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Relationship between psychiatric disorders and social habits dependence: A review

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Abstract

Globally, psychiatric disorders are most prevalent and are diagnosed very late. Prolonged psychiatric ailments will lead to the addiction towards social habits such as Alcohol, Tobacco and Cannabis etc. Therefore addiction towards these social habits may further worsen the Neuro-chemistry of CNS which is in the state of imbalance previously. This will lead to decreased quality of life mentally and physically. By measuring the degree of dependence and managing it in the psychiatric patients who were addicted to social habits will be helpful in management of psychiatric distress. It is important to create the awareness among health care professionals as well as every individual in early recognition of addiction symptoms will be effective in slowing down of disease process.

Keywords: Schizophrenia, depression, panic attack, substance abuse, cognitive behavioural therapy

Introduction

Mental Health is defined as a state of well-being in which every individual realizes his or her own potential, copes with normal stress of life, can work productively and fruitfully, and is able to make a contribution to her or his community ^[1].

Definitions

Psychiatric disorder

- It is a behavioral or psychological syndrome or pattern that occurs in an individual.
- The consequences of disorder are clinically significant distress (e.g., a painful symptom) or disability (i.e., impairment in one or more important areas of functioning).
- Must not be merely an expectable response to common stressors and losses (for example, the loss of a loved one) or a culturally sanctioned response to a particular event (for example, trance states in religious rituals).
- That reflects an underlying psychobiological dysfunction.
- That is not solely a result of social deviance or conflicts with society.
- That has diagnostic validity using one or more sets of diagnostic validators (e.g., prognostic significance, psychobiological disruption, response to treatment).
- That has clinical utility (for example, contributes to better conceptualization of diagnoses, or to better assessment and treatment) ^[2].

Substance abuse

Substance abuse refers to the use of alcohol, tobacco, cannabis and illicit drugs. It can lead to a dependence syndrome (a cluster of behavioral, cognitive and physiological phenomena that develop after repeated substance use and that typically include a strong desire to take the drug, difficulties in controlling its use, persisting in its use despite harmful consequences, a higher priority given to drug use than to other activities and obligations, increased tolerance, and sometimes a physical withdrawal state) ^[3].

Psychiatric disorders

Schizophrenia

Definition: It is a serious, chronic mental disease that affects a person's mood, understanding of reality, and ability to think clearly ^[4].

Epidemiology: Globally 20.9 million people are suffering from Schizophrenia ^[5], 2.3 - 2.7 per thousand people are in India ^[6].

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Etiology ^[7]

Obstetric complications: bleeding during pregnancy, gestational diabetes, emergency cesarean section, asphyxia, and low birth weight.

Genetic factors: Risk of illness is approximately 10 % for a first-degree relative and 3 % for a second-degree relative. In the case of monozygotic twins, the risk of one twin having schizophrenia is 48 % if the other has the disorder, whereas the risk is 12 % to 14 % in dizygotic twins. If both parents have schizophrenia, risk that they will produce a child with schizophrenia is approximately 40%.

Environmental stressors: Childhood trauma, minority ethnicity, residence in an urban area and social isolation.

Social stressors: Discrimination or economic adversity may predispose individuals toward delusional or paranoid thinking.

Pathophysiology ^[8]

Neuroanatomical & Neurofunctional theory: Changes in the mesolimbic pathway (the neuronal projection from the ventral

tegmental area (VTA) to the nucleus accumbens, amygdala and hippocampus) is associated with positive symptoms, whereas negative and cognitive impairment symptoms are associated with changes in the mesocortical pathway (the projection from the VTA to areas of the prefrontal cortex).

Dopamine theory: Amphetamine releases dopamine in the brain and can produce a behavioural syndrome indistinguishable from an acute schizophrenic episode in humans. Also, hallucinations are a side effect of L-dopa therapy for Parkinson's disease. In animals, dopamine release causes a specific pattern of stereotyped behavior that resembles the repetitive behavior sometimes seen in schizophrenic patients. Potent D2 receptor agonists, such as bromocriptine will produce similar effects in animals, and these drugs, like amphetamine, exacerbate the symptoms of schizophrenic patients.

Glutamate theory: In humans, NMDA receptor antagonists such as phencyclidine, ketamine and dizocilpine (can produce both positive and negative psychotic symptoms). It has therefore been postulated that schizophrenia may result from disruption of glutamatergic neurotransmission.

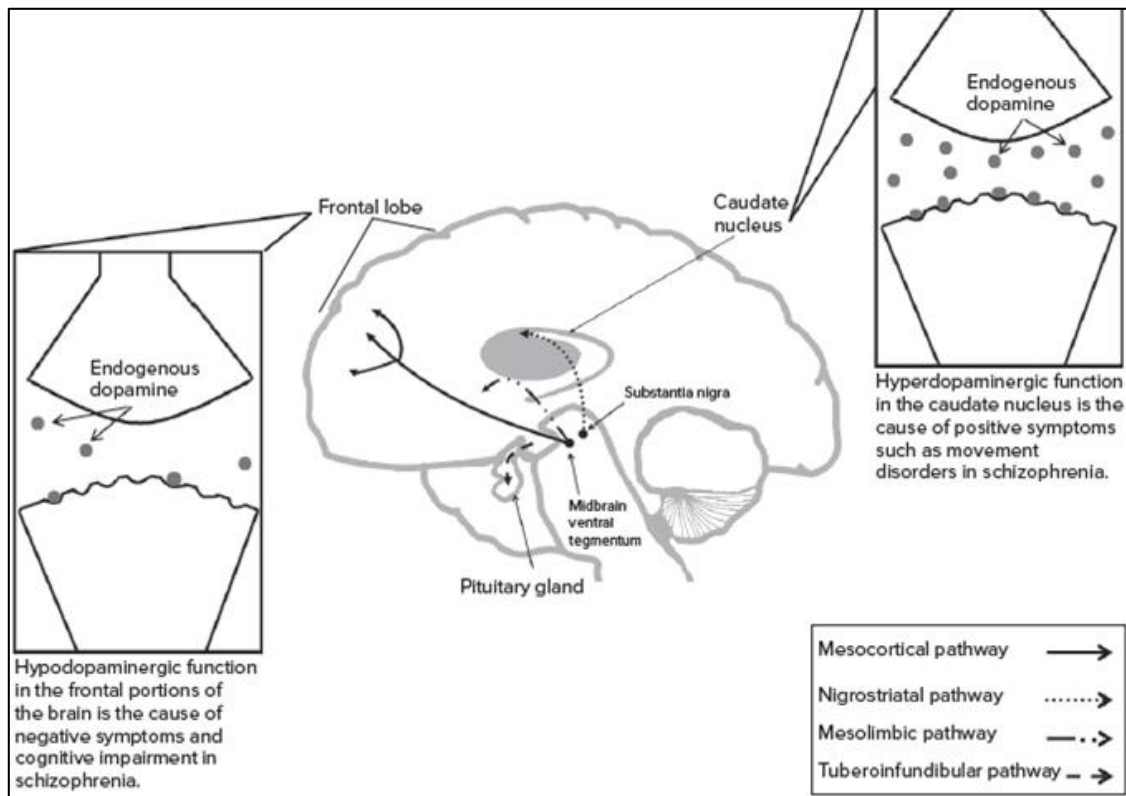


Fig 1: An overview of pathophysiology of schizophrenia ^[6].

Diagnostic tests ^[9]

DSM-IV-TR Diagnostic Criteria for Schizophrenia

A. Characteristic symptoms: Two or more of the following, each persisting for a significant portion of at least one-month

Period

- (1) Delusions
- (2) Hallucinations
- (3) Disorganized speech
- (4) Grossly disorganized or catatonic behavior
- (5) Negative symptoms

B. Social / occupational dysfunction: For a significant portion of the time since onset of the disorder, one or more major areas of functioning such as work, interpersonal relations, or self-care are significantly below the level prior to onset.

C. Duration: Continuous signs of the disorder for at least 6 months. This must include at least 1 month of symptoms fulfilling criterion A (unless successfully treated). These 6 months may include prodromal or residual symptoms.

D. Schizoaffective or mood disorder has been excluded.

E. Disorder is not due to a medical disorder or substance use.

F. If a history of a pervasive developmental disorder is present, there must be symptoms of hallucinations or delusions present for at least 1 month.

Pharmacotherapy

Goals ^[9]

- 1) The choice of anti-psychotic medication is based on the following:
 - Past Medical History of patient or family member response
 - ADR profile of Anti-Psychotic Agent (APA)
- 2) Minimum effective dose should be used.
- 3) Emphasizing an adequate time as a primary variable in determining response.
- 4) Limiting augmentation medications to nonresponsive patients.

Typical APAs ^[10]

- Perphenazine 8 -16 mg PO as divided doses BD
- Trifluoperazine 2.5 mg PO BD should be increased gradually to 15 – 20 mg PO OD

- Fluphenazine 2.5-10 mg PO in 2 - 3 divided doses / day
- Haloperidol 0.5-5 mg PO BD
- Thiothixene 2-5 mg PO BD
- Loxapine 30-50 mg PO BD
- Thioridazine 200-800 mg PO daily in 2 - 4 divided doses
- Pimozide 2 mg PO OD increased by 2 - 4 mg every week

Atypical APAs ^[10]

- Clozapine 150-300 mg PO BD
- Risperidone 1-4 mg PO QD
- Olanzapine 5-10 mg PO QD
- Quetiapine 150-750 mg per day as 2-3 divided doses
- Ziprasidone 20-40 mg PO BD
- Aripiprazole 10-15 mg PO QD
- Paliperidone 6 mg PO QAM
- Iloperidone 5-10 mg SL BD
- Lurasidone 40-160mg PO QD

Guidelines

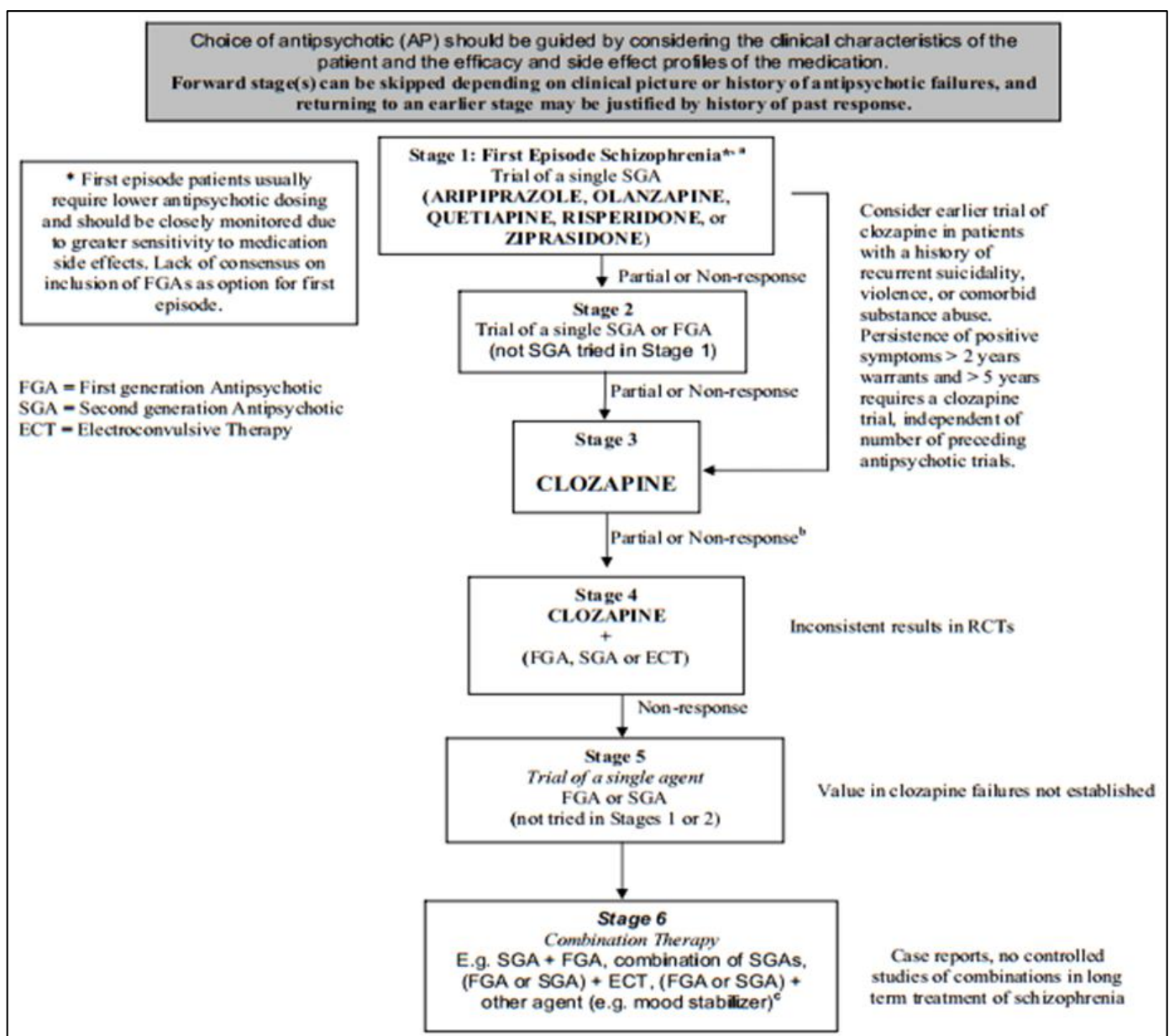


Fig 2: Guidelines for management of schizophrenia ^[11].

Patient counselling

Patient counselling is done by various psychotherapeutic approaches such as ^[12].

- Cognitively oriented skill training
- Integrated psychological therapies
- Schematic cognitive therapy
- Cognitive-behavioral therapy
- Neuropsychologically oriented therapy
- Sociocognitive techniques

Major Depressive Disorder (MDD)

Definition: It is a chronic mental disorder that causes depressed mood (dysphoria) and loss of interest in activities that were rather pleasurable in the past (anhedonia) for a duration of at least two weeks ^[13].

Epidemiology: Over 300 million people of all ages in the world are suffering from depression ^[14], 57 million people are in India ^[15].

Etiology ^[16]

Biological factors: Genetic and familial predisposition, alteration in the neural structures and sleep dysregulation.

Psychological factors: Cognitive schemas, beliefs, assumptions, pessimism, low self-esteem, ruminative response style and negative cognitive style.

Social factors: Marriage and relationship issues, low social support and low income.

Chronic diseases: cardiovascular problems, diabetes mellitus, cancer, disability.

Pathophysiology ^[8]

Monoamine theory

Decreased synaptic concentrations of norepinephrine and / or serotonin causes depression. (Explained by reserpine).

Dysregulation theory

An impairment in the regulatory or homeostatic mechanisms.

- An erratic basal output of the neurotransmitter system.
- A disruption in normal periodicities (circadian rhythm).
- A less selective response to environmental stimuli.
- Perturbation of the system resulting in a delayed return to baseline.

Neuro-endocrine findings

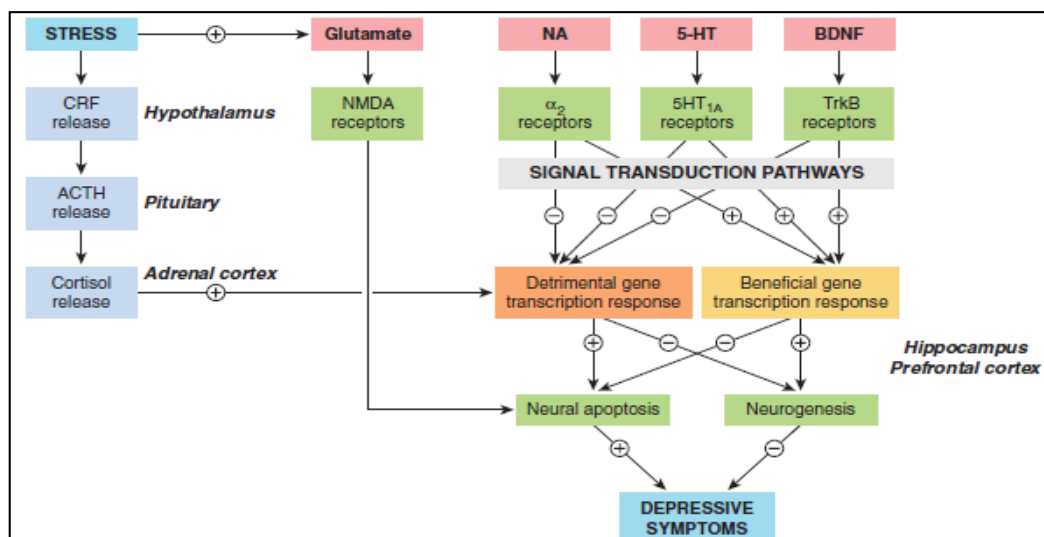


Fig 3: Simplified diagram showing mechanisms believed to be involved in the pathophysiology of depression ^[8]

Thyroid hormones

Thyroid hormones (TH) imbalance are implicated in the pathophysiology of neurodegenerative and psychiatric conditions. These hormones are very essential for brain development, maturation and have been shown to promote neurogenesis, in particular, in the hippocampus. Hypothyroidism has been linked to depressive like behavior in that it impaired hippocampal neurogenesis which resolved with hormone replacement. Animal studies also revealed that thyroid hormone causes an increase in serotonergic neurotransmission which supports the fact that TH supplementation has been beneficial in management of refractory cases of depression ^[17].

Diagnostic tests

DSM IV TR Scale for MDD ^[18]

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of

interest or pleasure.

Note: Do not include symptoms that are clearly caused by a general medical condition or mood-incongruent delusions or hallucinations.

1. Depressed mood most of the day nearly every day.
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day nearly every day.
3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5 % of body weight in a month), or decrease or increase in appetite nearly every day.
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt nearly every day.
8. Diminished ability to think or concentrate, or

indecisiveness, nearly every day.

10. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.
- A. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- B. The symptoms are not due to the direct physiologic effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
- C. The symptoms are not better accounted for by bereavement (i.e., after the loss of a loved one), the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

Pharmacotherapy

Goals ^[9]

- To reduce the symptoms of acute depression
- To facilitate the patient's return to a level of functioning before the onset of illness
- To prevent further episodes of depression

I) Selective serotonin reuptake inhibitors

Citalopram: 20 - 60 mg PO OD

Escitalopram: 10 - 20 mg PO OD

Fluoxetine: 20 - 60 mg PO OD

Fluvoxamine: 50 - 300 mg PO OD

Paroxetine: 20 - 60 mg PO OD

Sertraline: 50 - 200 mg PO OD

II) Serotonin / Nor - epinephrine reuptake inhibitors

Venlafaxine: 75 - 225 mg PO OD

Duloxetine: 30 - 90 mg PO OD

III) Aminoketone

Bupropion: 150 - 300 mg PO OD

IV) Triazolopyridines:

Nefazodone: 200 - 600 mg PO OD

Trazodone: 150 - 300 mg PO OD

V) Tetracyclics

Mirtazapine: 15 - 45 mg PO OD

VI) Tricyclics

Tertiary amines

Amitriptyline: 100 - 300 mg PO OD

Clomipramine: 100 - 250 mg PO OD

Doxepin: 100 - 300 mg PO OD

Imipramine: 100 - 300 mg PO OD

Secondary amines

Desipramine: 100 - 300 mg PO OD

Nortriptyline: 50 - 200 mg PO OD

VII) Monoamine oxidase inhibitors

Phenelzine: 30 - 90 mg PO OD

Selegiline (transdermal): 6 - 12 mg

Tranylcypromine: 20 - 60 mg PO OD [19]

Guidelines

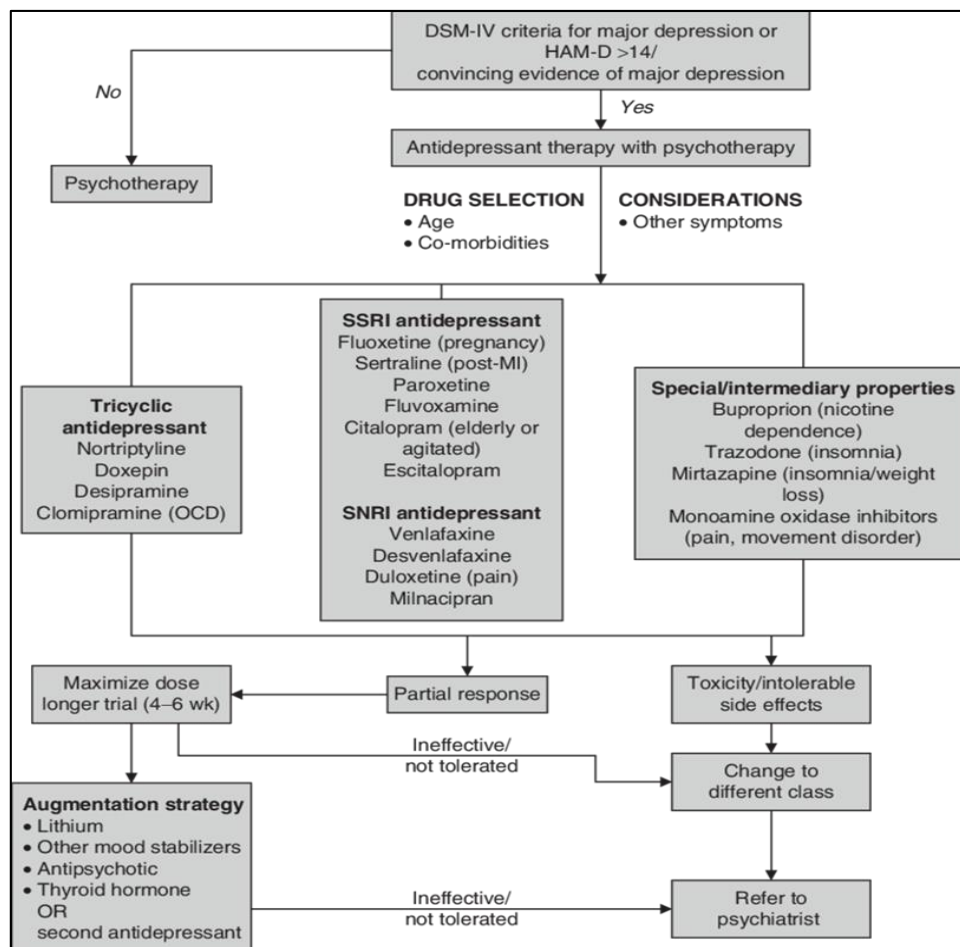


Fig 4: Guidelines for management of MDD ^[19]

Patient counselling

Patient counselling is done by various psychotherapeutic approaches such as ^[20]:

- Psychotherapy
- Cognitive behavioral therapy
- Psychodynamic psychotherapy
- Interpersonal therapy
- Emotion focused therapy
- Group therapy
- Exercise
- Relaxation

Panic Disorder

Definition: It is defined as a discrete period of intense fear or discomfort that has an abrupt onset, reaches peak in 10 minutes, and is accompanied by at least 4 or 13 of at least somatic symptoms ^[21].

Epidemiology: 1.5 - 4 % of the world's population are

suffering from panic disorder, In India approximately 10 per 100,000 population per year patients are affected ^[22, 23].

Etiopathogenesis

The cause of panic disorder is unknown. Several factors probably contribute to its development, and no biological test is available. Genetic and early family factors, or both, are important. Patients inherit a sensitive “central nervous system fear mechanism, centered in the amygdala”.

A psychological model suggests that panic attacks and panic disorder represent “fear of fear.” In this model, physical sensations associated with anxiety—feeling dizzy or faint, or having a pounding heart, shortness of breath, or chest pain—are interpreted as indicating a dire consequence. This leads to hyper vigilance about bodily sensations, increased arousal of the sympathetic nervous system, more physical sensations, and heightened anxiety, which spirals into a panic attack.

Panic disorder can be caused by traumatic events, excessive caffeine, and misuse or withdrawal of drugs or alcohol ^[23].

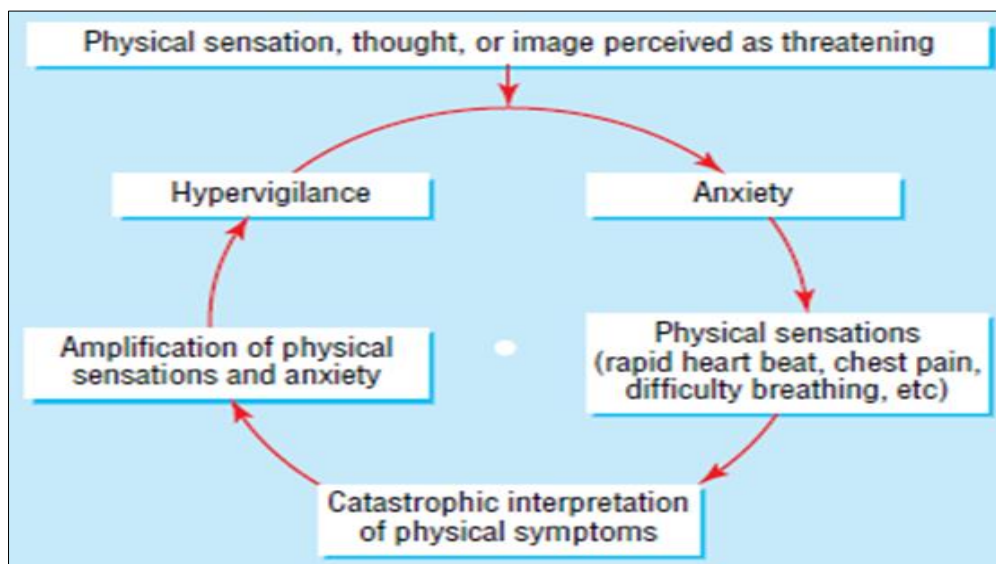


Fig 5: Cognitive model of panic: symptoms, hyper vigilance, and anxiety spiral into panic attack. Patients develop “fear of fear” and avoid situations where they think they will have a panic attack ^[23].

Diagnostic tests

DSM-IV criteria for panic disorder and agoraphobia ^[41].

Panic attack

A discrete period of intense fear or discomfort in which four (or more) of the following symptoms developed abruptly and reached a peak within 10 minutes:

- Palpitations, pounding heart, or accelerated heart rate
- Sweating
- Trembling or shaking
- Sensations of shortness of breath or smothering
- Sensation of choking
- Chest pain or discomfort
- Nausea or abdominal distress
- Feeling dizzy, unsteady, light headed, or faint
- Derealisation (feelings of unreality) or depersonalization (being detached from oneself)
- Fear of losing control or going crazy
- Fear of dying
- Paraesthesias
- Chills or hot flashes

Agoraphobia

- Anxiety about being in places or situations from which escape is difficult (or embarrassing) or in which help may not be available. Consider the diagnosis of a specific phobia if avoidance is limited to one or a few specific situations, or social phobia if avoidance is limited to social situations
- The situations are avoided or endured with great distress or anxiety about having a panic attack

Panic disorder without agoraphobia

Recurrent, unexpected panic attacks, one of which at least is followed by one month (or more) of at least one of:

- Persistent concern about having additional attacks
- Worry about the implications of the attack or its consequences
- A significant change in behavior related to the attacks
- Panic attacks are not due to drug misuse, prescribed drugs, or a medical condition
- The panic attacks are not better accounted for by another disorder
- Absence of agoraphobia

Panic disorder with agoraphobia

Characterized by the above criteria for panic disorder but with agoraphobia:

- Anxiety about being in places or situations from which escape might be difficult (or embarrassing)
- Avoiding these situations or enduring them with great distress or anxiety

Agoraphobia without history of panic disorder

It is characterized by the criteria for agoraphobia but not panic disorder.

Pharmacotherapy**Goals**

- The goal of therapy in panic disorder is remission.
- Patients should be free of panic attacks, have no or minimal anticipatory anxiety and agoraphobic avoidance, and no functional impairment ^[9].

Selective serotonin reuptake inhibitors

Citalopram: 20–60 mg PO OD

Escitalopram: 10–20 mg PO OD

Fluoxetine: 10–30 mg PO OD

Fluvoxamine: 100–300 mg PO OD

Paroxetine: 20–60 mg PO OD

Sertraline: 50–200 mg PO OD

Serotonin norepinephrine reuptake inhibitors

Venlafaxine: 75–225 mg PO OD

Benzodiazepines

Alprazolam: 4–10 mg PO OD

Clonazepam: 1–4 mg PO OD

Diazepam: 5–20 mg PO OD

Lorazepam: 2–8 mg PO OD

Tricyclic antidepressant:

Imipramine: 75–250 mg PO OD

Monoamine oxidase inhibitor

Phenelzine: 45–90 mg PO OD. ^[42]

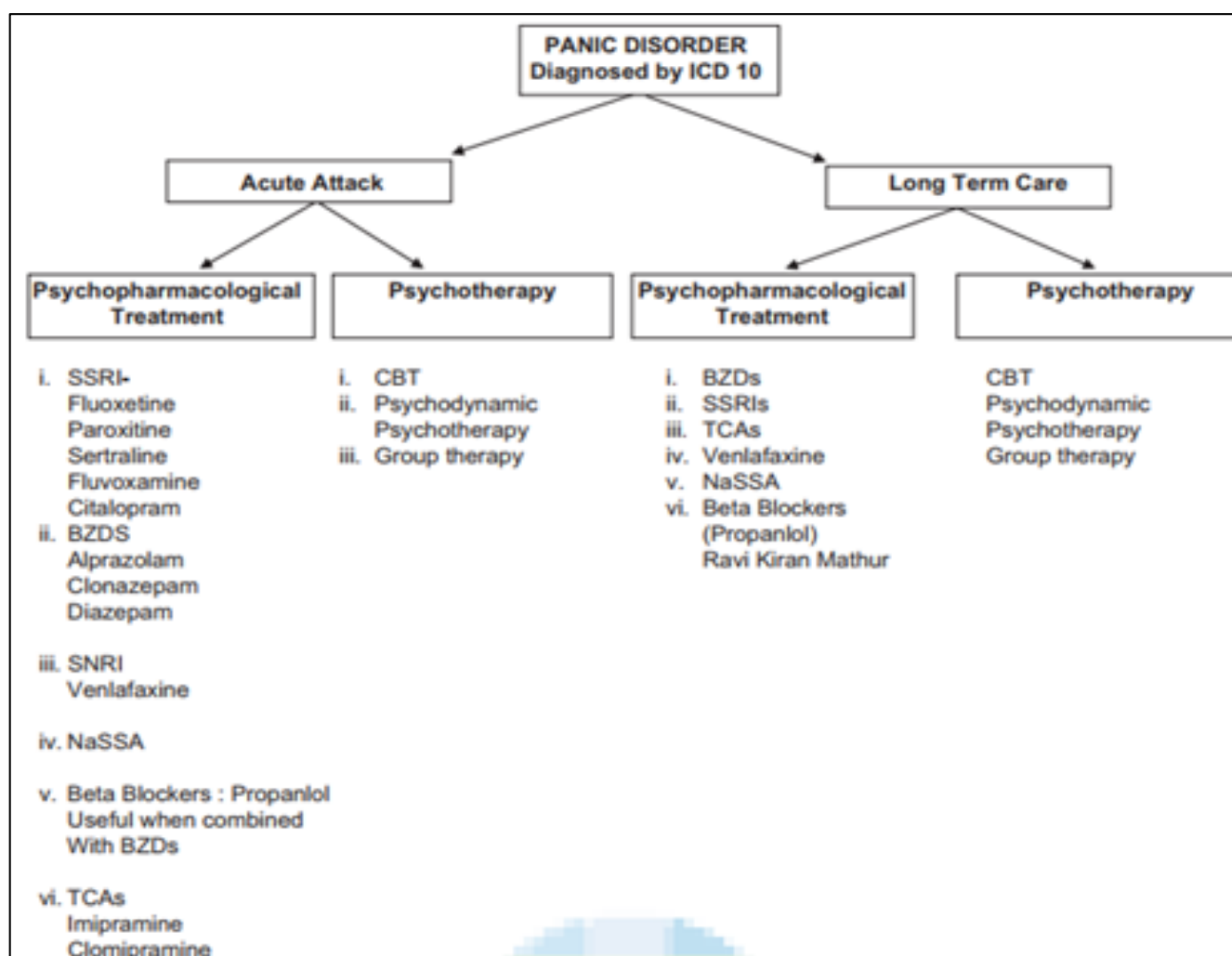
Guidelines

Fig 6: Guidelines for management of Panic Disorder ^[26].

Patient Counselling

Patient counselling is done by various psychotherapeutic approaches such as ^[27].

- Psycho education (PE)
- Supportive psychotherapy (SP)
- Physiological therapies (PT)
- Behaviour therapy (BT)
- Cognitive therapy (CT)
- Cognitive behavior therapy (CBT)
- Third-wave CBT (3W)
- Psychodynamic therapies (PD).

Substance Abuse Disorders

Alcohol Dependence Syndrome

Definition: A cluster of behavioral, cognitive, and physiological phenomena that develop after repeated alcohol use and that typically include a strong desire to take the alcohol, difficulties in controlling its use, persisting in its use. Despite harmful consequences, a higher priority given to alcohol use than to other activities and obligations, increased tolerance and sometimes a physical withdrawal state ^[28].

Epidemiology: Globally, 3 million deaths every year result from alcohol abuse which represents 5.3 % of all deaths. Overall 5.1 % of the global burden of disease and injury is attributable to alcohol, as measured in disability-adjusted life years (DALYs). Alcohol consumption causes death and disability relatively early in life. In the age group 20–39 years approximately 13.5 % of the total deaths are alcohol-attributable ^[29].

2.7% of population (29 million individuals) in India is affected by alcohol dependence ^[30].

Etiology ^[9]

Novelty seeking
Impulsivity
Being single
Aggression
Depression
History of sexual abuse
Having deceased parents
Urbanicity
Religious background

Pathophysiology

1. **GAB Amergic Theory:** It acts by enhancing gamma aminobutyric acid (GABA)-nergic function through interaction with GABA A receptors and associated chloride ion channels.
2. **Glutamate Theory:** NMDA receptors mediate neurotoxicity by increasing permeability to calcium and regulating neuronal long-term potentiation. It is postulated that in the acute form of ethanol use, NMDA receptor function is inhibited, while chronic ethanol uses results in up-regulation of NMDA receptors.

Changing the balance among glutamate and GABA signaling establishes a state of hyperexcitability that is manifest upon cessation of drinking and that may contribute to the clinical manifestations ^[31].

Signs & Symptoms

Alcoholism condition in where an individual consumes large amounts of alcohol over a long period of time. It is characterised by:

- A pathological desire for alcohol intake
- Black-outs (amnesia) during intoxication
- Withdrawal symptoms on ceasing alcohol intake

Peripheral neuropathy (Tingling & Numbness)

Wernicke's syndrome (encephalopathy, uncoordinated muscle movement, and eye muscle weakness)

Korsakoff's syndrome (amnesia)

Arrhythmias in binge drinking ^[31, 32].

Diagnostic tests

DSM V TR Scale

1. Had times when you ended up drinking more, or longer, than you intended?
2. More than once wanted to cut down or stop drinking, or tried to, but couldn't?
3. Spent a lot of time drinking?
4. Wanted a drink so badly you couldn't think of anything else?
5. Found that drinking—or being sick from drinking—often interfered with taking care of your home or family? Or caused job troubles? Or school problems?
6. Continued to drink even though it was causing trouble with your family or friends?
7. Given up or cut back on activities that were important or interesting to you, or gave you pleasure, in order to drink?
8. More than once gotten into situations while or after drinking that increased your chances of getting hurt (such as driving, swimming, using machinery, walking in a dangerous area, or having unsafe sex)?
9. Continued to drink even though it was making you feel depressed or anxious or adding to another health problem? Or after having had a memory blackout?
10. Had to drink much more than you once did to get the effect you want? Or found that your usual number of drinks had much less effect than before?
11. Found that when the effects of alcohol were wearing off, you had withdrawal symptoms, such as trouble sleeping, shakiness, restlessness, nausea, sweating, a racing heart, or a seizure? Or sensed things that were not there ^[33]?

Severity

Mild: The presence of 2 to 3 symptoms.

1) Moderate: The presence of 4 to 5 symptoms.

2) Severe: The presence of 6 or more symptoms ^[33].

Pharmacotherapy

- 1) Disulfiram: 250 mg–500 mg PO OD
- 2) Acamprosate: 999 mg–1,998 mg and higher PO OD
- 3) Naltrexone: 50 mg–100 mg PO OD

4) Mood stabilizers

- A) Lamotrigine-25 mg PO every other day
- B) Topiramate, -25 mg PO OD
- C) Carbamazepine- 400 mg PO OD
- D) Valproic acid- 15 mg / kg PO OD

5) Anti-depressants

- E) Clomipramine- 25 mg PO OD
- F) Bupropion- 150 mg PO OD
- G) Doxepin- 75 mg PO OD
- H) Maprotiline- 75 mg PO OD
- I) Fluoxetine- 20mg PO OD ^[9, 31, 32].

Guidelines

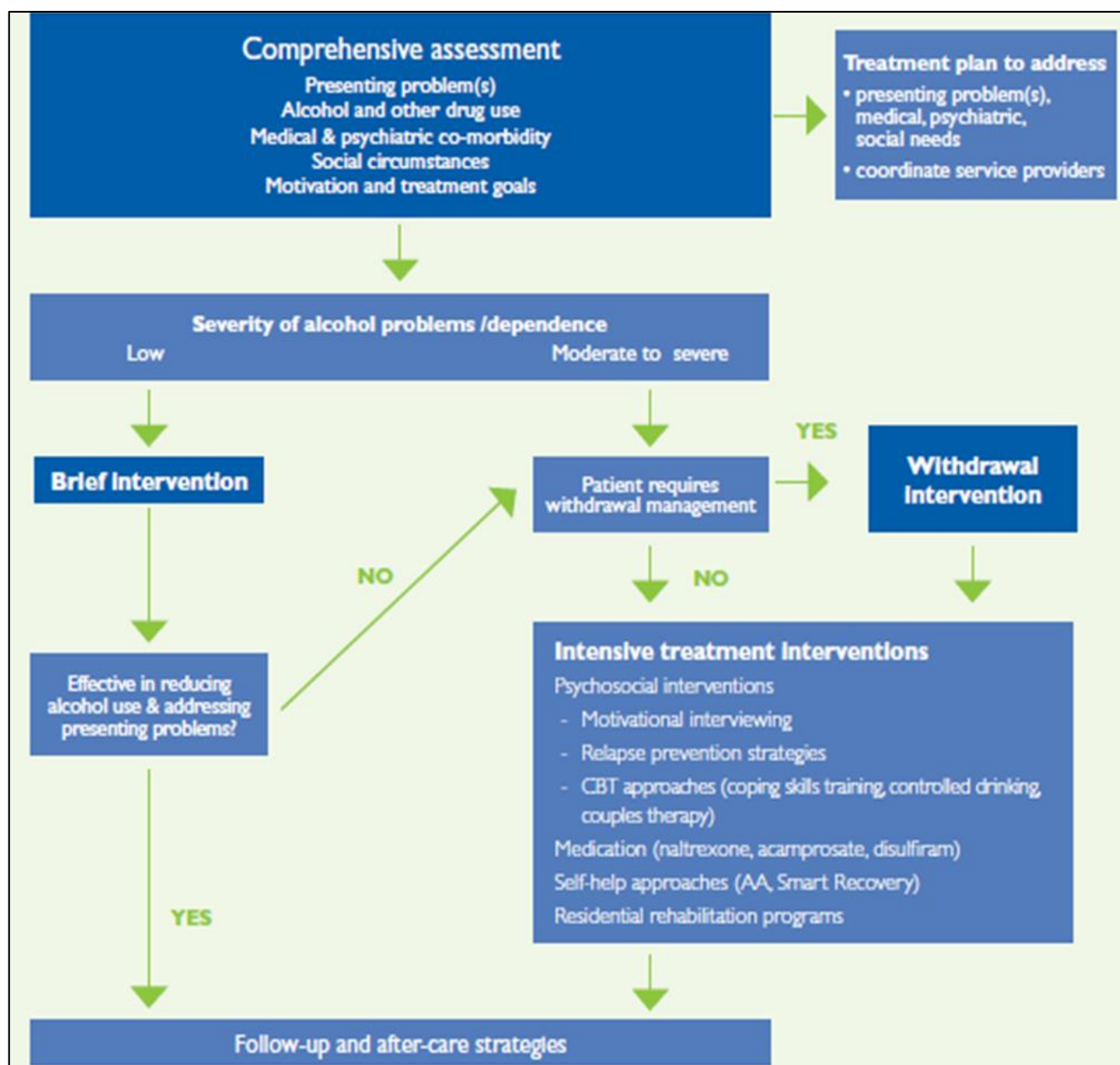


Fig 7: Guidelines for management of alcohol dependence syndrome ^[34]

Patient counselling

Patient counselling is performed by various approaches such as:

- Aversion therapy
- Psychotherapy
- Group therapy
- Alcoholic anonymous
- Behaviour therapy
- Cue therapy
- Relaxation training
- Contingency management
- Skills training
- Cognitive behavioral therapy
- Family & Couple therapy ^[35].

Nicotine dependence syndrome

Definition: A cluster of behavioral, cognitive, and physiological phenomena that develop after repeated nicotine

use and that typically include a strong desire to take the nicotine, difficulties in controlling its use, persisting in its use. Despite harmful consequences, a higher priority given to nicotine abuse than the other activities and obligations, increased tolerance, and sometimes a physical withdrawal state ^[28].

Epidemiology: In 2015, over 1.1 billion people smoked tobacco. Almost 30 % of the Indian population older than age 15 years uses some form of tobacco ^[36].

Etiology ^[37].

Presence of a smoker in the household
Easy access to cigarettes
Perceived parental approval of smoking
Comorbid stress and psychiatric disorders
Low self-esteem/self-worth
Poor academic performance
Boys: High levels of aggression and rebelliousness
Girls: Preoccupation with weight and body image

Forms of tobacco ^[31]

Smoked
Cigarettes
Cigars
Beedies
Pipes
Kreteks

Smokeless

Chewing tobacco
Moist & Dry snuff

Pathophysiology

1. Nicotine produces paradoxical effects, acting as both a stimulant and a depressant.
2. As a stimulant, it has been shown to increase attention, memory, information processing, and learning.
3. It has also been shown to alleviate anxiety, depression, and pain.
4. Nicotine stimulates the Dopaminergic pathways of the mesolimbic system in the brain, an area that is involved in reinforcement for other drugs of abuse.
5. It binds to the nicotinic acetylcholine receptors in the brain (nAChRs), causing the release of dopamine in the nucleus accumbens and the subsequent release of neurotransmitters, resulting in a variety of physiological effects, including behavioral arousal and neural activation.
6. Release of dopamine, norepinephrine, and serotonin is associated with pleasurable feelings and also with appetite suppression.
7. The excess release of acetylcholine associated with

nicotine consumption is related to improved attention, increased vigilance in the performance of repetitive tasks, and memory improvements ^[31, 32].

Diagnostic tests

DSM IV TR Scale

Dependence is a maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same

12-month period

Tolerance, as defined by either a need for markedly increased amounts of the substance to achieve the desired effect, or markedly diminished effect with continued use of the same amount of substance.

- 1) Withdrawal, as manifested by either the characteristic withdrawal syndrome for the substance, or the substance being taken to relieve or avoid withdrawal symptoms.
- 2) Taking larger amounts of the substance or over a longer period than was intended.
- 3) A persistent desire for or unsuccessful efforts to cut down on substance use.
- 4) A great deal of time being spent in activities necessary to obtain or use a substance.
- 5) Abandonment or reduction of important social, occupational, or recreational activities because of substance abuse ^[38].

Severity



Fig 8: Severity of nicotine dependence ^[38]

Stage - 1 (PC) = Precontemplation.

Stage - 2 (C) = Contemplation.

Stage - 3 (PR) = Preparation.

Stage - 4 (A) = Action.

Stage - 5 (M) = Maintenance ^[38]

Pharmacotherapy

Nicotine Replacement therapy

Gums: 4 mg SL OD.

Patch: 7 mg EA OD.

Inhaler: 0.5 mg (2 doses / hour).

Second-line medications

Bupropion: 150 mg PO BD.

Clonidine: 0.2 mg PO OD.

Nortriptyline: 75 mg PO OD.

Varenicline: 1 mg PO OD ^[9].

Guidelines

(5Rs: Relevance, Risks, Rewards, Road blocks, Repetitions)

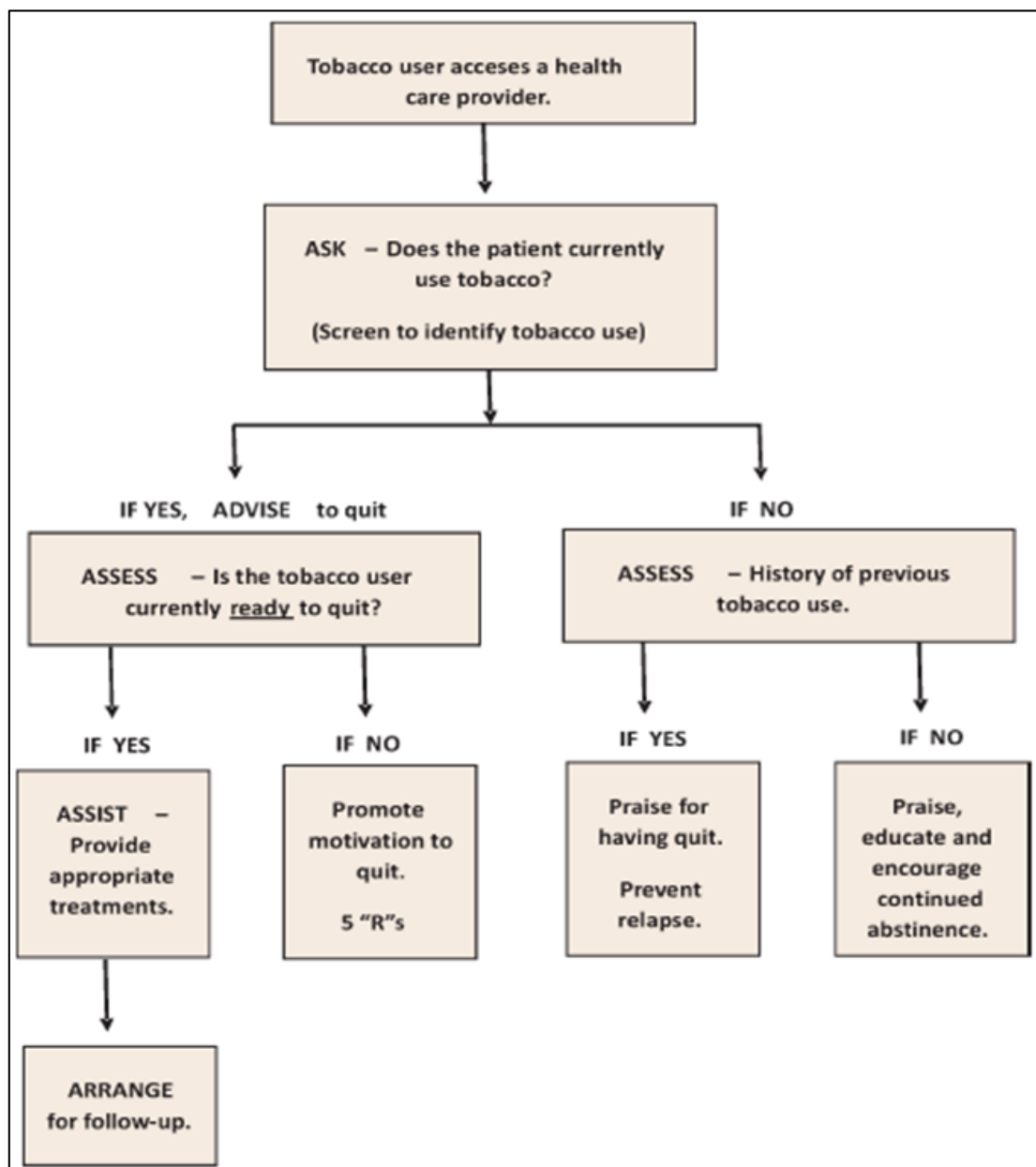


Fig 9: Guidelines for management of alcohol dependence syndrome [39].

Patient counselling

Patient counselling is performed by various approaches such as:

1. **Unassisted method:** “Cold Turkey” is the mostly frequently used method.
2. **Substitute for cigarette:** Electronic cigarette shaped like a cigar or cigarette to satisfy the habitual tactile craving significantly decrease tar & carbon monoxide.
3. **Alternative approach:** Acupuncture, aromatherapy, hypnosis, herbs.
4. **Method used for children and adolescents:** Motivational enhancement, psychological support, and youth anti-tobacco activities such as sports involvement, family communication; school-based curricula such as life-skills training are promising.
5. **Biochemical feedback:** estimation of carbon monoxide, cotinine level during follow-up.
6. **Cut-down to quit:** Gradual reduction involves slowly reducing one’s daily need of nicotine by applying NicoBloc drops on cigarette filter which absorbs 99% of tar and nicotine [40].

Link between psychiatric disorders and alcohol dependence

Four (non-exclusive) hypotheses that can be used to explain the relationship are:

- A. The substance use disorder and another mental health disorder may represent two or more independent conditions.
In such circumstances, both disorders may occur through chance alone or as a consequence of the same predisposing factors (e.g. stress, personality, childhood environment, genetic influences) that affect the risk for several conditions. Substance use disorders and other psychiatric disorders may represent different symptomatic expressions of similar pre-existing neurobiological abnormalities. Some studies had revealed the key roles of biological and genetic or epigenetic factors in the patient’s vulnerability to the manifestations. But it has always to be considered that genes and neural bases are linked with the environment.
- B. The psychiatric disorder other than substance use disorder is a risk factor for drug use and the development

of a co morbid substance use disorder.

In this situation, different pathways can be considered. In the 'self-medication hypothesis', the substance use disorder develops as a result of attempts by the patient to deal with problems associated with the mental health disorder (e.g. Schizophrenia, Depression, GAD). In this case, the substance use disorder might become a long-term problem, or the excessive use of alcohol or an illicit drug might abate when the pre-existing mental health disorder is addressed appropriately. However, the psychiatric disorder could increase the risk of overt and rhythmic use of substances, leading to the development of a substance use disorder that might continue even when the pre-existing psychiatric condition is appropriately treated or remits.

- C. The substance use disorder could trigger the development of a psychiatric disorder in such a way that the additional disorder then runs an independent course.

Drug use can function as a trigger for an underlying long-term disorder. It is the most important process underlying the association between cannabis use and schizophrenia. It is well known that cannabis use in vulnerable adolescents can facilitate the development of a psychosis that runs as an independent illness.

- D. The temporary psychiatric disorder is produced as a consequence of intoxication with, or withdrawal from, a specific type of substance, also called a substance-induced disorder.

Momentary psychiatric conditions (e.g. psychosis with features resembling schizophrenia) may be manifested as a consequence of intoxication with specific types of substances (e.g. stimulants, such as amphetamines and cocaine) or withdrawal conditions (e.g. depressive syndromes associated with the cessation of stimulant use). The latest evidence of similar patterns of comorbidity and risk factors in individuals with substance-induced disorder and those with independent non-substance-induced psychiatric symptoms suggests that the two conditions may share underlying causal or etiological factors. Furthermore, there are some other studies suggests that, previous induced disorders have been diagnosed as independent disorders after a follow-up period. These findings suggest that substance-induced disorders may be a transitory state prior to an independent disorder ^[41].

Situations where the psychiatric disorder is precipitated:

1) In Anxiety Patients

A. It occurs by 2 mechanisms

A. Underlying anxiety leads to increased alcohol use, which changes the physiology of the brain and that in turn leads to a depletion of the neurotransmitters to reduce anxiety naturally. Hence, the individual feels more anxious and needs more alcohol to 'numb' their anxiety. On the long-term effect, this can lead to an individual becoming tolerant of alcohol – that is, they need increasingly large amounts of drink to experience the same reduction in their anxiety.

B. Patients use alcohol to self-medicate is that it is difficult to maintain exactly the amount of alcohol needed to reduce the negative feelings.

Keeping the optimum balance of alcohol to reduce anxiety is almost impossible because the effect of alcohol on the brain is such that after the initial 'euphoria' or stimulation from the first drink, alcohol acts as a depressant and the feelings of anxiety may rapidly return. Increased drinking to cope with

those feelings leads to a rapid increase in the levels of alcohol in the blood and may become counter-productive.

2) In depression patients

Regular drinking changes the chemistry of the brain and, of particularly, depletes the levels of the neurotransmitter serotonin. This leads to the cyclical process of drinking to relieve depression, becoming more depressed as levels of serotonin become more depleted, thus needing more alcohol to medicate the depression. Increased alcohol consumption can also affect social relationships and work life, which in turn can contribute to depression ^[42].

5. Conclusion

Social habits are highly prevalent in psychiatric patients. Detoxification and Rehabilitation should be provided to these patients. Motives for the addiction towards these social habits should be evaluated. It is necessary to abstain from social habits to prevent further complications. Clinical Pharmacist plays a major role by screening and risk assessment in psychiatric disorders, conducting comprehensive programmes & educating other health care professionals and care takers.

Ach: Acetyl Choline

ACTH: Adreno Cortico Trophic Hormone

ADS: Alcohol Dependence Syndrome

APA: Anti-Psychotic Agent

BD: Twice daily

BDNF: Brain Derived Neurotrophic Factor

BZD: Benzodiazepine

CBT: Cognitive Behavioral Therapy

CNS: Central Nervous System

COPD: Chronic Obstructive Pulmonary Diseases

CRF: Corticotropin Releasing Hormone

DA: Dopamine

DSM: Diagnostic Statistical Manual

EA: External Application

ECT: Electro Convulsive Therapy

FGA: First Generation Anti-psychotic

GABA: Gamma Amino Butyric Acid.

NDS: Nicotine Dependence Syndrome

NA: Nor adrenaline

Acronyms

NaSSA: Noradrenergic and Specific Serotonin Antidepressant

NE: Norepinephrine

NMDA: N-methyl-D-aspartate

OD: Once daily

PMHx: Past Medical History

PO: Per Oral

QAM: Every Morning

QD: Four times a day

QHS: Every other Day

QOL: Quality of Life

SGA: Second Generation Anti-psychotic

SL: Sub Lingual

SNRI: Selective nor epinephrine Reuptake Inhibitor

SSRI: Selective Serotonin Reuptake Inhibitor

TCA: Tricyclic antidepressant

TD: Thrice daily

TH: Thyroid Hormone

VTA: Ventral Tegmental Area

5-HT: 5-Hydroxytryptamine

References

1. Mental health. https://www.who.int/features/factfiles/mental_health/en/. 2019.
2. Dan J. Stein, Katharine A. Phillips, Derek Bolton, K.W.M Fulford, John Z. Sadler, and Kenneth S. Kendler. What is a Mental/Psychiatric Disorder? From DSM-IV to DSM-V. *Psychol. Med.* 2010; 40(11):1759-1765.
3. Substance abuse. https://www.who.int/topics/substance_abuse/en/. 2019.
4. Staff of U.S. Pharmacist Journal. Schizophrenia. *U.S. Pharmacist*. 2011; 36(11):25-26.
5. Fiona J Charlson, Alize J Ferrari, Damian F Santomauro, Sandra Diminic, Emily Stockings, James G Scott *et al.* Global Epidemiology and Burden of Schizophrenia: Findings from the Global Burden of Disease Study. *Schizophrenia Bulletin*. 2018; 44(6):1195-1203.
6. Roshan Lal Dewangan, Promila Singh, Tanmay Mahapatra, Sanchita Mahapatra. Demographic and clinical correlates of social cognition in schizophrenia: Observation from India. *Indian Journal of Psychological Medicine*. 2019; 40(2):143-155.
7. Krishna R Patel, Pharm D RPH, Jessica Cherian, Pharm D RPH, Kunj Gohil, Pharm D RPH, Dylan Atkinson. Schizophrenia: Overview and Treatment Options. *Pharmacy and Therapeutics*. 2014; 39(9):638-645.
8. HP Rang, MM Dale, JM Ritter, RJ Flower, G Henderson, Rang Dale's Pharmacology. Seventh Edition. Elsevier, China. 2012; 1:531-607.
9. Joseph T DiPiro, Pharm D FCCP, Robert L Talbert, Pharm D FCCP, BCPS CLS, Gary C Yee *et al.* Pharmacotherapy A Pathophysiologic Approach, Seventh Edition. Volume I, The McGraw-Hill Companies, United States of America, 2008, 1021-1201.
10. Kiran Panesar, BPharm S, Pharm SR, CP Schizophrenia Managing Symptoms With Antipsychotics. *U.S. Pharmacist*. 2012, 37(11):1-12.
11. Tami Argo, M Lynn Crismon, Alexander L Miller, Troy A Moore, Sherrie D Bende, Brandon Suehs. Schizophrenia Clinician's manual. Texas Department of Health Services, 2008.
12. William Spaulding, Jeffrey Nolting. Psychotherapy for Schizophrenia in the Year 2030: Prognosis and Prognostication. *Schizophrenia Bulletin*. 2006; 32(1):94-105.
13. Netsanet Fekadul, Workineh Shibeshi, Ephrem Engidawork. Major Depressive Disorder: Pathophysiology and Clinical Management. *Journal of Depression and Anxiety*. 2017; 6(1):1-7.
14. Depression. <https://www.who.int/news-room/fact-sheets/detail/depression>, 2019.
15. Gangadhar Gururaj, Senthil Amudhan, Santosh Loganathan, Arvind. Depression in India – Let's talk, 1st edition, World Health Organization, Geneva. 2017; 1:1-38.
16. Al Qahtani A, Al Qahtani N. Prevention of Depression: A Review of Literature, *Journal of depression and anxiety*. 2017; 6(4):1-6.
17. Mary Anne Koda-Kimble, Lloyd Yee Young, Brian K Alldredge, Robin L Corelli, B Joseph Guglielmo, Wayne A Kradjan *et al.* Applied therapeutics: The clinical use of drugs, Ninth edition, Lippincott Williams & Wilkins, California, 200; 76(1):82-23.
18. Elizabeth A O'Connor, Evelyn P Whitlock, Bradley Gaynes, Tracy L Beil. Screening for Depression in Adults and Older Adults in Primary Care: An Updated Systematic Review. *Evidence Synthesis*, 2009, 1-161.
19. Alan J Gelenberg, Marlene P Freeman, John C Markowitz, Jerrold F Rosenbaum, Michael E Thase, Madhukar H Trivedi, *et al.* Practice Guideline for the Treatment of Patients With Major Depressive Disorder, 2010, 1-152.
20. Christine Knauss, Margot J Schofield. A Resource for Counsellors and Psychotherapists Working with Clients Suffering from Depression, 2009; 1-27.
21. Rasim Somer Diler. Panic Disorder in Children and Adolescents. *Yonsei Medical Journal*. 2003; 44(1):174-179.
22. Panic Disorder. <https://www.omicsonline.org/depression-anxiety/panic-disorder-scholarly-journal.php>, 2019.
23. C Barr Taylor, Panic disorder, *British Medical Journal*. 2006; 332:951-955.
24. Samantha Barton, Charlotta Karner, Fatima Salih, David S Baldwin, Steven J Edwards. Clinical effectiveness of interventions for treatment-resistant anxiety in older people: a systematic review. *Health Technology Assessment*. 2014; 18(50):1-59.
25. Jessica L, Sobnosky. Overview and Management of Anxiety Disorders. *U.S. Pharmacist*. 2014; 39(11):56-62.
26. Shiv Gautam, Akhilesh Jain, Manaswi Gautam, Vihang N Vahia, Anita Gautam. Clinical Practice Guidelines for the Management of Generalised Anxiety Disorder (GAD) and Panic Disorder (PD). *Indian J Psychiatry*. 2017; 59(1):67-73.
27. Pompoli A, Furukawa TA, Imai H, Tajika A, Efthimiou O, Salanti G. Psychological therapies for panic disorder with or without agoraphobia in adults: a network meta-analysis (Review). *Wiley*. 2016; 4(11004):1-3.
28. AS Henderson, AS Henderson, Z Lipowski, Shen Yucun, Shen Yu-cun, E Strömberg *et al.* The ICD-10 Classification of Mental and Behavioural Disorders, 11th edition, Volume 1, WHO, Geneva, 2018, 1-267.
29. Alcohol. <https://www.who.int/news-room/fact-sheets/detail/alcohol>, 2019.
30. Atul Ambekar, Rakesh Kumar Chadda, Sudhir Kumar Khandelwal, Ravindra Rao, Ashwani Kumar Mishra, Alok Agrawal. Magnitude of Substance Use in India, 1st Edition, Minister of Social Justice & Empowerment Government of India, New Delhi. 2015; 1:1-68.
31. VV Pillay. Modern Medical Toxicology. 4th Edition, Volume 1, Jaypee Brothers Medical Publishers, 2013, 1-590.
32. Krishan Vij. Textbook of Forensic Medicine and Toxicology, 5th Edition, Elsevier, 2011, 1-593.
33. American Psychiatric Association Committee, Alcohol Use Disorder: A Comparison between DSM-IV and DSM-5, NIH Publication, 2016, 13-7999.
34. Paul Haber, Nicholas Lintzeris, Elizabeth Proude, Olga Lopatko, Quick Reference Guide to the Treatment of Alcohol Problems, Commonwealth of Australia. 2009, 3-5626.
35. Ram Chander Jiloha. Non-Pharmacological Treatment of Alcohol Dependence. *Delhi Psychiatric Journal*. 2007; 10(2):119-122.
36. Soni Preeti, Raut DK. Prevalence and Pattern of Tobacco Consumption in India. *International Research Journal of Social Sciences*. 2012; 1(4):36-43.
37. Laura L Jones1, Ahmed Hashim, Tricia McKeever,

- Derek G Cook, John Britton, Jo Leonardi-Bee. Parental and household smoking and the increased risk of bronchitis, bronchiolitis and other lower respiratory infections in infancy: systematic review and meta-analysis. *Respiratory Research*. 2011; 12(5):1-11.
38. Jonathan M Samet, Soon-Young Yoon. Gender, Women, the Tobacco Epidemic, 1st Edition, WHO, Manila, 2010, 137-149.
39. Rajkumar, Jagdish Kaur, Pratima Murthy, Smita Deshpande, Naseem Shah,. Tobacco Dependence Treatment Guidelines, Ministry of Health and Family Welfare, New Delhi, 2011, 1-22.
40. SM Gawali, SM Vaidya. Quitting smoking – How to go about it. *Journal, Indian Academy of Clinical Medicine*. 2012; 13(4): 311-315.
41. EMCDDA. Co morbidity of substance use and mental health disorders in Europe (Perspectives on drugs), 1st Edition, European Monitoring Centre for Drugs and Drug Addiction, Lisbon. 2016; 1:1-9.
42. Andrew McCulloch, Cheers. Understanding the relationship between alcohol and mental Health, 1st Edition, Volume 1, Mental Health Foundation, London. 2006; 1:1-40.