Kleptomania After Head Trauma

Two Case Reports and Combination Treatment Strategies

Anat Aizer, MD, Katherine Lowengrub, MD, and Pinhas N. Dannon, MD

Abstract: The purpose of this paper is to add to the growing number of reports about kleptomania occurring in relation to brain injury as well as to present the authors' findings regarding treatment strategies. The authors present two case reports of patients who developed the new onset of kleptomania after closed head trauma. Both patients had comorbid psychiatric symptoms associated with the kleptomania. Antidepressant monotherapy was not beneficial in reducing kleptomania in either patient. Kleptomanic behavior was successfully treated in both patients, however, through combination treatment using an antidepressant agent together with adjunctive cognitive behavioral therapy or adjunctive naltrexone. In one patient, single photon emission tomography showed a perfusion deficit in the left temporal lobe. Various hypotheses regarding this finding and the etiopathology of kleptomania are discussed. Review of current work in the field suggests that kleptomania is a heterogeneous disorder that shares features of both impulse and addiction disorders as well as affective spectrum disorders.

Key Words: kleptomania, head trauma, neuroimaging, treatment strategies

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Kleptomania is currently classified in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) of the American Psychiatric Association as an impulse control disorder not elsewhere classified,[1] and in the International Classification of Diseases (ICD) of the World Health Organization it is classified under the heading of habit and impulse disorders[2] together with pathologic gambling, pyromania, and trichotillomania. Both classification systems are based on the core symptom, which is the recurrent failure to resist the impulse to steal despite the ego-dystonic nature of the impulse and awareness of the wrongfulness of the act. The kleptomanic individual differs from an ordinary thief in that the act of stealing is performed to achieve emotional relaxation and not personal gain.

From The Rehovot Community Mental Health & Rehabilitation Center, Tel Aviv University, Rehovot Israel.

Reprints: Pinhas N. Dannon, MD, The Rehovot Community Mental Health & Rehabilitation Center, Tel Aviv University, Remez St 80, Rehovot 76449 Israel (e-mail: pinhasd@post.tau.ac.il).

Kleptomania is presumed to be rare, and most studies have suggested that women comprise about 66% of the reported cases. The symptoms may typically begin during adolescence or young adulthood, although late diagnosis after many years of suffering is the rule. Kleptomanic patients may experience repeated arrests and legal problems with resulting loss of self-esteem and impairment in social functioning.[3]

The specific etiopathology of kleptomania is not known. Serotonin dysregulation has been postulated as a contributory mechanism in the pathophysiology of impulse control disorders. Serotonergic abnormalities, for example, have been well documented in patients with pathologic gambling (PG) and trichotillomania.[4,5] The serotonin hypothesis of impulse disorders is consistent with findings in kleptomania comorbidity studies. It has been demonstrated that kleptomania is associated with high rates of psychiatric comorbidity, especially major depression.[6–9] High rates of mood and anxiety disorders as well as substance use have been consistently demonstrated in first-degree relatives.[8,9] Hudson and Pope[10] have introduced the term "affective spectrum disorders" to refer to kleptomania and other disorders of impulse control that have a high comorbidity with affective disorders. McElroy et al[11] and Hollander and Wong[12] suggested the syndrome is associated with strong obsessive and compulsive features and therefore may be considered part of the obsessive–compulsive spectrum disorders. According to the theory of the obsessive-compulsive spectrum disorders, kleptomania is described as belonging to a cluster of impulsive-style disorders that also includes PG, trichotillomania, compulsive buying, and self-injurious behavior. These comorbidity studies and phenomenologic studies, however, have not consistently supported this hypothesis.

Given the evidence of serotonin dysregulation in both the impulse control disorders and the obsessive–compulsive spectrum disorders, pharmacologic intervention for kleptomania has centered on the use of selective serotonin reuptake inhibitors (SSRIs). There have been several placebo-controlled trials of SSRIs in the treatment of PG that have shown a decrease in gambling behavior.[13] The evidence for the effectiveness of SSRIs in impulse control disorders is only preliminary, however, with many double-blind trials (particularly for compulsive buying and PG) some failed to demonstrate their effec-

tiveness. A small series of case reports have shown SSRIs to be effective in treating kleptomania.[14,15]

Advances in the pharmacologic treatment of drug addiction and related disorders of impulse control have led to new insights into treatment strategies for kleptomania. According to the opioid/dopamine hypothesis, the mu-opioid system is involved in the processing of reward, pleasure, and pain, and is mediated by dopamine (DA) neurons in the mesolimbic pathway. The mu-opioid receptor antagonist naltrexone has been effective in treating urge-driven behaviors in the field of addictive disorders.[16] Interestingly, preliminary studies have shown naltrexone to be effective in controlling urges associated with PG.[17] Recent open-label studies of naltrexone in the treatment of kleptomania demonstrated that naltrexone reduced urges to steal and kleptomanic behavior.[18,19] Pharmacologic studies, therefore, suggest that kleptomania is a heterogeneous disorder that shares features of impulse control disorders, addictive disorders, and affective spectrum disorders.

A review of the literature shows that there have been several case reports of kleptomania associated with organic brain disease. Kleptomania has been described in relation to frontal lobe dysfunction,[20] dementia,[21] presenile cortical atrophy,[22] a right parietal tumor,[23] subarachnoid hemorrhage with a consecutive basal forebrain lesion,[24] and metabolic disturbance secondary to an insulinoma.[25] We present 2 patients in whom kleptomania developed after head trauma.

CASE REPORT 1

Mr. L., a 43-year-old former army major who was married with two children, presented to our clinic for psychiatric evaluation 2 years after sustaining head trauma in an automobile accident. Two years previously, while on the highway, he lost control of his car and collided into a road barrier. At the time of the impact, he sustained blunt trauma to his frontotemporal area and was unconscious for 5 to 10 minutes. He was hospitalized for 48 hours, during which neurologic examination including EEG and head CT scan were reported as within normal limits.

Soon after the accident, the patient started to show bizarre behavior at work. Mr L. behaved aggressively to his colleagues and was found to be "very harsh" to the soldiers. In this same period, he suffered from headaches, insomnia, fatigue, and dizziness, together with complaints of impaired concentration and memory. The patient was referred to a neurologist, and the results of all neurologic tests including a brain MRI were within normal limits. Due to his continued low performance at work, he was discharged from the army and received an early retirement plan that gave him economic stability.

Soon after discharge from the army, his family reported the onset of stealing. He was apprehended by a local storeowner while in the act of stealing a colorful shiny magnet and again by another storeowner when he tried to steal a shiny toy star. The patient said that he intermittently felt an urge to steal "shiny objects" and was unable to control this urge. He compared his behavior with a "crow" that steals shiny things. Afterward, he kept the stolen material in his closet. He said that feelings of guilt prevented him from using the stolen objects. He also reported feeling a rapid heart beat and a sense of pleasure during the act of stealing.

On referral to our clinic, the patient received a complete psychiatric evaluation. Based on symptoms of insomnia, restlessness, suicidal thoughts, anhedonia, and depressed mood, the patient was diagnosed as suffering from major depression. The patient was also given diagnoses of personality change due to head trauma and kleptomania according to DSM-IV and ICD-10 diagnostic criteria. The patient was treated with citalopram 5 mg/day, and over a period of 2 weeks, the dosage was gradually increased up to 40 mg/day. Adjunctive clonazepam up to 1 mg/day was given on an as-needed basis. After 8 weeks of treatment, the patient and his family reported a gradual improvement in mood and anxiety symptoms but he was again caught in the act of stealing a colorful glass object. The patient did not accept the higher dosage of citalopram treatment or any other drug augmentation, but agreed to receive cognitive behavioral therapy (CBT) on a weekly basis. After 3 months of combined treatment with citalopram and CBT, he reported a full remission in mood symptoms and kleptomanic behavior. His family also reported an improvement in his aggressive behavior. At the 14-month follow-up visit, he was asymptomatic.

CASE REPORT 2

Mr. N., a 34-year-old Cyprus resident, married with five children, was sent to Israel by his physician for a psychiatric consultation. Thirteen months previously, he had fallen from the first floor level (height of 3–4 m) while changing a broken window. According to anamnesis, the patient suffered loss of consciousness immediately after the fall and remained unconscious for 3 days while in the intensive care unit. A CT scan of the brain did not show a hemorrhage, but brain MRI showed a contusion in the left temporal lobe. The patient was referred to the orthopedic department for the treatment of multiple fractures. After discharge to home, he started intensive physical therapy, and the physical therapist noticed that Mr. N. had stolen small change from his wallet. Over the ensuing months, the patient was caught multiple times in the act of stealing small amounts of money from different people and local stores. This behavior caused embarrassment to his family because Mr. N. had previously been a successful businessman. The patient did not use the stolen money for his own benefit.

The patient was referred to a local psychiatrist because of the stealing behavior and received multiple drug treatments that included up to 800 mg/day carbamazepine, 1000 mg/day valproic acid, 20 mg/day paroxetine, 150 mg/day venlafaxine combined with 900 mg/day lithium, and up to 2 mg/day risperidone without success. The kleptomanic behavior contin-

ued, and the patient stopped all medications because of the various side effects of the drugs. He remained without psychotropic medication for 3 months before he came to Israel for psychiatric consultation.

At the time of his psychiatric evaluation at our clinic, he complained about his impulsive stealing behavior. He explained his behavior as an impulse that he could not control and reported guilt feelings after the act of stealing. He also complained of depressed mood, loss of energy, decreased appetite, and insomnia, all of which had been present for the past 6 months. He noted that the venlafaxine had helped him with the mood symptoms but not with the kleptomanic behavior. The patient completed a brain imaging workup that included single photon emission computed tomography (SPECT) of the brain with 825 MBq Tc HMPAO intravenous administration, and results showed decreased perfusion involving the left temporal lobe (Fig. 1). After our evaluation, the patient was diagnosed with kleptomania and mood disorder due to head trauma. The patient received venlafaxine in the first phase of the treatment and the dosage was increased up to 150 mg/day in 12 days. After 8 weeks of treatment, the patient and his family reported a remission in depression but kleptomanic behavior remained. A dose of 25 mg naltrexone was added to the drug regimen, and the dosage was gradually titrated up to 100 mg/day. After 12 weeks of naltrexone–venlafaxine treatment, the patient reported a full response. At the first year follow-up visit, he and his family reported continued full remission.

DISCUSSION

Closed head injury is known to be associated with psychiatric disturbances including mood disturbance, apathy, emotional lability, and impulsive and aggressive behavior. We have described two cases of kleptomania that followed closed head trauma, and the successful use antidepressants in combination with CBT or naltrexone.

In the first case, the patient developed mild intellectual impairment accompanied by a personality change characterized by aggressive behavior and impulsivity. The patient subsequently developed the new onset of kleptomania and depressed mood together with neurovegetative symptoms. Despite the fact that brain MRI showed no abnormalities, we hypothesize that the patient suffered from frontal lobe injury based on the clinical picture of personality change with poor impulse control. Frontal lobe lesions remain the best known example of the effects of regional cerebral damage on personality and are presumed to play a causative role in the development of the emotional lability and impulsivity.[26] Furthermore, lesions in the frontal and ventromedial prefrontal lobes have been shown to be associated with faulty decision making and poor planning, and thus have been indirectly linked to addictive and impulsive disorders.[27] We hypothesize, therefore, that a frontal lobe abnormality may have played a role in the onset of both personality changes and comorbid kleptomania in our first patient.

Interestingly, adjunctive CBT appears to be an element that was successful in this patient. The CBT used with this patient consisted of 3 components: (1) cognitive restructuring to correct irrational and dysfunctional beliefs that precede impulsive behavior, (2) problem-solving skills aimed at generating alternative responses to stress, and (3) relapse prevention in which the patient was taught to identify and avoid high-risk situations. Although, to our knowledge, there have been no reported cases of CBT in the treatment of kleptomania, CBT has been shown to have efficacy in the treatment of PG,[28] as well as in major depressive disorder and a range of anxiety disorders.

In our second patient, kleptomania developed after a severe closed head injury in which the patient was unconscious for 3 days. This patient did not exhibit signs of a personality change, but instead complained of depressed mood and neurovegetative symptoms. MRI showed a contusion of the left temporal lobe, and SPECT showed a perfusion deficit in the left temporal lobe.

Temporal lobe lesions are not typically described in the pathophysiology of addictive and impulsive behavior. It is not surprising, however, that our second patient showed evidence of temporal lobe damage, for in head trauma, the temporal lobes are commonly concussed against the bony confines of the middle fossa. This trauma causes a physiologic disruption of hippocampal function, which in turn disturbs memory storage and retrieval.[29] Although temporal lobe lesions are known to be associated with memory deficits, there does not appear to be a form of personality change that is specific for temporal lobe dysfunction.

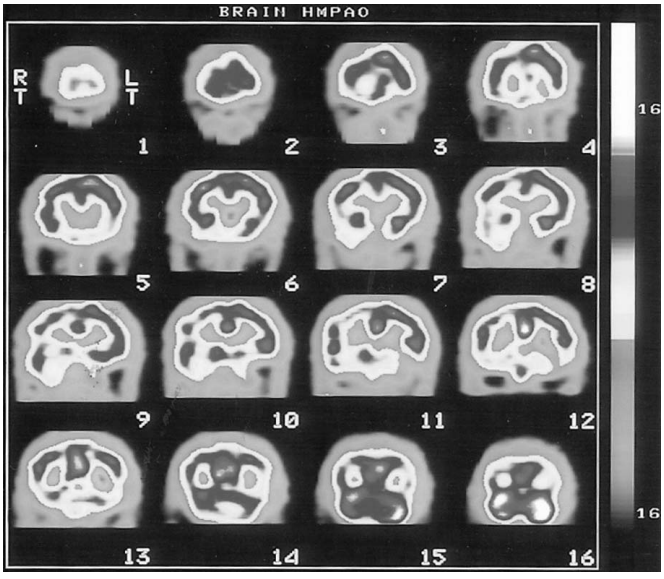

FIGURE 1. Case Two Spect Study.

There are several hypotheses regarding how temporal lobe damage may be associated with disorders of impulse control. Over 40 years ago, evidence from animal experimental work demonstrated that disturbances in the limbic system are associated with aggressive behavior.[30] The limbic system includes regions of the cerebral hemisphere, diencephalon, and midbrain that collectively mediate emotion and the behavioral expression of emotion. Important limbic structures within the cerebral hemisphere include the hippocampal formation and the amygdaloid complex. The hippocampal formation, which is located in the medial temporal lobe, receives afferent input from the cingulate gyrus and sends efferent connections through the hypothalamus and back to the cingulate gyrus. This circuit is thought to play a critical role in memory, the processing of motivational information, and the regulation of emotion. The amygdaloid complex, located in the rostral temporal lobe, has important efferent projections to the ventral medial nucleus (VMN). The VMN mediates diverse functions associated with appetitive behaviors. Lesions in the VMN have been associated with impulsivity and an increase in stimulus-driven behavior.[27]

Neurobiologic models of drug addiction have implicated the cingulate gyrus, VMN, and related limbic circuits, with the experience of craving and the loss of self-directed behaviors leading to compulsive drug administration.[27] In addition, both the hippocampus and the amygdala, with their complex interneuronal connections, are thought to play a role in the processing of pleasure and reward.[31,32] Drawing from the drug addiction model, therefore, we hypothesize that lesions in the limbic structures of the temporal lobe may either trigger or exacerbate the craving and binging behavior seen in kleptomania and other impulse control disorders.

The kleptomanic behavior in our second patient responded dramatically to combination therapy with venlafaxine and naltrexone. Venlafaxine is a serotonin and noradrenaline reuptake inhibitor that has both noradrenergic and serotonergic properties. In this patient, monotherapy with venlafaxine was helpful in treating depressive symptoms but was ineffective in treating the kleptomanic symptoms. We hypothesize that the use of adjunctive naltrexone helped to reduce the sense of pleasure associated with kleptomanic behavior in this patient. Preliminary studies showing the efficacy of naltrexone in the treatment of kleptomania[15,16] lend support to the idea of a temporal lobe abnormality causing kleptomania, for naltrexone is thought to exert it therapeutic effect through the modulation of γ-aminobutyric acid neuronal input to DA neurons in the limbic structures of the temporal lobe.[13]

Anatomically, the hippocampus is a structure with dense cortical folding, and this is thought to be indicative of the extensive neuronal connections with cortical areas. We hypothesize that hippocampal lesions may cause abnormalities of neuronal circuitry between cortical regions. It is possible that the abnormal neuronal circuitry may, in turn, be a causative factor in the pathology of kleptomania and other disorders of impulse control. This hypothesis would be consistent with the heterogeneity and high psychiatric comorbidity seen in impulse control disorders.

Based on results of the SPECT study performed on our patient, we offer a hypothesis as to how a lesion in the limbic structures of the temporal lobe may play a causative role in this disorder. The validity of our hypothesis is significantly limited by the fact that it is based on the SPECT study of one patient. Furthermore, we cannot rule out the fact that the lesion may have existed a priori and may be an incidental finding because there are no perfusion scans prior to the trauma for comparison. A further limitation of our study is the fact that no A-B-A design was used to assess the benefit of our combination treatment strategies. Because the patients were not taken off the medication to see if symptoms returned, there are possible confounders in assessing what was beneficial. Also, the kleptomania diagnosis was made according to the authors' clinical judgment and experience.

Kleptomania is an impulse control disorder with an underlying pathology that remains to be elucidated. Serotonin systems have been implicated in kleptomania and other disorders of impulse control, but the evidence for the effectiveness of SSRIs is unclear. We note that kleptomanic symptoms did not respond to monotherapy with an antidepressant agent in either of our patients. This finding supports the idea presented in the relevant literature that antidepressant monotherapy is ineffective in the treatment of impulse control disorders. Our successful use of combination therapy involving antidepressants (to reduce impulsivity) in combination with naltrexone (to reduce activity on the reward system) or CBT (to enhance self-control) may represent a direction for the future treatment of kleptomania.

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