

Case Report: Kleptomania and Other Psychiatric Symptoms After Carbon Monoxide Intoxication

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Abstract

Carbon monoxide (CO) intoxication is usually a serious condition, which can result in neurological disturbances or death. In some patients with CO intoxication, but not usually, a biphasic pattern can be seen. In this condition, after antitoxic treatment, patients may completely recover and after a short recovery period, neurological and/or psychiatric symptoms appear again. This condition is known as delayed encephalopathy and its occurrence rate is between 0.06% and 11.8%. Herein, we report a case with delayed encephalopathy after CO intoxication, which began with neurological symptoms and continued with obsessive-compulsive disorder, depression, kleptomania, and psychotic disorder. The 41-year-old female patient had no psychiatric or neurological symptoms or disorders prior to CO intoxication. Increased signal intensity changes in the basal region of the left temporal lobe (including the cortex and subcortical white matter), globus pallidus (bilateral), and cerebellar cortical and subcortical white matter (bilaterally symmetrical) was detected on axial T2-weighted magnetic resonance imaging (MRI). In addition, there were atrophic changes in both cerebellar hemispheres. To the best of our knowledge, this is the first case of kleptomania described after CO intoxication in the literature. We discuss the organic etiology of kleptomania and the other psychiatric symptoms of this patient in the light of recent research. We concluded that the kleptomania seen in this patient was related to concurrent lesions in the temporal lobe and globus pallidus; in other words, her kleptomania may have been related to dysfunction simultaneously seen in both the temporolimbic and frontal-subcortical circuits.

Key Words: Carbon monoxide poisoning, kleptomania, globus pallidus, frontal-subcortical circuits

INTRODUCTION

Carbon monoxide intoxication is a serious condition, which can result in neurological disturbances or death. Psychiatric and neurological symptoms include speech disorders, delirium, epileptic seizures, Parkinsonism, agnosia, ataxia, apraxia, and amnesic disturbances (Krigman and Boulding, 1983; Vlerge et al., 1989). In 40% of the cases, more permanent changes, such as moderate amnesic disorders and personality changes were reported. (Jefferson, 1976). Behavioral problems, irritability, hostility, loss of interest, and anhedonia are also frequent in these patients (Lugaresi et al., 1990; Myers et al., 1985). Neurological and psychiatric symptoms in carbon monoxide (CO) intoxication are generally dependent on basal ganglions (especially the globus pallidus), frontal lobe, and cortical periventricular white matter (Deckel, 1994). In some patients with CO intoxication, but not usually, a biphasic pattern can be seen. In this condition,

after antitoxic treatment, patients may fully recover, and following a short recovery period, neurological and/or psychiatric symptoms might reappear (Çelebisoy and Aydemir, 1996; Vlerge et al., 1989). While symptoms are persistent in some patients, other patients fully recover. This condition is called delayed encephalopathy and its recurrence rate is between 0.06%-11.8%; however, this rate can increase up to 11.8% in inpatients (Choi, 1983; Schaumburg and Spencer, 1986).

In delayed encephalopathy, after the acute phase, psychiatric symptoms, such as periodic depression (due to basal ganglion, white matter, and prefrontal cortex lesions), major depression, social withdrawal, personality change, apathy, perception disturbances, obsessive-compulsive disorder, catatonia, and psychosis (Cummings and Cunningham, 1992; Jefferson, 1976; Laplane et al., 1989; Olson, 1984), as well as neurological symptoms, such as headaches, cerebral ataxia, tonus increase, mild cognitive

deficiencies, and parkinsonism appear (Schaumburg and Spencer, 1986). In this article, we present a case of delayed encephalopathy following CO intoxication, which began with neurological signs then continued with obsessive-compulsive disorder, depression, kleptomania, and psychotic disorder.

Case

A 41-year-old married woman was followed-up in the anesthesia intensive care unit for 6 days due to CO poisoning, from a hot water heater in March 2004, where she received hyperbaric oxygen treatment. She was discharged after a full recovery and remained free of symptoms for approximately one month. In April 2004, she started to experience forgetfulness, nervousness, and disordered speech, and with an increase of these symptoms she presented to the neurology outpatient clinic at our university hospital with the diagnosis of minimal cognitive disorder at which time piracetam and pentoxifyllin treatments were initiated. Magnetic resonance imaging (MRI) of the patient's brain revealed secondary ischemic gliotic atrophic changes due to CO intoxication. An anxious mood and irritability were observed during the requested psychiatric consultation; however, a specific psychiatric diagnosis was not considered. In August 2004, she presented to a psychiatrist with suspected obsessions, and interrogation and control compulsions. She was diagnosed with obsessive-compulsive disorder (OCD) and 20 mg/day citalopram and 1 mg/day risperidone were initiated. Although the patient did not use the medications systematically, her symptoms completely disappeared within 2-3 months. After a 4-5-month asymptomatic period, the patient began to steal various objects from shops. The patient reported that she was ashamed of her stealing behavior, but could not control the impulse to steal. She also reported that she generally stole objects that she did not need and would not use, and that this situation was not related to financial difficulties. Moreover, the patient started to suspect that her husband was being unfaithful because he began arriving home late at night and she would spend the whole day struggling with suspicious thoughts about him. After a short time she became convinced that her husband was being unfaithful despite evidence to the contrary and her husband's reassurance. The patient became increasingly irritable and nervous and she started to fight with family members and began to threaten suicide. In June 2005, 75-mg/day sertraline treatment was initiated by her previous doctor. Although her depressive symptoms decreased, other symptoms were not alleviated. She was referred to the psychiatric outpatient

clinic due to delusional thoughts and was subsequently hospitalized in the inpatient psychiatric department for differential diagnosis and treatment.

The psychiatric assessment conducted during her hospitalization revealed she had difficulty concentrating. Spontaneous, short-term, and long-term memory was normal, but it was observed that she was experiencing mild forgetfulness and distraction. Anxious and depressive mood, difficulty in sleeping, intense hostile feelings toward her husband, irritability, delusions regarding her husband's infidelity, attempts to confirm the delusional thinking, and impulsive stealing behavior (kleptomania) were also observed. Her judgment, reality testing, and abstract thinking were sufficient, while her insight was evaluated as insufficient. The patient's birth was normal and on time, her motor and mental development were also normal, and her childhood and youth were non-problematic. She had no history of mental or physical illness, surgery, or seizures and no family psychiatric history.

There were no pathological findings in the blood count, blood sedimentation speed, and urine assessments. Thyroid function tests, vitamin B12 and folic acid levels, and EEG were all normal. Neurological examination revealed no pathological findings besides forgetfulness. Her Hamilton Depression Rating Scale (HAM-D) score was 10. The mini mental state exam score conducted to evaluate her cognitive functioning was 28/30. MRI examination of her brain revealed ischemic and necrotic lesions. Increased signal intensity changes in the basal region of the left temporal lobe (including the cortex and subcortical white matter), globus pallidus (bilateral), and bilateral cerebral cortical and subcortical white matter was detected on axial T2-weighted MRI. In addition, there were atrophic changes in both cerebral hemispheres. In the MRI findings, focal parenchymal signal increase, which included the cortex and the subcortical white matter, was detected in the left temporal lobe (Figure 1) In addition, signal increase in the bilateral globus pallidus (Figure 2) and atrophy in both cerebral hemispheres was also detected. In the light of these findings, as there was evidence that the depressive and psychotic symptoms were due to the direct physical effects of CO intoxication, the following DSM-IV diagnoses were considered: mood disorder due to CO intoxication, presented with depressive characteristics; psychotic disorder due to CO intoxication, presented with delusions. In addition, based on the stealing behavior of the patient, she was also diagnosed with kleptomania, according to DSM-IV (American Psychological Association, 1994).

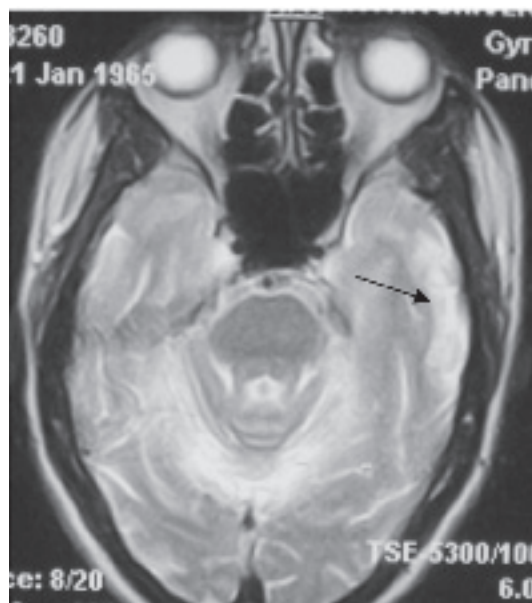


Figure I. Focal parenchymal signal increase, which included the cortex and the subcortical white matter, was detected in the left temporal lobe with MRI (compatible with necrosis).

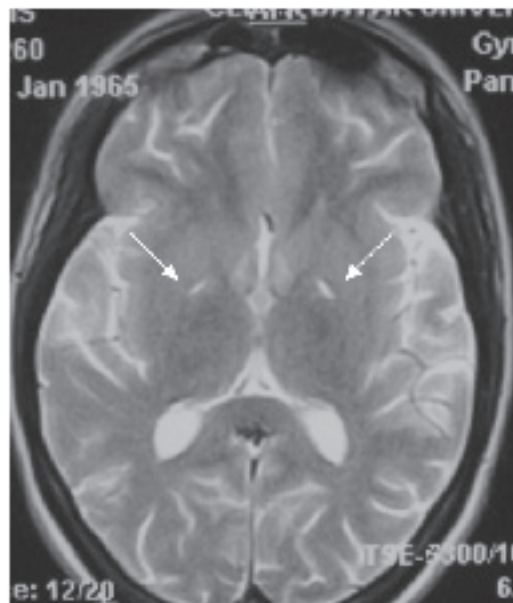


Figure II. Signal increase in the bilateral globus pallidus in MRI (compatible with necrosis).

It was observed that an anxious mood was most prominent in the patient. Her treatment included 75 mg/day sertraline and 2 mg/day risperidone during her hospitalization. The dosage of risperidone was gradually increased to 4 mg/day, and during follow-up, it was observed that although the delusions persisted, she no longer had the symptoms of depression and anxiety; therefore, sertraline treatment was stopped. On the 30th day of hospitalization, the prolactin level of the patient was 185.8 mg/ml and she had galactorrhea. Risperidone was gradually stopped and quetiapine treatment was initiated. When the dosage of quetiapine reached 600 mg/day, the symptoms disappeared and the dosage of quetiapine was stabilized at 600 mg/day. The relatives of the patient reported a marked recovery during her weekend stays, which was also confirmed in the hospital. The patient was discharged from the hospital to be followed-up as an outpatient at the psychosis outpatient clinic. During the follow-up, there was no kleptomania behavior, delusional thinking decreased, and she displayed marked memory deficiency; therefore, her quetiapine dosage was decreased to 400 mg/day and her symptoms remained stable.

DISCUSSION

When compared to the other conditions that result in anoxia in CO intoxication, delayed encephalopathy is more common (Choi, 1983). In delayed encephalopathy, an asymptomatic period follows the acute phase,

which proceeds with consciousness disturbance (Chang et al., 1992; Zagami et al., 1993). The appearance of late neurological symptoms is reported to occur between 4 and 18 days, and 2 and 40 days in various publications (Deckel, 1994; Jibiki et al., 1991; Schaumburg and Spencer, 1986). Generally, a good prognosis is reported for delayed encephalopathy. Choi (1983) reported that 75% of delayed encephalopathy cases fully recovered within one year. In our case, a biphasic phase was also observed. Approximately 1 month following the acute phase and recovery, neurological deficits, such as speech disorder and memory dysfunction appeared, and in the following months other psychiatric symptoms appeared.

CO intoxication results in various structural deficiencies in the brain tissue. Bilateral ischemic lesions and necrosis in the grey matter, especially in the globus pallidus, are the most frequently seen changes (Chang et al., 1992; Krigman and Boulding, 1983). Similar necrotic lesions can be seen in the Purkinje cells of the cerebral cortex, in the dentate nucleus, and in the cortex (Nardizzi, 1979). In computerized brain topographies and MRI scans of patients with CO intoxication, bilateral necrotic areas in the frontal white matter and centrum semiovale are typically observed, in addition to bilateral globus pallidus lesions (necrosis) (Chang et al., 1992; Choi, 1983; Vleregge et al., 1989). Similarly, the most prominent MRI finding in the presented case was hyperintense lesions (necrotic) in the globus pallidus on the periventricular basal ganglion level (Figure 2).

In CO intoxication, EEG changes due to functional deficiencies in the brain can be observed. It was reported that slow wave activity or thorn waves that show lateralization are generally observed in EEGs (Neufeld et al., 1981; Karakurum et al., 2005); however, these changes appear mostly during the acute phase and disappear in the following months (Gorman et al., 2005; Neufeld et al., 1981). Denays et al. (1994) examined 12 patients and showed that EEGs were normal in 9; but when EEG mapping and SPECT methods were used, one- or two-sided regional deficiencies were found in 8 of these patients. It was not possible to see the acute phase EEG readings of the presented case. The reason for not finding any pathological findings in the EEG taken during her hospitalization might have been the amount of time that passed after the intoxication and being unable to detect the changes in the deeper structures of the brain with the particular EEG method used.

Previous publications have reported that decreases in the density of the temporal lobe were associated with the severity of depression (Elderkin-Thompson et al., 2003; Robinson et al., 1999). In addition, it is known that in depressive patients, the density of the globus pallidus decreases and the frequency of pallidal lesions is high, especially in patients with secondary depression, and the risk of depression is high in patients following bilateral pallidotomy (Lacerda et al., 2003; Lauterbach et al., 1997; Green et al., 2002; Merello et al., 2002). In the presented case the observed lesions in the left temporal lobe and globus pallidus (Figures 1 and 2), and the appearance of depressive symptoms after CO intoxication suggests that her depressive disorder was due to these lesions.

The role of basal ganglions in the etiology of psychotic findings is known. In particular, circuits that involve the globus pallidus were suggested to be responsible for psychotic symptoms (Kayahan et al., 2005). Based on the presented patient's history and brain imaging, there was a possibility that the observed psychotic findings may have been due to the damage in the basal ganglions, which is compatible with the findings of Olson (1984) and Lauterbach et al. (1994). In addition, the relationship of obsessive-compulsive symptoms and the limbic system, chiefly the globus pallidus, is known (Kayahan et al., 2005). In the presented case, we thought that the obsessive-compulsive symptoms were related to the lesion in the globus pallidus and because the placement of the lesion was similar to the lesion placement described in cases by Cummings and Cunningham (1992) and Laplane et al. (1989). The lesion in the globus pallidus was also thought to be the cause of the mild memory deficits

in the presented case (Lauterbach et al., 1994; Soukup et al., 1997).

Another psychiatric condition observed in the presented patient during hospitalization was kleptomania. Kleptomania is an impulse control disorder, which is defined as the inability to resist the impulse to steal unneeded objects (Goldman 1992). With its characteristics, the stealing impulse is similar to the obsessions seen in OCD. Similar to compulsions, stealing cannot be controlled and serves to reduce anxiety. The prevalence of OCD and mood disorders in the families of patients with kleptomania is high. Lately, some authors have placed kleptomania on the OCD spectrum (Hollander and Wong, 1995). As with all other impulse control disorders, kleptomania may be related to neurological disorders and brain diseases in some cases (Gossling and Rosin, 1994). There are reports of the appearance of kleptomania in cases involving functional frontal lobe deficiency in which the right frontolimbic area is affected (Nyffeler and Regard, 2001). In a presentation of 2 cases with kleptomania following closed head trauma, left temporal region deficiency was associated with kleptomania in one of the cases (Aizer et al., 2004). The SPECT imaging of a patient with another impulse control disorder, trichotillomania, showed an increase in blood flow in the left temporal region (Özcan et al., 1997). In the light of these findings, the left temporal lobe lesion observed in the presented case might have played a role in the disruption of the patient's impulse control.

On the other hand, it was found that a part of the temporal lobe is attached to the limbic system and that this temporolimbic region is related to pleasure, and sexual and aggressive behaviors, and that biological changes detected in aggressive behaviors (such as decreased 5-HIAA levels of brain spinal cord fluid, desensitized prolactin response in 5-HT agonists, decreased serotonergic functions, and obscure neurological findings) are similar to those observed in many impulsive conditions, including kleptomania (Doksat and Savrun, 2001; Doruk and Uzun, 1997; Kısa et al., 2005). It is more correct to evaluate the brain as a unitary of functions when the etiologies of psychiatric disorders are explained; particular circuits are formed with the connection of frontal cortical regions to other brain regions. Interruption of the frontal temporal cycle results in a behavioral syndrome specific to the cycle. The main structure of all cycles originate in the frontal lobes, project to striatal structures (caudat, putamen, and ventral striatum), project from the striatum to the globus pallidus, and from the substantia nigra to specific thalamic nuclei. The cycles are

closed with the projection from these specific thalamic nuclei back to frontal region. The orbitofrontal cycle plays a role in the processing and integration of limbic impulses due to the orbitofrontal cortex's close relationships with para-limbic structures. Patients with orbitofrontal cortex function disorder display poor impulse control and explosive anger. Again, it has been theorized that OCD symptoms point to increased functioning of orbitofrontal mechanisms. Globus pallidus lesions may give rise to OCD symptoms by decreasing the inhibition on the thalamus and increasing thalamocortical stimulation. It was suggested that the indirect pathway in the orbitofrontal-subcortical cycles of normal individuals appropriately inhibits the thalamus and that this function degenerates in OCD, and that excess stimulation of the thalamus appears (Tural and Önder, 2001). Various studies have demonstrated the influence of the globus pallidus in some disorders associated with poor impulse control (such as attention deficit hyperactivity disorder (ADHD), Tourette's Disorder, OCD) (Kayahan et al., 2005; Peterson et al., 2003). In consideration of the debate concerning that OCD and impulse control disorders may be part of the same spectrum, and thus, may have a common pathophysiological base, it was believed that the kleptomania behavior in the presented case might

have been related to the lesions in the globus pallidus and the temporal lobe.

In the light of all this information and the case findings, including brain imaging, the lesions in the globus pallidus (which caused an increase in thalamocortical activity) and the temporal lobe were considered to have contributed to the observed kleptomania. Another finding that supports these ideas is that kleptomania has not been previously reported in many CO intoxication cases despite the existence of pallidal lesions. To the best of our knowledge, this is the first kleptomania case, described after CO intoxication in the literature. Kleptomania may be related to dysfunction simultaneously seen in both the temporolimbic and frontal-subcortical circuits.

In conclusion, we have presented a case with delayed hypoxic encephalopathy after CO intoxication, which presented with many psychiatric conditions over time. In addition, to the best of our knowledge, this case is the first kleptomania case described after CO intoxication. In the evaluation of clinical condition associated with any brain lesion, the related pathways should be considered in addition to the functions of the independent region.

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