Polyneuropathy Caused by Chronic Exposure to Trichloroethylene

Key words: Trichloroethylene—Metal degreasing—Chronic poisoning—Polyneuropathy

Trichloroethylene is widely used as a solvent in industry. It is mostly used for degreasing metal parts before painting or gilding, and there are many reports on its toxicity. Reports on the chronic toxicity of trichloroethylene, however, are much fewer than those on its acute toxicity or sequelae of acute poisoning. Nevertheless, the chronic neurotoxicity of trichloroethylene has recently received much attention from the viewpoint of occupational health, as well as its carcinogenicity and its association with pneumatosis cystoides coli. There is increasing evidence that senile dementia or cerebral atrophy might be caused by chronic exposure to trichloroethylene. There are also several reports that peripheral nerve function could be impaired by chronic occupational exposure to trichloroethylene. Recently, we experienced a case of polyneuropathy which was assumed to have been caused by chronic exposure to trichloroethylene.

The patient, a 51-year-old woman, had been employed in a factory where metal screws and washers for automobiles were manufactured. The patient had been exposed to a high concentration of trichloroethylene for about 12 years. Her chief complaints were distal dominant paresthesia and pain in the extremities, paresthesia around the mouth, and headache in the occipital region. The course of her main symptoms and her history of exposure to trichloroethylene are shown in Fig. 1. The patient started work at the factory in July 1970. Since that time up to August 1982, she had been engaged in dipping baskets containing metal screws and washers (about 10–15 kg) into an open bath of warm (80°C) trichloroethylene as shown in Fig. 2. Soon after the start of employment at the factory, she had experienced symptoms of fatigability and sleepiness. It became difficult for her to prepare her family’s dinner without sleeping for 30–60 minutes after returning home from the job, and she had suffered heavy headaches after degreasing the screws and washers. She felt drunk and dizzy during the cleaning of the trichloroethylene bath, and this sometimes necessitated an about 30-min rest outdoors. She noticed a cool sensation in her feet and hands after 1972, and at times she had to sleep wearing a pair of socks in bed even in the summer because of this sensation. After 1975, it became necessary for only the metal screws to be degreased in the factory, but the amount of work was increased. In 1977, she noticed distal dominant paresthesia in the feet and hands, a sensation of swelling in the soles and paresthesia around the mouth, and these symptoms became gradually aggravated. The pain in her soles became very severe around 1979, and she ordered many pairs of shoes expecting that this would relieve
She noticed hypersensitivity to daylight, and sometimes suffered from diplopia, which did not improve upon wearing of spectacles for the aged. Around 1981, her husband and children noticed that her snore had become louder and that she looked very tired. In November 1981, it became difficult for her to stretch her left 3rd and 4th fingers, and she was diagnosed as having tendovaginitis in the left hand, for which she underwent surgery. After the
operation, she stopped handling heavy materials, and her work in degreasing the metal screws was reduced. However, the symptoms were further deteriorated in 1982 and she noticed that her grasping power in both hands had weakened. She therefore visited Nagoya National Hospital in August 1982. She had no particular past medical history except for appendicitis in 1955, and no particular family history except for her grandfather's heart disease and asthma. The first medical examination in the hospital demonstrated the following findings: Her muscle strength and tendon reflexes of the extremities were moderately to severely weakened; coordination was slightly clumsy and slow; gait was slightly paretic; ophthalmological examination showed no abnormalities except for hypermetropia; psychological examination showed that her impressibility was slightly to moderately impaired; EMG showed complex NMU voltage and high amplitude voltage at full voluntary contraction; motor nerve conduction velocities (MCVs) of extremities were slightly delayed; MCV in the N. tibialis was 40.3 m/sec (normal range 41.3–55.6), and SCV in the N. ulnaris and suralis were 57.4 m/sec (63.2–77.6) and 35.4 m/sec (39.1–51.9), respectively; findings of laboratory examinations demonstrated no abnormalities except for slight increases in serum cholinesterase (1.34 μH), triglyceride (204 mg/dl), Na (150 mEq/l) and Cl (110 mE/l) in blood.

The degreasing process in the factory had usually been maintained by the patient alone, and sometimes assisted by the factory manager. The degreasing was carried out for 5–6 hours a day, 6 days a week, and the cleaning of the trichloroethylene bath took about 15 minutes, once a day, twice a week. Trichloroethylene used in the factory was analyzed by gas chromatography, and its purity was more than 99%. Concentrations of trichloroethylene in the air of the workplace were measured when the factory manager was degreasing the metal screws. The air samples were collected in 5-liter Tedlar bags at the positions indicated in Fig. 2. The samples were analyzed by FID gas chromatography, and the results are shown in Table 1. The concentrations in the breathing zone of the worker while bending over the bath were very high (579 and 792 ppm), while the concentration about 10 cm below the upper rim of the bath was much higher (2099 ppm). The worker unavoidably breathed the trichloroethylene vapor

<table>
<thead>
<tr>
<th>Sampling place</th>
<th>Concentration (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) worker's breathing zone while standing in front of the bath</td>
<td>16</td>
</tr>
<tr>
<td>(2) worker's breathing zone while bending over the bath</td>
<td>792</td>
</tr>
<tr>
<td>(3)</td>
<td>579</td>
</tr>
<tr>
<td>(4) 5-10 cm beneath rim of the bath</td>
<td>2099</td>
</tr>
<tr>
<td>(5) outdoors, near a ventilation fan</td>
<td>131</td>
</tr>
<tr>
<td>(6) one meter distant from the bath</td>
<td>4</td>
</tr>
</tbody>
</table>

(Numbers in parentheses indicate the positions shown in Fig. 2)
about 10 cm or more below the rim while cleaning the bath, and the patient had not worn any respirator or protective gloves. The cooling pipe of the trichloroethylene bath was not provided with water.

Trichloroethylene is well known to have a narcotic effect and to cause multiple cranial nerve lesions, especially trigeminal analgesia. Severe trigeminal nerve analgesia and multiple cranial nerve lesions caused by trichloroethylene anesthesia have been reported, but these complications were considered to be produced by dichloroacetylene formed in closed circuits with soda lime, and not by trichloroethylene itself. However, there have been several reports on peripheral nerve impairment in the extremities due to chronic occupational exposure to trichloroethylene. For example, Ohtahara et al. investigated 6 workers chronically exposed to high concentrations (420–990 ppm) of trichloroethylene in a dry cleaning factory. They found paresthesia of extremities in all of them, and emphasized that peripheral nerve involvement might be important in chronic trichloroethylene poisoning. Shimizu et al. reported a male worker, 48 years old, who had been exposed to trichloroethylene for about 10 years, and who had developed paresthesia and muscle weakness in the extremities, spontaneous pain in the lower extremities and hyperesthesia around the mouth, and that EMG examination revealed a remarkably decreased NMU and a high amplitude action potential at full voluntary contraction. However, pathohistological findings of the peroneal nerve showed only a slight axonal degeneration in contrast to the severe clinical signs. Tanabe et al. exposed rats to 2000 ppm trichloroethylene, 8 hours a day, 6 days a week for 16 weeks. The nerve conduction velocity in tail was impaired after 14 and 16 weeks, but the impairment was much less severe than that caused by n-hexane. From these reports, it can be inferred that the peripheral nerve impairment caused by trichloroethylene is less severe than that occurring in n-hexane polyneuropathy but accompanied by more severe central nervous system involvement. However, it can be considered that peripheral nerve impairment is one of the important signs in chronic trichloroethylene poisoning.

REFERENCES

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