Surgically Proven Desquamative Interstitial Pneumonia Induced by Waterproofing Spray

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Abstract

We herein describe the first case of desquamative interstitial pneumonia (DIP) induced by waterproofing spray, which was proven by a surgical lung biopsy. A 45-year-old male smoker heavily used a waterproofing spray gas, and presented with chills and fever that was followed by progressive dyspnea. Because steroid pulse therapy did not improve his symptoms, he was referred to our hospital. High-resolution chest CT showed diffuse pan-lobular ground-glass opacities in both lungs. A video-assisted thoracoscopic lung biopsy revealed a DIP pattern. Acute short-time exposure to waterproofing spray can thus be a potential cause of DIP.

Key words: waterproofing spray, desquamative interstitial pneumonia, thermal decomposition product, video-assisted thoracoscopic surgery


Introduction

Waterproofing spray is a readily available product that can be used to cover fabric surfaces for water-repellent treatment at home, and is widely used because of its convenience. Poisoning accidents after inhaling waterproofing spray gas have been reported (1-3). Although it is well known that chest radiography shows ground-glass opacities in such cases, the histopathological pattern of the lung tissue has been unclear. In a few previous reports, transbronchial biopsy revealed alveolitis with eosinophilic or neutrophilic infiltration (1-3). We herein report a case of surgically proven desquamative interstitial pneumonia (DIP) induced by waterproofing spray, in whom inhaling thermal decomposition products may have caused acute lung injury.

Case Report

A 45-year-old Japanese man was admitted to a general hospital because of progressive chest pain and dyspnea. He had a 38 pack-year history of cigarette smoking for 25 years. He worked in the manufacturing of wooden materials. He had no family history of lung disease. His chest X-rays had been free of an abnormal findings at every yearly routine physical examination. Three weeks before admission, he had used a waterproofing spray gas in a closed small entrance space. He smoked immediately after using the spray gas, and subsequently developed chills and a fever of over 39.5°C within two hours. After this event, he stopped smoking. There was no deterioration of his symptoms, but dyspnea on exertion persisted.

Chest radiography showed bilateral ground-glass opacities, and bronchoalveolar lavage was performed on the fourth day after admission. The total cell count in the bronchoalveolar lavage fluid was 17.5×10⁵ cells/mL. The cell fraction was 85.0% macrophages, 4.0% lymphocytes, 8.0% neutrophils and 3.0% eosinophils. The lymphocyte CD4/CD8 ratio was 1.20. No pathogens were detected in the fluid. Steroid pulse therapy (methylprednisolone infusion at 500 mg/day) was administered for two days starting on the fifth day after admission, after which oral prednisolone (0.5 mg/kg/day) was continued for one week. However, the steroid therapy did not improve his symptoms or radiological
A physical examination on his first visit revealed no palpable surface lymph nodes, no clubbing, no skin rash and no fine crackles on chest auscultation. He had a slightly high respiratory rate (20 breaths/min), a normal heart rate (90 beats/min), a blood pressure of 125/78 mmHg and a body temperature of 37.0°C. His oxygen saturation rate was 97% on ambient air. The pulmonary function test showed a forced vital capacity (FVC) of 3.55 L (83.4% of the predicted value) and a reduced diffusing capacity for carbon monoxide (DLco) at 74.9% of the predicted value. Chest X-rays showed ground-glass opacities in the bilateral upper and middle lung fields (Fig. 1). High-resolution chest CT showed diffuse panlobular ground-glass opacities in both lungs without a honeycomb appearance, and no traction bronchiectasis. An arterial blood gas analysis gave a partial pressure of arterial carbon dioxide of 38.9 Torr, a partial pressure of arterial oxygen of 97.1 Torr, a base excess of 1.6 and a pH of 7.430 on room air.

The laboratory test results showed increased serum C-reactive protein (0.48 mg/dL), lactate dehydrogenase (291 IU/L) and IgE (965 IU/mL, reference range: <170 IU/mL) levels. The white blood cell was within the normal limits (7,560 cells/μL, with 69.7% neutrophils, 3.3% eosinophils, 0.7% basophils, 4.5% monocytes and 21.8% lymphocytes). The results of serological tests, including the antinuclear antibodies, other specific autoantibodies and IgG, were all within the normal limits. All microbiological studies were negative.

A video-assisted thoracoscopic lung biopsy was performed one month later because of prolonged radiological abnormalities. A histopathological examination showed numerous pigmented macrophages within the distal airspaces, which is consistent with a DIP pattern (Fig. 2). Alveolar septal thickening and interstitial inflammation were mild, and the formation of granulomas and Masson bodies was not observed. There was no conspicuous air vesicle wall thickening or inflammatory cell infiltration.

During the course of observation without steroid therapy after his first visit to our hospital, the dyspnea gradually subsided. In addition, no further deterioration in his respiratory status was noted after he resumed working. High-resolution chest CT showed an improvement in the bilateral ground-glass opacities after three months, and the pulmonary function test results showed normal lung function, with a FVC of 3.74 L (85.0% of the predicted value) and a DLco of 22.8 L (104.3% of the predicted value).

**Discussion**

To the best of our knowledge, this is the first case report in the English literature to present surgically proven DIP that was induced by waterproofing spray inhalation. DIP is classically characterized by an insidious onset, and is usually associated with cigarette smoking, sometimes with occupational exposure and drug reactions (4, 5). However, no case reports of DIP that was related to waterproofing spray have previously been published.

For our case, the following three differential diagnoses were considered on the basis of the radiological findings: lung injury due to inhalation of waterproofing spray gas, hy-
persensitivity pneumonia or drug-induced pneumonia. The important factors for the diagnosis of the present case were i) he smoked immediately after using the waterproofing spray, ii) no granuloma formation or Masson bodies were observed in the histopathological examination and iii) he had no history of drug use. Although the effects of smoking and occupational exposure were possibilities (6), our diagnosis of lung injury with a DIP pattern induced by a waterproofing spray was based on a history of inhalation of waterproofing spray gas, visible changes on diagnostic imaging and a cytological examination of the bronchoalveolar lavage fluid and histopathological studies of the lung tissue. Because no recurrence was noted after he resumed working, it also reinforced the exclusion of occupational exposure.

Waterproofing spray is made of water-repellent material, solvent and an injection agent. The water-repellent material attaches to the outer surface, the solvent vaporizes and the sprayed gas has a waterproofing effect. In this case, the fluororesin contained a water-repellent material, 1,1,1-trichloroethane as the solvent, and liquefied petroleum gas was the injection agent. Poisoning accidents by inhaling the thermal decomposition products of fluororesin have been associated with symptoms such as fever, chills and sore throat (7). Heating causes 1,1,1-trichloroethane to change into phosgene (COCl₂), and phosgene inhalation can damage alveolar cells and alveolocapillary barriers (8).

The video-assisted thoracoscopic lung biopsy revealed a DIP pattern in the lung in our present case. Although the typical findings of interstitial inflammatory changes, such as alveolar spaces filled with numerous pigmented macrophages, were confirmed, the thickening of the alveolar septa was mild. There was no definite damage to the alveolar or bronchiolar epithelium. The limited findings of transbronchial damage may have been due to the effects of the earlier steroid therapy. Because there were no underlying pathological findings of dust inhalation, we believe that inhaling the waterproofing spray gas was the primary cause of his disease.

In addition to its diagnostic interest, the clinical course for this case was also worth reporting because of the poor response to steroid therapy. Corticosteroids are commonly used to treat such patients (9). However, a failed clinical response to steroid therapy was observed in our patient. This may have been due to the exposure to thermal decomposition products. These thermal decomposition products that are generated immediately after using spray gas and handling fire are known to have a stronger pulmonary toxicity than directly inhaling the gas (7). Although DIP often responds well to steroid therapy (4), the indications for steroid therapy remains undefined for this type of disease.

One of the limitations associated with this case report is the difficulty in evaluating the influence of smoking cessation. However, it is difficult to assume that smoking-related DIP was already present in our case, because the radiological examination did not show emphysema, and the histopathological findings of transbronchial damage were limited. Lung injury induced by inhalation of waterproofing spray gas is believed to occur independently of smoking habits and underlying pulmonary disease. In addition, a previous case report of a couple suffering acute respiratory illness due to waterproofing spray exposure has been documented, and heat degradation products from cigarettes caused the husband to present with more severe symptoms than the wife (10).

In conclusion, we herein presented the first case of DIP that was induced by waterproofing spray, which was confirmed by a surgical lung biopsy. Inhaling the spray gas and thermal decomposition products may have led to the poor improvement in dyspnea and ground-glass opacities.

The authors state that they have no Conflict of Interest (COI).

References