process suggesting radiation pneumonia (Fig 1, B). In spite of the administration of a high dose of adrenocortical hormones, respiratory failure worsened. The patient died of respiratory failure 50 days after the commencement of radiotherapy.

The levels of the serum markers LDH, P3P, CRP, and KL-6 antigen, which indicate the activity of interstitial pneumonia, are shown in Figure 2. The level of CRP was increased gradually to 22.8 mg/dl, but no more elevation was observed afterward. The LDH level was abnormally high just two days before the patient's death. The P3P level was abnormally high during radiotherapy, but the level temporarily decreased and then became elevated again two days before the patient died. In contrast, the KL-6 antigen level rose to 810 U/ml when the patient complained of dysphagia, but decreased into the normal range when radiotherapy was discontinued. However, the KL-6 level was increased again at the time when radiation pneumonia was diagnosed and rose remarkably to 16,640 U/ml two days before the patient's death.

Histologic examination of the autopsy specimens of the lung revealed the presence of hyaline membranes, regenerating type II pneumocytes, and fibrotic changes in the interstitium. Immunohistologic study of the lung sections showed that KL-6 antibody reacted with regenerating type II pneumocytes and macrophages or type II pneumocytes in air spaces but did not react with interstitial components (Fig 1, C), as we reported previously.1

**DISCUSSION**

Radiation pneumonia is one of the factors that restrict the use of radiotherapy against lung cancer. There are only a few methods for diagnosing and assessing the disease activity of radiation pneumonia—chest radiography, 67Ga citrate scintigraphy,4 spirography, and evaluation of serum markers, such as LDH5 and P3P.4

DeRemee6 first reported the use of LDH as an indicator of the presence and disease activity of interstitial pneumonia. The peptide released during the conversion of type III procollagen to type III collagen, P3P, is a potential marker of fibroblast activity. Serum P3P has been reported as a marker of disease activity of interstitial pneumonia,5 particularly of radiation pneumonia.8

The monoclonal antibody that we developed, KL-6, recognizes a carbohydrate antigen expressed on type II pneumocytes.4 Soluble KL-6 antigen is a circulating mucin-like glycoprotein, which can be detected by sandwich assay using the monoclonal antibody KL-6. This antigen has been identified as a potential indicator of the extent of the disease activity of interstitial pneumonia.1 The findings in this patient suggest that serum KL-6 antigen may be a more sensitive marker of radiation pneumonia than LDH or P3P and may reflect the histologic changes of the disease more sensitively than CRP does.

**REFERENCES**

3 DeRemee RA. Serum lactic dehydrogenase activity and diffuse interstitial pneumonitis. JAMA 1966; 204:1193-195

**Pulmonary Toxicity following Exposure to Methylene Chloride and its Combustion Product, Phosgene**

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Chemical paint removers containing methylene chloride are widely used in domestic and industrial settings where exposure to a heat source with conversion to phosgene is possible. We describe a case of noncardiogenic pulmonary edema and subsequent hyperreactive airways following such an exposure. In addition, the various problems that have been associated with exposure to methylene chloride and phosgene are reviewed. (Chest 1992; 101:600-61)

**MC = methylene chloride**

M ethylene chloride, CH$_2$Cl$_2$ (MC), or dichloromethane, is a common ingredient of many paint removers that are employed by individuals renovating older homes or repairing furniture. While warnings to use this substance in well-ventilated areas are common, they are often used in poorly ventilated areas and may be exposed to a heat source to facilitate paint removal. The industrial and domestic use of MC is equally widespread. The authors would like to stress the need to better inform individuals exposed to this substance concerning the risks of MC inhalation, the risk of phosgene production when exposed to a heat source, and the need to ensure adequate ventilation during use.

**CASE REPORT**

A 34-year-old man presented to the Emergency Department with complaints of dyspnea and vague discomfort in the midchest region. Medical history was unremarkable. The patient had been using a nationally advertised brand of paint remover, consisting of MC (>80 percent by weight) as well as small amounts of methanol propanol and ethylene glycol monobutyl ether, while refinishing the woodwork in a 180 × 360-cm foyer without windows. The patient applied the product with a paint brush and then scraped it off with the aid of an electric hot air gun. After working for 8 h and using 504 g of the product, he developed headache, cough, and chest discomfort. These symptoms persisted despite leaving his home, so he came to the Emergency Department for further evaluation. At that time the vital signs were stable, and the chest roentgen-

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The possibility of carcinogenicity in animals has also been raised. There are additional reports suggesting severe pulmonary injury and death due to phosgene (a combustion product of MC) poisoning caused by the use of this chemical near a heat source. Open flames seem worse than electric heating devices with potential lethal phosgene concentrations noted within 5 to 10 minutes in a small enclosure.

In conclusion, MC is a widely used substance both in domestic and industrial settings, and it seems likely that the problems associated with its use are underreported. Of the 26 cases of MC poisoning previously reported and reviewed, 13 occurred in a domestic setting and 11 were in an industrial setting. Three of the acute domestic poisonings were intentional intoxications. Four deaths have been reported.

A thorough patient history should prove helpful in diagnosing MC exposures, and we would recommend the routine addition of carboxyhemoglobin determination when patients are examined for a diagnosis of suspected solvent poisoning. Suggestions for safe use would include the avoidance of prolonged contact to the skin or eyes as well as avoiding the vapors by using the product outdoors or in areas with active exhaust and ample ventilation. Heat sources should be avoided with risk of phosgene production noted. When possible, alternatives to MC should be considered.

Our patient developed headaches as an initial complaint consistent with carbon monoxide intoxication. This resolved over 24 h, but progressive dyspnea and chest discomfort with diffuse alveolar infiltrates and a widened A-a gradient developed. Pulmonary edema has been reported following exposure to MC as well as phosgene, which we believe was generated when the MC was acted on by a hot air gun. Subsequently, hyperreactive Airways were documented and believed to be due to his event. We would like to stress the need for complete medical evaluation following toxic exposures and observation of the patient for 24 to 48 h because of the potential for delayed clinical problems.

**REFERENCES**

5. Hughes JP. Hazardous exposure to some so-called safe solvents. JAMA 1954; 156:234-37

**FIGURE 1.** Chest roentgenogram on hospital admission revealing bilateral alveolar infiltrates.