Case Study

Acute Pulmonary Injury due to Exposure to a High Concentration of Trichloroethylene Vapor

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We present the case of a 24-year-old male factory worker who became unconscious while he was cleaning the wall of a chamber where vacuum tubes had been washed using 100% trichloroethylene solution. He was rescued about one hour after he had been exposed to a high concentration of trichloroethylene vapor while he was lying unconscious in the left decubitus position on the floor of the chamber.

At the time of admission to our institution, his laboratory data and chest X-ray suggested acute pulmonary injury. His laboratory data showed elevation of white blood cell count (12,700/µl), neutrophil, 69.2%; lymphocyte, 19.2%; monocyte, 2%; CD4/CD8 ratio, 1.9. The histologic examination of a sample from transbronchial lung biopsy showed some lymphocyte infiltrates, fibrous thickening of the interalveolar septa and vacuolation of the cytoplasm of alveolar type II pneumocytes. Urinalysis one day after admission demonstrated a high concentration of trichloroacetic acid. The patient recovered from pulmonary injury and was discharged from ICU on the 15th day after admission.

To our knowledge, this case is the first report of acute pulmonary injury in relation to long standing exposure to a high concentration of trichloroethylene in a human. In Japan, workers sometimes work with a towel covering their noses and mouths. We suggest they should use a respirator as the towel habit is potentially dangerous.

Case

The patient was a 24-year-old healthy man who was engaged in room cleaning work. He became unconscious while he was cleaning without a respirator the dust from the wall of a chamber where vacuum tubes had been washed with 100% TCE solution. He had done the same work sometimes in the past, but he had experienced nothing abnormal. When he was rescued after being exposed to TCE vapor for about one hour, he was found unconscious in the left decubitus position on the floor of the chamber. Subsequently, he was immediately brought to our hospital by ambulance.

He had been working with a floorcloth moistened with a 100% TCE solution at a depth of about 2.5 m in the cylindrical chamber, which has a diameter of 90 cm, and a height of 4.85 m. The concentration of TCE vapor was 3,000~4,000 ppm at the level of his mouth and 15,000 ppm at the floor of the chamber. The maximum allowable concentration at the workplace is less than 25 ppm in Japan. When rescued, he was feeling drowsy (Glasgow Coma Scale 6). Vital signs were as follows: blood pressure, 100/72 mm Hg; pulse rate, 72/min with a regular rhythm; and oxygen saturation was 99% supported by mask saturation 10 L/min. When admitted to our hospital, arterial blood gases revealed acute lung injury (P/F oxygen ratio was less than 300). Electrocardiogram findings were within the normal range. Laboratory data showed elevation of white blood cell count (12,700/µl) and creatine kinase (334 U/l) but transaminase, amylase and bilirubin were normal. A chest radiograph showed an interstitial shadow in the whole left lung. A computed tomograph (CT) revealed ground glass opacity of the left upper lobe. A bronchoalveolar lavage (BAL) and transbronchial lung biopsy (TBLB) were performed the next morning since the interstitial shadow revealed by pulmonary high resolution CT scan had not disappeared. Macroscopically, the bronchial mucosa appeared somewhat swollen and BAL fluid was slightly blood-tinged. The analysis of BAL fluid was total cell count, 4.8 x 10³/ml; alveolar macrophage, 89%; lymphocyte, 8%; neutrophil, 3%; CD4/CD8 ratio, 1.9. The histologic specimen obtained by TBLB showed edema of the bronchial mucosa, some red blood cells in the alveolar space and vacuolation of the cytoplasm of alveolar type II pneumocytes (Fig. 1). The concentration of trichloroacetic acid measured by the method of gas chromatography in the 24-h urine was 128.6 µg/ml on the first admission day, 699.0 µg/ml on the second, 467.9 µg/ml on the third and 284.0 µg/ml on the fourth; that of...
serum chloral hydrate, a metabolite of TCE measured by the method of headspace-capillary gas chromatography mass spectrometry, was 7.5 µg/ml, 18.0 µg/ml, 7.2 µg/ml and 2.0 µg/ml on the same respective days (Fig. 2). The patient recovered from the pulmonary injury by oxygenation and treatment with antibiotics, he was discharged from the ICU on the 15th day after admission.

Discussion

Trichloroethylene (TCE) is an organochlorine agent characterized by its strong odor. It is a colorless liquid, very soluble in organic solvents and lipids. Because it efficiently removes grease, it is at present widely used in industries as a solvent agent and cleaner\(^1\). TCE is well known to cause liver disorder and to have an anesthetic effect, but there have been few reports regarding acute lung injury caused by TCE\(^2-4\).

Urine trichloroacetic acid and serum chloral hydrate are very valuable as an indexes of biochemical exposure\(^5\). Our patient was massively exposed to TCE according to the laboratory data which revealed high concentrations of both compounds. Forkert et al. reported\(^6\) that TCE produces bronchiolar damage when administered to mice. Much the same, Kurasawa reported\(^7\) selective damage of pulmonary nonciliated bronchiolar epithelial (Clara) cells by TCE in rats. Using Maclura pomifera agglutinin stain, he observed that TCE caused highly selective damage of nonciliated bronchiolar epithelial (Clara) cells, which became flat, and time-course studies conducted with exposure to 8,000 ppm of TCE to ethanol-treated rats demonstrated that maximal Clara cells damage occurred between 8 and 22 h after exposure. As the patient’s lung specimen was obtained about 25 h after exposure, there is a possibility that vacuolation of the cytoplasm of alveolar type II pneumocytes was the result of long standing exposure to a high concentration of trichloroethylene.

Nakajima et al. reported that acute trichloroethylene poisoning induced the elevation of white blood cell count, transaminase, amylase, lactate dehydrogenase and creatine kinase in a human\(^8\), but in our case, the level of transaminase and amylase were normal.

Our patient was asymptomatic 20 months after the accident, but we must follow up on his case, because recently, it has been reported that TCE induces pulmonary fibrosis in mice\(^9\) and cancer in animals\(^10\).

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