433 Teams

PSYCHIATRY

Neuro-Cognitive Disorders

Lecture contents:

- Introduction
- Delirium
- Dementia
- Amnestic syndrome
  - Wernicke-Korsakoff's syndrome
- Head injury
- Capacity vs. Competency

Manual of Basic Psychiatry

Blue slides

Doctor's notes

Important

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Introduction

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

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

2. 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!

Cognitive Disorders

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

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

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

In the DSM-5 classification:
Neurocognitive Disorders
1-Delirium
2-Mild Neurocognitive Disorders (new category).
3-Major Neurocognitive Disorder.

1- Delirium هذيان

Case: **patr1** 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.

Patr2 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.

Definition:

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

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

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%.** **"40 to 50 percent of patients who are recovering from surgery for hip fractures"**

Delirium is under-diagnosed especially when patient is hypoactive, somnolent, or with minimal features. Such cases may be **misdiagnosed as depression**.

Diagnostic criteria (simplified): Acute Co Co Cause

A. **Co**nsciousness is disturbed (i.e., awareness of the environment is impaired but patient is not in coma).
B. **Co**gnitive 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).

Delirium has bad prognosis:

- May progress to stupor, coma, seizures or death, particularly if untreated.
- Increased risk for postoperative complications, longer postoperative recuperation, longer hospital stays, long-term disability.
- **Elderly** patients 22-76% chance of dying during that hospitalization
- Several studies suggest that **up to 25% of all patients with delirium die within 6 months**.

Causes: **"I WATCH DEATH"** 44% estimated to have 2 or more etiologies.

Infections: encephalitis, meningitis, sepsis." UTI in females, pneumonia in males".

Withdrawal: ETOH, sedative-hypnotics, barbiturates

Acute metabolic: acid-base, electrolytes, liver or renal failure

Trauma: brain injury**, burns**

CNS pathology: hemorrhage, seizures, stroke, tumor (don't forget metastases)

Hypoxia: CO poisoning, hypoxia, pulmonary or cardiac failure, anemia

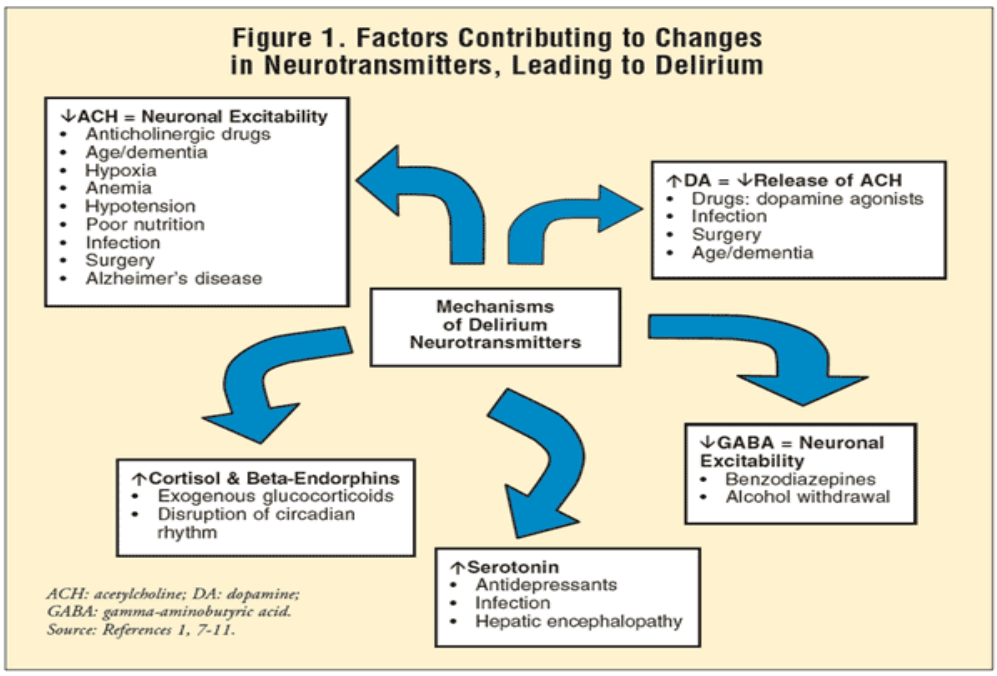
Deficiencies: thiamine, niacin, **B12**

Endocrinopathies: hyper- or hypo- adrenocortisolism**, hyper- or hypoglycemia**.

Acute vascular: hypertensive encephalopthy and shock

Toxins or drugs: pesticides, solvents, medications, (many!) drugs of abuse anticholinergics, narcotic analgesics, sedatives (BDZ).

Heavy metals: lead, manganese, mercury.



Life threatening causes of delirium (WHHHIMP):

- Wernicke's encephalopathy, Hypoxia, Hypoglycemia, Hypertensive encephalopathy Intracerebral hemorrhage, Meningitis/encephalitis and Poisoning

Risk Factors:

Extremes of age, Number of medications taken, Preexisting brain damage (e.g., dementia "delirium on top of dementia", cerebrovascular disease, tumor), History of delirium, **Alcohol dependence**, Diabetes, Cancer, Sensory impairment and Malnutrition.

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. / Brain scan (CT or MRI), Lumbar puncture and CSF examination**.**

Differential Diagnosis (DDx):

1- **Dementia**.

	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

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).

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

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.

Summary

- Delirium is common and is often a harbinger of death- especially in vulnerable populations.
- It is a sudden change in mental status, with a fluctuating course, marked by decreased attention.
- It is caused by underlying medical problems, drug intoxication/withdrawal, or a combination.
- Recognizing delirium and searching for the cause can save the patient's life.

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

Dementia الخرف

Case: 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.

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: 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.

Late stages

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.

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, 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.

5. Others:

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.

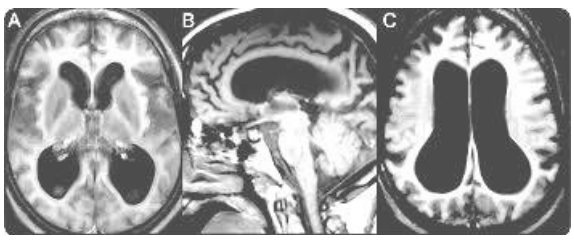
Course and Prognosis: (depend on the cause).

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.

Investigation:

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



Pic1 Alzheimer's dementia : (cortical atrophy+ wide sulci, gyri, & ventricles).

Pic2 Vascular dementia: multiple infarcts

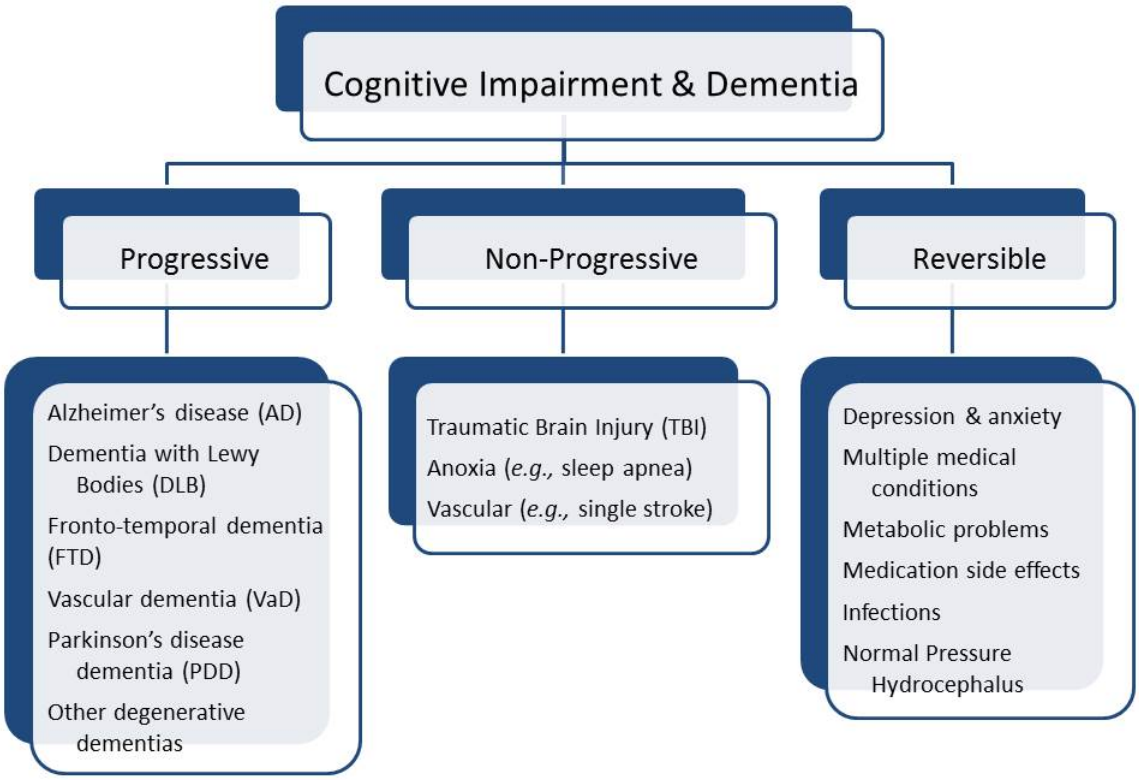
Ddx:

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, 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):

I- 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). Also available as a skin patch
Galantamine (Reminyl): 4mg twice/day, can be increased gradually to 12mg twice/day. (S/E: similar to rivastigmine).

II- 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.

Amnestic (Amnesic) Syndrome

"Was not mentioned by the doctor"

Case 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**.

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).

Wernicke – Korsakoff's syndrome

It starts as an acute syndrome then progresses to a chronic syndrome.

Wernicke encephalopathy: Ophthalmoplegia, Ataxia, Impairment of memory and impaired consciousness

Korsakoff psychosis: Peripheral neuropathy, Chronic memory defect and Irritability.

Treatment:

-Identify and reverse the cause if possible.
-Thiamine supply (if due to thiamine deficiency).
-Supportive medical measures; fluids & nutrition. **(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 because of underlying brain tissue injury, side effects from anticonvulsant medications, or seizure-specific psychiatric disturbances.

HEAD INJURY "Neuro-psychiatric Aspects"

"Was not mentioned by the doctor"

Case: 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.

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.

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.

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 "mentioned by the doctor only"

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.

Valid Informed Consent:

Permission voluntary given by a competent person without any elements of force, deceit, coercion after explanation and disclosure of

1. Purpose and details of procedure or treatment
2. Risks, Benefits and available alternative treatment/s
3. The right to withdrawal consent verbally or in written forms at any time.

Exceptions:

- Life threatening situation
- Patient who waive their rights to disclose and consent **(do not want to be informed)**
- Instances where " disclosure' may be harmful to the patient " Therapeutic privileges"

Rules of capacity:

- Being mentally ill doesn't in itself imply a loss of capacity or competency.
- Having Capacity or being Competent should be presumed until proven otherwise.

Steps in Mental Capacity Assessment:

A.

- General perspective or specific (Psychiatric hospitalization, ECT)
- Find out the best language of communication
- Determine if patient has adequate information on which to base a decision
- MMSE: attention, concentration, memory
- Inform the patient about the nature of the disorder, AND the risk and benefit of the PROPOSED treatment, and of ALTERNATIVE treatments or of NO treatment

B.

- Repeat information number of times and in different ways.
- Let the patient paraphrase or restate the understanding.
- Evaluate nature of questions that patient asks regarding treatment plan
- **Periodical Reassessment of capacity (if any change in clinical conditions or, mental status such as in delirium or any modifications in treatment plan)**

C.

- If patient has "severe deficit" in understanding information-→ No Capacity to make informed consent or make decision→ **Arrange a process for " a substitute decision maker".**

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