Pulmonary Reactions Caused by Welding-Induced Decomposed Trichloroethylene*

Bengt Sjögren, M.D.; Nils Plato; Rolf Alexandersson, M.D.; Anders Eklund, M.D., F.C.C.P.; and Claes Falkenberg, M.D.

This is the report of a welder who performed argon-shielded electric arc welding in an atmosphere containing trichloroethylene. He developed immediate respiratory symptoms, pulmonary edema 12 hours after exposure, and recurring dyspnea ten days after exposure. These pulmonary reactions might be explained by inhalation of decomposition products of trichloroethylene such as dichloroacetyl chloride and phosphene. (Chest 1991; 99:237-38)

TIG = tungsten inert gas; BAL = bronchoalveolar lavage

Welding in air contaminated with trichloroethylene might generate phosgene and dichloroacetyl chloride which after inhalation may cause pulmonary reactions. Such case reports seldom occur and hopefully this reflects the true incidence of this type of lung injury.1 The present case demonstrates the hazards of welding in an atmosphere containing trichloroethylene.

CASE REPORT

A 50-year-old man, free of medication, had worked as a welder for the last 25 years and was regarded as skilled. He had smoked about 40 cigarettes daily between 1960 and 1984 but he no longer smoked. The same year he was treated for a pneumonia caused by streptococci.

On July 30, 1987, he welded a stainless steel detail with his tungsten inert gas (TIG) equipment. Welding with TIG is an electric arc welding method using argon as a shielding gas. The steel had previously been washed with trichloroethylene that was often used as a degreaser. Almost instantly after starting this welding operation he experienced a sweetish flavor and severe dyspnea. He moved some yards away from the work bench but remained in the room. After some minutes he felt better and returned to his welding activities. This TIG-welding procedure was performed without any local exhaust ventilation as this would have disturbed the shielding effect of the argon gas. The duration of this welding procedure was six to ten minutes. There were no respiratory symptoms during the rest of the day or early evening.

During the night he experienced severe dyspnea and was admitted to an intensive care unit (ICU) with a clinical and roentgenologic picture of pulmonary edema (Fig 1). His temperature was 36.0°C and the white blood cell count (WBC) was 21,700/μm. The arterial Po2 and PCO2 were initially 23 mm Hg and 59 mm Hg, respectively, despite 8 L of oxygen per minute on mask. Some hours later the Po2 had increased to 48 mm Hg. He was also treated with diuretics, heparin, and penicillin G (benzylpenicillin) 3 g intravenously four times daily. The pulmonary edema could not be explained by cardiac dysfunction as there was no cardiac enlargement and the pulmonary vessels were of normal size.

On Aug 4, 2 g of ampicillin four times daily was added as three blood cultures had shown growth of enterococci, sensitive to ampicillin. The next day he was transferred to an ordinary medical ward. Two days later treatment was started with prednisolone 80 mg daily.

On Aug 9, he was readmitted, due to pulmonary edema with respiratory failure, to the ICU for treatment with a respirator with positive end-expiratory pressure. His temperature was 36.5°C and the WBC was 15,100/μm. The Po2 was 35 mm Hg and the PCO2 was 38 mm Hg immediately prior to respirator treatment. Artificial respiration was continued for five days.

Bronchoscopy was performed on Aug 11. The right main bronchus had an ordinary appearance but on the left side the mucosal membrane was edematous. Bronchoalveolar lavage (BAL) was performed in the right upper lobe with 200 ml (4 × 50 ml) of sterile saline solution; 80 ml was recovered. The differential cell count was as follows: monocytes/macrophages, 40 percent; lymphocytes, 8 percent; and neutrophils, 51 percent. Normal values in our laboratory (medians and interquartile ranges) are as follows: monocytes/macrophages, 89 percent (84 to 94 percent); lymphocytes, 9 percent (5 to 12 percent); and neutrophils, 1 percent (0.6 to 2 percent), respectively.1 Bronchial secretion showed sparse growth of Klebsiella oxytoca and very sparse growth of β-lactam-producing Staphylococcus epidermidis. Cultures regarding legionellae, Mycoplasma and Mycobacterium tuberculosis were all negative. Pneumocystis carinii was not found.

As the patient’s condition improved the dose of prednisolone was

*From the Departments of Occupational Medicine (Dr. Sjögren and Mr. Plato) and Thoracic Medicine (Dr. Eklund), Karolinska Hospital, Stockholm, Sweden; the Department of Work Science, The Royal Institute of Technology (Dr. Alexandersson); the Department of Internal Medicine, St Göran Hospital (Dr. Falkenberg); Stockholm, and the National Institute of Occupational Health (Dr. Sjögren), Solna, Sweden.

FIGURE 1. Chest roentgenogram (bedside) taken on July 31, 1987, about 12 h after exposure.

FIGURE 2. Chest roentgenogram taken on Sept 11, 1½ months after exposure.
Table 1—Lung Function Measurements before and after Exposure to Decomposed Trichloroethylene*

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*Percent of predicted values are given within parentheses.

redruced stepwise. When he left the hospital on Sept 17, he was taking 30 mg of prednisolone daily. The chest roentgenogram gradually became normal (Fig 2).

One and a half year after the exposure the welder experiences dyspnea when he walks fast or when he pulls elk to the camp during the hunting season. He has no cough. Lung function data are given in Table 1.

Exposure Data

Inhaled trichloroethylene is biotransformed to trichloroethanol and trichloroacetic acid. In the present case trichloroacetic acid was assessed about 100 h after exposure. At this time the urinary concentration was 120 µmol/L.

Discussion

This welder has been exposed to trichloroethylene as indicated by the urinary concentrations of trichloroacetic acid. He was not taking chloral hydrate, a sleeping drug biotransformed to trichloroacetic acid. The estimated eight-hour time-weighted average concentration of trichloroethylene would have been about 15 ppm and an exposure period of two hours would correspond to an air concentration of 60 ppm. However, it must be pointed out that the urinary level of trichloroacetic acid is only roughly correlated to the air level of trichloroethylene.

The TIG welding of stainless steel generates an intensive ultraviolet light. This radiation might photochemically decompose trichloroethylene to phosgene and dichloroacetyl chloride. The estimated air exposure of 60 ppm of trichloroethylene corresponds to more than 10 ppm of phosgene per minute when TIG welding of stainless steel is performed. Doses of phosgene above 150 ppm × min can produce clinically significant and life-threatening pulmonary edema. The present exposure to phosgene was at least 10 ppm during six to ten minutes corresponding to 60 to 100 ppm × min. However, this estimation of phosgene exposure is approximate and the level might have been higher.

The immediate breathing discomfort experienced by our patient when welding was started might well be explained by exposure to dichloroacetyl chloride, which is a very irritating substance.

Phosgene is not a potent irritant and a latency of 8 to 24 hours has been reported as typical between exposure to this gas and the development of pulmonary edema.

Differential cell counts on BAL fluid recruited from rats exposed to phosgene have shown 60 to 70 percent of polymorphonuclear leukocytes (PMNs) two to three days after exposure. This is in agreement with the lavage findings in our patient although the BAL was performed 12 days after exposure. The PMNs contain large amounts of proteolytic enzymes such as collagenase and elastase, and they produce oxygen radicals capable of damaging the pulmonary tissue. It could be argued that this patient had had bactere mia and that pneumonia also might result in elevated neutrophil counts in lavage fluids. However, pneumonia seems unlikely as cultures on bronchial secretions failed to show any significant growth of bacteria. Furthermore, when the patient was admitted to the hospital the second time a decrease in the WBC had occurred although steroids were administered and he was afebrile.

The relapse of dyspnea ten days after exposure is not the rule among patients exposed to phosgene. It might be explained by a superimposed pulmonary infection seeded from the blood as three blood cultures were positive. However, the recurring dyspnea seen in this patient is very similar to the recurring dyspnea sometimes seen after two to three weeks among patients exposed to nitrogen dioxide.

This second phase of dyspnea is caused by bronchiolitis obliterans. Cross and colleagues exposed rats to 0.5 to 4 ppm of phosgene for 10 to 480 minutes. The earliest detectable lesions they found involved the respiratory bronchiole, which had thickenings and increased cellularity of its wall. Progression of the lesion was marked by lateral extension of the thickening and an increased cellularity to the walls of the evaporating alveoli. Subsequently there was peripheral extension of these changes to the alveolar ducts. Because of the similar pulmonary changes seen after exposure to phosgene and nitrogen dioxide, we suggest that the relapse of dyspnea in the present patient could be a clinical expression of these histologic changes. The role of dichloroacetyl chloride remains unclear in this respect.

In conclusion, three respiratory effects have been observed in this patient after inhalation of photochemically decomposed trichloroethylene. First, there was an almost instantaneous dyspnea probably caused by dichloroacetyl chloride. Second, pulmonary edema probably caused by phosgene developed several hours after exposure. Third, there was recurring dyspnea a week later, possibly caused by pneumonia, pulmonary edema, or bronchiolitis obliterans or by a combination of these conditions.

References


2. Eklund A. Alveolitis in sarcoidosis: studies on markers of disease activity; thesis. Department of Thoracic Medicine, Karolinska Hospital, Stockholm, Sweden, 1986


Welding-Induced Decomposed Trichloroethylene (Sjögren et al)