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Subacute Chorea Induced by Organic Solvents

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Abstract

Introduction: Chronic exposure to organic solvents may result in a variety of neurologic complications like dementia, cerebellar dysfunction, pyramidal syndrome, cranial nerve abnormalities, and neuropathy. **Clinical Presentation:** We report an unusual case of subacute chorea induced by occupational exposure. Brain magnetic resonance imaging showed diffuse white matter T2 hyperintensity. The screening of basic blood tests, and CSF studies to eliminate alternate diagnoses, were normal. The patient received 1000 mg/day of intravenous methylprednisolone for 3 days, with cessation of professional activity. We observed a regression of neurological symptoms after 3 months of follow-up. **Conclusion:** This case highlights the diversity of acute or chronic neurological complications of solvents.

Keywords

Acute Chorea, Diffuse White Matter T2 Hyperintensity, Organic Solvent Exposure

1. Introduction

Chorea is a hyperkinetic abnormal movement characterized by involuntary brief, random, and irregular movements, giving it a dancelike appearance. The etiology of adult-onset sporadic chorea is diverse and can be categorized into acquired and genetic causes. It is crucial to identify acquired causes, as many of them are treatable. However, the toxic causes are uncommon and evidence is mostly based on case reports or series. The organic solvent exposition is rarely reported as a cause of chorea. We describe a case of acute chorea after the organic solvent exposition.

2. Case Report

A 54-year-old man was admitted to the emergency department for the subacute

onset of generalized abnormal movements associated with the behavioral disorder and personality changes, 2 weeks before admission. He had no medical history and no remarkable family history. He worked as a professional painter for 15 years, with recently overexposure to solvents in a confined workspace without protection.

His general medical examination findings are body temperature of 36.6˚C, blood pressure of 122/77 mmHg, and regular pulse rate (75 bpm). The neurological examination revealed mutism with frontal lobe syndrome and generalized spontaneous movements that are irregularly timed, randomly distributed, abrupt, and predominantly proximal and making walking impossible, suggestive of chorea. His cranial nerves were unremarkably normal. The deep tendon reflexes were also normal.

Brain magnetic resonance imaging (MRI) showed diffuse symmetric hyperintensities in periventricular white matter sparing U-fibers, involving internal capsules, on T2-weighted and diffusion-weighted sequences. There were also more marked hyperintensities T2-weighted and hypointense T1-weighted on bilateral globus pallidus (Figure 1).

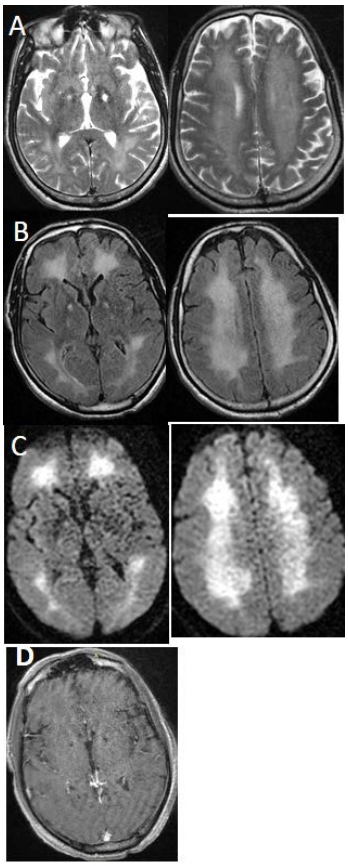

Figure 1. Brain MRI (A) axial T2-weighted, (B) axial fluid attenuation inversion recovery (FLAIR) images, and (C) proton density-weighted show bilateral symmetric extensive hyperintensities in the periventricular white matter, in the posterior limb of the internal capsule, in the centrum semiovale and the globus pallidus. Axial T1-weighted image with contrast (D) shows hypointense bipallidal lesion.

Inflammatory markers were not elevated (erythrocyte sedimentation rate 6 mm/hr and C-reactive protein 2 mg/L). Cerebrospinal fluid (CSF) analysis showed albuminocytologic dissociation with hyperproteinorachia at 1100 mg/L and normal white cell count (1Element /mm^3). Oligoclonal bands were absent.

The EEG showed a diffuse slowing down of the cerebral activity without any epileptic pattern.

Nerve conduction studies in Electromyography are normal

A computed tomography (CT) of the chest, abdomen, and pelvis, and a panel of tumor markers (anti-Hu, anti-Ri, anti-Yo, and anti-Ma antibodies) in the serum and CSE were negative. The Whole-body positron emission tomography (PET) was not done.

Given the radiological imaging, the normality of the blood test, and knowledge of the patient's overexposure to solvents in a confined workspace during 10 days, with no protective measures, preceding the neurological symptoms by 5 days, subacute solvent encephalopathy was the most probable diagnosis.

The patient received 1000 mg/day intravenous methylprednisolone for 3 days, in addition to a daily dose of 10 mg of Memantine with cessation of professional exposure.

Gradual improvement with complete regression of chorea after 2 weeks, progressive resolution of the frontal syndrome was noticed after 6 weeks. At 3 months follow-up, he had a normal neurological examination.

Control MRI showed mild regression of white matter abnormalities with moderate brain atrophy (Figure 2).

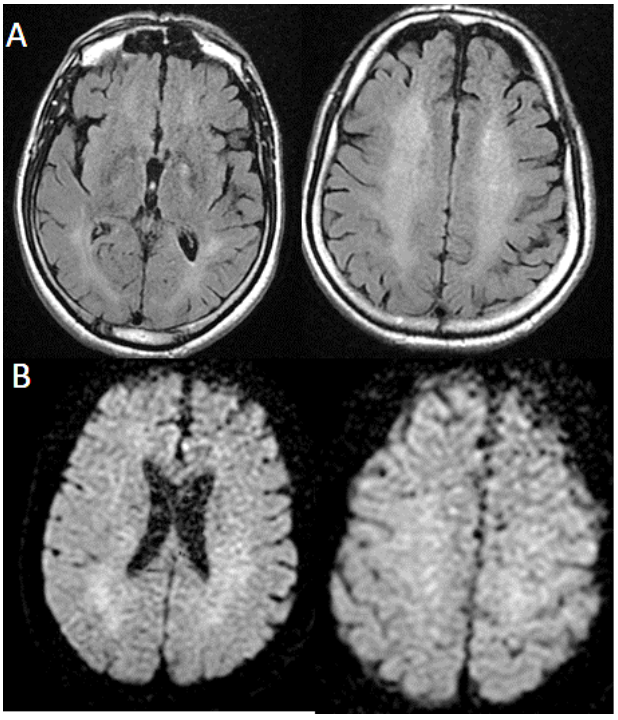

Figure 2. Brain MRI showing mild regression of hyperintensities and apparition of brain atrophy on the axial FLAIR (A) and proton density-weighted images (B).

3. Discussion

Organic solvents are volatile and lipophilic substances with low molecular weight. They are contained in many paint adhesives, inks, and cleaning products. The chemical properties of organic solvents rapidly penetrate the central nervous system (CNS) after inhalation [1]. The myelin of the enter ral and peripheral nervous system is rich in lipids, making it sensitive to damage by lipophilic substances such as volatile solvents [2].

In many countries, organic solvents are the most abused substances and are a public health problem in young people. Neurological complications due to illicit or occupational exposure to solvents are poorly defined. Chronic exposure to solvents can induce neurological toxicities such as demyelinating neuropathy, seizures, progressive encephalopathy, pyramidal syndrome, cerebellar syndrome, parkinsonism, and cranial nerve abnormalities [1] [2] [3]. The risk of experiencing acute neurological effects is also possible, in cases of high exposure (consumer overdose; confined workplace, or no personal protective equipment) [2] [3]. However, subacute movement disorders due to solvent exposure are uncommon, are rarely reported in the literature.

The most common causes of sporadic chorea are vascular disease, infectious diseases (AIDS, borreliosis…), metabolic disorders (non-ketotic hyperglycaemia or hyponatremia), autoimmune disease (systemic lupus erythematosus, Gougerot-Sjögren syndrome…), and genetic causes [4] [5].

The occurrence of Chorea is not rare after exposure to drugs such as neuroleptics, anticonvulsants, psychostimulants, and substance abuse (Heroin, Cocaine…) [1] [6] [7] [8]. However, chorea is an exceptional manifestation of organic solvent neurotoxicity unlike the parkinsonian syndrome [8].

The possible explanation for its occurrence in cases of solvent exposure is the increased dopamine concentrations in rat striatum and NMDA antagonism [8].

CT scan findings of the chronic solvent-exposed subject show cerebral atrophy correlated to long-term exposure [1] [10]. The MRI findings were mainly brain atrophy and the focal or extensive white matter hyperintensity on T2-weighted, and proton density-weighted (DW) sequences [10]. T2 hypointensity of the thalami and basal ganglia commonly attributed to the accumulation of iron, have also been reported [10]. Brain MRI is essential to exclude differentials diagnosis before concluding solvent-induced encephalopathy [1] [9].

Certain similarities between the lesions seen in brain MRIs of solvent abusers and reported findings of multiple sclerosis may suggest that demyelination is a neuropathological element in solvent neurotoxicity [10]. Therefore, white matter change is considered to represent damage in myelin similar to the demyelination reported in solvent abusers' autopsy reports [3].

Electroencephalogram (EEG) is abnormal in about fifty percent of the patients. EEG abnormalities that have been reported are slow activity and excessive beta activity, but there is no specific pattern to solvent toxicity [1].

Cerebrospinal Fluid Protein (CSF) analysis is necessary to exclude differential

causes. The albuminocytologic dissociation in CSF was frequently reported in the literature supporting the demyelination hypothesis [2].

Electromyography abnormalities that were observed in individuals exposed to solvents include a mixed sensory/motor neuropathy or inflammatory demyelinating polyradiculoneuropathy [2].

There are no specific treatments. Early diagnosis and cessation of exposure to solvents is the most effective therapy [1]. The hypothesis evoked in the literature, that the solvents caused the demyelinating lesions of the nervous system, explains the use of high doses of methylprednisolone in some cases [3].

Avoiding solvent abuse and preventing occupational exposure through the use of protective equipment and better ventilation of workplaces are the most effective way to prevent the neurotoxicity of solvent.

4. Conclusion

Solvent neurotoxicity in adults is well known with diverse clinical features. However, chorea is rarely described. Solvent neurotoxicity might not be apparent from routine toxicology tests and the diagnosis requires an exposure history, neurological symptoms, MRI findings, screening of basic blood tests, and CSF studies to discard alternate diagnoses. Improvement or stabilization of neurological symptoms after removal from exposure is another diagnosis support [1] [4].

Consent for Publication

Written informed consent was obtained from the patient's legal guardian(s). A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Conflicts of Interest

The authors declared no potential conflicts of interest concerning the research, authorship, and/or publication of this article.

References

[1] Rutchik, J.S. and Wittman, R.I. (2004) Neurologic Issues with Solvents. *Clinics in Occupational and Environmental Medicine*, **4**, 621-656. https://doi.org/10.1016/j.coem.2004.03.010

[2] Thulasirajah, S., Michaud, J., Doja, A. and McMillan, H.J. (2020) Inhaled Solvent Abuse Mimicking Chronic Inflammatory Demyelinating Polyradiculoneuropathy. *Child Neurology Open*. https://doi.org/10.1177/2329048X20934914

[3] Spina-França, A. (2008) Optic Neuritis Due to Solvent Abuse. *Arquivos de Neuro-Psiquiatria*, **66**, 108-110. https://doi.org/10.1590/S0004-282X2008000200041

[4] Cardoso, F. (2004) Chorea: Non-Genetic Causes. *Current Opinion in Neurology*, **17**, 433-436. https://doi.org/10.1097/01.wco.0000137533.53620.59

[5] Piccolo, I., Defanti, C.A., Soliveri, P., Volontè, M.A., Cislaghi, G. and Girotti, F. (2003) Cause and Course in a Series of Patients with Sporadic Chorea. *Journal of*

Neurology, **250**, 429-435. https://doi.org/10.1007/s00415-003-1010-7

[6] Brust, J.C. (2010) Substance Abuse and Movement Disorders. *Movement Disorders*, **25**, 2010-2020. https://doi.org/10.1002/mds.22599

[7] Miyasaki, J.M. (2011) Chorea Is Caused by Toxins. *Handbook of Clinical Neurology*, **100**, 335-346. https://doi.org/10.1016/B978-0-444-52014-2.00026-4

[8] Pappert, E.J. (2005) Toxin-Induced Movement Disorders. *Neurologic Clinics*, **23**, 429-459. https://doi.org/10.1016/j.ncl.2004.12.007

[9] Hurley, R.A. and Taber, K.H. (2015) Occupational Exposure to Solvents: Neuropsychiatric and Imaging Features. *The Journal of Neuropsychiatry and Clinical Neurosciences*, **27**, 1-6. https://doi.org/10.1176/appi.neuropsych.270101

[10] Keski-Säntti, P., Mäntylä, R., Lamminen, A., Hyvärinen, H.K. and Sainio, M. (2009) Magnetic Resonance Imaging in Occupational Chronic Solvent Encephalopathy. *International Archives of Occupational and Environmental Health*, **82**, 595-602. https://doi.org/10.1007/s00420-008-0368-3