Clinical Case Report

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Unilateral brachial plexus injury following carbon monoxide intoxication

A case report

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Abstract

Rationale: We report a 45-year-old male patient with unilateral brachial plexopathy following acute carbon monoxide (CO) intoxication.

Patient concerns: The patient suddenly developed severe left upper extremity weakness and cognitive impairment after CO intoxication.

Diagnoses: He showed severe weakness in the left upper extremity and decreased cognitive function during physical examination. Whole body bone scan revealed increased radioisotope uptake in the left shoulder, suggesting rhadomyolysis. Brain magnetic resonance imaging showed high signal intensity in both globus pallidus. The electrodiagnostic finding was compatible with incomplete left total brachial plexopathy (axonopathy).

Interventions: High oxygen therapy and massive intravenous normal saline infusion were administered immediately after acute CO intoxication. Since then, intensive rehabilitation treatment has been provided.

Outcome: Despite having received medical and rehabilitation treatment, the patient has not recovered severe weakness in the left upper extremity and decline in cognition.

Lessons: Localized swelling in the left shoulder caused by rhabdomyolysis may be a key mechanism in developing unilateral brachial plexopathy after acute CO intoxication. The early diagnosis and treatment of rhadomyolysis might be important preventing peripheral neuropathy. An electrodiagnostic study may be helpful for diagnosis of peripheral neuropathy after CO intoxication and prediction of patient's prognosis.

Abbreviations: CMAP = compound muscle action potentials, CO = carbon monoxide, EDS = electrodiagnostic study, MRC = medical research council, MRI = magnetic resonance imaging.

Keywords: axonopathy, brachial plexopathy, carbon monoxide intoxication, electrodiagnostic study, peripheral neuropathy, rhadomyolysis, unilateral

1. Introduction

Carbon monoxide (CO) is a colorless, odorless, tasteless, and nonirritant gas produced primarily as a result of incomplete combustion of any carbonaceous fossil fuel. Exposure to high concentrations of CO can be lethal and CO intoxication is the most common cause of death from poisoning globally. CO intoxication is also a leading cause of severe neuropsychological impairments. However, peripheral neuropathy has rarely been reported after CO intoxication and is commonly reported as occurring in the lower extremities.[1] Isolated involvement of both upper extremities has been described in only a few patients, which were related to root damage, and only one case of bilateral brachial plexopathy following CO intoxication has been reported.[2] We present the first description of unilateral brachial plexopathy combined with rhabdomyolysis after CO intoxication.

2. Case presentation

A 45-year-old man was admitted to our emergency center with altered consciousness. He had been discovered by police in his vehicle in an unconscious state inhalation of burning coal. Upon arrival at the emergency center, he remained confused and disorientated with tachycardia and hypoxemia, and moderate swelling was noted on his left shoulder. His blood test showed elevated carbon monoxide hemoglobin (17.4%), creatinine kinase (14,608 IU/L), creatinine kinase myoglobin (19.9 ng/mL), and myoglobin (above 3000 ng/mL). His urine color was red-to-brown. On brain magnetic resonance imaging (MRI), a hyperintense signal was detected in bilateral globus pallidus on T2-weighted images (Fig. 1). High oxygen therapy and massive intravenous normal saline infusion were administered immediately, and input and output volume was strictly checked to correct plasma volume imbalance. The next day, he returned to an alert state but complained of left upper limb weakness. Manual muscle tests revealed 2 medical research council (MRC) grade in left shoulder flexor and extensor, left elbow flexor and

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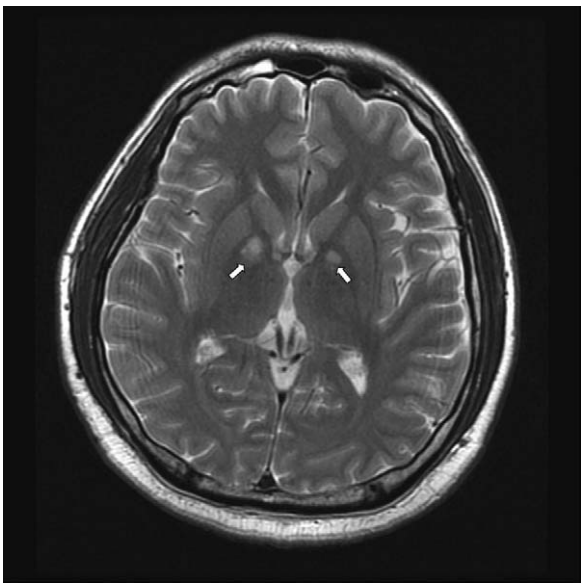

Figure 1. Brain magnetic resonance image at immediately after CO intoxication revealed high-signal intensity in both globus pallidus (arrows) on T2-weighted image. CO = carbon monoxide.

extensor, and left wrist flexor and extensor, and a 3 MRC grade in left finger flexor and extensor. There was decreased sensation to pinprick and light touch and an absence of deep tendon reflexes in the left upper limb. A whole body bone scan with Tc-99m HDP was taken 4 days after CO intoxication. This demonstrated increased radioisotope uptake in the soft tissue of both shoulders and right distal thigh area suggesting rhabdomyolysis was due to CO intoxication, and the left shoulder had more radioisotope uptake than the right one (Fig. 2). Follow-up blood tests became within normal range, and electrodiagnostic study (EDS) was performed 3 weeks after CO intoxication. A motor nerve conduction study showed a normal nerve conduction velocity for left median, ulnar, and radial nerves, a prolonged onset latency of compound muscle action potentials (CMAP) for the left median and musculocutaneous nerves, and a decreased amplitude of CMAP for left median, ulnar, radial, axillary, or musculocutaneous nerves. Sensory nerve action potential was not recorded for left median, ulnar, superficial radial, and lateral antebrachial cutaneous nerves (Table 1). Needle electromyography revealed abnormal spontaneous activities including positive sharp wave and fibrillation potential, and discrete recruitment pattern in all examined upper limb muscles including left infraspinatus, deltoid, biceps brachii, triceps brachii, flexor carpi radialis, first dorsal interosseous, and abductor pollicis brevis muscles (Table 2). Our electrophysiological interpretation was left incomplete total brachial plexopathy with axonal involvement. Four weeks after CO intoxication, the patient's cognition decreased suddenly and the Mini-Mental State Examination score was rated at 4. Follow-up brain MRI showed symmetric distinct patch hyperintensity in both globus pallidus and cerebral white matter on T2-weighted images, suggesting delayed CO intoxication. There was no improvement in left upper extremity muscle strength and cognitive function until 7 weeks after CO intoxication. Thereafter, he was transferred to local rehabilitation hospital but readmitted to our hospital due to prostatitis and aspiration pneumonia 11 months after CO intoxication. At that time, there was no interval change of left upper extremity weakness, impaired cognitive function, and follow-up EDS finding.

3. Discussion

CO is a toxic gas produced by the incomplete combustion of carbon-containing compounds, such as those within heating units, in which partial combustion of oils, coal, wood, kerosene, and other fuels generate CO. It is easily absorbed through respiration into the lung and competes with oxygen for binding to hemoglobin. The affinity of hemoglobin for carbon monoxide is 200 to 250 times greater than its affinity for oxygen. CO poisoning has previously been associated with amnesia, encephalopathy, dysarthria, Parkinsonism, peripheral neuropathy, bullous skin lesions, supranuclear gaze palsy, cerebral hemorrhage, cardiotoxicity, and muscle necrosis with renal failure. Among them, central nervous system involvements of CO intoxication are well known, but little has been reported about peripheral neuropathy.

A few reports of neuropathy after CO intoxication have been announced over the past few decades. They include peripheral neuropathy, encephalopathy, optic neuropathy, and unilateral diaphragmatic paralysis.[3–8] There was a large-scale clinical study of neuropathy caused by CO intoxication.[6,7] Among 2759 patients who were admitted to a single hospital due to CO intoxication, peripheral neuropathy was diagnosed using electromyography and a nerve conduction study in 23 subjects. The incidence of peripheral neuropathy was 0.8% and all cases except 2 involved the lower extremities. It was reported that peripheral neuropathy after CO intoxication usually affected both sexes in young adults, was confined to the lower parts of the body, displayed motor and sensory symptom, involved nerve root and peripheral nerve, and was fully recovered within 3 to 6 months.

Rahmani et al[2] reported a patient with reversible bilateral brachial plexus injury after acute CO poisoning. A nerve conduction study showed decreased amplitude of motor and sensory nerves, and electromyography revealed a reduced recruitment pattern without denervation potentials in both upper extremity muscles. The patient's neurological damage was completely restored after hyperbaric oxygen therapy. The prognosis of peripheral neuropathy after CO intoxication was excellent in most cases because peripheral nerve biopsy revealed demyelination, and an electrodiagnostic study showed demyelination with preservation of the axon.[1,7] To our knowledge, our case is the first report on unilateral brachial plexopathy with poor prognosis after CO intoxication. The muscle power of the patient's left upper extremity did not recover at 11 months after CO intoxication because the initial electrodiagnostic finding showed axonopathy. Therefore, an electrodiagnostic study is necessary to diagnose peripheral neuropathy after CO intoxication and determine its prognosis.

CO binds with hemoglobin to make carboxyhemoglobin in the blood and inhibiting oxygen uptake, leading to muscle hypoxia. Hypoxic muscle damage results in ATP depletion and Na/K-ATPase and Ca^{2+} ATPase pump dysfunction. If these electrolytic pumps fail to function, intracellular concentration of sodium and chloride will increase and result in osmotic swelling. As the concentration of calcium increase, the lipase and proteases are activated and lead to muscle cell death.[11] When the muscle cells die, myoglobin increases in plasma, results in renal tubular obstruction, and causes acute renal failure.[12,13] These are the pathophysiological mechanism of rhabdomyolysis that follow

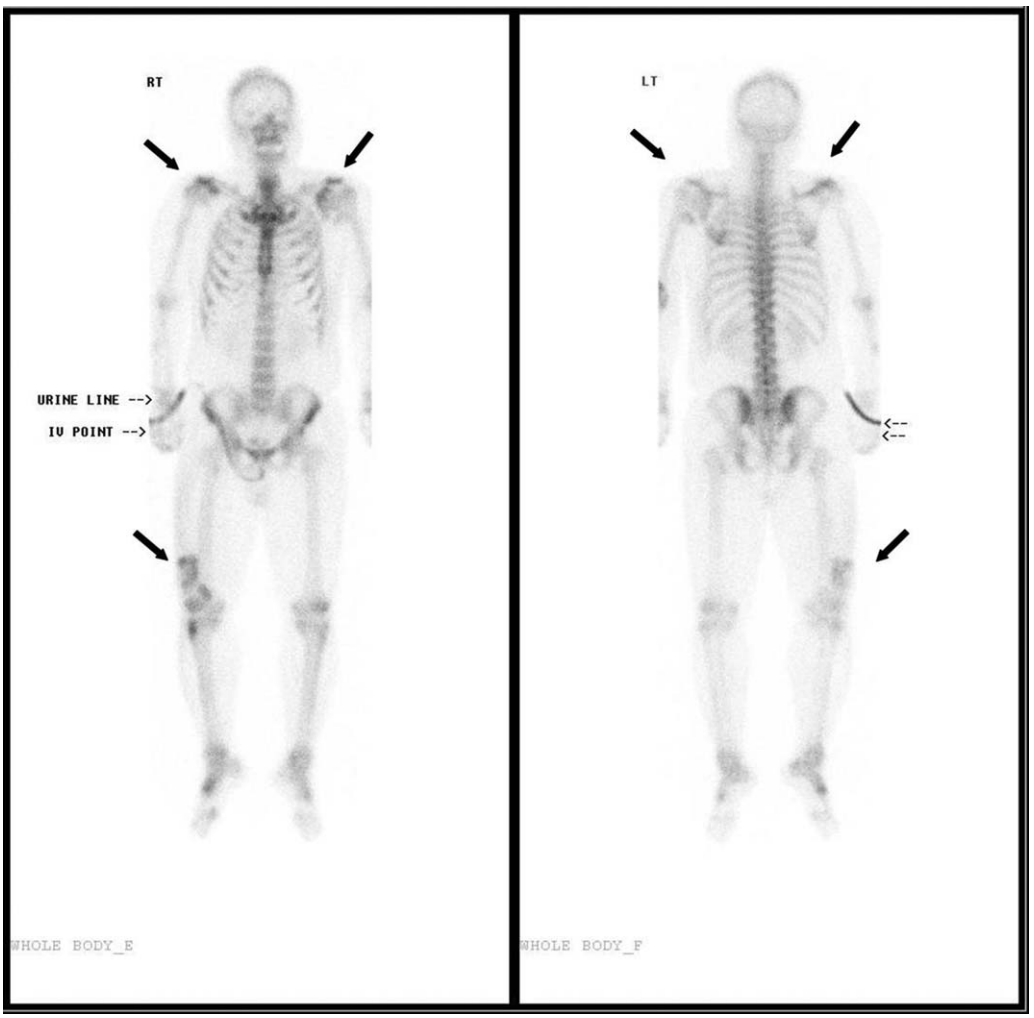

Figure 2. Whole body bone scan showed increased radioisotope uptake in both shoulders and right lower thigh (arrows).

CO intoxication. In our case, swelling was clinically detected on left shoulder and the patient's blood test showed elevated level of creatinine kinase, creatinine kinase myoglobin, and myoglobin. Whole body bone scan revealed that radioisotope uptake was mostly seen in the left shoulder. These findings are compatible with rhabdomyolysis in the left shoulder.

The pathophysiological mechanism contributing to peripheral neuropathy after CO intoxication can be multifactorial, different

Table 1

A nerve conduction study in the patient with incomplete left total brachial plexopathy after carbon monoxide intoxication.

	Right			Left		
Nerve	**DL, ms**	**Amp, mV**	**Velocity, m/s**	**DL, ms**	**Amp, mV**	**Velocity, m/s**
SNAP						
Median	3.44	20.5			No response	
Ulnar	2.55	27.2			No response	
Radil	2.14	26.5			No response	
LAC	1.20	18.5			No response	
CMAP						
Median	3.75	16.8	58.8	4.79	3.7	51.0
Ulnar	2.50	15.8	60.3	2.81	9.3	61.4
Radial	1.77	6.6	63.1	1.98	1.6	65.9
Axillary	2.81	6.1		3.23	2.7	
MC	4.22	9.5		6.30	0.3	
SSC	2.45	1.9		2.76	1.8	

Amp = amplitude, CMAP = compound muscle action potential, DL = distal latency, LAC = lateral antebrachial cutaneous nerve, MC = musculocutaneous nerve, SNAP = sensory nerve action potential, SSC = suprascapular nerve.

Table 2

Electromyographic findings in the patient with incomplete left total brachial plexopathy after carbon monoxide intoxication.

Muscle	ASA		MUAP	Interference pattern
	PSW	Fib		
Bilateral				
Cervical paraspinalis	None	None	Normal	
Left side				
Deltoid	3+	2+	Normal	Discrete
Biceps brachii	3+	2+	Normal	Discrete
Triceps brachii	2+	2+	Normal	Discrete
Flexor carpi radialis	3+	2+	Normal	Discrete
First dorsal interossei	2+	1+	Normal	Discrete
Abductor pollicis brevis	2+	2+	Normal	Discrete
Infraspinatus	2+	1+	Normal	Discrete

ASA = abnormal spontaneous activity, Fib = fibrillation potential, MUAP = motor unit action potential, PSW = positive sharp wave.

over time in the same patient, and even differ from patient to patient. Two important mechanisms were proposed in developing peripheral neuropathy after CO intoxication. First, generalized factors including hypoxia caused by CO and its subsequent ischemia, petechial hemorrhages, and cytotoxic effect of CO itself should be considered. Second, localized factors, including direct nerve compression or venous occlusion causing edema, circulatory disturbances in unconscious patient, and soft tissue necrosis might be contributor.[3,7,9,10] In our reported case, the localized edema in the left shoulder, due to rhadomyolysis, may be considered as the main mechanism of unilateral brachial plexopathy, and cytotoxic effect of CO and direct nerve compression may be involved.

A delayed neuropsychiatric syndrome can be presented in patients after acute CO exposure. This manifests in various ways, including personality change, cognitive impairment, psychosis, and Parkinsonism.[10] Our patient showed sudden decreased cognitive function 4 weeks after CO intoxication and this was diagnosed as CO encephalopathy. Thereafter, impaired cognition did not improve until a year after CO intoxication.

The novelty of our report is that it is of the first case of unilateral brachial plexopathy involving left upper extremity with poor prognosis and that the electrodiagnostic finding showed axonopathy involving total brachial plexus. Local swelling of the left shoulder due to rhabdomyolysis may be a key mechanism in development of unilateral brachial plexopathy. Therefore, the early diagnosis and treatment of rhadomyolysis might be important to prevent peripheral neuropathy after acute CO intoxication and an electrodiagnostic study can be helpful for physicians to diagnose peripheral neuropathy and predict a patient's prognosis.

4. Method

This was a case report. Ethics committee or institutional review board approval was not obtained. It was not necessary for the case report. The patient signed informed consent for the publication of this case report.

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Author contributions

Conceptualization: Dong Rak Kwon.
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