**Case Study**

**Chronic Occupational Exposure to Organic Solvents and Magnetic Resonance Signal Changes in the Brain White Matter**

---A Case Report---

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**Key words:** Organic solvent, Occupational exposure, Magnetic resonance imaging (MRI), Central nervous system (CNS)

Chronic toxic effects of organic solvents on the central nervous system (CNS) is one of the major concerns for exposed workers. Although the World Health Organization has proposed diagnostic criteria, mainly focusing on psychological evaluation, for solvent-induced chronic toxic encephalopathy, definite diagnosis of the disease can seldom be made in cases of occupational exposure, given the non-specificity of signs and symptoms and difficulties in defining the exposure. On the other hand, it is generally easier to establish the association between exposure and clinical findings in solvent sniffers because of much more intensive exposure to relatively limited kinds of solvents compared to occupational cases.

Magnetic resonance imaging (MRI) has been validated as a useful measure to assess irreversible CNS injury caused primarily by toluene, but there have been few reports on MRI findings in patients suffering from chronic solvent intoxication resulting from occupational exposure. Here we report on a patient who had been exposed to organic solvents during his work, developed dysfunction of the CNS, and whose MRI findings were comparable to those of sniffing cases.

**Case History and Clinical Findings**

A 54-year-old man visited our clinic complaining of hyposmia, hypogeusia, poor memory, lack of concentration, and personality changes. He noticed diminished sense of taste and smell when he was 52 yr old after 12 yr of daily exposure to organic solvents on the job. He soon came to suffer from the initial symptoms, which gradually became prominent. About two months later, he was admitted to a hospital for syncope following nausea, headache, and vomiting during his work. No information was available on the airborne concentration or biological monitoring of the solvents during his work and on the day of admission. He had to quit his job on account of the persistent initial symptoms and was finally referred to our clinic.

He was alert and almost maintained complete orientation, but showed signs of left visuo-spatial agnosia, constructional apraxia, and disturbance of short- and long-term memory. Examination of the cranial nerves revealed optic nerve atrophy, bilateral concentric contraction, and bilateral hyposmia. He had poor coordinating movement with his extremities but did not look ataxic. Muscles were mildly hypertonic in the upper and lower extremities. He was normotensive. WAIS-R (Wechsler Adult Intelligence Scale-revised) scores were 65, 55 and 56 for verbal IQ, performance IQ and full scale IQ, respectively. Computed tomography showed decreased X-ray absorption in white matter surrounding the posterior horns of the lateral ventricles and in subcortical white matter in the parietal lobe. T2-sized MRI manifested increased signal intensity in the white matter in the same area. Signal intensity in the basal ganglia was normal. A proton-weighted imaging showed diminished gray/white differentiation (Fig. 1). Moderate cortical atrophy was observed in the front-parietal lobe in T1-sized imaging. These findings had not changed since his admission. The brain angiogram, electroencephalogram, auditory brainstem response, somatosensory evoked potentials, and peripheral nerve conduction velocities were normal. He had no history of drinking alcohol or sniffing any organic solvents.

**Occupational History and Description of Solvent Exposure**

We interviewed the patient for detailed information about his occupational history and exposure to organic solvents during his work. He started to work for a bicycle shop at the age of 16 after receiving education for 9 yr. When he was 32 yr old, he began to work as a welder and switched jobs again at the age of 35 to mixing skimmed milk with water in a food company. Since the age of 40, he had worked for a medium-sized company producing trim for automobiles.

He worked on a dashboard line with several other workers who were engaged there for a shorter period than he was. The procedure involved coating molds with 10 %v/v silicone dissolved in xylene by using a mop or a spray gun, pouring liquid resin (180–200°C) into the molds, and taking the hardened resin products out of them. The workers on the line then smoothed the product surface.
and painted the discolored areas with black ink mixed with thinner (the ingredients unknown). They made about 40 dashboards per hour and worked 9.5–11.5 h a day. The smell of the silicone-dissolving solvent and marking ink permeated the workplace, but they did not use any protective masks to protect against the solvents. The workplace had a few windows which were open in summer and closed in winter. No local ventilation equipment had been installed. Since the design of the workplace had already changed and all of the patient’s co-workers had quit their jobs by the time he visited our clinic, the investigation of his on-site solvent exposure was impossible and no information was available about the co-workers’ state of health.

Discussion

The present patient was diagnosed with chronic solvent intoxication based on the following information: 1) He had been exposed to the solvents for 12 yr at fairly high concentrations at work; 2) the symptoms appeared after he began to work with solvents and gradually became prominent; 3) the optic nerve atrophy found was suspected to result from exposure to methanol, one of the ubiquitous solvents in workplaces; 4) other organic diseases which could show hyperintensity lesions on T2-weighted MRI (e.g., brain infarction and adrenoleucodystrophy) were ruled out because his brain angiogram was normal and the appearance and distribution of the MRI lesions were inconsistent with those diseases.

The MRI findings reported here were similar to those of the sniffers inhaling almost pure toluene; T2-weighted imaging shows increased signal intensity in white matter with loss of the gray/white differentiation. Although the solvent identified in the present case was xylene, it was possible that he was exposed also to toluene because the thinner mixed with black ink, another source of exposure, was supposed to contain toluene which is the most common constituent of solvent products.

Thuomas reported that all of 32 occupationally intoxicated cases revealed decreased signals in the basal ganglia on T2-weighted images, whereas diffuse hyperintensity of the white matter with loss of the gray/white differentiation was observed in 11 cases. The former finding is not specific to chronic solvent intoxication, since it can also be found in other diseases such as multiple system atrophy and hyperglycemia. Moreover, such a finding can be well detected only with MRI systems which use a stronger magnetic field (1.5T ≥). Accordingly, although further studies are necessary to establish the dose-response relationship in the solvent-induced MRI findings, the present case suggests that the increased signal intensity on T2-weighted images in white matter with loss of the gray/white differentiation comes about as a result of occupational exposure as well as solvent sniffing, and that these MRI findings are valuable in making a diagnosis of chronic solvent intoxication in exposed workers, especially when detailed information about the exposure is not available.

The manufacturing process described in this paper is not extraordinary in the automobile industry. Attention needs to be paid to workers in medium- and small-sized industries where appropriate occupational health services are not provided.
References


