433 Teams

PSYCHIATRY

Lecture 3

Neuro-Cognitive Disorders

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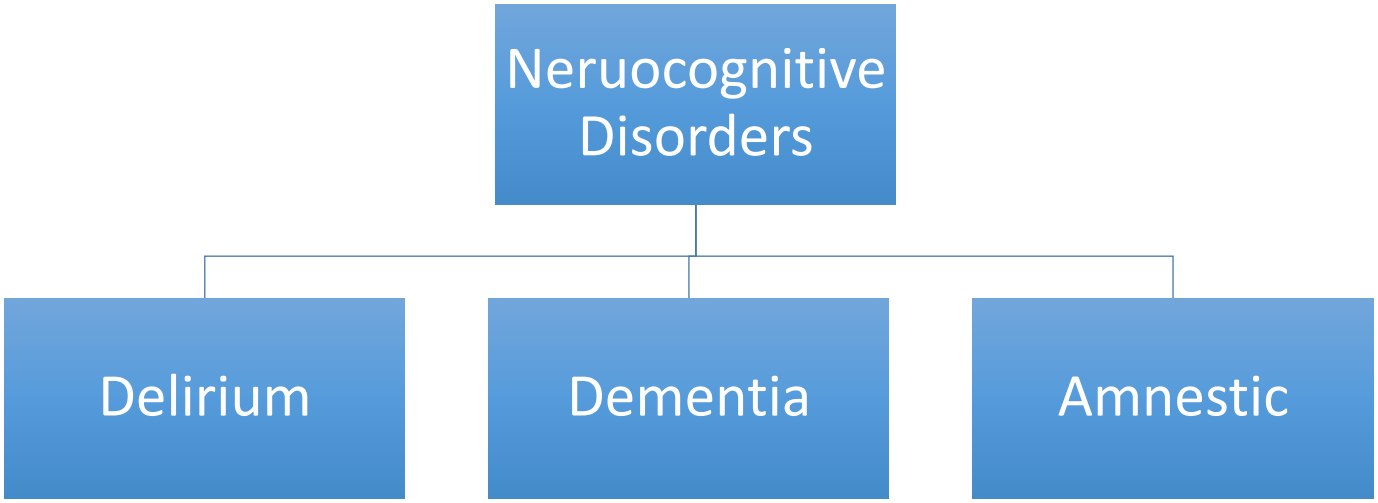
INTRODUCTION

- 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 not a treatment of cognitive disorders.



***In the DSM-5 classification:**

Neurocognitive Disorders

- **Delirium**
- **Mild Neurocognitive Disorders (new category):** 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.
- **Major Neurocognitive Disorders** (it includes dementia and amnestic disorder.

Case Study :

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.

What is Delirium?

Acute onset of **fluctuating** cognitive impairment (global)and a **disturbance of consciousness**.

Delirium is a syndrome, not a disease, and it has many causes, all of which result in a similar pattern of signs and symptoms.

Delirium is acute brain failure.

Classically, delirium has a **sudden onset** (hours or days).Rapid improvement when the causative factor is identified and eliminated .

Abnormalities of mood, perception, and behavior are common psychiatric symptoms.

Reversal of sleep-wake pattern (sleep during the day and awake at night) .

Tremor, asterixis, nystagmus, incoordination, and urinary incontinence are common in delirium.

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.

Diagnostic Criteria (Simplified)

Mnemonic *Acute Co Co Cause*

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

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

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

4. Caused by a physical problem (e.g. hypoxia, hypoglycemia, infection...).

Is delirium a serious condition & why?

Yes. 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.

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

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.

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.

Causes of delirium (I WATCH DEATH):

I	**Infections**: encephalitis*, meningitis*, sepsis
W	**Withdrawal**: ETOH (Wernicke's encephalopathy *), sedative-hypnotics, barbiturates
A	**Acute metabolic**: acid-base, electrolytes, liver or renal failure
T	**Trauma:** brain injury, burns
C	**CNS pathology**: hemorrhage*, seizures, stroke, tumor (don't forget metastases)
H	**Hypoxia***: CO poisoning, hypoxia, pulmonary or cardiac failure, anemia
D	**Deficiencies**: thiamine, niacin, B12
E	**Endocrinopathies**: hyper- or hypo- adrenocortisolism, hyper- or hypoglycemia*
A	**Acute vascular**: hypertensive encephalopathy* and shock
T	**Toxins or drugs:** pesticides, solvents, medications, (many!) drugs of abuse, anticholinergics, narcotic analgesics, sedatives (BDZ)
H	**Heavy metals*:** lead, manganese, mercury

*Life threatening causes of delirium

Types of Delirium

Hyperactive (30%) The most clear and least controversial.

Hypoactive (24%) 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)

Mixed (46%) The classic waxing and waning pattern. Commonly seen in surgical patients (agitated at times, with alternating episodes of hypoactivity).

Mini-mental state exam

Tests orientation, short-term memory, attention, concentration, constructional ability.

30 points is perfect score

< 20 points indicated probably ORGANIC etiology (delirium or dementia).

Not helpful without knowing baseline, but is useful for following over time.

Investigations

Electrolytes	Arterial blood gas or Oxygen saturation
CBC	Urinalysis +/- Culture and sensitivity
EKG	Urine drug screen
CXR	Blood alcohol
EEG- not usually necessary	

The most important part of the workup for delirium is to have the suspicion. As with most other diagnoses, the answer can often be found in the history and interview. Speaking with family members to see if the new behavior is a change from baseline is a huge help.

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 (see dementia later in this chapter).

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).

Management & Treatment

A. 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.

B. 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.

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

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

*Rarely, some antipsychotics are associated with torsade de pointes arrhythmia by lengthening the QT interval; avoid or monitor this by ECG monitoring.

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.

Case Study :

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.

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

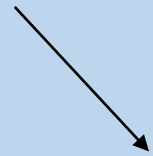
Epidemiology: The prevalence of moderate to severe dementia in the general population is 5 % > 65 years, 20- 40 % in > 85 years of age. In outpatient general medical practices, it is 15 - 20 %, and 50 % in chronic care facilities. Affective symptoms, including depression and anxiety, are seen in 40 to 50% of demented patients. Delusions and hallucinations occur in .30%

In early stages	In late stages
Cognitive impairment may not be apparent. Features include : - 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.

Causes of dementia

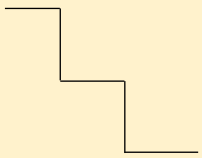
1. Alzheimer's disease (50 to 60% of dementias): Progressive ***downhill*** deterioration of intellectual functioning due to a degenerative process affecting the whole cortex, especially cholinergic neurons.



Downhill course

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.



Stepwise course

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

***Lewy Body Disease**:* a dementia clinically similar to Alzheimer's disease and often characterized by 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).

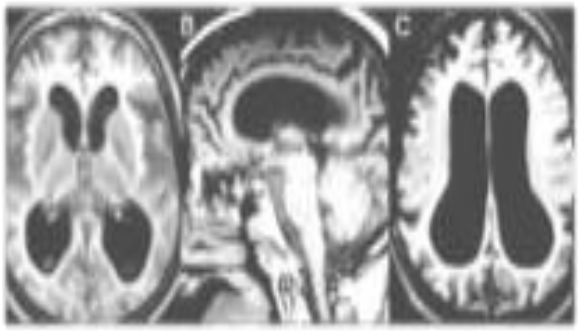
***Huntington's chorea**:* global intellectual impairment with extra pyramidal features. *Creutz Feldt–Jakob's disease. AIDS dementia . Pick's disease* (dementia of frontal lobe type).

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

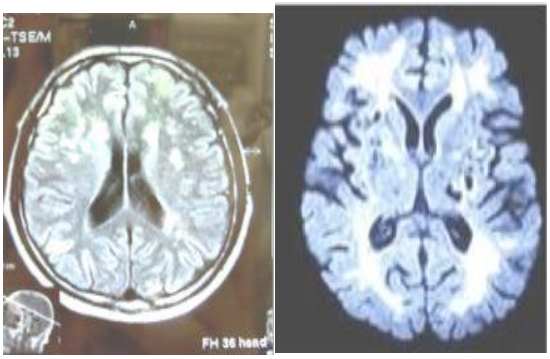
Dementias are classified as **cortical** and **subcortical** depending on the site of the cerebral lesion. A subcortical dementia occurs in vascular dementia, Parkinson's disease, normal pressure hydrocephalus, Huntington's disease and Wilson's disease. The subcortical dementias are associated with psychomotor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

Investigations

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



*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.

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

Management & Treatment

Supportive measures

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

Specific measures

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

Cognitive-enhancing medications

- Mainly for Alzheimer's dementia
- ***Cholinesterase Inhibitors*** *:*
- Donepezil(Aricept) S/E: diarrhea, weight loss, bradycardia, and syncope.
- Rivastigmine(Exelon) S/E: anorexia, somnolence, and dizziness.
- Galantamine (Remiryl) S/E: anorexia, somnolence, and dizziness.
- **NMDA glutamate receptors antagonist**: Memeantine*(Ebix)* 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
- **Drugs with high anticholinergic activity & Benzodiazepines should be avoided.**

Case Study :

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 **immediat**e 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).

Causes of Amnestic Syndrome

1-Head injury lesions (hippocampus, posterior hypothalamus and nearby midline structures).

2-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*)

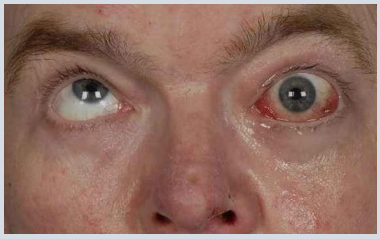
Wernicke encephalopathy (Acute)

•Clinical triad:

1-Encephalopathy: Impairment of memory and consciousness.

2- Ophthalmoplegia

3- Gait Ataxia



***Korsakoff psychosis* (Chronic)**

Peripheral neuropathy.

Chronic memory defect.

Irritability.

Delirium Tremens

a psychotic condition typical of withdrawal in chronic alcoholics, involving tremors, hallucinations, anxiety, and disorientation.

Treatment & Prognosis

-Identify and reverse the cause if possible.

-Thiamine supply (if due to thiamine deficiency) BEFORE glucose (because if glucose is given first , it will use an important substrate of thiamine which facilitate the action of many enzymes of TCA cycle , that will result in further depleting of thiamine -----> worsen wernicke's encephalopathy and also prevent TCA cycle from using any further glucose) , check Magnesium.

-Supportive medical measures; fluids & nutrition.

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 because of underlying brain tissue injury, side effects from anticonvulsant medications, or seizure-specific psychiatric disturbances.

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. outcome.

B. Chronic Consequences:

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.

Emotional disturbances: depressive, anxiety and phobic features are common, and associated with somatic complaints such as headache, fatigue and, dizziness.

Personality changes: There may be irritability, reduced control of aggressive impulses, Sexual disinhibition and some coarsening of behavior and premorbid personality traits, particularly after frontal lobe injury.

Psychotic features: psychotic features related to depression (non-dominant frontal damage). Paranoid Psychosis (temporal lobe damage). **Social consequences:** many patients and their relatives experience severe distress of head injury, and have to make substantial changes in their way of life.

Medico-legal aspects: compensation issue is more likely to contribute to disability if the patient feels someone else is at fault, financial compensation is

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.

Capacity vs. Competency

- **Clinical** vs. **Legal** term that denotes the ability to make rational and reasonably well informed decisions by a particular patient (vs. person) in their treatment and/ or life decision/s
- **Capacity** is a *clinical* determination that addresses the integrity of *mental functions.*
- **Competency** is a *legal* determination that addresses societal interest in restricting a person's right to make decisions or do acts because of incapacity.

Features	Delirium	Dementia
Onset	Acute	Insidious
Course	Fluctuating	Progressive
Duration	Days to weeks	Months to years
Consciousness	Altered	Clear
Attention	Impaired	Normal, except in severe dementia
Psychomotor changes	Increased or decreased	Often normal
Reversibility	Usually	Rarely
Treatment	Treat the underlying cause + Haloperidol (to control mental and physical disturbance)	Support + Specific measures + Cholinesterase Inhibitors or NMDA

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