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Case Report

Glue Sniffing Neuropathy in an 18-Year-Old Male Butcher with a History of Weed Smoking: Case Report

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ABSTRACT
Glue sniffing neuropathy, a consequence of volatile substance abuse, presents with peripheral nerve damage. We present a case of an 18-year-old male butcher with a history of chronic glue sniffing and occasional marijuana use. He presented with a three-month history of progressive muscle weakness, small muscle wasting, hypotonia, hyporeflexia, and ascending quadriparesis. This case highlights the neurological repercussions of inhalant abuse, particularly in adolescents.

Keywords: Glue Sniffing Neuropathy, Volatile Substance Abuse, Peripheral Neuropathy, Adolescents, Substance Cessation, Rehabilitation.

INTRODUCTION

Deliberate inhalation of volatile hydrocarbons leads to mood elevation and the low cost, ready availability, and ease of use contribute to its popularity among adolescents. Volatile hydrocarbons are contained in glues, solvents, lighter fluid, gasoline and paints. Most inhalants can be abused because the propellants are volatile hydrocarbons. The methods of inhalation commonly are sniffing, huffing and bagging.[1,2] Glue sniffing neuropathy, a rare neurological disorder, results from chronic inhalation of volatile organic copounds. It manifests with peripheral nerve damage, cognitive impairment, and psychiatric disturbances.[1,2,3] In this report, we describe a case of glue sniffing neuropathy in an 18-year-old male with a history of glue sniffing and marijuana use. We emphasize the clinical features, diagnostic approach, management challenges, and the significance of early intervention.

Case Presentation

An 18-year-old male butcher presented with a three-month history of progressive muscle weakness, particularly in the distal extremities initially, the patient developed distal weakness which was insidious in onset and gradually progressive in nature. The patient initially noticed weakness while climbing stairs due to which patient stopped using stairs later, the patient noticed difficulty in gripping the chappals, the weakness later progressed to proximal muscles due to which patient noticed difficulty raising from the chair and difficulty in getting up from squatting position.The weakness progressed gradually with the lower limb being affected more than the upper limb. Slowly, the patient started noticing weakness in the upper limbs in the form of difficulty in opening the jar, difficulty in buttoning and unbuttoning of his shirt and difficulty in combing hair. patient also noticed numbness over the bilateral palms and soles due to which patient had multiple cuts over the fingers while cutting the meat at his shop, which made the patient to seek medical help.He had a chronic history of glue sniffing, predominantly with adhesives from his family's butcher shop, and occasional marijuana use.

Physical examination revealed small muscle wasting, hypotonia, hyporeflexia, and sensory loss in a stocking-glove pattern. Motor strength was diminished (3/5 MRC scale), with difficulty in fine motor tasks. The bilateral upper extremities were less affected than the lower limbs. Muscle strength in the bilateral proximal upper limbs was intact, while finger flexion and extension displayed a grade of 4+. Light touch sensation, proprioception, and pain and temperature sensation were grossly intact. Serologic test results revealed no definite abnormalities. The values were as follows: creatine kinase, 143 IU/L (normal range, 0 to 185); vitamin B12, 594.8 pg/mL (normal range, 232 to 1,245); folic acid, 4.1 ng/mL (normal range, 4.6 to 18.7); and homocysteine, 11.3 µmol/L (normal range, 5.0 to 15.0), HIV -non reactive. Motor nerve conduction studies (NCS) revealed delayed onset latencies and mildly slowed motor velocities, along with conduction

blocks in the bilateral median and ulnar nerves (Table 1). Sensory NCS indicated a slight sensory deficit, characterized by low amplitudes in the right median and bilateral ulnar nerves(Table 1).Needle electromyography demonstrated mild-to-moderate denervation potentials and reduced interference patterns in the right first dorsal interosseous, tibialis anterior, and vastus lateralis muscles. These electrodiagnostic findings are indicative of diffuse motor-dominant sensorimotor neuropathy with some demyelinating characteristics. RI ruled out central nervous system involvement. Laboratory tests were unremarkable. Glue sniffing neuropathy was diagnosed based on history, examination, and electrophysiological findings. The patient was referred to higher centres for further evaluation and treatment. One month after his initial discharge, the patient's motor weakness had regressed to pre-treatment levels, and he reported aggravated symmetric distal paresthesia. During his second hospitalization, a follow-up electro diagnostic study was conducted, revealing the progression of diffuse, motor-dominant sensorimotor neuropathy. Compared to the initial study, motor NCS showed greater delays in onset latencies and reduced amplitudes in the bilateral upper and lower extremities (Table 2). Sensory NCS of the distal upper and lower extremities could not be obtained (Table 2).Due to progressive worsening patient was further referred to higher centres for multidisciplinary rehabilitation but showed limited neurological recovery over the course of his treatment.

Table 1: Findings of the Initial Nerve Conduction Study

Nerve site	Onset latency (ms)		Amplitude (mV)		CV (m/sec)	
	Left	Right	Left	Right	Left	Right
Motor NCS						
Median Wrist Elbow Axilla	4.3* 9.5 12.1	4.2* 8.5 11.5	6.8 3.5 2.8*	7.8 2.4* 2.5*	- 42* 55	- 44* 53
Ulnar Wrist Below Elbow Above Elbow Axilla	3.1* 8.2 10.2 11.5	3.1* 9.5 11.5 13.8	12.5 11.5 7.2* 7.4*	11.2 9.2 6.5* 6.6*	- 45* 55 58	- 35* 45 65
Peroneal Ankle Below Fibula	6.2 14.5	4.5 14.8	10.5 3.5	11.5 3.6	- 36	- 39
Tibial Ankle Knee	4.5 14.4	4.5 14.5	10.5 3.5	11.5 3.4	- 36	- 41
Sensory NCS						
Median Finger-Wrist Palm-Wrist Wrist-elbow Elbow-axilla	3.5 2.2 3.6 1.8	3.1 2.1 3.5 1.8	9.3 15.4 25.6 110.5	6.4* 11.9 26.4 189.5	38 35 56 58	38 36 55 59
Ulnar Finger-Wrist Palm-Wrist Wrist-elbow Elbow-axilla	2.5 1.4 3.6 1.9	2.8 1.5 2.9 2.6	2.4 15.2 15.6 58.2	3.7* 11.6 26.5 52.1	36 38 55 59	24 36 54 56
Sural Claf	-	-	10.5	15.5	36	41

CV: conduction velocity; NCS: nerve conduction study. *Abnormal value.

Table 2: Findings of the Follow-Up Nerve Conduction Study

Nerve site	Onset latency (ms)		Amplitude (mV)		CV (m/sec)	
	Left	Right	Left	Right	Left	Right
Motor NCS						
Median Wrist Elbow Axilla	6.5* 11.5 14.8	6.1* 14.5 16.5	0.6 0.5* 0.4*	0.8* 0.5* 0.4*	- 43* 51*	- 28* 55
Ulnar Wrist Below Elbow Above Elbow Axilla	4.5* 9.5 12.5 14.8	3.6* 9.6 13.5 16.5	3.1* 2.2* 2.5* 1.5*	1.6* 1.8* 1.7* 1.3*	- 44* 45* 45*	- 40* 33* 39*
Peroneal Ankle Below Fibula	9.0* 20.6	7.6* 19.5	1.5* 0.5	1.4* 0.3	- 28*	- 28*
Tibial Ankle Knee	6.1* 16.5	5.8* 16.5	3.2* 0.5	2.2* 0.6	- 38*	- 32*
Sensory NCS						
Median Finger-Wrist Wrist-elbow Elbow-axilla	NR* 3.6 1.6	NR* 3.6 1.4	NR* 15.6 95.6	NR* 20.6 150.6	- 45 65	- 49 69
Ulnar Finger-Wrist Wrist-elbow Elbow-axilla	NR* 3.3 2.6	NR* 3.6 1.1	NR* 11.8 20.6	NR* 14.5 60.5	- 49 43	- 48 72
Sural Claf	NR*	NR*	NR*	NR*	-	-

CV: conduction velocity; NCS: nerve conduction study. *Abnormal value, NR: No response

DISCUSSION

Glue sniffing neuropathy, commonly known as n-hexane neuropathy has been well documented. Exposure to n-hexane in industrial solutions is known cause neuropathy, but inhalation of n-hexane present in the vapors is less well recognized as a neurotoxin to peripheral nerves. however that type of patients not seen that frequently. As there is acute to subacute worsening, the differential diagnosis also includes GBS. Glue sniffing neuropathy, a consequence of chronic inhalant abuse, is characterized by peripheral nerve damage. The primary neurotoxic agents are volatile organic compounds found in glues, paints, and other household products. Inhalants such as toluene, a common component in glue, act as neurotoxins by disrupting neuronal membranes, impairing axonal transport, and inducing oxidative stress. This process leads to axonal degeneration and demyelination, which in turn manifests as neuropathy.[1,2,3,4]

Glue sniffing, a form of volatile substance abuse, is a significant public health concern in India, particularly among vulnerable populations such as street children.[5,6] A scoping review and meta-analysis focusing on the South Asian Association for Regional Cooperation (SAARC) region found that India reported the highest incidence of glue sniffing, with a prevalence rate of 57% among certain groups.[7] However, specific data on the prevalence of glue sniffing-induced neuropathy in India are limited. The overall prevalence of peripheral neuropathy in various Indian community studies varies widely, ranging from 0.05% to 24%. This variation is influenced by factors such as diabetes mellitus, nutritional deficiencies, infections, and exposure to toxins, including substances used in glue sniffing.[8]

Given the high prevalence of glue sniffing in certain populations, it is plausible that a proportion of peripheral neuropathy cases in India are associated with this practice. However, due to the lack of specific studies

quantifying glue sniffing-induced neuropathy, the exact prevalence remains undetermined. Further research is necessary to elucidate the extent of this issue and to develop targeted interventions for affected populations.[1,2,3,4]

Glue sniffing neuropathy is primarily caused by chronic exposure to volatile organic solvents, particularly toluene, which is a neurotoxic agent found in many adhesives. These substances cause direct toxic damage to peripheral nerves and can also affect the central nervous system. The pathophysiology involves multiple mechanisms, including axonal dysfunction, myelin damage, and oxidative stress.[8]

The clinical course of glue sniffing neuropathy varies based on the severity and duration of the inhalant abuse. Common symptoms include progressive muscle weakness, particularly affecting the distal extremities, as well as small muscle wasting and sensory disturbances. In our patient, the initial symptoms began with distal weakness, progressing to more generalized motor deficits. Sensory loss followed a stocking-glove distribution, which is often seen in polyneuropathies. Autonomic dysfunction, such as postural hypotension and gastrointestinal disturbances, may also occur but was not observed in our patient. Cognitive impairment and psychiatric disturbances, including mood disorders and memory deficits, are additional features of the condition but were not present in this case. The delayed onset and insidious progression of symptoms often result in delayed diagnosis and management, underscoring the importance of early recognition and intervention.[8]

The diagnosis of glue sniffing neuropathy is primarily based on a thorough clinical history, neurological examination, and electrophysiological studies. In our case, the patient's history of chronic glue sniffing and muscle weakness provided the initial clues. Nerve conduction studies and electromyography (EMG) are crucial for confirming the diagnosis and assessing the extent of nerve damage. In this patient, these studies revealed sensorimotor polyneuropathy with reduced conduction velocities and prolonged latencies, consistent with axonal degeneration and demyelination.[8,9]

MRI of the brain and spinal cord is typically performed to rule out central nervous system involvement, but in the case of glue sniffing neuropathy, findings are usually nonspecific. Our patient's MRI results were normal, supporting the diagnosis of a peripheral neuropathy rather than a central nervous system disorder. Laboratory tests, including blood counts and metabolic panels, are important for excluding other potential causes of neuropathy, but our patient's results were unremarkable.

The management of glue sniffing neuropathy requires a multidisciplinary approach that addresses both the neurological consequences of inhalant abuse and the underlying psychosocial factors contributing to substance dependence. The cornerstone of treatment is the cessation of inhalant abuse, which can be challenging in cases of long-term addiction. Behavioral interventions, including substance abuse counseling and cognitive-behavioral therapy, are essential for achieving and maintaining abstinence. Pharmacological therapies, such as antidepressants or anxiolytics, may be required to address coexisting mood disorders and prevent relapse. Symptomatic treatments may be necessary to manage pain and discomfort associated with peripheral neuropathy. These can include the use of pain medications, such as gabapentin or tricyclic antidepressants, to control neuropathic pain. Orthotic devices may be used to assist with mobility and improve posture, while assistive devices such as wheelchairs may be necessary in more severe cases of muscle weakness.[8,9]

The prognosis of glue sniffing neuropathy is variable, with some patients showing significant improvement following the cessation of inhalant abuse and appropriate rehabilitation, while others may experience persistent neurological deficits despite treatment. The extent of recovery is influenced by factors such as the duration of inhalant abuse, the degree of nerve damage, and the timeliness of intervention.[8,9,10] In our case, the patient showed limited neurological recovery, which is consistent with reports in the literature indicating that the outcomes for glue sniffing neuropathy are often suboptimal. Long-term monitoring is necessary to assess for ongoing symptoms, prevent relapse into substance abuse, and address any emerging psychosocial issues that may impede recovery. It is also important to monitor for complications related to peripheral neuropathy, such as falls, infections, and contractures.

CONCLUSION

Glue sniffing neuropathy is a rare but serious neurological disorder that results from chronic inhalant abuse, particularly in vulnerable populations such as adolescents. This case

highlights the neurological consequences of glue sniffing and the importance of early diagnosis and intervention. Comprehensive management, including the cessation of inhalant abuse, supportive care, and rehabilitation, is essential for optimizing outcomes and reducing the long-term impact of the disorder. Preventive measures, including education and awareness campaigns, are needed to reduce the prevalence of inhalant abuse and mitigate the risk of developing glue sniffing neuropathy in adolescents.

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