Published online: 2019-09-26

Kleptomania: Beyond serotonin

Sir,

Kleptomania has been described as an inability to refrain from the urge to steal things for reasons other than personal use or financial gain. Decades of research show that both dopamine and serotonin are the primary neurotransmitters involved in the pathogenesis and management of kleptomania, respectively. Selective serotonin reuptake inhibitors (SSRIs) have been used as first-line agents for treating kleptomania. On the contrary, cases of kleptomania had been paradoxically induced in three adults who were started on SSRIs for depression.[1] A recent Indian paper also reports a similar manifestation while on fluvoxamine treatment for obsessive compulsive disorder.[2] Restlessness and impulsivity have been recognized as symptoms of behavioral activation during initiation of treatment with SSRIs. Emergence of kleptomania post SSRI treatment may be partly explained by the same phenomenon in which there could be effective depletion of synaptic serotonin post acute administration of SSRIs through serotonin-mediated action on its auto-receptor. While the role of serotonin is murky, the role of dopamine is increasingly getting clearer especially in neurological disorders. Kleptomania is seen as an emergent side effect of the use of dopamine agonists in Parkinson's disease (PD). Other impulse control disorders (ICDs) like pathological gambling, compulsive shopping, compulsive eating and hypersexuality have also been reported with the use of dopamine agonists. This phenomenon is explained by the overdose theory.[3] In PD, ventral striatal dopamine is preserved relative to dorsal striatal activity; thus, dopaminergic treatment titrated to alleviate motor dorsal striatal deficiencies may result in an "over-dosing" in ventral cortico-striatal cognitive and limbic pathways. And as such there is preliminary evidence for the benefit of atypical antipsychotics for treating impulse regulation disorders.[4]

At the same time, serotonergic and dopaminergic systems are not mutually exclusive. Could an alteration in dopamine levels post SSRI initiation explain the emergence of kleptomania in rare cases? SSRIs are known to sensitize dopamine (D2) receptors.[5] On the other hand, could the apparent effectiveness of SSRIs in kleptomania be because of their dopaminergic modulation rather than their primary effect on the serotonergic system?

Kleptomania shares certain distinct features with substance use disorders, unlike other impulse control disorders like intermittent explosive disorder. This possible association between the two has resulted in naltrexone being tried as a treatment strategy for kleptomania with favorable results.[6] Further supportive evidence can be obtained from the rare finding of new onset alcohol dependence linked to treatment with SSRI.[7] This intriguing phenomenon can be considered similar to the earlier mentioned reports of emergence of kleptomania after taking SSRIs. Probably the mechanism, although still speculative, may be similar in both phenomena. So along with the erstwhile serotonin and dopamine, treatment of kleptomania is further complicated with the involvement of opioid and glutamatergic system.[8]

Kleptomania is often been found comorbid with other psychiatric disorders including depression, addiction and personality disorders. Various structural brain lesions have been associated with kleptomania including head trauma.[9] Decreased white matter microstructural integrity in the inferior frontal brain region has also been identified in patients with kleptomania.[10] Functional anatomy of impulse control disorders including kleptomania has recently been reviewed.[11]

While medications that affect the serotonergic system have been most widely studied for the treatment of kleptomania, their clinical effects have been modest or inconsistent or even paradoxical. Medications that affect dopaminergic neurotransmission have received less research attention. Kleptomania is the final common manifestation of various underlying pathogenic mechanisms which calls for rationalization of treatment based on co-morbid symptoms and personality traits rather than blanket use of one agent. Extension of this suggestion to other impulse control disorders requires renewed research interest and further elaboration. Developments in neurobiology and pharmaco-genetics, coupled with newer pharmacological models involving the opioid and glutamate systems will surely further our understanding of the pathophysiology and pharmacotherapy of kleptomania.

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	DOI: 10.4103/0976-3147.145244