433 Teams

PSYCHIATRY

Psychotic Disorders

*All of the information was collected from manual of psychiatry 2014 in addition to Dr.alosaimi lecture.

Psychiatry.team433@gmail.com

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.

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

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

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.

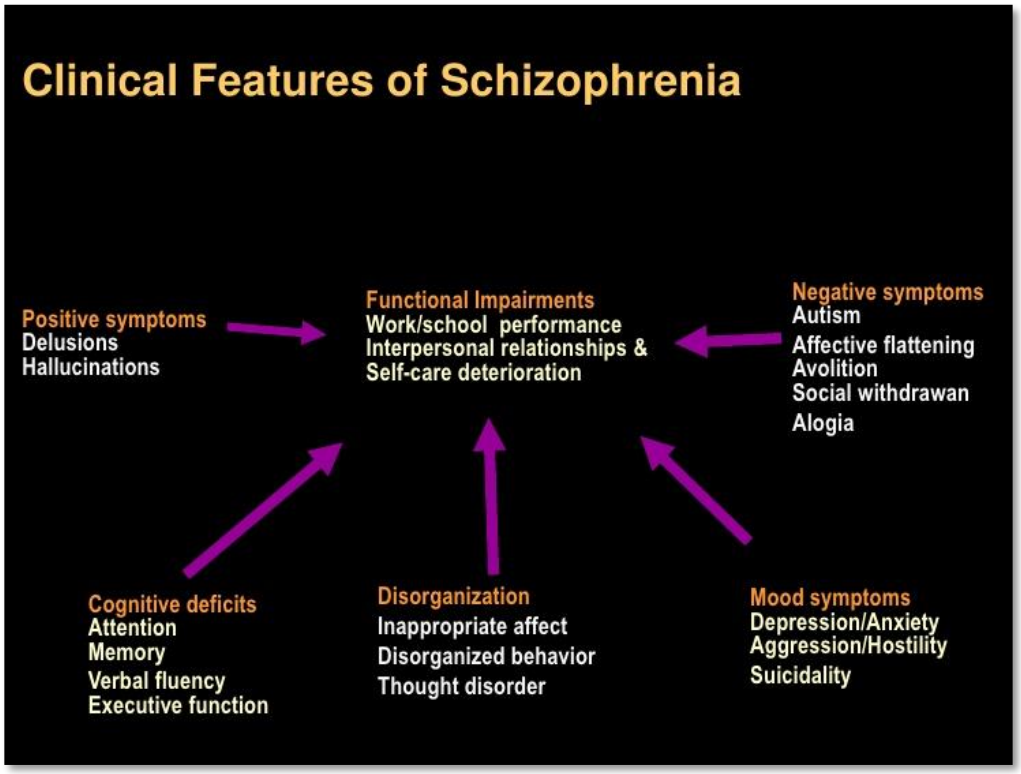
Schizoaffective Disorder: Concurrent presence of mood disturbance (depressive or manic episodes) and psychotic features (delusions or hallucinations, for at least **2** weeks in the absence of prominent mood symptoms during some phase of the illness).
DDx; mood disorders, substance-induced psychosis, 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 derogatorycontent) • 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?

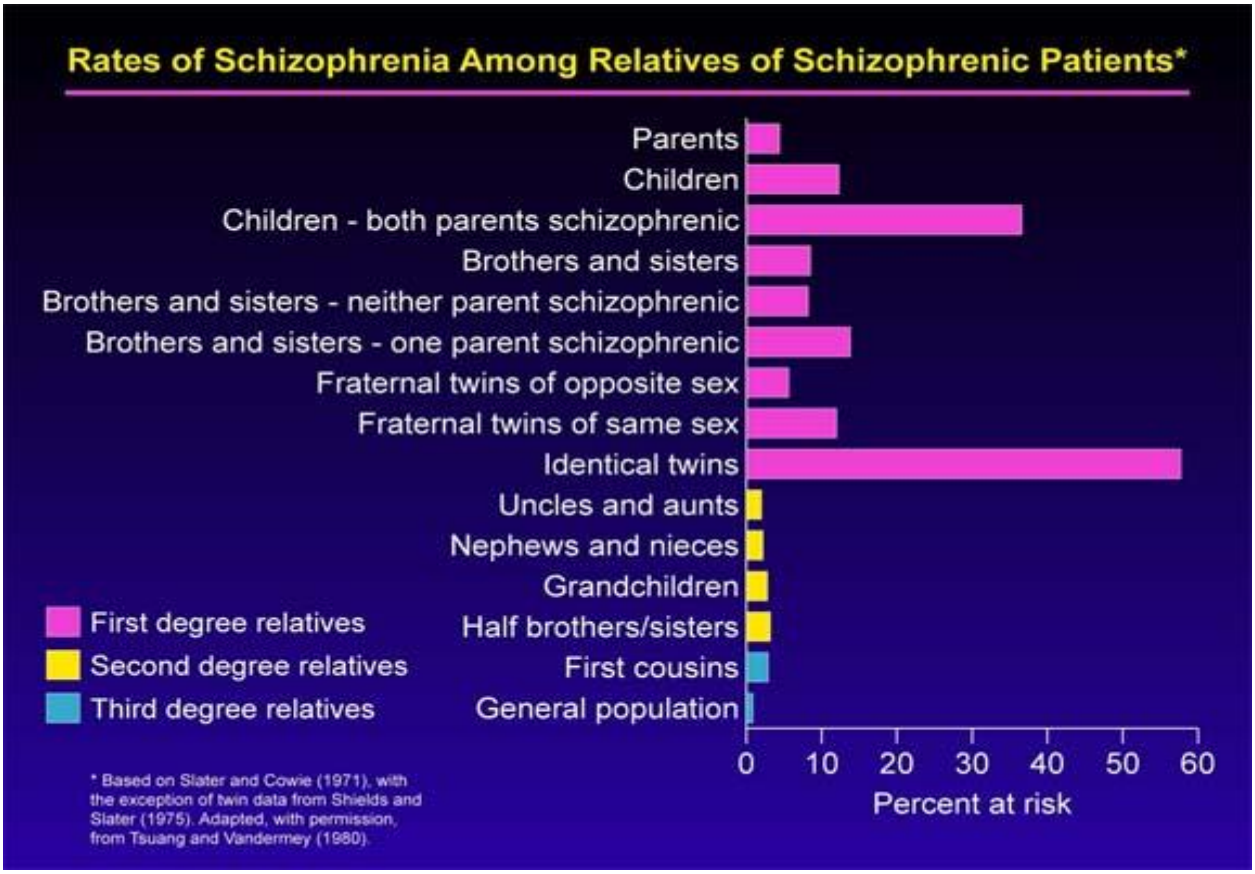
No single etiological factor is considered causative. The model most often used is that the person who develops schizophrenia has a specific biological vulnerability (or diathesis) that is triggered by stress and leads to emergence of schizophrenic symptoms.

Etiology:

1. **Genetic: (there are several genes involve in schizphrnia. Take look at them in the lecture)**

- 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- Neurobiological*:*

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.

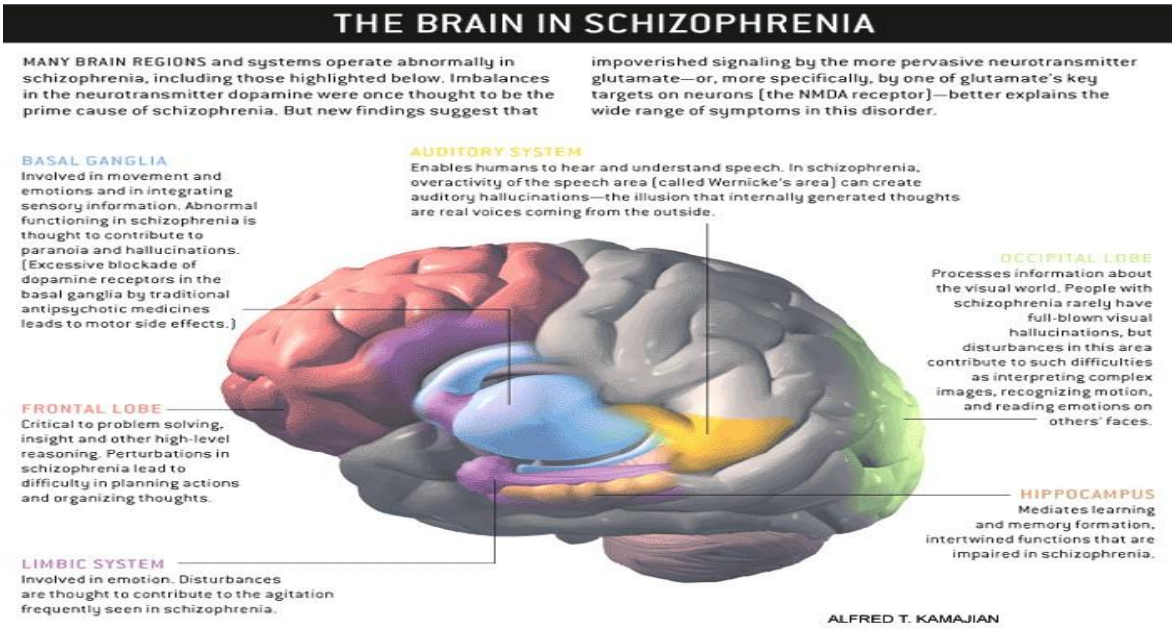
E. GABA hypothesis: the loss of inhibitory GABAergic neurons could lead to the hyperactivity of dopaminergic neurons. Some patients with schizophrenia have a loss of GABAergic neurons in the hippocampus.

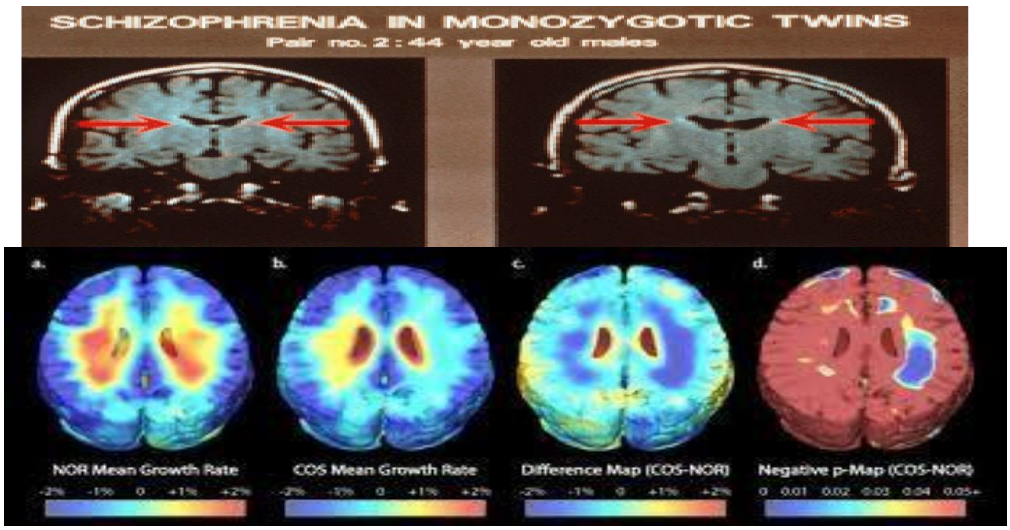
3. Neuropathology and Neuroimaging *:*

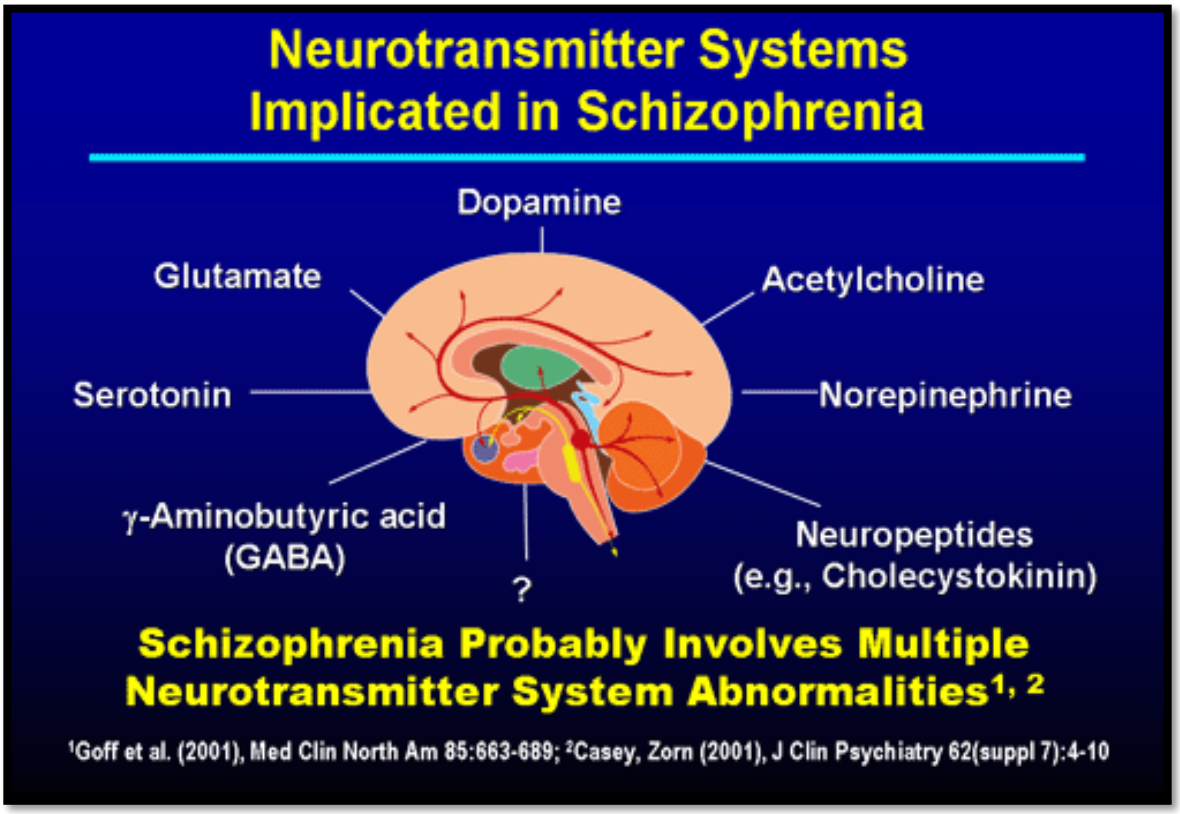
- CT scan studies: Cortical atrophy in 10 - 35 %. Enlargement of the lateral and third ventricles in 10-50%.
- Findings correlate more with negative features and with cognitive impairments.
- MRI and PET (Positron Emission Tomography): Abnormal frontal, parietal and temporal lobe structure and metabolism**.**

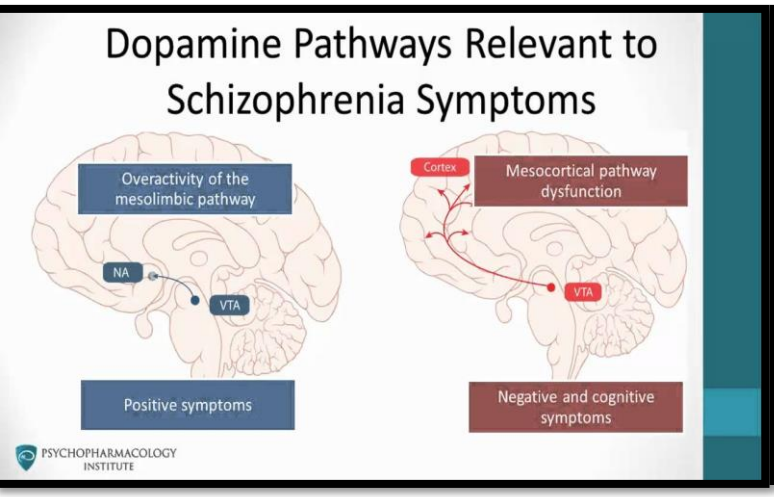
4. Psychosocial and Environmental:

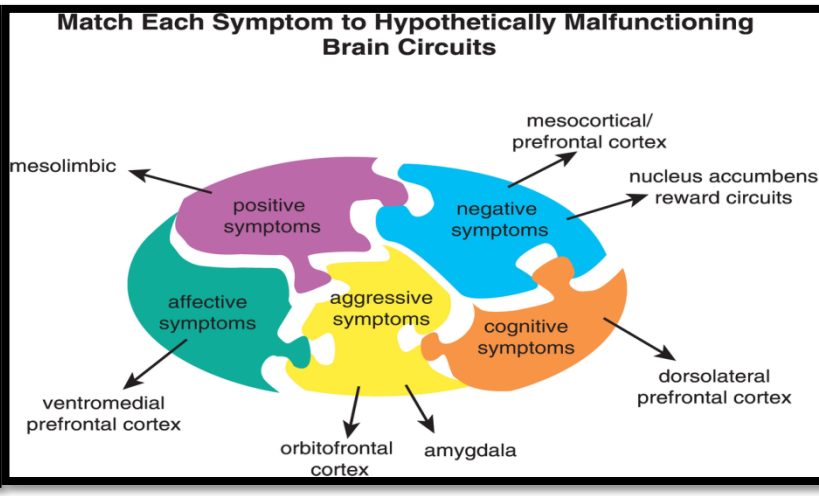
A*. Life Events*: Life stressors, particularly in the three months before onset, can induce schizophrenia in those who are vulnerable. ***B****. High Expressed Emotions (EE)* of the family (critical comments and emotional over-involvement. Patients whose families have high expressed emotions have higher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











VTA : VENTRAL TEGAMENTAL AREA

NA: NORADRENALINE

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

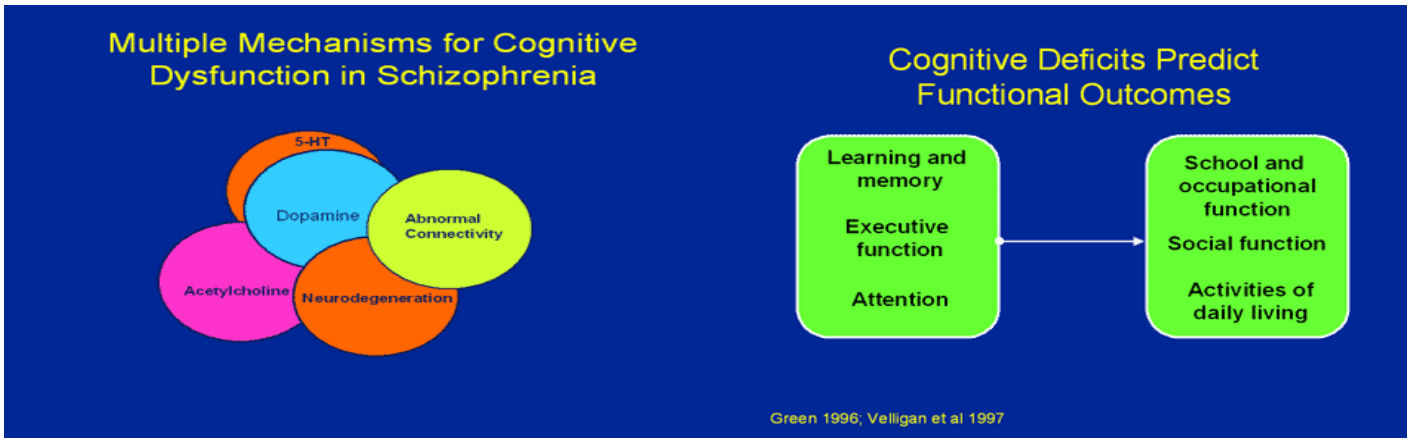
Hospitalization	Medications	Psychosocial
It is usually indicated in the acute phase in order to: -**Clarify diagnosis** (rule out possible organic causes). - **Control the disturbed behavior**. -**Protect patient and / or others** (risk of dangerousness or suicide; close 1:1 nurse observation).  -) for 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). 2Illness-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.



DDx of Causes:

1. Brief psychosis /schizophreniform disorder /acute schizophrenia.
2. Substance abuse (intoxication / withdrawal).
3. Acute organic brain syndrome (e.g. delirium).
4. Mood disorders; mania - severe agitated depression.
5. Personality disorders (e.g. borderline personality disorder).

Medications:

Major Tranquilizers e.g. :

Olanzapine 5-10mg IM,

(Haloperidol 5 - 10 mg IM or

Chlorpromazine 50 - 100 mg IM.)

Benzodiazepines: e.g. diazepam 5-10 mg (slow IV infusion to avoid the risk of respiratory depression). However, benzodiazepines may aggravate hostile behavior in certain susceptible people (release of inhibitory mechanisms).

Hospitalization:

For further assessment and treatment.

Approach

–Arrange for adequate help.

–Appear calm and helpful.

–Avoid confrontation.

–Take precautions:

- Never attempt to evaluate an armed patient.
- Other persons should be present (security guards or police officers).
- Keep the door open for an unavoidable exit.
- Restraints if needed by an adequate number of people using the minimum of force.
- Carefully search for any kind of offensive weapon.

–Aim to save patient and others.

- Anticipate possible violence from hostile, threatening behavior and from restless, agitated abusive patient.

–Do not bargain with a violent person about the need for restraints, medication or psychiatric admission.

- Reassure the patient and encourage self-control and cooperation.

youtube.com/watch?v=8zXsNEf7DuI

Restraint Technique: Enough staff should be available. If restraint becomes necessary, assign one team member to the patient's head and to each extremity. Be humane but firm, and do not bargain, start together to hold the patient and accomplish restraint quickly.

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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.

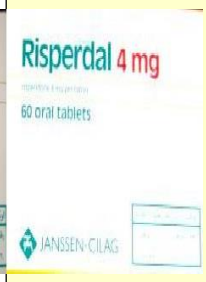
 # Antihistaminergic S/E; sedation and weight gain.

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.

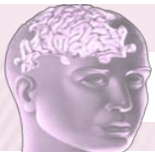
Adverse effects; Less EPSE, antiadrenergic, anticholinergic S/E. but there is a high risk of metabolic syndrome (see below).

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.

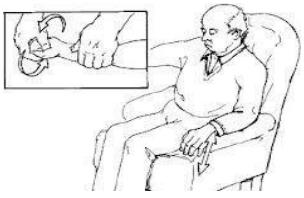
- Risperdal consta:25-50 mg./2weeks.
-Zuclopenthixol decanoate (Clopixol) : 200 – 600 mg. /month.
- Flupenthixol decanoate (Depixol – Fluanxol): e.g. 20 – 100 mg / month.
-Haloperidol decanoate (Haldol) : 200 – 400 mg. / month.
-Fluphenazine decanoate (Anatensol – Modecate): e.g. 25 – 75 mg / month.

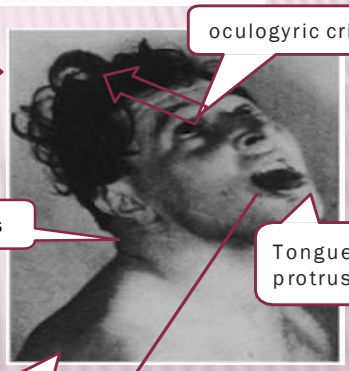


Extra-Pyramidal Side Effects (EPSE)

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)





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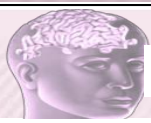

Extra-Pyramidal Side Effects (EPSE)

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.

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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.

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ANTIADRENERGIC

Postural hypotension.

Inhibition of ejaculation.

ANTICHOLINERGIC

Blurred vision

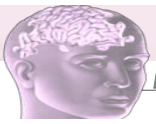
Precipitation of closed – angle glaucoma.

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,

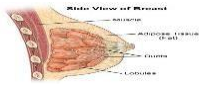
(4) hypertension.

(5) an elevated fasting blood glucose level.

It increases risk of cardiovascular disease and type II diabetes.

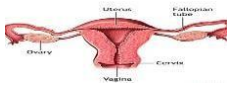
Other

Hyperprolactinemia.



Galactorrhea.

Amenorrhea.



Low libido.

Sedation

(antihistamine effect).

Weight gain.

Toxic Effect:

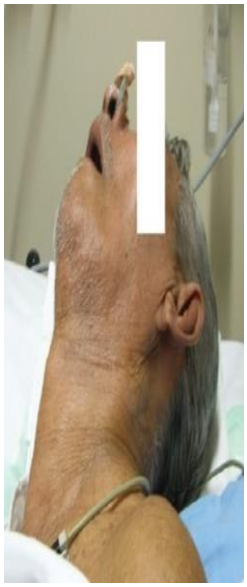
Neuroleptic Malignant Syndrome (NMS) see Psychiatric Emergencies.

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).

- MCQS

1.A 27-year-old man was brought to outpatient psychiatry clinic with 2 months history of hearing voices commenting on his actions, persecutory delusion, and disorganized behavior without disturbed mood. However, he returned normal with no medications. The most likely diagnosis is:

a. Brief psychotic disorder.
b. Schizophreniform disorder.
c. Schizoaffective disorder.
d. Schizophrenia.

2. A 36-year-old male was brought to the emergency department by his parents who gave a 9-year history of mental illness. He is treated with a monthly injection at a mental hospital. Parents are worried about their son's mutism, rigid limbs, and clouding consciousness. The most appropriate management step is:

a. Olanzapine IM.
b. Brain CT-Scan.
c. CPK blood test.
d. Brain MRI.

3.A 31- year-old woman delivered 3 weeks ago, she then gradually became paranoid, agitated aggressive, restless and insomniac. The most appropriate treatment is:

a. Escitalopram.
b. Fluoxetine.
c. Carbamazepine.
d. Quetiapine.

4.A 22-year-old college student has one year history of poor academic performance, poor self-care, posturing, rigidity and lack of motivation. The most likely diagnosis is:

a. Catatonic schizophrenia.
b. schizoaffective disorder.
c. Schizoid personality disorder.
d. Paranoid personality.

5.A 23-year-old single female developed hallucinations, paranoid delusions and disorganized behavior. She was treated with risperidone 4 mg/day. She has amenorrhea for 4 months. Your best initial step would be:

a. Bromocriptine 10 mg.
b. Discontinue risperidone.
c. Reduce risperidone to 3 mg.
d. Change to clozapine 200 mg.

Answers:

1	2	3	4	5
B	C	D	A	C

Done By: Hamad Aldossari