

Case Report

Cerebellar Ataxia and MRI Abnormalities Associated with Glue Sniffing

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ABSTRACT

Glue sniffing, particularly pattex paste or betax glue, is an important social problem among teenagers and young adults. The chemical constituent of pattex paste, toluene, is a neurotoxin. We report a case of 22-year-old male patient who sniffed glue on a nearly daily basis for more than eight years. Three years following the start of his substance abuse, he began to develop slowly progressive tremor, slurred speech and unsteadiness on walking, which became quite severe, over the last four months prior to admission. Clinical examination revealed slurred speech and bilateral cerebellar signs with ataxic gait. CT

scan brain showed signs of brain atrophy. MRI brain revealed high signal changes in the subcortical and deep white matter, as well as diffuse periventricular and occipital lobe lesions. It also showed low signal changes in the thalami with cortical and cerebellar atrophy. These findings were suggestive of toxic encephalopathy. After he stopped glue sniffing, the patient's symptoms regressed significantly and he became more ambulant. In conclusion, this case demonstrates the possible reversibility of neurological manifestations of glue sniffing.

KEYWORDS: cerebellar signs; glue sniffing; neurotoxin; substance abuse

INTRODUCTION

Glue sniffing, involving solvent abuse among young adults and children, is a growing health problem in developing countries. Commercial contact cements such as spray paints or glue or pattex pastes contain n-hexane and toluene based products. They are popular among glue sniffers because of their euphoric effect, easy availability and relatively low cost. Glue sniffing becomes an alternative to alcoholic drinks, whenever not available, because it produces tranquility, relaxation, hallucinations and euphoria. N-hexane or toluene (methylbenzene) present in these products are neurotoxic and may result in persistent neurological abnormalities. Some patients abusing such products, however, showed spontaneous recovery, while others continue to have chronic residual deficits. In general, the management of such patients is challenging due to poor cooperation and their tendency to relapse to their former habit once discharged or substitute with other substances like alcohol or drugs.

We report herewith, a case of 22-year-old male patient with history of chronic glue sniffing, resulting in cerebellar signs and abnormal MRI changes in the brain.

CASE REPORT

A 22-year-old male patient, from a large joint family, presented with progressive difficulty in

walking and tremors of both hands especially during action. He gave history of chronic solvent abuse, in the form of glue sniffing (betax glue) for more than eight years on almost daily basis. He consumed a 50 gms glue tube, by inhaling the vapour coming out intermittently, which resulted in feeling of euphoria, relaxation overactivity, laughing, dancing, and sensation of flying in air, with visual and auditory hallucinations.

Three years after beginning his abuse of betax glue, he developed hand tremors, slurred speech and abnormal movements of both hands, which affected his dressing and eating ability. He also developed unsteadiness on walking, which was slowly progressive until the time of presentation in 1998.

Clinical examination revealed, a conscious, well-oriented patient, with a minimental score of 22/30. Fundi were normal. He had head titubation with staccato speech. His motor power was 4+ /5 in all four limbs with normal deep tendon reflexes and flexor plantar responses. He had coarse action tremors on stretching the arms, severe bilateral limb ataxia involving both upper and lower limbs, and was unable to walk without support because of unsteadiness. No extrapyramidal signs were demonstrable.

All blood tests including renal and liver function tests were normal. Serum anti HIV 1&2

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Fig. 1: FLAIR MR axial image (TR 11004, TE 184, T1 2200) showing bilateral diffuse and multiple discrete high signal lesions (arrows).



Fig. 2: T2 weighted MR axial image (TR 4000, TE 105) showing hypointensities in both Thalami and right putamen, dilated ventricles and atrophic changes (arrows).



Fig. 3: FLAIR MR axial Image (TR 11004, TE 184, T1 2200) showing enlarged Cerebellar Sulci, indicating cerebellar atrophy and dilated temporal horns (arrows).

were negative. Serum copper and caeruloplasmin levels were normal and there was no evidence of acanthosis. EMG and nerve conduction studies were normal with no evidence of peripheral neuropathy. EEG was abnormal showing focal slow and sharp wave activities in both frontotemporal areas with secondary generalization. Somatosensory evoked potential (SSEP) study revealed a slightly prolonged central conduction time with normal peripheral conduction, suggestive of slowing of conduction in the central sensory pathway. Visual evoked potential study (VEP) was within normal limit and brain stem auditory evoked potential study (BAEP) was normal. CT scan brain, revealed moderate dilatation of the ventricular system with prominent cerebrospinal fluid spaces and cerebellar foliae, indicating cerebellar atrophy. MRI brain axial T2 and FLAIR images showed subcortical and deep white matter lesions, as well as diffuse periventricular high signal lesions, more in the occipital lobes (Fig. 1). Low signal lesions were also seen in the lateral parts of both thalami and right putamen with dilated ventricles (Fig. 2). The FLAIR MR Axial Image showed enlarged cerebellar sulci indicating cerebellar atrophy, with prominent subarachnoid spaces and enlarged temporal horns (Fig. 3). The MRI findings were consistent with toxic leukoencephalopathy and brain atrophy.

After admission, the patient stopped sniffing glue, and was started on physiotherapy and enrolled in a rehabilitation program. On follow up, four months later, his gait and limb ataxia improved and he became more ambulant, however, he continued to have minimal bilateral hand tremors, with cerebellar signs.

DISCUSSION

Volatile substance abuse (VSA) (glue sniffing, inhalant abuse, solvent abuse) has now been reported from most parts of the world, mainly among adolescents, individuals living in remote communities and those whose occupations give ready access to abusable substances^[1]. Inhalant abuse, in general, is associated with mortality and morbidity, including social, educational and economic deprivation in adolescents and young adults^[2]. Glue sniffing is the common term but refers generally to the use of a variety of substances including spray paints, thinners, nail varnish removers, gasoline, marking pens and lighter fluids. Toluene and N-hexane are the most frequently involved neurotoxins^[3,4]. VSA gives rise to dose-related effects similar to those of other hypnotic sedatives. Small doses can rapidly lead to euphoria and other disturbances of behaviour, similar to those caused by ethanol (alcohol) and may also induce delusions and hallucinations. High doses may produce life-threatening effects such as convulsions and coma. Death may ensue indirectly after, for example, inhalation of vomit or from direct cardiac or CNS toxicity. Drunken behavior, unexplained listlessness, anorexia and moodiness may result from VSA^[1].

The inhaled vapours are drawn into the lungs and absorbed into the blood stream. The toxic compounds are lipophilic, attaching to the fats in the nervous system where they can be stored for a long time. They are also stored in the liver and kidneys and there are reports of oliguric acute renal failure and renal acidosis following solvent abuse^[5]. The neurotoxicity of hydrocarbons like toluene is dose related. The allowed legal maximum level for pure toluene vapour is 200 ppm. Above this level,

fatigue, headache, paresthesia and slowed reflexes appear. Levels above 600 ppm cause confusion or delirium. Euphoric effects appear above 800 ppm. Chronic abusers are usually exposed to levels well above 1000 ppm for a longer period^[3]. Multiple components in the mixtures can act in a synergistic or additive manner. Pure toluene is thought to act principally centrally, causing dementia and cerebellar ataxia while Aliphatic hydrocarbons such as n-hexane, which are found in many glue compounds (including glue made from neutral crepe rubber), are thought to cause predominantly peripheral nerve damage^[4,6,7,8]. The betax glue is contact cement containing a inflammable liquid petroleum mixture, and contains mainly toluene (methylbenzene). In the presented case, glue sniffing resulted in damage to the central nervous system, predominantly involving the cerebellum, which improved following cessation of substance abuse. The CNS involvement was demonstrated by abnormal electrophysiological and radiological tests. Since the patient had never been exposed to alcohol, his gross cerebellar deficits, which were temporarily related to his substance abuse, are certainly related to the habit of betax glue sniffing .

There are many earlier reports of solvent abuse, particularly toluene-based products, causing diffuse central nervous system and white matter changes^[9,10]. Rosenberg et al^[7] described diffuse cerebral, cerebellar and brainstem atrophy in patients with toluene abuse, including loss of differentiation between the grey and white matter and increased periventricular white matter signal intensity, on T2 weighted MRI images. The patients with diffuse white matter changes showed hippocampal atrophy with thinning and atrophy of corpus callosum^[9,11].

Some patients developed severe changes in the brainstem and cerebellum to cause neurological symptoms well before obvious brain atrophy. Caldemeyer et al reported diffuse brain atrophy, white matter T2 hyperintensity lesions and T2 hypointensity lesions involving the basal ganglia and thalami^[10,12]. There was no definite clinical evidence of damage to the basal ganglia and thalamus despite the MR imaging finding of T2 hypointensity lesions. Autopsy of brain in one patient who died due to acute toluene overdose revealed diffuse ill-defined myelin pallor, maximum in cerebellum, periventricular and deep cerebral white matter^[7]. There was mild gliosis and scant perivascular macrophage collections were seen. These findings suggest that the pathological and MRI abnormalities are due to either increased water content of the white matter or subtle toluene-induced metabolic changes in myelin. Katzelnick et al^[13] observed reversibility of psychiatric symptoms

in a chronic solvent abuser in one patient when he stopped abusing solvent. No similar reports regarding reversibility of neurological deficit clinically were found.

According to one study, the effects of MRI abnormalities on WAIS-R (Wechsler Adult Intelligence Scale-Revised) in substance abusers showed that there was a decline in verbal as well as performance IQ. The patients with white matter changes showed significantly lower performance IQ^[14]. In our patient, MRI imaging showed white matter changes, periventricular high signal lesions, cerebellar atrophy and low signal lesion in both thalami. Clinically, however, there were no correlating signs and symptoms other than persistent cerebellar deficits. There were no gross decline in verbal and performance IQ in our patient.

CONCLUSION

In conclusion, glue sniffing, particularly betax glue inhalation, affects the central nervous system, predominantly cerebellar system, causing persistent cerebellar ataxia as demonstrated in our patient. The clinical picture is often associated with radiological changes demonstrated on MRI consistent with toxic leukoencephalopathy. However, there is no clear anatomical correlation between the clinical findings and the MRI changes, consistent with other studies reported in literatures. Finally, this case also demonstrated the reversibility of the neurological symptoms and signs with discontinuation of glue sniffing, in addition to the possibility of reversibility of psychiatric symptoms as observed in one case report.

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