing or cultural activities. For Māori, cultural activities and utilising te reo Māori are considered protective factors that optimise a person's functioning within their whānau and community.[1] Encourage Māori patients to continue with their roles within the whānau and on the marae, where possible.[1]

Information for patients about staying active and engaged following a dementia diagnosis is available from: **www.alzheimers.org.nz/information-and-support/support/living-well-with-dementia/staying-involved-and-active**

Pharmacological interventions: the role of dementia medicines

Acetylcholinesterase inhibitors

An acetylcholinesterase inhibitor such as donepezil (oral, funded), rivastigmine (transdermal patches funded with Special Authority approval – see: "Rivastigmine patch brand change", oral not funded) or galantamine (oral, not funded) may be considered in people with Alzheimer's-type dementia, vascular dementia where subcortical ischaemic changes are prominent and dementia associated with Parkinson's disease/Dementia with Lewy Bodies (unapproved indication). Acetylcholinesterase inhibitors should not be prescribed to people with mild cognitive impairment.[2]

The treatment effects of acetylcholinesterase inhibitors are generally modest; not all patients will respond to treatment and it is not possible to predict response. There is no evidence that acetylcholinesterase inhibitors prevent the progression of dementia, however, some people may have a temporary improvement in cognition and functionality. A meta-analysis of 43 RCTs including over 16,000 people with Alzheimer's disease reported that acetylcholinesterase inhibitor treatment resulted in small to moderate improvements in cognitive function, global symptoms and functional capacity.[20] Data on neuropsychiatric symptoms are mixed, but suggests that there may be benefits for some symptoms (such as apathy and psychosis) but not others (such as anxiety and aggression).[20]

Cautions to acetylcholinesterase inhibitor use include sick sinus syndrome or other supraventricular conduction abnormalities, e.g. atrioventricular or sinoatrial block, due to an increased risk of bradycardia. Perform an ECG in all patients prior to initiating treatment to check for pre-existing conduction abnormalities.[21, 22]

Rivastigmine patch brand change

The funded brand of rivastigmine patches is changing from Exelon to Generic Partners. From 1 February, 2020, the Generic Partners patches are available fully funded alongside the Exelon patches. From 1 April, 2020, the subsidy on the Exelon patches will reduce and they will no longer be funded from 1 July, 2020.

Key points to discuss with patients about the brand change:

- Generic Partners patches have the same active ingredient (rivastigmine) as other brands
- The new brand is just as safe and works in the same way as the old brand
- The Generic Partners patches are clear, whereas the Exelon patches are beige. This will not change how the medicine works.

For further information see: **www.pharmac.govt.nz**

Acetylcholinesterase inhibitors can cause dose-related cholinergic effects, e.g. nausea, bradycardia, vomiting, diarrhoea, dyspepsia, urinary incontinence, dizziness.[21] Treatment should be initiated at a low dose and titrated upwards, if tolerated. Patients who have intolerable nausea or vomiting with donepezil tablets can be prescribed transdermal rivastigmine patches.

N.B. The acetylcholinesterase inhibitors available have similar effectiveness; most people will be initiated on donepezil as it is fully funded. If donepezil is not effective, rivastigmine or galantamine may be trialled, taking into consideration the affordability of non-funded treatments. Only one acetylcholinesterase inhibitor should be used at a time.

Treatment effectiveness, adverse effects, adherence and symptom progression should be assessed one month after initiating a acetylcholinesterase inhibitor, and again at three months and six months, if treatment has been tolerated.[23] Family and caregivers are well-placed to observe treatment response and adverse effects, however, it is recommended that an objective measure, e.g. Mini-ACE, is also used to monitor treatment effectiveness.[23] There is limited guidance on the recommended duration of acetylcholinesterase inhibitor treatment. If the patient experiences significant adverse effects, has poor adherence to treatment or monitoring requirements, is no longer showing benefit from treatment or has not benefitted from treatment, the medicine should be discontinued.[23] It is recommended that the dose is stepped down over two to three weeks rather than stopping abruptly, and if there is significant decline, re-start the medicine promptly.[23]

Refer to the NZF for further information on cautions, dosage, switching from oral to transdermal formulations and monitoring of acetylcholinesterase inhibitor treatment: **www.nzf.org.nz/nzf_2879**

Memantine

Memantine (not funded) may be considered for people with moderate Alzheimer's disease who are intolerant of or have a contraindication to an acetylcholinesterase inhibitor, or who have severe Alzheimer's disease.[2, 3] Memantine may also be used in combination with an acetylcholinesterase inhibitor by people with moderate to severe Alzheimer's disease.[2, 3] As with acetylcholinesterase inhibitors, the effects of memantine are variable; some may experience benefit, e.g. improved function or slowed rate of decline, while others will not. Memantine is contraindicated in people with a history of seizures and should be avoided in people with severe hepatic or renal impairment.[21] Common adverse effects include constipation, hypertension, dyspnoea, headache, dizziness and drowsiness.[21]

Refer to the NZF for further information on cautions and dosage: **www.nzf.org.nz/nzf_2879**

Further Resources

Future articles will cover the management of behavioural and psychological symptoms as dementia progresses and the role of primary care in supporting patients and their families during the palliative phase of dementia care.

Dementia resources for primary care health professionals are available here:

- A free online course on dementia diagnosis and management from the Goodfellow Unit: **www.goodfellowunit.org/group/111**
- A series of presentations on managing the different stages of dementia from PHARMAC: **www.youtube.com/watch?v=vKYIEhHRijU**
- A Goodfellow Unit podcast with Professor Ngaire Kerse on living well with dementia: **www.goodfellowunit.org/podcast/living-well-dementia**

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