An Elderly Lady with Parkinsonism

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Citation

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Abstract

Parkinsonism is a syndrome consisting of variable combination of tremor, rigidity, bradykinesia and a characteristic disturbance of gait and posture. It may be caused by many etiological factors and is an important cause of neurological disability in the elderly population. Symptoms of Parkinson's disease are caused by loss of nerve cells in the pigmented substantia nigra pars compata and the locus coeurulus in the mid brain. Loss of dopaminergic cells in the substantia nigra leads to striatal dopamine depletion. This results in reduced thalamic excitation of the motor cortex. The exact etiology of Parkinson's disease is not known but exposure to many toxic substances also can lead to this clinical picture. We are reporting here a case of delayed neurological sequelae of carbon monoxide poisoning, with Parkinsonism as the predominant clinical feature and excellent response to treatment with Levodopa and Bromocriptine.

INTRODUCTION

Carbon monoxide (CO) is a great imitator[1]. The symptoms of Carbon monoxide poisoning are vague and varied and providing insufficient treatment can lead to neurological sequelae. Common source of carbon monoxide is smoke from any type of fire. It produces toxicity by five mechanisms[1].

1. Directly binding carbon monoxide to hemoglobin
2. Shifting the oxygen – hemoglobin dissociation curve
3. Carbon monoxide binding to myoglobin
4. Carbon monoxide binding to cytochrome oxidase system
5. Brain lipid peroxidation

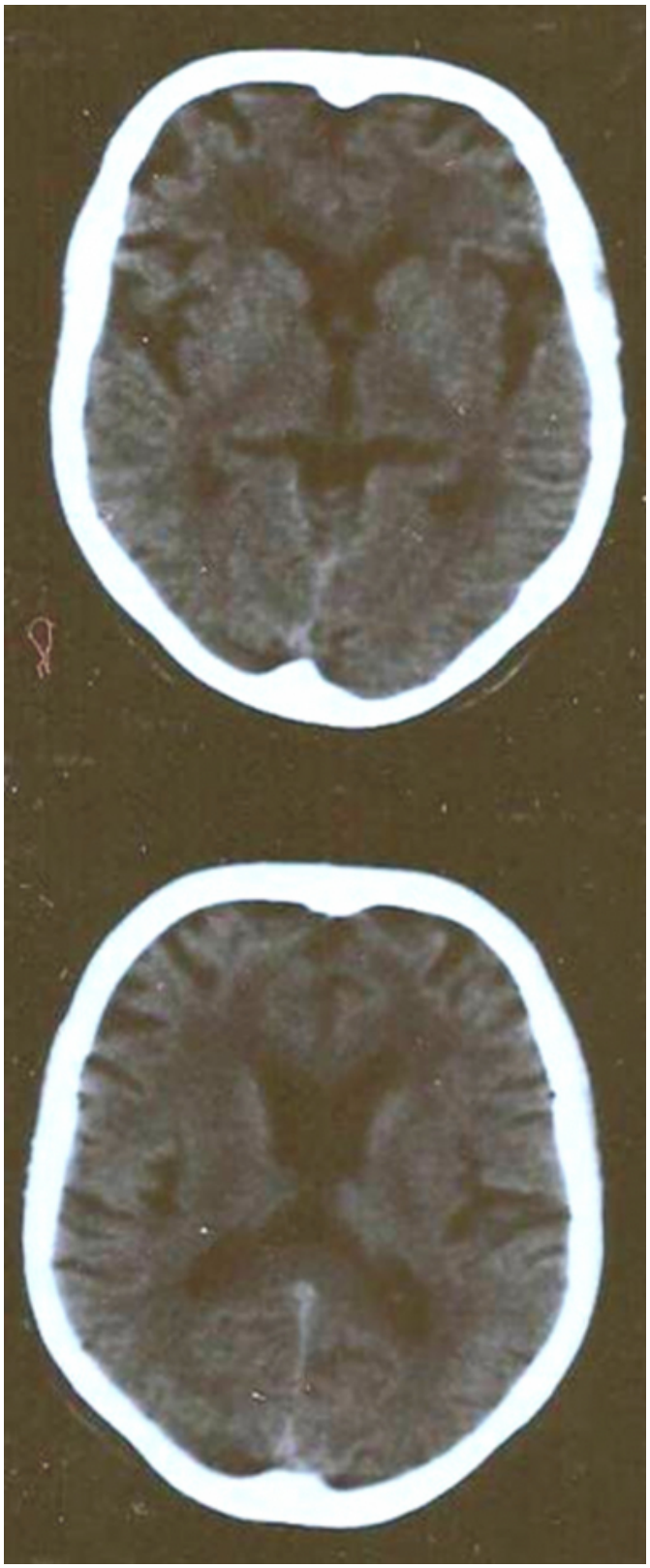
A syndrome of delayed neurological sequelae can occur with appearance of symptoms within 2 to 3 weeks of acute exposure in 10 to 30% cases[2]. These are common in patients with decreased levels of consciousness at the time of emergency room evaluation and those who develop hypotension during hypoxic period[3] and older patients. The most common symptoms are cognitive dysfunction, urinary and fecal incontinence and gait disturbance. Other symptoms include memory loss, disinhibition, disorientation, plasticity, rigidity, aphasia and personality changes. Complete recovery occurs in two third of cases[1]. Parkinsonism is a recognized complication of carbon monoxide poisoning which has been described to occur from 3 to 240 days after exposure[4] but the reported incidents varies widely [5,6,7]. We are reporting here a lady who presented with delayed neurological complications of carbon monoxide poisoning in the form of Parkinsonism and responded well to treatment with levodopa and bromocriptine.

CASE REPORT

A 65-year-old Yemeni lady was admitted with inability to move the limbs, inability to speak and eat since two days. The patient was treated elsewhere for Carbon Monoxide poisoning one month before coming to our hospital. Both she and her husband were sleeping in a closed room when the fire happened. Her husband died during the episode. She did not improve after treatment in the local hospital and sought medical help in Saudi Arabia. She gave past history of diabetes and hypertension which were poorly controlled. On examination the patient was conscious but dysphasic, BP – 150/110 mm Hg. Neurological examination revealed right-sided weakness. CT scan of Brain (Figure 1) was reported normal showing only age-related changes.

Figure 1

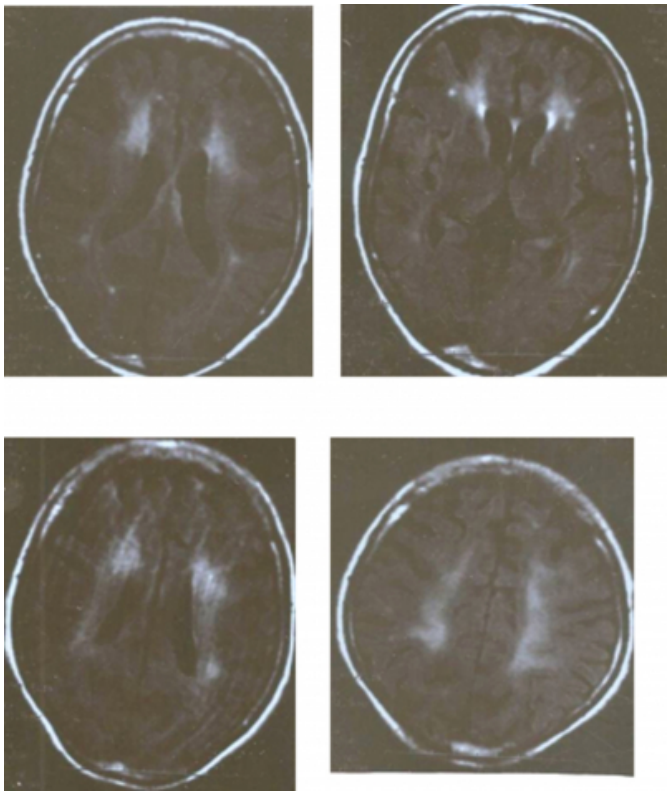
Figure 1: CT scan of Brain showing age-related changes



She was diagnosed as reversible ischemic neurological deficit and discharged after three days on aspirin 100mg once daily. Diabetes and Hypertension were controlled at the time of discharge. She was readmitted three weeks later with history of paucity of movements, disturbed level of consciousness for the past fifteen days with sphincter disturbances. She was unable to move because of bilateral rigidity. On examination the patient was conscious but confused with generalized rigidity. She had slightly exaggerated deep tendon reflexes with flexor plantar responses. The MRI of brain done was reported as showing low intensity signals of right frontal subcortical region at T1 becoming bright at T2 weighted images; sharply demarcated both lentiform nuclei and right putamen, presumably early demyelination consistent with sequel of carbon monoxide poisoning (Figure 2).

Figure 2

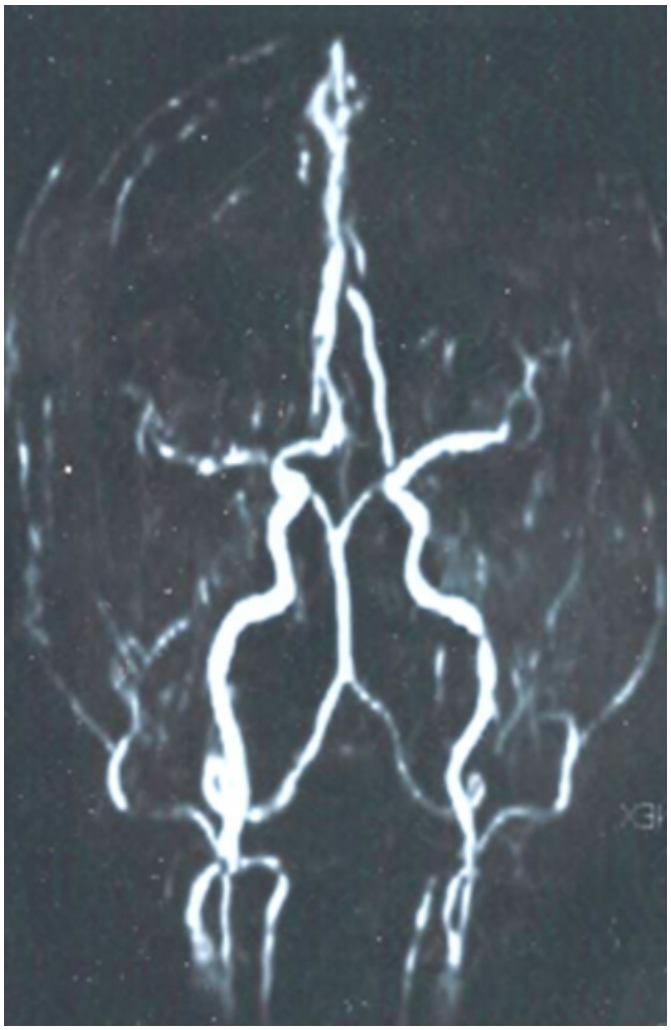
Figure 2: MRI Scan of brain showing areas of demyelination



MRA was reported as normal (Figure 3).

Figure 3

Figure 3: MRA showing normal vessels



Based on the history and physical findings, the patient was diagnosed to have delayed neurological sequelae of Carbon monoxide poisoning with Parkinsonism as the predominant component. She was started on Levodopa (Sinemet) 110mg TID and Bromocriptine 2.5mg OD increased to b.i.d. later.

Medication for diabetes and hypertension were continued. Her mobility increased dramatically and she was able to walk without support and regained sphincter control. Her dysphasia improved and she was able to communicate well. She was discharged after two weeks on Sinemet 110 mg t.i.d and Bromocriptine 2.5 mg b.i.d.

DISCUSSION

Parkinsonism is a well-known complication of carbon monoxide poisoning. Carbon monoxide converts Xanthine dehydrogenase to Xanthine oxidase which reacts with hypoxanthine and eventually produces superoxide oxygen free radical; superoxide formation leads to lipid peroxidation and resultant neuronal damage. This mechanism strongly suggests that carbon monoxide induced brain injury is similar to a post-ischemic reperfusion phenomena [8]. The common symptoms are headache, dizziness, agitation, stupor, seizures and coma, which follow acute poisoning. Additional neurological findings may include hemiparesis, memory deficits, visual impairment, personality changes, emotional lability, loss of reading and writing skills, ataxia and parietal lobe dysfunction. Disorientation, slurred speech, involuntary movements, cortical blindness, akinesia and chorea can occur. Unusual symptoms include mutism, tick disorders, Korsakoff syndrome, peripheral neuritis, dementia, psychosis and Wernicke's aphasia. Long-term psychiatric and neurological sequelae are grossly apparent in 11% of survivors of significant carbon monoxide toxicity and memory impairment occurs in upto 43%[1]. The most common personality change is characterized by emotional lability. Resolution of neurological sequel may take up to two years [1].

In one series the latency before the appearance of Parkinsonism varied from 2 to 26 (median 4) weeks, but Parkinsonism developed within 1 month after an acute insult in the majority of the patients [9]. In our patient initial presentation was about one month after the acute exposure but clear-cut evidence of Parkinsonism appeared about three months from the date of exposure to carbon monoxide.

Parkinsonism has been reported to occur in 9.5% of Carbon monoxide poisoned persons in series of 242 patients[9]. MRI of the brain is more sensitive than CT for detecting abnormalities secondary to carbon monoxide toxicity. Kwon O.Y et al advocate diffusion weighted magnetic resonance imaging(DWMRI) for diagnosis of delayed post-anoxic encephalopathy (DPE)[10]. Pathological features of carbon monoxide poisoning include necrosis of the globus pallidus, lesions of white matter with demyelination, lesion of the “spongy” cerebral cortex and necrotic lesions of the hippocampus[11]. Some of these features were present in the MRI scan of our patient. Initial management in the acute stage of poisoning with hyperbaric oxygen can prevent the incidence of neurological sequel.

Because carbon monoxide poisoning has no pathognomonic signs or symptoms, high level of suspicion, particularly among primary care clinicians and emergency medicine specialist is essential for initial diagnosis. When diagnosis of carbon monoxide poisoning has been established, a detailed neurologic examination and neuro-psychological tests

should be performed. The Carbon Monoxide Neuro Psychological Screening Battery is a frequently used tool that takes 30 minutes to administer and provides a base line for assessing subsequent changes in mental status [12].

Our patient had all the clinical feature of delayed neurological sequelae of carbon monoxide poisoning which were missed at the first presentation but confirmed during the second admission. The MRI scan was suggestive but not confirmatory. The sphincter disturbances on presentation could partly be attributed her immobility and inability to get up and walk freely.

Parkinsonism, which is one of the delayed neurological complications responds to treatment with Levodopa. Although there were some reports that treatment with any drug is not effective [8], some authors have reported good response in patients who were treated with Bromocripitine alone or in combination with Levodopa [13]. Our patient was given combination treatment with Levodopa and Bromocriptine and got dramatic improvement.

SUMMARY

A 65-year-old lady with delayed neurological complications of carbon monoxide poisoning is reported. Parkinsonism was the main presenting feature. She responded very well to treatment with levodopa and bromocriptine.

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References

1. Earl J. Reisdorft, MD, FACEP, Sid M. Shah, MD; Carbon Monoxide Poisoning; EMR text book
2. Norris CR, Trench JM, Hook R. Delayed Carbon Monoxide encephalopathy: Clinical and Research implications. J Clin Psychiatry 1982; 43: 294 - 295.
3. Ginsberg MD. Delayed neurological deterioration following hypoxia. In: Fahn S. Davis JN, Rowland LP, eds. Cerebral Hypoxia and its consequences. Advances in Neurology. New York: Raven Press; 1979:21-44.
4. Armin Ernst, M.D., and Joseph D. Zibrak, M.D., Carbon Monoxide Poisoning, New Eng JM; 339: 22: 1603 - 1608
5. Hart IK, Kennedy PGE, Adams JH, Cunningham NE. Neurological Manifestation of Carbon Monoxide Poisoning. Postgrad Med J 1988; 64: 213-6
6. Sawa GM, Watson CPM, Terbrugge K. Chiu M. Delayed Encephalopathy following carbon monoxide intoxication. Can J Neuol Sci 1981; 8: 77-9
7. Choi IS. Delayed Neurologic sequelae in carbon monoxide intoxication, Arch Neurol 1983; 40:433-5
8. Thorn SR, Dehydrogensase conversion to oxidase and lipid peroxidation in brain after carbon monoxide poisoning. J Appl Physiol 1992; 73: 1584 - 1589.
9. Choi IS; Parkinsonism after carbon monoxide poisoning, European Neurology 2002; 48:30-33
10. O.Y Kwon et-al; delayed post anoxi encephalopathy after carbon monoxide poisoning
11. Horowitz AL, Kaplan R, Sarpel G. Carbon monoxide toxicity: MR imaging in the brain. Radiology 1987; 162:787 - 788
12. Mcssicrs LD, Myers, RAM. A Neuropsychological screening battery for emergency assessment of carbon monoxide poisoned patients. J.Clin Psychol 1991; 47: 675-84.
13. Tack E. de Reuck J. The use of brmocriptine in Parkinsonism after carbon monoxide poisoning; PMD:3690934 [PubMed-indexed for MEDLINE].

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