

Definition, etiopathogenesis, clinical manifestations, nonpharmacological and pharmacological management of the diseases associated with Psychiatric Disorders

Depression (Major Depressive Disorder – MDD)

Definition:

- Depression is a common psychiatric disorder characterized by persistent low mood, loss of interest, and impaired functioning lasting at least two weeks, accompanied by cognitive, emotional, and somatic symptoms.

Etiopathogenesis:

Biological factors:

- **Neurotransmitter imbalance:** Decreased serotonin, norepinephrine, and dopamine levels
- **Genetic predisposition:** Family history of depression
- **HPA axis dysfunction:** Chronic stress increases cortisol levels
- **Neuroplasticity and neuroinflammation** theories

Psychosocial factors:

- Stressful life events (e.g., bereavement, trauma)
- Childhood adversity
- Lack of social support
- Personality traits (e.g., pessimism)

Clinical Manifestations:

- **Core symptoms** (at least 5 for ≥ 2 weeks, one must be depressed mood or anhedonia):
 - Depressed mood
 - Loss of interest or pleasure (anhedonia)
 - Fatigue or low energy
 - Changes in sleep (insomnia or hypersomnia)
 - Appetite/weight changes
 - Poor concentration
 - Psychomotor retardation or agitation
 - Feelings of worthlessness or guilt

- Suicidal thoughts or behavior

Subtypes:

- Melancholic, atypical, psychotic, postpartum, seasonal

Non-Pharmacological Management:

➤ Psychotherapy (first-line for mild to moderate cases):

- Cognitive Behavioral Therapy (CBT)
- Interpersonal therapy (IPT)
- Supportive therapy

➤ Lifestyle modifications:

- Regular exercise
- Sleep hygiene
- Nutrition
- Stress management

➤ Social support and psychoeducation

Pharmacological Management:

Antidepressants (minimum duration: 6–9 months):

- **SSRIs** (first-line): Fluoxetine, sertraline, escitalopram
- **SNRIs**: Venlafaxine, duloxetine
- **TCAs**: Amitriptyline, nortriptyline (more side effects)
- **Atypical antidepressants**: Mirtazapine, bupropion
- **MAOIs** (rarely used due to dietary restrictions)

Others:

- **Mood stabilizers** (if bipolar disorder suspected)
- **Antipsychotics** (for psychotic depression)

Electroconvulsive Therapy (ECT):

- For severe, suicidal, or treatment-resistant depression

Anxiety Disorders (Generalized Anxiety Disorder – GAD)

Definition:

- Generalized Anxiety Disorder is characterized by excessive, uncontrollable worry about various aspects of daily life, lasting for at least 6 months, and accompanied by physical and psychological symptoms.

Etiopathogenesis:

Neurobiological factors:

- Dysfunction in GABA, serotonin, and noradrenaline systems
- Amygdala hyperactivity

Psychological/Environmental:

- Personality (e.g., neuroticism)
- Childhood trauma or overprotective parenting
- Chronic stress or illness

Comorbidity:

- Often coexists with depression, substance use, or other anxiety disorders

Clinical Manifestations:

- Excessive worry (health, finances, work, relationships)
- Restlessness or feeling on edge
- Muscle tension
- Fatigue
- Difficulty concentrating
- Sleep disturbances
- Irritability
- Physical symptoms: headache, nausea, palpitations, sweating

Non-Pharmacological Management:

- **CBT:** Gold standard – targets cognitive distortions
- **Relaxation techniques:** Breathing exercises, meditation, yoga
- **Exercise:** Reduces stress hormones
- Sleep hygiene and routine
- Limit caffeine and stimulants
- Psychoeducation

Pharmacological Management:

- **SSRIs:** Escitalopram, paroxetine
 - **SNRIs:** Venlafaxine, duloxetine
 - **Buspirone:** Non-benzodiazepine anxiolytic
 - **Benzodiazepines:** Short-term use (e.g., lorazepam, clonazepam) – risk of dependence
 - **Beta-blockers:** Propranolol (for physical symptoms like tremors)
 - **Pregabalin:** Used in some chronic anxiety disorders
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Psychosis

Definition:

- Psychosis is a mental disorder characterized by a loss of contact with reality, involving hallucinations, delusions, disorganized thinking, and abnormal behaviors.
- Seen in conditions like **schizophrenia**, severe mood disorders, and drug-induced states.

Etiopathogenesis:

Biological basis:

- Dopamine hypothesis: Hyperactivity in mesolimbic pathway
- Genetic vulnerability (e.g., family history of schizophrenia)
- Brain structural abnormalities (enlarged ventricles, cortical thinning)

Environmental factors:

- Early life stress or trauma
- Urban upbringing, migration
- Substance abuse (especially cannabis, amphetamines)

Clinical Manifestations:

Positive symptoms:

- **Delusions** (fixed false beliefs): paranoid, grandiose, somatic
- **Hallucinations** (usually auditory): voices commenting or commanding
- Disorganized speech or behavior

Negative symptoms:

- Apathy, lack of motivation



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