

# Neurocognitive Disorders

An introduction – CNS Block

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# Objectives

- ▶ At the end of this lecture, student should be able to
  1. Know the cognitive functions & the neurocognitive disorders.
  2. Understand delirium and know how to detect it.
  3. Understand dementia and know how to detect it.
  4. Know other neurocognitive disorders (amnesic syndrome/ TBI).

# Cognition

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

**Delirium.**

**Mild Neurocognitive Disorders.**

**Major Neurocognitive Disorders: Dementia-  
Amnestic syndrome.**

**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). It is *not* a treatment of cognitive disorders.

# Delirium

- ▶ Delirium is a global impairment of cognitive functions and awareness of the surrounding (consciousness).
- ▶ Acute, severe, & reversible.
- ▶ Usually associated with disturbances of the following:
  - Perception (hallucinations/illusions).
  - Thinking (delusions).
  - Affect/Mood (perplexity/ irritability).
  - Behavior (agitation/aggression).

The patient may be dangerous to himself or others.

Thus, delirium is one of the serious emergencies.

**Epidemiology:** more common among elderly and children. M=F. Among hospitalized patients about 10 %, post burn patients 20%, intensive care unit 30%.

## Types of delirium

### Hyperactive (30%)

The most clear and least controversial in diagnosis.

### Mixed (46%)

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

### Hypoactive (24%)

- . Classically, these patients present with symptoms that resemble depression (lethargy, slowness, decreased level of alertness, and decreased speech production).
- . A large percentage of these patients are inappropriately diagnosed as depressed.
- . The most difficult type to identify.

# Etiology

- ▶ Metabolic disturbances/ electrolyte imbalance.
- ▶ Endocrinopathies (e.g. hypoglycemia, hyperglycemia).
- ▶ Medications (multiple drugs with multiple interactions).
- ▶ Infections: systemic (e.g. septicemia), specific (e.g. encephalitis).
- ▶ Organ failure: e.g. hepatic encephalopathy, uremia, hypoxia.
- ▶ Neurological diseases: seizure / head trauma.
- ▶ Substance abuse: intoxication or withdrawal (e.g. delirium tremens ).

▶ **DDx:**

- ▶ Other neurocognitive disorders (may coexist) e.g., stroke.
- ▶ Acute psychosis (no disturbance in awareness of the environment).

▶ **Course and Prognosis:**

- ▶ The course is usually short self-limiting (7-10 days).
- ▶ If not treated may progress rapidly into death or dementia.
- ▶ When treated, it usually resolves rapidly.
- ▶ However, some residual deficit may persist. Patients may have another episode later in their life.

# Treatment

- ▶ Detect the cause (s) & treat it properly, e.g. infection, electrolyte imbalances
- ▶ Control mental and physical disturbance with antidopaminergics, e.g. haloperidol (1 mg oral, IV, or IM), quetiapine 25mg, or Olanzapine (5mg oral or IM) 2- 3 times/day.
- ▶ IM 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.
- ▶ Limit benzodiazepines (or give with extreme caution) because their effects may increase disorientation, drowsiness and ataxia with possible falls, head trauma and fractures.
- ▶ Keep the patient in a quiet, well lit-room; avoid over and under stimulation. Frequently reorient, reassure and explain procedures clearly to the patient.



# Dementia

- ▶ A chronic global impairment of cognitive functions without disturbed consciousness.
- ▶ **Features:** The essential feature is a loss of intellectual abilities of sufficient severity to interfere with social or occupational functioning or both.
  - Memory impairment (short-term memory first then, in advanced stages long-term memory is affected).
  - Thinking and speech: inappropriate repetition of the same thoughts (perseveration) with vague and imprecise speech.
  - Shrinkage of social interaction with other.
  - Disorientation: particularly to time and place and when advanced to person (can't identify relatives).
  - Judgment impairment.
  - Psychotic features: hallucinations and delusions.

# Dementia

- ▶ **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).

- ▶ **The most common causes of dementia :**

- ❑ Alzheimer's disease: continuous deterioration of intellectual functioning due to degenerative process affecting the whole cortex, especially cholinergic neurons.
- ❑ Vascular (multi-infarct) dementia: 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., Parkinson's D., metabolic causes: severe B 12 deficiency, hypothyroidism.

# Dementia

► **DDx:**

1. *Normal aging*: age-related cognitive decline (the course is not progressively deteriorating), no loss of social or occupational functioning.
2. *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.
3. *Delirium*: the onset is rapid and consciousness is impaired.

Some demented patients may develop delirium. Diagnosis of dementia *cannot* be made before delirium clears.

# Dementia

**Course and Prognosis:** usually progressive deterioration (slow downhill in Alzheimer's dementias and stepwise in vascular dementia). Some patients become double incontinent.

## Treatment:

- ▶ 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.
- ▶ Specific measures: identify and correct any treatable or controllable condition e.g.: hypothyroidism, vitamin B12 deficiency, hypertension, diabetes.
- ▶ Medications: 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.

# Dementia

► **Memory-enhancing medications (mainly for Alzheimer's dementia)**

*Cholinesterase Inhibitors:*

Donepezil, Rivastigmine, or Galantamine.

*Memantine:* an N-methyl-D-aspartate (NMDA) receptor antagonist , protects neurons from neurodegenerative process induced by glutamate excitotoxicity.

# Amnestic Syndrome

- ▶ It is a major NCD but focal impairment of *short-term memory* (hippocampal pathology).
- ▶ It leads to social and occupational impairment.
- ▶ It's old terminology is Wernicke–Korsakoff's syndrome, which starts as an acute syndrome (Wernicke's encephalopathy) characterized by impairment of memory, ataxia, ophthalmoplegia and impaired consciousness. Then followed by Korsakoff's disorder (chronic short-term memory defect, peripheral neuropathy and irritability).

## Etiology:

- ▶ The most common cause is thiamine (vitamin B<sub>1</sub>), deficiency associated with alcohol abuse. Thiamin is essential for the enzyme transketolase which is important for glucose metabolism.
- ▶ Other causes of thiamine deficiency include gastric carcinoma and persistent vomiting (e.g. typhoid fever).

# Amnestic Syndrome

## Treatment:

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

## Prognosis:

- ▶ If is provided promptly, prognosis is good.
- ▶ Otherwise, the course is usually chronic and may be progressive.
- ▶ Psychiatric symptoms & seizures may arise as a result of underlying brain tissue injury.

# Traumatic Brain Injury (TBI)

TBI is a 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

The neuropsychiatric effects of head trauma include:

► **A. Acute consequences:**

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



# Traumatic Brain Injury (TBI)

## ▶ **Chronic Consequences:**

- ▶ Lasting cognitive impairment.
- ▶ Emotional disturbances / Personality changes: There may be irritability, reduced control of aggressive impulses, sexual disinhibition.
- ▶ Psychotic features: delusions/ hallucinations.
- ▶ Social consequences:
- ▶ Medico-legal aspects: Compensation

# Traumatic Brain Injury (TBI)

## ► Treatment:

- ❑ A plan for long-term treatment should be made as early as possible after head trauma.
- ❑ 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.