
Unilateral Diaphragmatic Paralysis Secondary to Carbon Monoxide Poisoning*

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Complications of carbon monoxide poisoning include peripheral neuropathy, which is usually confined to the lower extremities. We report a case of carbon monoxide-associated neuropathy resulting in unilateral diaphragmatic paralysis. Possible mechanisms of injury are described. (Chest 1990; 97:498-99)

Carbon monoxide poisoning accounts for 3,500 deaths per year in the United States, nearly half of the deaths from all poisonings. Nonlethal complications include pulmonary edema, central nervous system dysfunction with psychomotor difficulties, and neuropathy. Peripheral neuropathy has been described but is almost exclusively confined to the lower extremities. We report a case of transient unilateral diaphragmatic paralysis associated with carbon monoxide poisoning.

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Figure 1. Admission chest x-ray film revealing an elevated right hemidiaphragm.

Case Report

A 56-year-old white man was admitted to our hospital after having been found unconscious in his automobile which had been left in operation in a closed garage. He had a previous history of depression with a past medical history of hypertension, non-insulin-dependent diabetes mellitus and a cerebrovascular accident. Initial physical examination revealed that he was afebrile, had spontaneous but labored respirations and was unresponsive to painful stimuli. His initial arterial blood gas values on 5 L of oxygen by nasal cannula revealed mild hypoxemia but no acidosis. His carboxyhemoglobin level, measured approximately 2 h after being found, was 10.2 g percent (Instrumentation Laboratories CO-oximeter model 282). The white blood cell count was 15,200/cu mm with a normal differential cell count. Serum glucose level was 283 mg percent and serum electrolyte values were normal. Chest x-ray film showed an elevated right hemidiaphragm (Fig 1) while a comparison film taken 22 months previously was normal (Fig 2).

He was treated with high flow oxygen. Within 24 h his carboxyhemoglobin level had fallen to 1.3 g percent and his neurologic examination was normal. Fluoroscopy revealed paradoxic motion of the right hemidiaphragm and an abdominal ultrasound did not reveal any mass in the right upper quadrant of his abdomen. On day 4 he was considered medically stable and was transferred to

Figure 2. Normal chest x-ray film of patient taken 22 months prior to admission.
the psychiatric unit. He remained afibrile throughout his hospital stay. Serial chest X-ray films were obtained and by day 12 suggested that the paralysis had resolved (Fig 3). Fluoroscopy confirmed normal mobility of his right hemidiaphragm.

**DISCUSSION**

Carbon monoxide is a tasteless, odorless gas which is produced by incomplete combustion of organic compounds. Ten thousand Americans seek medical attention or lose at least one day of normal activity each year because of carbon monoxide intoxication. Inhaled carbon monoxide is absorbed rapidly through the lungs, producing tissue hypoxia by competing with oxygen for binding sites on hemoglobin, with an affinity 250 times that of oxygen. Carbon monoxide also shifts the oxyhemoglobin dissociation curve to the left, causing oxygen carried by the red blood cells to be more tightly bound to hemoglobin.

In addition to tissue hypoxia, carbon monoxide may cause injury by impairing tissue perfusion. By binding to myoglobin within muscle cells, carbon monoxide potentially may cause myocardial depression and hypotension.

Sone et al. reviewed 67 patients with acute carbon monoxide poisoning, finding that 30 percent had abnormal chest X-ray films, including ground glass appearance, perihilar haze, peribronchial and perivascul bar cuffing and interstitial edema. Of those patients who died, 67 percent had pulmonary edema or hemorrhage at autopsy. In 2,759 patients with carbon monoxide intoxication, Choi et al. reported 23 cases of peripheral neuropathy which was limited almost exclusively to the lower extremities. The neuropathy typically was associated with local edema and a pathologic finding of demyelination.

Table 1 lists the chief causes of unilateral diaphragmatic paralysis. The fact that this paralysis was transient, that no other causes were documented by history, physical examination or radiologic studies, and that there was a clear association with carbon monoxide inhalation tend to discount causes other than carbon monoxide. In acute carbon monoxide poisoning, peripheral nerve injury may be explained by four mechanisms. The hypoxia induced by carbon monoxide may cause ischemic nerve injury, exposure to high partial pressures of carbon monoxide itself may grossly impair the peripheral nerve, petechial hemorrhages may occur in peripheral nerves as it does in other organs, finally, direct nerve compression or venous obstruction with ensuing edema and circulatory compromise may occur in the unconscious patient, leading to peripheral nerve injury.

In general, peripheral neuropathy is a rare, but well-documented sequel of carbon monoxide poisoning. Though no case of unilateral phrenic nerve paralysis has been previously described in association with carbon monoxide poisoning, this case reflects the fact that this may be a potential complication. The prognosis for recovery is excellent in cases showing a peripheral neuropathy, as in this case.

**REFERENCES**

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**Table 1—Causes of Unilateral Diaphragmatic Paralysis**

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