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Case Report

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N-hexane neuropathy: more frequent than expected

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ABSTRACT

Organic solvents that are widely used in industrial production and abused amongst teenagers for recreational purposes may cause some neurological abnormalities, including polyneuropathy. Poorly ventilated working environments and working without protective garments are the most common reasons for intoxication. N-hexane toxicity often causes sensorimotor mixed type demyelinating neuropathy, but axonal degeneration is not rare. We report five cases that had mainly axonal motor polyneuropathy due to n-hexane containing glue usage, with clinical and electrophysiological data. Clinical and electrophysiological examinations revealed predominantly motor findings. One patient had delayed P100 potential, two had abnormal conduction in fasciculus gracilis. "coasting" phenomenon was observed in one patient. Neuropathy due to toxic agents is a significant public health problem. Workingplace conditions must be strictly controlled if toxic materials are present. Patients who had electromyography compatible with polyneuropathy should be questioned for harmful exposure. Asymptomatic workers should also be examined. Patients should be followed for the possibility of the "coasting" effect.

Keywords: N-hexane, polyneuropathy, electrophysiology, organic solvents

INTRODUCTION

N-hexane is an aliphatic hydrocarbon derivative, which is widely used in industrial settings and abused for recreational purposes amongst teenagers.[1] It is used as a solvent in vegetable oil extraction, and in cleaners, degreasers, glues, spray paints, paint thinners, coatings, silicones, and greases. Workers in the fields of food processing, printing, manufacturing, and painting often use these n-hexane-containing products and automotive repair industries, as well as anywhere petroleum distillates are used.[2-4] Because of the volatile property of this substance, working in insufficiently ventilated close workplaces may cause slow progressing, sensorimotor polyneuropathy with axonal degeneration and secondary demyelination.[5-9] Cranial neuropathies and blurred vision due to maculopathy have been observed.[3,8,10] In glue-sniffers or patients with n-hexane neuropathy caused by addictive inhalation, the neurological manifestations are characterized by subacute, predominantly motor polyneuropathy. Nerve conduction abnormalities may fulfill the formal criteria for demyelinating polyradiculoneuropathy.[4] However, there are also reported cases with pure motor nerve involvement. The rate of polyneuropathy development is wide. The rate of polyneuropathy due to volatile substances, including n-hexane, is between 7% and 62%.[8]

We would like to present five cases of n-hexane neuropathy here that were referred to our electrophysiology laboratory from different neurology departments. Clinical and electrophysiological data of these patients were discussed in relation to literature.

CASES PRESENTATION

We evaluated five patients who had prior n-hexane exposure in their workplaces with polyneuropathy. Two of them worked in a shoe manufacturing, while other three worked in same factory in which adhesive bands were produced. All patients underwent extensive search concerning the etiology of polyneuropathy, which included complete blood count, renal and liver functions, electrolytes, glucose, vitamin B12, folic acid, antibodies for hepatitis and human immunodeficiency viruses, serum and urine protein electrophoresis and tumor markers. They all had chest radiographs and spinal magnetic resonance imagings (MRI). Electrophysiological examinations included motor and sensory nerve conductions (CNV), needle electromyography (EMG) of distal and proximal limb muscles and in two cases evoked potentials (**Table 1**). Two of the patients also had control examinations after termination of exposure (**Table 2**). None of them had nerve biopsy or urine 2.5 hexanedione analysis.

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Table 1. Results of needle electromyography and nerve conduction studies

	Case 1	Case 2	Case 3	Case 4	Case 5
Median motor (right)					
Distal latency (msec)	5.3	5.3	5	4.2	4.4
Proximal latency (msec)	11.7	10.8	9.3	9.3	10.5
Distal amplitude (μV)	4.5	16.1	6.6	5.2	16.7
Proximal amplitude (μV)	3.7	14.6	6.0	4.6	10.1
Velocity (m/sec)	43.8	43.6	53.4	47.4	38.0
Ulnar motor (right)					
Distal latency (msec)	3.8	4.0	3.0	3.4	3.2
Proximal latency (msec)	10.7	9.2	7.6	7.9	7.9
Distal amplitude (μV)	7.1	12.0	9.3	7.2	13.8
Proximal amplitude (μV)	6.1	12.1	8.1	6.0	14.6
Velocity (m/sec)	40.6	46.2	52.2	55.6	35.9
Tibial motor (right)					
Distal latency (msec)	8.2	5.8	6.8	6.5	7.3
Proximal latency (msec)	24.8	16.6	18.2	16.6	17.6
Distal amplitude (μV)	0.6	12.5	3.7	2.7	10.9
Proximal amplitude (μV)	0.5	7.4	2.5	2.5	6.6
Velocity (m/sec)	31.3	38.9	35.5	40.6	36.4
Peroneal motor (right)					
Distal latency (msec)	7.5	4.3	5.9	5.4	4.2
Proximal latency (msec)	20.4	13.7	15.4	13.9	14.1
Distal amplitude (μV)	0.2	4.2	5	1.6	8.4
Proximal amplitude (μV)	0.2	3.2	4.3	1.6	8.1
Velocity (m/sec)	34.1	36.2	35.3	41.2	27.3
Median sensory (right)					
Latency (msec)	3.6	3.5	3.1	2.6	2.7
Amplitude (μV)	14.0	21.0	15.0	13.7	12.9
Velocity (m/sec)	46.7	44.8	52.0	48.5	41.4
Ulnar sensory (right)					
Latency (msec)	3.9	4.1	3.3	2.5	2.7
Amplitude (μV)	13.0	29.0	18.0	10.6	12.7
Velocity (m/sec)	46.9	45.3	14.0	56.2	42.9
Sural sensory (right)					
Latency (msec)	4.7	3.6	3.9	3.5	3.3
Amplitude (μV)	2.7	12.0	14.0	8.0	9.6
Velocity (m/sec)	39.5	41.4	43.5	42.6	41.4
Needle EMG	Acute and chronic denervation findings with reduced recruitment	Slight chronic denervation findings	Acute and chronic denervation findings	Acute and chronic denervation findings	Mild reduction of recruitment

EMG: Electromyography

Case 1

Forty-one years old male patient presented with weakness of legs, numbness and burning sensations of feet. Forty-one-year-old male patients presented with weakness in their legs, numbness, and burning sensations in their feet. He has been working in shoe manufacturing for twenty years, six days in a week and nine hours daily. His initial complaints have been numbness in the feet and hands for ten years. Loss of dexterity of hands while performing fine movements supervened. He had weakness in his legs for six months. Three years ago, he had psychotic disorder diagnosis and used haloperidol and biperiden until six months ago. He had weakness of both lower limbs; distal parts were prominently affected. Deep tendon reflexes were reduced in the upper limbs and absent in the lower ones. Sensory examination revealed short stoking fashion loss of superficial touch sensation and impairment of vibration in lower limbs. He had widespread atrophy prominently affecting intrinsic hand and feet muscles. Primary reflexes were not present. He had lumbar spinal MRI because of his bilateral dropped feet, which showed no relevant abnormality. Other previously mentioned laboratory investigations were in normal limits. Electrophysiological examination disclosed widespread, symmetrical sensory-motor axonal neuropathy, which affected motor fiber predominantly. He also had prolonged distal motor latency in his legs, which suggested demyelination. The traces are shown in **Figure 1**.

Table 2. Control electrophysiological data of three patients

	Case 2	Case 4	Case 5
Duration after discontinuation of exposure	1 month	6 months	3 months
Median motor (right)			
Distal latency (msec)	3.64	3.5	4.2
Proximal latency (msec)	8.96	8.0	9.2
Distal amplitude (μV)	8.15	12.2	2.6
Proximal amplitude (μV)	6.33	11.0	1.7
Velocity (m/sec)	47.0	53.3	46.0
Ulnar motor (right)			
Distal latency (msec)	2.64	3.5	3.2
Proximal latency (msec)	8.49	7.3	7.9
Distal amplitude (μV)	7.14	10.5	2.3
Proximal amplitude (μV)	5.62	10.7	1.6
Velocity (m/sec)	43.6	60.0	42.0
Tibial motor (right)			
Distal latency (msec)	6.5	4.2	6.2
Proximal latency (msec)	18.65	15.0	17.5
Distal amplitude (μV)	7.14	5.0	1.8
Proximal amplitude (μV)	5.62	5.3	1.4
Velocity (m/sec)	43.6	38.4	35.8
Peroneal motor (right)			
Distal latency (msec)	4.4	4.2	5.1
Proximal latency (msec)	15.0	13.4	13.0
Distal amplitude (μV)	2.9	2.8	2.2
Proximal amplitude (μV)	1.79	3.8	1.2
Velocity (m/sec)	30.7	36.7	36.7
Median sensory (right)			
Latency (msec)	2.04	3.0	3.0
Amplitude (μV)	27.2	12.0	5.6
Velocity (m/sec)	66.2	52.0	46.2
Ulnar sensory (right)			No response
Latency (msec)	2.26	3.3	
Amplitude (μV)	16.9	10.0	
Velocity (m/sec)	64.2	53.8	
Sural sensory (right)			
Latency (msec)	3.72	3.7	3.0
Amplitude (μV)	8.30	6.9	4.1
Velocity (m/sec)	44.4	42.9	44.0
Needle EMG	Acute and chronic denervation findings with reduced recruitment	Acute and chronic denervation findings	Acute denervation findings

EMG: Electromyography

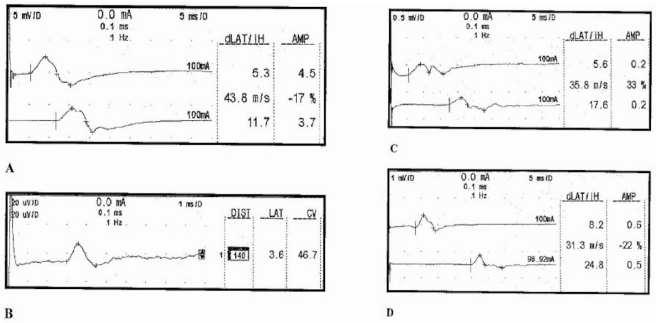

Figure 1. Nerve conduction studies of case 1. Motor nerve conduction (A) and sensory nerve conduction (B) of median nerve, peroneal (C) and tibial (D) motor nerve conductions

Case 2

Twenty-eight years old male patient presented with difficulty of climbing stairs, walking difficulty while carrying weight, and pain the legs and arms for twenty days. He has been working in a factory producing adhesive bands for five months. He had no personal or family history of any diseases concerning neuropathy. Clinical examination revealed only walking difficulty on heels. Electrophysiological investigations showed motor neuropathy affecting both upper and lower limbs. One month after discontinuation of working electrophysiological examination showed motor neuropathy with axonal degeneration with prominent decrease in amplitudes. One

month after discontinuation of working electrophysiological examination, motor neuropathy with axonal degeneration and a prominent decrease in amplitudes were observed. These findings suggested the "coasting" phenomenon. The Results of the second electrophysiological investigation are shown in Table 2.

Case 3

Thirty-three years old male patient presented with difficulty of walking. He has been working in the factory as case 2, which produced adhesive bands for seven years. He had no previous history of any illness. Clinical examination revealed weakness in both lower limbs affecting distal muscles more than proximal ones. He had normoactive deep tendon reflexes with regular sensory examination. Cranial and cervical MRIs were normal besides the asymptomatic protrusion of intervertebral discs. Nerve conduction studies were compatible with axonal motor neuropathy. Mild prolongation of P100 latencies on both sides were detected (right: 127 msec, left: 127.8 msec) (Figure 2). Conductions in fasciculus cuneatus were normal while in fasciculus gracilis were mildly slowed.

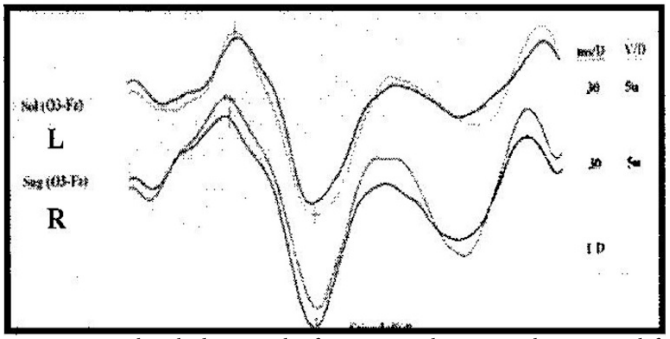

Figure 2. Visual evoked potentials of case 3. P100 latencies right 127 msn, left 127.8 msn

Case 4

Forty-one years old male patient who worked at the same factory as case 2 and case 3 presented with difficulty of walking, pain and burning feelings in hands and feet. He has been working in same factory for nine years. Three years ago, he was followed for uncertain sarcoidosis diagnosis for a year without any treatment. He had no family history concerning neuropathy. Clinical examination revealed weakness in both lower limbs, which affected distal limb muscles prominently. Deep tendon reflexes were hypoactive with impairment of vibration sense in lower limbs with preservation of position sense. Cervical spinal MRI disclosed no relevant abnormality. He had chest computed tomography with normal results and he had no clinical sign of sarcoidosis. Electrophysiological examination disclosed severe axonal motor neuropathy with widespread denervation of muscles and decreased muscle action potential amplitudes. In addition, median nerve motor conduction velocity was reduced. Visual evoked potential (VEP) findings were normal. Conductions in fasciculus cuneatus were normal bilaterally while there was conduction block on both sides in fasciculus gracilis (Table 1). After discontinuation of exposure for six months, his pain and weakness complaints showed some improvement, but he still had difficulty to lift his feet from the ground. He has been taking Gabapentin 300 mg/day for neuropathic pain. His neurologic examination revealed full muscle strength in arms and proximal leg muscles while he had paresis of distal feet and toe muscles. His deep tendon reflexes, and position sense were normal, while vibration sense was impaired in lower limbs and was more prominent on the right. Electrophysiological examination findings showed axonal motor neuropathy affecting lower limbs (Table 2). When findings were compared to the first examination, motor CNVs were slower and muscle action potential amplitudes were slightly higher.

Case 5

Fifty-two years old male patient who had been working in shoe manufacturing for forty years presented with inability to walk. His first complaints were tingling sensations in his legs, which began four months before the examination. He had no complaints until then. Involuntary contraction of leg muscles followed sensory complaints. Within two months, he was unable to walk due to severe loss of muscle strength. He had brisk tendon reflexes and quadriparesis that was prominent in the lower limbs and distal parts. He had to stock glove-type loss of sensation. He had stocking glove type loss of sensation. Right first dorsal interosseous muscle was atrophic. He stopped working after the appearance of his first complaints. The first EMG examination performed within the first month after his ailment showed mild slowing of motor conduction velocities. The second examination disclosed decreased compound muscle action potentials with mild slowing of motor conduction velocities and sensory abnormalities. Needle EMG showed findings that are compatible with acute axonal degeneration (Table 2).

DISCUSSION

We evaluated five patients who had prior n-hexane exposure in their workplaces with polyneuropathy. In this case series, we found more positive neuropathy findings than expected, which may be due to be our center to which the patients were referred may have increased the likelihood of diagnosis.

The first n-hexane related toxic neuropathy was reported in Japan in 1963 but it has been known since 1957, when the first cases of the so-called shoemakers' toxic polyneuropathy were reported.[13,17] As it was pointed out previously neurotoxic effects of n-hexane are attributable to the toxic metabolites. In humans, as well as and experimental animals, the initial alteration is an accumulation of 10-nm neurofilaments in the distal preterminal axon. The exact mechanism of n-hexane neurotoxicity has not yet been identified but an interference with neuronal axoplasmic flow has been suggested.[1] Axon was degenerated by multifocal giant axonal swellings containing cytoskeletal component accumulations and fragments of mitochondrial and endoplasmic reticular fragments.[14]

The loss of large, myelinated fibers with focally enlarged "giant" axons filled with these neurofilaments, and associated thinning of the overlying myelin have been reported. It has been reported that axonal swellings in the proximal parts of nodes of Ranvier begin multifocally and spread to the more proximal and distal sites of axons, causing the breaking down of nerve fibers. These areas are the accumulation sites of structural elements. The local swelling process also results in paranodal myelin retraction and occasional segmental demyelination of overlying myelin sheath with electrophysiological properties of demyelinating neuropathy.[4,15] Axonal swellings filled with the neurofilaments in large myelinated axons are followed by Wallerian-like degeneration of the distal axon.[18] Animal studies have documented a relationship between slowing of axonal

transport and focal axonal swelling.[2] Electrophysiological studies of n-hexane polyneuropathy indicate primary axonal degeneration and focal demyelination with and without conduction blocks.[5,6,8,9,19] Polyneuropathy due to occupational exposure shows mild subacute progression with sensory and motor symptoms. Symptoms are usually worsen after discontinuation of exposure for a few months, which is described as "coasting" phenomenon.[2,8,9,20,21] Deterioration of muscle strength and sensory deficit is common and become maximal within two-three months. The longest and thickest nerves are more susceptible to the toxic effects therefore symptoms usually affect legs first. Initial complaints consist of numbness, burning and tingling sensations in the toes and fingers, followed by distal limb weakness. The symptoms of polyneuropathy are usually symmetrical. Sensory impairments include reduced sensation of temperature, pinpricks, light touch, and vibration as well as position sense in the distal limbs. The muscle wasting usually occurs in the intrinsic hand and foot muscles; and muscle weakness often involves the extensor and flexor muscles of forearms and legs. Extensor muscles are more severely affected than flexors.[2] Electrophysiological abnormalities have also been reported in asymptomatic workers who had n-hexane exposure for more than one year.[8]

The main complaint of our five cases was walking difficulty. Accompanying sensory disturbances were numbness and burning sensation in the feet in the first case, pain in the legs and feet in the second patient, burning sensation and impaired vibration sensation in the fourth case. Sural nerve sensory nerve action potentials amplitude was low and CNV was slow in our first patient. Other than these findings, there was no sensory abnormality in any of the patients. We found electrophysiological abnormalities to mostly involve motor nerves. The study conducted by Kutlu et al.[3] mainly revealed a decrease in the amplitude of the compound nerve action potential. There are also several reports that multifocal motor conduction block with prominent slowing of the nerve conduction velocity in patients who have been exposed to n-hexane.[3] In another study from Turkiye, motor deficits were the main clinical manifestation, but the EMG study revealed a sensorimotor neuropathy in all patients.[9]

In our case series, the first case had almost twenty years of exposure while the second case had only five months. In the literature there are conflicting views concerning the effects of exposure times. In some studies, exposure time has been found closely correlated with clinical and electrophysiological abnormalities while in some studies it was denied. In some studies, exposure time has been found to be closely correlated with clinical and electrophysiological abnormalities; in others, it was denied.[11,22] Effects of age are not clear in our patients. The youngest had more severe electrophysiological abnormalities than older ones. The logical explanation is that the severity of abnormalities is correlated with the exposure intensity and personal differences of metabolism. In Turkiye, the patients who had nervous system dysfunction due to n-hexane exposure usually admitted during February and March like previously reported in other countries.[3,17] During winter months, factory doors and windows are closed because of cold weather, causing insufficient air circulation and accumulation of the volatile toxic substances in the air.[3] The quality of working environment seems crucial.

Needle EMG findings of our cases showed denervation potentials as the evidence of axonal injury in all patients but the case two, which has the shortest exposure time.

The neurotoxic effects of n-hexane and metabolites include headache, sleep disturbances, mental impairment, and spastic gait.[2,13] Hyperreflexia with spasticity may be observed in patients with polyneuropathy several months after recovery of peripheral neuropathy. The sequelae of spasticity may persist even several years after n-hexane exposure.[2] Evoked potential studies showed prolonged central conduction time supporting the central effects of n-hexane and metabolites.[23]

Even though our patients did not report any visual complaints, two of them had visual evoked potential examinations. Case 3 had prolonged p100 latency (Figure 2), while case 4 had normal examination results. Blurred vision, impaired color vision, retinal or macular changes have been reported in literature. Periodical clinical and electrophysiological scanning might be recommended to detect early changes in subclinically affected patients.

Currently, there is no specific antidote for n-hexane intoxication. Therefore, only treatment for it is prevention of further excessive exposure.[6,9,11,13] After cessation of the exposure, the deterioration may continue.[8,9,20] Likewise, our case 2 worsened clinically two months after he stopped working. The second electrophysiological examination showed increased axonal degeneration. Case 4 also continued to deteriorate despite termination of exposure for six months.

In occupational medicine, preventing diseases before they occur is more effective and easier. In occupational medicine, it is more effective and easier to prevent diseases before they occur. Because of that, safety of working places is very important. Until the education of workers to use safe and proper equipment is achieved; protective clothing (gloves), proper air conditioning, removing contaminated objects should be put in practice. People who were exposed to toxic substances should be periodically screened with clinical and electrophysiological examinations even if they are asymptomatic. Measurement of urinary 2, 5 hexanedione is also useful for detecting the toxic levels of exposure.[24] These would be most helpful to prevent associated diseases before they become decapacitating; therefore, would reduce the healthcare expenses and increase the rate of productivity.

Detailed patient history, toxic substance exposure, appropriate laboratory and electrophysiological examinations are required for correct diagnosis. After all the required examinations are performed, control examinations should be scheduled. Also, it is important that both sensorimotor polyneuropathy and motor neuropathy to be detected in n-hexane neuropathy. So, the patient should be asked about occupational and environmental toxic agent exposure during examination.

CONCLUSION

Neuropathy due to toxic agents is an important public health problem. Working place conditions must be strictly controlled if toxic materials are present. Patients who had electromyography compatible with polyneuropathy should be questioned for toxic exposure. Asymptomatic workers should also be examined. Patients should be followed for the possibility of "coasting" effect.

ETHICAL DECLARATIONS

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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