Diagnosis and management of Dementia

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Dr. Jahnavi Kedare
Associate Professor
Dept. of Psychiatry,
T. N. Medical College
B. Y. L. Nair Hospital, Mumbai

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Prevalence of Dementia

- Lower than developed countries
- 1.4-4.4% according to various studies
- Vascular risk factors, Type II Diabetes, high prevalence of smoking, anticipated lifestyle changes – prevalence rate may increase in near future.

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- Normal Aging: Speed of information processing and psychomotor performance decline.
- Mild Cognitive Impairment: Complaints of poor memory, normal activities of daily living and nlrmal general cognitive function
- Dementia: Impairment of multiple cognitive abilities, including memory, sufficient to interfere with self-maintenance, work or social relationships.

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Major Neurocognitive Disorders

- **Diagnostic Criteria**

A. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual motor, or social cognition) based on: Concern of the individual, a knowledgeable informant, or the clinician that there has been a significant decline in cognitive function; and A substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

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B. The cognitive deficits interfere with independence in everyday activities (i.e., at a minimum, requiring assistance with complex instrumental activities of daily living such as paying bills or managing medications).

C. The cognitive deficits do not occur exclusively in the context of a delirium

D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia).

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Specify whether due to:

- **Alzheimer's disease**
- **Frontotemporal lobar degeneration**
- **Lewy body disease**
- **Vascular disease**
- **Traumatic brain injury**
- **Substance/medication use**
- **HIV infection**
- **Prion disease**
- **Parkinson's disease**
- **Huntington's disease**
- **Another medical condition**
- **Multiple etiologies**
- **Unspecified**

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Mild Neurocognitive Disorder

Diagnostic Criteria

A. Evidence of modest cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on: Concern of the individual, a knowledgeable informant, or the clinician that there has been a mild decline in cognitive function; and A modest impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

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B. The cognitive deficits ***do not interfere with capacity for independence in everyday activities*** (i.e., complex instrumental activities of daily living such as paying bills or managing medications are preserved, but greater effort, compensatory strategies, or accommodation may be required).

C. The cognitive deficits do not occur exclusively in the context of a delirium.

D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia).

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Specify whether due to

- **Alzheimer's disease**
- **Frontotemporal lobar degeneration**
- **Lewy body disease**
- **Vascular disease**
- **Traumatic brain injury**
- **Substance/medication use**
- **HIV infection**
- **Prion disease**
- **Parkinson's disease**
- **Huntington's disease**
- **Another medical condition**
- **Multiple etiologies**
- **Unspecified**

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Specify:

- **Without behavioral disturbance:** If the cognitive disturbance is not accompanied by any clinically significant behavioral disturbance.
- **With behavioral disturbance** *(specify disturbance):* If the cognitive disturbance is ac-companied by a clinically significant behavioral disturbance (e.g., psychotic symptoms, mood disturbance, agitation, apathy, or other behavioral symptoms).

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Subtypes

- Based on etiological/pathological entity or entities underlying the cognitive decline.
- These subtypes are distinguished on the basis of a combination of time course, characteristic domains affected, and associated symptoms.
- For certain etiological subtypes, the diagnosis depends substantially on the presence of a potentially causative entity, such as Parkinson's or Huntington's disease, or a traumatic brain injury or stroke in the appropriate time period.
- For other etiological subtypes (generally the neurodegenerative diseases like Alzhei-mer's disease, frontotemporal lobar degeneration, and Lewy body disease), the diagnosis is based primarily on the cognitive, behavioral, and functional symptoms.

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Specifiers

- Psychotic Features
- Mood disturbances,
- Agitation
- Behavioral Symptoms: Sleep disturbances
- Apathy
- Other important behavioral symptoms: wandering, disinhibition, hyperphagia, and hoarding.

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Differential Diagnosis of Dementia

- Delirium
- Amnestic Disorders
- Depression
- Schizophrenia
- Bipolar Mood Disorder
- Mental Retardation
- Factitious disorder

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Clinical Features

- Cognitive Impairment: Memory, Language and aphasia, Orientation, Agnosia, Activities of daily living, Apraxia, Problem solving abilities, Executive functioning, Social and Community functioning, Intellectual functioning, Judgement
- Functional Impairment
- Neuropsychiatric manifestations

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- Behavioral disturbances
- Mood changes
- Anxiety
- Personality change
- Psychosis
- Sleep disturbances

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Assessment

The first part of clinical assessment is aimed at establishing the cause of dementia syndrome.

Early identification of reversible causes could improve the treatment outcome.

Investigations should be targeted at identifying the treatable causes of the dementia syndrome.

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Diagnosis

- History
- Physical Examination
- MSE with an emphasis on Attention and concentration, recent and remote memory, language, executive functioning, visuospatial skills
- Neuropsychological testing
- Investigations
- Scales

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- Detailed history
- Informant
- Present complaints
- Past history
- Family history
- Substance Use
- Stressors

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Medical evaluation

- Blood chemistries: Correctable or contributory causes
- Complete blood count and differential count
- Electrolytes, blood urea nitrogen, creatinine, proteines, and liver function tests
- Blood sugar
- Folate
- Vitamin B12 level
- Homocysteine
- Lipid profile
- Thyroid-stimulating hormone
- Serological tests for syphilis, HIV if indicated

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Neuropsychological Tests

- Screening with scales like MMSE, ACE-R, MoCA, Mattis Dementia Rating Scale
- CDRS
- Luria- Nebraska Battery
- NIMHANS Battery
- AIIMS Battery
- PGI Battery

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Neuro-psychological testing for various cognitive domains

Assessments
Global cognitive status
Dementia Rating Scale–2
Mini-Mental State Examination
Montreal Cognitive Assessment
Intellectual ability
Selected subtests from WAIS-III[a] or WAIS-IV
Premorbid intellectual level estimation
National Adult Reading Test—Second Edition
Wide Range Achievement Test—3, Reading subtest
Wechsler Test of Adult Reading
Attention and processing speed
Digit span subtest (WAIS-IV, WAIS-III, or RBANS)
Trail Making Test, Part A
Digit Symbol-Coding subtest (WAIS-IV, WAIS-III)
Executive function
Wisconsin Card Sorting Test
Trail Making Test, Part B
Stroop Interference Test
Delis-Kaplan Executive Function System

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Learning and memory

Verbal learning and memory

California Verbal Learning Test (CVLT, CVLT-II)

Hopkins Verbal Learning Test—Revised

Rey Auditory Verbal Learning Test

Logical Memory subtest (WMS-IV, WMS-III, WMS-R)

Nonverbal memory

Visual Reproduction subtest (WMS-IV, WMS-III, WMS-R)

Rey-Osterrieth Complex Figure, Immediate and Delayed Recall

Visuospatial functioning

Block Design subtest (WAIS-IV, WAIS-III)

Clock drawing

Rey-Osterrieth Complex Figure, Copy

Language

Boston Naming Test (60-, 30-, and 15-item versions)

Verbal fluency (letter fluency [FAS, CFL] and category fluency [animals, fruits])

Vocabulary subtest (WAIS-IV, WAIS-III)

Boston Diagnostic Aphasia Examination—Third Edition

Multilingual Aphasia Examination—Third Edition

- Neuroimaging: Infarcts, Mass lesions, Tumours, Hydrocephalous, Correlate findings with clinical picture
- Computed tomography (CT) or
- Magnetic resonance imaging (MRI)
- PET/SPECT

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Diagnostic questions in dementia most relevant to neuroimaging

I. Structural imaging most valuable

- Is there a mass lesion?
- Is there cerebrobascular disease?
- Is there ventricular enlargement?
- Is there generalizes volume loss?
- Is there focal volume loss?

II. Molecular imaging most valuable

- What is the pattern of hypometabolism?
- Is there loss of dopaminergic neurons?
- Are there amyloid plaques?

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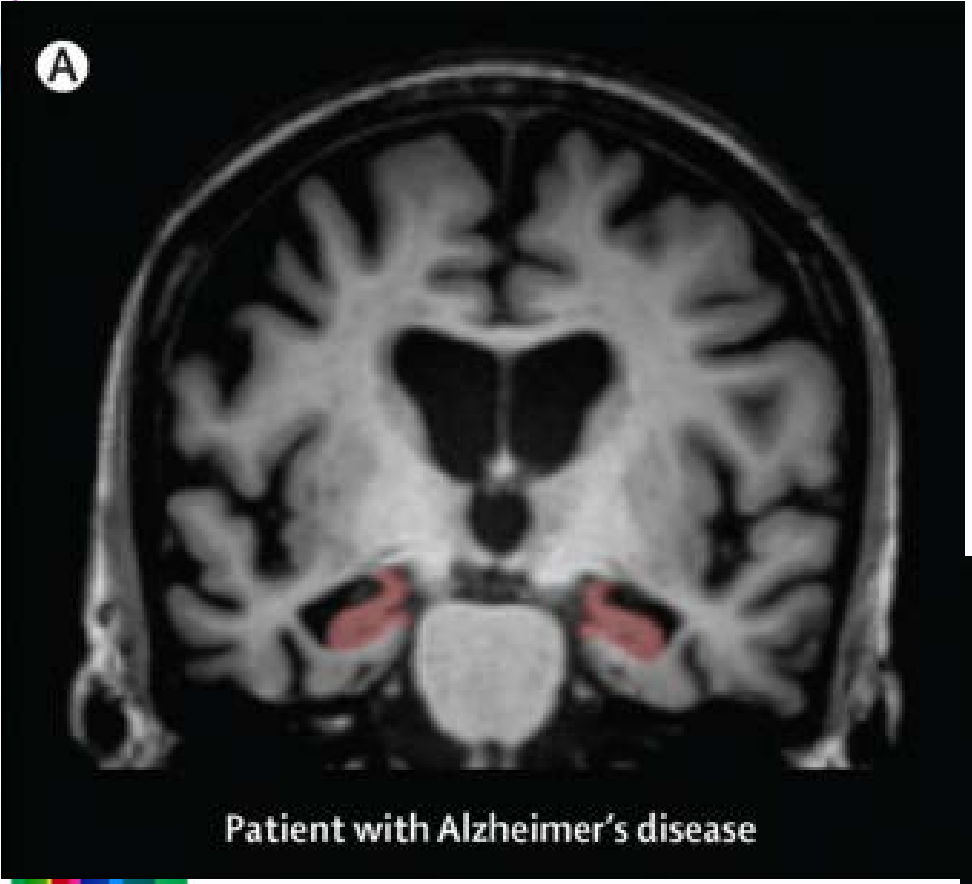
A
Patient with Alzheimer's disease


B
Healthy control

Typical patterns of regional cerebral glucose metabolism in common dementing diseases

Disease	Pattern of glucose hypometabolism
Alzheimer disease	Symmetric or asymmetric bilateral temporoparietal and posterior cingulate; lesser frontal association cortex; sparing of primary sensorimotor and visual cortex
Vascular dementia	Multifocal cortical and subcortical, correlating with structural imaging lesions
Parkinson disease with dementia and dementia with Lewy bodies	Symmetric or asymmetric bilateral temporoparietal, posterior cingulate, and visual cortex; lesser frontal association cortex; sparing of primary sensorimotor cortex
Huntington disease	Caudate nucleus and lesser frontal association cortex
Progressive supranuclear palsy	Caudate nucleus, putamen, thalamus, pons, primarily superior and anterior frontal cortex; sparing of cerebellum
Corticobasal degeneration	Asymmetric frontal, temporal, and parietal cortex and thalamus contralateral to limb apraxia

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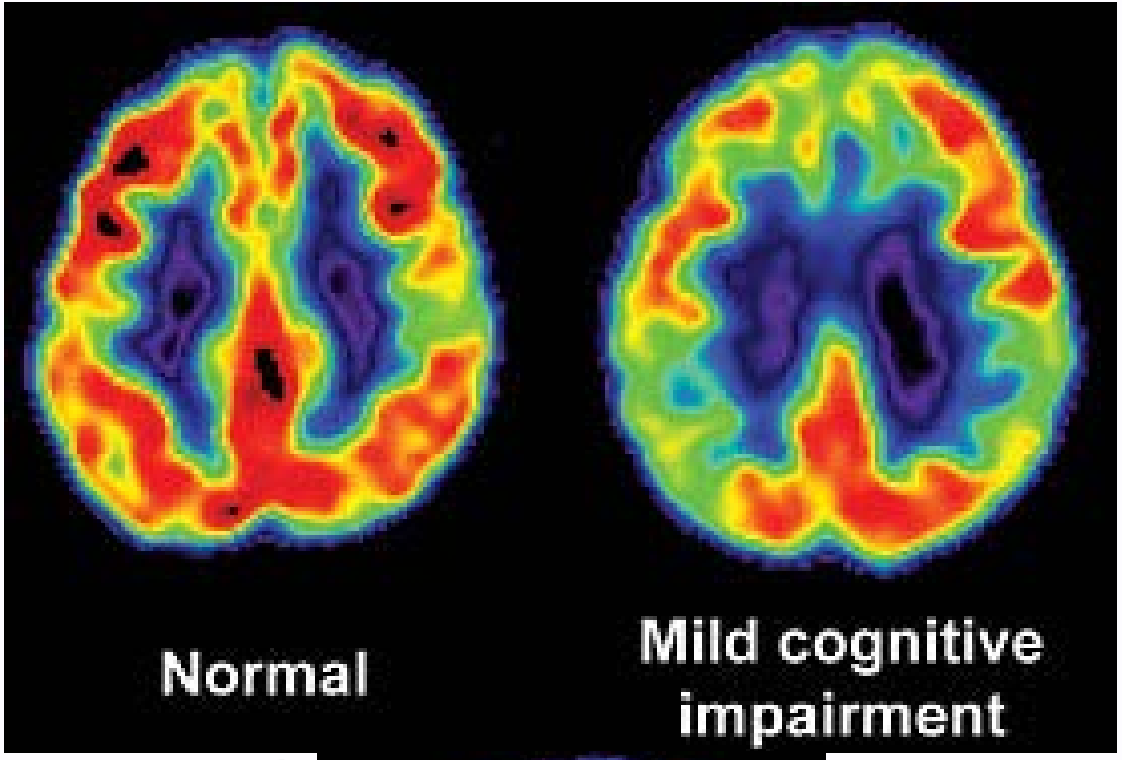

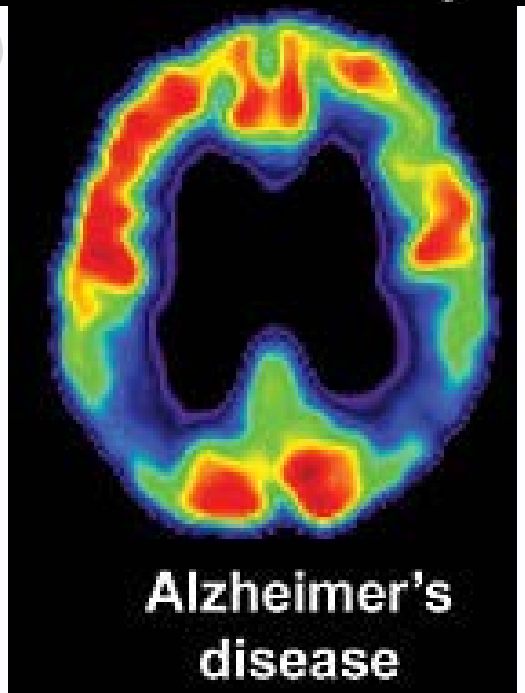

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- Assessment of the following will help in planning the management
- Daily function, including feeding, bathing, dressing, mobility, toileting, continence and the ability to manage finances and medications
- Cognitive status using a reliable and valid instrument (e.g. the MMSE)
- Other medical conditions
- Behavioral problems, psychotic symptoms, or depression

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- Reassessment should occur every 6 months or more frequently with any sudden decline or behavioral change.
- Identify the primary caregiver and assess the adequacy of family and other support systems.
- Assess the patient's decision-making capacity.

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- Caregiver's needs and risks should be assessed and reassessed on a regular basis.

- Assess the patient's and family's culture, values, primary language, literacy level and decision-making process

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Subtypes

- ***Alzheimer's disease***
- ***Frontotemporal lobar degeneration***
- ***Lewy body disease***
- ***Vascular disease***
- **Traumatic brain injury**
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- **HIV infection**
- **Prion disease**
- **Parkinson's disease**
- **Huntington's disease**

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NINCDS- ADRDA criteria for Alzheimer's type

- A.Alzheimer's disease is characterized by a progressive decline and ultimately loss of multiple cognitive functions, including both:
- Memory Impairment- Impaired ability to learn new information or to recall previously learned information
- And at least one of the following:
- Loss of word comprehension ability, aphasia
- Loss of ability to perform complex tasks involving muscle coordination, apraxia
- Loss of ability to recognize and use familiar objects.
- Loss of ability to plan, organise and execute normal activities.

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- B. The problems in A represent a substantial decline from previous abilities and cause significant problems in everyday functioning.
- C. The problems in A begin slowly and gradually become more severe.
- D. The problems in A are not due to :
- Other conditions that cause progressive cognitive decline, among them stroke, Parkinson’s disease, Huntington’s chorea, brain tumour etc.
- Other conditions that cause dementia, maong them, hypothyroidism, HIV infection, syphilis, defficiencies in niacin, Vit. B12 and folic acid

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- E. The problems in A are not caused by episodes of delirium.
- F. The problems in A are not caused by another mental illness.

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Criteria for probable Alzheimer's disease

- Dementia established by clinical examination, documented by a standard test of cognitive function e.g. MMSE, and confirmed by neuropsychological tests
- Significant deficiencies in two or more areas of cognition e. g. word comprehension and task completion ability
- Progressive deterioration in memory and other cognitive functions
- No loss of consciousness
- Onset from age 40-90, typically after 65.
- No other diseases or disorders that could account for loss of memory and cognition.

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A diagnosis of probable Alzheimer's disease is supported by

- Progressive deterioration of specific cognitive functions: Language, motor skills and perception
- Impaired ADL and altered patterns of behaviour
- A family history of similar problems
- The following lab tests:
- Normal CSF,
- Normal EEG, Evidence of cerebral atrophy on CT scan
- Other features consistent with Alzheimer's disease:
- Plateaus in the course of illness progression
- CT findings normal for the person's age
- Associated symptoms including depression, insomnia, incontinence, delusions, hallucinations, wt. loss, sex problems, significant verbal, emotional and physical outbursts

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- Other neurological abnormalities, especially in advanced disease, including increased muscle tone and a shuffling gait
- Features that decrease the likelihood of Alzheimer's disease:
- Sudden onset
- Such early symptoms as seizures, gait problems, loss of vision and coordination

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NINDS – AIREN criteria for of vascular dementia

I. The criteria for the clinical diagnosis of ***probable*** vascular dementia include *all* of the following:

- *Dementia* defined by cognitive decline from a previously higher level of functioning and manifested by impairment of memory and of two or more cognitive domains (orientation, attention, language, visuospatial functions, executive functions, motor control, and praxis), preferable established by clinical examination and documented by neuropsychological testing; deficits should be severe enough to interfere with activities of daily living not due to physical effects of stroke alone.
  Exclusion criteria: cases with disturbance of consciousness, delirium, psychosis, severe aphasia, or major sensorimotor impairment precluding neuropsychological testing. Also excluded are systemic disorders or other brain diseases (such as AD) that in and of themselves could account for deficits in memory and cognition.

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- *Cerebrovascular disease*, defined by the presence of focal signs on neurologic examination, such as hemiparesis, lower facial weakness, Babinski sign, sensory deficit, hemianopia, and dysarthria consistent with stroke (with or without history of stroke), and evidence of CVD by brain imaging (CT or MRI) including *multiple large vessel infarcts* or a *single strategically placed infarct* (angular gyrus, thalamus, basal forebrain, or PCA or ACA territories)
- *A relationship between the above two disorders*, manifested or inferred by the presence of one or more of the following: (a) onset of dementia within 3 months following a recognized stroke; (b) abrupt deterioration in cognitive functions; or fluctuating, stepwise progression of cognitive deficits.

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II. Clinical features consistent with the diagnosis of ***probable*** vascular dementia include the following:

(a) Early presence of gait disturbance (small-step gait or marche a petits pas, or magnetic, apraxic-ataxic or parkinsonian gait); (b) history of unsteadiness and frequent, unprovoked falls; (c) early urinary frequency, urgency, and other urinary symptoms not explained by urologic disease; (d) pseudobulbar palsy; and (e) personality and mood changes, abulia, depression, emotional incontinence, or other subcortical deficits including psychomotor retardation and abnormal executive function.

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- **III.** Features that make the ***diagnosis of vascular dementia*** uncertain or unlikely include (a) early onset of of memory deficit and progressive worsening of memory deficit and other cognitive functions such as language (transcortical sensory aphasia), motor skills (apraxia), and perception (agnosia), in the absence of corresponding focal lesions on brain imaging; (b) absence of focal neurological signs, other than cognitive disturbance; and (c) absence of cerebrovascular lesions on brain CT or MRI.

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IV. Clinical diagnosis of ***possible*** vascular dementia may be made in the presence of dementia (section I-1) with focal neurologic signs in patients in whom brain imaging studies to confirm definite CVD are missing; or in the absence of clear temporal relationship between dementia and stroke; or in patients with subtle onset and variable course (plateau or improvement) of cognitive deficits and evidence of relevant CVD.

V. Criteria for diagnosis of ***definite*** vascular dementia are (a) clinical criteria for *probable* vascular dementia; (b) histopathologic evidence of CVD obtained from biopsy or autopsy; (c) absence of neurofibrillary tangles and neuritic plaques exceeding those expected for age; and (d) absence of other clinical or pathological disorder capable of producing dementia.

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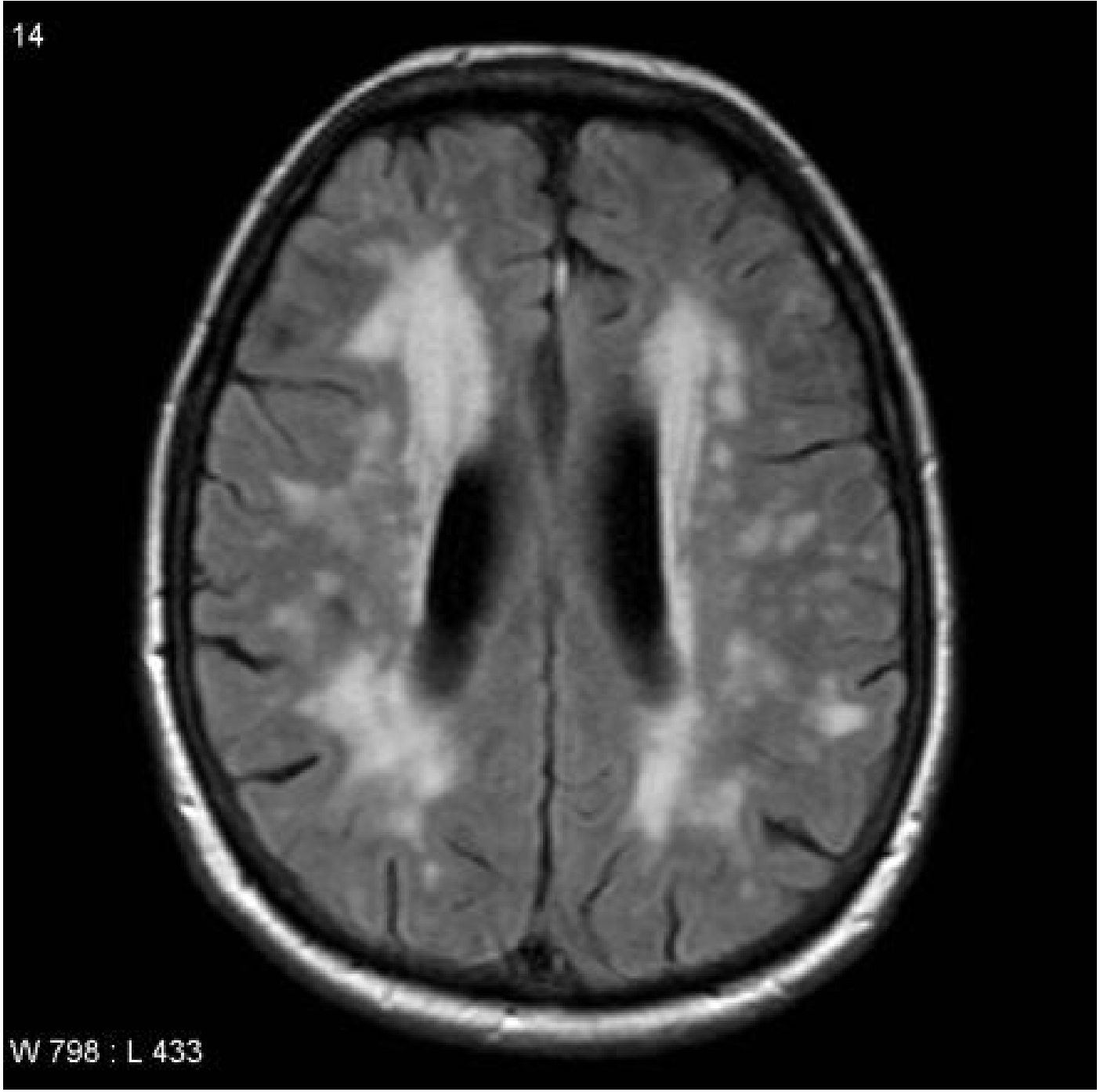

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- **Consensus Criteria for the Clinical Diagnosis of Probable and Possible Dementia With Lewy Bodies**
- **1.** The central feature required for a diagnosis of dementia with Lewy bodies is progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational function. Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression. Deficits on tests of attention and of frontal-subcortical skills and visuospatial ability may be especially prominent.

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- 2. **Two** of the following core features are essential for a diagnosis of **probable** dementia with Lewy bodies, and **one** is essential for **possible** dementia with Lewy bodies:
- Fluctuating cognition with profound variations in attention and alertness
- Recurrent visual hallucinations that are typically well formed and detailed
- Spontaneous motor features of parkinsonism

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- 3. Features supportive of the diagnosis are:
- Repeated falls
- Syncope
- Transient loss of consciousness
- Neuroleptic sensitivity
- Systematized delusions
- Hallucinations in other modalities
- 4. A diagnosis of dementia with Lewy bodies is less likely in the presence of:
- Stroke disease, evident as local neurologic signs or on brain imaging
- Evidence on physical examination and investigation of any physical illness or other brain disorder sufficient to account for the clinical picture
- (McKeith IG, Galasko D, Kosaka K, et al: 1996.)

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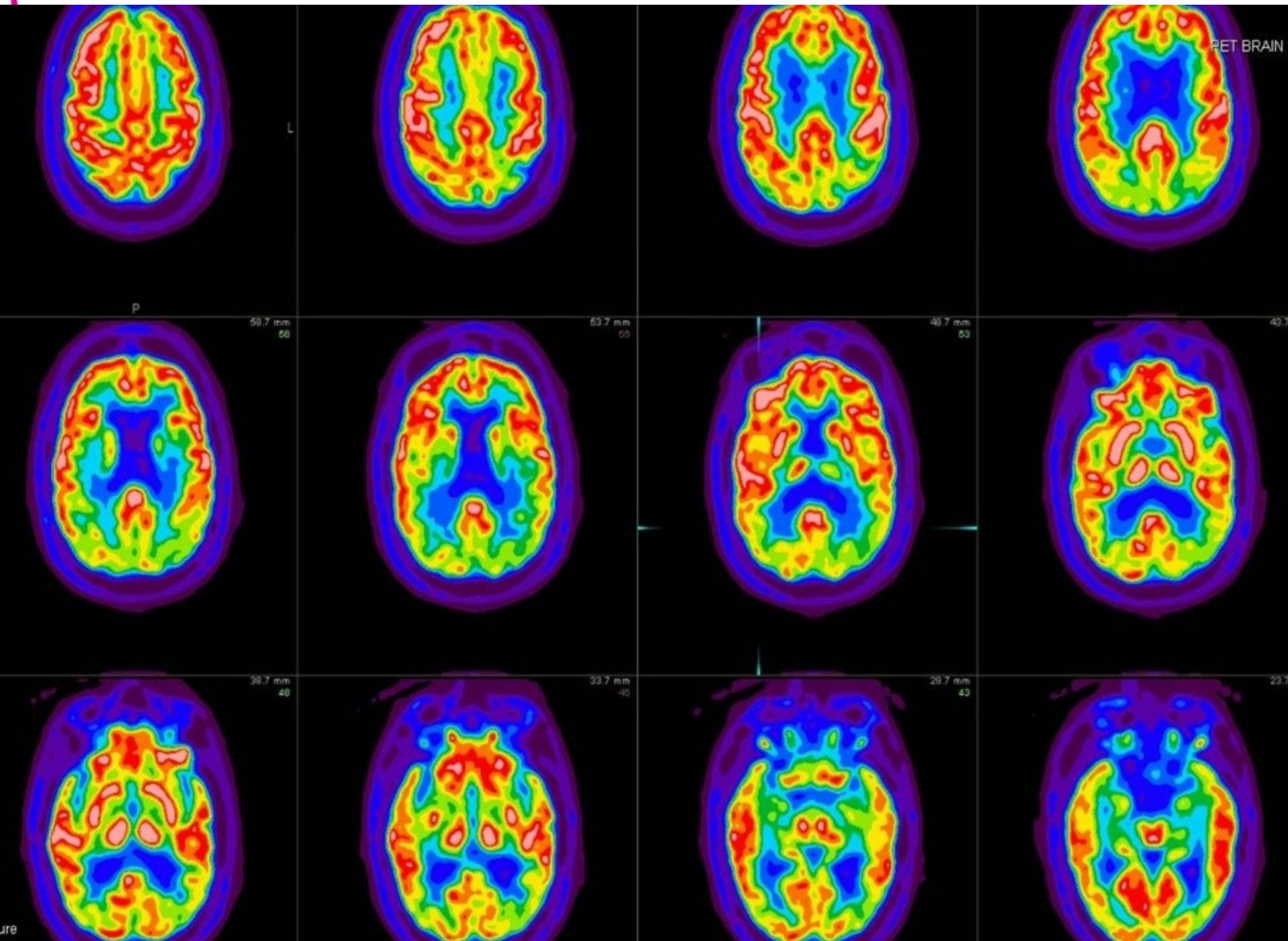
PET BRAIN
L
P
58.7 mm
58
53.7 mm
48.7 mm
53
38.7 mm
48
33.7 mm
28.7 mm
43

Clinical Diagnostic Criteria for Frontotemporal Dementia

- 1. The developement of behavioral or cognitive deficits manifested by either
- a) Early and progressive change in personality, characterized by difficulty in modulating behaviour, often resulting in inappropriate response or activities, or
- b) Early and progressive change in language, characterized by problems with expression of language or severe naming difficulty and problems with word meaning.
- 2. The deficits in 1a) and b) cause significant impairment in social and occupational functioning and represent a significant decline from a previous level of functioning.

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- 3. The course is characterized by a gradual onset and continuing decline in function.
- 4. The deficits in 1a) or b) are not due to other nervous system conditions, systemic conditions or substance induced conditions.
- 5. The deficits do not occur exclusively during delirium.
- 6. The disturbance is not better accounted for by a psychiatric diagnosis.

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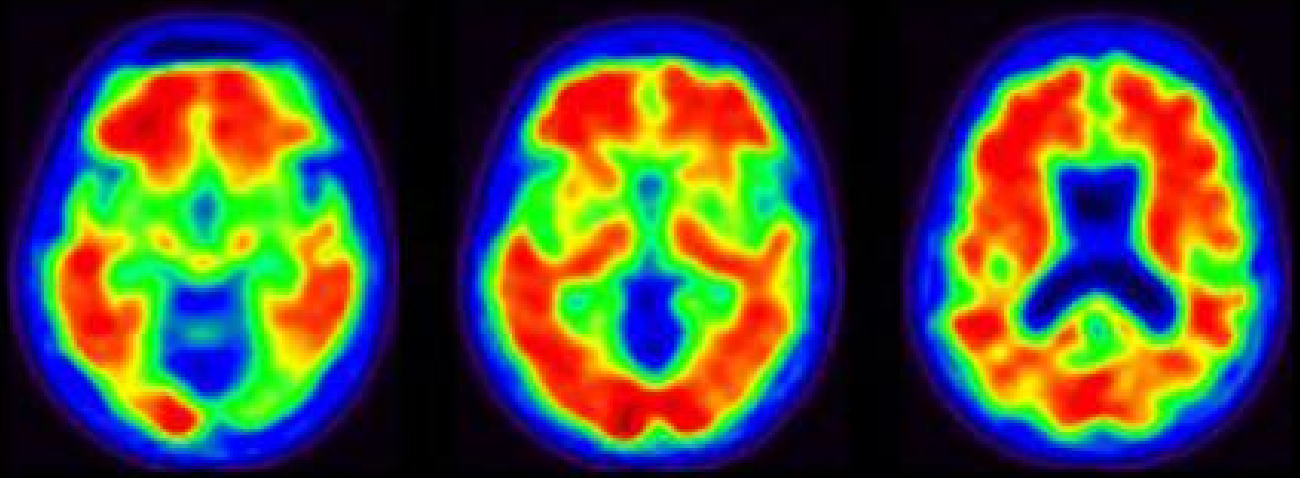
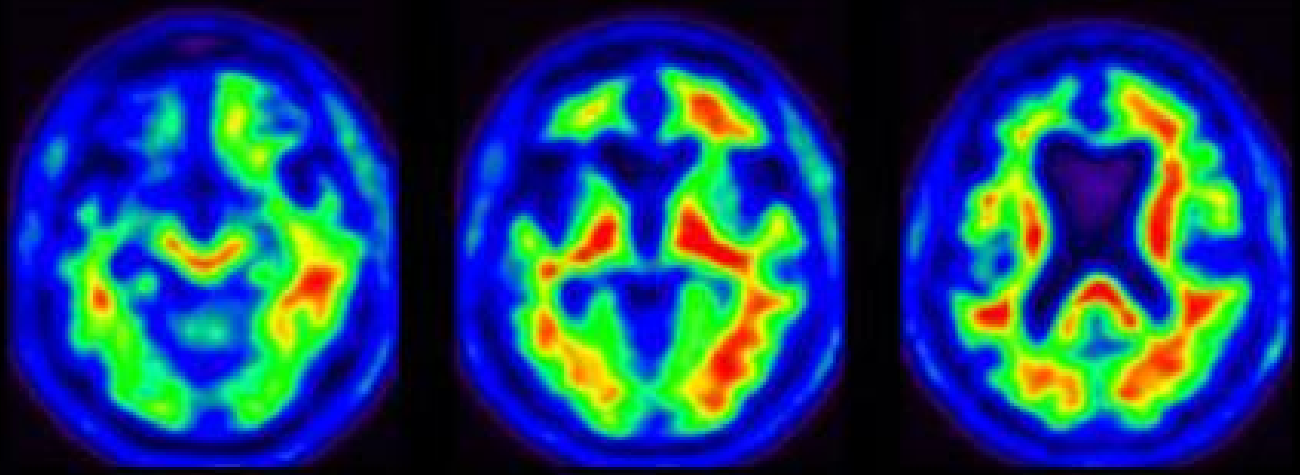
Amyloid PET Imaging in Alzheimer's Disease and Frontotemporal Dementia

Alzheimer's Disease:
Amyloid PET positive



Frontotemporal Dementia:
Amyloid PET negative



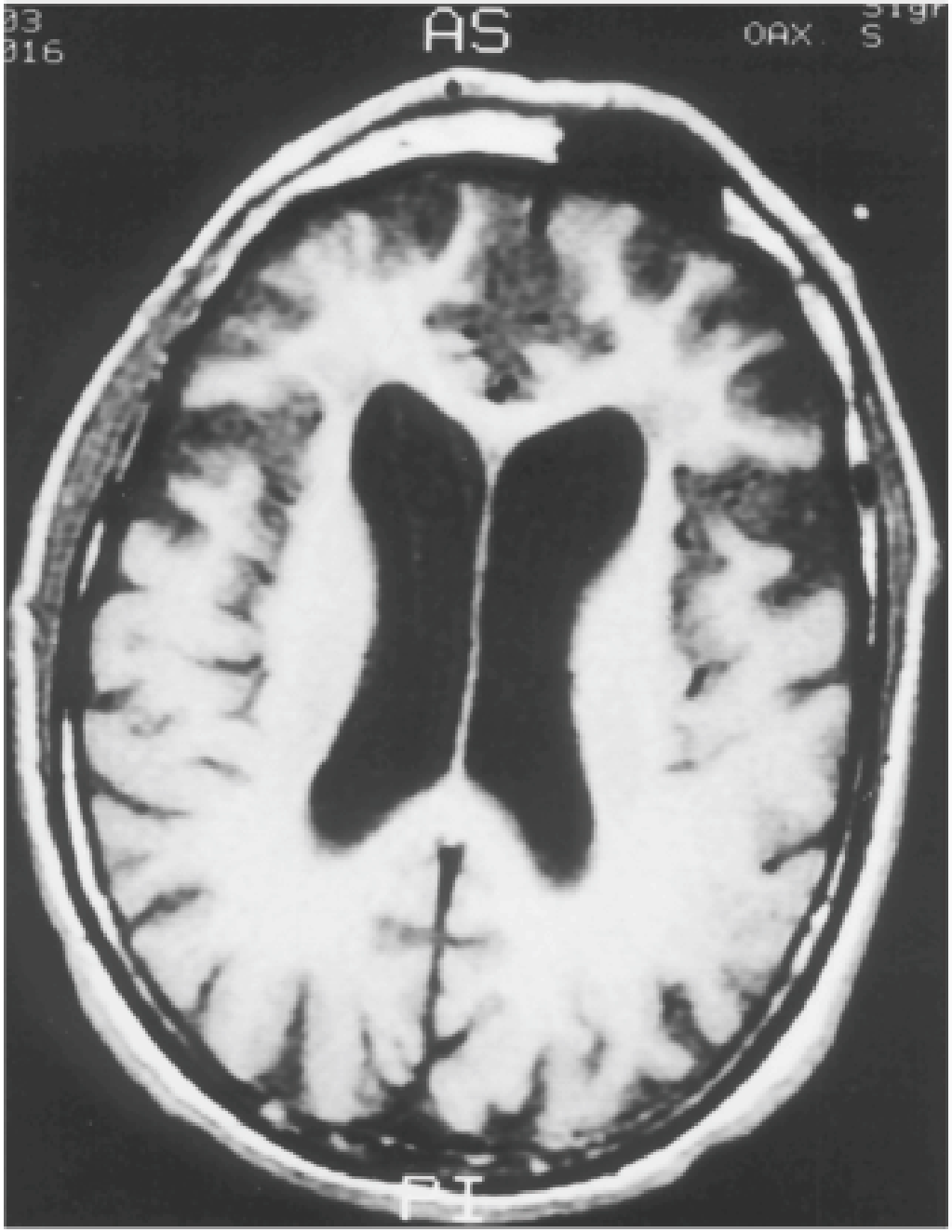


Fig 1. MRI scan (axial plane) demonstrating frontal atrophy with ventricular enlargement.

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Management of Alzheimer's Disease

General Measures

- Information & Education
- Communication & consent
- Caregiver Support
- Respite Care

Management of Symptoms of Alzheimer's Disease

- Management of Cognitive Symptoms.

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Agents Useful for Reducing the Signs of Dementia

Drug	Donepezil	Rivastigmine	Galantamine	Memantine
Dose	5mg to 10 mg daily	1.5 mg twice daily to 6 mg twice daily	4 mg twice daily to 12 mg twice daily	5 mg daily to 10 mg twice daily

- Cholinesterase inhibitors, have been shown to be effective in patients with mild to moderate dementia.

Guidelines for prescribing cholinesterase inhibitors.

A. Prescription only for patients- a) fulfilling criteria for probable AD, b) Duration of illness being more than 6 months, and c) with MMSE more than 10 (i.e., mild or moderately severe dementia).

B. Three phase evaluation of response- a) early (2 weeks) for assessing tolerance and side effects, b) later (3 months) for cognitive state, c) continued (6 months) for disease state.

C. Stop treatment- a) if early evaluation shows poor tolerance or compliance, b) if deterioration continues at pre-treatment rate after 3-6 months of treatment, c) if even after reaching maintenance dose accelerating deterioration continues, d) if a drug-free period of 3-4 weeks suggests that the drug is no longer helping.

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- Vitamin E: One study demonstrating delay in poor outcome though no effect on cognitive decline
- Further evidence of increased morbidity and mortality in a dose dependent manner.
- No longer recommended
- NSAIDs: Not recommended as except for one study no further evidence of benefit and risk of side effects is high.
- Hormone replacement therapy : Contradictory results
- No evidence for use of estrogen
- Ginko Biloba: Currently not recommended
- MAO-B inhibitor: Selegiline- Currently not recommended
- Ergoloids: Hydergine: No role in treatment
- Antiamyloid Therapies: Under investigation

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Mangement of Behavioural and Psychological Symptoms of Dementia.

- Non-Pharmacological Management of BPSD :
- Look for treatable factors like unmet needs, pain, hunger, wetness etc.
- All of these factors can initiate or sustain BPSD.
- Identification and removal of these factors
- A predictable environment is always better tolerated by patients.
- Distracting and redirecting the patient to some other activity can terminate certain undesirable behaviours.
- communication strategies: short and simple sentences, nonverbal communication
- Avoid confrontation, Use praise and encouragement.

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Pharmacological Management of BPSD

- For drug treatments the '3T' (Target, Titration, Ttime) approach is good practice:
- (1) drug treatments should have a specific **target** symptom
- (2) the starting dose should be low and then be **titrated** upwards
- (3) drug treatments should be **time** limited.

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Type and Drug	Initial Daily Dose	Final Daily Dose (Range)	Targeted Symptoms
Atypical antipsychotic			Psychosis and agitation
Risperidone	0.5 mg daily	1.0 mg (0.75-1.5 mg daily)	
Olanzapine	2.5 mg daily	5.0 mg (5-10 mg daily)	
Quetiapine	25 mg daily	200 mg (50-150 mg twice a day)	
Ziprasidone	20 mg daily	40 mg (20-80 mg twice a day)	
Aripiprazole	10 mg daily	10 mg (10-30 mg daily)	
Neuroleptic			Psychosis and agitation
Haloperidol	0.25 mg daily	2 mg (1-3 mg daily)	
Mood stabilizer			
Divalproex sodium	125 mg twice a day	500 mg (250-500 mg twice a day)	Agitation
Carbamazepine	200 mg twice a day	400 mg (200-500 mg twice a day)	
Selective serotonin-reuptake inhibitor			Depression, anxiety, psychosis, and agitation
Citalopram	10 mg daily	20 mg (20-40 mg daily)	
Escitalopram	5 mg daily	10 mg (10-20 mg daily)	
Paroxetine	10 mg daily	20 mg (10-40 mg daily)	
Sertraline	25 mg daily	75 mg (75-100 mg daily)	
Fluoxetine	5 mg daily	10 mg (10-40 mg daily)	
Tricyclic antidepressant			Depression
Nortriptyline	10 mg daily	50 mg (25-100 mg daily)	
Desipramine	10 mg daily	100 mg (50-200 mg daily)	
Serotonin and noradrenergic-reuptake inhibitor			Depression and anxiety
Venlafaxine	25 mg twice a day	200 mg (100-150 mg twice a day)	
Noradrenergic and specific serotonergic antidepressant			Depression
Mirtazapine	7.5 mg daily	15 mg (15-30 mg daily)	

- Management of Impairments in Activities of Daily Living
- **Management of Co-morbidity:**
- sensory deprivation should be minimized by treating cataract and removing earwax and/or providing hearing aids.
- Dental and oral hygiene should be maintained;
- constipations, pneumonia, urinary infections should be treated;
- and nutrition should be improved

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- Falls and fractures should be prevented by taking appropriate precautions.
- Periodic review of the patients prescription, elimination of all non-essential drugs, minimizing the number of medications on prescription, and appropriately dosing the medications.

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- . Maintaining a daily routine,
- monitoring fluid balance, nutrition and body weight,
- training and regularization of toilet habits,
- meticulous management of incontinence and
- ensuring adequate and undisturbed sleep at night,

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- Management of the extra personal environment:
- providing a clean and levelled flooring without too many obstacles on it;
- padded clothing to minimize injuries in cases of falls;
- well lit rooms
- modifying the toilets to make them more user-friendly for the patients and providing them close to the patient's bed;
- ; grab rails in toilet, stairways and, if required, in the rooms;
- regularizing meal-timings and sleeping hours;

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- **Plan for Future Care:**
- Long term care
- Caregiver issues
- Resources
- Institutionalization

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STRATEGIES TO IMPROVE FUNCTIONAL PERFORMANCE AND REDUCE PROBLEM BEHAVIORS

Strategy	Strength of evidence
• <u>To improve functional performance</u>	
– Behavior modification,modification, scheduled toileting, prompted voiding to reduce urinary incontinence.....	.....**Strong**
– Graded assistance, practice and positive reinforcement to increase functional independence.....	.....**Good**
– Low lighting levels, music and simulated nature sounds to improve eating behaviors.....	.....**Weak**
– Intensive multi-modality group training may improve activities of daily living.....	....**Weak**

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Strategy	Strength of evidence
• To reduce problem behaviors	
– Music, particularly during meals and bathing	.....Good
– Walking or other forms of light exercise	.....Good
– Simulated presence therapy, such as use of videotapes of family	.....Weak
– Massage	....Weak
– Comprehensive psychosocial care programs	.....Weak
– Pet therapy	....Weak
– Utilizing commands issued at the patient's comprehension level	.....Weak
– Bright light, white noise	.....Weak
– Cognitive remediation	.....Weak

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Management of Vascular Dementia

- Primary Prevention:
- Controlling vascular risk factors- Treatment of HT, DM, Hyperlipidemia, Atrial fibrillation, carotid stenosis
- Antiplatelet therapy in patients with TIA and stroke
- Healthy lifestyle changes
- Treatment of Sleep apnoea

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Unique Markers for Diagnosis and new treatment modalities

- Declining CSF Aβ42 levels occur at least 20-25 yrs prior to clinical dementia
- MCI of Alzheimer's: MRI -hippocampal and entorhinal atrophy, 18F-FDG-PET- Reduced metabolism in temporoparietal cortex, posterior cingulate cortex
- AD: MRI- Pronounced atrophy of hippocampus and volumetric studies show atrohy in middle temporal lobe
- Genetic: Mutations in amyloid precursor protein(APP) or presenilin 1 or 2 genes, trisomy 21- Measurable changes 20 yrs before the onset

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- Also, numerous single nucleotide polymorphisms:
- Individuals with one copy of APOE4- 2-3 fold increase in lifetime risk of AD
- Homozygous individuals 10 fold increase
- APOE2- lower risk of AD
- Biofluid markers: e.g. Blood samples stained with Oil Red O, distinguishes between healthy and demented individuals

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Treatment advances

- Amyloid based treatments
- Secretase targeted treatments
- Metabolic approaches- insulin nasal sprays
- Aβ aggregation inhibitors
- APOE dependent Aβ clearance- Bexarotene, human studies awaited
- Enzymatic clearance
- Aβ immunotherapy

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- Secondary Prevention:
- Rigorous rehabilitation after stroke may help in improving cognition.
- Donepezil, Galantamine, Rivastgmine
- Memantine especially useful in severe dementia

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Treatment of dementia with Lewy Bodies

- Nonpharmacological:
- Strategies to increase levels of arousal and attention may be useful.
- Cognitive deficits: Cholinesterase inhibitors
- Memantine: Not recommended as may cause worsening of symptoms
- Neuropsychiatric symptoms: Donepezil and other cholinesterase inhibitors, second generation antipsychotics
- Treatment of Parkinsonian features: L-Dopa, direct dopamine agonists

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Treatment of Frontotemporal Dementia

- Currently No treatment for cognitive deficits and no treatment for progression
- Insufficient data for cholinesterase inhibitors or memantine
- Insufficient data on treatment of behavioral symptoms
- Symptomatic use of antidepressants, antipsychotics and anticonvulsants
- Nonpharmacological management of behavioral symptoms

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