Lumbosacral Plexopathy Due to Carbon-Monoxide Intoxication: Case Report

Karbonmonoksit Zehirlenmesine Bağlı Lumbosakral Pleksopati

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ABSTRACT Carbon-monoxide (CO) is a common cause of human poisonings. Peripheral nerve involvement due to CO poisoning is rarely reported. Here we report a patient with unilateral lumbosacral plexopathy, following acute CO intoxication. A 51-year-old man was found unconscious due to CO inhalation and was admitted to emergency unit. After he became conscious, he complained of weakness and burning pain at his right lower extremity. Laboratory and lumbar magnetic resonance imaging revealed no abnormality. Electroneurographic findings were compatible with an asymmetric lumbosacral plexopathy in his right lower limb. After 9 months, favorable improvement was observed in both clinical and electroneurographic parameters, with the help of an intense rehabilitation. To our knowledge, this is the first case of plexopathy reported following CO intoxication. Plexopathy should be rule out among survivors with weakness and dysesthesia.

Key Words: Carbon monoxide poisoning; electromyography; peripheral nervous system diseases

ÖZET Karbonmonoksit (CO) insanlarda ölümcül gaz zehirlenmelerinin en sık rastlanan sebebidir. CO zehirlenmesine bağlı periferik sinir tutulumu bildiren az sayıda yayın mevcuttur. Bu makalede, akut CO zehirlenmesini takiben unilateral lumbosakral pleksopati gelişen bir olgu sunulmuştur. Ellibir yaşındaki erkek hasta acil servise CO inhalasyonuna bağlı bilinç kaybı ile getirildi. Bilinci açılan hastanın sağ alt ekstremitesinde güçsüzlük ve yanıcı ağrı yakınması oldu. Laboratuvar ve manyetik rezonans görüntülemesinde herhangi bir anormallik yoktu. Elektronörografik bulguları sağ alt ekstremitede asimetrik lumbosakral pleksopati ile uyumluydu. Yoğun rehabilitasyon programının da yardımıyla 9 ayın sonunda hem klinik hem de elektronörografik parametrelerde anlamlı düzelme gözlendi. Bildiğimiz kadarıyla bu vaka literatürde CO zehirlenmesi sonrası lomber pleksopati bildirilen ilk olgudur. CO'e maruziyet sonrasında güçsüzlük ve dizestezi tarifleyen hastalarda ayırıcı tanıda pleksopati de akla gelmelidir.

Anahtar Kelimeler: Karbon monoksid zehirlenmesi; elektromiyografi; periferik sinir sistemi hastalıkları

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Carbon-monoxide (CO) intoxication is a very serious cause of lethal poisonings. This condition is frequently encountered in our country, due to common use of coal and gas heaters. Approximately 10 000 cases of CO poisoning have been reported annually.[1]

CO affects nearly all the systems and tissues of the body such as nervous system, heart, kidney, skeletal muscle, etc.[2,3] Survivors of severe CO intoxication can develop long term neurological sequelae. Loss of memory

and orientation, anterograd amnesia, parkinsonism and speech problems can be seen.[4,5] Peripheral neuropathy is one of these neurological manifestations, with 0.84% incidence.[6] To our knowledge, we present the first reported case of lumbosacral plexopathy as a peripheral neuropathy due to CO intoxication.

CASE REPORT

A 51-year-old male patient was admitted to emergency unit by the development of unconsciousness after spending approximately 7 hours with a coal heater. There was no sign of trauma. His prior medical history and family history were nonspecific. He was internalized to the intensive care unit. Second day of the follow up, an evident improvement was observed in his consciousness state. During this period, his major complaints were burning pain, numbness and weakness radiating to the right leg.

After discharge from neurology clinic, the patient was referred to rehabilitation unit for muscle strength decrease in his right lower extremity. Besides, he was unable to walk without support and had neuropathic pain. His neurological examination disclosed a severe weakness, abolished deep tendon reflexes and hypoesthesia in L5-S1 dermatomes on right side. Muscle power of right foot dorsiflexion and plantar flexion was about 0/5. Also hip extension was 3/5. Hip flexion, knee flexion and extension showed a slight weakness (4/5). Neurologic examination of the left lower extremity revealed no abnormality. Additionally, rest tremor and a moderate rigidity were observed in both of the upper extremities. Laboratory findings, including total creatine phosphokinase (CK), 56 IU/L (normal range: 30-200 IU/L); total blood count, erythrocyte sedimentation rate, liver and renal function tests and urine analysis, were normal.

His first electromyography investigation was performed at the 2nd month of the intoxication; compound muscle action potentials of right tibial and peroneal nerves could not be recorded. Additionally, sensory nerve action potential of right sural and peroneal nerve was unobtainable (Table 1).

Needle EMG demonstrated acute denervation in peroneal, tibial, superior and inferior gluteal nerve innervated muscles. Absence of motor unit potential was observed in affected muscles. Needle EMG of paraspinous muscles was normal (Table 2).

Cranial magnetic resonance imaging (MRI) (Phillips 1.5T Achieva) showed bilateral diffuse restricted diffusion in the white matter consistent with periventriculer-subcortical hypoxic ischemic damage. Electroencephalogram was normal. MRI scans with T1-T2 in sagittal plane, T2 and fat-suppressed T2 in axial plane, fat-suppressed T2 in coronal plane, contrast-enhanced fat-suppressed T1-weighted in coronal, and axial displayed normal lumbar spine and lumbosacral plexus. The patient was enrolled in neurologic rehabilitation program. Exercises for strength, stretching and balance and electric stimulation for affected muscles were performed. After 9 months of rehabilitation program, the patient became independent for walking with spring ankle foot orthosis, and improvement in muscle power of the right hip extensors, from 3 to 4, foot dorsiflexors and plantar flexors, from 0 to 1 in the manual muscle test, was observed. Control EMG revealed diminished acute denervation potentials and increased reinnervation potentials in affected muscles especially prominent at proximal muscles.

DISCUSSION

Carbon monoxide is an odorless, colorless gas that can cause serious morbidities, even sudden death. It has 210 times higher hemoglobin-binding affinity than oxygen, therefore eventually reduces oxygen transfer to the peripheral tissues. Also, it causes a leftward shift in oxyhemoglobin dissociation curve which makes hemoglobin to release oxygen harder and leads to tissue hypoxia.[7]

Among the survivors of CO intoxication, symptoms range in a wide spectrum according to the carboxyhemoglobin (COHb) levels. While 10-20% COHb levels lead to a slight headache, coma state is observed at the level of 50-60%.[2] The most common symptoms are myocardial and neurological involvement.[8] The incidence of neurological sequelae due to CO intoxication is ranging between

TABLE 1: Nerve conduction studies.

Nerve	Sites	Recording site	Initial						Month 9					
			Latency (ms)		Amplitude (motor:mV sensory:µV)		Velocity (m/s)		Latency (ms)		Amplitude (motor:mV sensory:µV)		Velocity (m/s)	
			R	L	R	L	R	L	R	L	R	L	R	L
Motor														
Medianus	Wrist	APB	2.8	3.5	5.4	4.5								
	Elbow		6.9	8.1	5.7	3.0	54.9	49.8						
Ulnaris	Wrist	ADM	2.4		10.9									
	B. Elbow		5.4		10.7		63.3							
	A. Elbow		6.8		10.2		57.1							
Tibialis	Ankle	AHB	Ø	3.4	Ø	10.5			4.0	5.2	0.4	8.6		
	P. Fossa		Ø	11.6	Ø	6.3	Ø	45.1	13.7	13.2	0.2	9.8	34.0	41.9
Peroneus	Ankle	EDB	Ø	4	Ø	4.26			Ø	2.8	Ø	9.8		
	F. Head		Ø	9.2	Ø	3.34	Ø	52.9	Ø	9.2	Ø	9.5	Ø	51.6
	P. Fossa		Ø	11	Ø	2.79	Ø	47.2						
Sensory														
Medianus	Wrist	2.digit	2.4		15		54.2							
Ulnaris	Wrist	5.digit	2.0		19		60.0							
Suralis	Mid calf	L. Malleol	Ø	2.7	Ø	11	Ø	51.9	Ø	2.2	Ø	22	Ø	50.0
Peroneus	Mid calf	Ankle	Ø	3.12	Ø	10.7	Ø	55.5	Ø	3.1	Ø	10.4	Ø	54.8
Saphenus	Mid calf	Ankle	2.9	2.9	2.0	2.2	43.3	44.8						

R: Right; L: Left; ABP: Abductor pollicis brevis; ADM: Adductor digiti minimi; AHB: Abductor hallucis brevis; EDB: Extensor digitorum brevis; B. Elbow: Below elbow; A. Elbow: Above elbow; F. Head: Fibular head; P. Fossa: Popliteal fossa; Ø: Absent.

1 and 47%.[9,10] Carbon monoxide intoxication has several central nervous system complications including behavioral, cognitive and motor dysfunctions.[5,8,11] Although there are few reports regarding peripheral nervous system involvement due to CO intoxication, no reported case of plexopathy as neurological sequelae among the published data in English language could be found.[12-16] To our knowledge, this is the first report of lumbosacral plexus involvement due to CO intoxication.

In this case there might be several causes of weakness: (1) Compression of peripheral nerves due to inappropriate position of the body before the patient refers to the emergency unit or due to hemorrhages and edema; (2) Rhabdomyolysis; (3) Compartment syndrome due to subfascial edema; (4) Ischemic nerve damage related to hypoxia induced by CO.[6,13,17,18]

The patient had spent 7 hours unconsciously and we got no information about his lying position. However, we can conclude with some clues. Compression is known to cause predominantly demyelinating neuropathy. Because electroneurographic evaluation revealed axonal damage of plexus, and there was no sign of edema or hemorrhage in the MRI scan of the plexus, the possibility of compression can be excluded.

TABLE 2: Initial needle EMG.

Muscle	Fibrillations	PSWs	MUPs
Paraspinalis L5	None	None	Normal
Paraspinalis S1	None	None	Normal
Vastus lateralis	None	None	Normal
Gluteus medius	+4	+4	No activity
Tibialis anterior	+4	+4	No activity
EHL	+4	+4	No activity
GCN caput medialis	+4	+4	No activity

PSWs: Positive Sharp Waves; MUPs: Motor Unit Potantials; EHL: Extansor hallucis longus; GCN:Gastrocnemius.

Rhabdomyolysis was reported as another cause of weakness after CO intoxication.[13,18] Assessing CK level is the most reliable and sensitive indicator for muscle injury.[19] In this case, serum level of CK was in normal range. Additionally, distribution of in-

volved muscles was not diffuse and referred to a precise location. This specific presentation also excludes compartment syndrome.

There have been published data regarding peripheral neuropathy due to CO intoxication. Choi found CO-induced peripheral neuropathy in 0.84% of his cases. In that study, the lower-extremity involvement was more prevalent and major complaints were numbness, tingling and burning pain. Weakness, abnormal EMG and NCS findings were detected at 9 of those patients and three of them were determined as lumbosacral radiculopathy.[6] Garcia and Maestro have also reported an asymmetric neuropathy with electrophysiological abnormalities in both lower extremities developed following CO intoxication.[15] Meanwhile, Mimura et al. concluded that 16% of CO-intoxicated patients had peripheral nerve symptoms.[20] Kuo et al. presented a case with symmetrical femoral neuropathy due to CO intoxication which was confirmed by EMG findings.[13] Also Yarar et al. published a case with tibial and peroneal neuropathy with nerve conduction study abnormalities subsequent to CO poisoning.[16] However, there was no needle EMG finding in their case. Different from previous case reports of CO-induced peripheral neuropathy, our EMG results suggest a lumbosacral plexus involvement.

Human pathogenesis of peripheral neuropathy due to CO intoxication is still unknown. However, there are a number of experimental animal studies. Petajan et al investigated sequelae of hypoxia, due to CO, in rats. They found that peripheral nerve conduction velocities were not affected until the COHb level of 60%. After this threshold, NCV gradually decreased and a total conduction block was observed after the level of 70%. The researchers also observed that peripheral nerve response to anoxia happened in three-phases. In acute phase, the conduction block occurred due to impairment of axonal mechanisms of oxidative metabolism. In subacute period, loss of nerve conduction was thought to be caused by anoxia-induced axoplasmic flow loss. During the third stage, conduction was not totally lost but slowed and the distal latencies were normal. Authors explained this by involvement of Ranvier nodes and the dying-back phenomenon.[21] Pankow et al. demonstrated that the sciatic motor conduction velocity of rats was reduced by 33%, 24 hrs after acute carbon monoxide inhalation (COHb level is about 60%). This reduction was significant even 4 weeks after intoxication.[22] We estimate that our patient's blood COHb level was higher than 50-60% as he was in coma state. Our results are consistent with Petejan's results, regarding nerve conduction block.

Petajan's findings were supported by another study investigating histopathological changes in peripheral nerves after CO intoxication. This study included concurrent nerve conduction studies and pathological investigations. Samples, taken immediately after exposure, revealed an intact myelin sheath but damaged Ranvier nodes. This injury was more prominent in large myelinated fibers than in small myelinated ones. The authors concluded that after peak impairment in 7 to 10 days, a repair period began in 14 lasting to 21 days. When the repair began, an improvement in nerve conductions was also observed.[23]

In this case we observed significant improvement within 9 months. Previously reported peripheral neuropathies with CO intoxication showed evident improvement as well. Choi reported full recover of all cases within three to six months.[6] Also Garcia et al. noticed significant improvement in a short time period. His case showed normal clinical and electroneurographic findings at the first year follow up.[15] Kuo et al. observed good progression after an intense rehabilitation program.[13] Although our case had plexus involvement (different from the previous cases), he also showed significant improvement within 9 months consistent with previous reports.

In conclusion, this case is the first one that demonstrated lumbosacral plexopathy as neurological sequelae after CO intoxication up to date, to our knowledge. Marked improvement was observed during the rehabilitation program. Plexopathy should be taken into consideration in CO-intoxication-survivors with weakness and dysesthesia in extremities (which demonstrates distribution of a certain plexus). EMG examination is also a necessity in such cases.

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