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Peripheral Mononeuropathy Following Acute Carbon Monoxide Intoxication: A Diagnostic Challenge

Karbon Monoksit Zehirlenmesi ve Periferik Sinir Mononöropati Birlikteliği: Olgu Sunumu

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Due to social and economic reasons, one of the most important problems worldwide is the threat posed by carbon monoxide (CO) in home heating systems. In a retrospective study reported by Keles at al. (1), 323 patients were diagnosed with CO poisoning between January 2002 and December 2003 in a tertiary care university emergency department. All poisonings were unintentional and most of the patients have presented during the winter months.

Because of their high metabolic rate, the brain and the heart are most susceptible to CO toxicity (2). Physiopathology and pathology of central nervous system (CNS) complications following CO intoxication are well described in the literature (3,4). However, peripheral neuropathy has rarely been reported and the pathogenesis of neuropathy still remains unclear (5-10).

A previously healthy 58-year-old woman was admitted to our outpatient clinic with a complaint of weakness and numbness in her right foot. Three weeks earlier, she was found unconscious in her house with severe hypoxia and carboxyhemoglobin level of 45%. She was treated successfully with ventilatory support and ten sessions of hyperbaric oxygen therapy. When she recovered consciousness, neurological examination revealed severe weakness of the right ankle and toe dorsal flexion. Careful examination also showed weakness in the right knee flexion. The right Achilles tendon reflex was absent. She had no low-back pain and no pathological reflexes. Neurologic examination was otherwise normal. Laboratory studies were normal. Magnetic resonance imaging (MRI) of the lumbar spine and the brain, which was taken four weeks after the incidence, was normal. Electroencephalogram did not show any focal abnormalities. Electrophysiological studies were carried out with a Medelec® Synergy Multimedia electromyograph. Although bilateral peripheral nerve conduction parameters of the lower limbs were within our laboratory limits, distal motor latency of the right common peroneal nerve was relatively prolonged and compound muscle action potential amplitude was relatively lower compared to the left. In addition, the F-wave of the right common peroneal nerve could not be obtained. Similarly, sensory nerve action potential amplitude of the right superficial peroneal nerve was relatively lower compared to the left. Needle electromyography (EMG) showed marked denervation potentials and a reduced recruitment pattern especially in the right tibialis anterior, peroneus longus and biceps femoris muscles. The gastrocnemius medialis muscle was moderately affected. Needle electromyography of the left lower extremity and bilateral lumbal paraspinal muscles were normal. She was included in an intense rehabilitation program with a diagnosis of sciatic nerve neuropathy; her complaints decreased significantly after 3 months and a complete clinical recovery was reached 6 months later. Repeated electrophysiological studies at 6-month follow-up revealed normal peripheral nerve conduction studies with an improved recruitment pattern in the involved right lower extremity muscles.

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A variety of neurological sequelae develop after CO poisoning as a result of the involvement of the CNS. Several mechanisms of CO-mediated toxicity have been suggested. One hypothesis is that CO-induced tissue hypoxia may be followed by reoxygenation injury to the CNS. Delayed neurological sequelae may also occur in patients from days to weeks after the poisoning event showing the characteristic symptom triad of mental deterioration, urinary and/or fecal incontinence, and gait disturbance (11).

The incidence of peripheral neuropathy has been reported in less than 5% of the cases with acute CO poisoning (7,8). Choi (7) reported 20 cases of peripheral neuropathy after CO intoxication during a six year period. In his study, electrophysiological studies of 20 cases were performed and 6 showed denervation potentials on needle EMG but normal nerve conduction studies, while 14 had abnormal findings both on nerve conduction studies and needle EMG. All of the 15 patients who were followed for 1 year, recovered within 3-6 months. Various pathogenic mechanisms such as ischemia due to hypoxia, petechial hemorrhages, or CO itself have been accused for the changes found in the peripheral nerve (6). In addition to these, compression from spending hours or even days on a hard surface may be considered as an important contributing factor for the peripheral nerve abnormality in some cases (6,8). Although histologic examination of clinically affected human peripheral nerves in cases of CO intoxication has shown demyelination with preservation of axons (5), Choi (7), from his electrophysiological studies suggested a concomitant axonal involvement. Furthermore, Grunette et al. (12) have reported reversible loss of normal axonal and Schwann cell structure at the node of Ranvier in peroneal and caudal ventral nerves of rats exposed to CO. Similarly, electrophysiolgic findings in our patient suggested both demyelination and axonal involvement.

Identifying the cause of a lower extremity neurologic symptom such as acute foot drop can be challenging. Post-injection sciatic nerve injury is a common cause of sciatic mononeuropathy, however, our patient did not receive any intramuscular gluteal injections during the intensive care unit stay or after discharge. The patient did not show any signs of Parkinsonism such as bradykinesia or cognitive dysfunction suggesting CNS lesion. Brain MRI immediately after CO intoxication may be normal since hiperintensity in the basal ganglia can be detected 7-8 weeks after the incident. However, since our patient's detailed neurological examination at discharge was normal and her weakness recovered within weeks, there was no need to repeat brain MRI. She did not suffer from any endocrine or metabolic disease associated with neuropathies. Similarly, the patient did not present sepsis or multiorgan failure suggesting critical illness neuropathy, during intensive care unit stay. The patient had no local swelling, which is thought to be an important contributing factor for the development of neuropathy after CO intoxication (8), on the affected lower limb. The patient's spine MRI and paraspinal needle EMG was negative for nerve root disease. The patient's electrodiagnostic studies suggested a reversible unilateral peripheral mononeuropathy of the sciatic nerve, especially the peroneal component, following acute CO poisoning. Previously, Choi (7) described the following characteristics of peripheral neuropathy after CO intoxication, most of which were also present in our case: it equally affects both sexes, exclusively involves lower extremities, has motor and sensory symptoms and complete recovery occurs within 3 to 6 months. The exact mechanism of CO-induced neuropathy still remains controversial; it has been proposed that CO toxicity is due to combination of tissue hypoxia and direct CO-mediated damage at cellular level (10,13), however, as in our case, compression neuropathy could not be ruled out.

In conclusion, domestic CO poisoning is a serious public health problem which demands greater efforts in public and medical education. The true incidence of peripheral neuropathy is unknown and many cases probably go unrecognized. Clinicians should be on high alert and look for any such or similar neurological complications in the early phase. Careful neurologic examination and electrophysiologic studies are essential for its differential diagnosis and follow-up.

Conflict of Interest:

Authors reported no conflicts of interest.

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