WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 10, Issue 12, 428-443. Review Article ISSN 2277– 7105

MECHANISM AND CLASSIFICATION OF ANTIDEPRESSANT DRUG: A REVIEW

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Article Received on
26 July 2021,

Revised on 16 August 2021,
Accepted on 06 Sept. 2021

DOI: 10.20959/wjpr202112-21690

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ABSTRACT

Antidepressant drug those which can decrease symptoms in depression. Even though they don't cure the illness but can rest the symptoms. Then the uses of drugs without correctly prescription form psychiatrists overdose may reason of side effect, even is some instance death may be happen so we can use these drug inside the limit. New generation of antidepressant drug (ADs) are generally used as the first of treat of important depressive illness and the considered tolerate than tricyclic agent. This review are want to supply into the pharmacology, mechanism, material and method. The Depression being a condition of the sadness may be specify as a psychoneurotic disorder distinguish by a mental and functional activity, unhappiness, hopelessness and generation of suicidal tendencies. It is a ordinary disorder create significant morbidity and mortality world wide. Some developmental researches have a argued present diagnostic criteria for major depressive disorder (MDD) may are not exactly distinguish true instance of disorder form a usual adaptive stress response. When the pharmacological perturbation that stop the reverse forces reasons of a monoamine levels to a overshoot their equilibrium levels. Since depressive symptoms are the less than monoaminergic control, this is the overshoot have reasons a resurgence of depressive symptoms.

KEYWORDS: Antidepressants, Desmethylvenlafaxin, Antimuscarinic, Antihistaminic, Veniataxine.

INTRODUCTION

Depression is the successful state of the unfavorable mood and a low arousal. Millions human being are afflicted by the depression each year. About 6.6% of adult or about the 13 million of a adult in US people, are evaluate to have a episode that a meet present day. There are the two panoptic divergent approaches to the connection in between stress or depression. In several way connection must the mediated by brain. But a neurological due to of depression are generally to accept to the unknown. Several researches convinced that depressive symptoms. Many articulation of a monoamine disorder of a hypothesis propose that the forebrain surface of monoamine neurotransmitter are exhaust in the depressive disorder in depressive disorder. Specially serotonin (5-HT), norepinephrine (NE) and to minor extent, dopamine (DA) even so, several have advise that monoaminergic transmission possibly enhanced in depression. The precise way of association mediate of depression and a forebrain monoamine surface is not the vital present paper. It is a tolerable to say that a neurochemical disorder hypothesis proposes that a monoamines especially (5- HT) and NA, are perturbed is a several direction. The antidepressant is the first line of treat of the unipolar important depression and the number of more psychiatric disorder, equally as obsessive compulsive disorder. In 2011 the antidepressant is most distribute drug in the united states. The antidepressant treat can be used for some indication antidepressant, anxiolytics. Which have always involve education and counselling. Earlies studies intimate that the patient which advanced cancer and antidepressant have be reticent for important depressive disorder cases since patient who don't have a important depressive disorder. Serotonin is usually delivered by activated platelets begin increase platelet aggregation. When drug are estimate to determine their applicability to demonstrate based on clinical practice. It is major to asses their efficiency in aimless. Depression is a mental and functional activity, unhappiness decrease in activity. In the female the depression emanate also due to over extra working load and family responsibilities children care strained connection and care to the old parent. In adding these all indices the psychological biological hormones part, serious contribute in depression.[3,4,5,6,7,8]

Fig. 1: Antidepressant drug.

Pharmacology[14]

1) Depression

Antidepressants increase transmission of one or more monoamines. Some have additional actions they may contribute towards their advantageous and/or disagreeable effect. Although increased monoamine transmission occurs within hours and the antidepressant effect is lower to appear because this require normalization of receptor sensitivity and neuroplasticity. The neuroplasticity is the ability of the CNS to adapt structurally and functionally in response to external stimuli and is mediated by the nerve growth factor. In depression, neuroplasticity impaired in the limbic and prefrontal cortex circuits that regulate mood, attention, energy, appetitie and sleep. Depression refractory to one of antidepressants that can respond the following switch to another antidepressant and the use of combination treatment that targets different, or multiple monoamine.

2) Anxiety and Panic Disorder

Antidepressant and benzodiazepines that inhibit the amygdalas so called fear circuits through the 5HTA and GABAA Receptor respectively. The threat sensor which integrates sensory information with the contextual information. The fear response is required for the amygdalas effect or pathway to activates the relevant circuis.

3) Pain

The analgesic effect of antidepressant are also enhanced due to monoamic transmission example-in the descending pain modulation pathway. The pain modulation pathway that can induce both analgesia and hyperalgesia.

Antidepressant drug and their classification[1,2,3]

Antidepressants are those days which help in the reduction in symptoms of depressive transmission example-in the descending pain modulation pathway. pain modulation pathway that can induce both analgesia and hyperalgesia. disorders and the altering chemical imbalance of neurotransmitters in the brain. The change in mood and the behaviour is due to the chemical imbalance. The Communication link between neurons in the brain are the Neurotransmitteas. Neurotransmitteas are vesiclet found in the nerve cells. The reuptake means the neurotransmitter such as serotonin, noradrenaline and dopamine or norepinephrine are released by the exonic end of one nerve and that received the nerve. The antidepressant inhibit the reuptake of neurotransmitters through the selective receptor. These receptors increase the concentration of specific neurotransmitters around the nerves in the brain. The selective Serotonin reuptake inhibitor (SSRI)that affects the brain serotonin level. The antidepressants are recover the signs of depression and also exert same side effects. The antidepressants are used in the treatment of the Symptoms including not only depression, nervous ness, OCD, some disorder, between in childhood, manic-depressive disorders, diabetic peripheral neuropathic pain, Social fretfulness.

The antidepressants are divided into following group

1) Tricyclic antidepressants (TCAs).
2) Selective serotonin-reuptake inhibitors (SSRIs).
3) Monoamine oxidase inhibitor (MAOIs).
4) Serotonin-norepinephrine reuptake inhibitor (SNRI).
5) Atypical antidepressants.

1) Tricyclic Antidepressants (TCAs)

The SARS for the TCAs are compiled in details in the edition of the that text. The interested reader is referred to the compilation, The features of that can be visualized by the consulting in the structures of imipramine and desipramine. The overall arrangement of that features that fully extended trends conformation of a ß arylaminel The TCAs are the structurally related to the each other and Consequently, Possess related biological properties that can be summarized as the characteristic of this group. The Compounds have anti cholinergic property that are usually higher in the dimethyl amino compounds. The TCA are extremely lipophilic and accordingly very highly bounded outside the CNS. Where as they have the anticholinergic and noradrenergic effects, the both central and peripheral side effect are often

unpleasant and Sometimes that are dangerous. The overdose is complicated because the agents are highly protein bound that dialysis is ineffective.

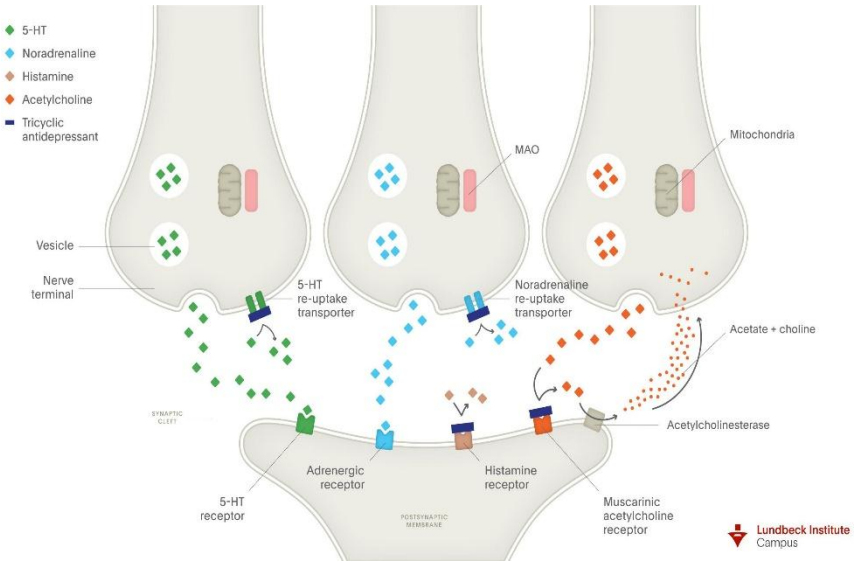

Fig. 2: Tricyclic Antidepressants.

2) Selective serotonin-reuptake inhibitors (SSRIs)

Starts are acknowledge as a selectively inhibit serotonin transporter, fluoxetine (prozac, selfemra), paroxetine (paxit, pexeva), setraline (Zoloft), citalopram (celexa) and escitalopram (Lexapro) these are the several examples of SSRIs, the working of SSRIs causes sudden increase in serotonin in the somatodendritic area of serotonergic neurons which are located in the raphe nucleus which leads to desensitization of somatodendritic serotonin- 1A auto receptors, due to this the flow of neural impulse increases, as a result the level of serotonin increases which is release by axon terminals, as a result it increased the serotonin which is librated from the axon terminals which is leads up to desensitization of the postsynaptic serotonin receptors, desensitization of these receptors may come up with the therapeutic actions of SSRIs, might possibly consider for the enlargement of forebearance to intense reaction of SSRIs, these agents can cause powerful at the same time gental disinhibition of 5-HT neurotransmission in the CNS and it is suggested by pharmacological analysis of SSRIs, the effect of antidepressants are transmitted which is coming from or originated from midbrain raphe to prefrontal cortex, SSRIs shows some side effects like anxiety, sleep disturbance, sexual dysfunction and gastrointestinal disturbance, it is considered as a lethal the 5- HT_2 and 5- HT_3 receptors definitely serotonergic pathway are responsible, serotonin and dopamine shows a correlation between them namely serotonin helps to slow down the sexual functioning as well as dopamine helps to build up sexual functioning, sexuale

dysfunctioning can be seen due to the negative effects on sexual functions which are arbitrate through 5 - HT_2 receptors, hence $5HT_2$ antagonists can alter SSRIs to produce sexual dysfunction. To better recognize for what reason sufferer may cease antidepresaant treatment. In real fact of each person conducted a CATI at three and six month behind them patient started SSRI for modern effect of antidepresaant during maintenance and treatment, the aim was to report reasons for cessation or changing the antidepressant initially in treatment.

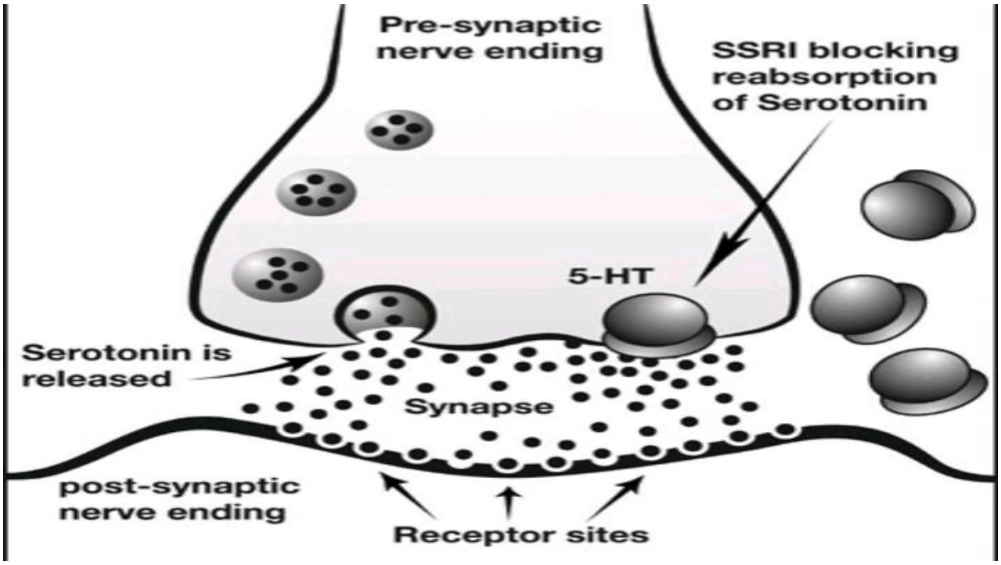

Fig. 3: Selective serotonin-reuptake inhibitors (SSRIs).

3) Monoamine oxide inhibitors (MAOIs)

Antidepressant therapy usually implies therapy of the unipolar type and is centered on the chemical agents that are the MAOIs, the monoamine reuptake inhibitor and auto receptor desensitizes and antagonist. The electroshock therapy is one more option. The highest curve rate is achieved with electroshock therapy. The 60-70% response rate of MAOIS and monoamine reuptake inhibitor. The history of MAOI development illustrates is the role of serendipity. The presence of clinically useful irreversible inactivates can be considered mechanism. based inhibitors of MAO. They are converted by MAO to agents that are inhibit the enzyme and that enzyme formed from reactants that bond covalently. A reversible inhibitor of MAO-A that is considered on effective antidepressant and Permits metabolism of dietary tyramine. Metabolism of the drugs are implicated in the activity. The clinically MAOI antidepressants are nonselective between inhibiting metabolism of NE and 5-HT.

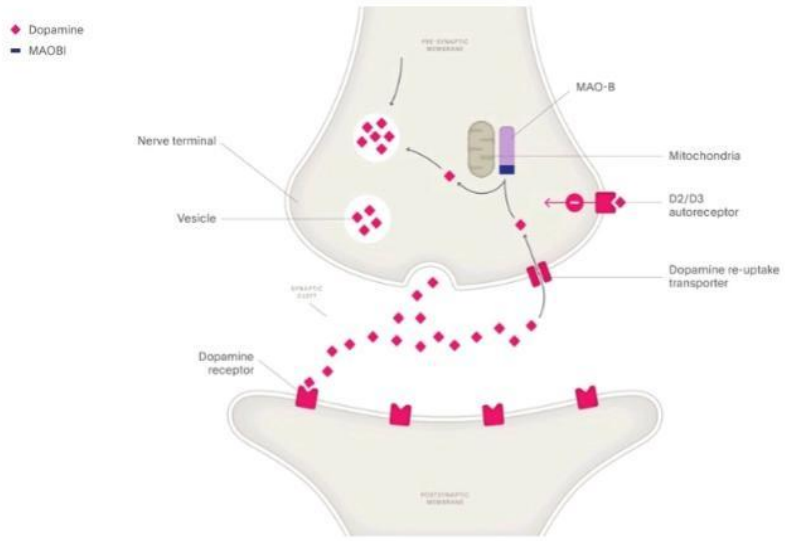

Fig. 4: Monoamine oxide inhibitors (MAOIs).

4) Serotonin and noradrenaline reuptake inhibitor (SNRIs)

1. Veniataxine

The novel antidepressants mentioned to as SNRI. Thus it discourage uptake the two NA and 5-HT yet, in conversely to elderly TCAs, doesn't collaborate with a cholinergic, adrenergic or histaminergic receptor either posses sedative property. These trials posses displayed it be as a successful antidepressant as a TCAs and a numerous work is a several. A speedy short of an action is claimed.

2. Desvenlafaxine

It is a Desmethylvenlafaxin, an energetic metabolite of a venlafaxine with same action, uses and a side effect.

3. Duloxetine

A new brand of SNRI same to the venlafaxine. It is a lightly sedating with several antimuscarinic effect. Yet not an antihistaminic, side effect, counting g.i and a sexual problem are a clement, yet agitation insomnia and jump up BP can happen in few. Antidepressant efficacy is a similar to TCAs. Duloxetine is a specially point in a diabetic and more of a neuropathic pain, fibromyalgia and strain of urinary incontinence in female. It is used of a conservation therapy on panic disorder.

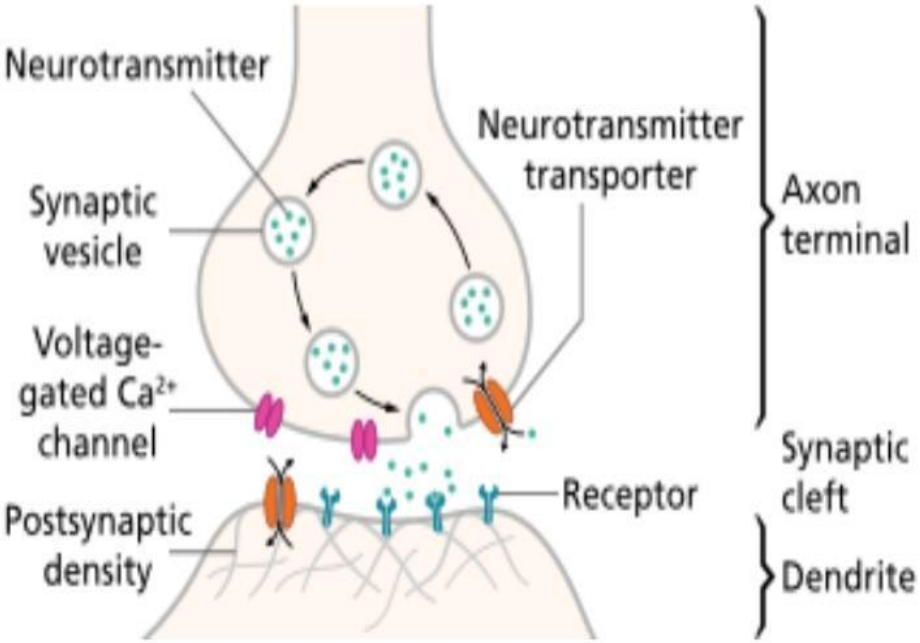

Fig. 5: Serotonin and noradrenaline reuptake inhibitor (SNRIs).

5) Atypical antidepressant

1. Trazodone

It is a earliest atypical antidepressant, small expertly blocks 5-HT insult, however has important Alpha adrenergic and feeble 5-HT antagonistic actions. But is metabolite is a powerful 5-HT_2 blocker. Antidepressant effect is the moderate. It is a sedative but is not a antichonergic, reason bradycardia sooner aside form tachycardia doesn't alliance with a intracardiac conduction. According smaller prone to a reason of arrhythmia.

2. Mianserin

It is individual isn't inhibiting the other NA or 5-HT comeback, yet blocks presynaptic alpha receptor through greater release and a turnover of NA of brain that may exist amenable for the antidepressant effect. Antagonistic step at 5-HT_2, 5-HT_1 also H_1 receptor have as well as been shown. It is a sedative-relieves related anxiety and a defeat panic attack.

3. Mirtazapine

This an antidepressant represent by new drug mechanism viz. auto- (on NA neurons), hetero (on 5-HT neurons) receptor increase the pair both NA and 5-HT discharge. The increase NA forward expanding firing of a serotonergic raphe neurons via. Alpha 1 receptor. Careful ameliorate of antidepressant 5-HT receptor steps is reach simultaneous blocked of 5-HT_2 and 5- HT_3 receptor.

4. Bupropian

This constraint of DA and NA comeback has desire rather apart form sedative property. It is metabolized keep amphetamine like compound that can source of presynaptic release of DA and NA. A sustain free formulation in the market as an aid the smoke cessation. In the clinical trials it ancient establish to yield equal to smoking abstinence.

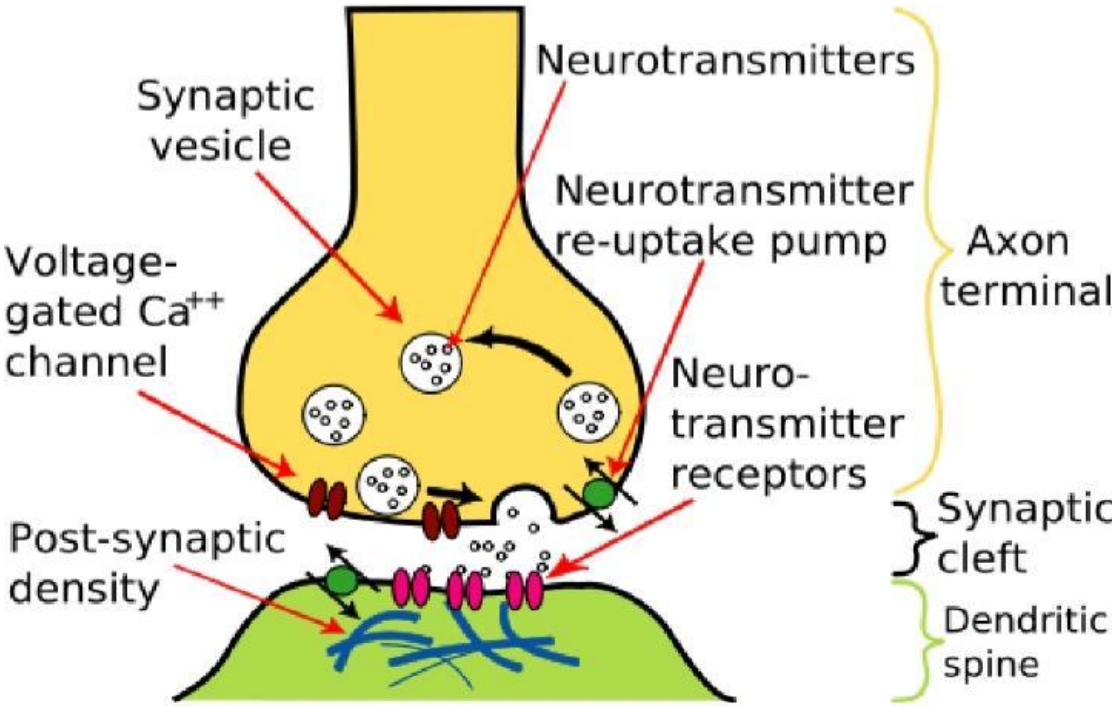

Fig. 6: Atypical antidepressant.

Mechanism

Neurotransmitters are the endogenous chemicals which transmits signal from one neuron to another target neuron across the synapse. Serotonin, dopamine and melatonin are important chemicals in the brain. When the nerves are robbed of those neurotransmitter, they can not send message to different nerve hence, depression is occur. The message passed through the neuron are exhibited as behaviour, emotion, apeptide, pleasure, temperature, sleep, thoughts etc. The mechanism of action of SSRIs (selective serotonin reuptake inhibitor) and SNRIs (serotonin norepinephrine reuptake inhibitor) are different. SSRI has different serotonin reuptake inhibitor like fluoxetine, paroxetine and sertraline. Those inhibitors have selective effect on citalopram and fluvoxamine on the serotonin reuptake pump (5-HTT). While 5-HTT and the NET (norepinephrine transporter) are blocked by SNRIs. By blocking these transporter prevents the neuron from vaccuming up excess neurotransmitter and it stimulate the postsynaptic receptor by staying for lot of time in synapse. Monoamines are classify as

serotonin, monoamine neurotransmitter and dopamine. MAOIs increase neurotransmitter level by inhibiting MAO. Cyclic antidepressants usually block the serotonin and norepinephrine neurotransmitter which are present in brain. These chemicals used to treat the depression. SRMs (serotonin receptor modulators) is used to treat irritable intestine syndrome. Serotonin is important for initiation of peristaltic and humor reflex and in modulation of visceral sensation. Lithium is used to treat manic depressive patient.[9]

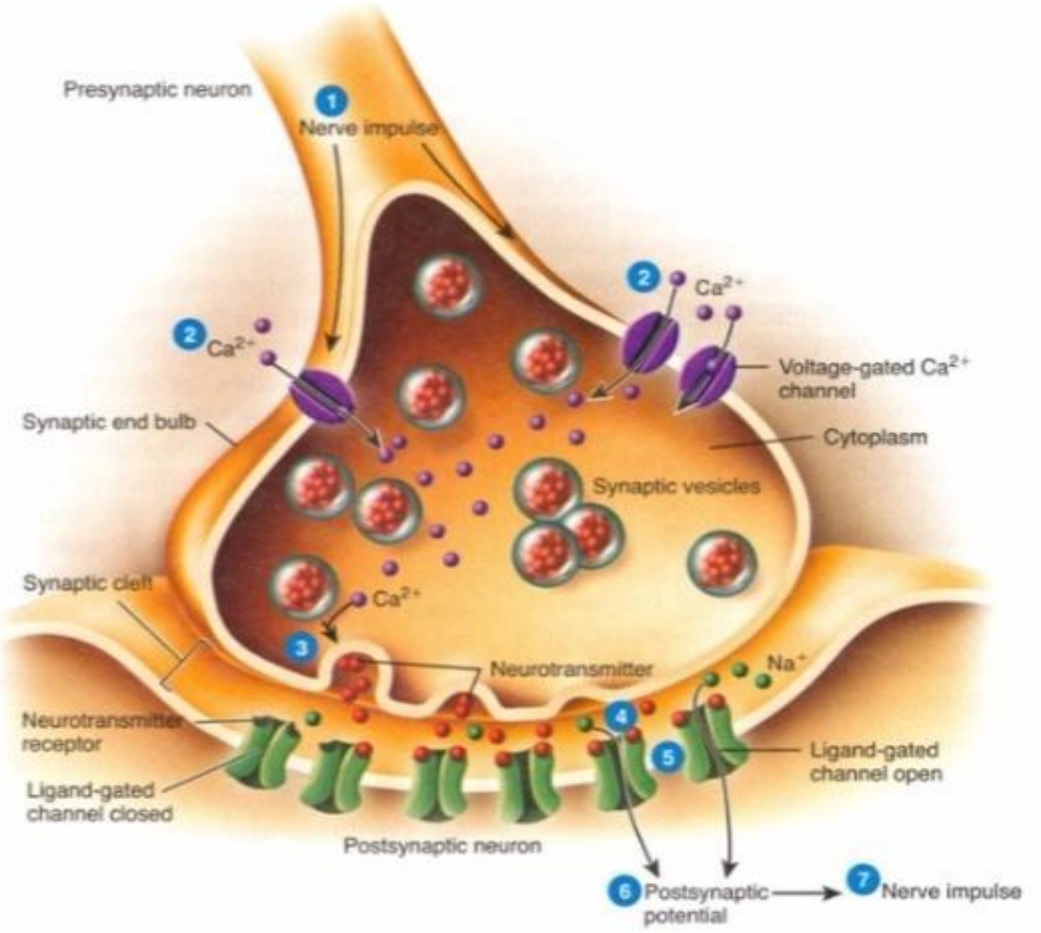

Fig. 7: Mechanism of action of antidepressant.

Method and material

Antidepressants drugs are defined as per international anatomical therapeutic chemical classification system (code: N06 A) by the WHO. The antipsychotic drugs (code:N05A) lithium used as a mood stabilizer for bipolar disorder and quetiapine, a atypical antipsychotics were used to treat symptoms in depressed patients. In recent study, antidepressants drug were classified as selective serotonin reuptake inhibitor (SSRI), norepinephrine reuptake inhibitor, tricyclic, tetracyclic, monoamine oxidase inhibitors. The

retrospective study of prescription pattern and drugs use was carried out using prescription formed from the out-patient clinic of the federal Neuro-psychiatric hospital, Nigeria. Those patients having antidepressants medication in their prescription regardless of clinical treatment format (psychotherapeutic agents) were included in the study without repetition of the patients prescription. The prescription were analyzed by using WHO core drug indicator and comparative cost of drugs.[10,11]

Main adverse events related to use of antidepressants[12]

1. Gastrointestinal (vomiting, nausea, GI bleeding)

serotonin act as a important regulatory role in the motor and sensory effects of the gastrointestinal (GI) tract. Serotonin agent that effects on central "setrons" that means 5-HT3 antagonists receptor may caused due to vomiting & nausea. Most of the side effects connected with the utilize of selective serotonin reuptake inhibitors (SSRIs) and(SNRIS) means serotonin noradrenaline reuptake inhibitors. Greater levels of serotonin in G.I tract the major main adverse effects of selective serotonin reuptake inhibitors treatment are gastrointestinal. Escitalopram was small effects similarly to causes G.I side effects.

2. Hepatotoxicity and Hypersensitivity reaction

certain antidepressant drugs can harmful your liver over time including MAO inhibitor, tetracyclic or tricyclic such as bupropion, duloxetine and agomelatine. Main two different mechanism involved in antidepressant liver toxicity, first one component of metabolism and immunological allergic reaction or pathways. A Hepatotoxicity with weakness and body rashes as clinical toxicology. Small latency period time is (1-6 weeks) is a act on immuno-allergic pathophysiological action. In other way less of hypersensitivity action then greater latency period time is (1month to 1year) is a action idiosyncratic metabolism reaction.

3. Weight Gain and Metabolic Disturbance

Increase in body weight through antidepressant therapy take place through both sensitive and maintaining stage of therapy. Metabolic distraction and weight gain both the familiar adverse effects through antidepressant therapy. Mirtazapine drug is posterity antidepressant major stability assign with beneficial weight gain in the first or primary phase of therapy. The relationship between antidepressant utilize and (DM) diates mellitus persist indecisive.

4. Central Nervous system

Acquired demyelinating syndrome can short the seizure threshold. The lower factors are including alcohol, menstruation, stress and deprivation etc. weakness and migraine general adverse effects interrelation with the utilize of antidepressants in greater retroactive division of adolescents and mature person. In central nervous system (CNS) basis of antidepressants drugs are include in sertraline, fluoxetine, paroxetine etc.

5. Bleeding

Every serotonin antidepressant agent have been relation with greater risk of bleeding problem. Similarly mechanism of action responsible for adverse drug reaction is depletion of serotonin reuptake inhibitors by thrombocyte, other side mechanism have compromise. Fluoxetine, sertraline and paroxetine are greater risk of thrombocyte dysfunction. Certain antidepressants such as Mirtazapine and venlafaxine also have been relation with greater risk of bleeding.

Antidepressant Discontinuation Syndrome[13]

1) Antidepressant Discontinuation Syndrome

It is also known as Antidepressant Withdrawal Syndrome. About 20% of patients developed it due to sudden stoppage, interruption, remarkable reduction or discontinuation of Antidepressant medications which are used continuously for at least a month. The Symptoms are normally mild and may seen in two to four days. The symptoms last for one to two weeks(sometimes for a year). If same or similar drug is started the symptoms resolve within one to three days. The clinical and also the Sociodramatic factors for increased susceptibility have not been identified. Among the Serotonin reuptake inhibitors, paroxetine is associated with the highest incidence of syndrome and fluoxetine with the lowest. Due to venlafaxine's short half life, the syndrome may occurs more frequently following cessation and symptoms may be severe.

2) symptoms are ambiguous and variable

The Failure to identification of Antidepressants Discontinuation Syndrome may result in psychiatric misdiagnosis.

A) Somatic Symptoms

Malaise, Dizziness, light-headedness, nausea, vertigo, fatigue, headache, trimer, muscle spasm, diarrhea, sweating, Hallucination.

B) Psychological Symptoms

Anxiety, insomnia, emotional blunting, irritability. Daws are diverse and variably indicated; the acronym 'FINISH' Flue Like Symptoms (Lethargy, fatigue, headache, sweating) Insomnia (Vivid dreams or nightmares) Nausea (Vomiting) Imbalance (Light headedness, vertigo) Sensory disturbances (Blurring, tingling, shock like sensations) Hyperarousal (Irritability, anxiety, agitation, jerkiness).

3) The Syndrome Needful To Be Distinguished From Relapse

Numerous antidepressants work by an altering the method sure chemicals in a brain alike as serotonin, dopamine and norepinephrine transmit signals required in managing emotions and a moods. Ending antidepressants is thought to interchange hence these brain chemicals function, which may lead to relapse in certain peoples.

Difference between discontinuation syndrome and relapse

Discontinuation Syndrome

Symptoms begin with in a some days. Dizziness, nausea, headache, or body aches are ordinary. Symptoms rule later 1 to 3 weeks.

Relapse

Symptoms slowly evolve over time. Commonly no acute outset of physical symptoms. Symptoms obtain lesser over time.

Side effect[9]

Antidepressants causes consist headache, nausea, vomiting, dizziness, sleep, disturbance, seizure and psychosis. Avoid the use of antidepressants drug on children and adolescents without consultations of doctor because it cause psychiatric disorder. Antidepressants are used to change the behaviour and thinking in children and adolescence.

Table no. 1: Side effect of Antidepressant drug.

		Possible Effect	
		Therapeutic	**Adverse**
1	Norepinephrine Transporter	Antidepressant	a) It causes tremor & tachycardia b) blockade of antihypertensive effect of guanethidine & guanadrel is occur
2	Serotonin Transporter	Antidepressant	a) It affect on gasterointestinal disorder b) Depending on dose it increase or decreases anxiety c) It also affect sexual dysfunction

			d) By interacting with tryptophan, monoamine oxidase inhibitor and fenfluramine causes extrapyramidal adverse effect
3	Muscarinic Receptor	Antidepressant	a) Muscarinic receptor affect on eye & cause blurred vision & narrow angle-Gluacoma b) It also cause dry mouth, constipation c) It causes memory dysfunction & urinary retention
4	Dopamine Transporter	Antidepressant Anti-parkinsonian	a) It activate the psychomotor b) It causes precipitation or aggravation of psychosis
5	Dopamine D_2 receptor	Amellaration of signs & symptoms of psychosis	a) It show extrapyramidal disorder like tardive dyskinesia, parkinsonism, akathisia, etc. b) By affecting endocrine system it causes elevation of prolactin which also cause gynecomastia, galactorrhea, menstural changes.
6	Histamine H_1 receptor	Sedation	a) It causes drowsiness, sedation weight gain

CONCLUSION

Nowadays depression ia critical mental situation in human life, but it can be treated by various accessible treatments, the antidepressant which are used in treatment of depression are selectively used along with safety without any side effects, for the proper medication of the patient is based on the mental condition of patient like symptoms, how the patient react to the medicines, what will be the likely side effects, psychological condition, gestation, lactation, based on the type and requirment of depression the antidepressant can be classified.

Examples

1) Tricyclic antidepressant (TCAs).

2) Selective serotonin reuptake inhibitors (SSRIs).

3) Monoamine oxidase inhibitors (MAOIs).

4) Serotonin norepinephrine reuptake inhibitors (SNRIs).

5) Atypical antidepressant.

Acknowledgment

Firstly, I would like to express my sincere gratitude to my advisor proff. Shrikhrishna baokar {M.PHARM (Pharmaceutical Analyasis)} for the continuous support of my subject (Mechanism and Classification of Antidepressant drug: A reveiw) for his patience, motivation, enthusiasm and immense knowledge. His valuable information helped me in all the time of research and writing of this article. I would not have imagine having a better

advisor and mentor for my this study. Besides my advisor, I would like to thank you rest of my article committee.

Mr.Tejas Tembare

Miss. Mayuri Gophane

Miss. Mayuri Thorat

Miss. Shraddha Shinde

Miss. Srushti Shingate

For their encouragement, insightful comments and hard question, in their working on diverse exciting project. We were working together and for the Sleepless Night. Last but not least I would like to thank you my family ; my parents for supporting me and spirituality throughout my life. I would like to express my gratitude for World journal in pharmaceutical research who gives this chance to publish our review article.

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