Myoclonic Encephalopathy after Exposure to Trichloroethylene

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Abstract: Trichloroethylene is a widely-used industrial solvent that is absorbed through the digestive or respiratory tracts or cutaneously. It has a selective tropism for the cardiovascular and central nervous systems and may cause death due to cardiac arrest or neurological sequelae. We present the case of a 25-yr-old woman who was exposed to trichloroethylene in the workplace for 18 months and who developed a disabling myoclonic encephalopathy. Non-toxicological causes were excluded. Although the exposure ceased, the disease progressed with thalamic and cerebellar involvement. The patient, who had only a partial response to symptomatic treatment, suffered severe limitations in the activities of daily living and was registered as permanently disabled due to a work-related disability.

Key words: Trichloroethylene, Trichloroethylene poisoning, Myoclonic encephalopathy, Neurological sequelae

Introduction

Trichloroethylene (TEC) is a colourless, non-inflammable fluid with a sweetish aroma, which is converted into extremely-toxic agents such as phosgene, chlorhydric acid and chlorine when it comes into contact with hot surfaces or flames. It is a solvent widely used as a degreaser of metal products and has also been used for dry-cleaning1). TEC can be absorbed by the cutaneous, digestive and respiratory routes. Most of the TEC absorbed metabolises to trichloroethanol and trichloroacetic acid and is eliminated by the kidneys. Its half-life is around four days.

Myoclonic encephalopathy caused by TEC toxicity has been previously reported. We report a case of myoclonic encephalopathy after exposure to TEC in a patient employed in a shoe shop, who used it to remove labels from shoes and stains from the carpet and floor of the workplace.

Case Report

The patient was a 25-yr-old woman with an unremarkable medical record, who had smoked 10 cigarettes a day for six years and did not drink alcohol or use addictive substances.

At 21 yr of age she was employed as a shop assistant by a shoe shop located in Barcelona (Spain), where she worked for 18 months. During the sales period, she had to replace the old price tag on the shoes with new ones by rubbing them with TEC on a rag until it was removed. She used a mean of 1.5 l of TEC (purity 99.9%) per day. This work was carried out in a small room (thirty cubic meters) in the basement of the workplace without any ventilation system. The patient had no individual protection (gloves, mask, etc). This exposure to TEC lasted 4 to 8 h daily for a period of 8–10 d. In addition, the patient spent 15–30 min every two days cleaning the carpet of the shoe shop with a rag impregnated with TEC, rubbing the floor and the carpet to remove sticky paper, chewing gum and spots of fat. During the final weeks
of her employment, the patient began to complain of intense headaches, asthenia, irritability and upper respiratory tract irritation that she associated with the use of the solvent. For this reason, she spontaneously decided to leave her job, and found employment in a clothes shop, where she had no contact with chemical agents. She continued to suffer headaches.

Two weeks after leaving the shoe shop, the patient lost consciousness at home and was admitted to the Emergency Department (ED) of the local hospital. She presented with involuntary myoclonic-type movements in all four limbs and left-sided weakness. The physical examination was normal except for anomalous movements in all four limbs, predominantly on the left side. The blood count and blood biochemistry were normal. Brain computed tomography (CT), magnetic resonance imaging (MRI), electroencephalogram (EEG), electromyography (EMG) and somatosensory evoked potentials were within normal limits. Cerebral perfusion, SPECT (Single Photon Emission Computed Tomography) and psychometric studies were also normal. Administration of clonazepam 3 mg daily allowed partial control of the myoclonic movement and the patient was discharged after five days.

In the following six weeks, the patient continued to suffer from almost constant headaches, moderate asthenia and weakness in the left limbs, although the myoclonic movements had greatly improved. A cerebral PET (Positron Emission Computed Tomography) scan (Fig. 1) during follow-up detected a reduction in global cerebral metabolic activity and a deficit of focal metabolism in the right subthalamus with mild thalamic asymmetry. At the cortical level, there was reduced metabolism in the right hemisphere. There were no deficits or asymmetries in the basal ganglia. A further neurophysiologic study showed electromyographic activity compatible with spontaneous and action myoclonias and normal corticospinal conduction.

One month later, the patient was admitted to the ED due to increasingly intense headaches, confusion, speech difficulties and unstable gait. The neurological examination showed positive Romberg’s sign, myoclonic movements in the left arm and poor coordination in the left leg. The fundus oculi examination was normal. The confusion remitted spontaneously. Additional tests excluded prion encephalopathy, Creutzfeldt-Jakob disease and myoclonus epilepsy associated with ragged-red fibres. A further neurological examination showed a static, dynamic ataxia that prevented the patient walking without help. Further EEG, brain MRI, blink reflex, auditory, visual, and somatosensory evoked potential tests were normal.

Treatment with sodium valproate was started and the headaches, myoclonus and dynamic ataxia improved, allowing the patient to walk with help. Neuropsychological tests showed cognitive deterioration with a deficit of hand-eye coordination and associative memory.

Two years after the diagnosis of myoclonic encephalopathy secondary to occupational exposure to TEC, the patient continues to suffer from continuous headaches, asthenia, motor difficulties and unstable gait. The involuntary movements are well-controlled by treatment with sodium valproate, clonazepam and a muscle relaxant.

Discussion

Myoclonic encephalopathy has been reported in a series of toxic conditions due to bismuth, methyl bromide, manganese, lead, mercury, colloidal silver, rodenticides containing alpha-chloralose, drugs such as dopa, lamotrigine, carisoprodol, cefuroxime and lithium, and mushroom ingestion. Myoclonic encephalopathy has been reported in various other conditions, such as anoxic encephalopathy following cardiac arrest, head trauma, stroke, tumors, and degenerative or metabolic (especially uremia) central nervous system diseases.

TEC may cause damage to both the central and peripheral nervous system. It is a highly volatile, liposoluble solvent that may cause central nervous system depression with great affinity for the cranial nerves, most frequently the trigeminal, optic and facial nerves and, exceptionally, other cranial nerves, leading to polyneuropathy, cerebellar tremor and extrapyramidal deficits. Cases of central vestibular involvement have also been described. Reported sequelae of acute poisoning include headaches, amnesia, psychosis, limb weakness and hemiparesis.

Chronic exposure may lead to diffuse cerebral damage.

Fig. 1. A cerebral PET detected a reduction in global cerebral metabolic activity and a deficit of focal metabolism in the right subthalamus with mild thalamic asymmetry. At the cortical level, there was reduced metabolism in the right hemisphere.
which manifests as headaches, asthenia, anorexia, vertigo, severe memory loss, depression, emotional disorders and, in more serious cases, clinical manifestations compatible with dementia. Asymmetrical neurological involvement means that in some cases the symptoms are compatible with multiple sclerosis. TEC potentiates the hepatotoxic effect of ethanol and may cause an anti-abuse effect when alcohol is ingested before during or after exposure. TEC is a human cancerigen. Damage to other systems may include contact dermatitis and skin burns, disorders of the cardiac rhythm and pulmonary, hepatic and renal alterations.

The use of TEC, even sporadically, in badly-ventilated spaces without personal protection can lead to severe or even fatal intoxication. In spite of its high level of toxicity, TEC is widely used and easily available in Spain and Europe for use as a stain remover, solvent and grease remover. According to data from the ECSA (European Chlorinate Solvent Association www.eurochlor.org), more than 52,000 tons of TEC were used in Europe in 2002.

Values of free trichloroethanol in blood (biological limit: 4 mg/l), trichloroacetic acid in urine (biological limit: 100 mg/g creatinine) and trichloroacetic acid + trichloroethanol in urine (biological limit: 300 mg/g creatinine) should be determined to aid diagnosis of the poisoning and biological control of exposed workers. In the case presented here, no toxicological analysis was available because the elimination half-life of TEC is very short and the patient did not go to the hospital until a relatively long time after the exposure. Neither were environmental analyses of TEC in the workplace available, as the exposure did not occur on strictly industrial premises but rather in a small shop which used TEC sporadically as a solvent and was not subject to obligatory environmental monitoring.

This case emphasizes a possible unusual neurological presentation of occupational exposure to TEC.

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