A Case of Acute Respiratory Distress Syndrome Caused by Waterproofing Spray Inhalation

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We report the case of a 62-year-old man who used approximately one can of waterproofing spray in an enclosed room and, then, smoked a cigarette. He developed a fever of 39 °C with respiratory distress and was transported by ambulance to his usual doctor. Since his respiratory state was very severe, he was transferred to our hospital. The patient had a smoking habit of 20 cigarettes per day for approximately 42 years. Chest computed tomography (CT) on arrival showed ground glass opacity (GGO) in the bilateral lungs with emphysematous change. We diagnosed the patient with acute respiratory distress syndrome (ARDS) because of severe hypoxemia. Based on the symptoms' progress, the cause of ARDS was thought to be lung injury due to waterproofing spray inhalation, and treatment was accordingly initiated.

Several reports have described lung injury caused by waterproofing spray inhalation; however, severe cases that progress to ARDS are rare. We believe that the aggravation was caused by smoking after inhaling the waterproofing spray and pre-existing pulmonary lesions, such as emphysema. Education regarding the precautions to be taken when using waterproofing spray is necessary.

Key words: Waterproofing spray, Fluororesin, Pulmonary surfactant, emphysema, Smoking

INTRODUCTION

Fluororesin is implicated in lung injury due to waterproofing spray inhalation. It has been reported that the water-repellant effect of fluororesin competes with that of pulmonary surfactants, causing alveolar collapse [1]. In lung injury caused by waterproofing spray inhalation, alveolitis is pathologically accompanied by macrophage infiltration [2]. Several reports have described lung injury caused by waterproofing spray inhalation; however, severe cases that progress to acute respiratory distress syndrome (ARDS) are rare. In this case, we think that the lung injury was exacerbated by smoking a cigarette immediately after using the waterproofing spray and the existence of emphysema.

CASE REPORT

A 62-year-old man used one can of waterproofing spray in an enclosed room to treat four raincoats belonging to his friends who wanted to wear them to golf in the rain. Then, he immediately smoked a cigarette in the same room. Two hours later, he developed a fever of 39 °C with respiratory distress and was transported by ambulance to his family doctor. He was found to have severe hypoxemia and chest x-ray revealed ground glass opacity (GGO) in the bilateral lung fields.

Three hours after using the waterproofing spray, the patient was transferred to our hospital. Medical history was taken, including autonomic dystonia and a smoking habit of 20 cigarettes per day for approximately 42 years. His vital signs on hospital arrival were as follows: Glasgow Coma Scale at E4V5M6, blood pressure of 138/82 mmHg, heart rate of 116 beats/min, respiratory rate of 26 breaths/min, body temperature of 39 °C, and oxygen saturation of 91% (with oxygen provided at 15 L/min). Physical findings at arrival revealed no heart murmurs, no difference between the left and right breathing sounds, and no clear crackles on auscultation. Furthermore, we found no signs of heart failure, such as pedal edema. Chest x-ray and chest computed tomography (CT) on arrival showed GGO in the bilateral lungs, with emphysematous changes primarily in the pulmonary apex regions and dorsal lung areas, indicating the existence of chronic obstructive lung disease (Figs. 1-a, b and c). Laboratory data on arrival, including arterial blood gas analysis and bronchoalveolar lavage fluid data, are presented in Table. 1.

Based on the imaging results, PaO_2/FiO_2 of 61.7 mmHg, and absence of findings suggestive of cardiogenic pulmonary edema, we diagnosed the patient with ARDS. The cause of ARDS was thought to be lung injury caused by waterproofing spray inhalation based on the symptoms' progress, and treatment was accordingly initiated. After admission, respiratory management with a reservoir facemask was applied, which did not promote oxygenation successfully. Therefore, artificial ventilation was administered using noninvasive positive pressure ventilation (NPPV) from hospital days 2 to 7; respiratory management was performed from hospital day 8 using tracheal intubation

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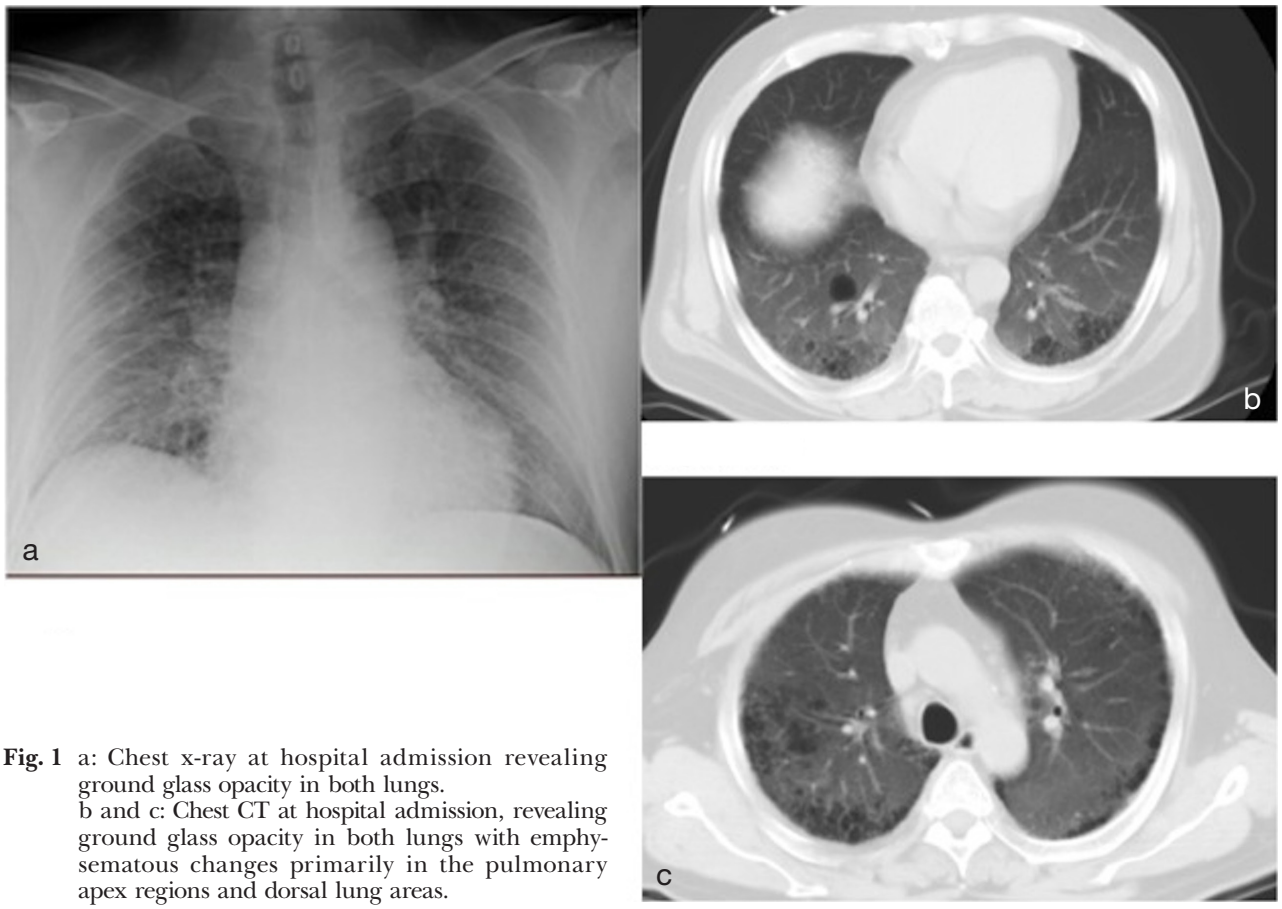

Fig. 1 a: Chest x-ray at hospital admission revealing ground glass opacity in both lungs.
b and c: Chest CT at hospital admission, revealing ground glass opacity in both lungs with emphysematous changes primarily in the pulmonary apex regions and dorsal lung areas.

Table 1 Laboratory data

<Hematology>		<Biochemistry>		<Blood gas analysis(15l RM)	
WBC	21,230/μl	Alb	3.8g/dl	pH	7.43
RBC	486×10⁴/μl	T-Bil	1.0mg/dl	PaO2	61.7 mmHg
Hb	15.2g/dl	AST	32IU/l	PaCO2	34.3 mmHg
Ht	45.2%	ALT	21IU/l	HCO3-	22.5mmol/l
Plt	15.5×10⁴/μl	LDH	332IU/l	BE	-1.2mmol/l
		ALP	194IU/l		
<Serology>		γGTP	48IU/l	<Bronchoalveolar lavage fluid data>	
CRP	2.22mg/dl	Na	142mEq/l	Recovery Rate	60%
		K	4.1mEq/l	Total cell count	0.4×10⁵/ml
		Cl	109mEq/l	Macrophages	10.0%
				Lymphocytes	3.0%
				Neutrophil	82.0%
				Others	3.0%
				CD4+/CD8+	0.61

RM: reservoir mask

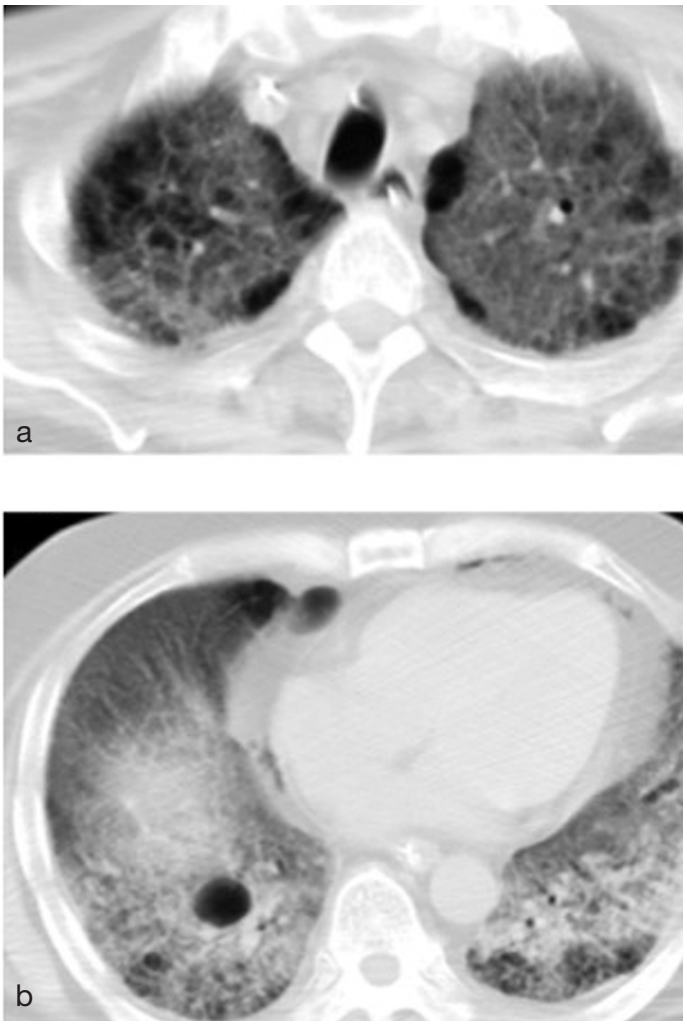

Fig. 2 a and b: On chest CT on day 16, the GGO findings were worse than on chest CT on arrival.

and artificial ventilation. On day 28, tracheotomy was performed, and the treatment modality included respiratory management using artificial ventilation and antibiotic administration due to signs of infection were observed. In addition, respiratory physiotherapy and strict fluid management were prescribed. On days 6–8, steroid pulse therapy with methylprednisolone at 1 g/day was administered, which was switched to 1 mg/kg/day and subsequently tapered. Chest CT on day 16 showed worsening GGO in the bilateral lungs (Figs. 2-a, b).

Because the patient developed hypercapnia with tachypnea caused by reduced ventilator volume due to alveolar collapse, it was difficult to wean him from mechanical ventilation; however, weaning was achieved on day 63. On day 130, the patient was transferred for rehabilitation.

DISCUSSION

Waterproofing spray contains two types of water repellant constituents: fluororesin and silicon resin. It has been demonstrated in a mouse model that fluororesin competes with pulmonary surfactants, causing alveolar collapse, increasing airway resistance, and reducing expiratory flow rate [1]. In cases of fluororesin spray inhalation, macrophages accumulate in the alveoli and pathological changes occur, include thickening of the alveolar wall and interstitial inflammation [2].

In the present case, the type of waterproofing spray used was not known and, thus, its constituents are unclear; however, fluororesin was most likely involved based on the clinical history, because the reduced ventilatory volume suggests alveolar collapse caused by surfactant dysfunction and carbon dioxide retention.

Pulmonary surfactants are found in the alveoli and peripheral bronchi, and their attenuated activity leads to decreased airway patency and increased airway resistance. The main constituents of pulmonary surfactants include phospholipids and proteins (four types: SP-A, -B, -C, and -D). SP-B activity weakens the lung surface tensile strength and prevents alveolar collapse, whereas reduced SP-C activity is said to have a major impact on respiration. Both SP-B and SP-C are hydrophobic and are hidden in phospholipids [3]. Fluorine compounds have an affinity for proteins and phospholipids due to their polar hydrophobicity [4]; therefore, they are thought to exhibit pulmonary surfactant antagonist activity.

The treatment for lung injury caused by waterproofing spray inhalation is essentially based on alleviating symptoms. Table 2 shows the clinical course and laboratory data from past cases. In half of the cases, steroid

Table 2 Clinical data and therapy of previous and present cases caused due to waterproofing spray

	Author	Age/ Sex	P/ F ratio or SPO_2 (RA)	Smoking habit	Smoking after using WPS	Steroid	Days to improve	CT findings emphysematous change
1.	Hayashi et al.[7]	24/F	374	+	+	−	10	−
2.	Kobayashi et al. [8]	28/M	255	+	+	+	7	−
3.	Kobayashi et al. [8]	27/F	452	−	−	−	1	−
4.	Hashimoto et al. [9]	57/M	SPO_2: 97%	+	+	+	7	−
5.	Hashimoto et al. [9]	59/M	SPO_2: 97%	+	+	−	12	−
6.	Sato et al. [10]	51/M	328	+	+	−	15	−
7.	Shimizu et al. [11]	49/M	105	+	+	+	60	+
8.	Kikuchi et al. [12]	30/M	$SPO2_2$ 97%	+	Unknown	−	7	−
9.	Takano et al. [13]	36/F	269	+	Unknown	+	7	−
10.	present case	62/M	63	+	+	+	130	+

RA: room air. WPS: waterproofing spray.

therapy was performed. While cases No. 1, 5, and 6 without steroid therapy took 10 to 15 days to improve, cases No. 2, 4 with steroid therapy took about only 7 days to improve. In case No. 7 and the present case, the patients were in a state of advanced hypoxemia at the onset. It took 60 days in case No. 7 and 130 days in the present case to get better, although steroid therapy was performed in both cases. Therefore, we think that steroid therapy may be effective in mild lung injury cases caused by waterproofing spray inhalation.

All the cases except No. 3 were of patients with a smoking habit. In case No. 3, the patient only had nausea and was not in a state of hypoxemia, and spontaneously improved soon afterwards. All the patients with a smoking habit, except for cases No. 8 and 9, smoked a cigarette immediately after using waterproofing spray. It is unknown whether patients No. 8 and No. 9 smoked immediately after using waterproofing spray because it was not mentioned in those reports apparently. In case No. 7 and the present case, the patients' condition became considerably severe and there were emphysematous findings in the CT images. Considering these factors, smoking a cigarette immediately after using waterproofing spray and the existence of pulmonary lesions, such as emphysema, are thought to exacerbate lung injury caused by waterproofing spray inhalation.

Concerning the first exacerbation factor, it has been reported that fluororesin creates fumes upon heating; inhaling these fumes causes fever and lung injury, a condition known as polymer fume fever [5, 6]. Concerning the second exacerbation factor, we think that the patient already had alveolar wall destruction with peripheral airway obstruction and mucus retention, and this exacerbated the acute injury due to fluororesin inhalation.

We encountered a patient whose injury caused by waterproofing spray inhalation progressed to ARDS. Most lung injuries caused by waterproofing spray inhalation can spontaneously recover; however, smoking immediately after using the spray and the existence of underlying conditions, such as emphysema, can exacerbate the lung injury.

Over the past few years, the incidence of lung injury caused by waterproofing spray inhalation has increased. While waterproofing spray bottles do carry a warning label, the dangers involved are not yet understood fully. Hence, we believe that further action to promote awareness of such risks is warranted.

CONFLICT OF INTEREST

There were no conflicts of interest.

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