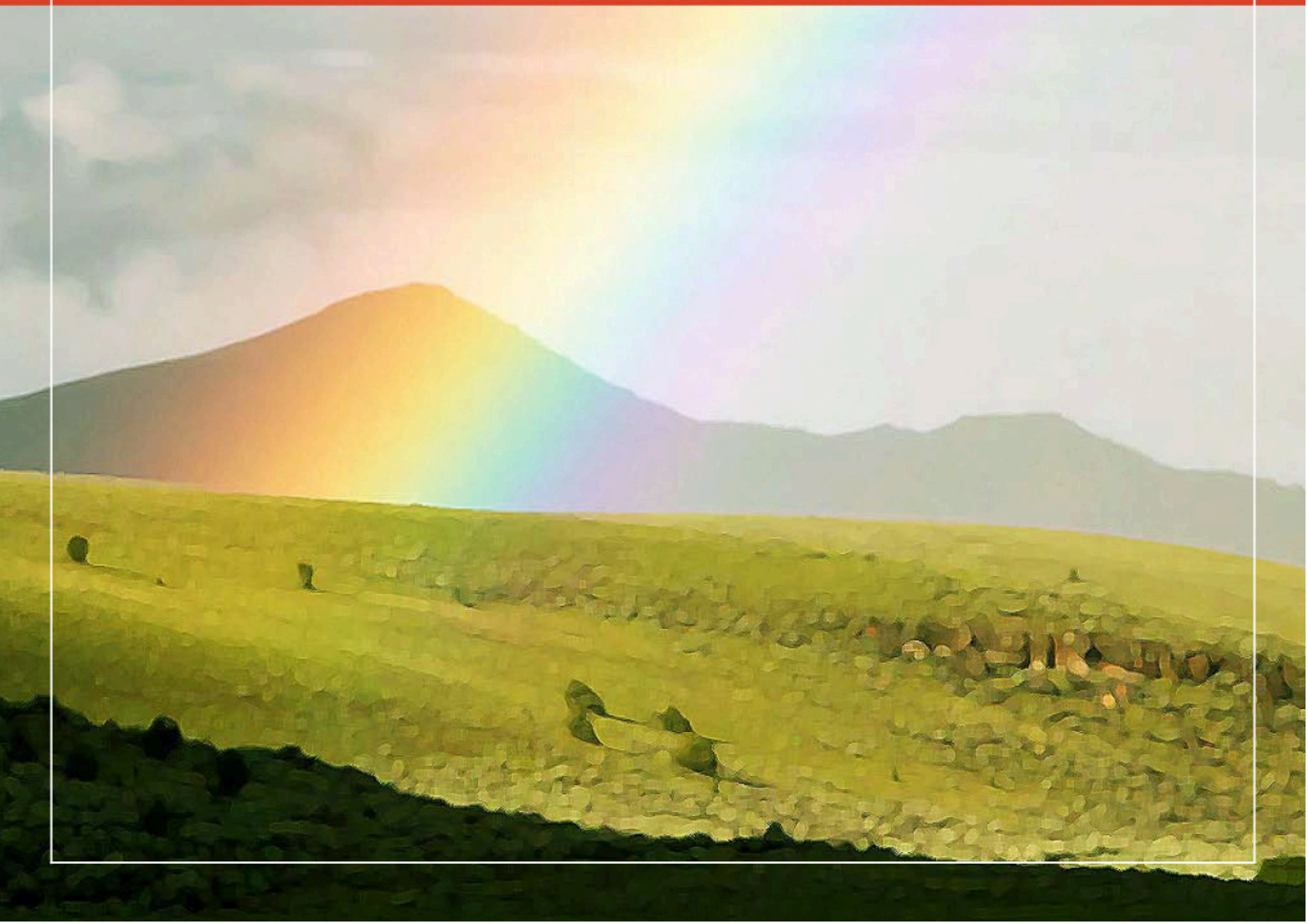
College of Family Physicians Singapore

The Singapore Family Physician

Permit No. MICA(P):163/01/2011

Vol 37(3)(Supplement 1)July–September 2011

Dementia



CONTENTS

Permit No. MICA (P) 163/01/2011
JOURNAL OF THE SINGAPORE FAMILY PHYSICIAN
Printed by Tri-Art Printers Pte Ltd

College of Family Physicians Singapore
23rd Council (2011-2013)

President	A/Prof Lee Kheng Hock
Vice President	Dr Tan See Leng
Censor-in-Chief	A/Prof Tan Boon Yeow
Honorary Secretary	Dr Pang Sze Kang Jonathan
Honorary Treasurer	Dr Tan Tze Lee
Honorary Editor	Adj Asst Prof (Dr) Tan Ngiap Chuan
Assistant Honorary Secretary	Dr Leong Choon Kit
Assistant Honorary Treasurer	Dr Rukshini Puvanendran
Council Members	Dr Ang Choon Kiat Alvin
	Dr Chng Shih Kiat
	Dr Koh Choon Huat Gerald
	Dr Lim Fong Seng
	Dr Siew Chee Weng
	Dr Tham Tat Yean
	Dr Eu Tieng Juoh Wilson
	Dr Yee Jenn Jet Michael

College Secretariat

Executive Director	Dr Pang Sze Kang Jonathan
Curriculum Director	Dr Tan Lip Pin
Manager	Ms Jennifer Lau
Corporate Communications Executive	Ms Stella Teh
Programme and IT Executive	Ms Ariel Heng
Programmes and Training Executive	Mr Ang Chee Yen
Administrative Assistant	Ms Anica Goh
Administrative & Accounts Assistant	Mr Ryan Tan

DEMENTIA UPDATE 2011

Adj Asst Prof (Dr) Tan Ngiap Chuan

SFP2011; 37(3) (Supp 1) : 3

Dementia represents a late stage of disease along a continuum of cognitive impairment. With a fast aging population in Singapore, the number of local patients with established dementia is expected to rise to 53 000 by 2020. Dementia is largely related to neurodegenerative diseases such as Alzheimer's Disease (AD) and vascular dementia (VD). Increasingly AD and VD are noted to occur concurrently. Attempting to identify the dementia aetiology is to seek out potentially reversible causes of dementia and to pick the appropriate treatment for irreversible dementia. Reducing the vascular risk factors such as optimizing diabetic and blood pressure control tend to retard the amyloid cascade and stabilize the cognitive function of patients with vascular cognitive impairment. It is also clinically important to screen for other reversible causes such as vitamin B12 or Folate deficiency, which can be rectified.

Evidences show that the pathological changes in the brain begin many years prior to the clinical display of dementia. The challenge for physicians is to identify these early changes of disease, which have been described as "mild cognitive impairment (MCI) and cognitively impaired not demented (CIND). Studies showed that patients with MCI deteriorated to dementia by about 12% annually and 80% at six years of follow up. Distinction between these two conditions can be difficult as they represent a continuum of the disease and is a subject of intense research.

Evaluation begins with physician taking history of the pattern of cognitive decline, progression, significant alcohol intake and medication use. Physical examination should target at detecting neurological deficits, extra-pyramidal signs, movement disorders and gait abnormalities. It is important to distinguish dementia from delirium, which can be achieved using the Confusion Assessment Method (CAM) with its high degree of sensitivity and specificity.

Functional assessment is carried out via self-reporting or through feedback from family members or caregivers. The subjective approach aims to detect memory impairment and deficits in one other cognitive domain (aphasia, apraxia, agnosia and executive dysfunctioning. The objective approach uses performance-based instruments such as mental status tests. Locally validated tools include Elderly Cognitive Assessment Questionnaire (ECAQ), Abbreviated Mental Test (AMT) and the Chinese Mini Mental Status Examination (CMMSE). The cut-off scores serve as a screening instrument for dementia but can be confounded by language barriers, advanced age and low education. Functional difficulties can be assessed at three levels: community functioning, home functioning and self-care.

Combining both subjective and objective methods appears to be the way ahead. Even if this fails and diagnosis is inconclusive, neuropsychological tests by clinical psychologists is another option.

Neuroimaging (such as CT or MRI brain, PET scans) are helpful in the differential diagnoses of dementia and to exclude structural lesions such as cerebral infarcts and subdural hematoma. Whether it is required for all patients with dementia remains a subject of debate.

Management of dementia is multi-faceted. Behavioural and psychological symptoms of dementia (BPSD) are common. Non-pharmacological interventions are usually first line management for mild to moderate symptoms.

Pharmacological treatment aims to reverse or stabilize the underlying disease by addressing the reversible causes or risk factors, improve cognitive symptomatology and treat BPSD. Acetyl cholinesterase inhibitors (ChEI) and N-methyl D-aspartate (NMDA) are primarily symptomatic in their mode of action. Cochrane review has shown that Gingko biloba was not effective in preventing dementia. Anti-psychotic, anti-depressants, anti-convulsants and benzodia-zepines are other groups of drugs to manage BPSD. Cost and side effects are important considerations with the use of medications, including long term use of ChEI and NMDA; the choices depend on underlying aetiology and stages of dementia severity. The CMMSE, AMT and ECAQ can be used to monitor the benefits of these symptomatic treatments.

Caregivers assume critical roles in the management of patients with dementia. Support for caregivers not only reduces caregiver depression, lessen their burden of care, improve their health and quality of life (QOL), it also impacts on patients' care, improve their QOL, medication compliance and decreases rates of institutionalization. Most local caregivers of dementia patients are women, middle-aged and mostly children or spouses of the patients. Many families also engage foreign domestic helpers, who facilitate the physical caregiving whilst family members make decisions on medical care and provide financial support.

Age, gender, healthcare status, kin relationship and ethnic background are factors that influence caregiver performance. As dementia progresses, caregivers experience increasing suffering compared to the patients; they encounter different

TAN NGIAP CHUAN, Honorary Editor, Singapore Family Physician

genres of problems at different stages of the disease. Stressors can impact on the caregivers' emotional well-being, physical health, employment status, income and financial security

Family Physicians (FP) too, play pivotal roles in the comprehensive management of dementia. Caregiver's positive experience with their FP is the first key step in the holistic care of these patients. In the early stage of dementia, FP can explain the diagnosis, educate the families/caregivers on the course of the disease over time, suggest adaptation measures, advise financial, legal planning and advance medical directives (in relation to Mental Capacity Act), and set up of a support system for the caregiver.

During the middle stage characterized by increasing BPSD, FP can screen for caregiver stress, mode change, frustration and burnout and identify resources to help them cope with the increased burden of care. In the late stage, FP can support caregivers to manage end of life issues, facilitates referral, coordinate care with other community healthcare providers and handle bereavement.

FP should be aware of the inclusion of dementia under the Chronic Disease Management Programme, which offers the caregivers another option to financially support drug therapy and long term care of the patients with dementia. Working towards achieving positive experience with patients and their caregivers should be the common goal of all FP in the management of dementia in the community.

Distance Learning Course on "Dementia"

- Overview of Family Practice Skills Course on Dementia
- Unit 1 : Overview & Diagnosis of Dementia
- Unit 2 : Behavioural and Psychological Symptoms of Dementia
- Unit 3 : Pharmacological Treatment of Dementia
- Unit 4 : Family Caregivers and Caregiving in Dementia
- Unit 5 : Chronic Disease Management Programme on Dementia
- Unit 6 : User Information for e-Service Clinical Data Submission

OVERVIEW OF FAMILY PRACTICE SKILLS COURSE ON DEMENTIA

A/Prof Goh Lee Gan

SFP2011; 37(3) (Supp 1) : 6-7

INTRODUCTION

This dementia skills course serves to update primary care medical practitioners on the early diagnosis of dementia and management. Dementia has been included as one of the diseases in the chronic diseases management programme (CDMP) that is eligible for use of the Medisave of the patient and immediate family members. The perspective of dementia nowadays is to regard it as a chronic disease rather than a palliative problem. Viewed this way, there is stage-specific management strategies and there is incentive to diagnose the condition early in order to improve its outlook. Thanks are due to the Ministry of Health for sponsoring this family practice skills course. This training course for Family Physicians is organised as part of the CDMP initiatives. Do mark this course on your calendar.

COURSE OUTLINE AND CME POINTS

This Family Practice Skills Course is made up of the following components. You can choose to participate in one or more parts of it. The CME points that will be awarded are also indicated below.

Components and CME Points

- Distance Learning Course – 6 units (6 Core FM CME points upon attaining a minimum pass grade of 60% in Distance Learning Online MCQ Assessment).
- 2 Seminars (2 Core FM CME points per seminar).
- 2 Workshops (2 Core FM CME points for workshops).
- 10 Readings – read 5 out of 10 recommended journals (maximum of 5 CME points for the whole CME year).

Distance Learning Course

Unit 1 : Overview & Diagnosis of Dementia
Dr Nagaendran Kandiah, Dr Chong Mei Sian

Unit 2 : Behavioural and Psychological Symptoms of Dementia
Dr Ng Li-Ling

Unit 3 : Pharmacological Treatment of Dementia
Dr Lim Wee Shiong

Unit 4 : Family Caregivers and Caregiving in Dementia
Dr Dennis Seow, Dr Philip Yap Lin Kiat

Unit 5 : Chronic Disease Management Programme (CDMP) on Dementia
Dr Chong Mei Sian

Unit 6 : User Information for e-Service Clinical Data Submission Extracted from CDMP Handbook for Healthcare Professionals 2011

COURSE TOPIC DETAILS

Unit 1: Overview & Diagnosis of Dementia

- Introduction.
- Epidemiology.
- Etiology & risk factors.
- Mild cognitive impairment.
- Assessment.
- Investigatsions.
- Management.
- Conclusions.

Unit 2: Behavioural and Psychological Symptoms of Dementia

- Introduction.
- Definition.
- Assessment.
- Management.

Unit 3: Pharmacological Treatment of Dementia

- Introduction.
- Overview.
- Cholinesterase inhibitors.
- NDMA antagonists.
- Common issues in the use of dementia-specific drugs.
- New frontiers in dementia management.

Unit 4: Family Caregivers and Caregiving in Dementia

- Introduction.
- Caregivers.
- Caregivers' experiences with GPs.
- Optimal care and the health care triad.
- Management and support of caregiver.
- Role of the GP in caregiving for the caregiver.
- Conclusions.

Unit 5: Chronic Disease Management Programme (CDMP) on Dementia

- Update on use of Medisave for CDMP.
- Inclusion of Dementia into CDMP.
- Treatment algorithm for dementia.

GOH LEE GAN, Associate Professor, Division of Family Medicine, University Medicine Cluster, National University Health System
Senior Consultant, Institute of Family Medicine, College of Family Physicians Singapore

- Essential care components for dementia follow-up management in Dementia Disease Management Programme.
- Patient education and monitoring.
- Guidelines for continuing care.
- Recommended investigations, drugs and therapies.

Unit 6: User Information for e-Service Clinical Data Submission

- System requirements.
- Getting started.
- Clinical Indicators report submission.
- Searching clinical indicator reports.
- FAQs.
- Summary of Medisave for CDMP.

FACE-TO-FACE SESSIONS

Seminar 1: 17 September 2011, 2.00pm – 4.00pm

Unit 1 : Overview & Diagnosis of Dementia
Dr Nagaendran Kandiah, Dr Chong Mei Sian

Unit 2 : Behavioural and Psychological Symptoms of Dementia
Dr Ng Li-Ling

Workshop 1: 17 September 2011, 4.20pm – 6.20pm

Workshop A - Cognitive and Functional Assessments
Dr Mark Chan, Dr Nagaendran Kandiah

Workshop B - Non-pharmacological Management of Behaviours
Dr Ng Li-Ling, Dr Aaron Ang

Seminar 2: 18 September 2011, 2.00pm – 4.00pm

Unit 3 : Pharmacological Treatment of Dementia
Dr Lim Wee Shiong

Unit 4 : Family Caregivers and Caregiving in Dementia
Dr Philip Yap Lin Kiat

Unit 5 : Chronic Disease Management Programme (CDMP) on Dementia
Dr Chong Mei Sian

Workshop 2: 18 September 2011, 4.20pm – 5.20pm

Chronic Disease Management Programme (CDMP) for Dementia
Dr Chong Mei Sian

UNIT NO. 1

OVERVIEW & DIAGNOSIS OF DEMENTIA

Dr Nagaendran Kandiah, Dr Chong Mei Sian

ABSTRACT

Dementia represents a late stage of disease along the continuum of cognitive difficulties and hence clinicians should aim to identify the prodromal stages of dementia in evaluating the individual who presents to the clinic with cognitive complaints such as forgetfulness or confusion. The prevalence of dementia is on a rising trend with the rapidly ageing population in Singapore. Early diagnosis of dementia is important as early therapeutic interventions may palliate substantially, if not reverse, the significant emotional and economic costs of the illness. A thorough history, cognitive evaluation along with suitable investigational studies is necessary for early diagnosis. The ability to diagnose dementia at the earliest stages has been greatly improved with the use of biomarkers such as medial temporal atrophy on MR imaging and cerebrospinal fluid beta amyloid levels. A 4-step approach to dementia evaluation, incorporating local data, where possible can be used: The first step requires the exclusion of delirium as the cause of the forgetfulness or confusion; the second step involves establishing the diagnosis of dementia; the third step assesses for the behavioural, functional and social problems associated with dementia; and the final step, with the use of a focused history, physical examination, investigations and selected use of neuroimaging, attempts to establish the aetiological diagnosis of the dementia. The management of dementia requires a multidisciplinary approach. While acetyl cholinesterase inhibitors can slow cognitive deterioration, research for newer disease modifying drugs which target the underlying pathology is ongoing.

Keywords: Integrated care; Elderly; Chronic conditions

SFP2011; 37(3) (Supp 1) : 8-14

INTRODUCTION

Dementia is a brain disorder that affects millions of people, mostly older adults. Dementia should be viewed as a "late stage" in the continuum of cognitive difficulties and hence clinicians should aim to identify the earliest stages of dementia.

With the rising trend in the prevalence of dementia, especially with Singapore's rapidly greying population, this is an area of intense research in the area of therapeutics as well as the diagnosis of dementia in the earlier stages, even in the preclinical dementia state as with early diagnosis, because the affected patients are then more amendable to benefit from treatment advances.

Dementia is now included as a disease in the chronic disease management programme where Medisave can be used to pay primary care visits.

The diagnosis of dementia requires the presence of dysfunction in memory and other cognitive domains which are progressive, resulting in a decreased level of function[1]. At the stage of dementia the pathological changes in the brain are often well established and profound. Alzheimer's disease (AD) is the most common cause of dementia and the pathological hallmarks of AD include b-amyloid plaques and neurofibrillary tangles.

There is evidence to show that these pathological changes begin many years prior to the onset of dementia[2]. The challenge for physicians would be to identify subtle changes in cognition when the pathological changes are only beginning to develop. These earlier stages of disease have been described using several terminologies including mild cognitive impairment (MCI) and cognitively impaired not demented (CIND)[3,4]. It is crucial that clinicians are able to identify these earliest stages of cognitive impairment as intervention is most likely to be effective when initiated at this early stage.

EPIDEMIOLOGY

In Singapore the prevalence of dementia and cognitive disorders is likely to increase rapidly over the coming years. We have the fastest ageing population in the Asia-Pacific region with15-20% of the total population being above the age of 65 by the year 2030. At the present time it is estimated that we have about 25 thousand patients with dementia and this number is set to increase to 53 thousand by 20205-7. The prevalence of MCI is presently unclear but based on western prevalence rates of 18.5% at age 50-60 and 35-38% at age greater than 60, it is estimated that we currently have 75-100 thousand subjects with MCI8-9.

ETIOLOGY AND RISK FACTORS

Dementias are largely neurodegenerative conditions including Alzheimer's disease, vascular dementia (VD), Lewy Body dementia, Frontotemporal dementia (FTLD), dementia associated with Parkinsonism and Creutzfeldt-Jakob disease. However reversible causes such as normal pressure hydrocephalus, neurosyphilis, B12 deficiency, folate deficiency and Hashimoto's encephalopathy need to be considered and excluded. AD represents the most common cause of dementia followed by vascular dementia.

NAGAENDRAN KANDIAH, Consultant, Department of Neurology, National Neuroscience Institute

CHONG MEI SIAN, Senior Consultant, Department of Geriatric Medicine, Tan Tock Seng Hospital

The main pathological hallmarks of AD are the Beta-amyloid plaques and neurofibrillary tangles. The risk factors for the development of this pathology include advanced age, family history, vascular risk factors and APOE4 genotype[10-11]. It is also increasingly evident that AD and vascular pathology often coexist and manifests as mixed dementia. Optimization of vascular risk factors such as diabetes mellitus and hypertension is believed to slow the amyloid cascade resulting in stabilization of cognitive function among patients with vascular cognitive impairment.

MILD COGNITIVE IMPAIRMENT

Cognitive changes in the elderly occur over a continuum, ranging from normal ageing at one end of the spectrum to dementia at the other end. There has been intense interest in the intermediate stage between normal ageing and dementia. Of the various classification systems, the Mayo Clinic's mild cognitive impairment (MCI) has received the most attention. Its pathological validity is supported by conversion rates to dementia of approximately 12% annually and 80% at six years of follow-up. Originally, MCI diagnosis required the presence of memory complaint (preferably corroborated by an informant), objective memory impairment for age, essentially preserved general cognitive function, normal functional activities and no dementia. (Chong, 2008)[12]

The heterogeneity within MCI has lead to the proposal of a new classification system, based predominantly on neuropsychological profiles and includes amnestic or single memory MCI, multiple-domain MCI and single non-memory MCI. However, the existing clinical criteria for diagnosis of MCI are subjective, variable in operationalisation, and highly dependent on clinical judgment. They are also unable to reliably predict who amongst those with MCI would progress to dementia. Thus, the differentiation between normal cognitive aging and MCI (especially the early stages of MCI) would be extremely challenging using only clinical methods. This has prompted research into the use of more objective neuroimaging (structural and functional), cerebrospinal fluid (CSF), genetic and molecular biomarkers which reflect AD pathogenesis, to complement clinical approaches towards an early and accurate diagnosis of AD. Initial drug trials have not shown clinical benefit, likely related to the heterogeneity of this MCI entity.

Clinical research in accurate characterisation of MCI is of paramount importance in tandem with the concurrent development of disease-modifying therapies to identify those MCI subjects who would stand to gain most from early intervention. These issues currently render MCI to be mainly a research entity at this moment and preclude their current use in routine clinical practice. As such, the discussion below will focus mainly on established dementia.

ASSESSMENT

The evaluation of dementia should be targeted at individuals in whom there is some suspicion of cognitive impairment. This includes subjects with memory or other cognitive complaints, this could either be self-reported or noticed by family members or caregivers; subjects in whom the physician has suspicion of cognitive impairment during the consultation despite the absence of memory or cognitive complaints; subjects who are at increased risk for dementia, such as those with strong family history of dementia and elderly subjects who need to make an important decision (such as making a will, sale of flat, handling complicated financial matters) and in whom mental competency is in question. It is important to note that forgetfulness is not a part of normal aging, while normal older persons might take a longer time to recall, they should still be able to function independently and maintain social functioning should they be given more time to do so.

The evaluation of cognitive impairment should been done via a multifaceted approach, focusing not only on the cognitive complaints, but also on the functional and social consequences of these cognitive changes. This would help the clinician diagnose dementia early, assess for the complications of dementia and establish the aetiology of the dementia and manage accordingly.

With a patient presenting with forgetfulness or confusion, we can use a 4-step assessment to evaluate the cognitive complaint:

(i) Is the forgetfulness or confusion acute or chronic?
(ii) If the forgetfulness or confusion is chronic, is it dementia?
(iii) If it is dementia, what are the complications? (iv) If it is dementia, what is the aetiology?

(i) Is the forgetfulness or confusion acute or chronic?

If the cognitive complaints is of an acute nature, with a rapid onset and short duration (lasting from few hours to days), it would be important to exclude delirium.

Delirium is defined by the Diagnostic and Statistical Manual of Mental Disorders – fourth edition (DSM-IV); however, this may be difficult to apply in clinical practice. The Confusion Assessment Method (CAM) is a brief and structured assessment commonly used in clinical setting to diagnose delirium. It requires the presence of 3 of the following 4 features: presence of acute change in mental status and fluctuating course with inattention, coupled with either the presence of disorganized thinking or altered level of consciousness. CAM has been shown to have 94-100% sensitivity and 90-100% specificity in the identification of delirium with good inter-observer reliability (kappa test 0.81-1.0). If the cognitive complaints are assessed to be secondary to delirium, the underlying precipitating factors (such as sepsis, stroke disease or drug causes) should be looked out for and the patient would require hospitalisation to manage the delirium and the underlying medical illness.

One must also be mindful that acute confusional state can sometimes be superimposed on chronic confusion. If the

Table 1. DSM-Iv Clinical Criteria for Diagnosis of Dementia

Cognitive domain	Questions
Amnesia	Any forgetfulness? Did it start gradually or suddenly? Is it progressively worse? And if so, is it smoothly declining or showing a step-wise/ fluctuating decline? Is it over short-term or long-term matters?
AND declines in one of the following domains:	
Aphasia	Any word-finding difficulty or other difficulties with communication?
Apraxia	Any problems with buttoning or dressing? Any difficulties with using utensils during mealtimes?
Agnosia	Any problems recognising familiar faces or familiar items?
Executive dysfunctioning	Any problems handling money (loose change)? Any change in general problem-solving abilities? Is one's work getting to be more disorganised?
Of sufficient severity to cause significant impairment in social or occupational functioning	As a result of the above, is he becoming less independent in the - community? - home-care? - self-care level?

forgetfulness or confusion is of a subacute nature, developing over a period of week to few months, conditions such as stroke disease, space-occupying lesion, Creutzfeld-Jakob disease and hydrocephalus have to be excluded.

(ii) Is it dementia?

If the cognitive complaints are of a chronic nature, it is first important to exclude depression and late-onset psychiatry disorders. The diagnosis of dementia is then assessed via a clinical approach, either subjectively (looking for features of cognitive decline in the subject) or objectively (testing the subject's cognitive abilities using validated performance-based assessments).

Subjective approach

The DSM-IV criteria for dementia are often used as the gold standard for clinical diagnosis of dementia. It requires the presence of memory impairment, together with deficits in one other cognitive domain (aphasia, apraxia, agnosia and executive dysfunctioning). Examples of practical questions to be asked to the patient's informants with regards to these cognitive domains are shown in Table 1.

The Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE) is a 26-item test that enquires about the subject's memory, cognition and language ability of the last 10 years. The strength of this instrument is its assessment of cognitive changes over a period of time, instead of a single point in time and also it is independent of the subject's premorbid ability or past educational attainments. This has also been validated locally among elderly Chinese subjects with an optimal cut-off score of 3.3/3.4 with 94.3% sensitivity and 94% specificity.

Objective approach

This is an observer-based approach using either performance-based instruments, such as mental status test (brief screening instruments), or a more detailed neuropsychological tests, which is usually administered by clinical psychologists.

There are several mental status tests which have been validated locally. These include the Elderly Cognitive Assessment Questionnaire (ECAQ), Abbreviated Mental Test (AMT) and the Chinese Mini Mental Status Examination (CMMSE); more recently, a single question screen on progressive forgetfulness, clock drawing test (CLOX) and Brief Informant Screening test.

Table 2. Locally Validated Bedside Screening Instruments for Dementia

Elderly Cognitive Assessment questionnaire (ECAQ)	
Items	Score
Memory	
1. I want you to remember this number. Can you repeat after me (4517). I shall test you again in 15 mins.	1
2. How old are you?	1
3. When is your birthday? OR in what year were you born?	1
Orientation and information	
4. What is the year?	1
5. Date?	1
6. Day?	1
7. Month?	1
8. What is this place called? Hospital/Clinic	1
9. What is his/her job? (e.g. nurse/doctor)	1
Memory Recall	
10. Can you recall the number again?	
Total score	
Abbreviated Mental Test (AMT)	
Items	Score
What is the year?	1
What is the time? (within 1 hour)	1
What is your age?	1
What is your date of birth?	1
What is your home address?	1
Where are we now?	1
Who is our country's Prime Minister?	1
What is his/her job? (show picture)	1
Memory phrase "37 Bukit Timah Road"	1
Count backwards from 20 to 11	1
Recall memory phrase	1
Total score	

The ECAQ (Table 2) is a 10-item cognitive test which assesses memory and information-orientation. Using a cut-off score of 5/6, it has 85.3% sensitivity with 91.5% specificity for identifying cognitive impairment.

The 10-item AMT (Table 2) and 28-item CMMSE has also been validated locally. For mild cognitive impairment, AMT's cut-off score is 7/8 (81% sensitivity with 89% specificity) and CMMSE cut-off score of 20/21 (sensitivity 83%, specificity 94%). The CMMSE is more useful in those with higher educational attainment as the AMT has a ceiling effect on these individuals.

It is important to keep in mind that these cut-off scores serve as a screening instrument for dementia; where some subjects may score low on cognitive screening test and have no dementia, while others may score very well but have dementia. Language barriers, advanced age and low education may confound the results and provide false-positive scores. We recommend a combined subjective and objective approach and acknowledge the challenges in diagnosing dementia in a certain group of patients.

Neuropsychological testing is useful in detecting subtle cognitive difficulties which is not picked up by the brief screening instruments. They should be performed on subjects who have memory complaints but do not yet satisfy criteria for dementia; depressed subjects who present with memory complaints to help in determining whether the memory complaints is due solely to the depression or whether they have concomitant dementia; and subjects in whom decision-making capacity is being assessed. Psychometric testing can be a useful adjunct in the latter scenario. In addition, neuropsychological testing may be helpful in dementia aetiologic differentiation. Neuropsychometric batteries have been validated locally in the elderly Chinese and the Vascular Dementia Battery test has also been validated in the Singapore population.

Neuropsychological tests are also useful in individuals in whom the diagnosis of dementia is inconclusive (such as those subjects with performance below 1SD or 1.5SD below age and education adjusted norms) and serial monitoring for performance decline over time is useful in establishing the diagnosis.

(iii) what are the Dementia complications?

The complications of dementia can be broadly divided into behavioural and psychological symptoms, functional problems and social problems (discussed in subsequent chapters). These should be evaluated in all patients with dementia as these issues are the major cause of stress on the caregiver and assessment would enable the clinician to target subsequent management effectively.

Functional difficulties can be assessed at 3 levels: community functioning, home functioning and self-care. They are generally affected with the progression of dementia in a descending order and also allow these functional deficits to serve as markers of dementia severity. It is important when asking for functional deficits to ask for a change in the level of function, i.e. whether the patient is functioning at the same level as before and whether the patient is as independent as before. It is also important to make sure that these difficulties result from cognitive difficulties and not physical disabilities.

The severity of dementia can be staged using the Diagnostic and Statistical Manual of Mental Disorders-3rd revised-edition (DSM-III-R) criteria where mild dementia is defined as impairment for work and social activities with the capacity for independent living remaining largely intact. Moderate dementia takes place when independent living is hazardous and would require some degree of supervision. Severe dementia is characterized by impaired activities of daily living such that continual supervision is required. Other formal functional assessment scales include Clinical Dementia Rating Scale (CDR), Functional Assessment Staging (FAST), Barthel Index and Blessed Dementia Scale (BDS).

(iv) what is the Dementia aetiology?

Having determined the cognitive impairment to be chronic and having met clinical criteria for dementia, as well as assessing for the complications of dementia, the final step of the clinical evaluation involves determining the dementia aetiology.

The types of dementia can be broadly divided into 2 categories – irreversible and reversible causes (Table 3). The aim of determining dementia aetiology is to rule out potentially reversible causes of dementia and selecting appropriate treatment strategies for the irreversible dementias. This is done via clinical history and physical examination, followed by laboratory investigations and neuroimaging. There are guidelines and practice parameters developed for evaluating of dementia etiology and also more specific criteria for diagnosis of the more common Alzheimer's disease (AD) and vascular dementia (VD).

In the history, it is important to ask for the nature of the cognitive decline (sudden or gradual), progression – either gradually progressive (more suggestive of AD) or stepwise/fluctuating course (suggestive of VD). A history of significant alcohol ingestion and medication use (such as antipsychotics, antidepressants, anticholinergic agents and sedative-hypnotic

Table 3. Types of Dementia

Irreversible causes
• Degenerative causes – Alzheimer's disease (AD), frontotemporal dementia, diffuse Lewy body dementia.
• Cerebrovascular disease – vascular dementia (VD).
• Prion-associated disorders (Creutzfeld-Jakob disease).
• Neurogenetic disorders.
Potentially reversible causes
• Infectious disorders – meningitis, encephalitis.
• Toxic or metabolic causes – hypothyroidism, vitamin B12 deficiency, alcohol-related syndromes.
• Neoplastic causes.
• Hydrocephalus – obstructive or normal pressure hydrocephalus.

agents) and history of medical, neurological and psychiatric illness is important.

A targeted physical examination should be performed, looking for focal neurological deficits (such as visual field defects, hemiparesis, hemisensory loss, asymmetric deep tendon reflexes or unilateral extensor plantar responses). It is also important to examine for extrapyramidal signs such as rigidity and bradykinesia, movement disorders and gait abnormalities as these may point to certain aetiologic diagnosis.

Dementias which are related to metabolic abnormalities are thought to be reversible. The most commonly recommended haematological tests are: full blood count, urea and electrolytes, serum calcium, serum glucose, thyroid function tests and vitamin B12 levels. We do not advise routine testing for neurosyphilis given the problems in interpreting the results of testing. Serum Venereal Disease Research Laboratory (VDRL) testing detects only 75% of tertiary syphilis and CSF VDRL may be negative in 30-70% of cases and neurosyphilis. Thus we recommend testing only when patients exhibit clinical features of neurosyphilis.

Other biomarkers which can help in establishing dementia diagnosis include apolipoprotein-E e4 allele, CSF-tau and β-amyloid for AD, CSF 14-3-3, neuron-specific enolase and electroencephalogram for Creutzfeld-Jakob disease. However, these are not performed routinely.

Neuroimaging is useful in the differential diagnosis of dementia and are also necessary in the diagnostic criteria in AD and VD. This may be helpful in justification of aggressive management of vascular risk factors in those patients found to have cerebrovascular disease on neuroimaging. They are also useful in detection of very early dementia as the functional and structural brain changes takes place before clinical manifestation of cognitive deficits. They consist of either structural imaging techniques (computed tomography (CT) scan of head and magnetic resonance imaging (MRI)) or functional neuroimaging techniques (Positron emission tomography and single-photon emission tomography).

Whether all patients with dementia require a structural imaging is an important clinical question, for which there is no consensus. The value of neuroimaging is the identification of cerebral infarcts and clinically important surgical brain lesions (SBLs) such as subdural haematomas, cerebral tumors and normal pressure hydrocephalus. The Canadian Consensus Conference on the Assessment of Dementia (CCCAD) has outlined the criteria for undertaking a CT scan, only if certain conditions are met (Table 4).

We also believe that the functional stage of the dementia is also relevant and important, over and above the duration of cognitive symptoms. In a patient with advanced dementia of long duration (>2 years), we believe that a brain scan is not warranted to detect potentially reversible SBLs. However, if the patient's dementia is still mild and moderate (even after 2 years), a brain scan is indicated.

Summary Of Approach To Patient With Memory Complaint

- Is the memory complaint acute or chronic?
  Rule out delirium.
- If it is chronic, is it dementia?
- If it is dementia, what are the complications?
  Behavioural, functional, social aspects of dementia
- What is the aetiology?
  Clinical evaluation (history, clinical examination, laboratory tests, + neuroimaging)
  To rule out reversible causes.
  If irreversible cause, clinical criteria in the differential diagnosis of dementia aetiology.

INVESTIGATIONS

We are now fortunate to have a wide range of investigational tools including CT brain, MRI brain, PET scans, cerebrospinal fluid (CSF) studies and genotyping. With the availability of such tools which have been demonstrated to have reliable sensitivity and specificity the diagnosis of dementia and MCI should move away from being a "diagnosis of exclusion" to a "diagnosis of inclusion". Structural brain imaging with MRI is useful to evaluate for hippocampal atrophy which is the hallmark of

Table 4. Canadian Consensus Conference Criteria for Performing Cranial CT in Patients with Dementia

CT is recommended if one or more of these criteria are present.

- Patients are less than 60 years old.
- Rapid (e.g. over 1-2 months), unexplained decline in cognition or function.
- Dementia of relatively short duration (< 2 y).
- Recent, significant head trauma.
- Unexplained neurologic symptoms (e.g. new onset of severe headache or seizures).
- History of cancer, especially of a type or at a site associated with metastasis to the brain.
- Use of anticoagulants or history of bleeding disorder.
- History of urinary incontinence and gait disturbance early in the course of dementia (suggestive of normal pressure hydrocephalus).
- Presence of any new localizing signs on physical examination (hemiparesis, babinski's sign).
- Unusual or atypical cognitive symptoms or presentation (e.g. progressive aphasia).
- Gait disturbance.

AD while disproportionate atrophy of the frontal lobes may be indicative of frontotemporal dementia[13]. MRI is also valuable in demonstrating white matter disease and lacunar infarctions which are suggestive of vascular dementia. Special MRI sequences such as the diffusion weighted imaging (DWI) can demonstrate diffusion abnormalities which are highly specific for Creutzfeldt-Jakob disease. These advanced neuroimaging techniques will have increasing importance once MCI is accurately characterized and disease-modifying treatments have been shown to be effective. CSF studies of beta amyloid, total tau and phospho-tau have been demonstrated to have a high specificity for the diagnosis of AD. CSF examination is also valuable in managing reversible conditions such as encephalitis and autoimmune encephalopathies. PET scans also can help distinguish between AD and FTLD based on the pattern of glucose hypometabolism.

MANAGEMENT

Management of cognitive disorders requires a multidisciplinary approach including pharmaceutical and non-pharmaceutical management of the patient, caregiver support and provision of long term nursing care. The mainstay of pharmaceutical management includes acetyl cholinesterase inhibitors[14]. Patients who are initiated on AchEIs should be offered the highest tolerable dose for an adequate length of time. Switching from one AchEI to another or switching from an oral formulation to a patch delivery may need to be considered for patients who develop intolerable side effects.

Memantine, a NMDA receptor antagonist may be useful for patients with moderate to severe AD. In view of the increased risk of cardiovascular and cerebrovascular events with both typical and atypical antipsychotics, these drugs should be reserved for patients with severe behavioral symptoms. Several disease modifying agents are now in phase 3 clinical studies. They target the amyloid cascade or the production of tau and preliminary studies have demonstrated promising results.

CONCLUSIONS

Dementia represents a late stage of disease along the continuum of cognitive impairment. Early diagnosis of dementia is important as early therapeutic interventions may palliate substantially, if not reverse, the significant emotional and economic costs of the illness. A 4-step clinical approach could be a succinct framework to aid the family physician in evaluating the individual who presents to the clinic with cognitive complaints such as forgetfulness or confusion. Management of cognitive disorders requires a multidisciplinary approach including pharmaceutical and non-pharmaceutical management of the patient, caregiver support and provision of long term nursing care.

REFERENCES

1. American Psychiatric association. Diagnostic and statistical manual of mental disorders (1V-TR), 4th edition-text revision. Washington, DC 2000.
2. Price JL, Morris JC. Tangles and plaques in nondemented aging "preclinical" Alzheimer's disease. Ann Neurol 1999;45:358-68.
3. Petersen RC. Mild cognitive impairment as a diagnostic entity. J Intern Med. 2004;256:183-94.
4. Ebly EM, Hogan DB, Parhad IM. Cognitive impairment in the nondemented elderly. Results from the Canadian Study of Health and Aging. Arch Neurol. 1995;52:612-9.
5. Chiam PC, Ng TP, Tan LL, Ong PS, Ang A, Kua EH. Prevalence of dementia in Singapore--results of the National Mental Health Survey of the Elderly 2003. Ann Acad Med Singapore. 2004; 33:S14-5.
6. Asia Pacific Members of Alzheimer's Disease International. Dementia in the Asia Pacific Region: The epidemic is here. Alzheimer's disease International 2006. Available at: http://www.accesseconomics. com.au/publicationsreports/search.php
7. Inter-Ministerial Committee on Health Care for the Elderly 1999
8. Barker A, Jones R, Jennison C.A prevalence study of age-associated memory impairment. Br J Psychiatry. 1995;167:642-8.
9. Richards M, Touchon J, Ledesert B, Richie K. Cognitive decline in ageing: are AAMI and AACD distinct entities? Int J Geriatr Psychiatry. 1999; 14:534-40.
10. Graff-Radford NR, Green RC, Go RCP, et al. Association between apolipoprotein E genotype and Alzheimer's disease in African American subjects. Arch Neurol 2002; 59:594-600.
11. Casserly I, Topol E. Convergence of atherosclerosis and Alzheimer's disease: inflammation, cholesterol, and misfolded proteins. Lancet 2004; 363:1139-46.
12. Chong MS. Making the diagnosis of dementia. Singapore Family Physician 2008; 35(1):66-72.
13. Jack CR Jr, Petersen RC, Xu YC, Waring SC, O'Brien PC, Tangalos EG, Smith GE, Ivnik RJ, Kokmen E. Medial temporal atrophy on MRI in normal aging and very mild Alzheimer's disease. Neurology. 1997 Sep;49(3):786-94.
14. Doody RS, Stevens JC, Beck C, et al. Practice parameter: management of dementia (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2001;56:1154-66.

FURTHER READINGS

1. Clinical Practice Guidelines for Dementia (April/2007). <http://www.moh.gov.sg/mohcorp/publications.aspx?id=16970>
2. Chong MS, Sahadevan S. An Evidence-based Clinical Approach to the diagnosis of dementia. Annals Academy of Medicine Singapore 2003;32:740-8.
3. Knopman DS, DeKosky ST, Cummings JL, Chui H, Corey-Bloom J, Relkin N, Small GW, Miller B, Stevens JC. Practice parameter: Diagnosis of dementia (an evidence-based review) Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2001; 56:1143-53.
4. Feldman HH, Jacova C, Robillard A, Garcia A e tal. Diagnosis and treatment of dementia: 2. Diagnosis. CMAJ 2008; 178(7):825-36.
5. Chertkow H, Massoud F, Nasreddine Z, Belleville S et al. Diagnosis and treatment of dementia: 3. Mild cognitive impairment and cognitive impairment without dementia. CMAJ 2008; 178(10) 1273-85.
6. Peterson RC. Conceptual overview. IN: Petersen RC, ed. Mild cognitive impairment: Aging to Alzheimer's disease. New York, NY: Oxford University Press, Inc; 2003:1-14.

LEARNING POINTS

- **Cognitive dysfunction manifests along a continuum ranging from mild cognitive impairment to dementia.**
- **The strongest risk factors for AD are age, family history and APOE genotype.**
- **While dementia is often secondary to a neurodegenerative pathology, other reversible causes such as normal pressure hydrocephalus needs to be excluded.**
- **Investigative tools such as MRI and CSF studies can help establish a diagnosis of mild cognitive impairment and early dementia.**
- **The four-step approach to dementia evaluation consists of:**
  - **Exclusion of delirium as the cause of the forgetfulness or confusion.**
  - **Establishing the diagnosis of dementia.**
  - **Assessing for the behavioural, functional, and social problems associated with dementia.**
  - **Establishing the aetiological diagnosis of dementia.**
- **Management of cognitive disorders requires a multidisciplinary approach including pharmaceutical and non-pharmaceutical management of the patient, caregiver support and provision of long term nursing care.**

UNIT NO. 2

BEHAVIOURAL AND PSYCHOLOGICAL SYMPTOMS OF DEMENTIA

Dr Ng Li-Ling

ABSTRACT

Behavioural and psychological symptoms of dementia (BPSD) are common in dementia. They cause significant distress to people with dementia and their carers. In managing BPSD, medical causes such as delirium must be excluded. Non pharmacological management, such as environmental and behavioural interventions are effective first line strategies. Medication may be useful in moderate to severe BPSD but must be used carefully in view of the risk of side-effects.

SFP2011; 37(3) (Supp 1) : 15-16

INTRODUCTION

Dementia is a devastating disease and leads to tremendous suffering for people with dementia and their families. In addition to the cognitive deficits of dementia the behavioural and psychological symptoms of dementia (BPSD) are an integral part of dementia. In the original description of Alzheimer's disease 100 years ago, prominent symptoms of paranoia, screaming and hallucinations were present. BPSD, sometimes referred to as non-cognitive or neuropsychiatric symptoms of dementia, is common and occurs in up to 90% of patients over the course of the disease. It is a significant cause of distress in people with dementia as well as their carers and if untreated can lead to premature institutionalization.

DEFINITION

BPSD refers to the symptoms of disturbed perception, thought content, mood or behaviour that frequently occur in patients with dementia (Consensus Conference, International Psychogeriatric Association). Table 1 lists some of common BPSD.

ASSESSMENT

A comprehensive diagnosis of dementia must include an assessment of cognitive and behavioural symptoms as well as the needs of the family. In the initial assessment any medical causes for the behavioural symptoms must be sought and laboratory tests to exclude treatable causes are necessary. (See Table 2)

MANAGEMENT

The main objectives in the management of BPSD are to maximise functional independence, improve the quality of life of patients, minimise caregiver stress and distress, and help families cope with the behaviours.

NG LI-LING, Senior Consultant, Division of Psychological Medicine, Changi General Hospital

Table 1: Common Behavioural and Psychological Symptoms of Dementia

BPSD	Common examples
Anxiety	Repeatedly asking questions of an upcoming event Fear of being left alone Worries about their finances
Depressive mood	Pervasive depressed mood or loss of pleasure Self deprecatory statements Expressing wish to die
Hallucinations	Seeing people in the home who are not really there Hearing deceased people call their names
Misidentifications	Not recognizing their image in the mirror Mistaking carers for other people Misidentification of events on TV or Radio as if they were real
Delusions	People are stealing things House is not one's home Spouse or caregiver is an impostor Spouse is unfaithful
Apathy	Lack of interest in daily activities Decrease in social interaction Decrease in emotional responsiveness Decrease in initiative
Negativism	Refusal to co-operate Resistance to care
Disinhibition	Crying Impulsiveness Verbal aggression Sexual disinhibition – stripping, masturbation
Sleeplessness	Night-time wandering
Agitation	Complex phenomenon Defined as socially inappropriate verbal, vocal of motor activity may include the following:
Physically aggressive behaviours	Hitting Pinching Kicking & biting Slapping Grabbing
Restlessness	Pacing
Screaming	Calling for help, asking to go home, cursing
Wandering	Shadowing/stalking of carer Aimless walking Excessive activity Repeatedly trying to leave the house

Table 2: Some Common Causes of BPSD

Causes	
Delirium	Due to infections, medication, dehydration, metabolic causes etc
Constipation	Faecal impaction
Pain	Arthritis, toothache
Discomfort	Uncomfortable clothing, ingrown toe nail
Sensory impairment	Faulty hearing aid

After comprehensive assessment and treatment of underlying medical causes specific BPSD are identified. The general principles in management are:

- to understand the cause of the behaviour disturbance e.g. environmental factors, stressful tasks or caregiver reactions.
- decide if the symptoms need to be treated.

- formulate a management plan with the caregiver.
- implement specific strategies.
- review care plans regularly.

General advice for caregivers includes; maintaining a calm familiar environment with a regular routine, organising an activity programme that is appropriate to the person with dementia or arrange for the person with dementia to attend a dementia day care centre. Caregivers need support and can seek help from family support groups and counselling centres.

Table 3: Examples of Non-Pharmacological Interventions

Symptom	Interventions
Agitation and aggression	Use a calm approach to the person Speak in a soft voice Distract if possible – offer a drink, talk about a pleasant activity, hand massage Use music or audio or video tapes
Wandering	Reassure when the person appears lost Use large written signs with clear words or symbols If there is a risk that they wander out of the house use identity bracelets with a contact number Allow access to safe wandering places e.g. a garden that is enclosed Use digital locks at exit doors Use artificial partitions or visual barriers to hide exit areas Electronic alarm systems may be useful Handphones with GPS tracking are available
Sleeplessness	Maintain a regular activity and exercise programme Avoid day time naps and caffeine in the evenings Sleep hygiene

Non-pharmacological Management

Non-pharmacological interventions are usually first line management for mild to moderate BPSD and it has been shown that environmental and behavioural interventions in conjunction with caregiver education, training and support are effective. Some examples of interventions in the care plan for people with BPSD are listed in Table 3.

Pharmacological management

Medication is indicated if non-pharmacological interventions have failed or when the symptoms are moderate or severe and has an adverse impact on the person with dementia or his caregiver.

Guidelines to pharmacotherapy:

- Treat only moderate or severe BPSD with medication.
- Use lower doses especially in the elderly.
- Target specific behaviours e.g. hallucinations, delusions, aggression.
- Start with one drug at a time.
- Be aware of adverse effects and drug sensitivity.
- Regular reviews of medication effects and side-effects.
- Make sure use of medication is time limited.

REFERENCES

1. Behavioral and Psychological Symptoms of Dementia (BPSD) Educational Pack, International Psychogeriatic Association, 2002
2. Hort J et al. EFNS guidelines for the diagnosis and management of Alzheimer's disease. European Journal of Neurology 2010;17:1236-48.
3. The 36 hour Day, Nancy Mace and Peter Rabins.

Table 4. Pharmacological Interventions

Drug	Use	Daily dose range	Comments
Anti-psychotics	Hallucinations Delusions Agitation Aggression	Haloperidol (0.5-2 mg) Risperidone (0.5-2 mg) Olanzapine (5-10 mg) Quetiapine (25-150 mg)	Extrapyramidal side effects Over sedation Atypical anti-psychotics associated with possible raised risk of cerebrovascular adverse events and prolongation of Q-T interval
Anti-depressants	Depression	Fluoxetine (20-30 mg) Fluvoxamine (50-150 mg) Escitalopram (10-20 mg) Paroxetine (20-30 mg) Mirtazapine (15-45mg)	
Cholinesterase inhibitors	Apathy Hallucinations	Donepezil (5-10mg) Rivastigmine (6-12 mg) Galantamine (16-24 mg)	Nausea GIT symptoms
Anti-convulsants	Agitation Aggression	Sodium Valproate (400-1000 mg)	Monitor liver function
Benzodiazepines	Insomnia Anxiety Agitation	Lorazepam (0.5-2 mg)	Excessive sedation Risk of falls

LEARNING POINTS

- **Exclude delirium and psychiatric disorders such as depression as the cause of behavioural problems.**
- **Non pharmacological management of BPSD with environmental and behavioural interventions, is the first line of treatment.**
- **When using medication for moderate to severe BPSD, use the lowest dose and regularly review treatment.**

UNIT NO. 3

PHARMACOLOGICAL TREATMENT OF DEMENTIA

Dr Lim Wee Shiong

ABSTRACT

Pharmacotherapy is a vital part of the multi-pronged strategy in dementia management. All dementia patients should be evaluated for suitability of pharmacological strategies to address the underlying disease, enhance cognitive symptomatology, and treat attendant behavioural complications. Once a definitive diagnosis of dementia has been made, the choice of symptomatic treatment hinges mainly on dementia etiology and stage of severity. While skilful use of symptomatic treatment can offer tangible but modest benefits in many cases, the decision to initiate such costly treatment should be individualized and always made in conjunction with the patient and caregiver. In future, disease-modifying treatment which goes beyond a primary symptomatic effect to target the underlying disease process may be available.

SFP2011; 37(3) (Supp 1) : 17-23

INTRODUCTION

An executive report in 2006 highlighted the threat of an impending epidemic of dementia in the Asia-Pacific region in line with the greying demographic trend[1]. This has implications for Singapore, which has one of the most rapidly aging populations in the region. There is a compelling need for primary care physicians to be trained in the care and management of dementia patients to meet the projected burgeoning demand. From the standpoint of pharmacological management, it is foreseeable that the primary care physician would be involved in one of two ways:

- initiation of treatment in a newly diagnosed dementia patient, or
- more commonly, continuation of treatment in dementia individuals whose treatment regimes have been initiated and stabilized by the hospital-based dementia specialist.

LIM WEE SHIONG, Senior Consultant, Department of Geriatric medicine, Tan Tock Seng Hospital

OVERVIEW

In the past, dementia was often perceived as a terminal illness for which the main focus of treatment is palliation. Increasingly, there is a paradigm shift towards treating dementia as a chronic disease, not unlike conditions like diabetes mellitus and heart failure, where specific treatment goals can be formulated depending on the stage of the disease (Figure 1). For instance, in the early stages of disease, the management aims would be to delay progression of disease, maintain optimal functioning, prevent emergence of behavioural symptoms, and address relevant psychosocial issues (e.g. development of advanced directives, assessment of driving safety, and diagnostic disclosure to employer/other family members).

Seen in this light, it is important to appreciate that pharmacotherapy is only one of the tenets of a comprehensive multi-pronged strategy for dementia management that should encompass other aspects such as a well-established diagnosis, education of patient and carer, non-pharmacological measures and comprehensive caregiver psychosocial intervention.

Pharmacological treatment can be broadly conceptualized into three broad categories:

1. Reverse or stabilize the underlying disease.
2. Improve cognitive symptomatology, and
3. Treat behavioural and psychiatric symptoms associated with dementia.

As behavioural and psychiatric symptoms associated with dementia are covered in Unit 2, the rest of the article shall focus on the first two aspects of pharmacotherapy.

(1) Reverse or stabilize the underlying disease

Pharmacological strategies to address the underlying disease include treating identifiable reversible causes, reduction of established risk factors, and disease modifying measures to slow the rate of disease progression (Table 1).

Figure 1: Paradigm shift in dementia treatment

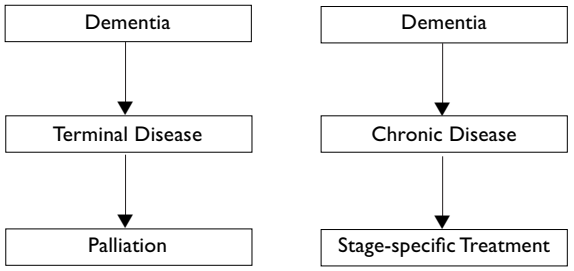


Table 1. PHARMACOLOGICAL STRATEGIES TO ADDRESS UNDERLYING DISEASE

1. Treating identifiable reversible causes • Treat depression (pseudodementia). • Replace deficiency states (e.g. B12 deficiency, hypothyroidism). • Correct metabolic abnormalities (e.g. hypercalcemia, hypoglycemia). • Treat infections (e.g. neurosyphilis, HIV-associated dementia).
2. Reduction of vascular risk factors • Hyperlipidemia, hypertension, diabetes mellitus, smoking, obesity. • Homocysteine-lowering agents e.g. folate, pyridoxine, B12. • Anti-platelet agents for secondary stroke prevention. • Anti-coagulation for atrial fibrillation and cardioembolic strokes.
3. Ancillary treatment to slow rate of disease progression • Lack of evidence in trials so far involving NSAIDS, cyclooxygenase-2 inhibitors, low dose prednisolone, oestrogen replacement therapy and statins. • High dose Vitamin E is not recommended.

It is now established that vascular risk factors are putative not only in vascular dementia (VaD), but also in Alzheimer's disease (AD); thus, vascular risk factors should be assiduously sought for and managed in all dementia cases. While a search for reversible causes should be undertaken in all newly diagnosed dementia patients, in truth, only a small percentage of potentially reversible abnormalities are truly reversible, most notably conditions such as depression and hypothyroidism. There is concomitant neurodegenerative causes such as AD in many of these patients. Moreover, when significant neuronal damage has occurred, treatment of potentially reversible causes often arrests the underlying pathophysiology but does not reverse the dementia.

Trials involving NSAIDS, cyclooxygenase-2 inhibitors, low-dose prednisolone and estrogen replacement therapy have yielded null findings. High dose vitamin E (2000 IU per day) is currently not recommended as ancillary treatment for dementia, because the debatable marginal benefits are mitigated by concerns about safety, especially in doses above 400 IU/day[2]. The LEADe study reported no benefit in cognition or global function when Atorvastatin 80mg/day was given to patients with mild to moderate Alzheimer's disease who were taking donepezil.[3] Subgroup analysis in the OmegaAD study also suggests a slower decline in Mini Mental State Examination (MMSE) in very mild AD (MMSE>27) treated with omega 3 fatty acid[4], although two RCTs reported no overall effect of omega 3 fatty acid supplementation on cognitive performance among cognitively healthy older adults[5].

(2) Medications for improving cognitive symptomatology

Currently, the established modalities for dementia treatment are considered to be primarily symptomatic rather than disease-modifying in their mode of action. There are two main classes (Table 2):

- *Cholinesterase Inhibitors (ChEIs)* based on the cholinergic hypothesis, which states that many of the cognitive, functional and behavioural symptoms derive from an absolute or relative deficit in brain acetylcholine activity, and;
- *N-methyl D-aspartate (NMDA)* receptor antagonists, which protect against glutamate-mediated excitotoxicity.

Other less established treatment options for dementia include:

- Ginkgo biloba, which exhibits "inconsistent and unconvincing benefits" based on a 2007 Cochrane systematic review of 35 clinical trials and 4247 participants[6]. The recently published Ginkgo Evaluation of Memory (GEM) study also concluded that ginkgo was not effective in preventing dementia in elderly individuals with mild cognitive impairment (MCI) or normal cognition[7]. Practitioners who prescribe ginkgo should be aware of the variability of active ingredient among preparations and the potential for drug interactions, such as increased bleeding risk when combined with warfarin and antiplatelet agents.
- Selegiline and piracetam, which are both not recommended for the treatment of core cognitive symptoms of dementia.

CHOLINESTERASE INHIBITORS

ChEIs form the mainstay of dementia treatment. Most of the published data on ChEIs are derived from randomized controlled trials of mild-to-moderate stages of AD. A recent study demonstrated the benefit of donepezil in the more severe stages of AD (MMSE<10)[8]. In general, ChEIs confer modest improvement in (1) cognition and global functioning of short-term duration (6 to 9 months), (2) activities of daily living (best described as a slowing of decline rather than an actual improvement), and (3) neuropsychiatric symptoms (delay in emergence of symptoms, improvement in apathy, and variable patterns of improvement for milder degrees of anxiety, depression and hallucination).

Patients who received higher doses had a better long-term outcome than those who received placebo or low doses. Although the placebo and low-dose groups did show improvement when switched to high doses during the open-label extensions of double-blind pivotal trials, they did not "catch up" with the group that received high-dose ChEI since trial inception, suggesting that ChEIs provide greater benefit when started as soon as dementia is diagnosed, rather than waiting until symptoms become more prominent. In some open-label studies, the duration of benefit was observed to persist for as long as three years[9].

Trials of mixed dementia and VaD reported significant improvement in cognition and global function but the benefit in activities of daily living and behaviour was less obvious. Studies of rivastigmine in Parkinson's disease dementia (PDD) and dementia with Lewy bodies (DLB) also demonstrated cognitive, neuropsychiatric and functional benefits without worsening of motor symptoms[10-11].

There are currently three ChEIs regularly used for the symptomatic treatment of dementia (Table 2). There is very little to choose between them in practice in terms of core efficacy. The clinical relevance of pharmacodynamic characteristics (such as selectivity for acetylcholinesterases versus butyrylcholinesterases [rivastigmine], and allostearic modulation of nicotinic receptors [galantamine]) has not been established. Moreover, the few comparative studies are small, industry sponsored, inconsistent in results, and offer little basis to make a clinical choice[12]. Thus, the choice of ChEI therapy will depend on the experience of the clinician, tolerance to side effects, ease of use, and the clinical profile of the individual to be treated (such as co-morbid diseases and drug interactions) (Table 3). For patients who require medications to be crushed due to swallowing difficulties, the capsule formulations (rivastigmine and galantamine PR) should be avoided.

The side effects of the three ChEIs are broadly similar (Table 4). The most common side effect is gastrointestinal (nausea, vomiting, diarrhea, anorexia), which is dose-related, transient, and often circumvented to a large extent by a slower titration and taking the medication with food. Although cardiovascular side effects (such as symptomatic bradycardia and syncope) are generally not frequent, ChEIs should generally be avoided in those with significant bradycardia, sick sinus syndrome or cardiac conduction disturbances. Other uncommon side effects that have been reported (with donepezil, in particular) include muscle cramps, insomnia and vivid dreams; the latter can be avoided by ingestion of donepezil in the morning. Weight should be regularly monitored as weight loss is not uncommon. The latest addition to the therapeutic armamentarium is the transdermal patch for rivastigmine. The matrix patch enables smooth continuous delivery of the drug into the bloodstream over 24 hours, resulting in less fluctuation between peak and trough drug levels than with the capsule formulation. This resulted in 3-fold decrease in side effects such as nausea and diarrhoea whilst maintaining comparable efficacy when compared with equivalent doses of the capsule formulation[13]. Skin tolerability is good and skin irritation generally is limited to mild reactions such as erythema and itch[13.] The patch is available in two doses: 4.6mg/24 hours and 9.6 mg/24 hours (Table 2). It should be applied every 24 hours at a consistent time each day to the upper back, upper arm or chest; application to other body sites may result in reduced absorption. Indications for the patch include: gastrointestinal side effects during titration to higher doses, non-compliance and when a smooth drug delivery is desired (e.g. presence of co-morbidity such as epilepsy).

NMDA ANTAGONISTS

Although memantine has been used in Germany for over 20 years, it is only in recent years that it has been approved in the US and UK for the symptomatic treatment of moderate-to-severe AD. Memantine appears to be beneficial alone or in combination with donepezil for moderately advanced AD[14]. In

Table 2: DOSING RECOMMENDATIONS OF DEMENTIA DRUGS IN CLINICAL USE

Medication	Forms	Starting Dose	Titration	Example of titration
(1) Cholinesterase inhibitors				
Donepezil (Aricept®)	Tablet (5mg, 10mg)	2.5 – 5mg once daily	Increase to 10mg/day after 4-8 wks	2.5mg om → 5mg om → 10mg om
Rivastigmine (Exelon®)	Capsule (1.5mg, 3mg, 4.5mg, 6mg) Patch (4.6mg/24h, 9.5mg/24h)	1.5mg bid after meals 4.6mg/24h once daily	Increase by 1.5mg bid every 2-4 wks up to 6mg bid Increase to 9.5mg/24h after 4 wks	1.5mg bid → 3mg bid → 4.5mg bid → 6mg bid 4.6mg/24h → 9.5mg/24h
Galantamine (Reminyl®)	IR Tablet (4mg, 8mg, 12mg)* PR Capsule (8mg, 16mg and 24mg)* Solution (4mg/ml; 100ml bottle)†	4mg bid after meals‡	Increase by 4mg bid every 4 wks up to 12mg bid‡	4mg bid → 8mg bid → 12mg bid‡
(2) NMDA antagonists				
Memantine (Exiba®)	Tablet (10mg)	5mg once daily	Increase by 5mg every 1-2 weekly up to 10mg bid Increase by 5mg every 1-2 weekly up to 20mg om	5mg om → 5mg bid → 10mg om 5mg at 2pm → 10mg bid 5mg om → 10mg om → 15mg om → 20mg om

* IR: immediate release; PR: prolonged release once-a-day formulation.

† Solution can be mixed with non-alcoholic beverage, but must be consumed immediately.

‡ Dose expressed in terms of immediate release formulation. To calculate the equivalent dosing for the PR formulation, simply add up the total daily dose e.g. galantamine 4mg IR tab bid = galantamine 8mg PR cap once daily; galantamine 8mg IR tab bid = galantamine 16mg PR cap once daily.

an industry sponsored study in moderately severe AD patients (MMSE 5-14) on stable doses of donepezil, the addition of memantine 20mg a day slightly improved cognitive, functional and global scores in comparison with patients adding placebo[15]. The cost-effectiveness of memantine therapy in moderately advanced AD remains to be established. There is also evidence of benefit in mild to moderate AD and VaD, but of a smaller magnitude compared with ChEI therapy. A small randomized controlled study of PDD and DLB patients reported that memantine produced cognitive and global benefits, although there were earlier case reports that memantine can worsen confusion in patients with DLB.

The initial dose is 5mg once a day, with 5mg increments at intervals of at least one week until a maximum of 10mg twice a day is achieved (Table 2). A recent study reported that a once-daily 20mg regime titrated over 4 weeks is equally efficacious and better tolerated compared with the b.i.d. dosing (Table 2)[16]. Memantine should be used with caution in patients with epilepsy and renal impairment, and the clinician should be aware of interactions involving commonly prescribed medications such as dextromethorphan and L-dopa (Table 3).

Memantine is generally better tolerated (especially gastrointestinal-related side effects) than ChEIs. Common adverse events such as dizziness, headache, fatigue, hallucinations and confusion tend to be transient (Table 4). In clinical experience, the side effects that are most likely to lead to discontinuation are restlessness and hyperexcitation.

COMMON ISSUES IN THE USE OF DEMENTIA-SPECIFIC DRUGS

1. How should I decide whether to start symptomatic dementia treatment?

Dementia-specific treatment should only be contemplated in patients with a definitive diagnosis of dementia. ChEI therapy did not delay progression to dementia nor confer any consistent cognitive, global or functional benefits in the pre-dementia stage of mild cognitive impairment (MCI); there was also a higher prevalence of side effects (including cases of sudden deaths) in the treatment group[17]. Thus, ChEIs are presently not recommended in the treatment of MCI.

Because the costs of ChEI and memantine therapy are not subsidized, the greatest challenge of whether to initiate cognitive enhancers resides in the cost-effectiveness, especially in the more severe stages of dementia where the benefit of costly symptomatic treatment is going to be even more marginal. In the AD 2000 study, despite the small but measurable improvements in cognition and activities of daily living, there were no benefits for donepezil in institutionalization, progression of disability and cost savings for health and social services[18]. Thus, treatment decisions regarding the use of symptomatic treatment need to be individualized for each patient, with a conjoint decision reached after careful discussion of the pros and cons of treatment. For instance, where financial resources are limited, the opportunity cost of employing a maid to look after a patient requiring help with activities of daily living may override the modest benefits of symptomatic therapy.

Fig 2. ALGORITHM FOR PHARMACOLOGICAL TREATMENT OF COGNITIVE SYMPTOMS OF DEMENTIA

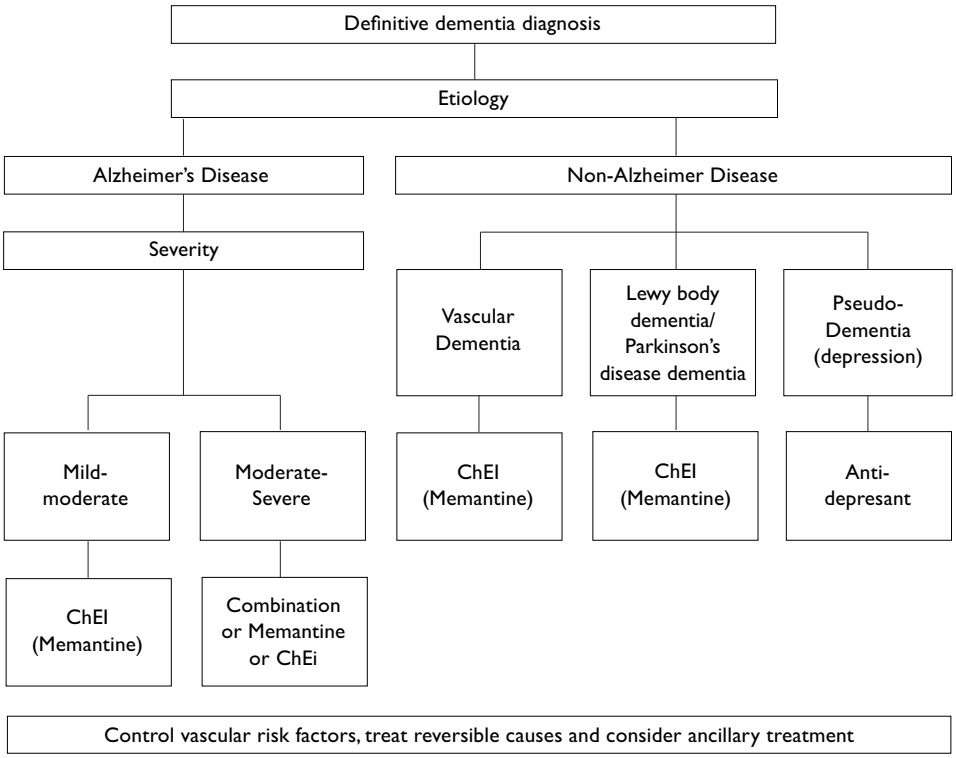


Table 3: IMPORTANT PRESCRIBING INFORMATION OF DEMENTIA DRUGS IN CLINICAL USE

Medication	Dose adjustment		Significant drug interactions
	Hepatic impairment	Renal impairment	
Donepezil	None	None	None
Rivastigmine	None	None	None
Galantamine	Child-Pugh score 7-9: max 16mg/day Child-Pugh score 10-15: use not recommended	Moderate renal impairment: max 16mg/day CrCl < 9ml/min: use not recommended	Amitriptylline, ketoconazole, prosac (fluoxetine), faverin (fluvoxamine) and paroxetine decrease galantamine clearance.
Memantine	None	CrCl 40-60 ml/min: 10mg/day Severe: use not recommended	Concomitant use of amantadine, ketamine or dextromethorphan should be avoided. Effects of L-dopa and dopaminergic agents may be enhanced. Caution is recommended with patients suffering from epilepsy.

Table 4: SIDE EFFECTS OF DEMENTIA DRUGS

Cholinesterase inhibitors

Common	*Less common*
• Nausea	• Weight loss
• Vomiting	• Fatigue
• Diarrhoea	• Bradycardia
• Anorexia	• Urinary incontinence
• Abdominal pain	• Vivid dreams, insomnia
• Headache	• Muscle cramps
• Dizziness	

Memantine

Common	*Less common*
• Headache	• Anxiety
• Dizziness	• Vomiting
• Fatigue	• Cystitis
• Diarrhoea	• Increased muscle tone
• Hallucination	
• Confusion	

To avoid unrealistic expectations, it is important to communicate with the patient and his caregiver/family from the onset that:

– The medications are not a cure.
– The medications do not work for everyone. The principle of one-thirds generally applies: one-third improve, one-third remain stable, while the remaining one-third deteriorate at a rate as if untreated.
– Although there may be a response in terms of modest improvement or "stabilization", symptomatic therapy does not prevent progression of disease and cognitive decline will continue even with treatment.
– The medication will be discontinued if the patient does not respond after an adequate trial of 3-6 months.

2. Which modality should I choose?

Once a definitive diagnosis of dementia has been made, the choice of treatment modality is dependent on 2 key factors (Figure 2):

- Etiology of dementia, which can be broadly classified into AD and non-AD categories.
- Stage of dementia severity, which can be easily ascertained using functional-based scales such as the DSM-IIIR criteria (Table 5).

For AD individuals, ChEIs remain the preferred modality in the mild-moderate stages. Memantine is an option if ChEIs are contraindicated, not tolerated, or if there is disease progression despite an adequate trial of ChEI therapy. In the moderate-severe stages, although combination therapy appears to have the best benefit, the cost remains prohibitive. Memantine has more robust data of benefit in the more severe stages compared with ChEI[14-15].

With regards to non-AD etiologies, the choice of treatment depends on the underlying etiology. ChEI therapy is the preferred modality in vascular dementia, as well as the synucleinopathy-based dementias such as DLB and PDD. While memantine offers a viable option in vascular dementia, it should be used with great caution in DLB and PDD, since there are reports of worsening confusion and behaviour (delusions and hallucinations) with memantine therapy in this group of dementias[19]. Conversely, there are reports of worsening behaviour in patients with frontotemporal dementia treated with ChEIs[20].

3. How do I monitor the benefits of symptomatic treatment?

A range of improvement above baseline may be observed in the first 6-9 months, which can be monitored by the use of clinical methods or standardized rating scales. The former involves a clinical global impression of change after assessing the cognitive, functional and behavioural domains via interview with the patient and caregiver. The latter involves either: (a) brief mental status tests such as the Chinese MMSE, Abbreviated Mental Test (AMT) and Elderly Assessment Cognitive Questionnaire (ECAQ), or (b) more detailed psychometric testing. After 9-12 months, a lesser decline can be observed, which can be documented by patient and caregiver interview for cognitive, functional and behavioural (emergence of neuropsychiatric symptoms) features.

When a patient does not appear to be responding to ChEI therapy, and this is not due to non-compliance or other confounding conditions such as delirium, the options[12,21] include:

- Increasing the dose.
- Switching to another ChEI.
- Switching to memantine.
- Adding on memantine (i.e. ChEI-memantine combination).
- Drug holidays can be associated with clinical deterioration that may not revert to baseline even on resumption of therapy, and hence, should be discouraged.

Table 5: CRITERIA FOR THE STAGING OF DEMENTIA SEVERITY

DSM III-R* criteria
Mild: although work or social activities are significantly impaired, the capacity for independent living remains, with adequate personal hygiene and relatively intact judgement.
Moderate: independent living is hazardous, and some degree of supervision is necessary.
Severe: activities of daily living are so impaired that continual supervision is required (e.g. unable to maintain minimal personal hygiene, largely incoherent or mute).

*DSM III-R: Diagnostic and Statistical Manual of Mental Disorders, third edition, revised.

4. When should symptomatic treatment be stopped?

A trial of treatment withdrawal should be considered when the harm outweighs the benefit. Examples include intolerable or serious side effects, and progression of disease despite optimizing treatment. This should be undertaken only after careful discussion with the patient and caregiver. When attempting withdrawal, it is important to monitor closely for any deterioration so that the medication can be quickly reinstated to regain the same level of symptomatic effect.

NEW FRONTIERS IN DEMENTIA TREATMENT

Recent advances in understanding disease pathogenesis have led to the development of new therapeutic approaches that might modify the underlying specific disease process (i.e. disease-modifying treatment as opposed to current symptomatic treatment). For instance, in Alzheimer's disease, a wide array of anti-amyloid and neuroprotective therapeutic approaches are under investigation on the basis of the hypothesis that amyloid beta (Aβ) protein plays a pivotal role in disease onset and progression and that secondary consequences of Aβ generation and deposition, including tau hyperphosphorylation and neurofibrillary tangle formation, oxidation, inflammation, and excitotoxicity, contribute to the disease process. Investigations are currently underway to evaluate the effectiveness of disease-modifying agents that might block the cascade of events comprising AD pathogenesis, such as anti-amyloid strategies, anti-tau strategies, limiting oxidation and excitotoxicity, and controlling inflammation[22]. With the advent of disease-modifying therapy, there will be an increasing emphasis on accurate clinical characterization in the earlier stages of disease such as MCI, and the development of methods and trial designs to effectively identify and test promising candidate agents[23].

REFERENCES

1. Asia Pacific Members of the Alzheimer's disease International. Dementia in the Asia Pacific Region: The epidemic is here. Alzheimer's Disease International 2006.
2. Miller ER, Pastor-Barriuso R, Dalal D, et al. Meta-analysis: high dosage vitamin E supplementation may increase all-cause mortality. Ann Intern Med 2005;142:37-46.
3. Feldman HH, Doody RS, Kivipelto M, et al. Randomized controlled trial of atorvastatin in mild to moderate Alzheimer disease. Neurology 2010;74:956-64.
4. Freud-Levi Y, Eriksdotter-Jo¨nhagen M, Cederholm T, et al. Omega-3 Fatty Acid treatment in 174 patients with mild to moderate Alzheimer Disease: OmegAD Study. A Randomized Double-blind Trial. Arch Neurol 2006;63:1402-8.
5. van de Rest O, Geleijnse JM, Kok FJ, et al. Effects of fish oil on cognitive performance in older subjects: A randomized controlled trial. Neurology 2008;71:430-8.
6. Birks J, Grimley Evans J. Ginkgo biloba for cognitive impairment and dementia. Cochrane Database Syst Rev 2007;(2):CD003120..pub2.
7. DeKosky ST, Williamson JD, Fitzpatrick AL, et al. Ginkgo Evaluation of Memory (GEM) Study Investigators. Ginkgo biloba for prevention of dementia: a randomized controlled trial. JAMA 2008;300:2253-62.
8. Winblad B, Kilander L, Eriksson S, et al. Donepezil in patients with severe Alzheimer's disease: double-blind, parallel-group, placebo-controlled study. Lancet 2006;367:1057-65.
9. Winblad B, Wimo A, Engedal K. A 3-year study of donepezil therapy in Alzheimer's disease: effects of early and continuous therapy. Dement Geriatr Cogn Disord 2006;21:353-63.
10. McKeith I, Del Sen T, Spano P, et al. Efficacy of rivastigmine in dementia with Lewy bodies: a randomized, double-blind, placebo-controlled international study. Lancet 2000;356:2031-6.
11. Emre M, Aarsland D, Albanese A, et al. Rivastigmine for dementia associated with Parkinson's disease. N Engl J Med 2004;351:2509-18.
12. Overshott R, Burns A. Treatment of dementia. J Neurol Neurosurg Psychiatry 2005;76(S):v53-v59.
13. Winblad B, Grossberg G, Frolich L , et al. IDEAL. A 6-month double-blind, placebo-controlled study of the first skin patch for Alzheimer's disease. Neurology 2007;69(Suppl 1):S14-S22.
14. Reisberg B, Doody R, Stoffler A, et al. Memantine Study Group. Memantine in moderate-to severe Alzheimer's disease. N Engl J Med 2003;348:1333-41.
15. Tariot PN, Farlow MR, Grossberg GT, et al. Memantine treatment in patients with moderate to severe Alzheimer disease already receiving donepezil: A randomized controlled trial. JAMA 2004;291:317-24.
16. Jones RW, Bayer A, Inglis F, et al. Safety and tolerability of once-daily versus twice-daily memantine: a randomized, double-blind study in moderate to severe Alzheimer's disease. Int J Geriatr Psychiatry 2007;22:258-62.

17. Raschetti R, Albanese E, Vanacore N, et al. Cholinesterase inhibitors in mild cognitive impairment: A systematic review of randomised trials. PloS Med 2007;4(11):e338. doi:10.1371/journal.pmed.0040338.
18. Courtney C, Farrell D, Gray R, et al. Long term donepezil treatment in 565 patients with Alzheimer's disease (AD 2000): randomized double-blind trial. Lancet 2004;363:2105-15.
19. Ridha BH, Josephs KA, Rossor MN. Delusions and hallucinations in dementia with Lewy bodies: worsening with memantine. Neurology 2005;65:481-2.
20. Mendez MF, Shapira JS, McMurray A, et al. Preliminary findings: Behavioural worsening on donepezil in patients with frontotemporal dementia. Am J Geriatr Psychiatry 2007;15:84-7.
21. Hake AM. The treatment of Alzheimer's disease: the approach from a clinical specialist in the trenches. Sem Neurol 2002;22:71-4.
22. Salloway S, Mintzer J, Weiner MF, et al. Disease modifying therapies in Alzheimer's disease. Alzheimer's & Dementia 2008;4:65-79.
23. Cumming JL, Doody R, Clark C. Disease-modifying therapies for Alzheimer's disease. Challenges to early intervention. Neurology 2007;69:1622-34.

LEARNING POINTS

- **All dementia patients should be evaluated for suitability of pharmacological strategies to address the underlying disease, enhance cognitive symptomatology, and treat attendant behavioural complications.**
- **Once a definitive diagnosis of dementia has been made, the key factors determining choice of symptomatic treatment are dementia etiology and stage of severity.**
- **The pre-requisite to skilful use of symptomatic treatment is a firm knowledge of the pharmacokinetic and dosing properties, side effect profile and expected benefits of such medications.**
- **The decision to initiate costly symptomatic treatment should be individualized and always made in conjunction with the patient and caregiver.**

UNIT NO. 4

FAMILY CAREGIVERS AND CAREGIVING IN DEMENTIA

Dr Dennis Seow, Dr Philip Yap Lin Kiat

ABSTRACT

Caregiver interventions have been shown to reduce caregiver depression, burden of care, and improve their health and quality of life. Caregiver support also benefits the person with dementia (PWD). It is important to recognize that caregivers too need caring. Caregivers of PWD are usually middle-aged daughters and sons followed by spouses. Foreign domestic helpers also play a pivotal role in Singapore. Stressors arising from caregiving change at different stages of the disease. As the disease progresses into the advanced stages, stress from having to deal with behavioural problems can lessen as the burden from coping with functional impairments increases. For this reason, caregiver interventions should be stage appropriate. There is a need to work towards creating a positive experience in the GP consultation with the important elements of early diagnosis, providing stage specific information and interventions, and up-to-date information on dementia resources available in the community.

SFP2011; 37(3) (Supp 1) : 24-29

INTRODUCTION

Caregivers are an integral part of the support and care of the person with dementia (PWD). Caregiver interventions been shown to reduce caregiver depression, burden of care and improve their health and quality of life.

More importantly, intervening through the caregiver also impacts quality of life, behavioural changes, medication compliance and rates of institutionalization in PWD as has been shown in several studies. In dementia care, two tenets are espoused: (1) Treatment through both pharmacological and non-pharmacological means (2) Treating the PWD as well as the caregiver.

The importance of caregivers cannot be over-emphasised. In Singapore, based on the findings of a study conducted by Alzheimer's Disease International in Asia Pacific[1], the prevalence of dementia in 2020 and 2050 will approximate 53,000 and 187,000 respectively. By including caregivers in the tally, this means an additional 53,000 and 187,000 caregivers and/ or families being affected as well.

DENNIS SEOW, Consultant, Department of Geriatric Medicine, Singapore General Hospital

PHILIP YAP LIN KIAT, Senior Consultant, Department of Geriatric Medicine, Khoo Teck Puat Hospital

It is paramount in dementia care not to neglect the caregiver. He/she is often the silent patient or sufferer. Caregivers too need caring. Caring for caregivers includes: (1) continual assessment of their needs, (2) support in the form of education, empowerment and enablement, (3) Helping them look after their own health.

In ageing Singapore, General Practitioners (GPs) will play an increasing role in meeting the healthcare needs of the silver generation. In addition, care of PWD by GPs will gain increasing importance in light of the fact that there will be too many patients and insufficient specialists to meet the need.

Can GPs make a difference to dementia care? A study by Fortinsky[2] showed that when the symptoms of dementia emerge, patients and caregivers often turn first to their primary care physician for answers to questions about memory loss and to obtain a diagnosis. As GPs are in regular contact with their patients, they are in a position to recognize early signs of cognitive decline in them. They also have the benefit of having a long-standing relationship with their patients. Therefore, a GP is well poised to provide holistic care for the PWD and his caregiver. GPs will have an increasingly important role in contributing to the care of PWD and their caregivers in the years to come.

CAREGIVERS

Local studies have shown that the majority of caregivers are women[3,4,5,32]. Caregivers are usually middle-aged and mostly children followed by spouses[4,5,32]. Many caregivers rely on other family members for additional help. About half hold a full-time or part time job[3]. In the Chinese family, there is also a hierarchy of expectation that the relative will be a caregiver in the order of: spouse, daughter, daughter-in-law, son and other kin[3]. As a reflection of changing social norms and disintegration of the extended family, quite often it is usually the unmarried daughter or son who is left to care for the older patient.

Besides family members, most families engage the help of a foreign domestic helper (usually from Philippines, Indonesia or Myanmar). This is especially true in Singapore where a local study showed about 50% of families of PWD engage foreign domestic help[32]. This has led to a dichotomy of caregiving responsibilities. The foreign domestic helper does the physical caregiving while the children provide financial support and make the decisions regarding care In large families, it is not uncommon for the PWD and foreign domestic helper to rotate and stay in the homes of different children for certain periods of time. For smaller families, the foreign domestic helper is sometimes the only person who resides with the PWD in a one or two room Housing Development Board (HDB) flat. It is thus important to look into the needs of domestic helpers as they often assume the role of the main caregiver and may be

more aware of cognitive and behavioural changes in the PWD in the course of the illness.

Factors that affect caregiver performance

Demographic characteristics that influence caregiver performance include: age, gender, healthcare status, kin relationship and racial/ethnic background (Table 1)[6]. Older spouses have more caregiver stress and burden as they themselves are often beset with ill-health or even become cognitively impaired themselves. Women and wives tend to have more psychological stress in caregiving[7,8]. The relationship to the PWD also matters. Daughters-in- law who have a difficult relationship with their mothers-in-law often have more caregiver stress[3]. With regards to ethnicity and caregiving, not much is known locally; although Malay families appear more willing to take up caregiving roles for their relative with dementia..

Table 1. Demographic characteristics that influence caregiver performance[6]

- Age.
- Gender.
- Healthcare status.
- Kin relationship.
- Racial/ethnic background.

Stressors from caregiving

As dementia progresses, caregivers can experience greater burden. (Table 2). A local study[4] done in 1999 on the burden of caregiving in mild to moderate dementia revealed that even in the earlier stages of dementia, 48% of caregivers reported the caring process to be a difficult one. More importantly, these difficulties were pertinent enough to be significantly associated with the intention to institutionalize the PWD. Behavioural problems featured more prominently than functional disabilities in relation to the caregivers' experience of burden. The converse was seen in another local study[10] done on patients with more advanced dementia. Taken together, these studies suggest that as dementia progresses and behavioural problems lessen in intensity, functional impairments become more pronounced. Caregivers therefore encounter the changing issues that emerge at different stages of the disease.

Understanding the background, personality and life history of the PWD plays a crucial role in helping the caregiver understand the reasons behind his behaviour. Often, behavioural issues may seem bizarre but with thoughtful reflection of the circumstances surrounding the emergence of the behavior in the PWD in the light of his past, one can often find meaning and understanding. This insight gained can direct the caregiver to find means to offer comfort and solace to the PWD who may be feeling threatened, insecure and vulnerable when he exhibits seemingly "difficult behavior".

The impact of caregiving on the caregiver can also be felt in indirect ways (Table 3). Caregivers are often torn between the needs of the patient and that of their nuclear families. Primary caregivers may suffer restricted social lives and have less time for career pursuits, hobbies and other social activities. This can lead feelings of disenchantment, disdain and even despair. Caregiver burnout can thus result and this needs to addressed early (Table 4).

Impact of caregiving on Caregivers

The impact of caregiving on the caregivers can be divided into 4 categories:

(1) Impact on Emotional Well-Being

In a previous study on Chinese families of PWD in Singapore, behavioural symptoms were significantly related to caregiver stress. Overseas studies also paint a similar picture, more than 40% of family and other unpaid caregivers of PWD rate the emotional stress of caregiving as high or very high. In general, up to one-third of family caregivers experience symptoms of depression. However, in the local study, 47% of caregivers who had caregiving problems experienced significant depression.

The notion that nursing home placement would bring relief of stress may actually not be the case in some families. One study found that family caregiver stress and depression were just as high after the placement as before placement. While the physical burden of caregiving may be relieved with institutionalization of the PWD, the emotional burden of guilt and feeling that one is not doing enough for the PWD often persists.

Table 2. Stressors arising directly from caregiving (primary stressors)[9]

Pertaining to the PWD:
- Severity of cognitive problems.
- Functional disability.
- Behavioural problems.
- Resistiveness to care.

Table 3. Stressors arising indirectly from caregiving (secondary stressors)[9]

Pertaining to the caregiver:
- Restriction of social life/leisure time.
- Role strain and role conflict
- Financial strain.
- Family conflict.

Table 4. Factors associated with caregiver burnout[6]

- Feeling overwhelmed, angry or frustrated by caregiving responsibilities.
- Feeling frustrated or angry with the PWD.
- Feeling that life or health has suffered since becoming a caregiver.
- Feeling that one is not doing a good job.
- Feeling that one's efforts do not matter or are futile.

(2) Impact on the Caregiver's health

In a local study[3] involving 50 family caregivers of Chinese PWD, 56% had poorer self rated health based on the General Health Questionnaire (GHQ) and that correlated significantly with incontinence, delusion, hallucination, agitation, sleep disturbance and depression in the PWD.

Caregivers of PWD are more likely than non-caregivers to report their health to be fair or poor[11,12].

Caregivers are also more likely than non-caregivers to have high levels of stress hormones,[12,13,14,15], reduced immune function[12,16], slow wound healing[17], new onset of hypertension[18] and coronary heart disease[19]. The impact on health can also be demonstrated at the chromosomal level: caregivers of Alzheimer's disease patients have significantly shorter telomeres on average than other people of the same age and gender[20].

(3) Impact on the Caregiver's employment

Many caregivers often have to reduce working hours, take time off or quit work because of caregiving responsibilities. One study found that 57% of caregivers were employed full time or part time. Of those employed, two-thirds had to go in late, leave early or take time off because of caregiving; 18% had to take leave of absence; 13% had reduced hours; and 8% turned down promotions[21]. Clearly, loss of income and employment adds to the caregiver burden as well.

(4) Impact on Caregivers' finances

Locally, many caregivers exhaust their finances, including their medisave accounts, in providing care for the PWD throughout the disease course. Besides food and basic necessities, other out-of-pocket expenses include medications, day care, foreign domestic helper employment, nursing home stay, home medical and nursing services as well as ancillary services such home help and meals delivery.

Positive aspects of caregiving

The positive aspects of caregiving are often overlooked. Physicians can help the caregivers identify and emphasise the positive aspects of caregiving[6]. Cohen found that 73% of her subjects could state at least one positive aspect of caregiving[22]. A local study on caregiving gains identified 3 areas of gains: (1) Personal growth (2) Gains in relationship and (3) Higher level gains[23]. Caregivers can derive personal satisfaction and meaning in caregiving in knowing that their actions can promote positive situations and avoid negative ones[24]. They also gain new perspectives and a sense of purpose in life. The degree of meaningfulness in caregiving was also correlated with the presence of depression in a study by Noonan and Tennstedt[25].

GPs can certainly help the caregiver identify the positive aspects of caregiving. This will boost morale of caregivers and also provide opportunities for the GPs to detect low moods, burnout and depression[9] amongst caregivers, especially when they are persistently pessimistic and unable to see the positive in providing care for the PWD.

CAREGIVERS' EXPERIENCES WITH GPs

Caregivers report mixed experiences with GPs. A positive experience can bring about earlier detection and diagnosis of dementia, appropriate early intervention, reduction of caregiver stress and contribute to the overall holistic care of the PWD and caregiver alike. A negative experience often brings much frustration and stress on caregivers besides delay in diagnosis and treatment.

A small novel study done on GPs in Australia in 2008 focused on patients' and caregivers' experiences with GPs in settings where GPs provided a wide range of services in the absence of dementia specialist services[26]. The themes explored included diagnosis, cognitive testing, dementia knowledge, caregiver support, treatment, medication compliance. Below are some of the findings.

Diagnosis

Twenty-five percent (5/20) respondents reported prompt diagnosis by their GPs. The rest had delays of 1-8 year intervals between onset of symptoms and diagnosis. Three patients were aware something was wrong but only one was offered investigations. Two were frustrated when the diagnosis was initially refuted by their GPs.

Dementia Knowledge

Out of 4 respondents, two had positive comments on their GPs' ability to offer prompt diagnosis and access to support. Two had negative comments which were attributed to difficulties in accessing help and GPs' lack of knowledge about dementia.

Caregiver support

The interviews focused on caregiver support, discussing on issues ranging from the help they received to the frustration of being unable to access help. Many positive comments demonstrated that the most reliable, up-to-date source of information about dementia support services came from other caregivers who had firsthand knowledge of pitfalls and benefits, and not from the GP. A quarter (n=5) of the interviews produced negative comments about the services received, demonstrating the significant impact of negative experiences.

'Not a damned thing happened for us. That was the hard part because she had no help. You didn't know what help there was.' (A daughter)

Medication compliance

Medication compliance was an issue in nearly half the cases (n=9). This was a major problem when the patient was self caring.

This study showed that the diagnosis of dementia may often be missed in routine consultations. More importantly it also showed that patients in the early stages may be aware of their condition and thus it was important to listen to them. With regards to dementia knowledge, "most PWD trusted their GPs to be informed about the disease and deficiencies in GP knowledge led to delayed diagnosis and consequently less optimal support and management."….."Negative comments were also received when GPs failed to identify the disease or arrange for support." "Caregivers appreciated a diagnosis that explained what was happening, even when providing a prognosis was difficult." For caregiver support, "PWD and caregivers expected their GPs to offer appropriate care and access to dementia services and wished for GPs to be better informed about support services." It also showed that many older persons (and caregivers) valued a GP who could inform them.

Locally, some may have similar experiences with their GPs and this reinforces the view that GPs are well placed to initiate early support, diagnosis and treatment. In addition, medication compliance is a constant issue with PWD and thus caregivers need to be encouraged and supported to take an active part in the assisting with administering medication.

OPTIMAL CARE AND THE HEALTH CARE TRIAD

In Singapore today, GPs have a wealth of resources to draw from to help in providing care and care to PWD and their families. Against a setting of limited consultation time in primary care, evolving symptoms with disease progression in the PWD, possible negative attitudes towards dementia diagnosis and treatment, inadequate reimbursement and lack of incentive for in-depth consultations, the quality of interaction between the GP, PWD and caregiver(s) is most critical for optimal dementia care. A review by Holmes and Adler[27] provided a few pointers that could enhance this interaction.

These include (1) being alert to the cognitive and behavioural changes in the PWD (e.g. missed appointments, poor compliance with medications, frequent telephone calls to the clinic, missed payments and a family member accompanying the PWD to the clinic visit when there was none before), (2) involving persons with early dementia in their own care, (3) identification of a principal caregiver, (4) progressive involvement of the caregivers in the care plan as the disease progresses. The relationship of the GP with the PWD and caregiver thus forms a critical "health care triad"[2,28] which is essential for optimal dementia care and management[29].

MANAGEMENT AND SUPPORT OF CAREGIVER

When and how?

The needs of the PWD change throughout the course of the illness, this means that support and intervention for the caregiver would also need to be different at various stages of dementia. These key stages are elaborated herein.

(1) Diagnosis and disclosure.
(2) Early stage disease.
(3) Middle stage disease.
(4) Final stage disease.
(5) Bereavement.
(6) Referral and use of community resources.

(1) Diagnosis & Disclosure

Patients and families want an accurate and clearly explained diagnosis and desire to better understand the course of the illness over time[30]. "Specifically, caregivers want their physicians to listen to their concerns, devote more time to discussing diagnosis and what it means, and include the PWD even if he or she may not fully understand."[30] Research has documented that these factors are closely linked to with caregiver satisfaction[6].

The disclosure process should be tailored to the patient and caregiver dyad. While most physicians and caregivers prefer to focus on discussions on memory problems and safety issues rather than the term Alzheimer's disease; most families want more specific information regarding the diagnosis and prognosis as mentioned above[30].

(2) Early stage disease

Accepting and adapting to the role of a caregiver is the primary goal for most caregivers at this stage[6]. Caregivers can be in denial during this stage and fearful of grappling with the unknown. Time taken to educate and empower the caregiver certainly helps the caregiver to cope better. Simple explanations with written materials, brochures and books, and information from caregiving websites are useful. Repetition of important information over several visits is also helpful. Referrals to caregiver support programmes is a good way for caregivers to seek peer support and advice.

Other care initiatives that can established with the caregiver at this stage include:

- Adaptation.
- Financial, legal planning and advance directives.
- Establishment of support system for the caregiver.

<u>Adaptation</u>

Becoming a caregiver is often unplanned, life-changing and a long term event. Spouses or children have to discard old roles and take on new ones, for example a son becoming the caregiver and decision maker for the father. Emotional support & empathy are crucial at this stage.

<u>Financial, legal planning and advance directives</u>

Advice should also be given to the PWD and caregiver on sorting out financial issues such as bills, CPF / bank accounts, and insurance, With the enactment of the Mental Capacity Act, PWD who are still mentally competent can assign health care decision making designees (known as donees). Other considerations include advance medical directives, will and estate planning.

Establishment of support system for the caregiver

Helping the caregiver look after him/herself is also important. GPs can play a role in involving extended family members and friends in caregiving so as to relieve the burden on the primary caregiver(s). Besides caregiver support groups, caregivers can be encouraged to seek support through religious or voluntary groups and even close neighbours.

(3) Middle stage disease

This stage is characterized by the emergence of more behavioural / personality changes in addition to progressive cognitive and functional decline. Most caregivers face significant burden and need more help at this stage. However, some caregivers may not see that they need more help and accepting help from others also presents an issue. The local caregiver study[3] revealed that Chinese caregivers relied more on family support and less on psychogeriatric services for fear of 'losing face'. Hence, caregivers may delay seeking help till a crisis or burnout occurs.

GPs are well placed to offer assistance. GPs need to be on the alert for caregiver distress, depression and burnout (Table 4). The ability of the caregiver to cope depends on his personal coping resources as well as the amount and quality of formal and informal support[3]. Early referral to the appropriate caregiver resources is recommended and the GP can help the caregiver select the service appropriate for his needs. These resources can be specific to the PWD or primarily targeted at caregivers. Regular contact with the GP or attending specialist can help the caregiver tide over difficult periods.

(4) Late stage disease

At this stage, patients are often debilitated and require round-the-clock care for their activities of daily living. Caregivers are faced with decision making and preparation for various end-of-life issues and trust their physician to guide them in making difficult choices. These issues include do-not-resuscitate orders, tube feeding, rational use of medications and specialist palliative care.

(5) Bereavement

Bereavement on the part of the family caregiver often begins from in the earlier stages of dementia when the PWD progressively ceases to be the person he used to be. Depression is prevalent especially among caregivers who experience loss of companionship and a treasured relationship[6] as the PWD becomes increasingly foreign and distant. Studies show that even after death, caregivers can still have grief reactions up to 3 years after death[9] of the PWD. GPs can provide counsel and support for the caregiver trying to come to terms with the losses in dementia.

(6) Referral and use of community resources

Besides information from hospital-based memory clinics, the websites of Alzheimer's Disease Association of Singapore (www.alzheimers.org.sg) and the Agency for Integrated Care (ww.aic.sg) provide much information on community resources and services. ADA also runs a helpline for caregivers and the general public.

Additional tips in meeting the needs of the caregiver

- Establish contact and liaise with the specialist to gain greater understanding of the needs of the PWD and his caregiver.
- Understand the life history and personality of the patient. This is cardinal to providing person centred care[31]. Oftentimes, one can understand the reason behind certain behavioural issues in the PWD in the light of his past. This can help the caregiver achieve greater understanding of the PWD, cope better and in turn reduce caregiver stress.
- Provide information to caregivers appropriate to their situation and relevant to the problems consistent with the patient's stage of dementia. Divide important information into "bite-sized" portions over several visits.
- Offer a listening ear to the caregiver and allow time for him/her to ventilate; this can be therapeutic for the caregiver.
- Enquire about the caregiver's health and coping regularly as some caregivers may not volunteer information about their own well-being.

CONCLUSION

Caregiver interventions have been proven to improve caregiver coping and reduce caregiver depression and burden. Caregiver support also benefits the PWD. It is important to recognize that caregivers too need caring. The GP has an indispensable role in holistic dementia care. We need to make strides towards a more proactive role by GPs in dementia care in Singapore.

REFERENCES

1. Dementia in the Asia Pacific Region: The Epidemic is here. Report by Access Economic Pty Limited for Asia Pacific Members of Alzheimer's Disease International. 21st September 2006. (http://www.accesseconomics.com.au/publicationsreport/search.php?searchby=year&searchfor=2006)

2. Fortinsky RH. Health care triads and dementia care: integrative framework and future direction. Aging Ment Health. 2001;5:S35-48.

3. Kua EH, Tan SL. Stress of caregivers of dementia patients in the Singapore Chinese family. Int J Geriatr Psychiatry. 1997;12:466-9.

4. Lim PPJ, Sahadevan S, Geok KC, Anthony P. Burden of caregiving in mild to moderate dementia: An Asian Experience. Int J Geriatric Psychiatry. 1999;11:411-20.

5. Yap LKP, Seow CCD. Caregiving in dementia. Poster presentation in National Healthcare Group Annual Scientific Congress 2004. Abstract in Ann Academy of Medicine Singapore. 2004;33(Suppl):S148.

6. Morrison AS, Rabins PV. Comprehensive review of caregiving for Health Care Professional. In: Morris JC, Galvin JE, Holtzman DM, editors. Handbook of Dementing Illnesses Second Edition. Informa Healthcare. 2006;Chapter 22:pages 394-403.

7. Fitting M, Rabins P, Lucas JM, Eastham J. Caregivers for dementia patients: A comparison of husband and wives. Gerontologist. 1986;26:248-52.

8. Collins C, Jones R. Emotional distress and morbidity in dementia carers: a matched comparision of husband and wives. Int J Geriatr Psychiatry. 1997;12:1168-73.
9. Anehensel C, Pearlin I, Mullan J, Zarit S, Whitlatch C. Profiles in caregiving. The Unexpected Career. San Diego, CA: Academia Press, Inc. 1995.
10. Sahadevan S, Lim PPJ, Choo PWJ. Dementia in the hospitalized elderly – A study of one hundred consecutive cases in Singapore. Int J Geriatr Psychiatry. 1999;14:261-5.
11. Schulz R, O'Brien AT, Bookwala J, Fleissner K. Psychiatric and physical morbidity effects of dementia caregiving: Prevalence, correlates and causes. Gerontologist. 1995;35(6):771-91.
12. Vitaliano PP, Zhang J, Scanlan JM. Is caregiving hazardous to one physical health? A meta-analysis. Psychological Bulletin. 2003;129(6); 946-72.
13. Lutgendorf SK, Garand L, Buckwalter KC, Reimer TT, Hong S-Y, Lubaroff DM. Life stress, mood disturbance, and elevated Interluekin-6 in healthy older women. Journal of Gerontology: Medical Sciences. 1999;54A(9):M434-9.
14. Von Kanel R, Dimsdale JE, Mills PJ, Ancoli-Israel S, Patterson TL, Mausback BT et al. Effect of Alzheimer caregiving stress and age on frailty markers Interleukin-6, C-Reactive Protein and D-Dimer. Journal of Gerontology: Medical Sciences. 2006;61A(9):963-9.
15. Kiecolt-Glaser JK, Glaser R, Gravenstein S, Malarkay WB, Sheridan J. Chronic stress alters the immune response to influenza virus vaccine in older adults. Proc of Nat Acad of Sci. 1996;93:3043-7.
16. Kiecolt-Glaser JK, Dura JR, Speicher CE, Trask OJ, Glaser R. Spousal caregivers of dementia victims: Longitudinal changes in immunity and health. Psychosomatic Medicine. 1991;53:345-62.
17. Kiecolt-Glaser JK, Marucha PT, Mercado AM, , Malarkay WB, Glaser R. Slowing of wound healing by psychological stress. Lancet. 1995;346(8984):1194-6.
18. Shaw WS, Patterson TL, Ziegler MG, Dimsdale JE, Semple SJ, Grant I. Accelerated risk of hypertensive blood pressure recordings among Alzheimer caregivers. Journal of Psychosomatic Resaerch. 1999;46(3):215-27.
19. Vitaliano PP, Scanlan JM, Zhang J, Savage MV, Hirsch IB, Siegler I. A path model of chronic stress, the metabolic syndrome and coronary artery disease. Psychosomatic Medicine. 2002;64:418-35.
20. Damjanovic AK, Yang Y, Glaser R, Kiecolt-Glaser JK, Nguyen H, Laskowski B et al. Accelerated telomere erosion is associated with a declininn immune function of caregivers of Alzheimer's disease patients. Journal of Immunology. 2007;179:4249-54.
21. Alzheimer's Association and National Alliance for Caregiving. Families Care: Alzheimer's Caregiving in the United States, 2004, accessible at www.alz.org.
22. Cohen C, Olantonio A, Vernich L. Positive aspects of caregiving: rounding out the caregiver experience. Int J Geriatric Psychiatry 2002;17:184-88
23. Netto NR, Goh YNJ, Yap LKP. Growing and gaining through caring for a loved one with dementia. Dementia 2009; 8:245-61.
24. Nolan M, Grant G, Keady J. Understanding family care. A Multidimensional model of Caring and Coping. Philadelphia: Open university Press, 1996.
25. Noonan A, Tennstedt S. Meaning in caregiving and its contribution to caregiver well being. Gerontologist. 1997;37:785-94.
26. Millard F. GP management of dementia. A consumer perspective. Aust Fam Phys. 2008 Jan/Feb;37(1/2):89-92.
27. Holmes SB, Adler D. Dementia Care: Critical Interactions among Primary Care Physicians, Patients and Caregivers. Prim Care Clin Office Practice. 2005;32:671-82.
28. Haug MR. Elderly patients, caregivers and physicians: Theory and research on health care tirds. J Health Soc Behav. 1994;35:1-12.
29. American Medical Association. In: Gattman R, Seleski M, editors. Diagnosis, management and treatment of dementia: A practical guide for physicians. Chicago: American Medical Association;1999.
30. Boise L, Connell C. Diagnosing dementia :What to tell the patient and family. Geriatrics and Aging. 2005;8(5):48-51.
31. Kitwood T. Dementia Reconsidered. Open University Press, Buckingham 1997.
32. Profiling the dementia family carer in Singapore. [last accessed on 7 Sept 2011] . http://www.alz.org.sg/about-dementia/research

LEARNING POINTS

- **Support for caregivers has been shown to reduce caregiver depression, burden of care, and improve their health and quality of life.**
- **Caregiver interventions also benefit PWD.**
- **Caregivers of PWD are usually middle-aged daughters and sons, followed by spouses. Foreign domestic helpers often provide direct care to the PWD.**
- **Information given to caregivers should be tailored to their specific needs.**
- **GPs can work towards a more proactive role in dementia care in Singapore.**

UNIT NO. 5

CHRONIC DISEASE MANAGEMENT PROGRAMME ON DEMENTIA

Dr Chong Mei Sian

ABSTRACT

From 1 Nov 2011, Dementia will be included into the CDMP. This is expected to bring about better health outcomes for patients who will have better control of their conditions with close supervision from their doctors. Together with Bipolar disorder to be added in, there will be a total of 10 chronic diseases that could use Medisave for chronic disease management. For new diagnosis of dementia or suspected cognitive impairment, when in doubt, it is advisable to consult or refer to a geriatrician/ psychiatrist/ neurologist for confirmation as these diagnoses carry long term medical and legal implication. Existing patients with dementia in the RHs or IMH are recommended to be assessed by geriatricians/ psychiatrists/their primary care physician to be suitable for follow-up in the community by GP clinics or polyclinics, which are participating in Shared Care or GP Partnership Programmes. Clinics enrolled under the Medisave for CDMP are required to provide all the essential care components detailed in the DMP. The basis for diagnosis and management of dementia should conform to the prevailing MOH Clinical Practice Guidelines. There is a list investigations, drugs and therapies for the evaluation and management of dementia for which Medisave use can be allowed. As part of the national effort under this Programme, the Health Promotion Board has prepared Patient Education Booklets for dementia. Participating medical institutions must monitor the quality of care that patients receive.

SFP2011; 37(3) (Supp 1) : 30-41

UPDATE ON USE OF MEDISAVE FOR CHRONIC DISEASE MANAGEMENT PROGRAMME (CDMP)

The use of Medisave for chronic disease management programme (CDMP) was implemented on 1 Oct 2006 for Diabetes. This was extended to three additional diseases in Jan 2007, namely Hypertension, Lipid Disorders and Stroke. Asthma and Chronic Obstructive Pulmonary Disease (COPD) were added in Apr 2008. Since 1 Oct 2009, CDMP was also extended to cover Schizophrenia and Major Depression.

Starting with just over 7000 patients in Oct 2006, the CDMP has grown and as of Dec 2010, there are about 112,000 patients in this Programme, with an annual Medisave withdrawal of about S$27 million in 2010.

CHONG MEI SIAN, Senior Consultant, Department of Geriatric Medicine, Tan Tock Seng Hospital

Submission of clinical data is an essential component of the Programme. Participating clinics are required to monitor the quality of care that patients receive and submit clinical data to the Ministry of Health (MOH). To facilitate quality improvement, the clinical data submitted had been routinely fed back to the clinic via the online CDMP outcome reports through the Mediclaim system since 2008.

INCLUSION OF DEMENTIA INTO CDMP

From 1 Nov 2011, Dementia will be included into the CDMP. This is expected to bring about better health outcomes for patients who will have better control of their conditions with close supervision from their doctors.

It is recognised that the treatment of chronic diseases is costly when administered collectively over a long period. However, this Programme will help reduce out-of-pocket payments and also reduce the barriers for patients to seek medical treatment. With the implementation of the CDMP for Dementia, GPs will be able to take on a greater role in the management of chronic diseases of their patients.

TREATMENT ALGORITHM FOR DEMENTIA

For new diagnosis of dementia or suspected cognitive impairment, when in doubt, it is advisable to consult or refer to a geriatrician/ psychiatrist/ neurologist for confirmation as these diagnoses carry long term medical and legal implication.

Patients who are already enrolled under the existing DMPs (i.e. Diabetes Mellitus, Hypertension, Lipid Disorders, Stroke, Asthma or COPD, Schizophrenia and/or Major Depression) but who also suffer from dementia, should, in addition, be enrolled into the programme. See Figure 1.

Patients who are assessed to be suitable for community follow-up will be able to use Medisave to pay for management of all these ten chronic diseases (existing rules and regulations for Medisave claims apply). Clinical outcomes will be tracked for all the DMPs that the patient has been enrolled into.

Existing patients with dementia in the restructured hospitals (RHs) or Institute of Mental Health (IMH) are recommended to be assessed by geriatricians/psychiatrists/their primary care physician to be suitable for follow-up in the community by GP clinics or polyclinics, which are participating in Shared Care or GP Partnership Programmes.

ESSENTIAL CARE COMPONENTS FOR DEMENTIA FOLLOW-UP MANAGEMENT IN DEMENTIA DISEASE MANAGEMENT PROGRAMME

Clinics enrolled under the Medisave for CDMP are required to provide all the essential care components detailed in the DMP. The basis for diagnosis and management of dementia should

Figure 1. ENROLLING PATIENTS WITH MULTIPLE CHRONIC DISEASES
(source: Annex 2-A of CDMP Handbook 2011 page 11)

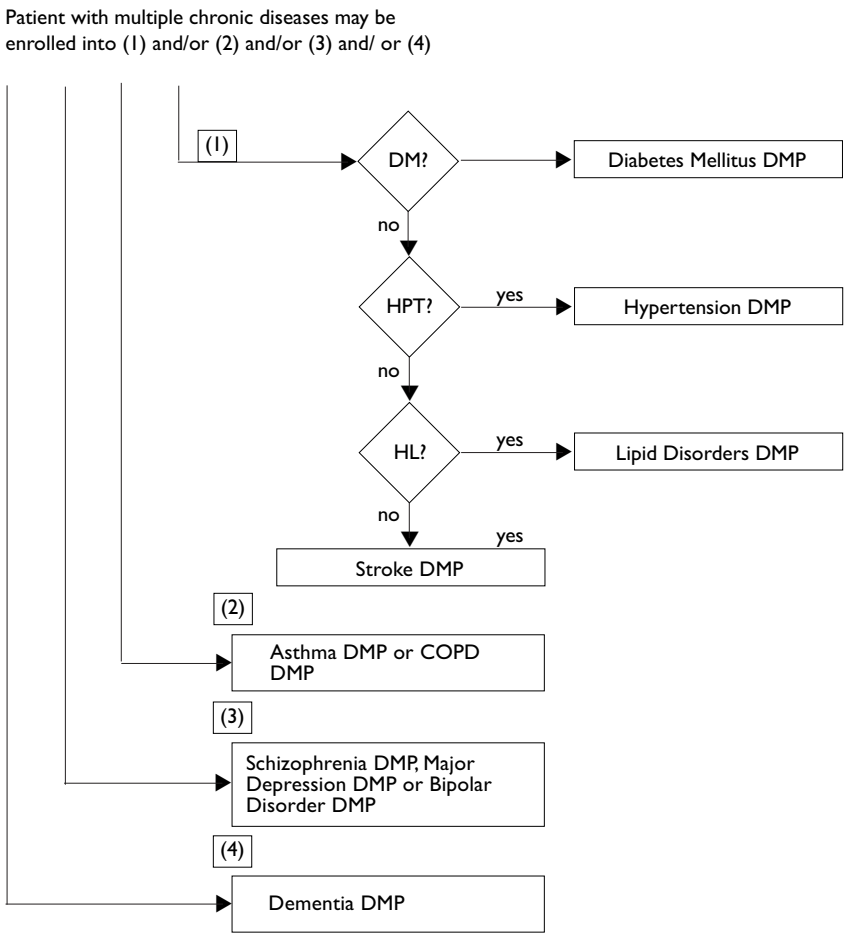
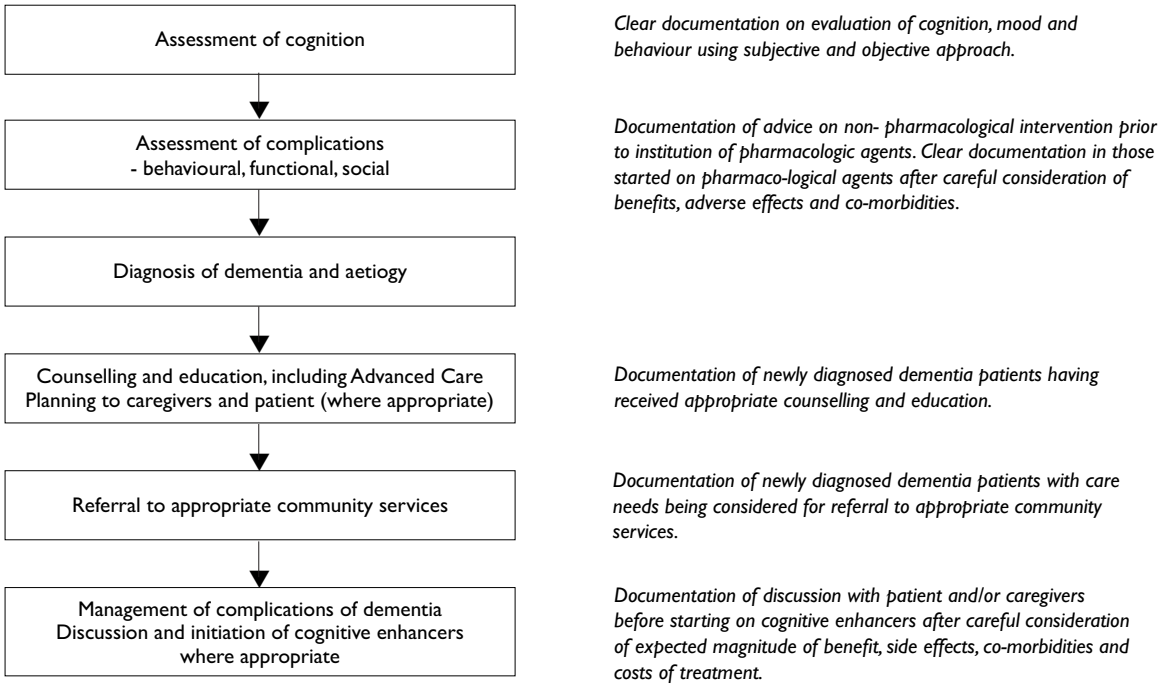


Figure 2. TREATMENT ALGORITHM FOR DEMENTIA



conform to the prevailing MOH Clinical Practice Guidelines. Shared Care Programmes or GP partnership programme with an RH must provide the essential care components for the continuing evaluation and management of dementia and bipolar disorder as set out in the Tables 2.1 and 2.2.

The care components in each DMP are recommended by the Clinical Advisory Committee appointed by MOH. These care components are recommended based on current available medical evidence

Some clinics have found it administratively easier to package their services for their patients. Packages should contain the care components detailed in the DMPs. Additional components, if any, can only be offered as add-ons.

Figure 2 shows the treatment algorithm for dementia. Details regarding each of the essential care components can also be found in the MOH Clinical Practice Guidelines, available at http://www.moh.gov.sg/mohcorp/publications.aspx?id=16266.

Medisave can also be used for doctor follow-up, nurse follow-up evaluation, physiotherapy, occupational therapy, speech therapy, home visit evaluation as clinically indicated and ordered by the attending doctor but not for home meal delivery, transport or other non-medical aspects of care.

PATIENT EDUCATION AND MONITORING

As part of the national effort under this Programme, the Health Promotion Board has prepared Patient Education Booklets for dementia.

These materials will be distributed to all CDMP clinics for the doctors to use in patient education. Specialist Outpatient Clinics (SOCs) and Polyclinics will also use the same materials to facilitate integration of care across the various care settings.

It will be useful to explain the contents of the patient education booklet to the caregiver and patient (if appropriate) as this will help enhance the doctor-patient relationship.

GUIDELINES FOR CONTINUING CARE

To facilitate integration of care across the various levels so that patients are able to continue and receive the appropriate management of their conditions, MOH has developed the following guidelines:

Referral from Specialist to Primary Care

- Suitable patients must be assessed by specialist to be stable and suitable for community follow-up.
- They should have a clear diagnosis of dementia.
- The caregivers should have been counselled on their condition, natural history and progression of illness.
- The patients should not have significant behavioural issues or significant caregiver stress. If they have behavioural issues, these should be stable before transfer to their primary care physician.
- If prescribed antidepressant and/or antipsychotic agents, the patients should be on stable doses of these medications for at least 3 months.

Referral from Primary Care to Specialist

- GPs should refer for specialist's review, patients in whom diagnosis of dementia is uncertain. GPs should also refer for specialist's review, complicated cases of bipolar disorder such as co-morbidities, pregnancy, patients 19 years or younger or other complications which in the family physician's opinion would require specialist opinion.
- Patients who, under special circumstances, require specialist opinion for medication titration for their condition (i.e. side effects or complications from conventional medication).

Clinical Indicators for Dementia

Participating medical institutions must monitor the quality of care that patients receive. The following are for management of dementia patients after establishing diagnosis:

a) Documentation in follow-up of dementia patients
   - Documentation of assessment of memory.
   - Documentation of assessment of mood and behaviour.
   - Documentation of assessment of functional and social difficulties (if any).
   - Documentation of assessment of rehabilitation needs.

b) Consultation for CDMP Dementia

c) For patients on cognitive enhancers, objective documentation of memory assessment must be performed, by way of a bedside cognitive screening instrument (such as the Mini-Mental State Examination (MMSE) or Chinese Mini Mental State Examination (CMMSE).

d) Blood test for sodium and liver function tests (only for patients on SSRIs or mood stabilisers).

e) Full blood count (for patients on mood stabilisers or considered anti-platelet therapy).

f) Clinical parameters (HR/BP) (especially for patients on cholinesterase inhibitors and antidepressants or antipsychotic medication).

g) Physical examination of extrapyramidal side effects (for patients on antipsychotics).

h) Electrocardiogram (especially for patients being considered for or on cholinesterase inhibitor. Also for patients on antipsychotics).

For those patients with stroke and dementia:

- Documentation of thromboembolism risk assessment.
- Clinical evaluation including atrial fibrillation, cardiac mumurs and need for anti-thrombotic therapy.
- Documentation of rehabilitation need assessment.

The Clinical Practice Guidelines details the good clinical practices required in dementia evaluation and management. The documentation of the important care component process in dementia evaluation and dementia management is captured in the first two clinical parameters to indicate good clinical dementia care.

As following up patients to detect complications early and prevent the morbidity and mortality associated with complications is an important aspect of care for dementia patients, the Consultation for CDMP Dementia (at least twice per year) is a key care compliance indicator for the Programme.

For dementia patients who are prescribed antidepressants or antipsychotic medications, biochemical tests should be performed at least once yearly.

For dementia patients who are prescribed cholinesterase inhibitors and antipsychotic agents, they should have clinical parameters taken during consultation visits and if there are concerns, electrocardiogram should be done. Recent evidence has shown association of cardiac rhythm abnormalities with cholinesterase inhibitor use.

Table 2.3 summarises the clinical indicators for patients with dementia required for submission via electronic channels to MOH:

RECOMMENDED INVESTIGATIONS, DRUGS AND THERAPIES

Tables 2.1 to 2.3 lists the investigations, drugs and therapies for the evaluation and management of dementia disorder for which Medisave use can be allowed.

REFERENCE FOR FURTHER READING

MOH. Chronic disease management programme handbook for healthcare professionals, 2011.

Table 2.1. ESSENTIAL CARE COMPONENTS FOR DEMENTIA FOLLOW-UP MANAGEMENT IN DEMENTIA DISEASE MANAGEMENT PROGRAMME

	Essential Component*	Minimum Recommended Frequency (per year)	Remarks
A1	Assessment of memory (if on cognitive enhancers to document MMSE/CMMSE scores)	At least once yearly or as clinically indicated	Enquiring about memory and/or performing cognitive screening test
A2	Assessment of mood and behaviour	At least once yearly or as clinically indicated	Enquiring about mood and behaviour and initiating appropriate non-pharmacological and/or pharmacological treatment where appropriate
A3	Assessment of social difficulties and and caregiver stress	At least once yearly or as clinically indicated	Assessment and referral to care co-ordinator or medical social worker or appropriate community services
A4	Functional needs assessment	As indicated	To initiate if there are concerns with regards home safety, driving safety, reports of recurrent falls, functional decline, swallowing difficulties
A5	Clinical parameters (HR/BP)	At least once yearly or as clinically indicated	Especially patients on cholinesterase inhibitors and antidepressants or antipsychotics which might affect cardiac rhythm
A6	Blood test for sodium and liver function tests	At least once yearly or as clinically indicated	Only for patients on SSRIs
A7	Full Blood count	At least once yearly or as clinically indicated	For patients on mood stabilisers or antiplatelet
A8	Physical examination for extra-pyramidal side-effects	At least once yearly or as clinically indicated	Only for patients on antipsychotics
A9	Electrocardiogram	As indicated	Especially patients who are being considered for cholinesterase inhibitor and/or on cholinesterase inhibitor but concerns regarding heart rhythm and patients on antipsychotics

**The diagnosis of dementia needs to be already established*

Table 2.2: ADDITIONAL CARE COMPONENTS FOR PATIENT WITH DEMENTIA AND STROKE

Essential Component		Minimum Recommended Frequency (per year)	Remarks
S1	Thomboembolism Risk Assessment	Annually	Clinical evaluation including atrial Fibrillation, cardiac Mumurs and need for anti-thombotic therapy
S2	Rehabilitation need assessment	As clinically indicated	

Table 2.3 CLINICAL INDICATORS FOR PATIENTS WITH DEMENTIA FOR SUBMISSION VIA ELECTRONIC CHANNELS TO MOH

Clinical Indicator	Frequency
Documentation of: i. assessment of memory ii. assessment of mood and behaviour iii. assessment of functional and social difficulties (if any) iv. assessment of rehabilitation needs	At least once yearly or as clinically indicated
Consultation for CDMP Dementia	Twice yearly
For patients on cognitive enhancers, documentation of objective assessment of memory (MMSE or CMMSE testing or other validated instruments)	At least once yearly or as clinically indicated

Table 2.4 – DOSING INFORMATION FOR DEMENTIA PATIENTS*

Drug class	Drug name	Examples of brand names	Usual adult starting dose	Usual adult dose range (per day)	Max. adult recomm. dose (per day)
SSRI	Escitalopram	Lexapro®	5 – 10 mg/day	10 – 20 mg	20 mg
	Fluoxetine	Prozac®	10 – 20 mg OM	20 – 60 mg	80 mg
	Fluvoxamine	Faverin®	25 – 50 mg/day	50 – 300 mg	300 mg
	Paroxetine	Seroxat CR®	10 – 12.5 mg/day	12.5 – 50 mg	75 mg
	Sertraline	Zoloft®	25 – 50 mg/day	25 – 200 mg	200 mg
SNRI	Duloxetine	Cymbalta®	30 – 60 mg/day	30 – 60 mg	120 mg
	Venlafaxine	Efexor XR®	75 mg/day	75 – 225 mg	225 mg
NASSA	Mirtazapine	Remeron Soltab®	15 – 30 mg/day	15 – 45 mg	45 mg
RIMA	Moclobemide	Aurorix®	150 mg/day	150 – 600 mg	600 mg
Cholinesterase Inhibitors	Donepezil	Aricept®	2.5 – 5 mg once daily {Tablet (5 mg, 10 mg)}	5 – 10 mg	10 mg
	Rivastigmine	Exelon®	1.5 mg bid after meals {Capsule (1.5mg, 3mg, 4.5mg, 6 mg) Transdermal patch (4.6mg/24 hours, 9.5mg/24 hour)}	6 – 12 mg 4.6mg – 9.5mg (Transdermal patch)	12 mg
	Galantamine	Reminyl®	8 mg once daily after meals {PR Capsule (8mg, 16 mg and24 mg)[2] Solution (4mg/ml; 100 ml bottle)[3]}	16 – 24 mg	24 mg
NMDA Antagonists	Memantine	Ebixa®	5 mg once daily {Tablet: 10 mg, Solution: 10 mg/g oral drops (10 drops = 5 mg)}	20 mg/day (CCT[4] >60) 10 mg/day (CCT 40-60)	20 mg
Others	Bupropion	Wellbutrin SR®	150 mg OM, increase to 150 mg BD on day 4 if well tolerated	150 – 300 mg	300 mg
	Tianeptine	Stablon®	25 – 50 mg/day in 2 – 4 divided doses	25 – 37.5 mg	50 mg
	Trazodone	Trittico®	25 – 150 mg/day in divided doses	50 – 300 mg	600 mg

[2] PR: prolonged release once-a-day formulation. The immediate-release formulation has been phased out.
[3] Solution can be mixed with non-alcoholic beverage, but must be consumed immediately.
[4] Creatinine clearance

Abbreviations:
- SSRI: Selective Serotonin Reuptake Inhibitor:
- SNRI: Serotonin and Noradrenaline Reuptake Inhibitor
- NASSA: Noradrenaline and Specific Serotonin Antidepressant
- RIMA: Reversible Inhibitor of Monoamine Oxidase

Important Notes:
- For details, please consult the manufacturers most current product literature or other standard references.
- Lowest effective doses should be used. Elderly patients should be carefully initiated at lower doses of a suitable antidepressant. Individualized dosing for any antidepressant should be based on an in-depth evaluation of the individual patient's therapy requirement with considerations to issues such as contraindications, warnings, precautions, adverse reactions and interactions with other drugs.
- There are many adverse drug interactions with antidepressant drug use, please refer to drug literature for details. Some examples of potential clinically significant interactions with general medicines when initiating/increasing an antidepressant dose can be:
- Triptans (e.g. Sumatriptan), St. John's Wort: Risks of serotonin syndrome with SSRIs and related antidepressants.

- Insulins, oral hypoglycaemic agents: Risks of hypoglycaemia with some antidepressants (e.g. Fluoxetine)
- Theophylline, Clozapine: Risks of toxicity with Fluvoxamine
- Digoxin: Risks of toxicity with Fluoxetine
- Anticonvulsants: Levels affected by many antidepressants. Seizure threshold reduced by TCAs, bupropion.
- Warfarin: Risks of bleeding with many antidepressants (e.g. Fluvoxamine)
- Precautions when switching antidepressants: Other antidepressants should not be started until at least 2 weeks after Moclobemide has been stopped. Moclobemide should not be started until at least 1 week after a TCA or SSRI or related antidepressant has been stopped (2 weeks in the case of Sertraline, and at least 5 weeks in the case of Fluoxetine). Combinations of SSRIs and related antidepressants may cause serotonin syndrome, hypotension and drowsiness.

References:
British National Formulary Vol. 57 (Mar 2009) & Geriatric Dosage Handbook (11th Ed)
MICROMEDEX (DRUGDEX) Healthcare Series Vol. 140 (2009)
American Hospital Formulary System (2009 Edition)
Manufacturers' Product Information

Table 3.1: RECOMMENDED INVESTIGATIONS FOR PATIENTS RECEIVING SELECTED PHARMACOTHERAPY

S/N	Investigation	Indication
DEMENTIA		
1	Full Blood Count	Patients on mood stabilisers. Patients for consideration or on antiplatelet agent
2	Renal Panel (U/E/Cr)	Patients on antidepressants or mood stabilisers
3	Liver Function Test	Patients on antidepressants, atypical antipsychotics, mood stabilisers
4	Electrocardiogram	Patients for consideration or on cholinesterase inhibitors and antipsychotics (both typical and atypical) and in whom there is concern with regards to cardiac rhythm abnormalities

TABLE 3.2: LIST OF MEDISAVE CLAIMABLE DRUGS FOR TREATMENT OF PSYCHIATRIC CONDITIONS

S/N	Drug	S/N	Drug
1	Amisulpride	24	Lithium*
2	Amitriptyline	25	Maprotiline
3	Aripiprazole	26	Memantine#
4	Benzhexol	27	Mirtazepine
5	Benztropine	28	Moclobemide
6	Bupropion	29	Nortriptyline
7	Carbamazepine*	30	Olanzepine
8	Chlorpromazine	31	Paliperidone
9	Clomipramine	32	Paroxetine
10	Clozapine	33	Perphenazine
11	Donepezil	34	Quetiapine
12	Dothiepin	35	Risperidone
13	Doxepin	36	Rivastigmine #
14	Duloxetine	37	Sertraline
15	Escitalopram	38	Sodium Valproate*
16	Fluoxetine	39	Sulpiride
17	Flupenthixol	40	Tianeptine
18	Fluphenazine	41	Trazodone
19	Fluvoxamine	42	Trifluoperazine
20	Galantamine#	43	Trimipramine
21	Haloperidol	44	Venlafaxine
22	Imipramine	45	Ziprasidone
23	Lamotrigine	46	Zuclopenthixol

Notes
NB: The list will automatically include any other new psychiatric drugs (excluding benzodiazepams) that are approved by the Health Sciences Authority (HSA)
**Mood stabilizers*
 # Drugs which are specific for the treatment of dementia

TABLE 3.3: LIST OF ALLOWABLE THERAPIES FOR TREATMENT OF PSYCHIATRIC CONDITIONS

- Psychological therapy in specific cases
- Electro-convulsive therapy (ECT)
- Occupational Therapy
- Physiotherapy
- Speech therapy

CMMSE scoring sheet

Attention (forward digit span): 4719 582036 (1) Intact (2) Impaired [ ]

ITEMS	(61) CMMSE
What day of the week is it?	(1)
What is the date today?	(1)
What is the month?	(1)
What is the year?	(1)
Where are we now?	(1)
What floor are we now?	(1)
In which estate are we?	(1)
In which country are we?	(1)
* Repeat the following words: "Lemon, Key, Balloon"	(3)
Subtract $7 from $100 and make 5 subtractions	(5)
* Can you recall the three words	(3)
What is this? (show a pencil)	(1)
What is this? (show a watch)	(1)
Repeat the following: a) "No ifs, ands or buts" (English) b) "Forty-four stone lions" (Chinese)	(1)
Follow a 3-stage command: "Take this piece of paper, fold it in half, and put it on the floor."	(3)
Say a sentence of your choice	(1)
Read & obey what is written on this piece of paper. "Raise your hands"	(1)
Copy this drawing on a piece of paper	(1)
TOTAL SCORE	(28)

LEARNING POINTS

- **From 1 Nov 2011, Dementia will be included into the CDMP.**
- **This is expected to bring about better health outcomes for patients who will have better control of their conditions with close supervision from their doctors.**
- **For new diagnosis of dementia or suspected cognitive impairment, when in doubt, it is advisable to consult or refer to a geriatrician/ psychiatrist/ neurologist for confirmation as these diagnoses carry long term medical and legal implication.**
- **Existing patients with dementia in the RHs or IMH are recommended to be assessed by geriatricians/ psychiatrists/their primary care physician to be suitable for follow-up in the community by GP clinics or polyclinics, which are participating in Shared Care or GP Partnership Programmes.**
- **Clinics enrolled under the Medisave for CDMP are required to provide all the essential care components detailed in the DMP.**
- **The basis for diagnosis and management of dementia should conform to the prevailing MOH Clinical Practice Guidelines.**
- **There is a list investigations, drugs and therapies for the evaluation and management of dementia for which Medisave use can be allowed.**
- **As part of the national effort under this Programme, the Health Promotion Board has prepared Patient Education Booklets for dementia.**
- **Participating medical institutions must monitor the quality of care that patients receive.**

SLAS ☐ 1 ☐ 2 **Date:** _____

SUBJECT NO: ☐☐☐☐ **Name:** _____

MINI MENTAL STATE EXAM
迷你精神状况测试
PEPERIKSAAN KEADAAN ROHANI MINI

Instructions: Read the instructions for each item to the participant **word for word** as provided. Due to colloquial differences between the Chinese dialects, some minor deviations from verbatim instructions is acceptable only for Hokkien and Cantonese. However, examiners are recommended not to deviate overly from the provided instructions to avoid giving too much or too little information to the participants and potentially biasing their performance. For each of the 30 items, check the appropriate box (correct or incorrect) and record the subject's verbatim response in the spaces provided.

Orientation/Orientasi

Correct	Incorrect	
☐	☐	1. What is the year? 现在是哪一年? Sekarang tahun apa? _____
☐	☐	2. What is the month? (OK to accept Chinese calendar equivalents, but ask if subject knows Western calendar equivalent) 现在是几月? Sekarang bulan apa? _____
☐	☐	3. What is the date today? 今天几号? Apakah tarikh hari ini? _____
☐	☐	4. What day is today? 今天是星期几? Hari ini hari apa? _____
☐	☐	5. Without looking at your watch, what time is it? 不要看表，现在几点钟? Jangan melihat jam; sekarang pukul berapa? Subject's response _____ Current time _____
☐	☐	6. What area are we in? 我们在哪一个地区? Kita berada di kawasan mana? _____
☐	☐	7 What building are we in now? If necessary, ask for name or block number of building. 我们现在在哪一个建筑物? If necessary, 这个建筑物叫什么名/ 是什么号码? Sekarang kita berada di bangunan apa? If necessary, tanyakan nama bangunan atau nombor blok. _____
☐	☐	8. What floor are we on? 我们现在在几楼? Sekarang kita berada di tingkat berapa? _____
☐	☐	9. What country are we in? 我们现在在哪个国家? Kita berada di negara apa? _____
☐	☐	10. Which part of Singapore is this place (North, South, East, West or Central)? 这个地方在新加坡的那个方向，东，南，西，北或中? Di manakah kedudukan tempat ini di Singapura? (Utara, selatan, timur, barat atau pertengahan) _____

Immediate Recall / 即时回忆 / Pengingatan Kembali Segera

"I'm going to name three objects. When I am through, I want you to repeat them."
"我要说三样东西的名称。当我讲完后，我要你再重复一遍，
"Saya akan sebutkan tiga benda. Selepas ini, saya ingin anda ulanginya lagi."

The first repetition determines his/her score (0-3), but keep saying them until he/she can repeat all three, up to six trials.

Correct	Incorrect				
☐	☐	11.	Ball	Bola	柠檬
☐	☐	12.	Flag	Bendera	锁匙
☐	☐	13.	Tree	Pokok	气球
_____		13a.	Number of trials (Range = 1-6)		

"Please remember them as I will ask you to repeat them again later on."
"请把他们记住因为过后我会要你重复一次。"
"Cuba mengingatinya kerana saya akan menyuruh anda sebutkan benda-benda itu sebentar lagi."

Attention / 注意力/ Perhatian

"Subtract 7 from 100 and keep on subtracting 7 from each answer until I tell you to stop. Tell me your answer for each subtraction".
"请从一百减去七，然后从所得到的数目再减七，一直这样的计算下去。把每个答案都告诉我，直到我叫你停为止"。
"Sila tolak 7 dari 100 dan terus menolak 7 dari setiap jawapan yang didapati sampai saya berhenti. Berikan jawapan setelah setiap tolakan."

Each answer must be independently compared to the prior answer to ensure that a single mistake is not unduly penalised.

Correct	Incorrect			
☐	☐	14.	93	_____
☐	☐	15.	86	_____
☐	☐	16.	79	_____
☐	☐	17.	72	_____
☐	☐	18.	65	_____

Delayed Recall / 延缓回忆 / Peringatan Kembali Perlambatan

"Can you tell me the three objects that I asked you to remember earlier?"
"现在请告诉我， 刚才我叫你记住的三样东西是什么？ "
"Cuba namakan tiga benda yang saya suruh ingatkan tadi."

Correct	Incorrect				
☐	☐	19.	Ball	Bola	柠檬
☐	☐	20.	Flag	Bendera	锁匙
☐	☐	21.	Tree	Pokok	气球

Language / 语文 / Bahasa

Correct	Incorrect	
☐	☐	22. Show the subject a wrist watch and ask "**What is this**?" If subject gives a function say, "**Yes, but what is this called**?" or "**What is its name**?" **"这是什么？"，"是的，但是它叫什么？" 或 "它的名字是什么？"** **"Apakah ini?", "Ya, tetapi ia dipanggil apa?"** or **"Apakah nama nya?"**
☐	☐	23. Repeat for pencil / 铅笔 / pensil.
☐	☐	24. Say: "**I will say this once only, please listen carefully and repeat after me: An apple a day keeps the doctor away.**" **"现在我要说一句话，请听清楚后跟我重复一遍。 我只能说一遍，所以好好地听这句话是： 家家有本难念的经."** **"Saya akan menyatakan sekali sahaja, sila dengar baik-baik dan ikut apa yang saya cakap: marah,merah,murah."**

Hold a piece of paper in front of subject, do not allow him/her to take it until all three commands are given and say **"Listen carefully, take the paper in your right hand, fold it into half and put it on the floor."**

"请听清楚，用你的右手拿着张纸，把它折成一半后放在地板上。"

"Dengar baik-baik, ambil kertas dengan tangan kanan anda, lipatnya setengah dan letak di lantai."

Correct	Incorrect	
☐	☐	25. Takes paper in right hand.
☐	☐	26. Folds paper in half.
☐	☐	27. Puts paper on floor

Correct	Incorrect	
☐	☐	28. Present the piece of paper which reads 'Close your eyes' and say: **"Read this and do what it says"** "读这个，并按上面说的去做" **"Baca ini dan patuhi/lakukan apa yang tertulis"** *Score correct only if the subject actually closes his/her eyes.*
☐	☐	29. Say: **"Say a complete sentence"** The sentence must have a noun, a verb, and be meaningful.If needed, prompt the subject: **"For example, say something about the weather"** Write down the sentence provided. **"**请讲一个完整的句子。**" , "**比如，讲一个关于天气的句子。**"** **"Sebutkan sebuah ayat lengkap" , "Misalnya, bina sebuah ayat berkenaan cuaca."**

Note down the sentence _____

Construction / 图案构画 / Pembangunan

30. Present the subject with the Construction Stimulus page.
Say, **"Copy this design" / "照着纸上的图案来画" / "Cuba lukis gambar ini".**

Do not allow erasure. The subject may request a second attempt. (Clearly label the first and second attempts.)

☐ **Correct** ☐ **Incorrect**

Languages/Dialects used _____

Remarks: _____

The subject is having the following problem(s) at the time of interview:

- ☐ 0. Mute
- ☐ 1. Cannot see
- ☐ 2. Paralysed
- ☐ 3. Illiterate
- ☐ 4. Tired
- ☐ 5. Cannot hear

Total Score:

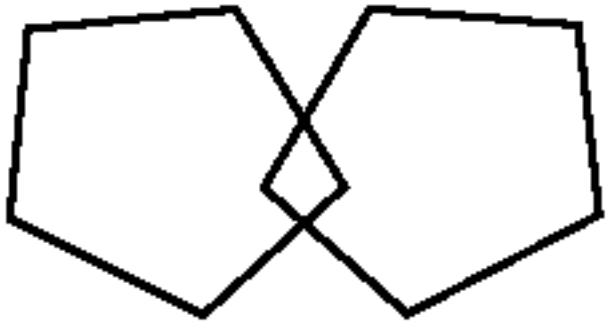
Assessor: _____

Close your eyes

关/闭上眼睛

關/閉上眼睛

Tutup Mata



UNIT NO. 6

USER INFORMATION FOR e-SERVICE CLINICAL DATA SUBMISSION

EXTRACTED FROM CDMP HANDBOOK FOR HEALTHCARE PROFESSIONALS 2011

ABSTRACT

The User Manual in the Chronic Disease Management Programme Handbook describes the steps in the clinical data submission. The e-Service Clinical Data submission requires an Internet-enabled computer. The user needs an user account to log in at the URL page. The Clinical Data submission e-Service allows submission of new reports. It also allows retrieval of submitted reports through the "search" function. The Frequently answered questions (FAQs) that accompanies this reading explains: clinical matters; registration matters; Medisave claims, reimbursement, billing; and data submission, clinical improvement and audit matters.

SFP2011; 37(3) (Supp 1) : 42-58

1. SYSTEM REQUIREMENTS

In order to use the e-Service Clinical Data Submission, an Internet-enabled computer with the followings is required:

- The minimum recommended hardware configuration is:
  - Pentium III MHz Processor with 256MB RAM.
  - At least 200 MB free hard disk space.
- System Software Requirements
  - Windows XP.
  - Internet Explorer 6.0 and above.
  - Broadband Internet Connection.
- Other Requirements
  - RSA token card.
  - MediClaim user account.

2. GETTING STARTED

User Account

- You will be using your MediClaim system user account to access the e-Service. The MediClaim account is the same one used for the submission of claims.
- If you do not have an account for the claims submission, you will need to approach MOH for the creation of a new account.

Accessing the e-Service

- The web URL to access the MediClaim system is: https://access.medinet.gov.sg. Screen 1 shows the login screen. Fill and press login.
- Upon successful login to the MediClaim system, you will be able to see the Clinical Indicators data collection e-Service in the left hand menu as shown on Screen 2. All users with access to the Chronic Disease Claim Form e-Service will have access to the Clinical Indicators Data Collection e-Service.
- Click on the functions available:
- Submission is used to submit a new report.
- Search is used to retrieve submitted reports.

3. CLINICAL INDICATORS REPORT SUBMISSION

This function is used to submit clinical data on patients who have used their Medisave under the CDMP. A new submission can be made each time there is additional indicator information for the patient either on a per visit basis or consolidated over a few visits. All submissions are distinct and will be used for analysis by MOH on a cumulative basis.

To submit a new set of clinical data for a patient to MOH, click on the "Submission" sub-menu. Screen 3 will appear.

Actions

- Select the Identification Type and enter the Patient NRIC/FIN.
- Select the chronic disease applicable to this patient. You can select one or more diseases, as applicable.

Click on [Next] to proceed to the Clinical Indicator Form.

The Clinical Indicator Form consists of 4 sections:

- Patient Details.
- Known Medical History.
- Clinical and Assessment Indicators.
- Attending Physician Information.

3A. Patient details

This section details the patient's basic bio-data. If it is your first submission for the patient, only Patient NRIC, Name, Date of Birth, Sex, Race, and Current Smoker is required. See Table 1.

For subsequent submissions, only the Patient NRIC and Name are mandatory.

In the event of differences between two submissions, the data from the latest submission will be considered as the up-to-date information.

3B. Known medical history

- This section details the patient's medical history.
- If it is your first submission for the patient, please enter all the details.
- For subsequent submissions, you can omit the details if there are no changes.

Table 1. Patient Details to fill in

	Data Item	Remarks
1	Patient Name	Patient's name as in NRIC.
1.	Patient NRIC/FIN	Will be copied from previous screen.
2.	Date of Birth	Patient's date of birth (enter in DDMMYYYY format).
3.	Sex	Gender of patient.
4.	Race	Ethnic group of patient.
5.	Height (m)	Patient's height in metres (e.g. 1.75) and must be between 0.10 and 2.50 (inclusive) or 9.99 if not measurable.
6.	Current Smoker	Whether patient is a current smoker.
8.	Year Started Smoking	Year that patient started smoking (enter in YYYY format).

- If you are unsure whether you have submitted the information, it is recommended you fill in the details.

Enter the relevant medical conditions for the patient. If a particular condition is selected, then the year of diagnosis is mandatory. You only need to fill in medical conditions that apply to the patient.

Depending on the medical condition indicated, different treatment sections will be available for input (see Table 2).

3C. Clinical indicators and assessment

- This section enables you to enter the indicator measurement and assessment done on the patient over any period.
- Only measurements and assessments not reported previously need to be entered in this section.
- Initially there will be no clinical indicators added to the report.
- Fill in all the clinical indicators and use the [Add Indicators] button to save them. See Table 3 for the range of values to fill in.
- There must not be any unsaved data left in the Clinical Indicators Section before submitting the form.

After saving the data, you can use the delete button to remove any mistakes. By default, the data displayed is sorted by date of visit and indicators. You can also click on the "Indicators" and "Date" headers to sort the data according to your preference.

3D. Attending physician information

- This section details the physician attending to the patient. It is required for each submission.
- If there is more than one physician attending to the patient, the main physician information should be entered here. Table 4 shows the details to be filled in.

3E. Report submission

- Once you have completed the data entry, you can submit the report to MOH by clicking on the [Submit] button.
- If you are not yet ready to submit, you can click on the [Save Draft] button and retrieve the report later from the search function for submission. See Table 5.

4. SEARCHING CLINICAL INDICATOR REPORTS

After you have submitted a report or created a draft, you can retrieve the reports at a later stage using the search function. This function allows you to specify search criteria and retrieve all reports matching the criteria. To access this function, click on the "Search" sub-menu under the "Clinical Indicators" main menu as shown on Screen 2.

At least one of the search criteria must be entered before you can proceed with the search. The search is case insensitive. Table 7 show the criteria for searching and the reports that will be retrieved.

All submissions made by your clinic which matches the criteria will be displayed as shown on Screen 9.

If the number of search results is too large, you can either specify more restrictive search criteria or use the page number to navigate through the results.

Table 2. Fill in treatment details

Medical Condition	Diabetes Treatment	Hypertension Treatment	Lipid Disorder Treatment	Asthma Treatment	COPD Treatment	Depression Treatment	Schizophrenia Treatment	Bipolar Disorder Treatment	Dementia Treatment
Diabetes	Available	Available	Available	X	X	X	X	X	X
Hypertension	X	Available	Available	X	X	X	X	X	X
Lipid Disorders	X	X	Available	X	X	X	X	X	X
Asthma	X	X	X	Available	X	X	X	X	X
COPD	X	X	X	X	Available	X	X	X	X
Major Depression	X	X	X	X	X	Available	X	X	X
Schizophrenia	X	X	X	X	X	X	Available	X	X
Bipolar Disorder	X	X	X	X	X	X	X	Available	X
Dementia	X	X	X	X	X	X	X	X	Available
None of the above	X	X	X	X	X	X	X	X	X

Table 3. The list of Clinical Indicators and Assessments to fill in

Clinical Indicators	Remarks
Glucose - HbA1c (%)	Value must be between 0.1 and 40.0 (inclusive).
Blood Pressure - Diastolic BP	Value (in mmHg) must be between 20 and 200 (inclusive) and must be smaller than Systolic BP reading.
Blood Pressure - Systolic BP	Value (in mmHg) must be between 30 to 300 (inclusive).
Lipids – LDL-C	Value (in mg/dL) must be between 1 and 999 (inclusive). Value (in mmol/L) must be between 0.1 and 30.0 (inclusive). If measurement is attempted but not measurable due to high Triglyceride (TG) value, a reading of 999 (mg/dL) should be entered.
Lifestyle - Weight (kg)	Value (in kg) must be between 1.0 and 300.0 (inclusive) or 999 if not measurable.
Smoking - Cigarettes smoked per day (average)	Value must be between 0 to 1000.
Asthma - ACT Score	Value must be between 5 and 25 (inclusive) for patients who are aged 12 years and above. Value must be between 0 and 27 (inclusive) for patients who are aged between 4 to below 12 years old. Value must not be entered for patients who are aged below 4 years old.
CGI – Severity of Illness	Only for CDMP Mental Health Programme patients. Value must be between 1 and 7 (inclusive).
CGI – Global Improvement	Only for CDMP Mental Health Programme patients. Value must be between 0 and 7 (inclusive).
Assessments/Screening	
DM - Eye Screening	Select and enter date of assessment if done. If assessment is not done during the reporting period, you need not enter anything. If the exact date of assessment is not known, please key in the date as 0101(for DDMM). e.g. for an assessment done in 2006 you can key in 01012006. If the known date is March 2006, you can enter as 01032006
DM - Foot Screening	
DM - Nephropathy Screening	
Stroke - Thromboembolism Risk Assessment	
Inhaler Technique Assessment (Asthma & COPD only)	
Influenza Vaccination Assessment (COPD only)	
Fasting Lipids Blood Test Fasting Glucose Blood Test (Only for CDMP Mental Illness Programme – Schizophrenia Patients on Atypical Antipsychotics)	
Consultation for CDMP Mental Health (Only for CDMP Mental Illness Programme Patients)	
Assessment of memory Assessment of mood and behaviour Assessment of functional and social difficulties Assessment of rehabilitation needs For patients on cognitive enhancers, documentation of objective assessment of memory (MMSE or CMMSE testing or other validated instruments)	

Table 4. Physician Information to be filled in

	Data Item	Remarks
1.	Doctor Name	Full Name of Doctor.
2.	Registration Number	The Doctor's MCR Number.
3.	Speciality/Training	Select the appropriate value from the drop down list if applicable.
4.	Healthcare Establishment	The Healthcare Establishment which is making the submission. It is tied to the user ID of the person making the submission and is defaulted based on the user's ID establishment.
5.	Role	Indicate the role applicable.
6.	Name of Primary Physician	Only applicable when "None of the Above" is selected.

Table 5. The buttons at the bottom of screen

Button	Function Description
Submit	Submits the form after completion. Deletes any existing drafts saved previously.
Save Draft	Saves the unfinished form inputs as a draft for completion in the future.
Close	Closes the current form and returns to the main menu.

6. SUMMARY OF MEDISAVE FOR CDMP

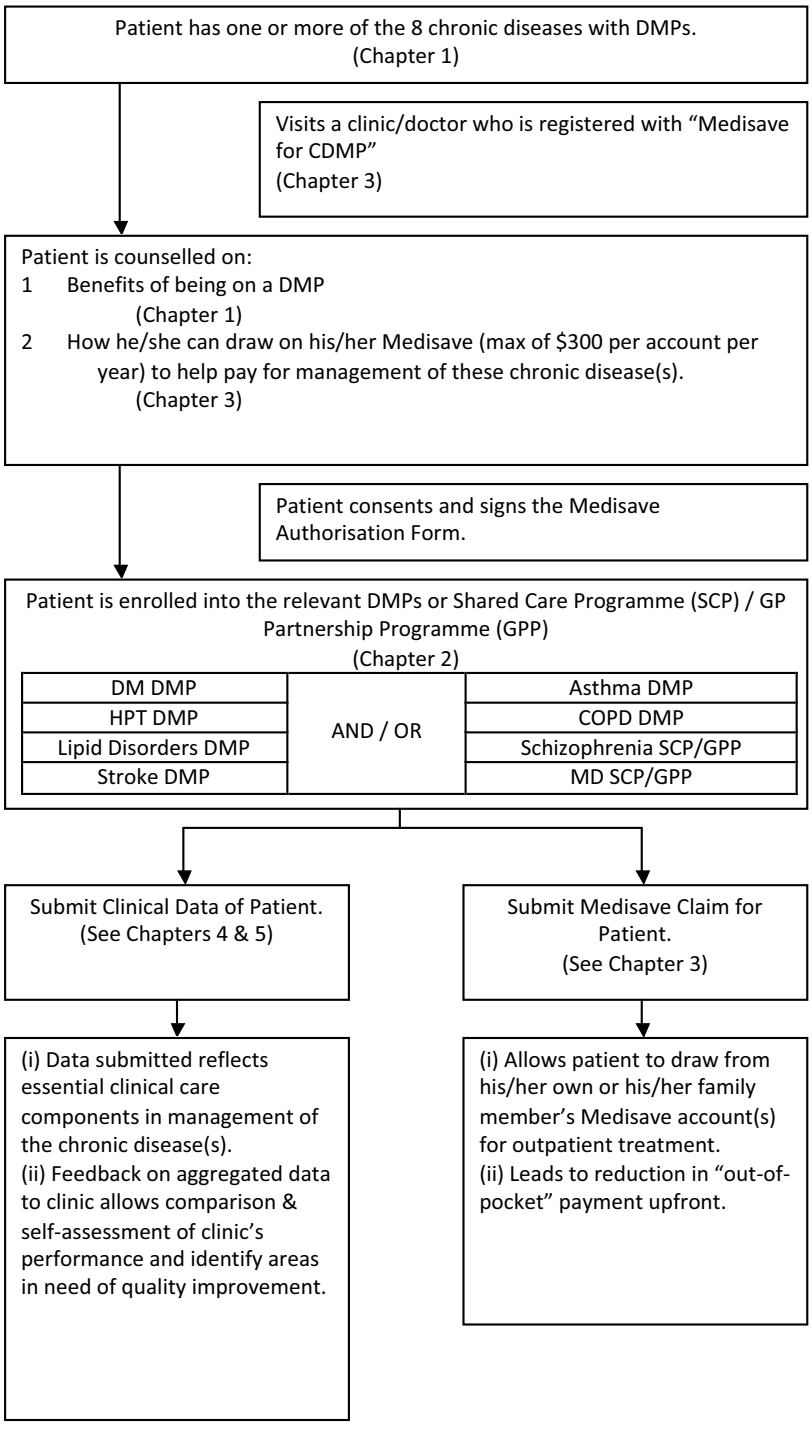


Table 7. Criteria for searching and the reports that will be retrieved

	Criteria	Remarks
1.	Patient Name	All reports where the patient name matches are retrieved A partial name is allowed, e.g. if Mark is entered, reports for all patients with Mark in their names are retrieved.
2.	Patient NRIC/FIN	All reports where the patient NRIC matches are retrieved.
3.	From Date	All reports submitted from this date (inclusive) are retrieved.
4.	To Date	All reports submitted up to this date (inclusive) are retrieved.
5.	Sort By	Specifies the sorting sequence for the results.

Click on the Patient Name hyperlink to view the report submitted.

After retrieving the report, you can also proceed to "Amend" it if there was any mistake in the previous submission, or delete it altogether.

When the [Amend] button is clicked, the selected record will be displayed in editable mode as shown on Screen 10.

Upon entering a valid year, a list of patient NRIC numbers will be generated. The report generated below shows the record of a patient who had a claim submitted but with no submission of any clinical indicator.

Button	Action
Amend	Re-submits all the data in the report
Close	Closes the form

CIDC Clinic Reports

- This function provides standard report(s) for use by clinics. One report is currently available and additional reports may be added in future releases.
- To access this function, click on the CIDC Clinics Reports under the Reports menu button. A page displaying all the available reports and their description will be loaded.

List of NRICs for patients for whom Clinical Indicators have not been submitted. This report enables the clinics to have a listing of all the patients' NRICs for whom the clinics had made claims in the specified year but no clinical indicator reports were submitted within a fixed period of 12 months from the claim submission date of each patient. This report is built in to assist doctors and clinics to keep track of the outstanding clinical indicator reports they would require to submit with each claim.

Click on the report title from the list of available reports as shown on Screen 12. A report page with a textbox would appear for the user to key in the year of the requested report, as shown below.

Troubleshooting

Enabling of Pop Ups

Certain screens within the application will be displayed as pop up windows. In order to access the full system functionality, you need to enable pop up windows for the MediClaim website. To enable this feature, follow the steps below:

- Select Tools>Pop-up Blocker> Pop-up Blocker Settings…
- Enter "*.medinet.gov.sg" and "*.moh.gov.sg", then click [Add] button.

Fallback Procedures

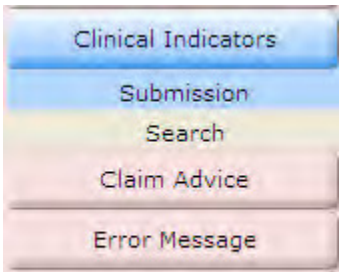
- In the event that the submission cannot be done online immediately, you can keep a record of the information and submit it at a later date.

Contact Information for Queries Related to Clinical Data Collection and Submission

- For online e-service related technical queries, please e-mail to mediclaim@ncs.com.sg, or contact NCS at: 6776 9330 (Mon - Fri, excluding public holidays, 8:30am to 6:00pm).
- For clinical data collection and submission issues related feedback, please email to moh_cds@moh.gov.sg (preferred method), or contact at: 6325 1757 (Mon - Fri, excluding public holidays, 8:30 am to 6:00 pm).

Screen 1 – MediClaim login screen

Screen 2 – Menu



Screen 3 –Submission screen – for new report

Clinical Indicators > **Submission**

New Submission:

Patient ID Type * SINGAPORE PINK / BLUE NRIC

Patient NRIC/FIN *

(as entered in Medisave claim form)

Diseases *

☐ Diabetes ☐ Hypertension ☐ Lipid Disorder
☐ Stroke ☐ Asthma ☐ COPD
☐ Major Depression ☐ Schizophrenia ☐ Bipolar Disorder
☐ Dementia

Next

Condition	Care Components Per Year
Diabetes mellitus	• Two blood pressure measurements • Two bodyweight measurements • Two haemoglobin A1c (HbA1c) tests • One serum cholesterol level (LDL-C) test • One smoking assessment • One eye assessment • One foot assessment • One nephropathy screening test
Hypertension	• Two blood pressure measurements • One bodyweight measurement • One smoking assessment
Lipid Disorder	• One serum cholesterol level (LDL-C) test • One smoking assessment
Stroke	• Two blood pressure measurements • One serum cholesterol level (LDL-C) test • One smoking assessment • One clinical thromboembolism risk assessment
Asthma	• One inhaler technique assessment • One smoking assessment • Two Asthma Control Test (ACT) scores
COPD	• One inhaler technique assessment • One smoking assessment • One bodyweight measurement • One influenza vaccination
The following care components are only for CDMP Mental Health Programme Patients:	
Major Depression	• One Clinical Global Impression (CGI) Scale for each item (severity, improvement) • Two consultations for CDMP Mental Health
Schizophrenia	• One Clinical Global Impression (CGI) Scale for each item (severity, improvement) • Two consultations for CDMP Mental Health • One blood test for fasting lipids • One blood test for fasting glucose
Bipolar Disorder	• One Clinical Global Impression (CGI) Scale for each item (severity, improvement) • Two consultations for CDMP Mental Health • One blood test for fasting lipids • One blood test for fasting glucose
Dementia	• One assessment of memory • One assessment of mood and behaviour • One assessment of functional and social difficulties (if any) • One assessment of rehabilitation Needs • Two consultations for CDMP Mental Health • For patients on cognitive enhancers, documentation of objective assessment of memory (MMSE or CMMSE testing or other validated instruments)

Screen 4 – Clinical Indicator Form

Patient Details:

***Patient Name:** * Tan Ah Kun

Patient NRIC/FIN: S1234567D

Date of Birth (DDMMYYYY): 14041971

Sex: (•) Male ( ) Female

Race: Chinese

Height (Metres): 1.62 *(use 9.99 if not measurable)*

Current Smoker ( ) Yes (•) No

Year Started Smoking(YYYY)

** denotes a mandatory field*

Known Medical History:

Medical Condition	Diagnosis Year	Medical Condition	Diagnosis Year
[x] Diabetes	2007 (YYYY)	[ ] Hypertension	(YYYY)
[ ] DM Retinopathy	(YYYY)	[ ] Lipid Disorder	(YYYY)
[ ] DM Nephropathy	(YYYY)	[ ] Cerebrovascular Accident (CVA)	(YYYY)
[ ] DM Foot Complications	(YYYY)	[ ] Coronary Heart Disease (CHD)	(YYYY)
[x] Asthma	(YYYY)	[x] COPD	(YYYY)
[x] Major Depression	2007 (YYYY)	[x] Schizophrenia	2007 (YYYY)
[ ] Bipolar Disorder	(YYYY)	[ ] Dementia	(YYYY)

Clinical Indicators:

Date of Visit (DDMMYYYY):*

Blood Pressure (Systolic/Diastolic): /

LDL-C mg/dL

HbA1c (%):

Weight (kg) *(use 999 if not measurable)*

Smoking Assessment #

Cigarettes smoked per day (average) ##:

ACT Score (Asthma only):

DM - Eye Assessment:

DM - Nephropathy Assessment:

DM - Foot Assessment:

Stroke - Thromboembolism Risk Assessment:

Inhaler Technique Assessment (Asthma & COPD only):

Influenza Vaccination Assessment (COPD only):

The following care components are only for CDMP Mental Health Programme Patients:

CGI - Severity of Illness:

CGI - Global Improvement:

Consultation for CDMP Mental Health (Indicate the patient attendance):

For patients on cognitive enhancers, documentation of objective assessment of memory (MMSE or CMMSE testing or other validated instruments):

Assessment of Functional and Social Difficulties (if any):

Fasting Lipids Blood Test ###:

Fasting Glucose Blood Test ###:

Assessment of Memory:

Assessment of Mood and Behaviour:

Assessment of Rehabilitation Needs:

** denotes a mandatory field*

 # For current smokers, smoking cessation advice should be given;
For non- or ex-smoker, please reinforce the benefits of not smoking cigarettes

Applicable to current smokers only

Only for patients on Schizophrenia and Bipolar Disorder - Atypical Antipsychotics Medication. To check the box if test is done

Add Indicators *Click to add clinical indicators (only those performed)*

Attending Physician Information:

Doctor Name:*

Registration Number:*

Specialty/Training: Please select if applicable

Healthcare Establishment:

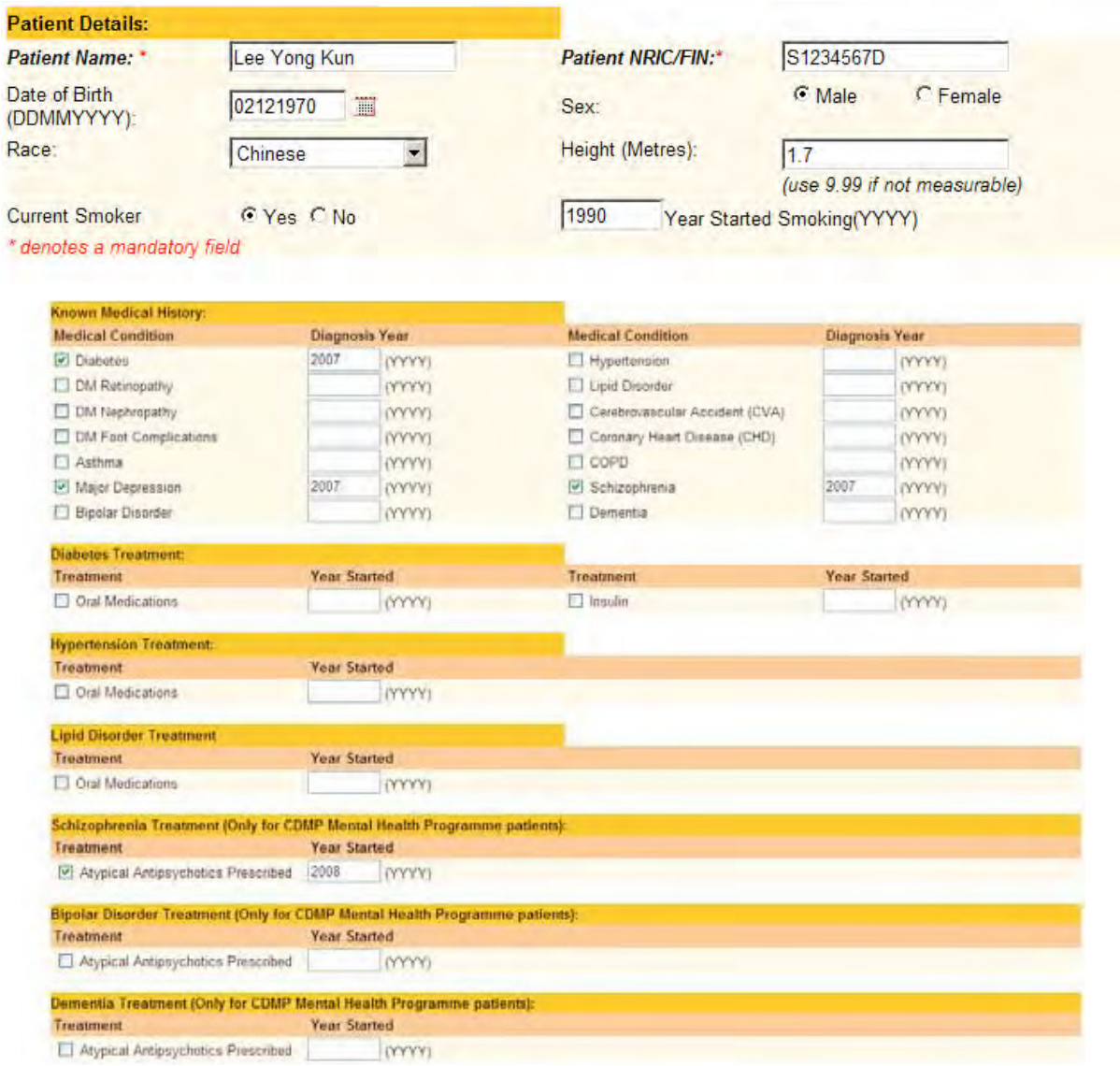
Role:* (•) Attending Doctor is the patient's regular primary physician
( ) The Clinic is the patient's regular primary provider
( ) None of the Above

Date of Submission: 06-Jan-2008

** denotes a mandatory field*

Submit Save Draft Close

Screen 5– Known Medical History and Treatment Sections



Patient Details:

Patient Name: *	Lee Yong Kun	***Patient NRIC/FIN:****	S1234567D
Date of Birth (DDMMYYYY):	02121970	Sex:	(•) Male ( ) Female
Race:	Chinese	Height (Metres):	1.7 (use 9.99 if not measurable)
Current Smoker	(•) Yes ( ) No	1990	Year Started Smoking(YYYY)

** denotes a mandatory field*

Known Medical History:

Medical Condition	Diagnosis Year	Medical Condition	Diagnosis Year
☑ Diabetes	2007 (YYYY)	☐ Hypertension	(YYYY)
☐ DM Retinopathy	(YYYY)	☐ Lipid Disorder	(YYYY)
☐ DM Nephropathy	(YYYY)	☐ Cerebrovascular Accident (CVA)	(YYYY)
☐ DM Foot Complications	(YYYY)	☐ Coronary Heart Disease (CHD)	(YYYY)
☐ Asthma	(YYYY)	☐ COPD	(YYYY)
☑ Major Depression	2007 (YYYY)	☑ Schizophrenia	2007 (YYYY)
☐ Bipolar Disorder	(YYYY)	☐ Dementia	(YYYY)

Diabetes Treatment:

Treatment	Year Started	Treatment	Year Started
☐ Oral Medications	(YYYY)	☐ Insulin	(YYYY)

Hypertension Treatment:

Treatment	Year Started
☐ Oral Medications	(YYYY)

Lipid Disorder Treatment

Treatment	Year Started
☐ Oral Medications	(YYYY)

Schizophrenia Treatment (Only for CDMP Mental Health Programme patients):

Treatment	Year Started
☑ Atypical Antipsychotics Prescribed	2008 (YYYY)

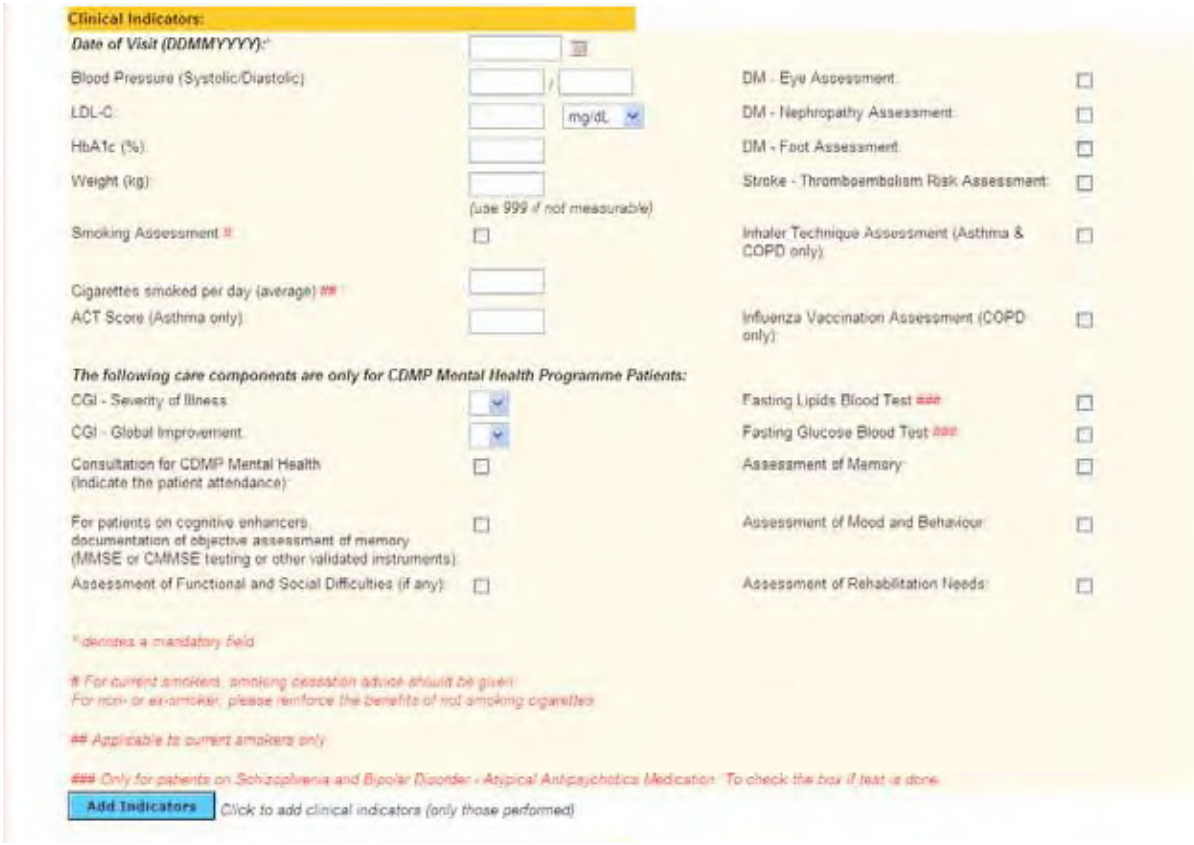
Bipolar Disorder Treatment (Only for CDMP Mental Health Programme patients):

Treatment	Year Started
☐ Atypical Antipsychotics Prescribed	(YYYY)

Dementia Treatment (Only for CDMP Mental Health Programme patients):

Treatment	Year Started
☐ Atypical Antipsychotics Prescribed	(YYYY)

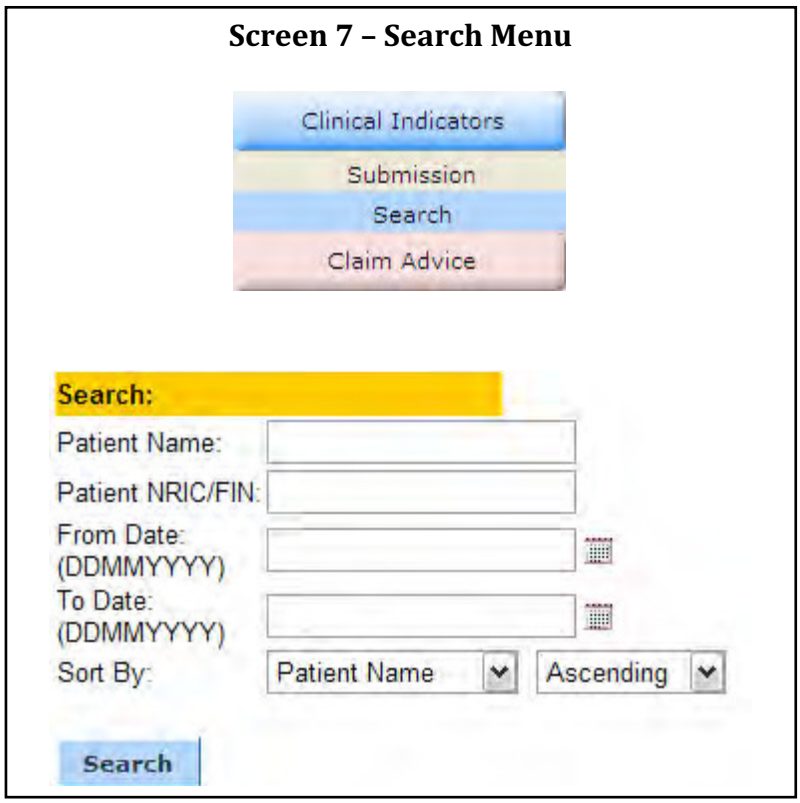
Screen 6 – Filling in the Clinical Indicators



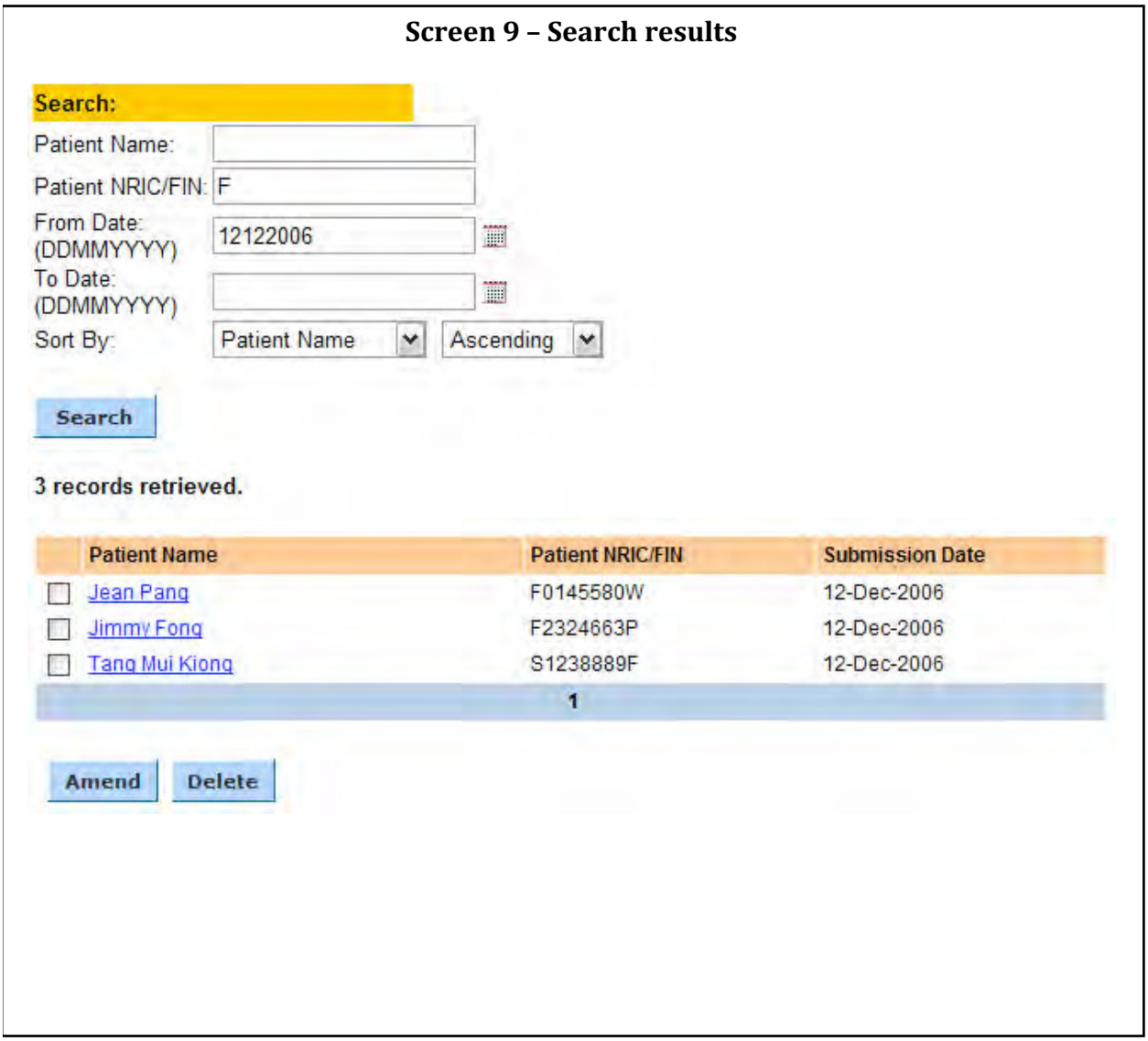
	Date	Indicators	Value
☐	11-May-2007	Systolic BP(mmHg)	150
☐	11-May-2007	Diastolic BP(mmHg)	100
☐	11-May-2007	LDL(mg/dL)	40
☐	11-May-2007	HbA1c(%)	30
☐	11-May-2007	Weight(kg)	90
☐	11-May-2007	Cigarettes smoked per day(Avg)	10
☐	11-May-2007	DM-Eye Assessment	Y
☐	11-May-2007	DM-Nephropathy Assessment	Y
☐	11-May-2007	DM-Foot Assessment	Y
☐	11-May-2007	Stroke-Thromboembolism Risk Assessment	Y

Delete Indicators *Click to delete selected clinical indicators*

Screen 7 – Search Menu



Screen 9 – Search results



Screen 10 – Editable page of patient record

Patient Details:

Patient Name: * Tan Ah Kun

Patient NRIC/FIN: * S1234567D

Date of Birth (DDMMYYYY): 14041971

Sex: (•) Male ( ) Female

Race: Chinese

Height (Metres): 1.62 *(use 9.99 if not measurable)*

Current Smoker ( ) Yes (•) No

Year Started Smoking(YYYY)

** denotes a mandatory field*

Known Medical History:

Medical Condition	Diagnosis Year	Medical Condition	Diagnosis Year
☑ Diabetes	2007 (YYYY)	☐ Hypertension	(YYYY)
☐ DM Retinopathy	(YYYY)	☐ Lipid Disorder	(YYYY)
☐ DM Nephropathy	(YYYY)	☐ Cerebrovascular Accident (CVA)	(YYYY)
☐ DM Foot Complications	(YYYY)	☐ Coronary Heart Disease (CHD)	(YYYY)
☑ Asthma	(YYYY)	☑ COPD	(YYYY)
☑ Major Depression	2007 (YYYY)	☑ Schizophrenia	2007 (YYYY)
☐ Bipolar Disorder	(YYYY)	☐ Dementia	(YYYY)

Diabetes Treatment:

Treatment	Year Started	Treatment	Year Started
☐ Oral Medications	(YYYY)	☐ Insulin	(YYYY)

Hypertension Treatment:

Treatment	Year Started
☐ Oral Medications	(YYYY)

Lipid Disorder Treatment

Treatment	Year Started
☐ Oral Medications	(YYYY)

Asthma Treatment:

Treatment	Year Started
☐ Preventer	(YYYY)

Schizophrenia Treatment (Only for CDMP Mental Health Programme patients):

Treatment	Year Started
☑ Atypical Antipsychotics Prescribed	2008 (YYYY)

Bipolar Disorder Treatment (Only for CDMP Mental Health Programme patients):

Treatment	Year Started
☐ Atypical Antipsychotics Prescribed	(YYYY)

Dementia Treatment (Only for CDMP Mental Health Programme patients):

Treatment	Year Started
☐ Atypical Antipsychotics Prescribed	(YYYY)

Clinical Indicators:

Date of Visit (DDMMYYYY): *

Blood Pressure (Systolic/Diastolic): /

LDL-C: mg/dL

HbA1c (%):

Weight (kg): *(use 999 if not measurable)*

Smoking Assessment # :

Cigarettes smoked per day (average) ## :

ACT Score (Asthma only):

DM - Eye Assessment: ☐

DM - Nephropathy Assessment: ☐

DM - Foot Assessment: ☐

Stroke - Thromboembolism Risk Assessment: ☐

Inhaler Technique Assessment (Asthma & COPD only): ☐

Influenza Vaccination Assessment (COPD only): ☐

The following care components are only for CDMP Mental Health Programme Patients:

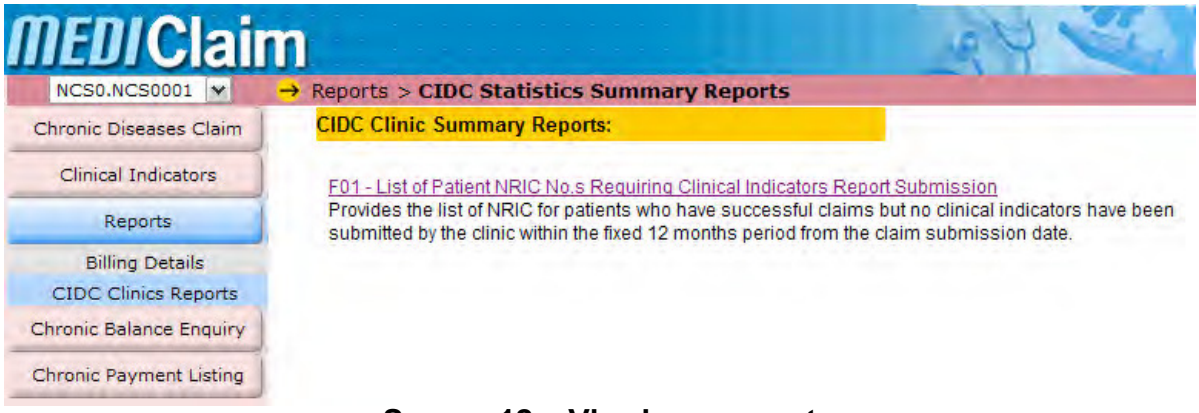
CGI - Severity of Illness:

CGI - Global Improvement:

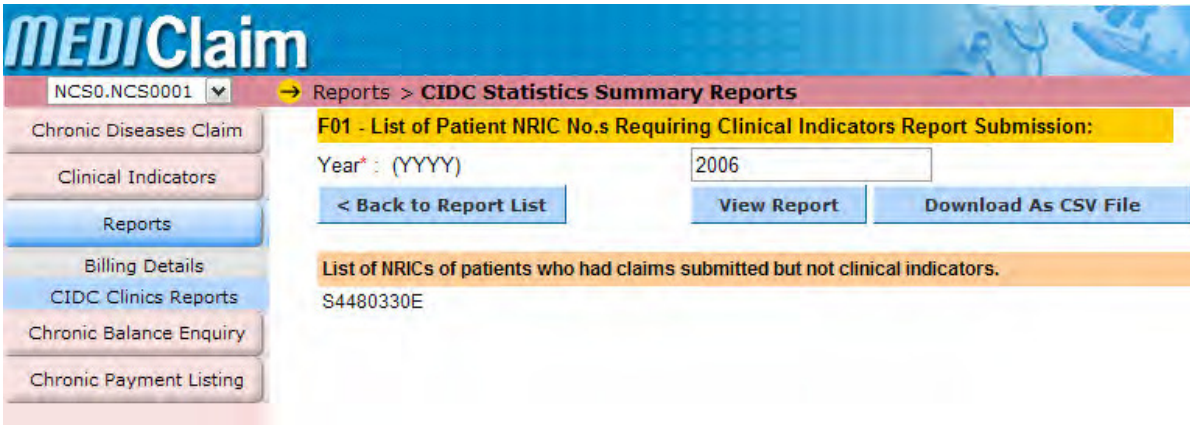
Fasting Lipids Blood Test ###: ☐

Fasting Glucose Blood Test ###: ☐

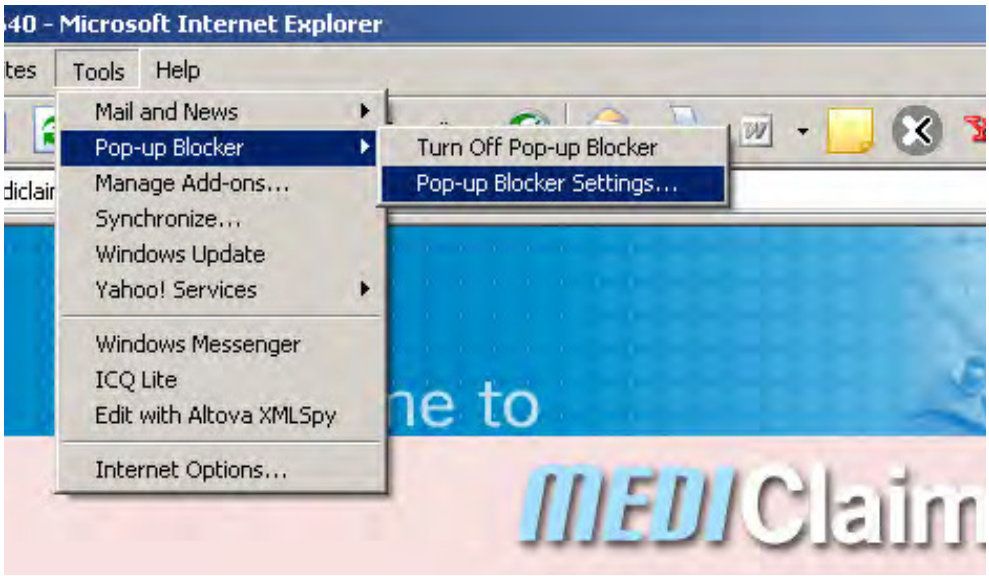
Screen 11 – CDC Reports



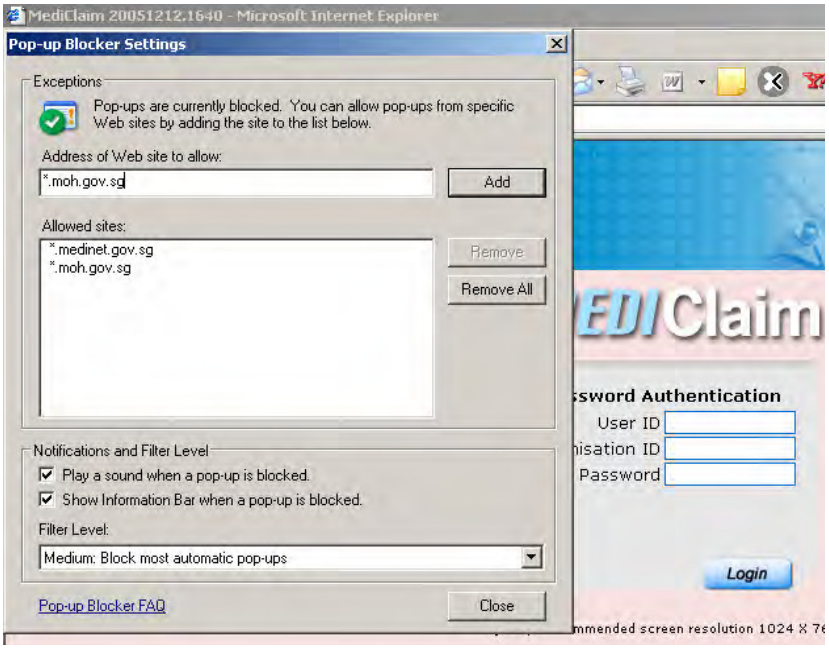
Screen 12 – Viewing a report



Screen 13 – Internet Explorer Menus



Screen 15 – Configuring Pop Up Blocker



5. FREQUENTLY ASKED QUESTIONS

A. CLINICAL MATTERS:

For Doctors who have already registered into the Programme

Q1. Which chronic diseases are currently included under this Programme?

Diabetes Mellitus, Hypertension, Lipid disorders, Stroke, Asthma, COPD, Schizophrenia, Major Depression, dementia and bipolar disorder are currently included under this Programme.

Q2. I have a patient with Diabetes, Hyperlipidaemia and Asthma, which DMPs should I enrol him into?

Enrol him into both Diabetes AND Asthma DMPs. He will then be able to use Medisave to co-pay for the total bill for the treatment prescribed for all 3 conditions. However, the doctor will also need to submit outcome data based on the essential care components of diabetes and asthma. (Please refer to Chapter 3 for details.)

Q3. My patient has DM, however, he also has symptoms and signs of Hypothyroidism. Can I use his Medisave to co-pay the thyroid function test?

No. In this instance, thyroid function test was done to screen for an associated disease and not for monitoring of the primary condition or its complication. Hence, it is suggested that his bill be itemised so that the patient can use cash to pay for the thyroid function test and Medisave to co-pay the rest of the bill which is related to DM care components. (Please refer to Chapter 3)

Q4. Who decides on the stipulated clinical care component?

The clinical care components were drawn from the Clinical Practice Guidelines, with inputs from professional bodies, which include leading specialists in the respective fields and respected primary care physicians. They were also endorsed by the Clinical Advisory Committee.

Q5. What if the patient has symptoms suggestive of both COPD and Asthma? Which DMP should I enrol him into?

For patients whose signs and symptoms are not so distinct between the two conditions, spirometry or/and bronchodilator reversibility testing may be performed to help classify the patient into one of the two diagnoses or to differentiate these conditions from other diseases that may mimic its presentation.

It is important to try to classify the patient into the correct DMP as this will help to determine the management of the patient and also prevent any issues with respect to the Medisave claims.

(Please refer to the Clinical Practice Guidelines for more information on diagnosis and management of Asthma and COPD).

Q6. Can the patient use Medisave to pay for pulmonary rehabilitation?

Yes, if and only if

a) the patient has been diagnosed to have COPD, AND
b) It is clinically deemed to be beneficial for the patient.

B. REGISTRATION MATTERS

For Doctors & Clinics which wish to be registered into the Programme:

Q1. What are the requirements to be on the Programme?

Clinics that wish to participate in the Programme must agree to:

a) Participate in a shared care or GP partnership programme with a Restructured Hospital.
b) Provide treatment to chronic disease patients through evidence-based DMPs. These DMPs will include MOH-recommended key treatment components.
c) Treat patient medical information with confidentiality.
d) Submit to MOH, with the informed consent of patient, data on patient care delivery on an annual basis or as specified by MOH, for the purpose of medical audits. Relevant aggregated performance data will be published to assist patients in making informed choices.
e) Be accredited under the Medisave for CDMP.
f) Be periodically reviewed and audited, both clinically and administratively. Any clinic/hospital that fails to satisfy the minimum standards of clinical performance set by MOH, will be asked to withdraw from the Programme. (see Chapter Two: Clinical Programme).

Q2. How do I register for the CDMP Programme?

Clinics who are already in the CDMP Programme need not re-register for the Programme.

For clinics who are not in the Programme, they must submit the following forms for registration:

a) E-Application for Clinics to Participate in the Medisave for Chronic Disease Management Programme (by MOH).
b) Direct Authorisation Credit Form (by CPF Board).
c) GIRO Form (MediClaim charges by NCS).
d) GIRO Form (Medisave charges by CPF Board).

The E-Application website can be accessed via http://www.moh.gov.sg/mmae/overview.aspx

Clinics participating in the Programme will also have to sign a Deed of Indemnity with the CPF Board.

Doctors need to be individually registered under the Programme in order to process Medisave claims for their patients. Doctors can do so by submitting the Application Form for Medical Professionals.

Q3. My clinic is already participating in CDMP. Can I make Medisave claims for my patient who is suffering from schizophrenia, major depression, dementia or bipolar disorder?

In addition to participating in CDMP, your clinic will also need to be participating in a shared care or GP partnership programme with a restructured hospital before Medisave claims for patients with psychiatric illnesses can be made. This is part of an additional quality assurance framework in place to ensure quality of care for patients.

Q4. How do I register for a shared care or partnership programme with a restructured hospital?

You may register via MOH's MMAE website (http://www.moh.gov.sg/mmae/overview.aspx) by selecting the "Chronic Disease Management Programme (CDMP) – Shared Care Programmes".

Q5. What will be the cost of registration and start-up?

Apart from computer hardware and Internet access subscription (which may already be in place), there is a one-time non-refundable cost of $171.20 for the security token to access the Medisave claims system. This security token is required only when using the MediClaim e-service.

You or your staff will need to attend a half-day training session on Medisave claims process, guidelines on Medisave use and the use of the MediClaim system. This training session is free-of-charge.

Q6. How do patients sign up for the Programme?

To qualify, patients need to be certified by a doctor to suffer from at least one of the approved chronic diseases. The certification is made by the doctor when the patient fills out the Medisave Authorisation Form that allows the doctor to make Medisave claims on the patient's behalf.

C. MEDISAVE CLAIMS, REIMBURSEMENT, BILLING

For Doctors & Clinics that wish to be registered into the Programme:

Q1. In total, how much can patients claim from Medisave for chronic disease treatments?

Patients can claim up to $300 per Medisave account per year for outpatient treatment of the approved chronic diseases, regardless of the number of diseases they might have.

Q2. Whose Medisave account(s) can a patient make use of, other than his own?

Patients can use their own Medisave account(s) and the account(s) of their immediate family members (i.e. parents, children, spouse). In addition, patients who are Singapore citizens or PRs can also use the Medisave accounts of their grandchildren. Claims can be made once the family member has signed the relevant Medisave Authorisation Form.

Q3. What will be the exact level of deductible and co-payment? Are the levels different for packages and individual visits?

There is a $30 deductible and 15% co-payment of the bill balance for each claim that the patient has to pay in cash, regardless if the claim is for an individual visit or packaged treatment.

Q4. Who should submit Medisave claims?

Any of the permanent staff of a Medisave-accredited clinic who has attended the training sessions, i.e. doctors, nurses, counter staff, clinic managers etc, can submit the Medisave claims.

Q5. If the patient sees me for both a chronic disease and an acute illness at the same time, can the entire bill be claimed?

Medisave can only be used for treatment related to the 10 chronic diseases listed, subject to a cap of $300. If patient attendance is purely for an acute or unrelated condition, Medisave deduction is not allowed even though the patient may have a chronic condition. Checks will be made during audits to ensure that claims are related to approved chronic conditions.

Q6. How does the annual cycle of the $300 limit apply? Is it calculated based on the time that the patient first seeks treatment under the scheme?
The $300 annual limit is reset at the start of each calendar year i.e. 1 Jan to 31 Dec.

Q7. Will Medisave use be allowed for purchasing equipment (e.g. blood pressure monitoring equipment, glucometer or strips, etc.)?
No. In line with existing Medisave guidelines, Medisave use does not cover equipment purchase, whether for chronic disease treatment or other uses.

Q8. How will I know if the patient has sufficient balance left for claims?
An enquiry function to check the withdrawal limit and overall account balance is available via the MediClaim e-Service. Clinics may use this function to check the remaining balance of the Medisave account holder with his/ her consent.

Alternatively, you can request for the Medisave holders to show you a print-out or electronic statement of their current Medisave balance. They can obtain their current Medisave balance from the CPF Board's website (www.cpf.gov.sg) under My CPF Online Services - My Statement, by logging in with their SingPass. You may wish to ask your patients to bring along a copy of the Medisave balance of the Medisave payers if you do not have a computer terminal at your clinic.

Q9. If the Medisave balance is insufficient to cover the costs, can the patient top up the difference in cash?
Yes.

Q10. Can the bill be split among two or more accounts according to a given percentage?
Yes, a claim can be shared by a maximum of 10 Medisave accounts.

Q11. What is the cost of making Medisave claims?
The current cost is $2.91 (exclude GST) per transaction and has to be paid in cash. The cost is levied on the clinics and not the patients. However, some clinics may decide to pass on this cost to their patients.

Q12. Why is there a transaction cost of $2.91?
The transaction cost consists of a $2.44 charge from CPF Board for processing each Medisave account and a $0.47 charge from NCS for use of the MediClaim system.

Q13. Can I transfer the cost per transaction ($3.11 with GST) to the patient?
You may choose to do so. However, medical institutions deciding to charge out the operational transaction cost should list this item in the bill as "Medisave processing fee". This fee has to be paid in cash. Should medical institutions decide to charge out additional administrative fees on top of what MOH/CPFB charged out to them, they are required to separately attribute it to their own business administrative charges, instead of lumping it as "Medisave processing fee".

Q14. Will patients have to pay the full amount upfront and then be reimbursed or can they make partial payment based on estimated Medisave payout?
This decision will lie upon the individual clinics. However, clinics should explain to their patients on the mode of payment clearly so as to avoid any confusion or unhappiness.

Q15. Can I accumulate several bills to be submitted in a single claim for the whole year so as to decrease the cost per transaction?
Yes. The deductible and co-payment is based on a per claim basis. You will need to enter the visit date and bill details for each visit within the single claim.

Q16. How will refunds for Medisave withdrawals be handled (e.g. if a patient opts out of a package)?
The clinic will have to amend the approved Medisave claim through the MediClaim system to return the money back to the relevant Medisave accounts. CPF Board will liaise with the clinics to debit and credit the amounts accordingly. Medisave will have first claim on any refunds. As for the amount of cash co-payment collected previously ($30 deductible and 15% co-payment on the bill balance), the clinic can refund the amount to the patient in cash.

Q17. If patients have signed up for the Programme, can they opt out of it at a later date? Do I need to refund the amount that he had paid up for a package?
Patients can opt out at a later date by informing the clinic from which he/she is receiving care. In terms of refund, it is a private arrangement between the provider and the patient. Patients should find out the provider's policy on refunds before signing up for packages. However, funds withdrawn from Medisave must be reimbursed to the Medisave accounts first.

Q18. Is Medisave withdrawal dependent on the patient having only one specific primary care provider?
No. Patients are encouraged to have continuity of care with one family physician but they are free to choose and switch providers. Hence, they can make Medisave claims at any Medisave-accredited clinic.

Q19. How will claims be made if a patient is referred to an unaccredited provider?
Medisave claims will not be allowed at an unaccredited clinic. However, the referring party can make arrangements to bill on behalf of his unaccredited partners. The referring party is expected to bear full responsibility for any such arrangements made.

Q20. How will the scheme apply to Permanent Residents and Foreigners?
Current Medisave rules apply. Patients can be Permanent Residents or Foreigners. As long as they have Medisave accounts or their immediate family members with Medisave accounts, they are eligible for the scheme.

Q21. How will the scheme apply to those who have employer medical benefits or an existing comprehensive insurance plan?
Claims can be made under employer plans. This also applies to pensioners. Employer medical benefits or an existing comprehensive insurance plan can be used to cover the cost of the deductible and co-payment. Any amount in excess of the employer medical benefits or the insurance plan can be paid using Medisave. Clinics will have to liaise directly with their partnering employers for payment under employer plans as per their current arrangements.

Q22. What is the process of making Medisave claims like? Will it involve a huge change in my clinic operations?

The process is as follows:

1) The clinic/doctor should explain the following to patients suffering from any of the approved chronic diseases and their immediate family member(s) whose Medisave account(s) is/are being used (if any):
   - the treatment components.
   - the cost of treatment.
   - estimated amount that can be claimed from Medisave.
   - the out-of-pocket cash payment that the patient will need to make.
   - the charging of transaction fees.

2) When the patient and/or his/her immediate family member(s) have decided to use Medisave for the bill, each Medisave account holder who wishes to make use of his/her Medisave account need to sign a Medisave Authorisation Form (MAF) to authorise the CPF Board to deduct his/her Medisave savings for the treatment of the patient. The authorisation can be made on a per treatment basis or over a period of months. It then stands until revoked in writing. Clinic/Medical institution staff should witness the signing and verify the relationship(s) to the patient as stated in the MAF.

3) Clinics/Medical institutions can then submit the Medisave claims electronically to the CPF Board for processing via the MediClaim System.

4) Payment will be made daily to Medisave-accredited medical institutions via InterBank Giro (IBG) on the 3rd working day after the approval date of the Medisave claims.

Q23. Can GPs who are contracted by nursing homes to provide outpatient care for their residents help the ones suffering from one of the six listed chronic diseases make Medisave claims?

Yes, if the GP and his/her clinic are on the Programme. He/She can help the nursing home patients to make a Medisave claim for their outpatient chronic disease treatment(s) through his/her clinic.

D. DATA SUBMISSION, CLINICAL IMPROVEMENT AND AUDITS

Q1. Why is the patient's medical and treatment history required?

The data collected will provide a better profile of patients on CDMP. This information will be useful for fine-tuning for programme planning and management purposes.

Q2. Must the medical history be captured at each visit?

The items in the medical history data will only need to be captured once but should be updated as and when there are changes.

Q3. How do I record the actual year of diagnosis of patients with long standing chronic diseases?

The estimated year of diagnosis for the patient's chronic condition can be recorded if the exact year is not known.

Q4. Will data on all clinical parameters be required at every visit?

No. Only data on assessments or tests performed during the visit need to be captured.

Q5. Would I need to repeat HbA1c or LDL cholesterol if my patient is able to produce the results of a test done elsewhere?

You can submit the relevant details of your patient's test results that have been performed elsewhere instead of repeating the test. If you do so, please keep a copy of the record of the test results.

Q6. What if the patient is lost to follow up?

Please note it down in your clinical documentation. Alternatively, if you are using the web-based e-Service for data submission, you may also document the information using the textbox available under the Patient Participation Module present on the navigation bar. If you are using CMS for data submission, please contact your CMS provider for more details on capturing of this type of information electronically.

Q7. What if the patient refuses certain tests?

Tests are performed, when indicated, as part of the proper management of the chronic disease. As such, the physician should inform the patient as to the rationale and provide other key information regarding these tests. If the patient refuses the tests, please note this response in the patient's clinic notes.

Q8. If I missed the previous deadline for submission of clinical data, do I still need to submit the data for that period?

Yes, you should still submit the relevant data for that period as well as the current data.

Q9. Which healthcare provider should submit clinical data if the patient makes Medisave claims at three different healthcare providers during one year?

It would be appropriate for each provider to collect relevant data for the care that has been provided, and to submit the data. If they are not able to make the submission, they should forward the data to the primary physician who is coordinating the care of the patient's chronic condition so that he/she may be updated and make the submission.

Q10. If a patient starts making Medisave claims from June onwards, must I submit clinical information captured before June?

You can capture the relevant clinical data of the patient. However, for the purpose of assessing the care process and outcome of the chronic condition, the period of one year (taken from the date when the patient first enrolled into the CDMP for the chronic condition) will be used.

Q11. My patient claimed Medisave for treatment of a chronic condition when he first consulted me on 5 Jan 2009, but paid cash for three subsequent visits (in Mar, Jul, Oct 2009) for the same chronic condition. Would I still need to submit clinical data for the latter three visits?

Yes, you should continue to submit the patient's clinical data on this chronic condition for one year from 5 Jan 09.

Q12. Can the clinical data submitted be shared by different healthcare providers within the same clinic / institution / cluster?

This will depend on the electronic Clinic Management System (if any) that is used by the healthcare institution.

Q13. If I have already fulfilled the number of care components for the chronic condition, do I still need to submit clinical data subsequently?

The care components are the essential aspects of medical care that are recommended for management of the chronic diseases. The data submission system allows you to submit more than the recommended number of care components.

Q14. Will clinical data submitted be shared with the providers?

The clinical data received will be used to monitor the success of the CDMP, and also to give feedback routinely to the registered clinics for quality improvement. The release of data back to the clinics had been effected in phases. Clinical data submitted have been routinely fed back to the clinic as the online CDMP outcome reports via the Mediclaim system from the first quarter 2008 onwards. In these reports, a clinic will be able to compare its performance against the aggregated local and national performance. Over time, each clinic will also be able to track its own performance trends.

Q15. What will the clinical quality improvement process be like?

The clinical data that is monitored is useful for clinical quality improvement in the care of chronic conditions. When meaningfully used, it will empower patients to take charge of managing their chronic condition as guided and supervised by their family physician. This can improve compliance with the recommended care of the chronic condition(s) with better longer term outcomes.

Q16. What will the clinical audit process be like?

Periodic on-site audits will be carried out to ensure accuracy of clinical data submission and to ensure that minimum standards of performance are met. Due consideration will be given so that such audits do not disrupt clinic operations and patient care processes.

FURTHER READING

MOH. Chronic Disease Management Handbook for Healthcare Professionals 2011.

LEARNING POINTS

- **The User Manual in the Chronic Disease Management Programme Handbook describes the steps in the clinical data submission.**
- **The e-Service Clinical Data submission requires an Internet-enabled computer. The user needs an user account to log in at the URL page.**
- **The Clinical Data submission e-Service allows submission of new reports.**
- **It also allows retrieval of submitted reports through the "search" function.**
- **The Frequently answered questions (FAQs) that accompanies this reading explains: clinical matters; registration matters; Medisave claims, reimbursement, billing; and data submission, clinical improvement and audit matters.**

ASSESSMENT OF 30 MCQs

FPSC NO : 43
MCQs on DEMENTIA
Submission DEADLINE : 28 OCTOBER 2011

INSTRUCTIONS

- **To submit answers to the following multiple choice questions, you are required to log on to the College On-line Portal (www.cfps2online.org).**
- **Attempt ALL the following multiple choice questions.**
- **There is only ONE correct answer for each question.**
- **The answers should be submitted to the College of Family Physicians Singapore via the College On-line Portal before the submission deadline stated above.**

1. After Alzheimer Disease, which of the following is the most common etiology of dementia?
- **A. Creutzfeldt-Jakob disease.**
- **B. Vascular dementia.**
- **C. Neurosyphilis.**
- **D. Dementia associated with Parksonism.**
- **E. Frontotemporal dementia.**

2. About the clinical features of delirium, which of the following is NOT a feature?
- **A. Acute change in mental status.**
- **B. Fluctuating level of attention.**
- **C. Disorganised thinking.**
- **D. Apathy.**
- **E. None of the above are features.**

3. In the diagnosis of dementia, which of the following associations is CORRECT?
- **A. Apraxia and "Any difficulty in communication?"**
- **B. Agnosia and "Is one's work getting more disorganised?"**
- **C. Executive dysfunctioning and "Any problems with handling loose change?"**
- **D. Agnosia and "Any difficulties in using utensils during meal times?"**
- **E. Apraxia and "Any problems recognising familiar faces?"**

4. In using ECAQ, a 10-item cognitive test, to assess memory and information orientation, a cut-off score of X has 85.3% sensitivity and 91.5% specificity for identifying cognitive impairment. What is X?
- **A. 5/6.**
- **B. 6/7.**
- **C. 7/8.**
- **D. 8/9.**
- **E. 9/10.**

5. In using CMMSE, a 28-item cognitive test for assessing mild cognitive impairment, a cut-off score of X has 83% sensitivity and 94% specificity for identifying cognitive impairment. What is X?
- **A. 24/25.**
- **B. 23/24.**
- **C. 22/23.**
- **D. 21/22.**
- **E. 20/21.**

6. About common behavioural and psychological symptoms of dementia, which of the following associations is CORRECT?
- **A. Hallucinations and "Expressing the wish to die".**
- **B. Depressed mood and "Hearing deceased people call their names".**
- **C. Apathy and "Resistance to care".**
- **D. Anxiety and "Worries about their finances".**
- **E. None of the above.**

7. About common causes of behavioural and psychological symptoms in dementia, which of the following is LEAST LIKELY to be a cause?
- **A. Medications.**
- **B. Faecal impaction.**
- **C. Arthritis.**
- **D. Music.**
- **E. Faulty hearing aid.**

8. About the use of non-pharmacological intervention in a person with behavioural and psychological symptoms of dementia (BPSD), which of the following statements is CORRECT?
- **A. Aggression and agitiation is best dealt with by a firm, strong voice.**
- **B. Non-pharmacological intervention is usually the first line management for mild and moderate BPSD.**
- **C. Use of digital locks at exit doors are ethically incorrect.**
- **D. Electronic alarm systems are the most effective measures.**
- **E. Day time naps have been found to reduce episodes of BPSD at night.**

9. About the use of pharmacological management in a person with behavioural and psychological symptoms of dementia (BPSD), which of the following statements is CORRECT?
   A. "Start high and reduce" is the strategy of pharmacoogical treatment of BPSD.
   B. Treat only moderate or severe BPSD with medication.
   C. Medications should be given on a long term to prevent recurrence of BPSD.
   D. Once the symptoms of BPSP are stabilised, half the dosage of medications.
   E. Routinly start with two different types of medications to achieve faster resolution of symptoms.

10. Madam Tan, 88 years old has BPSD. An anti-pscyhotic is being considered for her hallucinations. Which of the following would you prescribe?
   A. Mirtazapine.
   B. Quetiapine.
   C. Risperidone.
   D. Olanzapine.
   E. Haloperidol.

11. About reversible causes of dementia, only a small percentage is truly reversible, most notably X besides pseudo-dementia. What is X?
   A. Hypothyoidism.
   B. B12 deficiency.
   C. Neurosyphilis.
   D. HIV-associated dementia.
   E. Neoplastic causes.

12. Trials to find medications for improving dementia have been ongoing. Which of the following has convincing evidence of preventing dementia?
   A. High dose vitamin E.
   B. Low dose prednisolone.
   C. Atorvastatin.
   D. Estrogen replacement therapy.
   E. All of the above have yielded null findings.

13. A patient with dementia is on donepezil. Which of the following is a side effect of the medication?
   A. Constipation.
   B. Increased appetite.
   C. Tachycardia.
   D. Somnolence.
   E. Vivid dreams.

14. Memantine is being considered for a patient with dementia. Which of the following is a side effect of this medication?
   A. Hepatic impairment.
   B. Reduction of effect of L-dopa.
   C. Hyperexcitation.
   D. Worsening of behavour with frontotemporal dementia.
   E. All of the above are correct.

15. Madam T, aged 80 is on donepezil. She is not responding to the therapy. Non-compliance has been excluded. Which of the following is the action to take?
   A. Increase the dose.
   B. Switch to another ChEI.
   C. Switch to memantine.
   D. Add memantine.
   E. All the above are correct.

16. About advice on symptoms of memory loss and diagnosis, who is the healthcare provider that the patient and caregiver first turn to?
   A. The lay counsellor.
   B. The specialist physician.
   C. The primary care physician.
   D. The geriactician.
   E. Friends and relatives.

17. About caregivers in Singapore for people with dementia, which of the following statements about this group of people is CORRECT?
   A. The caregivers are mostly elderly.
   B. The caregivers are usually a married son or daughter.
   C. About half of the caregivers hold a full time or part time job.
   D. The majority of caregivers are men.
   E. About 70% of families of people with dementia engage foreign domestic help to provide the caregiving.

18. On the burden of caregiving in people with mild and moderate dementia, X% of caregivers reported the caregiving process was a difficulty one. What is X?
   A. 48.
   B. 53.
   C. 58.
   D. 63.
   E. 78.

19. On the impact of burden of caregiving on caregivers, a local study showed that a prevalence of X% of caregivers who had caregiving problems experienced depression. What is X?
   A. 37.
   B. 47.
   C. 57.
   D. 67.
   E. 77.

20. The interventions of the family physician in caring of caregivers of people with dementia is stage specific. Which of the following associations of stage of dementia and intervention of the family physician is **CORRECT**?
    A. Diagnosis and disclosure stage and advice to the patient and caregiver on financial, legal planning and advance directives.
    B. Early disease stage and specific information on diagnosis and prognosis.
    C. Bereavement on the part of the family caregiver and counsel and support from the family physician.
    D. Late disease stage and management of caregivers for distress, depression, and burnout.
    E. Middle disease stage and dealing with issues of do-not-resuscitation disorders.

21. In the use of Medisave for chronic disease management of dementia, which of the following care components is **NOT** allowable?
    A. Home meal delivery.
    B. Speech therapy.
    C. Occupational therapy.
    D. Physiotherapy.
    E. Home visit evaluation.

22. About the guidelines for referral of patients with dementia from specialists to primary care physicians for follow up in the community, which of the following statements is **CORRECT**?
    A. Suitable patients must be assessed by the specialist to be financially stable.
    B. The referred patients must have a provisional diagnosis of dementia.
    C. If the patients have behavioural issues, they cannot be referred to the primary care physician.
    D. The caregivers should have been counseled on the natural history of dementia.
    E. If the patient is prescribed antidepressants and/or psychotic agents, he or she must be on stable doses of these medications for at least one month.

23. For patients with dementia, with regards to the clinical indicators for submission via electronic channels to MOH, which of the following frequency of submission is **CORRECT**?
    A. Documentation of assessment of memory – at least twice yearly.
    B. Documentation of mood and behaviour – at least quarterly.
    C. Consultation for dementia – twice yearly.
    D. For patients on cognitive enhancers, documentation of objective assessment of memory – at least quarterly.
    E. Documentation of assessment of rehabilitation needs – at least twice yearly.

24. With regards to the dosing of medications for people with dementia, which of the maximum adult recommended dose per day is **CORRECT**?
    A. Memantine – 20 mg.
    B. Fluoxetine – 45 mg.
    C. Mirtazapine – 80 mg.
    D. Rivastigmine – 10 mg.
    E. Donepezil – 12 mg.

25. About the recommended investigations for people with dementia receiving selected pharmacotherapy, which of the following associations of investigations and pharmacological agent is **CORRECT**?
    A. Full blood count – patients on anticholinesterase inhibitors.
    B. Liver function test – patients on atypical antipsychotics.
    C. Renal panel (Urea/electrolytes/creatinine) – patients on anticholinesterase inhibitors.
    D. Electrocardiogram – patients on antidepressants.
    E. None of the above associations are correct.

26. About the use of Medisave for chronic disease management programme (CDMP), which of the following mental health diseases is **NOT** included?
    A. Schizophrenia.
    B. Major depression.
    C. Borderline personality disorder.
    D. Dementia.
    E. Bipolar disorder.

27. As one of the requirements of use of the MediClaim system, the doctors and clinic staff of the participating clinic needs to attend a X-day training session on the Medisave Claim process, guidelines on Medisave Use and use of the MediClaim system? What is X?
    A. Two.
    B. One and a half.
    C. One.
    D. Half.
    E. Quarter.

28. Mr A, aged 75 years old has dementia. He is a Permanent resident. The son wishes to claim part of the medical care of the father for dementia through the Medisave chronic disease management programme. Which of the following Medisave accounts **CANNOT** be used?
    A. Patient's son.
    B. Patient's daughter-in-law.
    C. Patient's daughter.
    D. Patient's spouse.
    E. Patient's grandson.

29. A patient's medical bill to be claimed through Medisave chronic disease progarmme can be shared by a maximum of X Medisave accounts. What is X?

A. 10.
B. 8.
C. 5.
D. 3.
E. 2.

30. In the use of a Medisave account for payment for chronic disease management, there is a deductable of $X and a 15% co-payment of the bill balance for each claim that the patient has to pay in cash, regardless if the claim is for an individual visit or packaged treatment? What is X?

A. $10.
B. $15.
C. $20.
D. $25.
E. $30.

READINGS

- A Selection of Ten Current Readings on Topics Related To Dementia

A SELECTION OF TEN CURRENT READINGS ON TOPICS RELATED TO DEMENTIA –

some available as free full-text and some requiring payment

Selection of readings made by A/Prof Goh Lee Gan

READING 1 – Frontotemporal dementia

Cardarelli R, Kertesz A, Knebl JA. Frontotemporal dementia: a review for primary care physicians. Am Fam Physician. 2010 Dec 1;82(11):1372-7. Review. PubMed PMID: 21121521.

URL: http://www.aafp.org/afp/2010/1201/p1372.pdf (payment required)

Cardarelli R, Kertesz A, Knebl JA. University of North Texas Health Science Center, Fort Worth, TX 76107, USA.

ABSTRACT

Frontotemporal dementia (FTD) is one of the most common forms of dementia in persons younger than 65 years. Variants include behavioral variant FTD, semantic dementia, and progressive nonfluent aphasia. Behavioral and language manifestations are core features of FTD, and patients have relatively preserved memory, which differs from Alzheimer disease. Common behavioral features include loss of insight, social inappropriateness, and emotional blunting. Common language features are loss of comprehension and object knowledge (semantic dementia), and nonfluent and hesitant speech (progressive nonfluent aphasia). Neuroimaging (magnetic resonance imaging) usually demonstrates focal atrophy in addition to excluding other etiologies. A careful history and physical examination, and judicious use of magnetic resonance imaging, can help distinguish FTD from other common forms of dementia, including Alzheimer disease, dementia with Lewy bodies, and vascular dementia. Although no cure for FTD exists, symptom management with selective serotonin reuptake inhibitors, antipsychotics, and galantamine has been shown to be beneficial. Primary care physicians have a critical role in identifying patients with FTD and assembling an interdisciplinary team to care for patients with FTD, their families, and caregivers. PMID: 21121521 [PubMed - indexed for MEDLINE]

READING 2 – Vitamin B12 deficiency

Langan RC, Zawistoski KJ. Update on vitamin B12 deficiency. Am Fam Physician. 2011 Jun 15;83(12):1425-30. PubMed PMID: 21671542.

URL: http://www.aafp.org/afp/2011/0615/p1425.pdf (payment required)

Langan RC, Zawistoski KJ. St. Luke's Hospital, Bethlehem, PA, USA.

ABSTRACT

Vitamin B12 (cobalamin) deficiency is a common cause of megaloblastic anemia, a variety of neuropsychiatric symptoms, and elevated serum homocysteine levels, especially in older persons. There are a number of risk factors for vitamin B12 deficiency, including prolonged use of metformin and proton pump inhibitors. No major medical orga-

nizations, including the U.S. Preventive Services Task Force, have published guidelines on screening asymptomatic or low-risk adults for vitamin B12 deficiency, but high-risk patients, such as those with malabsorptive disorders, may warrant screening. The initial laboratory assessment of a patient with suspected vitamin B12 deficiency should include a complete blood count and a serum vitamin B12 level. Measurements of serum vitamin B12 may not reliably detect deficiency, and measurement of serum homocysteine and/or methylmalonic acid should be used to confirm deficiency in asymptomatic high-risk patients with low normal levels of vitamin B12. Oral administration of high-dose vitamin B12 (1 to 2 mg daily) is as effective as intramuscular administration in correcting the deficiency, regardless of etiology. Because crystalline formulations are better absorbed than naturally occurring vitamin B12, patients older than 50 years and strict vegetarians should consume foods fortified with vitamin B12 and vitamin B12 supplements, rather than attempting to get vitamin B12 strictly from dietary sources. Administration of vitamin B12 to patients with elevated serum homocysteine levels has not been shown to reduce cardiovascular outcomes in high-risk patients or alter the cognitive decline of patients with mild to moderate Alzheimer disease. PMID: 21671542 [PubMed - in process]

READING 3 – Treatment of Alzheimer disease

Winslow BT, Onysko MK, Stob CM, Hazlewood KA. Treatment of Alzheimer disease. Am Fam Physician. 2011 Jun 15;83(12):1403-12. PubMed PMID: 21671540.

URL: http://www.aafp.org/afp/2011/0615/p1403.pdf (payment required)

Winslow BT, Onysko MK, Stob CM, Hazlewood KA. Swedish Family Medicine Residency, Littleton, CO, USA.

ABSTRACT

Alzheimer disease is the most common form of dementia, affecting more than one-third of Americans older than 85 years. It is characterized by progressive memory loss and cognitive decline. Amyloid plaque accumulation, neurofibrillary tau tangles, and depletion of acetylcholine are among the pathologic manifestations of Alzheimer disease. Although there are no proven modalities for preventing Alzheimer disease, hypertension treatment, omega-3 fatty acid supplementation, physical activity, and cognitive engagement demonstrate modest potential. Acetylcholinesterase inhibitors are first-line medications for the treatment of Alzheimer disease, and are associated with mild improvements in cognitive function, behavior, and activities of daily living; however, the clinical relevance of these effects is unclear. The most common adverse effects of acetylcholinesterase inhibitors are nausea, vomiting, diarrhea, dizziness, confusion, and cardiac arrhythmias. Short-term use of the N-methyl-D-aspartate receptor antagonist memantine can modestly improve measures of cognition, behavior, and activities of daily living in patients with moderate to severe Alzheimer disease. Memantine can also be used in combination with acetylcholinesterase inhibitors. Memantine is generally well tolerated, but whether its benefits produce clinically meaningful improvement is controversial. Although N-methyl-D-aspartate receptor antagonists and acetylcholinesterase inhibitors can slow the progression of Alzheimer disease, no pharmacologic agents can reverse the progression. Atypical antipsychotics can improve some behavioral symptoms, but have been associated with increased mortality rates in older patients with dementia. There is conflicting evidence about the benefit of selegiline, testosterone, and ginkgo for the treatment of Alzheimer disease. There is no evidence supporting the beneficial effects of vitamin E, estrogen, or nonsteroidal anti-inflammatory drug therapy. PMID: 21671540 [PubMed - in process]

READING 4 – Early dementia – optimal management in general practice

Workman B, Dickson F, Green S. Early dementia--optimal management in general practice. Aust Fam Physician. 2010 Oct;39(10):722-6. PubMed PMID: 20890472.

URL: http://www.racgp.org.au/afp/201010/201010workman.pdf (free full text)

Workman B, Dickson F, Green S. Monash University, and Rehabilitation and Aged Services, Southern Health, Victoria. barbara.workman@monash.edu.au

ABSTRACT
BACKGROUND: The assessment and management of dementia is complex. General practitioners are often the first point of contact for people with dementia, and their families. General practitioners have a key role in providing quality primary care in terms of the identification, assessment, provision of information, referral and ongoing management.
OBJECTIVE: This article discusses the role of the GP in the diagnosis and management of people with dementia.
DISCUSSION: It is important GPs are aware of the importance of early detection of dementia. Dementia is a complex condition. It develops slowly and early signs of dementia are very subtle. Difficulty in detecting the transition between normal aging and the onset of dementia and the lack of a definitive diagnostic tool often precludes early diagnosis. Evidence based recommendations are available to assist GPs in the diagnosis and ongoing management of people with dementia.
PMID: 20890472 [PubMed - indexed for MEDLINE]

READING 5 – Finances in older patient with cognitive impairment

Widera E, Steenpass V, Marson D, Sudore R. Finances in the older patient with cognitive impairment: "He didn't want me to take over". JAMA. 2011 Feb 16;305(7):698-706. Review. PubMed PMID: 21325186.

URL: http://jama.ama-assn.org /content/305/7/698.full.pdf+html (free full text)

Widera E, Steenpass V, Marson D, Sudore R. VA Medical Center 181G, 4150 Clement St, San Francisco, CA 94121, USA. eric.widera@ucsf.edu

ABSTRACT
Financial capacity can be defined as the ability to independently manage one's financial affairs in a manner consistent with personal self-interest. Financial capacity is essential for an individual to function independently in society; however, Alzheimer disease and other progressive dementias eventually lead to a complete loss of financial capacity. Many patients with cognitive impairment and their families seek guidance from their primary care clinician for help with financial impairment, yet most clinicians do not understand their role or know how to help. We review the prevalence and impact of diminished financial capacity in older adults with cognitive impairment. We also articulate the role of the primary care clinician, which includes (1) educating older adult patients and their families about the need for advance financial planning; (2) recognizing signs of possible impaired financial capacity; (3) assessing financial impairments in cognitively impaired adults; (4) recommending interventions to help patients maintain financial independence; and (5) knowing when and to whom to make medical and legal referrals. Clearly delineating the clinician's role regarding identification of financial impairment could establish for patients and families effective financial protections and limit the economic, psychological, and legal hardships of financial incapacity on patients with dementia and their families. PMID: 21325186 [PubMed - indexed for MEDLINE]

READING 6 – Making decisions for people with dementia

Livingston G, Leavey G, Manela M, Livingston D, Rait G, Sampson E, Bavishi S, Shahriyarmolki K, Cooper C. Making decisions for people with dementia who lack capacity: qualitative study of family carers in UK. BMJ. 2010 Aug 18;341:c4184. doi: 10.1136/bmj.c4184. PubMed PMID: 20719843; PubMed Central PMCID: PMC2923693.

URL: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2923693/pdf/bmj.c4184.pdf (full free text)

Livingston G, Leavey G, Manela M, Livingston D, Rait G, Sampson E, Bavishi S, Shahriyarmolki K, Cooper C. Department of Mental Health Sciences, University College London, London W1W 7EJ. g.livingston@ucl.ac.uk

ABSTRACT

OBJECTIVE: To identify common difficult decisions made by family carers on behalf of people with dementia, and facilitators of and barriers to such decisions, in order to produce information for family carers about overcoming barriers. DESIGN: Qualitative study to delineate decision areas through focus groups and complexity of decision making in individual interviews.

SETTING: Community settings in London.

PARTICIPANTS: 43 family carers of people with dementia in focus groups and 46 carers who had already made such decisions in individual interviews.

RESULTS: Family carers identified five core problematic areas of decision making: accessing dementia related health and social services; care homes; legal-financial matters; non-dementia related health care; and making plans for the person with dementia if the carer became too ill to care for them. They highlighted the difficulties in making proxy decisions, especially against active

resistance, and their altered role of patient manager while still a family member. Families devised strategies to gain agreement in order to ensure that the person with dementia retained dignity.

CONCLUSIONS: The following strategies helped with implementation of decisions: introducing change slowly; organising legal changes for the carer as well as the patient; involving a professional to persuade the patient to accept services; and emphasising that services optimised, not impeded, independence. To access services, carers made patients' general practice appointments, accompanied them to the surgery, pointed out symptoms, gained permission to receive confidential information, asked for referral to specialist services, and used professionals' authority to gain patients' agreement. End of life decisions were particularly difficult. They were helped by knowledge of the person with dementia's previous views, clear prognostic information, and family support. Information sheets to help carers to overcome barriers to proxy decision making have been developed; their impact in practice has yet to be evaluated.

PMCID: PMC2923693 PMID: 20719843 [PubMed - indexed for MEDLINE]

READING 7 – Caregiver care

Collins LG, Swartz K. Caregiver care. Am Fam Physician. 2011 Jun 1;83(11):1309-17. PubMed PMID: 21661713.

URL: http://www.aafp.org/afp/2011/0601/p1309.pdf (payment required)

Collins LG, Swartz K. Jefferson Medical College, Thomas Jefferson University, Philadelphia, PA, USA. lauren.collins@jefferson.edu

ABSTRACT

In 2009, nearly 66 million Americans (three in 10 U.S. households) reported at least one person providing unpaid care as a family caregiver. More adults with chronic conditions and disabilities are living at home than ever before, and family caregivers have an even higher level of responsibility. Caring for loved ones is associated with several benefits, including personal fulfillment. However, caregiving is also associated with physical, psychological, and financial burdens. Primary care physicians can aid in the identification, support, and treatment of caregivers by offering caregiver assessments-interviews directed at identifying high levels of burden-as soon as caregivers are identified. Repeat assessments may be considered when there is a change in the status of caregiver or care recipient. Caregivers should be directed to appropriate resources for support, including national caregiving organizations, local area agencies on aging, Web sites, and respite care. Psychoeducational, skills-training, and therapeutic counseling interventions for caregivers of patients with chronic conditions such as dementia, cancer, stroke, and heart failure have shown small to moderate success in decreasing caregiver burden and increasing caregiver quality of life. Further research is needed to further identify strategies to offset caregiver stress, depression, and poor health outcomes. Additional support and anticipatory guidance for the care recipient and caregiver are particularly helpful during care transitions and at the care recipient's end of life. PMID: 21661713 [PubMed - indexed for MEDLINE]

READING 8 – Building capacity for dementia care

Lee L, Kasperski MJ, Weston WW. Building capacity for dementia care: training program to develop primary care memory clinics. Can Fam Physician. 2011 Jul;57(7):e249-52. PubMed PMID: 21753083; PubMed Central PMCID: PMC3135463.

URL: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3135463/pdf/057e249.pdf (free full text))

Lee L, Kasperski MJ, Weston WW. The Centre for Family Medicine, 10 B Victoria St S, Kitchener, ON N2G 1C5. joelinda5@rogers.com

ABSTRACT

PROBLEM BEING ADDRESSED: Currently, dementia care provided by family physicians is suboptimal and access to specialist resources is limited. With the aging population, there is a need for system-wide, programmatic interventions to improve the diagnosis and management of patients with memory difficulties. The development of primary care memory clinics addresses this need.

OBJECTIVE: The Memory Clinic Training Program aims to develop highly functioning interprofessional memory clinics that assist family physicians in providing improved care for patients with dementia and other forms of cognitive impairment.
PROGRAM DESCRIPTION: The interprofessional training program consists of a 2-day case-based workshop, 1 day of observership and clinical training at the Centre for Family Medicine Memory Clinic, and 2 days of on-site mentorship at each newly formed memory clinic.
CONCLUSION: The Memory Clinic Training Program is an accredited, comprehensive program designed to assist family practice groups with developing primary care memory clinics. These clinics aim to transform the current limited practice capability of individual family physicians into a systematic, comprehensive, interprofessional health care service that improves capacity and quality of primary care for patients with cognitive impairment and dementia.
PMCID: PMC3135463
PMID: 21753083 [PubMed - in process]

READING 9 – Preventing post stroke cognitive impairment

Ankolekar S, Geeganage C, Anderton P, Hogg C, Bath PM. Clinical trials for preventing post stroke cognitive impairment. J Neurol Sci. 2010 Dec 15;299(1-2):168-74. Epub 2010 Sep 19. PubMed PMID: 20855090.

URL: http://www.sciencedirect.com /science/article/pii/S0022510X10004193 (payment required)

Ankolekar S, Geeganage C, Anderton P, Hogg C, Bath PM. Stroke Trials Unit, University of Nottingham, Nottingham NG5 1PB, UK.

ABSTRACT
Post stroke dementia (PSD) develops in up to 40% of patients and often co-exists with Alzheimer's disease in the elderly. Unsurprisingly, the combination of stroke and dementia is associated with considerable morbidity and mortality, and is devastating to patients and carers. Limited trial evidence suggests that lowering high blood pressure reduces the development of cognitive decline, vascular dementia and PSD, although whether this relates to the magnitude of BP reduction or specific drug classes remains unclear. Biological plausibility and/or existing studies suggest that other types of drug treatments might also be effective, including choline esterase inhibitors, lipid lowering agents, antiplatelet agents, and selective serotonin reuptake inhibitors. Preventing cognitive decline and dementia post stroke is critical and large definitive trials are now needed PMID: 20855090 [PubMed - indexed for MEDLINE]

READING 10 – Reducing incidence of dementia

Ritchie K, Carrière I, Ritchie CW, Berr C, Artero S, Ancelin ML. Designing prevention programmes to reduce incidence of dementia: prospective cohort study of modifiable risk factors. BMJ. 2010 Aug 5;341:c3885. doi: 10.1136/bmj.c3885. PubMed PMID: 20688841; PubMed Central PMCID: PMC2917002.

URL: http://www.ncbi.nlm.nih.gov.libproxy1.nus.edu.sg/pmc/articles/PMC2917002/pdf/bmj.c3885.pdf (free full text)

Ritchie K, Carrière I, Ritchie CW, Berr C, Artero S, Ancelin ML. Inserm, U888 Nervous System Pathologies: Epidemiological and Clinical Research, La Colombière Hospital, 34093 Montpellier Cedex 5, France.karen.ritchie@inserm.fr

ABSTRACT

OBJECTIVE: To estimate the percentage reduction in incidence of dementia that would be obtained if specific risk factors were eliminated.

DESIGN: Prospective seven year cohort study.

SETTING: General population, Montpellier, France.

PARTICIPANTS: 1433 people aged over 65 with a mean baseline age of 72.5 years (SD 5.1).

MAIN OUTCOME MEASURES: Diagnosis of mild cognitive impairment or dementia established by a standardised neurological examination.

RESULTS: Cox models were constructed to derive hazard ratios and determine confounding and interaction effects for potentially modifiable risk factors for dementia. Mean percentage population attributable fractions were calculated with 95% confidence intervals derived from bootstrapping for seven year incidence of mild cognitive impairment or dementia. The final model retained crystallized intelligence (population attributable fraction 18.11%, 95% confidence interval 10.91% to 25.42%), depression (10.31%, 3.66% to 17.17%), fruit and vegetable consumption (6.46%, 0.15% to 13.06%), diabetes (4.88%, 1.87% to 7.98%), and apolipoprotein E epsilon4 allele (7.11%, 2.44% to 11.98%).

CONCLUSIONS: Increasing crystallised intelligence and fruit and vegetable consumption and eliminating depression and diabetes are likely to have the biggest impact on reducing the incidence of dementia, outweighing even the effect of removing the principal known genetic risk factor. Although causal relations cannot be concluded with certainty, the study suggests priorities that may inform public health programmes. PMCID: PMC2917002 PMID: 20688841 [PubMed - indexed for MEDLINE]

ORIGINAL PAPER

- **Hyperthyroidism In The Elderly**

HYPERTHYROIDISM IN THE ELDERLY

Dr Low Lian Leng, A/Prof Lee Kheng Hock

SFP2011; 37(3) (Supp 1) : 72-75

INTRODUCTION

Hyperthyroidism in the elderly is not uncommon and often presents in an atypical manner. Signs and symptoms are often non-specific and may be easily attributed to aging or diseases in other organ systems, leading to delayed diagnosis and complications. Drugs such as beta-blockers may also mask the signs of hyperthyroidism. The diagnosis can be easily made and treatment leads to a euthyroid state. Atrial fibrillation and other cardiovascular complications can be avoided and prognosis is excellent. Family physicians are often the first point of contact for these patients and will be managing such cases in their practice. We therefore need to be familiar with the presentation, diagnosis and treatment.

We describe a case that illustrates the non-specificity of symptoms in hyperthyroidism in elderly patients.

CASE DESCRIPTION

An 85-year-old lady Chinese lady was admitted to Singapore General Hospital for giddiness of a few weeks' duration. This was associated with occasional dyspnoea, palpitations, and loss of weight of unquantifiable amount and duration.

Her past medical history included hypertension for which she was on atenolol 100 mg, enalapril 5 mg, and hydrochlorothiazide 12.5 mg to be taken every morning. There was no family history of autoimmune disorders.

On examination, she was noted to have a left thyroid nodule. Physical examination did not reveal signs of thoracic outlet obstruction. She was not tachycardic and her heart rate was regular. Thyroid eye signs were absent.

Table 1 shows the results of investigations done at time of admission. There were elevation of T3 and T4. Thyrotropin (TSH) was suppressed. Her thyroid auto-antibodies (thyroid receptor auto-antibodies, anti-thyroid peroxidase, anti-thyroglobulin) were negative.

Table 1. LABORATORY RESULTS OF PATIENT ON ADMISSION

Investigations	Results	Units	Normal Values
Free Thyroxine (T3)	10.1	PMOL/L	3.2-5.3
Free Thyroxine (T4)	31.6	PMOL/L	9.6-19.1
Thyrotropin (TSH)	0.023	MU/L	0.36-3.24
Hemoglobin	10.4	G/DL	12-16
WBC	7.2	109/L	4.0-10.0
Platelets	170	109/L	140-440
Troponin T	<0.01	UG/L	Less than 0.03
Total Cholesterol	2.99	MMOL/L	Less than 5.20
HDL Cholesterol	1.12	MMOL/L	More than 1.00
Triglycerides	0.56	MMOL/L	Less than 1.70
LDL Cholesterol	1.62	MMOL/L	Less than 2.60
Pro-BNP	1184	PG/L	Less than 150
Iron	13	UMOL/L	11-27
Total Iron Binding Capacity	46	UMOL/L	39-60
Serum Folate	>45.3	NMOL/L	4.8-37.4
Serum B12	313	PMOL/L	145-637
Electrocardiogram	Multiple atrial premature complexes		
Chest X-Ray	Normal		

A thyroid uptake scan was performed and showed an enlarged left thyroid lobe containing several hot nodules, the largest nodule measures 2.8 x 1.8 x 3.2 cm. The right thyroid lobe was barely discernible.

Oesophago-duodenoscopy and colonoscopy to investigate the loss of weight did not reveal any gastro-intestinal malignancy.

A diagnosis of toxic nodular goiter with hyperthyroidism was made. The patient was started on oral carbimazole 10mg twice a day. A referral was made to the cardiology department in view of the multiple premature atrial complexes on her electrocardiogram.

She was advised by the cardiologist to continue on beta-blockers and to observe for signs and symptoms of heart failure. A follow-up appointment with the endocrinologist was arranged for her to discuss the option of radioactive iodine ablation.

CASE DISCUSSION

Thyroid Function in Normal Aging

The thyroid gland produces thyroxine (T4) and triiodothyronine (T3). With age, the thyroid gland undergoes moderate atrophy and develops nonspecific histopathologic abnormalities: fibrosis, increasing numbers of colloid nodules, and some lymphocytic infiltration. Production of T4 decreases by about 30% between young adulthood and advanced age.[1] The decrease in T4 is

LOW LIAN LENG. Resident, Department of Family Medicine and Continuing Care, Singapore General Hospital

LEE KHENG HOCK, Head and Senior Consultant, Department of Family Medicine and Continuing Care, Singapore General Hospital

considered to be physiologic compensation for decreased use of the hormone by tissue and not a manifestation of primary thyroid failure.

The body's decrease in use of T4 correlates with the age-related decline in lean body mass, suggesting that the mass of metabolically active, protein-rich tissue (i.e., muscle, skin, bone, and viscera) decreases, which may lead to reduced use and catabolism of thyroid hormones. Thyroid hormone levels rise subtly, and thyrotropin (TSH) output decreases. T3 and T4 output decreases, and serum T4 levels return to normal. When stimulated by increased TSH, the healthy aged thyroid gland can increase its hormone production normally.

Serum T3 and free T3 levels decrease moderately with age. This decline is thought to be due to a combination of decreased monodeiodination of the outer ring of T4 and decreased pituitary secretion of TSH.

Prevalence of hyperthyroidism in the elderly

In elderly patients, the prevalence of overt hyperthyroidism is 2%[2] and 10 to 15% of patients are older than 60 years.[1]

Etiology and Pathophysiology

Unlike in younger adults where Graves' Disease is more common, in elderly patients, hyperthyroidism is more often due to multinodular goitre[3]. Adenomas autonomously produce and secrete excessive thyroid hormone even though TSH production is fully suppressed. Another common cause of hyperthyroidism among elderly patients is iodine-induced hyperthyroidism, often from the use of amiodarone, a cardiac drug containing iodine that deposits in tissue and delivers iodine to the circulation over very long periods of time. The thyroid gland has the unique function to concentrate iodine to thousand times that of blood and hence is affected. Transient hyperthyroidism from subacute thyroiditis is less common and rarely clinically significant as it resolves within weeks.

Atypical presentation

Atypical presentation is common in elderly patients with hyperthyroidism. Only about 25% of hyperthyroid patients aged 65 years of age present with typical symptoms and signs[3]. These age-related differences in symptoms are the result of the aging process and of concomitant disease and medications that modifies the effects of excessive thyroid hormone. For example, cardiac disease and heart failure is common in elderly persons, so the possibility of underlying hyperthyroidism may not be suspected. Gastrointestinal (GI) symptoms may be confused with GI malignancy. A decreased number or affinity of catecholamines receptors results in a decreased response to catecholamines in the elderly.

The classical triad of hyperthyroidism in older patients is tachycardia, weight loss, and fatigue. Constipation is present in more than 20% of elderly patients, while diarrhoea is uncommon. Sweating, hyper-reactive reflexes, nervousness and anxiety are also far less common in elderly hyperthyroid patients. Table 2 shows the clinical features of hyperthyroidism in elderly patients. Note then seven signs of hyperthyroidism which are seen commonly in younger patients but are seen less often in older patients: hyperactive reflexes, increased sweating, heat intolerance, tremor, nervousness, polydipsia, and increased appetite[4]. The thyroid gland is normal in size or impalpable in about 40% of cases, enlarged and nodular in 35%, and enlarged and diffuse in 25%.

Table 2. CLINICAL FEATURES OF HYPERTHYROIDISM IN ELDERLY PATIENTS

More Common	Less Common
Tachycardia	Diarrhoea
Fatigue & weakness	Nervousness and Anxiety
Atrial Fibrillation	Hyperkinesis
Arrhythmias	Sweating
Angina, Heart Failure	Hyperreflexia
Constipation	Ocular Signs i.e. Exophthalmos
Changes in Appetite	
Neuropsychiatric Symptoms i.e. Apathy,	
Weight loss	
Smaller Thyroid Gland	
Multinodular Gland	

Complications

The most common complication in elderly patients is atrial fibrillation, which occurs in 27% of elderly hyperthyroid patients at presentation. There is a statistically significant association between atrial fibrillation and hyperthyroidism.5 Risks for heart failure and early death are increased if atrial fibrillation does not convert to normal sinus rhythm when euthyroidism is restored. Atrial fibrillation also carries a high risk of embolic stroke. Other important complications are depression (called apathetic thyroidism), myopathy, and osteoporosis.

Diagnosis

Laboratory diagnosis of hyperthyroidism is usually straightforward if there is a high index of suspicion. Most elderly hyperthyroid patients have increased serum concentrations of unbound thyroxine (T4) and tri-iodothyronine (T3), and reduced concentration of thyrotropin (TSH).[6]

Serum TSH measurement is the best single test for the diagnosis of hyperthyroidism. However it is important to remember that TSH level may be low in some normal elderly individuals as well as patients receiving glucocorticoid therapy and patients with non-thyroidal illness eg. sick euthyroid state. About 1% of patients have normal amounts of free T4 in serum and raised values of T3; this is called T3 toxicosis. Serum T4, T3, and thyroglobulin levels are on the average lower in older

patients with hyperthyroidism than in younger patients.

Drugs in particular amiodarone should be considered when interpreting an abnormal thyroid function test and can be associated with both hyper and hypothyroidism.

To determine the etiology of hyperthyroidism, investigations such as a thyroid uptake scan, thyroid ultrasound, or Fine Needle Aspiration Cytology (FNAC), thyroid antibodies can be considered.

Prognosis and Treatment

The prognosis for hyperthyroidism in the elderly is excellent. Treatment usually leads to a euthyroid state. If hypothyroidism results, it is treated easily with levothyroxine sodium. The three treatment strategies for hyperthyroidism namely, medication to suppress the gland, surgery to remove the hyper functioning tissue, and radioactive iodine (RAI) to destroy the gland are still applicable to the elderly although the preferred choice depends on the etiology.

In multinodular toxic goitre, surgery[8] may be preferred as the response to 131I therapy is often delayed and incomplete.

When hyperthyroidism is due to subacute thyroiditis, Hashimoto's disease, or acute radiation damage, the only effective treatment is to give beta-blockers and to closely observe the patient for complications. Antithyroid drugs and 131I are not helpful because they do not decrease the uncontrolled output of hormone from damaged thyroid follicles.

The usual treatment of iodine-induced hyperthyroidism is high doses of antithyroid drugs and a beta-blocker. Treatment may be difficult because the large store of thyroid hormone in the gland blunts the effect of antithyroid drugs, and the large pool of iodine throughout the body markedly decreases the uptake of 131I.

Treatment of choice for most elderly patients with Graves' disease or a single autonomous nodule is radioactive sodium iodide (131I). It is preferred because it is easy to administer and it avoids any age-related postoperative complications of surgery. Antithyroid drugs (e.g., propylthiouracil, carbimazole) are effective in the treatment of Graves' disease if the patient's adherence with the regimen is good. However, in patients with uninodular toxic goitre, antithyroid drugs are slow to take effect and rarely lead to permanent remission. Unlike in Graves' disease, post-ablative hypothyroidism does not arise routinely, since the isotope is not concentrated in the contra lateral suppressed thyroid tissue.[7] Long-term antithyroid drug treatment for Graves' Disease usually lasts 1 to 2 years. Antithyroid drug therapy is usually successful if the patient is compliant. As mild hyperthyroidism and a small thyroid gland are characteristic in elderly patients, the chance of permanent remission is enhanced. If hyperthyroidism recurs after antithyroid drug treatment, 131I should be considered.

Antithyroid drugs as primary therapy in elderly persons are administered in the same way as in younger persons. Propylthiouracil may be initiated at 150 to 300 mg/day orally in divided doses every 8 hourly. The dosage can be adjusted based on serum TSH levels. Carbimazole can be started at 15 to 40 mg/day and given as a single daily dose. Propranolol and other beta-blockers can be added on to manage sympathetic symptoms of hyperthyroidism. Beta-blocker related bradycardia may occur once the patient returns to a euthyroid state and needs to be observed for. In patients with atrial fibrillation and high thyroid hormone levels, cardioversion should not be attempted until a euthyroid state is achieved. Once it is, the atrial rhythm spontaneously reverts to normal in about two thirds of patients. Psychiatric symptoms usually resolve when the patient becomes euthyroid but should be treated if necessary. Standard measures to prevent osteoporosis are indicated, particularly in elderly women.

Subclincal hyperthyroidism

This merits discussion here. Subclinical hyperthyroidism is defined as a below-normal TSH concentration concurrent with normal triiodothyronine and T4 levels. Patients are usually euthyroid without the specific signs or symptoms associated with overt hyperthyroidism. Causes of subclinical hyperthyroidism include endogenous overproduction (Graves disease and nodular thyroid disease), excessive levothyroxine administration, and adverse effects of medications such as amiodarone. Progression to overt disease occurs at a rate of 1-2% per year when TSH is less than 0.1 mU/L and up to 5% per year in patients with multinodular goitre.[9,10] Current recommendations suggest to repeat the thyroid function tests within 2 to 3 months, or as early as 2 weeks later if the patient has atrial fibrillation or established cardiac disease.[8] Evidence supports treatment in patients with a TSH less than 0.1mU/L, multinodular goitre, or symptoms (including known cardiac and skeletal complications)[11]. General treatment options include antithyroid medications, radioactive iodine and partial thyroidectomy.

CONCLUSION

Hyperthyroidism in this elderly patient presents with weight loss as the cardinal complaint, and on examination there was a left thyroid nodule. There was no tachycardia and the pulse was regular. Laboratory investigations confirmed hyperthyroidism and thyroid scan showed multiple hot nodules.

REFERENCES

1. Kennedy JW, Caro JF. The ABC of managing hyperthyroidism in the older patient. Geriatrics 1996; 51:22-32.

2. Maugeri D, Russo MS, Carnazzo G, et al. Altered laboratory thyroid parameters indicating hyperthyroidism in elderly subjects. Arch Gerontol Geriatr 1996; 22:145-53.

3. Juan J. Díez. Hyperthyroidism in patients older than 55 years: an analysis of the etiology and management. Gerontology 2003; 49:316-23.
4. Trivalle C, Doucet J, Chassagne P, et al. Differences in the Signs and Symptoms of Hyperthyroidism in Older and Younger Patients. J Am Geriatr Soc 1996; 44: 50-3.
5. Cobler JL, Williams ME, Greenland P. Thyrotoxicosis in institutionalized elderly patients with atrial fibrillation. Arch Intern Med 1984; 144: 1758-60.
6. Thorkild Friis et al. Thyrotoxicosis in the elderly Scandinavian Journal of Primary Health Care, Volume 3, Issue 1 February 1985, pp 7-8.
7. Szabolc I, Podoba J, Feldkamp, et al. Comparative screening for thyroid diseases in old age in areas of iodine deficiency, long term iodine prophylaxis and abundant iodine intake. J Clin Endocrinol (oxf) 1997 Jul; 47(1): 87-92.
8. Todd CH. Management of thyroid disorders in primary care: challenges and controversies. Postgrad Med J 2009;85:655-9.
9. Surks MI, Ortiz E, Daniels GH et al. Subclinical thyroid disease: scientific review and guidelines for diagnosis and management. JAMA 2004;291:228-38.
10. Toft AD. Subclinical hyperthyroidism. N Engl J Med. 2001;345:512-6.
11. GharibH, Tuttle RM, Baskin HJ, et al. Consensus statement: subclinical thyroid dysfunction: a joint statement on management from the American Association of Clinical Endocrinologists, the American Thyroid Association, and The Endocrine Society. J Clin Endocrinol Metab.2005;90:581-5.

Authors are invited to submit material for publication in The Singapore Family Physician on the understanding that the work is original and that it has not been submitted or published elsewhere. Your article will be considered for publication on the understanding that they have to be approved by the Editorial Board via a double-blinded peer-review process and subject to revision. Authors are encouraged to consult the recommendations in the Uniform Requirements for Manuscripts Submitted to Biomedical Journals (http://www.icmje.org/index.html) which the SFP is in accord with.

The following types of articles may be suitable for publication: case reports/study, original research works, audits of patient care, protocols for patient or practice management, letters to the Editor, CME and review articles. The article should be written in British English, and not be more than 2000 words in length. This must be submitted in an electronic form and of a format that is compatible with major word processor applications. Submissions in Microsoft Word in Word 1997-2003 format (.doc) is preferred, later versions (.docx) will not be accepted.

From 31 January 2010 all articles submitted for publication must be submitted electronically through the SFP Editorial Manager, our online submission and peer-review system which can be accessed at www.editorialmanager.com/sfp/default.asp.

All instructions for registration and submission can be found at the webpage. Authors and reviewers can follow clearly the progress of the manuscript submission and review process by logging into the SFP Editorial Manager. An online users' guide, authors' and reviewers' instructions are also located at the website in case of queries and difficulties. Any problems encountered logging in can be addressed to sfp@cfps.org.sg.

RECOMMENDED FORMAT FOR THE MANUSCRIPT

The submission should comprise of the following:

1. Title Page
2. Summary/Abstract
3. Text/Manuscript (anonymised version)
4. Tables
5. Illustrations
6. Authors Agreement/Copyright Assignment Form
7. Patient's Consent Form, if necessary

Each one of these sections should start on a fresh page.

Authors are advised to ensure the anonymity of study subjects and patients by removing any and all information that could compromise their privacy from the submission.

The Title Page

- The title should be short and clear.
- Include on the title page first name, qualifications, present appointments, type and place of practice of each contributor.
- Include name, address and telephone number of the author to whom correspondence should be sent.
- Insert at the bottom: name and address of institution from which the work originated.

The Summary/Abstract

- The summary should describe why the article was written and present the main argument or findings.
- Limit words as follows: 200 words for major articles; 200 words for case reports.

Key Words

- Add, at the end of summary in alphabetical listing, keywords of up to 8 in number which will be used for article indexing and retrieval.

The Text (full complete)

The text should have the following sequence:

- **Introduction:** State clearly the purpose of the article.
- **Materials and methods:** Describe the selection of the subjects clearly. Give References to established methods, including statistical methods; provide references and brief descriptions of methods that have been published but are not well known. Describe new or substantially modified methods, giving reasons for using them and evaluate their limitations. Include numbers of observations and the statistical significance of the findings where appropriate.
  Drugs must be referred to generically; all the usual trade names may be included in parentheses.
  Dosages should be quoted in metric units.
  Laboratory values should be in SI units with traditional unit in parentheses.
  Do not use patients' names, initials or hospital numbers to ensure anonymity.
- **Results:** Present results in logical sequence in the text, table and illustrations.
- **Statistics:** Describe statistical methods which can be easily understood and verified by the reader. Use technical terms in its proper place, and where possible quantify readings and indicate errors of uncertainty and confidence intervals.
- **References** should be cited consecutively by numbers in parenthesis and listed at the end of the article in numerical order. Where there are more than three authors, the first three should be named and then followed by et al. This should be written in the style as follows:

  Elder JT, Travakkol A, Klein SB, et al. Protooncogene expression in normal and psoriatic skin. J Invest Dermatol, 1990;94:19-20.

  Authors may wish to familiarise themselves with the AMA style for the citing of references for BioMedical publications at www.amamanualofstyle.com.

Tables

- Tables should be submitted on a separate page. Label them in roman-numeric sequence [I, II, III, etc] and ensure they are clear and with explanatory legends as required.

Illustrations

- Illustrations must be submitted in a separate page, and should be provided whenever appropriate. Illustrations should be cited in the text. When required, it is the author's responsibility to obtain permission to reproduce illustrations. Authors need to ensure that photographs, illustrations and figures do not contain any information that will reveal the identities of the patients and authors. Captions to the figures should be provided.

Anonymised Text

As the article will be subjected to a double-blinded peer review process, all identification of names and institutions have to be removed from this version to facilitate the peer review process.

Revised Manuscript Submission

Manuscripts may be returned to their respective authors for revision. This will be accompanied by an Editor's email for which comments and recommendations may be made. The authors are advised to read and to take note of these comments carefully and to revise their articles accordingly. The authors need to reply to the editor's email to outline their response before the resubmission of the revised manuscript. They should exclude the identity of the authors and their institutions, as the email may be redirected to the reviewers during the resubmission process. The resubmitted manuscripts should include the revised complete version, as well as the anonymized version as before.

Proofs

Prior to publication, the Editorial Team will copyedit the article to fit the format of the Journal. The author will be sent the copyedited proof of the article, and the author should read carefully the proof and give comments and/or confirmation within 48 hours of receiving the proof. This will greatly facilitate the SFP to proceed to printing without delay, or to have to go to print without the corresponding author's comments.

Reprints

1 complimentary copy of the article will be given to the authors. Additional copies can be ordered at authors' rates at time of printing.

Correspondence & Enquiries should be addressed to:

The Honorary Editor
The Singapore Family Physician
College of Family Physicians Singapore
College of Medicine Building
16 College Road #01-02
Singapore 169854
Tel: 6223 0606 Fax: 6222 0204
Email: sfp@cfps.org.sg

Circulation

The Singapore Family Physician is published quarterly. It is circulated to all Fellows, Collegiate Members, Ordinary Members and Associate Members of the College of Family Physicians Singapore, and to private and institutional subscribers. It is also published online and can be found at www.cfps.org.sg.

The journal is also circulated to all relevant government, professional, medical and academic organisations in Singapore, sister Colleges overseas and to the World Organization of National Colleges and Academies of General Practitioners/Family Physicians (WONCA)