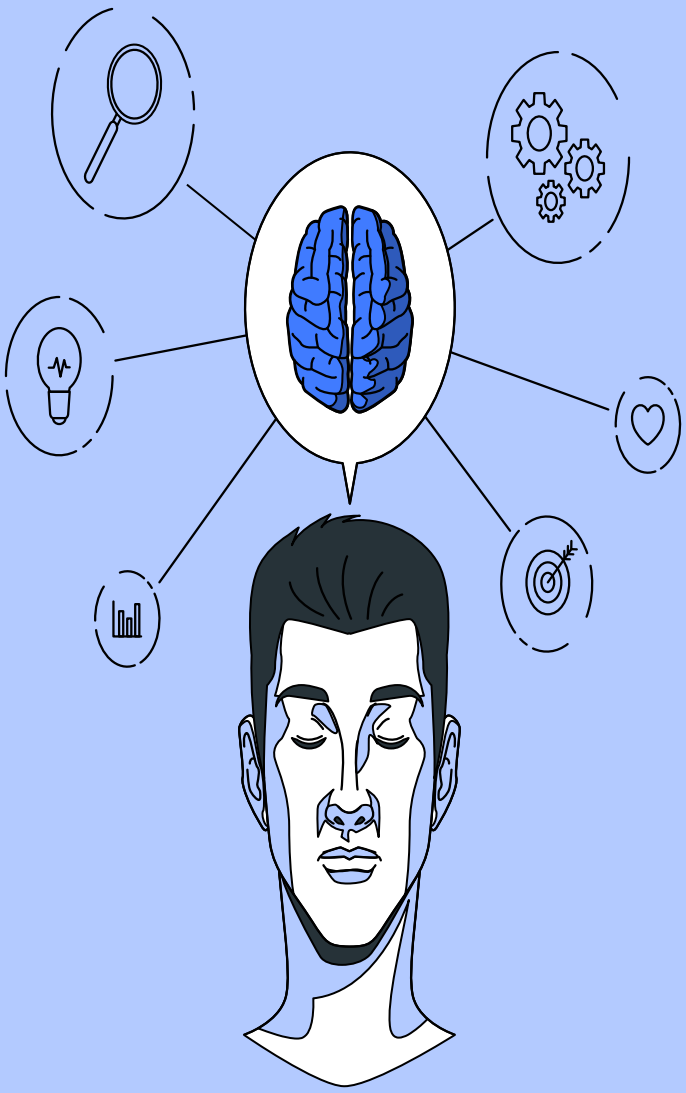


Psychotic Disorders

- Objectives: No objectives are given
- Sources: Dr. Mohammed Aljaffer Slides
- Done by: Mashael Alkahtani and Norah Alkadi

- Color index: Golden notes - Dr. notes - extra

Imp BEFORE studying

- Summary (found in female drive) is enough with dr's notes.
- This is based on females' slides which is different from males'.

Case

Complaint: Nasser is a 28 year-old single male who was brought to the emergency room by his family. They noticed gradual changes in his behavior 9 month when he started eating only canned food but not cooked food made by his family. He started to become agitated and they have noticed that He talks to himself and stares occasionally on the roof of his room. He is afraid of being poisoned.

Past hospitalization: He had two brief psychiatric hospitalizations in the last 3 years . Precipitated by anger at his neighbor and voices commenting about his behavior.

Childhood: Nasser was a healthy child, but he was a bedwetter. He was slower to develop than his siblings.

What are the possible etiological reasons?

What are the ddx?

What are the main symptoms and signs?

Notes:

- Psychosis is a symptom not illness (impaired reality testing includes: hallucination, paranoia.
- Paranoia: المبالغة في الشكوك
- Never diagnose a psych illness before ruling out secondary causes for e.g hallucination due to hypoglycemia
- Myxedema madness: severe hypothyroidism. An important feature is psychosis

SCZ

Schizophrenia:
Schizo means split
Phrenia means mind

Prevalence and incidence world wide:

- It is not a single disease
- Prevalence 0.6-1.9%
- Annual incidence of 0.5-5.0 per 10,000

Age and Gender:

- Females 25-35 years old
- Males 10-25 years old
- Onset is adolescents
- Gender only differ in onset

Features of SCZ:

Features do not mean diagnosis (not all features are in the diagnostic criteria); The first three are present in the criteria. They are essential and very important because they're what makes us human beings.

1. Positive symptoms
2. Negative symptoms
3. Disorganization Speech e.g circumtiality or Behaviour for e.g irritability disinhibition.
4. Cognitive deficits not a diagnostic criteria but is imp because cognitive function is what makes us human.
5. Mood symptoms not in the diagnostic criteria so we don't mix SCZ with mood disorders + it is common with SCZ.
6. Function anything that doesn't affect patient's function is not a psychiatric disorder.

Causes of SCZ:

Unknown

Theories:

Stress-Diathesis Model:

- Not a strong theory.
- Symptoms: Vulnerability due to many factors e.g genetics and social.
- Biological and psychosocial and environmental.

Genetic Factors:

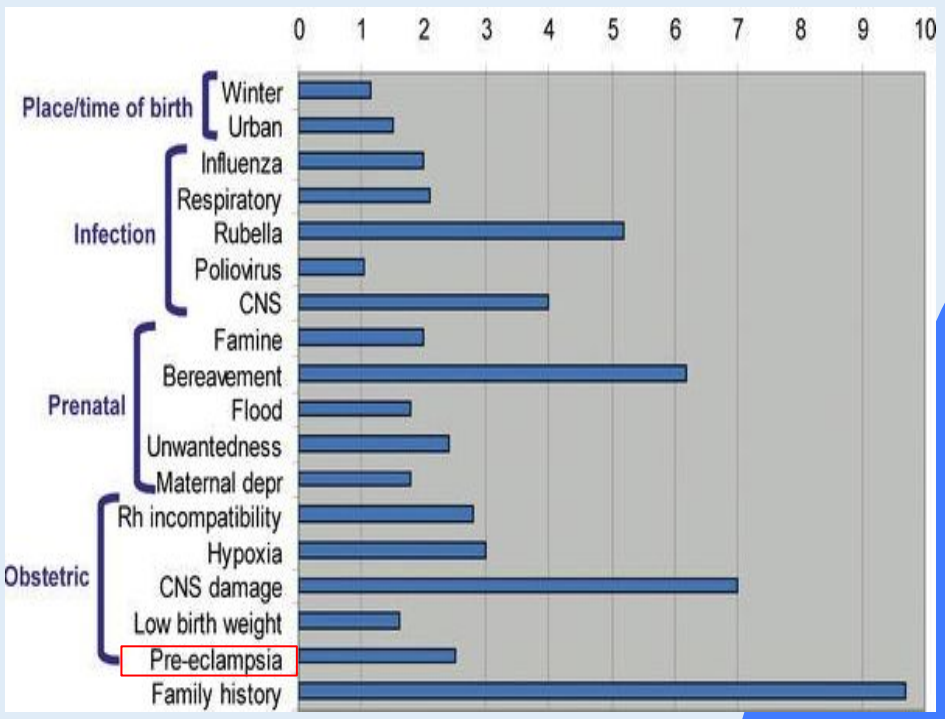
- Genetic factors have no role in treatment. Role is only in 1. Counseling and prevention 2. Early detection.
- Family studies.
- Twin studies.Identical twins is the most common genetic risk follow by child with both parents with SCZ.
- Chromosome.

Neurobiology:

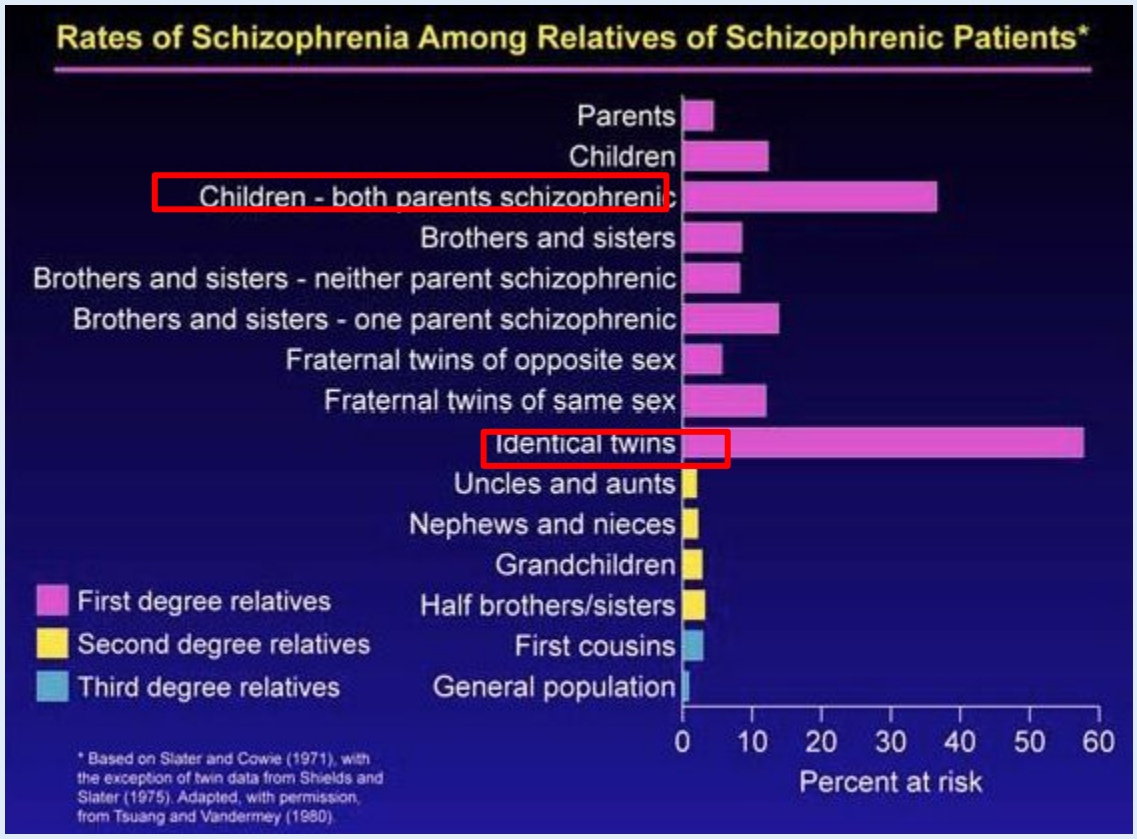
- Strongest theory it is a neurotransmitter problem
- Treatment depends on this theory
- Areas of the brain
- Dopamine
- Other

Neuropathology:

- Not strong it is changes in brain structure
- Structures or connections
- Limbic system
- Basal ganglia
- Cerebellum



Notes:
The most important risk factor is family history.
Other are not that reliable for example winter. Most people get married in the summer and have babies at winter.
Another example is that the study related to rubella was done when there was a rubella outbreak so it's unreliable.



Schizophrenia: genes plus stressors

Schizophrenia is mostly caused by various possible combinations of many different genes (which are involved in neurodevelopment, neuronal connectivity and synaptogenesis) plus stressors from the environment conspiring to cause abnormal neurodevelopment. There is also abnormal neurotransmission at glutamate synapses, possibly involving hypofunctional NMDA receptors .

Stephen M The Genetics Of Schizophrenia Converge,Upon,The NMDA Glutamate Receptor, *CNS Spectr*. 2007

TABLE.
Susceptibility Genes for Schizophrenia

Dysbindin	Erb-B4
Neuregulin	FEZ1
DISC-1	MUTED
DAOA	MRDS1
DAAO	BDNF
RGS4	Nur77
COMT	MAO-A
CHRNA7	Spinophylin
GAD1	Calcyon
GRM3	Tyrosine hydroxylase
PPP3CC	$Dopamine_2$ receptor
PRODH2	$Dopamine_3$ receptor
AKT1	

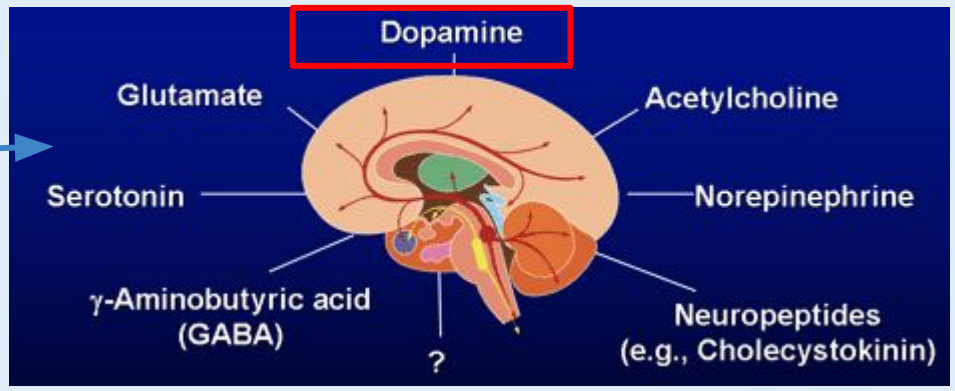
DISC-1=disrupted in schizophrenia-1; DAOA=D-amino acid oxidase activator (G72/G30); DAAO=D-amino acid oxidase; RGS4=regulator of G-protein signalling 4; COMT=catechol O methyl transferase; CHRNA7=α-7 nictonic cholinergic receptor; GAD1=glutamic acid decarboxylase 1; GRM3=glutamate receptor, metabotropic 3; BDNF=brain derived neurotrophic factor; MAO-A=monoamine oxidase A.

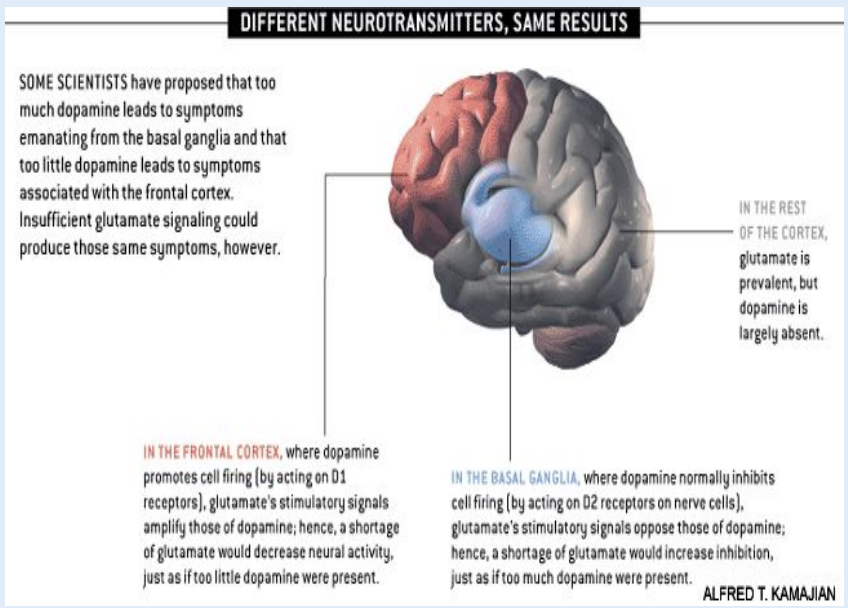
Stahl SM. *CNS Spectr*. Vol 12, No 8. 2007.

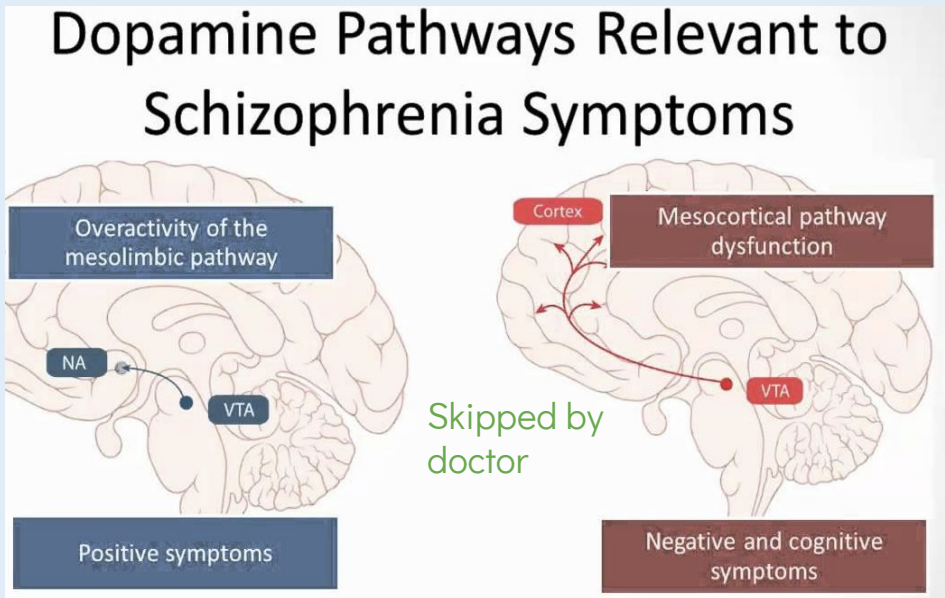
Neurotransmitter Systems Implicated in Schizophrenia

Don't skip very imp to understand

Schizophrenia probably involves multiple neurotransmitter system abnormalities.







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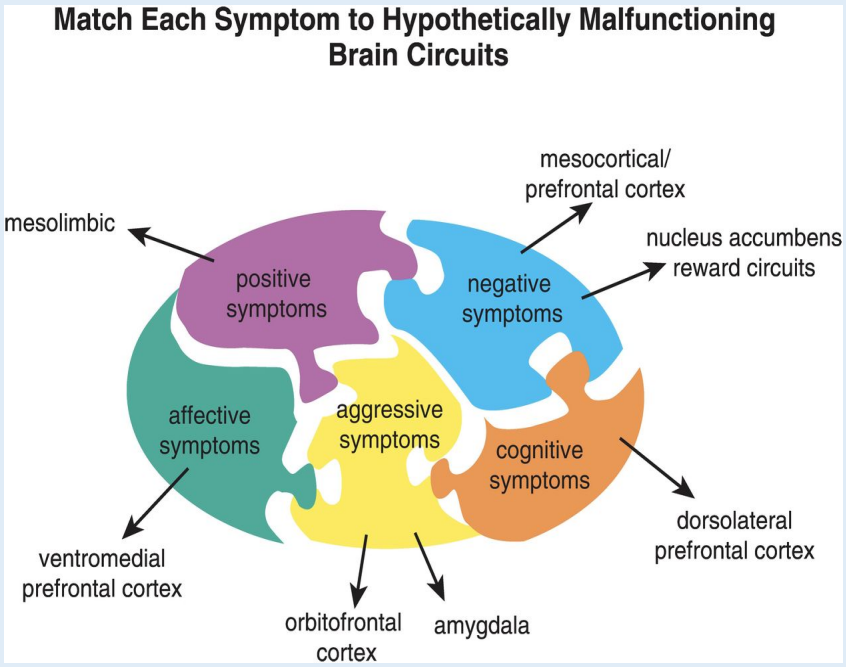

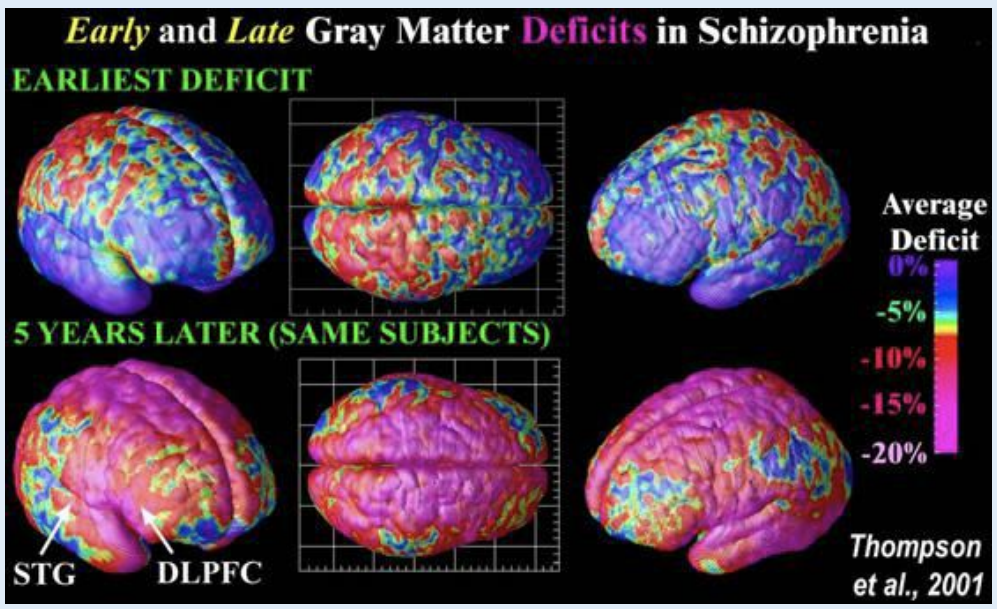

Early loss of cognitive function most in 1st 5 years then plateau. i Start RX as early as possible.
In the picture the pink means loss of cognitive see the picture below in 5 years almost all gone.

What else?

1. Psychoneuroimmunology
2. Psychoneuroendocrinology
3. Psychosocial factors (evidence - high expressed emotion family (EE)

Cognitive Deficits in Schizophrenia

Multiple Mechanisms for Cognitive Dysfunction in Schizophrenia:

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1. Dopamine
2. 5-HT
3. Acetylcholine
4. Neurodegenerative
5. Abnormal connectivity

Cognitive Deficits Predict Functional Outcomes:

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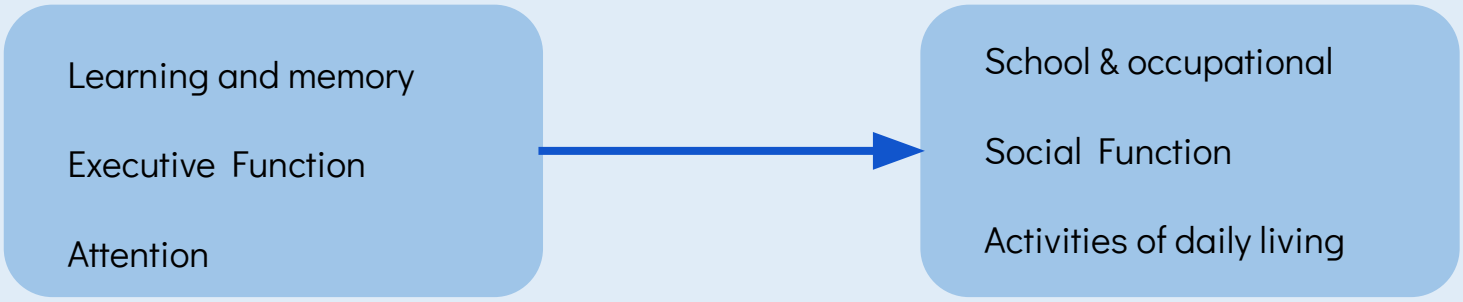


DSM-5 criteria for Schizophrenia	
A	≥ two characteristic symptoms for one month, at least one of them is (1),(2) or (3) 1. Delusions which is a false fixed belief 2. Hallucinations Can be all 5 sense. Absence of stimuli (as opposed to illusions) 3. Disorganized speech (frequent derailment or incoherence) 4. Grossly disorganized or catatonic behavior 5. Negative symptoms (diminished emotional expression or lack of drive (avolition)
B	Social, Occupation or self-care dysfunction
C	Duration of at least *6 months* of disturbance (include at least one month of active symptoms that meet Criterion A; in addition of periods of prodromal and residual symptoms).
D	Schizoaffective & mood disorder *exclusion*
E	The disturbance is not due to Substance or another medical condition.
F	If there is history of autism spectrum disorder or a communication disorder of childhood onset, schizophrenia diagnosis is made only if delusion or hallucinations plus other criteria are present.

Issue of illusion> Interpretation

Issue of hallucination> Stimuli.

Psychosis: impaired reality testing.

Clinical Features of Schizophrenia

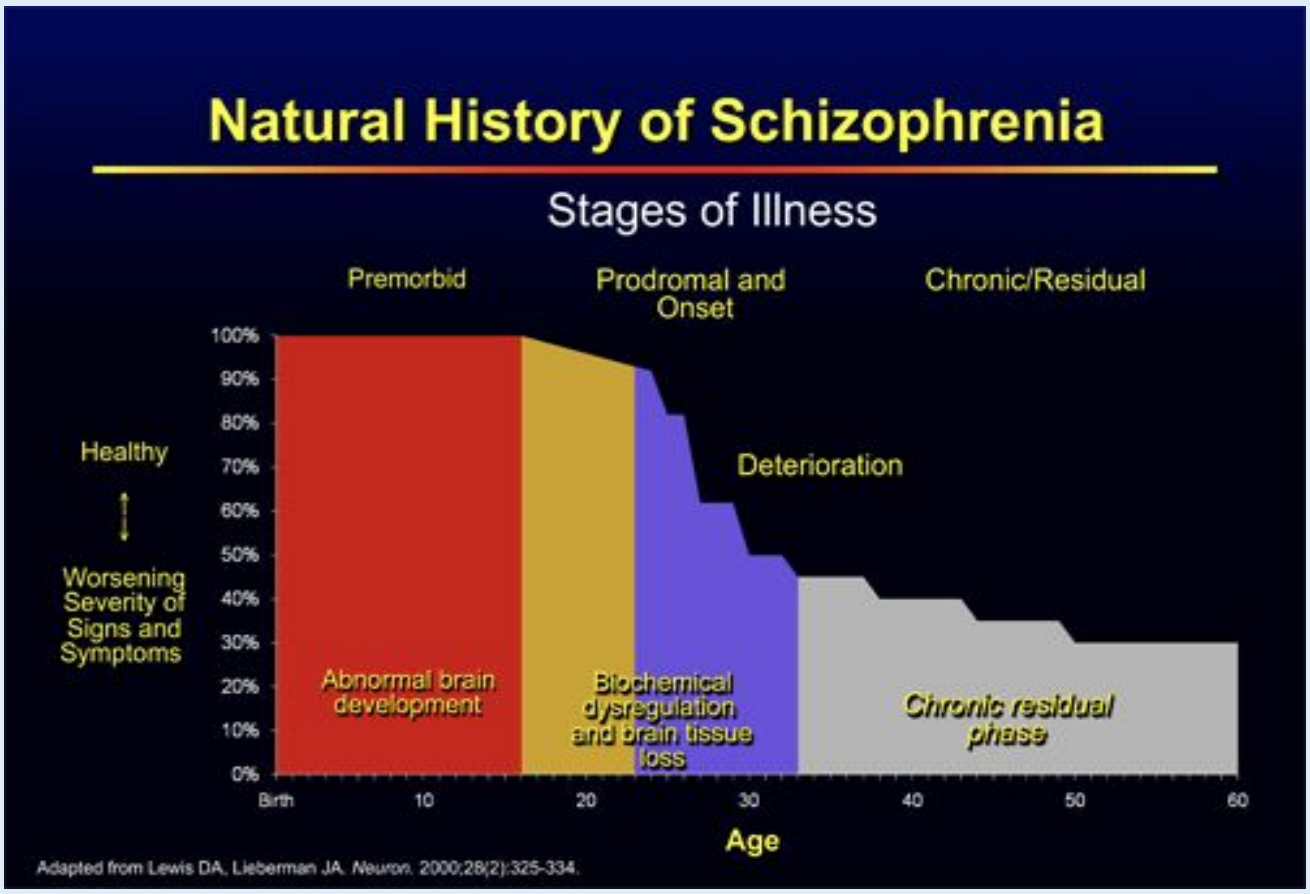
- No single clinical sign or symptom is **pathognomonic** for schizophrenia
- Patient's history & mental status examination are essential for diagnosis.
- Premorbid history includes schizoid or schizotypal personalities, few friends & exclusion of social activities.
- **Prodromal features** include obsessive compulsive behaviors , attenuated positive psychotic features

Mental Status Examination

- Appearance & behavior
- Mood, feelings & affect
- Perceptual disturbances
- Thought: 1. Thought content
  2. Form of thought
  3. Thought process (thought blocking, poverty of thought content, poor abstraction, perseveration)
- Impulsiveness, violence, suicide & homicide
- Cognitive functioning
- Poor insight and judgment

Course

- Acute exacerbation with increased residual impairment
- Full recovery: very rare
- Longitudinal course: downhill



Prognosis very imp

Good prognostic factors	Poor prognostic factors
1. Late age of onset 2. Acute onset 3. Obvious precipitating factors 4. Presence of mood component 5. . Good response to Tx 6. Good supportive system presence of positive symptoms	1. Young age of onset 2. Insidious onset 3. Lack of P.F. 4. Multiple relapses 5. Low IQ 6. Poor premorbid personality 7. Negative symptom 8. Positive family history

Differential Diagnosis

Primary Psychiatric disorders:

- Schizophreniform disorder
- Brief psychotic disorder
- Delusional disorder
- Schizoaffective disorder
- Mood disorders
- Personality disorders (schizoid, schizotypal & borderline personality)
- Factitious disorder
- Malingering

Secondary Psychiatric disorders:

- Substance-induced disorders.

  There needs to be:

1. Evidence of substance use.
2. Symptoms get better within 1 month.
3. Complete cure within 6 months.

- Psychotic disorders due to another medical disorder
  - Epilepsy (complex partial).
  - CNS diseases.
  - Trauma.
  - Others.

Other Psychotic disorders vs. SCZ:

1. Brief psychotic disorder

- Meets diagnostic criteria for SCZ but duration less than 4 weeks.

2. Schizophreniform disorder

- Meets diagnostic criteria for SCZ but duration more than 4 weeks less than 6 month.

3. Schizoaffective

- Has to have all of the following three:

1. Psychosis more than 6 months (which meet the diagnostic criteria of SCZ).
2. Has affective mood disorder (bipolar or depression) for at least 20-25% duration of psychosis.
3. Two week of pure psychosis (w/out mood symptoms)

4. Delusional disorder

- Main component is delusion (+/- mild hallucinations that are RELATED to the delusion topic)

5. DIP

- Has to have these two :

1. Evidence of drug use (WEED)
2. Symptoms improve within 1 month from stopping the drug

6. MIP

- A medical cause causing psychosis for e.g MS
- MIP has a longer duration unlike medical induced delirium which comes acutely

7. MDD with Psychotic features

- MDD is major depressive disorder.
- Mood signs and symptoms appears first then psychosis.
- If psychosis occured first then it is a psychotic disorder such as brief psychotic, scz and etc.

NOTE: SCZ vs Mania

- Duration: SCZ 6 months - Mania 1 week
- The sxs mood then psych or psych then mood

Antipsychotic Medications

Conventional Antipsychotics

Chlorpromazine
Fluphenazine
Haloperidol
Loxapine
Molindone
Perphenazine
Pimozide
Prochlorperazine
Thiothixene
Thioridazine
Trifluoperazine

- Know extrapyramidal symptoms and neuroleptic syndrome
- Know meds work on dopamine

Atypical Antipsychotics

Aripiprazole
Clozapine
Olanzapine
Paliperidone
Quetiapine
Risperidone
Ziprasidone

NOTES: Clozapine is used as a 3rd line due to serious side effects:

1. Agranulocytosis
2. Myocarditis
3. PE
4. Seizure
5. BBefore

Before starting do CBBC, ECG, CXR. No need for EEG

- Typical antipsychotics have more extrapyramidal side effects.
- Atypical antipsychotics have less extrapyramidal but more metabolic syndrome side effects such as obesity.

DSM-5 Diagnostic Criteria for Schizoaffective disorder:

- An uninterrupted period of illness that includes either a major depressive disorder or a manic episode along with at least two active symptoms of schizophrenia (hallucinations, delusions, disorganized speech, severely disorganized or catatonic behaviors, negative symptoms like decreased emotional expression or movement)
- Delusions or hallucinations occur at least two weeks without major depressive or manic symptoms at some time during the illness.
- The major mood symptoms occur for most of the duration of the illness.
- The illness is not the result of a medical condition or the effects of alcohol, other drugs of abuse, or a medication.

Substance-Induced psychiatric Disorder

- Potentially severe, usually temporary.
- Context of substances of abuse, medications, or toxins of any of the 10 classes of substances.
- Clinically significant presentation of a secondary psychiatric disorder.
- Evidence in history, PE, MSE and labs of: 1. Develop during or within 1 month of use 2. Capable of producing mental disorder seen
- Not an independent mental disorder: 1. Preceded onset of use 2. Persists for substantial time after use (more that a month after off of substance use)

Treatment

Indications for hospitalization

1. Diagnostic purpose
2. Patient & other's safety
3. Initiating or stabilizing medications
4. Establishing an effective association between patient & community supportive systems

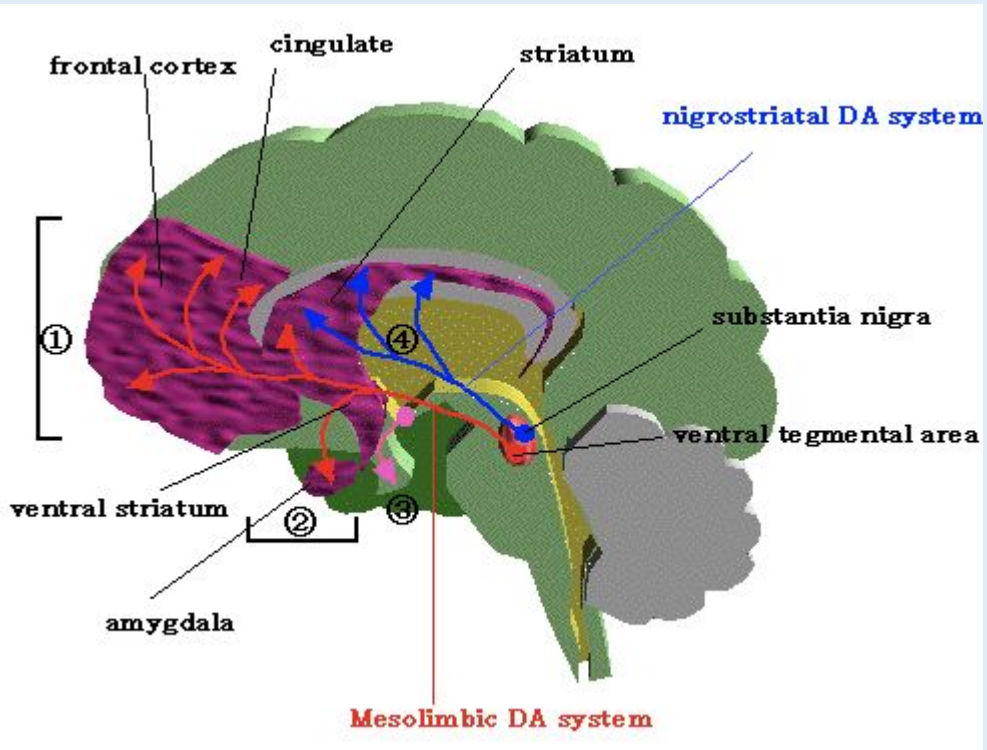
Biological therapies

- Antipsychotic medications are the mainstay of the treatment of schizophrenia. Generally, they are remarkably safe.
- Two major classes:

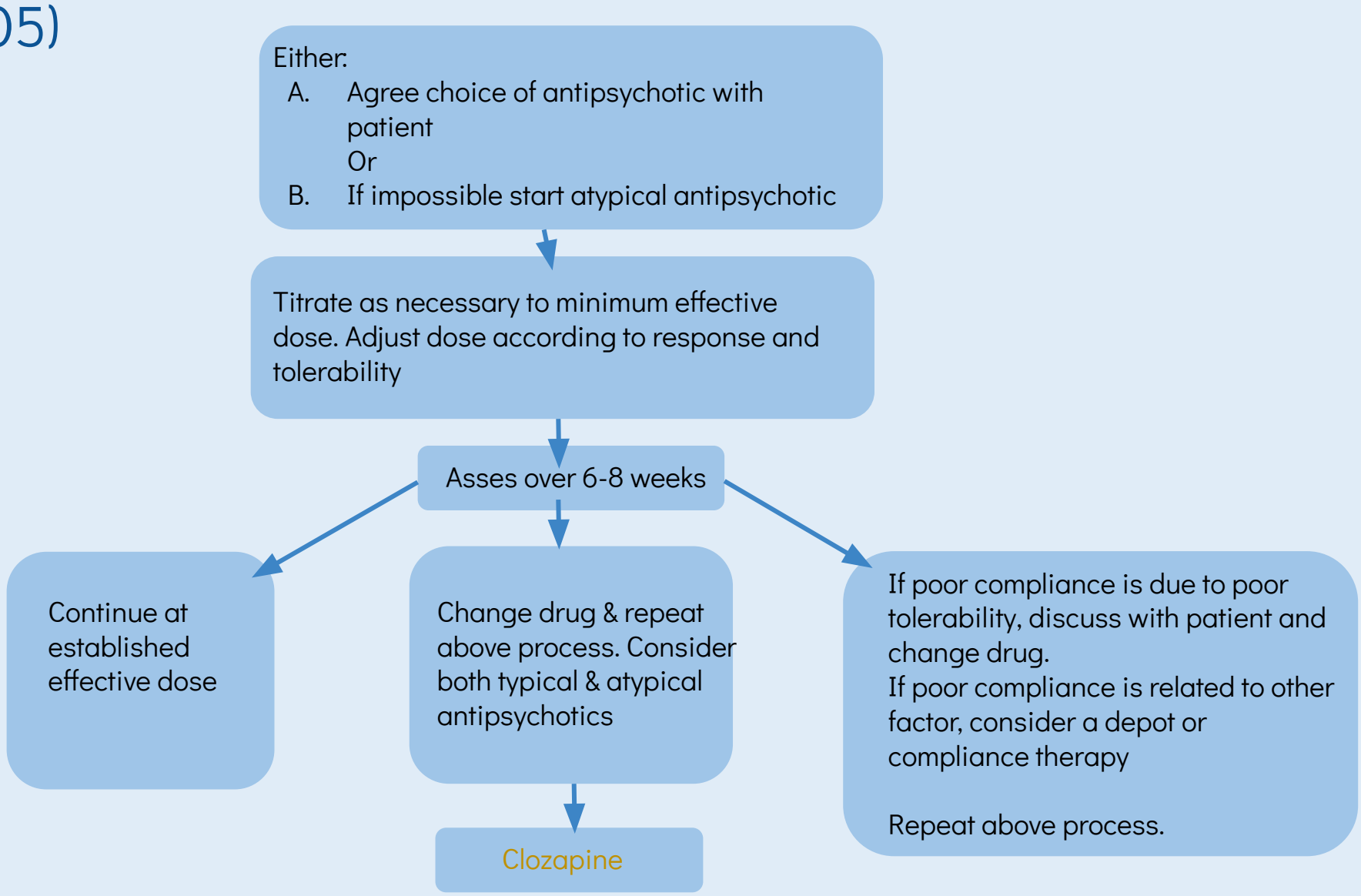
1. Dopamine receptor antagonists (haloperidol, chlorpromazine)
2. Serotonin-dopamine receptor antagonists (Risperidone, clozapine, olanzapine).

- Depot forms of antipsychotics eg. Risperidone Consta is indicated for poorly compliant patients.
- Electroconvulsive therapy (ECT) for catatonic or poorly responding patients to medications

Antipsychotics and dopamine system



Pharmacological Treatment Algorithm Adapted from the Maudsley prescribing Guidelines (Taylor et al, 2005)



Receptor Blockade & Antipsychotic Side Effects

Receptor Type	Side Effects
D2 (dopamine)	EPS, prolactin elevation
M1 (Muscarine)	Cognitive deficits, dry mouth, constipation, increased heart rate, urinary retention, blurred vision.
H1 (Histamine)	Sedation, weight gain, dizziness
α1	Hypotension
5-HT2A (5-HT= serotonin)	Anti-EPS (extrapyramidal symptoms)
5-HT 2C (5-HT= serotonin)	Satiety blockade

First generation antipsychotics

Extrapyramidal effects:
Dystonia
Pseudoparkinsonism
Akathisia
Tardive dyskinesia

Sedation

Hyperprolactinemia

Reduced seizure threshold

Postural hypotension

Anticholinergic effects:
Blurred vision
Dry Mouth
Urinary retention

Neuroleptic malignant syndrome

Weight gain

Sexual dysfunction

Cardiotoxicity (including prolonged QTc)

Second generation antipsychotics

Olanzapine:
Weight gain
Sedation
Glucose intolerance and frank
Diabetes Mellitus
Hypotension

Risperidone:
Hyperprolactinemia
Hypotension
EPS at higher doses
Sexual dysfunction

Amisulpiride:
Hyperprolactinemia
Innsomnia
Extrapyramidal effects

Quetiapine:
Hypotension
Dyspepsia
Drowsiness

Clozapine

Sedation	Glucose intolerance and diabetes mellitus
Hypersalivation	
Constipation	Nocturnal enuresis
Reduced seizure threshold	Rare serious side effects: Neutropaenia 3% Agranulocytosis 0.8% Thromboembolism Cardiomyopathy Myocarditis Aspiration pneumonia
Hypo & hypertension	
Tachycardia	
Pyrexia	
Weight gain	

ANTIPSYCHOTICS: SAFETY AND TOLERABILITY[1]

Item	Typical Neuroleptic	Clozapine	Risperidone	Olanzapine	Quetiapine	Ziprasidone	Aripiprazole
EPS	+ to +++	±	± to +++*	± to +*	±	± to +*	± to +
TD	+++	±	± to ++	± (?)	± (?)	± (?)	± (?)
Somnolence	± to +++	+++	±	++	++	±	±
Prolactin	+++	±	+++	±	±	±	±
Weight	± to ++	+++	+	+++	++	±	±
Dyslipidemia	± to +	+++	+	+++	++	±	±
DM	± to +	+++	+	+++	++	±	±
QTc	+	++	+	+	+	++	±
Orthostatic BP↓	± to +++	+++	++	+	++	±	±

*Dose-related.

Key: ±=none-to-minimal; +=mild; ++=moderate; +++=marked; ?=no data, compared to placebo rates.

EPS=extrapyramidal symptoms; TD=tardive dyskinesia; DM=diabetes mellitus; QTc=corrected Q-T interval; BP=blood pressure.

Side effects of Atypical Antipsychotics

Shift in risk perception

Prior Safety Concerns:

1. Neurological side effects (EPS +TD)
2. Weight gain
3. Insulin resistance
4. Hyperglycemia
5. CVD
6. Hyperlipidemia
7. QTc

Current Safety Concerns:

1. Weight gain
2. EPS
3. Diabetes
4. Insulin resistance
5. QTC
6. Hyperglycemia
7. CVD
8. Dyslipidemia

Drug	Weight Gain	Risk for Diabetes	Worsening Lipid Profile
Clozapine	+++	+	+
Olanzapine	+++	+	+
Risperidone	++	D	D
Quetiapine	++	D	D
Aripiprazole *	+/-	-	-
Ziprasidone*	+/-	-	-

- += increase effect , -= no effect, D= discrepant results , *=newer drugs with limited long-term data

Neuroleptic Malignant Syndrome (NMS):

- Uncommon but potentially fatal complication of antipsychotic.
- Typically occurs soon after an antipsychotic is started or dose is increased but may occur late.
- Risk factors include depot antipsychotics, intramuscular administration, rapid increase in dose of antipsychotic, high doses of antipsychotics, dehydration, malnutrition, iron deficiency, underlying brain abnormalities and agitation.
- Diagnostic triad- Fever> or equal to 38 C (100.4 F, muscle rigidity, mental status changes.
- Autonomic instability and hyperthermia are the major causes of morbidity and mortality .
- Common lab abnormalities include high CPK or myoglobinuria, high WBC, metabolic acidosis.
- Ensure other medical causes have been excluded
- Management includes discontinuing antipsychotics, lithium and dopamine blocking antiemetic agents and providing supportive care, most commonly in an ICU. Although older references recommend use of bromocriptine or dantrolene, more recent references show no advantage for these agents

Psychosocial therapies

- Social skills training
- Family oriented therapies Group therapy
- Individual psychotherapy
- Assertive community treatment
- Vocational therapy

Manual of Basic Psychiatry by Prof. Al-Sughayir

Ahmed is a 28-year-old single man was brought by his father to Emergency Department with 7 months progressive history of: 1. Talking to himself with giggling and grimacing. 2. Staring at the roof of his room. 3.Over-suspiciousness (e.g. his family may poison his food). & 4.Agitation.
Past history: Several psychiatric hospitalizations because of disturbed behavior and perception (hearing non-existent distressing voices commenting on his action).

Fahad, what do you know about **psychotic**

Psychotic Disorders are mental illnesses characterized by gross impairment in reality testing and personal functioning as evidenced by disturbances in thinking (delusions), perception (hallucinations), or behavior (e.g. violence). Examples: schizophrenia, severe mood disorders, delusional disorders.

What are the ***clinical features*** of psychotic disorders?

Defects in	Examples
Behavior	Abnormal movements/posture/smile/laughter
Perception	Hallucinations
Thinking	Delusions/concrete thinking/loose association
Insight	Denial of mental illness
Judgment	Reckless/dangerous decisions

Not all mental functions are defected in all patients.

Great! that means you have reviewed (signs & symptoms). Pay more attention to ***delusions*** and ***hallucinations*** (the main signs of psychosis).

Fahad, tell us about **DDx** of psychotic disorders

Well, there are organic & functional causes of psychosis. I can simplify them in the table below:

Psychosis due to medical/organic causes:

Delirium/dementia/ CNS infections / frontal lobe pathology / temporal lobe epilepsy.
Medications (e.g. steroids, bromocriptine, L-dopa).
Autoimmune D. (e.g. SLE).
Substance-induced psychosis e.g.: stimulants, cannabis, alcohol…e.t.c
Features: like functional psychosis (but hallucinations; visual > auditory + cognitive impairment)

Functional psychosis:

1.Brief Psychotic disorder.
2. Schizophreniform disorder.
3. Delusional disorders.
4. Schizophrenia.
5. Schizoaffective

Personality Disorders:

Paranoid, schizoid, schizotypal, and borderline personality disorders may *co-occur* with psychotic disorders.

Brief Psychotic Disorder: an acute and transient psychotic condition that lasts ≥ 1 day but ≤1 month and not induced by an organic cause. Common features include paranoid delusions, hallucinations, emotional volatility, odd behavior, & screaming. It may be triggered by stress (e.g. death of a relative). Remission is full, and the individual returns to the premorbid level of functioning. It occurs among young (20- 40 years) > old patients. Comorbidity: personality disorders (most commonly, borderline personality disorders, paranoid, schizoid, schizotypal). Patients have a biological or psychological (inadequate coping mechanisms)vulnerability for the development of psychotic symptoms. **DDX**: substance-induced psychosis, manic episode, and PTSD (see later). **Management**: brief hospitalization for protection, evaluation, & antipsychotic treatment; e.g. haloperidol 10 mg or olanzapine 10 mg). ECT for postpartum psychosis. **Prognosis**: varies some patients show no further major psychiatric problems and others progress to mood disorders or schizophrenia.

If the onset is within 4 weeks after delivery, it is called **"Postpartum Psychosis"**. It is uncommon (about 1 in 500 birth). The most common form is affective psychosis (70 %). It begins 2-4 days after delivery. More frequent among primiparous women, those with family history of psychiatric illness and those with previous major psychiatric disorders. The clinical features include disturbed mood, perplexity, excitement, restlessness (or withdrawal), excessive guilt, disturbed thinking and suicidal and infanticidal threats. Schizophrenia-like psychosis occurs in about 25 % of cases who usually remain chronically ill. About 5 % of patients develop delirium. **Treatment:** hospitalization **ECT** (Its rapid effect enables the mother to care for her baby). Drugs: antipsychotics (e.g. risperidone 4 mg).

Schizophreniform Disorder: Similar features to those of brief psychotic disorder but the duration is **>** 1 month & **<** 6 months. **DDX**: manic episode, substance-induced psychosis.
Management: brief hospitalization for protection, evaluation, & antipsychotic treatment; e.g. risperidone 4mg. for 3- to 6-month course. Patients respond to antipsychotic treatment much more rapidly than patients with schizophrenia. **Prognosis**: recurrence is high as well as progression to schizophrenia.

Delusional Disorders: ≥ 1- month systematized delusion(s) (such as being persecuted, followed, loved at a distance, or deceived by spouse). Patients usually do not have prominent or sustained hallucinations. Patients' moods are consistent with the content of their delusions(a patient with grandiose delusions is euphoric). **Types: Persecutory type:** delusions that the person (or someone to whom the person is close) is being malevolently treated in some way**. Grandiose type:** delusions of inflated worth, power, knowledge, identity, or special relationship to a deity or famous person**. Jealous type:** delusions that the individual's sexual partner is unfaithful**. Erotomanic type:** delusions that another person, usually of higher status, is in love with the individual. **Somatic type:** delusions that the person has some physical defect or general medical condition. **Mixed type:** delusions characteristic of more than one of the above types but no one theme predominates. **Unspecified type**. **DDx**; schizophrenia, schizoaffective, mood disorder, organic psychosis. **Treatment**: in-patient or outpatient, antipsychotics oral or depot (long acting IM injections e.g. clopixol 200 mg / month) if patient is not compliant with oral medications. Insight-oriented, supportive, and cognitive therapies are often effective. **Prognosis**: varies depending on many factors (type o delusion, personality, psychosocial stresses, and treatment).

Affective (Mood) Disorders: Manic episode or major depressive episode with psychosis. Note that in mood disorders: hallucination and delusions are mood – congruent and usually develop after the mood disturbance. The course of mood disorders is fluctuating: (repeated episodes of mood disturbance with normal periods in between) see later; module -4; Mood Disorders.

High mood		mania			
Normal mood					
Low mood				depression	

Schizoaffective Disorder: Concurrent presence of mood disturbance (depressive or manic episodes) and psychotic features (delusions or hallucinations, for at least **2** weeks in the absence of prominent mood symptoms during some phase of the illness).

High mood		mania			
Normal mood			**+ 2 w (delusion or hallucinations)**		
Low mood				depression	

DDx; mood disorders, substance-induced psychosis, delusional disorders, and schizophrenia. **Treatment:** an antipsychotic (e.g. olanzapine 10 mg) for psychotic features, mood stabilizer (e.g. sodium valproate 500 mg twice/day), and when depressed an antidepressant (e.g. fluoxetine 20 mg) can be added**. Prognosis:** it has a better prognosis than schizophrenia and a worse prognosis than mood disorders.

Schizophrenia: ≥ **6** months duration of disturbance (including the prodromal and residual phases). ≥ 1month period of psychotic features with **2** out of **5**: delusions/hallucination/disorganized speech (e.g. incoherence) / or disorganized behavior/ catatonic features or negative features (e.g. flat affect). Significant functional impairment (occupational, social, academic...etc.) Exclusion of other psychotic disorders (see above; the differential diagnosis).

There are no specific limited pathognomonic features for schizophrenia. The best starting point is to study simplified descriptions of two variants; the acute and the chronic presentations of schizophrenia.

Epidemiology: Worldwide lifetime prevalence is about 1 %. Worldwide, 2 million new cases appear each year. Incidence is about 20 per 100,000 per year. The lifetime risk of developing schizophrenia is about 1%. Most common between 15 - 35 years. Paranoid type: later onset than other types. Sex ratio is 1: 1
Median age at onset: Males = 28 years, Females = 32 years.

Acute Schizophrenia	Chronic Schizophrenia
Presence of ***active/positive*** features : • Prominent Delusions (paranoid - bizarre) • Prominent Hallucinations: (3rd or 2nd but with derogatory content) • Disorganized thinking and speech. • Disturbed behavior +/- aggression. • Incongruity between affect thinking and behavior.	Presence of ***negative features*** : • Poor self-care and hygiene. • Lack of initiative and ambition. • Social withdrawal. • Poverty of thought and speech. • Restricted or apathetic affect. • Cognitive deficit. • Loose association >>> Word salad. Delusions and hallucinations become less prominent.

What is the cause of schizophrenia?

No single etiological factor is considered causative. The model most often used is that the person who develops schizophrenia has a specific biological vulnerability (or diathesis) that is triggered by stress and leads to emergence of schizophrenic symptoms.

Etiology:

1. **<u>Genetic:</u>**

- Single gene (serotonin receptor on chromosomes 5, D4 dopamine receptor gene on chromosome11).
- Polygenic theory appears to be more consistent with heterogeneity of the presentation of schizophrenia.
- Consanguinity:
  - Incidence in families is higher than in general population.
  - Monozygotic twin concordance rate is greater than dizygotic concordance rate (50 % , 15 % respectively).
  - Adoptive Studies: Test for genetic versus environmental influence by examining rates of schizophrenia in adopted away offspring and of normal parents. (10 % from schizophrenic parents versus 0 % from normal parents).
- Family Studies :

Morbid Risk	Relationship to Schizophrenic
14 %	Child of one schizophrenic parent
46 %	Child of two schizophrenic parents
10 %	Sibling
5 %	Parents

2. **<u>Neurobiological:</u>**

A. Dopamine hypothesis : schizophrenic symptoms are in part a result of increased dopamine activity in mesolimbic & mesocortical pathways.

B. Serotonin hypothesis: abnormal serotonin metabolism in some patients.

C. Disturbed balance between dopamine and serotonin as supported by the new generation of antipsychotics (dopamine-serotonin antagonists).

D. Glutamate hypothesis:

a- Glutamate hyperactivity causes glutamate-induced neuro- toxicity.

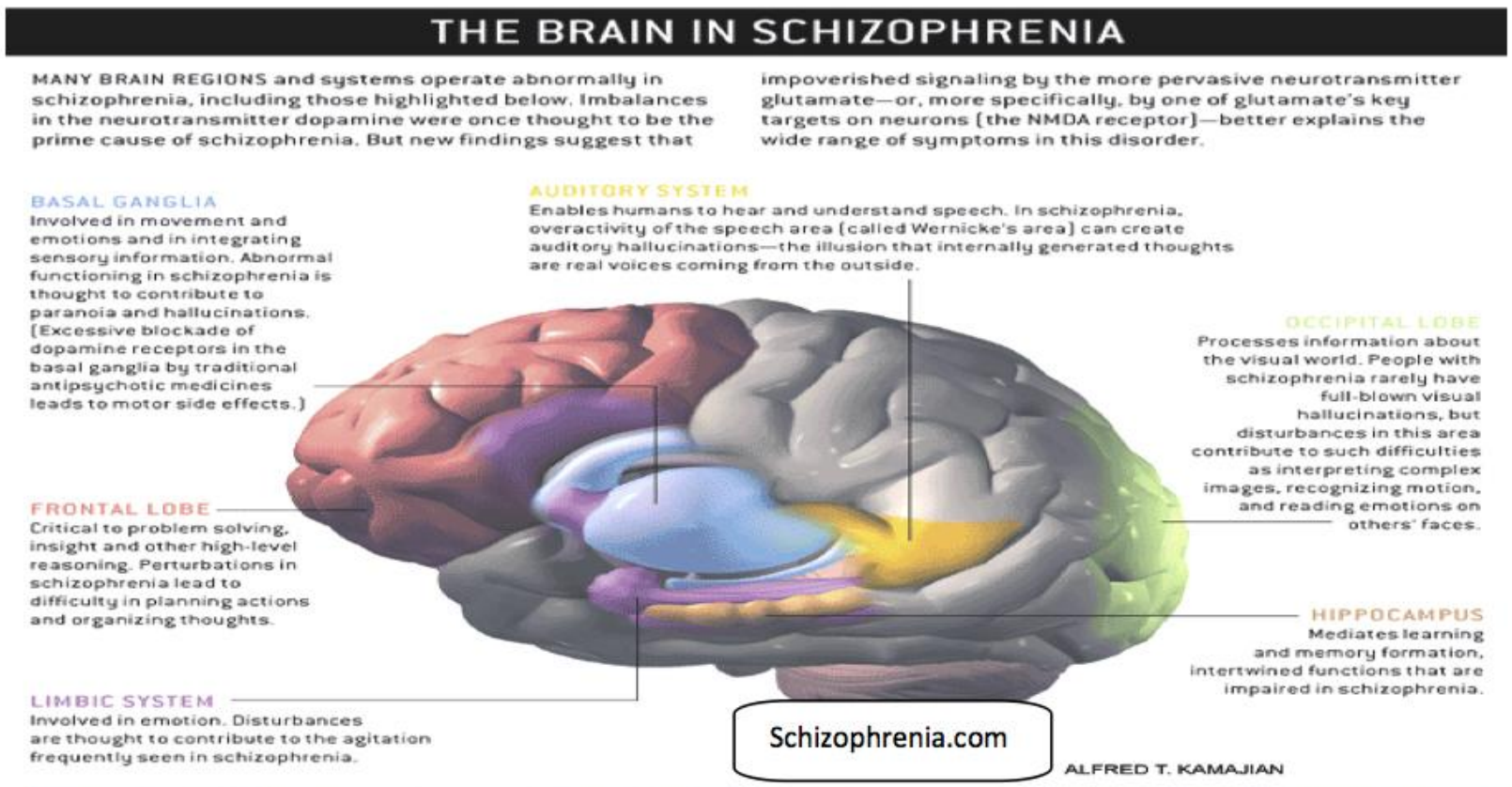
b- Glutamate hypoactivity. It has been implicated because ingestion of phencyclidine, a glutamate antagonist, produces an acute syndrome similar to schizophrenia.

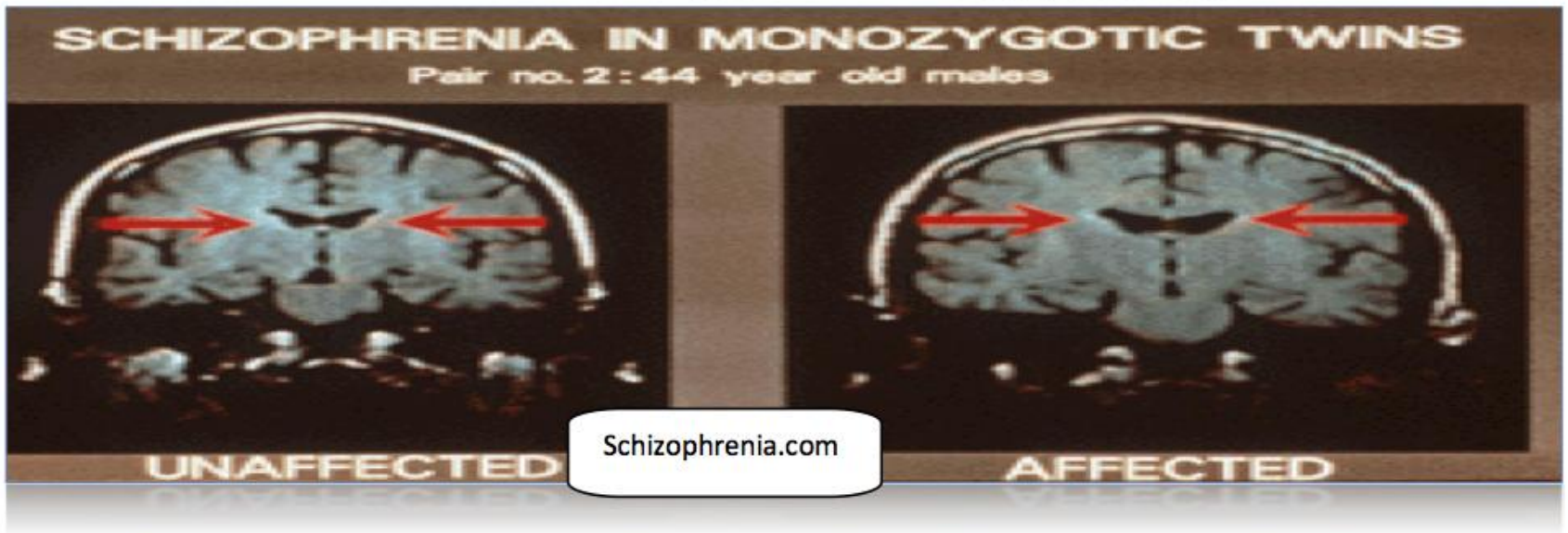
E. GABA hypothesis: the loss of inhibitory GABAergic neurons could lead to the hyperactivity of dopaminergic neurons. Some patients with schizophrenia have a loss of GABAergic neurons in the hippocampus.

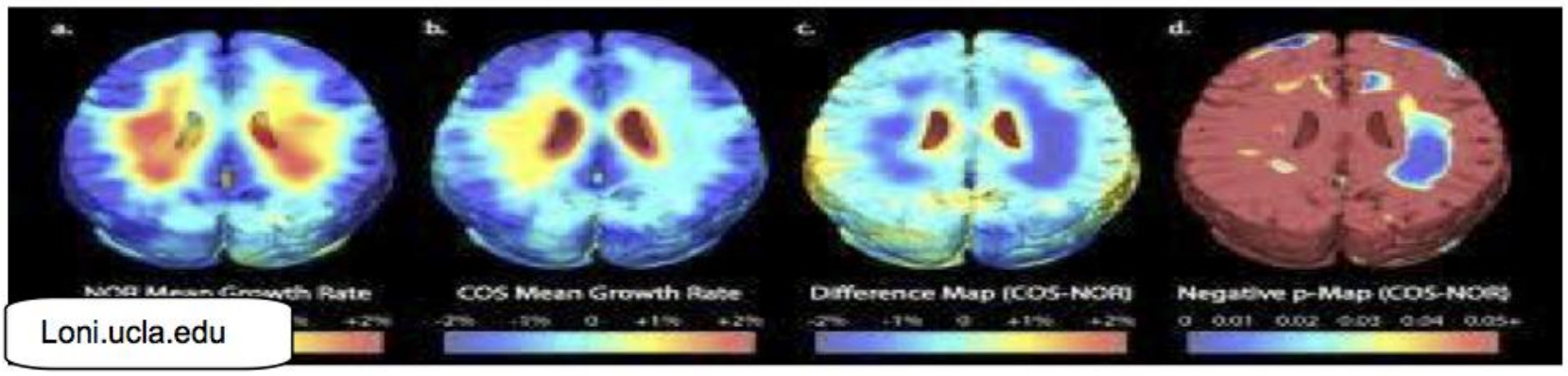
3. **Neuropathology and Neuroimaging :**
   - CT scan studies: Cortical atrophy in 10 - 35 %. Enlargement of the lateral and third ventricles in 10-50%.
   - Findings correlate more with negative features and with cognitive impairments.
   - MRI and PET (Positron Emission Tomography): Abnormal frontal, parietal and temporal lobe structure and metabolism.

4. **Psychosocial and Environmental:**

A. *Life Events*: Life stressors, particularly in the three months before onset, can induce schizophrenia in those who are vulnerable. ***B.*** *High Expressed Emotions (EE)* of the family (critical comments and emotional over-involvement. Patients whose families have high expressed emotions have higher relapse rate than those whose families have low expressed emotions.







Management: Bio-Psycho-Social approach / Multidisciplinary team.

Hospitalization	Medications	Psychosocial
It is usually indicated in the acute phase in order to: - **Clarify diagnosis** (rule out possible organic causes). - **Control the disturbed behavior**. -**Protect patient and / or others** (risk of dangerousness or suicide; close 1:1 nurse observation).  -Give electroconvulsive therapy (ECT) for catatonic type, those with concomitant depression and in resistant cases.	***Antipsychotics:*** e.g. haloperidol (10 - 20 mg) or olanzapine (10 - 20 mg). Adjust the dose based on the response and side effects. See antipsychotics down for further details.	-**Family therapy**, education, and explanation can significantly reduce relapse rate and high-EE family interaction can be diminished. Compliance may also be enhanced. -**Supportive therapy** and counseling. -**Rehabilitation** (Community - based process): 1-Social skill training (e.g. self-care). 2-Illness-management skills (e.g. when to take medication). 3-Vocational rehabilitation (for more stable cases). -Token economy: Positive and negative reinforcement are used to alter patient's unacceptable behavior.

Course and prognosis: Patient may recover from the active psychotic phase but complete return to normal level of functioning is very unusual. The common course is one of acute exacerbations with increasing residual impairment between episodes. The longitudinal course is that of downhill nature (disintegration of personality and deterioration of mental abilities and psychosocial functioning).

Prognostic Factors:

Good Prognostic Factors	Bad Prognostic Factors
• Late onset • Acute onset • Obvious precipitating factors • Good premorbid personality • Presence of mood symptoms (especially depression) • Presence of positive symptoms • Good support (married, stable family)	• Young age at onset • Insidious onset • No precipitating factors • Poor premorbid Personality • Low IQ • Many relapses • No remission in 3 years • Poor compliance • Negative symptoms • Poor support system • Family history of schizophrenia • High EE family

In general; third>>good prognosis, third>>poor prognosis, & third>>Intermediate prognosis.

Community Psychiatry ------- **أسئلة شائعة من أقارب المرضى**

هل يستطيع المريض العقلي الزواج ؟

- يختلف الحال باختلاف ظروف المرضى (شدة الحالة واستجابتها للعلاج و وجود الدعم الأسري والمتابعة الطبية المستمرة) فمن المرضى من يناسبه الزواج ويكون عامل استقرار لحالته ومنهم من قد يزيد حالته شدة.
- يعد الزواج وتكوين أسرة ورعايتها عبئا نفسيا على كثير من المرضى ، وبعضهم ليس لديه دافع قوي لذلك (في حين يبالغ بعض الناس في المجتمع في أن الزواج هو الحل الأمثل للمريض العقلي).

هل يستطيع المريض العقلي أن يتوظف؟

- يختلف الحال باختلاف ظروف المرضى (قدرات المريض وطبيعة أعراضه ونحو ذلك).
- رغم وجود عبء نفسي في العمل إلا إن له آثارا نفسية إيجابية إذا تم اختياره بما يلائم وضع المريض.

هل يعفي المرض العقلي المريض من المسؤولية القانونية و الجنائية؟ وهل يحجر عليه؟

- *ليس كل مرض عقلي يسلب المريض مسؤوليته عن تصرفاته وأخطائه أو يبرر الحجر عليه.*
- لكل حالة وضعها الخاص بها، تنظر فيها الجهات المختصة (لجان شرعية أمنية طبية) مع الاهتمام بالتقييم الطبي النفسي للحالة العقلية للمريض بكافة الوظائف العقلية (خصوصا إدراك حقيقة الواقع والاستبصار بالمرض والقدرة على تمييز الأمور وضبط التصرفات).

رابط فيلم تثقيفي عن الفصام إصدار وزارة الصحة https://www.youtube.com/watch?v=dPaR7Z7DGeA

Antipsychotic Medications (Neuroleptics)

Indications:

A.Functional psychosis: schizophrenia, schizoaffective disorders, schizophreniform disorder, brief psychotic disorder, mania, postpartum psychosis, psychosis with depressed mood, and delusional disorders.

B.**Organic psychosis**: psychosis induced by medications, substance abuse, delirium, and dementia.

C.Violence/aggression, agitation, and excitement.

First Generation Antipsychotics [FGAs] (Also called conventional, typical, or traditional antipsychotics).

Chlorpromazine (Largactil) was the first drug (in the mid-1950s) that significantly reduced symptoms of psychosis. Then, other drugs with similar clinical effects were introduced; haloperidol, sulpiride, ...).

Mechanism of action;, high blockade of dopamine receptors type 2 (D2).

Therapuetic effect: in the **mesolimbic** pathwayD2 blockade reduces *active* psychotic features. This may take up to 6 weeks to appear).

Adverse effects: (may appear within hours - weeks)

 # Antidopaminergic *S/E; 1.* In Nigrostriatal tract >>> EPSE (because of the resulting hypercholinergic effect, which manifests in skeletal muscle spasms. These side effects, in contrast to Parkinson's disease, are better treated with anticholinergic medications rather than dopaminergic drugs). **2. In Tuberoinfundibular tract >>> hyperprolactinemia** (dopamine inhibits prolactin release from the anterior pituitary. Thus, antidopaminergics induce excessive prolactin secretion, which lead to gynecomastia and amenorrhea. Some gynecologists prescribe dopaminergic medications (e.g. bromocriptine) to reverse amenorrhea in psychotic females, which may aggravate their psychosis). **3. In Mesocortical tract >>>** reduced concentration, low initiation, lack of motivation, and restricted affect.

 # Anticholinergic S/E; dry mouth, constipation, urinary retention, poor erection, blurred vision, and precipitation of closed-angle glaucoma.

 # Antiadrenergic S/E; postural hypotension and inhibition o ejaculation.

 # Antihistaminergic S/E; sedation and weight gain.

Second Generation Antipsychotics [SGAs] (Also called novel or atypical antipsychotics, serotonin-dopamine antagonists). **SGAs**; olanzapine (zyprexa), quetiapine (seroquel), clozapine (leponex), risperidone (risperdal), & paliperidone (invega).

Mechanism of action; blockade of dopamine and serotonin (5HT A_2) receptors.

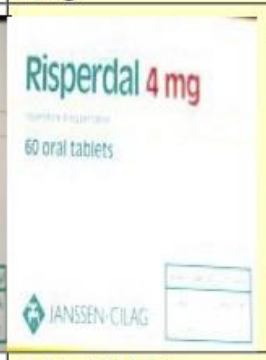
Therapeutic effects; More specific for the mesolimbic than nigrostriatal dopamine system >>> less EPSE. In the mesocortical tract blockade of 5HT A_2 enhances dopamine function (5HT inhibits dopamine) >>> improve negative symptoms of psychosis: low initiation, lack of motivation, and restricted affect. They improve *both positive and negative* symptoms of psychosis and can help some *resistant* cases.

Adverse effects; Less EPSE, antiadrenergic, anticholinergic S/E. but there is a high risk of metabolic syndrome (see below).

Third Generation Antipsychotics

Dopamine System Stabilizers [DSS].

Aripiprazole: Unlike the SDAs, it is not a D_2 antagonist, but is a partial D_2 agonist; in mesolimbic it competes with dopamine (functional antagonism) >>> less active symptoms. However, in the mesocortical tract it acts like the SDAs. It does not increase weight and is usually non-sedating but somnolence may occur in some patients. Side effects include agitation, anxiety , headache, insomnia, dyspepsia, , and nausea. Seizures have been reported. Prolactin elevation does not typically occur. Aripiprazole does not cause significant QT_c interval changes.

Medication / Side effects	First GAs	Second GAs				Third GAs
	Haloperidol (Haldol) 10 mg	Risperidone (Risperdal) 4 mg	Olanzapine (Zeprexa) 10 mg	Quetiapine (Seroquel) 200mg	Clozapine (Leponex) 400mg	Aripiprazole (Abilify) 15 mg
						
	18 SR / 25 tablets	582 SR / 60 tablets	314 SR / 28 tablets	665 SR/ 30 tablets	156 SR/ 50 tablets	525 SR / 28 tablets
EPSE	++ to +++	0 to ++ (> 6 mg)	0	0	0	0 +
Sedation	+ to + + +	+	+	+	+ + +	0
Weight gain	0 to ++	+	+++	+	+++	0
Prolactin increase	+ + to + ++	+ to + +	0 to +	0	0 to +	0
Orthostatic hypotension	+ to + + +	+	+	0	+ to + + +	0
Agranulocytosis	0	0	0	0	+ + +	0
Prolonged QT In ECG	0 to + +	+	0	+	0	0
Seizures	+	0	+	0	+++	+
Anticholinergic S/Es	++ to +++	0	+	0	+++	0
Advantages	Effective on active symptoms Cheap.	Effective on negative features	Effective on negative features	No hyperprolactinemia	Effective in resistant cases.	No wt gain No hyperprolactinemia
Disadvantages	Severe EPSE + Many other S/Es	Hyperprolactinemia + metabolic syndrome	Metabolic syndrome	Metabolic syndrome	Metabolic syndrome + Agranulocytosis (check WBCs). + High risk of seizures	Insomnia + Agitation

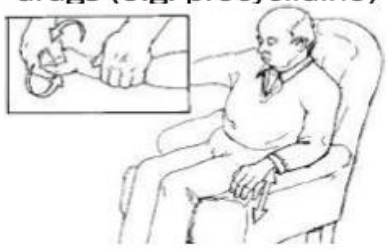
DEPOT (SLOW RELEASE) ANTIPSYCHOTICS: These are long-acting antipsychotic drugs, given as deep intramuscular injections to patients who improve with drugs but cannot be relied on to take them regularly by mouth (i.e.poor compliance). Such patients usually suffer from either; chronic schizophrenia, delusional disorders, or schizoaffective disorder. A test dose is usually given (¼ - ½ the dose) to check patient's tolerability. Depot injections are released slowly in 1 – 8 weeks.

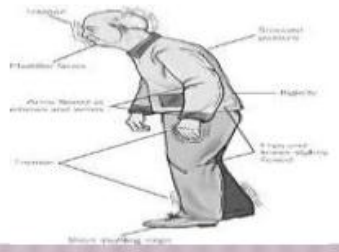
- Risperdal consta:25-50 mg./2weeks.
-Zuclopenthixol decanoate (Clopixol) : 200 – 600 mg. /month.
- Flupenthixol decanoate (Depixol – Fluanxol): e.g. 20 – 100 mg / month.
-Haloperidol decanoate (Haldol) : 200 – 400 mg. / month.
-Fluphenazine decanoate (Anatensol – Modecate): e.g. 25 – 75 mg / month.

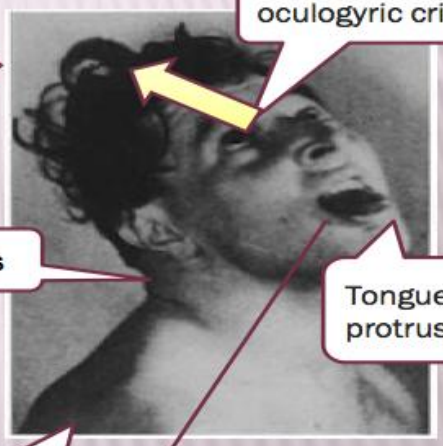
Extra-Pyramidal Side Effects (EPSE)

1- Acute dystonia: appears within days after Rx. Severe painful spasm of neck muscles (torticollis), ocular muscles (oculogyric crisis) muscles of the back (opisthotonus) and tongue protrusion. Treated with anticholinergic drugs (e.g. procyclidine 5 – 10 mg IM or P.O.).

2- Parkinsonism: appears within weeks after treatment, its features: stooped posture, akinesia, muscle rigidity, masked face, and coarse tremor. Treated with anticholinergic drugs (e.g. procyclidine)







شد وألم شديد في عضلاتي من المسؤول DA / Ach؟ عالجوني بسرعة.

Extra-Pyramidal Side Effects (EPSE)

3- Akathisia :

Inability to keep still + unpleasant feelings of inner tension.
Appears within days – weeks.
Generally disappears if the dose is reduced.
Benzodiazepine or beta-blockers may help in the treatment, whereas anticholinergics have no therapeutic effect.

4- Rabbit Syndrome:

Rapid perioral tremor.

5- Tardive Dyskinesia:

It occurs in about 10 – 20 % of patients on long-term antipsychotics for several years. *Features*: chewing, sucking or choreo-athetoid movements of the facial neck and hand muscles.

Super-sensitivity of dopamine receptors.

No specific treatment, the only agreed treatment is to discontinue the antipsychotic drug when the patient's state allows this.

ANTIADRENERGIC

Postural hypotension.

Inhibition of ejaculation.

ANTICHOLINERGIC

Blurred vision

Precipitation of closed – angle glaucoma.

Dry mouth.

Constipation

Urinary retention.

Poor erection.

Metabolic syndrome

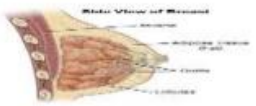
(with atypical Rx)

The syndrome is diagnosed when a patient has three or more of the following five risk factors:

(1) abdominal obesity,
(2) high triglyceride level,
(3) low HDL cholesterol level,
(4) hypertension.
(5) an elevated fasting blood glucose level.

It increases risk of cardiovascular disease and type II diabetes.

Others:



Hyperprolactinemia.

Galactorrhea.

Amenorrhea.

Low libido.

Sedation

(antihistamine effect).

Weight gain.

Toxic Effect:

Neuroleptic Malignant Syndrome (NMS) see Psychiatric Emergencies.

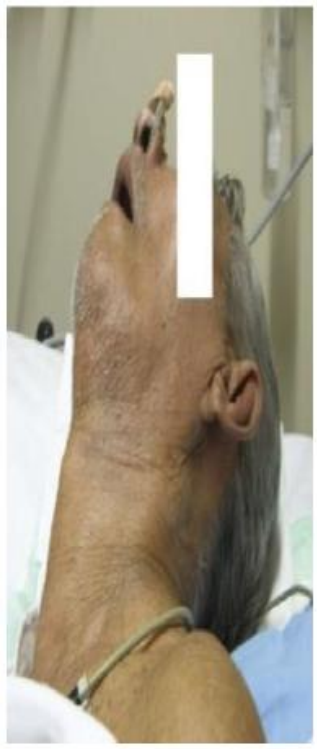
Neuroleptic Malignant Syndrome (NMS): idiosyncratic reaction to antidopaminergics

Features:

Muscle rigidity (trunk, limbs, neck, and throat), sweating and hyperthermia (check temperature), autonomic instability: fluctuating pulse, BP and respiration, akinesia, and clouding consciousness. **Onset** after 2 – 28 days , lasts 5 – 10 days after oral antipsychotics. **Incidence** is higher in males > 40 years.

Laboratory Abnormalities (Secondary Features) Raised CPK (creatinine-phosphokinase), raised potassium, neutrophilia.

Treatment (should be in Medical Intensive Care Unit " MICU"): Stop antipsychotics, monitor vital signs, support respiratory functions, reduce body temperature (cooling), treat secondary infection (if any), rehydrate, bromocriptine (30 – 60 mg / day) to enhance dopaminergic activity, Dantrolene (5 – 10 mg / day) to reduce muscle rigidity, and supportive treatment: dialysis may be required, to reduce the risk of acute renal failure that may result from excessive amounts of myoglobin in the blood following muscle tissue destruction due to excessive prolonged very severe muscle rigidity.



ELECTROCONVULSIVE THERAPY (ECT)

Indications for ECT:

1. Schizophrenia (catatonic, resistant to drugs).
2. Depression:
   - – Depressive disorder with suicidal risk.
   - – Depressive stupor or marked retardation.
   - – Depressive disorder with delusions
   - – Inability to take drugs :
     - First trimester of pregnancy.
     - In the elderly.
     - In physical diseases e.g. renal failure.
3. Post-partum psychosis.
4. Schizoaffective disorder.
5. Mania and mixed affective states.

History and Concept: Patients with concomitant schizophrenia and epilepsy were found to improve in psychosis following repeated fits. It was therefore, thought that there is an antagonism between schizophrenia and epilepsy. In 1938 Cerletti administered an electrically – induced fit to a catatonic vagrant schizophrenic patient who then showed reasonable improvement. Later, anesthesia was introduced and convulsions were modified using muscle relaxing agents.

Psychiatric disorders that may show deterioration or no response to ECT: Phobic disorders, conversion disorder, Primary hypochondriasis (not due to depression), depersonalization disorder.

Precautions: Recent research showed no absolute contraindications to ECT. At one time raised intracranial pressure was considered as the only absolute contraindication to ECT. Remember that not all space occupying lesions produce raised intracranial pressure. **Relative Contraindications: A- To anesthesia and muscle relaxants. B- To ECT itself:** Cardiac infarct in the preceding 3 months (some references extend it to 2 years). Other cardiac diseases including arrhythmias. History of cerebral infarction. Brain tumor.

Mode of Action of ECT: The exact mode of action is unknown. The current hypothesis: the beneficial effect which depends on the cerebral seizures (not on the motor component) is thought to result from neurotransmitter changes probably involving serotonin and noradrenaline transmission.

ECT Preparations: Explanation to the patient (or his caretakers).ECT consent by the patient or his caretaker. Hospital admission for full physical assessment (fitness for anesthesia and ECT). Fasting (midnight).Oxygenation to overcome succinylcholine-induced apnea, to facilitate seizure activity and to reduce memory impairment. Muscle relaxant to reduce the consequent motor effects (severe muscle contraction may lead to bone fracture). Placing a mouth gag in patient's mouth to prevent tongue or lip bites. Machine and electrodes preparations. Decreasing scalp's resistance with jelly or normal saline.

ECT Procedure:

- Bilateral (most commonly used procedure)
  - – One electrode on each side of the head (fronto-temporal position).
  - – It gives a rapid response.
  - – Bi-frontal position can be used; it produces less memory impairment therapeutically ineffective.
- Unilateral:
  - – Both electrodes are placed on the non – dominant side.
  - – It produces less memory impairment but less effective than bilateral.
- ECT is usually given **2 – 3 times a week** with a total of **6 – 12** sessions, according to response and progress. Response begins usually after 2 – 4 sessions. If there is no response after 8 sessions, it is unlikely that more sessions will produce a useful change.
  In depressed patients, antidepressants should be started towards the end of the course of ECT to reduce the risk of relapse.

youtube.com/watch?v=9L2-B-aluCE

Side Effects of ECT: (ECT in general is a safe procedure)

- Headache (due to temporary increase in intracranial pressure).
- Body aches and myalgias (due to muscle contraction)
- Memory impairment (both retrograde and anterograde amnesia).
  - – Duration varies (days – several months).
  - – May be due to neuronal hypoxia during seizure.
- It may induce mania in certain susceptible depressed patients.
- Bone fracture and tongue or lip injury.
- Very rarely death (in patients with cardiovascular disease).

Misconceptions about ECT: Dangerous procedure/causes serious brain damages/involves a high voltage (110 – 220 V) current. Some traditional healers tried 110 V current with some patients assuming that it is the same procedure used by psychiatrist (ECT).

Questions:

Q1: male with schizophrenia on medications, was brought to the emergency department because of 2 days history of muteness, rigid limbs and clouding consciousness. What is the most important initial step to reach the diagnosis?

A. Brain MRI B. Brain CT scan C. Check serum CPK level D. Chest X-Ray

Ans: C

Q2: Which of the following is a good prognostic factor of schizophrenia?

A. Absence of hallucinating . B. Insidious onset c. Early age onset .D. Presence of precipitating factor .

Answer: D

Q3: Which of the following is considered a good prognostic factor of schizophrenia?

A. Gradually onset . B. presence of hallucinating . C. High family emotions . D. Restricted affect

Answer: B

Q4: A patient comes to the emergency because he had low conciseness and muscle rigidity so what is the finding in neuroleptic malignant syndrome?

A . Low K B . Low Na C . High neutrophils D . Low muscle enzyme

Answer: C.

Q5: Which of following statements is applied on schizoaffective more than schizophrenia?

A. Presence of hallucination B. Presence of delusion C. More response to antipsychotics medications

D. Poor prognosis than schizophrenia

Answer: C

Q6: Patient on anti-psychotic medication cannot still on bed or chair and tell you I want to relax but I can't. what is the treatment?

A. Benzodiazepines B. Procyclidine C. Dopamineagonist D. SSRI

Answer: A

Q7: Patient on anti-psychotic medication cannot still on bed or chair and tell you I want to relax but I can't. what is the treatment?

A. Benzodiazepines B. Procyclidine C. Dopamineagonist D. SSRI

Answer: A

Q8: A 40 years old man has a 7 years history of persistent firmly held belief that he has been followed by his boss at work through a hidden camera. But he doesn't have any hallucinations. What is the diagnosis?

A. Delusional disorder B. Schizophrenia C. Bipolar disorder D. Schizoaffective disorder

Answer: A

Questions:

Q9- A 41-year-old woman has a history of continuous auditory hallucinations for 9 months of which she had intermittent episodes of disturbed mood, what is the most likely diagnosis

A. Schizophrenia. B. Schizoaffective disorder. C. Schizophreniform disorder. D. Schizoid personality disorder.

Answer: B

Q10: While evaluating a 26-year-old woman, she indicated that she feels as if she heard voices of her relatives inside her head without their presence. What is the psychopathology?

A. Pseudo- hallucination. B. De-realization. C. Illusions. D. Hallucinations.

Answer: A

Q11: 33 years old woman had one major depressive episode and 2 manic episodes during the last 4 years. During the past 5 years, she was complaining from auditory hallucinations. What's the diagnosis?

A. Bipolar disorders. B. Delusional disorders. C. Schizoaffective disorder. D. Schizophreniform.

Answer: C

Q12: A 44-year-old man has intermittent muscle rigidity, muteness, immobility and unresponsiveness for 9 months. What is the most likely diagnosis?

A. Schizoaffective disorder B. Delusional disorder C. Schizophrenia D. Acute dystonia

Answer: C

Q14: A 44-year-old man has intermittent muscle rigidity, muteness, immobility and unresponsiveness for 9 months. What is the most likely diagnosis?

A. Schizoaffective disorder B. Delusional disorder C. Schizophrenia D. Acute dystonia

Answer: C

Q15: A 67 years old patient being treated for his schizophrenia developed painful muscle spasms and tongue protrusion. What is the mechanism of action of the drug used to manage this case?

A. Dopaminergic B. Anti-dopaminergic C. GABA stimulation D. Anticholinergic

Answer: B

Q16: Patient presented with a case of Resistant psychosis (not responsive to other 2nd gen antipsychotics)

A. Mirtazapine B. fluvoxamine C. Clozapine D. Buspirone

Answer: C

Questions:

Q17: Which part of the brain is function to maintain posture and body balance?

A. cerebellum B. pons C. basal ganglia D. temporal lobe

Answer: A

Q18: Which dopamine pathway includes the reward system,sex drive and pleasure feelings?
A. nigrostriatal tract B. tuberoinfundibular tract C. mesolimbic tract D. mesocortical tract
Answer: C

Q19: Which of the following indicates bad prognosis in schizophrenia ?
A. high expressed emotions family B. presence of mood symptoms C. acute onset D. late onset
Answer: A

Q20: Which of the following is antidopaminergic effect does the nigrostriatal tract affect?
A. reduced concentration B. dry mouth C. muscle spasms D. amenorrhea
Answer: C

Q21: Which of the following antipsychotic drugs causes agranulocytosis ?
A. haloperidol B. clozapine C. olanzapine D. risperidone
Answer: B

Q22: Which of the following antipsychotic drugs is the most to cause weight gain?
A. haloperidol B. Quetiapine C. olanzapine D. risperidone
Answer: C

Q23: 38 years old man diagnosed with schizophrenia, lack of motivation and poor concentration. Which one of the following Dopamine pathways regulate psychological reward?
A. Mesolimbic pathway B. Mesocortical pathway C. Nigrostriatal pathway D. Tuberoinfundibular pathway
Answer: B

Q24: Major neurotransmitter cause dependency and carving?
A. Dopamine B. Serotonin C. GABA D. Substance P
Ans: A

Q25: Which of the following brain structures is responsible for analysis of dimensions?
A. Parietal lobe. B. Occipital lobe. C. Basal ganglia. D. Cerebellum.
Answer: B

Q26: A 24-year-old schizophrenic lady on 20mg/day Haloperidol seen at the emergency department complaining of up-rolling of the eye balls and arching of the back What is the pathophysiology of this condition?
A. High dopamine. B. High serotonin. C. Low dopamine. D. Low serotonin.
Answer: C

1-Q71: What part of the brain maintains consciousness?
A. Occipital lobe B. Reticular formation C. Cerebellum D. Basal ganglia
Answer: B

Q34: A 35.year.old female nurse has borderline personality disorder. She asked you about the receptor responsible for regulation of emotional reaction to stress and impulsive behavior?
A. 5HT1AB. 5HT2A C. 5HT2c D. 5HT3
Answer: A

Questions:

Case 3:

Video link: https://www.youtube.com/watch?v=rwv88mUyd20 From 0:42 - 2:30

Scenario: “A 21 year old guy complaining that his roommate is stalking him and that the FBI hired the roommate to watch him”

Q1:Mention two psychopathological features:

1-Persecutory delusions

2-3rd person hallucinations

3- paranoid delusions

Q2:Mention 2 differential diagnosis:

1-substance-induced psychosis

2-schizophreniform

Q3:Mention one lab test you’ll do for this patient:

Blood drug levels – urine toxicology