TRANSIENT TIC DISORDER FOLLOWING CARBON MONOXIDE POISONING

Z. ALİOĞLU (1), C. BOZ (1), A. SARI (2), M. AYNACI (3)

(1) Department of Neurology.
(2) Department of Radiology.
(3) Department of Paediatrics, Karadeniz Technical University Medical School, Nöroloji Anabilim Dalı, 61080, Trabzon, Turkey.

SUMMARY

We report a 12-year-old male patient who developed transient motor and vocal tics twelve days after carbon monoxide (CO) poisoning. Cranial magnetic resonance image (MRI) of the patient showed bilateral symmetric hyperintensity in the caudate nucleus and putamen. Tic disorder was successfully treated with haloperidol. Thirty-three months after CO poisoning, the patient was asymptomatic and MRI revealed atrophy in caudate nucleus and putamen. The mechanism of tic disorder in CO intoxication is discussed.

Key words: Tic, Carbon monoxide, MR imaging.

INTRODUCTION

Carbon monoxide (CO) is an odourless, tasteless, colourless and nonirritant gas that frequently causes death or neuropsychiatric abnormalities, including parkinsonism, chorea, Gilles de la Tourette’s syndrome, seizure, focal cortical deficits, dementia, organic affective and personality disorders and psychoses. In most cases, neuropsychiatric sequelae develop acutely following CO intoxication, but some patients recover completely from acute CO poisoning to develop delayed neurologic or psychiatric findings within several days or months [1, 4, 5].

Tics are brief involuntary movements (motor tics) or sounds produced by moving air through mouth, nose or throat (vocal tics) [19]. Tic disorder usually is idiopathic, rarely it may appear as a result of neurologic insult or following neuroleptic medications [7, 10, 19].

In this article, we report the case of a patient who developed transient tic disorder twelve days after CO intoxication that had bilateral diffuse lesions in the putamen and caudate nucleus at MRI.

CASE REPORT

A 12-year-old boy was found unconscious after exposure to a gas heater in a closed bathroom. He was admitted to the emergency room of our hospital. The patient had no history of neuropsychiatric disorders. He was unconscious and responsive to painful stimuli. The patient’s vital signs revealed tachycardia and tachypnea. Routine laboratory examination results were within normal limits. Arterial blood gases revealed mild hypoxia (Pao2 = 60 mmHg). EEG showed bilateral diffuse theta and delta waves. Supplemental oxygen was administered through a nonrebreather mask. Three days later, he was conscious, but his orientation in time and place was impaired. On the fifth day neurologic examination revealed normal mental status and he was discharged. One week after discharge, he developed involuntary movements of the left arm and leg. In the ensuing week, smiling, speech arrest, throat clearing and humming appeared. The patient was readmitted. Neurological examination was notable for smiling, lip smacking, humming, speech arrest and bilateral shoulder shrugging, arm jerking and foot dorsiflexion. At this time, routine laboratory examination and visual evoked potentials were within normal limits. Proton density weighted and T2-weighted magnetic resonance image showed bilateral symmetric hyperintensity in the caudate nucleus and putamen (figure 1). Haloperidol was started (2 mg/day) for treatment of involuntary movements. Seven days later, the severity of involuntary movements had decreased and they completely resolved twenty days after the second admission. Thirty-three months after CO poisoning, the patient was asymptomatic. At this time, cranial
MRI revealed mild atrophy of the caudate nucleus and severe atrophy of the putamen (figure 2).

DISCUSSION

Transient tic disorder is diagnosed by criteria that include onset before age 21, single or multiple motor and/or vocal tics, no history of Tourette’s syndrome or chronic motor or vocal tic disorders, the tics occur many times a day for at least 2 weeks, but no longer than 12 consecutive months [19]. This patient fulfills the criteria for transient tic disorders except for preceding CO poisoning.

Pathophysiology of CO intoxication is complex. CO reacts readily and reversibly with hemoglobin that results in carboxyhemoglobin (CO-Hb). CO-Hb interferes with the transport of oxygen from alveoli to tissues. The resulting hypoxia is the main cause of morbidity and mortality [4, 6]. Furthermore, CO has a direct toxic effect at the cellular level by blocking mitochondrial respiration [6]. Various neurologic sequelae may develop after CO intoxication. Delayed neurologic sequelae (DNS) is characteristic of anoxic encephalopathy after CO poisoning [1]. The pathophysiologic explanation of DNS is speculative. It is uncertain whether secondary hemorrhage or vasogenic edema causes the delayed onset of symptoms [15]. In animals, low concentrations of CO could cause perivascular oxidative changes. This relatively subtle vascular insult may lead to DNS [18]. Extrapyramidal complications of CO intoxication are usually a parkinsonian syndrome or an akinetic-rigid syndrome and occasionally choreothetosis or Tourette’s syndrome [13, 16]. Pulst et al. [13] reported a patient with Tourette’s syndrome due to CO intoxication who had patchy low-density lesions in the basal ganglia at CT. The involvement of the basal ganglia in CO intoxication may be due to the end arterial vascular supply to this structure [5]. Neuropathological and neuroradiological studies in CO intoxication demonstrate bilateral necrosis or ischemia of basal ganglia. These changes are usually seen in pallidum, occasionally they are seen in putamen, substantia nigra, thalamus and caudate [5]. Diffusion-weighted MR [17] and FLAIR sequences [9] may be useful for early identification of the changes of acute CO poisoning.

Neuroanatomical and neurobiological studies demonstrated that involvement of specific cortico-striato-thalamo-cortical circuits leads to expression of Tourette’s syndrome and related disorders. Sensorimotor cortical projections to striatum are organized in the dorsolateral caudate and dorsolateral putamen. Projections from striatum to external segment of globus pallidus and from external segment of globus pallidus to subthalamic nucleus are inhibitory in character. Striatum lesions may cause minimal inhibition of external segment of globus pallidus, therefore it leads to excessive inhibition of subthalamic nucleus. Tourette’s syndrome may be associated with increased inhibition of the subthalamic nucleus [8].

MRI study of Tourette’s syndrome patients showed that mean volumes of the caudate, lenticular and globus pallidus nuclei were smaller than those of controls [12]. Also, bilateral globus pallidus lesions on MRI in a patient with Tourette’s syndrome was reported [2].

When a patient is found unconscious in the bathroom with gas heaters or in a room with poorly functioning heating systems and his/her MRI revealed bilateral basal ganglia lesions, appropriate therapy should be given early to prevent delayed neurologic sequelae. Hyperbaric oxygen therapy hastens the resolution of symptoms in carbon monoxide poisoning [3]. The patient presented in a coma state in our emergency room. Coma is an undisputed indication for hyperbaric oxygen therapy [3]. We administered normobaric oxygen therapy because there was not hyperbaric oxygen treatment in our hospital.

Thirty-three months after CO poisoning, our patient was asymptomatic and MRI revealed atrophy in caudate nucleus and putamen. Clinical and neuroradiological outcome of CO intoxication was reported in a
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study [11]. They stated that 4 patients with both MRI abnormalities and neurological sequelae improved in both clinical features and MRI findings during 3 years. A case with progressive neurological and psychological deterioration that began 17 years after recovery from CO intoxication was also reported [14].

In this paper, we described the unusual association between carbon monoxide intoxication and subsequent transient tic disorders. This report supports that striatum has an important role in the development of tic disorders.

REFERENCES