Chlorinated Solvents, Welding and Pulmonary Edema

To the Editor:

Sjögren et al (Chest 1991;99:237) reported a case of toxic pulmonary edema probably caused by phosgene formed during tungsten inert gas welding of stainless steel in the presence of trichloromethane. In a similar case involving methyl chloroform (MCF), we extensively investigated the phosgene theory but with a frustrating outcome, illustrating the inherent problems of retrospective exposure assessment.

A 62-year-old skilled welder who was a smoker was admitted to hospital on March 1, 1981 with fever (38.6°C) and severe respiratory distress. Initially, myocardial infarction or Legionella pneumonia was suspected, but these diagnoses were later discarded. Instead, toxic pulmonary edema with secondary fibrosis was suspected. The patient was treated in a respirator with positive end-expiratory pressure, but his condition remained unstable and he could not be disconnected from the respirator until the end of April. However, due to recurrent gastrointestinal bleeding (probably due to the massive steroid treatment in combination with metabolic stress) the patient died on May 20. The underlying cause of death based on a medicolegal autopsy was phosgene intoxication.

When this case was revealed to us one week after hospital care, several attempts were made to find the cause. However, we were not able to interview the welder in person.

During three consecutive days ending February 27, the patient had been welding mild (carbon) steel items covered with a drawing oil containing 1.7 percent chlorine. A consumable electrode and a 4:1 mixture of argon/carbon dioxide metal inert gas was used. Lack of time forced him to use an MCF formula stabilized with dioxane for on-site degreasing between welding operations. The welding was performed in a corner of a voluminous (approx. 40,000 cu cm) workshop with fairly simple general and local ventilation. On the night between February 27 and 28 he started to feel “uneasy”. At lunchtime on February 28 he was shivering and his body temperature was 40.5°C. According to his wife, he had recognized symptoms of “intoxication” (possibly alluding to metal fume fever). The situation progressed with a severe dyspnea and some instances of diarrhoea; the next morning he was taken to the hospital.1

During a work site reconstruction one month after the incident, no phosgene could be detected using reagent tubes (Dräger; detection limit 0.05 ppm), not even at high MCF levels (maximum 740 ppm).1 In a separate experiment with the reagent tubes, phosgene could not be detected until the MCF concentration reached 1,000 ppm during welding.

According to Dahlberg et al, 100 ppm exposure to unstabilized MCF during the type of welding relevant to this case would yield approximately 1 ppm phosgene.2 Consequently, a second reconstruction of the incident, assisted by the National Board of Occupational Safety and Health, was undertaken one year later. No phosgene could be found by gas chromatography and electron capture detection even at a maximal MCF level of 250 ppm.3 Ozone or nitrous oxides were not detected either. Thus, we could not verify Dahlberg’s findings, possibly due to insufficient sampling or analysis or other shortcomings.

Although no technical support for a toxic phosgene exposure could be found in this particular case, the overall evidence suggests that this nevertheless was an example of toxic pulmonary edema following welding in the presence of a chlorinated solvent. The combination should always be discouraged.

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REFERENCES


How Long Do Patients with Cor Pulmonale Secondary to Pulmonary Fibrosis Survive?

To the Editor:

The mortality rate in patients with cor pulmonale secondary to chronic obstructive pulmonary disease (COPD) is well documented. The 6-months-1 year survival rate (despite intensive medical treatment) is generally very poor: approximately 30 to 35 percent.14

To our knowledge, there is very little information on the mortality rate of patients with cor pulmonale secondary to pulmonary fibrosis (PF) alone or in association with COPD. Pulmonary fibrosis is a common sequela of repeated and improperly treated lower respiratory bacterial, viral, protozoal, and mycobacterial infections during childhood and early adulthood, especially in developing...