GLUE SNIFFING NEUROPATHY AND REVIEW OF LITERATURE

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ABSTRACT

Glue sniffing neuropathy commonly known as n-hexane neuropathy. It is well documented that industrial exposure to n-hexane causes neuropathy, however it is less well recognized that inhalation of n-hexane present in the vapors can also cause neuropathy. However such patients are not seen that frequently. The acute worsening also generates differential diagnosis of GBS. Most of literature is reported from west .We report such case for the first time from Saudi Arabia. A 35 year old male presented to us with progressive numbness followed by weakness in both legs since last three weeks. Over next two week he became chair bound and in the beginning of third week he also stated to feel numbness in both the hands and some weakness was also noted in hands. His past history was significant for carpet cleaning glue sniffing for many years. His exam was significant for distal weakness feet greater than hands, deep tendon reflexes were absent all over. All sensory modalities showed glove and stocking pattern. Nerve conduction velocities showed slowing. His CSF exam was normal. We conclude that n-hexane is neurotoxic when inhaled to excess and, that the neuropathy has characteristic electrophysiological and pathological features.

Keywords: n-hexane, neurotoxic, guillain barre syndrome.

INTRODUCTION

Deliberate inhalation of volatile hydrocarbons leads to mood-elevation (euophoria)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 or bagging 1,2.

Hydrocarbons are commonly found in dry cleaning solutions, paint, spot remover, rubber cement, glue and solvents. Prolonged abuse of hydrocarbons results in white matter degeneration (leukoencephalopathy) and atrophy 3,4. In addition, prolonged exposure to certain hydrocarbons (eg, n -hexane) which is present in Glue results in peripheral neuropathy, blurred vision, sensory impairment, muscle atrophy, & parkinsonism 5.

CASE PRESENTATION

We report a case of a 35 year old male with progressive numbness followed by weakness in both legs since last three weeks. According to patients he was alright three weeks back when he started to feel numbness in his feet and in three to four days he noticed difficulty in walking and climbing up. Over next two week he became chair bound and in the beginning of third week he also stated to feel numbness in both the hands and some weakness was also noted in hands. No bowel and bladder involvement noticed.

Examination showed a normal higher mental functions, no cranial nerve involvement. Deep tendon reflexes were absent all over. On power assessment mild weakness in small muscle of hand (4/5) foot dorsi flexion and extension had 2/5 Hip and knee extension was 3 by 5. Plantars were non reactive. All the sensory modalities showed glove and stocking pattern. His past history was significant for glue sniffing for many years.

His nerve conduction showed demylinating type of neuropathy. Lumber puncture was done to rule out possibility of GBS .The CSF was normal.

The nerve conduction report was suggestive of sensory motor demylinating neuropathy wit axonal component.

NERVE CONDUCTION STUDY

Table1.1: Motor nerve conduction study

site	nerve	onset laten	Normal	Amplitude	norm Amp	Velosity	Norm Vel	F waves
Left								
Wrist	median	4.3	<4.5	6.7	> 4.5	36	>49	29.5
Elbow		11.6		4				
Right	Median							
Wrist		4.3	<4.5	7.2	>4.5	34	>49	28.77
Elbow		12		4.1				
Right	peroneal							
Ankle		6.9	<6.6	0.3	>2	30	>38	
Tibialis		19.5		0.2				
right	Tibial							
Ankle		9.1	<6.0	0.5	>4	30	>41	
Knee		25		0.3				
left	Ulnar							
wrist		5.9	<3.6	6.7	>5	44	>53	
B elbow A		12.1		5.5				
elbow		19.6		3.5				
axilla		23.8		2.9				
right	ulnar							
wrist		3.8	<3.6	5	>5	38	>53	
B elbow		10.2		5.1		27		
A elbow		15		3.7				

Table 1.2: Sensory Nerve Conduction Study

Site	Nerve	peak	norm peak	Amplitude	Norm Peak	Velosity	Norm velo
Right	median						
Wrist	2nd digit	3.8	<3.6	35.2	>15	34	
Right	ulnar						
Wrist	2nd digit	3.7	<3.1	23.7	>10.0	46	>38

EMG

	Muscle	spont act	CMAP	Int pat	Recuri
right	Ant Tibial	normal	normal	normal	normal
right	Gastroc	normal	normal	normal	normal

DISCUSSION

Glue sniffing neuropathy commonly known as n hexane neuropathy has been well documented. Exposure to n-hexane in industrial solutions is known to cause neuropathy, but inhalation of n-hexane present in the vapors is less well recognized as a neurotoxin to peripheral nerves However such patients are not seen that frequently. As there is acute to sub acute worsening, the differential diagnosis also includes GBS. Keeping this in mind on must inquire about addiction history. The treatment option also varies as it consists of stopping the abuse and rehabilitation. Plasmapherisis and IV IG G are not helpful. The damaged nerves are slow to recover and residual deficits may be left behind.

Review of literature showed mostly old case reports as with time it became a well established case. However the rarity of its occurrence or encounter makes it interesting. The earliest report was published in 1975, where 18 children in West Berlin had a toxic poly neuropathy due to sniffing glue thinner. The symptoms reached peak in 11 to 21 months. Clinically they presented as symmetrical, progressive, ascending, mainly motor, polyneuropathy with pronounced muscle atrophy. Seven patients presented with tetraplegia. Even after 8 months all patients still had a motor deficit. Nerve biopsy showed paranodal axon swelling, dense masses of neurofilaments and secondary myelin retraction. In the same year case of a young man with a long history of addictive glue-sniffing was published6. He developed severe distal symmetrical polyneuropathy several months after using cement containing n-hexane and gradually improved several months after when he switched to another cement containing no n-hexane7. Radial Nerve biopsy showed marked segmental distention of axons by neurofilamentous masses with secondary thinning of myelin sheath, paranodal myelin retraction. Nerve conduction velocities were found to be slow.

The other paper was published in 1985 in Australian and New Zealand Journal of Medicine, where three young men were reported, who developed severe, sub acute, predominantly motor peripheral neuropathy resulting from the deliberate inhalation of glue vapor. Weakness began after several years of daily glue sniffing. weakness was noted in both proximal and distal muscles. Prominent muscle wasting noticed at the time of presentation. Deterioration continued for several weeks after glue sniffing was stopped. Peripheral nerve conduction was markedly slow and there was extensive denervation in the muscles. Sural nerve biopsy showed changes as that mentioned above8. Classically the clinical presentation is that of poly neuropathy but N hexane toxicity may also affects cerebellum and it may even cause optic neuropathy and hearing loss9.

So in conclusion, while examining patients with neuropathy, one may also consider n hexane polyneuropathy in differential and good addiction history should be obtained so that cause of polyneuropaty can be detected.

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