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Theme:

MENTAL HEALTH DAY

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Dr. D.Y.PATIL COLLEGE
OF PHARMACY,
AKURDI, PUNE

VISION

To impart quality education to the Students and mould them into proactive multifaceted Pharmacists.

MISSION

To establish a centre of Academic excellence and research in Pharmacy Education and thereby produce professionally competent and ethically sound Pharmacist to cater to the needs of the global society.

PROGRAM EDUCATIONAL OBJECTIVES (PEOs)

After graduation students will

1. Reflect critical thinking and problem solving skills through their Pharmaceutical knowledge, expertise and competency in industry, higher studies and research.
2. Practice ethics and values in their profession.
3. Contribute effectively in various fields of social healthcare system.
4. Inculcate leadership and entrepreneurship capabilities through effective communications, appropriate time management and self-upgradation.

EDITORS DESK:

Mr Pavankumar Wankhade

Dr. Nikita Saraswat

Mrs. Kajal Bhagat

The technological advancements, like media and internet, have surely helped the pharmacist patients as well as the clinicians. However, many traditional disease issues, remain the same. Yet the woes of treating the same continue and the pharmacist try to find and develop better ways to alleviate the sufferings of the patient. The advancements in science and technology continue, and different modes of new treatments and diagnostic modalities continue to emerge. Yet the common diseases, historically seen in the patients, are still the subject of medical research, books, journals and the medical industry as a whole. The current issue of the journal highlights on Mental health related disease. In the issue we published the papers about detail information about mental health disorder. Thus, this particular issue will be helpful, for all pharmacist to understand their role better.

-Mr. Pavankumar Wankhade. Assistant Professor. Dr D. Y. Patil College of Pharmacy, Akurdi.

Pharma Tech magazine of current issue emphasize on Global public health days which offer great potential to raise awareness and understanding about health issues and mobilize support for action, from the local community to the international stage. There are many world days observed throughout the year related to specific health issues or conditions. So our students and faculty member wrote article related on Mental Health Day which would be helpful for Pharmacist to improve awareness regarding Global Mental Health Day

-Dr. Nikita Saraswat, Assistant Professor, Dr. D. Y. Patil College of Pharmacy, Akurdi.

The current issue focuses upon the topic of utmost priority that is “Health”, addressing the current need of an hour and spreading awareness about some common diseases, our students and faculty members have put forward some informative articles. World Mental Health Day is an opportunity for people and communities to unite behind the theme “Mental health is a universal human right” to improve knowledge, raise awareness and drive actions that promote and protect everyone's mental health as a universal human right.

-Mrs. Kajal Bhagat , Assistant Professor, Dr. D. Y. Patil College of Pharmacy, Akurdi.

“We would like to express our gratitude and heartfelt thanks to our beloved Principal Dr. Niraj Vyawahare for constant support and motivation. We are also grateful to our Vice Principal Dr. (Mrs). Shilpa Chaudhari, all the teaching, non-teaching staff and our students.”

Our organisation feels special and privileged in presenting this issue. Thank you all once again

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BINGE EATING DISORDER (BED)

Bhagwat Patil, Vishal Mhetre , Dr.Pallavi M. Chaudhari
Department of Pharmaceutics, Dr. D.Y. Patil College of Pharmacy, Akurdi Pune

ABSTRACT

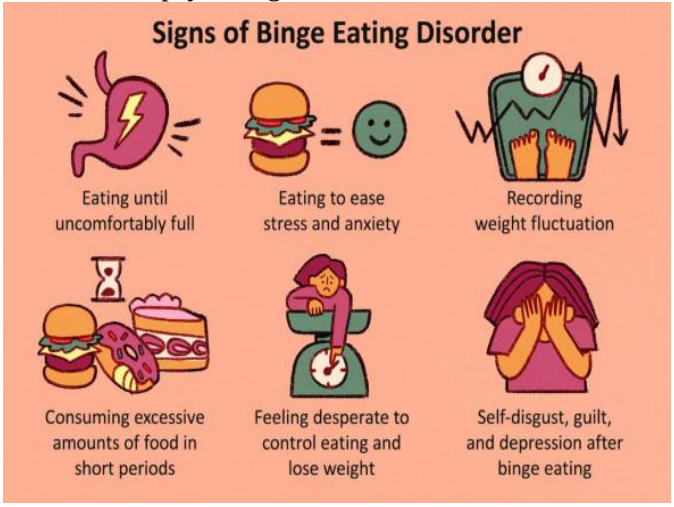
The phrase binge eating was coined over 35 years ago to describe problematic behaviors that occur in a subset of overweight individuals and as a subset of bulimia nervosa. The defining characteristics of patients with BED are the consumption of objectively large amounts of food in discrete periods of time, a lack of control during eating episodes, and a lack of compensatory overeating. BED affects approximately 30% of obese individuals seeking treatment. Binge eaters represent a distinct subgroup among the obese with a higher prevalence of comorbid psychiatric disorders as well as higher rates of pathological eating compared to normal obese women. Several studies have suggested that individuals use binge eating as a means of coping with emotional stress. In addition, binge eating disorder represents a distinct subgroup among bulimia nervosa with lower comorbid psychiatric disorders, eating pathology, and purging behaviors. This paper summarizes this proposed syndrome and discusses whether it is a distinct subgroup of obese patients or a distinct subgroup of patients with bulimia nervosa, or a new diagnostic category. This diagnostic dilemma is discussed in light of the clinical and empirical literature. Clinical and therapeutic implications are presented, and ideas for future research are suggested.

INTRODUCTION

Binge eating disorder (BED) is a condition characterized by episodes of eating larger than normal amounts of food in a short period of time. These episodes occur every week for three months. It is an individual diagnosis different from bulimia nervosa. Binge eating is associated with a variety of psychological and non-psychological problems with some degree of disruption to daily life and several serious disorders. Its comorbid conditions include general health disorders such as obesity, diabetes, hypertension, and chronic pain. It is most common in, but not limited to, obese individuals. A common complaint of people with binge eating disorder is weight gain. The prevalence of this disorder increases with increasing body weight, and obesity is a common comorbidity. [1][2]

ETIOLOGY

Binge eating disorder can result from numerous psychological, social, cultural, and



biological factors. Some of the risk factors for binge eating disorder include.[3][4]

1) Childhood obesity
2) Loss of controlled eating in childhood
3) Perfectionism
4) Conduct problems
5) Substance abuse
6) Family weight concerns and eating problems
7) Family conflicts and parenting problems
8) Parental psychopathology
9) Physical and sexual abuse
10) Mental health impairment

CAUSES AND RISK FACTORS

The exact causes of Binge Eating Disorder (BED) are not fully understood, and it is likely that a combination of genetic, biological, psychological, and environmental factors contribute to its development.[5]

Here are some key factors that may play a role in the onset of BED:

Genetics: There is evidence to suggest a genetic predisposition to eating disorders, including BED. Individuals with a family history of eating disorders may be at a higher risk of developing BED.

1.Biological factors: Imbalances in neurotransmitters, chemicals in the brain that regulate mood and appetite, may contribute to BED. Serotonin, in particular, is thought to play a role in

regulating food intake, and alterations in its function have been linked to binge eating behaviors.[5]

2. Psychological Factors:

Low Self-Esteem: Individuals with BED often struggle with low self-esteem and negative body image, fueling a cycle of emotional distress and binge eating.

Perfectionism: Setting unrealistically high standards and feeling a need to meet them can contribute to stress and trigger binge episodes when these standards aren't achieved.

Mood Disorders: Conditions like depression, anxiety, or other mood disorders are often co-occurring with BED, suggesting a complex interplay between mental health and eating behaviors.

3. Social and Cultural Factors:

Societal Pressures: Cultural ideals emphasizing thinness and certain body types may contribute to body dissatisfaction and a desire to achieve these ideals.

Diet Culture: Constant exposure to dieting messages and weight-loss strategies can lead to a preoccupation with food and body image.

Childhood Trauma: Experiencing abuse, neglect, or other traumatic events during childhood may increase the risk of developing BED.

4.Environmental Factors:

Family Environment: Family attitudes toward food, weight, and appearance can influence an individual's relationship with food.

Childhood Dieting: Early exposure to restrictive diets or messages about body weight and appearance can impact eating behaviors later in life.

Teasing or Bullying: Negative social experiences related to body image, weight, or eating habits may contribute to the development of BED.

5. Dieting and Restrictive Eating:

Repeated Dieting: Engaging in frequent restrictive eating patterns can lead to a cycle of deprivation and overeating.

Weight Stigma: Experiencing discrimination or prejudice based on weight can contribute to emotional distress and dysfunctional eating patterns.

6. Coping Mechanisms:

Emotional Regulation: Using food as a way to cope with stress, anxiety, sadness, or other emotions is a common feature of BED.

TREATMENT

The treatment of Binge Eating Disorder (BED) often involves a combination of therapeutic, medical, and lifestyle interventions. Here are common approaches:[6]

1. Psychotherapy:

Cognitive-Behavioral Therapy (CBT): Focuses on identifying and changing thought patterns and behaviors associated with binge eating. CBT is considered the most effective psychological treatment for BED.

Interpersonal Psychotherapy (IPT): Addresses interpersonal issues and relationships that may contribute to binge eating episodes.

2. Medication:

Selective Serotonin Reuptake Inhibitors (SSRIs): Antidepressants like fluoxetine (Prozac) have been found to be effective in reducing binge eating episodes.

Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs): Medications like venlafaxine (Effexor) may also be prescribed.

3. Nutritional Counseling:

Registered Dietitian: Working with a dietitian can help establish regular eating patterns, promote balanced nutrition, and address any nutritional deficiencies.

4. Self-Help Strategies:

Establishing Regular Eating Patterns: Eating balanced meals at regular intervals can help prevent the cycle of binge eating.

Coping Skills Development: Learning healthier ways to cope with stress and emotions, reducing reliance on food as a coping mechanism.

5. Support Groups:

Group Therapy: Joining support groups or therapy sessions with others experiencing BED can provide a sense of community, understanding, and shared coping strategies.

6. Lifestyle Changes:

Physical Activity: Incorporating regular physical activity, with the guidance of healthcare professionals, can contribute to overall well-being.

Sleep Management: Ensuring adequate and quality sleep can have positive effects on mood and eating behaviors.

DIFFERNTIAL DIAGNOSIS

Diseases that fall under the differential diagnosis of binge eating disorder include:

• Bulimia nervosa - Binge eating disorder differs from bulimia nervosa in that there is no compensatory behavior (laxative abuse, fasting, or self-induced vomiting) after eating to prevent weight gain.[7]

• Anxiety Disorders – Anxiety disorders are also associated with binge eating; However, a patient will only receive a diagnosis of binge eating disorder when they have binging episodes every week for three months.

• Kleine-Lewin syndrome - episodes of binges associated with excessive sleep.

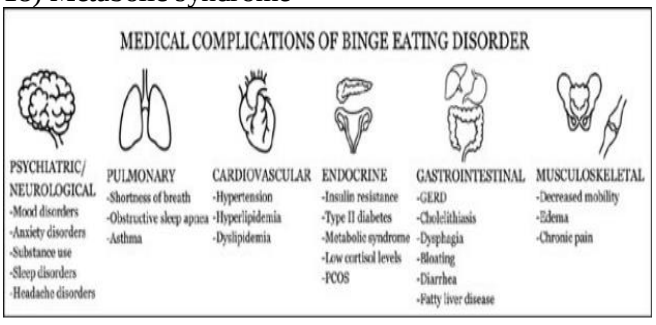
• Mood disorders – Episodes of binges occur with other psychological features of a mood disorder.

Complications in bed

Most patients with binge eating disorder are obese. Binge eating disorder and obesity often coexist and share complications.[8]

Complications of these two disorders include:

1) Muscle pain
2) Pain in neck, shoulder and lower back
3) Impairment resulting from physical health problems after adjustment for BMI
4) Hypertension
5) Diabetes
6) Asthma - Breathing disease
7) Coronary artery diseases and heart failure
8) Hyperlipidemia
9) Weight gain
10) Menstrual disorders (amenorrhea, oligomenorrhea)
11) Imbalance of cortisol hormones (diminished cortisol response to stress test and decreased urinary cortisol levels)
12) Cancer (colon, breast, endometrial, gall bladder and others)
13) Osteoarthritis
14) Sleep apnea
15) Obesity hypoventilation syndrome
16) Non-alcoholic fatty liver disease
17) Gall bladder disease
18) Metabolic syndrome



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BULIMIA NERVOSA DISEASE: A REVIEW

Ms. Bhumika Sonar, Ms. Jayantika Pawar, Mrs. Pranita S. Shankaratti
Department of Pharmaceutical chemistry, Dr. D.Y Patil College of Pharmacy. Akurdi, Pune

ABSTRACT:

Bulimia nervosa is a severe mental disorder followed by episodic binge-eating. Along with efforts to purge all the unnecessary calories through forceful vomiting and excessive compulsive exercise. The person suffering from anxiousness about weight gain is prone to the disease. The person goes through anxiety, fear of gaining weight, preoccupied with body shape, feeling loss of control during eating. Bulimia nervosa is mainly seen in women as compared to men. Usually it starts in adolescence, in the early stage of adulthood. Over-all its treatment revolves around the following: a) through medication and b) through psychological therapy.

INTRODUCTION:

Bulimia nervosa is a condition that happens most usually in juvenile females, portrayed by guilty pleasure in voraciously consuming food, and unseemly compensatory ways of behaving to forestall weight gain. [1,2] The Diagnostic criteria of the diagnostic and statistical manual of mental disorders-5th edition (DSM-V) for bulimia nervosa are as follows:

Episodes of binge eating:

1) Patients are eating segments more critical than what the vast majority would consume in a comparable period (normally under 2 hours) and under similar circumstances.

2) During eating episodes, the patient lets completely go and can't control the servings he consumes. To establish a proper diagnosis, binging episodes should occur once a week for at least 3 months. [3]

ETIOLOGY

The exact cause of bulimia nervosa is unknown, but there are several factors. In particular, abnormalities in the internal functioning of the insula may contribute to the binge eating behaviour associated with this condition. A 2016 study found that patients with anorexia and bulimia nervosa have many serious illnesses and significant changes in the structure and efficiency of white matter connectivity, especially in the power of desire and sweet ways.

Other studies have shown that it is possible to change the function of the brain's internal functional structures. [4]

EPIDEMIOLOGY:

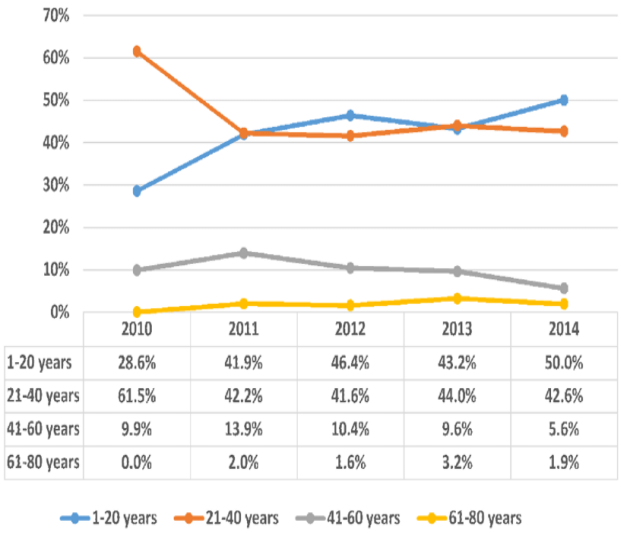
Bulimia nervosa can affect both men and women, but it affects women more frequently. The average age of onset was12.4 times. The frequency of bulimia nervosa in the United States is 0.9 among adolescents,1.5 among ladies, and 0.5 among males. The frequency of bulimia nervosa has not been established in developed countries, but estimates in North America, Australia and Europe range from0.1 to1.3 for men and from0.5 for women to2.0 for women. [5]

HISTORY AND PHYSICAL:

A check of frames in cases with bulimia nervosa shows sore throat, a changeable regular cycle, blockage, migraine, prostration, dormancy, stomach torment, and bulging. While directing an actual test on a case with anatomized or thought bulimia nervosa, get the position, weight, important fleshly functions, and orthostatic blood pressures. It is likewise important to look at a case's skin, mouth, and mid-region. [6] A neurological assessment is pivotal for checking for essential neurological reasons for weight reduction or hurling previous to diagnosing bulimia nervosa. Normal factual test signs related to bulimia nervosa incorporate hypotension, dry skin, parotid organ enlarging, dental decomposition, and calluses on the rearward part of the hand (known as" Russel's sign.") Bulimia nervosa can likewise be related to going bald, edema, and epistaxis. [6]

PREVALENCE:

- Over time, the incidence rates of bulimia nervosa and anorexia nervosa decreased and remained relatively stable, respectively.
- Anorexia nervosa is becoming more common in younger people (less than 15 years old).
- Up to 4% of females and 0.3% of males experience anorexia nervosa during their lifetime, while up to 3% of females and more than 1% of males experience bulimia nervosa.
- According to recent research, the mortality risk is five times higher for both bulimia nervosa and anorexia nervosa.
- Anorexia nervosa and bulimia nervosa do affect men, of all ages, and in non-Western nations in addition to the traditionally studied group of young girls in Western nations.



	2010	2011	2012	2013	2014
1-20 years	28.6%	41.9%	46.4%	43.2%	50.0%
21-40 years	61.5%	42.2%	41.6%	44.0%	42.6%
41-60 years	9.9%	13.9%	10.4%	9.6%	5.6%
61-80 years	0.0%	2.0%	1.6%	3.2%	1.9%

Fig 1: Prevalence of Bulima Nervosa [23]

EVALUATION:

Evaluation of a patient with bulimia nervosa must include:

• Liver function test.

• Urine analysis.

• In extreme events, acquire serum magnesium and phosphorous as well as the electrocardiogram.

• Female patients ought to go through a pregnancy test. Testing for luteinizing hormone, prolactin, beta-HCG, and a follicle-stimulating hormone should be done on female patients with secondary amenorrhea to look for other potential causes of amenorrhea.

• Lab tests are accessible to test for stool or pee bisacodyl, emodin, aloe-emodin, and rhein. Nonetheless, a positive test for a stool or pee diuretic isn't important to lay out the finding. [7,8]

COMPLICATIONS:

A mental illness known as bulimia nervosa has the potential to have serious repercussions. The frequency and mode of purging by the patient can reveal the type and severity of medical issues associated with bulimia nervosa, unlike anorexia nervosa, were complications stem from weight loss and malnourishment. [6,7] Bulimia nervosa associated complications are as follows:

• Hypertrophy of the saliva glands (sialadenosis) and swollen cheeks: these conditions
usually affect the parotid gland, although they can also induce swelling in the submandibular
salivary gland. [16]• Mucosal and submucosal esophageal tears close to the gastroesophageal junction are known as Mallory-Weiss syndrome. This condition can occasionally be preceded by gastroesophageal intussusception, which is brought on by the patient's stomach contracting violently as they vomit. Esophageal rupture associated with Boerhaave syndrome can result from Mallory-Weiss tears. [17,18]

• Barrett's esophagus may become more likely in people with GERD, or gastroesophageal
reflux disease. [18,20]

• Cardiac arrhythmia: self-induced vomiting can result in hypokalemia. One possible cause of QTc prolongation is hypokalemia. Ipecac misuse is also linked to additional cardiac
problems, such as congestive heart failure and even death. [19,22]

• Diabetes: Bulimia nervosa was linked to a relative risk of type 2 diabetes, according to a meta-analysis of ten studies. [21]

TREATMENT AND MANAGEMENT:

The essential target of treatment is a suspension of the pigging out and vomiting conduct. Particular serotonin reuptake inhibitors like fluoxetine, citalopram, and sertraline have been displayed to diminish side effects of bulimia nervosa. Fluoxetine is the main FDA supported medicine for bulimia nervosa. Apparently, a higher portion (60 mg) is essentially better compared to a fake treatment in diminishing the recurrence of gorge and regurgitating episodes.

Proof for other drug classes to treat this condition is restricted. Trazodone has fundamentally decreased the recurrence of gorging episodes when contrasted with fake treatment. Due to their lethality and potential side effects, monoamine oxidase inhibitors and tricyclic antidepressants are reserved for resistant cases. [9] Bupropion ought not be utilized in patients with bulimia nervosa in light of the expanded gamble of epileptic episodes. Topiramate, one of the antiepileptic medications, has been shown to reduce binge eating, but side effects like weight loss and cognitive problems should be closely monitored. Clinical preliminaries of mental social treatment and relational psychotherapy have likewise exhibited an advantage for patients with bulimia nervosa. Patients with bulimia nervosa ought to be evaluated for suicidality and comorbid mental ailment as they are at a higher gamble of other mental illnesses than everyone. [7,8] Numerous general medical issues, such as metabolic alkalosis, dehydration, constipation, and cardiac arrhythmias, can result from bulimia nervosa. In addition to stopping the purging behavior, saline treatment is recommended for fluid volume depletion, which is the most frequent cause of metabolic alkalosis in bulimia nervosa patients. Consider intravascular
administration for inpatients; however, these patients need to be watched for indications of volume

overload. Similar measures are taken to address dehydration linked to bulimia nervosa.

Intravenous saline has no part in the infrequent occurrences of average or increased fluid volume with alkalemia in a bulimia nervosa patient. Exercise, proper hydration, and dietary

fiber are treatments for constipation related to bulimia nervosa or caused by stopping laxatives. [10,11,12] Low doses of lactulose or powdered polyethylene glycol may be administered if laxatives are still required. Consider getting a cardiology consultation if a patient has significant or symptomatic cardiac issues from bulimia nervosa, which are typically brought on by electrolyte imbalances. [13]

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OBSESSIVE COMPULSIVE DISORDER

Ms. Kiran Heera Makhija
Department of Pharmacology, Dr. D.Y Patil College of Pharmacy. Akurdi, Pune

1. Introduction:

Obsessive-compulsive disorder (OCD), once thought to be an uncommon condition, is now acknowledged as a prevalent early-onset brain disorder that can be severely disabling and potentially treatable. [1]

OCD is a mental illness in which sufferers must do routines to ease their discomfort and deal with unwanted thoughts. It would be preferable to refer to them more precisely as obsessive- compulsive spectrum disorders, which are marked by constant behavioral or mental activity that takes up the majority of a person's time in an effort to counteract intrusive mental content. All of these actions are incompatible with the person's ideals or "ego-dystonic".[2]

The cause of obsessive-compulsive disorder (OCD), a very variable and common disorder, is unknown. Like other psychiatric diseases, OCD most likely results from a complex interplay between environmental and genetic risk factors. With obsessive-compulsive disorder (OCD), the person experience undesirable thoughts and feelings on a regular basis (obsessions), which push you to engage in repetitive actions (compulsions). The repetitive activities can seriously hinder everyday duties and social relationships. Typically, medication and psychotherapy are used to treat OCD. The prognosis for OCD is better the earlier it is identified and treated.[1,2]

Obsessions, which are defined as recurring or persistent thoughts, urges, or ideas that are deemed invasive or inappropriate, and compulsions, which appear as repetitive actions or mental activities frequently developed in reaction to an obsession, are typical symptoms of OCD. Fear of contamination, the desire for order or symmetry, pathological doubt, and recurring violent or horrifying visions are a few of the more prevalent obsessions. Washing, checking, counting, organizing, and repeating words or actions are among the most prevalent obsessive activities.[1] To counteract certain obsessive thoughts, an OCD patient with a

contamination obsession, for instance, can wash their skin until it becomes raw for hours on end.

It should come as no surprise that obsessions and compulsions take a lot of time and can seriously impair day-to day activities and professional performance.[3]

The WHO listed OCD within the ten medical illnesses associated with greatest worldwide disability.In The Diagnostic and Statistical Manual of Mental Disorders (DSM-5, which was published by the American Psychiatric Association (APA) in 2013, obsessive-compulsive disorder sits under its own category of obsesive compulsive and related disorders where the following subcategories were placed:[4,5]

- ✓ Obsessive-compulsive disorder (OCD)
- ✓ Body dysmorphic disorder (BDD)
- ✓ Hoarding disunder
- ✓ Trichotillomania
- ✓ Excoriation (skin-picking)
- ✓ disorderSubstance/medication-induced
- ✓ obsessive-compulsive and related disorder
- ✓ Obsessive-compulsive and related disorder as a result of another medical condition
- ✓ Other specified obsessive-compulsive and related disorder
- ✓ Unspecified obsessive-compulsive and related disorder.

2. Epidemiology:

In the US, the lifetime prevalence of OCD is between 2 and 3 percent. OCD onset occurs in two distinct phases: throughout infancy and late adolescence/early adulthood. Just 15% of instances

start beyond the age of 35, and two thirds of cases start before the age of 25. Approximately one-third of cases begin in early adolescence or childhood. Males typically experience a more malignant course and an earlier beginning. Before seeking therapy, symptoms could exist for years, and people who are impacted frequently endure their suffering in silence. 15% of patients experience worsening during the disease's chronic waxing and waning course, while 5% experience episodes of interepisode healing.[5,6,7]
An epidemiological study from India is the only one available. The study discovered a 0.6% lifetime frequency. When compared to the 2-3% rate found in the research conducted inNorth America and Europe, this percentage is significantly lower. However, a Taiwanese study found a similarly low prevalence, ranging from 0.5-0.9%.[8]

3. Etiology:

3.1 Genetic/Familial Theories: Twin research has revealed that concordance rates between monozygotic and dizygotic twins are higher. If a first-degree relative has Tourette's or OCD, the prevalence of OCD is increased. Additionally, there is proof that in certain Tourette's syndrome families, the prevalence of both OCD and Tourette's are higher in the biological relations. This implies that OCD and Tourette's may be different phenotypic manifestations of the same underlying genetic abnormality in these families. [9]

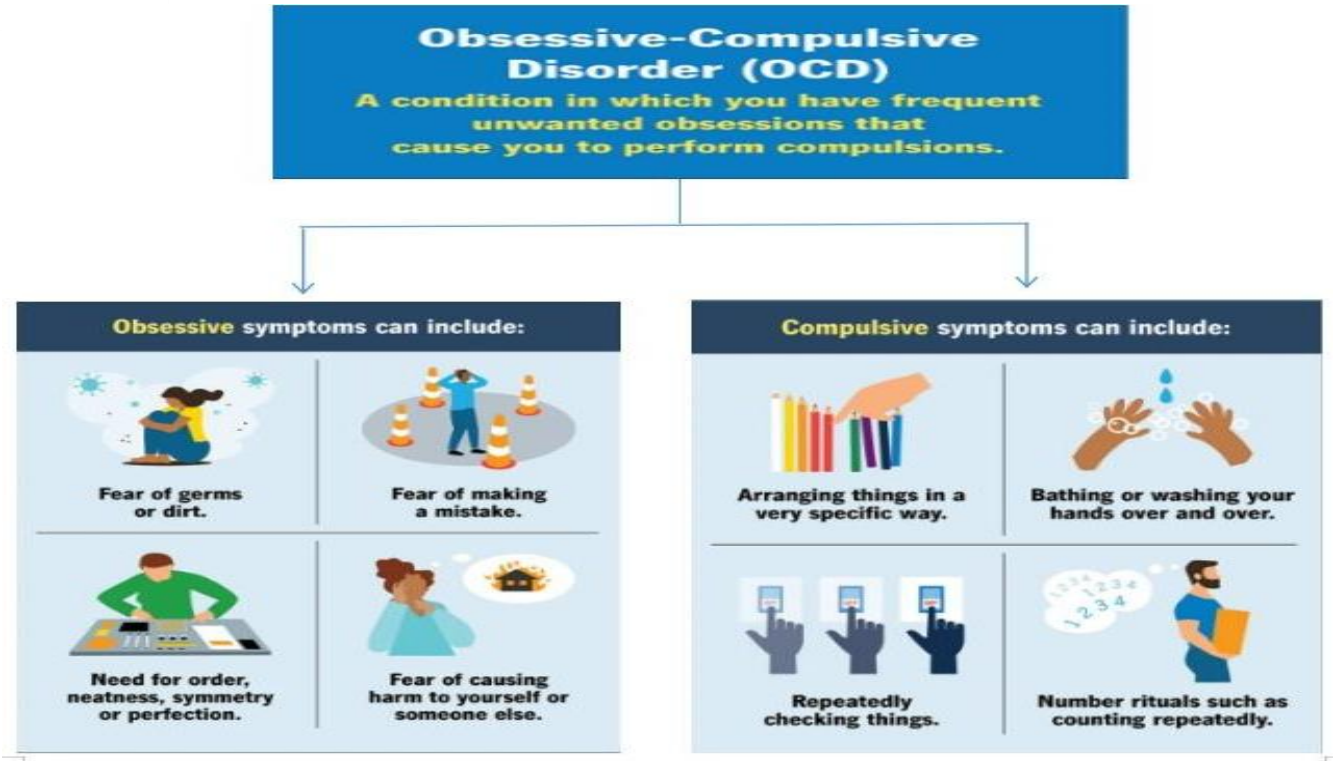
3.2 Theories of Behavior: Classical instrumental conditioning model of OCD in two stages: When anxious thoughts are combined with mental stimulation, obsessions arise.
Compulsions are neutral actions that are encouraged because they have been linked to a decrease in anxiety.

3.3 Neurobiological Theories: A growing body of research from imaging, medication, and behavioral investigations suggests that the pathophysiology of hyperactivity in frontal- subcortical thalamic circuits

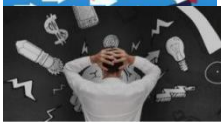
4. Signs & Symptoms of OCD:
The symptoms could be classified as obsessive and compulsive symptoms. In psychology, obsessions are repeated and unwanted thoughts that cause anxiety. Compulsions are repetitive behaviors people perform to temporarily relieve this anxiety.[10]

4.1 Typical Obsessions: [11]
- ✓ Fear of being contaminated or unclean by other people or the surroundings
- ✓ Fear of catching a sickness or infection, such as AIDS
- ✓ Fear that a disaster will occur if something is not done in a certain manner.
- ✓ Fear of breaking the law.
- ✓ Recurring distressing sexual thoughts or images
- ✓ Fear of thinking sinful or blasphemous thoughts
- ✓ Fear of blurting out insults or profanity
- ✓ Extreme concern with order, symmetry or exactness
- ✓ Recurrent intrusive thoughts of certain sounds, images, words or numbers
- ✓ Intense need to know or remember

4.2 Fear of losing/discarding something important
Typical Compulsions:
- ✓ Overdosing or customary hand washing
- ✓ Prolonged or ritualized showering, brushing teeth, or toilet routine
- ✓ Dressing and undressing repeatedly
- ✓ Repeated cleaning of household objects
- ✓ A need to put things in a specific order or arrangement
- ✓ Repeatedly checking locks, switches, faucets, appliances
- ✓ confirming that their acts have not caused harm to anyone
- ✓ Need to tell, ask, confess
- ✓ Repeating certain actions (e.g. going through doors)
- ✓ Checking that the patient did not make a mistake
- ✓ Constant seeking of approval or reassurance
- ✓ Touching certain objects in a particular way
- ✓ Repeated counting to a certain number or a multiple of that number.
- ✓ Hoarding useless objects

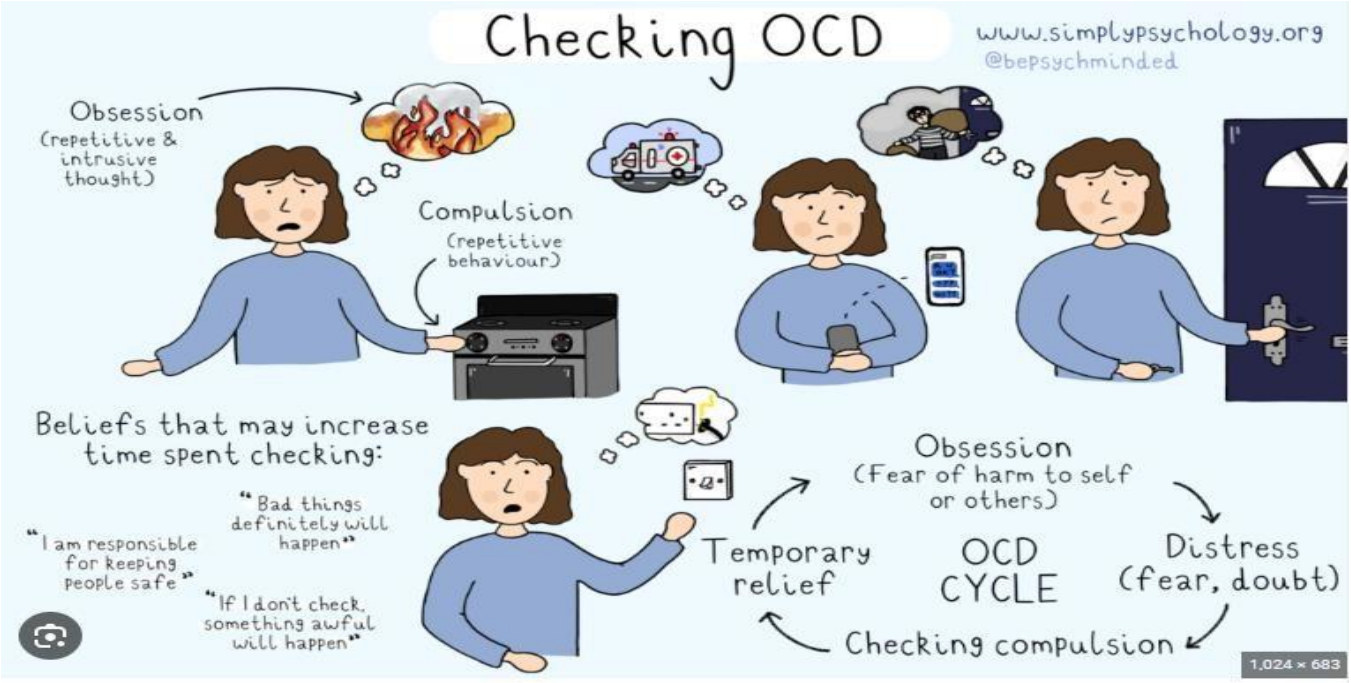


4.3. Early Signs of OCD: [11,12,13]

Repetitive Behaviours: Repeating actions until they are "JUST RIGHT" or starting the things or tasks all over again to get it done more perfectly.	
Rule-Driven: Maintaining a rigid system that dictates your behaviour. Having feelings like the things have to be done in certainorder and in certain amount of time.	
Resistant to Change: Finds it difficult/distressing when things don't go as expected.	
Uncontrollable Intrusive Thoughts: Unwanted ideas, cravings, or pictures that keeps coming back.	
Simple Routine tasks becomes time-consuming: Simple day-to-day tasks seems to be time consuming because ofspending to much time on activities such as washing hands, leaving the house etc.	
Excessive Worries and Doubts: Always having feelings of extreme fears about bad things happening or doing something wrong. Fear of doing something wrong.	
Reassurance Seeking or Re-checking: Excessive checking of things, such as ensuring that the doors are locked multiple times. Finds difficult to tolerate uncertainty.	

5. Diagnosis:

There is no specific test available for the diagnosis of OCD but it frequently coexists with other conditions including depression (>50%), dysthymia, anxiety disorders such panic disorder and social phobia, problems related to eating and hypochondria. Schizophrenia frequently manifests as OCD symptoms as well. There is a significant overlap with other repetitive behaviors, like Tourette's Syndrome (of which less than 50%have OCD but 25% also have tics). [14,15] The hallmark of obsessive-compulsive personality disorder (OCPD) is a strict obsession with rules, sometimes to the point that people miss the purpose of an activity. In individuals, OCPD is typically exhibited without OCD and vice versa; however, in families, OCD and OCPD may coexist. [16,17]The healthcare professionals confirms the OCD on the basis of signs, symptoms and medical and mental health history. The doctors usually use Diagnostic and Statistical Manualof Mental Disorders (DSM-V) to diagnose OCD. [17,18]



6. Management & Treatment Options for OCD:

6.1 First-line treatment:

Psychotherapy:
Psychotherapy, also referred to as talk therapy, is a word used to describe a range of therapeutic approaches intended to assist you in recognizing feelings, ideas, and behaviors. [19]

Cognitive Behavioral Therapy (CBT): With CBT, a therapist will work with you to analyze and comprehend your feelings and ideas. CBT can help cease negative habits and damaging beliefs over the course of several sessions, possibly substituting healthier coping mechanisms. [20]

Exposure and Response Prevention (ERP):
CBT is one kind of ERP. A therapist exposes you to the scenarios or pictures that make you anxious during ERP, and you are asked to fight the want to carry out a compulsion.[21] Your therapist might, for instance, ask you to handle unclean objects but forbid you from washing your hands afterward. You discover that your nervous ideas are only that thoughts and not always reality if you remain in the dreaded circumstance without anything unfavorable happening. [22,23]

Pharmacotherapy: American Psychiatric Association (APA) Practice Guidelines classify serotoninergic drugs as a first-line treatment for OCD, along with cognitive behavioral therapy (CBT) and SSRIs. Numerous pieces of evidence support the links between OCD and serotonergic dysfunction. [24]

6.2. Second-line Treatment:
Clomipramine[24,25] Venlafaxine

6.3. Add-on Treatment: [24,25]
Anti-depressants Anti-dopaminergics
Glutamatergic Agents.

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Personality Disorder: Crack-up craziness delusions depression

Mrs. Kajal Bhagat
Department of Pharmacognosy, Dr. D.Y Patil College of Pharmacy. Akurdi, Pune

Abstract:
Personality disorder is a group of mental illness. Recent research o contributions show the individuals being suffering from long lasting, the behaviour is done non consciously[8], misinterpretation between the real life, stubborn, intense change in a person's emotional state, no alertness, overthinking and misrepresented. The individual is not treated at earlier stage this leads to psychologically damage, high risk of suicide and depression. Unable to recollect the negative pattern is going on. There are specific disorders of personality disorders- Paranoid, Schizoid, Schizotypal, Antisocial, Borderline, Histrionic, Narcissistic, Avoidant, Dependent and Obsessive–compulsive personality disorder. The earliest origins of personality disorders studied and elaborated by Hippocrates and further it carried by modern foundational works of Pritchard, Schneider, and Horney2. The studies and research are still going on and as such clear data is not available. People with personality disorders often have a hard time understanding emotions and tolerating distress. And they act impulsively. The treatment should be taken once it is detected because this makes it hard for them to relate to others, causing serious issues, and affecting their family life, social activities, work and school performance, and overall quality of life[1].

Keywords: Diagnosis, personality disorder, treatment

1. Introduction:

The term "Personality Disorder" implies there is something not-quite-right about someone's personality. The most noticeable and significant feature of these disorders is their negative effect on interpersonal relationships. A person with an untreated personality disorder is rarely able to enjoy sustained, meaningful, and rewarding relationships with others, and any relationships they do form are often fraught with problems and difficulties. To be diagnosed with a "personality disorder" does not mean that someone's personality is fatally flawed or that they represent some freak of nature. Many types of disorders are evidenced by a complete and total deviation from normal and healthy functioning (e.g., epilepsy). However, personality disorders cannot be understood independently from healthy personalities. Since everyone has a personality (but not everyone has epileptic seizures), personality disorders reflect a variant form of normal, healthy personality. Thus, a personality disorder exists as a special case of a normal, healthy personality in much the same way as a square is a special case of the more general construct of a rectangle[3].

2. History and evolution:

The history of personality disorders described by the Hippocrates in 1952, about 400 BC, about the four fundamental body fluids mainly focused on personality patterns. His theory was physiologically based, and also associated with environmental features like climate and temperature. The modern history of personality disorders further appraised by Pritchard. In 1835 he delicately described what is now the antisocial personality disorder followed by the modern foundational works of Pritchard, Schneider, and Horney.

Continuously the research carried out and by looking to the current situation of personality disorder the Schneider in 1950,he has first published his taxonomy in 1923. He indicated that the present Axis II disorders into many respects. First, he did not view ‘‘psychopathology personalities’’ as necessary precursors or it may lead to severe mental disturbances but he noticed that it has coexistent entities. Second, developing in childhood and continuing into adulthood. Third, observed different psychopathologic personalities in psychiatric individuals.such as depression, anankastic, attention-seeking, labile, and affectionless. The five versions of the Diagnostic and Statistical Manual of Mental Disorders (DSM), first published in 1952. Complete

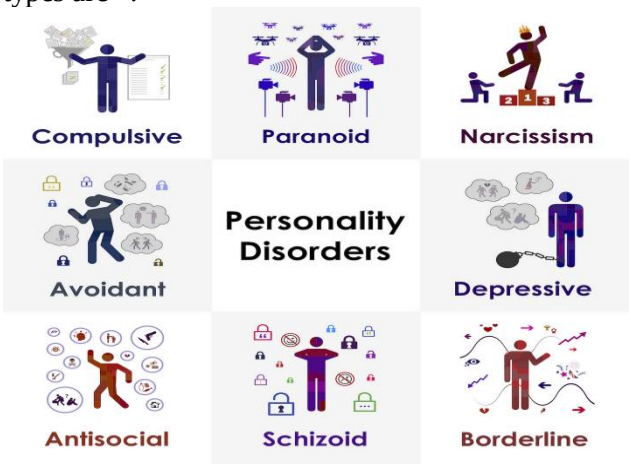
historical overview of the European origins of personality disorders by Berrios in 1993. Moreover from the recent studies many of individuals have several personality disorder diagnoses.

3. Literature survey:

In the region of 50% of individuals are solicited by personality disorders. 63% in a depressed elderly mixed inpatient and outpatient sample and lifetime rate of personality disorders is between 10% and 13%. These were based on community surveys and large samples of relatives and through various methods including self-report inventories and clinical interviews. In our recent study, we assessed personality disorders in young and old chronically mentally ill inpatients and found high prevalence rates for both groups: 66% for the younger sample and 58% for the older sample. Personality disorders are also difficult to diagnose properly. In fact, agreement between diagnosticians has been scandalously poor for personality disorders and the changing nature of the taxonomy of this particular form of psychopathology[2].

4. Types of personality disorder:

Some of the most common Personality Disorders are discussed below. There are 10 types of personality disorders. They are grouped into three different categories called clusters. The clusters and types are[3]:



4.1.Cluster A personality disorders involve unusual and odd thoughts and behaviors[3]. It includes:

- ✓ **Paranoid personality disorder,** in which a person has paranoia (an extreme fear and distrust of others). They may think that someone is trying to harm them.
- ✓ **Schizoid personality disorder,** in which a person prefers to be alone and is not interested in having relationships with others.
- ✓ **Schizotypal personality disorder,** in which a person has unusual thoughts and ways of behaving and speaking. They are uncomfortable having close relationships with others.

4.2.Cluster B personality disorders involve dramatic and emotional thoughts and behaviors that can keep changing. It includes:

- ✓ **Antisocial personality disorder,** in which a person has a long-term pattern of manipulating, exploiting, or violating the rights of others.
- ✓ **Borderline personality disorder,** in which a person has lots of trouble managing their emotions. This makes them impulsive and uncertain about how they see themselves. It can cause a lot of trouble in their relationships.
- ✓ **Histrionic personality disorder,** in which a person is dramatic, has strong emotions, and always wants attention from others.
- ✓ **Narcissistic personality disorder,** in which a person lacks empathy and wants to be admired by others. They think that they are better than others and that they deserve special treatment.

4.3. Cluster C personality disorders involve anxious and fearful thoughts and behaviors. It includes:

- ✓ **Avoidant personality disorder,** in which a person is very shy and feels that they are not as good as others. They often avoid people because they fear rejection.
- ✓ **Dependent personality disorder,** in which a person depends too much on others and feels that they need to be taken care of. They may let others treat them badly because they are afraid of losing the relationship.
- ✓ **Obsessive-compulsive personality disorder,** in which a person needs control and order. They are perfectionists and can be inflexible. Although some of the symptoms are similar, this is not the same thing as obsessive-compulsive disorder (OCD).

5. Causes:

Most experts agree that there is no single causes of personality disorder, it is likely to caused by a combination of factors. Personality disorders usually begin when someone is in their teens or early adult years. The cause is unknown. However, genes and childhood experiences such as abuse and trauma.

- ✓ **Genetics-** genes inherited may make people more vulnerable, given certain environmental factor.
- ✓ **Neurotransmitters-** neurotransmitters can have a significant effect on mood and behaviour.
- ✓ **Neurobiology-** study of the nervous system and how the brain works
- ✓ **Environmental factors-** events that happened in a persons past like family, abuse, lack of validation.

In the past, some believed that people with personality disorders were just lazy or even evil. But new research has begun to explore such potential causes as genetics, parenting and peer influences.

6. Signs and Symptoms:

6.1.Common signs of a personality disorder include:

- ✓ Strange or unpredictable behaviour.
- ✓ Suspicion and distrust (not trusting others)taking risks.
- ✓ Extreme mood swings or emotional outbursts.
- ✓ Difficulty with relationships.
- ✓ Problems at school or work avoiding other people
- ✓ Need for instant gratification (immediate pleasure or reward)
- ✓ Feeling empty and emotionally disconnected
- ✓ Difficulty[7] maintaining stable and close relationships, especially with partners, children and professional carers
- ✓ Periods of losing contact with reality
- ✓ Being[7] overwhelmed by negative feelings like distress, anxiety, worthlessness or anger.

7. Diagnosis:

- A physical exam. Your doctor may do a physical exam and ask questions about your health.
- A mental health evaluation. Your doctor may refer you to a mental health professional.
- Comparing your symptoms to standard guidelines.
- Neuropsychological testing.
-International Personality Disorder Examination.
- NEO Five-Factor Inventory.
- Thematic Apperception Test.
- Global Assessment of Functioning scale.
- Adult Attachment Interview

8.Treatment:

- ✓ **Psychotherapy**

Psychotherapy, or talk therapy, may help in managing personality disorders. During psychotherapy, you and a therapist can discuss your condition, as well as your feelings and thoughts. This can provide you with insight on how to manage your symptoms and behaviors that interfere with your daily life. There are many types of psychotherapy. Dialectical behavior therapy can include group and individual sessions where people learn how to tolerate stress and improve relationships. Cognitive behavioral therapy aims to teach people how to change negative thinking patterns so they can better cope with everyday challenges.

- ✓ **Medications**

There aren't any medications approved for the treatment of personality disorders. However, certain types of prescription medications might be helpful in reducing various personality disorder symptoms, such as:
, which can help improve a depressed mood, anger, or impulsivity

- ✧ mood stabilizers, which prevent intense mood changes and reduce irritability and aggression
- ✧ antipsychotic medications, also known as neuroleptics, which can help reduce symptoms of psychosis like hallucinations and delusions
- ✧ anti-anxiety medications, which can help relieve anxiety, agitation, and insomnia.

8. Risk factors:

Although the specific causes of personality disorders are not known, some factors seem to increase the risk of having one:

- ❖ **Specific personality traits.** This includes always trying to stay away from harm, or the opposite — a strong need to seek out new

activities that get the adrenaline pumping. It also includes poor impulse control.

- **Early life experiences.** This includes a home environment that is not stable, predictable or supportive. It also includes a history of trauma — physical neglect or abuse, emotional neglect or abuse, or sexual abuse.

9. Conclusion:

- Recent technological advancements and improvements to diagnostic methodologies have enabled researchers to study personality and personality disorders as never before.
- The most important aspect of treating a personality disorder is determining the condition in the first place, it will be easier for you to seek and stick with treatment.
- The treatment that works for individuals, should continue for improvement in the symptoms.
- As a result, we now have a much greater understanding of these disorders.
- Furthermore, this research has facilitated the development of several highly effective treatments for personality disorders that are evidenced-based.
- As research continues, these treatment approaches will be further refined.

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PANIC DISORDERS

Ms. Prachi Dhonde, Mrs. Somthane P. N

Dr. D. Y. Patil College of Pharmacy, Akurdi

Introduction:

Anxiety disorders like panic disorder are marked by sudden, recurrent bouts of extreme terror coupled with physical symptoms such dizziness, shortness of breath, chest discomfort, and palpitations. or abdominal distress.1

What causes panic disorder?

Panic disorder is a common mental health problem. Though it can sometimes start in childhood, it usually does so in adolescence or early adulthood. It is twice as common in women as in men. There may be a genetic link. It tends to run in families. Panic disorder may be an overreaction of the body's normal survival instincts and behaviours. In people with panic disorder, the body may be more sensitive to hormones that trigger excited feelings in the body[2].

The debilitating mental health condition known as panic disorder (PD) is characterized by sudden, frequent panic attacks, anxiety about the consequences of the attacks, and behavioral changes brought on by the episodes. PD can occur with or without agoraphobia and is associated with high levels of psychiatric comorbidity and severe role impairment. A number of recent studies have focused on the neurobiology of common psychiatric disorders, including anxiety disorders, and underlying cognitive impairments associated with them. The debilitating mental health condition known as panic disorder (PD) is characterized by sudden, frequent panic attacks, anxiety about the consequences of the attacks, and behavioral changes brought on by the episodes. These impairments may also act as measurable symptoms of underlying neurobiological dysfunction. Several studies have found structural brain abnormalities in patients with anxiety disorders, including patients with PD. Patterns of impairments in executive function have been reported in a number of recent reviews of neuropsychological performance in OCD. Executive function impairments have also been implicated in Post Traumatic Stress Disorder ,however PD has been less well researched.[3]

In PD, imaging studies have indicated abnormalities in specific brain regions compared to controls, including different metabolic activity in the hippocampal and parahippocampal areas and abnormalities in the temporal lobe structures. Brain abnormalities such as these may lead to learning and memory deficits, if present in panic disordered individuals. However Reiman and colleagues have noted that in their work, similar regional blood flow patterns have been seen in panic disordered patients as in healthy controls with anticipatory anxiety. It remains unclear whether abnormalities seen relate to structural differences or effects of state or trait anxiety.[4]

The co-occurrence of anxiety and depression may influence neuropsychological performance. Although anxiety alone was not found to affect memory performance, when anxiety and depression were reported together by veteran participants, a significant negative effect on memory performance was seen. Overall, current research suggests that state or trait anxiety alone in the absence of depression may not have a large impact on neuropsychological test performance in PD.[5]

What are the symptoms of panic disorder?

Panic episodes can also occur in various kinds of anxiety disorders. Panic disorder is often diagnosed if an individual has experienced four or more panic attacks and is always afraid of getting another one. Symptoms of a panic attack may include:[6]

- Pounding heart
- Sweating
- Trembling or shaking
- Shortness of breath
- Sense of choking
- Nausea or belly pain
- Dizziness or light-headedness
- Feeling unreal or disconnected from oneself
- Fear of losing control

- Fear of "going crazy" or dying
- Numbness
- Chills or hot flashes
- Chest pain and other symptoms that mimic a heart attack

Panic disorder can be upsetting and disabling. An attack can last from a few minutes to an hour or sometimes longer.The symptoms of a panic attack may look like other mental health conditions. Always see your healthcare provider for a diagnosis. Individuals with panic disorder usually have a series of intense episodes of extreme anxiety during panic attacks. These episodes usually last ten minutes, though they might endure anywhere from one to five minutes, twenty minutes to over an hour, or until someone steps in to provide assistance. An hour is the maximum duration for panic attacks, although the severity and signs of panic can differ. [7]

In certain instances, the assault might carry on unabatedly at a high intensity or appear to be getting worse. Although treating panic disorder can be difficult, there are a number of techniques that can assist people both reduce their symptoms and enhance their social interactions. Fast heartbeat, sweating, lightheadedness, dyspnea, trembling, and uncontrollable dread—such as the fear of losing control and going insane, the fear of death, or hyperventilation—are typical signs of panic disorder attacks.[7] Other symptoms are a sensation of choking, paralysis, chest pain, nausea, numbness or tingling, chills or hot flashes, vision problems, faintness, crying and some sense of altered reality. In addition, the person usually has thoughts of impending doom. Individuals experiencing an episode have often a strong wish of escaping from the situation that provoked the attack. The anxiety of panic disorder is particularly severe and noticeably episodic compared to that from generalized anxiety disorder. Panic attacks may be provoked by exposure to certain stimuli (e.g., seeing a mouse) or settings (e.g., the dentist's office). Nocturnal panic attacks are common in people with panic disorder. Other attacks may appear unprovoked. Some individuals deal with these events on a regular basis, sometimes daily or weekly. Limited symptom attacks are similar to panic attacks but have fewer symptoms. Most people with PD experience both panic attacks and limited symptom attacks.[8]

Key points about panic disorder

- An excessive reactivity to everyday pressures causing worry and anxiety is known as panic disorder.
- The reaction sets off a hyperkinetic reaction, which is followed by a severe fear that there will be another assault shortly. This may interfere with one's capacity to operate normally.
- Panic disorders may be crippling because they cause you to become so terrified of when your next panic attack may occur that you are unable to function normally. It is a prevalent disorder that frequently results in depression.
- Treatment involves use of anti-anxiety medicines and antidepressants along with cognitive behavioural therapy.[11]

The cause of panic disorder is unknown. Panic disorder often runs in families. Risk factors include smoking, psychological stress, and a history of child abuse. Diagnosis involves ruling out other potential causes of anxiety including other mental disorders, medical conditions such as heart disease or hyperthyroidism, and drug use. Screening for the condition may be done using a questionnaire.

Panic disorder is usually treated with counselling and medications. The type of counselling used is typically cognitive behavioral therapy (CBT) which is effective in more than half of people. Medications used include antidepressants, benzodiazepines, and beta blockers. Following stopping treatment up to 30% of people have a recurrence.

Panic disorder affects about 2.5% of people at some point in their life. It usually begins during adolescence or early adulthood, but may affect people of any age. It is less common in children and elderly people. Women are more often affected than men and it occurs more often in people of above average intelligence.[12]

How is panic disorder diagnosed?

Based on your symptoms, a mental health

specialist or your healthcare practitioner may diagnose you with panic disorder. Panic disorder is generally indicated by four or more panic attacks and a persistent dread of experiencing another one.

According to the DSM-IV-TR diagnostic criteria, panic disorder is defined as unexpected, repeated panic attacks that, in at least one case, are followed by a major and associated behavior change that lasts at least a month, a persistent fear of further attacks, or worry about the effects of the attack. Agoraphobia is present in one of the two forms. Diagnosis is excluded by attacks due to a drug or medical condition, or by panic attacks that are better accounted for by other mental disorders.[9]
The ICD-10 diagnostic criteria:
The essential feature is recurrent attacks of severe anxiety (panic), which are not restricted to any particular situation or set of circumstances and are therefore unpredictable.
The dominant symptoms include:

- sudden onset of palpitations
- chest pain or tightness
- shortness of breath or hyperventilation
- choking sensations
- dizziness
- feelings of unreality (depersonalization or derealization)
- secondary dread of going insane, dying, or losing control

If a person has a depressive disorder at the time the episodes begin, panic disorder should not be the primary diagnosis; in these cases, the panic attacks are probably secondary to depression. The Panic Disorder Severity Scale (PDSS) is a questionnaire for measuring the severity of panic disorder.[10]

Treatment

Treatment may include:

- Anti-anxiety and antidepressant medications
- Counseling, such as cognitive behavioral therapy

Treatment for panic disorders is usually quite effective. Your ability to discern that the symptoms are not life-threatening will improve with treatment. In order to lessen the severity and duration of the panic attack, you will also learn coping mechanisms and relaxation techniques. consequences of panic disorder: As the panic worsens and the episodes stay longer, you could find it extremely difficult to go about your daily business, hold down a job, or function in social settings. You might feel stuck or afraid to enter situations from which you might not be able to get out. Some people are unable to leave their homes out of concern that assistance won't come or that they'll be compelled to go somewhere that will incite an attack. People with this condition may also abuse alcohol or drugs to relieve stress.[16]

Panic disorder is a serious health problem that in many cases can be successfully treated, although there is no known cure. Identification of treatments that engender as full a response as possible, and can minimize relapse, is imperative. Cognitive behavioral therapy and positive self-talk specific for panic are the treatments of choice for panic disorder. Several studies show that 85 to 90 percent of panic disorder patients treated with CBT recover completely from their panic attacks within 12 weeks. When cognitive behavioral therapy is not an option, pharmacotherapy can be used.[17] SSRIs are considered a first-line pharmacotherapeutic option[18].

Medication

Appropriate medications are effective for panic disorder. First-line therapies for depression include selective serotonin reuptake inhibitors instead of benzodiazepines because of concerns about tolerance, dependence, and abuse of the latter. Few research have been conducted, and treating panic with medicine makes treating phobias much simpler, even while there is no evidence that pharmacological therapies can directly affect phobias (one example in Europe where only 8% of patients receive appropriate treatment).[19]

Medications can include:

- **Antidepressants (SSRIs, MAOIs, tricyclic antidepressants and norepinephrine reuptake inhibitors):** The most common SSRIs used while treating panic disorder include Prozac, Luvox, Zoloft, Paxil, Effexor, and Serzone with Prozac. These have been reported as the most effective in preventing panic attacks. The most researched and support tricyclic antidepressants are Tofranil and Anafranil. Pamelor, Norpramin, and Elavil are a few more tricyclic antidepressants that are prescribed for treatment. Compared to the two drugs previously listed, there hasn't been as much conclusive research on these, despite their potential usefulness in treating panic disorder. It is thought that the best effective medication for preventing panic episodes is an MAO inhibitor.

Phenelzine and Tofranil, two of the most commonly used of anti-panic MAOI medications, have been found to be the best with both being equally effective in treatment.[20]

- **Antianxiety agents (benzodiazepines):** The American Psychiatric Association states that benzodiazepines can be effective for the treatment of panic disorder and recommends that the choice of whether to use benzodiazepines The history and characteristics of each patient should be taken into consideration when prescribing antidepressants with anti-panic qualities or psychotherapy. Other experts believe that benzodiazepines are best avoided due to the risks of the development of tolerance and physical dependence.[21] The World Federation of Societies of Biological Psychiatry, say that benzodiazepines should not be used as a first-line treatment option but are an option for treatment-resistant cases of panic disorder. Despite increasing focus on the use of antidepressants and other agents for the treatment of anxiety as recommended best practice, benzodiazepines have remained a commonly used medication for panic disorder.[22] They reported that in their view there is insufficient evidence to recommend one treatment over another for panic disorder. The APA noted that while benzodiazepines have the advantage of a rapid onset of action, that this is offset by the risk of developing a benzodiazepine dependence.[23] The National Institute of Clinical Excellence came to a different conclusion, they pointed out the problems of using uncontrolled clinical trials to assess the effectiveness of pharmacotherapy and based on placebo-controlled research they concluded that benzodiazepines were not effective in the long-term for panic disorder and recommended that benzodiazepines not be used for longer than 4 weeks for panic disorder. Instead NICE clinical guidelines recommend alternative pharmacotherapeutic or psychotherapeutic interventions.[24] When compared to placebos, benzodiazepines demonstrate possible superiority in the short term but the evidence is low quality with limited applicability to clinical practice.[25]

Mechanism

Most anxiety disorders and panic disorder share a similar neuroanatomy. Neuropsychological, neurosurgical, and neuroimaging studies implicate the insula, amygdala, hippocampus, anterior cingulate cortex (ACC), lateral prefrontal cortex, and periaqueductal grey. Most studies reveal increased blood flow or metabolism during acute panic attacks, when reading emotionally laden phrases, and when at rest. The amygdala's hyperactivity is not always observed, though, particularly in research that chemically simulate panic attacks. Hippocampus hyperactivity has been observed during rest and viewing emotionally charged pictures, which has been hypothesized to be related to memory retrieval bias towards anxious memories. Abnormal introceptive processes—the belief that one's own body sensations are real—are thought to be connected to insulin hyperactivity during the onset and duration of acute panic episodes. "wrong" is a transdiagnostic finding (i.e. found across multiple anxiety disorders), and may be related to insula dysfunction. Rodent and human studies heavily implicate the periaqueductal grey in generating fear responses, and abnormalities related to the structure and metabolism in the PAG have been reported in panic disorder. The frontal cortex is implicated in panic disorder by multiple lines of evidence. Damage to the dorsal ACC has been reported to lead to panic disorder. Elevated ventral ACC and dorsolateral prefrontal cortex during symptom provocation and viewing emotional stimuli have also been reported, although findings are not consistent.[13]

Researchers looking at a few people who suffer from panic disorder speculate that these people might have a chemical imbalance in the limbic system, specifically in the GABA-A neurotransmitter. The amygdala, which controls the body's "fight or flight" response mechanism, receives misleading information from the decreased synthesis of GABA-A, which results in the physiological symptoms that lead to the disorder. Clonazepam, an anticonvulsant benzodiazepine with a long half-life, has been successful in keeping the condition under control. Recently, researchers have begun to identify mediators and moderators of aspects of panic disorder. One such mediator is the partial pressure of carbon dioxide, which mediates the relationship between panic disorder patients receiving breathing training and anxiety sensitivity; thus, breathing training affects the partial pressure of carbon dioxide in a patient's arterial blood, which in turn lowers anxiety sensitivity. Another mediator is hypochondriacal concerns, which mediate the relationship between anxiety sensitivity and panic symptomatology; thus, anxiety sensitivity affects hypochondriacal concerns which, in turn, affect panic symptomatology.[14]

Perceived threat control has been identified as a moderator within panic disorder, moderating the relationship between anxiety sensitivity and agoraphobia; thus, the level of perceived threat control dictates the degree This agoraphobia is caused by sensitivity to anxiety.Genetic variants in the galanin gene are another recently discovered moderator of panic disorder; these genetic variations modify the link between females with panic disorder and the level of severity of panic disorder symptomatology.[15]

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SCHIZOPHRENIA DISEASE: A REVIEW

Disha Wadave, Sakshi Babar, Dr. Shubhangi Daswadkar
Department of Pharmaceutical chemistry, Dr. D.Y Patil College of Pharmacy. Akurdi, Pune

ABSTRACT:

Schizophrenia is a common, severe mental illness that most clinicians will encounter regularly during their practice. This report provides an overview of the clinical characteristics, epidemiology, genetics, neuroscience, and psychopharmacology of schizophrenia to provide a basis to understand the disorder and its treatment. This educational review is integrated with a clinical case to highlight how recent research findings can inform clinical understanding.

INTRODUCTION:

Schizophrenia is long –term mental health condition. It causes a range of different psychological symptoms. Doctors often describe schizophrenia as a type of psychosis. This means the person may not always be able to distinguish their own thoughts and ideas from reality.Schizophrenic patients are unable to filter sensory stimuli and may have enhanced perception of sounds, colours and other features of their environment. While there is no cure for schizophrenia, research is leading to innovative and safer treatments. [1] Experts also are unraveling the causes of the disease by studying genetics, conducting behavioral research, and using advanced imaging to look at the brain's structure and function. These approaches hold the promise of new, and more effective therapies. [2]

INCIDENCE AND PREVALENCE:

Most schizophrenics, if untreated can gradually withdraw themselves from the interactions with other people and lose their ability to take care of personnel needs[3] 24 million people have schizophrenia but less than 33% of them receive treatment.90% of people diagnosed with schizophrenia have educational, personal, familial, social, and occupational issues in their day-to-day lives.1.5 for every 10,000 people is the yearly amount of novel schizophrenia cases diagnosed.Among young adults, 1 out of every 222 individuals, or 0.45% of the global population, have schizophrenia.60% of people with schizophrenia face discrimination, stigma from other individuals, and human rights abuses at some point. 2 out of 3 people suffering from psychosis never receive the proper healthcare they need to treat their illness, including those with schizophrenia. There is a multitude of options available for people with schizophrenia to get help, yet only 1 out of 3 individuals with the disorder ever recover in full.[4]

Women	Men
Peak ages of onset are 25-35 years	Peak ages of onset are 15-25 years
About 2/3 of cases are in the age group of 15-30 year	Very common in lower socio-economic groups

SIGNS AND SYMPTOMS:

Positive Symptoms	Negative symptoms	Cognitive Symptoms
Hallucinations	Anhedonia	Memory Issue
Delusion	Avolition	Inability to process social cues
Disorganized speech and thought	Blunted affect	Impaired sensory perception

PATHOPHYSIOLOGY:

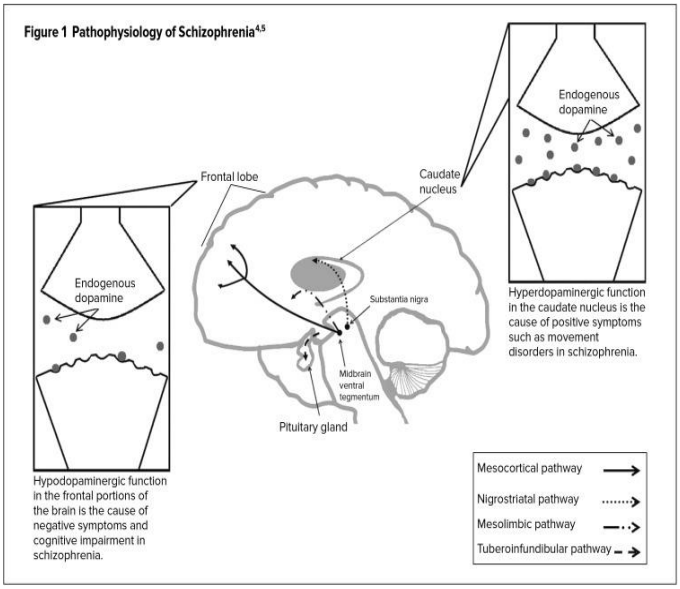

Figure 1 Pathophysiology of Schizophrenia[4,5]

While many factors have been associated with developing schizophrenia-including genetics, early environment, neurobiology, and psychological and social processes-the exact cause of the disease is unknown. Abnormalities in neurotransmission have provided the basis for theories on the pathophysiology of schizophrenia. Most of these theories center on either an excess or a deficiency of neurotransmitters, including dopamine, serotonin, and glutamate. Other theories implicate aspartate, glycine, and gamma-aminobutyric acid (GABA) as part of the neurochemical imbalance of schizophrenia.[5]

Abnormal activity at dopamine receptor sites (specifically D_2) is thought to be associated with many of the symptoms of schizophrenia. Four dopaminergic pathways have been implicated. The nigrostriatal pathway originates in the substantia nigra and ends in the caudate nucleus. Low dopamine levels within this pathway are thought to affect the extrapyramidal system, leading to motor symptoms. The mesolimbic pathway, extending from the ventral tegmental area (VTA) to limbic areas, may play a role in the positive symptoms of schizophrenia in the presence of excess dopamine.[1] The mesocortical pathway extends from the VTA to the cortex.[6]

Negative symptoms and cognitive deficits in schizophrenia are thought to be caused by low mesocortical dopamine levels. The tuberoinfundibular pathway projects from the hypothalamus to the pituitary gland. A decrease or blockade of tuberoinfundibular dopamine results in elevated prolactin levels and, as a result, galactorrhea, amenorrhea, and reduced libido.[7]

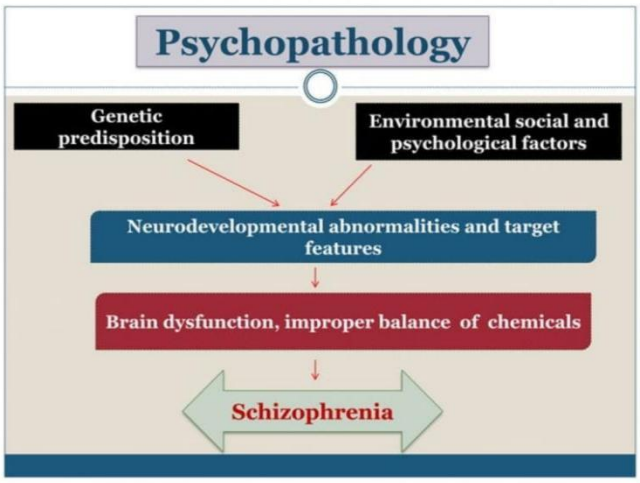

Fig no2:Psychopathology of Schizophrenia

DIAGNOSIS:

There is no single test for diagnosing schizophrenia. Due to the range of different symptoms that may be seen in the patient, the psychiatrist makes a diagnosis after a thorough clinical examination. As part of the examination the psychiatrist tries and unravels the changes in their behavior and biological functions (sleeplessness, lack of interest in eating or socializing). Information about the deviations in the patient's behavior is also collected from the family or caregivers.

A person is diagnosed with schizophrenia only if he or she has exhibited a combination of the above mentioned symptoms for at least a month.[8]

If you suspect someone you know has schizophrenia, it is best to consult a doctor. There are other mental health disorders such as addiction, bipolar disorder and depression that can be confused with schizophrenia because they also may cause delusions, hallucinations, and social withdrawal. Only a psychiatrist will be able to accurately diagnose whether the person has schizophrenia, or is suffering from another disorder.[9]

TREATMENT

Fig no 3: How to treat Schizophrenia

- Antipsychotics are the first line treatment for schizophrenia.
- Psychotherapy can also be helpful and may be used alongside complementary approaches to improve quality of life.
- Treatment adherence can lead to better outcomes, so it is essential to take your medications and stick to your treatment plan.

The medications doctors prescribe most often for schizophrenia are called antipsychotics. They ease symptoms such as delusions and hallucinations. The medications doctors prescribe most often for schizophrenia are called antipsychotics. They ease symptoms such as delusions and hallucinations .the medication doctors prescriber most often for schizophrenia are called antipsychotic. There are two groups of antipsychotics. Doctors called older group of medication "first -generation", or "Conventional" antipsychotics. Some common ones are:

- chlorpromazine (Thorazine)
- Fluphenazine (Prolixin)
- Haloperidol (Haldol)
- Perphenazine (Triladon)
- Thioridazine (Mellaril)
- Thiothixene (Navane)
- Trifluoperazine (Stelazine)[10]

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"WINTER'S SHADOW: UNDERSTANDING AND ADDRESSING SEASONAL AFFECTIVE DISORDER - A COMPREHENSIVE REVIEW"

Ms. Ashwini Gavit, Dr. Devendra Shirode
Department of Pharmacology, Dr. D.Y Patil College of Pharmacy. Akurdi, Pune

Introduction

Seasonal Affective Disorder (SAD) is a type of depression that occurs at a specific time of year, usually during the fall and winter months when daylight hours are shorter. Also known as winter depression or seasonal depression, SAD is characterized by a recurring pattern of depressive symptoms that typically begin in the late fall or early winter and subside in the spring or summer. The exact cause of SAD is not fully understood, but it is widely believed to be linked to the reduced exposure to natural sunlight during the darker months, which can disrupt the body's internal biological clock and lead to mood-related disturbances. This condition is more prevalent in regions with pronounced seasonal changes, where daylight hours vary significantly.1

Individuals affected by SAD may experience a range of symptoms, including low energy, changes in sleep patterns, weight gain, difficulty concentrating, and a persistent sense of sadness or hopelessness. While SAD shares similarities with major depressive disorder, its cyclical nature distinguishes it as a unique subset of mood disorders. Recognizing the symptoms and understanding the underlying factors contributing to SAD is crucial for effective diagnosis and management. Various treatment options, such as light therapy, psychotherapy, and medication, can be employed to alleviate the impact of SAD and improve overall well-being during the challenging winter months.[2-4] Despite its association with the changing seasons, Seasonal Affective Disorder is not solely limited to the colder months. In some rare cases, a less common form of SAD known as "summer depression" can occur, with symptoms peaking during the warmer months. However, the majority of individuals with SAD experience its effects in the fall and winter.[5] The primary theory behind the development of SAD revolves around the disruption of the body's circadian rhythm and the impact on neurotransmitters, particularly serotonin and melatonin. Reduced exposure to natural sunlight can disturb these biological processes, affecting mood, sleep, and overall well-being. Additionally, individuals with a predisposition to depression may be more vulnerable to the seasonal variations that trigger SAD.[6-8] The geographical prevalence of SAD is notable, with higher latitudes and regions farther from the equator reporting increased incidences. This geographical correlation is attributed to the diminished sunlight exposure that occurs in these areas during the winter months. Furthermore, gender and age can play a role, as SAD is more commonly diagnosed in women and tends to emerge in early adulthood.[9-10]

Diagnosing SAD involves a thorough evaluation of an individual's medical history, symptoms, and the timing of symptom onset. It's important to distinguish SAD from other types of depression and mood disorders to tailor appropriate interventions.[11] Treatment approaches may include light therapy, where individuals are exposed to bright artificial light mimicking natural sunlight, psychotherapy to address the emotional aspects of the disorder, and in some cases, medications such as antidepressants.[12-13]As our understanding of Seasonal Affective Disorder continues to evolve, researchers are exploring additional factors that may contribute to its development, including genetic predispositions and lifestyle factors. Increased awareness of SAD is essential for fostering a supportive environment and encouraging those affected to seek timely and effective interventions. By acknowledging the challenges posed by this seasonal variant of depression, we can work towards creating a more compassionate and informed approach to mental health care.[14]Living with Seasonal Affective Disorder requires a holistic approach that goes beyond clinical interventions. Lifestyle modifications and self-care strategies can also play a pivotal role in managing symptoms and improving overall well-being. Establishing a

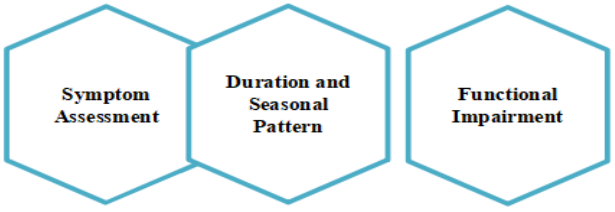
consistent daily routine, incorporating regular physical activity, and maintaining a healthy diet are essential components of a comprehensive treatment plan.[15]

Engaging in outdoor activities, even during the colder months, can be particularly beneficial. Exposure to natural light, even on overcast days, can help regulate circadian rhythms and alleviate some of the symptoms associated with SAD. Combining these lifestyle adjustments with therapeutic practices, such as mindfulness and stress-reduction techniques, may contribute to a more balanced mental state. Social support is another crucial aspect of managing Seasonal Affective Disorder. Connecting with friends, family, or support groups can provide a valuable network of understanding and encouragement. Sharing experiences with others who are facing similar challenges can help reduce feelings of isolation and foster a sense of community. For those affected by SAD, it's essential to monitor symptoms closely and communicate openly with healthcare professionals to adjust treatment plans as needed. Additionally, individuals with a history of SAD may benefit from proactive measures as the seasons change, such as starting light therapy before symptoms typically emerge. [16-17]As research into Seasonal Affective Disorder progresses, ongoing efforts to destigmatize mental health discussions and raise awareness about SAD are critical. Understanding that this condition is a legitimate and treatable form of depression can empower individuals to seek help and take an active role in managing their mental health.

Diagnosis

Diagnosing Seasonal Affective Disorder (SAD) involves a thorough assessment of an individual's symptoms, medical history, and the seasonal pattern of depressive episodes. Here are key components of the diagnostic process:

1. **Clinical Evaluation:**



- **Symptom Assessment:** A healthcare professional will conduct a comprehensive evaluation of the individual's symptoms. Common symptoms of SAD include low energy, changes in sleep patterns, weight gain, difficulty concentrating, irritability, and a persistent low mood.
- **Duration and Seasonal Pattern:** The clinician will inquire about the duration and seasonal occurrence of symptoms. To be diagnosed with SAD, there should be a consistent pattern of depressive episodes occurring during specific seasons, typically fall and winter.
- **Functional Impairment:** The impact of symptoms on daily functioning is considered. SAD can significantly affect a person's ability to carry out routine activities, maintain relationships, and perform at work or school. [18-20]

2. **Medical History:**

- **Psychiatric History:** Information about past episodes of depression or other mood disorders is essential. A history of recurrent depressive episodes that coincide with specific seasons supports the diagnosis of SAD.
- **Physical Health:** The clinician may inquire about the individual's overall physical health, as certain medical conditions and medications can contribute to or exacerbate depressive symptoms.

3. **Rule Out Other Conditions:**

- It's important to rule out other medical and psychiatric conditions that may mimic the symptoms of SAD. Conditions such as major depressive disorder, bipolar disorder, and certain medical illnesses can present with similar symptoms.

4. **Use of Diagnostic Criteria:**

- The diagnostic criteria for Seasonal Affective Disorder are outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), which is a widely accepted classification system used by mental health professionals. Meeting the specific criteria outlined in the DSM-5 is crucial for an accurate diagnosis.

5. **Self-Reported Measures:**

- Psychiatric rating scales and self-reported questionnaires may be used to assess the severity of symptoms and track changes over time. These measures can provide valuable information to

aid in the diagnosis and monitor treatment progress.

6. **Collaborative Approach:**

- The diagnostic process for SAD often involves collaboration between the individual and healthcare professionals, including psychiatrists, psychologists, or primary care physicians. The individual's input regarding their symptoms and the impact on their life is integral to the diagnostic process.It's important to note that diagnosing Seasonal Affective Disorder should be done by a qualified healthcare professional. If someone suspects they have SAD or is experiencing symptoms of depression, they should seek prompt evaluation and guidance from a healthcare provider. Early diagnosis and intervention can lead to effective treatment and improved quality of life. [21-23]

Treatment

Several effective treatments are available for Seasonal Affective Disorder (SAD), and the choice of treatment may depend on the severity of symptoms and individual preferences. Here are some commonly used approaches:

1. **Light Therapy (Phototherapy):** One of the primary treatments for SAD is light therapy, also known as phototherapy. This involves exposure to a bright light that mimics natural sunlight. Light boxes emit a specific intensity of light, typically 10,000 lux, and are designed to be used for a specific duration each day, usually in the morning. This exposure helps regulate the body's internal clock and improve mood.

2. **Psychotherapy (Cognitive-Behavioural Therapy):** Cognitive-behavioural therapy (CBT) is a form of psychotherapy that has been found effective in treating SAD. CBT focuses on identifying and changing negative thought patterns and behaviours associated with depression. It can help individuals develop coping strategies and establish healthier habits.

3. **Medications:** Antidepressant medications, particularly selective serotonin reuptake inhibitors (SSRIs) or serotonin-norepinephrine reuptake inhibitors (SNRIs), may be prescribed to alleviate symptoms of SAD. These medications work by increasing the levels of neurotransmitters in the brain that regulate mood.

4. **Dawn Simulators:** These devices mimic the natural sunrise and can be used as an alternative or complement to light therapy. Dawn simulators gradually increase the intensity of light in the room, simulating the rising sun and helping to regulate the body's circadian rhythm.

5. **Outdoor Activities and Increased Sunlight Exposure:** Encouraging individuals with SAD to spend more time outdoors during daylight hours can be a simple yet effective strategy. Even on overcast days, natural light exposure can positively impact mood and regulate circadian rhythms.

6. **Regular Exercise:** Engaging in regular physical activity has been shown to have antidepressant effects. Exercise can help alleviate symptoms of depression, including those associated with SAD. Both indoor and outdoor activities can be beneficial.

7. **Vitamin D Supplementation:** Some studies suggest a link between vitamin D deficiency and depressive symptoms. In regions with limited sunlight during the winter months, healthcare professionals may recommend vitamin D supplementation. [25-29]

It's crucial for individuals experiencing symptoms of Seasonal Affective Disorder to consult with a healthcare professional for a proper diagnosis and personalized treatment plan. Treatment effectiveness can vary from person to person, and a combination of therapies may be recommended for optimal results. Regular monitoring and adjustments to the treatment plan may be necessary based on individual responses and seasonal variations. In conclusion, Seasonal Affective Disorder is a complex and multifaceted condition that impacts individuals in predictable patterns related to seasonal changes. By combining medical interventions, lifestyle adjustments, and social support, individuals can navigate the challenges of SAD and work towards maintaining a fulfilling and balanced life throughout the year. Through continued research, education, and advocacy, the aim is to create a more supportive and informed society for those affected by Seasonal Affective Disorder.[30]

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Social Phobia: A Review

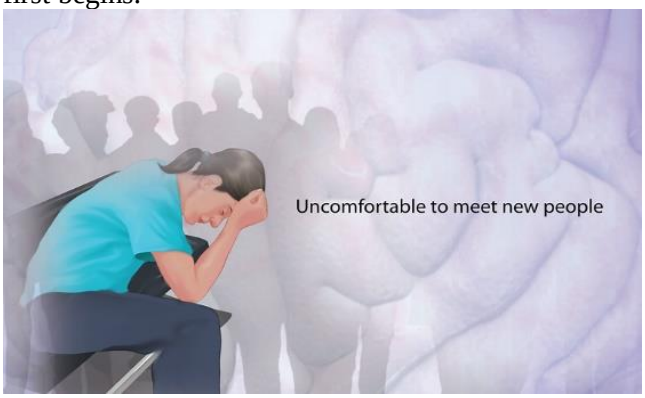
Samiksha Kumbhare, Tejashree Deokule, Poonam Rajaram Mulay
Department of Pharmaceutical Chemistry, Dr. D. Y Patil College of Pharmacy, Akurdi, Pune

ABSTRACT:

Social situations cause fear or anxiety in those who suffer from social phobia disorder. They might avoid going out, going to work, going shopping, or going anywhere else where people might be around if the feeling is so strong. Though it's unlikely, the person fears that they will be judged by others, offend someone, or somehow embarrass themselves. People can manage their symptoms and enhance their quality of life with the use of medication, counselling, and lifestyle changes.

INTRODUCTION:

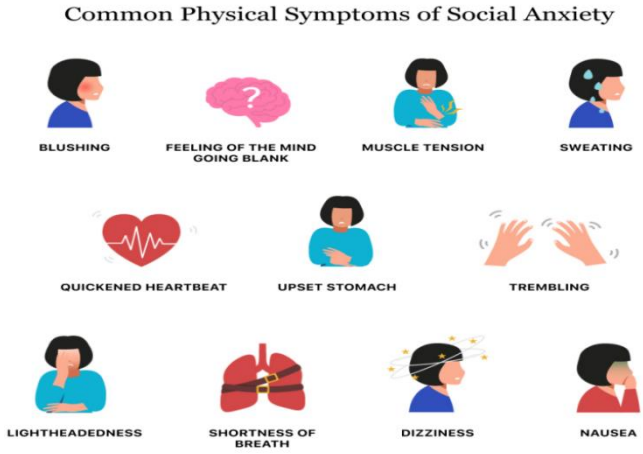
Social Phobia disorder sometimes known as social anxiety is a type of phobia disorder that causes anxiety or fear in social settings. A person suffering from this disorder finds it difficult to socialize, make new friends, and engage in conversation. They could have anxiety about being scrutinized or judged by others. Even if they know their worries are unfounded, they nonetheless feel helpless to face them head-on. Shyness and social anxiety are not the same thing. While shyness might make it challenging to socialize, learn, and work, it doesn't interfere with life as much as social anxiety does. Grocery shopping is one example of how social phobia can interfere with daily routines. It is overpowering and persistent.[1] Anxiety and Depression Association of America (ADAA) estimates that 15 million adults in the US suffer from social anxiety disorder. Teenage years are typically when it first begins.



SYMPTOMS:

When someone suffers from social phobia disorder, they could experience:

- Reddening.
- Sick stomach.
- Drenching in sweat.
- Quivering or swaying.
- An inflexible posture.
- Having trouble speaking.
- Feeling as though their thoughts wander.
- Feeling lightheaded or dizzy.
- Accelerated heart rate



Some examples of psychological symptoms are:

- Severe phobia preceding, duranting, and following a social interaction.
- Attempting to blend in with the background if you must attend, or avoiding social situations.
- Fear of looking foolish and feeling self-conscious.
- Fears that other people would see you as anxious or tense.
- Having to drink alcohol in order to cope with a social scenario.
- Anxiety-related absences from job or school.

Everyone experiences phobia at times, but people with social phobia are constantly afraid of being judged or humiliated in front of others.[2] They might avoid all social situations, such as:

- posing a query
- interviews for jobs
- shopping
- making use of public restrooms
- conversing on the phone
- consuming food in public

Some people experience selective or limited anxiety. They may be phobia only when eating in front of others or conversing with strangers, for example. People suffering from severe symptoms may avoid all social situations.[3]

CAUSES:

The exact cause of social phobia disorder is unknown, but it could be the result of a combination of factors.

According to scientists, physical, biological, and genetic factors are all likely to play a role.[4] Problems with neurotransmitter systems can cause hormonal imbalances in serotonin, dopamine, and glutamate. These brain chemicals aid in mood regulation.

Environmental factors may play a role, but only as part of a complex interaction that also includes biological and genetic factors, according to some experts.[5]

Among the factors that may play a role are a history of:

- negative peer interactions
- overbearing parenting styles
- having a shaky attachment style

Negative experiences can result in a form of post-traumatic stress disorder (PTSD), with social anxiety as a symptom.

Phobia disorders can be passed down through families.

DIAGNOSIS:

A physician will probably utilize criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) to determine whether social phobia disorder is likely. There is no specific medical test to diagnose social anxiety disorder.[6]

Most likely, they'll inquire about:

- Your signs and symptoms
- Your ancestry
- other medical issues
- The DSM-5 lists the following criteria for evaluating social phobia disorder:
- a fear of one or more social settings where one might be subject to other people's scrutiny
- being afraid to act in a way that might cause others to view you negatively or that might irritate or offend people
- Fear or anxiety is almost always evoked by a particular situation.
- The individual either stays away from the situation or shows up extremely nervous or afraid.
- The level of fear is excessive given the danger.
- Fear and anxiety are chronic conditions that typically last six months or longer.
- Anxiety and terror interfere with day-to-day existence
- The person's feelings of fear and anxiety cannot be explained by other symptoms or medical conditions.

They might also want to rule out additional ailments like:

- a problem involving substances
- a personality disorder
- worries about a physical trait, like a facial burn, or a health problem, like obesity[7]

TREATMENTS:

Social phobia disorder can be helped by a number of treatment approaches. Each person will respond differently to therapy. While some patients may only require one kind of treatment, others might require a combination.

Your primary care physician might recommend therapy or send you to a psychologist or other mental health professional.[8]

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Among the options are the following:

Counselling Therapy-

Talking, either one-on-one or in groups, is a key component of counselling. Online or in-person counselling sessions are offered.

Cognitive behavioural therapy (CBT):

CBT teaches you new techniques to deal with anxiety, like how to switch out negative thoughts for positive ones.

Acceptance and commitment therapy (ACT)

Teaches patients how to be more present and figure out how to live a life based on values in spite of unpleasant feelings. Patients learn these skills via mindfulness, acceptance, and behavioural techniques.[9]

Group therapy:

You can acquire social skills and methods for interacting with people in social situations by participating in group therapy or a support group. Collaborating with others can show you that you're not alone and allow you to role-play useful solutions.

Exposure therapy involves gradually confronting social situations with the assistance of a healthcare professional, as opposed to avoiding them.[10]

MEDICATON:

Medication can help you live a more normal life and manage your symptoms.

Some medications that are used to treat social phobia disorder include Reliable Source:

- selective serotonin reuptake inhibitors (SSRIs), like sertraline (Zoloft) and paroxetine (Paxil)
- selective norepinephrine reuptake inhibitors (SNRIs), such as venlafaxine (Effexor)
- Propanol.

HOME REMEDIES:

Natural and home remedies can complement the course of care that a particular prescription can be described.

The following are some recommendations for lowering stress and anxiety:

- respiration techniques
- meditation and mindfulness
- stress-reduction techniques like yoga and tai chi
- Steer clear of stimulants like caffeine.
- establishing a consistent sleeping schedule
- learning about the causes and effects of anxiety
- locating a friend, therapist, or family member with whom you can have an honest conversation
- recognizing the warning signals and when to get help
- eating a balanced diet and engaging in regular exercise to improve your general feeling of wellbeing.[11]

OUTLOOK & COMPLICATIONS:

More than one-third of those who suffer from social phobia wait to get treatment until after their symptoms have persisted for at least ten years, according to the ADAA. Individuals might not recognize their discomfort as a mental health problem or that there is support available to them.

In the absence of therapy, social phobia can have an impact on Reliable Source:

- performance in the workplace and in the classroom
- social dialogue
- associations
- self-worth.
- level of living

In addition to social phobia, up to 90% of people also suffer from other conditions like:

- misusing alcohol
- considering or making an attempt at suicide.[12]

Many people find that social anxiety and other mental health issues can be managed with the aid of medication, counselling, and lifestyle modifications.

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SPECIFIC PHOBIA: A REVIEW

Mayur Salunkhe, Mansi Varpe, Vikrant Bijjamwar, Miss Ankita Dudhal
Department of Pharmacology, Dr. D.Y Patil College of Pharmacy. Akurdi, Pune

ABSTRACT:

Phobias, characterized by excessive and irrational fears, represent a prevalent form of anxiety disorder affecting individuals across diverse demographics. This comprehensive review synthesizes current knowledge on the etiology, manifestations, and treatment approaches for various phobias. Drawing on evolutionary perspectives, genetic predispositions, and learned behaviors, we explore the origins of phobias, shedding light on their potential adaptive functions in ancestral environments. The manifestations of specific phobias, such as arachnophobia and acrophobia, are examined in detail, highlighting the considerable impact on individuals' daily lives and well-being.

In addressing treatment modalities, we survey contemporary therapeutic interventions, emphasizing the effectiveness of exposure therapy and cognitive-behavioral approaches. The role of pharmacological interventions is also explored, acknowledging their supplementary role in managing phobic symptoms. Additionally, this review underscores the importance of individualized treatment plans, considering the unique nature of each phobia and the diverse needs of affected individuals.

Furthermore, we discuss the challenges associated with phobia research, including the need for standardized diagnostic criteria and the exploration of cultural influences on phobic manifestations. By synthesizing existing knowledge and identifying gaps in understanding, this review aims to contribute to the ongoing discourse surrounding phobias, fostering a more nuanced and holistic approach to their recognition and management.

INTRODUCTION

Phobias, classified as anxiety disorders, constitute a pervasive aspect of human experience, reflecting an intricate interplay of psychological, biological, and environmental factors. Defined by excessive and irrational fears, phobias elicit profound emotional responses, often leading to avoidance behaviours that impact individuals' daily lives. This introduction provides an overview of phobias, delving into their prevalence, classifications, and the intricate web of factors contributing to their development.

1. **Definition and Classification: **

Phobias are characterized by intense and persistent fears of specific objects, situations, or activities, extending beyond the bounds of normal apprehension. The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) categorizes phobias into three main types: specific phobias, social anxiety disorder (social phobia), and agoraphobia. Specific phobias involve fear of particular objects or situations, while social anxiety disorder centers around fears of social scrutiny. Agoraphobia, on the other hand, involves anxiety about being in situations from which escape might be difficult.

2. **Prevalence and Impact: **

Phobias are remarkably prevalent, affecting individuals across diverse age groups, cultures, and backgrounds. Epidemiological studies reveal that specific phobias are among the most common psychiatric disorders, with a lifetime prevalence that underscores their widespread impact. The consequences of phobias extend beyond momentary discomfort, often leading to avoidance behaviors that can impede academic, occupational, and social functioning.

4. **Etiology and Factors Contributing to Phobias: **

The origins of phobias are multifaceted, involving genetic predispositions, evolutionary adaptability, and learned responses. While evolutionary perspectives posit that certain fears may have conferred survival advantages to our ancestors, contemporary theories emphasize the role of cognitive processes, such as negative reinforcement and information processing biases, in the development and maintenance of phobic reactions. Additionally, environmental factors, including traumatic experiences and cultural influences, contribute to the complexity of phobia etiology.

4. **The Adaptive Nature of Phobias: **

Despite their seemingly maladaptive nature in modern society, phobias may have served an adaptive function in ancestral environments. Rapid fear acquisition and retention of aversive stimuli may have enhanced survival by promoting quick and effective responses to potential threats. Understanding the adaptive roots of phobias offers insights into the intricate interplay of genetics,

evolution, and environmental pressures shaping human fears.
In navigating the labyrinth of phobias, researchers and clinicians alike seek to unravel the complexities surrounding their onset, manifestation, and treatment. This exploration serves as a foundation for subsequent sections that delve into specific phobias, treatment modalities, and the evolving landscape of phobia research.

INCIDENCE AND PREVALENCE

The incidence and prevalence of phobias can vary depending on the specific type of phobia and the population under consideration. Below are some general observations regarding the incidence and prevalence of phobias:

1. Lifetime Prevalence:
   - Phobias are among the most common mental health disorders, with a high lifetime prevalence.
   - Specific phobias are the most prevalent subtype, affecting approximately 7-9% of the global population at some point in their lives.
2. Specific Phobias:
   - Specific phobias are more prevalent than other types of phobias.
   - Estimates suggest that specific phobias affect around 12% of the population in a given year.
3. Social Anxiety Disorder (Social Phobia):
   - Social anxiety disorder has a lifetime prevalence of about 7-12%.
   - It often begins in adolescence and can significantly impact social and occupational functioning.
4. Agoraphobia:
   - Agoraphobia is less prevalent than specific phobias and social anxiety disorder.
   - It is estimated to have a lifetime prevalence of around 2% of the population.
5.Gender Differences:
   - Phobias, in general, are more common in females than males.
   - This gender difference is particularly pronounced for specific phobias and social anxiety disorders.
6. Age of Onset:
   - Phobias often have an early age of onset, typically in childhood or adolescence.
   - Some specific phobias may develop in adulthood, often in response to traumatic events.

It's important to note that these statistics are approximate, and prevalence rates can vary based on the criteria used for diagnosis, cultural factors, and regional differences. Additionally, many individuals with phobias may not seek professional help, leading to underreporting of cases.

Phobias can significantly impact an individual's quality of life, and understanding their prevalence helps guide public health efforts and mental health services. Early intervention and effective treatment, often involving psychotherapy such as exposure therapy or cognitive-behavioral therapy, can help individuals manage and overcome phobias. If you or someone you know is struggling with a phobia, seeking guidance from a mental health professional is recommended.

SIGNS AND SYMPTOMS

Phobias are anxiety disorders characterized by an intense and irrational fear of specific objects, situations, or activities. The signs and symptoms of phobias can manifest both psychologically and physiologically. It's important to note that the severity of symptoms can vary among individuals. Common signs and symptoms of phobias include:

1. **Intense Fear or Anxiety: **
   - The primary characteristic of a phobia is an overwhelming and persistent fear or anxiety related to a specific object or situation.

2. **Immediate Anxiety Response: **
   - The fear response is often immediate and intense, even when the feared object or situation poses little or no actual threat.

3. **Avoidance Behavior: **
   - Individuals with phobias typically go to great lengths to avoid the feared stimulus. This may involve changing routines, avoiding specific places, or refusing to engage in certain activities.

4. **Physical Symptoms: **
   - Phobias can trigger a range of physical symptoms, including rapid heartbeat, shortness of breath, trembling, sweating, and nausea. These symptoms are part of the body's "fight or flight" response.

5. **Panic Attacks: **
   - In some cases, exposure to the phobic stimulus can lead to panic attacks, which are sudden and intense periods of heightened anxiety.

6. **Recognizing Irrationality: **
   - Individuals with phobias often recognize that their fear is irrational, but they find it challenging to control or overcome.

7. **Anticipatory Anxiety: **
   - The anticipation of encountering the phobic stimulus can lead to anxiety well before the actual

exposure, affecting daily functioning and causing distress.

8. **Impact on Daily Life: **
- Phobias can significantly impact a person's daily life, social interactions, and occupational functioning. For example, a fear of flying might interfere with travel plans or career opportunities.

9. **Duration of Symptoms: **
- Phobic symptoms typically persist for six months or more and can cause significant distress.

10. **Interference with Relationships: **
- Phobias can strain relationships, especially if the fear leads to avoidance of social situations or activities.

Common types of specific phobias include fear of animals (e.g., spiders, snakes), natural environments (e.g., heights, thunderstorms), blood or injury, and situational fears (e.g., flying, enclosed spaces).

It's important for individuals experiencing these symptoms to seek professional help. Cognitive-behavioral therapy (CBT), exposure therapy, and sometimes medications are common approaches used to treat phobias. A mental health professional can provide a proper diagnosis and develop an individualized treatment plan based on the specific phobia and its impact on the person's life.

PATHOPHYSIOLOGY

The pathophysiology of phobias involves complex interactions between genetic, neurological, cognitive, and environmental factors. While the exact mechanisms are not fully understood, research suggests several key components that contribute to the development and maintenance of phobic responses. It's important to note that the pathophysiology may vary across different types of phobias.

1. **Genetic Factors: **
- There is evidence of a genetic predisposition to anxiety disorders, including phobias. Family studies and twin studies have shown a higher risk of phobias among individuals with a family history of anxiety disorders.

2. **Neurotransmitters and Brain Structure: **
- Neurotransmitters such as serotonin, gamma-aminobutyric acid (GABA), and norepinephrine play a role in regulating mood and anxiety. Imbalances in these neurotransmitters have been associated with anxiety disorders, including phobias.
- The amygdala, a part of the brain involved in emotional processing and fear response, is often implicated in phobias. Increased activity in the amygdala and alterations in its connectivity with other brain regions may contribute to the heightened fear response.

3. **Classical Conditioning: **
- Phobias often develop through classical conditioning, where a neutral stimulus becomes associated with a traumatic or fear-inducing event. For example, a person may develop a phobia of flying after experiencing severe turbulence during a flight.

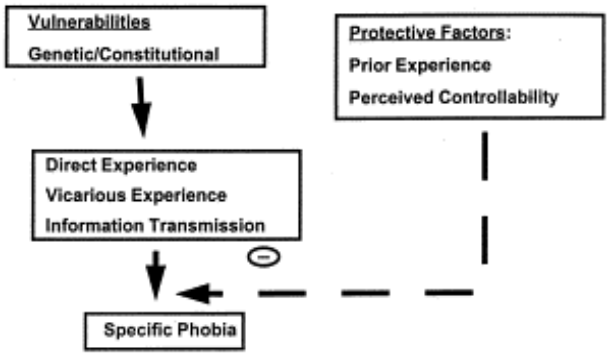
4. **Cognitive Factors: **
- Cognitive processes, such as biased information processing and catastrophic thinking, contribute to the maintenance of phobias. Individuals with phobias may have distorted perceptions of the likelihood and severity of harm associated with the feared stimulus.

5. **Memory and Recall: **
- The way individuals with phobias store and recall memories related to the phobic stimulus may contribute to the persistence of the fear. Memories of traumatic or fear-inducing events may be more vivid and easily triggered.

6. **Learned Responses and Reinforcement: **
- Avoidance behaviors, which are common in phobias, can be reinforced through negative reinforcement. If avoiding the phobic stimulus leads to a reduction in anxiety, the behavior is reinforced, making it more likely to persist.

7. **Environmental Factors: **
- Traumatic experiences or exposure to highly stressful events can contribute to the development of phobias. A person who experiences a traumatic event involving a specific stimulus may develop a phobia related to that stimulus.

Understanding the pathophysiology of phobias is crucial for developing effective treatment strategies. Therapeutic approaches often focus on disrupting maladaptive learning patterns, challenging distorted thought processes, and gradually exposing individuals to the feared stimulus in a controlled and supportive environment. Medications, such as selective serotonin reuptake inhibitors (SSRIs) or benzodiazepines, may be prescribed in some cases to alleviate symptoms. However, these medications are typically used in conjunction with psychotherapy for comprehensive treatment.

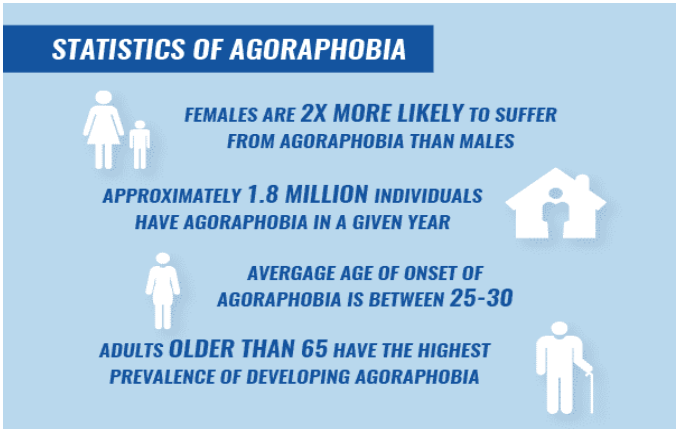


DIAGNOSIS

The diagnosis of phobias typically involves a comprehensive assessment by a mental health professional, such as a psychologist or psychiatrist. The diagnostic process aims to determine the presence of specific phobias, understand their impact on an individual's life, and rule out other potential mental health conditions. Here are the key components of the diagnosis:

1. **Clinical Interview: **
   - A thorough clinical interview is conducted to gather information about the individual's symptoms, their duration, and the impact on daily life. The clinician explores the nature of the fear, the circumstances that trigger anxiety, and any avoidance behaviors.
2. **Diagnostic Criteria: **
   - The diagnostic criteria for specific phobias are outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), which is widely used by mental health professionals. According to the DSM-5, specific phobias involve a marked and persistent fear of a specific object or situation, leading to avoidance or endurance with intense anxiety.

3. **Differentiation from Other Disorders: **
   - It is essential to differentiate specific phobias from other anxiety disorders, such as generalized anxiety disorder or panic disorder. The clinician assesses whether the fear is circumscribed to a specific object or situation or if it is part of a broader pattern of anxiety.
4. **Impact Assessment: **
   - The clinician evaluates the impact of the phobia on the individual's daily life, relationships, and overall functioning. This includes assessing whether the fear is causing sigificant distress or impairment.
5. **Duration of Symptoms: **
   - The symptoms of a specific phobia must persist for at least six months for a diagnosis to be made. This duration criterion helps distinguish transient fears from clinically significant phobias.
6. **Rule Out Medical Conditions: **
   - It is crucial to rule out any medical conditions or substances that might be contributing to the symptoms. Certain medical conditions or medications can mimic anxiety symptoms.
7. **Collateral Information: **
   - Collateral information from friends, family, or other sources may provide additional insights into the individual's experiences, behaviors, and the impact of the phobia on their life.
8. **Assessment Tools: **
   - Psychometric assessment tools, such as questionnaires and rating scales, may be used to quantify the severity of symptoms and monitor changes over time.
9. **Cultural Considerations: **
   - The clinician considers cultural factors that may influence the expression of fears and anxiety, ensuring that the diagnostic process is culturally sensitive.Once a diagnosis is established, treatment options can be discussed. Psychotherapy, particularly exposure therapy and cognitive-behavioral therapy (CBT), is a common and effective approach for treating specific phobias. In some cases, medications may be prescribed to alleviate symptoms, but they are often used in conjunction with psychotherapy.



TREATMENT

The treatment of phobias typically involves therapeutic interventions aimed at reducing and managing the excessive fear and anxiety associated with the specific object or situation. The two primary forms of evidence-based treatment for phobias are psychotherapy, particularly exposure therapy, and, in some cases, medications. Here are the main approaches to treating phobias:

1. **Psychotherapy: **
   - **Exposure Therapy: ** This is a widely used and effective form of psychotherapy for treating phobias. Exposure therapy involves systematic and gradual exposure to the feared object or situation in a controlled and supportive environment. Over time,

repeated exposure helps individuals learn that the feared stimulus is not as threatening as perceived, leading to a reduction in anxiety.

- **Cognitive-Behavioural Therapy (CBT): ** CBT is another effective therapeutic approach for phobias. It combines cognitive restructuring, which involves challenging and changing distorted thought patterns, with behavioral techniques, such as exposure therapy. CBT helps individuals identify and modify irrational beliefs about the feared stimulus.

2. **Medications: **

- **Selective Serotonin Reuptake Inhibitors (SSRIs): ** These antidepressant medications, such as fluoxetine or sertraline, may be prescribed to help alleviate symptoms of anxiety associated with phobias. SSRIs are often used in cases where psychotherapy alone is insufficient or when there is comorbidity with other anxiety disorders.

- **Benzodiazepines: ** These medications may be used for short-term relief of acute anxiety symptoms, but they are generally not recommended for long-term use due to the risk of dependence.

3. **Virtual Reality Therapy: **

- Virtual reality exposure therapy involves using computer-generated simulations to expose individuals to the feared stimulus in a virtual environment. This approach can be particularly useful when real-life exposure is challenging or impractical.

4. **Mindfulness and Relaxation Techniques: **

- Mindfulness-based interventions and relaxation techniques, such as deep breathing and progressive muscle relaxation, can help individuals manage anxiety symptoms associated with phobias.

5. **Group Therapy and Support Groups: **

- Group therapy provides a supportive environment where individuals with similar phobias can share their experiences and strategies for coping. Support groups may be facilitated by mental health professionals or organized by advocacy organizations.

6. **Self-Help Strategies: **

- Self-help strategies, including self-directed exposure exercises and the use of self-help materials, can be beneficial for individuals with milder forms of specific phobias.

Treatment plans are often tailored to the individual's specific phobia, the severity of symptoms, and their preferences. It's essential for individuals experiencing phobias to seek professional help to determine the most appropriate and effective treatment approach. Early intervention can lead to successful outcomes and an improved quality of life.

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Navigating the Shadows: Understanding and Confronting Depression

Riya Singh, Dr. Pallavi Chaudhari
Department of Pharmaceutics, Dr. D.Y. Patil College of Pharmacy, Akurdi Pune

Abstract:

Depression is a common mental health condition that affects people from a wide range of demographic backgrounds worldwide. This essay investigates its ubiquity, which is thought to affect millions of people globally. When examining the chemistry of depression, neurotransmitter imbalances—especially those involving dopamine, serotonin, and norepinephrine—become very important. Its onset is also greatly influenced by socioeconomic factors, environmental stressors, and genetic predispositions. A range of therapeutic approaches, such as psychotherapy, medication, and newer therapies like ketamine therapy and transcranial magnetic stimulation (TMS), have demonstrated effectiveness in treating depressive symptoms. To address this complex and widespread mental health issue, more research is needed into customized medicine, targeted therapy, and holistic approaches.

This article provides a succinct synopsis of depression, including its neurochemical components, contributing causes, prevalence, and a brief description of treatment options and the need for additional research.

Keywords: Depression, mental disorder, prevalence, causes, treatment, drugs

Introduction:

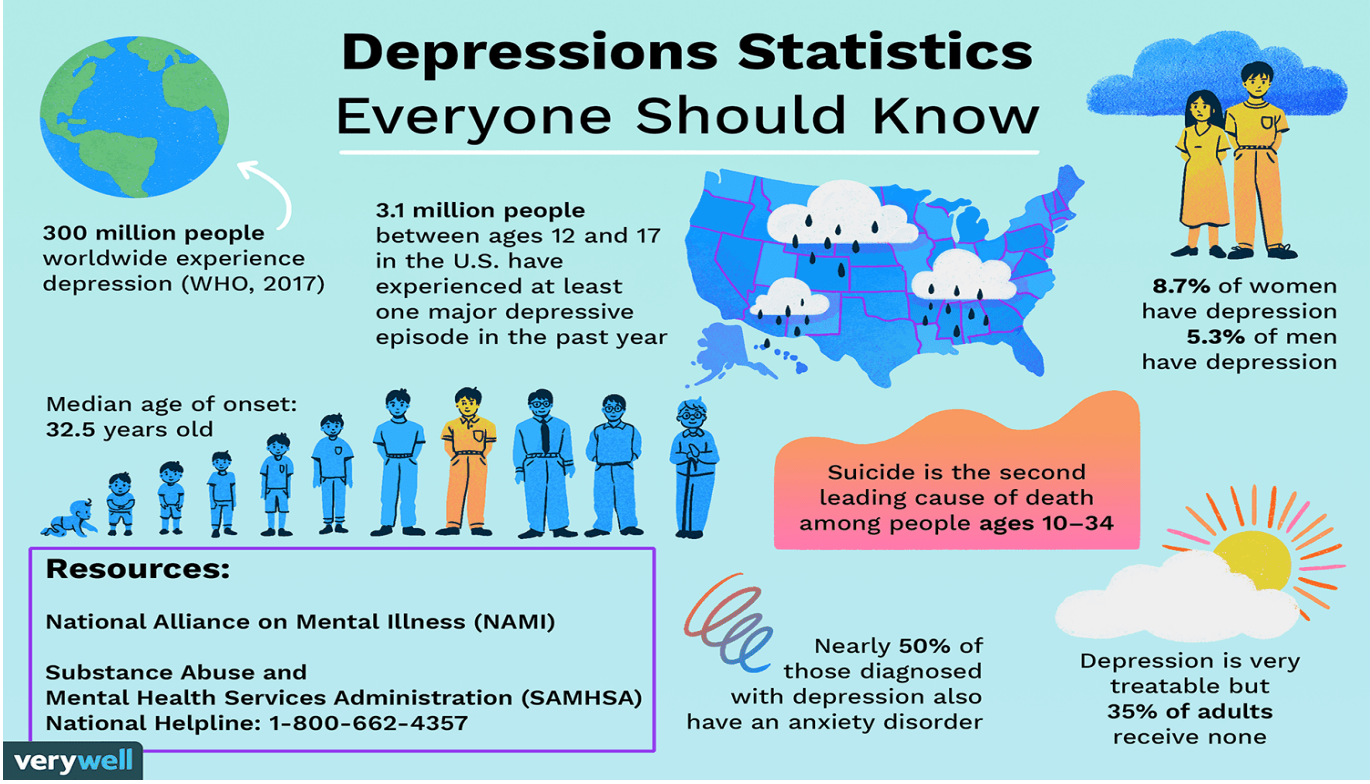
Because of its impact and frequency, depression is one of the main causes of disability worldwide. Research from the United States, Europe, and the United Kingdom point to and 12-month prevalence rates ranging from 3% to 10%1, whereas large-scale representative surveys conducted in the United States have yielded an estimated lifetime prevalence of 17%2. According to Eaton et al. (2008), depression is linked to significant personal suffering, increased mortality, and decreased quality of life and functioning3. According to the disability-adjusted life-year, it is currently the third most common cause of illness burden worldwide. It is also the most common cause in middle- class and wealthy nations. By 2030, it is expected to contribute the second most to the burden of disease worldwide.[4]

The current definition of depression, as held by the majority of psychiatrists and codified in the official ICD-10 Classification of Behavioral and Menial Disorders. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)5 and Clinical Descriptions and Diagnostic Guidelines (ICD 10)6 are basically two versions of a clinical syndrome, which is defined as the existence of several clinical features without requiring a particular etiology and, in a somewhat Meyerian manner, acknowledging the possibility of both psychological and biological causative factors. The DSM-IV does not include states in which the symptoms are characterized by significant functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or "better accounted for by bereavement," an imprecise criterion that is expanded by specifications of not persisting for longer than two months, is characterized by psychomotor retardation, suicidal thoughts, psychotic symptoms, severe functional impairment, or morbid obsession with worthlessness. There is disagreement over the usefulness of this exclusion.[7]

Prevalence of depression:

Depression disorders place a great deal of strain on the sufferer, even if treatment is effective and remission is reached. Rarely does remission result in the complete cessation of all symptoms. Remaining symptoms can continue to hinder performance and produce a great deal of distress, particularly if they involve social dysfunction or cognitive impairment. The constant threat of relapse and recurrence also significantly lowers quality of life in general.

A recent analysis indicated that while the rate of major depressive disorder recurrence in primary care patients was much lower (35% after 15 years), it was quite high (60% after 5 years, 67% after 10 years, and 85% after 15 years) in settings for specialist mental health treatment. Subclinical residual symptoms and the frequency of prior episodes were revealed to be the



most significant predictors of recurrence in other studies.[8]

Figure.1. Depression statistics worldwide

Chemistry behind depression:

Preclinical and clinical research point to a disruption in serotonin (5-HT) activity in the central nervous system as a significant contributing factor. Norepinephrine (NE), dopamine (DA), glutamate, and brain-derived neurotrophic factor (BDNF) are among the other neurotransmitters that have been linked.

The beneficial effects of selective serotonin reuptake inhibitors (SSRIs) point to a potential involvement of central nervous system 5-HT activity in the pathophysiology of major depressive disorder. In addition to increased neurotransmitter availability, research findings suggest a role for intracellular signalling, gene expression over time, and regulation of neuronal receptors.A type of major depressive disorder known as seasonal affective disorder usually appears in the autumn and winter and goes away in the spring and summer. Studies indicate that changes in circadian rhythm and sunlight exposure appear to be the triggers for seasonal affective disorder, which is also thought to be mediated by changes in CNS levels of 5-HT.

By upsetting the neural networks involved in emotion regulation, such as the frontostriatal pathways connecting the dorsolateral prefrontal cortex, orbitofrontal cortex, anterior cingulate, and dorsal cingulate, vascular lesions may exacerbate depression.

Depression has also been linked to other limbic circuit components, specifically the amygdala and hippocampus.9

Types of depression:

There is no one-size-fits-or types for depression. Among the varieties are:

- ✓ A persistently depressed mood, a loss of interest in activities, and other symptoms that linger for at least two weeks are characteristics of Major Depressive Disorder (MDD).
- ✓ Dysthymia, also known as chronic depression, is a milder but more persistent type of depression that lasts for two years or longer and is characterised by enduring depressive symptoms.
- ✓ Seasonal Affective Disorder (SAD): Depression brought on by seasonal variations; typically manifests in the autumn and winter as a result of less daylight.
- ✓ Some women have postpartum depression, which is characterised by intense emotions of

melancholy, worry, and tiredness following childbirth.
- ✓ Bipolar disorder is characterised by manic or hypomanic moments of heightened mood interspersed with depressive spells.
- ✓ Depression combined with psychotic symptoms, such as delusions or hallucinations, is known as psychotic depression.[10-12]

Causes of depression

There are several possible reasons of depression, such as:

- ✓ Biological Factors: Variations in the structure of the brain, genetics, hormone fluctuations, and imbalances in neurotransmitters can all have an impact.
- ✓ Psychological Factors: Depression may result from long-term stress, trauma, low self-esteem, or negative thought patterns.
- ✓ Environmental Factors: Stressful life experiences such as financial hardships, abuse, bereavement, or big life changes can set off depressive episodes.
- ✓ Medical Conditions: Mood swings and depression can be exacerbated by certain diseases, chronic pain, or side effects from medications.
- ✓ Genetic Predisposition: Family history is important; people who have relatives who suffer from depression may be more susceptible.

Comprehending these diverse origins aids in customising therapy and assistance tactics for those grappling with despair.[13-14]

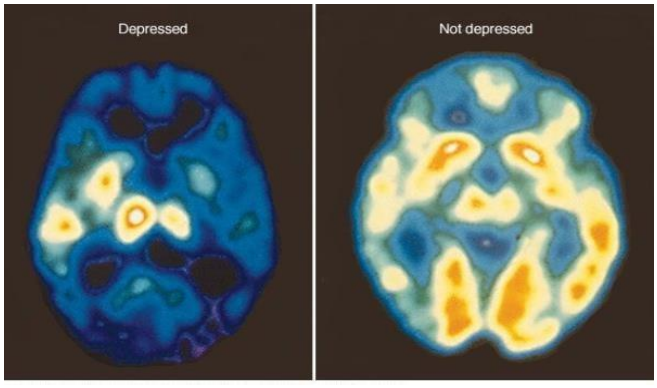


Figure.2 A typical comparison of depressed and non-depressed brain

Treatment for depression:

Different methods of diagnosis and treatment are necessary for these variances in depression. Of course! The following are a few typical therapies for depression:

- ✓ Counseling/Therapy: Cognitive-behavioral therapy (CBT), psychodynamic therapy, and interpersonal therapy (IPT) are some of the therapeutic modalities that can assist people in comprehending the ideas, emotions, and actions that are connected to depression.

- ✓ Medication: To treat depression, doctors frequently prescribe antidepressants like SNRIs (Selective Serotonin Reuptake Inhibitors), SSRIs (Serotonin and Norepinephrine Reuptake Inhibitors), and others.
- ✓ Lifestyle Modifications: Getting regular exercise, eating a healthy diet, getting enough sleep, and practising stress reduction can all greatly lift your spirits and lessen the symptoms of depression.
- ✓ Electroconvulsive Therapy (ECT): If other treatments have failed for severe depression, ECT may be a possibility. While the patient is sedated, the brain is briefly stimulated electrically.
- ✓ Transcranial Magnetic Stimulation (TMS): This non-invasive technique, which is frequently used when other therapies have failed, stimulates brain nerve cells using magnetic fields.
- ✓ Support Groups and Peer Support: Being a part of a support group or having a close-knit circle of friends and family can offer motivation and emotional support.

The intensity of the depression, personal preferences, medical history, and professional advice from healthcare providers all play a role in the treatment decision. For some people, it might also be advantageous to combine various strategies. Always seek the advice of a healthcare provider to ensure a proper diagnosis and to determine the most effective course of treatment.[15-17]

Drugs used for depression:

The absence of treatment for depression lowers quality of life. People who are depressed experience a lower sense of wellbeing and struggle to appreciate or even watch television. Anhedonia is one of the symptoms that many doctors and their senior patients mistake for aging, leading to the untreated diagnosis of depression. Pharmacotherapy especially enhances quality of life and has been demonstrated to be an effective treatment for depression in the elderly. Antidepressants have been demonstrated in numerous

studies to be a safe treatment for depression in later age. Compared to previous medications, such as monoamine oxidase inhibitors (MAOIs) and tricyclic antidepressants (TCAs), serotonin selective reuptake inhibitors (SSRIs) and other modern or second generation antidepressants seem to be more palatable and easier to use in the elderly.18-21

Herbal therapies for depression:

Numerous herbal remedies demonstrated a wide range of pre-clinical antidepressant effects. The known psychopharmacological actions of some antidepressant herbal medicines, such as H. perforatum, Rhodiola rosea (roseroot), and Crocus sativus (saffron), such as the inhibition of monoamine re-uptake (including serotonin, dopamine, and noradrenaline), enhanced binding and sensitization of serotonin receptors, monoamine oxidase inhibition, and neuro-endocrine modulation, show promise for the treatment of this disorder. GABAergic effects, cytokine modulation (particularly in depressive disorders with a co-occurring inflammatory disease), and effects on the opioid and cannabinoid systems are possible additional effects.Most phytomedicines have a variety of biological effects on the reuptake and receptor binding of different monoamines, frequently in addition to endocrine and psychoneuroimmunological regulation, making their antidepressant mechanisms of action less well defined than those of SSRIs. [22-25]

Ayurveda for depression:

Every sickness has a predominance of one humor over another, according to Ayurveda. The humor that controls all bodily and mental processes, vata, is vitiated, and this leads to the majority of the classic symptoms of depression. Prana vata is the motivator and controller of manas among the five types of vata. It possesses the quality of budhidharana, or the retention of intellect. Depression- related prefrontal lobe damage results in poor perception, inability to concentrate, distractibility, memory loss, and lack of initiation. As a result, cognitive, memory, and retention are compromised, leading to psychological problems. Manodharana, or mental control, is another characteristic of prana vata. Humor will therefore be vitiated when mental, emotional, and physical processes are impacted. Udana vata, which aids with motivation, energy, and memory, is also in charge of the disease's expression. The vata element can be used to explain the agitation, anxiety, and weight loss associated with mild to moderate depression. There aren't many symptoms associated with kapha, the humor that lubricates and supports the body and mind. In addition to the humors already stated, there is also vitiation of sadhaka pitta and vyana vata, which are found in the heart, or hridaya, the seat of consciousness that controls emotions.[26-28]

Open conversations- A key to tackle depression:

Understanding depression requires empathy—a willingness to acknowledge the pain that lurks beneath the surface. It's about recognizing that the darkness isn't a choice but a formidable force that demands resilience and support to overcome. Just as we wouldn't fault someone for succumbing to a physical ailment, we must extend the same compassion to those grappling with the invisible wounds of depression. The journey to healing begins with destigmatizing mental health, fostering open conversations, and creating safe spaces where individuals can share their struggles without fear of judgment. Depression thrives in isolation but with a supportive community, its power diminishes.

Recovery is not a linear path; it's a series of small victories, each step forward a testament to the strength within. Professional help, whether through therapy or medication, plays a crucial role in navigating the labyrinth of depression. Seeking assistance is not a sign of weakness but a courageous step towards reclaiming one's life. As we collectively strive to dismantle the barriers surrounding mental health, let us remember that empathy is the bridge that connects us. A simple act of kindness, a listening ear, or a reassuring presence can be a lifeline for someone grappling with depression.[29-30]

Conclusion:

In the face of this formidable foe, let us be beacons of understanding, compassion, and hope. By fostering a culture that prioritizes mental well-being, we can collectively unravel the shadows that shroud depression, allowing the light of resilience and healing to prevail.

Millions of people worldwide are impacted by the complicated and serious mental health illness known as depression. Recognizing its complexity, getting the right help, and raising awareness are still essential to treating and lessening its consequences on people and society at large.

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Dissociative Identity Disorder

Ms. Gayatri H. Patil, Dr. Sanket Kadam
Department of Pharmacy Practice, Dr. D.Y. Patil College of Pharmacy, Akurdi Pune

Introduction-

Dissociation is a general term that refers to the separation of any normally integrated psychological processes, encompassing both dissociative amnesia and the dissociative state. Dissociative Identity Disorder (DID), also known as Multiple Personalities Disorder (MPD) is one of the controversial disorders since the 20th century.

Dissociative Identity Disorder (DID), previously referred to as Multiple Personalities Disorder, has been historically misrepresented in the media and excluded from professional training. Dissociative identity disorder (DID) is classified by DSM-V as "presence of two or more distinct identities or personality states, each with its own patterns of perceiving, thinking, and relating to the environment and the self " where "at least two of these identities or personality states recurrently take control of the person's behavior".[1]

These disorders include dissociative identity disorder, dissociative amnesia, depersonalization/derealisation disorder, other specified dissociative disorder, and unspecified dissociative disorder. -Dissociative identity disorder is examined in the literature according to a variety of discourses, each of which suggest different ways of conceptualizing problems and therapeutic approaches. These discourses reviewed include: psychiatry, psychology, corporeality, feminism, social constructivism, anthropology, and postmodernism.

The first complete account of a patient with DID was written in 1865. In 1869, a system of ideas split off from the prominent personality was discovered by French neurologist Pierre Janet with hypnotizing methods, later termed "dissociation" by William James, the father of American psychology. In the 1970s, the diagnosis of DID rose dramatically after the publication of the top-rated book Sybil in 1973. In the 1970s alone, more cases of DID were reported than in all of history (since 1816), along with the famous case of Mary Reynolds. In 1980, the American Psychiatric Association officially recognized and designated DID a genuine emotional illness.[2]

Risk factors-

The Child's exposure to trauma was found as a major risk of developing a dissociative disorder. Hereditary factors mostly does not show a risk of prevalence. Psychiatric comorbidity have high rate of risk chances which may prevent clinicians from recognize the dissociative disorder in the overall population. Other risk factors include lack of socio and familial support, inter personal and environmental stress.[3]

Clinical features-[4]

The average number of personalities in BPD has been found to be between 15-18. Often only two or three of the personalities are present at the time of diagnosis. The others can be identified during the treatment process. Alter personalities may be of both sexes, of different races and ages, and associated with a separate community or family. The most common alter personality is the child alter personality. The prevalence of paediatric alter personality is 85-96%, while the prevalence of an alter personality of a gender different from the patient's gender has been found to be between 26-65% as a result of various studies.

Signs and symptoms-[5]

DID patients may suffer from symptoms associated with mood, anxiety, personality, eating, functional somatic, and substance use disorders, as well as psychosis, among others.3–8 DID can be overlooked due to both this poly symptomatic profile and patients' tendency to be ashamed and avoidant about revealing their dissociative symptoms and history of childhood trauma.

Pathophysiology-[6]

Ross, et al. concluded that there is a strong co-existence of DID and bipolar disorder, these kinds of studies just provide a hint of a possible association between the two conditions and any changes in the brain structure. DID has been found associated with some personality disorders with avoidant disorder (76%) being the most common; followed by self-defeating (68%); borderline (53%), and passive aggressive (45%) personality disorders.

Models-[7]

There are two major competing models of the origins of DD and corresponding dissociative Taylor & Francis symptoms:

1. Trauma related Model (TM) and

2. Fantasy Prone Model.

The trauma-related model states that severe early childhood abuse increases the risk for the development of dissociation. patients who had suffered childhood abuse had a significant reduction in hippocampal volume with a higher DES score also observed an increase in dissociative symptoms in survivors of childhood abuse. Despite the existing data supporting this theory, many psychiatrists and psychiatric researchers remain unconvinced.

The non-trauma related model, or Socio-cognitive model or Fantasy model, states that dissociative symptoms in DID are caused by simulation, suggestive psychotherapy, and/or sociocultural influences. This theory suggests that the development of DID is mediated by high fantasy proneness.

Diagnosis-[8]

The diagnosis is done via mental status examination, basic psychiatric interview, by screening people for dissociation or by differential diagnosis. There several screening tests performed to assess the presence of Dissociative Identity Disorder clinically such as-

1. The Dissociative Experiences Scale (DES)
2. The Somatic Dissociation Questionnaire (SDQ-20)
3. The Multidimensional Inventory of Dissociation (MID
4. A Structured Diagnostic Tool: The Dissociative Disorders Interview Schedule (DDIS)
5. A Semi-Structured Diagnostic Interview: The Structured Clinical Interview for Dissociative Disorders (SCID-D

Public Health Issues Associated with Dissociative Disorder-[9]

1. It involves disability or impaired functioning
2. Hospital and healthcare utilization
3. Chronic health issues
4. Self harm and suicidality
5. Re-victimization
6. Social services involvement

Treatment-[10]

There are various types of treatment given to patient diagnosed with Dissociative Identity Disorder like-

1. Psychotherapy - Individual therapy to address trauma and promote integration. Sessions may involve talking to various alters (personality states)
2. CBT (cognitive behavioral therapy) It addresses negative thought patterns and behaviors. Can be helpful for comorbid conditions like anxiety or depression.
3. TF-CBT (trauma-focused cognitive behavioral therapy) created to treat children and adolescents who have experienced trauma. TF-CBT is an evidence based treatment (EBT) and is currently the only EBT whose focus is solely on treating trauma in children and adolescents.
4. Hypnotherapy- Hypnosis is a way of communicating ideas in the context of a doctor-patient or therapist client relationship. It is a therapeutic tool for systematically amplifying dimensions of experience, and then associating those experiences to situations in ways that are useful to the patient.-
5. Medication- The antidepressants, anti-anxiety and anti psychotic drugs are mainly prescribed in Dissociative Identity Disorder
6. Group therapy- Provides a supportive environment for sharing experiences and coping strategies.
7. Inpatient treatment- It include alternative and holistic therapies, group and family support, exercise and nutrition.

Complications-[11]

People with DID are at increased risk of severe complications and associated conditions such as

1. Self-harm
2. Sexual dysfunction
3. Eating disorders
4. Depression or anxiety disorders
5. Suicidal thoughts and behavior
6. Insomnia, nightmares or other sleeping disorders.

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HARLEQUIN ICHTHYOSIS: A RARE SKIN DISORDER.

Snehal Gaikwad, Disha Wadave, Sakshi Babar, Ms. Pooja A. Palandurkar
Department of Pharmaceutics, Dr. D. Y. Patil College of Pharmacy Akurdi, Pune.

Introduction:[1,2]

Harlequin ichthyosis is a severe genetic disorder that affects the skin. Infants with this condition are born prematurely with very hard, thick skin covering most of their bodies. The skin forms large, diamond-shaped plates that are separated by deep cracks (fissures). These skin abnormalities affect the shape of the eyelids, nose, mouth, and ears, and limit movement of the arms and legs. Restricted movement of the chest can lead to breathing difficulties and respiratory failure in babies with harlequin ichthyosis. Affected infants also have feeding problems. The skin normally forms a protective barrier between the body and its surrounding environment. The skin abnormalities associated with harlequin ichthyosis disrupt this barrier, making it difficult for affected infants to control water loss, regulate their body temperature, and fight infections. Infants with harlequin ichthyosis often experience an excessive loss of fluids (dehydration) and develop life-threatening infections in the first few weeks of life. It used to be very rare for affected infants to survive the new-born period. Following the new-born period, the hard, skin plates are shed and the skin develops widespread scales and redness. However, with intensive medical support and improved treatment, babies with this disorder now have a better chance of living into childhood and early adulthood.

History:[1,2]

Odisha reported its first-ever case of a baby born with harlequin ichthyosis, a rare genetic condition, at a hospital in Berhampur in the state's Ganjam district April 22, 2021. The baby girl was born to a 30-year-old woman who has multiple congenital anomalies, Santosh Kumar Mishra, superintendent of the medical college and hospital, said. The facial features of the baby, including the mouth, eyes and ears were deformed, restricting breathing and eating. The baby was kept in the intensive care unit. The condition of the mother was good, Indira Palo, assistant professor at the

college and hospital's gynaecology department, said The disease affected one in three million births and is caused due to a mutated gene inherited from the parents. The disease sees the skin form large diamond-shaped plates across the body that are separated by deep cracks (fissures). The skin is dry and scaly, almost like fish skin and hence the term 'icthyosis', derived from 'ikthus', Greek for fish.

OTHER NAMES OF THIS DISEASE

- Autosomal recessive congenital ichthyosis 4B
- Harlequin baby syndrome ,
- HI
- Ichthyosis congenita, harlequin fetus type 3

Epidemiology:[3]

It is rarely found in girl child. There are few sporadic case reports in the literature indicating its rarity. However, it is a common presentation among healthy new-borns, and the incidence has been reported as high as 10%. It usually occurs from day 2 to day 5 of life and rarely occurs after three weeks. It was previously thought to be more common among premature neonates, but recent studies show a similar incidence among healthy term neonates

Etiology:[3]

Harlequin ichthyosis is caused by changes (mutations) in the ABCA12 GENE, which gives instructions for making a protein that is necessary for skin cells to develop normally. It plays a key role in the transport of facts (lipids) to most superficial layer of the skin (epidermis), creating an effective skin barrier.

Causes:[4]

- Variants (also known as mutations) in the*ABCA12*gene cause harlequin ichthyosis. The*ABCA12*gene provides instructions for making a protein that is essential for the normal development of skin cells.
- This protein plays a major role in the transport of fats (lipids) and enzymes in the outermost layer of skin (the epidermis).
- Some variants in the*ABCA12*gene prevent the cell from making any ABCA12 protein. Other variants lead to the production of an abnormally small version of the protein that cannot transport lipids properly.
- A loss of functional ABCA12 protein disrupts the normal development of the epidermis before and after birth, resulting in the severe skin abnormalities characteristic of harlequin ichthyosis.

Sign and Symptoms of Harlequin Ichthyosis:[4,5]

Signs and Symptoms In New Borns:

Babies with Harlequin ichthyosis are usually born prematurely. That means they may have a higher risk of other complications as well.The sign people usually first notice is hard, thick scales all over the body, including the face. The skin is pulled tightly, causing the scales to crack and split open.This hardened skin can cause a number of serious issues, including:

- Eyes not closing.
- Lips pulled tight, leaving the mouth open and making nursing difficult.
- Ears fused to the head.
- Small, swollen hands and feet.
- Imited mobility in arms and legs.
- Nursing difficulties.
- Breathing problems due to tight chest skin.

In Older Children & Adult:

Children with Harlequin ichthyosis may experience a delay in physical development. But their mental development is usually on track with other children their age.A child born with Harlequin ichthyosis will likely have red, scaly skin throughout their life.They may also have:

Sparse or thin hair as a result of scales on the scalp.

Unusual facial features due to stretched skin.

Reduced hearing from a build-up of scales in the ears.

Problems with finger movement due to tight skin.

Thick fingernails.

Recurring skin infections.

Overheating due to scales that interfere with sweating.

Diagnosis:[5]

Harlequin ichthyosis is usually diagnosed at birth based on appearance. It can also be confirmed through genetic testing. These tests can also determine if it is another type of ichthyosis. But genetic testing doesn't offer any information on disease severity or prognosis.

1. **Treatment:**[5,6]

With improved neonatal facilities, infants born today have a better chance of living longer, healthier lives but early, intensive treatment is vital. A new-born with Harlequin ichthyosis requires neonatal intensive care, which may include spending time in a heated incubator with high humidity. Tube feeding can help prevent malnutrition and dehydration. Special lubrication and protection can help keep eyes healthy.

Other initial treatments might include:

Applying retinoid to help shed hard, scaly skin.Applying topical antibiotics to prevent infection.Covering the skin in bandages to prevent infection.Placing a tube in the airway to help with breathing.Using lubricating eye drops or protective devices on the eyes

Precaution:[6,7]

1. There's no cure for Harlequin ichthyosis, so management becomes a crucial part of the equation after initial treatment & it's all about the skin.
2. Skin protects the body from bacteria, viruses, and other harmful elements in the environment.
3. It also helps to regulate body temperature and fluid loss.
4. That's why keeping your skin clean, moist, and supple is so important for children and adults with Harlequin ichthyosis.

5. Dry, tight skin can crack and become vulnerable to infection.
6. For maximized benefit, apply ointments and moisturizers right after a bath or shower, while the skin is still moist.

Conclusion:[6,7]

Harlequin syndrome is a rare autonomic disorder characterized by unilateral facial flushing and sweating with contralateral an hydrosis induced by exercise, heat, and emotion. It is usually idiopathic but could be the first manifestation of several serious underlying medical conditions. Medical or surgical treatments are not required for idiopathic Harlequin syndrome, but social and psychological factors may indicate sympathectomise or botulinum toxin injection.

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DEGOS DISEASE: A RARE GENODERMATOSIS

Miss Sonam Bendre, Miss Shital Rajad, Dr. Revan Karodi
Department of Pharmacognosy, Dr. D. Y. Patil College of Pharmacy Akurdi, Pune.

Introduction

Dowling-Degos disease (DDD) is an uncommon genetic skin condition characterised by flexural dark pigmented reticulate macules, comedo-like papules on the back and neck, and pitted perioral or face scars. Clinical and histological correlations are used to make the diagnosis. [1,2] In 1941, in an essay entitled "Multiple Hautrekrosen bei Thromboangiitis obliterans," Kohlmeier documented a case of a disease that has now been dubbed malignant atrophic papulosis (MAP) or Degos disease. In 1942, Degos identified it as a unique clinical entity.[3] Dowling Disease was initially described by Wilson Jones and Grice in 1978. (DDD). It is a rare condition that affects women between the ages of 20 and 40. It has a late onset and a benign progression; yet it is exceedingly unaesthetic. Clinically, it manifests as acquired reticulated hyperpigmentation, primarily in flexures that do not change or are affected by sun exposition. They have also been diagnosed with cribriform scars and perioral acne, with no prior history of acne. Furthermore, DDD patients with comedo-like lesions on the face, dorsum, and other places listed above are noticed.[4] Degos disease is classified as a vasculopathy or endovasculitis. It is a type of occlusive arteriopathy that involves small-caliber arteries. It is a progressive, small- and medium-sized artery occluding condition that causes tissue infarction and initially affects the epidermis. Degos disease manifests as a limited benign cutaneous form as well as a potentially fatal multiorgan, systemic version. Degos disease appears on the skin as erythematous, pink or red papules. When these papules heal, they leave pathognomonic scars with core, porcelain white atrophic centres. These papules typically have a telangiectatic rim.

Epidemiology

Dowling-Degos illness is a rare genodermatosis; less than 100 instances have been recorded in the literature. Both sexes are affected, with a slight female predominance reported by a few publications. DDD does not have a racial preference. DDD normally appears in adults, however it can appear in childhood as well.[6]

Etiology

Degos illness has no recognised cause. A virus, an immunological deficiency, or a clotting problem are all possible reasons.

Sign and Symptoms

The first signs of Degos disease are usually prominent skin lesions or a rash. Those who are affected acquire little elevated lumps or spots (papules) of various shape, generally on the trunk, upper arms, and upper legs. At first, only a few lesions may be seen. Ultimately, 10-40 lesions may form slowly, and in some cases, hundreds. Usually, the palms, soles, and face are spared. The lesions may itch at times (pruritis). Lesions begin as reddish or pink lumps and eventually degenerate (atrophy) to the point where older lesions have a crimson border with porcelain-white, atrophic centres.[3]

Causes

Degos disease's specific cause is unknown. The illness process causes the cells lining the arteries to grow, contributing to artery constriction or obstruction (arterial occlusion). When blood flow is restricted by narrowed or blocked arteries, areas of severely damaged tissue (necrosis) may occur (occlusive arteriopathy). The symptoms of Degos disease vary depending on where the blocked arteries and necrotic lesions are located.

Several theories have been offered to explain what causes Degos disease. There are three major theories: (1) viral infection, (2) problems in the body's blood clotting ability (primary disease of coagulation), and (3) autoimmunity, in which the immune system incorrectly assaults healthy tissue. Certain cases of Degos disease have been reported to run in families, implying a genetic predisposition to the ailment. This family type of Degos disease has no known method of inheritance. Surprisingly, this type of Degos disease is usually restricted to the skin (benign cutaneous Degos disease).[7]

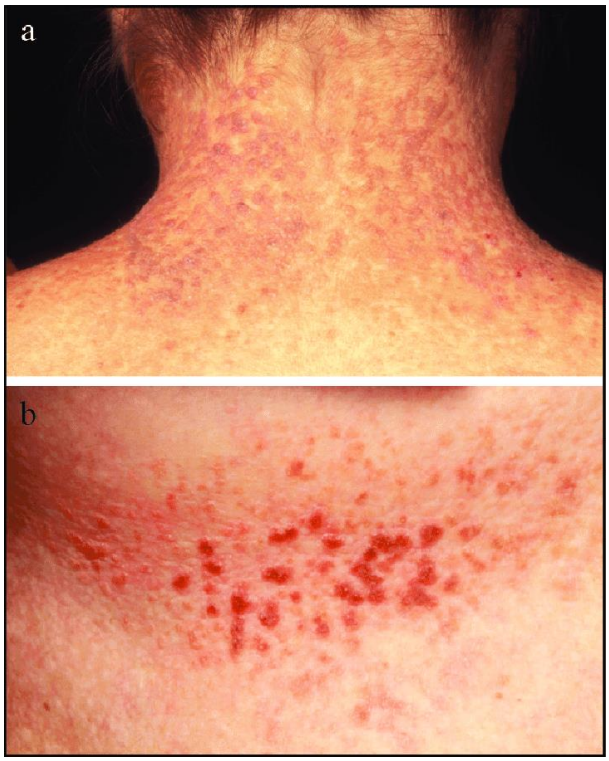
Diagnosis

A thorough clinical evaluation, a complete patient history, identification of typical features (e.g., skin lesions), and microscopic analysis of affected

skitissue that reveals specific alterations to that tissue are used to make a diagnosis of Degos disease. While the majority of tests are normal, no single laboratory test can be utilised to help diagnose Degos disease.

Pathophysiology

Degos disease's genesis and pathophysiology remain unknown. Degos disease has been categorised as a vasculitis, mucinosis, or thrombotic condition by some. Vasculitis, coagulopathy, and primary endothelial dysfunction are the most widely proposed explanations.[8] No circulating immune complexes, antiendothelial cell antibodies, or anticardiolipin antibodies are often identified. Yet, in rare circumstances, antiphospholipid antibodies of unknown relevance are discovered. Understanding atrophic papulosis is fraught with ambiguity. The variances in blood arteries in each area of the body (e.g., skin, brain, intestines) are likely to help explain the various illness phenotypes. Some experts believe that an initial endothelial cell abnormality combined with subsequent thrombosis causes infarctive alterations that appear as Degos disease. There is no evidence to establish particular antibody-mediated harm. Additionally, medications and toxic chemicals do not appear to induce Degos disease. Physical damage to blood arteries includes, at least in part, decreased fibrinolytic activity and changes in platelet function. Degos disease may not be classified as a vasculitis because there is little inflammation of the vessel walls and no immune complexes have been detected in the vessel walls. Three plausible reasons for this pathology have been proposed: immune disruption, viral infection, and abnormalities in the blood clotting system. An autosomal dominant mode of inheritance has been proposed in family situations, but this is questionable. Using polymerase chain reaction, no paramyxovirus was found in instances of Degos disease with just cutaneous lesions. The efficacy of eculizumab in the treatment of Degos disease must change our understanding of the disease. Degos disease may be a hematological/endothelial/clotting disease that involves C5 in some pathological cascade because eculizumab is a haematological medication that targets complement component 5 (C5). A hereditary abnormality similar to paroxysmal nocturnal hemoglobinuria (PNH) would not be unexpected. This topic has been extensively discussed by Scheinfeld.[9] Support for the idea that Degos disease is a genetic defect of the endothelial tissue (with or with out a viral



trigger) comes from Passarini who noted that in a patient suffering from systemic Degos disease who received an organ transplant, who died, on autopsy did not have any Degos disease-like changes in the transplanted tissue.[10] A 2013 Japanese study looked at the expression of stromal cell-derived factor (SDF)-1/CXCL12 in Degos disease. SDF-1/CXCL12 is secreted by bone marrow stromal and endothelial cells. Megakaryocyte precursors are activated by SDF-1/CXCL12. Platelet activation is co-stimulated by SDF-1/CXCL12. The researchers studied two patients with Degos disease, one with cryoglobulinemia, one with antiphospholipid syndrome, and two healthy controls. There was no staining in antiphospholipid syndrome, cryoglobulinemia, or control patients. Investigators observed strong SDF-1/CXCL12 staining penetrating inflammatory cells in Degos disease patients. These cells might be found in the perivascular, intravascular, and perineural tissues. This data lends credence to the hypothesis that Degos disease is, at least in part, an endothelial illness.[11]

Investigational Therapies

Individuals with Degos disease have been treated with drugs that restrict the action of platelets (specialised blood cells that cluster together to form clots to halt bleeding at an injury site). These medications are

known as anti-platelet or anti-coagulation medications. A combination of two of these medications (aspirin and dipyridamole) has been shown to improve skin and ocular lesions in two people with no systemic involvement. A number of drugs, including those that reduce the body's immune system (immunosuppressives), have been tested for Degos disease with no success. One case described in the medical literature showed that nicotine patches improved skin lesions and gastrointestinal issues. Some researchers have proposed for the use of intravenous immunoglobulin as a treatment for affected persons. Further research is needed to assess the long-term safety and efficacy of such therapy in people with Degos disease.[11]

Conclusion

To summarise, DDD is an autosomal dominant genodermatosis characterised by acquired pigmentation over the flexural sites, follicular comedone-like papules, and pitted perioral scars. This syndrome develops in maturity and progresses. Thus far, three primary genes have been linked to the pathophysiology of DDD: KRT5, POFUT1, and POGLUT1. This condition is part of a group of reticulate hyperpigmentation syndromes that also includes Galli-Galli disease, Haber syndrome, Dohi acropigmentation, and Kitamura reticulate acropigmentation. Sadly, this illness is degenerative, and most treatment options fail to entirely resolve the lesions.

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MAPLE SYRUP URINE DISORDER

Ms. Darshita Makhija, Dr. Shubhangi Jadhav
Department of Pharmacognosy : Dr. D. Y. Patil College of Pharmacy Akurdi, Pune

Introduction:

Maple syrup urine disease (MSUD) is a rare but serious inherited condition. It means the body cannot process certain amino acids (the "building blocks" of protein), causing a harmful build-up of substances in the blood and urine. Normally, our bodies break down protein foods such as meat and fish into amino acids [1], but in case of MSUD this does not happen. MSUD was first described in the medical literature in the 1950s and has since been the subject of extensive research. Several studies have explored the genetics of the disease, identifying mutations in the genes that code for the enzymes involved in amino acid metabolism [1,2]. Other studies have focused on developing new treatments, such as gene therapy and novel drugs that can help manage the symptoms of MSUD [3,4]. While MSUD is relatively rare, affecting approximately 1 in 185,000 newborns in the United States [5], it can have severe consequences if left untreated. Early diagnosis and treatment are crucial for preventing complications, such as intellectual disability, seizures, and even death. In this article, a comprehensive overview of maple syrup disease, including its causes, symptoms, diagnosis, treatment options, and challenges has been provided.

Causes:

MSUD is caused by mutations in one of three genes – BCKDHA/ BCKDHB/ DBT. These genes provide instruction for the human body to make enzymes (BCKDH complex enzymes) which are essential for breaking down amino acids including leucine, isoleucine, and valine. One of the main causes of MSUD is a deficiency in the enzymes responsible for breaking down certain amino acids. These amino acids (leucine, isoleucine, and valine) when are not properly metabolized, they can build up in the body and cause a range of symptoms (8). The symptoms of MSUD can vary in severity, but they typically include poor feeding and growth, lethargy, irritability, and developmental delays. Severe cases can also lead to seizures, coma, and even death (9). The genes BCKDHA, BCKDHB, and DBT are responsible for the branched-chain alpha-keto acid dehydrogenase complex (10). Type 1 MSUD is the most common and severe form of the disease and is caused by a deficiency in the branched-chain alpha-keto acid dehydrogenase complex. This complex is responsible for breaking down leucine, isoleucine, and valine, and when it is not functioning properly, these amino acids can build up in the body and cause a range of symptoms (11). Type 2 MSUD is caused by a deficiency in the E2 subunit of the branched-chain alpha-keto acid dehydrogenase complex, which is responsible for breaking down leucine. Type 3 MSUD is caused by a deficiency in the dihydrolipoamide dehydrogenase enzyme, which is responsible for breaking down isoleucine (12). MSUD is more common in certain populations, such as the Mennonite and Amish communities, where the incidence of the disease is much higher than in the general population (13). Treatment for MSUD typically involves a low-protein diet that is supplemented with a special formula to ensure that the body gets the nutrients it needs. Medications may also be prescribed to help remove excess amino acids from the body (14). With proper treatment, people with MSUD can live relatively normal lives. However, it is important to strictly adhere to the dietary restrictions and treatment plan in order to prevent complications and ensure the best possible outcome (14). Maple syrup urine disease (MSUD) is a rare genetic disorder characterized by deficiency of an enzyme complex (branched-chain alpha-keto acid dehydrogenase) that is required to break down (metabolize) the three branched-chain amino acids (BCAAs) leucine, isoleucine and valine, in the body. If untreated, life-threatening coma or respiratory failure can occur within 7 to 10 days and death can occur within the first two months.

Symptoms: [16]

All four types of MSUD have symptoms including:

1. Urine, sweat, or earwax that smells like maple syrup or burnt sugar. ...
2. Poor feeding, vomiting, loss of appetite, irritability.
3. Sluggish/slow/tiredness and weakness.
4. Changes in muscle tone – poor muscle tone, muscle tightness/tension.

Diagnosis: The diagnosis of MSUD typically involves a combination of clinical evaluation,

biochemical testing, and genetic analysis. Clinical features such as poor feeding, vomiting, seizures, and the characteristic sweet odor of the urine and sweat may raise suspicion for MSUD, but confirmatory diagnosis requires measuring the levels of branched-chain amino acids (leucine, isoleucine, and valine) and their keto-acid derivatives in blood and urine. Elevated levels of these amino acids and their metabolites can confirm the diagnosis.

Genetic testing can also be used to identify the specific mutations responsible for the condition, which can aid in making a definitive diagnosis and in counseling affected families about the likelihood of recurrence [1]. Newborn screening programs in many countries now include MSUD screening, which can detect the condition before symptoms appear, allowing for early intervention and improved outcomes. Treatment must be initiated promptly, even before confirmation of the diagnosis, to avoid life-threatening metabolic crises. At around 5 days old, babies are offered newborn blood spot screening to check for inherited conditions like MSUD [5]. This involves pricking your baby's heel to collect drops of blood to test. If your baby is diagnosed with MSUD, treatment should be given straight away to reduce the risk of serious complications. It is important to note that some forms of MSUD may not be detected by routine screening methods and may require additional testing, such as enzyme analysis or molecular genetic testing. Early diagnosis and treatment are crucial for preventing serious complications and improving the long-term outlook for individuals with MSUD [6]. Plasma amino acids (PAA) testing should be performed to assess for elevated levels of branched-chain amino acids (BCAAs) and to detect l-alloisoleucine (derived from l-isoleucine). The detection of l-alloisoleucine (also termed alloisoleucine) is diagnostic for maple syrup urine disease.

Treatment:

The main treatment for MSUD is a low-protein diet with low levels of the three amino acids. Babies with MSUD must be on a special formula as soon as possible. Then, they follow a special diet for the rest of their lives. Some also need to take nutritional supplements. The primary treatment for MSUD is a special diet that restricts the intake of certain amino acids, such as leucine, isoleucine, and valine[2]. This diet is usually combined with dietary supplements and regular monitoring of blood levels of amino acids and other metabolic products. In some cases, patients may require hospitalization for intravenous administration of amino acids and glucose to maintain normal blood levels during times of stress, such as illness or surgery. Early diagnosis and prompt treatment are crucial to prevent serious complications from MSUD. Genetic counseling and screening are also important for families with a history of the condition [8].

Role of Diet: Successful treatment of MSUD involves the following foods to avoid - High protein foods such as meat, fish, chicken, eggs, milk, cheese, yogurts, soya, nuts, bread, pasta and chocolate which are generally too high in leucine, isoleucine and valine and are not allowed in the diet [3]. The role of diet is crucial in the management of maple syrup urine disease (MSUD). The goal of the MSUD diet is to restrict the intake of these amino acids while ensuring adequate intake of other essential amino acids to support growth and development. Patients may require specialized medical formulas or dietary supplements to ensure adequate nutrition while adhering to the diet. Compliance with the MSUD diet can be challenging, especially during adolescence and adulthood when patients may be more likely to deviate from the prescribed diet. It is important to note that the MSUD diet is lifelong and requires close monitoring of blood levels of amino acids and other metabolic products. Patients may also require hospitalization for intravenous administration of amino acids and glucose during times of stress such as illness or surgery. In addition to dietary restrictions, individuals with MSUD should avoid foods and drinks containing artificial sweeteners and should consume foods low in protein. Nutritional counseling and support are important for maintaining long-term adherence to the MSUD diet.[2]

Complications: Maple syrup urine disease affects an estimated 1 in 185,000 infants worldwide. The disorder occurs much more frequently in the Old Order Mennonite population, with an estimated incidence of about 1 in 380 newborns [1]. Classic maple syrup urine disease (MSUD) is typically diagnosed in newborns, whereas nonclassic forms may manifest at any age [6]. Females with MSUD are capable of having normal children, but need to adhere strictly to the diet and be monitored carefully, particularly postpartum, by metabolic geneticists. Without treatment, one can expect intellectual disability, neurologic disturbances, and early death.

Conclusion: In summary, the diagnosis and management of Maple Syrup Urine Disease (MSUD) represent significant challenges in the field of inherited metabolic disorders. Early detection of the disease through newborn screening, followed by strict dietary management, is crucial for preventing

long-term complications. However, despite the significant progress in our understanding of the genetic and biochemical mechanisms underlying MSUD, there are still many unanswered questions, particularly regarding the molecular basis of the disease and the optimal approaches for treatment. Further research into the pathophysiology of MSUD, as well as the development of new therapeutic strategies, is necessary to improve outcomes for affected individuals. Advances in genetic engineering and gene therapy hold great promise for the future of MSUD treatment, but much work remains to be done before these approaches can be translated into effective clinical interventions. Overall, the study of MSUD provides a fascinating insight into the complexities of inherited metabolic disorders and underscores the importance of ongoing research in this field.

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KLINEFELTER SYNDROME

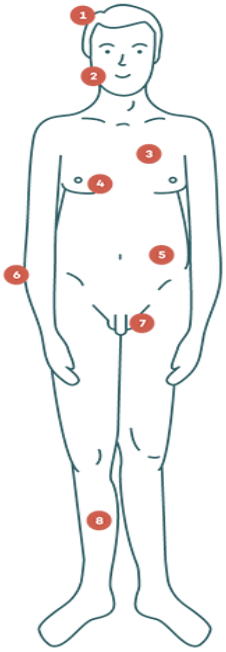
Bapat Sai Shrikant, Jadhav Varsha Rajendra, Dr. Mrs. Sonali P. Mahaparale
M. Pharmacy Quality Assurance Techniques: Dr. D. Y. Patil College of Pharmacy

The first ever Klinefelters syndrome (KS) awareness day was celebrated on 10th May 2022. [7] Sometimes KS is associated with language problems and learning disabilities. It is also called as 47 XXY.

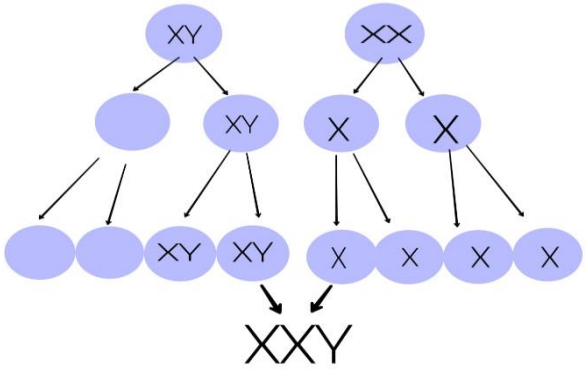
INTRODUCTION: Klinefelter syndrome is named after the Dr. Henry Klinefelter, who identified this condition in the early 1940s. Normally, every cell in a male's body, except sperm and red blood cells, contains 46 chromosomes, but males containing this syndrome have 47 chromosomes. Usually male contains XY chromosomes but in KS syndrome have an extra X chromosome, which makes them 47 XXY (karyotype). [2][6] Klinefelter syndrome is a congenital condition, which means it's present. from the time of birth. But many men (some say 70 to 80%) likely don't know they have this condition.

ETIOLOGY: KS occurs when a male baby is born with at one extra X chromosome. This imbalance is due to an error during egg or sperm development and causes in a male having an extra X chromosome in all of his body's cells. It is a genetic disorder. KS is found in about 1 in 500 to 1,000 newborn males.

SIGNS AND SYMPTOMS: In babies, symptoms are hernia, slower to learn to sit, crawling, and talk. Testies does not drop into scrotum. [5] Boys in their teenage have behavior changes like mostly stay quite, sensitive, unassertive. [6] Get taller than other normal boys (1) and have more belly fat than their peers (5). They are slow in learning, talking, reading, and have difficulties in hearing. [1] They have small penis size (7), less muscle tone, wider hips, varicose veins (8), etc. Male having KS shows signs low testosterone levels, low sex drive, infertility, breast development (4) (gynecomastia), reduced muscle mass (6), broad hips, weaker bones, reduced body hair (3) and facial hairs(2), etc. Following figure shows symptoms of KS with numbers listed 1-8 above.



PATHOPHYSIOLOGY: As shown in following figure, the XY nondisjunction in male fertilizes with normal womens X chromosome in meiosis II phase to produse trisomy XXY (47).



COMPLICATIONS: There are some complications such as heart disease, diabetes, osteoporosis, autoimmune disorders (including lupus), cancer (including breast cancer), lung disease, varicose veins, dental cavities, anxiety, and depression. [8]
DIAGNOSIS: Genetic test can diagnose KS. 1.

Karyotype Test - A small skin or blood sample is sent to a laboratory to find out if there is an extra X chromosome. [5] 2.In puberty a physical examination of the chest and testes such as small testes and enlarged breasts shows presence of KS.3. Sperm count test- It is done for reduced fertility and a hormone test for reduced testosterone. Only 10 % Trusted Source of cases are diagnosed in childhood, while on average, males with KS are not diagnosed until their mid-30s. Sometimes because of similar symptoms of other diseases, KS remains undiagnosed.

TREATMENT: KS can't be cure but can reduces symptoms by treatments.

1.Testosterone replacement therapy- It is done by using an injection, pills, gel, or a patch. It improves strength, body hair growth, energy, and concentration. Testosterone therapy does not improve testicle size or fertility. [3][5]

2. Fertility treatment - 95 - 99 % of males with KS are infertile because they can't produce enough sperm to fertilize an egg. For those males, an intracytoplasmic sperm injection (ICSI) is effective. During an ICSI, sperm is removed from the testicle and injected directly into the egg. [4][8]

3. Cryopreservation - If KS is diagnosed earlier, semen or testicular tissue can be preserved before the testicular damage starts, probably at puberty.

4. Breast reduction surgery- There is no approved drug treatment for overdeveloped breast tissue in males. Removal of the breast tissue by a plastic surgeon is effective but comes with the risks. But it reduces the chances of developing breast cancer. [8]

5. Psychological counseling and speech therapy, physical therapy, educational evaluation, behavioral therapy. [4][6]

CONCLUSION:

The Klinefelter's syndrome is frequent and, if not diagnosed (which seems to be the most common case), these men have higher risks to develop psychiatric disorders. The diagnosis of KS would be less frequently missed if doctors were more aware of, and attentive to, its key manifestations, particularly the small, firm testes, erectile dysfunction, and the comorbidities mentioned above. So the conclusion is that if the diagnosis were made more often, patients would more often be able to receive early treatment, which would improve their quality of life. Klinefelter syndrome 47 XXY was first described 70 years ago (1). With an incidence of 0.1% to 0.2% of male neonates (i.e. 1 to 2 per 1000), it is one of the commonest congenital chromosome disorders resulting in hypogonadism and genetically-determined infertility.

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MCARDLE DISEASE: AN OVERVIEW

Ms. Jyotsna Chopade
Department of Pharmaceutical Chemistry, Dr. D. Y Patil College of Pharmacy, Akurdi, Pune

What is McArdle disease?

McArdle disease is a rare muscle disorder. In this disease, the muscle cells can't break down a complex sugar called **glycogen.** It is part of a group of diseases called glycogen storage diseases. Another name for McArdle disease is glycogen storage disease GSD 5 or GSD V.Your cells use a simple sugar, called glucose, for energy. When you eat, your digestive system sends a large amount of glucose into your blood. This raises your blood glucose levels. Your body removes this extra glucose from the blood to stabilize the blood glucose level. Your body then converts the extra glucose into glycogen. It stores it in the liver, muscles, and other places in the body. Glycogen is a form of energy storage.When you haven't eaten in a while, the glucose level in your blood starts to drop.

This tells your body to start using some of the glycogen it saved up earlier. The glycogen gets broken down into the glucose so that your body has a steady supply. Your muscles need a constant supply of glucose to keep working well. In McArdle disease, your muscles can't break down the saved up glycogen. That's because an important substance needed for that process is missing from your muscle cells.

This means your muscles can't use the stored glycogen to get the glucose they need. The key missing substance in your muscles is an enzyme called **myophosphorylase.** An enzyme is a substance that helps speed up chemical reactions in the body. time, symptoms of this health problem appear by the time a person is age 15.

What causes McArdle disease?

McArdle disease is an inherited disease. It results from changes (mutations) in the gene for the enzyme muscle phosphorylase. Your muscle cells can't make this enzyme. So they can't break down glycogen into glucose. Your muscles need glucose to work their best. The gene change that causes McArdle disease usually passes down from a parent to a child. You generally have a pair of genes (1 from each parent) for each substance your body makes. In most cases, a person with McArdle disease needs to have 2 copies of a mutated gene. This is **recessive** inheritance. A person who has only 1 copy of the mutated gene may still have some symptoms of McArdle disease.

What are the symptoms of McArdle disease?

McArdle disease causes muscle symptoms. Generally, it can make it hard for you to exercise without becoming tired. You may find that this does not happen with gentle walking. But you may have trouble with strenuous exercise for more than a few minutes. You may notice that after a brief rest you feel a "second wind" that lets you exercise again. These symptoms can vary in intensity. Some common symptoms of McArdle disease are:

- Better able to do aerobic exercise after 8 to 10 minutes (second-wind phenomenon)
- Brownish red urine, especially after periods of activity
- Easily tiring during activity, with stiffness or weakness soon after starting exercise
- Muscle cramping
- Muscle pain
- Permanent weakness in the thigh or other muscles. This happens in a small portion of people with the condition.

Most of the time, people notice these symptoms before age 15. Often, people assume the symptoms are "growing pains" or due to some other cause. Different people may have symptoms of different severity. You may even notice that the symptoms seem worse or better at different times.

How is McArdle disease diagnosed?

Your healthcare provider first takes a health history. He or she asks about your recent symptoms, past health conditions, and your family health history. He or she usually does a thorough physical exam, including tests of your muscle strength. He or she may test your endurance or ability for sustained exercise. Some diagnostic tests include:

- Blood tests to check for muscle enzymes, such as creatine kinase
- DNA blood tests for known McArdle disease mutations
- Electromyography to measure the electrical activity of the muscles
- Forearm exercise test
- MRI studies of your muscles
- Muscle biopsy to examine the muscle cells for glycogen buildup
- Urine tests to check for myoglobin, which darkens the urine

You may first see your main healthcare provider who may then refer you to a specialist, such as a neurologist. While the symptoms often appear in childhood, McArdle disease is rare. Some people with the condition do not receive the diagnosis until later in adulthood.

How is McArdle disease treated?

There is no cure for McArdle disease. But you may be able to use certain diet and exercise strategies to help control the problem. A well-designed low or moderate exercise routine may help your body get the most out of your ability to use glucose. It is very important to work with your healthcare provider to create this plan, though. Overdoing exercise can harm the muscles and kidneys in people with McArdle disease. You can work with your health team to create the best care plan for your situation. Some therapies used to help manage McArdle disease are:

- Careful attention to a diet rich in carbohydrates
- Creatine supplements
- Eating or drinking prescribed amounts of sucrose before exercise
- Prescribed, moderate aerobic exercise plan
- Vitamin B-6 supplements
- Other medicines, such as ACE inhibitors

People with McArdle disease need to work with their care team to establish a safe exercise plan. For more information and support, you may also want to talk to the team about genetic counseling.

Key points about McArdle disease

McArdle disease (GSD 5) is a rare, genetic muscle disorder. It is a type of glycogen storage disease. It results from a lack of a key substance that the muscles need to break down glycogen into glucose for energy.

- Your muscles need a constant supply of sugar (glucose) to keep working properly.
- Glycogen is a long chain of sugars that your body breaks down into glucose as needed.
- McArdle disease is a deficiency of muscle phosphorylase. This is an important substance needed to break down glycogen in your muscle cells.
- The condition causes fatigue and muscle pain during exercise.
- The disease can lead to dark urine. Severe, uncontrolled McArdle disease can cause life-threatening kidney problems.
- You can work with your care team to make a diet and exercise plan that helps you control McArdle disease and its complications.
- Your care team can advise you if sucrose before exercise may be helpful.

TRANSVERSE MYELITIS (TM) – RARE NERVOUS DISABILITY

Mustafa Indorewala, Dr. Ramdas D. Shinde
Department of Pharmacy Practice, Dr. D. Y. Patil College of Pharmacy, Akurdi, Pune

Transverse myelitis, also known as TM, is a rare ailment with no known treatment, June 9 is designated as Transverse Myelitis Awareness Day in order to increase knowledge about the condition and assist individuals who are affected by it[1].

INTRODUCTION:

An uncommon neurological disorder called transverse myelitis (TM) is brought on by inflammation of the spinal cord. From your brainstem to your low back, your spinal cord travels through the middle of your spine in a cylindrical shape. It is a fragile structure made up of cells and nerve bundles that transmit signals from your brain to the rest of your body. Your spinal cord's myelin sheath, which protects the nerve cells, gets harmed as a result of inflammation. This interferes with the communication between your spinal neurons and the rest of your body, leading to problems like loss of sensation, uncontrollable movement, and incontinence. Anywhere throughout your spine, TM can occur near the spinal cord. The term "myelitis" refers to spinal cord inflammation. "Transverse" describes the sequence of changes in sensation and function; in TM, there is frequently a band-like sensation running down the middle of your body, followed by sensory changes. The majority of the time, TM is a single-event condition that manifests suddenly and subsides or stabilises[2].

EPIDEMIOLOGY:

According to conservative estimates, there are between 1 and 8 new cases of TM per million people each year, or roughly 1,400 cases in the US each year. While affecting people of various ages, this illness is most prevalent in those between the ages of 10 and 19 and 30 and 39. Moreover, children account for about 25% of instances. There is no connection between TM and gender or family. A small minority of people with TM develop recurring sickness, especially if there is a predisposing underlying illness. TM is monophasic in 75–90% of instances.[3]

ETIOLOGY:

Doctors frequently don't know the exact cause of it. Yet, they are aware that it can occur when your body is trying to combat an illness. or when your immune system unjustifiably targets healthy cells. It frequently involves: Inflammation-causing autoimmune diseases including lupus and Sjogren's syndrome.

Infections:

Infections caused by bacteria such syphilis, TB, and Lyme disease. **Fungal infections** of the spinal cord such as aspergillus, blastomyces, coccidioides, and cryptococcus can infect the spinal cord. Parasites that cause schistosomiasis, toxoplasmosis, cysticercosis, and strongyloid. Viral infections such as enterovirus, West Nile virus, and varicella zoster, which cause shingles and chickenpox. Multiple Sclerosis (MS)- Transverse myelitis, which causes myelin to be destroyed in your brain and spinal cord, may be the initial symptom of MS. It could also be a symptom of relapse[4].

SIGN AND SYMPTOMS:

Transverse myelitis signs and symptoms often appear over the course of a few hours to a few days, though they can occasionally advance gradually over several weeks. Below the damaged region of the spinal cord, transverse myelitis often affects both sides of the body, while occasionally only one side of the body is affected.

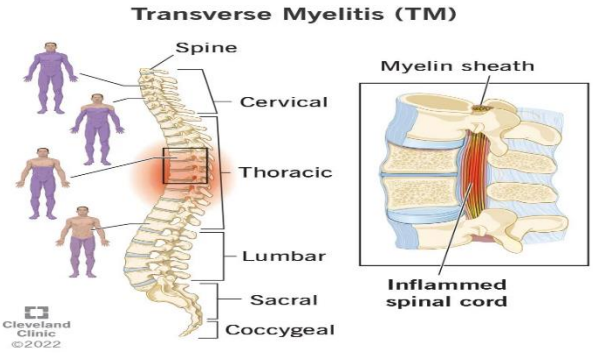
Typical warning signs and symptoms are:

Pain: Your lower back may become suddenly inflamed with transverse myelitis pain. Sharp pain may radiate around your chest or abdomen, down your legs or arms, or all over you. Depending on whether portion of your spinal cord is injured, different pain symptoms may occur.

Abnormal sensation: Transverse myelitis patients can experience numbness, tingling, coldness, or burning sensations. Some people are more sensitive to gentle contact from clothing or extremely high or low temperatures. Your chest, abdomen, or legs may feel as though they are being tightly wrapped.

Weakness in arms and legs: Some people feel their legs are heavy, or they stumble or drag one foot. Some could get severe paralysis or even total weakness.

bowel and bladder issues: Constipation, urine incontinence, increased urination frequency, and difficulty urinating are a few examples of this[5].



DIAGNOSIS:

Transverse myelitis is a neurological condition that causes inflammation of the spinal cord, leading to various symptoms like weakness or numbness in the legs or arms, back pain, and bladder or bowel dysfunction. To diagnose transverse myelitis, a doctor will typically start with a thorough medical history and physical examination. The doctor may also order various tests, including:

MRI Scan: This is one of the most important diagnostic tests used to confirm the diagnosis of transverse myelitis. An MRI scan can detect inflammation and other abnormalities in the spinal cord.[6]

Blood tests: These can help rule out other possible causes of symptoms, such as infections or autoimmune diseases.

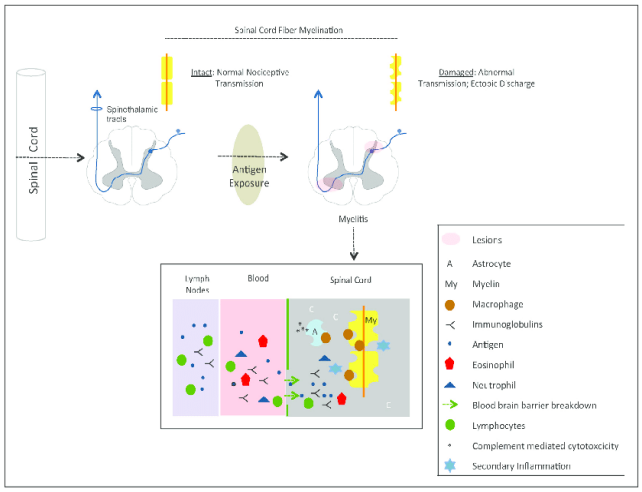
Lumbar puncture (Spinal Tap): This involves removing a sample of cerebrospinal fluid (CSF) from the lower back. CSF analysis can help detect signs of inflammation or infection in the spinal cord.

Electromyography (EMG): This test measures the electrical activity of muscles and nerves. It can help diagnose nerve damage or other neurological conditions that can cause similar symptoms.

Once a diagnosis of transverse myelitis is confirmed, the doctor may recommend treatment options, which may include medication to reduce inflammation, physical therapy to improve strength and mobility, and pain management techniques.

PATHOPHYSIOLOGY:

Schematic diagram of the pathogenesis of transverse



myelitis affecting pain pathways.

Top: macroscopic changes leading to altered spinal cord fiber myelination; in cases that affect the pain pathways (e.g., spinothalamic tracts), pain may be a major symptom in transverse myelitis.

Bottom: microscopic changes in the spinal cord. A series of events leading to demyelination are believed to start by exposure to antigens, activation of lymphocytes and immunoglobulins, followed by eosinophils and neutrophils and astrocyte pathology. Macrophages may 'attack' astrocytes and myelin, leading to local inflammation, oligodendrocyte death and myelin loss.

Exposed axons may thus have abnormal irritability (ectopic firing) and conduction patterns.[7] An inflammatory response in the spinal cord, which may activate immune cells and cause the production of different inflammatory mediators including cytokines and chemokines, is a component of the pathogenesis of transverse myelitis. These inflammatory mediators have the potential to harm the myelin sheath, which could result in demyelination and interfere with nerve transmission. The middle portion of the spinal cord, known as the thoracic area, is where injury to the spinal cord most frequently occurs, however it can happen at any level. The signs of transverse myelitis can differ depending on the location and degree of the inflammation and damage. In extreme circumstances, the injury may be irreversible and result in long-term impairment.

TREATMENT:

Intravenous steroids: Despite the lack of clinical trials that back an innovative strategy for treating TM

patients, it is widely accepted as the gold standard of care that those who are suspected of having acute myelitis receive high-dose intravenous methyl-prednisolone for 3-5 days, barring medical emergencies. The clinical course and MRI appearance after 5 days of steroid treatment are frequently taken into consideration when deciding whether to continue steroids or add a new therapy.

Plasma Exchange (PLEX): Patients with moderate to aggressive forms of TM who don't significantly improve after receiving intravenous and oral steroids may benefit from plasma exchange (PLEX). The usefulness of PLEX in treating TM has not been demonstrated in a clinical trial, although retrospective investigations of individuals with TM who had IV steroids followed by PLEX treatment demonstrated a positive outcome. Also, some patients with other autoimmune or inflammatory central nervous system illnesses have demonstrated success with PLEX. Early treatment is especially beneficial for patients, and PLEX is often started within days after commencing steroids. If treatment is initiated during the acute or subacute stages of myelitis or in patients who have active inflammation on their on MRI [8]

Additional sorts of immune-based therapy may be necessary for transverse myelitis patients who do not react to either steroids or PLEX and who still display active inflammation in the spinal cord. Immunosuppressive or immunomodulatory medications may be needed. Intravenous cyclophosphamide may be used in one of those methods (a chemotherapy drug often used for lymphomas or leukemia). Aggressive immunosuppression with cyclophosphamide may be beneficial for patients who initially appeared with aggressive forms of myelitis or who are particularly resistant to therapy with steroids and/or PLEX. The administration of this medication must involve a skilled oncology team, and patients must be closely watched because immunosuppression may result in problems. Other immune-based treatments such as anti-TNF inhibitors, B-cell modulators, and IV immunoglobulins (IVIG) have not been studied and are not recommended for the treatment of acute or subacute TM.

There may be no known aetiology for transverse myelitis, making it an idiopathic condition. Patients seldom experience a recurrence in these circumstances. Others may experience TM as a symptom of a different illness, such as neuromyelitis optica, multiple sclerosis, sarcoidosis, or lupus, to mention a few. In these circumstances, continued immunosuppressive or modulating drug therapy may be required. Aggressive rehabilitation and ongoing symptom control are essential components of the treatment strategy in either case.

PHARMACOTHERAPEUTICS:

The pharmacotherapeutics for transverse myelitis are aimed at reducing inflammation, relieving symptoms, and preventing complications. The treatment plan may vary depending on the severity and underlying cause of the condition. Here are some common pharmacological treatments used for transverse myelitis:

Corticosteroids: These medications, such as prednisone, are commonly used to reduce inflammation in the spinal cord. They work by suppressing the immune system and reducing the production of inflammatory mediators. Corticosteroids may be administered orally or intravenously, depending on the severity of the condition.

Immunosuppressants: These medications, such as azathioprine or mycophenolate mofetil, are used to suppress the immune system and reduce inflammation. They are often used in combination with corticosteroids, especially in cases where the patient does not respond well to corticosteroid treatment alone.

Intravenous Immunoglobulin (IVIG): This medication contains antibodies that can help reduce inflammation and improve immune system function. IVIG may be used as an alternative to corticosteroids or in combination with other medications.

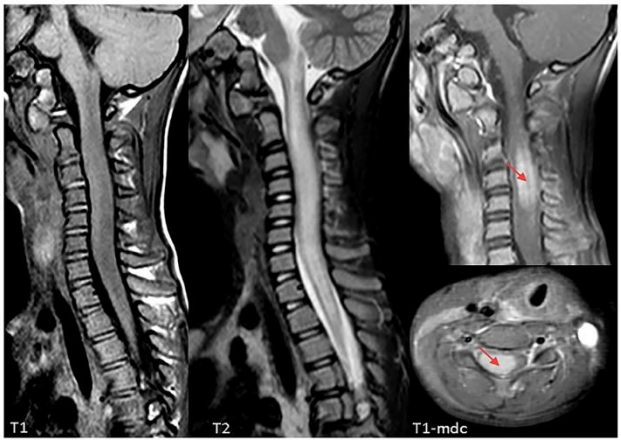
Plasma exchange (plasmapheresis): This procedure involves removing the plasma from the blood and replacing it with a donor plasma or a plasma substitute. It is used to remove harmful antibodies and other inflammatory mediators from the bloodstream.

Pain medications: These medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs) or opioids, may be used to relieve pain and discomfort associated with transverse myelitis.

It is important to note that while these medications can be helpful in managing the symptoms of transverse myelitis, they may also have potential side effects. It is crucial to work closely with a healthcare provider to monitor the effectiveness of the medications and manage any adverse effects.

CONCLUSION:

In conclusion, transverse myelitis is a neurological condition that require prompt diagnosis and treatment. Working closely with a healthcare



provider is crucial in managing the symptoms of the condition and preventing complications

Diagnosis of transverse myelitis typically involves a through medical history, physical examinations and various tests such as MRI scan, blood tests, lumbar puncture and electromyography. Treatment for transverse myelitis is focused on reducing inflammation reliving symptoms and preventing complications. The pharmacological treatment includes corticosteroids, immune suppressants, IVIG, plasma exchange and pain medication.

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WORLD SPINE HEALTH DAY

Tushar Gaud, Yashodeep Gore, Maharshi Ahirrao, Ms. Aishwarya Sambhaji Patil
Department of Pharmacology, Dr. D. Y. Patil College of Pharmacy

Spinal Cord

1. Introduction

The spinal chord is a part of the control the system it is a long pipe like structure from the medulla oblongata part of the brain consisting of a collection of nerve fibers , running through the vertebral column of the backbone it is segmented with pair of roots (dorsal and natural roots) consisting of nerve fibres joining to form the spina nerves.

2. Structure:

The spinal cord runs through a hollow case from the skull enclosed within the vertebral column. spinal nerve arises from different regions of vertebral column and are named accordingly, the regions are neck, chest, pelvic and abdominal. Cross function of spinal cord displays grey matter shaped like a butterfly surrounded by a white matter. Grey matter consist of the central canal at the central and its filled with a fluid called CSF (cerebrospinal fluid) it consist of horns (four projections) and forms the core mainly containing neurons and cells of the CNS. These are two dorsal and two ventral horns. A white matter consists of a collection of neurons permitting communication between different layers of CNS. a tract is collection of neurons and carried a specialized information. Spinal nerves act as mediators, communicating information to and from the rest of the body and the spinal cord we have pains of spinal nerves. Three layers of meninge surround the spinal cord and spinal nerves roots Arachnoid matter, Pia meter Durra maters. Durra matter consist of 2 layers periosteal and meningeal, epidural space is present between the two layers subarachnoid space like between the arachnoid mate and pia mater . it is filled with cerebrospinal fluid. Spinal cord nerves

The spinal cord can be grouped as

1. Cervical
2. Thoracic
3. Sacral
4. Lumbar
5. Coccygeal

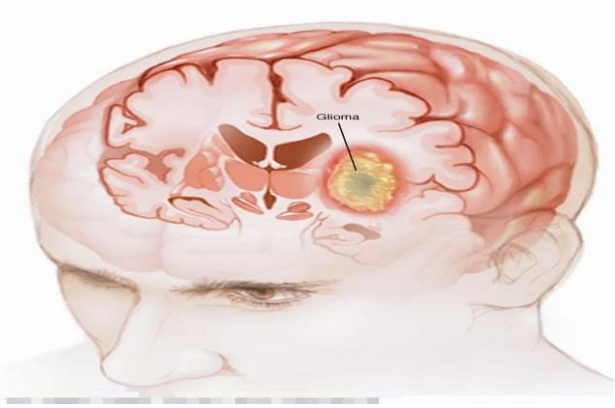
Cervical nerves	Cervical means the neck these are 8 cervical nerves they emerge from the cervical spine (c1-c8)
Thoracic nerves	Thoracic means of the chest there are 12 thoracic nerves that emerge from the thoracic (t1-t12) .
Sacral nerve	Sacral means of the sacrum the sacrum is a bony plate as the base of the vertebral column. There are 5 sacral nerves that emerge from the sacral bone (s1-s5)
Lumbar nerves	Lumbar means from lower back region there are 5 lumbar nerves that emerge from the lumbar spine (l1-l5)
Coccygeal	Coccygeal means of the tailbone these is one nerve that emerges from the coccygeal bone

3.Functions:

Important functions of spinal cord are mentioned below:

1. Forms a connecting link between the brain and the PNS. (peripheral nervous system)
2. Provides structural supper and builds a body posture.

3. Myelin present in the white mater acts as an electrical insulation.



4. Communicate massage from the brain to different parts of body.
5. Coordinates the body.
6. Receives sensory in formation from receptors appear towards the brain for processing.

Spinach health

A healthy spine supports the body while getting it more freely. It does this with the help of 3 natural causes. Strong, flexible muscles help too. They support the spine by keeping its causes properly aligned. The disks that acts as cushion to bones of spine also play a role in back.

4.Importance:

1. Spinal health its crucial because the spine controls the body's vital function it is the spine which provides the structural support.
2. To our body and helps us to maintain the upright posture, it protects spinal chord and new roots assists us to more and perform our everyday tasks.

Glioma

1.Introduction:

Glioma is growth of cells that starts in the brain (or) spinal chord.

The cells in a glioma are called as glial cells which look similar to healthy brain cells. Glial cells surrounds nerve cells and help them function.

This is the most common primary brain tumor.

Glioma is the disease which can affect all ages but this disease is mostly after seen adult.

There are many types of glioma. Some grow slowly and aren't considered to be cancers. Other are considered as cancerous

Malignant gliomas can invade healthy brain tissue and grows quickly.

2. Epidemiology:

In the United States there is an estimation of 80,000 newly diagnosed cases as primary brain tumor in each year. In the above cases gliomas are around one-fourth (i.e., 20,000) cases are present in each year there is around 12000 cases of glioblastomas are diagnosed (this is approximately 15% of the total newly diagnosed brain tumor).

3.Types of glioma:

Astrocytomas	A type of tumor that develop from a specific type of cell called astrocytes, which are a part of the connective tissue in the brain. These tumors are the most common type of primary brain tumor and account for almost half of all cases.
Brain stem gliomas	Also known as DI PGs or diffuse infiltrating brainstem

	gliomas, are uncommon tumors that occur in the brain stem.
Ependymomas	Tumors that arise from ependymal cells found in the lining of the ventricles or in the spinal cord Although they are rare and account for only 2-3% of primary brain tumors, they are more prevalent in children, making up around 8-10% of brain tumors in this age group.
Mixed gliomas	also known as oligo-astrocytomas, are tumors that consist of multiple types of glial cells. There is debate regarding whether they should be classified as a unique type of tumor, and this issue may be clarified by conducting genetic tests on the tumor tissue.
Oligodendrogliomas	Type of brain tumor that originates from the cells in the brain called oligodendrocytes which provide support to the nerve cells These
	tumors are typically located in the cerebrum, and they account for around 2 to 4 percent.
Optic pathway gliomas	Are a specific type of tumor that is typically low-grade and found in either the optic nerve or chiasm. These tumors tend to infiltrate the optic nerves, which are responsible for transmitting visual information from the eyes to the brain.

4.Pathophysiology:

Gliomas are a type of brain tumor that commonly present with headaches as the initial symptom. The cause of theseheadaches is believed to be due to the tumor's growth, which puts pressure on surrounding tissues, leading to inflammation, and swelling. The location of the tumor in the brain determines the specific symptoms a patient may experience, such as behavioral changes,speech problems, nausea, vomiting and changes in vision. Seizures are the second most common symptom and are thought to be caused by the tumor irritating the cerebral cortex, resulting in focal or generalized seizures. Other symptoms of gliomas may include tingling sensations, weakness, difficultywalking, and, in rare cases, a comatose state due to hemorrhage within the tumor.

5.Sign and symptoms:

Glioma symptoms depend on the location of the glioma. Symptoms also may depend on the type of glioma:

Common signs and symptoms of gliomas include:

1. Headache, particularly one that hurts the most in the morning.
2. Nausea and vomiting.
3. Confusion or a decline in brain function, such as problems with thinking and understanding information.
4. Memory loss.
5. Personality changes or irritability.
6. Vision problems, such as blurred vision, double vision or
7. Loss of peripheral vision.
8. Speech difficulties.
9. Seizures, especially in someone who hasn't had seizures before.

6.Risk Factors:

1. Age Factors:

2. Hormonal status, exogenous hormones and risk for developing gliomas:

3. Exposure to ionizing radiations.

7.Treatment:

1. Surgery:

Grade I: Gliomas at this stage can be cured through surgery

Grade 11: A complete removal of the tumor through surgery and regular. imaging tests are currently acceptable treatments.

Grade II: A complete removal of the tumor through surgery, along with chemoradiation therapy and regular imaging tests to monitor for recurrence are acceptable treatments

Grade IV (glioblastoma): A complete removal of the tumor through surgery along with chemoradiation therapy and regular imaging tests to monitor for recurrence, are acceptable treatments.

2. Chemoradiation: The Stupp protocol is currently the standard of care for Grade II-11- V gliomas. This protocol involves radiotherapy and chemoradiation therapy using temozolomide, with a total of 60 Gray to 2 Gray given per daily fraction over 6 weeks.

3. Treatments for Recurrence: Options for treating recurrent gliomas include re-operation with Gliadel wafers, as well as targeted therapies like angiogenesis inhibitors or immunotherapy. The effectiveness of these additional treatments is still being studied.

4. Other Treatments: Patients with high-grade gliomas are at risk of experiencing seizures, malignant edema, and complications related to immobility. As such, these patients require antiepileptic medications, deep venous thrombosis (DVT) prophylaxis, and steroids before, during, and after treatment to prevent cerebral edema.

9.Preventive measures:

There are two possible ways to prevent gliomas: stop them from forming in the first place or prevent them from progressing to higher-grade tumors. Both methods would reduce the number of deaths caused by gliomas over time. However, we need to understand the causes of gliomas before we can effectively prevent them. In this talk, I will discuss primary prevention, chemoprevention, and screening First, I will explain the chromosomal, genetic, and protein changes that are associated with different types of gliomas and the factors that can promote their development and progression. These include environmental factors, heredity, and infections. I will discuss how changes in genetic patterns can lead to different types of tumors and how identifying these patterns can help us diagnose and treat gliomas more effectively. The idea behind prevention is to stop specific gene mutations or changes in chromosome numbers that cause low-grade gliomas (WHO 2) from developing into higher-grade tumors WHO 3 and 4). By preventing these changes, we can reduce the occurrence of high-grade gliomas and improve patient survival rates. Alternatively, if we can cure low-grade gliomas and prevent these

changes from happening, we can also prevent some glioblastomas (WHO 4) from forming and improve survival rates.

However, to achieve these goals, we must be able to diagnose and treat gliomas at an earlier stage. This requires better screening methods and more effective treatments for low-grade gliomas.

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STONEMAN SYNDROME: A RARE DISEASE

Mrs Amruta Sapate
Department of Pharmaceutics, Dr. D. Y. Patil College of Pharmacy

Abstract:

Stoneman syndrome or fibrodysplasia Ossificans Progressiva (FOP) is an extremely rare (1in 2 million) genetic disorder characterised by ectopic ossification of the skeletal and connective tissues leading to progressive fusion of axial and appendicular skeleton. They may include bilateral hallux valgus, monophalangic great toes with short and stout first metatarsals, heterotopic ossification if muscle and connective tissues, short broad femoral necks, pseudo exostoses, short and stout first metacarpals, C2-C7 facet joint fusion, large posterior elements, and tall narrow vertebral bodies. The mutation affects the body's repair mechanism, causing fibrous tissue including muscle, tendons and ligaments to become ossified, either spontaneously or when damaged as the result of trauma. In many cases, otherwise minor injuries can cause joints to become permanently fused as new bone forms, replacing the damaged muscle tissue. This new bone formation known as "heterotopic ossification" eventually form a secondary skeleton and progressively restrict the patient's ability to move. Bone formed as a result of this process is identical to normal bone, simply in improper location.

Keywords: Fibrodysplasia Ossificans Progressiva; Hallux Valgus; Autosomal Dominant; stone man syndrome; Munchmeyer disease.

1. Introduction:

FOP is also called Munchmeyer disease or Myositis Ossificans Progressiva. It is an extremely rare connective tissue disease in which fibrous connective tissue such as muscle, tendons and ligaments turn into bone tissue. It is the only known medical condition where one organ system changes into another. It is a severe, disabling disorder with no current cure or treatment. FOP is caused by a mutation of the gene ACVR1 (active in receptor type 1A).

Medical reports describing individuals affected by FOP date back to Dr. Guy Patin in 1692. FOP was originally called myositis ossificans Progressiva and was thought to be caused by muscular inflammation (myositis) that caused bone formation. The disease was renamed by Victor A. McKusick in 1970 following the discovery that soft tissue other than muscles (e.g. ligaments) were also by the disease process.

The best-known FOP case is that of Harry Eastlack (1933-1973). His condition began to develop at the age of ten and by the time of his death from pneumonia in November 1973, six days before his 40th birthday, his body had completely ossified, leaving him to move only his lips. Eastlack never met another person with FOP during his lifetime. Eastlack donated his body to science and his skeleton is now at the Mutter Museum in Philadelphia. Genetic analysis revealed that the FOP gene located on chromosome 4 and mutation in this gene causes an over expression of a one morphogenetic protein (BMP4)

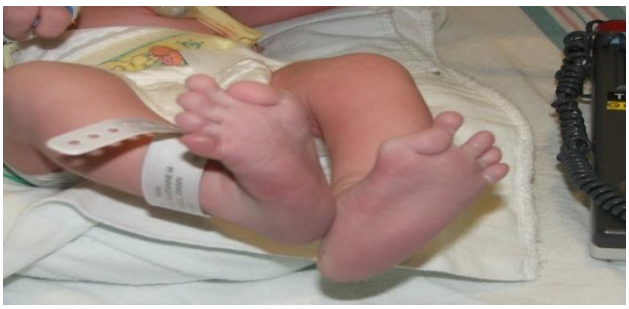
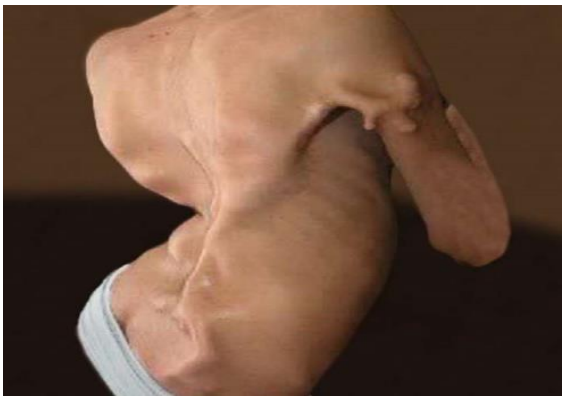


Fig: 1.1 FOP (Fibrodysplasia Ossificans Progressiva)



2. Causes of FOP:

A mutation of the ACVR1 gene causes fibrodysplasia Ossificans Progressiva. The ACVR1 gene gives your body instruction to make type 1 receptors for a protein called bone morphogenic protein (BMP) that reside in your muscles and cartilage. BMP controls how bones and muscles grow and develop. The mutation of ACVR1 causes symptoms of Fibrodysplasia Ossificans Progressiva because the receptor, like a light switch, is always on when it should turn off. Fibrodysplasia Ossificans Progressiva is an autosomal dominant condition, which means only one biological parent needs to pass the altered gene to the child for them to inherit it. If a parent has the gene that causes fibrodysplasia ossificans Progressiva, there is 50% chance that the child will inherit the conditions. Most cases of fibrodysplasia ossificans progressive occurs because of a new mutation of the ACVR1 gene (de novo). This happens randomly and there is no link to the gene being present in a person's family history.

3. Symptoms of FOP:

1. Gradual replacement of muscles, tendons and ligaments into bone (heterotopic ossification).

2. FOP starts at neck and shoulders in early childhood and progresses throughout the body over time.

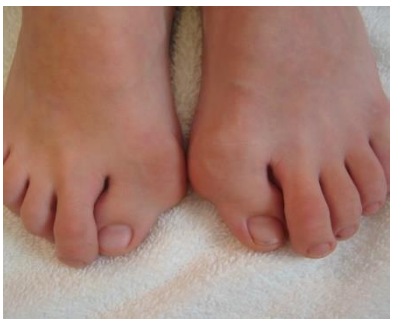
3. Swelling or an increase in size or shape of a body part of the body can be painful.

4. Symptoms arises during flare-ups, which are your body's reaction to trauma that could be from an injury, surgery or viral illness like flue.

5. When flare-ups occur, the bone morphogenic protein type1 receptors fails to stop producing protein, which cause new bone to form on muscles, tendons and ligaments.

6. In FOP malformed and short big toe that sometimes grows inward and over the second toe.

7. Decreased mobility (scooting instead of crawling, joint stiffness, locked joints.)



8. Difficulty in eating and speaking.

9. Hearing impairment.

10. Red to purple, painful and hot to the touch areas of the body that look like tumours.

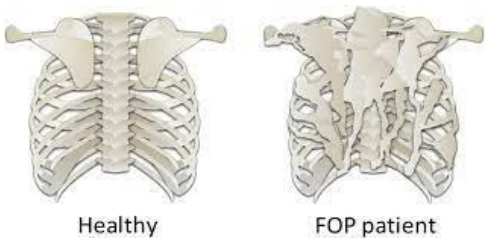


Fig: 3.1 Difference between healthy bone and bone with FOP

4. Diagnosis of FOP:

Diagnosis of a rare genetic disease can be a challenging task. At initial stages, cases of FOP can be confused with other aggressive muscular illness like juvenile fibromatosis, cancer and fibrous dysplasia. Diagnosis of FOP depends on medical history and clinical examination, only the symptoms

that makes it different from other condition is Malformation of the big toe.

- Spontaneous flare-ups of inflammation or soft tissue swelling.
- Increased flare-ups after injury, viral illness or immunizations.
- Difficulty moving.
- Frequent injury due to falling.

In some cases, excess bone formation may be seen by a series of physical examinations and laboratory test such as X-ray and MRI and genetic testing registry (GTR). Imaging tests will help the doctor to see the overall growth of the heterotopic bone whereas GTR will direct the doctor to choose appropriate genetic tests for this condition. GTR is conducted by a group of medical professionals that majorly deals with research and analysis of such rare conditions and it includes a series of questions for both patient and the medical team.

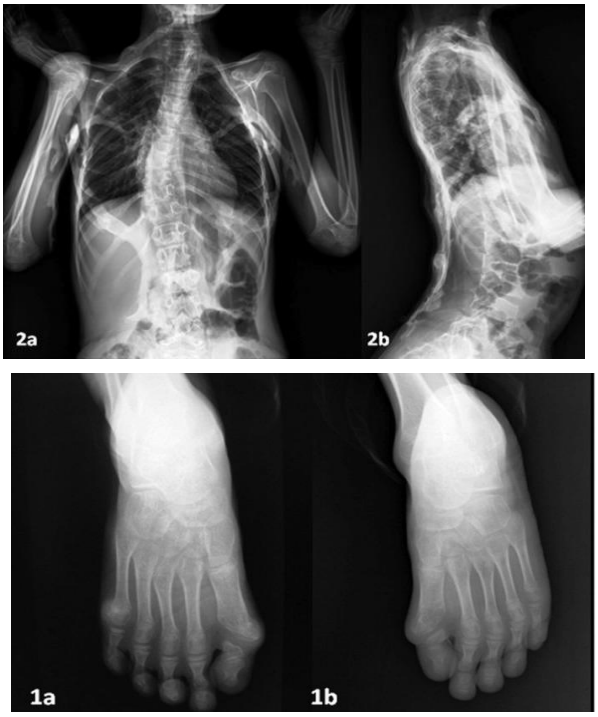


Fig: 4.1 X-Ray photocopy of change in Bone and Flair-ups toes

5. Management including treatment:

There is no Definitive treatment for FOP. Supportive medical management was provided.

2. The patient was advised to rest along with avoidance of exercise and traumatic events.

3. Some of supportive medication like Non-steroidal Anti-inflammatory agents, Bisphosphonate and glucocorticoids for symptomatic relief.

4. Sometimes COX2 inhibitors and leukotrienes inhibitors are other options.

5. Also patient should be advised to avoid precipitating factors.

6. Corticosteroids in the form of 500mg injections, methylprednisolone for 5 days was given to some patients for trials in case studies.

7. Surgical attempts to remove heterotopic bone will provoke explosive new bone growth and should not be attempted.

6. Prognosis:

The median lifespan is approximately 40 years of age. Most patients are wheelchair bound by the end of second decades of life and commonly die of complications of thoracic insufficiency syndrome.

Conclusion:

Fibro dysplasia ossificans progressiva is a rare disease, Fop should be reported in any child with an unexplained inflammatory mass, associated with heterotopic ossification of the soft parts and congenital bone abnormalities. His diagnosis is radio-clinical. The development is marked by the appearance of particularly disabling joint stiffness. No effective preventive or curative treatment is available to date.

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Dr. D. Y. Patil College of Pharmacy, D. Y. Patil Educational Complex, Sector 29, Nigidi Pradhikaran, Akurdi, Pune 411044.