NEUROCOGNITIVE DISORDERS

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Cognitive functions

- Attention
- Concentration
- Memory
- Speed of Processing
- Orientation

- Perseveration
- Impulsiveness
- Language Processing
- "Executive functions"

Neurocognitive Disorders

- Cognitive deficits are present in <u>many mental disorders</u> but only disorders whose core features are cognitive are included in the NCD category.
- The cognitive decline was <u>not present</u> from birth or very early in life therefore represent a decline from a previously attained level of functioning.

Topics:

- Delirium
- Major NCDs (dementias and amnestic syndrome).
- Mild NCDs.
- Seizure
- TBI.

Case no 1

- 81 year old man with diagnosis of Benign Prostate Hypertrophy and Hypertension, came in with 3 days history prior to admission of:
- low-grade fever and nocturia.
- Poor sleep. (Daughter gave him diphenhydramine for sleep).
- On day of admission, became confused, had high grade fever. No loss of consciousness, vomiting
- Past medical history: Hypertension
- Personal/ Social History: Smoke tobacco
- In hospital, diagnosed to have UTI and acute urinary retention.
- 6 hours after admission, became combative, agitated, confused. Pulled out IV and insisted on going home



Many terms are used to describe this disorder:

- Acute confusional state
- acute organic syndrome
- Acute brain failure.
- Acute brain syndrome.
- Acute cerebral insufficiency.
- Exogenous psychosis.
- Metabolic encephalopathy.
- ICU psychosis
- Toxic encephalopathy.

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) diagnostic criteria for delirium is as follows

- Disturbance in attention (ie, reduced ability to direct, focus, sustain, and shift attention) and awareness.
- Change in cognition (eg, 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.
- 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.

Clinical features

- Acute onset of mental status change with fluctuating course.
- Attentional deficits.
- Confusion or disorganized thinking.
- Perceptual disturbances.
- Disturbed sleep/wake cycle. (sundowning phenomena)
- Altered psychomotor activity.
- Disorientation and memory impairment.
- Behavioral and emotional abnormalities.
- Other cognitive deficits

Epidemiology

100%

• Delirium complicates at least 25% of all hospitalizations in the elderly



Prevalence of delirium

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Fong et al 2009
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Importance

- Previous studies 32%-66% of cases are unrecognized by Medical Staff
 - ↑ morbidity and mortality
 - 1 length of stay
 - ↑ rates of admission to long term care facilities
 - 20% of patients discharged post hip # still had evidence of delirium (Journal of American Geriatric Society 2001 May;49(5):678-9).

Types:

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%)	Waxing and waning pattern. Commonly seen in surgical patients (agitated at times, with alternating episodes of hypoactivity).

Causes: "I WATCH DEATH"

- I nfections
- ullet W ithdrawal
- A cute metabolic
- **T** rauma
- C NS pathology
- H ypoxia

- D eficiencies
- E ndocrinopathies
- A cute vascular
- **T** oxins or drugs
- H eavy metals

Predisposing risk factors

- >60 years of age
- Male sex
- Visual impairment
- Underlying brain pathology such as stroke, tumor, vasculitis, trauma, dementia
- Major medical illness
- Recent major surgery

- Depression
- Functional dependence
- Dehydration
- Substance abuse/dependence
- Hip fx
- Metabolic abnormalities
- Polypharmacy



TREATMENT OF THE UNDERLYING MEDICAL CAUSE

Non-Pharmacological

- Symptomatic measures involving attention to fluid and electrolyte balance, nutritional status, and early treatment of infections.
- Early mobilization
- environmental interventions.
- Reduce unfamiliarity by providing a calendar, a clock, family pictures, and personal objects.
- Maintain a moderate sensory balance in the patient by avoiding sensory overstimulation or deprivation.
- Minimize staff changes, limit ambient noise and the number of visits from strangers, and provide a radio or a television set, a nightlight, and where necessary, eyeglasses and hearing aids.
- Proper communication and support are critical with these patients

Pharmacological

- All the patient's medications should be reviewed, and <u>any unnecessary drugs</u> should be discontinued.
- These patients should receive the lowest possible dose and <u>should not get</u> drugs such as phenobarbital or long- acting benzodiazepines.
- Often used is haloperidol starting at 0.25 mg daily. Haloperidol may be repeated every 30 minutes, PO or IM.
- The atypical antipsychotics—risperidone, olanzapine, quetiapine, and aripiprazole—may be used at low doses.

Any Questions...

Just Ask!







Why changing names

- Dementia typically refers to degenerative d/o in elderly
- DSM expands category to d/o of younger
 - e.g. HIV, traumatic brain injury.
- More global.
- Individuals with substantial decline in a single domain can receive diagnosis

Characteristic :

- Significant Cognitive Decline
- Interfere with independence
- Not due to delirium
- Not due to other mental disorder

Cognitive domains:

- Complex attention
- Executive function
- Learning & memory
- Language
- Perceptual-motor
- Social cognition

Dementia:

- A chronic global impairment of cognitive functions without disturbed consciousness.
- The essential feature is a loss of intellectual abilities of sufficient severity to interfere with social or occupational functioning or both.

In early stages

- Gradual loss of social and intellectual skills (first noticed in work setting where high performance is required).
- Decrease in the range of interest and enthusiasm.
- Mood changes (depressed, irritable, or easily provoked).
- Multiple somatic complaints with vague psychiatric symptoms.

In late stages, cognitive disturbances emerge:

- Memory impairment (short-term memory first then, in advanced stages longterm memory is affected).
- Thinking and speech: inappropriate repetition of the same thoughts (perseveration) with vague and imprecise speech.
- Significant change in mood and behavior: shrinkage of social interaction with catastrophic reaction (agitation under stressful circumstances secondary to the subjective awareness of intellectual deficits)
- Judgment impairment.
- Psychotic features: hallucinations and delusions.
- Disorientation: particularly to time, and when severe to place and person.

Epidemiology:

- No gender difference
- Increasing age is the most important risk factor. It is primarily a disorder of the elderly (if < 65 years, it is called presenile dementia).
- 3.1/1000 person years at age 60-64
- 175.0/ 1000 person years at age 95+

The most common causes of dementia :

- <u>Alzheimer's disease</u>: continuous deterioration of intellectual functioning due to degenerative process affecting the whole cortex, especially cholinergic neurons.
- <u>Vascular (multi-infarct) dementia</u>: 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.
- Medical conditions: e.g. metabolic causes: vitamin deficiency (e.g. B12), hypothyroidism.

Differential diagnosis:

- Normal aging: age-related cognitive decline (the course is not progressively deteriorating), no loss of social or occupational functioning.
- Depression in the elderly (Pseudo-dementia): cognitive disturbance is relatively of rapid onset and preceded by depressive features. 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.
- Delirium: the onset is rapid and consciousness is impaired. Some demented patients may develop delirium. Diagnosis of dementia cannot be made before delirium clears.

Delirium vs. Dementia vs. Depression

Features	Delirium	Dementia	Depression
Onset	Acute (hours to days)	Insidious (months to years)	Acute or Insidious (wks to months)
Course	Fluctuating	Progressive	May be chronic
Duration	Hours to weeks	Months to years	Months to years
Consciousness	Altered	Usually clear	Clear
Attention	Impaired	Normal except in severe dementia	May be decreased
Psychomotor changes	Increased or decreased	Often normal	May be slowed in severe cases
Reversibility	Usually	Irreversible	Usually

Treatment:

First, Identify and correct any treatable or controllable condition e.g.: hypothyroidism, vitamin B12 deficiency, hypertension, diabetes. Supportive measures:

- provide good physical care (meals, hygiene, ...).
- encourage the family's involvement.
- support the care givers (they are prone to depression).
- keep in familiar settings if possible to avoid accidents, wandering away,...etc.

Symptomatic treatment:

- If agitated, aggressive, or insomniac: give a small dose of antidopaminergic drug (e.g. olanzapine 5mg, risperidone 2mg, or quetiapine 25mg). If depressed: give a small dose of antidepressant (e.g. escitalopram 5 mg or sertraline 25mg). Be aware of possible mental side effects of such medications e.g. confusion, over-sedation, risk of falling down.
- Memory-enhancing medications (mainly for Alzheimer's dementia); Cholinesterase Inhibitors :
- 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 6mg twice/day (S/E: anorexia, fatigue, somnolence, and dizziness)
- Galantamine (Reminyl):4mg twice/day & can be increased gradually to 12mg twice/day (S/E: similar to rivastigmine)
- Memantine (Epixa, Akatinol): an N-methyl-D-aspartate (NMDA) receptor antagonist, protects neurons from neurodegenerative process induced by glutamate excitotoxicity.

Amnestic syndrome :



Characteristic:

- It is a major NCD due to another medical condition.
- impaired short-term memory due to a specific brain insult, in the absence of generalized intellectual impairment.
- The immediate memory (frontal lobe function) is usually intact: i.e. digit span test is normal.
- It is often known as Wernicke Korsakoff's syndrome.

Wernicke's encephaopathy

- an acute syndrome
- Characterized by impairment of 1- memory
 - 2- ataxia
 - 3- ophthalmoplegia

Korsakoff's psychosis

- Chronic memory defect.
- Peripheral neuropathy
- Irritability

Major Causes of Amnestic Disorders

Systemic medical conditions

Thiamine deficiency (Korsakoff's syndrome)

Hypoglycemia

Primary brain conditions

Seizures

Head trauma (closed and penetrating) Cerebral tumors (especially thalamic and temporal lobe)

Cerebrovascular diseases (especially thalamic and temporal lobe) Surgical procedures on the brain Encephalitis due to herpes simplex

Hypoxia (including nonfatal hanging attempts and carbon monoxide poisoning)

Transient global amnesia Electroconvulsive therapy

Multiple sclerosis

Substance-related causes

Alcohol use disorders

Neurotoxins

*Benzodiazepines (and other sedative-hypnotics)

Many over-the-counter preparations

Treatment & prognosis:

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

Prognosis: Good vs Bad

Mild Neurocognitive Disorder

- 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:
- 1. Concern of the individual, a knowledgeable informant, or the clinician that there has
- been a mild decline in cognitive function; and
- 2. A modest impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.
- 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).
- Specify etiology whether due to: Alzheimer's disease, Lewy body disease, vascular disease, traumatic brain injury, substance/medication use, Parkinson's disease, Huntington's disease, another medical condition, multiple etiologies, unspecified.

Epilepsy:



Complex partial seizure:

- 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).
- 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.

Features: (depend on the site of the focus)

- Pre-ictal: irritability, lethargy and dizziness.
- Aura: epigastric discomfort associated with distortion of sensations; visual, auditory, gustatory, olfactory or tactile.

Ictus:

behavior disturbances: repetitive movements, e.g. chewing, grimacing, automatism.

confusion and disturbed consciousness.

fear, panic, derealization, memory disturbance.

thinking disturbances.

hallucinations .

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:
- 1- Psychosis: schizophrenia, mood disorder, brief psychosis, etc.
- 2- 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).

Management:

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

Traumatic Brain Injury





Definition:

- an insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness
- Area of function affected:
- 1- Cognitive
- 2- Sensory/perceptual
- 3- Seizures
- 4- Other physical changes
- 5- Social-emotional

Acute consequences:

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

amnesia for events in the time between the trauma and the resumption of normal continuous memory.

retrograde amnesia:

amnesia for events in the time between the trauma and the last clearly recalled memory before the injury. It is *not* a good predictor of outcome.

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 behaviour and premorbid personality traits, particularly after frontal lobe injury.

Con't

• Psychotic features:

Depression with psychotic features (associated with non-dominant frontal damage). Paranoid Psychosis (associated with 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 possible, low social status and in industrial injury.

Factors affecting the outcome of head trauma:

- Loss of consciousness and post-traumatic amnesia.
- Amount and location of brain damage.
- Premorbid personality and past psychiatric history.
- Development of seizures.
- Medico-legal factors e.g. compensation.

Management:

- Pharmacotherapy.
- physical therapy.
- occupational therapy.
- Recreation therapy.
- speech therapy.
- Cognitive rehabilitation especially during the first 6 months after injury.

