Objectives

- Definition of dementia
- Brief overview of dementia
- Dementia as both a clinical and social challenge
- Definition of non-cognitive symptoms of dementia
- Management of non-cognitive symptoms of dementia
Dementia (ICD-10, 1992)

* a syndrome due to disease of the brain, usually of a chronic or progressive nature, in which there is disturbance of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capability, language, and judgement. Consciousness is not impaired.
Dementia (ICD -10, 1992)

- Impairments of cognitive function are commonly accompanied, occasionally preceded, by deterioration in emotional control, social behaviour, or motivation. The syndrome occurs in Alzheimer’s disease, in cerebrovascular disease, and in other conditions primarily or secondarily affecting the brain.
Causes of dementia

- Alzheimer’s disease (AD) accounts for around 60% of all cases;
- Other common causes in older people include cerebrovascular disease (vascular dementia [VaD]) and dementia with Lewy bodies (DLB) (accounting for 15–20% of cases each).
- In cases of young onset, frontotemporal dementia (FTD) is also a common cause, second only to AD.
Causes of dementia

- Other degenerative diseases (for example, Huntington’s disease)
- Prion diseases (Creutzfeldt-Jakob Disease [CJD])
- HIV dementia
- Several toxic and metabolic disorders (for example, alcohol-related dementia).
- Dementia also develops in between 30–70% of people with Parkinson’s disease, depending on duration and age (Aarsland et al., 2003)
Causes of dementia

- Mixed cases of dementia, such as AZD and VaD, are encountered
- This is probably quite common in the local population, which has the risk factors of advanced age and high CND rates
- Each aetiological factor contributes to the clinical expression of disease (Snowdon et al., 1997)
Dementia vs. normal aging

- Dementia can be distinguished from the mild and variable cognitive decline associated with normal aging by the severity and global nature of cognitive impairment and the accompanying functional disability that result.
Presentation of dementia - AZD

* AZD
  * early sign is memory loss esp. for learning new information (hippocampus, medial temporal lobe)
  * later, higher cortical function affected: language, praxis, executive function
Presentation of dementia - VaD

- Can present after a single acute vascular event (such as a stroke)
- Can present insidiously
  - progressive attentional and executive/planning problems
  - gait disturbance and apraxia reflecting (‘subcortical’ frontostriatal dysfunction)
  - focal neurological signs are common*
Presentation of dementia - FTD, DLB

- FTD usually presents with language disturbance and/or behavioural difficulties (disinhibition or apathy)
- DLB characterized by recurrent visual hallucinations, fluctuating cognitive disturbance, and motor features of parkinsonism
  - Associated features in DLB are falls, disturbances of consciousness, autonomic dysfunction and rapid eye movement (REM) sleep behaviour disorder (McKeith et al., 2005).
Non-cognitive symptoms

- Behavioural/psychiatric symptoms in later stages
  - depression (AZD, VaD)
  - apathy (AZD, VaD)
  - agitation (AZD, VaD, FTD, DLB)
  - psychosis (hallucinations, delusions) (AZD, DLB)
  - wandering
  - aggression
  - incontinence
  - altered eating habits
Non-cognitive symptoms

- Important because they are frequent
- Often cause more disruption than cognitive symptoms
- May increase risk to client and caregivers more than cognitive symptoms
- Cause greater distress to caregivers and clients than non-cognitive symptoms
Management of non-cognitive symptoms

- Non-pharmacological management is essential
- Supervision consistent with the patient’s level of function is essential
- Environment that is adapted to patient’s needs
- Adequate support for assistance with activities of daily living, and maintaining remaining function wherever possible
Management of non-cognitive symptoms

- Non-cognitive symptoms and behaviours that pose a challenge can be the most difficult aspects of dementia syndromes to manage, both for caregivers and clinicians.

- Management can be pharmacological and non-pharmacological, and it is important to know when either type or a combination of both is necessary.

- Such behaviours include aggression, agitation, wandering, hoarding, sexual disinhibition, apathy and disruptive vocal activity such as shouting.
Management of non-cognitive symptoms

- There is also the possibility that a stressed carer behaves in ways that elicit more difficult behaviour from a person with dementia (Woods, 2001).

- Therapy must be individualized to the patient and their care situation wherever possible
Non-pharmacological management of non-cognitive symptoms

- Several non-pharmacological interventions exist
- Behavioural interventions have proved extremely effective for all categories of non-cognitive symptoms than psychological interventions, including depression and anxiety (Livingston et al, 2005)
Behavioural interventions

- Occupational/structured activities
- Environmental interventions/modifications
- Sensory enhancement/relaxation
- Social contact
- Staff training
**Behavioural interventions**

* Medical/nursing care interventions (including bright-light therapy/ sleep interventions, pain management, hearing aids, removal of restraints)
  – Combination therapies
  – Interventions involving working with carers
  – Multi-sensory stimulation
  – Psychodrama
  – Subjective barriers to prevent wandering
It is important to avoid compounding feelings of failure and humiliation where people with dementia have difficulty with interventions, activities and games (Pulsford et al, 2000).

Regular review of the effectiveness of interventions is necessary, as effective can change with time and with increasing severity of dementia.
Pharmacological interventions

- Those who develop one of the dementias are more likely than the general population to suffer from depression and/or psychosis (Robert et al., 2005).
- Many classes of drugs have been used to treat the non-cognitive symptoms of dementia, and much use has been off-label.
Pharmacological interventions

- Classes include
  - antipsychotics
  - anticonvulsants (mood stabilisers)
  - benzodiazepines
  - acetylcholinesterase inhibitors
  - NMDA antagonist
Pharmacological interventions

- The potential benefits of using these drugs, such as reduced levels of depression and neuropsychiatric symptoms, must be weighed against the potential risk of side effects and serious adverse events.

- In particular, a number of these drugs may cause confusion or worsen cognition, especially drugs that have anticholinergic properties, for example antipsychotics.
In people with AD or VaD, there is moderate- to high-quality evidence that atypical antipsychotic drugs (aripiprazole 15mg/day for 10 weeks, olanzapine 2.5 to 10 mg/day for 6 to 10 weeks, quetiapine 50 to 100 mg/day for 26 weeks, risperidone 0.5 to 2mg/day for 10 to 13 weeks) when compared with placebo produce small benefits in terms of reduced neuropsychiatric symptoms
Antipsychotics

- Typical antipsychotics appear to produce similar benefits to the atypical antipsychotics, although there is a paucity of head-to-head trials.

- There is evidence of increased risk of somnolence, hostility, confusion, fever/flu syndrome, abnormal gait, urinary incontinence, asthenia and peripheral oedema when compared to placebo.

- With regard to safety, all antipsychotics studied appear to increase the risk of death when compared to placebo.
In people with AD or VaD with clinically significant agitation, there is moderate-quality evidence suggesting that both antipsychotic drugs and benzodiazepine drugs administered by IM injection, when compared with placebo, may produce benefits in terms of reduced psychotic symptoms and aggression/agitation that outweigh the risk of adverse events.
Benzodiazepines

- The risk of excessive drowsiness, falls with resultant injury etc. that may occur with benzodiazepine use must be considered.
Acetylcholinesterase Inhibitors

* In people with AD, there is moderate-quality evidence suggesting that donepezil when compared with placebo, produces benefits in terms of reduced neuropsychiatric symptoms and agitation/aggression that outweigh the risk of adverse events.

* In people with VaD, there was insufficient evidence to determine whether acetylcholinesterase inhibitors, when compared with placebo, produce benefits in terms of neuropsychiatric symptoms that outweigh the risk of adverse events.
Memantine (NMDA antagonist)

- In people with AD, there is insufficient evidence to determine whether memantine produces clinically important improvements in neuropsychiatric symptoms.

- In people with VaD, there was insufficient evidence to determine whether acetylcholinesterase inhibitors, when compared with placebo, produce benefits in terms of neuropsychiatric symptoms that outweigh the risk of adverse events.
Mood stabilisers

There is currently **insufficient evidence** to support the use of mood stabilisers.

With regard to valproate, current evidence from one meta-analysis and five RCTs **did not strongly support** its efficacy for global BPSD, including agitation and aggression.
Mood stabilisers

- The single RCT investigating the effect of oxcarbazepine on agitation and aggression showed negative results.
- Thus far, among mood stabilizers, carbamazepine has the most robust evidence of efficacy.
## Summary


<table>
<thead>
<tr>
<th>Key Symptom</th>
<th>First Line</th>
<th>Second Line</th>
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<tbody>
<tr>
<td>Apathy</td>
<td>Sertraline, citalopram</td>
<td>Donepezil, Rivastigmine, Galantamine S; Galantamine S</td>
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<tr>
<td>Psychosis</td>
<td>Risperidone</td>
<td>Olanzapine, aripiprazole, memantine</td>
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<tr>
<td>Aggression</td>
<td>Risperidone, haloperidol</td>
<td>olanzapine, aripiprazole, memantine, carbamazepine, lorazepam</td>
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<tr>
<td>Moderate agitation/anxiety</td>
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<td>trazodone, lorazepam, memantine, mirtazapine</td>
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<tr>
<td>Severe</td>
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<td>aripiprazole, lorazepam</td>
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THANK YOU

Questions
References


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