Neurocognitive Disorders

Leader: Hossam Saleh Alawad

Done by: Khaled Alghamdi – Mohannad Alzahrany

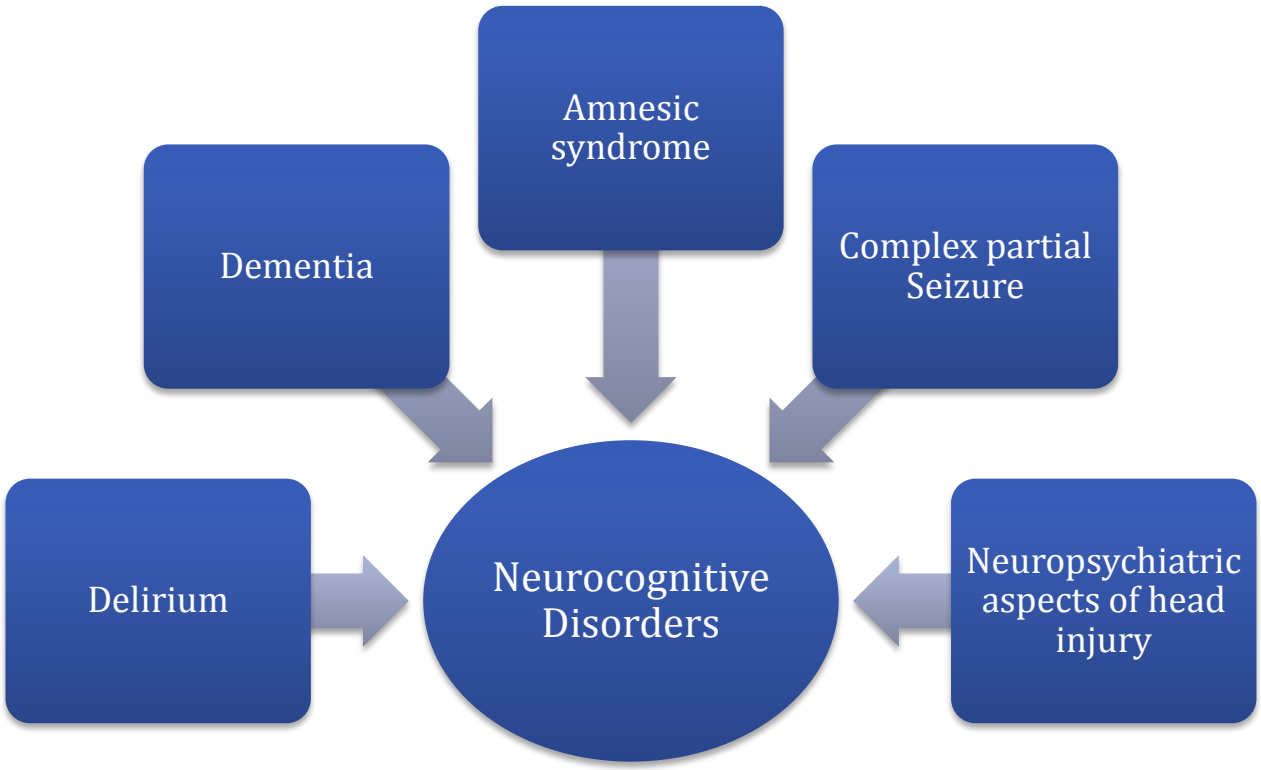
Revised by: Hossam Saleh Alawad

`Doctor's note Team's note Not important Important Book's note

Teams

جامعة الملك سعود
King Saud University

Mind Map



Introduction

In psychiatry, the words "cognition/cognitive" are used in 2 different contexts:

- **Cognitive functions:** attention, concentration, orientation, and memory. Disorders of which are called: “Cognitive disorders”

- **Cognitive Processes:** ways of thinking and conclusion formation.

- **Cognitive Therapy:** a type of psychotherapy that is concerned with detection and correction of wrong thoughts & thinking process (negative cognition) commonly seen in patients with anxiety & depressive disorders.

Cognitive Therapy is <u>not</u> a treatment of cognitive disorders.

- **Cognitive Disorders**

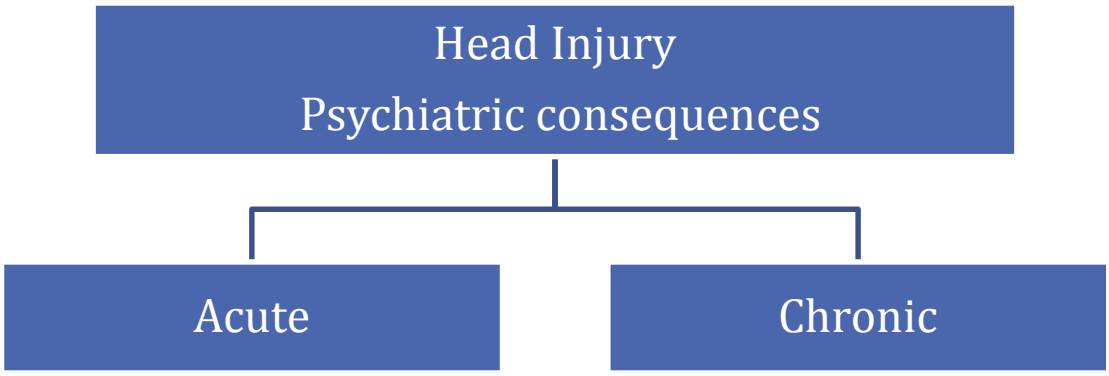
- **Delirium:** an acute global cognitive disorder with disturbed consciousness.

- **Dementia:** a chronic global cognitive disorder without disturbed consciousness.

- **Amnestic (amnesic) syndrome:** a specific disorder of short-term memory.

In the **DSM-5** classification:

- Delirium.
- Mild Neurocognitive Disorders (new category).
- Major Neurocognitive Disorders (dementia, amnestic syndrome).



1-Delirium (الهذيان-الهُذاء)

Mr. Hassan is a 75-year-old man was brought to the emergency department by his sons because of 3 days history of fluctuating consciousness, disorientation, disturbed perception, speech, thinking, and behavior. Recently he developed fever and urinary incontinence.

Definition: Acute transient reversible global cognitive impairment with impaired consciousness due to a medical problem.

Epidemiology: It may occur in anyone at any age but more in elderly and children. The highest rate of delirium is found in post-cardiotomy patients > 80%. In ICU 30%, post burn patients 20% & among hospitalized patients about 10%. Delirium is under-diagnosed especially when patient is hypoactive, somnolent or with minimal features. Such cases may be misdiagnosed as depression.

Mnemonic: Acute Co Co Cause

Diagnostic criteria (simplified):

A. Consciousness is disturbed (i.e., awareness of the environment is impaired but patient is not in coma).

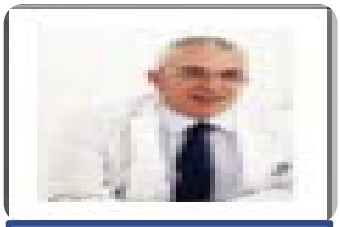
B. Cognitive functions are impaired +/- perceptual disturbances (illusions or hallucinations).

C. Acute onset with fluctuating symptoms (within hours during the day) & transient course (few days).

D. Caused by a physical problem (e.g. hypoxia, hypoglycemia, infection...others see causes).

youtube.com/watch?v=lJH1AoVuVS0

Abdulrahman



Dr

- Dr. is delirium a serious condition & why?

Yes, Abdulrahman. It is a very serious medical & psychiatric condition due to high risks of:

1- Death (b/o the serious nature of the associated medical conditions).
2- Suicide. 3- Violence. 4- Impaired judgment. 5- Psychosis.

- Dr. why does a delirious patient become suicidal or aggressive?

Due to the severe disturbance in the patient's perception, mood, thinking, and behavior. Patient may act on hallucinations, illusions or delusional thoughts as if they were genuine dangers (e.g., blood extraction by a nurse might be perceived as an attack). However, the clinical presentation differs from patient to patient. Some patients may be excessively somnolent, and some may fluctuate from one state to the other, usually restless at night and sleepy during the day with lucid intervals.

Mr. Hassan showed difficulty focusing, sustaining, and shifting attention. He was not cooperative during physical & mental status examinations. He was agitated, shouting, and tried to pull out his intravenous lines.

- Dr. is there a specific diagnostic investigation for delirium?

No, it is a bedside clinical diagnosis. Thus, good clinical skills are essential:

A. **History:** acute onset + medical disease + consciousness & cognitive disturbances.

B. **MSE:** proper assessment of mental functions.

- **Etiology:**

Abdulrahman, what are the common causes of delirium?

- Infections: e.g. UTI, chest infection, encephalitis, septicemia.
- Medications (e.g. anticholinergics). Anticholinergics have central and peripheral side effect, which cause delirium is a central side effect include impaired concentration, confusion, attention deficit, and memory impairment.
- Metabolic & electrolyte disturbances. E.g. Hypothyroidism and hyperthyroidism - Thiamine deficiency - Sodium (Na) disturbances.
- Endocrinopathies (e.g. hypoglycemia).
- Hypoxia; cardiac or respiratory failure.
- Renal failure; uremia.
- Hepatic failure; encephalopathy.
- CNS: seizure/head trauma/substance abuse (intoxication or withdrawal).

Regardless of the cause, the presentation is similar.

- **Risk factors:**

- Age ≥ 70 years. - Fever, DM, HTN, COPD and Organ failure.

- Past history of delirium. - Current history of dementia.

- Substance abuse. - Multiple medications. Drug interaction

Investigations:

Blood: CBC + differential WBCs. Blood chemistries (including electrolytes, renal and hepatic indexes, and glucose). Blood culture. Blood drug screen. Thyroid function tests. CPK.

Urine: Urinalysis. Culture & sensitivity. Urine drug screen.

Additional tests when indicated:
Chest XR/ECG/EEG (in seizure)/ Brain scan (CT or MRI).
Lumbar puncture and CSF examination.

Mr. Hassan's history revealed memory deterioration and time disorientation over the past 5 years.

Differential Diagnosis (DDx):

1- Dementia: occasionally, delirium occurs in a patient with dementia, a condition known as **beclouded dementia**. However, a dual diagnosis (i.e. dementia and delirium) can only be made when there is a definite history of preexisting dementia.

2- Substance abuse; alcohol, inhalants, sedatives, and opioids.

3- Amnestic syndrome.

4- Acute functional psychosis (brief psychosis, mania, and exacerbation of schizophrenia or schizoaffective disorder): patients usually experience no change in their level of consciousness or in their orientation. The hallucinations and delusions are more constant and better organized than those of patients with delirium.

5- Severe Depression: patients with hypoactive symptoms of delirium may appear somewhat similar to severely depressed patients, but they can be distinguished on the basis of an EEG (normal in depression). When a delirious patient is treated with tricyclic antidepressants (TCAs), his/her cognitive functions deteriorate further because of the anticholinergic effect of (TCAs).

Treatment: (It should be in a well-equipped medical rather than a psychiatric ward).

1. The cause should be searched for and treated properly, e.g. ensure electrolyte balances, enough oxygen, nutrition, and hydration. The referring physician should do this task.

2. Control mental and physical disturbance with antipsychotics e.g. Haloperidol (1mg oral, IV, or IM) or Olanzapine (5mg oral or IM) 2-3 times/day. Intramuscular administration may be preferable for some patients with delirium who are poorly compliant with oral medications or who are too sedated to safely swallow tablets.

3. Limit benzodiazepines (or give with extreme caution) because their effects may increase disorientation, drowsiness and ataxia with possible falls, head trauma and fractures.

4. Keep the patient in a quiet, well lit-room; avoid over and under stimulation. Frequently reorient, reassure and explain procedures clearly to the patient.

Types of delirium (Meagher 1996):

Hyperactive (30%)	Hypoactive (24%)	Mixed (46%)
The most clear and least controversial.	The most difficult type to identify. A large percentage of these patients are inappropriately diagnosed and treated as depressed. Classically, these patients present with symptoms that are commonly associated with depression (lethargy, apathy, decreased level of alertness, psychomotor retardation, and decreased speech production).	The classic waxing and waning pattern. Commonly seen in surgical patients (agitated at times, with alternating episodes of hypoactivity).

Course and Prognosis: The course is usually short (7-10 days). However, the symptoms of delirium usually persist as long as the causally relevant factors are present. The longer the patient has been delirious and the older the patient, the longer the delirium takes to resolve. Delirium may spontaneously clear or progress rapidly into dementia or into death; because of the serious nature of the associated medical conditions. When treated, it usually resolves rapidly. However, some residual deficit may persist. It is sometimes followed by depression. Prognosis is bad

Neurocognitive Disorders (DSM-5)

1. **Delirium:** The criteria for delirium have been updated and clarified on the basis of currently available evidence.

2. **Mild Neurocognitive Disorder:** it describes a less severe & less disabling level of cognitive impairment that requires compensatory strategies and accommodations to help maintain independence and perform activities of daily living. To be diagnosed with this disorder, there must be changes that impact cognitive functioning. These symptoms are usually observed by the individual a close relative, other knowledgeable informant such as a friend, colleague or clinician or they are detected through objective testing. This diagnostic category provides an opportunity for early detection and treatment of cognitive decline before patients' deficits become more pronounced and progress to major neurocognitive disorder (dementia) or other debilitating conditions. Its inclusion in the manual will help clinicians develop effective treatment plans as well as encourage researchers to evaluate diagnostic criteria and potential therapies. Recent studies suggest that identifying mild neurocognitive disorder as early as possible may allow interventions to be more effective. Early intervention efforts may enable the use of treatments that are not effective at more severe levels of impairment and may prevent or slow progression.

3. **Major Neurocognitive Disorder:** it includes dementia and amnestic disorder. However, the term dementia can be used in the etiological subtypes. An updated listing of neurocognitive domains is also provided in DSM-5, as these are necessary for establishing the presence of NCD, distinguishing between the major and mild levels of impairment, and differentiating among etiological subtypes.

2- Dementia

Aminah is a 73-year-old diabetic woman noticed to show a gradual loss of social skills, a decreased range of interest, multiple somatic complaints, and memory impairment.

Definition: a progressive impairment of cognitive functions occurring in clear consciousness.

Epidemiology: The prevalence of moderate to severe dementia in the general population is 5% > 65 years, 20-40% in > 85 years of age. In outpatient general medical practices, it is 15-20%, and 50% in chronic care facilities.

Affective symptoms, including depression and anxiety, are seen in 40 to 50% of demented patients. Delusions and hallucinations occur in 30%.

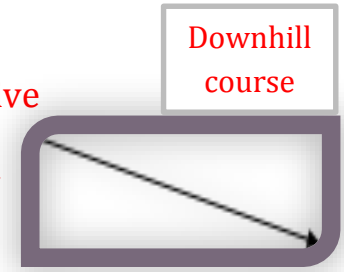
Features: The essential feature is a loss of intellectual abilities of sufficient severity to interfere with social or occupational functioning or both.

In early stages	In late stages
Cognitive impairment may not be apparent. <u>Features include:</u> - A gradual loss of social and intellectual skills (first noticed in work setting where high performance is required). - Mild memory impairment. - Subtle changes in personality. - Changes in affect (irritability, anger ...). - Multiple somatic complaints and vague psychiatric symptoms.	Cognitive disturbances emerge: - Increasing memory impairment (esp. recent memory). - Attention impairment. - Disorientation: particularly to time, and when severe to place and person. - Language: vague and imprecise speech with inappropriate repetition of the same thoughts (perseveration). - Impaired judgment. - Potential aggression (verbal & physical). - Psychotic features: hallucinations and delusions. - Emotional lability. - Catastrophic reaction marked by agitation secondary to the subjective awareness of intellectual deficits under stressful circumstances. **Sundowner Syndrome:** Drowsiness, confusion, ataxia, and accidental falls. It occurs in demented patients when external stimuli, such as light and interpersonal orienting cues, are diminished.

youtube.com/watch?v=uAlkCMfTASQ

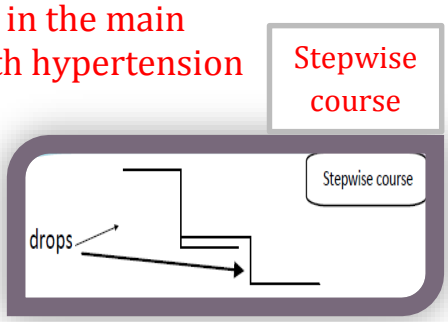
youtube.com/watch?v=_hRBPrfDQVI

Causes of dementia:



1. Alzheimer's disease (50 to 60% of dementias): Progressive downhill deterioration of intellectual functioning due to a degenerative process affecting the whole cortex, especially cholinergic neurons.

2. Vascular (multi-infarct) dementia (10 to 25% of dementias): Declining stepwise deterioration of intellectual functioning due to multiple infarcts of varying sizes or arteriosclerosis in the main intracranial vessels. It usually occurs in patients with hypertension or diabetes. Stepwise course (multiple drops). Onset: after a stroke, its sudden onset may resemble delirium. Some cases follow a stationary course.



3. Medical conditions (reversible conditions; 15% of dementias) e.g., metabolic causes: vitamin deficiency (e.g. B12, folic acid), hypothyroidism and TB affecting CNS.

4. Substance-induced dementia: e.g. alcoholic dementia.

5. Parkinson's Disease: it is a disease of the basal ganglia, commonly associated with dementia and depression. An estimated 20 -30% of patients with Parkinson's disease have dementia, and an additional 30 - 40% has measurable impairment in cognitive abilities.

6. Others:

- Lewy Body Disease: a dementia clinically similar to Alzheimer's disease and often characterized by Visual hallucinations, parkinsonian features, and extrapyramidal signs. Lewy inclusion bodies are found in the cerebral cortex. The exact incidence is unknown. These patients show marked adverse effects when given antipsychotic medications.

- Normal pressure hydrocephalus: Progressive memory impairment, slowness and marked unsteady gait (+ urine incontinence in late stages).

- Huntington's chorea: global intellectual impairment with extra pyramidal features.

- Creutz Feldt–Jakob's disease.

- AIDS dementia.

- Pick's disease (dementia of frontal lobe type).

- Binswanger's Disease (also known as subcortical arteriosclerotic encephalopathy): it is characterized by the presence of many small infarctions of the white matter that spare the cortical regions.

Dementias are classified as **cortical** and **subcortical** depending on the site of the cerebral lesion. A **subcortical** dementia occurs in vascular dementia, Parkinson's disease, normal pressure hydrocephalus, Huntington's disease and Wilson's disease. The **subcortical** dementias are associated with psychomotor retardation, movement disorders, gait incoordination, apathy, and akinetic mutism, which can be confused with catatonia.

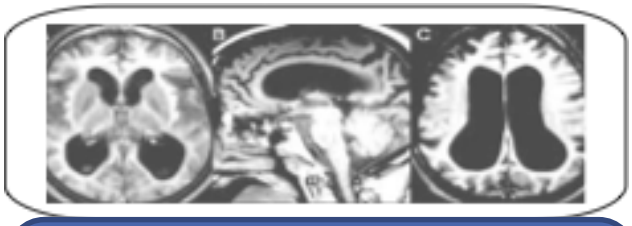
Course and Prognosis: (depend on the cause)

Alzheimer's dementia shows a progressive slow deterioration. The patient may become incontinent of urine and/or stool. Vascular dementia shows stepwise deterioration or stationary course after a massive stroke that is then followed by a good control of the risk factors e.g., HTN, DM ...etc.

Investigations:

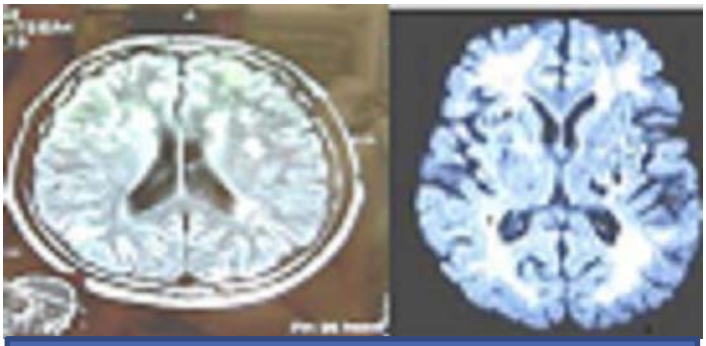
Essential workup to confirm dx / exclude treatable causes: B12 and folate blood levels. Thyroid Function Tests (TSH, T3 and T4). Brain CT or MRI.

- Dementia effect frontal and temporal lobes.
- Postmortem examination is the confirmatory of the type of dementia.



Alzheimer's dementia:

(Cortical atrophy+ wide sulci, gyri, & ventricles).



Vascular dementia:

Multiple infarcts.

Differential Diagnosis (DDx):

1. Normal aging: age-related cognitive decline (the course is not progressively deteriorating), no loss of social or occupational functioning.

2. Pseudo-dementia (Depression in the elderly): cognitive disturbance is relatively of rapid onset and proceeded by depressive features. Patient is aware of problems & often answers, "I don't know" compared to confabulation in demented patient. The differentiation is sometimes difficult as demented patients may also become depressed as they begin to comprehend their progressive cognitive impairment. EEG and CT scan are normal in pseudo-dementia. See major depressive episode (MDE) later.

3. Delirium: the onset is rapid and consciousness is impaired.

Treatment:

1. Supportive measures:

A- Provide good meals & hygiene. B- Encourage family's involvement. C- Support the caregiver. D- Keep in familiar settings if possible to avoid accidents, wandering away...etc.

2. Specific measures:

A- Identify and correct any treatable or controllable condition e.g. hypothyroidism, vitamin B12 deficiency, hypertension or diabetes.

B- Symptomatic treatment:
- Agitation, aggression: small doses of major tranquilizers (e.g. Olanzapine 5mg).
- Insomnia: a small dose of major tranquilizers (e.g. olanzapine 5mg) or benzodiazepine (e.g. lorazepam 1mg).
- Depression: small doses of antidepressant (e.g. citalopram 10 – 20 mg).

  Be aware of possible mental side effects of such medications (over-sedation, risk of falling down, head trauma & fractures and central anticholinergic activity that may cause delirium).

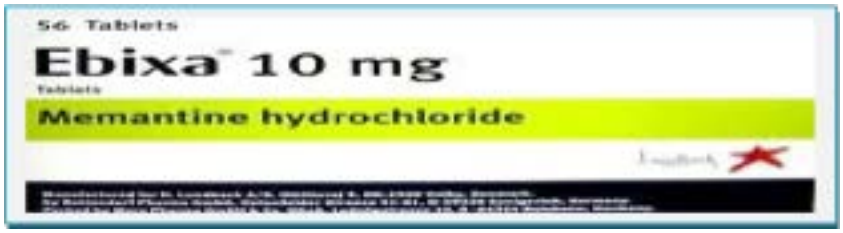
C- Cognitive-enhancing medications (mainly for Alzheimer's dementia).

1- <u>Cholinesterase Inhibitors:</u>

- Donepezil (Aricept): 5 mg at night & can be increased gradually to 10 mg. It is well tolerated. (S/E: diarrhea, weight loss, bradycardia, and syncope).
- Rivastigmine (Exelon): 1.5 mg twice/day & can be increased gradually to maximum 6 mg twice/day. (S/E: anorexia, fatigue somnolence, and dizziness). Also available as a skin patch.
- Galantamine (Reminyl): 4mg twice/day, can be increased gradually to 12mg twice/day. (S/E: similar to rivastigmine).

2- NMDA receptor antagonist; Memantine (Ebix):

An N-methyl-D-aspartate (NMDA) receptor antagonist protects neurons from neurodegenerative process induced by glutamate excito-toxicity. Memantine has been shown to have a modest effect in moderate-to-severe Alzheimer's disease and in dementia with Lewy bodies. It is in general well tolerated. Adverse drug reactions include confusion, dizziness, drowsiness, headache, insomnia, agitation, and/or hallucinations. Less common adverse effects include vomiting, anxiety, hypertonia, cystitis, and increased libido.



3- Amnestic (Amnesic) Syndrome

A 48-year-old alcoholic man displayed significant cognitive and behavioral problems. He had difficulty with learning new information and making appropriate plans.

Definition: impairment in the **short-term memory** (retention of new information; temporal lobe function) due to a specific organic cause, in the absence of generalized intellectual impairment. It leads to social and occupational dysfunctioning. The patient may show confabulation (filling memory gaps with incorrectly retrieved information). The insight is partially impaired.

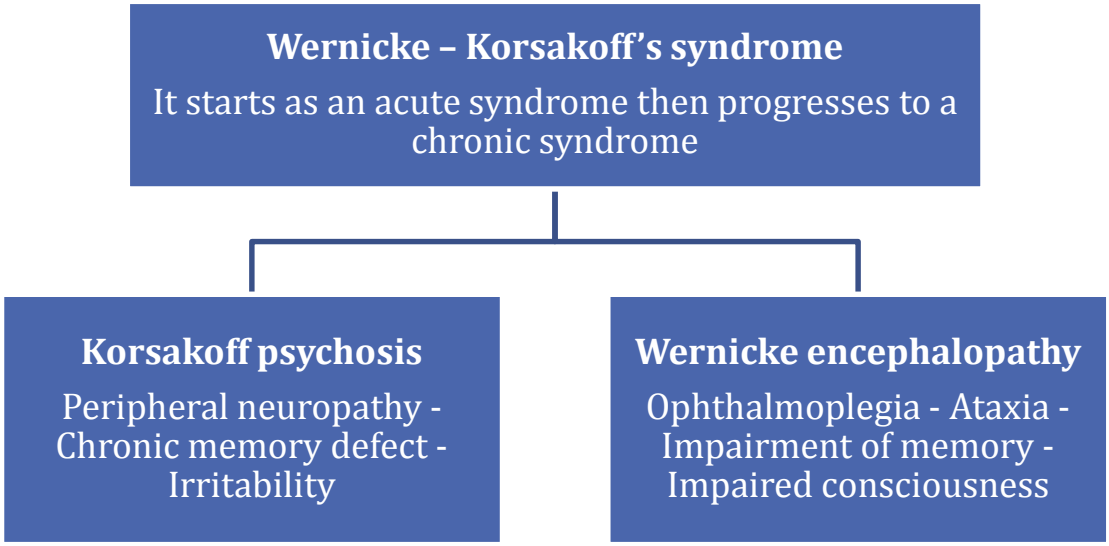
In contrast to delirium, the **immediate** memory is usually **intact** i.e. digit span test (frontal lobe function) is normal. In contrast to dementia, the **remote** memory is intact.

Clinical Assessment: memory assessment (normal registration and long-term memory but defected short-term recall).

Etiology:

- Head injury lesions (hippocampus, posterior hypothalamus and nearby midline structures).
-Thiamine (B1) deficiency, (associated with alcohol abuse, gastric carcinoma and persistent vomiting). Thiamine is essential for the enzyme transketolase, which is essential for glucose metabolism.

Amnestic Syndrome is most commonly found in alcohol use disorders (Wernicke – Korsakoff's syndrome, see below).



Treatment:

- Identify and reverse the cause if possible.
- Thiamine supply (if due to thiamine deficiency).
- Supportive medical measures (no specific treatment).

Prognosis:

If it is due to thiamine deficiency and thiamine is provided promptly, prognosis is good. Otherwise, the course is usually chronic and may be progressive. Psychiatric symptoms occur with increased frequency in patients with seizures as a result of underlying brain tissue injury, side effects from anticonvulsant medications, or seizure-specific psychiatric disturbances.

4- COMPLEX PARTIAL SEIZURE

Definition:

Episodic brief recurrent attacks stereotypic in nature associated with paroxysmal discharges of epileptic foci, usually located in limbic structure, particularly the temporal lobes (70% arise from temporal lobe; therefore, it is commonly called temporal lobe epilepsy).

Features: (depend on the site of the focus)

1- Pre-ictal: irritability, lethargy and dizziness.
2- Aura: epigastric discomfort associated with distortion of sensations; visual, auditory, gustatory, olfactory or tactile.
3- Ictus:
- Behavior disturbances: repetitive movements, e.g. chewing, grimacing, automatism.
- Confusion and disturbed consciousness.
- Fear, panic, derealization, memory disturbance.
- Thinking disturbances.
- Hallucinations.

Complex partial seizures, the most common focal seizures found in adults (30 % of all adult epileptics), may appear at any age; onset is usually in adolescence. Seizure may be triggered by bright lights, colors, noises, trauma or intense emotions.

Possible Causes:

- Perinatal injuries.
- Prolonged febrile convulsions (lead to mesiotemporal sclerosis).
- Trauma to the base of the skull.
- Hamartomas of temporal lobe, fibrosis or gliosis.
- Hippocambal sclerosis.
- Vascular malformation.

Diagnosis:

This type of seizure disorder may mimic and be confused with any psychiatric disorder:
- Psychosis: schizophrenia, mood disorder, brief psychosis, etc.
- Neurosis: panic disorder, generalized anxiety disorder, depersonalization disorder …etc.

Diagnosis is mainly clinical; EEG findings are not necessary for diagnosis. (EEG with sphenoidal or nasopharyngeal leads, shows temporal area spikes).

Treatment:

1- Proper assessment of all aspects of the patient's life.
2- Anticonvulsants e.g. carbamazepine (Tegretol) 200 - 400 mg twice or three times per day.
3- Neurology consultation is helpful.

5- HEAD INJURY - Neuro-psychiatric Aspects

Hamad is a 19-year-old male was involved in a road traffic accident lost consciousness for 5 days, and remained 3 weeks in the hospital. After discharge, his parents noticed that he became impulsive, disinhibited, and aggressive at times.

A. Acute consequences:

1. Impaired consciousness in varying duration (hours, days, weeks or months) long duration suggests poor prognosis.
2. Delirium (after severe head trauma).
3. Memory defects: on recovery of consciousness, defects of memory are usually present.

A. Anterograde (post-traumatic) amnesia: amnesia for events in the time between the trauma and the resumption of normal continuous memory. It is a good prognostic factor: probably full recovery when anterograde amnesia was less than 12 hours.

B. Retrograde amnesia: amnesia for events in the time between the trauma and the last clearly recalled memory before the injury. Final duration is frequently less than 1 minute. It is not a good predictor of outcome.

Factors affecting the outcome of head trauma:

1. Duration of loss of consciousness.
2. Duration of anterograde (post-traumatic) amnesia.
3. Amount and location of brain damage.
4. Premorbid personality and past psychiatric history.
5. Development of seizures.
6. Medico-legal factors e.g. compensation.

B. Chronic Consequences:

1. Lasting cognitive impairment: there is more likelihood of cognitive impairment when the injury has caused a prolonged post traumatic amnesia (of more than 24 hours). Cognitive impairment was particularly associated with parietal and temporal damage, especially on the left side. Recovery of function may be very slow and may continue over the years.
2. Emotional disturbances: depressive, anxiety and phobic features are common, and associated with somatic complaints such as headache, fatigue and, dizziness.
3. Personality changes:
a. There may be irritability, reduced control of aggressive impulses,
b. Sexual disinhibition and some coarsening of behavior and premorbid personality traits, particularly after frontal lobe injury.
4. Psychotic features: psychotic features related to depression (non-dominant frontal damage). Paranoid Psychosis (temporal lobe damage).
5. Social consequences: many patients and their relatives experience severe distress of head injury, and have to make substantial changes in their way of life.
6. Medico - legal aspects:
compensation issue is more likely to contribute to disability if the patient feels someone else is at fault, financial compensation is possible, low social status and in industrial injury.

Treatment:

A plan for long-term treatment should be made as early as possible after head trauma. The treatment of the cognitive and behavioral disorders is similar to the treatment approaches used in other patients. However, head trauma patients may be particularly susceptible to the side effects associated with antipsychotics; therefore, these drugs should be initiated in lower dosages than usual and should be titrated upward more slowly than usual. Aggression and impulsivity can be treated with anticonvulsants or antipsychotics. Treatment should include physical and psychological rehabilitation to which the clinical psychologist can sometimes contribute behavioral and cognitive techniques. Problems of litigation and compensation should be settled as early as possible. Continuing psychosocial help should be provided to patient and carers, by a special team.

Summary

	Delirium	Dementia
Onset	Acute	Gradual / insidious (except for vascular dementia caused by stroke)
Consciousness	Impaired	Intact
Course	Fluctuates / transient / clears within 7-10 days	Chronic / deteriorating

- Amnesic syndrome is Impaired memory (recent memory mainly) due to a specific organic cause, in the absence of generalized intellectual impairment.
- Complex Partial Seizure is episodic brief recurrent attacks stereotypic in nature associated with paroxysmal discharges of epileptic foci, usually located in limbic structure, particularly the temporal lobes (70 % arise from temporal lobe; therefore, it is commonly called temporal lobe epilepsy).
- Head injuries are common, with the majority being closed injuries (blunt trauma) occurring in motor accidents.

Causes of delirium	Causes of dementia
Infections (e.g. UTI, chest infection, encephalitis, septicemia)	**Alzheimer's disease** (50 to 60% of dementias) (downhill course)
Medications (e.g. anticholinergics)	**Vascular (multi-infarct) dementia** (10 to 25% of dementias) (stepwise course)
Metabolic & electrolyte disturbances. (e.g. Hypothyroidism and hyperthyroidism - Thiamine deficiency - Sodium (Na) disturbances)	**Medical conditions** (reversible conditions; 15% of dementias) (e.g., metabolic causes: vitamin deficiency, hypothyroidism and TB affecting CNS)
Endocrinopathies (e.g. hypoglycemia)	**Substance-induced dementia** e.g. alcoholic dementia
Hypoxia; cardiac or respiratory failure	**Parkinson's Disease** (it is a disease of the basal ganglia)
Renal failure; uremia	**Lewy Body Disease** (Visual hallucination)
Hepatic failure; encephalopathy	**Normal pressure hydrocephalus**
CNS: seizure/head trauma/substance abuse (intoxication or withdrawal)	**Huntington's chorea** (global intellectual impairment with extra pyramidal features)
	Creutz Feldt–Jakob's disease
	AIDS dementia
	Pick's disease (dementia of frontal lobe type)
	Binswanger's Disease (also known as subcortical arteriosclerotic encephalopathy)

MCQ's

1. **A 75-year-old man admitted in the surgical ward because of prostate carcinoma, urinary retention and urinary tract infection. At night, he became hostile, irritable, drowsy and uncooperative. The most likely diagnosis:**
    A. Adjustment disorder.
    B. Dementia.
    C. Acute stress disorder.
    D. Delirium.

2. **A 74-year-old woman known case of hypertension and diabetes mellitus developed dysarthria due to a transient ischemic attack. She has poor attention span and memory impairment for several months. The most likely primary diagnosis is:**
    A. Alzheimer's disease.
    B. Delirium.
    C. Vascular dementia.
    D. Amnestic syndrome.

3. **A 65-year-old woman uses antihistamine drugs for her chronic increasing insomnia. Last week she was commenced on Amitriptyline 50 mg by a GP for insomnia. Her husband found her disoriented, hallucinating and hyperthermic. Her face was flushed and her skin was dry. She developed:**
    A. Neuroleptic malignant syndrome.
    B. Serotonergic syndrome.
    C. Anticholinergic syndrome.
    D. Wernicke – Korsakoff's syndrome.

4. **A 45-year-old man presented with disorientation, ataxia and poor memory. He asked for a referral to a specialist in eye diseases. The most likely cognitive impairment in this patient is:**
    A. Short-term memory.
    B. Immediate memory.
    C. Recent memory.
    D. Orientation to time.

5. An 80-year-old man has progressive deterioration in memory, disorientation, and visual hallucinations. The most likely diagnosis is:

A. Alzheimer's disease.
B. Delirium.
C. Vascular dementia.
D. Amnestic syndrome.

Answers

1	2	3	4	5
D	C	C	A	A

Short Answer Questions

Case 1. A 77- year old diabetic woman brought by her son to the emergency department with 3 days history of fluctuating consciousness, pyrexia, dysarthria, amnesia, and disturbed behavior.

Q1. Mention two psychiatric differential diagnoses & justify each one.

Q2. Mention two clinical tests you would use to evaluate her cognitive functions.

Q3. Mention one medication used to control her disturbed behavior, the dose and the class.

Answers:

A1.

	Diagnosis	Justification
1	Delirium	Acute onset + confusion + pyrexia + disturbed behavior.
2	Vascular dementia – acute phase	Dysarthria + amnesia + old age + DM.

A2. Any 2 of the cognitive function tests (attention-concentration – orientation- memory)

1- **Attention test** (to spell a word backward e.g. World, to mention 5 words with the same letter, or by the digit span test (ability to repeat 7 digits e.g. 3,8,1,4,7,2,9 after an examiner dictates them slowly, first forward, then backward).

2- **Concentration test:** by naming the months of the year in reverse order or by subtracting serial 7s from 100 (serial 7s test): patient is asked to subtract 7 from 100 then to take 7 from the remainder repeatedly until it is less than seven.

A3. Haloperidol 0.5-2 mg (oral, IM or Slow IV); typical antipsychotic medication or olanzapine 5mg (oral or IM); atypical antipsychotic medication.

Case 2: A 68-year-old man seen at out-patient clinic because of 7 months history of increasing forgetfulness of very recent events, gradual loss of social skills, decreased range of interest, and multiple non-specific physical complaints. His history revealed chronic alcohol abuse.

Q1. Mention two psychiatric differential diagnoses & justify each one.
Q2. Mention 2 relevant history questions that would guide you to the diagnosis.
Q3. Mention one important lab. Investigation that you would request and the reason?

Answers:

A1.

	Diagnosis	Justification
1	Dementia (Alzheimer or vascular)	7 months h/o increasing forgetfulness of very recent events, gradual loss of social skills, decreased range of interest, and multiple nonspecific physical complaints.
2	Amnesic Syndrome	Chronic alcohol abuse + increasing forgetfulness of very recent events.

A2. Any two of the following:

Past history of:

1- Impaired consciousness/Ophthalmoplegia/Ataxia **(Wernicke encephalopathy)**

2- Peripheral neuropathy **(Korsakoff psychosis)**.

OR

Features suggestive of vascular dementia (stepwise course – neurological features …).

A3. Lab Investigation is B1 (thiamine). The reason: low B1 in alcohol abuse.

OR

Lab Investigation is B12 (or TFT). The reason: dementia due to low B12 or hypothyroidism.

Case 3: A 64-year-old hypertensive woman seen regularly at cardiology clinic presented with 3 months history of deterioration of mental functions, decreased range of interest, poor sleep, poor appetite, and multiple non-specific physical complaints**.**

Q1. Mention two psychiatric differential diagnoses & justify each one.

Q2. Mention 2 relevant clinical questions (history / MSE) that would guide you to the diagnosis.

Q3. Would you recommend Brain CT-scan? And why (justify your answer whether yes or no).

Answers:

A1.

	Diagnosis	Justification
1	Dementia (Alzheimer or vascular)	Old age - deterioration of mental functions.
2	Major Depressive Episode (Pseudo-dementia)	Decreased range of interest, poor sleep, poor appetite, and deterioration of mental functions.

A2. Any two of the following:

1- Which features started first; in MDE depressive features proceed cognitive disturbances & vice versa.

2- Onset: in MDE: cognitive disturbance is relatively of rapid onset.

3- Awareness: Patient with MDE (pseudo-dementia) is aware of his cognitive problems & often answers, "I don't know" compared to confabulation in demented patient.

A3. Yes, because brain CT scan is normal in pseudo-dementia and abnormal in dementia (wide sulci- ventricles- infarcts …).

For any suggestions:

Dr.7ossam1993@gmail.com