433 Teams

MEDICINE

22| Dementia and delirium

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Slides - Step-Up medicine - Kaplan Notes - Extre explanation - Doctor Notes

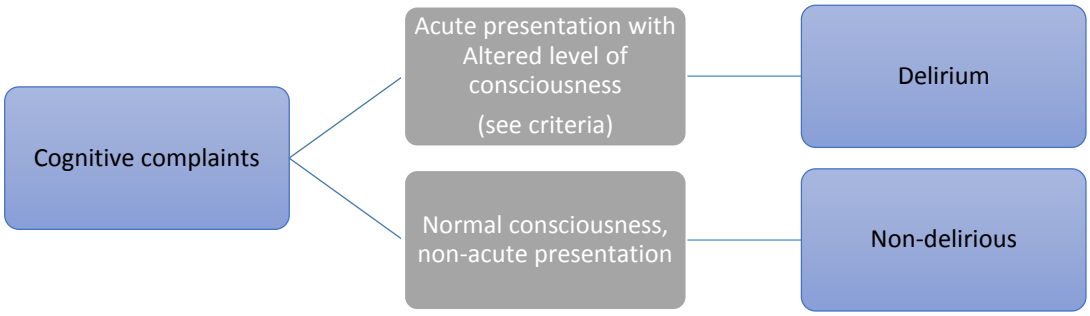
Objective: not given

1) Acute Confusional State (Delirium)

Delirium is an acute period of cognitive dysfunction due to a medical disturbance or condition.(When see a patient delirious and agitated this may give a hint its drug related)

- Elderly patients are especially prone to delirium
- **Delirium, usually encompasses: "Acute confusional state" and "encephalopathy**

Dementia or Delirium?



- **It is not normal to have delirium, while this statement is obvious, patients' who have symptoms of delirium are dismissed as being sleepy, tired, or just age related changes.**
- **BEING OLD ≠ Being confused or mentally impaired**

Causes

Causes of delirium include those of coma plus the following: "P. DIMM WIT." "SMAS HED"

1. P-postoperative state (compounded by pain medications)
2. D -dehydration and malnutrition
3. I -infection (sepsis, meningitis, encephalitis, urinary tract infection, and so on)
4. M -medications and drug intoxications—tricyclic antidepressants, corticosteroids,
   Anticholinergics, hallucinogens, cocaine
5. M -metals (heavy metal exposure)
6. W -withdrawal states (from alcohol, benzodiazepines)
7. I -inflammation, fever
8. T -trauma, burns

9.S -structural brain pathology: stroke, subdural or epidural hematoma, tumor, hydrocephalus, herniation, abscess

10.M -meningitis, mental illness (e.g., conversion disorder, catatonia—mimic coma)

11.A -alcohol, acidosis

12. S -seizures (postictal state), substrate deficiency (e.g., thiamine)

13. H -hypercapnia, hyperglycemia, hyperthermia; hyponatremia, hypoglycemia, hypoxia, hypotension/cerebral hypoperfusion, hypothermia

14.**E** -**e**ndocrine causes (Addisonian crisis, thyrotoxicosis, hypothyroidism); encephalitis, encephalopathy(hypertensive, hepatic, or uremic); extreme disturbances in calcium, magnesium, phosphate

15.**D** -**d**rugs (opiates, barbiturates, benzodiazepines, other sedatives); dangerous compounds (carbonmonoxide, cyanide, methanol)

Clinical features

1. In contrast to both dementia and psychosis, delirium is characterized by
√ Rapid deterioration in mental status (over hours to days)
√ Fluctuating level of awareness,
√ Disorientation,
√ Frequently abnormal vital signs.
2. Delirium may often be accompanied by acute abnormalities of perception, such as Hallucinations

3. Patients may not necessarily be agitated, but may have a slow, blunted responsiveness.

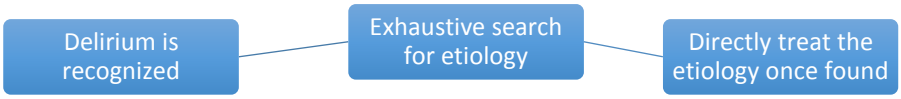
Diagnosis

Mental status examination—Mini-Mental Status Examination	Laboratory	Important clues to recognize delirium:
•**Diagnostic criteria for delirium is as follows:** •Disturbance in attention (reduced ability to direct, focus, sustain, and shift attention) and awareness. •Change in cognition (memory deficit, disorientation, language disturbance, perceptual disturbance) that is not better accounted for by a preexisting, established, or evolving dementia. •The disturbance develops over a short period (usually hours to days) and tends to fluctuate during the course of the day.	•chemistry panel •vitamin B12 •Thiamine •LP—Perform in any febrile, delirious patient unless there are contraindications (e.g., cerebral edema).	•Patient will not be able to give you a history •Rapid development of symptoms (hours or days). •Change in level of consciousness •When the patient appears awake, assess level of attention •Poor content of conversation and/or other cognitive deficits (memory loss, disorientation, abnormal language), neuropsychiatric symptoms such as hallucinations (visual, auditory somatosensory) and delusions of harm. •The opposite, hypervigilance, may occur in substance withdrawal (alcohol or sedative)

- There is evidence from the history, physical examination, or laboratory findings that the disturbance is caused by a direct physiologic consequence of a general medical condition, an intoxicating substance, medication use, or more than one cause

What can look like delirium?
- Non-convulsive seizures
- Sundowning behavior
- Dementia
- Psychiatric disorders
- Aphasias
- Transient Global Amnesia

Management



1. **Treat the underlying cause.**
2. **Haloperidol—for agitation/psychotic-like delirious behavior**
3. **Supportive treatment**

- The choice of the investigations should be guided by your history and clinical examination findings
- There many causes of delirium, so an initial investigation may include (but not limited to) the following:

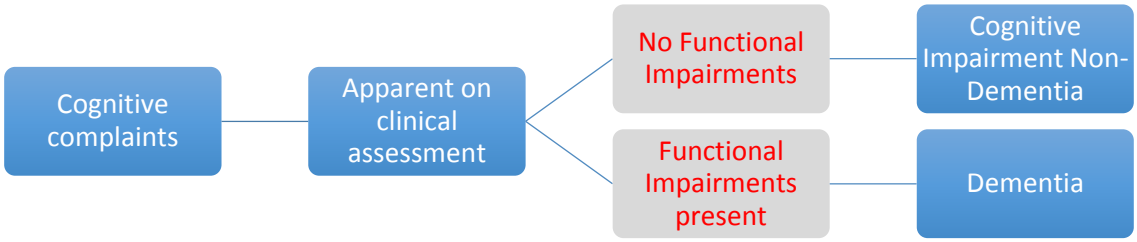
√ CBC, electrolytes, urea, creatinine, LFT, ESR, TSH +/- Auto-immune evaluation.
√ Arterial blood gases.
√ Urinalysis and toxicology screen.
√ Chest X-ray, EKG.
√ CT head, EEG, Lumbar Puncture.

2) Dementia

- Dementia is a progressive deterioration of intellectual function, typically characterized by preservation of consciousness.
- The most important risk factor for dementia is **increasing age**
  - **Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains*:**

- - Learning and memory.
- - Language.
- - Executive function (for ex. If you have a flight to Jeddah < can you book the flight from the website and pay with the credit card).
- - Complex attention (multi tasks ex. when you work on a project and then your brother come to you and ask you for something and you go back to your work).
- - Perceptual-motor (when used to do something and now he lost the skills to do it . not to be confused with apraxia is neurological condition characterized by loss of the ability to perform activities that a person is physically able and willing to do.)
- - Social cognition (pseudo dementia :when someone has a depression he might perform bad in cognitive scales and when you treat him by anti-depressant the impairment may go away . BUT Depression can coexists with dementia syndrome also like Alzheimer disease).
- The cognitive deficits interfere with independence in everyday activities.
- The cognitive deficits do not occur exclusively in the context of a delirium.
- The cognitive deficits are not better explained by another mental disorder (e.g. major depressive disorder, schizophrenia).

Dementia or Cognitive Impairment



■ Causes of dementia (reversible vs. irreversible) :

Potentially Reversible Causes of Dementia	Irreversible Causes of Dementia
Hypothyroidism	Alzheimer's disease
Neurosyphilis	Parkinson's, Huntington's
Vitamin B12/folate deficiency/thiamine deficiency	Multi-infarct dementia
Medications	Dementia with Lewy bodies, Pick's disease
Normal-pressure hydrocephalus	Unresectable brain mass
Depression	HIV dementia
Subdural hematoma	Korsakoff's syndrome
	Progressive multifocal leukoencephalopathy
	Creutzfeldt–Jakob disease

Major Dementias

- **Neurodegenerative**:
  - Alzheimer's Disease
  - Lewy Body Dementia
  - Parkinson's Disease Dementia
  - Frontotemporal Dementia
  - Huntington's Disease
- **Other**:
  - Vascular Dementia.
  - Normal Pressure Hydrocephalus.
  - Creutzfeldt-Jakob Disease.
  - Wernicke-Korsakoff Syndrome.
  - Secondary to infection or systemic illness.
  - Autoimmune disorders (eg: SLE).
  - Alcohol leading to wernicke-Korsakoff'ssyndrom, characterized by confabulations to compensate for amnesia.

Causes of dementia

1. Primary neurologic disorders

a. Alzheimer's disease	—accounts for 66% of all cases of dementia
b. Vascular dementia	•Multi-infarct dementia is a stepwise decline due to a series of cerebral infarctions • Binswanger's disease—insidious onset, due to diffuse subcortical white matter degeneration, most commonly seen in patients with long-standing HTN and atherosclerosis
c. Space-occupying lesions	, such as brain tumor or chronic subdural hematoma
d. Normal-pressure hydrocephalus	—triad of dementia, gait disturbance, and urinary incontinence; normal CSF pressure and dilated ventricles
e. Pick's disease	—clinically identical to Alzheimer's disease
f. Other neurologic conditions	MS, Parkinson's disease, Huntington's disease, Wilson's disease
g. Dementia with Lewy bodies	

2. Infections

a. HIV infection (AIDS-related dementia)

b. Neurosyphilis

c. Cryptococcal infection of CNS

e. Progressive multifocal leukoencephalopathy

d. Creutzfeldt–Jakob disease (spongiform encephalopathy)

3. Metabolic disorders

a. Thyroid disease (hypothyroidism or hyperthyroidism)

b. Vitamin B12 deficiency

c. Thiamine deficiency—common in alcoholics; if untreated can lead to Korsakoff's dementia (irreversible)

d. Niacin deficiency

4. Drugs andtoxins

a. Drug abuse; chronic alcoholism (may cause dementia independent from thiamine malnutrition

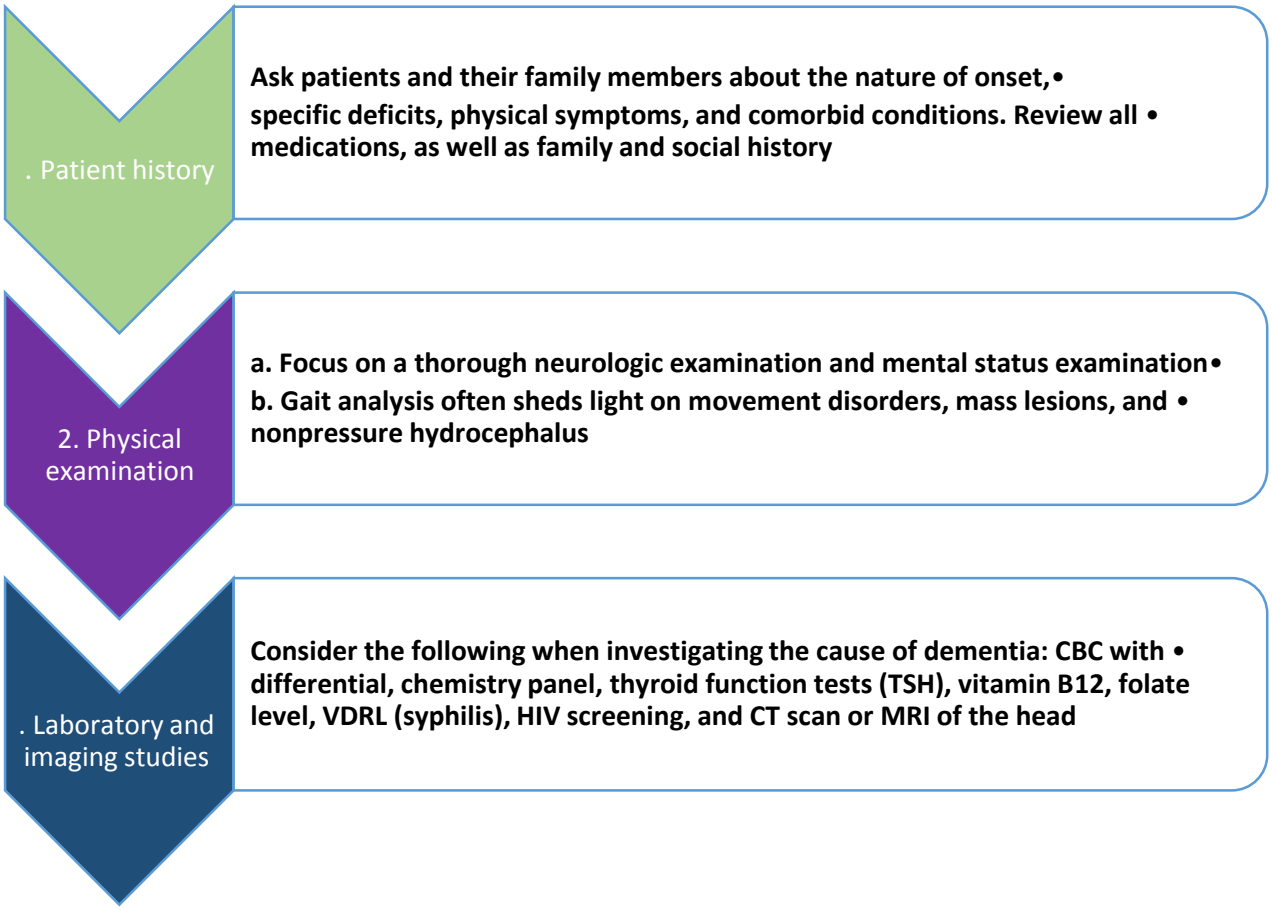
b. Toxic substances: aniline dyes, metals (e.g., lead)

5. Pseudodementia

Severe depression may cause a decline in cognition that is difficult to distinguish clinically from Alzheimer's disease but is responsive to antidepressant therapy.

For MCI "mild cognitive impairment" patients, the annual rate to develop AD "Alzaimer's disease" is 10-15% per year. In the normal population it is 1-2%.

Clinical approach to dementia



Treatment and management: (general principles)

1. Treat reversible causes.
2. Avoid and/or monitor doses of medications with adverse cognitive side effects (glucocorticoids, opiates, sedative hypnotics, anxiolytics, anticholinergics, lithium).
3. Treat/control comorbid medical conditions; e.g., diabetes, HTN, depression, visual and hearing impairment.
4. Pharmacologic therapy may include vitamin E, tacrine, and donepezil. The evidence supporting the efficacy of many pharmacologic treatments is inconclusive.
5. A multidisciplinary approach includes support groups for caregivers/families of patients with irreversible dementias.

➢ Dementia with Lewy Bodies:

- Second most common cause of "degenerative" dementia
- Dementia with Lewy bodies has features of both Alzheimer's disease and Parkinson's disease, but progression may be more rapid than in Alzheimer's disease. (Parkinson's Disease Dementia is similar to LBD. The difference is that a clear history of PD with NO cognitive impairment precedes the development of dementia by at least a year).
- Initially, visual hallucinations predominate. Other symptoms include extrapyramidal features and fluctuating mental status.
- Other symptoms include visual spatial impairment > short term memory, sensitivity to neuroleptics, REM sleep behavior disorder and autonomic dysfunction
- Diagnosis is primarily clinical
- Myocardial scintigraphy may be abnormal due to abnormal cardiac sympathetic innervation
- PET scan may show decreased occipital lobe metabolism
- Pathologically there are "Lewy Bodies" present in neurons, which are the result of abnormal synuclein protein accumulation.
- These patients are sensitive to the adverse effects of neuroleptic agents, which often exacerbate symptoms.
- Treatment is similar to that for Alzheimer's disease, with neuroleptic agents (for hallucinations and psychotic features). Selegiline may slow the progression of disease

➢ Vascular Dementia :

- Occurs secondary to:
  - A single stroke in a region important to cognition such as hippocampus or thalamus, or a large stroke that affects multiple lobes.
  - Recurrent strokes that accumulate over time, there is a step-wise development of cognitive deficits.
  - Slowly progressing cognitive deficits due to subclinical progressing of small vessel disease.
- Associated with vascular risk factors (HTN, DM, Hyperlipidemia, & smoking)
- Frequently coexists with Alzheimer's disease.

Frontotemporal Dementia

- Mean age of onset is 58
- Preferentially involves the frontal and temporal lobes, symptoms depend on the region (lobe) involved, therefore there are variants.
- Behavioral Variant (associated with personality changes, inappropriate social behaviors (disinhibited), lack of insight, Binging on certain foods, emotional blunting, rigid and cannot adopt to new situations, along with decreased attention modulation. MRI shows atrophy in the frontal lobes (may be asymmetric) Real stories EX. (Patient talk to women about sexual topics hygiene is bad , Apathy : patient watching tv while his wife in pain suffering from cholecystitis)
- Primary Progressive Aphasia(*broca's area* (slowly progressive unlike strokes)
- Semantic Dementia(*wernicke's area *(progressive loss of understanding words)
- Common pathological inclusions include hyperphosphrylated tau protein, TDP-43 protein, or FUS protein
  - FUS: Fused in Sarcoma
  - TDP: 43 kDtransactive response (TAR) DNA binding protein
- Progressive non-fluent aphasia: Patients present first with a non-fluent type of aphasia (similar to a Broca's lesion).
- MRI may show focal left frontal atrophy.
- Semantic dementia (temporal variant of FTD "frontotemporal dementia"): Usually have intact fluency, but comprehension is impaired and decreased naming ability.
- MRI may show focal left temporal atrophy.

➢ Normal Pressure Hydrocephalus:

- √ A rare disorder
- √ It classically presents with gait impairment, urinary incontinence along with the dementia. However these features are not unique to NPH.
- √ Dementia is of a subcortical type, where there is executive dysfunction, and psychomotor slowing first. Other features of cognitive impairment develop later on.
- √ The typical gait has been described as "magnetic", the patient may shuffle their feet on the ground with a normal or wide base, some may have some features of parkinsonism
- √ It usually results from impaired CSF absorption at the level of the arachnoid villi.
- √ In Secondary NPH, there is usually a history of a previous meningitis, inflammatory disorder, or subarachnoid hemorrhage. Idiopathic NPH is when there is no preceding explanation for the condition.
- √ Patients who present with gait impairment > cognitive impairments have better prognosis if identified early.
- √ Some patients will improve after a lumbar puncture that removes 30-50 cc of CSF. If this test is positive, than a CSF shunting procedure is performed.
- √ The MRI brain may also show dilated ventricles (however CSF pressure is normal).

➢ Creutzfeldt-Jakob Disease:

- Rare, 1 in a million.
- It is a prion disorder and can be transmitted (transmissible spongiform encephalopathy).
- Prions are abnormally formed proteins that induce pathological transformations in other proteins.
- It has been transmitted after the use of surgical equipment or growth hormones.
- CJD presents as a rapidly progressing dementia, disease duration usually 6 months. Myoclonic jerks may occur.
- Any picture of cognitive impairment may occur, as may other neurological symptoms like parkinsonism, ataxia, field defects, spasticity, hyper-reflexia, and + Babinski.
- MRI may show abnormal signal intensity in the basal ganglia and cortical ribbon.
- EEG shows characteristic periodic sharp wave complexes.
- No treatment, patients may die within a year.
- The bovine variant CJD has been linked to consumption of beef (UK outbreak in the 90s).

TABLE 5-3 Delirium Versus Dementia

Feature	Delirium	Dementia
Causes	• Infections (UTI, systemic infection) • Medications (narcotics, benzodiazepines) • Postoperative delirium (in elderly patients) • Alcoholism • Electrolyte imbalances • Medical conditions (stroke, heart disease, seizures, hepatic and renal disorders)	• Alzheimer's disease • Multi-infarct dementia • Pick's disease
Level of consciousness	Altered, fluctuating	Preserved
Hallucinations	Frequently present (visual)	Rarely present
Presence of tremor	Sometimes present (e.g., asterixis)	Usually absent unless dementia is due to Parkinson's disease
Course	• Rapid onset, **waxing and waning** • **"Sundowning"** (worsening at night) may be present	Insidious, progressive
Reversibility	Almost always reversible	Typically irreversible

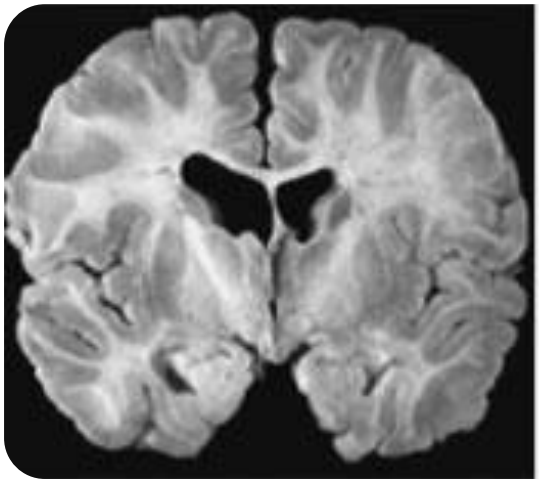
3) Alzheimer's Disease

■ Epidemiology :

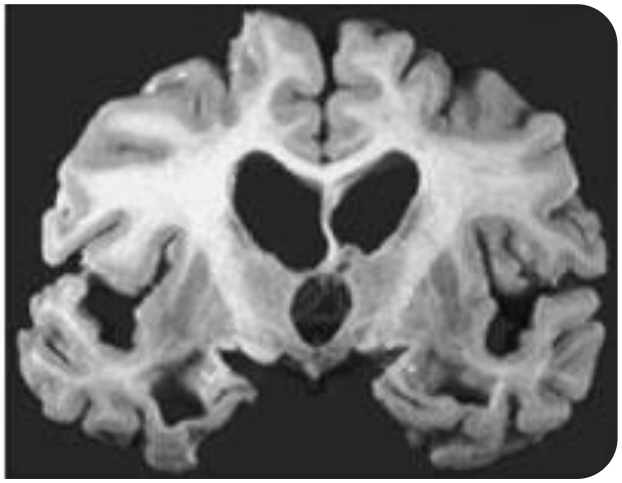
- Prevalence increases with age—Approximately 10% to 15% of individuals over age 65, and 15% to 30% of individuals over age 80 have Alzheimer's disease. However, many will die of other causes first.
- Uncommon under the age of 60.

■ Risk factors :

- Age.
- Family history (especially for early-onset Alzheimer's disease)
- Down's syndrome.
- (APOE ε4) The E4 allele for Apolipoprotein E on chromosome 19.
- Specific inherited types.
- Mid-life vascular risk factors (DM, HTN, Hyperlipidemia, Lack of exercise).
- Brain trauma.

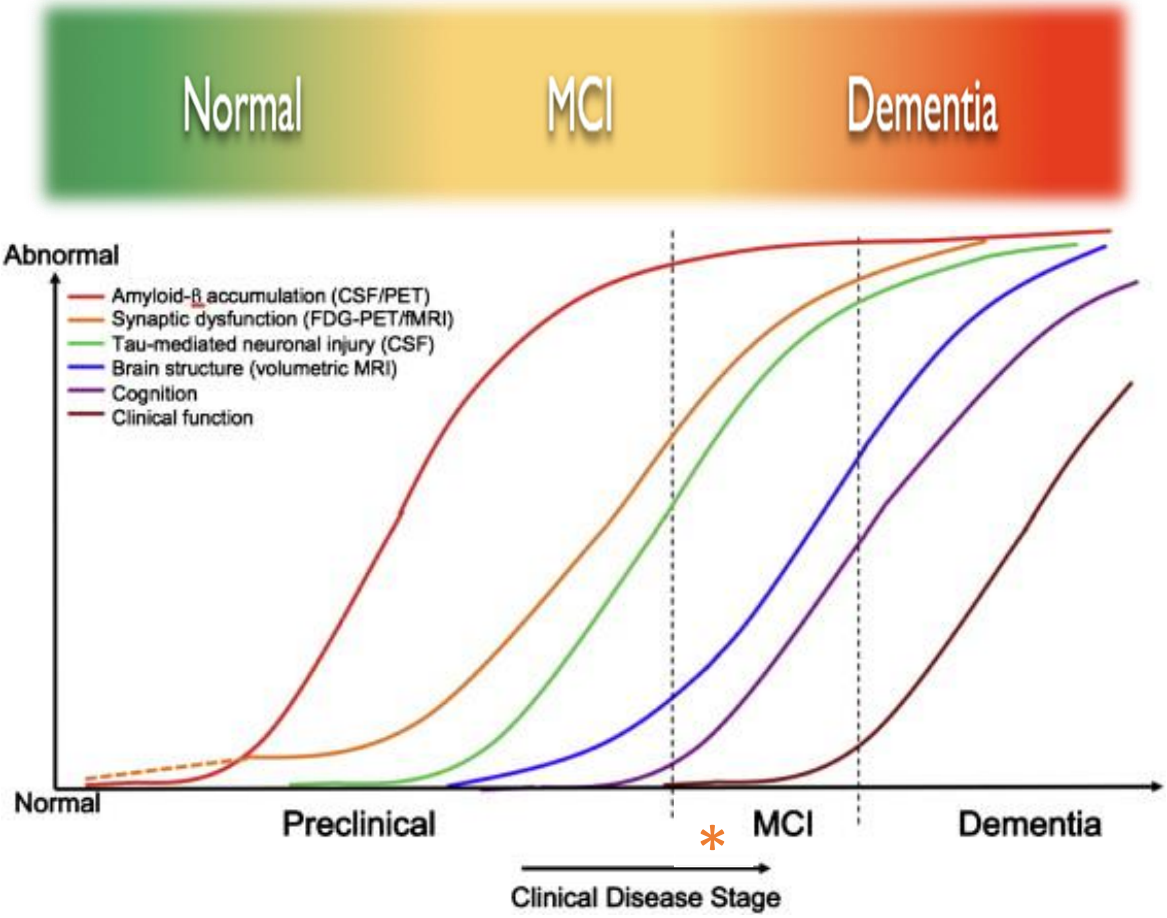


Normal brain



Brain atrophy in Alzheimer disease.

Mild Cognitive Impairment



* mild cognitive impairment

■ Pathophysiology

- Unknown, but a heritable component may be present.
- Chromosomes 21, 14, and 19 have been linked to Alzheimer's disease.
- Defects in the mechanisms for clearing amyloid beta results in its accumulation and form senile plaques
- Abnormal accumulation of hyperphosphorylated tau protein results in accumulation and the formation of neurofibrillary tangles.
- Tangles and plaques are pathological hallmarks in Alzheimer's disease.
- The resultant loss of neurons and synapses is responsible for the clinical profile ,the neuronal loss in the basal forebrain region is responsible for a cholinergic deficit.

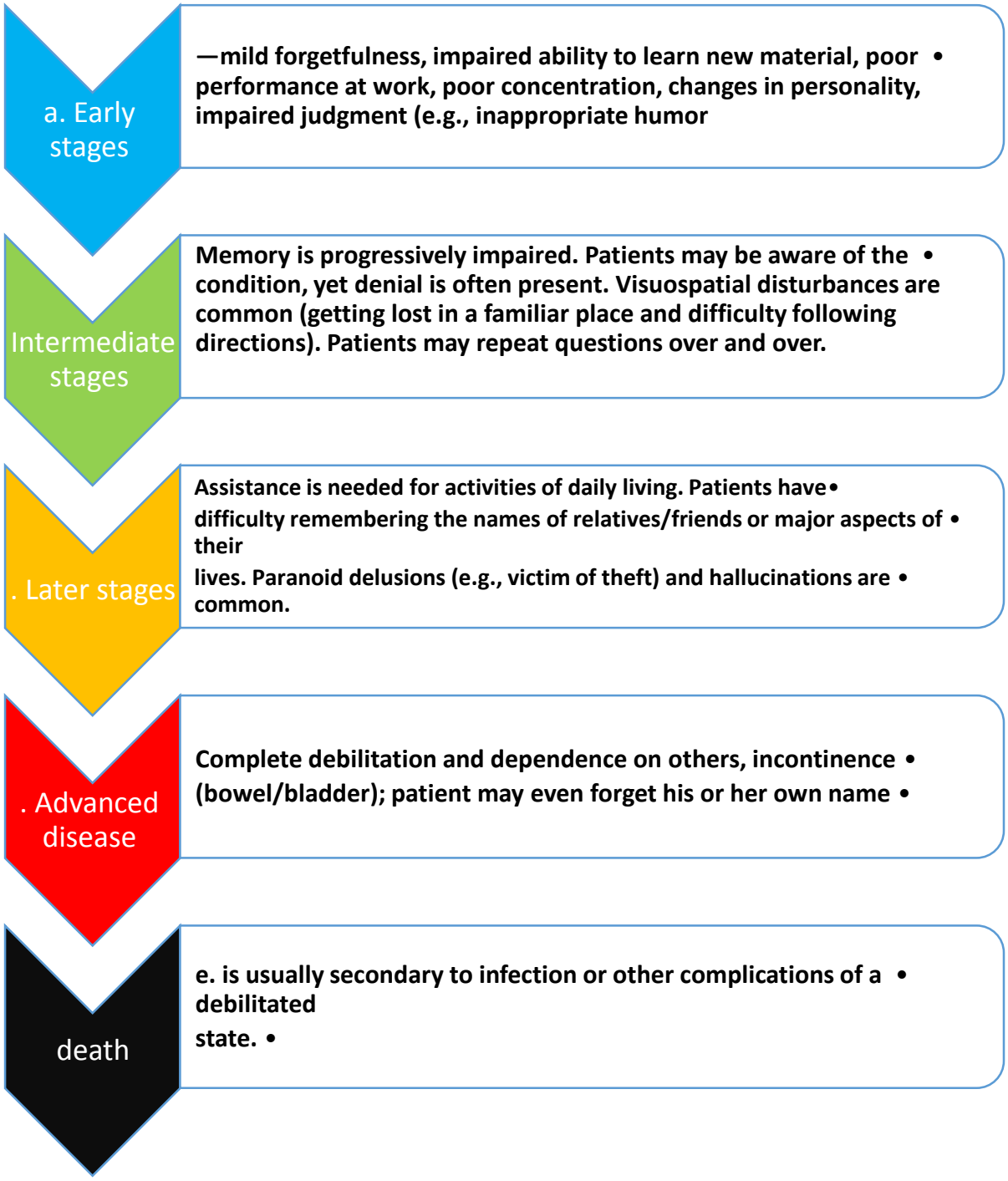
■ Pathology (noted at autopsy):

- Quantity of senile plaques (age-specific)—focal collections of dilated, tortuous neuritic processes surrounding a central amyloid core (amyloid beta-protein).
- Quantity of neurofibrillary tangles (age-specific).
- Bundles of neurofilaments in cytoplasm of neurons.
- Denote neuronal degeneration.

■ Clinical features :

- Begins insidiously but tends to progress at a steady rate
- The average time from onset to death is 5 to 10 years (with some variability).
- Decreased memory and new learning is the hallmark of the condition.
- Language impairment: Word finding difficulties with circumlocution and anomia.
- Executive dysfunction.
- Apraxia, Unawareness of illness.
- Visual-spatial impairments.
- Passivity, apathy > agitation.
- Delusions.
- Depression.
- Circadian rhythm disturbances (sundowning).
- Weight loss.

Stages:


a. Early stages
—mild forgetfulness, impaired ability to learn new material, poor performance at work, poor concentration, changes in personality, impaired judgment (e.g., inappropriate humor
Intermediate stages
Memory is progressively impaired. Patients may be aware of the condition, yet denial is often present. Visuospatial disturbances are common (getting lost in a familiar place and difficulty following directions). Patients may repeat questions over and over.
. Later stages
Assistance is needed for activities of daily living. Patients have difficulty remembering the names of relatives/friends or major aspects of their lives. Paranoid delusions (e.g., victim of theft) and hallucinations are common.
. Advanced disease
Complete debilitation and dependence on others, incontinence (bowel/bladder); patient may even forget his or her own name
death
e. is usually secondary to infection or other complications of a debilitated state.

Diagnosis:

- Alzheimer's disease is essentially a clinical diagnosis; exclude other causes first.
- Rely on history and cognitive/neuropsychological assessments that demonstrates a slowly progressing cognitive disorder which causes impairments in daily life.
- CT scan or MRI showing diffuse cortical atrophy with enlargement of the ventricles strengthens the diagnosis.
- Brain structure on MRI may demonstrate medial temporal atrophy bilaterally.
- PET scans can demonstrate decreased metabolism in temporal and parietal regions.
- Cerebrospinal fluid might show low Amyloid beta, and elevated Tau (not specific).

Treatment:

1. Cholinesterase Inhibitors—Brains of patients with Alzheimer's disease have lower levels of acetylcholine. Avoid anticholinergic medications! Options include donepezil, rivastigmine, and galantamine.
    - Drugs such as Donepezil, rivastigmine and galantamine which increase the presence of central nervous system acetylcholine help with cognitive and behavioral symptoms in Alzheimer's dementia.
    - Does not stop disease progression, but may provide transient clinical stability.
    - NMDA receptor antagonist, memantine, is helpful in advanced alzheimer's disease.
    - No treatment available for MCI .
    - Currently the first-line agent.
2. Certain dietary supplements (ginkgo, lecithin) have not been proven to be beneficial.
3. Vitamin E.
    - In one study, megadoses of vitamin E (2,000 IU/day) slowed disease progression and preserved function in people with moderately severe Alzheimer's disease.
    - Full benefit remains to be defined.

Notes:

- **Non-convulsive seizures** (they have a seizure but without convulsion . So their eyes staring and sometimes going to one side or twitching)
- **Sundowning behavior** (flips sleep awake cycle usually associate with people with dementia they tend to sleep through the day very deeply and awake through the night and they agitated/ and have behavioral symptoms)
- **Transient Global Amnesia**(it's a benign condition in which their is a loss of memory that last for 24h and then they will recover without change in the level of consciousness)

Extra:

Cortical Dementia Defined

- The cortex of the brain (the word *cortical* refers to the cortex) is the part most people are familiar with - in appearance at least. The characteristic twists and turns of the outer layers play an important role in processing information and functions such as language and memory.
- Cortical dementia is typically associated with the brain's gray matter. When these outer layers are affected, which is the case with Alzheimer's, frontotemporal dementia, Binswanger's disease and Creutzfeldt-Jakob disease, there are problems with memory, the inability to find the right words and in understanding what others are saying (aphasia).

Subcortical Dementia Defined

- As the term suggests, these are dementias believed to initially affect structures below the cortex (*sub* means below) and are more associated with the brain's white matter. Huntington's disease, Parkinson's dementia and AIDS dementia complex are three examples of conditions classified as subcortical dementia.
- It is more common to see changes in personality and a slowing down of thought processes in subcortical dementias. Language and memory functions often appear largely unaffected in the earlier stages of these dementias.

Case

73 Year old male retired judge,Presents with 1 year history of cognitive concerns, Trouble recalling names**(memory)**,He can completely forget a discussion**(memory)**,Forgets the location of previously placed tools, Only recalls fragments of a previous doctor visit 2 weeks earlier.Does not follow the dates as accurately as he used to and indicates that this is because he is retired. Sometimes he is repetitive with questions, Confusion about how to do things especially when tired, His ability to use household appliances is also affected **(Apraxia)** ,Tried putting on his shirt while still on the hanger**(Executive function)** , what is the most likely diagnosis?

Answer=>Alzheimer disease

MCQs

1. **An 80-year-old develops steady, progressive memory and cognitive deficit over 2 years. He has normal blood pressure and no focal neurologic findings, and workup for "treatable" causes of dementia is negative, what is the most likely diagnosis?**

A. Senile dementia of the Alzheimer type
B. Vascular (multi-infarct) dementia
C. Vitamin B12 deficiency
D. Dementia with Lewy bodies
E. Normal-pressure hydrocephalus

2. **A 70-year-old man with history of hypertension and diabetes presents with a stepwise loss of intellectual function. Prior episodes have been associated with unilateral weakness and difficulty swallowing. A unilateral Babinski sign is found on neurological examination, what is the most likely diagnosis?**

A. Senile dementia of the Alzheimer type
B. Vascular (multi-infarct) dementia
C. Vitamin B12 deficiency
D. Dementia with Lewy bodies
E. Normal-pressure hydrocephalus

3. **A 52-year-old man develops emotional lability, weight loss, and hallucinations. Over several months he develops a rapidly progressive dementia associated with quick jerks of his arms and legs that are precipitated by movement. An electroencephalogram is abnormal with diffuse slowing and periodic sharp waves. Cerebrospinal fluid analysis shows normal cell count, glucose, and protein. What is the most likely diagnosis in this patient?**

A. Alzheimer dementia
B. Wilson disease
C. Parkinson disease
D. Creutzfeldt-Jakob disease

answers: 1-A 2-B 3-D

1. **Answer is A,**The 80-year-old patient with progressive, steady memory loss and cognitive dysfunction over 2 years have not been found to have a reversible cause of dementia by standard workup. The great majority of such patients have senile dementia of the Alzheimer type. At present, there is no definitive method of premortem diagnosis, but characteristic histologic findings of neurofibrillary tangles and neuritic plaques would be noted at autopsy.

2. **Answer is B,** The 70-year-old with hypertension and previous focal deficits is most likely to have vascular dementia. This is associated with progressive stepwise deterioration, usually the result of recurrent bilateralcerebral infarcts. Focal findings, including hemiparesis, extensor plantar responses, and pseudobulbarpalsy, are common. Vitamin B12 deficiency can cause a dementing illness, often but not always in association with amacrocytic anemia and decrease in proprioception and vibratory sensation. Dementia with Lewy bodiescauses dementia with bradykinesia, visual hallucinations, and sensitivity to the side effects of anticholinergic drugs. Normal-pressure hydrocephalus (NPH) causes dementia, urinary incontinence, and gait disturbance. A workup for treatable causes of dementia should include a vitamin B12 level and CNS imaging to pick up NPH.

3. **Answer is d**. .Creutzfeldt-Jacob disease is a rare form of dementia thatis distinguished from other dementias by early personality change, a rapidly progressive course, thepresence of myoclonus (90% of patients), and distinctive EEG abnormalities (periodic sharp waves). Inthese patients the cerebrospinal fluid cell count, glucose, and protein levels are normal, but the 14-3-3protein is often present. The causative agent is thought to be a prion, which is a transmissible protein. Thedisease usually occurs sporadically though familial cases have been reported. Transmission has alsooccurred by consumption of contaminated beef as well as by transplantation of affected tissue such asdura mater, cornea, or pituitary gland. Creutzfeldt-Jacob disease is rapidly fatal with death occurring inmost cases within a year of symptom onset. Wilson disease and Alzheimer dementia also cause dementia,but they are much more slowly progressive and are not usually associated with myoclonus or periodicsharp waves on EEG. The movement abnormalities of Parkinson disease are tremor and bradykinesia, notmyoclonus. Some patients with multiple sclerosis develop ataxia, but myoclonus is not a feature, anddementia is uncommon.

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