AMERICAN JOURNAL OF PHARMTECH RESEARCH
Journal home page: http://www.ajptr.com/

Kleptomania - the Compulsion to Steal

Nasreen Sulthana[1]*, Meenu Singh[1], K Vijaya[1]
1. St. Paul's College of Pharmacy, Turkayamjal(V), Nagarjuna Sagar Road, R.R. Dist Hyderabad - 501 510, Telangana State, India.

ABSTRACT

Kleptomania is the inability to refrain from the urge to steal items and is done for reasons other than personal financial gain. It is a type of impulsive control disorder. Impulse control disorders (ICDs) are common psychiatric conditions in which affected individuals typically report significant impairment in social and occupational functioning, and may incur legal and financial difficulties as well. Kleptomania is characterized by repetitive uncontrollable theft of items that are of little use to the afflicted person. It is commonly associated with other anxiety and eating disorders, alcohol abuse, substance abuse and various mood disorders. Amygdala and nucleus accumbens are the major structures of brain involved in kleptomania. Serotonin and dopamine neuro transmitters and opioids are thought to be involved in the pathophysiology of kleptomania. Decreased serotonin and increased dopamine levels can lead to kleptomania. It can be effectively treated with behavioral or psychotherapy and pharmacological therapies. Selective serotonin inhibitors (SSRIs), tricyclic anti depressants (TCAs) and opioid antagonists are commonly used to treat kleptomania. Behavioral therapy involves Psychotherapy, Covert sensitization, aversion therapy, coping and support, self education, treatment of other mood disorders. This review summarizes the current information, associated disorders, and underlying pathophysiology regarding kleptomania, its treatment and prevention.

Keywords: kleptomania, impulsive compulsive disorder, amygdala, nucleus accumbens, dopamine, serotonin.

*Corresponding Author Email: nasrpharma31@gmail.com
Received 05 May 2015, Accepted 12 May 2015

Please cite this article as: Sulthana N *et al*., Kleptomania - the Compulsion to Steal. American Journal of PharmTech Research 2015.

INTRODUCTION

Kleptomania has been described as an inability to refrain from the urge to steal things for reasons other than personal use or financial gain[1]. Kleptomania is an uncontrollable, repetitive impulse to steal, even though the child is aware that the behavior is wrong, senseless. Often, the items stolen by someone with kleptomania are not even things that the person needs. Kleptomania could be an obsessive-compulsive spectrum disorder according to Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV).

Impulse control disorder (**ICD**) is a class of psychiatric disorders characterized by impulsivity failure to resist a temptation, urge or impulse that may harm one or others. These are characterized by problems in emotional and behavioral self control.

Classification of impulse control disorders

ICD are classified as

1. Pyromania: Pyromania is defined as an impulsive behavior leading to fire setting without an identifiable motive other than taking pleasure in viewing the fire and its effects[2] It is an impulse control disorder in which the person urges to start fire deliberately to relieve tension or for instant gratification. It is multiple, deliberate and purposeful fire setting.
2. Intermittent Explosive Disorder:

   It is characterized by several discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property [3]
3. Kleptomania:

   Kleptomania is characterized by an impulsive urge to steal purely for the sake of gratification
4. Sexual compulsion:

   It is concerned with an increased urge in sexual behavior and thoughts where the compulsion and lead to several consequences in individual's life[4.]
5. Internet addiction:

   It is characterized by excessive and damaging use of internet with increased amount of time spent chatting, web surfing, gambling, shopping or exploring pornographic websites[5.]
6. Compulsive shopping:

   It is concerned with frequent, excessive, irresistible urge to shop even if it is not required or cannot be afforded [6]
7. Tricotillomania:

   It is characterized by recurrent pulling out of one's hair for gratification, pleasure or relief of

tension, resulting in noticeable hair loss [7.]

Epidemiology

Kleptomania is a female-dominant disorder, and the exact causes remain unknown. One of the causes may be because of women's psychic conditions during pregnancy, puberty, and menopause. Women during these phases of life have deviant behavior and are more prone to crime [8]. It may be due to stressful childhood and low self-esteem or head trauma. It seems to be chronic, with exacerbations and remissions. The ratio of occurrence in males to females is 1:3.

Etiology

The behavior or mental disorder, kleptomania is caused to several conditions;

1. Psychological conditions

Low self esteem, stressful childhood and mood deviant conditions in females like pregnancy, disordered menstruation, bad marriages, menopause etc can lead to kleptomania. Also it is believed that women are more "materialistic' than men which contributed to feminization of kleptomania [9.] Deviant or dysfunctional sexuality can also lead to kleptomania in both males and females [10.]

2. Family history

A family history of mental illness, suicide, alcoholism or paralysis is also considered as a causative factor of kleptomania. High rates of mood disorder, alcohol use disorders and kleptomania in first-degree relatives of individuals with kleptomania have been reported [11, 12, 13, and 14.]

3. Biological conditions

Kleptomania may be caused due to imbalances in neurotransmitters like serotonin and dopamine [15.]

4. Associated disorders

Kleptomania is said to be associated with several other neural disorders like obsessive-compulsive disorder, bulimia nervosa, social phobia, anorexia nervosa, alcohol abuse, substance abuse and clinical depression [16, 17 and 18.]

5. Linked to the brain's opioid system:

Urges are regulated by the brain's opioid system. An imbalance in this system could make it harder to resist urges.

Risk Factors

Kleptomania is considered uncommon. However because many people with kleptomania never seek treatment, or they're simply jailed after repeated thefts, many cases of kleptomania may never be diagnosed. Kleptomania often begins during the teen years or in young adulthood, but in rare cases it begins in later adulthood.

Kleptomania risk factors may include:

- **Family history**: Having a first-degree relative, such as a parent or sibling, with kleptomania, obsessive-compulsive disorder, or a substance or alcohol use problem may increase your risk of kleptomania.
- **Being female.** About two-thirds of people with known kleptomania are women.
- **Having another mental illness.** People with kleptomania often have another mental illness, such as bipolar disorder, anxiety disorder, an eating disorder, substance use disorder or a personality disorder.
- **Head trauma or brain injuries.** People who've experienced a head trauma may develop kleptomania.

Symptoms

- According to DSM-V, kleptomania is characterized by [19]
- Repeated theft of objects that are unnecessary for either personal use or for their monetary value.
- An increase in tension, anxiety or arousal occurs leading up to the theft
- The tension is relieved and pleasure is felt while stealing or during the act of the theft
- Objects stolen are of little value and affordable.
- After stealing, the individual will then typically discard, hoard, secretly return or give them away. They have great levels of guilt, stress and remorse and privacy issues accompanying the act of stealing (Figure 1).

Left untreated, kleptomania can result in severe emotional, family, legal, work and financial problems. For example, one knows stealing is wrong but feel powerless to resist the impulse, may be wracked by guilt, shame, self-loathing and humiliation or may otherwise lead a moral, upstanding life and be confused and upset by compulsive stealing.

Examples of complications that kleptomania may cause or be associated with include:

- Compulsive gambling or shopping
- Arrest for shoplifting
- Imprisonment
- Alcohol and substance abuse
- Eating disorders
- Depression
- Anxiety

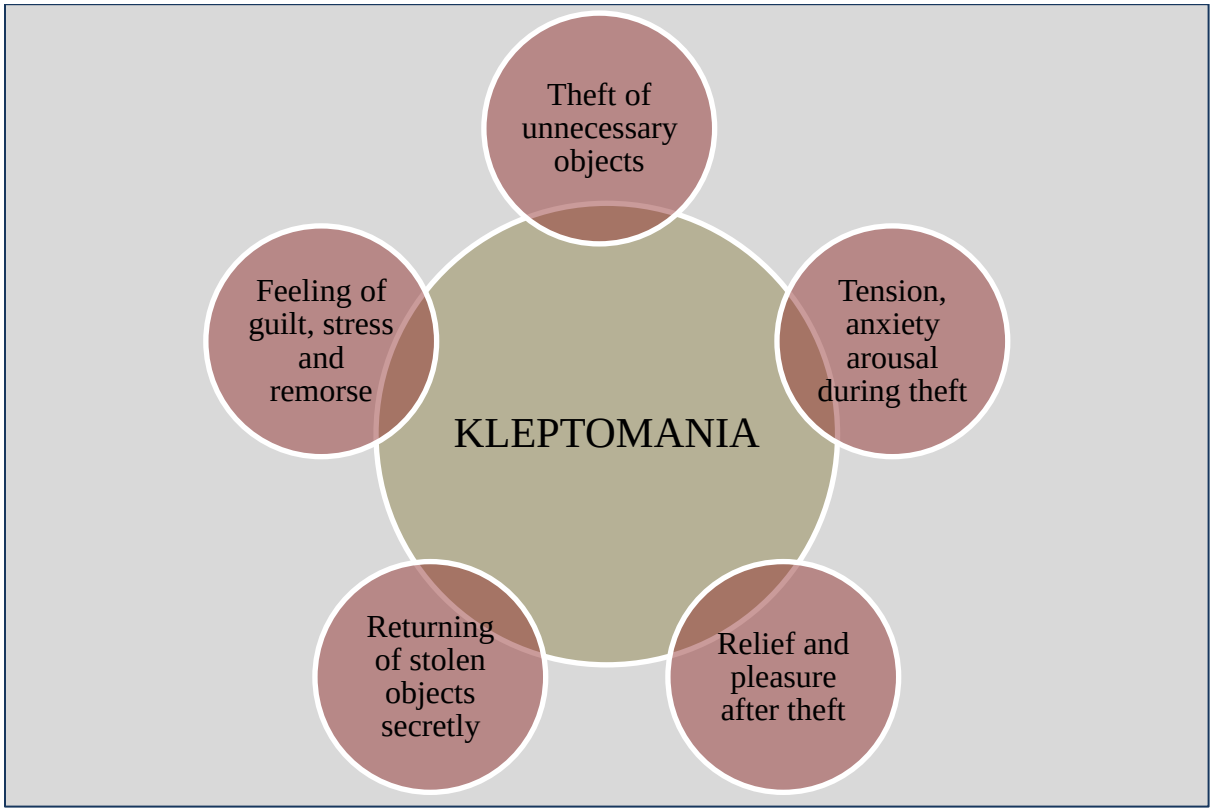

Figure 1: Symptoms of kleptomania

Pathophysiology

Behavioral addictions are characterized by dysfunction in multiple brain areas and neurotransmitter systems. The two main neurotransmitters associated with behavioral addiction are serotonin and dopamine.

Role of serotonin

Serotonin is said to be involved in every type of behaviour such as appetite, emotion, motor, cognitive and autonomic. It modulates the tone of nervous system like firing rate of serotonergic soma in raphe nuclei. Kleptomania is due to a defect in a molecule that transfers serotonin, which regulates moods and emotions[20]. The most common structure of brain involved in behavioural addiction is amygdala[21](Figure 2). It has sertotonergic, dopaminergic, opiodergic and nor adrenetrgic neurons. Over expression of Fos B gene is seen in the nucleus accumbens of amygdala in patients with kleptomania. Amygdala is involved in emotional significance and associated learning [22, 23 24.]

Serotonin receptors

5-HT1A receptors are present in raphe nuclei [25, 26.] These receptors are auto receptors which control the firing of serotonergic neurons. These receptors couple with Gi protein of G protein Coupled receptors. They inhibit adenylate cyclase enzyme and inhibit cAMP formation and

therefore are inhibitory in nature. Small amount of 5-HT3 receptors are also found in amygdala, hippocampus and fore brain regions. These receptors are ion channels which are selective to Na^+ or K^+ or Ca^{+2}. Kleptomania involves disruption of neurotransmitter serotonin [27.]

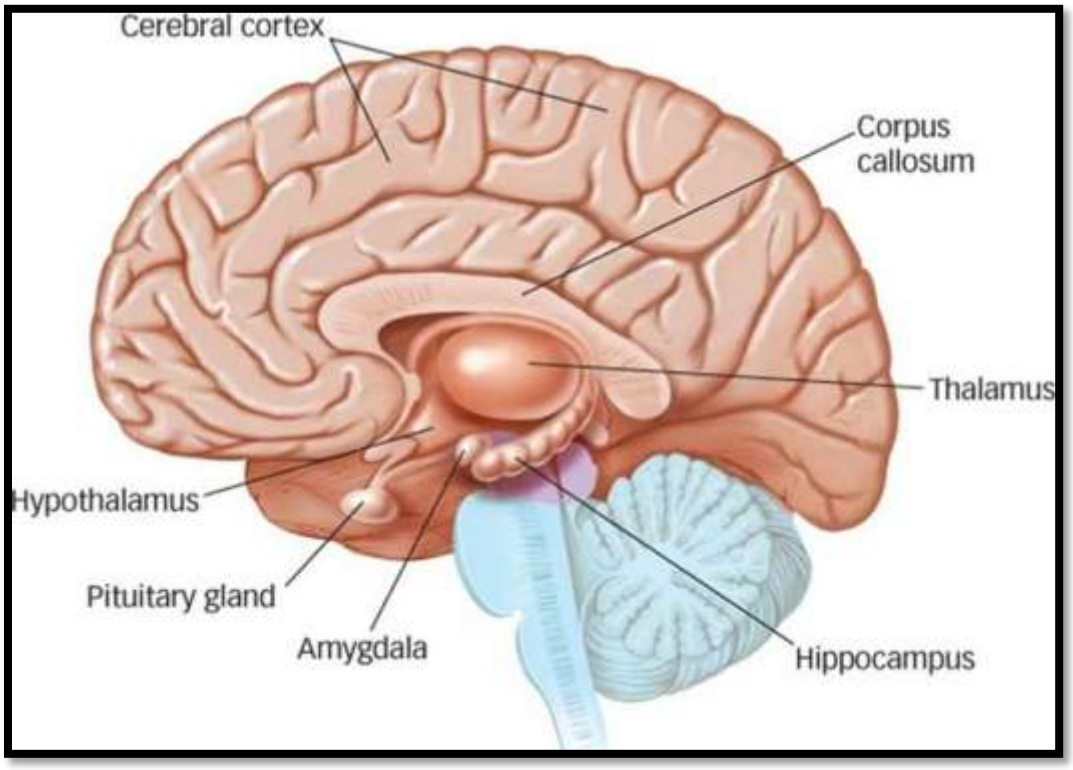

Figure 2: Structure of Brain showing amygdala

Role of dopamine

Dopamine in brain effects movement, cognition, pleasure and motivation. It is a primary neurotransmitter involved in reward pathway in brain. The reward system is a group of neural structures that are critically involved in mediating the effects of reinforcement. It is an essential neurotransmitter in basal ganglia responsible for subconscious contractions of skeletal muscles, cognitive functions such as attention, memory and planning and also regulates emotional behaviours[28]. When dopamine is released it gives the feeling of pleasure or satisfaction. This feeling of satisfaction becomes desired and the person will grow a desire for the satisfaction. To satisfy that desire the person will repeat behaviours that cause the release of dopamine. It is also observed that a gene related to dopamine is found twice as often in pathological gamblers than non gamblers [29.] Therefore it can be concluded that dopamine levels are genetic. Activation of the reward system is closely associated with an increased activity of dopamine pathways particular mesolimbic pathway which projects from the ventral tegmental area (VTA) to nucleus accumbens. Mesocorticolimbic nèurons play a central role in reward and other aspects of motivation.[30]

Dopamine receptors

Mostly D2 receptors are found to be involved in behavioural addiction [31]. These receptors couple with Gi protein of GPCR decreasing the formation of cAMP. These receptors are auto receptors which regulates the activity of dopaminergic neurons. These receptors reuptakes the neurotransmitter through DAT (dopamine transporter) and helps regulate the dopamine levels in brain. Blockade of DAT causes the synapse to flood with dopamine and hence increases dopaminergic signalling. If this occurs in nucleus accumbens, it increases D1 activity and decreases D2 receptor signalling mediating the rewarding stimulus in kleptomania [32.]

Role of opioids.

Opioid receptors are distributed throughout the brain and spinal cord and are known to mediate a number of activities including analgesia, species-typical behavior, and reward. Both endogenous opioids, which are naturally produced within the body, and exogenous opiates, which are produced outside the body, produce a variety of symptoms including pain relief, euphoria, respiratory depression (rarely clinically harmful), constipation, nausea, and vomiting. The effects are produced by opioids binding to opioid receptors throughout the body. opioids act at three distinct classes of receptors: kappa, delta, and mu, although it is likely that additional subtypes exist [33.] Since each class of receptor has a unique effect on the cell, the multitude of classes allows opioids to have a wide range of effects in the body. Most of the opioid receptors have common general structure. They are generally G-Protein Coupled receptors (GPCR) [34.] They occasionally act independent of G-Protein.

These receptors inhibit the activity of adenylate cyclase [35] which is an enzyme responsible for catalyzing numerous chemical reactions in neurons. All three types of receptors are present both pre synaptically and post synaptically [36.] When acting at presynaptic receptors, the peptides function as neuromodulators affecting the release of neurotransmitters. At postsynaptic receptors, the peptides act as neurotransmitters by directly altering membrane potentials. The overall effect of opioids on a particular tissue depends upon the concentration and location of particular opioid receptors in the area. The opioid system is connected with most neurotransmitter networks in the body. The interaction between the opioids and the dopaminergic system appears to be involved in addiction, tolerance, and withdrawal symptoms. The relevant interaction appears to occur along the mesolimbic projection, particularly in the ventral tegmental area (VTA) and nucleus accumbens (NA). Dopamine Involvement in Opioid Reinforcement at the VTA. Numerous studies suggest that the VTA, a known center of DA activity, is involved in opioid reward. opioids enhances the firing frequency of mesolimbic DA neurons projecting from the VTA [37,] which provides firm

evidence that opioids have an excitatory affect on dopamine. Not only do opioids have an excitatory effect on dopamine; the effects of opioids seem to be contingent upon dopamine activation. Dopamine antagonists, molecules that bind to the receptor and prevent it from being activated, block the effect of opioids by halting morphine-induced activities[38]. Dopamine involvement in Opioid Reinforcement at the NA. While dopaminergic neurons project from the VTA to structures throughout the brain, the neurons heading to the NA have been repeatedly implicated in the rewarding properties of opioids. Systemic administration (into the body at large) of opiates increase dopamine turnover in the NA [39] which suggests that opioids increase dopamine activity. It has been further demonstrated that opiates increase activity of early genes, c-fos, c-jun, and zif altering gene transcription[40] which suggests that opioids cause long-lasting and enduring changes in the cells of the NA (Figure 3). Although dopamine excitation likely increases the rewarding effect of opioids, it appears that reinforcement is not contingent upon dopamine activation. It is important to note that the animals will modify their behavior more to obtain opioids in the VTA[41] which suggests that the VTA activation produces a more rewarding effect than NA activation. It is interesting to note that opioid and dopamine agonists alike, both substances associated with addiction, depress overall excitation in the NA[42.]

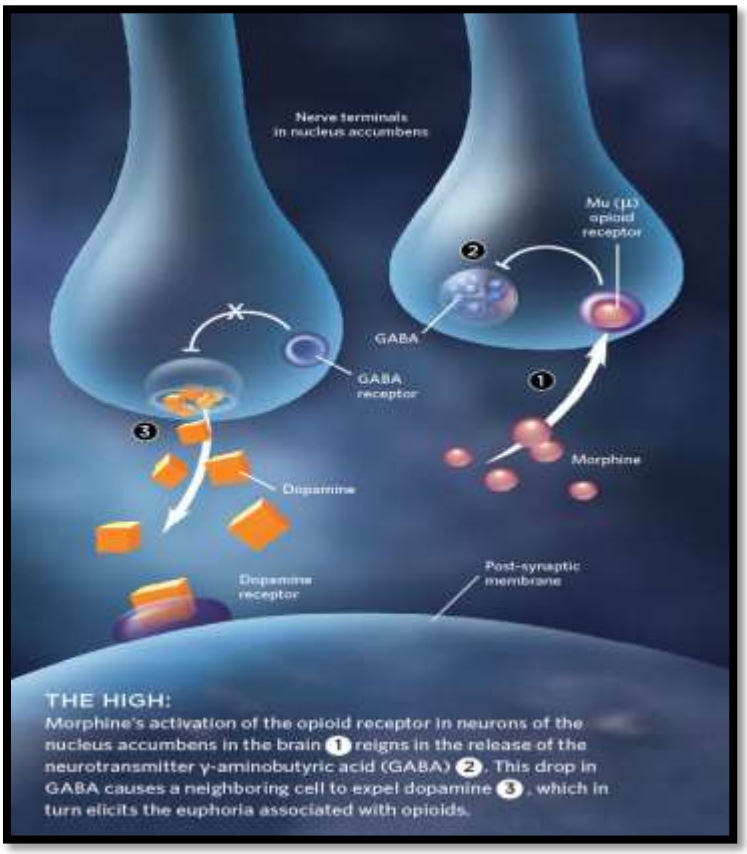

Figure 3: Relationship between opioids and dopamine.

Diagnosis and Comorbidity

Kleptomania seems to b likely associated with other disorders [16, 17, and 18.] Therefore stealing symptom can be seen in other disorders. Diagnosis of kleptomania is done by taking a screening test as follows [43.]

QUESTIONNAIRE FOR KLEPTOMANIA

	YES	NO
1. Do you steal or have urge to steal?	☐	☐
2. Do thoughts of stealing or urges to steal preoccupy you? That is, do you often about stealing or have urges to steal and wish the thoughts or urges occur less often?	☐	☐
3. Do you feel tense or anxious before you steal or when you have urges to steal?	☐	☐
4. Do you feel pleasure or a sense of calm when you steal something?	☐	☐
5. Has the stealing or urges to steal caused you much distress?	☐	☐
6. Has the stealing or urges to steal significantly interfered with your life in some way?	☐	☐

A patient who answers "yes" to questions 1 through 4 and to question 5 or 6 is likely to have kleptomania.

DSM IV criteria, American Psychiatric Association, 2000.

To better understand the symptoms and their effects, the following questions are to be asked

- At what age did you first experience an irresistible urge to steal?
- How often do you experience the urge to steal?
- Have you ever been caught or arrested for stealing?
- How would you describe your feelings before, during and after you steal something?
- What kinds of items do you steal? Are they things you need?
- From whom do you steal?
- What do you do with the items you steal?
- Does anything in particular seem to trigger your urge to steal?
- How is your urge to steal affecting your life, including school, work and personal relationships?

- Have any of your close relatives had a problem with compulsive stealing or with other mental health conditions such as depression, addiction or obsessive-compulsive disorder?
- Have you been treated for any other mental health problems, such as eating disorders? If yes, what treatments were most effective?
- Do you use alcohol or illegal drugs? How often?
- Are you currently being treated for any other medical conditions?

Differential diagnosis of kleptomania

As stealing is seen in other mental conditions, the following are the differences in symptoms between kleptomania and other mood disorders [43] (Table 1).

Table 1: differential diagnosis of kleptomania

S. No	Misdiagnosis	Patient with other disorder	Patient with kleptomania
1.	Bipolar disorder	They show elevated, expansive or irritable mood while stealing.	They show depressed mood when not stealing
2.	Border line personality disorder	They show long histories of unstable relationships, negative self image, inappropriate anger and psychotic like symptoms.	No such symptoms are seen.
3.	Antisocial personality disorder	No guilt or shame is seen	Show intense guilt and shame.
4.	Eating disorders	Disordered eating pattern is seen	Disordered eating pattern is not seen.

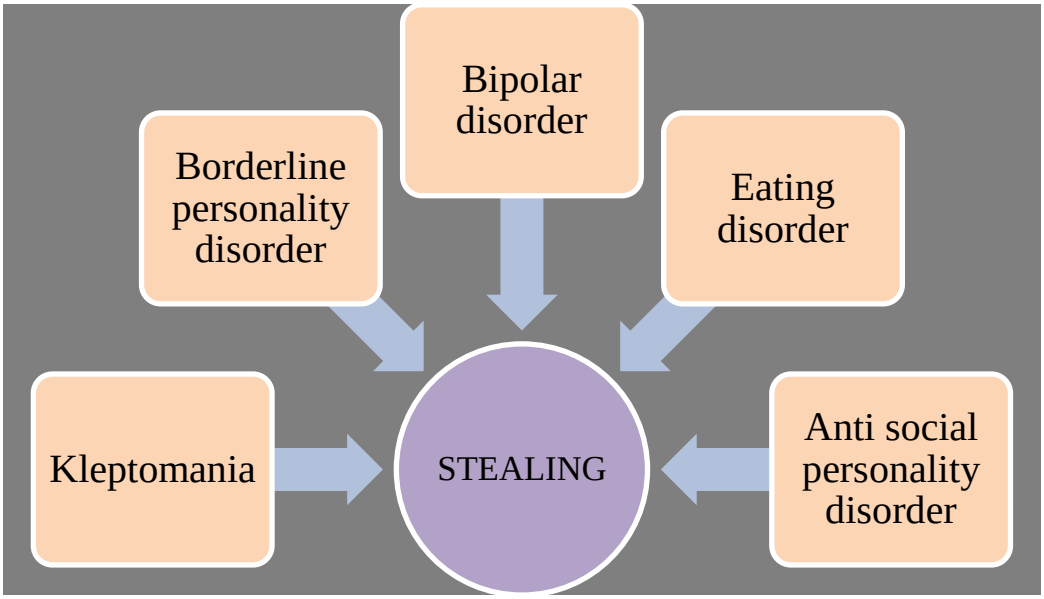

Figure 4: Comorbidity of Kleptomania.

Because of cormobidity with certain disorders every patient should be screened with a mood, Substance abuse, anxiety or eating disorder (Figure 4).

Treatment

Without treatment kleptomania can be ongoing, long term condition. The treatment strategies for kleptomania include pharmacotherapy, psychotherapy, combination therapy, self help groups and patient education[43.]

Pharmacotherapy

There are no FDA approved drugs for kleptomania. As the neurotransmitters dopamine and serotonin are involved treatment is based on the drugs related to these neurotransmitters. Commonly used drugs are [43:]

1. Selective Serotonin Reuptake Inhibitors (SSRI)
   Eg: Flouxetine, fluvoxamine, paroxetine
2. Tricyclic antidepressants
   Eg: imipramine, Nortryptiline
3. Opioid antagonists
   Eg: Nlatrexone.
4. Mood stabilizers
   Eg: lithium and valproate.

SSRI

SSRIs are the drugs that block the reuptake of serotonin in the brain which are commonly used as anti-depressants (Figure 5).

- Kleptomania is characterized by disruption of serotonin neurotransmitter. Therefore increasing serotonin levels is the aim of therapy. SSRIs inhibit the reuptake of serotonin by blocking the transporter [44.]
- Fluoxetine has affinity for monoamines but lack affinity for Acetylcholine, histamine and alpha adrenoreceptors.
- Fluoxetine, sertraline, citalopram exist as isomers and are formulated in racemic forms.
- Paroxetine and fluvoxamine are not optically active.
- Escitalopram is the s-enantiomer of citalopram
- SSRIs are highly lipophilic drugs.

Unlike TCAs antidepressants, SSRIs are highly selective: they act as weak inhibitors in the reuptake of non-serotonergic neurotransmitters such as norepinephrine, but act as strong inhibitors

in the reuptake of serotonin. Because of this selectivity, there are fewer side effects associated with SSRIs than with TCAs and their side effects are due to actions at other serotonin receptors in the central nervous system and the gut wall [44].

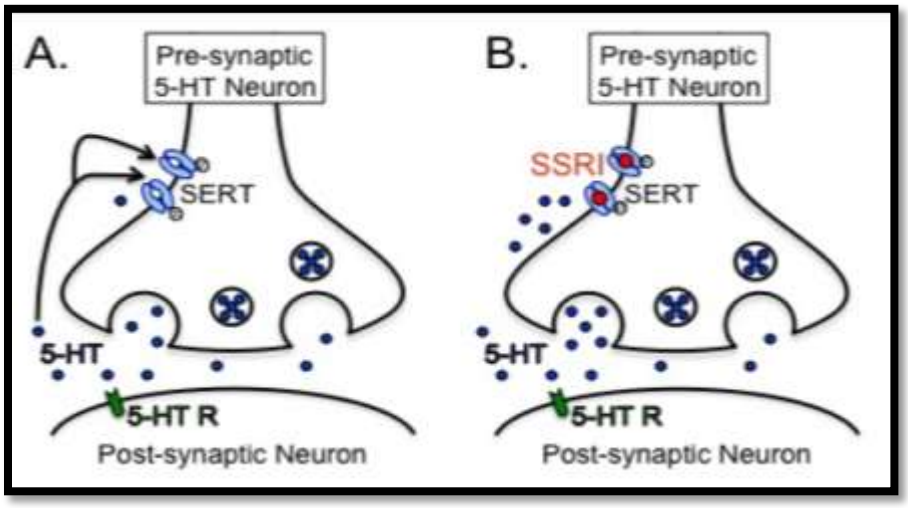

Figure 5: A: Serotonin (5-HT) released by the neuron is recycled by the serotonin transporter (SERT). B: SSRIs block the SERT and 5-HT is accumulated in the synapse.

SSRIs have variances in molecular structures. Selective serotonin reuptake inhibitors (citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, and sertraline) are structurally diverse but share a common mechanism of action.

Adverse effects of SSRIs:

SSRIs affect fewer sites of action than TCAs, and as a result cause fewer types of adverse effects. The SSRIs have a better overall tolerability profile than the TCAs in both acute and long-term treatment [45]. SSRIs induce significantly less anticholinergic, antihistaminergic and cardiotoxic side-effects than TCAs[46].

As a group, the SSRIs possess the following adverse effects:

- Nausea: The most common side effect associated with use of SSRIs is nausea.
- Sexual dysfunction: Sexual dysfunction includes decreased libido, orgasm difficulties, abnormal ejaculation and impotence.
- Diarrhea: Sertraline and fluoxetine are more frequently associated with diarrhea due to their greater specificity for serotonin receptors, while paroxetine has a lower incidence because of its antimuscarinic effects.
- Insomnia: SSRIs interfere with sleep architecture. Fluoxetine, paroxetine, and sertraline delay the onset of REM sleep, and fluoxetine and paroxetine increase awakenings and reduce REM sleep, slow-wave sleep, total sleep time, and sleep efficiency. In contrast, sertraline minimally

increases sleep efficiency and reduces nocturnal wakefulness time, which may benefit depressed patients with sleep disturbances [47].

- Anticholinergic effects: paroxetine causes a higher rate of anticholinergic effects, such as dry mouth, constipation, and cognitive disruption, compared with other SSRIs.
- Anxiety, agitation: Fluoxetine has been associated with highest rate of anxiety and agitation[1]. Escitalopram and paroxetine are less likely to cause insomnia than fluoxetine and sertraline. Escitalopram and citalopram have been associated with low rates of insomnia, anxiety, and other activating side effects.
- Weight gain: Weight gain is another troubling side effect. The SSRIs vary in their effect on the weight.
- Other adverse effects of SSRIs include: headache, nervousness, agitation, tachycardia, insomnia, drowsiness, increased appetite.
- The incidence of adverse effects varies according to the drug and patient may respond differently to certain SSRI or experience different adverse effect with different drugs.
- Withdrawal symptoms:

  SSRIs aren't considered to be addictive. However, stopping treatment abruptly or missing several doses can cause withdrawal-like symptoms, including nausea, headache, dizziness, lethargy and flu-like symptoms. This is also called as discontinuation syndrome.
- These drugs are not safe to use in pregnancy.

Tricyclic antidepressants

These are also antidepressant drugs.

Tricyclic antidepressants work by raising the levels of neurotransmitters serotonin and norepinephrine in the brain by slowing the rate of reuptake (reabsorption) by nerve cells (Figure 6). TCAs act as strong inhibitors in the reuptake of both norepinephrine and serotonin. Unfortunately, the TCAs also block histaminic, cholinergic, and alpha1-adrenergic receptor sites, and this lack of selectivity is what accounts for the unwanted side effects such as weightgain, dry mouth, constipation, drowsiness, and dizziness.

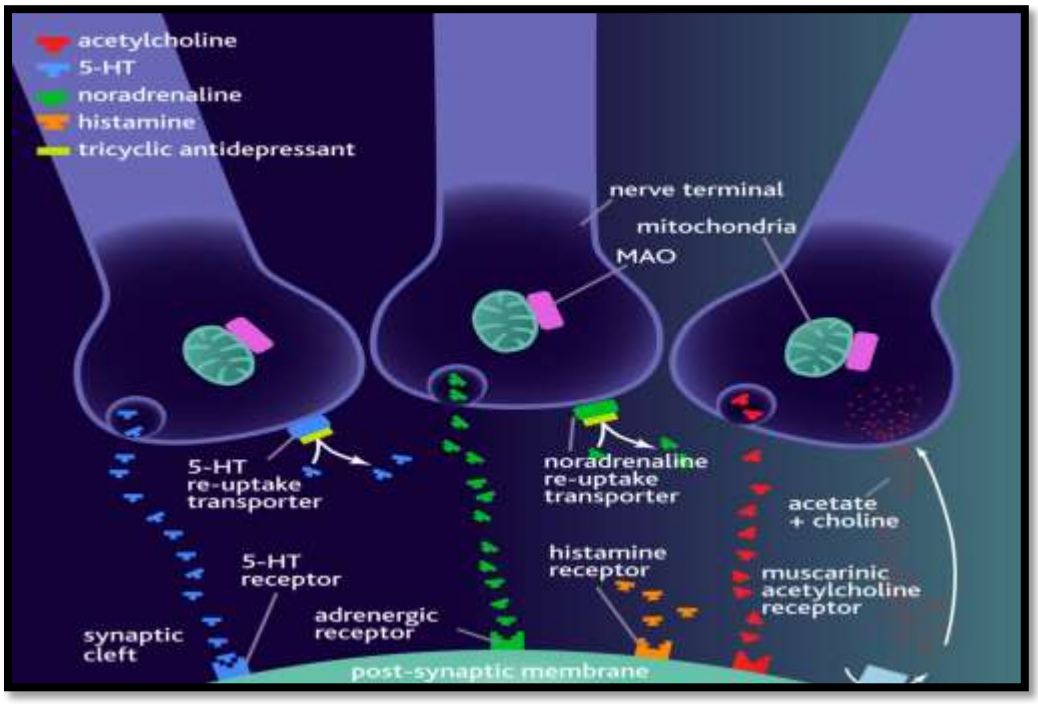

Figure 6: Mechanism of action of tricyclic anti depressants.

Tricyclic antidepressants (amitriptyline, amoxapine, clomipramine, dosulepin, doxepin, imipramine, lofepramine, nortriptyline, and trimipramine) are structurally similar.

Side effects

TCA s has high anti cholinergic, anti histaminergic and cardiotoxic side effects when compared to SSRIs.

- Cardiovascular effects. Tricyclic antidepressants have significantly higher rate of serious cardiovascular side effects[48.] Selective serotonin reuptake inhibitors as a class are less likely to affect cardiovascular parameters.
- Through a combination of anticholinergic activity, direct myocardial depressant activity and an effect on the adrenergic neuron, TCAs can cause a combination of arrhythmias (disturbances in cardiac rhythm or conduction), blood pressure abnormalities (orthostatic hypotension) and congestive heart failure [49]. The major side effects in therapeutic dosage include heart rate increase, postural hypotension and slight prolongation of the intraventricular conduction time and QT interval[50.]
- SSRIs also appear to affect the cardiovascular system [51, 52].
- Anticholinergic effects (dry mouth, blurred vision, drowsiness, constipation, and difficulty in urination). Dry mouth is a most common TCAs' side effect [53.] Tricyclic antidepressants produce a greater incidence of dry mouth, drowsiness, constipation and fatigue than SSRIs [54].

- Weight gain: Both SSRIs and TCAs can cause unwanted weight gain. However, there is tricyclic antidepressants may be more likely to cause weight gain and increased appetite than the selective serotonin reuptake inhibitors [55.]
- Sexual effects: Sexual dysfunction such as decreased sexual desire, erectile difficulties and delayed ejaculation is one of the most frequent and persistent SSRI adverse effect. These drugs are more likely to cause sexual dysfunction than the TCAs [56, 57.]
- Central Nervous System effects (headache, dizziness, agitation, insomnia and tremor). In contrast to the tricyclic antidepressants, SSRIs are more likely to cause headache, agitation, insomnia and tremor .
- Nausea: Nausea occurs more frequently with SSRIs. It is the most common adverse event reported during treatment with SSRIs[53, 54].
- Gastro-intestinal effects. Compared to TCAs, SSRIs have higher incidence of gastro-intestinal side effects [57.]

Table 2: comparison between the pharmacological effects and adverse effects of SSRIs and TCAs.

S.No	Pharmacological affect or adverse effect	SSRIs	TCAs
1	Serotonin reuptake	Present	Present
2	Anti cholinergic	Absent	Present
3	Noradrenergic reuptake	Absent	Present
4	Sexual dysfunction	More	Less
5	Nausea	More	Less
6	CNS effects	More	Less
7	Weight gain	Less	More
8	GIT effects	More	Less

Opioid antagonists:

Kleptomania is often characterized by uncontrollable cravings and excitement upon stealing an item. A reward-triggered release of dopamine is found to b implicated in this process [58.]Alteration i.e. in decrease in dopamine levels produces the feeling of pleasure associated with kleptomania. Therefore opioid antagonist such as naltrexone decreases dopamine neurotransmitter in the nucleus accumbens is found to be beneficial agent in dampening the excitement and cravings reported in kleptomania [59.]

Psychotherapy [42]:

A form of psychotherapy called cognitive behavior therapy helps in identifying unhealthy, negative beliefs and behaviors and replace them with healthy, positive ones. It involves targeting

urge and behavior and mechanisms for coping with urges and behavior. Cognitive behavior therapy includes the following techniques which may help to overcome kleptomania.

Covert sensitization:

It is a technique in which the patient pictures himself stealing and then facing negative consequences such as being caught.

Aversion therapy:

This is a mild painful technique which involves during an urge to steal, holding breath until the patient becomes uncomfortable.

Systemic desensitization:

This involves relaxation techniques which help in controlling urges to steal

Combination therapy:

Combined pharmacological and behavioral therapy is an optimum treatment strategy for kleptomania.

Coping and support[44:]

- Stick to the treatment plan:
  Medications should be taken as directed and patient should attend scheduled therapy sessions.
- Self Education:
  Patient should be aware of all the risk factors, treatment and triggering events of kleptomania.
- Discovering the driving force:
  The situations, thoughts and feelings that trigger the urge to steal should be identified in an individual.
- Treating substance abuse or other mental health problems: Addictions, depression, anxiety and stress can feed off each other, leading to a cycle of unhealthy behavior.
- Finding healthy outlets:
  Explore healthy ways to rechanneled urges to steal or shoplift through exercise and recreational activities.
- Relaxation and stress management: stress-reduction techniques as meditation, yoga or tai chi should be practiced.
- Focus on goal: Recovery from kleptomania can take time. Keep motivated by keeping recovery goals in mind and reminding them that they can work to repair damaged relationships and financial and legal problems.

Avoiding relapses

- It's not unusual to have relapses of kleptomania. To help avoid relapses, patient should stick to

- the treatment plan. If they feel urges to steal, they should contact their mental health provider or reach out to a trusted person or support group.
- Self-help groups:

  People with kleptomania may benefit from participating in self-help groups based on 12-step programs.

Prevention

There are no guidelines for the prevention of kleptomania.

- Healthy upbringing of children
- Positive intimate relationships
- Better management of stressful situations

To prevent the stealing problem, the patient should be accompanied by a friend. The friend's conscious mind will help stop the stealing tendency. Getting treatment as soon as compulsive stealing begins may help prevent kleptomania from becoming worse and prevent some of the negative consequences.

REFERENCES

1. Ajish G, Mangot. Kleptomania: Beyond serotonin, J Neurosci Rural Pract. 2014; 5 (1): S105–S106.
2. Candice Germain and Michel Lejoyeux. Pyromania: Clinical Aspects. Cambridge University 2015: 255-268.
3. Mattes JA. Comparative effectiveness of carbamazepine and propranolol for rage outbursts. J Neuropsychiatry Clin Neurosci 1990; 2:159- 64.
4. Barth RJ, Kinder BN. The mislabeling of sexual impulsivity. J Sex Marital Ther 1987; 1:15–23.
5. Shaw M, Black DW. Internet addiction: definition, assessment, epidemiology and clinical management. CNS Drugs 2008; 22: 353-365.
6. Dell'Osso B, Altamura AC, Allen A, Marazziti D, Hollander E. Epidemiologic and clinical updates on impulse control disorders: A critical review. European Archives of Psychiatry and Clinical Neuroscience 2006; 256 (8):464–475.
7. Ferra´o YA, Miguel E, Stein DJ. Tourette's syndrome, trichotillomania, and obsessive-compulsive disorder: how closely are they related? Psychiatry Res 2009;170:32–34.
8. Bullock SA, Potenza MN. Pathological gambling: neuropsychopharmacology and treatment. Curr Psychopharmacol 2012; 1:67-85.

9. Hodgins DC, Stea JN, Grant JE. Gambling disorders. Lancet 2011; 378(9806): 1874-1884.
10. Kendler KS, Chen XN, Dick D, et al. Recent advances in the genetic epidemiology and molecular genetics of substance use disorders. NatNeurosci 2O12;15(2):181-I89.
11. Grant JE, Kim SW. Clinical characteristics and associated psychopathology in 22 patients with kleptomania. Compr Psychiatry 2002; 43(5):378-84.
12. Presta S, Marazziti D, Dell'Osso L, Pfanner C, Pallanti S, Cassano GB. Kleptomania: clinical features and comorbidity in an Italian sample. Compr Psychiatry 2002;43(1):7-12.
13. Bayle FJ, Caci H, Millet B, Richa S, Olie JP. Psychopathology and comorbidity of psychiatric disorders in patients with kleptomania. Am J Psychiatry 2003; 160(8):1509-13.
14. Grant JE. Family history and psychiatric comorbidity in persons with kleptomania. Compr Psychiatry 2003; 44(6):437-41.
15. Brewer JA, Potenza MN. The neurobiology and genetics of impulse control disorders: relationships to drug addictions. Biochem Pharmacol 2008;75(l):63-75.
16. Bayle FJ, Caci H, Millet B, Richa S, Olie JP. Psychopathology and comorbidity of psychiatric disorders in patients with kleptomania. Am J Psychiatry 2003; 160(8):1509-13.
17. Grant JE. Family history and psychiatric comorbidity in persons with kleptomania. Compr Psychiatry 2003; 44(6):437-41.
18. McElroy SL, Hudson JI, Pope HG, Keck PE. Kleptomania: clinical characteristics and associated psychopathology. Psychol Med 1991;21(1):93-108..
19. Goldman M. Kleptomania: Making sense of the nonsensical. Am J Psychiatry 1991; 148:986–996.
20. Bechara A, Tranel D, Damasio H. Characterization of the decision making deficit of patients with ventromedial prefrontal cortex lesions. Brain- A journal of Neurology 2000; 123(11):2189-202.
21. Brewer, Judson A, Potenza, Marc N. The neurobiology and genetics of impulse control disorders: Relationships to drug addictions. Biochemical Pharmacology 2008; 75 (1): 63–75.
22. Robison AJ, Nestler EJ, Nestler. Transcriptional and epigenetic mechanisms of addiction. Nat. Rev. Neurosci 2008; 12 (11): 623–637.
23. Blum K, Werner T, Carnes S, Carnes P, Bowirrat A, Giordano J, Oscar-Berman M, Gold M, Werner, Carnes, Bowirrat, Giordano, Oscar-Berman, Gold. Sex, drugs, and rock 'n' roll: hypothesizing common mesolimbic activation as a function of reward gene polymorphisms. *J.* Psychoactive Drugs 2012*;* 44 (1): 38–55.

24. Olsen CM. Natural rewards, neuroplasticity, and non-drug addictions. Neuropharmacology 2011; 61 (7): 1109–22.
25. Hall H, Lundkvist C, Halldin C, Farde L, Pike VW, Mc Carron JA. Autoradiographic localization of 5-HT_{1A} receptors in the post-mortem human brain using [^{3}H] WAY-100635 and [^{11}C] WAY-100635. Brain Res 1997; 745:96-108.
26. Pedigo NW, Yamamura HI, Nelson DL. Discrimination of Multiple [^{3}H] 5-Hydroxytryptamine Binding Sites by the Neuroleptic Spiperone in Rat Brain. J Neurochem 1981; 36:220-226.
27. Hoyer D, Clarke DE, Fozard JR, Hartig PR, Martin GR, Mylecharane EJ, Saxena PR, Humphrey PP. International Union of Pharmacology classification of receptors for 5-hydroxytryptamine (Serotonin). Pharmacol. Rev 1994; 46 (2): 157–203.
28. Tortora GJ, Derricson BH. Principles of Anatomy and Physiology. 14th ed., 2013: 510-513.
29. Girault, Jean-Antoine, Greengard P. The Neurobiology of Dopamine Signaling. Archives of Neurology 2004; 61 (5): 641–44.
30. Brewer, Judson A, Potenza, Marc N. The neurobiology and genetics of impulse control disorders: Relationships to drug addictions. Biochemical Pharmacology 2008; 75 (1): 63–75.
31. Koob GF. Drugs of abuse: anatomy, pharmacology and function of reward pathways.Trends Pharmacol Sci 1992; 13:177–84.
32. Wise RA, Bozarth MA. A psychomotor stimulant theory of addiction. Psychol Rev 1987; 94:469–92.
33. Dhawan BN, Cesselin F, Raghubir R, Reisine T, Bradley PB, Portoghese PS, and Hamon M, International union of pharmacology classification of opioid receptors, Pharmac. Rev. 1996; 48: 567-591.
34. Satoh M and Minami M. Molecular pharmacology of the opioid receptors. Pharmacol. Ther 1995; 68: 343-364.
35. Childers SR. Opioid receptor-coupled second messenger systems. Life Science 1991; 48: 1991-2003.
36. Simon EJ. Opioid receptors and endogenous opioid peptides. Medicinal Res. Rev. 1991; 11: 357-374.
37. Matthews RT. and German DC. Electrophysiological evidence for excitation of rat VTA dopamine neurons by morphine. Neurosci. 1984; 11: 617-625.
38. Iwamoto ET. Locomotor activity and antinociception after potative mu, kappa, and sigma opioid agonists in the rat: Influence of dopaminergic agonists and antagonists. J. Pharmac. Exp. Ther. 1981; 217: 451-460.

39. Westernik BH. and Korf J. Regional rat brain levels of dihydrophenylacetic acid and homovanillic acid: Concurrent fluoimetric measurement and influence of drugs. Eur. J. Pharmacol. 1976; 38: 281-291.
40. Graybiel AM, Moratalla R., Robertson HA. Amphetamine and cocaine induce drug-specific activation of the c-fos gene in striosome-matrix compartments and limbic subdivisions of the striatum. Proc. Natn. Acad. Sci. USA 1990; 87: 6912-6916.
41. Olds ME. Reinforcing effects of morphine in the nucleus accumbens. Brain Res. 1982; 237: 429-440.
42. Pennartz CMA, Dolleman-van Der Weel MJ, Kitai ST, and Lopes da Silva FH. Presynaptic dopamine D1 receptors attenuate excitatory and inhibitory limbic inputs to the shell region of the rat NA studies in vitro. J Neurophysiol 1992; 67: 1325-1334.
43. Grant JE, Kim SW. Kleptomania: Emerging therapies target mood, impulsive behavior, Current Psychiatry 2002; 1: 1-6,
44. Katzung BG, Masters SB, Trevor AJ. Basic and Clinical Pharmacology. 11th ed., Mc Graw Hill: Lange 2009; 513-526.
45. Nurnberg HG, Thompson PM, Hensley PL. Antidepressant medication change in a clinical treatment setting: a comparison of the effectiveness of selective serotonin reuptake inhibitors. J Clin Psychiatry.1999; 60(9):574-9.
46. Pacher P, Ungvari Z, Nanasi PP, Furst S, Kecskemeti V. Speculations on difference between tricyclic and selective serotonin reuptake inhibitor antidepressants on their cardiac effects. Is there any? Curr Med Chem 1999; 6(6):469-80.
47. Rose JB. Tricyclic antidepressant toxicity. Clin Toxicol 1977; 11(4):391-402.
48. Roose SP, Laghrissi-Thode F, Kennedy JS. Comparison of paroxetine and nortriptyline in depressed patients with ischemic heart disease. JAMA 1998; 279(4):287-19.
49. Jefferson JW. A review of the cardiovascular effects and toxicity of tricyclic antidepressants. Psychosom Med 1975; 37(2):160-79.
50. Roos JC. Cardiac effects of antidepressant drugs- A comparison of the tricyclic antidepressants and fluvoxamine. Br J Clin Pharmacol 1983;15 (3):439S-445S
51. Pacher P, Kecskemeti V. Cardiovascular effects of selective serotonin reuptake inhibitor antidepressants. Orv Hetil 2004; 145(8):425-31.
52. Rodriguez de la Torre B, Dreher J, Malevany I, Bagli M, Kolbinger M, Omran H, Luderitz B, Rao ML. Serum levels and cardiovascular effects of tricyclic antidepressants and selective serotonin reuptake inhibitors in depressed patients. Ther Drug Monit 2001; 23(4):435-40

53. Trindade E, Menon D, Topfer LA, Coloma C. Adverse effects associated with selective serotonin reuptake inhibitors and tricyclic antidepressants: a meta-analysis. CMAJ 1998; 159(10): 1245–1252.
54. Kyle CJ, Petersen HE, Overo KF. Comparison of the tolerability and efficacy of citalopram and amitriptyline in elderly depressed patients treated in general practice. Depress Anxiety 1998;8(4):147-53.
55. Fava M. Weight gain and antidepressants. J Clin Psychiatry 2000; 61 (11):37-41
56. Steffens DC, Krishnan KR, Helms MJ. Are SSRIs better than TCAs? Comparison of SSRIs and TCAs: a meta-analysis. Depress Anxiety 1997;6(1):10-18.
57. Reimherr FW, Chouinard G, Cohn CK, et al. Antidepressant efficacy of sertraline: a double-blind, placebo- and amitriptyline-controlled, multicenter comparison study in outpatients with major depression. J Clin Psychiatry 1990;51:18-27
58. Grant JE, Kim SW. Adolescence Kleptomania treated with naltrexone: a case report. Eur Child Adolesc Psychiatry 2002; 11:92-95.
59. Grant JE, Kim SW. An open-labeled study of naltrexone in the treatment of kleptomania, J Clin Psychiatry 2002; 63:349-356.